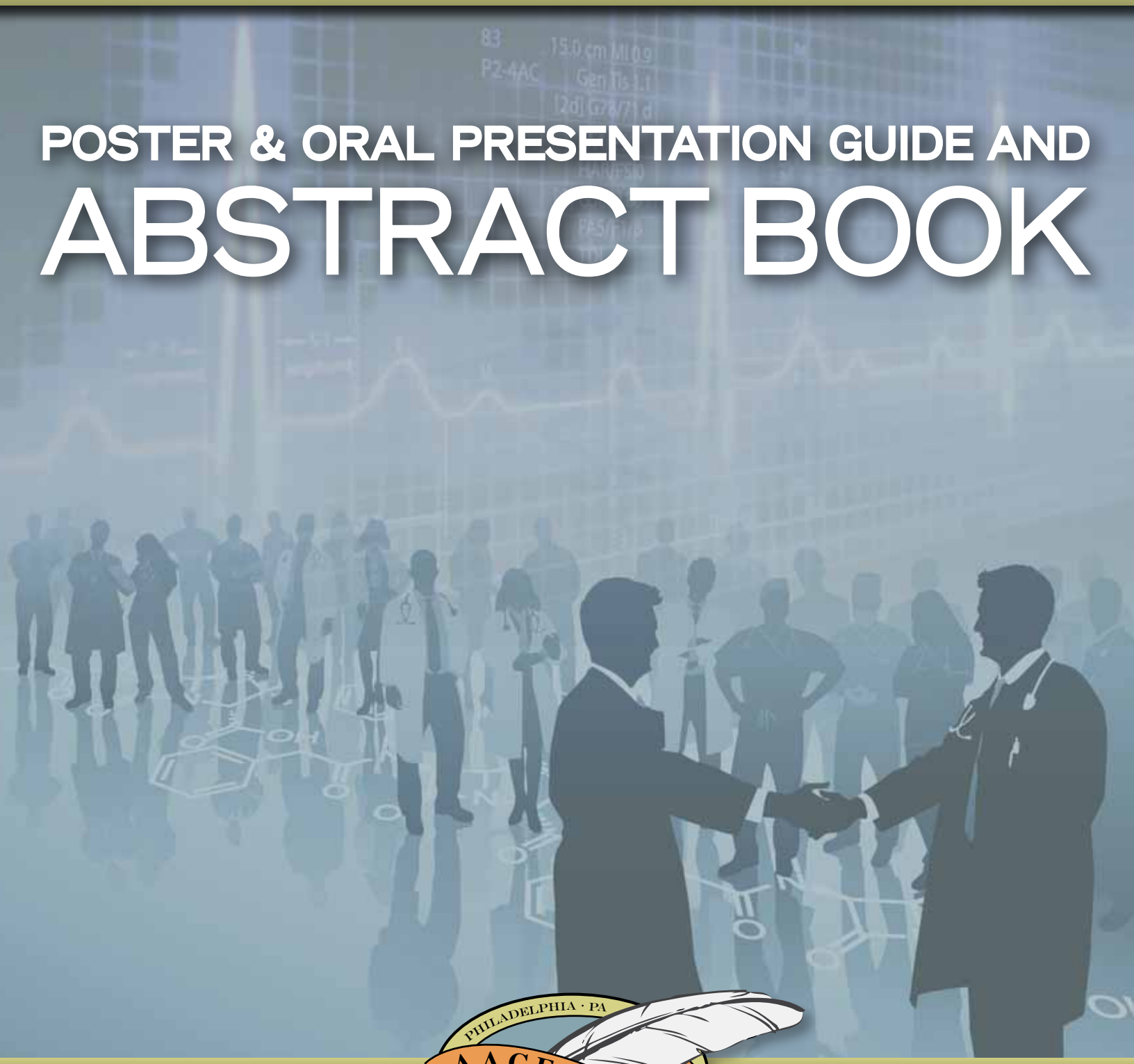
AMERICAN ASSOCIATION OF CLINICAL ENDOCRINOLOGISTS

POSTER & ORAL PRESENTATION GUIDE AND ABSTRACT BOOK



PHILADELPHIA · PA
AACE 2012
MAY 23-27

AACE 21ST ANNUAL SCIENTIFIC AND CLINICAL CONGRESS

MAY 23-27, 2012 • PHILADELPHIA, PA

MARRIOTT PHILADELPHIA DOWNTOWN & THE PENNSYLVANIA CONVENTION CENTER

AMERICAN ASSOCIATION OF CLINICAL ENDOCRINOLOGISTS

ABSTRACTS

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Poster and Oral Presentation Guide & Abstract Book

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TRAVEL GRANT WINNERS

Congratulations to the 2012 AACE Travel Grant Winners for their exceptional research and case presentations.

Domestic Fellow Travel Grant Winners	
Ritu Madan, MBBS	RELATIONSHIP OF EPICARDIAL FAT WITH OBESITY AND HYPERLIPIDEMIA
Owaise Mansuri, MD	GLYCEMIC CONTROL AT INITIATION OF HYPERBARIC OXYGEN THERAPY DOES NOT AFFECT DIABETIC LOWER EXTREMITY WOUND HEALING
Mallika Bhat, MD	DILEMMA IN MANAGEMENT OF WELL-DIFFERENTIATED THYROID CANCER IN PATIENTS ON HEMODIALYSIS
Sandra Mesliniene, MD	ELEVATED APOLIPOPROTEIN A-I GENE EXPRESSION IN VITAMIN D RECEPTOR KNOCKOUT MICE.
Megan Vanderlinde-Wood, MD	AGONISTIC AUTOANTIBODIES AS VASODILATORS IN ORTHOSTATIC HYPOTENSION: A NEW MECHANISM
International Fellow Travel Grant Winners	
Ankit Shrivastav, MD	PREVALENCE OF LATENT AUTOIMMUNE DIABETES IN YOUNG, NON OBESE , ADULT ONSET DIABETES PATIENTS WITH POOR RESPONSE TO ORAL INSULIN SECRETAGOGUES
International Physician Travel Grant Winners	
Sunil Kota, MD	EFFECTS OF GROWTH HORMONE THERAPY ON IGF-1 LEVELS IN GROWTH HORMONE-DEFICIENT INDIAN CHILDREN
Elliot Mitmaker, MD	QUALITATIVE MOLECULAR PROFILING OF CLINICALLY FAVORABLE RESECTED ADRENAL METASTASES
Innocent Okpe, MBBS	SOME METABOLIC PARAMETERS IN SIBLINGS OF TYPE 2 DIABETES PATIENTS IN NORTHERN NIGERIA: A FOCUS ON BLOOD LIPIDS AND BLOOD GLUCOSE
Ayotunde Ale, MD	THE PREVALENCE, CLINICAL AND BIOCHEMICAL CHARACTERISTICS OF BONE DISEASE IN HYPERTHYROID PATIENTS
Ashu Rastogi, MD	EFFICACY OF RAPID ESCALATION OF CABERGOLINE IN COMPARISON TO CONVENTIONAL DOSING IN PROLACTIN SECRETING MACROADENOMA
Domestic Resident Travel Grant Winners	
Haseeb Kazi, MD	TIME FOR CHANGE: DYSLIPIDEMIA MANAGEMENT BY INTERNAL MEDICINE HOUSESTAFF
Brandon Perry, MD	UTILITY OF THE HEMOGLOBIN A1C AS A SCREENING TOOL FOR GESTATIONAL DIABETES MELLITUS
Tulsi Sharma, MBBS	RECURRENT PLEURAL EFFUSION AFTER ZOLEDRONIC ACID IN A PATIENT WITH FIBROUS DYSPLASIA

GENERAL INFORMATION

AACE Exhibit
The AACE booth is the central location for all the information you need! Located in the center of the Exhibit Hall, you will find AACE Membership Services, AACE Impact Graphics™, AACE Endocrine Careers® Expo, AACE Legislative & Regulatory Advocacy, the American College of Endocrinology, AACE Coding Corner & Socioeconomics Resources, information on AACE Chapters, and more all conveniently located together. Stop by to join or renew your membership and learn about the many exciting activities and resources AACE offers. Knowledgeable AACE staff will be on hand to assist you so come with questions or just to say hello!

AACE Store
Leave Philadelphia in style! Stop by the AACE Store in booth 138 and pick up an AACE polo or Thyroid Awareness scarf. Browse the AACE merchandise as you charge your electronics at the nearby Charge-Up Station. Purchase AACE apparel, books, and training materials to help you in your practice.

Embargo Policy
AACE 2012 Abstracts are embargoed until the start of the General Poster Session on Thursday, May 24 at 9:30am.

Exhibit Hall – Exhibit Hall A, Level 200

Hours:
Thursday 9:30am – 2:00pm
Friday 9:30am – 2:15pm & 5:15 – 6:45pm (Wine and Cheese Reception)
Saturday 9:30 – 11:30am

Breaks:
Thursday 9:45 – 10:45am & 12:15 – 1:45pm
Friday 9:45 – 10:45am, 12:15 – 2:00pm & 5:15 – 6:45pm (Wine and Cheese Reception)
Saturday 9:45 – 10:45am

General Poster Session
Visit over 400 posters of top research and case presentations in endocrinology from around the world at the 2012 Poster Session in the Exhibit Hall. Posters will be displayed continuously during Exhibit Hall hours with presentations taking place on Thursday, May 24, for the Young Investigator Poster Competition from 9:45 am-10:45 am and 12:15 pm-1:45 pm; and on Friday, May 25 and Saturday, May 26 from 9:45 am-10:45 am.

Guided Audio Poster Tours 
Take a guided tour of select posters and hear directly from the authors. Expert moderators will lead attendees on 45-minute tours that include question and answer sessions with poster presenters. Daily tours will be held concurrently and will take place in the Exhibit Hall from 10:00 am–10:45 am on their respective days. To sign up for a tour, please visit the Poster Tour desk located at the front of the Exhibit Hall.

Product Theaters
Don't forget to attend the AACE Product Theaters, which are located within the Exhibit Hall to learn about some of the latest products and services available. Please refer to your Program for exact times.

Wireless Lounges and Cyber Cafe
Wireless internet access is available in WiFi hotspots located in the Exhibit Hall. Attendees traveling without a WiFi enabled device can access the internet in our Cyber Café located across from the registration desk on Level 200 of the Pennsylvania Convention Center. Charge-up stations are also available in WiFi hot spot locations.

2013 Annual Meeting
The AACE 22nd Annual Scientific & Clinical Congress will be held May 1-5, 2013 in Phoenix, Arizona at the Phoenix Convention Center and Sheraton Phoenix Downtown.

AACE 2012 POSTER SESSION SCHEDULE

All abstracts selected for poster presentation will be displayed continuously Thursday, Friday, and Saturday. Presenters will be available at their posters for questions and discussion with attendees during the scheduled poster session times.

Thursday, May 24, 2012

AACE Poster Session/Young Investigator Poster Presentations and Judging

9:30am – 2:00pm:	**Exhibit Hall Open**
8:00am – 9:00am:	Poster Set Up
9:45am – 10:45am:	Exhibit Hall Break/Poster Presentations/Young Investigator Poster Judging
10:00am – 10:45am:	Guided Audio Poster Tours
12:15pm – 1:45pm:	Exhibit Hall Break/Poster Presentations/Young Investigator Poster Judging

* Three Fellow-in-Training Poster Presenter Young Investigator Awards: $500.00, $250.00, and $100.00
* One Resident Poster Presenter Young Investigator Award: $250.00

Friday, May 25, 2012

Poster and Oral Presentation Awards Ceremony
General Session

8:00am – 8:15am:	Presentation of Awards

AACE Poster Session

9:30am – 2:30pm:	**Exhibit Hall Open**
9:45am – 10:45am:	Exhibit Hall Break/Poster Presentations
10:00am – 10:45am:	Guided Audio Poster Tours

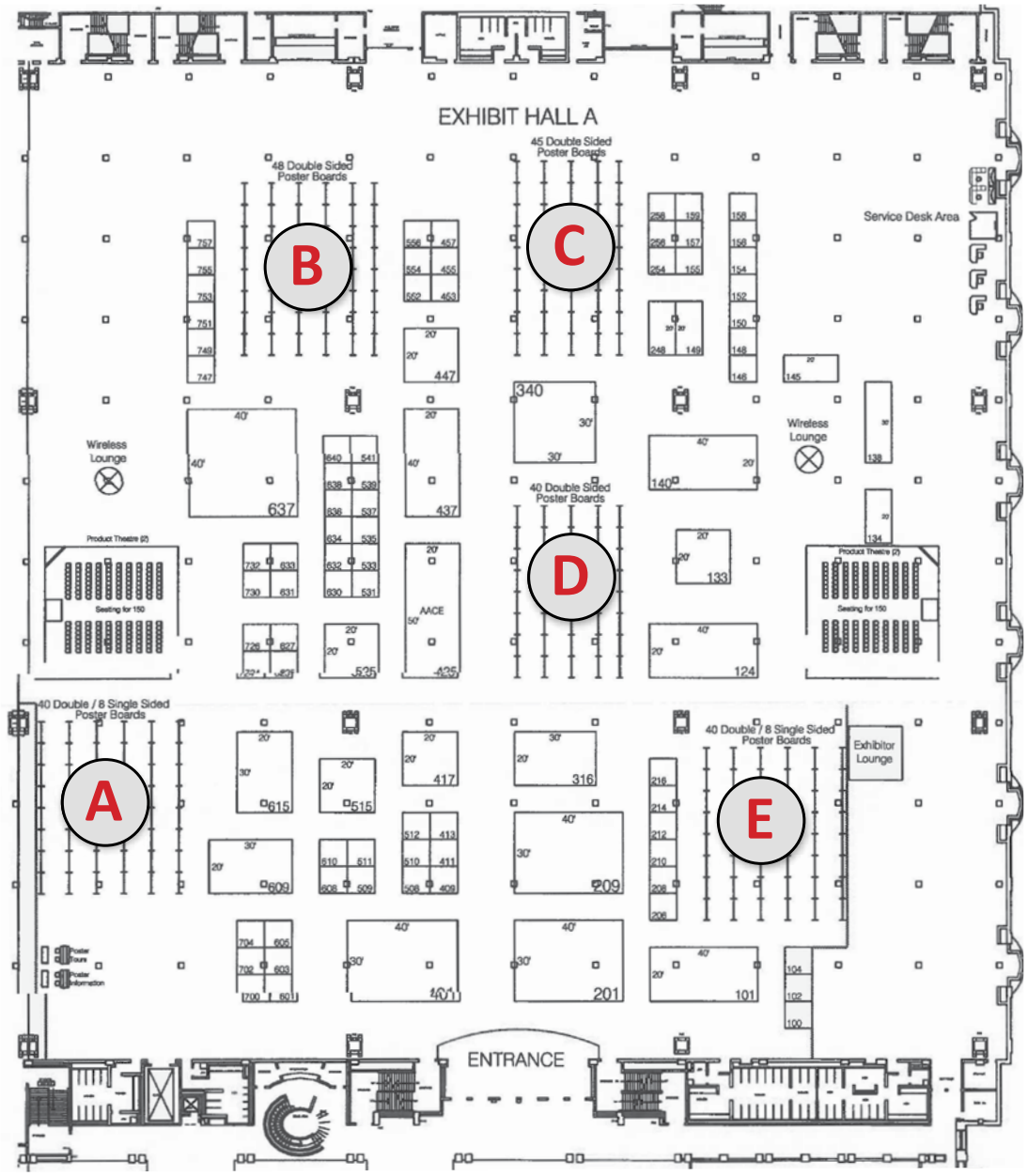
Saturday, May 26, 2012

AACE Poster Session

9:30am – 11:30am	**Exhibit Hall Open**
9:45am – 10:45am:	Exhibit Hall Break/Poster Presentations
10:00am – 10:45am:	Guided Audio Poster Tours

AACE POSTER SESSION MAP KEY

May 24-26, 2012 • Pennsylvania Convention Center • Exhibit Hall A



Poster Section A

Adrenal Disorders: . . . . . 100 – 136
Diabetes Mellitus: . . . . . 200 – 260

Poster Section B

Diabetes Mellitus: . . . . . 261 – 308
Hypoglycemia: . . . . . 400 – 409
Lipid Disorders: . . . . . 500 – 510
Metabolic Bone Disease: . . . 600 – 625

Poster Section C

Metabolic Bone Disease: . . . 626 – 649
Obesity: . . . . . 700 – 703
Other: . . . . . 800 – 841
Pituitary Disorders: . . . . . 900 – 919

Poster Section D

Pituitary Disorders: . . . . . .920 – 944
Reproductive Endocrinology: .1000 - 1016
Thyroid Disease: . . . . . 1100 -1138

Poster Section E

Thyroid Disease: . . . . . .1139 - 1217
Late Breaking: 1300

GUIDED AUDIO POSTER TOURS

 **Guided Audio Poster Tours** 

Take a guided tour of select posters and hear directly from the authors. Expert Moderators will lead attendees on 45-minute tours in question and answer sessions with poster presenters. Pre-register for the tours of your choice to ensure a spot as there is limited availability. Daily tours will be held concurrently and will take place in the exhibit hall from 10:00am – 10:45am on their respective days.

To sign-up for or attend a tour, visit the Poster Tours desk in the Exhibit Hall.

Poster Tours Schedule

	Thursday	**Friday**	**Saturday**
Tour 1	Diabetes	Adrenal	Adrenal
Tour 2	Diabetes	Diabetes	Diabetes
Tour 3	Diabetes	Diabetes	Diabetes
Tour 4	Diabetes	Diabetes/Hypoglycemia	Lipid Disorders
Tour 5	Other	Diabetes/Obesity	Metabolic Bone Disease
Tour 6	Pituitary Disorders	Metabolic Bone Disease	Other
Tour 7	Thyroid Disease	Metabolic Bone Disease	Pituitary
Tour 8	Thyroid Disease	Other	Thyroid Disease
Tour 9	Thyroid Disease	Reproductive Endocrinology	Thyroid Disease
Tour 10	n/a	Thyroid Disease	n/a
Tour 11	n/a	Thyroid Disease	n/a

GUIDED AUDIO POSTER TOURS

Thursday, May 23, 2012
10:00 am – 10:45 am

Tour 1 – Diabetes Mellitus

Poster #	Author	Title
210	Salah El Badawi	SCREENING MODELS FOR UNDETECTED DIABETES AND HIGH RISK FOR DIABETES IN THE UNITED ARAB EMIRATES
211	Gary Pepper	FAILURE OF SHORT TERM IPRO™ CGM TO IMPROVE GLYCOHEMOGLOBIN A1C LEVELS IN CLINICAL PRACTICE
212	Thomas Flood	LONG TERM TYPE 1 DIABETES IN A PRIVATE PRACTICE SETTING
213	Anthonia Ogbera	UNMASKING A LETHAL COMBINATION: DIABETES MELLITUS IN TUBERCULOSIS
214	Rosemarie Lajara	FLEXTOUCH®, A NEW PREFILLED INSULIN PEN: USABILITY STUDY VERSUS VIAL AND SYRINGE INVOLVING PHYSICIANS, NURSES AND PEOPLE WITH DIABETES
215	Joseph Henske	USE OF REAL-TIME CONTINUOUS GLUCOSE MONITORING FOR ENDURANCE ATHLETES WITH TYPE 1 DIABETES
216	Rinkoo Dalan	ETHNIC VARIATION IN THE CORRELATION BETWEEN FASTING SERUM GLUCOSE CONCENTRATION AND GLYCATED HEMOGLOBIN (HBA1C)

Tour 2 – Diabetes Mellitus

245	John Evans	A NOVEL ENDOCRINOLOGY-BASED WELLNESS PROGRAM TO REDUCE MEDICATION EXPENDITURES AND IMPROVE GLYCEMIC OUTCOMES
246	Marshall Tulloch-Reid	THE LIPID ACCUMULATION PRODUCT IS NOT BETTER THAN ANTHROPOMETRIC MEASUREMENTS IN IDENTIFYING INSULIN RESISTANT BLACK YOUTH
247	Ekenechukwu Young	PRE-DIABETES AND CARDIOVASCULAR RISK FACTORS IN URBAN AND RURAL WOMEN IN SOUTH-EASTERN NIGERIA
248	Kiran Singh	ROLE OF ACOUSTIC RADIATION FORCE IMAGING SONOELASTOGRAPHY IN DETECTION OF LIVER FIBROSIS AMONG PATIENTS WITH TYPE 2 DIABETES MELLITUS WITH COEXISTENT NONALCOHOLIC FATTY LIVER DISEASE
249	Andrea Klemes	A PREVENTIVE CARE MODEL IMPROVES MONITORING AND CONTROL OF DIABETES AND CARDIOVASCULAR DISEASE VERSUS TRADITIONAL PRACTICE.
250	Faria Afsana	STUDY OF CLINICAL AND BIOCHEMICAL PARAMETERS OF PULMONARY TUBERCULOSIS IN SUBJECTS WITH TYPE 2 DIABETES MELLITUS

Tour 3 – Diabetes Mellitus

269	Innocent Okpe	SOME METABOLIC PARAMETERS IN SIBLINGS OF TYPE 2 DIABETES PATIENTS IN NORTHERN NIGERIA: A FOCUS ON BLOOD LIPIDS AND BLOOD GLUCOSE
270	Anas Sabir	CLINICAL PROFILE OF TROPICAL DIABETIC HAND SYNDROME IN A TERTIARY HEALTH INSTITUTION IN NORTHERN NIGERIA
271	Miguel Pinto	MAURIAC SYNDROME. A CASE REPORT
272	Arinola Ipadeola	PATTERN OF DYSGLYCAEMIA AMONGST PERSONS WITH MULTI-DRUG RESISTANT TUBERCULOSIS IN IBADAN
273	J. Prendergast	IMPLEMENTING THE GOALS OF THE HITECH ACT IN CLINICAL PRACTICE.
274	Latha Dulipsingh	ASSESSMENT OF PAIN AND TREATMENT SATISFACTION IN PATIENTS WITH PAINFUL DIABETIC PERIPHERAL NEUROPATHY

Thursday, May 23, 2012
10:00 am – 10:45 am

Tour 4 – Diabetes Mellitus

287	Richard Bergenstal	IMPROVED PATIENT-REPORTED OUTCOMES WITH INSULIN DEGLUDEC 200 U/ML (IDEG U200) VERSUS INSULIN GLARGINE IN INSULIN-NAÏVE PEOPLE WITH TYPE 2 DIABETES
288	Jose Jimenez-Montero	RESULTS OF PERCUTANEOUS CORONARY ANGIOPLASTLY WITH STENT PLACEMENT IN DIABETIC PATIENTS WITH CORONARY ARTERY DISEASE
289	Mohsen Eledrisi	PATTERNS OF DIABETES THERAPY AND RATES OF GLUCOSE CONTROL IN SAUDI ARABIA
290	Ekenechukwu Young	TYPE2 DIABETES PRESENTING AS RECURRENT SEIZURES IN A NIGERIAN MALE
291	Ekenechukwu Young	PROFILE OF NEW-ONSET TYPE2 DIABETES MELLITUS IN THE ELDERLY
292	Paul Davidson	A NURSE-DIRECTED COMPUTER PROGRAM, WHICH RE-ADJUSTS S UBCUTANEOUS MULTIPLE DAILY INJECTIONS (MDI) OF INSULIN, LOWERS THE MEAN BG IN HOSPITAL PATIENTS BY 93 (MG/DL)

Tour 5 – Other

806	Donna Lawson	MODULATION OF ADIPONECTIN HOMEOSTASIS AS A POTENTIAL SUPPORT TO COMBINED VITAMIN D AND 1,25 DIHYDROXY VITAMIN D SUPPLEMENTATION IN VITAMIN D DEFICIENT CKD PATIENTS
807	Raymon Grogan	PARATHYROID HORMONE IS PRESENT IN THE TISSUE OF THE NECK OUTSIDE OF THE PARATHYROID GLAND AND SURROUNDING THE THYROID
808	Hammad Hussain	PERSISTENT HYPOCALCEMIA AFTER DENOSUMAB
809	Edward Ruby	BENIGN NON-FAMILIAL PHEOCHROMOCYTOMA PRESENTING WITH SYMPTOMATIC HYPERCALCEMIA FROM AN UNUSUAL ETIOLOGY
810	Luis Ospina	VALUE OF INTRAOPERATIVE PARATHYROID HORMONE MEASUREMENTS IN DETECTING UNSUSPECTED PARATHYROID ADENOMAS
811	Luz Prieto	AUTOIMMUNE POLYGLANDULAR SYNDROME IN A COMMUNITY BASED ENDOCRINE PRACTICE
812	Tuncay Delibasi	THE RELATION BETWEEN PLASMA LEPTIN LEVELS AND CAROTID INTIMA MEDIA THICKNESS WITH SEVERITY OF OBSTRUCTIVE SLEEP APNEA
813	Hemant Thacker	STATUS OF VITAMIN D DEFICIENCY OF PATIENTS IN A TERTIARY CARE CENTRE AND ITS CO- MORBIDITIES

Tour 6 – Pituitary Disorders

902	Edward Condon	OBSERVED MRI RESULTS IN MALES IDENTIFIED AS LOW TESOSTERONE WITH INAPPROPRIATE NORMAL RANGE LEUTINIZING HORMONE LEVELS
903	Monica Gheorghiu	EFFICACY OF PITUITARY RADIOTHERAPY ON GROWTH HORMONE (GH) SECRETION IN PATIENTS WITH ACROMEGALY
904	Satish Babu	A PROSPECTIVE STUDY OF CLINICAL AND RADIOLOGICAL IMPROVEMENT IN THYROTROPH PITUITARY HYPERPLASIA WITH TREATMENT OF PRIMARY HYPOTHYROIDISM
905	Satish Babu	A CASE OF ACTH SECRETING PITUITARY CARCINOMA
906	Satish Babu	THE 'PIT-VIPER' CONNECTION - A CASE REPORT OF HYPOPITUITARISM FOLLOWING VIPER ENVENOMATION
907	Dan Niculescu	NOCTURNAL PROFILES OF SERUM GROWTH HORMONE AND INSULIN IN ACROMEGALIC PATIENTS WITH OR WITHOUT SLEEP APNEA

Thursday, May 23, 2012
10:00 am – 10:45 am

Tour 7 – Thyroid Disease

1106	Amanda Laird	FINE NEEDLE ASPIRATION BIOPSY AND FINAL OPERATIVE HISTOPATHOLOGY: A THREE-YEAR REVIEW OF UTILIZATION OF THE BETHESDA CLASSIFICATION SYSTEM
1107	Angelo Carpi	LACK OF SIGNIFICANT INCREASED RATE OF THYROID CANCER DETECTION IN UNIVERSITY OF PISA HOSPITAL DEPARTMENTS FROM 1980 – 2010.
1108	Daniel Duick	THE IMPACT OF BENIGN GENE EXPRESSION CLASSIFIER TEST RESULTS ON THE PHYSICIAN DECISION-TO-OPERATE IN PATIENTS WITH THYROID NODULES WITH INDETERMINATE FNA CYTOLOGY
1109	Hjalmar Lagast	ESTIMATED PREVALENCE OF HYPOPARATHYROIDISM IN THE UNITED STATES USING A LARGE CLAIMS DATABASE AND DISEASE SEVERITY FROM PRIMARY MARKET RESEARCH
1110	Tarik Elhadd	A HITHERTO UNDESCRIBED CASE OF CEREBELLAR ATAXIA AS THE SOLE PRESENTATION OF THYROTOXICOSIS IN A YOUNG MAN. A PLAUSIBLE ASSOCIATION
1111	Marc Laufgraben	A RARE CASE OF MYXEDEMA COMA CAUSING REVERSIBLE HIGH-DEGREE ATRIOVENTRICULAR BLOCK
1112	Marc Laufgraben	MALIGNANT PLEURAL EFFUSION: A RARE COMPLICATION OF PAPILLARY THYROID CANCER

Tour 8 – Thyroid Disease

1130	Marlon Guerrero	REGIONAL DIFFERENCES IN STAGE OF PRESENTATION AND SURVIVAL OF INDIVIDUALS WITH THYROID CANCER
1131	MD Uddin	DIAGNOSIS OF SUBACUTE THYROIDITIES IS REALLY A PROBLEM!
1132	Michael Demeure	THE FREQUENCY AND BIOLOGIC SIGNIFICANCE OF BRAF V600E MUTATIONS IN PATIENTS WITH PAPILLARY THYROID CANCER.
1133	Miguel Pinto	METHIMAZOLE-INDUCED AGRANULOCYTOSIS. A CASE SERIES
1135	R. Harrell	INTEGRATIVE ENDOCRINE SURGERY PRACTICE IMPROVES EFFICIENCY IN THE DELIVERY OF ENDOCRINE SURGICAL CARE
1136	Reshmi Srinath	ACUTE THYROTOXIC PERIODIC PARALYSIS BEYOND THE ASIAN POPULATION: A DIAGNOSTIC CHALLENGE
1137	Ayotunde Ale	THE PREVALENCE, CLINICAL AND BIOCHEMICAL CHARACTERISTICS OF BONE DISEASE IN HYPERTHYROID PATIENTS

Tour 9 – Thyroid Disease

1163	Rinkoo Dalan	GRAVES DISEASE: EFFECTIVE DOSING OF CARBIMAZOLE (CMZ) AT DIAGNOSIS OF HYPERTHYROIDISM AND OPTIMAL SUBSEQUENT FOLLOW UP INTERVAL POST-CMZ INITIATION
1164	Saleh Aldasouqi	PENDRED SYNDROME: AN INTRIGUING DEVELOPMENTAL ANOMALY COMBINING GOITER, HYPOTHYROIDISM AND CONGENITAL DEAFNESS
1165	Saleh Aldasouqi	A UNIQUE COMBINATION OF HASHIMOTO'S DISEASE, GRAVES'DISEASE AND VITILIGO IN A PATIENT WITH VOGT-KOYANAGI-HARADA SYNDROME
1166	Saleh Aldasouqi	A UNIQUE CASE OF UNFOLDING GRAVES' DISEASE PROVIDES A SIMPLE MODEL FOR A BETTER UNDERSTANDING OF THE PATHOGENESIS OF THYROID AUTOIMMUNITY
1167	Saleh Aldasouqi	A PROPOSAL FOR ROUTINE ULTRASOUND SCREENING OF ALL PATIENTS WITH GRAVES' DISEASE IN VIEW OF INCREASED RISK AND AGGRESSIVENESS OF ASSOCIATED PAPILLARY THYROID CANCER: A CASE SERIES AND REVIEW OF THE LITERATURE
1168	Sandra Weber	PROGNOSTIC IMPLICATIONS OF BRAF MUTATION AND PAPILLARY THYROID CANCER
1169	Simona Fica	HUMERUS METASTASIS AS THE FIRST CLINICAL SIGN OF FOLLICULAR THYROID CARCINOMA

Friday, May 24, 2012
10:00 am – 10:45 am

Tour 1 – Adrenal Disorders

100	Sunil Kota	PHEOCHROMOCYTOMA COEXISTING WITH VASCULAR LESIONS
101	Issac Sachmechi	CASE OF DISSEMINATED HISTOPLASMOSIS IN IMMUNOCOMPITENT PATIENT WHO DEVELOPED ADRENAL INSUFFICIENCY WHEN TREATED WITH ITRACONAZOLE
102	Chee Kian Chew	ADRENOCORTICOTROPHIC INDEPENDENT CUSHING'S SYNDROME: BEWARE OF MALIGNANCY IN EVERY LARGE ADRENAL TUMOR
103	Sheryl Tugna	PHEOCHROMOCYTOMA PRESENTING WITH HEMOPTYSIS: CASE REPORT AND REVIEW OF OTHER UNUSUAL MANIFESTATIONS OF PHEOCHROMOCYTOMA
104	Elliot Mitmaker	QUALITATIVE MOLECULAR PROFILING OF CLINICALLY FAVORABLE RESECTED ADRENAL METASTASES
105	Donna Lawson	SUPPRESSION AND RECOVERY OF HPA FUNCTION AFTER A SINGLE EPIDURAL GLUCOCORTICOID INJECTION: DECONVOLUTION ESTIMATION OF ACTH AND CORTISOL SECRETORY DYNAMICS
106	Mini Mathew	PF4 ANTIBODY POSITIVITY IN THE SETTING OF BILATERAL ADRENAL HEMORRHAGE
107	Radha Devi	EAT MORE YET FEEL WEAK

Tour 2 – Diabetes Mellitus

218	Adam Maghrabi	HEMIBALLISM-HEMICHOREA: A RARE MANIFESTATION OF DIABETIC KETOACIDOSIS
219	Oluwatosin Kayode	DIABETIC FOOT CARE: AWARENESS AND PRACTICE AMONG PATIENTS ATTENDING A TERTIARY HOSPITAL IN LAGOS, NIGERIA
220	Allison Galloway	U-500 REGULAR INSULIN USE IN TYPE 2 DIABETIC PATIENTS: A RETROSPECTIVE STUDY
221	Rifka Schulman	ACHIEVING TIGHT GLYCEMIC CONTROL SAFELY IN PATIENTS WITH CHRONIC CRITICAL ILLNESS (CCI) USING A SUBCUTANEOUS INSULIN PROTOCOL
222	Remya Tharackal Ravindran	PATTERN OF CRITICAL CORONARY ARTERY STENOSIS IN DIABETIC VERSUS NON-DIABETIC PATIENTS
223	Sunil Kota	CAROTID INTIMA MEDIA THICKNESS IN TYPE-2 DIABETES MELLITUS WITH ISCHEMIC STROKE
224	Sunil Kota	ETIOPATHOGENETIC ASSOCIATION OF COEXISTING DISEASES IN TYPE 1 DIABETES MELLITUS
225	Sunil Kota	ILEAL INTERPOSITION WITH SLEEVE GASTRECTOMY/ DIVERTED SLEEVE GASTRECTOMY FOR TREATMENT OF TYPE 2 DIABETES

Tour 3 – Diabetes Mellitus

253	Rabia Rehman	TELE-ENDOCRINOLOGY: BRIDGING THE GAP IN ENDOCRINE CARE VIA TELE-MEDICINE.
254	Vasudev Govardhan Magaji	COMPARISON OF INSULIN INFUSION PROTOCOLS TARGETING BLOOD GLUCOSE(BG) 110-140MG/DL IN PATIENTS AFTER CARDIAC SURGERY
255	Amber Taylor	A RETROSPECTIVE STUDY ON METFORMIN USE AND NEUROPATHIC PAIN
256	Prajesh Joshi	COEXISTING AUTOIMMUNE DISEASE IN ADULTS WITH TYPE 1 DIABETES MELLITUS (T1DM).
257	Anteneh Zenebe	THE EFFECT OF CLINICAL PHARMACIST FOLLOW-UP AND DIABETES EDUCATION ALONG WITH MEDICATION IN ACHIEVING A1C GOAL: THE HOWARD UNIVERSITY HOSPITAL DIABETES TREATMENT CENTER EXPERIENCE
258	Brandon Perry	UTILITY OF THE HEMOGLOBIN A1C AS A SCREENING TOOL FOR GESTATIONAL DIABETES MELLITUS
259	Ayotunde Ale	THE PREVALENCE OF SLEEP DISORDER,RELATION WITH OTHER CARDIOVASCULAR RISKS AND IMPACT ON GLYCAEMIC CONTROL IN TYPE 2 DIABETES
260	Rajib Bhattacharya	COMMUNITY ACQUIRED PNEUMONIA IN ELDERLY DIABETICS (CAPED)

Friday, May 24, 2012
10:00 am – 10:45 am

Tour 4 – Diabetes/Hypoglycemia

302	Michael Tsoukas	THE IMPACT OF A BASAL-BOLUS INSULIN REGIMEN ON THE MANAGEMENT OF HYPERGLYCEMIA IN A HOSPITALIZED POPULATION
303	Cherie Lisa Vaz	NEW DIABETIC EMERGENCY: ACUTE RHABDOMYOLYSIS COMPLICATING HYPERGLYCEMIC HYPEROSMOLAR COMA: SUCCESSFUL MANAGEMENT OF A CASE AND INSIGHT INTO PATHOGENESIS.
304	Miguel Pinto	TYPE 2 DIABETES IN CHILDREN. A CASE SERIES FROM LIMA, PERU
403	Saleh Aldasouqi	HYPOGLYCEMIA WHILE FASTING FOR MORNING BLOOD TESTS: A FOLLOW UP OF THE CAPE GIRARDEAU HYPOGLYCEMIA EN ROUTE PREVENTION PROGRAM
404	Carla Romero	TRANSIENT RECURRENT HEMIPARESIS AS A PRESENTATION OF HYPOGLYCEMIA IN A PATIENT WITH INSULINOMA
405	Miguel Pinto	DOEGE-POTTER SYNDROME. A CASE REPORT

Tour 5 – Diabetes/Obesity

305	Sabyasachi Sen	VASCULAR REACTIVITY IMPROVES IN PRE-DIABETES PATIENTS, POST AEROBIC EXERCISE
306	Natia Potter	AMBULATORY BLOOD PRESSURE MONITORING (ABPM) AND VASCULAR STIFFNESS IN LEAN, OBESE, AND DIABETIC MINORITY YOUTH
307	Jaime Almandoz	A MULTIDISCIPLINARY LIFESTYLE INTERVENTION PRODUCES MARKED WEIGHT LOSS IN OBESE PATIENTS WITH TYPE 2 DIABETES MELLITUS
308	Jose Jimenez-Montero	MORTALITY RATES DUE TO DIABETES AND DIABETIC COMPLICATIONS IN COSTA RICA 2005 – 2010.
703	Pratima Sood	VIPOMA IN A PATIENT POST-GASTRIC BYPASS SURGERY

Tour 6 – Metabolic Bone Disease

602	Aashish Shah	TUMOR-INDUCED OSTEOMALACIA (TIO) CAUSED BY PRIMARY FIBROBLAST GROWTH FACTOR-23 (FGF-23) SECRETING NEOPLASM IN AXIAL SKELETON
603	Buvana Manickam	LOW SERUM VITAMIN D IS ASSOCIATED WITH METABOLIC SYNDROME IN AFRICAN AMERICAN AND CAUCASIAN AMERICAN MALE VETERANS.
604	Aliya Khan	ATYPICAL FEMORAL FRACTURES: RADIOGRAPHIC AND HISTOMORPHOMETRIC FEATURES IN 9 PATIENTS
605	Deepika Nallala	CALCIMIMETIC THERAPY FOR SEVERE SECONDARY HYPERPARATHYROIDISM REFRACTORY TO VITAMIN D REPLETION AFTER DUODENAL SWITCH SURGERY
606	Evelina Svrdlan	TUMOR-INDUCED OSTEOMALACIA ASSOCIATED WITH UNDIFFERENTIATED CARCINOMA OF THE PANCREAS WITH OSTEOCLAST-LIKE GIANT CELLS
607	Andrew Brackbill	A CASE OF EUGONADAL OSTEOPOROSIS IN A MALE WITH HEREDITARY HEMOCHROMATOSIS
608	Erica Kretchman	PARATHYROIDITIS INDUCED BY THERAPEUTIC RADIOACTIVE IODINE
609	Fadi Siyam	A CASE OF CARCINOID SYNDROME ASSOCIATED WITH HYPERCALCEMIA.

Friday, May 24, 2012
10:00 am – 10:45 am

Tour 7 – Metabolic Bone Disease

626	Michael Gonzales	RECOMBINANT HUMAN PARATHYROID HORMONE THERAP IN AN OLDER PATIENT WITH A GAIN OF FUNCTION MUTATION OF THE CALCIUM SENSING RECEPTOR-A CASE REPORT
627	Matheni Sathananthan	CLINICAL FEATURES OF SAPHO SYNDROME
628	Mini Mathew	PLACENTAL CALCIFICATION: A COMPLICATION OF HYPERPARATHYROIDISM IN PREGNANCY
629	Tulsi Sharma	RECURRENT PLEURAL EFFUSION AFTER ZOLEDRONIC ACID IN A PATIENT WITH FIBROUS DYSPLASIA
630	Mona Fouda	CELIAC DISEASE AND METABOLIC OSTEOPATHY: A UNIVERSITY HOSPITAL EXPERIENCE
631	Patchaya Boonchaya-anant	BONE MINERAL DENSITY IN PATIENTS WITH NONALCOHOLIC STEATOHEPATITIS
632	George Tsoukas	REAL-LIFE EFFECTIVENESS OF ZOLENDRONIC ACID IN PATIENTS WITH OSTEOPOROSIS: 5-YEAR EXPERIENCE
633	Ila Khanna	SYSTEMIC MASTOCYTOSIS- A RARE CAUSE OF OSTEOPENIA IN AN ADULT MALE
634	Mahshid Mohseni	RADIATION ASSOCIATED PELVIC FRACTURES: REPORT OF 4 CASES

Tour 8 – Other

814	Corina Galesanu	MULTIPLE ENDOCRINE NEOPLASIA TYPE 2A. STUDY OF A FAMILY OF THREE GENERATIONS WITH PEDIGREE ANALYSIS OF THE RET PROTO-ONCOGENE
815	Daniel Cosgrove	DISPROPORTIONATE RISE IN SERUM DHT LEVELS FOLLOWING TRANSDERMAL TESTOSTERONE TREATMENT
816	Yousef Altowaireb	RESOLVING DEPRESSION AND NECROLYTIC MIGRATORY ERYTHEMA 2 WEEKS FOLLOWING GLUCAGONOMA RESECTION
817	Cindy Huang	COMPARISON OF OBESE AND LEAN PRIMARY HYPERPARATHYROID PATIENTS
818	Pooja Aggarwal	HYPERCALCEMIA IN A PATIENT WITH MEN 1 SYNDROME
819	Swati Singh	RELATION OF SERUM CALCIUM LEVEL WITH METABOLIC RISK FACTORS AND CORONARY ARTERY DISEASE IN AFRICAN AMERICAN
820	Nidhi Bansal	MYSTERY TRAIL OF UNEXPLAINED HYPERCALCEMIA ENDS IN A FOOD SUPPLEMENT.
821	Nidhi Bansal	MYSTERY TRAIL OF RAISED SERUM HCG ENDS IN A LUNG MASS

Tour 9 – Reproductive Endocrinology

1000	Jennifer Cheng	AN UNUSAL CASE OF PURE XY GONADAL DYSGENESIS
1001	Yousef Altowaireb	VIRILIZATION DURING AN IVF PREGNANCY AND DELIVERY OF FEMALE TWINS WITH AMBIGOUS GENITALIA
1002	Maria Karaflou	EFFECT OF TIBOLONE AND RALOXIFENE ON SERUM MARKERS OF APOPTOSIS IN HEALTHY POSTMENOPAUSAL WOMEN
1003	Zdravko Kamenov	PATIENTS' PERCEPTION FOR CARDIOVASCULAR RISK FACTORS IN PCOS AND/OR OBESITY
1004	Sedigheh Soheilykhah	THE EFFECT OF DIFFERENT DOSES OF VITAMIN D SUPPLEMENTATION ON INSULIN RESISTANCE DURING PREGNANCY
1005	Tuncay Delibasi	HEART TYPE FATTY ACID BINDING PROTEIN LEVELS IN POLYCYSTIC OVARY SYNDROME PATIENTS
1006	Vijayaratna Chockalingam	THE USE OF OVARIAN VEIN SAMPLING TO FIND A RARE OVARIAN TUMOR

Friday, May 24, 2012
10:00 am – 10:45 am

Tour 10 – Thyroid Disease

1114	Jagriti Upadhyay	THYROTOXICOSIS FOLLOWING GAMMA KNIFE PARATHYROIDECTOMY
1115	Bhavika Bhan	INTRAMUSCULAR LEVOTHYROXINE: AN EFFECTIVE ALTERNATIVE FOR TWO PATIENTS WITH PROFOUND LEVOTHYROXINE MALABSOPRTION
1116	Brian O'Neill	PAPILLARY CARCINOMA OF AN OVARIAN TERATOMA DISCOVERED AFTER RADIOIODINE TREATMENT FOR PAPILLARY THYROID CARCINOMA
1117	Candice Rose	HYPOTHYROID MYOPATHY
1118	Celeste Cheryll Quianzon	A SURVEY ON INITIAL MANAGEMENT OF THYROID NODULES AMONG PRIMARY CARE PROVIDERS AND INTERNAL MEDICINE RESIDENTS
1119	David Cohen	CLINICAL RESPONSE TO INTRAVENOUS L-THYROXINE IN MYXEDEMA COMA
1120	Irina Ciubotaru	A UNIQUE CASE OF AUTOIMMUNE THYROID DISEASE IN A PREGNANT PATIENT
1121	Rod Marianne Arceo-Mendoza	SIGNET RING CELL FOLLICULAR ADENOMA OF THE THYROID

Tour 11 – Thyroid Disease

1139	Diep Nguyen	EXACERBATION OF GRAVES' DISEASE WITH THE ACUTE ADMINISTRATION AND INCREASED DOSE OF EPOPROSTENOL FOR PULMONARY HYPERTENSION: A CASE REPORT AND REVIEW OF THE LITERATURE
1140	Eleni Armeni	ARTERIAL STIFFNESS IS ASSOCIATED WITH HIGH-NORMAL TSH LEVELS IN HEALTHY POSTMENOPAUSAL WOMEN
1141	Erjola Balliu	ZENKER DIVERTICULUM PRESENTING AS THYROID NODULE: 2 CASE REPORTS
1142	Grace Chang	A RARE CASE OF EARLY ONSET THYROTOXIC PERIODIC PARALYSIS
1143	Grace Sun	HYPOTHYROIDISM AS A CAUSE OF HYPONATREMIA: FACT OR FICTION?
1144	Ivica Boban	FORTY-SEVEN YEARS OF FOLLOW-UP OF A PATIENT WITH TALL CELL VARIANT PAPILLARY THYROID CANCER
1145	Sayed Aamir	SUPPURATIVE THYROIDITIS DUE TO NOCARDIA ASTEROIDES IN AN IMMUNUSUPPRESSED PATIENT
1146	Ibrahim Ibrahim	RARE LIFE THREATENING COMPLICATIONS IN A COMMONLY USED DRUG

Saturday, May 25, 2012
10:00 am – 10:45 am

Tour 1 – Adrenal Disorders

108	Duarte Pignatelli	FAMILIAL CUSHING'S SYNDROME DUE TO A BILATERAL ACTH-INDEPENDENT MACRONODULAR ADRENAL HYPERPLASIA (AIMAH) RELATED TO THE ECTOPIC EXPRESSION OF BETA ADRENERGIC RECEPTORS
109	Elliot Mitmaker	THE PREVALENCE OF POSTOPERATIVE HYPOGLYCEMIA FOLLOWING ADRENALECTOMY FOR PHEOCHROMOCYTOMA: A RARE AND OFTEN FORGOTTEN COMPLICATION
110	Kwame Ntim	AN ELUSIVE NEUROENDOCRINE TUMOR; A CHALLENGING DIAGNOSIS IN A PATIENT WITH CUSHING'S SYNDROME
111	Zulfiya Shafigullina	HYPERCORTISOLISM IN YOUNG PATIENT WITH OBESITY
112	Shilpa Swamy	ADRENOMYELONEUROPATHY AND PRIMARY ADRENAL INSUFFICIENCY
113	Christopher Mulla	PHEOCHROMOCYTOMA PRESENTING AS ACUTE DECOMPENSATED HEART FAILURE

Tour 2 – Diabetes Mellitus

226	Saleh Aldasouqi	MORE PATIENTS WITH DIABETES RESORT TO MEDICAL ALERT TATTOOS AS AN ALTERNATIVE TO METAL MEDICAL ALERTS: A CALL UPON HEALTH ORGANIZATIONS TO DEVELOP REGULATIONS AND PRACTICE GUIDELINES FOR MEDICAL TATTOOING
227	Rafael Gonzalez-Rosario	INPATIENT MANAGEMENT OF DIABETES MELLITUS AMONG NONCRITICALLY ILL GENERAL MEDICINE PATIENTS AT THE PUERTO RICO UNIVERSITY HOSPITAL.
228	Karla Arce	INPATIENT BLOOD GLUCOSE CONTROL BEFORE AND AFTER IMPLEMENTATION OF AN INSULIN ORDER SET TO THE ELECTRONIC MEDICAL RECORD
229	Karla Arce	PREVALANCE OF UNDIAGNOSED DIABETES IN HOSPITALIZED PATIENTS.
230	Ofem Enang	PREVALENCE OF RISK FACTORS FOR GLUCOSE INTOLERANCE IN SOUTH EAST NIGERIA
231	Soni Srivastav	PREVALENCE OF ANTI-GAD ANTIBODIES IN PATIENTS WITH PANCREATIC DIABETES MELLITUS
232	M. Figaro	THE IMPACT OF DIABETES ON LENGTH OF STAY AND HOSPITAL COSTS AFTER ELECTIVE SURGICAL PROCEDURES

Tour 3 – Diabetes Mellitus

261	Jeremy Anthony	ADULT DIABETIC KETOACIDOSIS ASSOCIATED CEREBRAL EDEMA
262	Ignatius Ezeani	CASE SERIES ON TROPICAL DIABETIC HAND SYNDROME (TDHS)
263	Ahmet Ergin	EFFECTS OF THE IMPLEMENTATION OF A SUBCUTANEOUS GLARGINE PROTOCOL ON GLUCOSE CONTROL IN POSTOPERATIVE CARDIOTHORACIC PATIENTS
264	Vijayaratna Chockalingam	DIABETIC MYONECROSIS
265	Yin Oo	INSULIN RESISTANCE: CASE REPORT AND REVIEW ON MECHANISM, CURRENT CONCEPT AND MANAGEMENT
266	Sandra Barrow	INSULIN REQUIREMENTS IN DIABETIC PATIENTS WITH HEART FAILURE BEFORE AND AFTER LVAD

Saturday, May 25, 2012
10:00 am – 10:45 am

Tour 4 – Lipid Disorders

505	Daniel Okorodudu	PROFOUND FASTING CHYLOMICRONEMIA DURING PREGNANCY: COMPLEXITIES IN MANAGEMENT
506	Patricia Sareh	A CASE OF PEG-L-ASPARAGINASE-INDUCED HYPERTRIGLYCERIDEMIA TREATED WITH INTRAVENOUS HEPARIN BOLUSES
507	Kamran Rasul	PREVALENCE OF LOW HDL IN MARKEDLY OBESE PATIENTS
508	Nidhi Bansal	AGGRESSIVE CHOLESTEROL LOWERING WITH HIGH DOSE STATIN THERAPY IN GERIATRIC POPULATION: HOW SAFE IS THIS PRACTICE?
509	Miguel Pinto	RELATIONSHIP AMONG LIPID PROFILE, CALCIUM METABOLISM, AND OTHER CARDIOVASCULAR RISK FACTORS WITH CAROTID-WALL INTIMA-MEDIA THICKNESS IN PATIENTS WITH END-STAGE RENAL DISEASE IN HEMODIALYSIS
510	Anjana Harnoor	UNDIAGNOSED PANHYPOPITUITARISM PRESENTING AS SEVERE DYSLIPIDEMIA IN AN ADOLESCENT

Tour 5 – Metabolic Bone Disease

610	Petpring Prajuabpansri	PRIMARY HYPERPARATHYROIDISM MASQUERADING AS AMYOTROPHIC LATERAL SCLEROSIS
611	Ramesh Gadam	FRAX AS A PREDICTOR FOR OSTEOPOROSIS
612	Taral Shah	PARATHYROID CARCINOMA PRESENTING AS A GIANT CELL TUMOR
613	Subramanian Kannan	CHANGING SESTAMIBI AGENTS AND CONVERSION OF PARATHYROID SCAN OVER TIME IN DUAL AGENT PROTOCOL - ONE OF FEW CASE REPORTS IN LITERATURE
614	Sunil Wimalawansa	CHRONIC DISABILITIES MARKEDLY INCREASE BONE LOSS AND FRACTURES
615	Simona Ioja	DENOSUMAB INDUCED SEVERE HYPOCALCEMIA IN A CANCER PATIENT: MANAGEMENT OPTIONS
616	Shamsa Ali	A NOVEL ROBOTIC TECHNIQUE OF TRANANSAXILLARY GASLESS PARATHYROIDECTOMY FOR THE SURGICAL MANAGEMENT OF PRIMARY HYPERPARATHYROIDISM (PHPT) DUE TO PARATHYROID ADENOMA

Tour 6 – Other

800	Nikhil Gupta	COST CONSCIOUSNESS IN ENDOCRINOLOGY – LEVEL OF AWARENESS AMONG HEALTH CARE PROVIDERS AND NEED FOR EDUCATION
801	Matilda Malm	THE RACE TO STOP THE FALLS
802	Sarvpreet Ahluwalia	SEVERE HYPOCALCEMIA RELATED TO CHEMOTHERAPY IN BURKITT'S LYMPHOMA
803	Shveta Gandhi	A MIND'S DISGUISE: THYROTROPH HYPERPLASIA MIMICKING PITUITARY ADENOMA
804	Eleni Armeni	SUBCLINICAL ATHEROSCLEROSIS AND ARTERIAL STIFFNESS ARE ASSOCIATED WITH ENDOGENOUS TESTOSTERONE IN HEALTHY RECENTLY MENOPAUSAL WOMEN
805	Eleni Armeni	SUBCLINICAL ATHEROSCLEROSIS IS ASSOCIATED WITH MENOPAUSAL HOT FLUSHES IN HEALTHY YOUNG POSTMENOPAUSAL WOMEN

Saturday, May 25, 2012
10:00 am – 10:45 am

Tour 7 – Pituitary Disorders

920	Ranee Angeli Lleva	DELAYED EMERGENCE OF GROWTH HORMONE (GH) SECRETION FROM A LARGE, CYSTIC MACROPROLACTINOMA: A RARE TRANSFORMATION IN PITUITARY DISEASE
921	Uzma Shafqat	COMPLEX MANAGEMENT OF DDAVP RESISTANT DIABETES INSIPIDUS AFTER REMOVAL OF A THIRD VENTRICULAR TUMOR: IMPORTANCE OF CORRECTING ANTERIOR HYPOPITUITARISM
922	Archana Jarathi	IPILIMUMAB THERAPY RELATED ENDOCRINOPATHIES
923	Brian O'Neill	SQUAMOUS CELL CARCINOMA OF THE PITUITARY MIMICKING RATHKE'S CLEFT CYST
924	Mahshid Mohseni	RESISTANT PROLACTINOMA: AN UNUSUAL CASE OF PROLACTINOMA UNRESPONSIVE TO MEDICAL TREATMENT WITH HIGH DOSE DOPAMINE AGONISTS
925	Sarvpreet Ahluwalia	HYPOPITUITARISM SECONDARY TO ASPERGILLUS SELLAR ABSCESS
926	Sunil Kota	EFFECTS OF GROWTH HORMONE THERAPY ON IGF-1 LEVELS IN GROWTH HORMONE-DEFICIENT INDIAN CHILDREN
927	Ashu Rastogi	EFFICACY OF RAPID ESCALATION OF CABERGOLINE IN COMPARISON TO CONVENTIONAL DOSING IN PROLACTIN SECRETING MACROADENOMA

Tour 8 – Thyroid Disease

1122	Kavya Chitra Mekala	A CASE OF HYPERCALCEMIA IN METASTATIC FOLLICULAR THYROID CANCER
1123	Kristine Nicolas	HYPOTHYROIDISM-INDUCED RHABDOMYOLYSIS
1124	Kristine Parker	A CASE OF APATHETIC THYROID STORM WITH RESULTANT HYPERTHYROIDISM INDUCED HYPERCALCEMIA
1125	Lilah Morris	PREOPERATIVE ULTRASONOGRAPHIC THYROIDITIS HELPS PREDICT THE NEED FOR THYROID HORMONE REPLACEMENT (THR) AFTER THYROID LOBECTOMY
1126	Liliana Garcia	A NEW KINDRED WITH RESISTANT THYROID HORMONE SYNDROME: CHALLENGE IN MANAGEMENT
1127	Tadele Desalew	THROMBOCYTOSIS AND THYROID CANCERINOMA
1128	Manoj Mathew	A RARE CASE OF ACUTE PARALYSIS PRESENTING IN A YOUNG MALE FROM HYPOKALEMIA INDUCED BY THYROTOXICOSIS
1129	Manoj Mathew	A CASE OF FALSELY ELEVATED TSH LEVEL DUE TO HETEROPHILE ANTIBODY INTERFERING WITH THYROTROPIN IMMUNOASSAY

Tour 9 – Thyroid Disease

1171	Nidhi Bansal	A RARE CASE OF REVERSIBLE CARDIOMYOPATHY MANAGED SUCCESSFULLY.
1172	Paulina Cruz	A RARE CAUSE OF COUGH: METASTATIC THYROID DISEASE FROM RENAL CELL CARCINOMA
1173	Quang Ton	IMPENDING CARDIAC TAMPONADE WITH HYPERTENSIVE EMERGENCY AS PRIMARY PRESENTATION OF HYPOTHYROIDISM
1174	Ram Jhingan	A CURIOUS CASE OF DYSHORMONOGENETIC GOITER
1175	Hagop Kojanian	HOT NODULES AND THYROID CANCER
1176	Tulsi Sharma	OCCULT THYROID CANCER WITH DISTANT METASTASIS
1177	Megan Vanderlinde-Wood	ACTIVATING AUTOANTIBODIES TO THE β1 ADRENERGIC AND M2 MUSCARINIC RECEPTORS FACILITATE ATRIAL FIBRILLATION IN PATIENTS WITH GRAVES' HYPERTHYROIDISM
1178	Naga Nalini Tirumalasetty	ERLOTINIB ASSOCIATED EXACERBATION OF HYPOTHYROIDISM WITH PERICARDIAL TAMPONADE
1179	Rajib Bhattacharya	PERSISTENT AMIODARONE-INDUCED THYROIDTOXICOSIS (AIT)

ORAL PRESENTATION SCHEDULE

Thursday, May 24, 2012

Young Investigator Oral Presentations

1:45pm – 3:15pm: Young Investigator Oral Presentation Competition

Moderator: Dr. K.M. Mohamed Shakir

*Three Oral Presentation Young Investigator Awards: $500.00, $250.00, and $100.00

Oral Presentations

3:30pm – 5:15pm: Oral Presentations

Moderator: Dr. Bill Law, Jr and Dr. Sunil Wimalawansa

Friday, May 25, 2012

Poster and Oral Presentation Awards Ceremony
General Session

8:00am – 8:15am: Presentation of Awards

Clinical Research and Clinical Trials

2:15 – 4:45pm: Clinical Research & Clinical Trials Presentations

Moderator: Dr. Samuel Dagogo-Jack and Dr. Alan Garber

Sunday, May 27, 2012

Clinical Trials

10:15am – 11:00am: Clinical Trial Presentations

Moderator: Dr. Michael Gonzalez-Campoy

Late Breaking Clinical Trials

11:00am – 12:00pm: Late Breaking Clinical Trials Presentations

Moderator: Dr. Alan Garber

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ORAL PRESENTATIONS

YOUNG INVESTIGATOR ORAL PRESENTATION COMPETITION
Thursday, May 24th, 2012 • 1:45 - 3:15pm • AGENDA

Moderators:
K.M. Mohamed Shakir, MD, Chair,
Abstract Review Subcommittee
Harmeet S. Narula, MD, Chair,
Endocrine Training Support Committee

Judges:
H. Jack Baskin, MD, MACE
Lewis E. Braverman, MD, FACE
Hossein Gharib, MD, MACP, MACE
J. Michael González-Campoy, MD, PhD, FACE

1:45 – 1:50 pm **Welcome**
Harmeet S. Narula, MD, Chair, Endocrine Training Support Committee

1:50 – 1:55 pm **Introduction of Oral Presenters**
K.M. Mohamed Shakir, MD, Chair, Abstract Review Subcommittee

1:55 – 3:10 pm **Young Investigator Oral Presentation Competition:**

1) **RELATIONSHIP OF EPICARDIAL FAT WITH OBESITY AND HYPERLIPIDEMIA**
Ritu Madan, MBBS, Creighton University School Of Medicine, Omaha, Nebraska

2) **GLYCEMIC CONTROL AT INITIATION OF HYPERBARIC OXYGEN THERAPY DOES NOT AFFECT DIABETIC LOWER EXTREMITY WOUND HEALING**
Owaise Mansuri, MD, Southern Illinois University School of Medicine, Springfield, Illinois

3) **DILEMMA IN MANAGEMENT OF WELL-DIFFERENTIATED THYROID CANCER IN PATIENTS ON HEMODIALYSIS**
Mallika Bhat, MD, Westchester Medical Center, Valhalla, New York

4) **ELEVATED APOLIPOPROTEIN A-I GENE EXPRESSION IN VITAMIN D RECEPTOR KNOCKOUT MICE**
Sandra Mesliniene, MD, University of Florida, Shands Medical Center, Jacksonville, Florida

5) **AGONISTIC AUTOANTIBODIES AS VASODILATORS IN ORTHOSTATIC HYPOTENSION: A NEW MECHANISM**
Megan Vanderlinde-Wood, MD, University of Oklahoma, Oklahoma City, Oklahoma

6) **PREVALENCE OF LATENT AUTOIMMUNE DIABETES IN YOUNG, NON OBESE, ADULT ONSET DIABETES PATIENTS WITH POOR RESPONSE TO ORAL INSULIN SECRETAGOGUES**
Ankit Shrivastav, MD, Institute of Postgraduate Medical Education and Research, Kolkata, India

3:10 – 3:15 pm **Closing**
K.M. Mohamed Shakir, MD, Chair, Abstract Review Subcommittee

*Awards Ceremony will be held Friday, May 25 at 8:00am during the General Session in the Convention Center

ADDITIONAL SESSIONS

ORAL PRESENTATIONS • Thursday, May 24, 2012 • 3:30 pm-5:15 pm

3:30 PM	**Welcome** Bill Law, Jr., MD - Moderator Sunil Wimalawansa, MD - Moderator
3:30PM – 3:45PM	**Dapagliflozin Consistently Reduces Hba1c in Patients with Type 2 Diabetes Mellitus: Pooled Subgroup Analysis of Interaction between Baseline Parameters and Hba1c across 9 Clinical Trials** Elise Hardy, MD
3:45 PM–4:00 PM	**REDUCED RISK OF HYPOGLYCEMIA WITH INSULIN DEGLUDEC VS INSULIN GLARGINE IN PATIENTS WITH TYPE 2 DIABETES REQUIRING HIGH DOSES OF BASAL INSULIN: META-ANALYSIS OF FIVE RANDOMIZED TRIALS** Helena Rodbard, MD
4:00 PM–4:15 PM	**SIGNIFICANT WEIGHT LOSS (WL) WITH CONTROLLED-RELEASE PHENTERMINE/TOPIRAMATE (PHEN/TPM CR) IS ASSOCIATED WITH SIGNIFICANT REDUCTIONS IN CARDIOMETABOLIC PARAMETERS OVER 108 WEEKS** Nancy Bohannon, MD
4:15 PM–4:30 PM	**RANDOMIZED, PLACEBO-CONTROLLED, DOUBLE-BLIND, 24-WEEK STUDY OF LINAGLIPTIN 5 MG/DAY IN BLACK/AFRICAN AMERICAN PATIENTS WITH TYPE 2 DIABETES** James Thrasher, MD
4:30 PM–4:45 PM	**EFFECT OF EARLY WEIGHT LOSS ON TYPE 2 DIABETES THROUGH SURGICAL INTERVENTION 2 YEARS AFTER GASTRIC BANDING** Ted Okerson, MD
4:45 PM–5:00 PM	**LONG TERM FOLLOW UP OF PATIENTS WITH TYPE 1 DIABETES ON LIRAGLUTIDE AND THE EFFECT OF LIRAGLUTIDE AS ADDITIONAL TREATMENT IN OBESE PATIENTS WITH TYPE 1 DIABETES** Paresh Dandona, MD

CLINICAL TRIALS • Sunday, May 27, 2012 • 10:15 am-11:00 am

10:15 AM	**Welcome** Michael González-Campoy, MD - Moderator
10:15 AM–10:30 AM	**DECREASED PROGRESSION TO TYPE 2 DIABETES MELLITUS (T2DM) AFTER 1 YEAR OF TREATMENT WITH CONTROLLED-RELEASE PHENTERMINE/TOPIRAMATE (PHEN/TPM CR) IN OBESE SUBJECTS WITH PREDIABETES** W. Timothy Garvey, MD
10:30 AM–10:45 AM	**200 U/ML INSULIN DEGLUDEC IMPROVES GLYCEMIC CONTROL SIMILAR TO INSULIN GLARGINE WITH A LOW RISK OF HYPOGLYCEMIA IN INSULIN-NAÏVE PEOPLE WITH TYPE 2 DIABETES** Richard Bergenstal, MD
10:45 AM–11:00 AM	**DRAMATIC DIFFERENCES IN LONG-TERM CURE RATES FOLLOWING UNILATERAL VS. BILATERAL PARATHYROIDECTOMY: AN 18-YEAR, SINGLE-CENTER STUDY IN 15,500 PATIENTS** James Norman, MD

CLINICAL RESEARCH & CLINICAL TRIALS
Friday, May 25, 2012 • 2:15pm – 4:45pm

2:15pm

Welcome
Dr. Samuel Dagogo-Jack and
Dr. Alan Garber, Moderators

2:15 pm–2:30 pm

Dapagliflozin is Associated with Weight Reduction as a Secondary Benefit in Patients with Type 2 Diabetes Mellitus: Pooled Subgroup Analysis of 9 Clinical Trials

Afshin Salsali, MD

2:30pm – 2:45pm

Responses of Patients with Adult Growth Hormone Deficiency Treated with Growth Hormone Over 3 Years: Analysis of Results from The Answer Program®

Murray Gordon, MD

2:45pm – 3:00pm

Insulin Degludec Is Highly Efficacious Regardless of Diabetes Duration or Body Mass Index: A Cross-Trial Evaluation

Lawrence Blonde, MD

3:00pm – 3:15pm

Ultra-Long Pharmacokinetic Properties of Insulin Degludec in Younger Adults Are Preserved in Geriatric Subjects with Type 1 Diabetes

Stefan Korsatko, MD

3:15pm – 3:30pm

Patients Are More Likely To Reach A1c Target at Any Given Time during 26 Weeks' Treatment with Liraglutide Compared with both Sitagliptin and Exenatide

Robert Ratner, MD

3:30pm – 3:45pm

Changes in Body Composition Following Gastric Bypass or Gastric Banding

Helmuth Billy, MD

3:45pm – 4:00pm

Rapid Resolution of Diabetes-Related Risk Markers and Hypertension in Morbidly Obese Individuals with an Exercise-Centric Intense Lifestyle Intervention

Robert Huizenga, MD

4:00pm – 4:15pm

Primary Care Detection of CKD in Adults with Type-2 Diabetes in The Add-CKD Study

Lynda Szczech, MD

4:15pm – 4:30pm

Colesevelam Hcl: Glycemic and Lipid Parameter Effects in Patients with Type 2 Diabetes Mellitus (T2dm) Treated with Metformin-Based Therapy and A Statin

Harold E. Bays, MD

4:30pm – 4:45pm

Initial Combination of Linagliptin and Metformin in Patients with Type 2 Diabetes Mellitus: Efficacy and Safety in a 1-Year, Randomized, Double-Blind Extension Study

Maximilian von Eynatten, MD

LATE BREAKING CLINICAL TRIALS
Sunday, May 27, 2012 • 11:00am – 12:00pm

11:00am

Welcome
Dr. Alan Garber, Moderator

11:00am – 11:20am

Treatment of Obstructive Coronary Artery Disease with the Resolute Zotarolimus-Eluting Stent in Patients with Diabetes Mellitus

Scott Lee, MD

11:20am – 11:40am

In T2D Patients With Baseline A1c <8.0%, Liraglutide Achieves A1c Targets More Often Than Sitagliptin or Exenatide

Allen King, MD

11:40am – 12:00pm

Multicenter Validation of a Novel Gene Expression Classifier to Preoperatively Identify Benign Thyroid Nodules with Indeterminate FNA Cytology

Rick Lanman, MD

AMERICAN ASSOCIATION OF CLINICAL ENDOCRINOLOGISTS

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ABSTRACTS

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ABSTRACTS

ADRENAL DISORDERS

Abstract #100

PHEOCHROMOCYTOMA COEXISTING WITH VASCULAR LESIONS

Sunil Kota, MD, Siva Kota, Svs Krishna, Lalit Meher, Kirtikumar Modi

Objective: To report associated vascular lesions in pheochromocytoma and discuss possible mechanisms.
Methods: From 1990 to 2010, 50 patients were diagnosed with pheochromocytoma/ paragangliomas. Hospital charts were reviewed for coexistent vascular lesions.
Results: 50 patients (M: F= 35: 15) with mean age of 45.5±23.3 years, were diagnosed to have pheochromocytoma (42 adrenal and 8 extra adrenal). 7 patients (14%) had coexisting vascular lesions including renal artery stenosis (RAS) in 4, aortoarteritis in 1, aortic aneurysm in 1 and inferior vena cava thrombosis in 1. All of them harbored pheochromocytoma [adrenal in 6 patients (4- left, 1- right) and ectopic in one (at left renal hilum)]. RAS was suspected because of small ipsilateral kidneys in 2, delayed nephrogram in 1 and impingement of renal artery in 1 patient. A patient with RAS due to intimal fibrosis was offered percutaneous baloon angioplasty, other 3 improved after adrenalectomy and lysis of fibrous adhesive bands. Aortoarterits was treated with oral steroids. Inferior vena cava thrombosis was reversed with anticoagulants. The patient with abdominal aortic aneurysm was advised for annual follow up on account of its size of 4.5 cm and asymptomatic presentation.
Discussion: Pheochromocytoma has been described previously in coexistence with RAS, renal artery aneurysm, inferior vena cava thrombosis. Though the coexistence of the vascular abnormalities can reflect chance association, we propose certain causative factors. Mechanism for RAS in pheochromocytoma include 1) tumor compression, 2) catecholamine induced vasospasm, 3) periarterial adhesion, 4) associated atherosclerosis and fibromuscular dysplasia. Mechanism for associated aortoarteritis include 1) catecholamine induced endothelial damage and intimal fibrosis, 2) association with autoimmune conditions like SLE, Behcet's disease. Mechanisms for associated IVC thrombosis include 1) local compression leading to alteration in blood flow and stasis 2) sustained hypertension leading to vascular endothelial injury and hypercoagulbility, 3) association with autoimmune conditions like SLE, Behcet's disease and 4) An underlying anatomic abnormality or coagulation disorder. Mechanism for associated abdominal aortic aneurysm are 1) persistent exposure to high catecholamines induced vascular wall damage and weakening 2) Associated cigarette smoking, increasing age, hypertension and atherosclerosis 3) Coexistence of vasculitis like takayasu's disease, Giant cell arteritis and cystic medial necrosis due to marfan and ehlers danlos syndrome.
Conclusion: The state of catecholamine excess and various other coexisting factors can lead to simultaneous occurrence of uncommon vascular abnormalities.

Abstract #101

CASE OF DISSEMINATED HISTOPLASMOSIS IN IMMUNOCOMPITENT PATIENT WHO DEVELOPED ADRENAL INSUFFICIENCY WHEN TREATED WITH ITRACONAZOLE

Issac Sachmechi, MD, FACP, FACE, Kelash Kumar, MD, Chinmay Patel, Uday Shankar, Victoria Bellot

Objective: Histoplasmosis is the most prevalent endemic mycosis (Fungal infection) in the United States. Usually, the infection follows an asymptomatic and self limited. We describe disseminated Histoplasmosis in an immunocompetent patient from a non endemic area without lung involvement but involving the adrenal gland.
Case Presentation: We report a case of Disseminated Histoplasmosis with iatrogenic adrenal insufficiency in a 41 year old male from Bangladesh with an extensive travel history to western countries, presented with weight loss, intermittent loose watery stools, generalized weakness and anorexia for 4 months. On physical exam the patient was noted to have generalized wasting, had few mucosal papular lesions on the tongue. His labs were notable for hyponatremia: Low Na 126 meq/l (135-145meq/l), hypoalbuminemia: 1.7g/dl (3.4-5.4 g/dl) and prealbumin: 3.3mg/dl (normal 16-35mg/dl), AST 115 U/l, ALT 53 U/l, GGT 102, Alkaline phos: 489(30-115 U/l) and PT/INR: 12.8/1.15 and HIV negative. Chest x-ray was normal. His stools studies including C.difficile were negative. The Contrast computed tomography imaging of chest, abdomen and pelvis revealed bilateral adrenal enlargement. Blood cultures and fungal cultures were negative. The serum IgG for Histoplasma was positive. Biopsies of the skin, liver and colon revealed Histoplasma organisms and were consistent with disseminated Histoplasmosis. The patient was started on the intravenous Amphotericin B for two weeks,

with improvement in appetite and resolution of diarrhea. The patient was discharged home on Itraconazole 200mg PO daily. Two months after the discharge the patient readmitted from medical clinic on his follow up, with the hyponatremia, hyperkalemia, acidosis and generalized weakness. ACTH stimulation test revealed cortisol levels: 0.3Ug/dl at baseline & 0.6 Ug/dl at 30 minutes, consistent with primary adrenal insufficiency. The patient was started on hydrocortisone and fludrocortisones with resolution of weakness, weight gain and normalization of electrolytes.
Discussion: Itraconazole can cause adrenal insufficiency by inhibiting CYP3A in less than 2% of patients. This medication adverse effect may be due to subclinical adrenal insufficiency caused by Histoplasma infestation of adrenals with addition of Itraconazole inducing full blown adrenal insufficiency. The presence of bilateral adrenal enlargement raised the possibility of Disseminated Histoplasmosis while biopsies of the skin, colon and liver confirmed this diagnosis.
Conclusion: Patients with disseminated Histoplasmosis, treated with Itraconazole, should be closely monitored for adrenal insufficiency.

Abstract #102

ADRENOCORTICOTROPHIC INDEPENDENT CUSHING'S SYNDROME: BEWARE OF MALIGNANCY IN EVERY LARGE ADRENAL TUMOR

Chee Kian Chew, MD,
Rinkoo Dalan, MBBS, MRCP, FRCP (Edin), FAMS (Endocrinology)

Case Presentation: A 60 years old lady, with longstanding diabetes mellitus, hypertension and hyperlipidemia presented with clinical CS without virilisation. She had developed features of CS very rapidly in the preceding one month. Biochemically, 8am cortisol: 795 nmol/L (RI: 240-618), ACTH <2.0 pmol/L, 24 hours urinary free cortisol (UFC): 3015 nmol/day (RI: 59-413), 1mg overnight dexamethasone suppression test showed non-suppressible cortisol : 746 nmol/L and low dose dexamethasone suppression test also showed non-suppressible cortisol: 718 nmol/L. The CT scan showed a left adrenal, heterogenous 4.6x4.6x4.1 cm mass, with pre contrast attenuation of 26 HU, delayed attenuation of 52 HU and absolute washout of 64%. This confirmed ACTH independent CS secondary to left adrenal adenoma. She underwent a laproscopic adrenalectomy and the histology showed an unencapsulated adrenal cortical neoplasm with less than 25% scattered foci of clear cells , Fuhrman nuclear grade 3, 5 mitotic figures per 50 hpf, Weiss score 2/9 with evidence of tumor cells in the peripheries. These histopathological findings although suggested incomplete resection, didn't meet the criteria for adrenocortical carcinoma (ACC). Her post operative random cortisol was 31 nmol/L consistent with adrenal insufficiency and a remission. Although initial CT thorax, abdomen and pelvis did not show any evidence of metastasis, a CT scan just 6 months later showed recurrence of left adrenal tumor measuring 2.3x3.1 cm with mesenteric, retroperitoneal, peritoneal and lung metastasis. CT guided biopsy of left para-aortic lymph node confirmed metastasis. Biochemically, patient had hypercortisolism with 24 hours UFC of 1371 nmol/day indicative of a recurrence. She has been started on ketoconazole and palliative chemotherapy with mitotane.
Discussion: Although the histology in this case is more suggestive of benign tumor and the initial tumor surveillance studies were negative, she developed metastatic disease within 6 months. Despite complete resection in Stage I-III disease, approximately 40% of patients develop metastasis within 2 years. Adjuvant mitotane therapy may prolong recurrence-free survival in patients with radically resected ACC up to 42 months.
Conclusion: It is important to consider a possibility of ACC in every large adrenal adenoma associated with rapidly progressive CS.

Abstract #103

PHEOCHROMOCYTOMA PRESENTING WITH HEMOPTYSIS: CASE REPORT AND REVIEW OF OTHER UNUSUAL MANIFESTATIONS OF PHEOCHROMOCYTOMA

Sheryl Tugna, MD, Thelma Crisostomo

Objective: To report a case of pheochromocytoma presenting with hemoptysis and other unusual manifestations of pheochromocytoma.
Case Presentation: A 45 yr. old male was brought to ER with hemoptysis. There was no other bleeding manifestation. He is hypertensive for 10 years with systolic BP of 130-180 mmHg. 6 months ago, he started to have palpitations and tremors associated with headache and diaphoresis. At the ER, he was anxious and in respiratory distress. HR was 115 and BP was 200/100 mmHg. Nicardipine drip was started. Coagulation studies were normal. Serum Amylase was elevated at 418 U/L (N.V 28-100). CPK was high at 809 U/L (N.V 39-308) No lesion and mass noted on Bronchoscopy. Chest CT scan showed pulmonary hemorrhage and edema with a finding of left adrenal mass measuring 5.6 X 5.8 X 5.0 cm (APxWxH) exhibiting central hypodensity. Impression was Adrenal Incidentaloma to consider Pheochromocytoma. Urinary VMA and Metanephrine were elevated at 390 umol/24 hr.

(N.V 0-68) and 166 umol/24 hr. (N.V 0-5.5) respectively. By 3rd day, blood pressure stabilized to normal levels with resolution of hemoptysis.Chest Xray showed clearing of pulmonary edema. He was given oral anti-hypertensives. Systolic BP maintained at 110-130 mmHg. Left Adrenalectomy was done. Histopathologic result confirmed the diagnosis of Pheochromocytoma. Post-op, blood pressure was maintained at SBP 120-140 mmHg. 4 days after surgery, he was discharged.

Discussion: Pheochromocytoma usually presents with spell-signs and symptoms like headache, tremors, hypertension and diaphoresis. The unusual manifestations in our patient were hemoptysis, pulmonary edema, elevated serum Amylase and CPK.Bronchoscopy and CT scan of the Chest result were not able to identify the cause of hemoptysis in our patient. Frymoyer et.al proposed that the sudden catecholamine release could cause severe hypertension in pheochromocytoma. This may cause pulmonary venous hypertension, pulmonary edema and hemoptysis.Patient's hemoptysis and pulmonary edema resolved concurrently with normalization of blood pressure. The CPK level was elevated in the absence of myocardial infarction and muscle trauma. Bahtnagar et al suggested that catecholamine may cause vasoconstriction leading to ischemia of the muscle. Patient has elevated serum amylase in the absence of abdominal pain and radiographic evidence of pancreatitis. Review of literature has shown that the source of amylase was pulmonary endothelial cells under ischemic damage caused by vasoconstrictive effects of catecholamine.

Conclusion: Clinicians should be aware of various clinical presentations of Pheochromocytoma so as to have an early diagnosis and treatment before life-threatening complications develops.

Abstract #104

QUALITATIVE MOLECULAR PROFILING OF CLINICALLY FAVORABLE RESECTED ADRENAL METASTASES

Elliot Mitmaker, MD, Raymon Grogan, MD, Joe Kansopon, Avital Harari, Jimmy Hwang, Jessica Gosnell, Orlo Clark, MD, Quan-Yang Duh, Wen Shen

Objective: To determine the molecular profiles of resected adrenal metastases in order to explain the favorable clinical characteristics and outcomes in patients undergoing adrenalectomy for isolated metastatic disease with a 12-month disease-free interval.

Methods: Paraffin-embedded tissue blocks of metastatic adrenal tumors from 7 different primary sites (lung, breast, colorectal, renal cell, adrenocortical, melanoma and thyroid) were collected. A commercially available 92-gene assay was used to determine a metastatic gene expression profile for isolated adrenal metastasis. Pooled RNA/cDNA of adrenal metastases was compared to benign, non-functioning adrenal adenomas. Significance analysis of microarrays (SAM) determined differences in gene expression between metastatic and benign non-functioning adrenal tumors.

Results: A total of 18 genes were over-expressed in the adrenal metastases group as compared to benign adrenal controls (False Discovery Rate = 5%). Further analysis revealed overexpression of 9 genes (4 "pro-metastatic", 3 "anti-metastatic" and 2 chemokine genes) in the metastatic group with a greater than 2-fold expression difference. Four genes (FN1, MTA2, MMP2 and SET) were significantly under-expressed in the metastatic group, with a greater than 2-fold expression difference. All matrix metalloproteinase (MMP) genes were under-expressed while tissue inhibitors of MMPs were overexpressed in the adrenal metastases group.

Discussion: The adrenal glands are common sites of metastases from a variety of skin and solid organ cancers. Laparoscopic adrenalectomy for isolated adrenal metastases, once considered controversial, is now increasingly being performed for cure or palliation. We have previously demonstrated that patients with a disease-free interval of more than 12 months between initial diagnosis of primary cancer and detection of adrenal metastasis had slower-growing tumors and an improved overall survival. This study attempts to identify clinically favorable adrenal metastases at the molecular level.

Conclusion: Resected adrenal metastases from patients with a disease-free interval of more than 12 months have a favorable gene profile. We found that genes related to tumor proliferation were over-expressed in the metastatic group, while genes related to tumor invasion were generally under-expressed. Further understanding of the molecular profiles of adrenal metastasis could potentially provide prognostic information and allow for improved selection of patients with isolated adrenal metastases for adrenalectomy.

Abstract #105

SUPPRESSION AND RECOVERY OF HPA FUNCTION AFTER A SINGLE EPIDURAL GLUCOCORTICOID INJECTION: DECONVOLUTION ESTIMATION OF ACTH AND CORTISOL SECRETORY DYNAMICS

Donna Lawson, DO, Dakshin Gullapalli, Johannes Veldhuis, MD, Ali Iranmanesh, MD

Objective: This study was designed to assess the magnitude and duration of disrupted ACTH and cortisol secretory

patterns and time to recovery after epidural glucocorticoid injection.

Methods: Nine men (25-63 years) on 4 separate days (baseline, 1, 4, and 12 weeks). Triamcinolone (80 mg) was injected epidurally right after the 1st study session. During each visit, blood was collected in a fasting state at 10-min intervals for a period of 4 hours, with ovine CRH (1μg/Kg) injected after the 6th blood draw (min 60). ACTH (pg/mL) and cortisol (μg/dL) concentrations were measured in each blood sample, and their respective secretory properties were assessed by deconvolution analysis

Results: Mean (±SEM) pre-CRH baseline unstimulated plasma ACTH (6.1 ± 0.3 v 25.1 ± 1.2: P <0.0001) and cortisol (1.7 ± 0.1 v 12.9 ± 0.1: P < 0.0001) concentrations were significantly decreased at week 1. Although full recovery of ACTH occurred at week 4 (25.4 ± 2.6 v 25.1 ± 1.2: P = NS), respective mean cortisol levels of 10.9 ± 0.3 and 11.1 ± 0.1 at weeks 4 and 12, continued to be lower than the pre-treatment values (P=0.01). Corticotropic response to CRH stimulation was similarly blunted at week 1 with significant decreases in the 3-hr mean (±SEM) ACTH (11.1 ± 0.2 v 40.4 ± 3.4: P <0.0001) and cortisol (3.6 ± 0.2 v 21.3 ± 0.8: P < 0.0001) concentrations. While ACTH response to CRH normalized at week 4 (37.5 ± 2.4 v 40.4 ± 3.4: P=NS), cortisol response did not fully reverse by week 12 (19.6 ± 0.9 v 21.3 ± 0.8: P <0.01). Twenty-four hr urinary free cortisol (μg) was diminished at week 1 (8.7 ± 1.2 v 62 ± 6.1: P <0.0001), and did not normalize until week 12. Deconvolution analysis of post-CRH time series revealed altered pulsatile and basal secretory modes of ACTH and cortisol release with significant suppression at week 1, and recovery at week 4. Changes in pulsatile ACTH and cortisol secretion were primarily due to changes in the mass of hormone secreted per burst.

Discussion: Corticotropic function was uniformly suppressed within 7 days of epidural steroid injection. Pre- and post-CRH concentrations of ACTH and cortisol, and their respective secretory properties allowed a better understanding of underlying mechanisms, and the required time for the axis to recover.

Conclusion: Epidural glucocorticoid administration markedly represses HPA output via suppression of both basal and pulsatile modes of ACTH and cortisol release. The average recovery time appears to be 4 weeks for ACTH, and potentially 12 weeks or more for cortisol. These inferences warrant future confirmation in larger cohorts and over a longer time period.

Abstract #106

PF4 ANTIBODY POSITIVITY IN THE SETTING OF BILATERAL ADRENAL HEMORRHAGE

Mini Mathew, Pharm.D., D.O., Kamalpreet Singh, MD, Deepika Reddy

Case Presentation: 56 year old female underwent screening colonoscopy which led to diagnosis of Stage 4 Carcinoid tumor. Treatment consisted of right hemicolectomy with ileal resection. Postoperatively she was found to have bilateral pulmonary emboli and was started on heparin, which resulted in her developing heparin induced thrombocytopenia. She was placed on Coumadin, with her INR goal being closer to 2.0. CT of the abdomen showed possible adrenal hemorrhage, but she had no hypotension or abnormalities in sodium or potassium levels. Three and a half weeks later she presented to the hospital with complaints of nausea, vomiting and dizziness. Na was 126 and K was 5.7. Medical staff was unable to place IV access and so she was given IM Decadron. She reported improvement of symptoms within 2 hours. She underwent a cosynotropin stimulation test in the morning which showed a baseline ACTH of 1232 pg/mL and initial cortisol of 6.2 mcg/dL, at 30 minutes cortisol was 6.0mcg/dL and at 60 minutes was 5.5 mcg/dL. CT of the abdomen/pelvis showed bilateral adrenal hemorrhages. She was started on hydrocortisone and florinef and reported symptomatic improvement, repeat Na and K levels normalized.

Discussion: Bilateral adrenal hemorrhages are present in about 1% of routine autopsies. Major risk factors for adrenal hemorrhage include hypercoaguable states, sepsis, severe stress, and anticoagulant therapy. Heparin is the most common anticoagulant used in hospitalized patients and heparin induced thrombocytopenia (HIT) is a rare complication. HIT is caused by platelet-activating antibodies that recognize complexes of platelet factor four and heparin. This typically results in thrombocytopenia but bleeding is seldom an issue.

Conclusion: Bilateral adrenal hemorrhage should be suspected in patients with complaints of abdominal pain and hypotension with recent heparin exposure. The role of HIT in bilateral adrenal hemorrhage is likely underestimated as use of heparin is not always documented or PF4 antibodies are not ordered. The predominant thrombotic event is usually arterial but in this case it was venous. Adrenal insufficiency usually presents after a latency period weeks after initial episode of bilateral adrenal hemorrhage.

Abstract #107

EAT MORE YET FEEL WEAK

Radha Devi, MD, Parakkal Deepak

Objective: Megesterol acetate (Megace) is a synthetic progesterone derivative used in the treatment of cachexia and anorexia in metastatic cancers or AIDS. It is associated with side effects like thromboembolism, hypertension, gynecomastia and adrenal insufficiency. Patients with adrenal insufficiency may present with non-specific symptoms of fatigue, anorexia and decreased libido or hypotension and cardio vascular instability in times of stress. This case highlights the serious side effect of adrenal insufficiency caused by Megace.
Case Presentation: A 65 year old man with past medical history of coronary artery disease, hypertension, abdominal aortic aneurysm, and failed renal transplant on hemodialysis, was admitted to the hospital with complaints of altered mental status. He was placed on Megace 800 mg per day to treat decreased appetite and weight loss of 60 lb over the last 2 months. Physical exam revealed no mucosal or flexural hyper pigmentation with a normal blood pressure and neurological exam. Basic labs, thyroid function test and CT scan of the head were normal. An 8 am serum cortisol level was less than 6.2 microgram per deciliter(mcg/dl) and a low dose cosyntropin test was performed revealing cortisol level of < 1mcg/dl and ACTH level of 11 pg/ml indicating secondary adrenal insufficiency. A CT of the abdomen (done to rule out malignancy) revealed enlargement of his abdominal aortic aneurysm. Megace was discontinued and he was started on stress dose of steroids with hydrocortisone at 100mg intravenously every 8 hours, prior to undergoing endovascular repair of his aneurysm. He was discharged on a gradual taper of oral hydrocortisone with instructions not to resume megace on discharge.
Discussion: Megace is postulated to lead to a suppression of the hypothalamic pituitary axis by its glucocorticoid like action especially when used in doses more than 300mg per day. Patients may present with symptoms of acute adrenal insufficiency in times of stress or non specific symptoms of chronic adrenal insufficiency like fatigue. It is essential for physicians to be aware of this potentially life threatening side effect prior to prescribing this medication. Hence, patients receiving chronic high dose Megace therapy may need stress doses of glucocorticoids in times of stress. It has also been suggested that patients should be prescribed Megace as an appetite stimulant only for shorter periods and should be tapered off to avoid precipitating an adrenal crisis.
Conclusion: It is important to recognize the early symptoms and signs of adrenal insufficiency secondary to this under recognized etiology, in order to prevent morbidity and mortality.

Abstract #108

FAMILIAL CUSHING'S SYNDROME DUE TO A BILATERAL ACTH-INDEPENDENT MACRONODULAR ADRENAL HYPERPLASIA (AIMAH) RELATED TO THE ECTOPIC EXPRESSION OF BETA ADRENERGIC RECEPTORS

Duarte Pignatelli, MD, PhD, Jorge Lima

Objective: ACTH-independent bilateral macronodular hyperplasia (AIMAH) is a rare cause of Cushing's syndrome(CS). Recent studies demonstrated adrenal cortisol secretion to be regulated by ectopic membrane hormone receptors(HR) , but few reports described familial aggregation in these cases. We report a familial case in which three members of a family were confirmed as having this syndrome.
Methods: The clinical screening for potentially illegitimate HR was done according to A Lacroix protocol. The first case was diagnosed by postural testing and propranolol (Prop) suppression testing. His daughter had a clear response to isoproterenol and the affected son had AIMAH, but not Cushing's syndrome. Histological analysis confirmed the diagnosis of AIMAH in the father and his daughter that were the two only cases that were surgically intervened. Real-time PCR was also performed in samples obtained at surgery.
Case Presentation: The father was diagnosed as having a posture-sensitive, Prop-responsive, CS due to an asymmetric and bilateral adrenal hyperplasia. Many years after this case was diagnosed, his daughter also appeared with CS and an MRI also revealed the presence of bilateral AIMAH. One of her brothers was tested for the same disease and in spite of not having any CS characteristics or hypercortisolism he had bilateral AIMAH! A last brother was also studied but had neither CS nor AIMAH as detected by MRI. The father was initially submitted to unilateral adrenalectomy but later CS relapsed and he had to be treated with Prop. Remission then lasted for many years. Prop remarkably normalized the bl.pressure, the cortisol levels and its circadian rhythm. The response to Prop was much less efficient in the case of his daughter, She was also submitted to unilateral adrenalectomy with success (normalization of the signs and symptoms of CS as well as the cortisol levels
Discussion: This is one of the first cases of AIMAH to reveal a clear hereditary transmission. In the present case AIMAH was dependent on the ectopic expression of beta-adrenergic receptors. This was confirmed by the performance of Real-time PCR. The differences in the clinical expression in the different members of the family

namely the different response to the use of Propranolol in spite of both cases having an hyper-expression of β-adrenergic receptors, deserves consideration.
Conclusion: We conclude that heritability may be an important pathogenic cause of ectopic receptor expression in AIMAH cases and screening of family members of affected patients may reveal a much higher frequency of such cases and allow the design of appropriate genetic studies to be dome in muticenter studies.

Abstract #109

THE PREVALENCE OF POSTOPERATIVE HYPOGLYCEMIA FOLLOWING ADRENALECTOMY FOR PHEOCHROMOCYTOMA: A RARE AND OFTEN FORGOTTEN COMPLICATION

Elliot Mitmaker, MD, Raymon Grogan, MD, Robin Cisco, MD, Daniele Rottkamp, Jessica Gosnell, Orlo Clark, MD, Wen Shen, J. Blake Tyrrell, Quan-Yang Duh

Objective: To determine the prevalence of postoperative hypoglycemia in pheochromocytoma patients following adrenalectomy and to investigate the associated risk factors that predispose these patients to develop postoperative hypoglycemia.
Methods: We retrospectively reviewed all adrenalectomies performed between 1993-2011 at a single institution and identified 124 patients who underwent laparoscopic or open adrenal resections for pheochromocytoma.
Case Presentation: 81 of the 124 patients had serial glucose levels measured during the immediate postoperative period. Thirteen patients (13/81=16%) were diagnosed with non-insulin dependent diabetes mellitus preoperatively, while 3 patients had long-standing insulin dependent diabetes mellitus. Four patients (4.9%) developed hypoglycemia within 4 hours after adrenalectomy. These four patients were women and had an average body mass index (BMI) of 21, compared to a higher average BMI (27.4; p=0.055) among those who remained normoglycemic (range=70-199 mg/dL). None of the four patients with postoperative hypoglycemia had preoperative diabetes, although one patient had a preoperative HgA1C=6.9. These four patients were treated with intravenous 50% dextrose solution and became normoglycemic after 3 hours. All patients received preoperative alpha-blocking agents, as per our routine for preoperative preparation for pheochromocytoma. One patient who had received both alpha and beta adrenergic-blockade had severe postoperative hypoglycemia (glucose=14 mg/dL) and was unresponsive in the recovery room until after treatment with dextrose solution.
Discussion: Pheochromocytomas are rare catecholamine-secreting tumors of the adrenal medulla. Excessive catecholamine release has multiple physiologic effects, including abnormal glucose metabolism. Hyperglycemia and new onset diabetes are well described, but the problem of severe postoperative hypoglycemia following resection is under-recognized. Postoperative hypoglycemia following adrenalectomy for pheochromocytoma is rare but potentially serious. It is likely caused by the acute withdrawal of excessive catecholamines, coupled with preoperative alpha and beta-adrenergic blockade, blunting the normal mechanism of glucose regulation.
Conclusion: Postoperative blood glucose monitoring is recommended to identify this potentially fatal complication. Implementing a standardized hospital-based protocol for postoperative continuous blood glucose monitoring will lead to early recognition of this rare and easily reversible metabolic event.

Abstract #114

LATE ONSET CONGENITAL ADRENAL HYPERPLASIA (CAH) PRESENTING AS ISOLATED NOXIOUS BODY ODOR IN AN ADULT MALE - A SMELLY DIAGNOSIS!

Nisha Acharya, MD, Melissa Li-Ng

Case Presentation: A 43-year old male presented with intermittent noxious body for the past year. He noted some improvement on gluten-free diet but continued to have recurrence of this symptom despite daily showering and deodorant use. He noted hair thinning at the top of his scalp for the past 7 years. He otherwise felt well and denied erectile dysfunction, acne, and mood changes. He fathered a 4-year old daughter. He denied family history of endocrinopathies and infertility. Physical examination revealed a well-appearing male, blood pressure 118/81 mmHg, height 1.85 m, BMI 22.30 kg/m2. Testes were normal size & consistency, no palpable masses. Laboratory investigations including complete blood count, thyroid-stimulating hormone, hemoglobin A1C and comprehensive metabolic panel were unremarkable. Total and free testosterone as well as DHEA-S levels were within normal range. 17-hydroxyprogesterone (17-OHP), however, was elevated at 5.4 ng/ml (normal 0.4-1.8 ng/ml). Subsequently, ACTH stimulation showed an increase in 17-OHP levels from 7.4 ng/ml at baseline to 115.8 ng/ml at 60 minutes. The cortisol level increased from 9.4 ug/dl to 17.5 ug/dl at 60 minutes. These results were consistent with late onset CAH. Patient was offered dexamethasone therapy to decrease his body odor but he opted to forego any medical treatment at this time and was referred for genetic counseling.

Discussion: Late onset CAH due to CYP21A2 (21-hydroxylase) deficiency is characterized by signs of androgen excess later in life. The defective conversion of 17-hydroxyprogesterone to 11-deoxycortisol in patients with CYP21A2 deficiency causes decreased cortisol synthesis, which results in increased ACTH secretion. This in turn causes adrenal stimulation leading to increased production of androgens. Children with late onset CAH can present with premature adrenarche, which includes pubic hair, axillary hair and adult body odor. Men with late onset CAH can present with acne, infertility or testicular adrenal rest tumors. In the skin, androgen excess stimulates hyperplasia of sebaceous glands and apocrine glands. Sebaceous glands produce sebum while apocrine glands secrete a fatty, viscous sweat. Bacterial breakdown of sebum and apocrine sweat produces body odor.
Conclusion: Our case is unique because of the isolated finding of abnormal body odor as a presenting symptom of late-onset CAH. Thus it is important to think of late onset CAH in the differential diagnosis when evaluating a patient for body odor.

Abstract #111

HYPERCORTISOLISM IN YOUNG PATIENT WITH OBESITY

Zulfiya Shafigullina, MD, Ludmila Velikanova, Natalya Vorokchobina

Objective: The aim of this study was to investigate the features of adrenal steroid synthesis in young people with obesity.
Methods: We examined 40 patients (15-20 years old) with obesity (BMI 32.3 ±0.8). Twenty three subjects (57.5%) had arterial hypertension. Control group included 20 healthy subjects (15-20 years old, BMI 23.4 ±0.6) without obesity and hypertension. All patients underwent hormonal evaluation for circadian rhythm of plasma cortisol and ACTH secretion and also low dose (2 mg) dexamethasone suppression test (DST). Intermediates of steroido-genesis were assessed by means of high-performance liquid chromatography (HPLC) including measurement of plasma levels of cortisol (F), cortisone (E), corticosterone (B), 11-deoxycorticosterone (DOC), 11-deoxycortisol (S) and urinary excretion of free cortisol (UfF) and free cortisone (UfE).
Results: Among 40 patients with obesity, hormonal work-up showed high baseline cortisol in 30%, high ACTH in 27.5% and disturbed circadian cortisol rhythm in 22.5%. DST was normal in the majority of subjects although 6 patients (15%) had plasma cortisol concentrations above 60 nmol/L after this test. These patients were found to have significantly higher concentrations of F, B, UfF and UfE compared with the control group and normal indices of F/E and UFF/UFE. However, 7 patients (17.5%) had increased F/E and UfF/UfE indices and 5 showed lack of UfF suppression after 2 mg dexamethasone. Significant positive association was found between concentration of plasma cortisol by RIA, plasma cortisol by HPLC and fat mass ($P < 0.001$).
Discussion: It is known that young obese patients may show hyperactivity of hypothalamic-pituitary-adrenal (HPA) axis, which leads to a state of functional hypercortisolism. HPA axis may be dysregulated due to such reasons as puberty, stress, early life events and others. Also, over-expression of 11-beta-hydroxysteroid dehydrogenase type 1 in obese patients can result in increased conversion of cortisone (E) to cortisol (F) thus increasing glucocorticoid activity.
Conclusion: Our data confirms that young people with obesity often reveal functional hypercortisolism. Some patients have biochemical abnormalities indicating subclinical corticosteroid excess. These endocrine abnormalities in the young obese may place them at risk for glucose intolerance, diabetes, bone loss and other conditions.

Abstract #112

ADRENOMYELONEUROPATHY AND PRIMARY ADRENAL INSUFFICIENCY

Shilpa Swamy, MD,
Donald Richardson, MD, FACE, FACP

Objective: To describe an unusual cause of primary adrenal insufficiency.
Case Presentation: A 45 year old Japanese male was evaluated for adrenal insufficiency. The patient was diagnosed with adrenomyeloneuropathy at age 35 during further investigation of lower extremity weakness. Recent serum cortisol, drawn at 1pm, was 14mcg/dL with an ACTH of 69.2 pg/mL. No clinical signs of adrenal insufficiency were found but increased ACTH suggests impaired adrenal reserve.
Discussion: Adrenomyeloneuropathy (AMN) is a sub-set of X-linked Adrenoleukodystrophy (X-ALD), a group of disorders with abnormal accumulation of very long chain fatty acids (VLCFA) in the brain, adrenal cortex and Leydig cells of the testes. Responsible mutations affect the ABCD1 gene on chromosome Xp28, which encodes the protein involved in the import of VLCFA into the peroxisome. Adrenal pathology may be attributed to the combination of effects of VLCFA on membrane structure (increased micro-viscosity) and accumulation of cholesterol esterified by VLCFA (which are poor substrates for cholesterol hydrolases), impairing the response

of adrenal cortical cells to ACTH stimulation. The AMN phenotype represents ~45% of X-ALD. It presents in adulthood with spastic paraparesis and peripheral neuropathy as a consequence of spinal cord and peripheral nerve demyelination, as opposed to the childhood cerebral form which presents between 4-8yrs with cognitive dysfunction and progressive neurological deterioration. 8% of males present with adrenal insufficiency at any age and are at risk for X-ALD/AMN for life. Up to 50% of female heterozygotes may manifest an AMN-like syndrome. Adrenal insufficiency affects 80% of the childhood cerebral form, 50% of AMN and 1% of heterozygous women. Diagnosis is by plasma VLCFA assay, especially C26:0 and C24:0. Lorenzo's oil (a combination of mono-unsaturated fatty acids erucic acid and oleic acid, which blocks the endogenous synthesis of VLCFA) in asymptomatic cases may reduce the risk for neurological manifestations in cerebral X-ALD and slow the progression of AMN, but offers little once neurological impairment has set in (suspected to be due to failure of active ingredient to enter nervous system in significant quantity). Hematopoietic cell transplantation may be offered to boys with early cerebral involvement, and gene therapy is being studied.
Conclusion: Adrenoleukodystrophy, while only affecting between 1 in 17 to 21,000 males, is responsible for up to 50% of cases of Addison's disease in boys and young men. Once X-ALD is diagnosed, patients should be evaluated for adrenal insufficiency on an annual basis with serum ACTH and ACTH stimulation test.

Abstract #113

PHEOCHROMOCYTOMA PRESENTING AS ACUTE DECOMPENSATED HEART FAILURE

Christopher Mulla, MD, Paul Marik

Objective: Pheochromocytomas can have varied clinical manifestations, ranging from asymptomatic to hypertension with headaches and palpitations and very rarely to acute decompensated heart failure (ADHF) with multisystem organ failure.
Case Presentation: A 26-year-old woman was seen in the ER for chest pain. She was found to be hypertensive 179/121 mmHg and was diagnosed with a urinary tract infection. She was discharged with an antibiotic and thiazide diuretic. She returned 2 days later with epigastric pain, nausea, emesis and myalgias. On examination she remained hypertensive to 169/105 mmHg and tachycardic 125 bpm. She was given 1.5 L of fluid for presumed pre-renal azotemia and soon developed respiratory distress requiring intubation. A chest film demonstrated pulmonary edema. She was transferred to the ICU and she progressed to multisystem-organ failure. An echocardiogram established global left ventricular hypokinesis with a left-ventricular ejection fraction <10%. She was cautiously rehydrated with IV fluids and transiently treated with pressors followed by a gradual recovery. Her workup included right heart catheterization and biopsy for possible acute viral myocarditis that was negative. Her ejection fraction improved to 15%, she regained kidney function, was extubated, and eventually discharged home on a non-selective beta blockade with planned follow up at the CHF clinic. She returned to the ER 4 days later with an elevated BP, nausea and abdominal pain. A contrast CT scan of her abdomen revealed pancreatitis and an adrenal mass. During her treatment for pancreatitis she began to have paroxysms of headaches, nausea, emesis and abdominal pain with corresponding severe hypertension and tachycardia. Her urinary and serum catecholamine levels were elevated. She was rehydrated, treated with an alpha and beta adrenergic blocker followed by an adrenalectomy. Medical management prior to surgery was associated with improvement in left-ventricular ejection fraction to 55%.
Discussion: Pheochromocytomas are a rare growth of chromaffin cells which produce excess catecholamines. Failure to diagnose and treat this condition can lead to ADHF either due to stress induced cardiomyopathy or due to abrupt withdrawal of catecholamine signaling in a patient with desensitized adrenoreceptors and a reduced circulatory volume. Chronic catecholamine exposure and hypertension leads to excessive glomerular filtration and dehydration; therefore treatment includes fluid hydration. While surgical removal of offending tumor is definitive therapy, patients must first be stabilized with alpha and beta adrenergic blockade.
Conclusion: This case demonstrates that pheochromocytoma-induced ADHF can be reversed with medical therapy.

Abstract #110

AN ELUSIVE NEUROENDOCRINE TUMOR; A CHALLENGING DIAGNOSIS IN A PATIENT WITH CUSHING'S SYNDROME

Kwame Ntim, MBCHb, Daniel Wong, MD

Objective: Case report illustrating some of the challenges associated with localizing an ectopic ACTH dependent Cushing's syndrome.
Case Presentation: 40 y/o male was referred to our hospital for management of a perforated duodenal ulcer previously managed with an omental patch. He had a past history of Cushing's syndrome suspected to be secondary to an un-localized ectopic ACTH lesion. Past records indicated he had

been relatively well until a year ago when he was treated with fluticasone for allergic rhinitis. Two months later, he had cellulitis treated with antibiotics and subsequently with IM and oral steroids for metacarpophalangeal joint nodules. A couple of months after this, he is reported to have developed Cushingoid symptoms. His labs also showed hyperglycemia and hypokalemia. Endocrine work up demonstrated elevated ACTH 261pg/ml (7-50 pg/ml), 24 hr cortisol 1141mcg/24 (4-50 mcg/24) and 1 mg dexamethasone suppression test of 32.3mcg/dl (4-22 mcg/dl) with a normal pituitary MRI. Further work up at a tertiary center included a normal CT chest, abdomen and pelvis and CRH stimulation test. He was started on ketoconazole, spironolactone and insulin for diabetes. He then subsequently developed a perforated duodenal ulcer. Physical exam on admission was significant for moon facies, supraclavicular fat pads, buffalo hump, violaceous striae, central obesity and increased abdominal girth. Labs confirmed in our hospital were; 24 hour urine free cortisol of 1692mcg/24 (4-50 mcg/24), ACTH 200pg/ml (7.2-63 pg/ml). Repeat pituitary MRI and CT abdomen showed a 3mm suspicious pituitary mass and an ill defined mass in the uncinate process of the pancreas respectively. Patient underwent surgical removal of the mass. Pathology confirmed a neuroendocrine tumor. Post surgery ACTH level was 10pg/ml (7.2-63 pg/ml) though the initial pathology did not stain for ACTH.

Discussion: The symptoms and signs of hypercortisolism are non-specific making the diagnosis challenging. If established that the cause is endogenous, the next step involves identifying the underlying pathophysiology. Endogenous causes can be divided into ACTH-dependent (namely Cushing's disease, ectopic ACTH or ectopic CRH) or an ACTH-independent hypercortisolism secondary to hyper functioning adrenals. Inferior petrosal sinus sampling is useful in differentiating pituitary ACTH production from ectopic ACTH. The alternative is a CRH stimulation test. CT scans, MRIs, Octreotide scintigraphy are helpful in localizing ectopic sites. Surgical removal is often the first line of treatment.

Conclusion: Knowledge of the physiology of the Hypothalamus-pituitary-adrenal axis is essential in the evaluation, diagnosis and management of hypercortisolism.

Abstract #115

ADRENAL VENOUS SAMPLING: AN UNUSUAL METHOD FOR INVESTIGATING BILATERAL ADRENAL MASSES

Adam Maghrabi, MD, Saba Faiz, MD,
Tipu Faiz Saleem, MD

Objective: Subclinical Cushing syndrome (SCCS) is the most frequent hormonal abnormality in adrenal incidentaloma. Bilateral adrenal masses account for 10 to 15 % of adrenal incidentalomas. We are presenting a case of bilateral adrenal masses with SCCS secondary to ACTH-Independent Macronodular Adrenal Hyperplasia (AIMAH). We are also describing Adrenal venous sampling (AVS), a new emerging technique, which helps identify the source of cortisol secretion in this setting.

Case Presentation: 51 year old lady, was evaluated for bilateral adrenal masses found incidentally on an abdominal MRI, with loss of signal on chemical shift method indicating lipid content of the masses. Laboratory data showed lack of suppression of cortisol in response to both low and high dose dexamethasone with undetectable base line ACTH, normal 24 hour urine free cortisol and normal mid night salivary cortisol, suggestive of SCCS. Both MRI and CT were unable to differentiate the hyperplastic versus adenomatous nature of the masses. AVS was performed and blood cortisol and epinephrine levels were obtained from both adrenal veins (AV) and peripheral vein (PV). Results showed an AV: PV cortisol ratio of 5.85 and 5.04 on right and left side respectively, without significant lateralization. AVS results, coupled with the MRI and CT scan findings, favor the diagnosis of bilateral AIMAH. Robotic left adrenalectomy was performed since the left adrenal mass was larger. Pathology favored the diagnosis of AIMAH. ACTH and cortisol levels will be monitored to ensure cure and surveillance for recurrence.

Discussion: In this case, anatomical configuration of the bilateral adrenal masses on MRI and CT were not typical of hyperplasia or bilateral adenomas. The dilemma was: which gland is hypersecreting cortisol. AVS proved to be a useful tool. Adequate catheterization of the AV is ensured if the epinephrine level difference between AV and PV is more than 100. An AV: PV cortisol ratio of >4.1 may mean autonomous cortisol secretion, >6.5 points towards an adrenal adenoma, and between 4.1 and 6.5 (as seen in our case) may indicate hyperplasia. The combination of functional (AVS) and anatomical (CT) picture suggested AIMAH as the cause of the SCCS, which is a rare entity associated with either aberrant hormone receptors or genetic mutations.

Conclusion: AVS can be a useful tool to localize the source of the cortisol hypersecretion in ACTH-independent Cushing syndrome with bilateral adrenal masses. Furthermore, AVS can also help distinguish bilateral adrenal adenomas from AIMAH if the radiological findings are not clear.

Abstract #116

SEQUENTIAL ADRENAL GLAND HEMORRHAGE AND ACUTE MYELOID LEUKEMIA − IS THERE AN ASSOCIATION?

Preethi Sridhar, MD, Boby Theckedath, Janice Gilden, MD

Objective: To describe a patient on anticoagulation presenting with sequential adrenal gland hemorrhage, thrombocytopenia and anemia who was later diagnosed with Acute Myeloid Leukemia.
Case Presentation: A 63 year old gentleman with new onset atrial fibrillation was treated with enoxaparin and warfarin. Ten days later, he was admitted with left flank pain. His INR was 1.9 and platelet count 122,000/cubic mm. A CAT scan of the abdomen revealed an acute left adrenal hemorrhage. All anticoagulants were discontinued. He was discharged after 2 days of observation, since he was clinically and radiologically stable. One week later he was readmitted for fatigue, dizziness, hypotension, bradycardia, and orthostatic hypotension. A repeat abdominal CAT scan revealed a new right adrenal hemorrhage. Laboratory investigation showed a low serum AM cortisol of 0.93 mcg/dl, elevated ACTH at 57 pg/ml and a positive ACTH stimulation test (baseline cortisol = 5.62 mcg/dl, 30 minutes = 6.95 mcg/dl, 60 minutes = 8.07 mcg/dl). The patient was subsequently treated with hydrocortisone for adrenal insufficiency. The initial work up for persistent anemia and thrombocytopenia was negative, including repeatedly negative HIT antibodies. Heparin-induced thrombocytopenia was hence ruled out. Further work up with a bone marrow biopsy revealed Acute Myeloid Leukemia.
Discussion: Literature reports of the association between bilateral adrenal hemorrhage and acute leukemia are very rare. Bilateral adrenal hemorrhage has been increasingly reported as a complication of heparin-induced thrombocytopenia and anticoagulation therapy. In our patient, thrombocytopenia (not related to Heparin-Induced Thrombocytopenia and Thrombosis) and a sub-therapeutic INR are unlikely to have caused the bilateral adrenal hemorrhage.
Conclusion: Bilateral adrenal hemorrhage and adrenal insufficiency might occur in Acute Myeloid Leukemia and this association needs further investigation.

Abstract #117

SUPRAPHYSIOLOGIC RESPONSE TO ACTH STIMULATION TEST IN A 72 YEAR OLD MAN, UNMASKING SUBCLINICAL CUSHING'S SYNDROME

Marianna Antonopoulou, MD, Asya Perelstein, MD

Objective: To document a case of subclinical Cushing's syndrome in a 72 year old man with adrenal incidentalomas.
Methods: We present the diagnostic approach of a male patient with adrenal incidentalomas
Case Presentation: A 72 year old African American male with past medical history of hypertension, coronary artery disease (CAD), hyperlipidemia, spinal stenosis, and monoclonal gammopathy of uncertain significance (MGUS) had a CT scan of abdomen in 2009, showing right and left adrenal masses measuring 5x3.5 cm and 3.7x2.9 cm respectively. Patient underwent hormonal work up to rule out functioning adrenal tumors 3 times, including 24 hour urine cortisol and metanephrines, serum aldosterone, all of which were normal. The radiologist insisted that the CT findings are consistent with adrenal hyperplasia and since the patient was hypertensive, he underwent 250mcg ACTH stimulation test to rule out late onset congenital adrenal hyperplasia (CAH). The stimulation test revealed that 17-hydroxyprogesterone, 11-deoxycortisol increased to levels high enough to confirm CAH, but cortisol had exaggerated response too, making the diagnosis of CAH unlikely, where metabolism is shifted to precursors. Other causes of abnormal response to ACTH stimulation, including depression, medications, alcohol and obstructive sleep apnea were excluded. Subsequently patient underwent screening for Cushing's syndrome (CS) with overnight 1 mg dexamethasone and low dose (4 mg) suppression test. He did not suppress, making the diagnosis of subclinical CS (SCS) due to aberrant receptors likely. The full aberrant receptor work up could not be completed, because TRH and GnRH are not available. Also patient developed chest pain and underwent cardiac stent placement, so adrenalectomy was deferred. Patient is closely monitored for progression to overt CS.
Discussion: Our patient had been diagnosed in 2009 with MGUS; so far there are only 3 case reports of extramedullary plasmatocytoma arising from the adrenals. One was bilateral and one had functional abnormalities. Our differential diagnosis includes subclinical CS with aberrant receptors versus a functioning extramedullary plasmatocytoma. Unfortunately diagnosis remains uncertain without histologic examination.
Conclusion: Adrenal incidentalomas are often seen, as healthcare advances, more imaging studies are available

and the clinician is called to evaluate. As in our patient, there can still be a possibility of subclinical CS when using the screening tool of 24 urine cortisol. It is advisable to screen patients with the addition of overnight 1 mg dexamethasone suppression test, since even SCS can be a cause of increased morbidity and mortality.

Abstract #118

ADRENAL INSUFFICIENCY (AI) FOLLOWING INTRA-ARTICULAR STEROID INJECTION

Ava Port, MD, Stephanie Lee, MD, PhD

Case Presentation: An 83 year-old African American Female with a medical history significant for coronary artery disease, myocardial infarction, hypertension, dyslipidemia and osteoarthritis presented to the hospital with 3 weeks of progressive dizziness upon standing, fatigue, weakness, diminished appetite, and significant weight loss. Physical examination was remarkable for a fatigued appearance, temporal/quadriceps muscle wasting, and a marked drop in systolic blood pressure with postural change. Laboratory studies revealed mild hypokalemia and hypoalbuminemia, but were otherwise normal. She was admitted with a diagnosis of dehydration and given several liters of normal saline, with only modest improvement in orthostatic blood pressure. Further testing on hospital day 3 revealed a random morning Cortisol of 0.6 mcg/dL. A cosyntropin (ACTH) stimulation test was performed the next day at 8am, which showed low baseline Cortisol 0.7 mcg/dL and relatively low ACTH of 6 pg/dL, with blunted peak Cortisol response of 10 mcg/dL after 30 minutes, and 12.8 mcg/dL at 60 minutes. Additional labs included normal aldosterone/renin, TSH, prolactin, LH and FSH. Computed tomography imaging revealed normal pituitary and adrenal glands. An extensive medication review ruled out glucocorticoid exposure, with the exception of two steroid injections in the year prior to admission, including 20 mg Kenalog (triamcinolone acetate) into her knee joint about 10 months prior, and 80 mg Kenalog in her lumbar spine approximately 1 week before symptom onset. Her normal electrolytes, pituitary evaluation, imaging and ACTH suggested secondary AI, and the intra-articular steroid injection was felt to be the causative agent given lack of alternative explanation. She was started on Hydrocortisone 10 mg qAM + 5 mg qPM and discharged home with plan to slowly taper glucocorticoid dose. Symptoms resolved within 2 weeks of initiating hydrocortisone and weight returned to baseline at 2 month follow-up.

Discussion: Secondary AI is a well established side effect of systemic glucocorticoid therapy, but is rarely reported with intra-articular steroid use. AI following joint injection has been described in the literature as a transient phenomenon, with onset as early as 1 day post injection and typically resolving within 7-14 days, however, alterations in pituitary-adrenal axis function have been reported several months following injection.

Conclusion: This case highlights a rare but potentially life threatening complication of a widely used therapy, and illustrates the importance of considering a diagnosis of secondary adrenal insufficiency in a patient receiving intra-articular (non-systemic) glucocorticoid therapy.

Abstract #119

PARADOXICAL USE OF OF PRESSORS IN A PATIENT WITH EPINEPHRINE SECRETING PHEOCHROMOCYTOMA-DIAGNOSED ON ECHOCARDIOGRAM

Divyashree Varma, MBBS, Pratik Dalal

Objective: Pheochromocytomas are rare tumors of chromaffin cells, most commonly seen arising from the adrenal glands. Commonly, they secrete epinephrine, norepinephrine, or IL-6, which are responsible for certain features sometimes seen with pheochromocytomas. Pathognomonic symptoms include episodic hypertension, palpitations and diaphoresis; but pheochromocytomas can be notorious for presenting with subtle, atypical symptoms. We present a case that presented with typical features but also had some lesser known findings. We will also talk about what to avoid in suspected cases.

Case Presentation: A 59 years old gentleman with no past medical history presented with progressively worsening and more frequent "attacks" of shaking, palpitations, back pain, flushing and diaphoresis, followed by prostration and eventual resolution of the episode. Vitals signs showed a Blood pressure of 180/90mmhg & pulse of 150bpm. EKG showed Sinus Tachycardia, while an echo showed severe global hypokinesis with LVEF of 20%. Echo also showed a huge mass on the right adrenal. Labs showed creatinine of 1.9 and hemoglobin of 18.5 suggestive of severe volume depletion and a WBC count of 27000, mimicking infection, along with a troponin level of 0.34. The patient later became hypotensive with systolic blood pressure in the 70s and requiring pressor support. He also became confused, agitated and had to be intubated for airway protection. He became severely acidotic, and was later found to have multiple cerebral infarcts, some of which were assessed to be watershed. Epinephrine levels came out at 34649 with norepinephrine levels of 13028. BP gradually stabilized, patient was successfully extubated. Repeat echo showed normalization of EF. The patient was started on Doxazosin for alpha blockade prior to beta-blocker initiation and finally was successfully operated

with removal of the tumor. On admission, an abdominal examination, with manipulation of the mass seemed to have exacerbated an impending pheochromocytoma crisis.
Discussion: Fluctuations on blood pressure should raise suspicion for a pheochromocytoma. Alpha receptor stimulation at lower doses is thought to cause the hypertensive episodes, whereas beta stimulation at higher doses is thought to cause vasodilatation and hypotension, compounded by catecholamine induced cardiomyopathy. Hypotension resulted in watershed infarcts and neuropsychiatric symptoms.
Conclusion: We recommend avoiding repeated deep abdominal exams for fear of tumor manipulation and catecholamine surge.

Abstract #120

CASE REPORT OF MALIGNANT METASTATIC PHEOCHROMOCYTOMA

Kamran Rasul, MD, Robert Dubin,
Robert Richards, MD, Gabriel Uwaifo, MD

Objective: To report a case of malignant metastatic pheochromocytoma.
Case Presentation: A 22 year old male was admitted with uncontrolled hypertension. He had a history of right adrenalectomy at age 4 due to pheochromocytoma. He remained stable until age 12 when he developed hypertension again. Due to poor compliance with medications, his hypertension had remained uncontrolled. His mother and his maternal aunt also had history of surgery for pheochromocytoma. Plasma normetanephrine levels were elevated at 827 pg/mL. His calcitonin, intact PTH and calcium were normal. A CT scan of his abdomen showed two nodules in left adrenal measuring 1.7 X 1.5 cm with two nodules in periaortic chain, right external iliac lymphadenopathy, right sided bladder mass, and multiple nodules in seminal vesicles. An I-123 MIBG scan was performed and it showed intense localization in left adrenal, right side of urinary bladder and right iliac lymphadenopathy, consistent with pheochromocytoma. Patient was deemed not to be a candidate for surgical resection and FDA approval was obtained to treat patient with I-131 MIBG ablation therapy.
Discussion: Pheochromocytomas are chromaffin tumors arising in adrenal medulla. They are unilateral in 90% of cases. Bilateral pheochromocytomas are common in familial pheochromocytoma syndromes. Treatment is surgical resection. Chemotherapy has been used with a median survival of 3.3 years in a small study of 14 patients. Sunitinab was used in anecdotal case reports. Our patient has malignant recurrent metatastatic likely familial pheochromocytoma and I-131 MIBG ablation therapy was preferred over chemotherapy because of more clinical data with MIBG. I-131 MIBG ablation therapy is not currently approved by FDA for treatment of malignant metastatic pheochromocytoma. However several small case studies have shown improved survival with it (4.7 vs. 2.8 years in one study with 500 mCi). Dose ranges have been between 100 to 1690 mCi with more response seen at higher doses. Risk of hematological complications was 26% in one study with a dose of 600 mCi. High doses need stem cell harvest to be performed before ablation.
Conclusion: I-131 MIBG ablation therapy can be considered in a patient with metastatic malignant pheochromocytoma which is not amenable to surgery. However more data and clinical trials are needed.

Abstract #121

RAPID GROWING ANDROGEN SECRETING ADRENOCORTICAL MASS IN A PUERTO RICAN FEMALE

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Myriam Allende, Marielba Agosto, Meliza Martinez,
William Mendez, Carlos Alvarez

Objective: To describe a case of rapid growing androgen secreting adrenal tumor at unusual age in an adolescent female patient in Puerto Rico.
Case Presentation: Case of 20 years old female without past medical history evaluated in endocrinology clinics due to right adrenal incidental mass discovered in evaluation of right flank pain. Her menarche was at 12 years old and refers irregular menses, excessive body hair and acne. Physical examination showed normotensive normal weight female with body mass index of 22.8 kg/m2 and current positive findings: hirsutism at lip, chin, chest, back, abdomen, upper and lower extremities and mild deepening of the voice. No acanthosis nigricans, abdominal or axillary striaes, thin skin or skin bruising, clitoromegaly, frontal balding or galactorrhea. Abdominal ultrasound revealed right adrenal mass 4.9 x 4.4 x 4.8 cm. Total testosterone 171 ng/dL; Free testosterone 2.70 PG/ML; DHEA-S 1,000 UG/DL Prolactin 17.47 NG/ML; Cortisol 11.50 ug/dL.and negative urine collection for cathecolamines, metanephrines and vanillymandelic acid. Adrenal CTScan four months later reported large right suprarenal well-defined solid lipid poor mass measuring 8.0 x 7.8 x 7.9 cm that displaces the right kidney and causes mass effect on the overlying liver. On September 2011 she underwent right total adrenalectomy. Pathology report: Adrenal cortical neoplasm; tumor weight: 320 g; tumor size: 11 x 7 x 5 cm; negative for lymphovascular or sinusoidal invasion; negative for perineural invasion; absent necrosis, but extensive hemorrhage; the tumor capsule is rupture. Staging (T3NxMx).
Discussion: Adrenocortical carcinomas (ACCs) are rare,

aggressive tumors that may be functional or nonfunctional, and present as an abdominal mass or an incidental finding. The incidence is approximately one to two per million population per year. Can develop at any age, there is a bimodal age distribution, with disease peaks before the age of five and in the fourth to fifth decade of life. Androgen-secreting adrenal tumors are usually malignant. Less than 10 percent present with virilization alone, but the presence of virilization in a patient with an adrenal neoplasm suggests an ACC rather than an adenoma. In general, the level of aggressiveness and pace of disease progression are more rapid in adults than in children.

Conclusion: In spite nonfunctioning ACCs tended to progress more rapidly than functioning tumors and that the majority of adult patients with ACC have relatively advanced disease stage at initial presentation unusual clinical presentations and age onset of should be consider. The impact of clinical characteristics on outcome of ACC is controversial.

Abstract #122

MASSIVE ELEVATION OF LOW DENSITY LIPOPROTEIN IN A PATIENT WITH ADRENOCORTICAL CARCINOMA ON MITOTANE

Ha Nguyen, MD, Jane Mayrin, MD,
Marc Laufgraben, MD, MBA, FACE, FACP

Objective: Mitotane is commonly used to treat adrenocortical carcinoma (ACC), and has been associated with moderate changes in lipid levels. We present the case of a 62 year-old woman with dyslipidemia and ACC who was treated with mitotane and experienced a massive elevation in low density lipoprotein (LDL) cholesterol as well as significant elevations of high density lipoprotein (HDL) cholesterol and triglycerides.

Case Presentation: A 62 year-old post-menopausal female was found to have an 8.6 x 6.1 x 6.4 cm left adrenal mass. Her evaluation revealed elevated levels of androgens as well as estrogen. The patient underwent a left adrenalectomy with pathology demonstrating ACC. After surgery, she was started on Mitotane 1 gram twice a day (compounded in almond oil due to dysphagia to pills) and Hydrocortisone 15 mg AM, 5 mg PM. Prior to initiation of mitotane, her lipid profile while on simvastatin 20 mg daily demonstrated a total cholesterol 170 mg/dL (normal limit [nl] 125-200), LDL cholesterol 99 mg/dL (nl <130), HDL cholesterol 47 mg/dL (nl >46), and triglycerides 122 mg/dL (nl < 150). Following initiation of mitotane, her lipid profile (still on simvastatin 20 mg) demonstrated a massive increase of total cholesterol to 521 mg/dL and LDL cholesterol to >350 mg/dL, along with increases in HDL cholesterol to 72 mg/dL and triglycerides to 253 mg/dL. Her thyroid, liver and kidney function were normal. Adrenal androgens remained mildly elevated, but her estrogen level was appropriate for the post-menopausal state. A diagnosis of mitotane-induced hypercholesterolemia was made. Simvastatin was discontinued and rosuvastatin 10 mg daily was begun. At follow-up, her LDL had improved to 250mg/dl.

Discussion: Moderate elevations in LDL and HDL are not infrequent in patients taking mitotane, though massive elevations in LDL have been only rarely reported. A proposed mechanism is increased HMG-CoA reductase activity, with mitotane-induced inhibition of P450 enzymes responsible for oxysterol formation in the liver leading to decreased negative feedback on HMG-CoA reductase. HDL cholesterol level may be elevated due to the estrogen-like activity of mitotane itself. Almond oil has been shown to decrease LDL, and, therefore, the elevation in cholesterol is due to mitotane itself, not the oil it was compounded in.

Conclusion: Mitotane can cause massive elevation in LDL. Because Mitotane strongly induces CYP3A4, statins metabolized by other pathways, such as pravastatin or rosuvastatin, should be used to lower lipids.

Abstract #123

A CASE OF CUSHING'S SYNDROME PRESENTING WITH AORTIC DISSECTION

John Reyes-Castano, MD, Jennifer Swaner,
Shannon Sullivan, MD, PHD

Case Presentation: A 31yo African American man with a 5 year history of resistant hypertension was emergently transferred to our institution for treatment of a type A aortic dissection (TAAD). CT angiogram confirmed the TAAD and also demonstrated a 2cm right adrenal mass. After aortic dissection repair, the patient underwent complete biochemical evaluation of the adrenal incidentaloma. Biochemical testing was negative for pheochromocytoma and aldosteronism, however, 1mg dexamethasone suppression test and 24hr urine free cortisol were both consistent with CS. In fact, he had several phenotypic features consistent with CS, including central obesity, muscular atrophy of lower and upper extremities, moon facies, and prominent supraclavicular and dorsocervical fat pads. The patient was medically treated for hypertension and hyperglycemia. He received prophylactic treatment for opportunistic infections and for venous thromboembolism due to the increased risk in CS. Four weeks after aortic aneurysm repair, the patient underwent laparoscopic right adrenalectomy. Histopathology confirmed adrenal cortical adenoma with myelolipomatous change. He was treated

with stress dose hydrocortisone in the perioperative period, then quickly tapered to physiologic replacement dose HC.

Discussion: Cushing's Syndrome (CS) has been identified as a risk factor for aortic aneurysm dissection; however, historically, the association of these two conditions is rare. To our knowledge, there have been 9 reported cases of artery aneurysms associated with CS in the literature, 8 of which were dissecting aortic aneurysms. Our patient presented with TAAD requiring emergent surgical repair and a prolonged hospital stay that included cardiac rehab followed by laparoscopic right adrenalectomy. The mechanisms that lead to dissecting aneurysm in patients with CS are not well understood. Chronic hypercortisolemia has been demonstrated to cause atherosclerosis, hypertension and dissecting aneurysm in experimental models. One hypothesis is that hypercortisolemia disrupts aortic smooth muscle cells, ultimately leading to aneurysm formation. In hamsters, cellular metaplastic transformation of smooth muscle cells into fibroblast-like cells has been shown in the media of the aorta adjacent to cortisone-induced dissecting aneurysms.

Conclusion: Given the rare yet important association between CS and aortic aneurysm formation, CS should be considered in patients presenting with aortic aneurysm, with a biochemical evaluation for hypercortisolism in those with suspicious phenotypic features.

Abstract #124

ECTOPIC ACTH SYNDROME

Candice Rose, MD, Aimee Eidson, Rajib Bhattacharya

Case Presentation: A 38 year old woman presented to the ER with the complaint of shortness of breath. A CT scan showed a large mass in the lung with evidence of metastases. She underwent bronchoscopy with biopsy, which revealed small cell carcinoma. Two years later after multiple chemotherapeutic regimens, she developed rapid onset 50 pound weight gain, edema, and muscle cramping. Labs revealed random cortisol level of 82.9 mcg/dl (5-20) ACTH 318 pg/ml (10-60), potassium 2.6 mmol/L (3.5-5.1), CO2 36 mmol/L (21-30), and alkaline phosphatase of 388 U/L (25-110). She was later admitted to the hospital for hypokalemia and endocrinology was consulted for hypercortisolism. On physical exam she was noted to have moon facies, facial acneiform rash, increased abdominal girth, purple striae, muscle strength 3/5 in shoulders and thighs, and anasarca. An EKG revealed mild ST depression and low amplitude T-waves. A previous CT scan showed unremarkable adrenals. Spironolactone and potassium supplementation were increased. Increase in dose of spironolactone was limited by elevated liver enzymes. Prior to discharge, metyrapone was initiated at 250 mg four times daily. One month later random cortisol level had decreased to 40 mcg/dl. Shortly thereafter, she entered hospice care.

Discussion: Approximately 15% of cases of Cushings Syndrome are caused by non-pituitary tumors secreting ACTH, known as Ectopic ACTH Syndrome. Half of these cases are caused by small cell lung carcinoma. Serum levels of ACTH and cortisol can be very high, and time from symptom onset to presentation is usually less than 3 months. Primary treatment for ectopic ACTH production is surgical removal of the tumor. If the tumor is unresectable, chemotherapy and radiation may be of some benefit. The second step in treatment is the use of adrenal enzyme inhibitors such as ketoconazole and metyrapone. They inhibit the conversion of 11-deoxycortisol to cortisol, with ketoconazole also inhibiting the first step in cortisol synthesis. The last treatment option includes either surgical or medical adrenalectomy with mitotane. Our patient had end stage lung cancer with limited options. Metyrapone seemed to be effective but her overall prognosis was poor.

Conclusion: A 40 year old woman with history of small cell carcinoma presents with symptoms of excess cortisol due to ectopic ACTH production. Her tumors were unresectable and therefore therapy with adrenal enzyme inhibitors was initiated.

Abstract #125

INHALED CORTICOSTEROIDS AND CLINICALLY SIGNIFICANT HYPOTHALAMIC-PITUITARY-ADRENAL AXIS SUPPRESSION

Deepika Nallala, MBBS, MD, Chaitanya Mamillapalli, Michael Jakoby, IV, MD

Objective: Inhaled corticosteroids (ICS) are commonly used for management of chronic obstructive pulmonary disease (COPD). CS are less likely to cause systemic side effects than oral corticosteroids because 80 to 90 percent of a dose is absorbed through the upper gastrointestinal tract and inactivated by first pass hepatic metabolism. Although there is variability between ICS preparations and different inter-individual susceptibilities, high doses of ICS may lead to biochemical evidence of hypothalamic-pituitary-adrenal (HPA) axis suppression. Reports of clinical secondary adrenal insufficiency, however, are rare. We present a case of clinically apparent secondary adrenal insufficiency in a patient receiving high dose ICS for management of COPD.

Case Presentation: An 86-year-old woman was admitted to hospital for near syncope. Several weeks prior to admission, she experienced dizziness and weakness after standing that resolved in a seated or supine position. The

patient denied falls, chest pain, dyspnea, palpitations, or weight loss. She reported full compliance with her medical regimen which included olmesartan, nifedipine, furosemide, and isosorbide mononitrate. Inhaled fluticasone (1,000 mcg daily) had been prescribed for two years to manage COPD. The patient denied treatment with oral or articular corticosteroids. Examination was unremarkable with the exception of orthostatic blood pressure and heart rate changes. Orthostasis resolved after blood pressure medications were held and isotonic fluid was administered. Two consecutive 8:00 AM cortisol levels were unequivocally low (0.9 mcg/dL), and simultaneous ACTH measured on the second morning was 10 pg/mL (ref range 0-64). Thirty minute stimulated cortisol during a 250 mcg Cosyntropin stimulation test was 7 mcg/dL. FSH was unequivocally elevated, and simultaneous measurements of TSH and free T4 were unremarkable. Secondary adrenal insufficiency due to high dose fluticasone was diagnosed. The patient remained normotensive off anti-hypertensive agents, and she was given a prescription for prednisone to take during acute illnesses.

Discussion: Inhaled fluticasone has minimal effects on the HPA axis when prescribed at doses less than 500 mcg daily. Though data are limited, there is an apparent 5-10% risk of measurable HPA axis suppression at cumulative daily doses exceeding 500 mcg. Most cases appear to be subclinical, and the rate of clinical secondary adrenal insufficiency as unmasked by anti-hypertensive therapy in our patient is unknown.

Conclusion: The HPA axis should be evaluated promptly in patients receiving high dose ICS and a clinical presentation suspicious for secondary adrenal insufficiency.

Abstract #126

FALSELY ELEVATED URINARY DOPAMINE LEVELS DURING WORK-UP FOR PHEOCHROMOCYTOMA IN A PATIENT TREATED WITH LEVODOPA-CARBIDOPA

Nicoleta Ionica, MD, Sruti Chandrasekaran, MBBS, Elizabeth Streeten

Objective: Levodopa-carbidopa (L-dopa) can result in falsely positive testing for pheochromocytoma. L-dopa is converted by peripheral tissues to noradrenaline, adrenaline and their metabolites. We present a patient with Parkinson's disease treated with L-dopa who had falsely elevated urinary dopamine (DA) levels during evaluation for pheochromocytoma.

Case Presentation: A 69 year old African American man with a history of neurofibromatosis type 1 (NF-1) was found to have a 3 cm right adrenal nodule found on CT scan done to evaluate abdominal pain. Laboratory studies showed plasma free metanephrines of 539 pg/ml (0-205pg/ml) and normetanephrines of 449 pg/ml (0-148 pg/ml), 24 hour urine epinephrine 6mcg/24hr (0-20), norepinephrine 18 mcg/24 hrs (15-100). An abdominal MRI showed a 2.6 x 1.8cm nodule that was bright on T2 images, suspicious for pheochromocytoma. A laparoscopic right adrenalectomy was performed and pathology confirmed a benign pheochromocytoma. Six months after surgery, he was readmitted with abdominal pain and abdominal CT and MRI scan showed multiple small bowel masses. Plasma and urine metanephrines were normal but 24 hr urine DA was markedly elevated, 1994 and 1361pg/ml (normal 0 - 30pg/ml) on two separate collections. He had recently been diagnosed with Parkinson's disease, for which he was started on L-dopa, 25/100mg three times a day. Because of concern for a malignant DA secreting pheochromocytoma, an octreotide scan was done, which was negative. His L-dopa was stopped for a week and the repeat 24 hour urine DA was undetectable.

Discussion: L-dopa use for Parkinson's disease in our case resulted in markedly elevated urine DA levels, raising suspicion for persistent pheochromocytoma. Recent studies in the Parkinson's disease literature have shown significant increases in urinary DA levels up to 100 times normal in patients treated with L-dopa, with severity of elevation related to drug dose. L-dopa treatment can also falsely elevate urinary free adrenaline and metaadrenalines. Endocrinologists should be aware of the potentially dramatic effect of L-dopa on urinary catecholamines as a confounder in the evaluation of pheochromocytoma.

Conclusion: Our case illustrates the importance assessing urinary catecholamines off L-dopa in the evaluation of pheochromocytoma.

Abstract #127

AN UNUSUAL CASE OF AUTOIMMUNE POLYGLANDULAR SYNDROME (APS) II PRESENTING WITH NEUROLOGICAL SYMPTOMS AND HYPOGLYCEMIA DUE TO ADRENAL INSUFFICIENCY

Carla Romero MD, Nina Needleman, Agustin Busta

Case Presentation: A 24yo man, with alopecia at age 8, and DM1 at age 12, treated with an insulin pump, reported right hemiparesis and hemiparesthesia occurring upon waking up which resolved after eating. After the third episode, he turned off his insulin pump and went to the emergency department. On exam, he was found to have new skin hyperpigmentation. He was started on subcutaneous insulin. A 0.25µg cosyntropin test was

performed; cortisol was 4 µg/dl at 0, 30 and 60 minutes; baseline ACTH was 424 pg/ml. The test was repeated in the morning: cortisol was 6, 5, and 5 µg/dl at 0, 30 and 60 minutes respectively; and baseline ACTH of 2110 pg/ml. TSH and free thyroxine were normal. However, anti-TPO antibody was 115 IU/ml (nl 0 - 34.9 IU/ml). He was started on corticosteroid and mineralocorticoid replacement therapy, and was discharged with multiple subcutaneous insulin injections.

Discussion: Thomas Addison first described a syndrome including weakness, fatigue, anorexia, abdominal pain, orthostatic hypotension, salt craving, and hyperpigmentation. It has been reportedly caused by hereditary disorders, drugs, meningitis, TB adrenalitis, critical illness, and liver disease. In the Western world 80% of cases are autoimmune. APS has been rarely described, and most case reports show unusual presentations. There are three types; type I consists of 2 out of 3 of candidiasis, hypoparathyroidism, and autoimmune adrenal insufficiency; type II consists of adrenal insufficiency and either thyroid disease or DM1 associated with other disorders such as vitiligo and alopecia; and type III consists of autoimmune thyroiditis and other autoimmune disorders other than Addison's. Our patient was diagnosed with Addison's disease, and had previously been diagnosed with alopecia and DM1. He also had elevated anti-TPO antibodies, indicating Hashimoto's thyroiditis. Although currently euthyroid, he should be monitored regularly for the development of hypothyroidism. Our patient's presentation was unusual in that he presented with hemiparesis in the setting of hypoglycemia as a manifestation of hypocortisolism.

Conclusion: Neurologic deficits, such as hemiparesis, can be a presenting manifestation of low levels of glucose. In patients with autoimmune DM1 at a stable insulin dose and no recent lifestyle changes, other etiologies for hypoglycemia should be investigated, including adrenal insufficiency (Addison's disease).

Abstract #128

TWO CASES OF ADRENOCORTICAL CARCINOMA: DIAGNOSTIC AND TREATMENT CHALLENGES.

Anna Marino, MD, Philip Kern

Objective: Adrenocortical carcinoma is a rare tumor, accounting for 0.05 to 2% of all cancers. The prognosis is poor with 5-year survival rate in adults of 16-47%. Female patients are more likely to have an associated endocrine syndrome. We present 2 cases of adrenal carcinoma with Cushing's syndrome and discuss the challenges of evaluation and approach to the management depending on the stage.

Case Presentation: 1st case: 20-year-old female with quickly developing Cushing's syndrome during the previous year. She was found to have both clinical and biochemical findings of hypercortisolism, hyperandrogenism and unsuppressed cortisol and ACTH levels, even after high dose 48 hours dexamethasone suppression test. CRH level, chromogranin A and urine fractionated metanephrines were normal. CT scan revealed a left 7.5 x 4.6 cm adrenal mass with no additional findings to suggest metastatic disease. The patient underwent adrenalectomy. The pathology revealed adrenocortical carcinoma with invasion into the surrounding adipose tissue but negative staining for ACTH. Postop Cushing's symptoms have improved. The patient underwent adjuvant radiation therapy, and therapy with mitotane is considered. 2nd case: 48-year-old female diagnosed with adrenal carcinoma on biopsy of a liver metastasis. The patient had the stigmata of Cushing syndrome and hypercortisolism with suppressed ACTH on biochemical evaluation. Chromogranin A level was elevated. Abdominal and chest CT revealed 11.5 x 6 cm tumor in right adrenal gland with lung and liver metastases. The patient was not a candidate for surgery and started mitotane, which resulted in significant improvement in Cushing's symptoms. Unfortunately the imaging showed progression of the disease, and she plans to enroll in a phase II clinical trial.

Discussion: Both cases have features representing the challenges of diagnosis and treatment of adrenal cancer. The first case had a classical presentation of rapidly progressive Cushing's, but her biochemical profile was consistent with ACTH-dependent disease. Our hypothesis to explain this phenomenon is the loss of negative feedback from the tumor or the partial co-secretion of ACTH by the adrenal mass. The second case has classical biochemical features of adrenocortical cancer, and the main challenge is the management of non-operable disease. The application of novel strategies and participation in a clinical trial is the approach we chose in the second case.

Conclusion: Management of patients with adrenocortical cancer requires a multidisciplinary approach, and should involve surgeons, endocrinologists, oncologists and radiation oncologists. The prospective randomized trials are needed to provide future evidence-based recommendations.

Abstract #129

DISSEMINATED HISTOPLASMOSIS WITH BILATERAL ADRENAL ENLARGEMENT IN AN IMMUNOCOMPETENT HOST

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Objective: Adrenal Histoplasmosis with adrenal insufficiency can be fatal if undiagnosed and untreated.
Case Presentation: 76 year old male from Iowa with a medical history of amiodarone-induced pulmonary fibrosis, atrial fibrillation, heart failure and chronic kidney disease was admitted with complaints of black tarry stools, 30 pound weight loss and breathlessness. Examination was unremarkable except for bibasilar crackles. Pertinent labs: Hemoglobin 7 g/dl, white cell count 36,400/mm3, BUN 87 mg/dl, creatinine 2.5 mg/dl. Iron studies revealed iron deficiency anemia. Endoscopy showed esophagitis and gastritis. Patient refused colonoscopy. Hemoglobin and platelets started to decline. He required multiple blood transfusions. Dexamethasone was started for possible idiopathic thromocytopenic purpura versus drug induced (amiodarone) thrombocytopenia. He deteriorated clinically with anion gap metabolic acidosis (high lactate). Blood and urine cultures were obtained. CT scan showed calcified mediastinal lymph nodes, colonic diverticulae and bilateral adrenal gland enlargement with hemorrhage (hormonal work up was negative for what were thought to be bilateral adrenal incidentalomas a few months previously). Adrenal insufficiency was suspected during this admission and fludrocortisone was added. He sustained a fatal cardiac arrest. On autopsy, right and left adrenal glands weighed 56 and 66 grams respectively and were hemorrhagic. (Normal weight 4 to 6 grams). Histological staining of the adrenal glands, lungs, spleen and kidneys revealed histoplasmosis. Blood cultures grew histoplasma establishing the diagnosis of disseminated histoplamosis.
Discussion: Differential diagnosis of bilateral adrenal enlargement includes primary malignancies, metastasis, lymphoma, adrenal hemorrhage, sarcoidosis and infections. The initial work up to look for hypo- or hyperfunctioning glands should be performed even in an asymptomatic individual. CT findings of adrenal histoplasmosis are non-specific and include preservation of normal outlines, peripheral enhancement, central hypodense areas and calcifications. Percutaneous imaging-guided biopsy is considered as the most sensitive and highly specific test in evaluation of adrenal masses after biochemical evaluation and exclusion of pheochromocytoma. Adrenal histoplasmosis is treatable if antifungals are initiated in a timely fashion. Glucocorticoids and fludrocortsone must be added when adrenal insufficiency is present.
Conclusion: Disseminated histoplasmosis is rare in an immunocompetent host. Adrenal involvement in disseminated infection is seen in upto 80% and is usually unilateral. Bilateral adrenal gland involvement and hypoadrenalism are rare.

Abstract #130

ADRENAL MASS INCIDENTALLY DISCOVERED DURING ENDOSCOPIC ULTRASOUND (EUS)

Nirali Shah, M.D, Harmeet Narula

Objective: Adrenal nodules are often incidentally discovered during CT, PET scans and other abdominal imaging. We describe a case where an incidental adrenal mass was seen on Endoscopic Ultrasound (EUS) and discuss the appropriate use of this new modality for evaluation of adrenal masses.
Case Presentation: A 70-year-old man with no known malignancy was found to have an incidental adrenal nodule during EUS for evaluation of gallbladder polyps. A CT scan of the abdomen with contrast demonstrated a 3.5 x 3.3 cm left adrenal heterogeneous solid mass with 'no discernible or macroscopic fat content'. The gastroenterologist performed an EUS guided FNA revealing 'adrenocortical neoplasm', ruling out metastasis. He was then referred to the endocrinology clinic for further evaluation. Functional evaluation revealed it to be a non-functioning mass. Repeat noncontrast dedicated CT scan of the adrenals at 6 and 12 months, revealed stable left sided adrenal nodule with a benign imaging phenotype, compatible with an adrenal adenoma.
Discussion: Adrenal masses are common and often discovered during CT, MRI, PET or other abdominalimaging. EUS is a new imaging modality, increasingly being used by gastroenterologists for evaluation of various abdominal masses, including pancreatic neoplasms. EUS guided FNA has been used for evaluation of suspicious left adrenal masses, seen in patients with known primary malignancies (right adrenal is technically very difficult to approach by EUS). It is important that endocrinologists educate their gastroenterology colleagues to get a functional evaluation (to rule out a pheochromocytoma) before a biopsy of the adrenal mass to prevent a life-threatening crisis during the procedure. Also, a dedicated noncontrast CT or chemical shift MRI should be performed before doing invasive procedures like FNA of a mass.
Conclusion: Endocrinologists should be aware of this new modality, so they may educate their gastroenterology, oncology and hospitalist colleagues, for appropriate use of this new technology.

Abstract #131

ELEVATED CORTISOL LEVEL WITHOUT STIGMATA OF CUSHING'S SYNDROME

Bhavini Bhavsar, MBBS, M.D, Ved Gossain, MD, Dana Fletcher, DO

Objective: To report a case of markedly elevated cortisol levels in a patient without clinical stigmata of Cushing's syndrome.
Case Presentation: A 41-year-old Caucasian woman with history of hypothyroidism, hyperlipidemia, Polycystic Ovarian Syndrome (PCOS) and depression, was referred to us in December, 2008 for elevated 24-hour urine free cortisol level of 317 mg/24 hour (reference range: 3.5-45). She reported decreased libido, hair loss, irregular periods, anxiety and excessive facial hair. History was otherwise unremarkable except that she consumed a glass of wine everyday. Physical examination revealed BP122/80 mmHg, pulse rate 60/minute and BMI 21. A pea-sized thyroid nodule and increased hair on chin were noted. The remainder of the physical examination was normal. There were no clinical stigmata of Cushing's syndrome. A repeat 24-hour urinary cortisol was elevated at 320 mg/24 hour. After1 mg overnight dexamethasone, and after 2 mg dexamethasone for 2 days, serum cortisol levels were 20.6 and 12.1 mg/dL respectively. A year later she was seen by another endocrinologist and underwent inferior petrosal sinus (IPS) sampling. Baseline ACTH levels in left IPS, right IPS and peripheral vein were 285 pg/mL, 45 pg/mL and 18 pg/mL respectively. Five minutes after intravenous CRH, these levels were 865 pg/mL, 279 pg/mL and 27 pg/mL respectively. MRI of pituitary gland and CT scan of abdomen were normal. As recently as January 2012, no clinical stigmata of Cushing's syndrome were present.
Discussion: Major signs of Cushing's syndrome include centripetal obesity, supraclavicular fat pads, facial plethora, purplish striae and proximal myopathy. Although it is well known that Cushing's syndrome manifestations are slow to develop, our patient did not have any of these features at time of initial consultation and at 3 years of follow-up. She did have chronic facial hair and irregular periods, which can be attributed to history of PCOS. No causes for Pseudo Cushing's were present. Elevated cortisol levels, lack of suppression with dexamethasone and elevated ACTH levels in IPS compared to periphery in the absence of clinical features suggest Cortisol resistance syndrome, which results from glucocorticoid receptor gene mutations resulting in impaired glucocorticoid signal transmission and altered tissue sensitivity. It can be sporadic or familial. In familial cases, it is transmitted in autosomal dominant or recessive patterns depending on the type of gene mutations.
Conclusion: Cortisol resistance syndrome should be considered in patients with biochemical evidence of Cushing's syndrome but lack of clinical stigmata.

Abstract #132

CUSHING'S SYNDROME SECONDARY TO ADRENAL CARCINOMA IN 13 YEAR-OLD GIRL

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Objective: Background: Cushing's carcinoma is a rare disorder that has not been previously documented in a Nigerian. We report a case of adrenal carcinoma in a 13 year old girl - a rare occurrence in this age group.
Case Presentation: Case report: A 13 year old girl presented at the medical emergency because of worsening of her ill-health. She complained of 3month history of easy bruising, generalized hypo-pigmentation, cough, productive of yellowish sputum, chest pains and back pains. The patient had previously been seen by the gynecologist on account of congenital adrenal hyperplasia until she developed anasarca and proximal myopathy. On examination, patient is plethoric, dyspneoic, had Cushingoid phenotype, and generalized hypo-pigmentation and pitting pedal oedema. Pulse rate 124 beats/min, B.P 180/110mmHg ,R.R=40 c/min. Detailed examination revealed that she had tender hepato-splenomegaly and coarse crepitations in the right middle and lower zones of the lungs. Investigation showed PCV 52% electrolytes showed hypokalaemia and a RBS of 340mg. Screening test for Cushing syndrome showed elevated 24hour urinary cortisol >75.0 microgram/dl (20-70 microgram/dl). Further investigations done revealed Normal ACTH, elevated DHEAS, abdominal CT scan showed adrenal tumour with distant metastasis. Histopathology report confirmed an invasive adrenal carcinoma. She developed diabetes and hypertension and was on insulin and blood pressure lowering agents. She however did poorly whilst on admission and she deteriorated rapidly and subsequently died after 6 weeks on admission.
Discussion: Discussion: Adrenal Carcinoma is an uncommon cause of Cushing's syndrome. The diagnosis of cortisol - producing adrenal tumour in a female is suggested by clinical features of virilization and defeminization and biochemical parameters of elevated free urinary cortisol level, urinary 17-keto steroids DHEA sulfate.
Conclusion: Conclusion: All forms of Cushing 's syndrome should be properly investigated bearing in mind malignant causes to allow for early intervention.

Abstract #133

LIDDLE'S SYNDROME PRESENTING WITH HYPERTENSIVE ENCEPHALOPATHY

Sunil Kota, MD, Siva Kota, Svs Krishna, Lalit Meher, Kirtikumar Modi

Objective: Liddle's syndrome is a rare cause of secondary hypertension. We hereby wish to report a rare presentation with hypertensive encephalopathy in Liddle's syndrome.
Methods: Clinical and laboratory data are reported on a 35 year old lady, a known diabetic and hypertensive presenting with hypertensive encephalopathy.
Case Presentation: A 35 years old lady presented with headache, nausea, vomiting and 1 bout of generalized tonic clonic convulsion lasting for 2 minutes, without any accompanying focal neurologic deficits. She denied having any episodes of paroxysmal headache, palpitation and profuse sweating or any weakness of muscles, polyuria, swelling of body. For last 4 years despite receiving various combinations of calcium channel blocker, beta blocker, ACE inhibitor, angiotensin receptor blocker, alpha blocker and spironolactone and centrally acting sympathomimetics, her blood pressure was not controlled. There was no family history of hypertension. On examination her BP was 210/ 130 mm hg position without significant asymmetry or postural variation with palpable pulses in all extremities. There was no edema. Patient was confused without any focal neurologic signs, but had evidence of hypertensive retinopathy (retinal hemorrhages, exudates and papilledema). Rest of the systematic examination including CVS was normal. On investigation, she had hypokalemia (2.2 mmol/L) with normal serum sodium (137 mmol/L), high 24hr urine potassium (40 meq/L) and metabolic alkalosis (pH-7.47, HCO3-28 meq/L). Her urine examination, renal profile, serum cortisol and ACTH, 24 hour urine fractionated metanephrines, vanillyl mandelic acid and plasma metanephrines were normal. Her serum aldosterone level was low (30 pg/mL, normal 40-480 pg/ml) with low plasma renin activity (0.04 ng/ ml/ hr). CT scan of the brain was normal. Hypertensive emergency was treated with sodium nitroprusside infusion, resulting in gradual improvement of BP and patient's neurological status over 2 days. Patient was prescribed amiloride with oral potassium supplementation. 3 months later BP was stabilized at 130/ 80 mm hg with serum potassium at 3.6 mmol/L
Discussion: The combined picture of hypokalemic alkalosis with hyperkaliuria pointed to a state of mineralocorticoid excess, which could be excluded by virtue of low aldosterone levels. Hyporeninemic hypoaldosteronism in the backdrop of nonresponsiveness to spironolactone lead us to a provisional diagnosis of Liddle's syndrome. Response to amiloride with resultant normalization of blood pressure confirmed the diagnosis.
Conclusion: Liddle's syndrome presenting with hypertensive encephalopathy is a rare occurrence. Early diagnosis followed by appropriate treatment with amiloride is need of the hour.

Abstract #134

ATYPICAL PRESENTATION OF RECURRENT ADRENAL CELL CARCINOMA

Richard Hilliard, DO, Kimberly Rieniets, DO, Tyler Jenkins

Case Presentation: Aldosterone producing adrenocortical carcinoma (APAC) is a very rare malignancy, usually presenting in a clandestine manner with hypertension and potassium wasting. Our patient with APAC presented in June 2011 for chest pain. He was found to have mild hypertension, severe potassium wasting and metabolic alkalosis. The patient eventually revealed a long history of adrenal disease to include partial left adrenalectomy in 2004. During work up, Conn's syndrome with severe potassium wasting was diagnosed; Progressive hyperaldosteronism was noted with serum aldosterone levels ranging from 37 to 172 ng/dL. Adrenal CT imaging revealed a 2.2 x 2.4 x 2.4cm hyperdense nodule of his left adrenal gland and multiple other non-calcified and calcified nodules near the adrenal mass. Exploratory laparotomy with excisional biopsy of a mass and several diaphragmatic nodules was performed. Histology of a mass excised from the omentum revealed 6 mitotic figures per HPF and a paucity of vacuolated cells. Immunohistochemical stains had strong positivity for Calretinin, Vimentin and MART-1/melan A. Inhibin and Synaptophysin were focally blush positive. Pancytokeratin and Chromogranin were negative. Ki-67 proliferative index approached 15-20%. Modified Weiss criteria was >3, consistent with ACC. Clinical, biochemical, and histologic findings support APAC diagnosis.
Discussion: Discovery of adrenal tumors is increasing as imaging technologies advance. Adrenal masses may be benign or malignant adrenal tumors, metastatic tumors, infection, adrenal hemorrhage or adrenal hyperplasia. Roughly 50% of adrenal masses are benign adenomas. Adrenocortical Carcinoma (ACC) is a rare aggressive disease with an estimated incidence of one case per two million people annually with a female preponderance of 2.5:1. ACC's can present as non-functioning tumors or hyperfunctioning tumors. Cushing's syndrome with virilization is the most common presentation of hyperfunctioning ACC. APAC

account for 6% and Estrogen-producing tumors account for 2% of hyperfunctioning tumors, respectively. 10% of APAC are metastatic at diagnosis and 50% are metastatic by follow up. Therefore, it is essential to diagnose and resect this tumor early and to implement continuous surveillance since this malignancy has a high rate of recurrence and death.

Conclusion: We present a 62 YO male patient with a rare metastatic Aldosterone Producing Adrenal Carcinoma (APAC).

Abstract #135

RETROCARDIAC PARAGANGLIOMA: A CASE REPORT

Angela Boldo, MD, Sushela Chaidarun, Laura Trask, MD, Kathleen Belbruno

Case Presentation: We present a 24 year old female with 5 month history of hot flashes, sweating, episodic headache associated with nausea and new onset of hypertension. Her blood pressure was as high as 200/110mmHg and associated with pre-syncope symptoms but no documented hypotension or orthostasis. The patient has a history asthma and was on cyclobenzaprine and oral contraceptive pills at the time of presentation. She has no contributory family. Initial investigation showed a high 24 hour urinary normetanephrines 2837 mcg (40-412mcg) with normal epinephrine 7 mcg (2-24mcg) and high norepinephrine 779 mcg (15-100mcg). Cyclobenzaprine was stopped and the 24 hour urine showed similar results. The patient underwent a 123 I-MIBG scan with SPECT and abdominal/pelvis CT scan that were negative. For further investigation, patient underwent a Octreoscan that showed a focally intense activity projecting over the left posterior mediastinal region consistent with paraganglioma. This was confirmed with a cardiac MRI with gadolinium that showed a 3.1 x 2.1 x 2.1 cm homogenous lesion hyperintense on T2-weighted imaging. The lesion was retrocardiac with a course along the lateral wall of the aorta. She was started on phenoxybenzamine and subsequently metoprolol with good blood pressure control. Patient underwent surgery and the retrocardiac paraglanglioma was removed with complete resolution of her symptoms and hypertension.

Discussion: This case was unique for the rarity of a retrocardiac paraganglioma in a young patient with apparent sporadic disease. Genetic testing was negative for succinate dehydrogenase mutation/deletion and von Hippel-Lindau Disease. Even though her tumor was secreting high levels of norepinephrine, her MIBG scan was negative. Sensitivity is reported to be around 86% for 131I-MIBG for pheochromocytoma (higher with use of 123I-MIBG and SPECT), but only around 70% for paraganglioma and metastatic disease. False negatives may be seen with medications that interfere with MIBG uptake, small tumors, or large highly necrotic or dedifferentiated masses. On the other hand, octreotide scan has a complimentary role if the MIBG-scan is negative and sensitivity is around 70-80% for paraganglioma. FDG-PET has a sensitivity for pheo/paraganglioma of around 70% but as well as Octreoscan it has a role in MIBG-scan negative tumors.

Conclusion: In summary, this is a rare case of retrocardiac paraganglioma that was found to have a negative MIBG-scan and the patient's tumor was localized after an Octreotide scan. Therefore, Octreoscan, in this case, or possibly also FDG-PET can be very helpful to localize paragangliomas when abdominal morphological imaging (CT scan and MRI) and MIBG scan are negative.

Abstract #136

ROLE OF BILATERAL ADRENALECTOMY IN THE MANAGEMENT OF CUSHING'S SYNDROME DUE TO ECTOPIC ACTH

Kamalpreet Singh, MD, Deepika Reddy

Case Presentation: Cushing's disease and Cushing's syndrome due to ectopic ACTH productions are rare entities. Distinguishing between them can be challenging. MRI using pituitary protocol reveals a mass in roughly 60% of patients with Cushing's disease. The source of ectopic ACTH remains unidentified in approximately 10% of patients with Cushing's.

Discussion: 37 yr old male presented with uncontrolled Diabetes, hypertension, weight gain, and generalized fatigue. At presentation, physical findings showed a cushingoid appearance with moon facies, buffalo hump, supraclavicular fat pads, temporal fat, and violaceous stria. 24 hour urine cortisol was 1353.0 and plasma ACTH was184. Dexamethasone suppression test was consistent with non-suppressible cortisol levels. MRI of pituitary showed no discrete lesion. CT of chest/ thorax/ abd revealed a 6mm pulmonary nodule in left lobe and adrenal glands showed bilateral fullness without discrete nodule. CT of his chest at outside facility 5 months prior had reported the 6mm pulmonary nodule and he reported a positive smoking history. There was no interval increase in size of pulmonary nodule which would have been expected in setting of malignancy. He underwent an IPSS which did not lateralize at 10 minutes. His hospital course was complicated by development of DVT, fracture of humerus, bacteremia, and empyema which required chest tube placement. He was started on high dose Ketoconazale and cortisol initially trended downwards.

He was readmitted to the hospital with worsening abdominal pain, muscle weakness, and hypertensive emergency. It was still unclear whether this was an ectopic versus pituitary source. Biopsy of 6mm pulmonary nodule was non-diagnostic due to hypocellularity. He was considered to be a poor surgical canidate for open lung biopsy. Octreotide scan was ordered to help localize the ACTH source. Patient was clinically deteriorating from his hypercortisolism. Endocrine surgery was consulted and he undwerwent bilateral adrenalectomy. Post-op blood pressure remained elevated initially. Diabetes improved, no longer requiring insulin. His face appeared less round and plethoric, and his abdomen was less distended.

Conclusion: Bilateral adrenalectomy is useful in controlling hypercortisolism in some severely ill patients who have a presumed occult, ectopic source of ACTH. In this patient, bilateral adrenalectomy led to rapid control of hypercortisolism, that high dose ketoconazole was unable to achieve. This case illustrates the difficulty in locating the source of ACTH, the significant morbidity associated with hypercortisolism, and the need to aggressively reduce cortisol production using surgical means if necessary.

DIABETES MELLITUS

Abstract #200

DIABETES, ABDOMINAL AORTIC ANEURYSM AND THE EFFECT OF SMOKING

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Objective: The main objectives of this study were to assess the prevalence of diabetes in patients with abdominal aortic aneurysm (AAA) and to compare the mean AAA size in diabetic vs. non-diabetic patients who ever smoked.
Methods: A retrospective, comparative observational study of patients with a history of AAA seen in a community hospital setting during a one year period was conducted. The prevalences of some of the major cardiovascular risk factors (old age, diabetes, hypertension, hyperlipidemia, and smoking) and AAA size were analyzed. Only the largest available AAA size was taken into account. The mean size of AAA in diabetic smokers (DS) vs. non-diabetic smokers (NDS) was compared using the two-sample t-test with unequal variances. The one proportion z-test was used to compare the percentages of DS vs. NDS.
Results: 200 charts were reviewed. All patients whose health records had data available on the size of AAA were included (n=130), (mean age=79.4±8.9 years; 76% male, 23% female). Patients with no record of their AAA size were excluded (n=70). Prevalences of various cardiovascular risk factors in the total study population were as follows: diabetes: 25.3% (33/130); smoking: 60% (78/130); hypertension: 84.6% (110/130); hyperlipidemia: 27.6% (70/130); age over 65: 96.9% (126/130). Patients with an active or a former smoking history (n=78) was further subdivided into two groups: DS vs. NDS. The proportion of DS was significantly lesser than that of NDS {30.7% (24/78) vs. 71.7% (56/78); p<0.0001}. The mean age of patients in both groups did not differ significantly (DS vs. NDS: 77.7±7.7 vs. 77.6±9.2 years; p=0.9). Though the mean AAA size of DS was observed to be greater than that of NDS, this difference was not statistically significant (DS vs. NDS: 4.7±1.1 vs. 4.3±1.2 cms; p=0.15).
Discussion: Studies have also shown that hyperglycemia is associated with reduced AAA size. There have been very few studies that examined the prevalence of diabetes in patients with AAA. Smoking is a well know risk factor for the development of AAA. To our knowledge, no studies have compared the AAA size between DS vs. NDS and thereby assess if the adverse effect of smoking is offset by the protective effect of diabetes.
Conclusion: This study shows that diabetes is negatively associated with AAA since it had the lowest prevalence among the cardiovascular risk factors analyzed. Hypertension had the highest prevalence among the modifiable cardiovascular risk factors. The mean AAA size of DS was not statistically different from that of NDS. Since this was a retrospective study, the rate of growth of AAA could not be ascertained. Further prospective studies are needed to compare the rate of growth of AAA in DS vs. NDS.

Abstract #201

ASSOCIATION OF BODY MASS INDEX AND ABDOMINAL ADIPOSITY WITH ATHEROGENIC LIPID PROFILE IN NIGERIANS WITH TYPE 2 DIABETES AND/OR HYPERTENSION.

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Olufemi Fasanmade, MBBS, FWACP,
Christiana Amira, Njideka Okubadejo

Objective: To determine if either anthropometric index (BMI or WC) could be used to identify persons with pre-existing major cardiovascular risk factors (T2DM, HBP, or concomitant disease) who have lipid abnormalities associated with atherogenicity.
Methods: Using a prospective design, patients with T2DM, HBP, or concomitant disease, attending out-patient diabetes and hypertension clinics at a tertiary institution in Nigeria were evaluated. All patients were cholesterol-lowering oral medication naïve. Demographic and clinical data, and anthropometric measurements were documented. Fasting lipid profiles were measured in all cases. The cut-off points for defining dyslipidemia were: elevated TC (mg/dL) ≥200, elevated LDL-C (mg/dL) ≥100, low HDL-C (mg/dL) <40 for men and <50 for women, and high TG (mg/dL) ≥150mg/dL.
Results: We found a significantly higher mean BMI (kg/m2) in the HBP group (30.5±6.0) compared to T2DM (28.1±5.9) and concomitant HBP and T2DM groups (29.4±5.2) (ANOVA; P=0.02). The most frequent dyslipidemia was elevated LDL-C in 92 (96.8%) HBP, 73 (85.9%) T2DM and 79 (80.6%) concomitant disease. The frequency of low HDL-C was highest in T2DM (68.2%) compared to the other 2 groups (P=0.03).
Discussion: The major findings of this study are that although dyslipidaemia, abdominal adiposity, and obesity are all prevalent in our patients with HBP and T2DM, the only association of any lipid parameter with an anthropometric index was between TG and WC, and that occurred only in combined analyses of the three disease states. The possibility of a genetic basis for certain lipid traits (despite increased risk of insulin resistance) in persons of African ancestry exists. Sumner et al explored the biochemical basis of normal triglyceride levels despite the presence of insulin resistance in African Americans,

and showed that lipoprotein lipase activity remains high in the presence of insulin resistance. This enzymatic activity allows for the clearance of TG, a plausible explanation for the coexistence of insulin resistance and normotriglyceridaemia in African Americans.
Conclusion: Only TG levels were found to relate with any anthropometric index (WC in this case) in Nigerians with major cardiovascular risk factors in this study. Routine anthropometric indices do not appear to be reliable surrogates for atherogenicity measured by abnormalities in TC, LDL-C, and HDL-C.

Abstract #202

THE EFFECTS OF DEHYDROEPIANDROSTENDIONE ON INSULIN RESISTANCE IN PATIENTS WITH IMPAIRED GLUCOSE TOLERANCE

Afsaneh Talaei, PHD, Massoud Amini, MD, Mansour Siavash, MD, Maryam Zare

Objective: Dehydroepiandrosterone(DHEA) and Dehydroepiandrosterone-sulfate (DHEAS) are the most abundant steroid hormones in the body.Recently,DHEA-S has gained interest as anantidepressant ,with positive effects on autoimmune diseas,obesity,cancer, cardiovascular disease and diabetes.Its effect on insulin resistance is also assumed to be positive,but has not as yet been confirmed.
Methods: Participants were selected among relatives of diabetic patients who were reffered to the Isfahan Endocrine Research Center because of IGT test.Thirty IGT patients were treated randomly with DHEA (50 mg/day)or placebo by cross-over clinical trial for six months and insulin resistance between the beginning and the end of each three months treatment period was assessed.
Results: At the end of the first trimester,the mean changes from base in the drug group were:DHEA-S 5µmol/l(p=0.008); HOMA-IR,0.6(p=0.6); insulin, 7.1 pmol/l(p=0.3), FPG,0.5mmol/l(p=0.1). The changes in the placebo group were: DHEAS,0.08 µmol/l(p=0.6); HOMA-IR, 0.9(p=0.03)FPG, 0.8mmol/l(p=0.1); insulin, 25.1pmol/L(p=0.05). In the second three months, the mean changes in the drug group were: DHEAs, 4.5µmol/l(p=0.003): FPG,0.1mmol/l(p = 0.4); insulin, 4.3 pmol/l(p=0.2)HOMA-IR,0.3(p=0.1) and the changes in placebo group were: DHEAS,0.7µmol/l(p=0.5); FPG, 0.3mmol/l(p=0.3); insulin, 10.7pmol/l(p=0.1); HOMA-IR,0.6(p=0.03).
Discussion: patients who took DHEA in the first trimester decreased insulin resistance in the second trimester when they were on placebo.This observation might indicate that the wash-out period was not sufficient or that the effects of DHEA lasted longer even after discontinuation of the drug. The first trimester results indicating that daily DHEA to women with IGT favorably affected insulin sensitivity. Metabolism and secretion of many steroid hormones are altered in diabetes.Thus,in cases of poor glycemic control in both type 1 diabetes and also in type 2 diabetes,DHEA and DHEA-S are decreased.Furthermore DHEA has been shown to increase the number of pancreatic beta-cells and improve glycemic control by increasing insulin release in animals. In a study by Kawano DHEA improved endothelial function and insulin resistance while in another study,a strong inverse relation between insulin levels and atherosclerosis was observed. In another study, it was shown that taking DHEA for one year could improve insulin resistance and potentially prevent the development of diabetes.In another study,a two-week intake of DHEA by type 2 diabetic men improved glucose control and HOMA-IR.Other results however have shown that a two year intake of DHEA had no effect on insulin sensitivity.
Conclusion: The data indicate a possible but not clearly favorable effect of DHEA on insulin resistance.

Abstract #203

EVALUATION OF ANEMIA IN DIABETIC NEPHROPATHY AND CORRELATION OF IRON STATUS WITH DIFFERENT STAGES OF CKD

S.M. Ashrafuzzaman, MBBS, MD, Zafar Latif, FCPS

Objective: Anemia is extremely common in CKD affecting upto 95 % of the patients. Severity of anemia may vary at different stages of CKD in different population. Reduced Erythropoietin Synthesis by the kidneys plays the major role for anemia in CKD, though other factors also contributes for the development of Anemia in CKD.
Methods: A cross sectional study was done on 200 subjects of Diabetic Nephropathy. All the subjects has stage 3 (CCR < 60 ml/min/1.73 m2 according to Cock-Croft and Gault Equation from S. Creatinine) to Stage 5 CKD(CCR < 15 ml/min/1.73m2). Anemia is defined according to WHO criteria (Hb < 13 gm/dl in Men and < 12 gm/dl in women).
Results: Among 200 study subjects 43 % Male and 57 % Female. Age matched (Mean Age 55.37±11.07. Hb % is corrected for Sex . Among the CKD subjects anemia was found in 89%. Mean concentration of Haemoglobin was 10.24±1.60, 9.74 ±1.36, 9.04 ±1.37 in Stage 3, stage 4 and Stage 5 CKD respectively, which is statistically significant (p< 0.001). Mean S.Iron concentration was 13.55± 6.53, 11.08 ±5.75 and 10.84 ±7.75 respectively. Mean Serum Ferritin was 292.45 ±342.48 in Stage 5 CKD, which is higher than Stage 3 and Stage 4 CKD. TIBC was 39.37 ±15.96, 34.29 ±11.53, and 39.84 ±11.98 respectively. All are statistically Significant(p<0.04). According to PBF

63.7% are having Normocytic Normochromic anemia and 30.2 % with Non specific morphology. Rest(6.1%) are Microcytic hypochromic. Among the study subjects 60.5% are mildly anemic(Hb > 10 gm/dl), 29.5% Moderately anemic and 10.0% severely anemic(Hb< 7 gm/dl). Seventy four percent(74%) showed T-Sat ≥ 20 % and rest(26%) only constitutes T-Sat < 20 %.

Discussion: Many of the patients with DM and CKD are anemic. But some of them may EPO defient or inefficient As Iron is the essential raw material for hemoglobin synthesis, T-Sat is the best indicators for Iron available for Erythropoisis and stored Iron.

Conclusion: Anemia was found in 89 % at different stages of CKD patents. Serum Iron level gradually decreases from stage 3 CKD onwards reaching maximum at stage 5. T-Sat level also decreases accordingly. Thus in a developing country like Bangladesh, to make the treatment cost-effective, all patient with CKD should be screened for Iron profile and should be replaced if necessary, before giving Erythropoietin.

Abstract #204

PREDIABETES IN A HYPERTENSIVE POPULATION

Shashi Agarwal, MD

Objective: Diabetes mellitus is a common disease. It is estimated that it affects about 20 million Americans. It is also common in hypertensives, and increases their morbidity and mortality. It is estimated that 57 million people in the United States are prediabetic. These people have an increased propensity to develop premature adverse cardiovascular events. The prevalence of prediabetes in a hypertensive population has not been well studied.

Methods: We reviewed the charts of 277 consecutive hypertensive patients who had their HbA1c recorded. HbA1c was categorized as follows: Normal: <5.7 (less than 39 mmol/mol); pre-diabetes: 5.7% - 6.4% (39 - 46 mmol/mol); diabetes: 6.5% (47 mmol/mol) or higher.

Results: Of the 277 patients (males: 158; females: 119) (ages 16-92 years), 76 (27%) (M:52; F:24) had HbA1c <5.6; 127 (46%) (M:73; F:54) had HbA1c between 5.7-6.4; and 74 (27%) (M:33; F:41) had HbA1c >6.5.

Discussion: Most hypertensive patients with prediabetes have similar cardiovascular risk factors as patients with type 2 diabetes, such as dysglycemia, dyslipidemia, obesity, physical inactivity, insulin resistance, procoagulant state, endothelial dysfunction and inflammation. This places these patients at a high risk for premature cardiovascular morbidity and mortality.

Conclusion: We found an extremely high prevalence of prediabetes in our hypertensive population. Although established diabetes affected 27%, prediabetes was present in 46% of these patients. Hypertensive patients with prediabetes should be treated aggressively with lifestyle modifications. Further studies are needed to evaluate the protective role of specific anti-diabetic and anti-hypertensive agents in this population.

Abstract #205

DAPAGLIFLOZIN IS ASSOCIATED WITH WEIGHT REDUCTION AS A SECONDARY BENEFIT IN PATIENTS WITH TYPE 2 DIABETES MELLITUS: POOLED SUBGROUP ANALYSIS OF 9 CLINICAL TRIALS

Afshin Salsali, MD, Li Wei, Traci Mansfield, Catrin Wessman, Elise Hardy, Tjerk de Bruin, Shamik Parikh

Objective: Reduction of body weight is a fundamental goal in the overall management of patients with type 2 diabetes mellitus (T2DM) because of the association between excess body weight and insulin resistance. Dapagliflozin (DAPA), a selective inhibitor of sodium glucose cotransporter 2 (SGLT2), reduces plasma glucose independently of insulin secretion or action by increasing the excretion of excess glucose. DAPA also reduces body weight in patients with T2DM through the urinary loss of calories. This report analyzed body weight data pooled from 9 double-blind, randomized clinical trials of DAPA in patients with T2DM to determine if baseline parameters affect weight reduction.

Methods: Patients with T2DM (N=4047) received DAPA 2.5, 5, or 10 mg/d or placebo for 24 wk as monotherapy (NCT00528372, NCT00736879) or as add-on to metformin (NCT00528879, NCT00855166), glimepiride (NCT00680745), pioglitazone (NCT00683878), or insulin (NCT00673231), or as an initial combination with metformin (NCT00859898, NCT00643851). Adjusted mean change from baseline in total body weight, excluding data after rescue (LOCF), was analyzed by an ANCOVA model with treatment group, subgroup, and study as categorical factors, interaction between treatment group and subgroup, and baseline weight and study-by-baseline weight interaction as continuous covariates. Treatment by subgroup interactions were analyzed for baseline HbA1c, estimated glomerular filtration rate (eGFR), age, gender, race, ethnicity (US only), region, body mass index (BMI), and T2DM duration. *P* values are reported for treatment by subgroup interaction based on average treatment effect relative to placebo. A *P* value <0.1 indicated a potential interaction.

Results: Within individual studies, placebo-corrected change from baseline in body weight ranged from -0.46

to -2.16 kg in the overall population. No interaction of weight reduction with age, gender, baseline HbA1c, BMI, eGFR, or duration of T2DM was detected. Treatment by subgroup interactions were detected for geographic region (*P*=0.03), race (*P*=0.06), and ethnicity (*P*=0.09).
Discussion: North Americans had numerically greater weight loss compared with patients from Latin America, Europe, and the Asia/Pacific region. Asians, blacks, and whites tended to have greater weight loss with DAPA than those classified as other; however, there were small numbers of blacks and other patients within each treatment group. Non-Hispanic patients tended to have greater weight loss than Hispanic patients.
Conclusion: Overall, DAPA treatment reduces body weight in patients with T2DM, and this effect appears to be independent of age, gender, baseline HbA1c, BMI, eGFR, and duration of diabetes.

Abstract #206

RANDOMIZED, PLACEBO-CONTROLLED, DOUBLE-BLIND, 24-WEEK STUDY OF LINAGLIPTIN 5 MG/DAY IN BLACK/AFRICAN AMERICAN PATIENTS WITH TYPE 2 DIABETES.

James Thrasher, MD, Azazuddin Ahmed, MD, Kristen Daniels, Sanjay Patel, Jacqueline Whetteckey

Objective: Black/African Americans have high rates of type 2 diabetes mellitus (T2DM) yet are under-represented in clinical trials of oral antidiabetic drugs (OADs). Therefore, a trial of the recently developed OAD, linagliptin, was undertaken to specifically recruit African American or black patients with T2DM.
Methods: In this US, multicenter, randomized, placebo-controlled, double-blind trial (NCT01194830), T2DM patients who were treatment-naïve or on ≤1 OAD, and who reported their race as black or African American were randomized to 24 weeks linagliptin 5 mg/day or placebo.
Results: Of 592 patients screened and enrolled, 226 were randomized and received ≥1 dose of study drug (safety set: 106 linagliptin; 120 placebo) and 211 had a baseline and ≥1 on-treatment measurement (efficacy set: 100 linagliptin; 111 placebo). Linagliptin and placebo groups were well balanced for baseline characteristics: overall, 54% were men, mean age was 54 (SD 9.9) years, mean BMI 32.7 (SD 5.7) kg/m2, and 72% had hypertension. In the efficacy analysis set, mean (SE) baseline HbA1c was 8.60 (0.1)% and 8.68 (0.1)% for the linagliptin and placebo groups. Across groups, most patients were on metformin or a sulfonylurea, which was continued unchanged; 12% were treatment-naive. By 24 weeks, mean (SE) HbA1c changes were −0.84 (0.2)% with linagliptin and −0.25 (0.2)% with placebo (placebo-adjusted mean change, P=0.0005) and more patients in the linagliptin group achieved HbA1c <7.0% (26.0% vs 9.0%, OR 4.0, P=0.001) or an HbA1c reduction ≥0.5% (53.0% vs 29.7%, OR 2.8, P=0.0004). Both groups showed weight loss: mean (SD) with linagliptin -1.1 (3.8) kg, placebo -1.1 (7.6) kg. During the 24-week treatment period, 8/100 patients in the linagliptin group and 17/111 in the placebo group required rescue therapy (OR 0.5, P=0.13); AEs were reported in 62/106 (58.5%) and 74/120 (61.7%) of the 2 groups, respectively, most were mild or moderate and considered unrelated to study drug. Overall, the most common AEs were hyperglycemia (linagliptin 2.8%; placebo 9.2%) and nasopharyngitis (linagliptin 3.8%; placebo 5.0%). Serious AEs were reported in 1 patient in the linagliptin group and 2 in the placebo group. Investigator-defined hypoglycemia was rare (3 patients in the linagliptin group and 1 in the placebo group) and no event required external assistance.
Discussion: In this group of black/African Americans with T2DM, linagliptin 5 mg/day was associated with significant improvements in measures of hyperglycemia, and was well tolerated with an AE profile similar to placebo.
Conclusion: This study confirms linagliptin is an efficacious treatment option in black/African American patients with T2DM.

Abstract #207

200 U/ML INSULIN DEGLUDEC IMPROVES GLYCEMIC CONTROL SIMILAR TO INSULIN GLARGINE WITH A LOW RISK OF HYPOGLYCEMIA IN INSULIN-NAÏVE PEOPLE WITH TYPE 2 DIABETES

Richard Bergenstal, MD, Anuj Bhargava, Rajeev Jain, MD, Jeff Unger, MD, Søren Rasmussen, Henriette Mersebach, Stephen Gough

Objective: Insulin degludec (IDeg) is a new basal insulin that forms soluble multihexamers that dissociate slowly and steadily upon subcutaneous injection to produce an ultra-long and stable profile with a half-life >24 hours. The 200 U/mL formulation of IDeg (IDeg U200) contains equal units of insulin in half the volume compared to the 100 U/mL formulation, and thus allows larger insulin doses to be administered in a single injection (up to 160 U) with a prefilled pen device. This 26-week, open-label, treat-to-target trial compared the efficacy and safety of once-daily IDeg U200 with 100 U/mL of insulin glargine (IGlar), both in combination with oral antidiabetic drugs.
Methods: Insulin-naïve patients (≥18 years old) with type 2 diabetes (T2D) and HbA1c 7-10% ≥6 months (n=457; mean: 57.5 yrs old, diabetes duration 8.2 yrs, BMI 32.4 kg/m^2, HbA1c 8.3%, and fasting plasma glucose (FPG)

173.2 mg/dL) who qualified for intensified treatment were randomized to IDeg U200 or IGlar, both given OD in combination with metformin ± DPP-4 inhibitor in prefilled pen devices. Basal insulin was initiated at 10 U/day and titrated weekly to a FPG target of ≤90 mg/dL based on mean pre-breakfast plasma glucose values from the preceding 3 days.

Results: An equal proportion of subjects completed the trial (87% for both IDeg and IGlar). By 26 weeks, IDeg U200 effectively reduced HbA1c by 1.30%-points and was noninferior to IGlar (estimated treatment difference (ETD) IDeg-IGlar: 0.04%-points [95% CI: -0.11; 0.19]). Mean observed FPG reductions were significantly greater with IDeg U200 than with IGlar (-67 vs. -61 mg/dL; ETD: -7.59 [-14.09; -1.09], p=0.02). Rates of overall confirmed hypoglycemia (PG <56 mg/dL or requiring assistance) were numerically lower with IDeg U200 vs IGlar (1.22 and 1.42 episodes/patient-year, respectively; estimated rate ratio (ERR) IDeg/IGlar: 0.86 [95% CI: 0.58; 1.28], p=0.46). Rates of nocturnal confirmed hypoglycemia (occurring between 00:01-05:59) also were numerically lower with IDeg (0.18 vs. 0.28 episodes/patient-year, respectively; ERR: 0.64 [95% CI: 0.30; 1.37], p=0.25). Mean daily basal insulin dose was similar after 26 weeks (IDeg U200, 0.62 U/kg; IGlar, 0.66 U/kg). IDeg U200 was well tolerated and the rate of treatment-emergent adverse events, including injection site reactions, was similar across groups.

Discussion: Insulin degludec 200 U/mL allows patients who require larger daily doses of basal insulin and use prefilled pen devices to administer up to 160 U in a single injection.

Conclusion: In this trial in insulin-naïve patients with T2D, insulin degludec 200 U/mL improved glycemic control similar to IGlar with a low risk of hypoglycemia.

Abstract #208

GLYCEMIC CONTROL AT INITIATION OF HYPERBARIC OXYGEN THERAPY DOES NOT AFFECT DIABETIC LOWER EXTREMITY WOUND HEALING

Owaise Mansuri, MD, Parkash Bakhtiani, MBBS, Abhijeet Yadav, Chima Osuoha, Patricia Knight, Robert McLafferty, Michael Jakoby, IV, MD

Objective: Diabetic lower extremity ulcers are a major cause of disability and mortality, accounting for approximately two-thirds of all non-traumatic amputations performed in the United States. Hyperbaric oxygen (HBO) is increasingly used as an adjunct to antibiotics, debridement, and revascularization for therapy of chronic, non-healing wounds associated with diabetes mellitus. We investigated whether glycemic control at time of HBO therapy measured by hemoglobin A1c (HbA1c) has a significant impact on diabetic wound healing.

Methods: A multi-center, prospective cohort study assessing lower extremity wound healing rates among adult patients with diabetes mellitus treated with HBO was conducted at the Regional Wound Care Center in Springfield, IL and University Medical Center Hyperbaric Oxygen Center and Burn Care Unit in Las Vegas, NV. Patients underwent 20 sessions of HBO over the course of one month, and ulcer size (surface area and depth) and location were recorded at each visit. Transcutaneous oxygen pressures (TcPO2) were measured during each session. HbA1c was measured at first and last HBO treatments from capillary blood specimens using a Siemens DCA Vantage A1c Analyzer. Patient characteristics including age, gender, weight, type and duration of diabetes mellitus, current diabetes treatment regimen, hypertension, peripheral artery disease (PAD), tobacco use, ulcer duration, and additional wound care therapies (e.g. debridement, platelet derived growth factor) were determined.

Results: Complete data were collected for 22 patients who were included in the study analysis and divided into two groups based on pre-HBO HbA1c. Patients in the "controlled diabetes" group had HbA1c < 7.5%, and patients in the "uncontrolled diabetes" group had HbA1c ≥ 7.5%. Mean HbA1c in the "controlled diabetes" group (6.5 ± 0.8%, N=12) was significantly lower (P < 0.001) than in the "uncontrolled diabetes" group (8.8 ± 1.4%, N=10). Both groups were well matched across all other recorded characteristics. Wound volume was reduced by 65 ± 29% in the "controlled diabetes" group and 71 ± 30% in the "uncontrolled diabetes" group (P = 0.60). Wound healing was also unaffected by presence or absence of PAD, hypertension, tobacco use, weight, duration of diabetes, or ulcer duration.

Discussion: This study demonstrates that diabetic lower extremity wound response to HBO treatment is unaffected by pre-treatment glycemic control and several other clinical factors that may adversely impact wound healing.

Conclusion: HBO treatment should not be delayed if glycemic control is suboptimal at time that therapy is prescribed.

Abstract #209

PREVALENCE OF LATENT AUTOIMMUNE DIABETES IN YOUNG, NON OBESE , ADULT ONSET DIABETES PATIENTS WITH POOR RESPONSE TO ORAL INSULIN SECRETAGOGUES

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Objective: Latent autoimmune diabetes in adults (LADA) is defined as adult-onset diabetes with circulating islet antibodies but not requiring insulin initially. Diagnosing LADA has treatment implications because of the high risk of progression to insulin dependency. This study was done to observe the prevalence of LADA in young (25-40 years), adult onset (>20 years) and non obese (BMI < 25) diabetic patients having poor glycemic control with Oral insulin secretagogues.
Methods: One hundred young (25-40 years), adult onset (> 20 years) and non obese (BMI < 25) diabetic patients having poor glycemic control (HbA1c > 8.5 %) with optimal dosage of Oral insulin secretagogues (Sulphonylureas) were included in the study. None of the patient had e/o Ketoacidosis. Detailed clinical History, family history and Anthropometric measurements were taken. Ultrasound screening of pancreas was done for every patient. HbA1c, fasting C peptide, 1 hour post meal C peptide, Anti Glutamic acid decarboxylase antibodies (GADA) and Islet Cell Autoantigen 512 Antibodies (Anti-IA2) were estimated for every patient.
Results: The mean age of patients was 33 years and they had diabetes for an average of 6 years. On Autoantibody screening, 48 patients came out to be Autoantibody positive. 45 patients (45 %) were positive for GADA and 7 patients were positive for Anti IA2 antibodies. 4 of the 7 Anti IA2 positive patients were also having Anti GAD Antibodies. Patients who were Antibody positive had lower BMI , more complications and worse glycemic control. Family history of Diabetes was most predictive of absence of Antibodies.
Discussion: The high prevalence of LADA (48 %) in our study is among a selected subclass of diabetic with higher pretest probability for LADA. The prevalence in general diabetics will be much lower. However we can say that Antibody screening in this select group of diabetic (young, non obese, no family history) is warranted and will lead to better standards of care. The low positivity of Anti IA2 Antibody as compared to GADA has also been observed in other Indian studies and may be due to lower prevalence of HLA DR4. This study is ongoing and more patients are being recruited.
Conclusion: Young, non obese diabetes patients with adult onset diabetes, no family history and poor response to oral insulin secretagogues should be screened for Autoimmune Diabetes.

Abstract #210

SCREENING MODELS FOR UNDETECTED DIABETES AND HIGH RISK FOR DIABETES IN THE UNITED ARAB EMIRATES

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Objective: The prevalence of diabetes in the United Arab Emirates (UAE) is among the world's highest. We aimed to develop a simple non-invasive screening model for identifying adults with or at high risk of having undetected diabetes or glycaemia in the UAE.
Methods: Between November 2010 and January 2011 the Changing Diabetes® World Tour based on opportunity sampling screened 2466 individuals in the UAE aged between 30 and 75 years. Screening consisted of a self-administered questionnaire and measurement of anthropometry and HbA1c. After exclusion of those with known diabetes and missing data, the analyses included 1982 individuals. A risk score was created for the country on the basis of a backwards logistic regression of all relevant risk factors. Risk factors significant at a 10% level in a univariate analyses with HbA1c ≥6.0% as outcome were included in a multiple logistic regression using stepwise backward elimination and HbA1c ≥6.0% as outcome. A p value of <5% was considered significant in selection of factors to be included in the risk score. The risk factors in the final model were each assigned a score by multiplying the regression coefficients by 10 and rounded off to the nearest integer. Each participant was assigned a summed risk score calculated by adding the individual scores of the risk factors in the model.
Case Presentation: We found 171 cases of undiagnosed diabetes (HbA1c ≥ 6.5) and 230 cases of high risk of diabetes (HbA1c ≥ 6.0 and < 6.5). The final risk score included age in 5-year age groups, BMI in 5 kg/m2 groups, mother's diabetes status, gender and ethnic background. The area under the receiver operating characteristics curve (AUC) was 0.71 (95% CI: 0.68-0.73) and a sensitivity of 73% corresponded to a specificity of 56%. If applied for screening purposes the model would select 50% of UAE's population aged 30 to 75 as needing a diagnostic test for diabetes.
Discussion: If only the three most important risk factors were included in the risk score (age, BMI and mother's diabetes status), the AUC of the model was 0.69 (95% CI: 0.67-0.71), and a sensitivity of 78% corresponded

to a specificity of 49%. If this model was chosen, the proportion of people who would be referred to a diagnostic test would be 14% higher than if the more comprehensive model was used.
Conclusion: We developed a simple and non-invasive screening model which can be used in a stepwise screening strategy for diabetes and impaired glycaemia in the UAE. The diabetes risk score, based on information easily obtained through a simple questionnaire or interview, can be a good first line selection tool to find those most likely to benefit from a diagnostic HbA1c measurement.

Abstract #211

FAILURE OF SHORT TERM IPRO™ CGM TO IMPROVE GLYCOHEMOGLOBIN A1C LEVELS IN CLINICAL PRACTICE

Gary Pepper, MD, Kathryn Reynolds, Jaimie Steinsipar

Objective: To determine if short term (professional) CGM improves glycohemoglobin A1c levels in a mixed group of patients with type 1 and type 2 diabetes in the clinical setting of an office practice.
Methods: A retrospective analysis of 102 consecutive diabetics over the course of 10 months in a sub-specialty practice undergoing 3 day CGM utilizing the iPRO device. Glycohemoglobin A1c levels were measure prior to and up to 7 months post CGM procedure.
Results: Before CGM the average glycohemoglobin A1c levels was 7.7 ± 1.0 %, and after 7.8 ± 1.1 %. These values are not statistical different. A subgroup analysis of subjects using portable insulin pump devices also failed to show a significant glycohemoglobin A1c difference pre and post CGM.
Discussion: It is tempting to use new technologies to assist in management of diabetes, but the complexities involved in regulating glycemic control in a real life setting, appears to require more than is offered by this popular device. Short term CGM with the iPRO may be useful however, for other purposes including detecting asymptomatic or nocturnal hypoglycemia.
Conclusion: We are in agreement with the most recent recommendations by the American Association of Clinical Endocrinologist advising research to pinpoint patient groups which are the best candidates for CGM technology. As a first step, restricting short term CGM procedures to the groups most likely to benefit such as those with frequent nocturnal hypoglycemia or as a lead in to personal CGM use, seems advisable.

Abstract #212

LONG TERM TYPE 1 DIABETES IN A PRIVATE PRACTICE SETTING

Thomas Flood, M.D., F.A.C.E.

Objective: Present data on patients with Type 1 diabetes of 40-50 years duration.
Methods: Chart review
Case Presentation: Survival after 50 years of insulin requiring diabetes has been recognized as a benchmark of successful treatment and rewarded with medal recognition (Eli Lilly and Joslin Diabetes Center). As a group, these medalists are being increasingly recognized and many are evidencing exceptionally good health despite their many years of disease prompting calls for investigation of factors responsible for such quality survival. Data relevant to the 50 years patients (#11) is presented and expanded to include a much larger (#26) cohort of patients with Type 1 diabetes of 40 to 50 years duration under active follow up in a private practice. Full demographics to include age, duration of diabetes, control (A1C), treatment regimen, specific complications and years of follow up is presented in tabloid form for individual members of both cohorts. As a group, the 40 year cohort evidence an extremely high quality of complication free survival. There has been no significant visual loss, renal insufficiency, peripheral neuropathy or peripheral vascular disease in 25 of the 26 patients.
Discussion: Overall statistics of the 40-50 years cohort include: a) Mean A1C 7.3%. Range 5.5-9.3% with 10- <7.0%; 9-7.0-7.5%; 3- 7.6-8.0%; 4- > 8.0%. b) Mean age 62.4 years. Range 49-76 years old. c) Treatment: CSII-14; MDI-11; Split NPH-1. d) Retinopathy: Normal-11; NPDR-6; Laser Rx-9. e) Nephropathy: Normal Creatinine 24; Slightly increased creatinine 1; Dialysis 1. f) Neuropathy: 25/26 intact sensation to tuning fork and/or monofilament. g) Coronary Artery Disease: 23-asymptomatic; 2-CABG; 1- Stent. h) Peripheral Vascular Disease: 23/26 asymptomatic with intact pedal pulses. Other Treatment modalities: Statins: 16/26; Antihypertensives: 12/26 (10 with ACE or ARB alone; 3 with ACE or ABR plus other).
Conclusion: The high quality survival in this group of Type 1 patients of over 40 and less than 50 years duration suggests continued success once past the iconic 50 years, bench mark and potentially expands the pool of individuals eligible for investigation seeking factors above and beyond simple glycemic control which may be contributing to the good outcome.

Abstract #213

UNMASKING A LETHAL COMBINATION: DIABETES MELLITUS IN TUBERCULOSIS

Anthonia Ogbera, MBBS, FMCP, Sonny Chinenye, Adeleye Olufunmilayo, Olufemi Adewusi, Ayotunde Ale, Emmanuel Ezeobi, Olufemi Fasanmade, MBBS, FWACP

Objective: The global burden if diabetes mellitus (DM) and tuberculosis (TB) is immense and the burden of these disorders in Nigeria, a country with emerging high incidence rates of DM and TB is expected to be unacceptably high. The possible association between diabetes mellitus and tuberculosis has been shown in studies carried out in countries other than those from Africa. The objectives of this Report include documenting prevalence and incidence rates of DM in TB and also exploring for possible associations between these two disorders.
Methods: This was a cross sectional study carried out in persons with TB who were already on treatment with anti-TB drugs. The study subjects were recruited consecutively from 30 DOT centres in Lagos State. All Study subjects were screened for DM and suspicious blood glucose results were repeated.Clinical characteristics and TB relapse rates were compared between persons with DM and those without DM. Statistical tests used were Students t test and chi square.
Results: The study subjects were 1,687 in number and there were 1000 males thus making up 59% of the study population. The mean age and age range of the study subjects were 36.17 (13.4)years and 14-83years respectively. The incidence of DM in TB was found to be 4.5% of which the Male: Female ratio was 1.8:1 and the age range was 25 to 57 years. The mean age of persons found to have new onset TB was comparatively higher than those without TB and the difference was statistically significant (40.4 Vs 36.1 years, p=0.018). Positive family histories of DM in persons with new onset DM and persons without DM were comparable(1.4% Vs 4.3%, p=0.8). The prevalence of relapsed cases of TB was 5.7% and subjects with new onset DM were found to have higher numbers of relapsed TB cases compared to those without DM but this difference was not statistically significant.Sputum smear positivity was comparable in new onset DM and those without DM. A total of 75 persons with TB had a history of DM diagnosed prior to diagnosis and treatment of DM thus giving a total prevalence rate of DM in TB to be 8.7%. 70% of persons with DM were noted to have poor short term glycaemic control and hospitalisation was indicated for about a fourth of these patients.
Discussion: The presence of TB in DM may be responsible for poor glycaemic control which may negatively impact other systems. Although our reported incidence rate of DM in TB appears relatively low, it however translates into a significant number .
Conclusion: Age is an independent risk factor in the occurrence of DM in persons with TB and persons older than 20 years of age with TB should be screened routinely for DM.

Abstract #214

FLEXTOUCH®, A NEW PREFILLED INSULIN PEN: USABILITY STUDY VERSUS VIAL AND SYRINGE INVOLVING PHYSICIANS, NURSES AND PEOPLE WITH DIABETES

Rosemarie Lajara, MD, Jerome Thurman, MD, German Guerrero, Davida Kruger

Objective: FlexTouch® (FT; Novo Nordisk A/S, Bagsvaerd, Denmark) is a new prefilled insulin pen with no push-button extension through all doses, a low injection force and accurate and consistent dosing. It has been approved by the European Medicines Agency with currently marketed NovoRapid® (Novo Nordisk A/S, Bagsvaerd, Denmark) and will be available, upon approval, with insulin degludec: a new ultra-long-acting basal insulin with a flat and stable action profile, a half-life of greater than 24h and low day-to-day variability in glucose-lowering effect. Use of insulin pens as an alternative to vial and syringe (V&S) is increasing, as is the interest to assess how they may improve insulin delivery.
Methods: This subgroup analysis of a multicenter study assessed perceptions of FT vs V&S among 30 physicians, 30 nurses, 30 V&S-experienced people with diabetes (PwD) and 30 needle-naïve PwD for ease of use, confidence in the device, ease of learning and teaching to use, and overall preference. After performing test injections with both devices, participants rated them on a 5-point scale. For ease of teaching and learning to use, the results for each subgroup and for the total population are presented. For all other results only subgroups are presented here.
Results: In the total population, FT was rated higher than V&S for ease of teaching or learning to use (both $p<0.001$), and was preferred to V&S for teaching or learning to use (both $p<0.001$). Most physicians (87%) and all nurses (100%) preferred FT to V&S for ease of teaching. Most V&S-experienced (73%) and needle-naïve (83%) PwD preferred FT to V&S for ease of learning. More participants in each group rated FT 'very/fairly easy' than V&S for ease of use questions, including ease of depressing the push-button/plunger (FT vs V&S; physicians, 93% vs 80%; nurses, 97% vs 80%; V&S-experienced PwD, 93% vs 90%; needle-naïve PwD, 100% vs 77%), and ease of injecting three doses including the maximum dose of 80

units (FT vs V&S; physicians, 100% vs 40%; nurses, 87% vs 57%; V&S-experienced PwD, 90% vs 57%; needle-naïve PwD, 87% vs 43%). More participants were 'very/rather confident' in managing daily injections using FT than V&S (FT vs V&S; physicians, 100% vs 60%; nurses, 100% vs 70%; V&S-experienced PwD, 93% vs 90%; needle-naïve PwD, 90% vs 40%).
Discussion: These results suggest that physicians, nurses and patients, both needle-naïve and V&S-experienced, find FT easier to use and prefer it to V&S.
Conclusion: From the data we also infer that FT requires less time to train and that there is minimal resistance to transfer from V&S to FT.

Abstract #215

USE OF REAL-TIME CONTINUOUS GLUCOSE MONITORING FOR ENDURANCE ATHLETES WITH TYPE 1 DIABETES

Joseph Henske, MD

Objective: Demonstrate the utility of real-time continuous glucose monitoring (rt-CGM) during exercise for endurance athletes with type 1 diabetes.
Methods: Use of a rt-CGM device (DEXCOM) in a 34 year old male with a 19 year history of type 1 diabetes while training for and running the 2011 Chicago Marathon. Data was collected during training runs and used to optimize pump settings and race-day strategy. Data collected included start and finish blood glucose meter and CGM readings, minimum and maximum glucose levels, total carbohydrates consumed, basal rate percentage changes, and duration of exercise.
Case Presentation: Use of rt-CGM consistently during the training phase allowed: 1. Optimization of pre-exercise blood glucose. A blood glucose level of 160-180mg/dL before exercise was adequate to accommodate a fall in blood glucose during the first hour of exercise while avoiding hypoglycemia. 2. Optimization of basal rate reduction during exercise. A basal rate reduction of 50% from baseline reduced hypoglycemia with exercise and increased time in target glucose range. 3. Determination of optimal carbohydrate intake amounts and frequency to sustain energy and maintain target blood glucose. These can vary significantly depending on dynamic variables such as intensity and duration of exercise and "insulin-on-board." It was additionally observed that sensor adherence is important to address when exercise is performed in hot or humid conditions expected to increase perspiration in the sensor location. Sensor accuracy is optimized when the "insulin-on-board" is reduced by exercising in the fasting state.
Discussion: Glycemic variability is a major barrier to optimal performance in athletes with type 1 diabetes. Failure to reduce insulin levels, consume adequate carbohydrates, and recognize counterregulatory response signals can lead to dangerous hypoglycemia. Unguided carbohydrate loading, adrenaline responses, and excessive insulin reductions can lead to hyperglycemia, dehydration, and poor performance. Lack of a convenient, reliable method of monitoring blood glucose levels during exercise has historically been a barrier to exercise for individuals with type 1 diabetes. Rt-CGM, however, offers the ability to exercise safely with optimal performance.
Conclusion: Use of rt-CGM allows for improved confidence and performance during endurance exercise for athletes with type 1 diabetes. Use of rt-CGM during training allows an understanding of the glycemic response to exercise, adaptation of carbohydrate intake, and adjustment of insulin dosing. Addressing sensor adherence and insulin-on-board are important to maintaining sensor accuracy.

Abstract #216

ETHNIC VARIATION IN THE CORRELATION BETWEEN FASTING SERUM GLUCOSE CONCENTRATION AND GLYCATED HEMOGLOBIN (HBA1C)

Rinkoo Dalan, MBBS, MRCP, FRCP (Edin), FAMS (Endocrinology), Melvin Leow, Arul Earnest

Objective: A prospective cohort study in Singapore found that Malays have significantly higher HbA1c when compared to Chinese and Indians both at baseline and at subsequent follow up. It is possible that socioeconomic and cultural differences can influence the fasting serum glucose (FSG) and postprandial glucose (PPG) and alter their relative contributions to HbA1c. We aim to determine the relationship between fasting serum glucose (FSG) concentration and glycated hemoglobin (HbA1c) in the three ethnicities in Singapore after adjustment for demographic and therapeutic variables.
Methods: FSG, HbA1c and serum creatinine levels were simultaneously sampled from 479 patients with diabetes (315 Chinese, 90 Indians, 74 Malays) in this cross-sectional study between January to May 2008. We performed multivariate linear regression analysis using Stata V11.2.
Results: We found that there was a significant interaction effect between FSG and ethnicity on HbA1c. In other words, the correlation between FSG and HbA1c among the Chinese was 0.25 (95% CI:0.2-0.3), much lower than that among the Malays 0.38 (95% CI: 0.30-0.45 after adjustment for age, gender, serum creatinine concentrations, BMI, duration of diabetes, use of sulfonylureas, metformin and

insulin, the hemoglobin concentrations, MCV, MCHC and MCH. (P=0.005). Hence, for a given fasting glucose concentration, the predicted HbA1c will be higher in the Malays when compared to the Chinese. For eg, for a fasting glucose concentration of 7.0 mmol/L in a 50 years old male, with diabetes mellitus for 5 yrs, BMI=22, Hb=13.5g/dl, Creatinine 90 umol/L using metformin and a sulfonylurea, the predicted HbA1c will be 7.2% in the Chinese when compared to 8.4% in the malays.

Discussion: We showed that Malays had higher correlation between HbA1c and FSG relative to the Chinese amongst our patients. It is possible that Malays have higher PPG contributing to higher HbA1c due to metabolic and dietary factors. Other relevant factors including educational, socioeconomic, psycho-behavioural attributes, personal beliefs, attitudes and adjustment issues deserve further investigation. Future studies using continuous glucose monitoring system (CGMS) to elucidate the relative contributions by FSG and PPG to the daily blood glucose profile and the overall HbA1c by ethnicity are required.

Conclusion: The ethnic variation in HbA1c:FSG relationship may be related to differences in percentage contribution by the FSG to overall HbA1c between ethnic groups. The possibility of race exerting an independent effect will have to be addressed by future studies.

Abstract #217

THYROID DYSFUNCTION IN IRAQI DIABETIC PATIENTS

Fuad Alkurdi, MD

Methods: prospective study include type1 and type 2 DM,control group was healthy persons with age and sex match with patients. normal range for lab used was (0.95-5mU/l)for TSH, and (0.39-60mU/l) for patients

Results: The overall prevalence of hypothyroidism in study group was (19.2%) compared with (4.3 %) in control group ($p<0.05$), the prevalence of hypothyroidism in type 2DM was (17.1%), and it was (25%) in type 1 DM. The subclinical hypothyroidism composed (39.2%) of total number of affected patients, while overt hypothyroidism composed (60.8%). The diabetic females with hypothyroidism composed (59.2%) of total number, and males composed (40.8%).p=0.382 The prevalence of hypothyroidism is higher in age group (45-64 year) than age group (15-44 year). The prevalence of hyperthyroidism in study group was (7.1%) compared with (2.8%) in control. ($p<0.05$), and (55%) of them were subclinical. The vast majority of affected diabetic patients were overweight (46.65) or obese (43.4%). The mean BMI of diabetic patients with hypothyroidism was (29.04%), SD ±2.86.

Discussion: The present study confirmed the strong association between DM and hypothyroidism, and the prevalence of thyroid dysfunction in our study was higher than general population, and the result is consistent with those of previous studies (9, 10, 11, 12, 17), and the prevalence in our study was higher than most studies. The prevalence of hypothyroidism in our study was higher in diabetic females. And this result is consistent with previous studies (6, 8, 10, 17), but the relation of no statistical significance (p=0382). The prevalence of hypothyroidism was higher in type 1DM in compression with type 2DM, and this result is consistent with previous studies (6, 10, 17) Overt hypothyroidism prevalence in our study was more than subclinical one, and this result is in contrary with previous studies (10, 17), this result may explain by the age of study group (45.16 year, SD±10) and by the long time after the onset of diagnosis (mean 14.3 year SD±2.2) during which the progression to overt hypothyroidism might happened. In our study the vast majority of affected diabetic patients were with unhealthy weight (90%) and the mean BMI was (29.04), SD ±2.86, this finding is consistent with previous study (10). The prevalence of hyperthyroidism was (7.1%) compared with (2.8%) in control group ($p<0.05$), and this result is consistent with previous studies (6, 10, 15, 17).

Conclusion: Baseline serum TSH need to be check at time of diagnosis of DM, and annual thyroid screening for patients with levels more than 2.2mU per L is recommended.

Abstract #218

HEMIBALLISM-HEMICHOREA: A RARE MANIFESTATION OF DIABETIC KETOACIDOSIS

Adam Maghrabi, MD, Omar Akhtar, MBBS, Tipu Faiz Saleem, MD

Objective: Diabetes Mellitus is a common disorder with multiple complications and varied manifestations including CNS. Acute presentation with Hemiballism-Hemichorea has been described infrequently in the literature usually related to non-ketotic hyperglycemia. We describe here a case of new onset Hemiballism-Hemichorea associated with Diabetic ketoacidosis (DKA).

Case Presentation: A 72 year old male, with history of uncontrolled Diabetes Mellitus Type 2, and multiple past admissions for DKA, presented with uncontrollable left upper and lower extremity jerking movements for 2 days. Blood sugar was 871mg/dl, Anion gap was 25, Bicarbonate was 14, and HgbA1C 14.9. The patient was admitted to the MICU for management of DKA. Exam was unremarkable for any focal deficit, but showed left upper and lower extremity choreiform movements. CT scan of the head revealed an increased signal in the right basal

ganglia. There was no evidence of CVA, even on repeat imaging. MRI was not performed due to the presence of his pacemaker. EEG was negative. Aggressively treated with intravenous insulin and rehydration and Clonazepam. His symptoms significantly improved and then resolved with normalization of blood glucose levels. A diagnosis of Hemiballism-Hemichorea secondary to hyperglycemia was made after ruling out other secondary causes.
Discussion: Hyperglycemia, mostly non-ketotic, has been associated with Hemiballism-Hemichorea, which in rare instances, has been described with DKA as well. The pathophysiology is not well understood, many theories have been suggested, including ischemia, calcification, osmotic demyelination syndrome, and genetic predisposition. Blood glucose control has been associated with rapid resolution of the Hemiballism-Hemichorea in most reported cases, but rarely, it could persist for several months.
Conclusion: Hemiballism-Hemichorea is a rare manifestation of hyperglycemia. Hemiballism-Hemichorea in this setting can be usually reversed by optimizing glycemic control. Further research is needed to identify the underlying pathophysiological mechanism.

Abstract #219

DIABETIC FOOT CARE: AWARENESS AND PRACTICE AMONG PATIENTS ATTENDING A TERTIARY HOSPITAL IN LAGOS, NIGERIA

Oluwatosin Kayode, MBBS, Oluwakemi Odukoya, Ifedayo Odeniyi, MBBS, MWACP, FMCP, Olufemi Fasanmade, MBBS, FWACP, Augustine Ohwovoriole

Objective: To assess the awareness and practice of diabetic foot care among patients attending the diabetic clinic of Lagos University Teaching Hospital
Methods: A cross-sectional descriptive survey was carried among patients attending the diabetic clinic from January to March 2011 using a systematic random sampling method. Four hundred and nineteen patients were selected and interviewed using a pre tested interviewer administered questionnaire after written informed consent.
Results: The age of the patients ranged from 25-84 years with a mean of 55.2±12.5 years. There were more females (62%) than males 38%. Most of them were married (65%) with a mean year of diagnosis of 8.4± 6.5 years. Almost all the patients (97.1%) were aware that they needed to examine their feet regularly. However, 28.5% of them said that they never checked their feet, another 26.8% did so only occasionally. Only 32% said that they did so always. Similarly, almost all (96.4%) of them were aware that it is important for people with DM to wear the correct type of foot wear. However 8.1% said that they never wear the correct type of footwear while 46.8% did so only occasionally.
Discussion: Diabetic foot disease is a common complication of diabetes mellitus and is associated with significant morbidity and mortality among DM patients in Nigeria. Unfortunately podiatry services are almost absent in Nigeria. Increased awareness and practice of diabetic foot care is important in the prevention of DM foot disease for the promotion of optimal diabetic outcome.
Conclusion: There is a huge gap between awareness and practice of DM foot care. Possible barriers to adherence to optimal DM foot care should be explored in order to reduce the prevalence of DM foot as a complication of diabetes Mellitus.

Abstract #220

U-500 REGULAR INSULIN USE IN TYPE 2 DIABETIC PATIENTS: A RETROSPECTIVE STUDY

Allison Galloway, DO, Madona Azar

Objective: To evaluate the impact of U-500 insulin use on glycemic control, weight, and total daily insulin dose in patients with type 2 diabetes and severe insulin resistance.
Methods: A retrospective chart review of all patients followed at the Harold Hamm Diabetes Center in Oklahoma City who were transitioned to U-500 insulin due to severe insulin resistance (total daily dose>200 units) from May 2010 until present was performed. Patients for whom we had data before and three months after initiation of U-500 were included. The total daily dose of insulin, weight, and HbA1c were obtained by chart review and the average difference in these data before the initiation of U-500 and three months after the initiation of U-500 insulin were calculated and compared.
Case Presentation: Fifteen patients met inclusion criteria. After transition to U-500 insulin from other forms of insulin, mean reduction in HbA1c was 1.2% (± 2.02%) after three months of therapy. Thirteen of the fifteen patients gained weight and the average weight gain was 10.2 pounds (± 5.6 pounds). The total daily dose of insulin increased in 9 patients by an average 181 units per day and decreased in 5 patients by an average of 82 units per day. There was no report of severe hypoglycemia leading to an emergency room visit in any patient and no noted increased frequency of hypoglycemia reported by patients after switching to U-500 insulin.
Discussion: The number of patients in the United States with severe insulin resistance is growing as the population becomes increasingly obese. As insulin doses increase, adherence, injection site pain/leakage, and cost all become concerns. The use of more concentrated insulin formulations could help overcome some of those issues.

The use of U-500 insulin increased by 137% from 2007 to 2009 as clinicians, particularly diabetologists, became more familiar with its use. Previous algorithms have suggested starting U-500 insulin when a patient requires >200 units of insulin/day and his/her HbA1c is >8.5%. Despite these recommendations, physicians are often hesitant to initiate U-500 insulin because of lack of familiarity with this specific insulin, as well as concern for increased frequency of hypoglycemia. Although no large randomized clinical trials have been published, several case series and reports have shown improvement in HbA1c and lipids with the use of U-500, along with proving its safety when adequate instruction is given to patients. Our results support these findings.
Conclusion: In severe insulin resistance, U-500 insulin is a safe and effective option. Despite a significant improvement in glycemic control, it may promote weight gain and patients need to be educated on adequate dietary intake.

Abstract #221

ACHIEVING TIGHT GLYCEMIC CONTROL SAFELY IN PATIENTS WITH CHRONIC CRITICAL ILLNESS (CCI) USING A SUBCUTANEOUS INSULIN PROTOCOL

Rifka Schulman, MD, Chenbo Zhu, James Godbold, Jeffrey Mechanick, MD, FACP

Objective: Tight glycemic control (blood glucose [BG] 80-110 mg/dL) in the ICU using IV insulin has been associated with reduced morbidity and mortality in critically ill patients when rates of severe hypoglycemia (BG < 40 mg/dL) are low. The purpose of this study is to determine if tight glycemic control can be safely achieved using a sq insulin protocol in patients with Chronic Critical Illness (CCI; tracheostomy placed).
Methods: This is a retrospective case series of all patients admitted to The Mount Sinai Hospital Respiratory Care Unit (RCU) from June-December, 2010. The RCU uses point-of-care BG testing every 6 hours and multiple daily sq insulin injections with combinations of rapid, intermediate, and long acting insulin based on the total daily dose of insulin. Daily titrations of insulin are guided by an endocrinologist to target BG 80-110 mg/dL. BG levels were analyzed with respect to mean ± SD, hypoglycemia, and glycemic variability.
Results: Mean blood glucose (n=58) decreased from 139.1 mg/dL in the first 72 hours of the RCU stay to 128.4 mg/dL during the remainder of the RCU stay (P=0.001). Median incident rate of BG >180 mg/dL decreased from 0.129 in the first 72 hours to 0.065 thereafter (P=0.0114), and showed a decreasing trend over time in the RCU (P=0.0018). Although individual standard deviation (SD) of blood glucose values, a measure of glycemic variability, did not change significantly, group SD values decreased from 35.7 in the first 72 hours to 21.0 thereafter. The rate of severe hypoglycemia (BG < 40 mg/dL) was zero in this group for all time intervals. The ratio of incident blood glucose 40-59 mg/dL was 0.0053 in the first 72 hours and zero thereafter. A positive non-significant association was noted between glucose SD and length of stay in the RCU (Spearman correlation coefficient 0.166, P=0.19). A higher but non-significant mean Wilcoxon score for glucose SD was noted in patients expired in the RCU compared to those discharged alive, on or off the ventilator (40.67, 29.00, 30.53 respectively, P=0.18).
Discussion: A sq insulin protocol can be used to safely achieve tight glycemic control in CCI. The RCU CCI protocol involves less invasive and costly glucose monitoring, with only four daily point-of-care glucose values. Decreased BG SD reflects improved glycemic variability. While associations of BG means and SDs with RCU outcomes did not reach statistical significance in this ongoing study, a power analysis has demonstrated a minimum N of only 162 to reduce type-II error.
Conclusion: A sq insulin protocol utilizing 4 point-of-care glucose values daily can successfully achieve tight glycemic control while minimizing rates of hypoglycemia in CCI.

Abstract #222

PATTERN OF CRITICAL CORONARY ARTERY STENOSIS IN DIABETIC VERSUS NON-DIABETIC PATIENTS

Remya Tharackal Ravindran, MBBS, Sowjanya Bhagavatula

Objective: To analyze the pattern of critical coronary artery stenosis in diabetic vs. non-diabetic patients.
Methods: A retrospective, comparative observational study of patients who underwent PCI in a community hospital setting between years 2000 and 2010 was conducted. Data was collected from patients' initial PCI procedure. Patient demographics, critically stenosed coronary arteries (>70%), maximally stenosed coronary artery and the degree of maximal stenosis were noted. Percentages were compared using the z-test and means; with the t-test.
Results: PCI records of 250 random patients were analyzed. Patients with noncritical stenosis (n=23) and those with no record of their initial PCI (n=31) were excluded. Of the 196 included patients, 57% (112/196) were diabetic (DM) and 42% (84/196) were non-diabetic (NDM) {Mean age: 67.4±11.2 vs. 70.8±11.7 years;

p=0.08}. Diabetic females underwent first-time PCI at a younger age than non-diabetic females (mean age: 67.2±12.4 vs. 73.5±11.7 years; p=0.01). The left anterior descending artery (LAD) was the maximally stenosed one in both groups {59% (67/112) vs. 42% (36/84); p=0.01}. The degree of LAD stenosis was higher in the DM group (90.6±8% vs. 86.5±8%; p=0.0006). Right coronary artery (RC) was the next maximally stenosed one in both groups. However, greater proportion of non-diabetics had RC as the maximally stenosed artery {39% (33/84) vs. 23% (23/112); p=0.01}. The degrees of RC stenosis were not significantly different (90.3±9.1% vs. 88±9.6%; p=0.09). Similar proportion of diabetics and non-diabetics had circumflex artery (Cx) as the maximally stenosed one {15% (17/112) vs. 17% (15/84); p=0.7}. The degrees of Cx stenosis were not statistically different {90.1±8.9% vs. 89.7±6.7%; p=0.72}. Left main coronary artery was the maximally stenosed one in only two diabetic patients (2/196). There was no statistical difference between the proportion of non-diabetics with LAD vs. RC involvement {42% (36/84) vs. 39% (33/84); p=0.5}. However, the proportion of diabetics with LAD vs. RC involvement differed significantly {59% (67/112) vs. 23% (23/112); p<0.0001}. Frequency of multi-vessel critical stenosis did not differ significantly between the groups {54% (62/112) vs. 45% (39/84); p=0.2}.

Discussion: Over 228,000 diabetic patients undergo percutaneous coronary intervention (PCI) per year in the U.S. A review of medical literature revealed few studies that assessed the pattern of coronary artery involvement in diabetics undergoing PCI.

Conclusion: LAD was the maximally stenosed artery in diabetics as well as non-diabetics, followed by RC and Cx. Non-diabetics had near equal involvement of both LAD and RC; however diabetics had a greater propensity for LAD involvement.

Abstract #223

CAROTID INTIMA MEDIA THICKNESS IN TYPE-2 DIABETES MELLITUS WITH ISCHEMIC STROKE

Sunil Kota, MD, Siva Kota, Svs Krishna, Lalit Meher, Kirtikumar Modi

Objective: To find cut off point for carotid intima media thickness (CIMT) for ischemic stroke in type 2 diabetes mellitus(T2DM) patients.

Methods: 80 subjects in the age group of 30-75 years (M: F= 57: 23) were selected and divided into 3 groups A) subjects with T2DM and ischemic stroke, B) subjects with only T2DM and C) healthy subjects. Patients with cardio-embolic stroke, hemorrhagic stroke and stroke secondary to trauma, impaired coagulation or tumor were excluded. All the participants were subjected to B-mode ultrasonography of both common carotid arteries to determine CIMT along with history taking, physical examination and routine laboratory investigations including included FBS and PPBS, HbA1C, renal profile, lipid profile and microalbuminuria.

Results: 40 patients (M: F= 30: 10) were selected in group A with mean age 60.4 ± 10.2 years. Group B had 20 patients (M: F=12: 8) with mean age 56.8 ± 11.7 years and group C had 20 subjects (M: F= 15: 5) with mean age 51.3 ± 16.7 years. CIMT value greater than 0.8 mm was found to be associated with occurrence of stroke in group A subjects. Patients with T2DM with or without ischemic stroke were found to have significantly higher prevalence of increased CIMT (92.5 % in group A, 80% in group B and 20% in group C). The mean CIMT of study population was 0.84 ± 0.2 mm (Group A- 1.06± 0.2mm, Group B- 0.97± 0.26 mm and Group C- 0.73± 0.08 mm). The mean CIMT was not significantly different between T2DM patients with or without ischemic stroke (p-0.08). Type I variety (plaque with thin rim over the surface, but predominantly anechoic) was the most common among subjects with T2DM and ischemic stroke. However, the mean CIMT was significantly higher in diabetic subjects with and without stroke compared to healthy subjects (group A versus group C, p-0.003, group B versus group C, p- 0.03, combined group A & B versus group C, p- 0.006). Other parameters like higher age, smoking, hypertension, hyperlipidemia, low HDL cholesterol, the glycemic parameters, microalbuminuria and the duration of diabetes were independently and significantly related to CIMT.

Discussion: CIMT is used as a noninvasive tool for assessment of atherosclerosis. Rotterdam study has demonstrated CIMT > 1.2 mm as a risk for stroke in middle aged adults. CIMT is demonstrated as a surrogate marker for subclinical coronary artery disease (CAD) in diabetic patients. Our study highlights CIMT > 0.8 mm as a risk factor for occurrence of stroke in T2DM. T2DM patients with or without stroke are at equal risk for development of stroke, as evidenced by presence of CIMT > 0.8 mm.

Conclusion: A high CIMT (>0.8 mm) is a surrogate and reliable marker of higher risk of ischemic stroke amongst type 2 diabetic patients.

Abstract #224

ETIOPATHOGENETIC ASSOCIATION OF COEXISTING DISEASES IN TYPE 1 DIABETES MELLITUS

Sunil Kota, MD, Siva Kota, Svs Krishna, Lalit Meher, Kirtikumar Modi

Objective: Indian data citing association of type 1 diabetes mellitus (T1DM) with other diseases is scarce. We hereby profile the clinical association of such diseases among patients from our centre.
Methods: Consecutive patients of T1DM presenting to department of Endocrinology from May 1997 to December 2011 were retrospectively analyzed in context of associated clinical profile with reference to etiopathogenetic correlation.
Results: Among 260 patients diagnosed as T1DM, 21 (8%) had hypothyroidism, 4 (1.5%) had hyperthyroidism and 2 (0.7%) had primary adrenal insufficiency. Eighteen patients (7%) had celiac disease, 9 (3.5%) had Turner's syndrome, 5 patients (1.9%) had Klinefelter's syndrome, whereas Down's syndrome and Noonan's syndrome was present in 2 and 1 patients (0.7%) respectively. One patient each had Wolframs' syndrome and myasthenia gravis. Systemic lupus erythematosus and Rheumatoid arthritis were present in 3 and 1 patients respectively. Alopecia and vitiligo were present in 2 and 3 patients respectively. Total of 5 patients with cerebral palsy, 4 cases with deaf mutism, 4 cases with acute psychosis and 16 patients with depression were noted. Mean age of study patients was 20.8 ± 9.8 years (range, 3 to 23 years).
Discussion: Genetic predisposition, autoimmunity and viral infections are the main etiopathological factors implicated in the pathogenesis of T1DM. These coexisting diseases are attributed to organ specific antibodies like thyroid peroxidase, thyroglobulin with autoimmune thyroid disorders, endomysial antibody and tissue transglutaminase with celiac disease, muscarinic receptor with myasthenia gravis and 21- hydroxylase for addison's disease. Similarly patients with genetic disorders like Turner's syndrome, Down's syndrome, Klinefelter's syndrome etc are more prone for development of T1DM. Coexistence of T1DM with Noonan's syndrome is not reported yet. Since viral infections like coxsackie B, hepatitis C and HTLV-1 have been implicated in the etiopathogenesis of T1DM, there could be associated neurological disorders of probable or confirmed viral etiology with T1DM. Some such slow virus diseases can give rise to cerebral palsy or deaf-mutism in T1DM.
Conclusion: Various genetic, autoimmune and central nervous system diseases were the associated diseases encountered in our patients. These coexisting conditions render the care of diabetic patients more difficult and challenging. Routine screening is required for early diagnosis and treatment of associated co morbidities.

Abstract #225

ILEAL INTERPOSITION WITH SLEEVE GASTRECTOMY/ DIVERTED SLEEVE GASTRECTOMY FOR TREATMENT OF TYPE 2 DIABETES

Sunil Kota, MD, Surendra Ugale, Neeraj Gupta, Siva Kota, Kvs Kumar, Kirtikumar Modi, Viswas Naik

Objective: We evaluated the efficacy of ileal interposition (II) + sleeve gastrectomy (SG) / diverted SG (DSG) for control of type 2 diabetes (T2DM) and related metabolic abnormalities.
Methods: II+SG was performed on 43 patients (M: F=25:18). The inclusion criteria were T2DM > 1 year duration, age 25- 70 years, stable weight for 3 months and stimulated C-peptide level >1.5 ng/ml. 17 patients (M:F= 12: 5) were subjected to II+DSG, based on adverse clinical profile like longer duration of diabetes, lower BMI, poorer C-peptide response, requirement of ≥ 3 oral hypoglycemic agents (OHA) ± Insulin (> 100 IU/day), end organ impairment. The primary outcome was remission of diabetes (HbA1C < 6.5 % without OHAs/ Insulin) and secondary outcomes were reduction in OHA requirement and improvement in metabolic profile. Patients were followed up at 3 monthly intervals.
Results: Patients subjected to II+SG had mean age 47.2± 8.2years, DM duration 10.1± 9.2 years and BMI 33.2± 7.8 kg/m2. All patients had poorly controlled T2DM (HbA1C- 9.6 ± 2.1%). 30 (70%) patients had hypertension, 20 (46%) had dyslipidemia and 18 (42%) had microalbuminuria. Mean follow up was 20.2 ± 8.6 months (range: 4-40 months). Postoperatively glycemic parameters improved at all intervals ($p<0.05$). 20 patients (47%) had remission in diabetes and the remaining patients showed significantly decreased OHA requirement. 27 patients (90%) had remission in hypertension. There was a declining trend in lipids and microalbuminuria. Patients with duration of T2DM < 10 years, stimulated C-peptide > 4 ng/ ml and BMI > 27 kg/ m2 performed better. Patients subjected to II+DSG, had mean age 50.7± 8.1 years, DM duration 15.1 ± 5.8 years and BMI 29.2 ± 7.5 kg/ m2. 8 (45%) patients had hypertension, 7 (39%) had dyslipidemia and 7 (39%) had microalbuminuria. Mean follow-up data was 9.1 ± 5.3 months (range: 3-21 months). 12 patients had diabetes remission, and the remaining five showed significantly decreased OHA requirement. 7 patients (87.5%) had remission in hypertension. Significant decline was observed in the glycemic, lipid parameters and microalbuminuria at all intervals ($p< 0.05$). HbA1C reduction was higher than decline in BMI, justifying their weight loss independent glycemic benefits.
Discussion: The surgery addresses the foregut and hindgut mechanisms for DM control. The SG component

restricts calorie intake and induces ghrelin (orexin) loss. II leads to rapid stimulation of interposed ileal segment by ingested food resulting in augmented GLP-1 secretion. Accompanying improvement in hypertension, lipid profile and microalbuminuria justify its metabolic beneficial effects.
Conclusion: II+SG/ DSG can control Type 2 DM and associated metabolic abnormalities.

Abstract #226

MORE PATIENTS WITH DIABETES RESORT TO MEDICAL ALERT TATTOOS AS AN ALTERNATIVE TO METAL MEDICAL ALERTS: A CALL UPON HEALTH ORGANIZATIONS TO DEVELOP REGULATIONS AND PRACTICE GUIDELINES FOR MEDICAL TATTOOING

Saleh Aldasouqi, MD, Crystal Glassy, Matthew Glassy, Nicolas Kluger, Mamata Ojha, Sameer Ansar, MD, Bhavini Bhavsar, MBBS, M.D

Objective: We recently reported a patient with diabetes who elected to replace metal medical alerts with a permanent tattoo on his wrist, for the purpose of identification in emergency situations. We have encountered more patients with diabetes resorting to such technique recently, citing easy breakability of metal medical alerts, and persistently accruing expenses. No clinical practice guidelines are available in regards to this practice, given the potential risks and complications of tattooing, such as risk of transmission of communicable diseases, and the risk of poor healing and local infection in patients with poorly controlled diabetes. We present such a case we encountered recently, to draw the attention of diabetes specialists to this surfacing trend.
Methods: A case presentation.
Case Presentation: A 32 year old woman with type 1 diabetes since age 2, followed in our diabetes center, decided on her own to have a permanent tattoo on her wrist, depicting a traditional medical alert with the six-pointed "Star of Life", "Snake-Staff" and medical identification info. When asked about her rationale, she cited frustration having broken numerous metal necklaces and bracelets, with the accruing cost of the metal alerts, especially the jewelry types.
Discussion: This is the second case we report on patients with diabetes resorting to medical alert tattoos, in addition to few other unpublished cases we encountered. Furthermore, the internet is full of discussions about medical alert tattooing amongst patients with diabetes and their families. However, search of the medical literature retrieved no clinical practice guidelines addressing this piece in diabetes management.
Conclusion: Given the observed trend of patients with diabetes resorting to medical alert tattooing, we suggest that health organizations address this practice.

Abstract #227

INPATIENT MANAGEMENT OF DIABETES MELLITUS AMONG NONCRITICALLY ILL GENERAL MEDICINE PATIENTS AT THE PUERTO RICO UNIVERSITY HOSPITAL.

Rafael Gonzalez-Rosario, MD, Loida Gonzalez, Monica Vega, MD, Viviana Sanchez, MD, Milliette Alvarado Santiago, MD, Myriam Allende-Vigo, MD, MBA, FACP

Objective: To evaluate the adherence to current recommendations presented by the American Association of Clinical Endocrinologists and the American Diabetes Association for the treatment of Diabetes Mellitus (DM) in non-critically ill patients admitted to the Puerto Rico University Hospital.
Methods: In this retrospective observational study conducted at the PR University Hospital, 147 non-critically ill patients admitted to a General Medicine ward from Sep 1st 2010 to Aug 31st, 2011, with DM as a secondary diagnosis, were identified. Clinical data, including bedside glucose measurements and orders related to glucose management, was abstracted for the first 5 days of admission and for the last 24 hrs before discharge.
Results: The average age of patients was 58 ± 12 yrs. Sixty percent of the patients were men and 90% had type 2 DM. The mean glycated hemoglobin on admission was 8.19% with mean bedside glucose level of 226.5 ± 97.7 mg/dL. The mean percent of glucose readings >180 mg/dL per patient persisted above 50% for the first 5 days of admission. Nearly 60% of patients were hyperglycemic (mean bedside glucose > 180 mg/dL) during the first 24 hrs of admission, 64.6% had persistent hyperglycemia throughout the first 5 days, and 54.2% were hyperglycemic during the last 24 hrs. The mean last glucose value before discharge was 189.6 ± 73 mg/dL. The rate of hypoglycemia was low, 2.8%, as compared to the rate of hyperglycemia (56.7%). Regarding DM management, the mean total daily insulin per patient increased significantly in the first 5 days. The percent of patients prescribed basal/bolus therapy in the uncontrolled group (mean glucose >180 mg/dL) increased from 45% on admission to 63% in the last 24 hrs. However, there were still 37% of uncontrolled patients who remained with only basal therapy. In this same group, 64.2% of patients had an

increase in the insulin dose in the first 5 days of admission as compared to 15.4% in the controlled group. However, 28.4% of patients with uncontrolled glucose levels had no change in insulin dose and 7.4% actually had a decrease in the dose of insulin administered. Only 2.1% of patients in the uncontrolled group had an Endocrinology consult requested.

Discussion: The majority of patients presented uncontrolled DM upon admission and persistent hyperglycemia during hospitalization. Although most of the uncontrolled patients had their insulin regimen optimized, a significant portion had no change or even a decrease in insulin dose.

Conclusion: These findings are consistent with the phenomena of clinical inertia and negative therapeutic momentum previously reported in the inpatient management of DM and provide possible targets to improve inpatient glycemic control.

Abstract #228

INPATIENT BLOOD GLUCOSE CONTROL BEFORE AND AFTER IMPLEMENTATION OF AN INSULIN ORDER SET TO THE ELECTRONIC MEDICAL RECORD

Karla Arce, MD, Arnaldo Villafranca, Marlow Hernandez, Carmen Villabona

Objective: Poor glycemic control has been associated with adverse outcomes in hospitalized patients. We aimed to determine if implementation of a subcutaneous insulin order-set in the electronic medical records (EMR) will improve glycemic control and reduce incidences of hypoglycemia in the inpatient setting.

Methods: We conducted a retrospective study in our institution evaluating glucose readings in 256 randomly selected diabetic patients in the general wards from December 2007 to March 2011. The pre-intervention group was composed of 34 patients, 730 blood glucose values from December 2007 to March 2008 and the post-intervention group was composed of 222 patients, 2967 blood glucose values from Dec 2010 to March 2011. The glucose readings were subdivided as follows: hypoglycemic readings (<80), euglycemic readings (between 80 and 180), hyperglycemic readings (>180); and frequency of HbA1C testing. The results were analyzed using an independent sample t-test and chi square analysis.

Results: The pre-intervention group had a hypoglycemic rate of 9.6% compared to the post-intervention group with 3.8% ($p<0.05$) (95% CI: 0.27-0.51). Euglycemia in the pre-intervention group occurred 60% of the time vs. 65% ($p<0.05$) (95% CI: 1.01-1.41) in the post-intervention group. There was no statistical difference between the pre- and post-intervention groups in relationship to hyperglycemia and frequency of HbA1c measurement. The number needed to treat in order to prevent one hypoglycemic episode was 17, and the number needed to treat in order to increase euglycemic episodes by 1 was 24.

Discussion: Hypoglycemia has been associated with poor patient outcomes in both general wards and intensive care units. Patients with hypoglycemia have longer hospital stays and increased mortality both during and after admission. In addition, hypoglycemia has been strongly associated with mortality in patients with sepsis, bacteremia, liver disease, renal insufficiency and in elderly patients hospitalized with pneumonia. Studies have suggested that the benefits of tight glycemic control may be partially offset by the increased risk of hypoglycemia, therefore identifying at risk groups is crucial so that they can be monitored closely. The development of insulin order sets and documentation tools has had a dramatic effect on decreasing the incidence of hypoglycemia, improve glycemic control, increase adherence to evidence-based practices, and improve clinical outcomes in the hospital.

Conclusion: Implementation of the new insulin order-set within the EMR has led to significantly less hypoglycemic episodes (about 6%) and significantly more euglycemic episodes (about 4%) in the hospitalized patients on the general wards.

Abstract #229

PREVALANCE OF UNDIAGNOSED DIABETES IN HOSPITALIZED PATIENTS.

Karla Arce, MD, Divya Yogi-morren, M.D., Marlow Hernandez, Carmen Villabona

Objective: To determine the prevalence of undiagnosed diabetes and prediabetes in hospitalized patients determined by elevated random blood glucose levels of more than 140 mg/dl and an HbA1c > 6.5% for diabetes and HbA1c 5.7-6.4 for prediabetes.

Methods: Inpatient records from our institution, for the period of January 2009 to September 2011, were retrospectively reviewed. The study sample was composed of a random sample of inpatients without a diagnosis DM. We included male and female patients, age > 18 admitted from June 2009- June 2011. Patients with a diagnosis of DM, cancer, pregnancy, and glucocorticoid treatment were excluded. The collected variables included minimum blood glucose reading, maximum blood glucose reading, HbA1c (if obtained), and whether they were subsequently diagnosed with DM. Descriptive statistics were used to describe the study population with regards to HbA1C testing and DM diagnosis. A t-test was used to differentiate diabetics from non-diabetics with regards to minimum

and maximum blood sugars.

Results: A total of 3,321 randomized patients without a pre-hospitalization diagnosis of DM were analyzed. HbA1C was checked in 11% (N = 30) of patients with blood glucose > 140 (N = 286), compared to 0.3% (N = 8) in those with blood glucose less than 140 (N = 3036). 53% of patients (N = 16) who had an HbA1C checked had DM and 20% of patients (N= 6) had prediabetes.

Discussion: It is well known that patients with acute illness without a diagnosis of diabetes mellitus (DM) may develop stress hyperglycemia while hospitalized. Additionally, patients with newly diagnosed DM have a significantly higher mortality and a lower functional outcome than patients with a known history of DM or normoglycemia. Some studies have found that up to 18% of hospitalized patients had an elevated HbA1c of more than (>) 6.1 % without a prior diagnosis of DM. Our study was designed to identify this population of patients whose hyperglycemia may represent DM and to determine the percentage of these patients that have their HbA1c checked, thus enabling a diagnosis of DM to be made prior to discharge.

Conclusion: This study shows that 11 % of hospitalized patients with hyperglycemia had an HbA1c checked. Of the small proportion of patients tested, more that half received a diagnosis of diabetes and 20% had prediabetes. We understand that HbA1c testing in hospitalized patients has limitations. However, we must recognize the importance of ordering it in selected patients with hyperglycemia to diagnose DM early and to be able to institute proper follow up and treatment to prevent complications.

Abstract #230

PREVALENCE OF RISK FACTORS FOR GLUCOSE INTOLERANCE IN SOUTH EAST NIGERIA

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Objective: To determine the prevalence of risk factors for glucose intolerance among the inhabitants of a coastal Nigerian city.

Methods: In a cross sectional survey, a sample comprising 1134 subjects (645 males and 489 females) representative of the entire population of Calabar aged 15-79 was studied. Using the WHO STEPS instrument, information obtained included anthropometric indices (height in meters, weight in kilogram, waist circumference in centimetre, Hip circumference in centimetre), Blood Pressure in mmHg and Plasma glucose in mg/dl. An oral glucose tolerance test (OGTT) was performed on all participants. Waist circumference (WC) ≥ 102cm for males and ≥ 88cm for females defined Truncal adiposity. Body mass index (BMI) ≥ 30kg/m2 was regarded as generalised adiposity.

Results: The risk factors that were significantly associated with glucose intolerance were advancing age, WC, BMI and smoking. Others were physical inactivity, family history of diabetes mellitus and hypertension. The prevalence of hypertension was 33.3% (P<0.01), smoking 14.6% (P<0.01), Truncal obesity 38.8% (P<0.01), generalized adiposity 27.4% (P<0.01). Males had a higher mean waist circumference than females. Males had a higher prevalence of hypertension (37.2%) compared to (28%) in females. About 17.9% of all subjects with glucose intolerance were hypertensive. The mean BMI of the subjects was 27.6 ± 4.9. Females had a higher BMI than males.

Discussion: High prevalence of the risk factors for glucose intolerance has been reported in other studies and the odds for developing glucose intolerance increases with most of the modifiable risk factors for glucose intolerance including cigarette smoking, obesity and hypertension. The odds ratio for developing glucose intolerance was 1.02 times more for every year increase in age. The odds for developing glucose intolerance when physically active was halved.The odd ratio for developing glucose intolerance with cigarette smoking in this study was 1.5 times higher.

Conclusion: The prevalence of risk factors for glucose intolerance among residents of Calabar, South East Nigeria appears higher than the previous national prevalence.

Abstract #231

PREVALENCE OF ANTI-GAD ANTIBODIES IN PATIENTS WITH PANCREATIC DIABETES MELLITUS

Soni Srivastav, MD, Fernando Ovalle, MD

Objective: Pancreatic Diabetes (DM Type 3c) is an increasingly recognized phenomenon of DM due to inherited or acquired pancreatic disease (eg cystic fibrosis, acute or chronic pancreatitis) or pancreatic resection (total or partial). Anti-GAD antibodies (ab) are thought to be highly specific for DM Type1a. We set out to determine the prevalence of anti-GAD ab in patients with pancreatic form of DM.

Methods: In this study 1257 consecutive new patients referred for DM evaluation to our outpatient DM specialty clinic were evaluated prospectively and systematically. Clinical evaluation and biochemical testing was used to categorize specific subtypes of DM. These included 32 patients with DM secondary to a variety of pancreatic pathology, who were determined to have pancreatic DM. Patients with DMT1a and DMT2 were considered as

controls.

Results: In our study population 32 patients were determined to have pancreatic DM: 10 patients with cystic fibrosis (CF), 10 patients with chronic pancreatitis, 7 patients with pancreatectomy (total or partial), and 5 patients were labeled with 'mixed' diabetes (chronic pancreatitis and preexisting diabetes type 2). We also studied 222 patients with DM Type 1 and 614 patients with DM Type 2. The prevalence of Anti-GAD ab in these patients was as follows: in Type 1 DM 50% (110/222 patients), Chronic pancreatitis 30% (3/10 patients), status post pancreatectomy 14% (1/7), CF 10% (1/10), mixed (0%, 0/5) and Type 2 DM 4.2% (26/614).

Discussion: Antibodies to GAD are found in about 70% of type 1 diabetics at the time of diagnosis with a tendency to decrease with time after the onset of autoimmune destruction. The prevalence of Anti-GAD in the general population has been reported to be from 0.4-2.97%. Prevalence in first degree relatives of type 1 diabetics has been reported as 5-13%. Hardt et al also found that patients with non-alcoholic chronic pancreatitis had an increased prevalence of anti-GAD ab (8.3%) compared to normal subjects (2.5%). In our study we found that patients with pancreatic DM due to a variety of pathology had a higher prevalence rate of positive Anti-GAD ab. We speculate that various degrees of pancreatic cell damage expose cytoplasmic elements such as GAD to the immune system during non-autoimmune destruction of the islet cells in these forms of pancreatic injury. The presence of anti-GAD ab is likely only an epiphenomenon and unlikely to be playing a role in etiopathogenesis of pancreatic DM.

Conclusion: Our data suggests that anti-GAD ab may be positive in greater numbers than previously thought in pancreatic disorders other than DM Type1. This is important to keep in mind to avoid misinterpreting a positive anti-GAD ab in this patient population.

Abstract #232

THE IMPACT OF DIABETES ON LENGTH OF STAY AND HOSPITAL COSTS AFTER ELECTIVE SURGICAL PROCEDURES

M. Figaro, MD, Kyoungrae Jung, Dooyoung Lim, Rhonda BeLue

Objective: Longer length of stay (LOS) after elective surgery is associated with an increased use of health care resources and higher costs. The objectives of this study were to compare LOS after 13 major elective procedures between those with diabetes (DM) and those without and to test the hypothesis that comorbidities and perioperative complications predict greater costs and longer LOS for these procedures among diabetic patients.

Methods: Using extant data on 29,589 patients with DM and 793,827 patients without DM from the 2008 Healthcare Cost and Utilization Project (HCUP), Nationwide Inpatient Sample; thirteen elective operations were evaluated including thyroidectomy, hysterectomy, mastectomy, colectomy, cholecystectomy, amputations, fracture repairs and total joint replacements.

Results: Patients with DM were more likely to have HTN prior to surgery (72.7% vs. 46.1%) and had more chronic conditions on admission (mean of 7.6 vs. 3.6 diagnoses). There were significant differences between those with DM and those without in terms of mortality rate (1.42% vs. 0.93%); MI (1.6% vs. 0.59%), pneumonia (2.88% vs. 0.32%) or acute kidney injury (11.2 vs. 2.53%) respectively ($p > 0.0001$). There were significant increases in length of hospital stay (mean, 9.08 vs. 4.76 days; $p < 0.0001$) and hospital costs per patient ($19,547 vs. $15,873; $p < 0.0001$) for DM vs. non-DM and associated older age. Overall, DM costs were higher except for mastectomy ($9,210 vs. $9,722, $p=0.41$) and amputations (19,812 vs. 25,045 [non-DM], $p<0.0001$).

Discussion: Diabetes remains a morbid condition for those undergoing many elective procedures and is associated with increased length of stay and costs. For both common (joint replacement) and uncommon procedures (thyroidectomy), diabetic patients incurred greater rates of complications, had longer LOS and associated hospital costs. National policy initiatives will soon bundle payments for many elective procedures. This will likely compel healthcare systems to reevaluate their protocols and improve preoperative control of diabetes in order to lower the costs of elective surgery.

Conclusion: Patients with diabetes mellitus experienced significantly higher rates of surgical and systemic complications, higher mortality, and increased length of stay during hospitalization following 11 major elective procedures.

Abstract #233

DIABETIC KETOACIDOSIS -A PROTHROMBOTIC STATE-A LESSER KNOWN FACT

Pratik Dalal, MBBS, Divyashree Varma, MBBS

Objective: Diabetic ketoacidosis, a commonly encountered complication of insulin-dependent diabetes mellitus, has been found to be a prothrombotic state. This complication of thrombosis is more commonly seen in children and adolescents, but can sometimes overflow into the adult population. Though venous thrombosis is more common, cases of arterial thrombosis are known.

We present a case of an adult type 1 diabetic patient who, after presenting with DKA, went on to have thrombosis of his celiac and renal vasculature along with multiple pulmonary emboli.

Case Presentation: A 21 year old caucasian insulin dependent diabetic male, was admitted for DKA. Patient had historically been known to be complacent with his insulin regime with resultant caustic effects of hyperglycemia obvious in the form of neuropathy and gastroparesis. The DKA subsequently, resolved; however, patient remained nauseous and tachycardic , vomiting feculent material. Decompression of the GI tract with NG aspiration was urgently done. Abdominal X-ray showed gastric pneumatosis which needed to be differentiated from free air under the diaphragmn. Emergent surgery consult was placed for fear of gastric wall necrosis and possible perforation. CT scan of the abdomen showed thrombosis of common iliac vein, external iliac veins, left renal vein and the left gastric artery. CT scan of the thorax showed right segmental pulmonary emboli.

Discussion: The unfortunate patient above not only had DKA, but he also presented with gastric pneumatosis, and multiple venous and arterial thrombi. We urge physicians to keep in minds that DKA is prothrombotic state which can lead to dire circumstances if ignored. There are several reasons why DKA can lead to systemic thromboses. First, DKA is a hyperosmolar state. Both an elevated serum glucose and serum sodium increase the serum osmolarity causing hyperviscosity and slugglish blood flow leading to thrombosis formation. Second, a recent study showed that protein C activity was significantly decreased in DKA but normalized after treatment. However, protein S activity remained diminished despite treatment. vWF activity was increased in the first 120 hours of treatment. Among 2859 patients with diabetes and hyperosmolarity, 34 (1.2%) developed VTE during the hospitalization and 14 (0.5%) developed VTE within 91 days after discharge.

Conclusion: The risks are higher when central lines are used, which may be the case in an ICU setting. Therefore, shortness of breath and abdominal pain may be ominous signs in a patient presenting with DKA.

Abstract #234

RECURRENT DIABETIC MYONECROSIS PRESENTING AS FOOT PAIN: ROLE OF IMAGING IN DIAGNOSIS.

Richard Elias, MBBS, Norman Egger, Kimberly Amrami, Diana Dean, MD

Case Presentation: A 23-year-old woman with Type I diabetes mellitus presented to the emergency department with four days of left foot pain worsened by weight-bearing. Examination revealed erythema along the lateral aspect of her left foot and tenderness to palpation over this area and the anterior edge of calcaneus. Laboratory studies showed leukocytosis of 11.8 x 10(9)/L. HBA1c was 9.9%. X-ray was negative. MRI demonstrated abnormal signal and loss of normal muscle anatomy involving the abductor digiti minimi muscle. The myotendinous complex was intact and the muscle itself not disrupted. Findings were consistent with diabetic myonecrosis (DM). The patient had presented to medical care four years previously with right foot pain and erythema, underwent a foot x-ray which was unremarkable and was treated with oral and then IV antibiotics for presumed cellulitis. She represented to a physician with persistence of her symptoms. She underwent MRI which showed abnormal signal throughout the adductor hallucis muscle extending from the calcaneus to the base of the 1st metatarsal on the right. On both occasions the diagnosis of DM was made based on MRI without muscle biopsy and was managed conservatively.

Discussion: DM was first described in 1965 in two diabetic patients who had excision of thigh masses believed to be soft-tissue sarcoma. It is a rare manifestation of poorly-controlled diabetes mellitus which usually affects the thigh or less commonly the calf. This is the first reported case involving the feet. MRI is the principal imaging modality. The findings include isointense enlargement of the involved muscle on T1 imaging with obscuration of the fascial planes. On T2 images, affected muscle demonstrates increased signal intensity as well as surrounding edema. The major differential is infection such as pyomyositis. Findings suggestive of infection include prominent fluid collections demonstrating low signal intensity on T1 sequences and high-intensity on T2 or cellulitis involving the overlying hypodermis. Infection is often associated with reactive inflammatory changes in adjacent muscle groups while in DM abnormalities are generally limited to discrete groups.

Conclusion: DM is an uncommon condition generally affecting poorly-controlled diabetic patients. It is likely underdiagnosed or, as in our case, misdiagnosed as infection due to pain, erythema and leukocytosis. MRI is the principal imaging modality currently utilized for the diagnosis of DM. Clinicians should be mindful of this condition in consideration of diabetic patients who present with localized soft tissue pain or swelling.

Abstract #235

DAPAGLIFLOZIN CONSISTENTLY REDUCES HBA1C IN PATIENTS WITH TYPE 2 DIABETES MELLITUS: POOLED SUBGROUP ANALYSIS OF INTERACTION BETWEEN BASELINE PARAMETERS AND HBA1C ACROSS 9 CLINICAL TRIALS

Elise Hardy, Afshin Salsali, Li Wei, Traci Mansfield, Catrin Wessman, Tjerk de Bruin, Shamik Parikh

Objective: Dapagliflozin (DAPA), a selective inhibitor of sodium glucose cotransporter 2 (SGLT2), reduces plasma glucose independently of insulin secretion or action by increasing the excretion of excess glucose. This report analyzed glycated hemoglobin (HbA1c) data pooled from 9 double-blind, randomized clinical trials of DAPA in patients with type 2 diabetes mellitus (T2DM) to determine if reduction of HbA1c is dependent on baseline parameters.
Methods: Patients with T2DM (N=4047) received DAPA 2.5, 5, or 10 mg/d or placebo for 24 wk as monotherapy (NCT00528372, NCT00736879) or as add-on to metformin (NCT00528879, NCT00855166), glimepiride (NCT00680745), pioglitazone (NCT00683878), insulin (NCT00673231), or as an initial combination with metformin (NCT00859898, NCT00643851). Adjusted mean change from baseline in HbA1c, excluding data after rescue (LOCF), was analyzed by an ANCOVA model with treatment group, subgroup, and study as categorical factors, and interaction between treatment group and subgroup, baseline HbA1c, and study-by-baseline HbA1c interaction as continuous covariates. Treatment by subgroup interactions were analyzed for baseline HbA1c, estimated glomerular filtration rate (eGFR), age, gender, race, ethnicity (US only), geographic region, body mass index (BMI), T2DM duration, and combined eGFR and age. *P* values are reported for subgroup interaction based on average treatment effect relative to placebo; a *P* value <0.1 indicated a potential interaction.
Results: Control-corrected mean changes from baseline in the individual studies for HbA1c in the DAPA treatment groups (range, %) were: monotherapy, -0.35 to -0.84; +metformin, -0.28 to -0.54; +glimepiride, -0.44 to -0.68; +pioglitazone -0.40 to -0.55; +insulin, -0.45 to -0.60, and initial combination with metformin, -0.54 to -0.70. No interaction of change from baseline in HbA1c was detected for gender, race, ethnicity, region, BMI, or T2DM duration. Interactions were detected for baseline HbA1c ($P<0.0001$), eGFR category ($P=0.015$) and age ($P=0.054$).
Discussion: To determine whether the interaction observed for the age subgroup analysis was still present after controlling for baseline eGFR, an analysis was performed to evaluate the relationship between HbA1c and the joint effects of age and degree of renal impairment. The interaction *P* value was -0.29, suggesting that such a systematic difference in efficacy between age groups after controlling for eGFR, could not be established.
Conclusion: In conclusion, DAPA provides consistent efficacy across a range of subgroups of patients with T2DM. The magnitude of the reduction in HbA1c with DAPA is influenced by baseline HbA1c and eGFR, a determinant of the filtration of glucose.

Abstract #236

PERFORMANCE OF ESTIMATED GLOMERULAR FILTRATION RATE IN DIAGNOSING CHRONIC KIDNEY DISEASE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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Augustine Ohwovoriole

Objective: 1).To determine the accuracy of estimated Glomerular Filtration Rate(eGFR) in assessing kidney function in persons with diabetes. (2) To test the hypothesis that the use of lean body mass(LBM) in the calculation of eGFR produces more accurate results than using the actual weight of the subject.
Methods: The records of patients attending the Diabetes clinic of a tertiary hospital in Lagos who had creatinine clearance performed as part of annual review were studied. Demographic data, anthropometric measurements and co-morbidities were noted. LBM was determined using a validated equation. Creatinine clearance was determined in the standard way, and eGFR was obtained by using the Cockcroft and Gault equation. CKD was defined as GFR< 60ml/min. Data was analyzed with Epi-Info software.
Results: 35 patients, equally distributed gender-wise with an age range of 35-82 years were studied. There was agreement in the diagnosis of chronic kidney disease by the two methods at low and normal GFR ranges with sensitivity, specificity, positive predictive and negative predictive values of 92.3%,72.7%,66.7% and 94.1% respectively. There was however a discordance at higher than normal creatinine clearance values(hyper filtration stage of diabetic nephropathy). The use of LBM improved sensitivity and negative predictive value to 100%, however the specificty was very low (23.7%) with a tendency to underestimate GFR and over diagnose CKD.
Discussion: Screening for Chronic Kidney Disease(CKD) by determination of GFR is traditionally by creatinine clearance. This method which requires collection of a timed urine sample is inconvenient. Simpler but reliable methods are needed for the routine screening

for diabetic nephropathy. Estimated GFR obtained using the Cockcroft and Gault equation has been found to correlate well with creatinine clearance in various reports; however, no report was found on its diagnostic performance in persons with type 2 diabetes in Nigeria. Limitations in the use of the Cockcroft and Gault equation include its unreliability in the very obese and in edematous patients. We sought to circumvent these limitations by testing the hypothesis that the use of LBM in the Cockcroft and Gault equation will produce more accurate results than using the weight of the subjects. The use of LBM improved sensitivity to 100%, however specificity was very low (23.7%).

Conclusion: There should be caution in the use of eGFR as a surrogate for creatinine clearance using the Cockcroft and Gault equation in patients with DM as the method is unreliable at higher than normal creatinine clearance values. The use of LBM in doing the calculation of eGFR appears to underestimate renal function and thus overdiagnose CKD.

Abstract #237

INTERNAL MEDICINE RESIDENTS CONFIDENCE, KNOWLEDGE, AND COMFORT LEVEL IN MANAGING DIABETES MELLITUS

Renee Amori, MD, David Bernstein, Barbara Simon, MD

Objective: The high prevalence of diabetes mellitus requires a sufficiently prepared and trained physician workforce. With a nationwide shortage of endocrinologists, many patients must rely on non-endocrinologists to manage and treat their disease. An assessment of internal medicine trainee knowledge and confidence in managing diabetes can help design an educational curriculum targeting diabetes knowledge gaps.

Methods: A self selected subset of Internal Medicine residents at a single, three year training program participated in a voluntary 35 question online survey created by faculty to evaluate their experience in evaluating and managing diabetes mellitus. The survey included qualitative questions assessed with a modified Likert scale to appraise confidence levels, and quantitative questions to assess knowledge, which included diagnostic criteria, glycemic goals, and preventative screening.

Results: Data were analyzed by training year. The majority of respondents had completed an Endocrinology rotation, and did report more confidence in their management compared to those residents who had not. All residents had managed diabetes in the inpatient setting, and most had managed acute hyperglycemic emergencies. More than 85% of respondents had managed diabetes in the outpatient setting. Residents felt more confident in managing insulin in the inpatient setting versus outpatient. Residents also felt more confident in diagnosing type 2 versus type 1 diabetes. However, more than 50% of respondents did not use currently recommended terminology to classify types of diabetes. Third year residents reported the most confidence in outpatient management of type 2 diabetes. When asked to select the most cost effective insulin regimen for an uninsured patient, one in four respondents chose a basal-bolus analogue insulin regimen over a pre-mixed human insulin regimen. The majority identified an A1c of either 6.5% or 7% as a reasonable goal, but approximately 15% identified an A1c of 6% or less as a glycemic goal.

Discussion: Residents did improve their diabetes knowledge over the course of training. Limitations of these data include resident self-selection as the respondents were more likely to have completed an Endocrinology rotation.

Conclusion: Based on these results, education strategies should focus on reinforcing basic diabetes knowledge and treatment earlier during residency training. Educational focus areas could include proper medication selection, patient safety, and awareness of medical costs related to diabetes care and treatment. More ambulatory experience in insulin management may improve residents comfort levels with insulin management in the outpatient setting.

Abstract #238

DIABETIC KETOACIDOSIS IN A SICKLE CELL ANEMIA PATIENT ALSO PRESENTING IN VASO-OCCLUSIVE CRISIS

Olusegun Sheyin, MD

Objective: To report and discuss the management challenges of a case of diabetic ketoacidosis(DKA) in a sickle cell anemia(HbSS) patient who also presented in vaso-occlusive crisis(VOC) to the emergency unit of a tertiary hospital in Lagos.

Case Presentation: A 20 year-old female, known HbSS and type I diabetes mellitus patient previously well controlled on subcutaneous split mixed insulin, presented with a 1 week-history of cough, bone pains, polydipsia and polyuria. Examination findings included painful distress, dyspnea, pallor, icterus, dehydration, a sickle cell habitus and pyrexia of 38 degrees Celsius. Respiratory rate was 48 cycles per minute with bronchial breath sounds and crackles in the left lower lung zone. Pulse was 104 per minute with a blood pressure of 140/80mmHg. The remainder of the examination was unremarkable. Random Blood Sugar(RBS) was 388mg/dL. Urinalysis revealed ++ glucose and ++ ketones. Urine and sputum culture yielded no growth. Her chest radiograph showed consolidation in the left lower lung zone consistent

with a left lobar pneumonia. Serum chemistry revealed hypokalemia and a high anion gap metabolic acidosis. Anemia (hematocrit of 15.6%) and leukocytosis (31 x 109/L) were found on complete blood count. A diagnosis of DKA and VOC precipitated by chest infection was made. Treatment was with intravenous fluids 0.9% saline 500mls 6hrly initially and then 4.3% Dextrose in 0.18% saline 500mls 8hrly when her RBS became less than 250mg/dL. Hourly intramuscular soluble insulin was instituted with a change to dextrose-potassium-insulin infusion when her RBS fell below 250mg/dL. Intravenous ceftriaxone 1g daily and a 5 day-course of azithromycin were commenced. Pain was managed with pentazocine, diclofenac potassium, ketoprofen and Tramadol. Transfusion with 2 units of packed cells was also given. She was discharged on split mixed insulin 20 IU a.m. and 10 IU p.m. to the Sickle Cell and Diabetes clinics.

Discussion: To the best of my knowledge, no case of coexistent VOC and DKA has been reported in the literature. This co-existence may have a common precipitant such as an infection as is seen in this case. In addition, the presence of one of the conditions may precipitate or worsen the other, setting up a vicious cycle. Antibiotic use prior to presentation may explain the negative sputum culture. Furthermore, the presence of severe anemia limits aggressive fluid resuscitation in order to avoid precipitation of anemic heart failure. Therefore, red cell transfusion to near steady state hematocrit may be necessary.

Conclusion: With co-existent VOC and DKA, prompt and adequate management of each of the conditions is essential to reduce morbidity and mortality.

Abstract #239

PIOGLITAZONE INDUCED REVERSIBLE VALVULAR REGURGITATION

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Objective: Thiazolinediones (TZD) have beneficial effect on glycemia and cardiovascular risk factors. However edema with or without heart failure can occur in patients treated with TZD. Here we report the occurrence of pioglitazone induced reversible valvular regurgitant lesions.

Methods: Clinical and laboratory data are reported on a known case of Type 2 diabetes treated with pioglitazone.

Case Presentation: A 50 year-old lady, known diabetic for 5 years was treated with pioglitazone 30 mg/ daily. 5 months later she presented with occasional exertional dyspnea and gradually progressive swelling of feet and weight gain of 3 kg. There were no signs of left ventricular failure. HbA1c decreased to 6.8% in parallel with a reduction of hemoglobin by 2 gm/dl and an increase in BNP from 5 to 22 pmol/l. Transthoracic echocardiography revealed moderate mitral and aortic regurgitation, with a predominantly central jet. Valves were not deformed. Furthermore, ejection fraction was increased and left ventricular and atrial dimensions were also increased, compared to baseline values. Left ventricular filling pressure was slightly increased. There was no evidence of diastolic failure. Pioglitazone was substituted with sitagliptin 100 mg daily. All the laboratory indications of fluid retention (BNP and haemoglobin) were back to baseline values 5 months after discontinuation of pioglitazone. Echocardiography at 5 months was normal without any residual regurgitant lesions.

Discussion: The increase in plasma volume related to TZDs result from a reduction in renal excretion of sodium and an increase in sodium and free water retention. TZDs interact synergistically with insulin to cause arterial vasodilatation leading to sodium reabsorption and increase in extracellular volume and subsequent pedal edema. Increased sympathetic nervous system activity, altered interstitial ion transport, alterations in endothelial permeability and peroxisome proliferator activated receptor mediated expression of vascular permeability growth factor represent other possible mechanisms for edema with these agents. In our case, changes in haemoglobin and BNP with bipedal edema indicated fluid retention in the absence of pulmonary edema. Mitral regurgitation due to left ventricular dilatation tends to have central jet, as in our patient. Similarly aortic regurgitation can be explained by hypervolemic state. Dorkhan M, et al have reported occurrence of isolated mitral valvular regurgitation after 6 months of usage of pioglitazone 45 mg/day.

Conclusion: Five months treatment with pioglitazone could induce significant but reversible valve dysfunction. Awareness regarding safety profile of TZDs and carefully monitoring of patients receiving TZDs is needed.

Abstract #240

DO PREDIABETICS NEED INSULIN THERAPY?

Shashi Agarwal, MD

Objective: Prediabetes is three times as common as diabetes. Most patients with prediabetes not only progress to overt diabetes but experience higher cardiovascular events compared to those with normal glycemia. Although lifestyle changes have a positive impact and insulin sensitizing agents may be beneficial, the need for early initiation of insulin therapy in this population is not known. An understanding of the underlying patho-physiology may help in establishing a new treatment paradigm in these patients.

Methods: HbA1c and fasting insulin levels were collected in 50 consecutive patients being treated for hypertension. HbAic levels were regarded as being prediabetic if they measured 5.7% to 6.4% (39 - 46 mmol/mol). Fasting insulin level considered elevated if they measured 25 micro IU/mL or higher.
Results: Of the 50 patients (ages 29 to 80 years) there were 26 males and 24 females. Of these, 19(38%) had HbA1c levels below 5.7% and 31 (62%) had HbA1c levels between 5.7% and 6.4%. Of the 19 with normal HbA1c levels, 14 (74%) had normal insulin levels and 5 (26%) had elevated insulin levels. Of the 31 (62%) with prediabetes, 16 (52%) had normal insulin levels and 15 (48%) had elevated insulin levels.
Discussion: Diabetes mellitus is a common disease. According to the American Diabetic Association, it affects about 26 million Americans. It is estimated that 79 million people in the United States also suffer from prediabetes. Prediabetics have an increased risk of progressing to type 2 diabetes. They are also at a higher risk of developing cardiovascular and diabetes-related complications. Prediabetes is also associated with a significantly increased all-cause mortality. Prediabetes is common in hypertensive patients, and further increases their cardiovascular morbidity and mortality. The therapeutic approach to these patients is not very clear.
Conclusion: Prediabetes is common in patients with hypertension. Approximately one half of our prediabetic hypertensive patients had normal insulin levels, indicating probable associated insulin deficiency. Early insulin therapy in these patients, in addition to insulin sensitizing agents, may play a therapeutic role in restoring normal glucose tolerance. Further studies are needed to evaluate this suggested treatment paradigm.

Abstract #241

REDUCED RISK OF HYPOGLYCEMIA WITH INSULIN DEGLUDEC VS INSULIN GLARGINE IN PATIENTS WITH TYPE 2 DIABETES REQUIRING HIGH DOSES OF BASAL INSULIN: META-ANALYSIS OF FIVE RANDOMIZED TRIALS

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Objective: Insulin Degludec (IDeg), a new basal insulin that forms soluble multi-hexamers after sc injection, has an ultra-long, stable action profile with low hour-to-hour and day-to-day variability which may explain its low risk for hypoglycemia. A key finding in IDeg phase 3 clinical trials has been a consistent reduction in hypoglycemia risk compared to insulin glargine (IGlar). Little information is available on hypoglycemia risk in the subset of subjects using high basal insulin doses (>60 U/day). We have compared IDeg to IGlar in patients with type 2 diabetes (T2DM) who require >60 U/day of basal insulin.
Methods: The meta-analysis reported here compared glycosylated hemoglobin (HbA1c), fasting plasma glucose (FPG), and rates of overall confirmed and nocturnal confirmed hypoglycemia in a pooled population of T2DM subjects using >60 U of basal insulin at trial completion. Five phase 3A, open-label, randomized, treat-to-target, confirmatory 26- or 52-week trials with IDeg (N=2,262) vs IGlar (N=1,110) administered once daily in T2DM were included. Confirmed hypoglycemia was defined as self-measured blood glucose <56 mg/dl (plasma calibrated) or any episode requiring assistance (ADA 'severe' definition); nocturnal confirmed hypoglycemia was any confirmed episode with onset between 00:01-05:59, inclusive. Analysis of HbA1c and FPG was based on an ANCOVA model and analysis of hypoglycemic episodes was based on a negative binomial regression model.
Results: At end-of-trial, a substantial and similar observed percentage of IDeg- and IGlar-treated T2DM subjects required >60 U of basal insulin daily [IDeg, 35.1% (795/2262); IGlar, 33.7% (374/1110)]. Patients achieved similar mean HbA1c values at end-of-trial (estimated treatment difference (ETD) IDeg−IGlar: 0.05%, p=0.44, NS). End-of-trial mean FPG values were lower with IDeg than IGlar (ETD: −5.9 mg/dL, p<0.04). Overall confirmed and nocturnal hypoglycemia rates were lower in individuals requiring >60U of insulin on IDeg than on IGlar. There was a 21% lower rate of overall confirmed hypoglycemic episodes for IDeg (rate ratio (RR) IDeg/IGlar: 0.79, p=0.02) and a 52% lower rate of nocturnal confirmed hypoglycemic episodes for IDeg (RR: 0.48, p<0.0001).
Discussion: In this post-hoc meta-analysis, >30% of subjects with T2DM required >60 U/day of basal insulin. In these individuals, IDeg achieves similar HbA1c reduction with significantly less overall and nocturnal hypoglycemia compared to IGlar.
Conclusion: These findings are consistent with results for the overall trial population, confirming that IDeg is a safe and effective basal insulin choice for T2DM patients across the spectrum of insulin requirements.

Abstract #242

NEW ONSET DIABETES MELLITUS PRESENTING AS MIXED HYPEROSMOLAR HYPERGLYCEMIC SYNDROME AND DIABETIC KETOACIDOSIS

Amanda Long, DO, Aidar Gosmanov, MD, PhD, Samuel Dagogo-Jack, MD

Objective: Diabetic ketoacidosis (DKA) and hyperglycemia hyperosmolar syndrome (HHS) are often thought of as two distinct entities, but can be also characterized as points along a spectrum of disease. The overlap of DKA and HHS is reported to occur in up to 30% of patients with hyperglycemic crisis.
Case Presentation: A 73 year old African American woman presented to the hospital with complaints of coffee ground emesis, weakness, polyuria, and polydipsia for 2 weeks. She reported no known history of diabetes mellitus or chronic kidney disease. Initial labs showed a serum glucose of 1,137 (65-99mg/dL), bicarbonate of 12 (22-32mmol/L), , creatinine of 5.0 (0.6-1.3mg/dL), phosphorous of 8.4 mg/dL (2.5-4.9), anion gap of 36 (5-20), venous pH of 7.08 (7.35-7.45), positive serum and urine ketones, hemoglobin A1c of 10.9 % (4-6), and measured serum osmolality of 409 (270-290mOsm/kg). The presentation was consistent with mixed DKA and HHS. Treatment with IV fluids and insulin resulted in resolution of DKA and acute renal failure. Patient was discharged from the hospital on split mix insulin regimen with outpatient follow up.
Discussion: Diagnostic criteria for DKA includes a glucose greater than 250mg/dl, pH less than 7.3, bicarbonate less than 18mEq/L, and a ketonemia. DKA is often characterized by an anion gap metabolic acidosis. HHS criteria includes a serum osmolality greater than 320mOsm/kg, glucose greater than 600mg/dL, pH greater than 7.3, bicarbonate greater than 18-20mEq/L, and absence of ketonemia. The degree of beta-cell reserve and variable levels of counter-regulatory hormones determine whether patients present with DKA or HHS. However, the pathogenesis of mixed states remains unclear. In our patient, the presence of acute kidney injury, which is rare in DKA, suggests that perhaps prolonged HHS triggered beta-cell decompensation which resulted in development of DKA. As with HHS alone, the level of mortality is higher in mixed states when compared to DKA alone. New onset diabetes is a risk factor for the development of mixed DKA and HHS. The treatment of both DKA and HHS are similar, but the current American Diabetes Association (ADA) consensus statement does not address management of mixed states.
Conclusion: Mixed DKA and HHS occurs in up to 30% of patients with hyperglycemic crisis and has a high level of mortality. New onset diabetes appears to be a predictor of mixed states, but further work needs to be done to identify other risk factors. Additionally, further work should also be done to identify effective approaches in management of patients with mixed DKA and HHS, as this is not addressed in the current ADA consensus statement.

Abstract #243

FOURNIER'S GANGRENE IN A PATIENT WITH TYPE 1 DIABETES (T1D) AND DRUG- INDUCED AGRANULOCYTOSIS

Ayse Bag Ozbek, MD, Anand Viswanathan, Harmeet Narula, MD, FACP

Objective: To report a case of Fournier's Gangrene with Type 1 DM and drug-induced agranulocytosis.
Case Presentation: A 56 year old gentleman with longstanding T1D on insulin pump and long term antibiotic use (for infected surgical wound of shoulder) was admitted to the hospital with a skin rash & agranulocytosis (both likely secondary to Vancomycin and Cefepime). The patient was started on prednisone for his drug-induced agranulocytosis and skin rash, but soon thereafter developed a fever to 103 F and complained of severe testicular pain. He had diffuse scrotal edema, without any focal findings. A stat testicular sonogram showed no torsion or abscess. Due to his immunosuppressed state, aggressive antibiotics were initiated, and he was monitored closely with Urology. Within hours he became hypotensive (septic shock) and developed progressive scrotal wall swelling and discoloration of the scrotum, consistent with impending infarction of the scrotal skin. Patient underwent urgent surgical debridement, and received aggressive antibiotics and vasopressors in the ICU. Pathology results confirmed Fournier's Gangrene and the tissue cultures were positive for Pseudomonas Aeruginosa.
Discussion: Fournier's Gangrene is a rapidly progressing necrotizing fasciitis with a high mortality (without urgent surgical debridement, the mortality rate with antibiotics alone is 100 %. Hence the importance of early diagnosis and treatment. Endocrinologists should be aware of this rapidly progressive, life-threatening infection which may present with nonspecific signs and symptoms, like in our patient.
Conclusion: In a patient with T1D and fever, sudden onset, severe testicular pain should raise high suspicion of Fournier's Gangrene and emergent Urology consultation should be obtained for prompt and aggressive debridement.

Abstract #244

INSULIN DEGLUDEC IS HIGHLY EFFICACIOUS REGARDLESS OF DIABETES DURATION OR BODY MASS INDEX: A CROSS-TRIAL EVALUATION

Lawrence Blonde, MD, FACP, FACE, Lars Endahl, Nathan Lassota, Jeff Unger

Objective: A widely accepted treatment goal with any diabetes therapy is to lower HbA1c to levels of ≤6.5% or <7%. Not all patients are able to achieve these targets, for diverse reasons. We conducted a cross-trial evaluation to determine if certain baseline characteristics influence whether a patient achieves treatment goals with the new ultra-long-acting basal insulin, insulin degludec (IDeg).
Methods: Three phase 3a trials, including previously insulin-naïve T2DM patients as well as T1DM and T2DM patients on basal-bolus treatment, were utilized to assess whether certain baseline characteristics (HbA1c, diabetes duration, and body mass index (BMI)) differed between the entire IDeg-treated trial population and those individuals achieving HbA1c ≤6.5% or <7% with IDeg treatment. In trial A (trial 3668), a total of 228 T2DM patients previously insulin-naïve or receiving basal insulin treatment were given once-daily (OD) IDeg for 26 weeks. In trial B (trial 3582) and trial C (trial 3583), 744 and 472 previously insulin-treated patients with T2DM and T1DM, respectively, received IDeg OD for 52 weeks, with insulin aspart at mealtimes.
Results: The proportion of IDeg-treated patients achieving HbA1c ≤6.5% or <7% was 23% and 39%, respectively (trial A), 31% and 49%, respectively (trial B), and 24% and 40%, respectively (trial C). Mean baseline values (HbA1c, diabetes duration, and BMI) varied across trials, reflecting the different patient populations being studied. In all three trials, patients achieving HbA1c ≤6.5% or <7% had a lower HbA1c at baseline compared with the entire population treated with IDeg (values for ≤6.5% vs. <7% vs. entire population; trial A: 7.8 vs. 7.9 vs. 8.4%, trial B: 8.0 vs. 8.0 vs. 8.3%, trial C: 6.8 vs. 7.0 vs. 7.7%). Neither duration of diabetes nor BMI were systematically different for patients achieving HbA1c ≤6.5% or<7% compared to the entire population; diabetes duration (trial A: 9.0 vs. 9.9 vs. 10.3 years, trial B: 14.0 vs. 13.9 vs. 13.6 years, trial C: 19.1 vs. 19.9 vs. 19.1 years); BMI (trial A: 28.4 vs. 29.0 vs. 29.4 kg/m2, trial B: 32.9 vs. 32.8 vs. 32.2 kg/m2, trial C: 26.7 vs. 26.5 vs. 26.3 kg/m2).
Discussion: This evaluation of T2DM and T1DM patients suggests that neither diabetes duration nor BMI are factors that influence whether patients will achieve HbA1c ≤6.5% or <7.0% when initiating treatment with once-daily IDeg or switching to IDeg from another basal insulin. As expected, individuals achieving HbA1c ≤6.5% or <7% are characterized by a lower baseline HbA1c.
Conclusion: Based on the above studies, the likelihood of a patient achieving HbA1c targets with once-daily IDeg is not influenced by diabetes duration or BMI at baseline, but by baseline HbA1c.

Abstract #245

A NOVEL ENDOCRINOLOGY-BASED WELLNESS PROGRAM TO REDUCE MEDICATION EXPENDITURES AND IMPROVE GLYCEMIC OUTCOMES

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Objective: The aim of this study was to calculate diabetes-related medication expenditures before and after enrolling into a 16-week lifestyle intervention program.
Methods: The Wellness Life Program includes nutrition, fitness, and behavioral therapy. All educational sessions are provided by specialists: registered dieticians, personal fitness trainers, and a behavioral psychologist. Laboratory assessments are completed within 3 weeks prior to program initiation and within 1 week before program discontinuation. Patients are seen by their endocrinologist at least one time during the 16-week interventional period. Patients pay out of pocket to participate. Medication costs were calculated for a 30 day supply using average wholesale prices from Thomson Reuters 2010 Red Book.
Results: A total of 36 patients enrolled in the Wellness Life program, and 27 patients have Type 2 diabetes (Type 2 DM enrollees: mean age 63±7 years, body mass index (BMI) 40.3±9.4, hemoglobin A1c (HbA1C) 8.0±1.7). Prior to enrollment, 22% were on 1 antidiabetic agent, 45% were on 2 agents, 33% were on ≥3 agents, with a mean of 2 antidiabetic agents (19 patients using insulin therapy). Mean 30-day prescriptions costs were $412.54. By the end of the program, 30% of enrollees were on 1 antidiabetic agent, 48% were on 2 agents, and 22% were on ≥3 medications. Mean 30-day prescription costs were $269.62 following the interventional period, yielding an average reduction of $142.92 per patient per month. Insulin and oral medication doses were decreased on average by 46% and 16%, respectively. Average reductions in BMI and HbA1c were 3.07 and 0.7%, respectively. Patients with a baseline HbA1c >8 (n=12) experienced an average decrease of 1.3%.
Discussion: Type 2 diabetes typically requires lifestyle changes and multiple medications to improve

blood glucose, reduce the risk of microvascular and macrovascular complications, and produce a favorable impact on comorbidities. Medical expenditures for patients with diabetes are two times higher than patients without this medical condition. Normal physician office visits do not allow time to provide thorough patient education to improve fitness, eating, and life behaviors.

Conclusion: Employing a wellness program within an endocrinology practice can reduce anti-diabetic medication expenses. Recently, the Centers for Medicare and Medicaid Services (CMS) ruled that Medicare will now cover obesity counseling services. However, at this time only primary care providers will be covered. Wellness Life illustrates the successful use of a multidisciplinary team which includes endocrinologists and nutritionists to reduce body mass index and medication expenditures.

Abstract #246

THE LIPID ACCUMULATION PRODUCT IS NOT BETTER THAN ANTHROPOMETRIC MEASUREMENTS IN IDENTIFYING INSULIN RESISTANT BLACK YOUTH

Marshall Tulloch-Reid, MBBS, DSc, Trevor Ferguson, Rainford Wilks

Objective: The lipid accumulation product ([LAP = (Waist Circumference-65) × Triglycerides for men and (Waist Circumference -58) × Triglycerides for women]) has been proposed as a better marker of cardiovascular risk than simple measures of obesity. In this study we investigate the ability of the LAP to identify black youth with relative insulin resistance (a precursor of cardiovascular disease and type 2 diabetes) and compare it against traditionally used anthropometric measurements.

Methods: Trained nurses performed anthropometric measurements and collected fasting blood samples for glucose, insulin and lipid measurement in 18-20 year old youth. Insulin resistance was determined using HOMA, with participants in the highest sex-specific quartile considered insulin resistant. Receiver Operating Curve (ROC) analysis was used to assess the ability of the LAP to identify insulin resistant youth. The ROC area under the curve was used to compare the ability of the LAP to identify insulin resistant youth with that of the body mass index (BMI), waist circumference (WC) and waist-height ratio (WTHtR). The agreement, between sex specific quartiles for LAP and anthropometric measurements, was assessed using the kappa statistic.

Results: Data from 708 participants (316M; 392F, Age [Mean±SD]-19±0.5years, BMI - 23.1±5.2kg/m2, WC-74±12cm, WTHtR- 0.42 ±0.7, triglycerides - 0.58±0.26mmol/L) were analyzed. While the LAP identified insulin resistant men (ROC area under the curve [95%CI]=0.72[0.65, 0.79]) and women (0.73[0.67,0.79]) it was not superior to BMI, WC or WTHtR. LAP had moderate agreement with WC (Kappa = 0.61 M, 0.55 F) and WTHtR (Kappa = 0.46M, 0.47F) but poor agreement with BMI (kappa = 0.38M, 0.36F).

Discussion: The LAP combines waist circumference, a measure of central adiposity, with serum triglycerides, a simple index of circulating free fatty acids and may be indicative of an accumulation of metabolically active fat and not just central adiposity. Blacks are less likely to have elevated triglycerides compared to Caucasians and this may account for our not finding LAP superior to WC or WTHtR in identifying insulin resistant youth. There was poor agreement between LAP and BMI, a measure of general adiposity, despite both measurements having a similar ability to identify insulin resistant youth. Studies to identify and assess other markers of metabolically active fat that increase the risk of cardiovascular disease and type 2 diabetes in black populations are needed.

Conclusion: LAP was able to identify black youth with relative insulin resistance but did not perform better than traditional anthropometric measurements.

Abstract #247

PRE-DIABETES AND CARDIOVASCULAR RISK FACTORS IN URBAN AND RURAL WOMEN IN SOUTH-EASTERN NIGERIA

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Objective: The prevalence of diabetes mellitus is on the rise in Sub-Saharan Africa with its consequent burden on the health care systems of these emerging economies. Pre-diabetes predates clinical diabetes by many years and is known to increase cardiovascular risk in affected individuals and even more so in women, yet goes unrecognised without screening. This cross-sectional study was carried out to estimate the prevalence of pre-diabetes and other cardiovascular risk factors in Nigerian women and its association with residence in either a rural or urban area.

Methods: A total of 130 apparently healthy women; 65 living in an urban area in Enugu, SE Nigeria and 65 living in a rural area in the same state were sampled. Interviewer administered questionnaires were used to obtain demographic data including exercise habits or an active lifestyle, alcohol and tobacco intake. Blood pressure, BMI, waist circumference and biochemical parameters including FPG and total serum cholesterol were measured. Level of significance set at $p<0.05$.

Results: The mean ages of the urban and rural women

were 50.1± 9.6years and 50.9 ± 14.2 years respectively (p =0.73). The frequency of alcohol and tobacco use was low in both groups of women and they both had high levels of physical activity. The prevalence of pre-diabetes was 6.2% in the urban women and 1.5% for rural, while for diabetes was 15.4% and 7.7% with newly-diagnosed DM accounting for 7.7% and 1.5% respectively. Obesity was more prevalent in the urban women (mean BMI of 30.3 ± 5.4kg/m2 and 25.8 ± 5.6 kg/m2, waist circumference 97.0 ± 11.5cm and 86.9 ± 11.6cm, p <0.01). Mean total cholesterol was urban vs rural; 196.1 ± 45.9mg/dl and 190.5 ± 43.3mg/dl (p=0.84) while hypertension was present in 40% and 43.1% of urban and rural women respectively.

Discussion: The prevalence of pre-diabetes and diabetes was higher in the urban women and also notably higher than previous studies in the country. Urban women also had a higher prevalence of frank diabetes and newly-diagnosed diabetes. This rising trend in diabetes burden has been similarly reported in other developing countries especially in the urban areas. The higher prevalence of obesity in the urban dwellers appears to be the major risk factor for their developing both diabetes and pre-diabetes. Diet, rather than physical activity may have played a larger role in the development of obesity. Other cardiovascular risk factors were similar in both populations, suggesting that obesity and diabetes burden are the major contributors to the development of cardiovascular disease in our population.

Conclusion: The prevalence of obesity, pre-diabetes and diabetes is higher in our urban women than the rural dwellers.

Abstract #248

ROLE OF ACOUSTIC RADIATION FORCE IMAGING SONOELASTOGRAPHY IN DETECTION OF LIVER FIBROSIS AMONG PATIENTS WITH TYPE 2 DIABETES MELLITUS WITH COEXISTENT NONALCOHOLIC FATTY LIVER DISEASE

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Objective: To detect liver fibrosis in patients with diabetes with the help of a safe, efficient and non invasive method

Methods: The study was conducted on 75 patients with nonalcoholic fatty liver disease (NAFLD) in the age group 25-60 years, 37 of these had type 2 diabetes mellitus. All patients were subjected to ultrasound scan for elastography of the liver. Their liver echotexture and shear velocity measurements at four different sites were carried out by acoustic radiation force imaging (AFRI) sonoelastography. In both the groups clinical characteristics, history of drugs, alcohol & hepatitis, BMI, systemic examination including cardiovascular examination were recorded. Lab tests including LFTs, coagulogram, lipids, Hepatitis B & C markers, duration & status of diabetes - fasting & post prandial blood sugar & HbA1c were carried out. Upper GI endoscopy to detect oesophageal varices in patients with moderate to severe fibrosis was also done. Stages of Liver Fibrosis: F1 - 1.185 to 1.215; F2 - 1.215 to 1.54; F3 - 1.54 to 1.94; F4 - 1.94 & beyond.

Results: Total 12 patients showed evidence of fibrosis. Patients in the control group showed evidence of early fibrosis in 2(F1 & F2 one each) patients. While 10 patients in the diabetic group had evidence of moderate to severe fibrosis (3-F3, 7-F4). 6 out of 10 patients in this group had oesophageal varices.

Discussion: NAFLD is a common occurrence among patients with diabetes & obesity. It is also associated with insulin resistance, linking atherosclerosis & progressive liver damage. NAFLD may lead to chronic necrosis & inflammation culminating in cirrhosis. Management of diabetes has to be changed in the presence of liver cirrhosis as a few anti diabetic drugs are hepatotoxic. Liver biopsy is the gold standard for diagnosis of liver cirrhosis, but being invasive it cannot be recommended in every case. ARFI has shown promising results for detecting liver fibrosis that too without any harm and/or complications of liver biopsy. It should be the tool of choice for patients with contraindications to a biopsy. In our study, 6 out of 10 patients had oesophageal varices indicating advanced liver damage. Rest of the 4 patients were confirmed to have biochemical alterations of liver function. Our study highlights the importance of detecting liver fibrosis at an early stage in diabetic patients with NAFLD.

Conclusion: 1.Sonoelastography detected liver fibrosis in 10 out of 37 diabetics (27%) while 2 out of 38 controls (5.2%) had evidence of fibrosis. 2.In the diabetic group fibrosis was in the advanced stage (F3 &F4) while in control group it was in the early stage (F1&F2). 3.Sonoelastography can detect liver fibrosis in patients with normal serum transaminase levels.

Abstract #249

A PREVENTIVE CARE MODEL IMPROVES MONITORING AND CONTROL OF DIABETES AND CARDIOVASCULAR DISEASE VERSUS TRADITIONAL PRACTICE.

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Objective: Many Americans are not receiving recommended preventive and routine healthcare services in the current healthcare model. The purpose of the study

was to assess the impact of the MDVIP primary care model that focuses on personalized preventive healthcare and compare the rates of HEDIS effectiveness of care measures from the MDVIP model to national health plan results.

Methods: We performed a retrospective chart review of 15 MDVIP-affiliated Arizona physicians and used descriptive statistics (percentages) to compare the average rates of each of the HEDIS measures from our chart review to the 2008 national health maintenance organization (HMO) and preferred provider organization (PPO) health plans average rates published by the National Committee for Quality Assurance (NCQA).

Results: 357 members (41.5% men, 70.9% age > 65 years) identified as having a diagnosis of uncomplicated diabetes, hyperlipidemia, hypertension, or atrial fibrillation were included. A higher percentage of MDVIP members with Diabetes Mellitus had blood pressures <140/90 (83%) and LDL-C levels <100 (69%) as compared with national HMO plan rates (63% and 47%). The majority (>90%) of diabetic MDVIP members had "good" HbA1c (<8%) control, which was much higher than the national HMO (43%) and PPO (<25%) plan reported rates. 89.2% of all MDVIP members had a retinal exam (higher than the 90th percentile of commercial HMOs average of 73.7%), 92.5% had a foot exam within the past 12 months, and 70% had a urinary microalbumin test performed. A higher percentage of MDVIP members with cardiovascular conditions had LDL-C screenings (96%) performed compared with national HMO (89%) and PPO (>75%) plan rates. Approximately 50% of the MDVIP members with cardiovascular conditions had LDL-C levels <100, which is similar to national HMO plan reported rates (58%) but higher than national PPO plan reported rates (<30%). A higher percentage of MDVIP members had mammograms (87%) and DXA scans (90%) (Women only) and colonoscopies (85%) performed as compared with national HMO and PPO plan rates.

Discussion: MDVIP members with diabetes and cardiovascular disease had better hgbA1c, lipid and BP control than national benchmarks. They also had more preventive screenings.

Conclusion: The MDVIP primary care model delivers better preventive care services and better healthcare outcomes when compared to national health plans for patients with diabetes and cardiovascular disease. This improvement in outcomes and screening will ultimately lower health care costs.

Abstract #250

STUDY OF CLINICAL AND BIOCHEMICAL PARAMETERS OF PULMONARY TUBERCULOSIS IN SUBJECTS WITH TYPE 2 DIABETES MELLITUS

Faria Afsana, MBBS, Ashraf Jamil, Zafar Latif

Objective: Type 2DM is a global epidemic and recognized as a threat to pulmonary tuberculosis (PTB) control worldwide especially in developing countries. When tuberculosis is diagnosed in diabetic subjects these can effect each other in term of clinical presentation and course of disease. The aim of the study was to evaluate the demographic, clinical and biochemical parameters of newly detected PTB patients with type 2 diabetes .

Methods: Seventy two diabetic subjects with newly detected PTB were studied. Patients with fever, cough, hemoptysis and or weight loss were referred to pulmonologist from OPD, BIRDEM. After thorough clinical examination, blood sugar, complete blood count(CBC),ESR, sputum for bacteriological C/S and Acid fast bacilli(AFB) (3 sample),X-Ray chest was done. The diagnosis of PTB was based on a positive sputum AFB test, a suggestive CBC report or typical radiographic findings with high clinical probability. All patients are followed up at least at1st, 3rdand 6th month of antitubercular therapy.

Results: Mean age of study subjects was 46(19-75) years. Mean BMI was 19.9Kg/meter square.Mean ESR (mm in 1st hour) was 94.5 with 60% subjects having ESR >100. Mean fasting and post prandial blood sugar (mmol/l) was 13.69 & 21.02 respectively. Sputum for bacteriological C/S revealed no growth in 75% patients, 12.5% had klebsiella, 2.8% pseudomonus & candida each and rests were streptococcus. Chest X-Ray revealed cavity in47.2%, opacity in40.3%, both opacity and cavity in5.6%, pleural effusion in5.6%and 5.5% had no lesion. All patients were sputum AFB positive and among them 73.6% (n=53) had AFB positive in all 3 samples. Most of the patients (98%) with positive x-ray finding showed radiological improvement after 2 months of antitubercular treatment.

Discussion: PTB is common in developing countries as well as in diabetic subjects. Most of the PTB patients have features typical of tuberculosis. Most of them do not show any growth of organism in association of mycobacterium tuberculosis. About 95% of study subjects have Chest X-ray suggestive of TB. All the study subjects showed radiological improvement within a period of 2 months.

Conclusion: PTB is a common cause of uncontrolled diabetes. Most of them had high ESR. Klebsiella is the commonest associated organism in sputum of PTB patients. Pulmonary cavity and opacity is the commonest radiological observation among study subjects. Sputum AFB is a good diagnostic tool for PTB in diabetic subjects.

Abstract #251

A NEW APPROACH TO THE CARE OF HOSPITALIZED PATIENTS WITH TYPE 2 DIABETES: USE OF INCRETINS TO CONTROL HYPERGLYCEMIA WITHOUT UNDUE HYPOGLYCEMIA

Stanley Schwartz, MD

Objective: The rates of obesity, pre-diabetes, type 2 diabetes are increasing in epidemic proportions. They are associated with microvascular -macrovascular (cardiovascular) disease complications, but it is clear that aggressive control of hyperglycemia can decrease complication rates. Both ADA/AACE organizations recommend aiming for lowest sugar possible without undue hypoglycemia. The same situation obtains for the hospitalized patient with pre-diabetes, stress DM, or DM, with clear epidemiological data showing adverse outcomes are increased in proportion to the degree of hyperglycemia. Though several studies clearly show benefit of tight glycemic control in hospitalized patients, others do not. Most feel hypoglycemia is the main culprit of these negative studies, though clearly other factors may obtain. As such, the glycemic goals in hospital are in debate. Many however believe that tighter control would be a wonderful goal if it could be achieved without (any) undue hypoglycemia.
Case Presentation: We present a short case study of a patient without prior diabetes, s/p MI, resuscitation 4 times, intubated, NPO, whose IV insulin drip (24u/d) could be stopped by substituting a DPP-4 inhibitor.
Discussion: Our presentation reviews incretin-based therapies which may ontrol of hyperglycemia without undue hypoglycemia because of: glucose-dependent insulin secretion which results in infrequent hypoglycemia in absence of SU/glinides/insulin, marked benefit in reducing glycemic elevations from two stress hormones-glucagon and corticosteroids (ie: stress diabetes), reduced hypoglycemia even with insulin therapy, decreasing glycemic variability, eliminate need for bolus insulin in some patients even as may need basal insulin, eliminate need for insulin in some, and potential benefit on CV function, even acutely.
Conclusion: We offer a simple, practical guideline for using Incretins in concert with previous standard care to improve control without undue hypoglycemia in the hospital.

Abstract #252

PROFILES OF PARTICIPANTS ATTENDING A FREE DIABETES EDUCATION CLASS

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Objective: Diabetes Self Management Education (DSME) is a cornerstone of therapy for those with diabetes mellitus. Better patient outcomes have been associated with curriculums that reinforce diabetes education with multiple class sessions and personalized follow-up. However, many barriers exist to DSME among underserved communities including lack of insurance coverage for these services, lack of finances to pay out of pocket and a lack of time and resources attend multiple sequential classes, which leads to a high attrition rate after beginning a class series.
Methods: The Drexel Diabetes Education Programs holds a single monthly "Basics of Diabetes" class. The class is free and open to the public. The class content focuses on type 2 diabetes, but does not exclude participants with type 1 diabetes or glucose intolerance. The class draws 6-14 participants monthly. A survey was designed to assess class demographics, attendee's knowledge of diabetes goals, and self management activities. Recruitment including informed consent was done prior to each monthly class. The initial survey was administered prior to the beginning of each class, and survey respondents were contacted 4-6 weeks later by telephone for a repeat survey.
Results: Results presented here describe the population attending the class sessions based on the initial survey. Consent was obtained from 43 subjects representing 43% of class attendees. Respondents had a mean age of 54 years, and were 65% female. Specifying race was optional, and 22.6% did not report race. A total of 46.5% identified race as black or African American. Four respondents self-identified as having "pre-diabetes," and two self-identified as having type 1 diabetes. Over 45% had their diagnosis less <1 year, and 19% had their diagnosis >10 years. However, 69.8% of respondents identified this as their first diabetes education class regardless of duration of diagnosis. 39.5% of respondents live in zip codes with median household income <$35,000 per year.
Discussion: A free DSME class attracted participants with prediabetes, Type 1, and 2 diabetes. For many, this was their initial DSME experience despite having a long-standing diagnosis.
Conclusion: Though this data is limited by sample size, a single, a free diabetes education class removed a significant barrier to diabetes self management education, and attracted participants from underserved communities.

Abstract #253

TELE-ENDOCRINOLOGY: BRIDGING THE GAP IN ENDOCRINE CARE VIA TELE-MEDICINE.

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Objective: The prevalence of diabetes and other endocrine diseases continues to rise especially in rural areas of USA where the prevalence of diabetes is ~17% higher than in urban centers. This disparity is compounded by the lack of specialists in rural locations. Telemedicine (TM), the transfer of electronic medical data to distant locations using telecommunications like internet may be beneficial in providing care in remote areas. We investigated the efficacy of a telemedicine-based endocrine consultative service in improving outcome measures in endocrine patients in rural communities.
Methods: Patients from 5 rural areas in Tennessee (Somerville, Parsons, Savannah, Trenton and Dyersburg) were referred by their primary care providers (PCP) to the Tele-medicine Unit of the University of Tennessee, Memphis. Patients were interviewed and examined (inspection only) from the Tele-medicine studio. The studio and the remote sites were connected via video-conference using Polycom VSX 7000 video cameras, television monitors and internet to transmit video and audio electronic records. Recommendations regarding management were sent to PCP via fax. Laboratory evaluations were done by PCP as requested. Patients did not return for follow-up if PCP was comfortable with continuing their care.
Case Presentation: Sixty-six patients aged 53.8 ± 15.5 years, 73% of whom were females were seen over 2.5 years. Of the 66 patients, 35 (53%) had type 2 diabetes, 27 (41%) had hypertension, while 30 (45%) had dyslipidemia; 20 (30%) had thyroid disease, and 8 (12%) had osteoporosis or hypercalcemia. Other consultations included hyponatremia and polycystic ovarian syndrome. Some patients had more than one endocrine disorder. Data available for 20 diabetic patients showed significant improvement in 17 (85%) of them. After ~ 6 months of follow-up, A1c decreased from 9.1 ± 1.3 to 7.5 ± 1.4 %; ($P < 0.002$). Follow-up data were available in 4 patients with thyroid disease, all of whom had become euthyroid; 12 (70%) of 17 subjects with dyslipidemia who had available follow-up data showed improvement in lipid profile. Nearly all patients (97%) were comfortable with receiving care from a remote site through video-conference.
Discussion: TM based endocrine consultation using video-conference technology was effective in improving outcome measures in diabetes and other endocrine disorders. The potential growth of this strategy for effective control of diabetes with reduced need for face-to-face encounter time seems to be a promising solution
Conclusion: TM proved to be an acceptable means of providing affordable and accessible care in endocrine patients. Prospective studies on the application of TM in endocrine practice are recommended.

Abstract #254

COMPARISON OF INSULIN INFUSION PROTOCOLS TARGETING BLOOD GLUCOSE(BG) 110-140MG/DL IN PATIENTS AFTER CARDIAC SURGERY

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Objective: To compare the efficacy and safety of two different IV insulin infusion protocols (IVIIP) targeting BG 110-140mg/dL in cardiac surgery patients utilized in 2 hospitals of same hospital system due to limited data about IVIIPs targeting these revised glycemic goals published in 2009.
Methods: IVIIP1 consists of 4 algorithms to adjust insulin infusion rates(IIR) based upon BG and it's rate of change. IVIIP2 is a single table adjusting IIR based on rate of change BG and current IIR. Consecutive patients receiving IVIIP over 6 months were identified retrospectively. BG and IIR were recorded for 48 hours postoperatively or till IVIIP discontinuation. Patients with heart transplantation, on systemic glucocorticoids, enteral/parental nutrition and sepsis were excluded.
Results: There were 117 patients in IVIIP1 and 130 patients in IVIIP2.
BASELINE CHARACTERISTICS:IVIIP1 patients were older (65 vs. 61 years, $p=0.006$), had more CABG (68 vs. 46%;$p<0.001$) and fewer valve procedures(15 vs. 37%; $p<0.001$). There were no differences in baseline BG (149±40.6 vs. 151 ±38.1mg/dL, mean±SD), BMI (30±6.3 vs. 30±6.4 kg/m2), HCT(28 vs. 28%), %patients with diabetes(32 vs. 31%)and GFR<30 ml/min(5 vs. 6%) and median duration of ionotropes (18 (0.25-182) vs 29(0.06-642) hours[range]) between groups. INSULIN REQUIREMENTS: There was no difference in duration of insulin infusion (42(9-48) vs 40 (14-48) median hours[range]), total insulin dose (99 (15-376) vs. 114(12-457) median units [range]) or average insulin rates (2.59(0-21) vs. 2.96(0-25), median[range]) between groups. GLYCEMIC OUTCOMES: There was no difference in percent BG in 110-140mg/dL (44.27 vs 43.25), >180mg/dL (5.92 vs. 7.68), 40-69mg/dL(0.99 vs. 0.84), <40mg/

dL (0.03 vs. 0) and in the CV of BG (21± 6.5 vs. 21±6.1) between groups. There was no correlation between time to goal BG and duration of inotropic agents (r=0.03). IVIIP1 had shorter time to achieve goal (3.32[0.22-19.35] vs 5.03[0.92-19.80] median hours[range], p=0.018), fewer % BG 141-180mg/dL (21.37 vs. 27.85, p=0.001), lower mean BG (127+/-12.2 vs. 133+/-12.1 mean±SD, p<0.001) and more % BG 70-109mg/dL (27.42 vs. 20.37, p<0.001).
Discussion: Both IVIIP were safe and effective but with some differences in glycemic outcomes. It is not clear if differences were due to patient population or differences in the IVIIP. Adherence to IVIIP was not evaluated but may also have been a factor especially since BG targets had recently changed.
Conclusion: Both IVIIP were found to be safe and effective. Each hospital decided to continue the use of their existing IVIIP but identified areas for improvement.

Abstract #255

A RETROSPECTIVE STUDY ON METFORMIN USE AND NEUROPATHIC PAIN

Amber Taylor, MD, Magdalena Skudlinska, Prathima Guruguri, Emil Annabi, Theodore Price, Hussein Yassine

Objective: The oral hypoglycemic agent metformin is long approved and widely used for type 2 diabetes treatment. Its mechanism has never been fully elucidated; however, recent research has found that metformin exhibits anti-oxidative properties and activates adenosine monophosphate activated protein kinase (AMPK). We have recently shown that metformin and other AMPK activators reverse neuropathic pain in preclinical models in rats and mice. Therefore, we hypothesized that metformin might decrease lumbar radiculopathy pain in humans.
Methods: We performed a chart review on more than 2000 patients who sought care from a university pain specialist for lumbar radiculopathy between Jan 2008 and Nov 2011. They were asked to fill out a standardized questionnaire regarding their pain qualities. Patients coincidentally taking metformin at the time of the visit were considered the treatment group and were matched with subjects having lumbar radiculopathy but not taking metformin therapy. Patients with diabetic complications or pre-existing peripheral neuropathy were excluded. We matched the treatment and control groups on age, sex, BMI, socioeconomic status, and comorbidities. These included stroke, depression, diabetes, heart disease, and pain medications (in particular opioids, TCAs, and benzodiazepines). The outcomes were individual qualitative and quantitative pain scores, total pain experience, pain at the time of visit, and interference with daily functions. Responses of pain questionnaires were recorded and analyzed using Pearson chi square and t tests.
Results: We selected a total of 45 patients on metformin (age 59.3, BMI 33.2) and 58 controls (age 55.1, BMI 31.3), most of whom had diabetes. Mean number of pain medications taken were 2.5 in treatment group versus 2.7 in controls (p=0.56), with mean onset of pain 9.9 years ago on metformin and 10.9 years ago for those not on therapy (p=0.64). Individual subtypes of pain were analyzed, both "hot burning" and "aching" were more likely to be observed in those not taking metformin vs. treatment group (p=0.08, and 0.02 respectively). Those on metformin were less likely to experience interference with their working routine (p=0.1), interference with relations with others (p=0.09) and decreased enjoyment of life (p=0.02).
Discussion: Metformin therapy is associated with decreased severity of neuropathic pain independent of diabetes status.
Conclusion: These finding support mechanistic studies from preclinical models and warrant a prospective study of metformin on neuropathic pain.

Abstract #256

COEXISTING AUTOIMMUNE DISEASE IN ADULTS WITH TYPE 1 DIABETES MELLITUS (T1DM).

Prajesh Joshi, MD, Janet McGill, MD, Kavita Juneja, MD

Objective: The objective of this study was to evaluate prevalence of coexisting autoimmune diseases in adults with T1DM and to determine whether age, gender, race, duration of diabetes or age of onset of T1DM has an effect on the likelihood of developing additional autoimmune diseases.
Methods: We performed a cross-sectional study of 569 adult subjects of age more than ≥18 years with T1DM from Washington University and community endocrinology practices. All subjects completed a structured questionnaire either online, over the phone or using a paper questionnaire. Chart review was performed to confirm the diagnoses listed in >50% of questionnaires to validate the responses.
Results: Out of 569 subjects, 60.1% (342/569) were female. The mean age at the time of survey of respondents was 45 ±15.5 years with 37% (211/569) of them ≥50 years of age. At least one coexisting autoimmune disease was present in 34.3% (195/569) of T1DM subjects. Autoimmune diseases were more common in female (76.4%) than male (23.6%). Hypothyroidism (20%) was the most common coexisting autoimmune disease followed by hyperthyroidism (6%), pernicious anemia (3%), vitiligo

(2.8%), rheumatoid arthritis (2.3%), psoriasis (2.1%) and celiac disease (1.8%). The ratio of hypothyroidism to hyperthyroidism was higher in Whites (112/29) compared to African-Americans (1/5) at $p < 0.05$. The prevalence of coexisting autoimmune diseases increased with age; consequently many autoimmune diseases were diagnosed after the age of 30 in subjects with T1DM. At least one coexisting autoimmune disease was present in 14% of index cases with T1DM of ≤30 years incomparison to 52% of T1DM ≥60 years.

Discussion: T1DM is associated with both organ-specific and systemic autoimmune diseases, however data about the prevalence of coexisting autoimmune diseases in older ages, in African-Americans vs. Whites and in persons diagnosed with T1DM at older ages is sparse. This study confirmed the increasing prevalence of coexisting autoimmune diseases with advancing age in subjects with T1DM. We also demonstrated an ethnic difference in the type of thyroid disease likely to occur, with African-Americans at higher risk for hyperthyroidism compared to White patients.

Conclusion: In this cross-sectional study using structured questionnaire, the prevalence of coexisting autoimmune diseases in subjects with T1DM was found to increase with age. African-American patients with T1DM may be more susceptible to hyperthyroidism than Whites. Adult endocrinologists should continue to screen for autoimmune thyroid disease, and others as clinically indicated.

Abstract #257

THE EFFECT OF CLINICAL PHARMACIST FOLLOW-UP AND DIABETES EDUCATION ALONG WITH MEDICATION IN ACHIEVING A1C GOAL: THE HOWARD UNIVERSITY HOSPITAL DIABETES TREATMENT CENTER EXPERIENCE

Anteneh Zenebe, MD, Gail Nunlee-Bland, MD, Yassin Mustafa

Objective: Diabetes Mellitus is costly, affecting 285 million people globally. Diabetes education, involving nurse educators, nutritionists and physicians in a multidisciplinary team approach, plays a vital role in glycemic control. Pharmacists have become more active in improving medication compliance in diabetic patients.

Methods: This study was designed to examine the effect of clinical pharmacist intervention and diabetes education in reducing A1c. The study was conducted on a total of 273 diabetic patients attending the Howard University Hospital Diabetes Treatment Center. Patients 18 years or older with T2DM were included in the study. Patients had either diabetes education with a certified diabetes educator or medication adherence intervention with the clinical pharmacist, or both. Charts were reviewed from the electronic medical record retrospectively from January 2007 through August 2011. Pre and post-intervention A1c levels were compared. Statistical analysis using t-test, ANOVA and Bonferroni were used to analyse the data

Results: There were a total of 273 diabetic patients included in the study of which 190 patients had diabetes education alone, 22 patients had only pharmacy medication adherence intervention, and 61 patients had both interventions. The mean age was 58.95 +/- 12.53 years with 174 females. The average pre-intervention A1c was 9.15 +/- 2.62 % and post intervention A1c was 8.23 +/- 2.23% for the entire group.

Discussion: There were no significant differences between age or sex in the groups. There was no significant difference in baseline A1c between diabetes education alone and pharmacy medication adherence alone, but there was a significant difference in the combination pharmacy and diabetes education intervention group. However, when comparing the effectiveness of each intervention, significant decreases in A1c were found in diabetes education alone and diabetes education plus pharmacy intervention, ($p=0.019$ and $p=0.003$), respectively. There was no significant decrease in A1c in the pharmacy group alone ($p=0.224$). When using the Bonferroni multiple comparison test, the impact of A1c reduction in the combination diabetes education and pharmacy intervention group was due to the education.

Conclusion: In this retrospective study, diabetes education appears to be more effective in A1c reduction. We recommend further randomized controlled trials to compare the effectiveness of diabetes education and pharmacy medication adherence interventions in diabetes management.

Abstract #258

UTILITY OF THE HEMOGLOBIN A1C AS A SCREENING TOOL FOR GESTATIONAL DIABETES MELLITUS

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Objective: Gestational diabetes mellitus (GDM) has been defined as any degree of glucose intolerance with onset or first recognition during pregnancy. GDM correlates with maternal and fetal complications, including preeclampsia, fetal macrosomia, birth trauma, shoulder dystocia, and neonatal hypoglycemia. Early treatment that limits exposure to hyperglycemia mitigates GDM-associated morbidity. GDM diagnosis has traditionally

relied on algorithms based on the oral glucose tolerance test (OGTT). The glycosylated hemoglobin (HbA1c) level is a simple, low-cost test that has been validated as a screening tool for diabetes mellitus. We performed a study to evaluate the utility of the HbA1c as a screening tool for GDM.

Methods: In a study involving all women who presented to the outpatient obstetrics clinics of one large academic center, a HbA1c level was measured at the first prenatal visit. Per ADA criteria, women with HbA1c ≥ 6.5% were diagnosed with GDM. Those with HbA1c values between 5.7% and 6.4% underwent a 50 g non-fasting OGTT. GDM was diagnosed if the plasma glucose was ≥ 200 mg/dl 1 hour after glucose ingestion. Women with 1-hour plasma glucose levels between 140 and 200 mg/dl underwent a 100 g fasting OGTT. GDM was diagnosed if ≥ 2 plasma glucose levels were above normal limits (95, 180, 155, or 140 mg/dl at fasting, 1, 2, or 3 hours, respectively). Women with normal first trimester screening tests were rescreened at 24-28 weeks of gestation, per standard recommendations.

Results: First trimester HbA1c levels were measured in 1372 women. Among the women with no known history of diabetes, 136 had an abnormal HbA1c. Twenty-nine of these women were diagnosed with GDM, 17 in the first trimester and 12 later in gestation. To date, chart review of women with normal first trimester HbA1c values has identified 29 women who were diagnosed with GDM later in gestation.

Discussion: We used an algorithm that incorporates the HbA1c to screen for GDM among women presenting for routine prenatal care. Of the women diagnosed with GDM, 17 were identified early in pregnancy, allowing early treatment and potentially reducing maternal and fetal morbidity. An ongoing study focuses on combining the HbA1c with other factors such as age, family history of diabetes, ethnicity, and body mass index, to more accurately identify women at risk for GDM early in pregnancy. Further study is needed to validate this screening tool and to examine its cost-effectiveness.

Conclusion: This study demonstrates a potential role of the HbA1c in the early diagnosis of GDM. The future aim is to incorporate a more comprehensive risk factor profile to develop a valid, cost-effective screening tool.

Abstract #259

THE PREVALENCE OF SLEEP DISORDER,RELATION WITH OTHER CARDIOVASCULAR RISKS AND IMPACT ON GLYCAEMIC CONTROL IN TYPE 2 DIABETES

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Objective: Sleep dysfunction (SD) is linked with insulin resistance in type 2 DM.This disorder affects the neuroendocrine regulation of glucose metabolism and may be a potential cardiovascular risk factor.Therefore, this study is set out assess the qualitative, quantitative aspect of sleep dysfunction, atherogenic correlates and impact on glycaemic control.

Methods: This is a prospective study in which 150 patients with type 2 DM were randomly selected. Interviewer based questionnaire were administered. Sleep dysfunction was assessed using Pittsburgh Sleep quality Index and Epworth Sleepiness scale. Fasting venous blood and urine samples were analysed for biochemical indices and statistical analysis carried out using SPSS 17.p value < 0.05 is significant. The atherogenic indices between the study subjects with SD and those without SD were compared.

Results: The prevalence of sleep disorder was 53.8% with a male preponderance of 64%.The BMI (kg/m2) and neck circumference(inches) in the patients with sleep disorder were higher but statistically insignificant (28.77 + 5.6 vs. 28.38 + 7.93 and 13.53 +2.57 vs. 13.44 + 0.99 p=0.06). 50% of obese DM suffered from SD. Elevated LDL-C and reduced HDL-C levels(mg/dl) were significantly higher in subjects with SD compared to those without SD (136.39 + 36.22 vs.135.79 +37.9 p=0.05 and 44.38 + 13.27 vs. 47.71 + 14.44, p=0.02 respectively).The mean levels of uric acid (mg/dl) for male (M) and female (F) were high but statistically insignificant. (M=9.13 +1.28 vs. 5.99 +0.83, F= 7.72 + 1.58 vs. 4.96 +0.88, P>0.05). Although short and long -term glycaemic indices were higher in the SD but this is statistically insignificant (FBS=195.09 +54.8 vs99.7 +19.02, 2hpp= 234.96+88.73vs 110.66+16.53 and HBAic= 9.92+2.68vs8.22+2.11 p>0.05).Moreover,81% of subjects with SD had hypertension which was comparable to 68% without SD,p=0.24.

Discussion: The occurrence of SD in Type 2 DM was high. Male factor being a significant risk factor.33.8%(26) of subjects with SD were obese.There was significant association of SD with elevated LDL-C and reduced HDL-C. However,mean levels of uric acid,FBS,2HPP,HBAic and prevalence of hypertension were comparable in both groups.

Conclusion: SD is a prominent feature in type 2 DM and is strongly associated with male gender, elevated LDL-C, reduced levels of HDL-C and greater risk of cardiovascular complications.

Abstract #260

COMMUNITY ACQUIRED PNEUMONIA IN ELDERLY DIABETICS (CAPED)

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Objective: According to CDC data from 2009, pneumonia is the 8th most common cause of death in the United States and ranks 2nd among infectious causes of mortality. Over 5 million people develop pneumonia every year, mostly adults over age 65. Hyperglycemia has been correlated with increased morbidity in individuals admitted for cardiovascular causes but limited information is available for individuals admitted with hyperglycemia and community acquired pneumonia. In our study, we evaluated the association of hyperglycemia with mortality, length of stay and re-admission rates in patients over 65 admitted with community acquired pneumonia.
Methods: Observational data were extracted from admissions to The University of Kansas Hospital for community acquired pneumonia from January 1, 2008 to December 31, 2010. We examined the effects of both the first glucose measure upon admission and age on: hospital mortality, length of stay, and readmission for community acquired pneumonia within 30 days. Initially, our cohort had 857 hospital admissions. To analyze the 30-day readmission outcome, we removed subjects that died or were discharged to hospice on their initial hospitalization, leaving 797 subjects to analyze the readmission outcome. Multivariable results also adjusted for comorbid conditions.
Results: We found little impact of first glucose measures on in-hospital mortality ($p=0.94$), length of stay ($p=0.95$), and 30-day readmission ($p=0.56$). Subjects 65 years and older trended towards higher in-hospital mortality than those aged 40-64 ($p=0.13$). Age was also associated with increased length of stay, as those ages 40-64 had shorter hospital durations compared to those ages 65 and older ($p=0.01$). Thirty-day readmission rate was higher among subjects over 65 than for those ages 40-64 ($p=0.03$). Of the comorbid conditions, cancer ($p<0.01$) and heart failure ($p<0.01$) were associated with increased in-hospital mortality; cancer ($p<0.01$), chronic renal failure ($p=0.05$), and cirrhosis ($p=0.03$) were associated with longer lengths of stay; and cancer ($p=0.02$) and cirrhosis ($p<0.01$) were associated with higher 30-day readmission rates.
Discussion: Although age was associated with increased length of stay and 30-day readmission rate, our retrospective observational data revealed that first glucose measure upon admission did not predict hospital mortality, length of stay, and readmission for community acquired pneumonia within 30 days.
Conclusion: Further study needs to be done in a prospective fashion to verify these results and better understand the interaction between in-hospital hyperglycemia and community acquired pneumonia.

Abstract #261

ADULT DIABETIC KETOACIDOSIS ASSOCIATED CEREBRAL EDEMA

Jeremy Anthony, MD, Edward Chin, MD

Objective: Present a case of diabetic ketoacidosis (DKA) associated cerebral edema (CE) in an adult, discuss risk factors and management.
Case Presentation: An 18 year old man with poorly controlled type 1 diabetes mellitus presented with nausea, vomiting and hyperglycemia for one day. Evaluation showed arterial blood gas, pH 7.05 and PC02 of 17. He was treated with intravenous fluids and insulin for DKA which resolved within 24 hours. Forty-eight hours after admission, the patient became confused and vomited. He progressively lost consciousness and was intubated for airway protection. Head CT scan showed generalized cerebral edema. IV hydrocortisone was given and he was transferred. On arrival, he was unresponsive, areflexic and had fixed dilated pupils. Repeat CT scan showed progression of diffuse cerebral edema with interval development of uncal and tonsillar herniation. Invasive monitoring demonstrated an intracerebral pressure of 87 mmHg (normal 7-15). The patient received intravenous mannitol, continued to deteriorate and despite maximal therapy died within seven hours of arrival.
Discussion: CE is a rare, devastating complication of DKA with 95% of cases occurring in individuals under 20 years of age. DKA associated CE is associated with a 50-90% mortality rate and 30% of survivors suffer permanent neurologic damage. Clinically significant CE typically occurs 4 to 12 hours after DKA treatment initiation. Some DKA patients develop radiographic evidence of CE in the absence of clinical symptoms. Mental status changes or severe headache may be present for some time before clinical deterioration which may vary from progressive coma to gradual improvement followed by sudden loss of consciousness to respiratory arrest. Risk factors for developing clinically significant CE are blood pH less than 7.1, PCO2 less than 20 mmHg, low arterial bicarbonate concentration, bicarbonate use, prolonged acidosis prior to treatment, high rate of initial fluid administration, rapid correction of serum glucose and high serum BUN. Proposed etiologies include osmotic edema due to rapid correction of hyperosmolarity and brain cell ischemia induced edema due to vasogenic and inflammatory

mediators. Glucocorticoids and diuretics have not been universally successful in treatment. IV mannitol, within 5 to 10 minutes of neurologic deterioration, hyperventilation, and intracranial monitoring may improve outcome.
Conclusion: CE is the main cause of DKA associated death in children. While CE is rare in adults, mortality remains high. Risk factor awareness, prompt recognition, and immediate therapy are critical as patients transition from pediatric to adult care.

Abstract #262

CASE SERIES ON TROPICAL DIABETIC HAND SYNDROME (TDHS)

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Objective: To report 3 cases of TDHS in DM patients.
Case Presentation: First patient is F/47yr,diabetic of 8yrs duration that presented with insidious swelling of right index finger and fever both of 2wks duration.Examination revealed swelling of the index finger extending up to the radial side of the palm over the second metatarsal with differential warmth and tenderness over the index finger and palm.Wound swab yielded growth of Staph aureus sensitive to ofloxacin and ceftriaxone,while X-ray of affected hand showed soft tissue swelling.RBS=292mg/dl,HbA1c=12%.She had wound debridement.Second patient is a F/30yr recently diagnosed diabetic who presented with ulcer which started as a painful swelling on the right hand of 1wk duration.Examination revealed tender swelling of thenar aspect of right hand with associated swelling of the distal third of right forearm with differential warmth and pus collection in the thenar eminence.Wound swab yielded growth of Klebsiella species sensitive to imipenem and ceftazidine. HbA1c=14%, RBS=312mg/dl.She had wound debridement.Third patient is a M/53yr,diabetic of 2yrs duration who presented with 4 wks history of insidious ulcer in the middle finger of the right hand.Examination revealed an ulcer in middle phalanx of the mid-finger with gangrenous distal phalanx and necrotic tissue slough. X-ray of the affected hand showed soft tissue swelling with evidence of subcutaneous emphysema involving the right mid-finger. Wound swab yielded growth of Klebsiella sp. sensitive to Ofloxacin and Ceftriazone. RBS=262mg/dl,HbA1c=8.6%.He had disarticulation of the right middle finger.
Discussion: TDHS is a term used to describe diabetes complication of the hand affecting people in the tropics. It encompasses celluitis of the hand,swelling,fulminant sepsis and gangrene.It is less well recognized and reported than foot ulcers.It typically occurs in a female with type 2 DM,50-60years of age,with poor glycaemic control,history of mild trauma,low socioeconomic status,malnutrition and delay in treatment.They present with severe necrotizing infection,swelling gangrene,mostly mixed bacterial growth(Staph aureus,gram negative organism).Treatment involves hospitalization,hand elevation,broad spectrum antibiotics,optimization of glycaemic control,surgical drainage,amputation and rehabilitation.Outcome is poor with amputation,disability and death is not uncommon.
Conclusion: Prevention involves patient's education about hand care,good nutrition and early presentation.These cases emphasizes the importance of early recognition and treatment of TDHS by clinician in developing countries to prevent complications which have potential for socioeconomic burden.

Abstract #263

EFFECTS OF THE IMPLEMENTATION OF A SUBCUTANEOUS GLARGINE PROTOCOL ON GLUCOSE CONTROL IN POSTOPERATIVE CARDIOTHORACIC PATIENTS

Ahmet Ergin, MD

Objective: Hyperglycemia is common in critically-ill patients, especially after cardio-thoracic surgery and can be associated with bad outcomes. Glycemic control may improve these outcomes. Intravenous insulin infusion (IVII) is typically used in the immediate post-operative period and is subsequently transitioned to a subcutaneous (SC) insulin protocol. The objective of the study was to compare outcomes before and after the implementation of a protocol using SC glargine at transition from IVII.
Methods: This was a retrospective review of post-cardiothoracic surgery patients. The setting was cardiothoracic intensive care units in a single institution. In August 2006, the authors' institution started using glargine and supplemental rapid-acting insulin (SSI) at transition from IVII (group 1). Before that month, only supplemental insulin was used (group 2). The Primary outcome was first glucose (BG1) after discontinuation (DC) of IVII. Secondary outcomes were absolute difference between last glucose before DC of IVII (BG0) and BG1, mean glucose in first 24 hours after DC of IVII (BG-24), need for SSI, hypoglycemia as well as other outcomes.
Results: Ninety-three patients before August 2006 and 100 after that month were eligible. Seventy-eight had diabetes (DM) and 115 did not. Mean BG0, mean BG1, Mean BG-24, the difference between BG1 and BG0 and the difference between BG-24 and BG0 were not statistically different between the 2 groups. Among DM patients, those who had received glargine had lower mean BG1 and lower mean BG-24 than those who had not received glargine

(14.5 vs. 33.2 mg/dL, p=*** and 163.8 vs. 177.9 mg/dL, p=*** respectively). A higher proportion of patients with DM needed SSI compared to those without DM (81% vs. 35%, p<0.0001), the former group needed less cumulative doses of SSI than the latter (Mean 9.2 units vs. 3.8 units, p<0.0001). Among patients without DM who received glargine, 43% needed SSI compared to 29% of those who did not receive glargine (p=0.086), whereas in patients with DM who received glargine, 75.5% needed SSI compared to 89.7% of those who did not receive glargine (p=0.106). Only 1 patient had a glucose of 58 mg/dL; otherwise, no glucose <70 mg/dL was recorded. Four patients (2 in Group 1 and 2 in Group 2) had sternal wound infections; 2 had received glargine and 2 had not.

Discussion: In this cohort of retrospectively reviewed post-cardiothoracic surgery patients, glargine administered at the cessation of IVII did not provide any meaningful benefit to subsequent glucose control except in patients with DM

Conclusion: Prospective studies with larger number of patients will be needed to show clinically significant benefits of this intervention.

Abstract #264

DIABETIC MYONECROSIS

Vijayaratna Chockalingam, M.B.B.S

Objective: Diabetic myonecrosis is a rare microvascular complication of diabetes mellitus. Clinicians should be aware of this condition to avoid overzealous management. Our objective is to describe the pathogenesis; clinical, radiological features and treatment of diabetic myonecrosis.

Case Presentation: A 56-year-old African-American male with a 14-year history of type 1 diabetes mellitus, end-stage renal disease, status post-kidney transplant, peripheral neuropathy, and deep venous thrombosis presented with left thigh pain and swelling. Examination showed swelling, redness, left thigh tenderness, left knee joint swelling, and knee joint limitation of movement. The patient's left thigh circumference was 10 cm more than that of the right thigh. Laboratory tests were unremarkable except for hemoglobin of 10.9 gm/dL (reference range 12-16), creatinine of 1.5 mg/dL (0.5-1.4), and aldolase of 14.7 U/L (0-7.6). MRI of the left thigh showed large regions of increased T1 signal intensity in the vastus intermedialis and lateralis along with edema involving the entire anterior compartment of the left thigh. Small collection within the abductor magnus muscle with peripheral enhancement represented early myonecrosis. The patient underwent ultrasound-guided aspiration of the thigh. The aspirate showed few neutrophils, and no organisms, anaerobes, acid-fast bacilli, or fungi were identified. Muscle biopsy showed end-stage muscle. The patient was treated conservatively with rest and analgesics, and his diabetes was well-controlled in the hospital. The patient had gradual clinical improvement and was discharged home.

Discussion: Diabetic myonecrosis occurs in long-term, poorly controlled, type 1 diabetic patients with multiple microvascular complications. Microangiopathy and hypoxia-reperfusion injury causing severe inflammatory response and edema are the likely underlying mechanisms. Our patient had a classic presentation with involvement of the quadriceps and hamstrings. Patients can have normal or elevated creatine kinase. MRI, the radiological test of choice, will show increased intensity within the affected muscle, sub-cutaneous edema, and subfascial fluid. Muscle biopsy is the confirmatory test for diabetic myonecrosis and will show muscle necrosis, edema, and occlusion of arterioles and capillaries by fibrin. Conservative treatment with rest, analgesics, and aspirin is recommended.

Conclusion: Clinicians should consider diabetic myonecrosis as an etiology in poorly controlled diabetic patients with sudden onset muscle pain, initiate work-up with MRI, confirm with a muscle biopsy, and treat conservatively.

Abstract #265

INSULIN RESISTANCE: CASE REPORT AND REVIEW ON MECHANISM, CURRENT CONCEPT AND MANAGEMENT

Yin Oo, MD, Jocelyne Karam, MD, Christine Resta, MD

Objective: In hospitalized patients, development of transient insulin resistance related to different medical conditions like acute myocardial infarct (MI), sepsis and medications has been reported. However, majority of us rarely come across with a case of extreme insulin resistance. Here, we are reporting a case of extreme insulin resistance in a patient admitted with diabetic ketoacidosis (DKA) and MI.

Case Presentation: A 60 years-old Hispanic man with type 2 diabetes mellitus for 20 years admitted with DKA, acute renal failure and non-ST-elevation myocardial infarct. The initial anion gap was resolved after receiving 657 units of regular insulin over 24 hours. However, he was noted to be in DKA again on the 3rd day of hospitalization without evidence of recurrent ischemia or infection. His blood glucoses had been in high to 500s with insulin infusion rate of 76 units/hour from day 3 to day 4 of hospitalization. On day 5, his anion gap was closed. However, from day 5 to day 10 his insulin requirements were varied from 120 units/hour to 1 unit/hour. The work up for possible causes of

extreme insulin resistance and autoimmune work up were non-significant except positive insulin autoantibodies. On day 25 of hospitalization, he had coronary artery bypass graft (CABG) as coronary angiogram showed triple vessels coronary artery disease. Though we expected him to have increased insulin requirement in perioperative period of CABG, contradicts to our expectation, the insulin requirement and fluctuating in blood glucoses were significantly improved after CABG.

Discussion: The pathophysiological stressful conditions like DKA and MI cause insulin resistance by increasing the level of catecholamines and cortisol through activation of the sympathetic nervous system and hypothalamic-pituitary-adrenal axis respectively. In addition, the up-regulation of pro-inflammatory cytokines interferes with insulin signal transduction and creates insulin resistance. Although the role of low pH in terms of effect on insulin binding, biologic activity of insulin or rate of insulin degradation were not seem to explain the development of insulin resistance in DKA as per Yokoyama H et al, those areas should be re-examined. As insulin resistance resolved after CABG, the effect of clinically unapparent oxidative injury at cellular level on insulin action as described in the animal models by Ohta Yet al, should be explored further to understand the pathophysiology of extreme insulin resistance in MI patients.

Conclusion: In conclusion, the exact mechanism of development of extreme insulin resistance in DKA and MI is still unknown. However, we believe it is not solely due to excess of counter-regulatory hormones or inflammatory cytokines.

Abstract #266

INSULIN REQUIREMENTS IN DIABETIC PATIENTS WITH HEART FAILURE BEFORE AND AFTER LVAD

Sandra Barrow, MD, Dale Hamilton

Objective: Advanced heart failure is associated with insulin resistance and new onset diabetes mellitus. Left ventricular assist devices (LVAD) are approved for long-term heart failure therapy. Clinical observations suggested that insulin resistance improves after LVAD. This has now been recently reported in a small retrospective study by Uriel et al. Our study was aimed to confirm the metabolic effect of heart failure treatment with LVAD on a larger patient population and to establish preliminary data on a possible physiologic mechanism by distinguishing between improvement of insulin resistance in the liver versus the muscle and fat compartment.

Methods: We conducted a retrospective chart review and identified 30 patients with diabetes who received a Heart Mate II LVAD device at the Methodist Hospital in Houston, TX. Diabetes specific data collection included hemoglobin A1c before and after LVAD as well as fasting blood glucose, random blood glucose (average of 3) and daily insulin requirements at 48 hours before and at 7, 14, 21 and 28 days after LVAD. ALT levels were collected within 1 week prior and 1-3 months after LVAD and compared to ALT levels obtained from 31 non-diabetic patients with heart failure who also received LVAD therapy.

Results: Hemoglobin A1c was reduced from 8.56 +/- 1.8% to 6.78 +/- 1.01% (p=0.011) at follow up 7.3 +/- 3.4 months after LVAD. Fasting blood glucose averaged 122.8 +/- 41.8 mg/dl before LVAD and showed a reduction to 117.6 +/- 42 mg/dl (p=0.51) at 28 days after LVAD. Random blood glucose levels were decreased by 27.9 mg/dl averaging 170.9 +/- 54.8 mg/dl 48 before LVAD and 143.0 +/- 32.6 mg/dl after LVAD (p=0.056). Stable or even improved blood glucose control was achieved despite a reduction of daily insulin requirements by 44% (43.53 +/- 50.5 units vs. 24.23 +/- 24.4 units/day; p=0.036). ALT levels were significantly higher in the diabetic patient population when compared to the non-diabetic group before LVAD (92.8 +/- 137 U/l vs. 37.2 +/- 30 U/l; p= 0.03) and showed impressive improvement after LVAD to 29.8 +/- 32 U/l (p= 0.008).

Discussion: Our study confirmed that LVAD reduces insulin requirements and long term glucose control. Fasting glucoses are maintained despite lower insulin requirements. Before LVAD, ALT levels serving as an indirect marker for hepatic insulin sensitivity are higher in the diabetic heart failure group and improve significantly after LVAD suggesting reduced hepatic insulin resistance. Lastly, a downward trend in random blood glucoses was observed and may reflect improvement of peripheral insulin resistance as well.

Conclusion: Further studies are warranted to further characterize the pathomechanism of these effects.

Abstract #267

USER ACCEPTANCE OF THE MYSENTRY REMOTE MONITORING SYSTEM

John Welsh, MD, Kevin Kaiserman, Gnanagurudasan Prakasam, Fred Gunville, Robert Slover, Bruce Buckingham, Xuan Nguyen, Francine Kaufman, MD, Scott Lee, MD

Objective: Continuous glucose monitoring (CGM) sensors are typically connected to low-power radio frequency transmitters. The mySentry system consists of an outpost device placed near the patient which amplifies and relays pump and CGM data to a remote monitor so that parents

or caregivers elsewhere in the home can be alerted to abnormal glucose values or trends. We evaluated the usability and acceptance of the system.

Methods: Each enrolled family included a child (age 7-17) using a MiniMed Paradigm REAL-Time Revel sensor-augmented pump system (SAP) to treat type 1 diabetes, and a parent or caregiver. Surveys were conducted before and after 3 weeks' use of the mySentry system. Parents or caregivers rated their agreement with statements related to acceptability and usability of mySentry on a 7-point Likert scale. Responses ≥4 were considered affirmatory. Narrative comments regarding mySentry were also solicited from participants and from the study's investigators. No formal hypothesis testing was performed.

Results: Thirty-six children enrolled and 35 completed the study. Enrolled children (61.1% female) had a mean age of 11.9 years, mean age at diagnosis of 5.4 years, and mean age at initiation of pump therapy of 7.1 years. Most parents reported that their children had nocturnal low or high blood sugars (91.2% of responses ≥4) and being fearful of their unawareness of these excursions (80% of responses ≥4). All respondents found the mySentry controls easy to understand during set-up, found the mySentry alarms easier to hear than alarms from the pump, and found the display easy to read. All respondents reported that mySentry was easy to use, gave them greater confidence in managing their child's diabetes at night, and would recommend it to other parents or caregivers. Investigator surveys showed that mySentry could be expected to provide parents and caregivers with increased comfort at night and assist them in successfully managing their child's diabetes. Narrative comments included suggestions to allow for more extensive adjustment of the screen's brightness and to provide more robust inter-device communication. One severe adverse event occurred, and it was unrelated to the study device. There were no unanticipated adverse device effects.

Discussion: The mySentry system met all predefined criteria for acceptability, was easy to set up and use, and did not demonstrate any safety issues.

Conclusion: Remote monitoring of children's sensor-augmented pump systems was associated with an improved level of comfort and confidence felt by parents and caregivers at night.

Abstract #268

TREATMENT OF OBSTRUCTIVE CORONARY ARTERY DISEASE WITH THE RESOLUTE ZOTAROLIMUS-ELUTING STENT IN PATIENTS WITH DIABETES MELLITUS

Scott Lee, MD, Francine Kaufman, MD, Jorge Belardi, Martin Leon, Laura Mauri, Ian Meredith, Franz-Josef Neumann, Shigeru Saito, Patrick Serruys, Petr Widimsky, Stephan Windecker, Alan Yeung, Sigmund Silber

Objective: Current revascularization guidelines support the use of drug-eluting stents for the treatment of obstructive coronary artery disease in patients with diabetes, yet no drug-eluting stent has obtained a specific indication from the FDA for use in this high risk patient population. The Resolute zotarolimus-eluting stent comprises a thin-strut cobalt chromium bare-metal stent and a durable, biostable polymer that allows prolonged drug elution (up to 180 days) for treatment of patients with complex lesions and clinical characteristics, such as those with diabetes. Reports from the RESOLUTE Clinical program have shown the Resolute stent to be safe and effective in patients with complex clinical and lesion characteristics. We evaluated 1-year clinical outcomes in patients with diabetes treated with the Resolute stent.

Methods: We evaluated 878 patients with Type 2 diabetes and 1903 patients without diabetes from 5 RESOLUTE trials, from a prespecified analysis cohort to obtain the indication for use of the Resolute zotarolimus-eluting stent in patients with diabetes in the US. Patients were treated for 1 or 2 lesions in separate vessels.

Results: Insulin-taking patients comprised 28.5% of the cohort. Patients with diabetes compared to those without were older (65.2 ±10.2 vs 63.5±10.8 years, $p<0.001$); more likely female (33.6% vs 25.6%, $p<0.001$), had more hypertension (87.6% vs 73.1%, $p<0.001$) and hyperlipidemia (86.2% vs. 76.0%, $p<0.001$); more had a prior percutaneous coronary revascularization (34.6% vs 29.5%, $p<0.001$) or a prior coronary artery bypass grafting (10.5% vs. 7.4%, $p=0.006$). The existence of a prior myocardial infarction at baseline was similar between groups (24.9% vs 25.5%, $p=0.72$). At 1 year, the composite of cardiac death, myocardial infarction, and revascularization of the target vessel occurred in 57 (6.6%) patients with diabetes compared with 92 (4.9%) patients without diabetes ($p=0.09$). There were no differences in the rates of stent thrombosis between patients with diabetes and those without (both 0.3%). Adverse events in patients taking insulin were higher than those not taking insulin (composite; 10.6% vs. 5.0%, $p= 0.006$). There were no statistically significant differences in the

composite endpoint between patients not taking insulin, and patients without diabetes (5.0% vs 4.9%, p=NS). ***Discussion***: Patients with diabetes and obstructive coronary artery disease may benefit from the Resolute stent.
Conclusion: The Resolute drug-eluting stent was shown to be as safe and effective in non-insulin requiring patients with diabetes compared to patients without diabetes. Both groups had similar cardiovascular outcomes at one year.

Abstract #269

SOME METABOLIC PARAMETERS IN SIBLINGS OF TYPE 2 DIABETES PATIENTS IN NORTHERN NIGERIA: A FOCUS ON BLOOD LIPIDS AND BLOOD GLUCOSE

Innocent Okpe, MBBS, FMCP, A. Fasanmade

Objective: This report seeks to identify early metabolic derangement in the siblings of Type 2 Diabetes patients who are apparently healthy. ***Methods***: A total of 107 siblings of Type 2 Diabetes patients aged between 30 and 75 years were compared with 105 apparently healthy matched controls without known family history of Diabetes. Anthropometric indices, Blood pressure, serum lipid profile including the atherogenic index, and fasting and 2hour post glucose load estimates were measured. Test statistics used include the student- T test and the chi-squared test for proportions and the level of statistical significance was set at <0.05
Results: The mean waist circumference in both males and females and the mean BMI was higher for the siblings of Type 2 Diabetes patients than in those without a family history of Diabetes {91.7(13.2),95.4(11.0) VS 85.9(8.2),86.6(9.2) AND 30.3(5.8) VS 28.7(4.8) P=0.001 and 0.035 }respectively. Twenty eight percent (30) of the siblings of Type 2 DM patients had their Triglycerides raised above 1.7mmol/L as compared to 12% (13) in those without family history of Diabetes.(p=0.004). The LDL-C was found to be abnormal in 40.2% (43) of the siblings of Type2DM as compared to 25.7%(27) of the controls. 26.1% (28) of the siblings of Type 2DM had combined dyslipidemia as compared to 11.2%(12) of the controls. It was observed that the siblings of Type2DM patients had statistically higher mean FBS and 2HrPP glucose levels when compared with the controls(6.28[4.28] and 8.21[5.99] vs 4.49[1.14] and 5.44[1.15]). Only 43.9%(47) of the siblings of Type 2DM patients had their blood glucose within the normal range as compared to 84.8%(89) of the controls p=0.00001. A total of 54.2%(58) of the siblings of T2DM patients had glucose dysregulation compared to 15.2%(16) of the controls. P <0.05. Age, sex, BMI and the WC were found to be significant determinants of glucose dysregulation and dyslipidemia in this study.
Discussion: The anthropometric and metabolic parameters studied in this research reveals a statistically significant derangement amongst the siblings of Type 2 Diabetes patients when compared to the control group as corroborated in several other similar studies, but whether these findings are influenced by environmental factors or could be solely accounted for by the genetic make-up of these siblings of Type Diabetes patients remains to be determined.
Conclusion: Early metabolic derangements in apparently normal subjects who are at risk of Type 2DM include dyslipidemia and glucose dysregulation and these are readily accessible and affordable tools in the global war against the epidemic of Type2DM.

Abstract #270

CLINICAL PROFILE OF TROPICAL DIABETIC HAND SYNDROME IN A TERTIARY HEALTH INSTITUTION IN NORTHERN NIGERIA

Anas Sabir, MBBS

Objective: The objective of this study was to determine the pattern of clinical presentation, causes and outcome of patients with tropical diabetic hand syndrome as seen in a tertiary health center in Nigeria over a three year period.
Methods: All patients admitted at Usmanu Danfodiyo University Teaching Hospital (UDUTH) Sokoto, Nigeria, from January 2008 to December 2010 with tropical diabetes hand syndrome were included. The data retrieved from the case files of the patients included the age, gender, blood glucose, type of hand lesion, wound swab m/c/s, treatment offered and the outcome.
Results: Of the thirty one patients diagnosed with TDHS, 18(58.1%) were females while 13(41.9%) were males. The mean (SD) blood glucose level at initial presentation was 18.1(5.9) mmol/l. The mean (SD) age of the study patients was 44.8 (6.1) years. Nine (29.1%) patients had hand ulcerations, 8 (25.8%) had cellulitis, 10 (32.2%) had palmar abscess, while 4 (12.9%) had localized gangrene. Wound swab cultures of hand lesions obtained yielded polymicrobial growth that included Staphylococcus aureus (36.4%), Pseudomonas aeruginosa(18.2%) and Escherichia coli (45.5%). Thirteen (41.9%) patients were completely healed after treatment, while18 (58.1%) had residual deformities.
Discussion: Tropical diabetic hand syndrome (TDHS) is a complication affecting patients with diabetes mellitus in the tropics, characterized by localized cellulitis and ulceration of the hands, to progressive, fulminant hand sepsis, and gangrene affecting the entire limb. This study showed that TDHS occurred in patients with Poor

glycemic control of diabetes mellitus irrespective of their age and sex.
Conclusion: Although all the patients survived, permanent disability was present in 58.1% of patients. There is the need for TDHS to be recognized early by clinicians in developing countries and treated aggressively to prevent occurrence of such complications with their resultant socioeconomic effects.

Abstract #271

MAURIAC SYNDROME. A CASE REPORT

Miguel Pinto, MD, FACE, Helard Manrique

Objective: To describe a case of Mauriac syndrome in a poorly controlled type 1 diabetes patient.
Methods: We abstracted the clinical chart and reviewed the pertinent medical literature.
Case Presentation: A 17-year-old woman with history of type 1 diabetes diagnosed since 6 years of age with poor glycemic control and chronic malnutrition came to Emergency room because of abdominal pain, nausea, vomiting, and diarrhea. iN THE PAST, she was admitted several times due to diabetic ketoacidosis and her insulin therapy and follow-up was erratic. She weighed 25 kg (under 3rd percentile) and was 123 cm in length (under 3rd percentile). Physical examination showed severe growth impairment, cushingoid facies, and absence of secondary sexual characteristics. Laboratory results showed a glycated hemoglobin A1c of 11.6%, IGF-1 of 55 ng/mL (NR: 182-780), IGFBP3 of 2.2 µg/mL (NR: 2.9-7.8), and TSH of 6.68µU/mL (NR: 0.3-5). Creatinine, serum albumin, ALT, AST, ALKP, and total bilirubin were normal. The abdominal ultrasound revealed no hepatomegaly or ascites, and bone age was delayed (9.5 years).
Discussion: Mauriac syndrome may present in poorly controlled type 1 diabetic children and adolescents. It is characterized by growth failure, hepatomegaly and cushingoid features. Optimal glycemic control may play an important role in preventing its occurrence. Several plausible nonexclusive mechanisms are suggested for growth failure in the Mauriac syndrome. They include: insufficient tissue glucose availability, decreased circulating IGF-I, and relative growth hormone resistant state. On the other hand, cushingoid features are related to secondary hypercortisolism in poorly controlled diabetes and hepatomegaly is due to deposition of glycogen in the hepatocytes.
Conclusion: Mauriac syndrome is a rare complication of type 1 diabetes, it should be considered in type 1 diabetes children with growth retardation and liver disease.

Abstract #272

PATTERN OF DYSGLYCAEMIA AMONGST PERSONS WITH MULTI-DRUG RESISTANT TUBERCULOSIS IN IBADAN

Arinola Ipadeola, MBBS, Olusoji Ige, Modupe Kuti, Jokotade Adeleye, MBBS

Objective: The objective of this study was to determine the pattern of dysglycaemia amongst persons with multi-drug resistant tuberculosis (MDR-TB) at the University College Hospital, Ibadan.
Methods: 17 persons being managed for MDR-TB participated in the study after informed consent had been obtained. Anthropometric measurements (weight, height, and waist circumference) and blood pressure recordings were taken. Oral glucose tolerance test was performed on all patients except in persons known to have DM and analysis for glycated haemoglobin was also done. The results obtained were analyzed using SPSS package version 16.
Results: Persons studied consisted of 11(64.7%) males and 6(35.3%) females with a mean (SD) age of 35.8(8.4) years. The mean (SD) Fasting plasma glucose (FPG) was 75.1(18.3) mg/dl while the mean (SD) 2hours post glucose load was 98.2 (22.8) mg/dl. The mean (SD) HbA1c was 4.3(0.4) %. Four persons (23.5%) had different degrees of dysglycaemia. One person had previously diagnosed DM; another person was newly diagnosed to have DM, while 2 (11.8%) other persons had impaired glucose tolerance. Only 1(5.9%) person was found to be obese, 1was underweight while the rest had normal BMI. In addition, 1 person had truncal obesity while 6(35.3%) persons had elevated blood pressure.
Discussion: Tuberculosis and diabetes are twin epidemics in many middle and low income countries. Some studies also reported that persons with DM and tuberculosis (TB) are more likely to develop MDR-TB; however the mechanism is yet to be determined. Approximately a quarter of persons in this study with MDR TB had varying degrees of dysglycaemia. Self reported incidence of DM amongst other ethnic groups with MDR -TB is reported to vary from 29 to 37%. Although there are conflicting reports as regards the relationship between DM and MDR-TB, their co-occurence has serious public health implications for persons with diabetes mellitus, as well as the treatment and control of tuberculosis.
Conclusion: The association and effect of diabetes mellitus on MDR-TB is often under-reported. There is a need for awareness, screening and education for patients and health care providers, especially because of the epidemic of both diseases in many countries.

Abstract #273

IMPLEMENTING THE GOALS OF THE HITECH ACT IN CLINICAL PRACTICE.

J. Prendergast, MD, Evelyn Castillo-Profeta, Abraham Silvers

Objective: The Health Information Technology for Economic and Clinical Health (HITECH) Act established programs to improve health care quality, safety, and efficiency through promotion of health information technology including electronic health records (EHR). Great emphasis is placed on physicians to improve the quality of health care and to monitor their patients more efficiently. A typical endocrine practice sees a large number of elderly patients with several comorbidities; type 2 diabetic patients are a prime example.
Methods: Using EHR data, three comorbidities were measured in our diabetic patients: autonomic nervous system (ANS) disorders, HgA1c, and blood pressure (BP). Abnormal ANS is associated with coronary disease and may worsen arterial inflammation. The FDA recently cleared a medical device, the ANSiscope. It measures activity of the parasympathetic and sympathetic systems with every heartbeat. It is more sensitive than conventional techniques, easy to perform and, importantly, not affected by subject variability. Within 5-10 min. it 1) captures the percentage of autonomic dysfunction and 2) classifies the severity of dysfunction.
Results: 93 patients (64 male) over age 60 with type 2 diabetes were evaluated for ANS status, HgA1c and systolic and diastolic BP at their first visit and at their most recent follow up visit (average 6 months). 8 patients exhibited a decrease of 0.7% in HgA1c from the 5.9% measured at first visit. 38 patients, also with a low HgA1c at initial visit (5.7%), demonstrated an increase of 2.1%. 46 patients, with a higher initial HgA1c (7.7%), had a decrease of 2.2%. A lower HgA1c at initial visit does not mean physicians should be complacent about glucose control. Only 2 patients had ANS values at initial visit that would be considered normal. 38 patients were abnormal and 53 were very abnormal.
Discussion: Clearly, using HgA1c to predict clinical success in this patient population may not be optimal. By using EHR we confirmed the importance of ANS. Abnormal ANS was correlated with HTN as initial systolic BP was > 140 mmHg in 1/3 of the patients and more than half had BP between 120 and 140 mmHg.
Conclusion: ANS identifies patients in whom aggressive intervention should be undertaken to prevent eventual, but treatable, cardiovascular complications, despite what appear to be reasonable HgA1c. BP alone may no longer be optimal patient care in this era of EHR. The results justify the hopes of Congress that EHR will improve the quality and efficiency of health care.

Abstract #274

ASSESSMENT OF PAIN AND TREATMENT SATISFACTION IN PATIENTS WITH PAINFUL DIABETIC PERIPHERAL NEUROPATHY

Latha Dulipsingh, MD, FACE, FACP, Susan Zailskas, MSN, RN, CDE, Teresa McInnis, RN, CDE, CCRC, Aniello Marotta, PharmD

Objective: The purpose of this project was to describe pain experienced from painful diabetic peripheral neuropathy (pDPN) and evaluate current treatment satisfaction in patients followed through an endocrinology clinic.
Methods: Patients ≥ 18 years of age with a diagnosis of diabetes, with or without diabetic peripheral neuropathy, were offered a voluntary self-administered questionnaire, prior to their examination by the healthcare provider. Only patients who had nerve pain were asked to complete the entire questionnaire. In addition to basic information related to diabetes, patients were asked if they experienced nerve pain, the characteristics of the nerve pain, and questions related to treatment and satisfaction with current therapy.
Results: A total of 98 questionnaires were collected with 53.1% of the patients being female in gender. The mean age was 55.1 years, 75.6% had type 2 diabetes and the mean duration of diabetes was 16.3 years. Thirty-one patients (31.6%) reported neuropathic pain with 83.3% having experienced nerve pain for one year or greater and the majority (67.7%) reporting nerve pain in their feet. The percentage of patients who reported moderate to severe nerve pain, score of ≥4 on the 0-10 numeric pain rating scale, over the past week was 54.8% with 32.3% reporting nerve pain "All Day " in the past 24 hours. Overall health for past 4 weeks was described as being "Good" to "Very Good" in 45.2% of patients. Impact of nerve pain on quality of life described as "Somewhat" , "Quite a lot ", to "Very much" was noted by 61.3% of patients although 64.5% of patients reported that nerve pain did not cause them to avoid activities. Fifteen (48.4%) patients reported receiving medication for their nerve pain of which 66.7% reported being satisfied with their treatment. Of the satisfied patients, 40% reported severe nerve pain, scoring ≥7 out of 10, over the past week with 30% reporting pain "All Day".
Discussion: Painful diabetic peripheral neuropathy is a common complication of diabetes mellitus and can have debilitating consequences with a significant impact on quality of life. Based on our questionnaire we found about a third of patients with long standing diabetes having

pDPN of which about two thirds of them reported that it affected their quality of life. Surprisingly, almost half of the patients reported being satisfied with their treatment despite severe nerve pain.
Conclusion: It is important for healthcare providers to recognize barriers to pain control in patients with pDPN. Our findings will serve as a foundation for a pDPN provider-patient initiative focused at improving education and communication at our clinic.

Abstract #275

LIMITATIONS AND PITFALLS OF SELF MONITORING OF BLOOD GLUCOSE: A CASE STUDY.

Nidhi Bansal, MBBS, Liviu Danescu, Harminder Grewal

Objective: Self monitoring of blood glucose (SMBG) is integral to the management of diabetes mellitus. Blood glucose meters should be sufficiently accurate to allow patients and clinicians to monitor and manage diabetes.
Case Presentation: A 59 y/o lady with history of diabetes mellitus type 2, peripheral neuropathy & hypertension presented with an episode of unconsciousness. Her fingerstick glucose was found to be 36 mg% by EMS. She responded well to glucagon and dextrose with no permanent neurological sequelae. Detailed interview revealed that her PCP increased her basal and bolus insulin doses on multiple occasions in recent months in accordance with glucometer records to improve glycemic control. Her insulin supplies and usage also corroborated to the prescribed doses to allay any suspicion of malingering. She did not admit to any suicidal ideation either. Physical examination was unremarkable. Complete metabolic profile was normal. C- peptide levels were consistent with exogenous insulin administration. Blood test for sulfonylureas and anti insulin antibodies were also negative, thus leaving this medical puzzle unresolved. In hospital, her fingerstick glucose ranged between 80-186 mg% with only half of her home insulin dose. Her HBA1C level was 6.8% indicating a better glycemic control than that suggested by glucometer readings. Our multidisciplinary team for diabetic management checked her glucometer for accuracy and discovered a discrepancy of more than 100 points. These erroneous records resulted in administration of excessive insulin doses thus precipitating hypoglycemia.
Discussion: Glucometers are the standard of care in SMBG. The ADA mandates a maximum error of 5 % for all glucometers. Multiple sample-related, analysis-related, and data display-related factors can interfere with the accuracy of the instrument. Computer simulated models show that there is 10 fold rise in hypoglycemic episodes when there is a permitted error of > 10% for SMBG. The clinicians should always consider this possibility in their approach to patients with hypoglycemia.
Conclusion: Our case again highlights the importance of multidisciplinary approach (diabetes education, instrument checks) to minimize errors and optimize diabetes management.

Abstract #278

BRITTLE DIABETES IN HEPATITIS C VIRUS INDUCED PANCREATITIS

Ayoola Oladejo, MBBS, FWACP, Michael Olamoyegun, MBBS, Abimbola Alabi

Objective: To report a rare case of diabetes mellitus secondary to Hepatitis C virus induced chronic pancreatitis and the challenge of achieving an optimal glycemic control
Case Presentation: : Patient is a 33 year old male who presented at our facility on account of recurrent episodes of polyuria, polydipsia and diarrhea necessitating hospital admissions at various times. The random plasma glucose estimation at admission was 479mg/dl with no detected ketones in his urine samples and glycated hemoglobin was 11.4%. A possibility of chronic pancreatitis was entertained because of the unusual presentation and associated chronic diarrhea with both the plain abdominal X-ray and Ultrasonography revealing pancreatic calcification. There was no prior intake of alcohol or abuse of alcohol and no history of malnutrition in childhood. HIV Elisa antibody screening and Hepatitis B surface antigen were both negative. Serologic screening for hepatitis C showed antibody to hepatitis C virus. The management of diabetes posed a great challenge with fluctuating episodes of hyperglycemia and hypoglycemia despite the use of low dose insulin therapy.
Discussion: Pancreatic disorders are much rarer causes of diabetes mellitus as compared to conventional types 1 and 2 diabetes mellitus accounting for over 90% of diabetes. Alcoholism accounts for over 90% of chronic pancreatitis with few reported cases of hepatitis C virus induced pancreatitis in available literature. Hepatitis C virus is known to cause some extra-hepatic manifestations such as cryoglobinemia and porhyria cutanea tarda.
Conclusion: Hepatitis C virus induced pancreatitis is a rare extra-hepatic manifestation of hepatitis C virus infection. The secondary diabetes that results from chronic pancreatitis poses a great challenge in achieving an optimal glycemic control

Abstract #279

THE ROLE OF THE CLINICAL PHARMACIST IN THE CARE OF PATIENTS WITH TYPE 2 DIABETES (T2DM) MANAGED WITHIN AN ENDOCRINOLOGY SPECIALTY CENTER

Eyob Makonnen, MD, Pharm D, Laura Young

Objective: The complex nature of T2DM and its multiple treatment goals are difficult to achieve in the limited time between patients and providers, particularly in those patients who have poorly controlled T2DM. Clinical pharmacist (PharmD) based management has proven to be successful in management of T2DM in the primary care setting. No studies to date have evaluated the effect of PharmDs in endocrine specialty clinics. We performed a retrospective, observational analysis to determine the impact of PharmD co-management along with an endocrinologist in patients with poorly controlled T2DM on glycemic and blood pressure (BP) control.
Methods: A retrospective chart review was completed. Seventy-four patients seen at the UNC Endocrine clinic between Jan 2009 and Aug 2011 with an A1 c >9% and co-managed by a PharmD and endocrinologist were identified.
Results: On average patients met with the PharmD 2-3 times (range 1-10). The average A1c in the year prior to PharmD intervention was 10.8+2.0%; the average A1c in the year following initiation of PharmD co-management was 9.9+1.7% (paired t-test p<0.001). Sixty percent did not meet BP goals (<130/80 mmHg) in the year prior to the working with the PharmD while 70% did not meet BP goals following PharmD co-management. This was not statistically significant nor was the change in average systolic BP or diastolic BP following PharmD co-management.
Discussion: Similar to previous results, improvements in glycemic control were observed following PharmD intervention. No statistically significant difference in the percent of patients attaining BP goals following PharmD intervention was observed. Given the lack of a control group, and our inability to control for potential confounders in the current design, we are presently collecting similar outcomes for patients in our practice that were managed only by an endocrinologist during the same time frame. This data will be presented along with the above described finings during the poster session.
Conclusion: Our findings suggest that the impact of co-management of high risk patients with T2DM with a PharmD and an endocrinologist may positively impact glycemic control, while benefits on BP control are not apparent. In most cases patients followed by the pharmacist will have had more frequent clinic encounters and phone follow-ups which could indeed explain the improvement in blood glucose. In our case-control analyses we will examine this issue and control for this factor if it is found to be a significant confounder. Further investigation is warranted to determine the most appropriate and effective use of PharmD co-management within the endocrine setting.

Abstract #280

DIABETIC PEOPLE - INCREASINGLY HEAVIER

Zdravko Kamenov, MD, PhD, DMedSc,
Rumyana Parapunova, Rumyana Georgieva

Objective: The aim of this study was to analyze the phenotype evolution in diabetic patients during an 18 years interval.
Methods: This retrospective cross-sectional study, conducted at the University clinic of endocrinology of the Medical University-Sofia, consists of four consecutive periods of 3 years each, starting in 1990(1st period), 1995(2nd), 2000(3rd) and 2005(4th) years. The manually written hospital medical records, collected for 18 years, for 2031 patients >18 years old with DM1 and DM2, hospitalized for the first time, were analyzed. Data about gender, age, height, weight, BMI, waist (WC) and hip (HC) circumferences and W/H ratio, diabetes type, duration, glucemic control, micro-and macrovascular complications/co-morbidities was collected.
Results: The prevalence of DM2 was 84% and female were 55.6% of the patients. The age (mean±SD) in DM1 (mean 32,9±13,4) significantly increased (28,4±11,6 vs.35,1±13,1;p<0.001) but in DM2 (mean 60,0±11,9) decreased (60,7±11,7 vs.59,2±11,8;p<0.05) in the course of four periods. Women with DM2 (61,8±11,0) were considerably older than men (57,7±12,7 years; p<0.001). No gender age-difference was observed in DM1. Significant increase in height (+3.5 cm) and weight (+9.7 kg), resulting in a BMI increase of 2.2 kg/m2 (all p<0.001) was observed from 1st to the 4th periods, more pronounced in DM2 (74,0±14,6 vs. 84,5±19,4 kg and 28,2 ±5,6 vs. 30,5±6,3 kg/m2;p<0.001). WC and HC increased in the whole population - more demonstratively in DM2, where W/H increased from 0,92±0,11 to 0,95±0,08 (p<0.001). Obese were 26.6% of all patients in the 1st and 41.9% during the 4th period (p<0.001). Diabetes duration decreased in DM2 and increased in DM1. HbA1c was significantly (p<0.001) higher before 2000 year (9,9±2,4%), compared to the last decade (8,6±2,0 and 8,8±2,1%). Although the prevalence of arterial hypertension increased, those of coronary-, brain- and peripheral artery disease decreased. Compared to men, women with DM2 had higher prevalence of obesity

(46,3 vs. 32,0%;p<0.001), hypertension (86.7 vs. 77.8%; p<0.001) and dyslipidemia (61.2 vs. 55.0%;p<0.01).
Discussion: Obesity is a proven risk factor for diabetes type 2 (DM2). Less is known about the opposite link - how DM2 and its treatment impact the weight. Increasing efforts for tight glucemic control often induce hyperinsulinemia and weight gain, which amplify the underlying insulin resistance, facilitate the exhaustion of the beta-cells and accelerate diabetic complications.
Conclusion: A substantial increase in weight, BMI and prevalence of obesity was observed in diabetic patients in the course of time, which accompanied the improvement of glucemic control.

Abstract #281

HYPERGLYCEMIA INDUCED SEIZURES- AN ENDOCRINOLOGICAL SOLUTION TO A NEUROLOGICAL PRESENTATION

Niharika Singh, MD, Tahira Yasmeen, MD, Farah Hasan, MD

Objective: To present a case of new onset seizures due to hyperglycemia and discuss its management.
Case Presentation: A 55 year old female with history of hypertension was brought to the hospital for lethargy and an episode of aphasia. It was also noted that she had increased thirst and frequent urination for the last two weeks. Her blood sugar was found to be 458mg/dl with normal bicarbonate level and no anion gap. She was started on an insulin drip and her mental status improved. She was subsequently switched to a basal bolus insulin regimen. Within a few hours of this she developed two episodes of tonic clonic seizures. She was afebrile and her blood pressure was 130/90mm of Hg. Her blood sugar was now found to be 443mg/dl, bicarbonate was 16mmol/L and anion gap was 26mmol/L. Her electrolytes were within normal limits except corrected calcium level was 10.9mg/dl. Her renal and liver functions were normal except for a mildly elevated alkaline phosphatase of 138unit/L. Her HbA1c was found to be 14.8 and she did not have islet cell Ab IgG or Glutamic acid decarboxylase Ab. Brain MRI and EEG were both normal. There was no history of illicit drug use or personal or family history of seizures. She did not have any signs or symptoms suggestive of CNS infection. The patient's only medication was hydrochlorothiazide. She was given lorazepam and phenytoin for her seizures and started on an insulin drip and intravenous fluids for the treatment of Diabetic Ketoacidosis (DKA). Her hydrochlorothiazide was discontinued due to hypercalcemia, however, hypercalcemia was not considered the cause of her seizures. With the normalization of blood sugars the patient's mental status improved and she had no more episodes of seizures. She was later discharged on a basal bolus insulin regimen.
Discussion: Seizure(s) can be the presenting symptom of previously undiagnosed diabetes. It usually occurs in nonketotic hyperglycemia (NKH) but can occur in the presence of ketosis also. The majority of these patients are found to have type 2 diabetes. The most common seizure type described with this condition is partial seizure or partial seizure with secondary generalization. Brain imaging and EEG findings in between the seizure episodes are usually normal. It is important to recognize this entity as these seizures are refractory to anticonvulsant therapy but resolve once the hyperglycemia is controlled.
Conclusion: Seizures can be the presenting symptom of undiagnosed diabetes in adults. The mainstay of treatment of hyperglycemia induced seizures is normalization of blood glucose with insulin and rehydration whereas use of anticonvulsants is usually ineffective and unnecessary.

Abstract #283

TO EVALUATE AND COMPARE THE OUTCOME OF PREGNANCIES IN WOMEN WITH TYPE 1 DIABETES TREATED WITH CONTINUOUS SUBCUTANEOUS INSULIN PUMP OR MULTIPLE INSULIN INJECTIONS

Banshi Saboo, MD, Shashank Joshi, MD, FACP, FRCP, Hardik Chandarana, Smita Shah, Asha Shah

Results: The study was aimed to compare the outcome of pregnancies in women with type 1 diabetes treated with continuous subcutaneous insulin infusion pump (CSII) or multiple insulin injections (MDI). Total 14 patients were treated with insulin pump and 20 patients were treated with multiple injections; were mainly investigated for HbA1c, incidence of hypoglycemia, fetal outcome, rates of pregnancy induced hypertension and cesarean section. HbA1c with insulin pump was significantly better from that obtained with multiple injections. Hypoglycemic events were significantly less in CSII group as compared to MDI group. Moreover, severe hypoglycemia was not seen in CSII group, whereas there were numerous episodes of severe hypoglycemia in MDI group, few of them required hospitalization. Rate of early or mid pregnancy abortion was 10% treated with multiple insulin injections, while no abortion was seen in CSII group. Fetal prognosis was also better in pump treated patients, macrosomia was seen in 4 new borns in pump group whereas 12 in MDI group. The occurrence of pregnancy induced hypertension was similar in both groups. The rate of cesarean section was not influenced by therapeutic device, similar in both groups

Abstract #284

KNOWLEDGE BASED ACTION FOR DIABETES AWARENESS MOVEMENT

Banshi Saboo, MD, Shashank Joshi, MD, FACP, FRCP, Asha Shah, Hardik Chandarana

Results: The prevalence of type 2 diabetes mellitus (T2DM) in India is among the highest in the world. Rapid rise in prevalence of T2DM is a major concern of public health sector in India. Studies have confirmed that T2DM can be prevented or delayed by modifications in lifestyle. In India a low-cost, sustainable program is required which can raise mass awareness for prevention of T2DM. Raising awareness at a mass level or in whole society can transform lifestyle of all individuals, and it is indeed a very low cost, effective and sustainable model for prevention of such life style disorders. Therefore we propose a public awareness campaign KADAM (Knowledge based Action for Diabetes Awareness Movement) in Ahmedabad - 7th largest city of India, then same model can be replicated in different part of country. KADAM is aimed to develop awareness program about prevention of diabetes and its complications through various tools of mass communication. This project's innovations include training of low-cost lay interventionists, development and evaluation of culturally appropriate nutrition education and mass education programs for lay men. In addition, we will screen high-risk individuals and their lifestyle patterns, and will provide them appropriate lifestyle interventions. Through the screening we will be able to figure out incidence of T2DM, pre-diabetes and high risk individuals by gender, age, type and etiology. KADAM, in turn, will be helpful to devise appropriate therapeutic interventions and preventive strategies for people having diabetes or are at a high risk of diabetes. Further that intervention can be used by others in developing countries of Asia for prevention of diabetes.

Abstract #282

VALIDATION OF DIGITAL BLOOD PRESSURE DEVICES AGAINST MANUAL

Kamal Naser, MBBS, MD, MRCP

Objective: To determine the accuracy of different types of automated blood pressure devices by comparing with that of mercury sphygmomanometer readings.
Methods: Two hundred patients were studied at a Diabetes clinic in Sri Lanka. Five digital devices were tested comparing with that of mercury sphygmomanometer (40 patients for each digital device). Blood pressure and pulse rate (three measurements, averaged) were recorded in each patient with mercury sphygmomanometer and one of the study digital sphygmomanometers. Precision of measurement for mercury sphygmomanometer was 2mm Hg and for digital device was 1mm Hg. Data were descriptively analyzed by using Kappa measurement of agreement method and paired t-test.
Results: Median age in each group varied between 37 and 48 and most were females. Compared to sphygmomanometer readings, SBP readings were higher in all digital devices and the median pulse rate readings were also showing the same results. Kappa measure of agreement between digital devices and mercury device readings were calculated. In device A both SBP and DBP showed Kappa<0.3 but findings are not significant. The pulse rate showed Kappa >0.3 with a significant fair agreement with manual counting. In device B pulse rate showed very good agreement (Kappa-0.8, P<0.01), while Blood Pressure showed poor agreement between devices (P 0.085). Device C and E showed considerable agreement between the devices (P<0.01). The device D showed moderate to good agreement between the devices (P<0.01)) except for SBP. A paired-samples t-test was conducted to evaluate the mean differences between each of digital measure of blood pressure and pulse rates with a mercury sphygmomanometer. Significant mean differences were found in BP and pulse between electronic devices and manual readings in all five groups (device A to device E) except pulse rate on device B (p=0.053), SBP on device D (p=0.45) and DBP in device E (p=0.89). The mean BP and pulse rates were higher in digital devices except diastolic BP in device D and device D.
Discussion: Blood pressure is an important determinant of cardiovascular outcome in diabetes. In order to ensure that these new devices provide accurate data, they should be calibrated and validated periodically. Calibration ensures that measurements start from zero on all occasions.
Conclusion: Our finding revealed that "device A" not agreeing with sphygmomanometer reading (Kappa <0.3), "device E" showing good agreement and the rest of the devices showing agreement to some extent. There were significant differences in mean blood pressure and pulse rate between the readings of digital devices and manual sphygmomanometer.

Abstract #285

SWEET RASH: AN UNCOMMON PRESENTATION OF A COMMON DISEASE

Gavin Jackson, MD, Michael Lee, MD

Case Presentation: A fifteen year-old girl presented with necrobiosis lipoidica diabeticorum as her initial symptom of type one diabetes mellitus. She presented to her primary care physician with a six month history of bilateral anterior shin bruising. Routine labs revealed a non-fasting blood glucose of 385. She was subsequently referred to endocrine clinic where additional history revealed polydipsia, polyuria and nocturia, as well as a 30 pound weight loss over the previous twelve months. Further workup revealed positive glutamic acid decarboxylase-65 antibodies and a negative workup for type 3 Maturity Onset Diabetes of the Young. She was started on multiple daily injection treatment with long-acting and short-acting insulins. Despite improvement of her dysglycemia, the necrobiosis lipoidica lesions on the anterior shins persisted.
Discussion: Necrobiosis lipoidica diabeticorum is an unusual dermatologic manifestation associated, most commonly, with diabetes mellitus. It is seen in less than one percent of diabetics, but over 75 percent of cases occur in patients with diabetes, impaired glucose tolerance, or a family history of diabetes. The lesions tend to occur in patients with more substantial hyperglycemia, and are associated with an increased risk of retinopathy and nephropathy. Treatment involves correction of hyperglycemia, however specific treatment for the skin lesions have limited effecacy. Some of the treatments that have been used include topical and intra-lesional steroids, topical tacrolimus and anti-malarial drugs.
Conclusion: Our patient presented with necrobiosis lipoidica- which is particularly unusual as the presenting symptom of diabetes. In the case of our patient, the skin lesions led her to seek medical attention and hastened her diagnosis. This clearly decreases her risk of developing both short-term and long-term complications of uncontrolled diabetes. Recognition of these characteristic lesions and their association with diabetes mellitus is important for endocrinologists and primary care physicians alike.

Abstract #286

NEW-ONSET DIABETES MELLITUS IN A NIGERIAN MALE ON SALVAGE THERAPY FOR HIV INFECTION - A CASE REPORT

Andrew Uloko, MD, Musa Babashani, Ibrahim Gezawa, Fabian Puepet, MBBCh, FMCP, Rifkatu Mshelia, MD, Kabiru Sada

Objective: We report a case of diabetes mellitus in a Nigerian HIV-infected male on salvage therapy with a view to drawing the attention of clinicians to metabolic problems in HIV patients on Highly Active Anti-retroviral Therapy (HAART).
Methods: The case records of an HIV-infected 39-year old Nigerian male on HAART who developed diabetes mellitus was reviewed. Review of relevant literature was undertaken.
Case Presentation: A 39 year old Nigerian male with HIV-1 infection since 2006 was recently commenced on salvage therapy after clinical and virological failure on first and second line antiretrovirals (ARTs). Genotypic and phenotypic resistance testings by Polymerase Chain Reaction and direct bidirectional DNA sequencing confirmed HIV-1 group A, subtype G with Reverse Transcriptase gene resistance-associated mutations. He was subsequently commenced on salvage HAART consisting of DVR/r, Raltegravir and Tenofovir/ Emtricitabine (Truvada) with a good virology response (undetectable Viral Load after 3months of salvage HAART) and steady clinical improvement. However, by the 4th month he developed symptoms of diabetes mellitus (DM) with fasting plasma glucose (FPG) of 14 mmol/L and glycated haemoglobin (HbA1c) 7.6%; was commenced on lifestyle measures and metformin. His lipid profile, liver and renal function tests were normal. The glycaemic control became optimal three months later (FPG 5.6mmol/L, HbA1c 6.2%).
Discussion: The introduction of HAART has remarkably improved the morbidity and mortality of HIV-infected patients at the cost of increased risk of abnormalities of lipid and glucose metabolism. These complications may increase the patients' risk of cardiovascular disease, a rare occurrence prior to the era of HAART. Insulin resistance is implicated in the pathogenesis of lipodystrophy, dyslipidemia, hyperinsulinemia, and hyperglycemia. In a recent study from Jos, North-Central Nigeria, the prevalence rates of Impaired Fasting Glucose (IFG) (27.1%) and Diabetes Mellitus (11.2%) in HAART-treated patients were significantly higher than those in HAART-naïve patients (IFG 5.0%, DM 6.0%), $p<0.005$. Salvage therapy in HIV involves the use of ARTs after failure of first and second line therapy. The repeated use of ARTs

and later introduction of salvage therapy in this patient may account for the development of DM.
Conclusion: New onset DM can occur in HIV-infected persons on HAARTs (especially 2nd line ARVs). High index of suspicion and screening for diabetes is advised in HIV-infected persons receiving second line antiretroviral agents.

Abstract #287

IMPROVED PATIENT-REPORTED OUTCOMES WITH INSULIN DEGLUDEC 200 U/ML (IDEG U200) VERSUS INSULIN GLARGINE IN INSULIN-NAÏVE PEOPLE WITH TYPE 2 DIABETES

Richard Bergenstal, MD, Anuj Bhargava, Rajeev Jain, MD, Jeff Unger, MD, Torsten Christensen, Henriette Mersebach, Stephen Gough

Objective: Health status is a vital measure of overall treatment efficacy, satisfaction and adherence in people with diabetes. Insulin degludec, a new-generation basal insulin with an ultra-long action profile >24 hours, has the potential to improve patient outcomes. IDeg U200 contains equal units of insulin in half the volume compared with the 100U/mL formulation, and permits the delivery of larger insulin doses (up to 160U) in a smaller volume administered in a single injection with a prefilled pen device. This 26-week, open-label, randomized trial investigated the health status of insulin-naïve participants with type 2 diabetes (T2D) given IDeg U200 or 100U/mL insulin glargine (IGlar), both once daily.
Methods: Patient health status was assessed using the validated Short-Form 36 Health Survey, version 2 (SF-36,v2) questionnaire which has two summary measures: mental and physical well-being, each with 4 scales totalling 8 domains. Change in score from baseline was analyzed using ANOVA, with treatment, antidiabetes therapy at screening, sex and region as fixed factors, and age and relevant baseline values as covariates. Data were compiled from the intent-to-treat population, and missing data were replaced by last observation carried forward. While open-label designs can impose bias in patient reported outcomes, the impact here is likely negligible in an insulin-naive study population.
Results: At baseline, patients (IDeg, n=228; IGlar, n=229) had a mean age of 57.5 yrs; HbA1c 8.3%; FPG 173.2 mg/dL; and BMI 32.4 kg/m^2. Mean observed physical SF-36 scores improved by 1.3 with IDeg and by 1.2 with IGlar, while mean observed mental scores improved by 1.7 with IDeg and by 0.3 with IGlar. After 26 weeks, 2 of 8 domains in the SF-36 questionnaire significantly favored IDeg U200, including less bodily pain (ETD: 1.6[95% CI: 0.1; 3.2], p=0.04) and improved vitality (ETD: 1.5[95% CI: 0.1; 3.0], p=0.04). Five of the 6 remaining domains were numerically in favor of IDeg U200, albeit not significantly. IDeg effectively improved glycemic control (HbA1c and FPG) and the rates of overall confirmed hypoglycemia (PG <56 mg/dL or requiring assistance) and nocturnal confirmed (occurring between 00:01 and 05:59) were numerically lower with IDeg U200 (14% and 36% lower, respectively) compared to IGlar.
Discussion: Using the SF-36 questionnaire, IDeg U200 demonstrates significantly greater improvements in bodily pain and overall vitality vs IGlar, both important measures in determining the health status and treatment efficacy of patients with diabetes.
Conclusion: The lower injection volume (200 U/mL) may explain the observed improved outcomes with IDeg U200.

Abstract #288

RESULTS OF PERCUTANEOUS CORONARY ANGIOPLASTLY WITH STENT PLACEMENT IN DIABETIC PATIENTS WITH CORONARY ARTERY DISEASE

Jose Jimenez-Montero, MD, Patricia Monge-Ortega, Jorge Arauz-Chavarria

Objective: This is a descriptive, retrospective, observational study aimed to evaluate the clinical characteristics and outcomes of diabetic patients who had re-vascularization procedures.
Methods: We reviewed medical records of 268 patients with coronary heart disease admitted at Hospital San Juan de Dios from January to December 2009. Eligible patients were those who had diabetes, coronary artery disease, and a complete medical record and had percutaneous coronary angioplasty (PTCA).
Case Presentation: Sixty-two males and 26 females, (age 62.8±9.4 years old), with coronary artery disease (CAD) who underwent PTCA with stent placement (89% medicated) were included. Major coronary risk factors were: smoking (49,8%). hypertension (87,5%), and dyslipidemia (81.7%); LDL cholesterol ≤ 70 mg/dl was seen in 18.3% of the population, low HDL cholesterol (≤ 50 mg/dl) in 82 % of females and low HDL cholesterol (≤ 40 mg/dl) in 80 % of males. Fasting glucose > 130 mg/dl and glycosylated haemoglobin A1c (HbA1c), ≤ 7.0% were observed in 62% and 32% of the patients, respectively. On average there were 1,4 ± 0,6 vessels treated, and 1,7 ± 1,0 stents per patient. PTCA was successful in 96.6% of the patients. Complications related to PTCA occurred in 6.8%, representing a 26 % major cardiovascular adverse event after the PTCA and 39.1% required a new re-vascularization. Restenosis was observed in 4.5% of the patients. Two patients died during the first month due

to cardiogenic shock and a third patient died 12 months latter. Before PTCA aspirin and clopidogrel were used in 71.8% of the patients, 45 % were on statins and 42% received beta-blockers. Insulin and oral agents were used in 37% and 36 % of the patients before PTCA.

Discussion: Poor diabetic control and dyslipidemia were present our CAD patients. Previous studies showed HbA1c levels ≤ 7.0 % in 50 % in diabetic patients attended in primary clinical settings in Costa Rica; our patients had a more complicated clinical condition with hyperglycaemia and dyslipidemia and only 45 % were on statins before PTCA. After PTCA, thrombotic complications were similar to other series, but mortality and new re-vascularization procedures were slightly higher compared to other studies.

Conclusion: This is an observational study, which illustrates the clinical and re-vascularization management of diabetic patients with CAD in a general hospital in Costa Rica. Despite the limitations of this report, the results of the PTCA were similar to other studies and allowed us to improve the clinical management of these patients.

Abstract #289

PATTERNS OF DIABETES THERAPY AND RATES OF GLUCOSE CONTROL IN SAUDI ARABIA

Mohsen Eledrisi, MD, Buthina Alhaj, Mahmoud Mustafa, Daad Akbar, Shadia Matboli

Objective: Diabetes Mellitus is a common disease in Saudi Arabia with a prevalence of about 24 %. Data on the patterns of diabetes therapy and their relation to rates of glucose control among patients with type 2 diabetes in Saudi Arabia are limited. We aimed at examining the patterns of use of different medications for diabetes and their associated rates of glucose control.

Methods: We conducted a cross-sectional study at multicenter sites in the Eastern and Western provinces of Saudi Arabia. Patients aged 18 years and older with type 2 diabetes attending different outpatient clinics with a minimum follow up of 6 months were evaluated. Types of diabetes therapy-oral hypoglycemic drugs or insulin- and associated glycemic control assessed by levels of HbA1c were examined.

Results: A total of 1,107 patients were evaluated with a mean age of 52.6 ± 11 years, mean duration of diabetes since diagnosis 8.5 ± 7 years and mean HbA1c 8.2 ± 1.8 %. Therapy consisted of diet alone in 3 %, oral hypoglycemic drugs in 55.6 %, and insulin in 41.4 % (including 26 % on both insulin and oral agents) of patients. Acceptable glucose control (defined as HbA1c < 7 %) was observed in 33.3 % of patients on oral therapy and 10.2 % of patients on insulin, while poor glucose control (defined as HbA1c > 9.5 %) was found in 19.6 % of patients on oral therapy and 40.5 % of patients on insulin. Of all patients, adequate glucose control was observed in only 21 % (95 % confidence interval 21.1-27.8) of patients, while 24.5 % (95% CI 25.2-31.9) had poor glycemic control.

Discussion: We found that about two-third of patients on oral hypoglycemic agents had uncontrolled glucose (HbA1c levels > 7 %) and about one fifth (19.6 %) had poor glycemic control (HbA1c levels > 9.5 %).This indicates that a significant number of these patients require insulin; this delay in starting insulin deserves further study and may be related to physician and/or patient factors. For patients on insulin therapy only 10.2 % achieved acceptable glucose control; this may be related to both decreased patient's compliance with insulin as well as inadequate insulin doses.

Conclusion: A large percentage of Saudi patients with type 2 diabetes have uncontrolled glucose; this is marked by failure to prescribe insulin for many patients on oral drugs and the use of suboptimal doses for those on insulin. Public health, clinical and research efforts are needed to improve diabetes care at the patient, physician and health care system levels.

Abstract #290

TYPE2 DIABETES PRESENTING AS RECURRENT SEIZURES IN A NIGERIAN MALE

Ekenechukwu Young, MBBS, FWACP, Christian Okafor, Esther Ofoegbu

Objective: To report a case of previously-undiagnosed type2 diabetes mellitus presenting as recurrent seizures in a Nigerian male.

Case Presentation: : A 50 year old Nigerian male was admitted in the emergency with a history of recurrent generalised tonic-clonic seizures with focal onset from the right arm for two days. This was preceded by numbness and paraesthesiae of the right fingers. There was residual weakness of the right upper and lower limbs. The seizures gradually increased in frequency from about three times on the first day till he lapsed into status by the second day with loss of consciousness. There was no fever, headache, neck stiffness, vomiting, photophobia, drug or alcohol ingestion. He had no prior history of hypertension or DM or epiliepsy, no history of polyuria or polydipsia. Examination revealed an obese middle-aged man, (BMI 31.5kg/m2, WC 112cm), dehydrated, had tachycardia of 118/min, BP 140/80mmHg with a slightly displaced apex beat. No cranial nerve deficits were observed and he had Grade 4 power in the right upper and lower limbs with a slight reduction in tone. RBS was 468mg/dl. There was no ketonuria. Na 140meq/l, K 3.4, HCO3 26, Urea 4.2mmol/l, Creatinine 76µmol/l, Ca2 2.8mmol/l, PO4

1.1mmol/l. Serum osmolarity was 310.2mosm/kg, anion gap 10. Brain CT scan did not reveal any abnormalities. CBC was normal with Hb 14.7g/dl. Lipid profile showed dyslipidaemia with elevated total cholesterol 6.1mmol/l, low HDL 0.7mmol/l and high LDL 4.3 mmol/l. A diagnosis of HHS with status epilepticus was made and he was managed in the intensive care unit with IV Phenytoin , Oxygen and HHS protocol with adequate rehydration, insulin and potassium. Sodium valproate was added for further seizure control. By the fifth day, blood glucose control was stable, he had regained consciousness and seizures had abated. He was ambulant with only very slight residual weakness in the right lower limb. He was subsequently counselled by the diabetes educator and the dietician before discharge on pre-mixed (70/30) insulin.

Discussion: This is a rare case of first time presentation of diabetes with status epilepticus as a major feature masking a hyperglycaemic emergency. Similar reported cases include cases of recurrent partial seizures (epilepsia partialis continua). Hyperglycaemia and electrolyte imbalance are risk factors for seizures and should be immediately sought for in a patient presenting with seizures. Correction of the blood glucose will lead to seizure cessation, and the seizures are usually refractory to anti-epileptic drugs. Stroke and other focal neurological problems need to be excluded as in this case.

Conclusion: Severe hyperglycaemia can result in metabolic derangements leading to seizures.

Abstract #291

PROFILE OF NEW-ONSET TYPE2 DIABETES MELLITUS IN THE ELDERLY

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Arthur Onwuchekwa, Sunday Chinenye, MBBS, FWACP

Objective: :This was a cross-sectional study carried out in elderly patients developing diabetes for the first time. The aim of the study was to assess their common pattern of presentation and their clinical and biochemical characteristics.

Methods: All newly-diagnosed elderly (≥ 65 years) patients with type2 diabetes presenting at a tertiary facility in Port Harcourt Nigeria over a period of 3 months were assessed with interviewer-administered questionnaires. Their BP, BMI, and biochemical parameters including FPG, lipid profile and creatinine was measured. Nephropathy was assessed with the MDRD equation. Monofilament testing for neuropathy and a fundoscopy was also done.

Results: There were 19 males and 33 females with a mean age of 70.5±4.9years. Their presenting symptoms ranged from asymptomatic; being diagnosed while being investigated for other illness (46.2%),symptoms of DM (27%), visual symptoms (9.6%), and symptoms of complications such as paraesthesiae, leg oedema and stroke (17.2%). Only 15.4% of the patients carried out regular exercise at least two times a week. History of DM in a first degree relative was present in 57.7%. The mean BMI was 26.7+3.8kg/m2, with 42.3% overweight and 21.2% obese. Hypertension was present in 51.9% of the patients. The mean FPG was 8.3 ±3.9mmol/l. Good glycaemic control (FPG < 7.2mmol/l) was present in 42.3%.The mean total cholesterol was 190±34.9mg/dl. A total cholesterol level greater than 200mg/dl was present in 38.5% of the patients. The mean triglyceride was 106.8±35.6mg/dl and 14.9% had levels ≥ 150mg/dl, while the mean LDL was 128.1+34.5mg/dl with 55.8% having LDL ≥ 115mg/dl. Only 29.8% had HDL ≥ 45mg/dl. Complications were present in 30.8% of the patients of which 17.3% had neuropathy, 5.8% had eye complications and abnormal GFR was present in 77.5%.

Discussion: The high percentage of patients who were diagnosed in the course of investigation for other illnesses underscores the need for screening for diabetes in the elderly. This is because old age is a risk factor both for diabetes and cardiovascular disease. Hypertension was also common as expected as BP also rises with age with it being an additional risk factor for cardiovascular disease in them. Their overall poor glycaemic control may be due to the fact that their treatment was still being optimised, though the need to avoid hypoglycaemia may have led to the reluctance of their physicians to adopt aggressive measures to lower their blood glucose levels.

Conclusion: : Diabetes in the elderly is associated with a high prevalence of cardiovascular risk factors and complications and is frequently detected in the course of other illnesses.

Abstract #292

A NURSE-DIRECTED COMPUTER PROGRAM, WHICH RE-ADJUSTS SUBCUTANEOUS MULTIPLE DAILY INJECTIONS (MDI) OF INSULIN, LOWERS THE MEAN BG IN HOSPITAL PATIENTS BY 93 (MG/DL)

Paul Davidson, MD, Bruce Bode, MD, John Clarke, Harry Hebblewhite

Objective: To examine the performance of a computerized algorithm for adjusting subcutaneous MDI insulin regimens in hospital patients.

Methods: This algorithm determines a meal bolus by adjusting the dose given for the same interval of the previous day. Example: Breakfast Bolus = [Yesterday's Breakfast Bolus]*AF, where AF is an Adjustment Factor, that is governed by [Yesterday's pre-Lunch BG]. The

Basal dose is determined by an adjustment to yesterday's bedtime Basal dose, and is governed by the BG's from pre-Breakfast and MidSleep (3:00AM) earlier today. Adjustment for insulin dosing was made possible by the interfacing of the nurse (program-user) and the lab data system. The ADA-published correlation between A1c and BG was used to convert the numbers in both directions, as shown below.

Results: There were 30 patients in the Treatment Group. The average pre-study A1c was 9.7%, and its calculated equivalent BG (eAG) was 232mg/dl; these decreased to the final day's average BG of 139mg/dl and its calculated equivalent A1c of 6.5%. The change in the Treatment Group's Mean BG (final BG - initial eAG) was -93mg/dl; P<0.007. The equivalent change in A1c% was -3.2 percentage points; P<0.007. There were 31 patients in the Control Group. The average pre-study A1c was 8.5% and its calculated equivalent BG (eAG) was 198mg/dl; these decreased to the final day's average BG of 158mg/dl and its calculated equivalent A1c of 7.1%. The change in the Control Group's Mean BG was -40mg/dl The percent of all BG's < 50 was 0.3% for the Treatment Group and 0.5% for the Control Group. The percent of all BG's < 40 was 0.0% for the Treatment Group and 0.3% for the Control Group.

Discussion: The drop in the Mean BG of the Treatment Group was significantly greater than that of the Control Group; P<0.03

Conclusion: We suggest that this algorithm may be a valuable tool for adjusting subcutaneous MDI regimens. The algorithm is currently being incorporated into a server-based enterprise platform for continuous on-going dosing guidance.

Abstract #293

ANTI GAD ANTIBODY IS ASSOCIATED WITH IMPAIRED BRACHIAL ARTERY FLOW-MEDIATED VASODILATION IN YOUNG NONOBESE PATIENTS WITH ADULT ONSET DIABETES

Ankit Shrivastav, MD, Manojit Lodha, Jyotirmoy Pal, Satinath Mukhopadhyay, Subhankar Chowdhury

Objective: Flow-mediated dilatation (FMD), induced by occlusion of the brachial artery, is an index of endothelial-dependent vasodilatation (nitric oxide- dependent vasodilatation). It is a frequently used and relatively reliable method for detecting endothelial dysfunction. It is usually impaired early in patients with diabetes. This study was done to find if FMD was affected by the presence of Autoantibodies (Anti GAD and Anti IA2) in diabetic patient.

Methods: A total of 100 young (25-40 years) , non obese (BMI < 25) , adult onset diabetic patient were recruited for the study. Patients were rested in a temperature controlled quiet room for 30 min before scan. Scans were done using B-mode ultrasound imaging with a 10 -MHz linear array transducer. Brachial artery diameter was measured before inflating the cuff. Reactive hyperaemia was induced by inflating the cuff 50 mm of hg above the systolic blood pressure of the patient and maintained at that level for 5 minutes. Scans measuring post hyperaemia diameter were started 30 seconds before deflation and continued 60 seconds after inflation. The maximum diameter during this time was taken as the post hyperaemia diameter of the brachial artery. FMD% was expressed as the percentage change in the internal diameter of the brachial artery from the baseline diameter. HbA1c, fasting C peptide, 1 hour post meal C peptide, Anti Glutamic acid decarboxylase antibodies (GADA) and Islet Cell Autoantigen 512 Antibodies (Anti-IA2) were estimated for every patient.

Results: Flow-mediated dilatation was significantly lower in patients with positive Anti GAD antibody (11.7% ± 4.9%) than in those with negative Anti GAD antibody (14.57% ± 6.2%) (P < 0.001). However there was no significant difference in Flow-mediated dilatation in patients with positive Anti IA2 antibody (13.0% ± 4.6% vs 13.2% ± 5.8%).

Discussion: Flow-mediated dilatation of Brachial Artery is a reliable marker of endothelial dysfunction. In young non obese adult onset diabetics, presence of Anti GAD antibody was associated with decreased FMD ie more severe endothelial dysfunction in this study. This may be due to poorer glycemic control due to more severe insulinopenia in these patients. However the difference in HbA1c was not significant in the two groups. This is a preliminary report and the study is still ongoing.

Conclusion: In young, non obese patients with adult onset diabetes mellitus, presence of Anti GAD antibody is associated with increased endothelial dysfunction as shown by impaired brachial artery flow-mediated vasodilation.

Abstract #294

COMBINATION THERAPY DURING THE HOLY MONTH OF RAMADAN

MD Uddin, DEM, MD, Hajera Mahtab, MBBS, FCPS, Faruque Pathan, MBBS, MD, Zafar Latif, FCPS, Fazlarabbi Khan, MBBS

Objective: To evaluate the changes in bodyweight, glycemic control and hypoglycemic events in Muslim type 2 diabetes patients treated with Insulin Glargine

and Glimepride during fasting on Ramadan in type 2 Bangladeshi diabetes patients.

Methods: In these observational study 102 Muslim patients with type 2 diabetes who were on Insulin Glargine and Glimepride for 3.0 to 5.0 years and intended to fast during Ramadan were included with pre-designed screening process and counseling. Weight, BMI, glycemic status and hypoglycemic events were assessed before, during and after the period of Ramadan.

Results: The patient's mean age was 54.8 ± 9.4 years with average duration of diabetes 13.8 years. The mean weight and BMI were similar before and after Ramadan which was less during the month of Ramadan (68 ±10 vs. 67.3 ± 9.4 kg/m2, 26.1± 4.1 vs. 25.6 ± 4 kg/m2). Average daily dose of Glargine and Glimepride were 21.9 ± 10.3 IU and 3.1±1.5 mg which was reduced to 19.5 ±10.4 IU and 2.6±1.6 mg during Ramadan. Fasting blood glucose improved from 7.2±1.9 to 6.1±1.2 mmol/L during Ramadan. HbA1c level was also improved from 7.9±1.2 to 7±1% compared with before and after fasting in Ramadan. There was no severe hypoglycemia.

Discussion: Most of the T2 Diabetic Patients can fast during the holy month of Ramadan safely. But they need pre-Ramadan assessment and counseling for life style modification and drug adjustment to avoid complications during fasting.

Conclusion: Combination (Insulin Glargine and Glimepride) therapy is safe and effective before, during and after Ramadan.

Abstract #295

THE EFFECT OF RALOXIFEN ON RENAL FUNCTION IN POST-MENOPAUSAL WOMEN WITH DIABETIC NEPHROPATHY

Faranak Sharifi, MD, Seddigheh Abazari, Mahnaz Rahimi, Saeideh Mazloomzadeh

Objective: Experimental data suggest that activation of estrogen receptor pathway limits the progression of diabetic nephropathy. Selective estrogen receptor modulators (SERMs) may possess the optimal characteristics desirable in a drug designed for use in post-menopausal women. The aim of this study was to evaluate the effects of SERMs, raloxifen, on renal function in women with diabetic nephropathy after menopause.

Methods: Thirty seven post-menopausal women with type 2 diabetes mellitus and albuminuria included in a four-month double blind randomized placebo-controlled trial. The first group including 18 subjects received raloxifen tablets (60mg/d) and the second group (19 subjects) received placebo. Fasting plasma glucose,HbA1c, lipid profile, creatinin and urine albumin/creatinin ratio(ACR) were measured and GFRs were calculated at baseline and after the end of the study.

Results: The ACR was decreased significantly in the women on raloxifen(505 ± 115 µg/mg at the end of study Vs. 844±162 µg/mg at baseline,p: 0.007).The reduction in microalbuminuria with raloxifen was independent of other confounding factors like age, duration of diabetes or menopause, blood pressure and body mass index (BMI). No significant changes in ACR were seen in control group (607±109 µg/mg Vs. 347± 584 µg/mg, p: 0.4). Furthermore, raloxifen resulted in no significant changes in HbA1c, lipid profiles, GFR, BMI and systolic blood pressure.

Discussion: This study revealed a significant reduction in albuminuria and no change in GFR with Raloxifen in post-menopausal women with diabetic nephropathy. A reduction in albuminuria and glomerolosclerosis in diabetic rats with estradiol and also with raloxifen has been reported previously. These effects may be related to the inhibitory effect of these agents on synthesis of type I& IV of collagen and also TGF-β, angiotensin II receptor and endotelin I. Increased activation of nitric oxide synthetase and vascular endothelial growth factors may be other mechanisms of action for SERMs to improve glomerol functions.

Conclusion: Raloxifen may limit the progression of albuminuria in post-menopausal women with diabetes.

Abstract #296

ELECTRONIC DATA BASE (EDB) IMPROVES CARE OF PATIENTS WITH DIABETES BEFORE OVERT HYPERTENSION

J. Prendergast, MD, Evelyn Castillo-Profeta, Abraham Silvers

Objective: Slow insidious deterioration of small and large vessel arterial compliance (AC) occurs in patients with type 2 diabetes. This decrease is thought to be related to declining endothelial function. Being able to identify, and treat, this change before the subsequent increase in blood pressure would improve morbidity and mortality that otherwise accompanies hypertension (HTN).

Methods: We entered arterial compliance data, obtained non-invasively, for 768 sequential patients from our practice with, and without, HTN into our EDB to determine if this would improve the quality of healthcare delivery. 284 patients were normotensive controls, i.e., they did not have diabetes, 211 were normotensive type 2 patients, 129 were hypertensive and did not have diabetes and 144 here both hypertensive and had diabetes. Additional data for lipids, HgA1c, BMI, age, gender and ethnicity were noted. The majority was Caucasian, over weight and over

60 years old.
Results: Large and small vessel AC is clearly compromised in HTN patients with or without diabetes. However, small vessel AC is already reduced in diabetic patients who are not yet hypertensive.
Discussion: EDB information such as these dictates that we should be addressing AC in diabetic patients much earlier in their disease than previously thought. These data are an example of how our clinic has embraced the use of EDB to further the goals of the Hitech Act that was established to improve healthcare quality, safety and efficiency.
Conclusion: Clearly more intense follow up is needed to prevent the development of HTN in patients with diabetes and to aggressively treat HTN in those who have already developed it. AC, captured non-invasively, provide an additional tool for maximizing clinical care of patients with type 2 diabetes.

Abstract #297

EVIDENCE THAT USE OF TELECOMMUNICATION TECHNOLOGY DOES IMPROVE CARE OF DIABETIC PATIENTS

J. Prendergast, MD, Penelope Mayes, Abraham Silvers

Objective: Severely restricted availability of specialized diabetes care eventually leads to enormous strains on emergency rooms and otherwise avoidable medical costs. We adapted the Mayo Clinic Stroke Model for our practice. It is called the Promatoras-Telemedicine Care Provider interaction model and was developed to serve our expanding Hispanic diabetic patients who have more than twice the incidence of diabetes (18%) as do Caucasians (8.3%).
Methods: We gathered together a group of endocrinologists, nurses, primary care physicians and Promatoras (health promoters) to serve our Hispanic diabetes patients. The Promatoras have a long-standing history of serving this group with language translation, social customs, and financial transactions and is now helping to obtain meaningful medical care. In our model, a Promatoras becomes the primary educator and point of communication to patient or medical personnel overseeing each patient's home glucose monitoring, medical records, and medication. Telephone and email were used to communicate with patients and diabetes specialists. Between clinic visits, routine care, i.e., body weight, blood glucose and blood pressure were obtained or monitored by the Promatoras, shared over the Internet with the medical personnel and each patient was interviewed by audio and camera. 19 high-risk patients with high HgA1c entered our pilot study and 16 completed this 3.5 year evaluation.
Results: No patient experienced a diabetes related ER or hospital visit during this time. There were no vision or kidney problems. There was no deterioration in blood pressure and only a 5 pound weight increase (p=NS). The most important sign of improvement in the patient's medical condition was the significant and sustained decrease in HgA1c from 9.6% at baseline to 7.2%, a decrease of 21% (p=0.001).
Discussion: The cost savings and health benefits reflected in these outcomes should be evident. The success of the patient management style presented here relied on a social framework that has been part of the Hispanic community since early in the last century. Other at risk populations should be able to adopt this mixture of expert medical opinion, healthcare professional monitoring and local "volunteers", already invested in the well being of their community, to delay or prevent many health care problems.
Conclusion: Clinical medicine can and must adopt these and other social media tools to improve the care of patients and slow the escalating cost of healthcare.

Abstract #298

COMPARISON BETWEEN EXENATIDE AND SITAGLIPTIN IN NEW ONSET TYPE 1 DIABETES

Hari Kumar KVS, MD, DNB, P. Prusty

Objective: To study the effect of addition of incretin modulators to insulin requirement in new onset type 1 diabetes mellitus (T1DM)
Methods: Fifteen newly detected T1DM adult patients participated in this open label, randomized study for a period of one year. The patients were divided into 3 groups: Group1 (Insulin alone), Group 2 (Insulin and Exenatide), Group 3 (Insulin and Sitagliptin). The primary outcome was change in insulin requirement from onset of T1DM and secondary outcomes were preservation of c peptide secretion and risk of hypoglycemia at the end of one year observation (NCT01235819).
Results: The study participants consist of 12 males and 3 females with a mean age of 26.8 ± 3.5 yr. The decrease in insulin requirement was 16.7 ± 12.5, 39.8 ± 17.2, 21.2 ± 9.6 units in groups 1, 2 and 3 respectively at the end of one year (P=0.0431). The mean stimulated c peptide secretion was 0.34 ± 0.12, 0.45 ± 0.34, 0.44 ± 0.5 ng/mL at the end of the study period in 3 groups respectively (P=0.8656). Group 2 had the maximum percentage preservation in c peptide when compared with other groups. Two patients in group 2 and one patient in group 3 did not require insulin during last 9 months. The incidence of severe hypoglycemia was same in all the groups and none of the study participants had ketoacidosis during follow up.

Discussion: Incretin based therapies are known to have pleotropic benefits in diabetes mellitus including preservation of β-cell mass in animal studies. They have no demonstrable role in preventing the autoimmune destruction of β-cells, but may preserve β-cell integrity, longevity, proliferation and function. The data about the use of incretin modulators is plenty in type 2 diabetes but scanty in T1DM. We studied the use of exenatide and sitagliptin along with insulin in newly detected T1DM (less than 3 months from onset of the disease). Our data showed that use of the incretin modulators decreased the total insulin requirement with similar glycemic control between three groups.
Conclusion: Our preliminary data showed that the addition of exenatide at onset in patients of T1DM decreases the insulin requirement. Use of incretin modulators had no effect on endogenous insulin production and risk of hypoglycemia.

Abstract #299

AN UNUSUAL CASE OF DIABETIC AMYOTROPHY

Esti Charlap, MD, Patricia Dharapak

Objective: To report the occurrence of diabetic amyotrophy in a middle-aged man with long-standing uncontrolled type 1 DM.
Case Presentation: A 45 year old man with type 1 DM presented to the hospital with complaint of lower extremity weakness. He noted for the past year he had progressive weakness of his lower extremities to the point where he needed to hold on to walls or furniture to stand up. This was accompanied by a burning, shooting pain in his feet. He also complained of feeling dizzy and lightheaded upon standing. Review of systems was positive for erectile dysfunction and an unintentional 20-pound weight loss over a few months period. He had type 1 DM for ten years ago and was on an insulin pump. HbA1C was 19 six months ago. Finger sticks were frequently in the 400's. Blood pressure on admission was 125/76 sitting and 100/69 standing. Physical exam revealed 4/5 strength in his lower extremities, with positive leg drift bilaterally. He had decreased sensation to pin prick below his mid calves. HbA1c on admission was 12.1. CBC, electrolytes, and liver function tests were normal. ESR was mildly elevated at 15 mm/hr (normal 0-13 mm/hr). TSH, SPEP, and Vitamin B12 were normal. HIV test was negative. Rheumatologic work-up including ANA, rheumatoid factor, SSA, and SSB were negative. Tests for Lyme disease and syphilis were negative. Pelvic CT, MRI lumbar spine, and EMG were unremarkable. A trial of steroids did not improve his symptoms. A diagnosis of diabetic amyotrophy was made based on clinical signs and symptoms. He was discharged to acute rehab. One year later, he was confined to a wheelchair secondary to residual weakness, and autonomic dysfunction causing severe dizziness and orthostatic hypotension.
Discussion: Traditional features of diabetic amyotrophy include acute onset of pain followed by weakness involving the proximal leg, although distal leg weakness is not uncommon. This is associated with autonomic dysfunction and weight loss. Diagnosis is made on the basis of clinical signs and symptoms. There are no effective treatments. Some clinical improvement occurs, but complete recovery is uncommon. Although it typically occurs in older patients with a newly diagnosed type 2 DM that is under fairly good control, our patient had long standing Type 1 DM that was not well-controlled.
Conclusion: Although diabetic amyotrophy classically occurs in older patients with newly diagnosed type 2 DM that had been under fairly good control, it can occur in anyone with diabetes. This report highlights the importance of always including diabetic amyotrophy in the differential diagnosis of any diabetic patient presenting with weakness, even if they do not fit the typical profile.

Abstract #300

FIRST CASE OF SEVERE CUTANENOUS HYPERSENSITIVITY REACTION ASSOCIATED WITH LINAGLIPTIN (TRADJENTA)

Margie Banzuelo-Rio, MD, Sunil Asnani, MD, FACE

Objective: To report the first case of severe cutaneous hypersensitivity reaction associated with linagliptin (Tradjenta).
Methods: Clinical and laboratory findings of a case are presented, and the relevant literature is reviewed.
Case Presentation: A 70-year-old Caucasian male with history of type 2 diabetes for 35 years was recently started on linagliptin (Tradjenta) for uncontrolled type 2 DM. Six days after use, he developed a diffuse erythematous rash over his trunk associated with itching and myalgia. He presented to the ER 2 days later with additional complaints of high grade fever, fatigue and difficulty swallowing. Vital signs revealed fever of 101*F. Physical exam revealed diffuse painful blanching erythematous maculopapular rash that was worse on his trunk than his extremities. Ulcers were noted on lips and the oral cavity as well. Linaglpitin was discontinued and patient was started on intravenous glucocoticoids. Fever came down and rash improved with sloughing and occasional vesiculation over the next few days.
Discussion: Linagliptin (Tradjenta) is the newest member of the group of pharmaceutical agents which inhibit dipeptidyl peptidase IV (DPP-IV) enzyme resulting in

prolonged active incretin levels. We present here probably the first known case of severe cutaneous hypersensitivity reaction and possibly even Stevens- Johnson syndrome (SJS) associated with linagliptin (Tradjenta). Both sitaglipitin (Januvia) and saxaglipitin (Onglyza) have post marketing signals of serious cutaneous hypersensitivity reactions, including SJS. Because these reactions are reported voluntarily from a population of uncertain size, it is generally not possible to reliably estimate their frequency or establish a causal relationship to drug exposure. Onset of these reactions occurred within the first 3 months after initiation of the treatment, with some reports occurring after the first dose.
Conclusion: All DPP-IV inhibitors have the potential to cause severe cutaneous hypersensitivity reactions, including SJS. Clinicians and patients need to be aware of these adverse reactions to ensure timely intervention and discontinuation of the offending agent.

Abstract #301

RENAL AND BLOOD PRESSURE EFFECTS OF ALISKIREN IN DIABETIC HYPERTENSIVE PATIENTS: A REAL WORLD RETROSPECTIVE PILOT STUDY

Philip Levin, MD, James Mersey, MD, FACP, Lee Bromberger, Zachariah Koshy, Christopher Zacker

Objective: Assess real-world renal benefits of aliskiren in diabetic hypertensive patients at multiple sites.
Methods: Retrospective real-world chart view of 47 diabetic hypertensive patients treated for up to 2 years with aliskiren. Statistical analysis used was fixed effects estimates from mixed model.
Results: Measure: Time 0 (Baseline) to 2 year tx, Systolic BP: 152 baseline, 138 year 2 tx, P<.001 Diastolic BP -80 baseline, 73 year 2 tx, P<.008 Potassium: 4.3.Baseline, 4.3 year 2 tx, P<NS Creatinine: 1.2 Baseline, 1.26 year 2 tx, P<NS Log (ACR) Mc/CR: 3.9 Baseline, 3.3 year 2 tx, P<.045 Aliskiren significantly lowered SBP and DBP, Log ACR, and Log ACR micro/cre over a 2 year period. Potassium had an increase after 1 year but returned to baseline by year 2. Creatinine was not elevated significantly at year 2. Study population was 53% female, 55% white, 15% African American, median age of 62 years old, 83% type 2 and 17% type 1 diabetes. Duration of diabetes was 8 years and of hypertension 4 years. At baseline, 34% of patients were on any ACEI or combination and 78% on any ARB or combination, 64% were taking insulin with 66% taking oral anti-diabetic agents with a baseline HbA1c of 6.6.
Discussion: Aliskiren is a direct renin inhibitor affecting the RAAS system in regulation of blood pressure. Recent 6-12 month prospective controlled trials evaluated its renoprotection for diabetes patients with hypertension. This retrospective study evaluates real world data on 47 diabetic (type 1 and type 2) hypertensive patients at 4 US endocrinology/family practice centers, treated up to 2 years with addition of aliskiren to other antihypertensives. All patients took 2 or more antihypertensives prior to Aliskiren initiation with at least 6 months pretreatment data available.
Conclusion: In real world practice, Aliskiren demonstrated significant antihypertensive effects and renal benefits in diabetic patients treated up to 2 years.

Abstract #302

THE IMPACT OF A BASAL-BOLUS INSULIN REGIMEN ON THE MANAGEMENT OF HYPERGLYCEMIA IN A HOSPITALIZED POPULATION

Michael Tsoukas, MD, Kavya Chitra Mekala, M.D, Jason Lancaster, MaryBeth Hodge, MD

Objective: To analyze glycemic control in non-critically ill inpatients before and after implementation of a new basal-bolus insulin regimen order set from a correctional sliding scale order set.
Methods: An IRB approved, retrospective study was performed at our 317-bed academic medical center to compare glycemic control between 2 cohorts of patients after the introduction of a new basal-bolus insulin regimen order set. Patients with fasting blood glucose of >126 mg/dL (7.0 mmol/L) or random blood glucose level of >180 mg/dL (10.0 mmol/L) were considered eligible for inclusion. Patients were excluded if < 18 years of age, had less than 4 capillary blood glucose measurements (CBGM), admitted for < 24 hours, admitted to an ICU, found to be in DKA, administered IV insulin or parenteral nutrition. Admission type, admitting service, diabetic history, age, gender, creatinine, glycated hemoglobin, and outpatient glycemic regimens were recorded. A total of 162 patients were assessed for glycemic control over an 8-month period, (63 correctional insulin, 99 basal-bolus insulin). Primary endpoint was percentage of serial CBGM at or below goal, defined as CBGM <140 mg/dL (7.8 mmol/L).
Results: 1620 CBGM's were obtained, (1012 basal-bolus insulin, 608 correctional insulin). The percentage of patients in the basal-bolus insulin cohort achieving goal CBGM was 35.5% versus 40.7% for the correctional insulin cohort, p = 0.23. Additionally, the mean blood glucose values did not differ between cohorts, 172.9 mg/dL (basal-bolus) versus 178.9 mg/dL (correctional), Pr<f = 0.41. These findings held true even after adjusting

for age, sex, medication and renal function, where the mean CBGM was 152.1 (basal-bolus) versus 163.7 (correctional), Pr<f = 0.259.

Discussion: The basal-bolus insulin regimen is more physiological in comparison to using a correctional insulin scale alone. Efforts were taken to educate providers in the appropriate utilization of the new regimen. Despite this, results of our study suggest that the mean blood glucose control in inpatients has not significantly improved after implementing the new protocol. We suspect under-dosing of insulin and provider inertia toward titration of insulin doses as possible culprits.

Conclusion: The implementation of a basal-bolus insulin regimen in an inpatient setting did not improve patients' mean blood glucose control, as compared to correctional insulin, both in terms of mean serial CBGM and percentage of blood glucose measurements less than 140 mg/dL. More efforts need to be made in regards to management practices and physician compliance with new insulin order sets to improve overall utilization and patient outcomes.

Abstract #303

NEW DIABETIC EMERGENCY: ACUTE RHABDOMYOLYSIS COMPLICATING HYPERGLYCEMIC HYPEROSMOLAR COMA: SUCCESSFUL MANAGEMENT OF A CASE AND INSIGHT INTO PATHOGENESIS.

Cherie Lisa Vaz, MD, Ajay Chaudhuri

Objective: We describe a case of severe rhabdomyolysis (peak CK 48897u/ml & massive myoglobinuria) complicating hyperglycemic hyperosmolar coma.

Case Presentation: A 57-year-old male with type 2 diabetes mellitus, on metformin, presented with polyuria, polydipsia, generalized weakness and confusion. On admission he had GCS E2M3V1 and no signs of trauma or infection. Na 137meq/l, K 4.8meq/l, HCO3 22meq/l, Cl 93meq/l, BUN 116mg/dl, Cr 3.2mg/dl, glucose 1710mg/dl, pH 7.22, ketones +small, Mg 5.2mg/dl, P 3.3mg/dl, Ca 9.6mg/dl, TSH 1.47mcU/ml, Ethanol <5mg/dl, toxicology screen negative, osmolality 364mosm/kg. Initial Troponin 0.03ng/ml peaked at 0.12ng/ml 28 hours later. CPK initially 2379u/l, 21767u/l at 24 hours and peaked at 48897 u/l 28 hours later. CK isoenzyme 100% MM and 0% MB and BB. AST and ALT were initially normal, elevated on day 3 to 584u/ml and 160unit/ml respectively. Urine myoglobin was >10000ng/ml (Nl<1). TSH was 1.47mcU/ml(0.4-5). As he was on flexeril, serum & urine cyclobenzaprine level were checked & were negative. He received aggressive i.v. hydration with NS and Bicarb drip along with the insulin drip. Blood glucose control was achieved over 48 hours. His creatinine normalized in 28 hours. Sodium peaked the day after he presented to 166mg/dl and gradually corrected over the next 3 days. P and K remained normal and CPK trended down to 881u/l on day 7. Mental status returned to baseline by day 3.

Discussion: Hyperglycemic hyperosmolar state precipitates rhabdomyolysis which can aggravate acute renal failure. The pathogenesis of this nontraumatic rhabdomyolysis is multifactorial and includes inhibition of the Na pump by hyperosmolar state, acidosis, hypernatremia, and K deficiency, decrease in intramuscular energy supply due to insulin deficiency(1). The resultant fall in transmembrane potential and elevated intracellular calcium, activates proteases with subsequent leakage of muscle enzymes contributing to rhabdomyolysis(2). We also hypothesize that the prothrombotic state that is induced by severe hyperglycemia results in muscle tissue infarction with subsequent elevated CPK, further confirmed in our patient as 100% MM.

Conclusion: CPK is not routinely measured in hyperosmolar hyperglycemic states. We recommend routine monitoring of CPK in these patients with particular attention to cases with very high serum Na as without early recognition and treatment of severe rhabdomyolysis, patients could have potentially fatal outcomes. We also suggest that the CPK be re-measured a day after the peak blood glucose and serum osmolality given the temporal association that was demonstrated by our case.

1.Am J Nephrol 11: 447-450 2.Nephron 47: 202-204

Abstract #304

TYPE 2 DIABETES IN CHILDREN. A CASE SERIES FROM LIMA, PERU

Miguel Pinto, MD, FACE, Helard Manrique

Objective: To describe a case series of type 2 diabetes in children from two general hospitals from Lima, Peru.

Methods: We abstracted the clinical charts and describe the clinical and laboratory details at presentation.

Case Presentation: We report 27 cases of children (15 girls, mean age 14.5 years) which developed type 2 diabetes. At presentation, all patients were obese (mean BMI of 31.9), with acanthosis nigricans, 45% had family history of type 2 diabetes, and 18.5% presenting with diabetic ketoacidosis. The initial mean glucose level was 376 mg/dl (SD 159), HbA1c was 11.5% (SD 3), and C-peptide was 2.7 ng/dL (SD 1.8). Further work-up, showed that anti-GAD antibodies were negative (85% of the children) or inconclusive (15% of the children). The initial treatment included metformin (22% as monotherapy, 18.5% in combination with sulphonylurea, and 29.5% in combination with insulin), insulin alone (14.8%), and diet alone (11.1%). Intensive life style change

was started at the Diabetes Clinics of both hospitals. In five cases, a follow-up of 3-12 months showed that the mean HbA1c was 6.2% (SD 1.28), and 60% were treated with metformin, one child with glibenclamide, and one child with metformin plus NPH insulin.

Discussion: Type 2 diabetes has traditionally been viewed as a disorder of adults. However, as the prevalence of obesity in youth is increasing, type 2 diabetes is now occurring in children and adolescents. Currently, Peru is passing through its epidemiological transition, were infectious disease are coexisting with chronic diseases like obesity, diabetes, and cardiovascular diseases. In the long-term, these children have higher risk of developing complications at early ages.

Conclusion: The growing number of obese children and adolescents is related with early glucose homeostasis dysregulation and type 2 diabetes.

Abstract #305

VASCULAR REACTIVITY IMPROVES IN PRE-DIABETES PATIENTS, POST AEROBIC EXERCISE

Sabyasachi Sen, MD, MRCP, FACP, Anne Lagoy, Ashequl Islam, Sarah Witkowski

Objective: Literature confirms presence of endothelial cell dysfunction and poor vascular reactivity in diabetes compared to non-diabetes state. However there is lack of data on endothelial function in Pre-diabetes (defined as per Diabetes Care, Clinical Guideline, Jan'11). We decided to investigate degree of vascular reactivity in Prediabetes patient at baseline and whether weight neutral aerobic exercise impacts the reactivity.

Methods: We selected exercise-naive patients, 45-65 yrs, BMI 25-34.9, n=10 in each group and studied their endothelial function after 150min/week of aerobic exercise (similar to Diabetes Prevention Program) and after non exercise phase (6wks phase each), in a cross over design with a 4 week wash-out period between the 2 groups. Exercise was monitored using frequent phone calls and recorded by Accelerometers. For endothelial function assessment we looked at vascular reactivity, ie. flow mediated dilatation (FMD of brachial artery) and tried to co-relate with endothelial bio-inflammatory markers and BP, fasting lipids, insulin, glucose.

Results: FMD studies (undertaken by 3 observers, blinded to glycemic status) showed, mean FMD in non-exercise group of 5.7+0.6% and post exercise it improved to 11.2+0.9%. There was no weight loss noted between 2 groups, however, there was statistically significant reduction in leptin, IL-6, hs-CRP, TNFα, fasting Triglyceride, LDL and ApoB levels. Insulin sensitivity (HOMA) and Apo-A1 improved in the exercise group, though fasting glucose levels, HbA1C, HDL levels did not change.

Discussion: Prediabetic patients have poor vascular reactivity, similar to patients with diabetes. In Prediabetes patients, FMD measurements post exercise indicate reversal of poor vascular reactivity and along with biochemical inflammatory markers can serve as robust method for monitoring vascular reactivity and endothelial cell dysfunction in a "at risk of developing diabetes" population.

Conclusion: Pre-diabetic state is associated with poor vascular reactivity but it also may be the period when intervention such as regular aerobic exercise improves it almost close to that of a non-diabetic patient, inspite of no statistically significant weight loss, post exercise phase.

Abstract #306

AMBULATORY BLOOD PRESSURE MONITORING (ABPM) AND VASCULAR STIFFNESS IN LEAN, OBESE, AND DIABETIC MINORITY YOUTH

Natia Potter, MD, Rachna Walia, Arlene Mercado, MD, Nathaniel Winer

Objective: Our aim was to determine if children and adolescents who have obesity (OB) or type 2 diabetes (DM) of relatively short duration (mean 23 months) have impaired cardiovascular function compared with lean subjects (LN), using surrogate measures of evaluation.

Methods: We enrolled 100 African-Caribbean subjects (45 male/55 female), mean ages 14.6 to 15 years (range 11.8-18.5 years) and Tanner stage 4.2-4.9. Mean BMI for DM (n=39), OB (n=40), and LN (n=21) groups was 34.2, 40.3, and 20.8 m/kg2, respectively (P<.0001, DM and OB vs. LN). Mean hemoglobin A1c in LN and OB was 5.2% and 5.3%, compared to 8.3% in DM (P<.0001, DM vs. LN and OB).

Results: Blood pressure (BP) was recorded at 20-minute intervals over 24-hours (Spacelabs 70207). Mean 24 hour, daytime, and nighttime systolic BP was significantly higher in DM and OB compared to LN subjects (Mean: 120 and 117 vs.109 mm Hg; Daytime: 123 and 121 vs. 113 mm Hg; and Nighttime: 115 and 109 vs.101 mm Hg- P<.01 for all time periods. The nocturnal systolic dip in DM and OB did not differ from that of LN, whereas nocturnal diastolic BP decreased significantly in DM and OB, compared to LN (10.4 and 11.5 vs. 20.6 mm Hg-P<.01). Mean pulse pressure was significantly increased in DM and OB groups compared to LN subjects (54 and 51 vs. 45 mm Hg P<.01). Large artery stiffness did not differ significantly between groups (HDI Pulse wave CR-2000).

Small vessel elasticity was significantly greater in DM and OB than in LN subjects (10.4 and 10.9 vs. 7.8 mL/mm Hg x100 (P<.05, DM and OB vs. LN). Weight and BMI were strongly correlated with small artery elasticity (P<.0001 for both).

Discussion: Young persons with early type 2 DM and non-DM OB have increased 24 hour mean systolic BP, absence of the normal nocturnal systolic BP dip, widened pulse pressure, and greater small vessel elasticity, compared with LN controls matched for age and sexual maturation. The strong correlation of body size and small vessel elasticity in DM and OB youth may reflect higher circulating insulin and/or leptin levels leading to smooth muscle vasodilatation, which may later be reversed by factors which promote atherosclerosis, such as aging, hypertension, and dyslipidemia.

Conclusion: Adolescent DM and OB groups share adverse cardiovascular risk factors, which may be harbingers of adult cardiovascular events.

Abstract #307

A MULTIDISCIPLINARY LIFESTYLE INTERVENTION PRODUCES MARKED WEIGHT LOSS IN OBESE PATIENTS WITH TYPE 2 DIABETES MELLITUS

Jaime Almandoz, MB BCh, Lisa Howell, Karen Grothe, Brian Irving, Ekta Singh, MD, Robert Nelson, John Miles, MD

Objective: Overweight and obesity affect the majority of American adults and greatly increase the risk of type 2 diabetes mellitus (T2DM). Moreover, pharmacological therapy for T2DM often has the undesirable consequence of promoting weight gain. Weight loss (WL) in people with T2D tends to be more difficult to achieve and of lesser magnitude than in non-diabetic individuals. However, successful WL in T2DM improves glycemic control and is associated with reduced mortality.

Methods: We enrolled 13 obese subjects with T2DM on oral agents (9 men, 4 women, age 52±2 y, BMI 33±1 kg/m2, HbA1c 7.9±0.3%) in a 5 month multidisciplinary WL intervention. BodyMedia FIT activity monitors were worn continuously to provide data on energy expenditure, which combined with online calorie intake tracking helped subjects aim for and achieve a negative energy balance of 500-1000 kCal/d. Calorie deficit targets were encouraged more than specific expenditure or intake goals at fortnightly meetings with a study physician. Education regarding food choices was reinforced in 8 meetings with a study dietician. Behavioral modification and group support dynamic was provided weekly via a 3 month cognitive behavioral therapy program led by study psychologists. Body composition was determined at baseline and at 5 months with dual energy X-ray absorptiometry and single-slice (L2-L3) CT scans.

Results: This intervention resulted in WL of 13±2% (range 6-26%), and reductions in total fat mass and visceral fat area of 28±4% and 37±5%, respectively (both P<0.001). HbA1c decreased to 6.3±0.2% (P<0.001) and insulin sensitivity index improved from 2.0±0.3 to 5.7±0.8 (P<0.001). Sulfonylurea agents were discontinued in 11 of the 12 subjects taking them. Fasting triglycerides and total cholesterol decreased (167±21 to 96±20 mg/dL (P<0.001) and 185±10 to 165±7 mg/dL (P=0.03), respectively), and HDL-cholesterol increased from 43±4 to 50±5 mg/dL, P=0.027. There was a non-significant decrease in LDL-cholesterol (100±9 to 90±6 mg/dL, P=0.18) in the subjects, all of whom were already on statin therapy. Systolic blood pressure improved significantly (126±3 to 115±3 mmHg, P=0.005) as did VO_2 max (44±2 to 49±2 mL●min^{-1}●kg FFM^{-1}, P<0.001).

Discussion: This aggressive integrated lifestyle intervention produced WL greater than is ordinarily achieved in research studies or clinical practice. There was a significant reduction in the need for diabetes medications and improvement in metabolic parameters.

Conclusion: Our results demonstrate that an intensive but practical program, consisting of real-time monitoring of calorie balance with regular follow-up and behavioral modification support, can provide significant weight loss in obese subjects with T2DM.

Abstract #308

MORTALITY RATES DUE TO DIABETES AND DIABETIC COMPLICATIONS IN COSTA RICA 2005 - 2010.

Jose Jimenez-Montero, MD

Objective: In Costa Rica diabetes prevalence is 10.8%. Mortality due to diabetes increased (1990 to 2004) from 8 to 20 /100.000 habitants and length of hospitalization average 7 days. Along with the health reform, a national diabetes management program was lunched in 2007. This study analyzes mortality and morbidity due to diabetes and diabetic complications from 2005 to 2010 in this country.

Methods: The database from the Centro Centroamericano de Población and the International Classification of Diseases (CIE-10) were used to calculate death rates due to diabetes, specifically, death associated with diabetic nephropathy and peripheral vascular disease. Data of hospital discharges provided by the Department of Statistics of the Caja Costarricense de Seguro Social was employed to determine changes in morbidity from diabetes during the same timeframe.

Results: Compared to previous reports, mortality rate due to diabetes was reduced and remained stable at 9/100.000 habitants. Mortality rose after 60 years of age in both male and female populations. Death due to renal complications was lower in elderly diabetics in 2010 compared to 2005, on the other had no changes in peripheral vascular mortality was observed. Country based hospital discharges due to renal complications were 954 and 552 in 2005 and 2010, respectively and hospital discharges due to coronary artery disease showed a modest increase from 1123 to 1417, in same period. Deaths due to acute complications such as diabetic ketoacidosis remain low and tend to diminish over time.

Discussion: Fewer deaths due to renal complications occurred, specifically, in the elderly diabetic population and an approximately 40% reduction in hospital discharges due to renal complications was also noted. In this study it was not possible to calculate coronary heart disease mortality in diabetics, but hospitalizations due to coronary heart disease in diabetics increased.

Conclusion: Improvements in glucose control seen in Costa Rica in the last decades may have played a positive impact on diabetic nephropathy. Alternatively, since cardiovascular disease remains as the first cause of death in diabetics, one may speculate if the results of this study could be explained by an increased premature death due to cardiovascular mortality.

HYPOGLYCEMIA

Abstract #400

MEDICAL OPTIONS FOR TREATMENT OF INSULINOMA

Anda Gonciulea, MD

Objective: We describe a case of endogenous hyperinsulinemic hypoglycemia and review medical treatment options, including newer agents.
Methods: This is a review of literature looking at the medical treatment options for managing hypoglycemia in patients with insulinoma.
Case Presentation: An 88 year old male with a history of "borderline" diabetes (no medications) experienced multiple episodes of fasting hypoglycemia (range 46-65 mg/dl) over a two week period while hospitalized for a traumatic fall. He denied symptoms of hypoglycemia but of note, the patient finished a 5 lb bag of sugar every 2 weeks. CT and MRI of the abdomen (done on admission for trauma) suggested a possible 1.1 cm lesion in the neck of the pancreas, but this was not confirmed. Diagnostics: After a 9 hour fast, he became hypoglycemic at 46mg/dl with endogenous hyperinsulinism confirmed with: insulin level 13 μU/ml (n 2.6 - 24.9) , C-peptide 4.8 ng/ml (n 1.1-4.4), pro-insulin 180pmol/l (n 3-20) and betahydroxybutyrate 0.2 mmol/l (n <0.4). Prolactin, calcium, PTH and gastrin levels were all normal. The patient and family refused further evaluation and did not want surgical intervention. He was started on diazoxide and dietary therapy with good response to date.
Discussion: Insulinomas, though rare, are the most common functioning pancreatic endocrine neoplasms. 90-95% are benign. Surgical management with laparoscopic approach is often the treatment of choice for benign cases. Medical management is indicated in malignant disease, when surgery is refused or when surgical risks outweigh benefits. Diazoxide and octreotide analogues are first line treatment choices for hypoglycemia control, with steroids, phenytoin and calcium channel blockers as other possible options. Refractory hypoglycemic cases have responded to mTOR pathway inhibitors (sirolimus and everolimus) and radiolabelled somatostatin analogues (lutetium-177 octreotide with or without capecitabine) Our case of endogenous hyperinsulinemic hypoglycemia, in an elderly patient illustrates the need to fully understand medical treatment options. Further information on the newer agents will be discussed in detail.
Conclusion: Control of insulin hypersecretion and hypoglycemia may be difficult in patients with insulinomas. Diazoxide, octreotide analogues, steroids, phenytoin and calcium channel blockers are some of the treatment choices available for hypoglycemia control. Newer agents like mTOR pathway inhibitors and radiolabelled somatostatin analogues may be an effective option in situations where hypoglycemia is difficult to control with other available agents and have been shown to decrease tumor burden in cases of malignant insulinoma.

Abstract #401

CAUSES OF FASTING-EVOKED EN ROUTE HYPOGLYCEMIA IN DIABETES (FEEHD): A CASE SERIES AND RECOMMENDATIONS FOR PREVENTIVE MEASURES

Saleh Aldasouqi, MD, Archana Reddy, MD,
Ved Gossain, MD, George Hebdon, M.D.,
Wendy Kushion, Bhavini Bhavsar, MBBS, M.D,
Sameer Ansar, MD

Objective: In a recent observational study, patients with diabetes who fast overnight for laboratory tests were found to be at risk of developing hypoglycemia en route to and from laboratory facilities. In patients with hypoglycemia unawareness, severe hypoglycemia may progress without warning symptoms, and because these hypoglycemic events are not captured at the time of occurrence, the study findings raised concerns about patients' safety, including potential risk of traffic accidents while driving if hypoglycemia is severe enough to impair cognition. Due to design limitations in the aforementioned study, the causes or circumstances contributing to hypoglycemia could not be fully elucidated. This study was undertaken to better understand this overlooked safety problem in diabetes management.
Methods: A retrospective case series of laboratory-confirmed fasting hypoglycemia in patients with diabetes taking various anti-diabetic medications who fasted overnight for lipid profile tests.
Case Presentation: In a 4-month period, 3 patients with either type 1 or type 2 diabetes, developed hypoglycemia while fasting for laboratory tests, ranging in severity from mild (65 mg/dl) to critical (31 mg/dl). While fasting was the main denominator, specific causes of hypoglycemia in these cases included excessive basal insulin dose while fasting; taking regular and intermediate insulin without eating; and taking long-acting sulphonyluria in the evening before fasting. Hypoglycemic symptoms ranged from tremors and anxiety to asymptomatic.
Discussion: Patients with diabetes who fast for laboratory tests are at risk of hypoglycemia, that could be potentially hazardous to patients and to society if hypoglycemia results in traffic accidents.
Conclusion: This overlooked, underreported and potentially serious problem can be preventable by appropriate education and preparation of patients who are requested to fast overnight for laboratory tests.

Abstract #402

FASTING-EVOKED HYPOGLYCEMIA IN INSTITUTIONALIZED PATIENTS WITH DIABETES DUE TO LACK OF BASIC DIABETES KNOWLEDGE IN A MEMBER OF THE NURSING STAFF

Saleh Aldasouqi, MD, George Hebdon, M.D., Ved Gossain, MD, Sameer Ansar, MD, Bhavini Bhavsar, MBBS, M.D, Mamata Ojha, Cynthia Monroe

Objective: A recent study reported that ambulatory patients on antidiabetic medications who fast for laboratory tests are at risk for hypoglycemia, occurring en route, to and from laboratory facilities. The aforemnetioned study underscored a deficit in diabetes management knowledge amongst care providers. In this case report, we reoprt a case of diabetes-related hypoglycemia in a different context—institutionalized patients, where nursing staff handle diabetes management.
Case Presentation: An 88-year-old woman with diabetes, who resides in an assisted living facility, was scheduled for a fasting laboratory test, to be drawn by a visiting phlebotomist. She took her daily dose of 40 units of 70/30 mixed insulin at 8:00 AM, but the phlebotomist from the hospital laboratory did not arrive until 9:08 AM. Within 30 minutes of insulin administration, she began feeling shaky and anxious, and she asked the nurse to forgo the laboratory test, and to allow her to go to the cafeteria of the facility to eat breakfast, for fear of hypoglycemia. The nurse refused, despite the patient's insistence that she should eat following insulin administration. Following the blood draw, the patient finally took her breakfast and felt better. In retrospect, the glucose result was 26 mg/dl, while the fingerstick glucose was 184 mg/dl before taking insulin.
Discussion: This case illustrates a serious deficit of medical knowledge about insulin pharmacokinetics by a nursing staff member of a nursing home. Whether this knowledge deficit is isolated or more common is unknown. Diabetes education curricula are usually targeted towards patient education, but this case suggests the need for diabetes education amongst nursing staff. Hypoglycemia in residents of nursing homes can cause serious harm in view of advanced age, frailty and associated comorbidities.
Conclusion: Nursing staff in nursing homes, assisted-living facilities and other settings, such as prisons, should be adequately educated on the use of insulin and other anti-diabetic medications, in relation to hypoglycemia, especially that caused by fasting for laboratory tests.

Abstract #403

HYPOGLYCEMIA WHILE FASTING FOR MORNING BLOOD TESTS: A FOLLOW UP OF THE CAPE GIRARDEAU HYPOGLYCEMIA EN ROUTE PREVENTION PROGRAM

Saleh Aldasouqi, MD, Ahmad Sheikh, MD, Pamela Klosterman, Sheila Kniestedt, Lisa Schubert, Rosie Danker, Martin Grajower, MD, FACP

Objective: Periodically, laboratories call physicians' practices with critical fasting glucose results of patients who had already left the lab facility, creating nervousness about patient safety. Diabetes education programs lack guidelines to prepare patients fasting for lab tests. We undertook this prevention project to evaluate this overlooked problem, and to evaluate the effectiveness of a prevention program. Results of the pilot study (completed in September, 2009) which consisted of cohort A (described below) were previously presented at AACE 2011 annual meeting and were published in May, 2011 in the Diabetes Care journal. This follow up study was completed in September, 2011. This abstract is a combined report, including a comparison between the original cohort (A) and the follow up cohort (B), as described below.
Methods: A retrospective study consisting of chart reviews and telephone interviews of consecutive hypoglycemic events (glucose below 70 mg/dl). Cohort A consisted of patients prior to—and cohort B subsequent to—the implementation of a prevention program involving glucose monitoring and adjustment of anti-diabetic medications. Duration of each cohort was 21 months.
Results: In cohort A (n=55), of 35 patients on anti-diabetic medications who recalled fasting or probably fasting, there were 39 hypoglycemic events, as compared to 18 events in 17 patients in cohort B (n=22), indicating a 54% risk reduction. Frequency of critical hypoglycemia (< 50 mg/dL) was significantly reduced from 11 to 2 events (82%).
Discussion: This is the first study to evaluate iatrogenic, fasting-evoked hypoglycemia, an overlooked safety problem in diabetes management, and the effectiveness of a prevention program. This form of hypoglycemia can put patients with diabetes at risk of traffic accidents. In view of this concept, occurring en route, we refer to this form of hypoglycemia as FEEHD (Fasting-Evoked, En-route Hypoglycemia in Diabetes). We developed this acronym to refer to the need to revisit the actual need for fasting in ordering routine, regular lab tests, and if so, the need to carefully prepare patients. For example, resorting to Non-HDL lipid profiles, when appropriate. Study limitations include retrospective design and reliance on patients' recollection.

Conclusion: Diabetes clinicians and educators should properly instruct their patients on adequate glucose monitoring and adjustment of anti-diabetic medications, when fasting for lab tests, to avoid FEEHD, especially traffic accidents. We present guidelines which proved effective in our program, to help patients with diabetes and their clinicians avert this potentially harmful complication.

Abstract #404

TRANSIENT RECURRENT HEMIPARESIS AS A PRESENTATION OF HYPOGLYCEMIA IN A PATIENT WITH INSULINOMA

Carla Romero MD, Nina Needleman, Agustin Busta

Objective: To report hemiparesis as an early symptom in a patient with insulinoma.
Case Presentation: 32yo woman with no history of diabetes (DM), presented with perceptions of detachment from reality, left hemiparesis, and numbness for 6 months, which usually resolved in 1 or 2 hrs. She is being treated for depression and muscle weakness. Initial visit labs, blood glucose (BG) 48mg/dl (nl 74-106mg/dl). Creatinine, WBC, TSH, and liver panel normal. She is admitted after 12 hrs of fasting for hypoglycemia tests. Asymptomatic fingerstick (FS) 46mg/dl, labs: BG 53mg/dl, insulin <2µIU/ml (nl ≤29.1µIU/ml), C-peptide 0.73ng/ml (nl 0.8-3.1ng/ml), proinsulin 13.7pmol/l (nl ≤18.8pmol/l), and cortisol 8µg/dl (nl 4-22µg/dl); 2 hours later she became symptomatic with FS 46mg/dl, insulin <2 µIU/ml, C-peptide 0.98ng/ml, proinsulin 12.9pmol/l. She received an infusion of D5½NS overnight. The next day, D5½NS was stopped; and blood work re-sent when FS 50mg/dl. Insulin 23.5µIU/ml, C-peptide 5.1ng/ml, proinsulin 68.6pmol/l; sulfonylureas and metiglinides were negative. A CT scan showed an 8mm hypervascular round lesion in the pancreatic head, and she underwent a laparotomy. Pathology reported a 6mm well differentiated neuroendocrine tumor. After surgery, her hemiparesis and numbness completely resolved. She is no longer on antidepressants, and she is back to work. Her glucose levels have normalized.
Discussion: Hypoglycemic symptoms can occur when BG falls <70 mg/dl. One can experience anxiety, sweating, hunger, palpitations and other symptoms (Adrenergic symptoms). If BG <55mg/dl, one may experience confusion, fatigue, seizures, loss of consciousness, and focal neurologic deficits (Neuroglycopenic symptoms). Patients with hypoglycemia unawareness, or frequent hypoglycemic episodes, have a lower threshold for counterregulatory hormone release and sympathetic activation, usually when glucose <50mg/dl. Most hypoglycemic episodes occur in patients under treatment for diabetes. In non-diabetics, the hypoglycemia work-up should include drug side effects, food poisoning (such as ackee fruit), alcohol use, critical illness, hormonal and enzyme deficiencies, and pancreas β cell tumors (such as insulinoma).
Conclusion: In a young individual with neuropsychiatric complaints, the clinical workup must include glucose metabolism disorders. Insulinoma should be included in the differential, once other more common etiologies have been excluded.

Abstract #405

DOEGE-POTTER SYNDROME. A CASE REPORT

Miguel Pinto, MD, FACE, Rosa Ramirez-Vela

Objective: To describe a case of Doege-Potter syndrome and Pierre-Marie-Banberg syndrome due to intrathoracic tumour.
Methods: We abstracted the clinical chart and reviewed the pertinent medical literature.
Case Presentation: A 55-year-old woman with history of progressive dyspnea, weight loss, and episodes of palpitations, anxiety and diaphoresis came to Emergency room because of confusion. At presentation, plasma glucose was 30 mg/dL. Physical examination showed marked nail clubbing and decreased breath sounds in the right lower chest. Serum insulin and C-peptide were depressed, and chest x rays and chest CT scan showed a lobulated mass in the right chest suggestive of benign pleural tumour. She was treated with a continuous glucose infusion, and patient underwent thoracotomy with excision of the complete tumour. Microscopic examination of the tumour, revealed spindle cell proliferation arranged in short intersecting fascicles with areas separated by dense collagenous fibrous stroma. Inmmunohistochemistry was consistent with a solitary fibrous tumour. The episodes of hypoglycemia abated postoperatively. The dyspnea resolved within three months.
Discussion: Pleural solitary fibrous tumour (SFT) is an uncommon tumour. A recent review identified 48 cases presenting with hypoglycemia that have been published since 1981 in the English literature. Pleural SFT was considered to be of mesothelial origin, but recent evidence revealed it to be of mesenchymal histogenesis. At least, 80% of all pleural SFTs are benign, while the rest may show local recurrence and metastases. Hypoglycemia is rare in pleural SFT, occurring in approximately 5% of cases. Paraneoplastic hypoglycemia results from secretion of an unprocessed or incomplete high molecular weight form of insulin-like growth factor type II. Surgery is the treatment of choice. Hypoglycemia and finger-clubbing almost always completely resolve following surgical excision.
Conclusion: Doege-Potter syndrome is a rare complication of a pleural SFT. Complete surgical resection is associated with rapid resolution of episodic hypoglycemia and dyspnea.

Abstract #406

HYPOGLYCEMIA DUE TO ACUTE HEPATITIS. A CASE REPORT

Miguel Pinto, MD, FACE, Jose Pinto, Rosa Ramirez-Vela

Objective: To describe a case of hypoglycemia due to acute hepatitis in a young adult without diabetes.
Methods: We abstracted the clinical chart and reviewed the pertinent medical literature.
Case Presentation: A 25-year-old man with history of alcohol and marijuana abuse was admitted because of symptomatic hypoglycemia (43 mg/dL). In the previous nine days, he was complaining of malaise, fever, nausea and vomiting. Three days before admission, diarrhea and abdominal pain was added. He was medicated with acetaminophen, ciprofloxacin and dimenhydrinate without improvement. He stopped alcohol use in the previous two weeks. Physical examination was normal and laboratory results were ALT 998 U/L (NR: 21-72), AST 1234 U/L (NR: 17-59), LDH 4436 U/L (NR: 313-618), ALKP 868 U/L (NR: 38-126), GGT 446 U/L (NR: 15-73), albumin 2.9 g/dL, conjugated bilirrubin 0.7 mg/dL, and unconjugated bilirrubin 0.6 mg/dL. TSH, free T4, cortisol, insulin, and C-peptide were normal. Abdominal ultrasound showed acute diffuse hepatopathy with diminished echogenicity and normal intra/extrahepatic biliary ducts. He was treated with IV glucose and vitamin supplementation. HBsAg, anti-HAV (IgM), and anti-HCV were negative. He was discharged without complaints and no new episode of hypoglycemia was documented.
Discussion: Clinical hypoglycemia is a plasma glucose concentration low enough to cause symptoms and/or signs, including impairment of brain function. In healthy individuals, symptoms of hypoglycemia develop at a mean plasma glucose concentration of approximately 55 mg/dL. Hypoglycemia is an uncommon clinical event except in persons who use drugs that lower plasma glucose levels, particularly insulin or an insulin secretagogue, to treat diabetes mellitus. Drugs are the most common cause of hypoglycemia. Offending drugs include alcohol and fluoroquinolones. However, critical illnesses like hepatic, renal or cardiac failure also are related to hypoglycemia. In the case of acute hepatitis, hypoglycemia due to depleted hepatic glycogen storage can be present.
Conclusion: Hypoglycemia is a rare complication in persons without diabetes. Drugs are the most common cause of hypoglycemia. In this case, multiple factors were present (acute hepatitis, alcohol abuse, anorexia, and use of ciprofloxacin).

Abstract #407

ASSOCIATION OF ORAL ANTI-DIABETIC MEDICATIONS VERSUS BASAL-BOLUS INSULIN AND THE OCCURRENCE OF HYPOGLYCEMIA IN HOSPITALIZED, NON-CRITICALLY ILL TYPE 2 DIABETIC PATIENTS

Ha Nguyen, MD, Arthur Chernoff, Gentry King, Sherry Pomerantz

Objective: Hypoglycemia is associated with worse patient outcomes. The use of oral anti-diabetic medications (OADM) and hypoglycemia in the inpatient setting has not been well studied. The purpose of this study is to examine the occurrence of hypoglycemia with 2 commonly used inpatient diabetic regimens: OADM + sliding scale insulin (SSI) versus basal-bolus insulin (BBI) in non-critically ill type 2 diabetic patients.
Methods: This is a retrospective, case-controlled study conducted at a tertiary care hospital. All patients who had at least one hypoglycemic episode while admitted to the medicine service from October 2009 to July 2010 were identified. Demographic features, risk factors and the regimen used at the time of hypoglycemia were recorded. Patients with medical conditions that could directly cause hypoglycemia were excluded. A group of sex and age matched patients during the same hospitalization but who did not have hypoglycemia served as a control group.
Results: The study and the control group each included 240 patients. Demographic features between the study and control group had no statistically significant difference: mean age (67, 67.1), sex (female 60%, 62%), and race (black 81%, 78%). OADM + SSI was used in 21% of the study group and 36% of the control group. Bivariate regression analysis suggests the association of BBI use and hypoglycemia (odds ratio [OR] 2.12, 95% confidence interval [CI] 1.36-3.26, $p<0.001$). Other risk factors for hypoglycemia were identified: congestive heart failure (OR 1.77, 95%CI 1.20-2.63), diabetes > 10 years (OR 6.48, 95%CI 3.34-12.79), and serum albumin < 2.5 g/dl (OR 1.89, 95%CI 1.26-2.83). When a logistic regression analysis, adjusted for these additional factors, was performed, the higher odds of hypoglycemia with BBI use was no longer statistically significant (OR 1.52, 95%CI 0.91-2.53, $p=0.107$). However, diabetes > 10 years and serum albumin < 2.5 g/dl were significant risk factors for hypoglycemia (OR 6.05, 95%CI 3.01-12.13, $p<0.001$ and OR 1.69, 95%CI 1.04-2.74, $p=0.03$, respectively).
Discussion: This study suggests that a duration of diabetes for more than 10 years and albumin less than 2.5g/dl are significant risk factors for hypoglycemia in non-critically ill, hospitalized type 2 diabetic patients.
Conclusion: In this study, there was no statistically

significant difference between the use of oral anti-diabetic medications + SSI versus basal-bolus insulin and the occurrence of hypoglycemia. A prospective randomized study to confirm this finding would be helpful.

Abstract #408

SEVERE REACTIVE HYPOGLYCEMIA AS AN UNUSUAL SYMPTOM OF AMYLOIDOSIS

Faustino Macuha, MD, Harmeet Narula, MD

Objective: Amyloidosis is an uncommon infiltrative disorder which may rarely cause hyperglycemia & secondary diabetes due a reduced mass of insulin producing beta cells. However, hypoglycemia is rare in patients with amyloidosis. We present an unusual case of reactive hypoglycemia in a patient with amyloidosis.
Case Presentation: 70 year old non-diabetic male with Primary Amyloidosis with severe autonomic neuropathy, HTN, CAD, CHF (EF= 25%) was admitted with a CHF exacerbation. On the 5th hospital day, during discharge planning, patient had an episode of post-prandial hypoglycemia (<1 hour after eating.) Patient's mental status was altered with involuntary movements and diaphoresis. A bedside capillary glucose was noted to be 26mg/dl (Serum glucose was 19mg/dl). Patient was given intravenous D50 which alleviated his symptoms. He denied any surreptious insulin use or oral hypoglycemic intake. Workup revealed: elevated serum C-peptide at 17.5 ng/ml (normal 0.8-3.5); elevated serum insulin level at 204 (fasting 3-19 uIU/ml); Serum β-OH Butyric acid was low at 0.4mg/dl (normal 0.0-3.0); proinsulin 23.6 pmol/L (normal 2.1-26.8). Sulfonylurea screen was negative. ACTH stimulation test was negative for adrenal insufficiency. A 72-hour fast failed to reproduce hypoglycemia. However, the patient had multiple similar episodes of exclusively postprandial hypoglycemia. A diagnosis of reactive hypoglycemia related to amyloidosis was made. Patient was discharged on the 9th hospital day. On outpatient follow-up, dietary modification with frequent feedings provided adequate control of mild postprandial hypoglycemic symptoms.
Discussion: Amyloidosis refers to the extracellular deposition of fibrils of a variety of normal serum proteins . It may be primary (AL amyloidosis) or secondary (AA amyloidosis). Systemic effects depend on the type of precursor protein, tissue distribution, and amount of amyloid distribution. Hypoglycemia in a patient with amyloidosis may be related to hepatic involvement with amyloid, with reduced glycogen stores, leading to increased risk of fasting hypoglycemia. Our patient never had any fasting hypoglycemia, and had exclusively postprandial symptoms. Dysmotility from autonomic neuropathy and dysregulated pancreatic insulin release were likely responsible for the postprandial hypoglycemia in our patient.
Conclusion: Patients with amyloidosis may develop life-threatening hypoglycemia. Endocrinologists should be aware of this rare complication, to appropriately manage patients with this rare metabolic disorder.

Abstract #409

PATIENT CASE REPORT OF DAPTOMYCIN INDUCED HYPOGLYCEMIA

Maher Ghawji, MD, Maher Ghawji

Objective: We aim to recommend a close glucose observation of patient's under Daptomycin to avert a potentially life threatening hypoglycemia.Further in vitro studies are needed to examine the exact mechanisms of the B-cell activation and potential therapeutic application in finding a new line or class of drug in diabetes treatment.
Case Presentation: A patient's case report indicated that daptomycin is the main cause of this patient's induced hypoglycemia. Workup of the hypoglycemia revealed that the cause was due to pancreatic beta cell activation and excessive release of insulin.
Discussion: Daptomycin is a novel cyclic lipopeptide coumpound in cubicin used to treat skin and soft tissue infections. It fights against gram positive bacteria by acting on the cytoplasmic membrane and inhibiting RNA, DNA, protein synthesis thus causing cell death. We observed a patient's case who was given daptomycin for a duration of 5 days and displayed an unusual adverse effect not previosusly reported in literature. During this time of examination, the patient exhibited a dramatic drop in blood sugar. Once the daptomycin was withdrawn, his blood sugar returned to normal.
Conclusion: We concluded from blood tests taken from the patient that daptomycin was the cause of this unusual adverse symptom. The patient's blood sugar dramtically dropped to a critical point and his insulin levels remained unusually high for the 5 days he was on the drug. The day after the patient was taken off daptomycin, his blood glucose levels returned to a normal range.

LIPID DISORDERS

Abstract #500

DO AGE AND GENDER CORRELATE SIGNIFICANTLY WITH DYSLIPIDEMIA?

Shamsuddin Shaik, MD, Haseeb Kazi, MD, David Leh

Objective: Observationally, at our institution, younger individuals paradoxically appeared to have disproportionately higher abnormal lipid profiles, thus we hypothesize that advancing age does not directly correlate with dyslipidemia and subsequent coronary heart disease risk.
Methods: Data was obtained by retrospective review of 150 randomly selected admissions with the primary diagnosis of chest pain. Lack of lipid profile and chest pain as a secondary diagnosis was exclusion criteria. Data, obtained from the Internal Medicine residency patient lists at our institution from 2006 to 2008, was recorded and compared with ATP III Guidelines to classify risk factors and determine the degree of dyslipidemia. Selected demographic details (age, gender, etc.) were also obtained. Correlation of age and gender with dyslipidemia was examined by applying a binary logistic regression model.
Results: Of the patients selected for review, 66% (99/150) fulfilled the selection criteria. The mean age of the cohort was 59 years (range 29-97) and 51% of the subjects were females. The quartiles were evenly distributed around the age ranges, with Q1<46, Q2=46-56, Q3=56-72 and Q4>72. Based on the quartile split, prevalence of dyslipidemia in the four age groups was 73%, 67%, 71% and 53% respectively. Notably, 74% of men and 58% of women had dyslipidemia. Thus it appears that dyslipidemia may be better correlated with gender than age. A logistic regression model demonstrated a one-sided p-value of 0.095 for age and 0.035 for gender, with the usual significance threshold of 0.05. The odds ratios of 0.52 (gender) and 0.99 (age) indicate that women had a 48% reduced risk of dyslipidemia as compared to men, and that the risk goes down by 1% with each additional one year in age.
Discussion: Dyslipidemia increases morbidity and mortality related to coronary heart disease (CHD). ATP III Guidelines and Framingham scores determine the risk of CHD and risk factors are presumed to be related to advanced age. Given our study, gender was a greater correlate with dyslipidemia, with men at a significantly higher risk. Interestingly, the eldest patients (age > 72) had the lowest prevalence of dyslipidemia. This may be indicative of better personal care or optimal pharmacologic treatment for these patients.
Conclusion: Advanced age fails to demonstrate increased prevalence of dyslipidemia. Patient education and strict treatment regimens should be enforced early on to prevent adverse events in all patients with dyslipidemia.

Abstract #501

ELEVATED APOLIPOPROTEIN A-I GENE EXPRESSION IN VITAMIN D RECEPTOR KNOCKOUT MICE.

Sandra Mesliniene, MD, Margaret Gladysz, MD, Emad Naem, MD, Kent Wehmeier, Arshag Mooradian, Michael Haas

Objective: Apolipoprotein A-I (apo A-I) is the primary protein component of high-density lipoprotein (HDL). Though most epidemiological studies suggest that vitamin D levels are positively associated with plasma HDL levels, it is unclear how vitamin D regulates cholesterol metabolism and reverse-cholesterol transport (RCT).
Methods: Apo A-I and albumin levels were measured by Western blotting. Apo A-I, ATP-binding cassette protein A1 (ABCA1), scavenger receptor B type 1 (SR-B1), albumin, and glyceraldehyde-3-phosphate dehydrogenase (GAPDH) mRNA levels were measured by real-time quantitative PCR.
Results: To determine if the vitamin D receptor (VDR) regulates apo A-I gene expression and HDL synthesis in vivo, apo A-I protein levels were measured in two separate lines of VDR knockout mice. In both cases apo A-I protein levels were high in the homozygous knockout animals than in the wild type. Albumin levels were similar for knockout and wild type mice. Likewise, apo A-I mRNA levels were elevated in livers of VDR knockout mice. To determine if other hepatic genes involved in HDL synthesis and RCT are also regulated by the VDR, we measured ABCA1 and scavenger receptor B type 1 SR-B1 mRNA levels. Albumin and GAPDH mRNA levels were measured as controls. In homozygous VDR knockout mice, apo A-I and SR-B1 mRNA levels were significantly elevated relative to WT mice, while GAPDH, albumin and ABCA1 mRNA levels were similar in each group.
Discussion: Early studies in our laboratory demonstrated that 1,25-dihydroxy vitamin D represses hepatic apo A-I gene expression. Based on this observation, we used a genetic approach to determine if the VDR is involved in regulating apo A-I expression in vivo by ascertaining whether or not apo A-I levels are elevated in the plasma of VDR-null mice. This was observed in both lines of mice. The increase in hepatic SR-B1 mRNA levels in the VDR-null mice suggests that other genes involved in reverse-cholesterol transport and atherosclerosis may be regulated by vitamin D. These studies suggest that the relationship between VDR activation and dyslipidemia is complex and that there are genetic modifiers that dramatically alter the phenotype.

Conclusion: These results suggest that the VDR, either directly or indirectly, regulates expression of several genes involved in hepatic HDL synthesis and RCT.

Abstract #502

TIME FOR CHANGE: DYSLIPIDEMIA MANAGEMENT BY INTERNAL MEDICINE HOUSESTAFF

Haseeb Kazi, MD, Shamsuddin Shaik, MD, David Leh

Objective: To demonstrate that management of dyslipidemia does not improve with post-graduate level advancement.
Methods: Data was extracted by retrospective review of admissions for the primary diagnosis of chest pain, regardless of final diagnosis. Exclusion criteria included the lack of an ordered lipid profile or identification of chest pain as a secondary diagnosis. Data from 150 patients, from the Internal Medicine residency patient lists at our institution from 2006 to 2008, was recorded and compared with the ATP III Guidelines to classify risk factors and lipid profiles. For each patient, we identified whether appropriate treatment measures were followed and the post-graduate level of the house officer involved with the care. Appropriate treatment is defined as lifestyle modification or pharmacologic treatment.
Results: Among the 150 randomly selected patients, there were 99 cases (66%, 95%-confidence interval 58%-74%) where lipid profiles were abnormal. Appropriate treatment based on guidelines was not followed in 63 cases (42%, with a 95%-confidence interval 34%-50%). Of these cases where appropriate treatment was not followed, 94 cases were handled by interns (PGY-1) and 56 cases were handled by senior residents (PGY-2 and PGY-3). The error rate among interns was 38/94 (40.4%) and the error rate among senior residents was 25/56 (44.6%). A two sample comparison of binomial proportions resulted in a p-value of 0.614, indicating that the error rates among the two groups were statistically similar.
Discussion: Dyslipidemia is associated with increased morbidity and mortality in coronary heart disease (CHD) and its equivalents. ATP III Guidelines recommend treatments depending on major and minor risk factors. As a low Framingham score correlates with a lower 10-year risk of CHD, appropriate lipid management leads to improved outcome and fewer adverse/fatal events. Inadequate appreciation of cardiovascular risk stratification results in suboptimal patient care. Given our study, advancement in Internal Medicine post-graduate level fails to improve dyslipidemia management.
Conclusion: Lipid management should be promoted not only at the beginning of PGY-1, but needs to be reemphasized and monitored throughout training to ensure optimal patient care.

Abstract #503

DYSLIPIDEMIA IN NIGERIANS WITH NEWLY DIAGNOSED SYSTEMIC ARTERIAL HYPERTENSION

Doris Uchenna, MD, Jane Anyalechi

Objective: Hypertension is a formidable cardiovascular problem in Nigeria. It often coexists with dyslipidemia, thereby worsening the overall cardiovascular risk immensely
Methods: This study was conducted at the University of Port Harcourt Teaching Hospital. One hundred and fifty newly diagnosed hypertensives aged 18 - 60years were randomly selected. The same number of healthy non hypertensive age, sex and body mass index matched controls was also randomly selected. The fasting lipid profile in all study participants was determined.
Results: Total cholesterol > 5.2 mmol/l occurred in 73 (48.7%) and 34 (22.7%) of cases and controls respectively. Low density lipoprotein (LDL) cholesterol > 3.4mmol/l occurred in 67 (44.7%) and 24 (16%) of cases and controls respectively. Sixty five (43.3%) and 21 (14%) cases and controls respectively, had triglyceride levels > 1.7mm0l/l. Sixty one (40.7%) and 57 (38.0%) cases and controls respectively had high density lipoprotein (HDL) cholesterol levels < 1.0mmol/l. All the differences were statistically significant except for HDL-C.
Discussion: Co-morbid hypertension and dyslipidemia is quite prevalent as demonstrated by this study. Hypertension and dyslipidemia are related to atherosclerosis both in aetiology and prognosis. With co-morbid hypertension and dyslipidemia, the absolute cardiovascular risk is multiplicative rather than additive. The singular discovery of association of cholesterol with cardiovascular disease in the Framingham study has led to dietary changes and pharmacotherapy for dylipidemia, with reduction in the incidence of cardiovascular disease
Conclusion: The prevalence of dyslipidemia in Nigerians with newly diagnosed systemic hypertension is remarkable. It should therefore be sought in all hypertensives and tackled aggressively. Education about healthy life style modifications should be intensely encouraged.

Abstract #504

SEVERE HYPOTHYROISIM REVEALED BY EZETIMIBE INDUCED MYOPATHY

Emad Naem, MD

Objective: To report a case of severe hypothyroidism presenting with hand swelling and worsening myopathy after Ezetimibe therapy.
Methods: We present a case report, including clinical and laboratory data in a man with myopathy and concomitant severe hypothyroidism.
Case Presentation: A 40 year old male presented to rheumatology clinic for evaluation of elevated CK, rashes, fatigue, myalagia and upper and lower extremities edema. His condition got rapidly worse after he was started on Ezetimibe several weeks prior to his visit. He was found to have a significantly high CK and renal insufficiency. When Ezetimibe was discontinued, CK level improved. However, it remained elevated at a level similar to his CK level prior to starting Ezetimibe. His physical examination revealed a delayed relaxation phase suggestive of hypothyroidism. Diagnosis of hypothyroidism was confirmed with the thyroid function tests. Treatment with Levothryoxine resulted in significant improvement of patient's symptoms as well as normalization of the elevated CPK level and renal function.
Discussion: Treating this patient's hyperlipidemia with Ezetimibe resulted in worsening of his symptoms and further elevation in the CK level. When Ezetimibe was discontinued, CK level improved. However, it remained elevated at a level similar to his CK level prior to starting Ezetimibe. Only after starting Levothyroxine did the raised CK, and Cr fall towards normal values. This suggests that Ezetimibe can worsen myopathy and cause significant CK elevation in the setting of untreated hypothyroidism. Hypothyroidism is a well-recognized but often overlooked cause of dyslipidemia and myositis. There are several case reports of hypothyroidism induced myopathy that have been precipitated by statin therapy. To our knowledge, no case of myopathy with significant CK elevation associated with Ezetimibe in the setting of hypothyroidism has been previously reported.
Conclusion: Ezetimibe , like statin, may cause worsening of the hypothyroidism related myopathy. Hypothyroidism should be considered in patients with hyperlipidemia before starting hypolipidemic drugs. Treatment with levothyroxine will likely resolve the problem and prevent the myopathy that could be induced by these drugs.

Abstract #505

PROFOUND FASTING CHYLOMICRONEMIA DURING PREGNANCY: COMPLEXITIES IN MANAGEMENT

Daniel Okorodudu, MD, Matthew Crowley, MD

Objective: To present a pregnancy complicated by fasting chylomicronemia, eruptive xanthomas, and epigastiric pain, managed with very low fat diet, medications, plasmapheresis, and insulin infusion.
Case Presentation: We report a 40-year-old G4P1A2 woman at 25 weeks gestation who had 2 days of progressive mid-epigastric pain radiating to her back associated with nausea. Her past history included elevated triglycerides (TG average 450mg/dl) and type 2 diabetes mellitus (hemoglobin A1C 7.4% on presentation). She was taking gemfibrozil 600mg b.i.d. and rosuvastatin 10mg daily. Exam showed lipemia retinalis, scattered eruptive xanthomas , and diffuse epigastric tenderness. TG was 24,390 mg/dl with dilution to assess within the linear range. Amylase, lipase, and hepatic panel were normal. Abdominal ultrasound showed normal gall bladder; pancreas was not visualized due to bowel gas. Patient was presumptively managed for acute pancreatitis. Insulin infusion protocol was initiated. She received 3 daily plasma exchanges, lowering TG to 2,982 mg/dL. Rosuvastatin was stopped while omega-3 fatty acid ethyl esters 4 g daily was added to her gemfibrozil. Patient was educated on a very low fat diet. At discharge, her abdominal pain had resolved. Eruptive xanthomas improved over 2 weeks, and no new xanthomas have appeared. The fetus has been monitored biweekly and remains without abnormality except for borderline fetal growth restriction. The patient receives twice weekly plasmapheresis with TG range between 948 and 8,514mg/dl. She is on a 4-shot insulin regimen using Lantus and Novolog with mean pre-meal glucose of 100 and 1 hour postprandial 120.
Discussion: Fasting chylomicronemia in pregnancy may result in life threatening complications such as pancreatitis. Prior to pregnancy, our patient had only moderate hypertriglyceridemia, which makes recessive disorders involving lipoprotein lipase, apolipoproteins C-II or A-V, or GPI-anchored HDL-binding protein 1 highly unlikely. High estrogen levels of pregnancy likely exacerbated her hypertriglyceridemia. Strict avoidance of fat minimizes formation of new chylomicrons, while intensive insulin replacement reduces fatty acid transport to the liver and supports lipoprotein lipase. Endogenous plasma TG (in VLDL) might be less injurious to the pancreas than chylomicrons derived from exogenous fat. Nevertheless, continuing TG over 4000 mg/dl warrants repetitive plasmapheresis.

Conclusion: Management of severe hypertriglyceridemia in pregnancy is complex and not without risks. These include potential teratogenicity of lipid-modifying drugs, hemodynamic and metabolic instability during plasmapharesis, and impact of diet on the developing fetus.

Abstract #506

A CASE OF PEG-L-ASPARAGINASE-INDUCED HYPERTRIGLYCERIDEMIA TREATED WITH INTRAVENOUS HEPARIN BOLUSES

Patricia Sareh, MD, Kashif Munir, MD, Rana Malek

Objective: Peg-L-asparaginase (PLA) is commonly used for treatment of acute lymphoblastic leukemia (ALL). We describe a case of severe PLA-induced hypertriglyceridemia treated with intravenous (IV) heparin boluses.
Case Presentation: A 36 year-old Asian man with pre-B-cell ALL presented for maintenance chemotherapy. He was noted to have grossly lipemic blood. His triglyceride (TG) level was >2000 mg/dL. The patient presented with nausea, without abdominal pain or change in bowel movements. He had no other significant medical history, including any prior lipid abnormalities. He was on phase 3 of CALGB protocol, which included methotrexate and PLA (last given 3 weeks prior). His other medications were: acyclovir, pantoprazole, loratadine, monthly pentamidine, and ondansetron, lorazepam and oxycodone as needed. His alcohol intake was 1-2 servings per month. His brother had a history of hyperlipidemia. There was no family history of premature coronary artery disease. On physical exam, vital signs were normal and he had no abdominal tenderness or eruptive xanthomas. Laboratory studies showed: total cholesterol 885 mg/dL, TG > 2000 mg/dL (not further quantified due to the upper limit of assay), total bilirubin 1.6mg/dL, amylase 70 units/L and lipase 76 units/L. Hypertriglyceridemia was treated with fenofibrate 145mg daily, fish oil 2 g tid, and heparin 5000 units IV q12h. After 48 hours, TG level improved to 411 mg/dL. Long-term maintenance therapy with fenofibrate and fish oil was continued.
Discussion: PLA is known to cause elevation in TGs. Hypertriglyceridemia is likely due to increased endogenous VLDL synthesis and decreased lipoprotein lipase (LPL) activity, which is a key step in the removal of TG from TG-rich lipoproteins. The complications of severe hypertriglyceridemia include pancreatitis and hyperviscosity syndrome. There are no guidelines for treatment of PLA-induced hypertriglyceridemia, but case reports in the literature have described treatment with plasmapheresis, insulin and heparin infusions. In addition to fenofibrate and fish oil, heparin IV boluses were administered instead of continuous infusion due to ease of administration and monitoring. Heparin reduces TGs via release of LPL and hepatic lipase from the endothelium.
Conclusion: Hypertriglyceridemia is a potential complication of PLA therapy and can be successfully treated with a combination of fenofibrate, fish oil and IV heparin boluses. Using IV heparin boluses to treat PLA-induced hypertriglyceridemia should be considered as an alternative to continuous heparin infusion.

Abstract #507

PREVALENCE OF LOW HDL IN MARKEDLY OBESE PATIENTS

Kamran Rasul, MD, Robert Dubin, William Cefalu, Timothy Allerton, Gabriel Uwaifo, MD

Objective: To identify the prevalence of low HDL in a cohort of markedly obese, predominantly African American subjects enrolled in a multidisciplinary weight management program at LSUHSC, New Orleans , Louisiana.
Methods: A retrospective analysis of serum HDL was performed in 50 patients with BMI of more than 35, enrolled in the weight management program, who were seen from November 2010 to September 2011.
Results: Low HDL cholesterol (<40 mg/dL) was observed in 66% of patients with BMI of more than 35 kg/m2. All patients had Edmonton Staging System score 2 or greater, with class 3 obesity. Mean BMI was 56.3 kg/m2. Mean HDL was 38.8 mg/dL. 44% of patients were diabetics and the prevalence of low HDL in this population was 81%. Among non-diabetics, the prevalence was 53.5%. 76% of patients were females and the prevalence of low HDL in that group was 57.8%. Males were 24% and prevalence of low HDL in them was 91.6%. African Americans were 58% and prevalence of low HDL was 62% in this group. Caucasians were 38% and they had a prevalence of low HDL of 68%.
Discussion: Obese patients because of high prevalence of comorbidities such as diabetes, hypertension and dyslipidemia are at high risk of cardiovascular disease. Low HDL cholesterol levels are one of such cardiovascular risk surrogates. Severely obese patients have a high prevalence of low HDL. This is true even among African American subjects despite their historically higher reported HDL levels and this adds to the already high cardiovascular metabolic risk burden of these patients.
Conclusion: Markedly obese patients have high prevalence of Low HDL, which may be an important target of therapy beyond LDL and triglyceride lowering among markedly obese subjects so as to optimize their cardiovascular risk profile reduction.

Abstract #508

AGGRESSIVE CHOLESTEROL LOWERING WITH HIGH DOSE STATIN THERAPY IN GERIATRIC POPULATION: HOW SAFE IS THIS PRACTICE?

Nidhi Bansal, MBBS, Divey Manocha, Sharon Brangman

Objective: Use of high dose statin therapy (HDST) in patients with stroke has become the standard clinical practice after the SPARCL study in 2006. Although the mean age of population in that study was around 63 years, scientific evidence derived from the same has been extrapolated to much older patients in clinical practice. The goal of our study was to define the magnitude of side effects of HDST in the selected geriatric study population.
Methods: This single-center retrospective study was conducted at Upstate Medical University, Syracuse, NY. We reviewed electronic medical records of 120 patients between the ages of 65- 89 years to collect demographic, clinical, laboratory & adverse drug reaction data. Data were compared between patients on HDST(cases) vs. those on regular doses (controls) using Chi square, Fisher exact test & Student T test. P value <.05 was considered significant. HDST was defined as daily intake of ≥80 mg of simvastatin/ ≥40 mg of atorvastatin/ ≥10 mg of rosuvastatin.
Results: 60 cases were compared with age & sex matched 60 controls.67% cases were on simvastatin, 20% on atorvastatin & 13% were on rosuvastatin. Of these, 79% were initiated on statins for the first time upon hospital admission.Prevalence of elevated ALT (13.4%) and creatine kinase (16.6%) was significantly higher in cases. 14% cases reported myalgias, 9% had nausea & 6% had diarrhea. 27% cases had LDL <100 mg%. 40% cases had total cholesterol <160 mg%, 23% had serum albumin <3.5 g% and 6/8 had low pre albumin levels, all of which indicate poor nutritional status at baseline. Mean HbA1C ≥6.5% was noted in a significantly higher number of cases (41%) than controls (13%).
Discussion: Studies in adults on HDST show association with liver enzyme elevation(1.5%), myopathy(5- 7%)and rhabdomyolysis. Recent data also proposes an increased risk of neuropathy, memory loss,diabetes (upto 12%)and cancer.But evidence based literature on the magnitude of adverse effects of such regimens in geriatric patients is scanty.
Conclusion: When compared to other age groups, use of high dose statins in geriatric stroke patients is associated with significantly higher prevalance of liver dysfunction, elevated muscle enzymes, myalgias and diabetes. Thus dosing of statins in geriatric patients should be individualized.The increased risk may be related to age related changes in liver function, muscle mass, pancreatic islet reserve and poor nutritional status at baseline. There is also a potential risk of worsening diabetes control in post stroke period with continued use of statins and consequent need for more aggressive diabetic management in this large subgroup. This needs to be examined in larger prospective studies.

Abstract #509

RELATIONSHIP AMONG LIPID PROFILE, CALCIUM METABOLISM, AND OTHER CARDIOVASCULAR RISK FACTORS WITH CAROTID-WALL INTIMA-MEDIA THICKNESS IN PATIENTS WITH END-STAGE RENAL DISEASE IN HEMODIALYSIS

Miguel Pinto, MD, FACE, Rosa Ramirez-Vela, Javier Cieza, Felix Medina

Objective: To determinate the relationship among biochemical and clinical factors with carotid-wall intima-media thickness in patients with end-stage renal disease in hemodialysis.
Methods: Fifty adult subjects with end-stage renal disease (ESRD) and regular hemodialysis (>6 months, thrice a week) at the Cayetano Heredia University, Hemodialysis Center were included. Subjects with history of hypothyroidism, chronic liver disease or previous use of statins were excluded. Doppler carotid wall intima-media thickness (IMT) was used as marker of atherosclerosis. Baseline fasting plasma levels for total cholesterol, HDL cholesterol, triglycerides, calcium, phosphorus, PTH, and albumin were measured. Data were analyzed using Pearson's correlation coefficient and multiple linear regression for modeling the relationship among biochemical a clinical variables with IMT (dependent-variable). Subjects signed an informed consent and protocol was approved by the Ethics Committee of Cayetano Heredia University.
Results: The mean age (±SD) was 60 years-old (±19.6) and 42% were female. Hypertension was present in 90% of patients and diabetes was the cause of ESR in 38%. In the bivariate analysis PTH and total cholesterol were related with wider IMT. By multiple regression analysis, older age, smoking, and lower HDL cholesterol were related with IMT (p= 0.002).
Discussion: Cardiovascular disease and other complications of atherosclerosis are the most common cause of death in patients with ESR in maintenance hemodialysis. When compared to the general population, dialysis patients have approximately 10 times higher risk of dying from heart disease. On the other hand, carotid atherosclerosis and stiffness are independent prognostic

factors of cardiovascular morbidity and mortality in the general population and in ESRD patients. The determinants of atherosclerosis and cardiovascular disease in hemodialysis are multiple and consist of conventional risk factors, like hyperlipidemia, hypertension, diabetes and obesity; and specific factors attributed to uremic process or dialysis treatment such as the altered lipid profile, the existence of hyperparathyroidism, the increased levels of acute phase proteins and inflammation markers, all of which seem to play important role in the atherosclerotic process.

Conclusion: In patients with ESRD and hemodialysis, smoking, older age, and lower HDL cholesterol are related with wider IMT.

Abstract #510

UNDIAGNOSED PANHYPOPITUITARISM PRESENTING AS SEVERE DYSLIPIDEMIA IN AN ADOLESCENT

Anjana Harnoor, MD, Sandra Hardee, Fiona Cook, MD, Robert Tanenberg, MD, FACP

Objective: 1) In addition to thyroid hormone deficiency, multiple hormone deficiencies from panhypopituitarism may contribute to severe mixed dyslipidemia. 2) A normal TSH does not rule out hypothyroidism.

Case Presentation: We describe the case of a 17 year old Caucasian male who was referred to Endocrinology for evaluation and management of significant dyslipidemia. The patient had a traumatic brain injury (TBI) following an accident two years prior to presentation. His initial lipid profile at the time of screening prior to medications was TC 480, HDL 33, and TG 968. At the time of his initial consult he was taking fish oil and niacin. His lipid profile was TC 626, TG 1463.His physical exam revealed somewhat less than expected secondary sexual characteristics for his age. Height 5'7" Wt 164 lb BP 107/66 P 87.Further work up revealed TSH 2.38,FT4 0.40,Baseline cortisol 4.8,stimulated cortisol at 30 mins was 12.9mcg/dl and at 60 mins was 16.0mcg/dl,FSH 0.6,LH 2.4,Total testosterone 143 ng/dl,ACTH 31.6,IGF 1 64.These labs indicated that he had secondary hypothyroidism, adrenal insufficiency, mild hypogonadism and growth hormone deficiency, all attributable to panhypopituitarism secondary to TBI. Treatment with hydrocortisone 15 mg/d(10 mg qam and 5 mg qpm) followed one week later by levothyroxine 100 mcg a day was begun. One month after starting hydrocortisone and levothyroxine, his BP had improved to 115/74 .His Free T4 was now normal at 1.4 and his lipid profile improved .TC 369, TG 650. His mother noticed that his school performance had improved significantly. At this time he was started on growth hormone treatment at 0.4 mg/d. Two months later his lipid panel showed marked improvement: TC 221, HDL 38, LDL 120, and TG 315.Upon further follow up his lipid panel further improved:TC 138, HDL 34, LDL 55, TG 245.

Discussion: In the setting of TBI, lipid abnormalities can suggest multiple hormone deficiencies. Panhypopituitarism, a common complication of TBI, may have an insidious presentation depending on the degree of each hormonal abnormality. In our case the patient presented with a severe lipid abnormality that improved dramatically with the treatment of the underlying hormone deficiencies. It is well established that a normal serum TSH rules out primary hypothyroidism. However in secondary hypothyroidism the TSH is usually in the normal range and can be misleading. A low free T4 and a normal TSH should prompt evaluation for secondary hypothyroidism.

Conclusion: In our case the patient's TBI led to undiagnosed panhypopituitarism which contributed to his dyslipidemia. Hormone replacement (cortisol/thyroid/GH) improved the patient's lipid profile and overall quality of life.

METABOLIC BONE DISORDERS

Abstract #600

HYPERCALCEMIC CRISIS FROM PARATHYROID CANCER

Erica Kretchman, DO, Dalal Alromaihi, MD, D. Rao, MBBS, Eric Langer, DO, FACOI, Courtney Tabaka

Case Presentation: 34 year old male presented to a community hospital with complaints of rapid onset of abdominal pain and was diagnosed with pancreatitis. He had prior history of pancreatitis with gallbladder disease 3 years ago but was unable to complete work-up or surgery due to lack of insurance. After presentation he rapidly declined and required ventilator and pressor support. An emergent endocrine consultation was obtained due to calcium of 22 mg/dL and parathyroid hormone of greater then 1300 ng/dL. He had no personal or family history of hypercalcemia or hyperparathyroidism. On exam he had a significant goiter approximating 200 gms. He also presented in acute renal failure and note of osteitis fibrosa cystica. Hypercalcemia was managed with hemodialysis and Sensipar. When pancreatitis became necrotic he was transferred to a large referral hospital and managed with a new endocrinology team. His parathyroid hormone continued to trend down, but was still above 500. With the high suspicion of parathyroid cancer blind biopsies of his goiter were attempted, but revealed only thyroid tissue. His pancreatitis, sepsis, and multi-organ failure eventually caused his demise. On autopsy tissue diagnosis was consistant with parathyroid cancer.
Discussion: Parathyroid carcinoma is a rare cause of PTH dependent hypercalcemia. Our suspicion was high given the significant elevation in his parathyroid hormone and level of hypercalcemia. Even though he presented in acute renal failure, prior lab results had a normal serum creatinine. The bony lesions brought more certainty toward the diagnosis. The diagnosis of parathyroid cancer was made at autopsy.
Conclusion: This is a challenging yet textbook case of parathyroid carcinoma. We are anticipating reviewing interesting radiographic and pathologic images with the presentation.

Abstract #601

VITAMIN D DEFFICIENCY OBFUSCATES THE WORK-UP OF HYPERCALCEMIA

Stephanie Mayer, MD, Thomas Weber

Case Presentation: Patient is a 41 y.o. AA female who presents with total body pain and 4 days of left-sided headache. Labwork revealed an elevated ionized calcium of 1.45 mmol/L, chloride of 110 mmol/L, and creatinine of 1.1 mg/dl, suggesting volume contraction. History was further confounded by the use of hydrochlorothiazide. Following IV fluids, her ionized calcium normalized (1.26 mmol/L). Concomitant labs included intact PTH 57pg/ml, phosphorus 2.1 mg/dL and normal TSH. Repeat corrected calcium was 10.8 mg/dL. Hydrochlorothiazide was discontinued and the patient was referred to Endocrinology. She has no history of nephrolithiasis or bone fracture. Due to GI intolerance, her diet is sparse in dairy products (≤ one serving / week). She has no family history of hypercalcemia, osteoporosis, fractures or nephrolithiasis. She neither smokes nor drinks alcohol. Review of systems included ongoing fatigue as well as bony, knee, and back pains, constipation, depression, and occasional abdominal pain and loss of appetite. Her physical examination was unremarkable. Off of hydrochlorothiazide for 3 months, repeat blood work demonstrated a persistent corrected calcium elevation of 11.0 mg/dL, Ionized calcium 1.39 mmol/L, intact PTH 203 {ref. 14-72}, and severe 25,OH Vitamin D deficiency at 7 ng/ml. A 24 hour urine collection for calcium and creatinine was obtained.
Discussion: PTH-mediated hypercalcemia suggests either hyperparathyroidism (PHPT), primary or tertiary, or familial hypocalciuria hypercalcemia (FHH). In FHH, hypercalcemia occurs due to a mutation in the calcium sensing receptor that resets the set point for PTH secretion. Given the somatic nature and therefore renal effect of the mutation, patients with FHH have very low renal calcium clearance as measured by the fractional excretion of calcium (FeCa: {Ca x serum Cr} / {Serum Ca x Urine Cr}). Although her 24-hour urine calcium was normal (193 mg), her FeCa was very low and more consistent with FHH (0.00813) than PHPT. Generally, patients with FHH will have urine calcium excretion of < 0.01 and those with primary hyperparathyroidism (PHP) of > 0.02. Recent studies suggest that the presence of vitamin D deficiency is associated with worsened PTH hypersecretion, impairment of urinary calcium excretion and reduced sensitivity of FeCa measurement with respect to the detection of PHP. Indeed, after repletion of vitamin D to 19 ng/mL repeat 24 hour urine resulted in a higher FeCa of 0.0196.
Conclusion: Primary hyperparathyroidism may masquerade as FHH in the setting of profound vitamin D deficiency, necessitating judicious replacement and correction of D deficiency to properly diagnose and manage hypercalcemia in these patients.

Abstract #602

TUMOR-INDUCED OSTEOMALACIA (TIO) CAUSED BY PRIMARY FIBROBLAST GROWTH FACTOR-23 (FGF-23) SECRETING NEOPLASM IN AXIAL SKELETON

Aashish Shah, MD, Vivek Gupta, Kevin Wu, Gunjan Gandhi

Case Presentation: A 66-year-old female presented with non-traumatic, chronic dull-aching low back pain with radiation to the right leg. She denied history of nephrolithiasis or fractures. She was being treated for diabetes and hypertension. Physical exam revealed a waddling gait. MRI of the lumbar spine showed a focal lesion in L4 vertebra which was hypermetabolic on PET scan. CT guided biopsy of the lesion was nondiagnostic. A bone scan to evaluate possibility of metastatic disease only showed features of metabolic bone disease. Laboratory testing was remarkable for low serum phosphorous of 1.5 mg/dL (2.5-4.5 mg/dL). Additional abnormalities included elevated serum calcium of 10.6 mg/dL (8.9-10.1 mg/dL), inappropriately high PTH of 120 pg/mL (15-65 pg/mL), low 1,25 dihydroxy vitamin D of <8 pg/mL (18-78 pg/mL) and mildly elevated serum alkaline phosphatase of 162 u/L (45-115 u/L). She had frank phosphaturia, especially for level of serum phosphorous (772 mg, normal 0-1,099 mg). Parathyroid scan was normal. Plasma FGF-23 level was significantly elevated at 3,500 RU/mL (<180 RU/mL). Patient later sustained spontaneous right and left femur fractures. Repeat CT guided needle biopsy of L4 showed a low grade spindle cell neoplasm with positive FGF-23 mRNA expression by RT-PCR in paraffin embedded tissues, confirming the diagnosis of phosphaturic mesenchymal tumor.
Discussion: TIO causes increased excretion of renal phosphate, hypophosphatemia and osteomalacia. Although referred to as a paraneoplastic phenomenon, the tumors are usually benign. Localizing the tumor can be challenging: skeletal survey, MRI and recently, FDG-PET have been used. In addition, somatostatin receptor imaging, phosphate uptake bioassay and indium-111 octreotide scintigraphy have been utilized. Tumors that secrete FGF-23 are typically found in the appendicular skeleton, unlike in our patient. Most osteomalacia-associated phosphaturic mesenchymal tumors are a single histopathologic entity. FGF-23 inhibits phosphate transport in renal tubule and reduces calcitriol production. Tumors producing FGF-23 by frizzled-related protein 4 and matrix extracellular phosphoglycoprotein have been implicated as inducers of hypophosphatemia.
Conclusion: Only 13 cases of TIO with primary lesions in spine have been reported. These lesions were localized to cervical, thoracic or sacral spine. Our case is unique in that the FGF-23 secreting mesenchymal tumor was located in a lumbar vertebra and the diagnosis was confirmed by FGF-23 mRNA expression by RT-PCR . The patient has elected to undergo curative resection of the tumor with spondylectomy and front to back reconstruction with instrumentation.

Abstract #603

LOW SERUM VITAMIN D IS ASSOCIATED WITH METABOLIC SYNDROME IN AFRICAN AMERICAN AND CAUCASIAN AMERICAN MALE VETERANS.

Buvana Manickam, MD, Victoria Ryvkin, MD, Valeriu Neagu, MD, Subhash Kukreja, MD, Elena Barengolts, MD

Objective: Vitamin D and metabolic syndrome interaction has not been well studied; yet both define risk for developing cardiovascular disease (CVD). We assessed whether serum 25-hydroxyvitamin D (s25D) is associated with prevalent metabolic syndrome (MetS) and MetS components in a group of predominantly African American male (AAM) veterans at an urban veteran administration medical center (VAMC).
Methods: Male veterans were recruited, s25D was measured, and health surveys and medical chart reviews were completed. Vitamin D deficiency and insufficiency were defined as s25D < 20 and < 30 ng/ml, respectively.
Results: Among 923 men prevalence of MetS was 35% (n = 323). Race distribution was 65.5, 28.1, 5.5, and 0.9% for AAs, Caucasian Americans (CA), Hispanics, and other, respectively, reflecting diversity of our population. Majority of men were younger than 70 years (76%), retired (61%), considered themselves to be in good health (54.8%), and had low (< 30 ng/ml) s25D level (79%). Overall, 76% of males had a higher than normal BMI of 25 kg/m2, with 35% being overweight and 41% being obese. We compared men without (MetS-) and with (MetS+) metabolic syndrome. Some of significantly different ($p < 0.01$) comparisons (MetS- vs MetS+) included current alcohol use (28.7 vs 15.2%), been married (32.4 vs 41.9%), retired (57.3 vs 67.3%), and health self-perception been fair/poor (40.7 vs 53.1%). Mean (standard deviation, SD) level of s25D in MetS- compared to MetS+ was 22.3 (9.3) and 18.5 (7.6) ng/mL, respectively ($p < 0.001$). The serum 25D decreased (p for trend <0.05), as the number of components of metabolic syndrome increased. Prevalence of metabolic syndrome in AA (36.1%) and CA (30.6%) was similar while s25D level was lower in AA (mean [SD] 18.8 [11.8] ng/ml) vs CA (25.7 [13.3] ng/ml, $p<0.01$) men. In multivariate regression analysis of the entire

group, after controlling for other variables, independent determinants of metabolic syndrome included s25D level, current alcohol use, marital and working status, and self-perception of health. In the model, an increase in s25D of 1 ng/ml was associated with 4% decrease in odds of having metabolic syndrome.
Discussion: Low vitamin D is a significant predictor of metabolic syndrome in African American and Caucasian American male veterans. There is racial variability in vitamin D/metabolic syndrome interaction as well as important contribution of psychosocial risks to metabolic syndrome.
Conclusion: Complexities of interracial interactions and plausible bi- or multi-directional vitamin D/metabolic syndrome relations suggest that further research engaging approach defined as systems biology is warranted.

Abstract #604

ATYPICAL FEMORAL FRACTURES: RADIOGRAPHIC AND HISTOMORPHOMETRIC FEATURES IN 9 PATIENTS

Aliya Khan, MD, Adil Zaidi, Nazir Khan

Objective: This study describes characteristics and histomorphometric and radiographic features of atypical femoral fractures (AFF) as seen in 9 cases referred for evaluation.
Methods: All patients referred for evaluation of AFF were reviewed. Patients meeting the ASBMR criteria for AFF were further evaluated and tetracycline labelled bone biopsies were completed. Radiographs were reviewed by a musculoskeletal radiologist.
Case Presentation: All fracture lines were transverse or short oblique with thickened cortices. We report 9 cases of AFF in patients on long term bisphosphonate (BP) therapy. 6 of 7 fractures occurred without a fall or direct trauma to the femur with 1 case occurring after a fall from standing height. All patients were female; average age was 67 years (range 54-80 years). 2 of the 9 cases were of Chinese descent, 2 were East Indian with 3 being Caucasian. Average BP durations of us was 8.5 years (range 7-14 years). 5 of 9 patients were on alendronate alone, 2 patients were on risedronate. 1 patient had received 18 months of teriparatide, 3 years prior to AFF and had received a total of 10 years of BP use prior to teriparatide. Prodromal thigh or groin pain was seen in 5 of the 9 patients for 3 to 12 months prior to fracture. Proton pump inhibitor use was present in 1 patient for the previous 2 years. 1 patient was on prednisone for rheumatoid arthritis. Rheumatoid arthritis was present in 2 cases. Diabetes was not present in any of the 9 cases. Bilaterality occurred in 1 patient with the second AFF occurring 1 month after the first AFF. Renal function was well preserved with estimated glomerular filtration (eGFR) rates > 60 ml/min in all cases. 25 hydroxy Vitamin D levels were >75 nmol/L in 5 cases. 2 cases had mild Vitamin D insufficiency (>50 nmol/L).
Discussion: A large number of the AFF occurred in women of Asian descent (4 of 9). 6 of the 9 AFF occurred in the absence of a fall. Prodromal pain was commonly seen. Proton pump inhibitors were used in only 1 patient. Histomorphometric features included evidence of mineralization abnormalities and decreased bone formation.
Conclusion: AFF in association with long term BP use are being seen in a disproportionately large number of Asian women. Further evaluation of all AFF with identification of predisposing key clinical risk factors is needed. Improved understanding of the pathophysiology may be gained with further histomorphometric data in larger numbers of patients.

Abstract #605

CALCIMIMETIC THERAPY FOR SEVERE SECONDARY HYPERPARATHYROIDISM REFRACTORY TO VITAMIN D REPLETION AFTER DUODENAL SWITCH SURGERY

Deepika Nallala, MBBS, MD, Chaitanya Mamillapalli, Michael Jakoby, IV, MD

Objective: Duodenal switch surgery achieves weight loss through both restrictive and malabsorptive changes to the alimentary tract. Malabsorption is achieved through biliopancreatic diversion that bypasses the duodenum and proximal jejunum. Hypovitaminosis D and hypocalcemia may result and lead to secondary hyperparathyroidism (SHPT). Little is published regarding refractory SHPT in obese patients managed by bariatric surgery. We present a case of severe SHPT that persisted after vitamin D repletion but responded well to calcimimetic therapy.
Case Presentation: A 49-year-old woman underwent duodenal switch surgery six years before referral for vitamin D deficiency and osteoporosis. The patient reported feeling well and denied weakness or musculoskeletal discomfort. Calcium carbonate 2,000 mg daily and calcitriol 0.25 mcg twice daily were prescribed for treatment. Laboratory data at presentation were notable for 25-hydroxy-vitamin D (25-OH D) 4 ng/mL, intact parathyroid hormone (PTH) 826 pg/mL (ref range 10-65), serum calcium 8.4 mg/dL (ref range 8.6-10.2), albumin 3.7 g/dL (ref range 3.4-5.0), phosphorus 3.5 mg/dL (ref range 2.5-4.9), and creatinine 0.9 mg/dL (ref range 0.6-1.0). Hypovitaminosis D was treated with high dose ergocalciferol (initially 50,000 IU twice weekly, titrated

to 50,000 IU three times daily), and 25-OH D recovered to 41 ng/mL. Serum calcium consistently fell in the range of 8.5-9.0 mg/dL, and phosphorus ranged from 3.5-4.0 mg/dL. However, after nine months of therapy, intact PTH remained markedly elevated (1,337 pg/mL). The calcimimetic agent cinacalcet was started at 30 mg daily, and the drug was increased at two to three week intervals to a maximum dose of 120 mg daily. Calcium carbonate was advanced to 3,000 mg three times daily. Intact PTH fell over two-fold to 546 pg/mL at 90 mg cinacalcet, with no improvement in PTH on advancing to 120 mg. Serum calcium remained in the range of 7.8-8.7 mg/dL.

Discussion: Risk of hypovitaminosis D, hypocalcemia, and SHPT is significant in patients who undergo malabsorptive bariatric surgery. SHPT may occur in the absence of subnormal 25-OH D levels and may be refractory to vitamin D repletion. Though cinacalcet is used to manage SHPT due to chronic kidney disease, we are unaware of any reports documenting cinacalcet as therapy for SHPT caused by bariatric surgery.

Conclusion: This case demonstrates that calcimimetic agents can be used to safely and effectively lower PTH levels in vitamin D replete patients with refractory SHPT after biliopancreatic diversion with duodenal switch.

Abstract #606

TUMOR-INDUCED OSTEOMALACIA ASSOCIATED WITH UNDIFFERENTIATED CARCINOMA OF THE PANCREAS WITH OSTEOCLAST-LIKE GIANT CELLS

Evelina Svrdlan, MD, Ivance Albert Pugoy, MD, Marianne Ghobrial, MD, Katayoun Edalat Parsi, DO, Jagrati Mathur, Soe Naing, MD, MRCP, FACE

Objective: To describe a rare cause of tumor-induced osteomalacia (TIO)

Case Presentation: A 53 years old male presented with one year history of disabling bilateral knee pain. Initial evaluation revealed hypophosphatemia (1.5, NR: 2.5-4.5 mg/dl), normal serum calcium (9.3, NR 8.4-10.2 mg/dl), low 1,25-dihydroxyvitamin D (20.1, NR 25.1-66.1), normal 25-hydroxyvitamin D (56.1, NR 20- 100 ng/ml), normal PTH (64.7, NR 11.1 to 79.5 pg/ml) and high bone-specific alkaline phosphatase (62.3, NR 15-41.3 u/l). He also had low threshold for renal tubular reabsorption of phosphate (TmP/GFR) (0.389, NR 0.8-1.6 nmol/l) and high fractional excretion of phosphate (35.07%, NR 10-15%), indicating renal phosphate wasting. DXA bone scan showed low bone density with T score of -2.6 at L1-L4 spine and -2.0 at left femoral neck. These results were highly suggestive of TIO and therefore he had imaging studies in search of the tumor. Octreotide scan and CT chest were negative. However, CT abdomen revealed an ill-defined heterogeneous soft tissue mass of 4.1cm at the body of pancreas. He underwent pancreatectomy followed by radiotherapy. Pathology report showed undifferentiated carcinoma of the pancreas with osteoclast-like giant cells. His severe knee pain drastically improved and he was able to walk without crutches within a week after removal of the tumor. There was also rapid normalization of abnormal laboratory results within 2 weeks after surgery. However he had cancer recurrence 4 months later and succumbed to pancreatic cancer approximately 7 months after surgery.

Discussion: TIO, or oncogenic osteomalacia, is a rare paraneoplastic syndrome and is characterized by osteomalacia due to hypophosphatemia, renal phosphate wasting and low serum concentration of 1,25-dihydroxy vitamin D occurring in the presence of a tumor. These tumors are usually small, slow-growing and benign and secrete the phosphaturic hormone, fibroblast growth factor 23 (FGF-23). Our patient had the clinical and biochemical hallmarks of TIO. He was also found to have undifferentiated carcinoma of the pancreas with osteoclast-like giant cells, which is the rare tumor with aggressive biologic behavior. Tumor resection resulted in rapid and drastic resolution of symptoms and laboratory abnormalities.

Conclusion: We describe a patient with undifferentiated carcinoma of the pancreas with osteoclast-like giant cells, who presented with typical clinical features and biochemical abnormalities of TIO. After the tumor resection, all these abnormalities resolved drastically. To our knowledge, this is the first case of TIO associated with an aggressive pancreatic cancer in the literature.

Abstract #607

A CASE OF EUGONADAL OSTEOPOROSIS IN A MALE WITH HEREDITARY HEMOCHROMATOSIS

Andrew Brackbill, MD, Patrick Clyde, MD, K. M. Shakir, MD, FRCP, MACP,MACE,FACN

Case Presentation: Hereditary hemochromatosis(HH) decreases bone mineral density(BMD) in several ways, typically via hypogonadism. Roughly one quarter of phenotypically expressed HH patients will have osteoporosis. However, as in this case, some patients with hemochromatosis develop significant low bone mass despite normal serum Testosterone levels. This patient was discovered to have low BMD despite normal serum concentrations of testosterone, follicle-stimulating hormone (FSH) and Luteinizing hormone (LH). A 57-year-old Puerto Rican male with a several year history of hyperlipidemia, erectile dysfunction, and abnormal

liver associated enzymes was found to have worsening liver associated enzymes when his hypolipidemic drug was increased. Tests revealed elevated serum ferritin and iron saturation, consistent with Hemochromatosis. Liver biopsy demonstrated bridging fibrosis; genetic testing confirmed the patient was homozygous for the C282Y mutation, which causes HH. A BMD measurement performed one year prior to diagnosis of hemochromatosis showed osteoporosis with a lowest BMD of 0.631gm/cm2, T-score of -3.4 and a Z-score of -2.7 at the forearm. After diagnosis of HH, repeat BMD measurements revealed a significant decrease in Total Hip BMD of -5.2%. Lowest BMD remained roughly stable, however, at the forearm, with BMD of 0.635gm/cm2, T-score of -3.3 and Z-score of -2.6. The patient's FSH and LH levels were normal (5.6IU/L [nl. 1.4-18.1] and 5.2IU/L [1.5-9.3] respectively). Neither total nor free testosterone levels were low (1106ng/dL [nl. 250-1100] and 81pg/mL [35-155], respectively), consistent with a normal hypothalamic-pituitary-gonadal axis. Sex-hormone binding globulin was increased (94nmol/L [22-77]), accounting for slightly elevated total testosterone. Serum calcium (10.1mg/dL [8.7-10.4]), 25-hydroxy Vitamin D (32ng/mL [>30]), intact PTH (31.6pg/mL [13-75]), estradiol (32pg/mL [0-52]), cortisol (14mcg/dL [>10]), and prolactin levels were normal (4.4ng/mL [2.1-17.7]).

Discussion: HH typically causes low bone mass and osteoporosis via hypogonadism. However, other mechanisms clearly contribute as well. Iron overload itself appears to directly interfere with hydroxyappetite mineralization and with osteoblast function selectively. The majority of HH patients with iron overload, as reflected by elevated ferritin and iron saturation, will have low bone mass.

Conclusion: This case illustrates the importance of obtaining screening ferritin and iron saturation levels even in the absence of hypogonadism.

Abstract #608

PARATHYROIDITIS INDUCED BY THERAPEUTIC RADIOACTIVE IODINE

Erica Kretchman, DO, Courtney Soubliere, DO

Case Presentation: 38 year old female with history of MNG, hashimoto's thyroiditis since age 10, and celiac disease. The patient underwent total thyroidectomy and was diagnosed with stage one papillary thyroid cancer with postive surgical margins. Post-operative course was complicated with temporary hypocalcemia. Prior to her I-131 ablation her iPTH returned to normal. The patient was ablated with 75 mCi, during routine follow-up iPTH was evaluated elevated at 1369 pg/mL, this value was repeated one week later at 613 pg/mL, her calcium levels were 10 and 9 mg/dL respectively . The patient was asymptomatic from a parathyroid standpoint. Throughout one year her parathyroid hormone has remained elevated until resolution 15 months after ablative therapy.

Discussion: Trends of intact parathyroid hormone (pg/mL): 1/12/10 (pre-therapy) - 51.8, 3/3/10 (post-therapy) - 1369, 3/11/10 - 613, 4/13/10 - 289,11/30/10 - 127, 4/22/11 -105.8, 7/29/11 -48.2. Autoimmune reactions to parathyroid cells have been observed in human autoimmune polyendocrinopathy. Most cases are associated with Hashimoto's thyroiditis or other autoimmune conditions (Addison's). Focal lymphocytic infiltration of parathyroid glands has also been found at necropsy in up to 10% of patients assumed to be euparathyroid. Although evidence of parathyroid infiltration has been reported, there were no studies that mentioned parathyroid hormone elevation. When evaluating hyperparathyroidism after radioiodine treatment for Graves' disease, multinodular goiter or papillary thyroid carcinoma, the average latency time to the development of hyperparathyroidism after radioiodine treatment was 13.5 +/- 9.1 years and was found to be inversely correlated with age at radioiodine exposure. A separate study evaluated the parathyroid hormone levels were evaluated during the year post ablation they found serum parathyroid levels were decreased significantly at the 6th month and reached basal levels at the 12th month, and Serum Ca levels decreased below 2.1 mmol/l in several patients without clinical symptoms.

Conclusion: Given the evidence of a normal pre-therapy intact parathyroid hormone and significant elevation after therapy followed by resolution without development of hypercalcemia we have concluded that this unique patients hyperparathyroidism was caused by parathyroidits from exposure to radioactive iodine.

Abstract #609

A CASE OF CARCINOID SYNDROME ASSOCIATED WITH HYPERCALCEMIA.

Fadi Siyam, MBBS, Obai Abdullah, Stephen Brietzke, James Sowers

Case Presentation: A 55 year-old white male known with smoking and GERD was referred for management of severe diarrhea of 1 to 2 years duration interfering with daily activities and sleep along with weight loss of 30 pounds. Previous evaluation included an abdominal CT scan which demonstrated multiple nodular liver densities suggestive of metastases and a colonoscopy which yielded benign hyperplastic polyps. Upon examination, he appeared thin but was afebrile. Blood pressure was 96/66 mmHg with orthostatic tachycardia and mucus membranes were dry. The remainder of the physical examination was

normal. Serum chemistry profile revealed hyponatremia (130 mmol/L), hypokalemia (2.0 mmol/L), pre-renal azotemia (BUN 43 mg/dl, creatinine 1.67 mg/dl), hypophosphatemia (2.1 mg/dl) and calcium elevated at 11.2 mg/dl , with normal albumin and total protein. Initial management consisted of aggressive intravenous hydration with improvement in azotemia and blood pressure, but none in serum calcium. Profuse diarrhea persisted and was evaluated extensively with negative test results for fecal leukocytes, ova and parasites, Clostridium difficile toxins, celiac antibody panel and urinary 5-hydroxyindoleacetic acid. However, a Serum chromogranin A level was elevated (620 ng/ml). Biopsy of liver lesions demonstrated carcinoid tumor staining positively for chromogranin A and synaptophysin. Subsequently, a whole body octreotide scan showed uptake consistent with carcinoid tumor primarily involving the small bowel, with multifocal hepatic metastases. Workup of the persistent hypercalcemia was unrevealing: parathyroid hormone (PTH) was appropriately suppressed (11.1 pg/ml), PTH-related peptide < 2 pmol/L, 25- hydroxy Vitamin D normal at 31 ng/ml and 1,25-dihydroxy Vitamin D normal at 38 pg/ml.Serum and urine protein electrophoreses demonstrated no M-spike. However, the fibroblast growth factor-23 (FGF-23) was borderline elevated at 172 rU/mL (normal <180) .Following diagnosis of metastatic carcinoid tumor, therapeutic trial of octreotide resulted in improvement of diarrhea and normalization of serum calcium.

Discussion: Hypercalcemia has been reported in association with carcinoid tumors in a total of 16 cases since 1966. Putative explanations for the hypercalcemia included production of PTHrp; direct osteolysis by bone metastases; primary hyperparathyroidism as part of a multiple endocrine neoplasia syndrome and elevated calcitonin. None of these explained the hypercalcemia in the currently reported case. However, the FGF-23 was in the high normal range despite a low serum phosphate.

Conclusion: Thus, the most likely explanation of the elevated calcium was the inappropriately high FGF-23.

Abstract #610

PRIMARY HYPERPARATHYROIDISM MASQUERADING AS AMYOTROPHIC LATERAL SCLEROSIS

Petpring Prajuabpansri, MD, Milana Tedford, Robert Weinstein

Objective: The neuromuscular symptoms of hyperparathyroidism usually include non-specific weakness, fatigue, listlessness, and trouble concentrating. However, in some instances, the symptoms may be profound and suggest a primary neurological disorder such as amyotrophic lateral sclerosis. We report a case of hypercalcemia with elevated parathyroid hormone in a 70-year-old man who presented with neuromuscular signs and symptoms mimicking primary motor neuron disease. Following parathyroid surgery, there was remarkable recovery of the neuromuscular symptoms.

Methods: A 70-year-old Caucasian man with a history of a kidney stone 40 years ago presented with symmetrical proximal muscle weakness, atrophy, and foot drop resulting in frequent falls. He reported difficulty in speaking, polyuria, fatigue, anorexia, depression, and a 40-pound weight loss over the past few years. Examination revealed significant upper and lower limb proximal muscle atrophy. Muscle power was grade 3 in his proximal limbs and he failed to rise from a squat. His speech was slurred. The sensory examination was unremarkable. Reflexes returned normally in the upper and lower extremities but the Babinski sign was positive bilaterally. Magnetic resonance imaging of the brain and cervical spine showed changes appropriate for his age and mild cervical degenerative disease. The electromyogram and nerve conduction velocity were normal. Muscle biopsy showed neurogenic changes. Bone density at the radial diaphysis, distal radius, and total proximal femur was far below the normal peak adult range but at the lumbar spine, the density was normal. The serum calcium level was 11.1 mg/dL (normal is 8.6-10.2), parathyroid hormone was 344 pg/mL (normal is 12-88), and alkaline phosphatase was 118 IU/L (normal is 32-91). Neck ultrasound showed a right inferior neck mass measuring 2.3x2.5x2.7 cm and scintigraphic findings were consistent with a right inferior parathyroid adenoma.

Case Presentation: At neck exploration, the right inferior parathyroid gland was 4x3x2 cm and weighed 12.08 grams. Pathology revealed a chief cell adenoma. Shortly after recovery on the day of surgery, there were improvements in the neuromuscular symptoms and his speech was no longer slurred.

Discussion: Hyperparathyroidism can present with severe neuromuscular disease similar to that seen in patients with amyotrophic lateral sclerosis and recovery may be swift after parathyroid surgery.

Conclusion: It is important to rule out hyperparathyroidism as a cause of motor neuron disease.

Abstract #611

FRAX AS A PREDICTOR FOR OSTEOPOROSIS

Ramesh Gadam, MD, Kenneth Izuora, MD

Objective: To compare the prediction of 10 year fracture risk using the FRAX tool with and without the bone mineral density (BMD).

Methods: This is an ongoing cross-sectional study of post-menopausal women and men>50 years of age getting DXA scans as a part of their routine medical care. Included subjects have never been diagnosed or treated for osteoporosis. Following informed consent, a questionnaire on epidemiologic risk factors for fracture i.e. previous fracture, hip fracture in parents, current smoking, corticosteroid use, rheumatoid arthritis, secondary risks for osteoporosis, alcohol consumption is administered. Height and weight is measured by the investigator using medical scales. The FRAX score with and without BMD is calculated using the FRAX online tool and compared.
Results: Based on our power calculations, we plan to recruit 300 subjects for this study. Preliminary data from 67 female participants recruited to date, shows that 58 subjects (86.6%) had similar FRAX recommendations with or without the BMD. Eight subjects who did not meet treatment criteria with inclusion of BMD into the FRAX calculation were categorized as requiring treatment by the FRAX tool without their BMD and one subject that met treatment criteria with inclusion of their BMD in FRAX was excluded without the BMD.
Discussion: The FRAX tool uses epidemiologic risk factors with or without femoral neck BMD to estimate the 10-year probability of hip and other major osteoporotic fractures for patients with osteopenia. Our study aims to further explore the concept that the FRAX tool without the BMD is an accurate predictor of fracture risk in patients being evaluated for osteoporosis. Our findings so far suggest that for most subjects, the FRAX predictions with and without the BMD were similar. However, it is too early to draw conclusions at this point in the study.
Conclusion: 1. In most cases FRAX scores have similar prediction with and without the BMD. 2. For certain populations the FRAX tool alone may predict the ten year fracture risk accurately and may be more cost effective by reducing the frequency of BMD measurement with DXA scans.

Abstract #612

PARATHYROID CARCINOMA PRESENTING AS A GIANT CELL TUMOR

Taral Shah, MD, Sara Lubitz, MD, Stephen Schneider, Xiangbing Wang, MD, PhD, Louis Amorosa

Objective: Recognizing that bone giant cell tumors may be a sign of severe hyperparathyroidism, including carcinoma, may preclude bone surgery.
Case Presentation: A 36 year old white male noticed a right elbow mass. He was operated on at a referral center and was thought to have a giant cell tumor. A serum calcium value of 13.9 mg/dL on pre-admission testing was overlooked. Three months later, the patient's dentist noticed bony lesions suspicious of malignancy on X-ray. The patient had PET scan that showed two thyroid masses, confirmed on thyroid ultrasound (US), 4 x 2.4 x 2.3 cm on left and the 1.6 x 1.1 x 1.1 cm on right. Fine needle aspiration (FNA) biopsies were suspicious for follicular neoplasms, hurthle cell type on the left and neoplasm of undetermined significance on the right. Testing showed a serum calcium level of > 15 mg/dl, 24 hours urine calcium of 1265 mg/24 hours, phosphorus level of 2.3 mg/dL, and parathyroid hormone (PTH) level of 1139 pg/ml. The patient's otherwise benign physical exam was remarkable for nodular thyroid. A sestamibi scan clarified that the thyroid mass seen on the left by thyroid US was actually consistent with parathyroid adenoma. Hand X-ray showed sub-periosteal resorption. The patient had resections of the left parathyroid mass and right thyroid lobe. PTH dropped from 1318 to 88 pg/ml and calcium dropped from 12.2 to 10 mg/dl in less than 2 hours after surgery. Pathology result showed a well-circumscribed, 11 gm, 3.5 x 3 x 1.6 cm parathyroid carcinoma with foci of lymphovascular invasion and possible capsular invasion. Right thyroid lobectomy revealed a nodular goiter.
Discussion: Brown tumors and other signs of parathyroid bone disorder are now uncommonly found because of earlier diagnosis of hyperparathyroidism in generally asymptomatic patients. Brown tumors are seen in cortical bones. Patients with hyperparathyroidism may have giant cell granulomas, loss of lamina dura, and demineralization of the jaws. It is difficult to distinguish brown tumor from other giant cell lesions, histopathologically. Therefore, a clinical diagnosis of brown tumor is made based on the association with hyperparathyroidism. Furthermore, excising a brown tumor may not be necessary as the lesion may improve as hyperparathyroidism resolves.
Conclusion: This case represents classical findings consistent with parathyroid carcinoma: sub-periosteal resorption, brown tumor likely presenting as a giant cell tumor, markedly elevated levels of calcium and PTH, and cytology misdiagnosed as thyroid neoplasm. Finally, calcium level should be checked in all cases of giant cell tumor to rule out brown tumor.

Abstract #613

CHANGING SESTAMIBI AGENTS AND CONVERSION OF PARATHYROID SCAN OVER TIME IN DUAL AGENT PROTOCOL - ONE OF FEW CASE REPORTS IN LITERATURE

Subramanian Kannan, MD, Donald Neumann, Kresimira Milas, Angelo Licata, MD, PhD, FACP

Case Presentation: Technetium Parathyroid Scintigraphy (TPS) is the most popular noninvasive localization

procedure in patients with primary hyperparathyroidism (PHPT) because it can accurately localize 80% to 90% of the single adenomas. False negative scans are found in about 20-30% of cases. Reports of conversion of a negative scan to a positive are sparse and have occurred in dual phase TPS. This case highlights the conversion of the TPS from negative to positive in a dual agent subtraction TPS. Case: A 56 year postmenopausal (for 4y) Caucasian female was evaluated for elevated PTH [72 pg/ml (10-60)] and osteopenia (BMD L-spine T-score -1.5; Hip -1.3). She had a medical history of migraine and acid reflux but no renal stones, hypertension, or constipation. She used hormone replacement therapy for about 3 years. There was no family history of osteoporosis. Her daily calcium intake included 3-4 servings of dairy products a day, about 1000 mg of elemental calcium (Calcium Carbonate) and vitamin D 400-800 IU/day from MVI. Her serum calcium was 10.4 mg/dl (8.5-10.5), Phosphorus 3.6 mg/dl (2.5-4.5), 25-hydroxy vitamin D 37.6 ng/ml(31-80),1,25 vitamin D 68.7 pg/ml (25.1-66.1), urine N-telopeptide 70.1 nmolBCE/mmol Cr (14.4-75) and 24 hour urine calcium 263 mg. A TPS using Tc99m-Tetrofosmin (TETRO) showed no definite areas of abnormal parathyroid tissue. Patient refused surgical exploration, was started on Risedronate 35mg/week and subsequently monitored. In 2010, she suffered a Colles fracture of right wrist and then agreed to have surgery. A repeat TPS using Tc-99m Sestamibi (SES) revealed hypervascular parathyroid lesion in the right lower neck. She underwent successful removal of a right lower parathyroid adenoma which revealed predominantly clear cells and pockets of Oxyphil cells. ***Discussion***: Initial false-negative result scans may be related to technical differences in Technetium agents used or biological factors such as adenoma size, content of Oxyphilic cells, vascular perfusion, cell cycle phases, serum calcium levels, P-glycoprotein or multidrug resistance associated protein expression. We presume the conversion of TPS in our case may be related to difference in the Technetium agent used. Previous case reports of such conversion used TETRO in dual phase protocol. Conversion of TPS in dual agent subtraction protocol as in our case has not been reported before. ***Conclusion***: Multiple technical and biological factors may be involved in a conversion of negative TPS to positive. The need to repeat an initially negative TPS scan in patient followed up for PHPT has to be evaluated prospectively.

Abstract #614

CHRONIC DISABILITIES MARKEDLY INCREASE BONE LOSS AND FRACTURES

Sunil Wimalawansa, MD, PhD, MBA,FRCP,FACP, Patricia Graham

Objective: Adults and children with disabilities are at high risk for falls & fractures in comparison to non-disabled peers. In 2006, 16% of Americans reported disabilities and the rate is increasing. We hypothesize that Persons with Disability (PWD) are under-diagnosed & under-treated for osteoporosis, and not included in clinical research.
Methods: Informational literature review on pediatric and adult PWD with reference to BMD, falls, fractures & utilization of EMR.
Case Presentation: PWDs are grossly under-represented in bone research, frequently not evaluated for low BMD, and have low serum vitamin D levels, and increase fractures relative to non non-disabled peers. Disability & immobility are strong independent predictors of low BMD and fractures. Those with spina bifida, SCI, CP, MS, CVA, amputations, COPD & RA, and all patients with developmental disabilities have high incidence of low BMD. In addition, patients with childhood onset brain disorders & adults with developmental disabilities have lower BMD & low serum vitamin D levels (prevalence of 70 to 80%) and high fracture rates (2.7%) (local and NJ-DHSS/NJCHS data).
Discussion: While falls and injuries are common, fractures are a common cause of hospitalization in PWD, and occurs at a younger age. Even though risk factors for falls are common in PWD, they are rarely included in preventive care models. Unlike the FRAX, Garvan nomogram includes fall risk with 10-year hip fracture risk assessment and medication guidance. It is likely that, correction of calcium and vitamin D deficiency and perhaps, early medication use may reduce BMD loss and fractures in PWD. Optimized EMR can capture patients with low BMD and with high falls risks. Identification of such a sub-group would allow health care professionals to give special attention, screening, and treat PDWs promptly, leading to improve health, decrease fractures, and decrease health care cost.
Conclusion: More research is needed to define the etiology of low BMD and fracture risks in PWD. With FRAX or Garvan tool, one could establish uniform best practice parameters including safe exercise regimens, cost-effective screening, & early initiation of therapy. Such should be incorporated into rehabilitation strategies. Focus is to minimize injuries, eliminate disparity in health care, early screening for falls & low BMD, manage pain and disability, provide safe weight-bearing exercise strategies, home safety and psychosocial support for PWDs. The use of EHR system facilitates targeting PWD for preventative care.

Abstract #615

DENOSUMAB INDUCED SEVERE HYPOCALCEMIA IN A CANCER PATIENT: MANAGEMENT OPTIONS

Simona Ioja, MD, Victor Ciofoaia, MD, Rahim Rahimyar, Nancy Rennert, MD

Objective: To describe the mechanism and management of severe hypocalcemia associated with the RANK ligand inhibitor denosumab (Xgeva®/ Prolia®).
Case Presentation: A 75 year old male with metastatic prostate cancer developed generalized weakness and facial cellulitis three weeks after the first dose of denosumab. Comorbidities included stage IV chronic kidney disease, type 2 diabetes mellitus and seizure disorder controlled with phenytoin and carbamazepine. Prior to treatment with denosumab, serum calcium (Ca) was 8.8mg/dl, albumin 3.6 mg/dl and vitamin (Vit) D was not measured. Three weeks later, Ca level was 3.9mg/dl - corrected 4.4 (nl 8.4-10.7mg/dl), magnesium 1.7 mg/dl (nl 1.8 -2 .4, phosphorus 2.3 mg/dl (nl 2.7-4.5mg/dl), PTH 764pg/ml (nl 15-65 pg/ml) and QTc was prolonged on EKG (570ms). Ionized Ca was sent and 25-HO and 1.25 HO- Vit D levels were very low at 7.5 ng/ml and <8pg/ml respectively. Exam was remarkable for the absence of tetany or positive Chvostek 's/Trousseau's signs. Treatment with Ca and Vit D (including iv Ca drip) and the patient's gradual response will be detailed.
Discussion: Denosumab is a fully human monoclonal antibody against the osteoblast receptor activator of nuclear factor KB (RANK) ligand. It inhibits the interaction between RANK (osteoclast marker) and RANK ligand, with subsequent loss of osteoclast differentiation, activation and survival and promotion of bone formation. In different concentrations, Denosumab is FDA approved for treatment of osteoporosis (Prolia) in women with high risk of fracture and for prevention of skeletal-related events in patients with bone metastasis from prostate and breast cancer (Xgeva) as well as to increase bone mass in patients on androgen deprivation therapy in non-metastatic prostate cancer or adjuvant aromatase inhibitor therapy for breast cancer. Severe hypocalcemia (< 7mg/dl) has been reported in 3 - 5.5% of patients on denosumab and is more frequent in those with risk factors (Cr Cl < 30 ml/min, low Vit D and/or Ca levels prior to treatment). Vit D and Ca supplementation and monitoring are recommended prior to and during denosumab use. There is however, little guidance regarding management of denosumab induced severe hypocalcemia. Interestingly, our patient presented with very few clinical manifestations of hypocalcemia, possibly due to a protective effect of antiepileptics.
Conclusion: It is important to understand the risks and management of denosumab induced severe hypocalcemia, and other life-threatening complications, such as infections, as use of this drug will likely increase for both treatment of osteoporosis and bone metastases/fracture prevention.

Abstract #616

A NOVEL ROBOTIC TECHNIQUE OF TRANANSAXILLARY GASLESS PARATHYROIDECTOMY FOR THE SURGICAL MANAGEMENT OF PRIMARY HYPERPARATHYROIDISM (PHPT) DUE TO PARATHYROID ADENOMA

Shamsa Ali, MBBS, Salem Noureldine, Nicholas Lewing, Nicholas Avitabile, MD, Emad Kandil, MD

Case Presentation: Nine patients with confirmed PHPT underwent robotic parathyroidectomy for a parathyroid adenoma. Mean (±SD) age was 37.5 ±8.1 years. Curative resection was established in all nine patients with the aid of intraoperative monitoring of serum intact PTH levels. One patient required conversion to the cervical approach for bilateral exploration of multi-glandular disease. The mean (SD) total operative time was 119 (15.6) minutes; and mean console operative time was 33 (11.6) minutes. Mean blood loss (±SD) was 21 ±19.9 mL. There were no perioperative or postoperative complications. In addition there was no evidence of vocal cord palsy on postoperative laryngoscopy. Two patients were discharged within four hours after the procedure and the remaining patients were observed under a 23-hour status
Discussion: The cosmetic results were considered excellent due to the hidden anatomic location of the incision site. All patients were followed up for a period exceeding 6 months without any evidence of persistent or recurrent hyperparathyroidism. Robotic parathyroidectomy is feasible, safe and effective for the treatment of PHPT .
Conclusion: We believe that the use of robotic technology for endoscopic parathyroid surgeries could overcome the limitations of conventional endoscopic techniques in the surgical management of parathyroid lesions

Abstract #617

USE OF RISEDRONATE FOR PREVENTION OF BONE LOSS AFTER LUNG TRANSPLANTATION

Adrienne Barnosky, DO, Charles Alex, Barbara Sexton, Pauline Camacho, MD

Objective: Osteoporosis is a known sequela of lung transplantation. Immunosuppressive therapy and preexisting

low bone mass are factors that lead to decline in bone mass after transplantation. One study of osteoporosis after lung transplantation found bone loss to be most significant within the first 6 months post-transplant. Only one randomized, placebo-controlled, prospective study has shown efficacy in the prevention of transplant-induced bone loss after lung transplantation using bisphosphonates. Risedronate is a potent anti-resorptive agent proven to be effective in the prevention and treatment of glucocorticoid-induced osteoporosis. The aim of this study was to determine whether risedronate with calcium and vitamin D would lead to a reduction in bone loss compared to calcium and vitamin D alone in individuals after lung transplantation.

Methods: We conducted a small randomized, double-blind, placebo-controlled trial from 2002 to 2011 comparing bone mineral density (BMD) changes one year after lung transplantation. Sixteen patients who underwent lung transplantation (9 men, 7 women) at Loyola University Medical Center were evaluated at months 0, 1, 3, 6, 9, 12 after lung transplantation. All patients were given calcium, vitamin D, and either risedronate or placebo post-transplant. Bone densitometry was performed at baseline, 6, and 12 months after transplant and BMD between the risedronate group and the placebo group were analyzed.

Results: Out of 16 lung transplant recipients given calcium, vitamin D, and either risedronate or placebo, the lumbar spine BMD was not statistically significant at baseline ($p = 0.19$), 6 months, ($p = 0.28$), or 12 months ($p = 0.28$) between the 2 groups. The total hip BMD was also not statistically significant at baseline ($p = 0.21$), 6 months ($p = 0.29$), or 12 months ($p = 0.50$). Bone turnover markers (osteocalcin and urinary N-telopeptide) were obtained and showed dramatic lowering in bone formation after transplant.

Discussion: Our findings demonstrate that despite early intervention with calcium, vitamin D, and risedronate, a difference in BMD was not observed within the first 12 months after lung transplant when compared with controls. The lack of benefit observed in those patients treated with a bisphosphonate may have been secondary to small sample size, a follow-up duration of only 1 year, or the possibility that one year post-transplant, aggressive calcium and vitamin D supplementation provides enough protection that a loss in BMD is not observed.

Conclusion: Early intervention with calcium, vitamin D, and either bisphosphonate therapy or placebo during the first year after lung transplantation failed to show a difference in BMD when compared with controls.

Abstract #618

WHAT IS THE BEST VITAMIN D FORMULATION FOR THE TREATMENT OF HYPOPARATHYROIDISM: VITAMIN D PRECURSORS OR ACTIVE VITAMIN D PREPARATIONS?

Saleh Aldasouqi, MD, Bhavini Bhavsar, MBBS, M.D, Ala Elayyan, Sameer Ansar, MD, Deepthi Rao, MD, Shaza Khan, MD

Objective: Clinicians have different protocols for treating surgically-induced hypoparathyroidism (HPOPTH). Some use high doses of vitamin D2 or D3 while others use active metabolites, e.g., calcitriol, along with calcium supplementation. We discuss a case of HPOPTH treated with Vitamin D precursors with refractory hypocalcemia and large calcium supplementation that was conveniently managed with calcitriol.

Case Presentation: A 40-year old woman underwent thyroidectomy for a large obstructive goiter, and sustained permanent HPOPTH, with refractory hypocalcemia, with several visits to the ER. Symptomatic and severe hypocalcemia, as low as 5.5 mg/dl, required multiple intravenous calcium treatments. She was on an average total of 32 tablets of calcium daily (in the form of standard over the counter Tums and Calcium Citrate with D). In addition she was on vitamin D3, averaging 7000-9000 units daily. She had perioral numbness and positive Chvostek's sign. She reported 100 % compliance, but acknowledged the difficulty of swallowing the calcium tablets. Her calcium has rarely reached or exceeded 7.0 mg/dl over the prior 3 years. She was started on calcitriol, and within 6 weeks, her calcium was normalized, using 0.25 mcg of calcitriol. She was able to reduce her calcium requirements from 32 to only 2 tablets of calcium with vitamin D daily.

Discussion: Management guidelines are not clear about which vitamin D preparation is preferable in the treatment of HPOPTH. Our own observation has been that for many patients, achieving normocalcemia with D2 or D3 is more difficult, than with calcitriol, and is only achievable, with high doses of D2 or D3, and in some patients, at the expense of ingesting a large number of calcium tablets, which are difficult to swallow, posing a compliance challenge. Unlike vitamin D deficiency, the absence of parathyroid hormone (PTH) in HPOPTH results in ineffective renal activation of vitamin D, ineffective phosphorus excretion, as well as ineffective calcium mobilization from bones. If vitamin D precursors are used (D2, or D3), this requires very high doses, as high as 50,000 units daily. This high dose, and the long-duration of action of stored vitamin D would pose the risk of hypercalcemia, and calcinosis,

especially during illness. Our patient had difficulty in complying with the high calcium supplementation. She was able to reduce her requirement from over 30 tablets to 2 tablets of calcium, with just 0.25 mcg of calcitriol. We continued small dose of vitamin D to keep her serum levels in the physiologic range.
Conclusion: This case illustrates the inconvenience and difficulty of achieving normocalcemia with vitamin D2 or D3. We recommend active vitamin D preparations instead.

Abstract #619

HYPOCALCEMIA SECONDARY TO ZOLEDRONATE THERAPY IN A PATIENT WITH VITAMIN D DEFICIENCY

Narendranath Epperla, MD, Ram Pathak, MD

Objective: To present a case showing that hypocalcemia is common with Zoledronate (ZDA) therapy especially in the presence of inadequate vitamin D.
Case Presentation: A 54-year-old male undergoing chemotherapy for metastatic small cell lung cancer was started on ZDA for bony skeletal metastasis. Six days later, he presented with weakness, vomiting, and hand and foot numbness. He had not taken the recommended calcium and vitamin D supplements. His total serum calcium (Ca2+; 10.3mg/dl before ZDA therapy) had decreased to 7.7mg/dl. Ionized Ca2+ was 3.9mg/dl, creatinine 1.1mg/dl, 25-OH vitamin D 24 ng/ml and PTH was elevated at 104pg/dl. He received calcitriol (0.25mcg) and IV calcium gluconate. He was advised to continue the calcitriol and also take calcium (1200mg) and vitamin D (800U) twice daily. Seven days later, his symptoms had improved and his PTH levels normalized to 36pg/ml. The calcitriol was discontinued, but the calcium and vitamin D were continued. Two weeks later, he was asymptomatic and his total serum Ca2+ was normal (9.4mg/dl).
Discussion: In healthy individuals, osteoclast-mediated bone resorption constitutes a major defense against hypocalcemia. However, in medically-compromised patients, this mechanism may be disrupted, leading to hypocalcemia. Hypoparathyroidism from radiation or metastases to parathyroid glands have been reported to cause hypocalcemia. Potent bisphosphonates such as ZDA can also cause hypocalcemia. Zoledronate inhibits farnesyl pyrophosphate synthase, a key enzyme in the mevalonate pathway, leading to decreased production of isoprenoid lipids. This disruption leads to decreased osteoclast activity and increased cell death, which results in decreased elution of Ca2+ in the blood. Regardless of Ca2+ levels before administration, ZDA can decrease blood Ca2+. However, normal Ca2+ levels are rapidly restored provided factors involved in Ca2+ homeostasis are intact. Hypocalcemia during bisphosphonate therapy is mostly asymptomatic, mild and transient, and has been attributed to preexisting vitamin D deficiency, renal failure, hypoparathyroidism, or concurrent therapy with aminoglycosides. In studies involving the use of ZDA in patients with metastatic cancers, the incidence of severe hypocalcemia ranged from 0-2%. Our patient was not on aminoglycosides, his serum creatinine was normal, and PTH was elevated. Therefore, the hypocalcemia was likely contributed to by vitamin D insufficiency.
Conclusion: Vitamin D insuffiency/deficiency afflicts a large proportion of the elderly population. Its existence needs to be recognized before the start of bisphosphonate therapy, so that adequate calcium and vitamin D supplementation can be given to reduce the occurrence of hypocalcemia.

Abstract #620

VERY HIGH PREVALENCE OF VITAMIN D DEFICIENCY AMONG SAUDI FEMALE ADOLESCENTS

Riyad Sulimani, MBBS, Ashry Gad, Assim Alfadda, Suliman Alshehri, Abdulaziz Alothaman

Objective: To study the prevalence of vitamin D deficiency/insufficiency among Saudi adolescent females in intermediate and secondary (high) schools in Riyadh, Saudi Arabia.
Methods: This is a prospective cross sectional study in which adolescent Saudi females living in Riyadh, Saudi Arabia -latitude 24 39 North- were tested for the prevalence of vitamin D deficiency.Basic demographic data of the girls including their exposure to direct sunlight, drug history and life style was obtained. Fasting serum 25 (OH) vitamin D , calcium, alkaline phosphatase, phosphorous and parathyroid hormone levels were obtained during the winter months (December, January and February) and in summer (May and June). Vitamin D deficiency was defined as levels below 50 nmol/l while vitamin D insufficiency was defined as levels between 50- 75 nmol/l.
Results: A total of 1199 girls were studied. Their mean age was 16.3± 1.7 years.The majority were minimally exposed to sun (32.2% with no exposure and 35% with exposure for 1-10 minutes daily). Consumption of milk and dairy products was inadequate with mean calcium intake of 283.0±212.3 mg /day. 64.3% of the girls had sedentary life style. Vitamin D deficiency was present in 98.9% of the girls during winter increasing to 99.2% in summer. Vitamin D insufficiency was present in 1.1% of girls in winter and 0.8% in summer (p=1.00). Osteomalacia (defined biochemically by elevated alkaline phosphatase, raised parathyroid hormone levels with low 25 vitamin D

levels) was present in 14.5 % of the students.

Discussion: The results of the study document a very high (endemic) prevalence of vitamin D deficiency/ insufficiency among Saudi female adolescent students. The main risk factors appear to be related to minimal direct sun exposure and a low consumption of vitamin D.These results confirm previous reports also obtained from this part of the world. They also support global observations about the emerging high prevalence of vitamin D deficiency in many parts of the world including sun rich countries. The high prevalence of vitamin D deficiency was responsible for the relatively high frequency of metabolic bone disease in the form of osteomalacia among young females. Suggested causes include a lack of sun exposure due to hot climate and poor vitamin D intake.

Conclusion: Severe vitamin D deficiency is highly prevalent among Saudi adolescent females. Measures to overcome this problem are urgently needed. Fortification of dairy products and other food items should be encouraged. Adequate exposure to direct sunlight at homes and schools is also important.

Abstract #621

NORMAL INTACT PARATHYROID HORMONE LEVEL AS A CLUE TO CONCURRENT HYPERCALCEMIA OF MALIGNANCY IN A PATIENT WITH PRIMARY HYPERPARATHYROIDISM

Marc Laufgraben, MD, MBA, FACE, FACP, Batool Razvi

Objective: Although most patients with hypercalcemia will have a single etiology—most commonly primary hyperparathyroidism (PHPT) or hypercalcemia of malignancy--rare patients may have several concurrent causes. We present the case of an elderly woman with PHPT who developed hypercalcemia of malignancy.

Case Presentation: An 81 year-old female presented with lethargy and poor oral intake. Six months earlier, she presented with calcium (Ca) 12.2 (nl 8.6-10.2 mg/dL), phosphorous 2.3 (2.7-4.6 mg/dl) and creatinine 1.15 (nl 0.60-1.20 mg/dl). She was treated with pamidronate. Further evaluation showed intact parathyroid hormone (iPTH) 55 (nl 18-65 pg/mL), with Ca 10.5, PTH-related peptide (PTH-rp) 12 (nl 14-27 pg/mL) and 25-OH Vitamin D 11 (nl 30-100 ng/ml). Three months later, she was readmitted with Ca 12.6. Repeat iPTH was normal at 53 (with Ca 11.5); 25-OH Vitamin D was now normal. A thiazide diuretic was stopped. She was not a candidate for parathyroid surgery, and cinacalcet was initiated. On the present admission, Ca was 14.0 and iPTH 46. PTH-rp was rechecked and elevated to 34. She was treated with intravenous fluids, calcitonin, and pamidronate with improvement in serum calcium to 10.2. The patient was found to have metastatic lesions of an unknown primary malignancy in the lungs, liver, and pancreas. She was discharged to hospice care.

Discussion: Although patients with hypercalcemia due to PHPT classically present with elevated iPTH, "inappropriately normal" iPTH levels are seen in one-third of patients with PHPT in some series. Patients with parathyroid neoplasms or hyperplasia are felt to exhibit impaired suppression of iPTH secretion by calcium; the "set point" for suppression has been shifted to the right and higher levels of calcium are needed to decrease PTH secretion. This phenomenon may also result in incomplete suppression of iPTH when a concurrent cause of hypercalcemia supervenes in a patient with PHPT. In rare cases, PHPT has been described concurrently with sarcoidosis, with multiple myeloma, with chronic lymphocytic leukemia, and with metastatic carcinoma.

Conclusion: Although hypercalcemia with normal iPTH may be due solely to PHPT, we recommend further evaluation in such patients to exclude multiple concurrent causes of hypercalcemia. (As well, familial hypocalciuric hypercalcemia should be excluded in all patients with suspected PHPT.) This case also reinforces the importance of ongoing reevaluation of hypercalcemic patients who follow an unexpected course.

Abstract #622

SEVERE CARBOPLATIN-INDUCED HYPOMAGNESEMIC HYPOCALCEMIA IN A PATIENT WITH ACTH-SECRETING NEUROENDOCRINE TUMOR

Marc Laufgraben, MD, MBA, FACE, FACP, Hilary Whitlatch

Objective: Although hypocalcemia is a well-described side effect of cisplatin, there are few reports of severe hypocalcemia with carboplatin. We present the case of a 52-year-old woman with metastatic ACTH-secreting neuroendocrine tumor (NET) who developed severe hypocalcemia following carboplatin infusion.

Case Presentation: A 52-year-old woman with Cushing's syndrome due to metastatic ACTH-secreting NET presented with hypocalcemia. She was receiving treatment with metyrapone, dexamethasone, ketoconazole, and Octreotide LAR, and had begun chemotherapy with carboplatin and etoposide. Prior to Cycle 1, she had calcium (Ca) 8.6 (nl 8.5-10.5 mg/dl) and phosphorous (Phos) 5.6 (nl 2.7-4.8 mg/dl). Prior to administration of Cycle 2, her Ca was noted to be 6.5 with albumin 3.3. She was continued on Ca carbonate 600 mg/Vitamin D 400 IU three times a day and received Cycle 2 per protocol.

Lab tests nine days later revealed Ca 6.7 and albumin 3.5. She had minor abdominal cramping and was referred to the emergency department (ED). Her EKG was normal. She received two grams of IV Ca gluconate and was discharged. Eight days later, she was noted to have Ca 6.2 and albumin 3.6. She was referred back to the ED where her Ca was 6.3 with a magnesium (Mg) 0.6 (normal 1.3 -1.9mEq/l), Phos 7.0, and creatinine 1.1. She received IV Mg sulfate and IV Ca gluconate and was transferred to a tertiary care hospital. There, she denied muscle cramping or perioral paresthesias. She had chronic distal extremity paresthesias attributed to prior chemotherapy. Chvostek's and Trousseau's signs were negative. She had intact PTH 81 (nl 10-65 pg/ml) and 25-hydroxy Vitamin D 29 (nl 30-100 ng/ml). She received periodic IV boluses of Ca gluconate and Mg sulfate. By the time of discharge, Ca was 9.2, albumin 3.5, Phos 4.3 and Mg 1.5. She was discharged on Ca carbonate 1000 mg, vitamin D 400, and Mg oxide 800 mg —all PO TID. Her subsequent chemotherapy cycles included IV Mg and Ca repletion.

Discussion: Hypomagnesemic hypocalcemia, described frequently with cisplatin, is an unusual side effect of carboplatin. Cisplatin-treated patients are believed to develop hypomagnesemia due to renal Mg wasting from tubular dysfunction.. We hypothesize that the reduced nephrotoxicity of carboplatin results in less renal tubular damage and less Mg wasting. Hypomagnesemia causes hypocalcemia through both impairment of PTH secretion and induction of PTH resistance. Repletion of Mg restores normal PTH secretion and action.

Conclusion: Practitioners should be aware of the potential for severe hypomagnesemic hypocalcemia in patients treated with carboplatin.

Abstract #623

LIGHT CHAINS THAT BREAK BONES

Paulina Cruz, MD, Tahira Yasmeen, MD, Niharika Singh, MD, Farah Hasan, MD

Objective: Osteoporosis is a disease characterized by low bone mass predisposing the individual to fractures. In the US, it is estimated that 9.1 million women and 2.8 million men have osteoporosis, which translates to about $17 billion in direct medical costs. Significant gaps exist between what is known and its application in the community.

Case Presentation: A 63 year-old white female presented to the Emergency Department with a two-month history of back pain and a 1-day history of right side chest pain. Chest pain was pleuritic, associated with dyspnea. A vertebral compression fracture was diagnosed 2 months prior for which she received analgesics and ibandronate; the latter was discontinued after 6 weeks due to dyspepsia. She had menopause at age 50 and a 50 pack-year history of smoking. Her medications included calcium, vitamin D and phenytoin. On physical examination, she was in no acute distress, vitals signs were stable, respirations were 20. An incidental 3-cm thyroid nodule was found in the right lobe. Strength in the lower extremities was 4/5, sensation was intact. Back exam did not reveal focal tenderness but range of motion was extremely limited due to pain. Rest of examination was unremarkable. Relevant laboratory and imaging findings were: normal CBC, TSH 6.5, T4 1 ng/dl, calcium 9.8 mg/dl, globulin 3.2 g/dl, 25(OH)D 36.5 ng/ml, PTH 5 pg/ml, urine electrophoresis with immunofixation detected free monoclonal kappa light chains. CT of chest revealed multiple pulmonary emboli in the right middle and lower lobes. CT of lumbar spine showed progression of compression fractures at L1, L2 and L4. US Duplex of lower extremities was negative for DVT. DEXA scan showed a T-score of -2.9. Patient underwent kyphoplasty with vertebral body biopsies that confirmed the diagnosis of multiple myeloma.

Discussion: Many primary care physicians do not evaluate patients for secondary causes of osteoporosis. At the very least a 25(OH)D level, PTH, chemistries, CBC and TSH should be done on patients with low T scores. Unexplained or refractory vitamin D deficiency should prompt the evaluation for celiac disease even in the absence of GI symptoms. Attention should be given to the patients pharmacological history as there are well recognized and commonly used drugs that can cause secondary osteoporosis such as glucocorticoids, anticonvulsants and long-term heparin. Our patient had been started on therapy for osteoporosis without any workup for secondary causes prior to admission.

Conclusion: It is imperative to recognize risk factors for osteoporosis along with high-risk individuals to avoid delays in diagnosis and possibly prevent associated morbidity.

Abstract #624

RELATION OF VITAMIN D LEVELS WITH BONE MINERAL DENSITY AND PARATHYROID HORMONE IN ADULTS WITH LOW BONE DENSITY

Sunil Kota, MD, Siva Kota, Svs Krishna, Kirtikumar Modi, Lalit Meher

Objective: To investigate the relationship among serum 25-hydroxyvi¬tamin D [25(OH)D] levels, parathyroid hormone (iPTH) levels, and bone mineral density (BMD).

Methods: Adults with or without prevalent fragility fractures and with low BMD at the total hip or lumbar

spine underwent assessment. Multivariate regression models were used to investigate the relationships among serum 25(OH)D, iPTH, and BMD.

Results: 102 patients (M: F= 38: 64) with mean age of 62.5 ± 6.4 years were inducted into the study. 44 patients had osteopenia and osteoporosis was present in 58 patients. The mean values for serum 25(OH)D and IPTH levels were 21.3 ± 0.5 ng/ ml and 53.1 ± 22.3 pg/ ml respectively. In 84.3% of patients, serum 25(OH)D levels were below 30 ng/mL. There was no association between 25(OH) D levels and BMD at the total hip, or lumbar spine (P = .473, and .353, respectively). Serum iPTH levels were negatively associated with BMD at the total hip (P = .019) and the lumbar spine (P = .02). Both at the total hip and lumbar spine, iPTH levels, male sex, BMI, and age were found to be significant predictors of BMD. Patients with higher BMI had significantly lower BMD and T score. At levels <30 ng/mL, 25(OH)D was negatively associated with iPTH (P = .041).

Discussion: Our study has reaffirmed the previous studies citing high prevalence of vitamin D deficiency. Vitamin D deficiency is a risk factor for osteoporosis. Some studies have reported a positive correlation between 25(OH) D levels and BMD at all sites measured, others finding a correlation at the femoral neck but not at other sites. When patients with osteoporosis were included, a positive correlation between 25(OH)D levels and BMD at the femoral neck but not at the lumbar spine was found in one study; another study found an association between serum 25(OH)D and BMD at the trochanter only—and only in patients with a serum 25(OH)D concentration below 10 ng/mL. Similar study from south east Asia including patients with low BMD revealed no association between 25(OH)D levels and BMD at the femoral neck, total hip, or lumbar spine.

Conclusion: Among our cohort of patients with low BMD, no direct relationship between serum 25(OH)D levels and BMD at total hip and lumbar spine was observed. A negative correlation existed between iPTH and 25(OH)D at serum 25(OH)D concentrations <30 ng/mL, and serum iPTH levels showed a significant negative association with BMD at the total hip and lumbar spine. These significant negative associations between iPTH levels and BMD at the total hip and lumbar spine underscore the critical role of this hormone in bone metabolism and health. Advancing age, male sex, BMI are other signifcnat predictors for BMD both at total hip and lumbar spine.

Abstract #625

THE EFFICIENCY OF IBANDRONATE AFTER 6 MONTHS OF ORAL / I.V. ADMINISTRATION FOR OSTEOPOROSIS IN POSTMENOPAUSAL WOMEN

Mara Carsote, MD, Diana Loreta Paun, MD, Nicoleta Totolici, Rodica Petris, Catalina Poiana, MD, PhD, Constantin Dumitrache, MD

Objective: Ibandronic acid is a bisphosphonate used in the treatment of osteoporosis. It is available as 150 mg film-coated tablet for once monthly oral administration or 3 mg intravenously every 3 months. This study evaluates the efficiency of ibandronate after 6 months of oral / i.v. administration in women with osteoporosis.

Methods: This is a retrospective study including postmenopausal women that were registered in The National Program of Osteoporosis. Two groups were formed: one of 100 women with oral administration and another group of 50 women who received intravenously ibandronate. All patients received vitamin D. The average age was 64.51+12.1 vs. 65.77+8.23 yrs. The postmenopause yrs were 13 vs. 18 yrs. The av. T score was -3.43 + 0.66 SD vs. -3.5+0.7SD. We investigated:, calcemia, phosphatemia, alkaline phosphatase, vitamin D, markers of bone formation (osteocalcin) and bone resorption (cross-laps) and the presence of fragility fracture.

Results: 30% of the women from first group were not previously treated with other antiresorbtives . The number of patients with hip or wrist fracture was 20 (20%), respective 8 (13%). The biological parameters improved with treatment: calcemia- 9.12+ 0.47mg/dl →9.30+ 0.40mg/dl (p=0,02), cross-laps-0.501+0.34ng/ml→0.250+0.159ng/ml (group 1) // 0.364+0.33ng/ml→ 0.283+0.2ng/ml (group 2) (p=0.005).

Discussion: Both groups of women improved their biological parameters but it is difficult to establish if the administration of ibandronic acid is more important than the patients' profile.

Conclusion: Ibandronate is an efficient drug in the treatment of postmenopausal osteoporosis but a period of 6 months is not enough to appreciate the superiority of oral or intravenous administration.

Abstract #626

RECOMBINANT HUMAN PARATHYROID HORMONE THERAP IN AN OLDER PATIENT WITH A GAIN OF FUNCTION MUTATION OF THE CALCIUM SENSING RECEPTOR-A CASE REPORT

Michael Gonzales, MD, David Lieb, MD, Joseph Aloi, MD, Donald Richardson, MD, FACE, FACP, John O'Brian, MD, Romesh Khardori, Christopher Mulla, MD

Objective: To describe a case of hypocalcemia in a patient with a gain-of-function mutation in the calcium-sensing receptor that was undetected until adulthood and successfully treated with recombinant parathyroid hormone
Methods: Clinical findings and laboratory data are presented followed by a review of pertinent literature.
Case Presentation: A 55 year-old hospitalized woman was seen for hypocalcemia (7.1 mg/dl, albumin 4.0 g/dl) that could not be corrected despite repeated doses of intravenous calcium gluconate. She complained of chronic muscle cramping and paresthesias and had no prior history of neck surgery, neck irradiation, or autoimmune disease. She was a well-appearing female with no dysmorphic features or skin changes. Laboratory testing revealed hyperphosphatemia (6.5 mg/dl), hypomagnesemia, and hypovitaminosis D (21.8 ng/ml). Her parathyroid hormone concentration was inappropriately low at 14.2 pg/ml. Her PTH and calcium concentrations remained low despite repletion of magnesium and treatment with calcitriol and oral calcium replacement. A 24-hour collection for urinary calcium showed inappropriate hypercalciuria (240 mg/24hr). Previous records showed her hypocalcemia to be chronic. Several family members had also complained of muscle cramping. A congenital cause of her hypoparathyroidism was considered. Genetic testing confirmed heterozygosity for a gain-of-function mutation in the calcium-sensing receptor gene associated with autosomal dominant familial isolated hypoparathyroidism (ADH). Recombinant parathyroid hormone (teriparatide) 20 micrograms subcutaneously twice daily resulted in normalization of her calcium and phosphorus concentrations.
Discussion: The CaR detects changes in the serum calcium level and modulates key systems for homeostasis. Activation of the CaR lowers PTH secretion and PTH mRNA production in parathyroid cells. Activating mutations that cause ADH, increase receptor sensitivity to ligands causing a left shift in the PTH-dose response curve to both Ca2+ and Mg2+. Primary hypoparathyroidism is not typically treated by replacing the missing hormone. Conventional therapy with calcium, calcitriol, or other vitamin D analogs normalizes serum calcium with the risk of nephrocalcinosis. Recombinant PTH is a promising treatment for hypoparathyroidism, particularly for patients with ADH as it may help normalize urinary calcium excretion.
Conclusion: Teriparatide is an effective treatment for patients with hypoparathyroidism due to gain-of-function mutations in the calcium-sensing receptor. ADH can be insidious in presentation and diagnosis missed unless the index of suspicion is high.

Abstract #627

CLINICAL FEATURES OF SAPHO SYNDROME

Matheni Sathananthan, MD, Robert Wermers, MD

Objective: To evaluate clinical, radiographic, and laboratory features of patients with a diagnosis of synovitis, acne, pustulosis, hyperostosis and osteitis (SAPHO) syndrome.
Methods: Medical records from 1/1/1996-11/26/2010 were searched for a diagnosis or inclusion in the clinical notes of SAPHO syndrome. Inclusion criteria (Benhamou) included at least one of the following: skin manifestations of severe acne, palmoplantar pustulosis, hyperostosis, or chronic recurrent multifocal osteomyelitis involving the axial or peripheral skeleton. Exclusion criteria included septic osteomyelitis, infectious palmoplantar pustulosis, infectious chest wall arthritis, palmoplantar keratodermia, diffuse idiopathic skeletal hyperostosis or osteoarticular manifestations of retinoid therapy.
Case Presentation: Sixteen patients with clinical and radiographic features of SAPHO syndrome met inclusion and exclusion criteria from the 99 patients initially identified. Eleven patients (68.8%) were female and 5 (31.2%) were male ranging in age from 14-70 years at time of presentation (mean 43.4 years). The majority were Caucasian (n = 13, 81.3%). The duration of symptoms ranged from 2 days to 23 years at time of presentation (mean 6 years). The majority of patients presented with either chest or sternoclavicular joint pain (n = 10, 62.5%). Eight patients (50%) had manifestations of severe acne, 12 (75%) had palmoplantar pustulosis, 1 (6.3%) had chronic recurrent multifocal osteomyelitis and 13 (81.3%) had hyperostosis. Hyperostosis was present at the following sites; sternocostoclavicular - 10 (76.9%) and spine - 3 (23.1%). Fourteen patients were smokers or had a history of smoking (87.5%). Erythrocyte sedimentation rate was elevated in 8/14 (57.1%) patients in whom it was measured. HLA-B27 antigen was positive in 2/7 patients (28.6%) where it was performed.
Discussion: The majority of our patients presented with chest or sternoclavicular joint pain. Various imaging

modalities were used in evaluation of our cohort, and multiple treatment regimens were prescribed with varying results.

Conclusion: Hyperostosis and palmoplantar pustulosis, the most common clinical manifestations of SAPHO syndrome, often remain unrecognized based on the long duration of symptoms prior to diagnosis. Although not previously reported, smoking may be an important etiologic factor for this condition, but further research is needed to confirm this observation.

Abstract #628

PLACENTAL CALCIFICATION: A COMPLICATION OF HYPERPARATHYROIDISM IN PREGNANCY

Mini Mathew, Pharm.D., D.O., Veronica Piziak, Deepika Reddy, Kamalpreet Singh, MD

Case Presentation: Primary hyperparathyroidism is rare in pregnancy but morbidity due to its complications is reported in 67% of affected mothers and 80% of affected fetuses. Pregnancy itself alters levels of calcium and calcitropic hormones like PTH. Serum calcium is usually lower in pregnancy. In contrast, our patient presented with hypercalcemia.

Discussion: Patient is a 40yr old pregnant female G6P2-0-3-2, who was transferred to our hospital for severe hypercalcemia with a serum calcium of 14.5mg/dL. She had been complaining of hyperemesis throughout pregnancy. At 17 weeks gestation, an amniocentesis and ultrasound showed a small for gestational age female fetus. At about 25 weeks gestation, routine labs showed a Calcium of 11.7 mg/dL. A repeat Calcium done a week later showed a Ca 12mg/ dL with an albumin of 3.1gm/ dL. At 28 weeks, the patient complained of losing muscle control of 50% of the left side of her face and was started on a ten day prednisone taper for possible Bells Palsy. A repeat fetal ultrasound at 30 weeks gestations, showed a thickened placenta with surface calcifications, small fetal thorax circumference and short femur and humerus. Serum calcium level was 14.5 mg/dL. The patient was admitted to our hospital for therapy of hypercalcemia. Physical examination was unremarkable except for Blood pressure of 155/77. Upon admission to the hospital calcium was 15.3 and PTH was 245 pg/mL. Aggressive IV hydratiion with NS IV at 200cc/hr was started. Despite good urine output, the Calcium did not improve. Calcitonin therapy then lowered the calcium to 11.8mg/ dL. Calcitonin was continued as needed. At 32 weeks gestation after steroid prep, induction resulted in the delivery of a female infant. The patient did not breast feed and was given cinacalcet after delivery. A sestamibi scan showed a parathyroid adenoma in the region of the left thyroid lobe and a parathyroidectomy was performed. On the following day her Calcium was 8.9 and cinnacalcet was dicontinued with no recurrence of hypercalcemia.

Conclusion: Calcium is not routinely checked during pregnancy. Patient's hyperemesis and muscle weakness may have been due to the hypercalcemia. In cases of maternal hyperparathyroididsm, the fetus should be followed closely because they can develop hypocalcemia from suppressed PTH. Even though this patient presented with severe hypercalcemia, an excellent outcome was achieved for both the mother and the fetus by a team approach using high dose calcitonin and parathyroidectomy.

Abstract #629

RECURRENT PLEURAL EFFUSION AFTER ZOLEDRONIC ACID IN A PATIENT WITH FIBROUS DYSPLASIA

Tulsi Sharma, MBBS, Jennifer Kelly

Objective: Multiple serious adverse effects have been attributed to bisphosphonate use; some resulting from off-label uses, but others seen at the recommended dosage and infusion time.

Case Presentation: We present the case of a 41 year-old male with a history of polyostotic fibrous dysplasia since 1986. He had increasing bone pain for which he received IV zoledronic acid 5mg infusion in April 2011. He complained of progressive dyspnea over the next few weeks. Workup revealed large bilateral exudative pleural effusions. He underwent a therapeutic thoracentesis and left drain placement which was removed over the next 2 days. His symptoms recurred the following month, he was again found to have massive pleural effusions. He had a repeat thoracentesis and more than 1800ml of exudative fluid was removed. Since that time, his effusions have been recurrent requiring repeated thoracentesis. His course over the last few months has been complicated by development of pleural fibrosis and trapped lung as a result of his effusions. He has recently undergone a right sided VATS (video-assisted thoracic surgery) with parietal pleurectomy and pulmonary decortication in July 2011 for the pleural thickening. Pleural biopsy showed evidence of fibrotic adhesions. Rib lesion biopsy showed fibrous dysplasia without any evidence of malignancy. Despite a complete and exhaustive work-up, no specific cause has been identified for this effusion. Is this related to the use of zoledronic acid?

Discussion: Exudative pleural effusion has wide differential and thinking out of the ordinary can sometimes help if no

obvious cause is found. Although uncommon, a number of medications have been reported to cause exudative pleural effusions. This is especially the case for newer drugs with which we do not have long term data and experience. Our patient has a long history of fibrous dysplasia but did not have any prior pleural effusions. The occurrence of recurring effusions with recent use of zoledronic acid points to a diagnosis of this being a rare medication side-effect. A detailed search of literature shows that a few cases have been reported to the FDA after use of zoledronic acid but none have been published. Dyspnea is a listed side effect occurring in 22-27% patients but the etiology is not elucidated.

Conclusion: As the clinical indications for bisphosphonate use continues to expand, it is important for clinicians to prevent, recognize, and manage any possible complications effectively and expeditiously. This case raises awareness to be vigilant for the possibility of a pleural effusion after the use of zoledronic acid as dyspnea is a frequently reported complication.

Abstract #630

CELIAC DISEASE AND METABOLIC OSTEOPATHY: A UNIVERSITY HOSPITAL EXPERIENCE

Mona Fouda, MBBS, FRCP

Objective: To report on metabolic osteopathy in celiac disease (CD) patients in Saudi Arabia where vitamin D deficiency is common.

Methods: A retrospective analysis of the registry on histopathology of celiac disease starting from 1996 as well as a survey of the serological testing done since its introduction in 2003, conducted in a major teaching hospital. The criteria of the diagnosis of CD patients were as follows: a) Patients with duodenal biopsies reported by the histopathologist to be diagnostic of CD and complied with Marsh-Oberhuber system type III (a,b,c), or b) Positive IgA - Antiendomysial antibodies (EMA). Both positive tests had to be supported by strong clinical suspicion of the disease and the final diagnosis given by the treating physician was CD with no alternative diagnosis attached to them on follow-up. Biochemical parameters and imaging tests were recorded.

Case Presentation: The total numbers of patients were 114, 65 of whom were children (aged 15 years or younger). The male to female ratio was 1:1.9. A total of 88 patients (94%) were diagnosed by histopathology while 92 (85%) had positive serology. There were 82 patients who had both tests performed and 61 (74%) had both tests positive. The 25 Dihydroxy vitamin D3 [25(OH)2D3] was low in 100% of adults and in 70% of children who did the test, while all the groups who had a PTH value measured (11 patients) had high level (100%). Dual X-ray absorptiometry (DXA) was performed only in 19 patients with osteopenia/osteoporosis reported in 79% of them. A bone scan was performed in only 14 patients and an abnormal finding compatible with metabolic bone disease reported in 64% of them.

Discussion: Although only a small number of our patients had a bone imaging test, 100% of children and 75% of adults had low bone mass on DXA, and 33.3% of children versus 72.7% of adult had rickets/osteomalacia with or without pseudo-fractures on T-99m bone scan. The overall evidence of bone loss was almost 80% on DXA and 64% had abnormal bone scan compatible with metabolic bone disease. This is much higher than reported in other Middle Eastern countries. Out of small number of our patients who did the test, 100% had abnormal PTH values with vitamin D3 low in 100% of adults and almost 70% of children who performed the test. The group as a whole had near 85% prevalence of vitamin D deficiency, the highest reported so far in a Middle East celiac group.

Conclusion: Metabolic osteopathy and vitamin D deficiency are higher in Saudi CD patients than reported elsewhere. The physicians' awareness of importance of bone screening for metabolic bone disease and bone imaging is seriously low.

Abstract #631

BONE MINERAL DENSITY IN PATIENTS WITH NONALCOHOLIC STEATOHEPATITIS

Patchaya Boonchaya-anant, MD, Elvin Hardy, Brian Borg, Alan Burshell, MD

Objective: Several studies have shown that patients with cirrhosis/end-stage liver disease (ESLD) have lower bone density and higher prevalence of osteoporosis compare to age-matched population. Hyperinsulinemia and insulin resistance is typically associated with increased bone density. We hypothesized that patients who have nonalcoholic steatohepatitis (NASH) of the liver and underlying insulin resistance may have higher bone density (BMD) than patients with other causes of cirrhosis.

Methods: This is a retrospective chart review study of all patients with ESLD who underwent liver transplantation evaluation at Ochsner Clinic Foundation during 2009-2011. Patients who had been treated for osteoporosis or taking bisphosphonate, teriparatide, or denosumab prior to transplant evaluation were excluded. Patients were categorized to three groups based on the etiology of their liver disease as nonalcoholic steatohepatitis (NASH), alcoholic cirrhosis and viral hepatitis (HCV+HBV).

Lumbar spine, total hip and femoral neck BMD data were obtained within 2 years prior to transplantation and compared between groups using student t-test.

Results: The characteristic of patients are shown in Table 1. Sixty-three patients met inclusion criteria for the study: 15 patients with NASH, 17 with alcoholic cirrhosis, and 31 with HCV + HBV. The BMD values, T-score and Z-score in the three groups are shown in Table 2. There were no statistical differences in BMD values, T-score or Z-score between NASH and alcoholic cirrhosis groups at any sites. The BMD values, T-score and Z-score were higher in NASH group than HCV + HBV group at all sites. The overall prevalence of osteopenia and osteoporosis were 44% and 12% respectively. The majority of patients with alcoholic cirrhosis and HCV + HBV had osteopenia, 59% and 51% respectively. Sixty percent of patients with NASH had high Z-score (>1SD).

Discussion: Our study showed a high prevalence of low bone density among patients with end-stage liver disease awaiting liver transplantation. Patients with NASH had higher bone density than patients with viral hepatitis. We found no statistical differences in bone density between patients with NASH and alcoholic cirrhosis. Significant number of patients with NASH had high Z-score especially at lumbar spine which may indicate different underlying process such as the effect of body weight, hyperinsulinemia and insulin resistance.

Conclusion: Patients with nonalcoholic steatohepatitis may have different bone mineral density pattern compared to patients with other causes of cirrhosis. Future prospective study will be needed to clarify the underlying mechanism.

Abstract #632

REAL-LIFE EFFECTIVENESS OF ZOLENDRONIC ACID IN PATIENTS WITH OSTEOPOROSIS: 5-YEAR EXPERIENCE

George Tsoukas, MD, Emmanouil Rampakakis, Philip Tsoukas, John S. Sampalis, Michael Tsoukas, MD

Objective: Although the efficacy of zolendronic acid in bone metabolism has been demonstrated in several controlled clinical trials, data from longitudinal observational studies are scarce. Such studies are essential in order to assess the real-life effectiveness of therapeutic interventions and demonstrate true population-based benefits.

Methods: This was a retrospective chart review database analysis with data obtained from four clinics on patients initiating treatment with zolendronic acid. Changes in bone mineral density (BMD), ionized calcium (iCa), serum parathyroid hormone (PTH), bone formation markers (osteocalcin, alkaline phosphatase) and the bone resoprtion marker type I collagen C-telopeptide were assessed using linear mixed models with repeated measures.

Results: A total of 291 patients were included with a mean (SD) age of 65.0 (10.2) years. Among these, 262 (90.0%) patients were female. At baseline, the mean (SD) T-score was -2.9 (1.0) and -2.3 (0.8) for the spine and femur, respectively, and the mean (SD) zolendronic acid dose was 4.3 (0.78) mg. After one year of treatment with zolendronic acid, patients experienced clinically and statistically significant improvements in spine (% change = 2.09) and femur (% change = 2.05) bone mineral density (BMD), which were further augmented over five years (trend over time: Pspine = 0.003, Pfemur<0.001). Interestingly, significant differences in spine BMD were observed based on the type of previous osteoporosis treatment (P=0.009); patients having previously used only calcium and vitamin supplements experienced the maximal benefit followed by patients previously treated with osteoporosis hormone replacement therapy or selective estrogen receptor modulators (HRT/SERMs), or oral bisphosphonates (OBPs). Percent changes over time in iCa, PTH, and bone biochemical markers upon adjustment for baseline values were not statistically significant.

Discussion: The results of this real-life observational study demonstrate that long-term treatment with zolendronic acid over five years is effective in improving spine and femur bone mineral density in patients with osteoporosis, regardless of previous osteoporosis treatment. However, patients previously primed with calcium and vitamin supplements experienced the maximal clinical benefit.

Conclusion: Long-term treatment with zolendronic is effective in improving spine and femur bone mineral density in patients with osteoporosis in a real-life setting. Patients previously primed with calcium and vitamin supplements experienced the maximal clinical benefit.

Abstract #633

SYSTEMIC MASTOCYTOSIS- A RARE CAUSE OF OSTEOPENIA IN AN ADULT MALE

Ila Khanna, MD, Faryal Mirza, MD, Diane Whitaker Worth

Objective: To present a unique case of osteopenia in a male secondary to systemic mastocytosis

Case Presentation: This is a 53 year old Caucasian male who reports noticing a rash on his body starting in 1992. The rash was more prominent on his lower extremities, inner thighs, back and abdomen. It typically got worse after a hot shower but there was no associated flushing or pruritus. He denied any tachycardia, diarrhea or fatigue.

He underwent a dermatologic evaluation and a skin biopsy. On physical exam he was noted to have multiple scattered red-brown freckle like macules over trunk and upper body. Biopsy results were consistent with urticaria pigmentosa or maculopapular cutaneous mastocytosis. Laboratory evaluation with CBC, liver function tests and serum tryptase level were normal initially and he was managed conservatively with phototherapy and regular follow up of tryptase levels. In 2010, serum tryptase level was found to be elevated at 119 ng/ml (range 2-23 ng/ml) and the patient subsequently underwent a bone marrow biopsy which confirmed the diagnosis of systemic mastocytosis. He also had a bone density test done which revealed osteopenia and he was referred to the osteoporosis center for further evaluation.

Discussion: Osteopenia and osteoporosis in an adult male are less common than women and therefore secondary causes should be pursued thoroughly in this population. Systemic mastocytosis (SM) is one cause of secondary osteoporosis. It is a rare disorder characterized by excessive mast cell accumulation in one or multiple organs. Mast cells contain a variety of vasoactive mediators such as heparin, leukotrienes, histamine, prostaglandins, cytokines and platelet activating factor. The release of these mediators in patients with mastocytosis can be triggered by a variety of factors such as certain medications, exercise, surgical procedures, alcohol ingestion, infections and emotional stress. It is subdivided into cutaneous matocytosis which is limited to the skin and systemic mastocytosis with extra cutaneous organ involvement. Osteopenia and osteoporosis can be unique manifestations of systemic mastocytosis. Its pathogenesis is thought to be secondary to effects of mast cell mediators such as histamine, heparin and cytokines (TNF, IL-6 and TGF beta) on bone turnover. Recent study done by Barete et. Al showed that osteoporosis is the most prevalent bone manifestation in systemic mastocytosis. In addition, they also showed that bisphosphonate therapy is efficient at improving lumbar spine density associated with SM.

Conclusion: This case highlights the importance of evaluating for presence of osteopenia or osteoporosis in patients with systemic mastocytosis.

Abstract #634

RADIATION ASSOCIATED PELVIC FRACTURES: REPORT OF 4 CASES

Mahshid Mohseni, MD, Pooya Hosseinzadeh, Tipu Faiz Saleem, MD, Saba Faiz, MD, Mateen Hotiana, MD, Abid Yaqub, MBBS

Objective: High dose radiation is used for treatment of variety of malignancies. Radiation has known deleterious effects of bone metabolism and has been associated with insufficiency fractures. Treatment of these fractures are challanging due to high non union rate and concerns about the use of antiresportive and anabolic agents in this group of patients.

Case Presentation: Case 1: 76 year old female presented with bilateral sacral and pubic rami fractures two years after radiation for cervical cancer. She had been on bisphosphonates previously. Bisphosphonates were stopped and patient was offered Forteo (PTH) but patient decided not to proceed due to the increased risk of sarcoma development. The fracture was treated non operatively and the patient's symptoms resolved. Case 2: 76 year old female presented with bilateral sacral insufficiency fractures one year after radiation for rectal cancer. The patient was on bisphosphonates before and it was discontinued after the fracture. The fracture was treated non operatively and the patient's symptoms improved. Case 3: 65 year old female with a history of radiation for endometrial cancer presented with bilateral pubic rami fractures. Due to osteoporosis on DEXA scan she was placed on IV bisphosphonates. The follow up has showed improvement in symptoms, bone mineral density, and no evidence of new fractures. Case 4: 82 year old male presented with pubic rami fractures 7 years after radiation for prostate cancer. He had been on bisphosphonates that were stopped for one year. Patient became asymptomatic and the fracture showed radiographic healing after one year.

Discussion: Insuufiecency fractures are seen as a result of radiation treatment for malignancies. Surgical treatment is not recommended for pelvic insufiecency fractures and there is no proven effective medical treatment. Antiresoprtive therapy can delay fracture healing and has been associated with fractures with long term use. Anabolic agents have been associated with increase chance of sarcoma development in irradiated bone. The medical treatment of these fractures is currently evolving

Conclusion: Radiation associated fractures are difficult to treat due to high rate of non union and paucity of literature on treatment guidelines. The medical treatment of these fractures should be decided on case by case basis and the advantages and disadvantages of each treatment should be discussed with the patient.

Abstract #635

A BREAK IN BISPHOSPHONATE THERAPY

Ila Khanna, MD, Pamela Taxel

Objective: To present a unique case of atypical femoral fracture in setting of extensive bisphosphonate use.

Case Presentation: A 78 year old postmenopausal female

with history of osteopenia on bone mineral density was treated with alendronate for eleven years from 1999 to 2010 by her primary care physician. Past medical history was significant for hypertension, hyperlipidemia and glaucoma. There was no history of fractures, steroid use, endocrine disorders or kidney stones. On presentation, the patient reported right anterior thigh pain and difficulty walking for several weeks with no associated trauma. Initial x-rays of the right hip were unremarkable for any fractures, and she was treated for trochanteric bursitis and muscle strain. Due to the persistence of symptoms, a bone scan was performed at the request of an orthopedic surgeon 9 months after her initial complaint. This revealed a subtrochanteric stress fracture in the right hip. However, prior to her scheduling the surgery, the patient incurred a low trauma fall from a standing height and was found to have an impacted angulated fracture through the midshaft of the right femur, consistent with an atypical subtrochanteric fracture.

Discussion: Bisphosphonates are currently the first line treatment for postmenopausal osteoporosis. Over the last five years, a number of reports have shown an association between long term bisphosphonate therapy and atypical low-energy fractures defined as fractures sustained in a fall from a standing height or less. These have been associated with a prodrome of pain in the groin or thigh for days to months preceding the fracture. There is circumferential cortical thickening and development of cortical stress lesions that may precede a complete transverse or oblique fracture of the subtrochanteric femur. Mechanism of action for this remains poorly understood although it has been hypothesized to be secondary to reduction of bone remodeling and accumulation of micro-cracks leading to stress fractures. The American Society for Bone and Mineral Research has defined a diagnostic criteria to improve the recognition and incidence assessment of atypical femoral fractures. Although bisphosphonate therapy has been implicated as a major risk factor, the absolute risk remains small compared with the beneficial effects of its treatment.

Conclusion: This case highlights the need for the astute clinician to be aware that a simple complaint of thigh or groin pain could suggest the possibility of an atypical fracture particularly in a patient with a long term history of bisphosphonate exposure. It also points to the importance of reassessment of the need for anti-resorptive therapy at regular intervals.

Abstract #636

A HIGH PREVALENCE OF VITAMIN D DEFICIENCY IN MEDICAL OUTPATIENTS IN CENTRAL VALLEY, CALIFORNIA

Ivance Albert Pugoy, MD, Evelina Svrdlan, MD, Marianne Ghobrial, MD, Katayoun Edalat Parsi, DO, Abhishek Sawant, Paul Mills, Soe Naing, MD, MRCP, FACE

Objective: To determine the prevalence and predictors of vitamin D deficiency (VDD) in medical outpatients in Central Valley, California

Methods: This cross sectional retrospective study was conducted at the Ambulatory Care Center at University of California, San Francisco, Fresno Medical Education Program, CA. The outpatients from the clinics of Internal Medicine and Family Practice, who had serum 25 hydroxyvitamin D [25(OH)D] measured from July 1, 2009 to June 30, 2011 for any reason, were included in the study.

Results: We studied 536 patients with a mean age of 55.4 (SD±14.8) years. 62% were female; and Hispanic, Caucasians and African Americans comprised 55%, 21% and 15%, respectively. 42.7% had diabetes mellitus (DM) and mean BMI was 31.88 (SD±13.4). 16.4% had 25(OH)D <10mg/dl (severe VDD), 56.6% had <20mg/dl (VDD) and 85.4% (458 of 536 patients) had <30mg/dl. Those with VDD were younger (54.01 vs 57.13 years; p=0.006), had higher body weight (187.7 vs 181.1 lbs), lower total calcium (9.3 vs 9.5mg/dl; p=0.003), higher triglyceride (169.5 vs 135mg/dl; p=0.003), higher LDL (102.6 vs 92.7 mg/dl; p=0.021), higher HbA1c (7.17 vs 6.61%; p=0.004), lower hemoglobin (12.2 vs 12.8mg/dl; p <0.001) and higher TSH (5.45 vs 2.13mU/L; p=0.015) than those without VDD. 25(OH)D level was negatively correlated with weight, PTH, HbA1c, triglyceride, LDL, TSH, and positively correlated with total and corrected serum calcium, serum albumin, GFR and hemoglobin levels. In multivariate regression analysis, the presence of DM (OR 4.25, 95% CI 1.46-12.39), LDL (OR 1.025, 95% CI 1.00-1.04) and hemoglobin levels (OR 0.61, 95% CI 0.44-0.85) were independent predictors of VDD.

Discussion: Low vitamin D level has been implicated as a risk factor for several clinical disorders. With the abundant sunlight exposure in Fresno at Central California (36.77° N latitude and 267 total days with sun), prevalence of VDD is not expected to be high. However this study has demonstrated that over half of the patients had VDD and the vast majority of the patients had 25(OH)D level <30mg/dl. Those with DM, higher LDL or lower hemoglobin were particularly at increased risk for VDD. Sun avoidance, use of sunscreen or protective clothing, inadequate dietary

vitamin D intakes and associated medical conditions may be some of the possible risk factors contributing to VDD. ***Conclusion***: Vitamin D deficiency was highly prevalent in the medical outpatients in this sunny and temperate region. The presence of DM, LDL and hemoglobin levels were independent predictors of VDD.

Abstract #637

A SATISFYING SEARCH FOR DIAGNOSTIC CERTAINTY

Sameer Ansar, MD, George Hebdon, M.D., Andrew Saxe

Objective: We describe a case of hypercalcemia with elevated 1,25- dihyroxyvitamin D (1,25(OH)2D) in a patient ultimately diagnosed with occult sarcoidosis.
Case Presentation: A 67 year old female presented complaining of weakness and feeling unwell and was found to have a calcium level of 17.57 mg/dl. Three months prior to this her calcium was 9.42 mg/dl. She had no history of kidney stones or family history of endocrine disorders, did not take lithium or hydrochlorothiazide. She had no risk factor for tuberculosis A thyroid profile, cortisol and liver function tests were normal. Her albumin was 3.7 mg/dl, plasma 25-OHD 26 ng/dl, 1,25(OH)2D 151 pg/ml, PTH 10.3 pg/ml, PTH related Peptide (PTHrp) < 0.5 pmol/L, ACE level 18 U/L, and free retinol 80.4 mcg/dl. She had a negative computed tomography of her chest and, along with the low PTHrP made malignancy less likely. Serum protein electrophoresis was normal ruling out multiple myeloma. The normal ACE level and a negative CT chest largely excluded sarcoidosis and other granulomatous disease Given the elevated 1,25(OH)2D we continued to suspect granulomatous disease. A FDG/PET scan revealed increased activity in the submandibular region. A biopsy showed non-caseating granulomata with negative AFB and GMS stains consistent with sarcoidosis. She started prednisone 40 mg daily and her calcium and 1,25(OH)2D decreased to 10.72 mg/dl and 51 pg/ml respectively.
Discussion: There has been debate over how best to evaluate and manage patients such as the one we describe, particularly about when to empirically start steroids for suspected, but undiagnosed, sarcoidosis. Often one is reasonably certain of the etiology of hypercalcemia with the help of history, physical examination, PTH, 25 OHD, and a complete metabolic panel. In patients with elevated 1,25(OH)2D the diagnosis is narrowed down to granulomatous disease, fungal infections, tuberculosis, foreign body reactions and lymphomas. In our patient the elevated 1,25(OH)2D identified the mechanism, however the etiology remained unclear. Sarcoidosis remained high on the differential even in the face of a normal ACE level (as ACE level is neither sensitive nor specific for the diagnosis of sarcoidosis). Consequently a FDG PET was performed leading to a biopsy which secured the diagnosis ***Conclusion***: If an elevated 1,25(OH)2D mediated cause of hypercalcemia is suspected, it is important to target the underlying cause. We believe steroids should be used only with caution until a firm diagnosis of sarcoidosis is made. Sarcoidosis without lung involvement and even a normal ACE level should be kept in mind while working up hypercalcemia. FGT PET is a useful tool in the workup of Sarcoidosis.

Abstract #638

A SPONTANEOUS INSUFFICIENCY FRACTURE IN AN ADULT WITH X-LINKED HYPOPHOSPHATEMIA

Vaishali Patel, MD, Leigh Eck, MD

Objective: X-linked hypophosphatemia (XLH) is a disorder of renal phosphate wasting. It is the most common form of heritable rickets. Patients present in childhood with bowing of the legs; children are treated with activated vitamin D and phosphate salts until growth is complete. Treatment in adults is not straightforward with limited clinical trial data to base treatment decisions. Generally, adults with XLH are not treated unless they are symptomatic. We present a patient with XLH who presented with leg pain and was found to have a spontaneous fracture of the femur.
Case Presentation: A 49-year-old male with history of XLH diagnosed in childhood presented to our adult endocrine clinic with complaints of bone pain. He had been treated with calcitriol and phosphate in childhood with therapy continued until age 32. He had previously discontinued therapy on his own due to concerns for toxicity. On presentation, he noted myalgias as well as leg and back pain. He denied any recent fall or trauma to his skeletal system. On examination, he was of short stature, had bilateral bowing of the legs, and limited mobility due to pain. Lab revealed a low normal phosphate and 1, 25 dihydroxyvitamin D, normal serum calcium, and upper range normal parathyroid hormone level. Due to pain, radiology was undertaken with evident incomplete right femur fracture with a periosteal reaction. Due to this insufficiency fracture with planned orthopedic procedure, our patient was initiated on calcitriol 0.25 mcg twice daily and subsequently, elemental phosphorus 250mg per day was added with subsequent slow titration to three times daily. He underwent surgical correction of right femoral fracture. Careful laboratory monitoring was undertaken with a serum calcium, phosphorous, creatinine, alkaline phosphatase and 24 hour urinary collection for calcium and creatinine obtained four weeks after initiation of therapy.

His leg pain, muscle weakness, myalgias improved with treatment.

Discussion: The goals of treatment in adults with XLH are to reduce pain symptoms, to reduce the extent of osteomalacia, and to improve fracture healing or surgical recovery. Randomized clinical trial data supporting the utility of treatment in adults is limited and treatment can be associated with toxicity.

Conclusion: The major risks of long-term therapy with calcitriol and phosphorus in adults with XLH are similar to those in children: hypercalcemia, hypercalciuria, nephrolithiasis, nephrocalcinosis, and, potentially, chronic kidney disease. Managing XLH in adults is complicated; involving a clinician with experience treating XLH is prudent.

Abstract #639

EFFECT OF DIMETHYL SULFOXIDE ON BONE HEMOSTASIS WITH OBESITY

Omar Akhtar, MBBS, Abid Yaqub, MBBS

Objective: Obesity is a global epidemic that affects virtually all age and socioeconomic groups. This disorder is characterized by elevated state of inflammation which has been shown to negatively regulate bone quality. The objective of this study was to evaluate the effect of anti-inflammatory agent on bone hemostasis in obese Zucker rat model.

Methods: Twelve 4-week old obese Zucker rats were randomly assigned to control or dimethyl sulfoxide (DMSO)-treated groups (N = 6). DMSO (0.09 g / kg body weight / day) was given via drinking water for 6 months. Lean Zucker rats (N = 6) were used to control for the effects of obesity. At 30 weeks of age animals were euthanized and the femur and tibia of both hind limbs were dissected. Bone mineral density (BMD) was measured using a GE Lunar iDXA densitometer equipped with a specific animal software enCORETM 2011. Serum was collected for the determination of the pro-osteoblastic marker osteocalcin and bone degradation marker pyridinoline crosslinks (PYD) via ELISA.

Results: Compared to the obese control animals, DMSO treatment increased the BMD of both femur and tibia by 26.8% and 23.7%, respectively ($p \leq 0.05$). BMD density in the obese treated animals was not different from that observed in the lean Zucker control animals. Serum PYD in obese Zucker control was significantly higher than that of lean Zucker animals (+149.7%; $p \leq 0.05$). Interestingly, chronic DMSO ingestion reduced circulating PYD in obese animals to a level equivalent to that of lean control animals ($p = 0.1$). Serum osteocalcin was not statistically different between three groups.

Discussion: DMSO is a widely used polar aprotic solvent with clinically-proven anti-inflammatory activity. Chronic DMSO ingestion was able to increase BMD in both tibia and femur of obese Zucker rats and decrease circulating PYD levels, supporting a beneficial effect of anti-inflammatory agents on bone health with obesity. The improved BMD by DMSO may be mediated by attenuating bone degradation.

Conclusion: Chronic DMSO treatment can improve bone hemostasis in the obese Zucker rat model.

Abstract #640

ELEVATION OF SERUM PARATHORMONE LEVELS WITH NORMOCALCEMIA AFTER PARATHYROIDECTOMY FOR PRIMARY HYPERPARATHYROIDISM

Sunil Kota, MD, Siva Kota, Svs Krishna, Lalit Meher, Kirtikumar Modi

Objective: Persistent elevation of serum parathyroid hormone (PTH) despite normocalcemia is documented in 8- 40% of patients after parathyroidectomy. We hereby report our experience from a tertiary centre in south India, to determine clinical significance of postoperatively elevated PTH levels and review relevant literature.

Methods: We conducted a retrospective case series study and reviewed clinical and laboratory data of all the patients who underwent surgery for primary hyperparathyroidism (PHPT) from January 2005 to September 2011.

Results: Total of 67 patients were diagnosed as PHPT. Twenty one subjects (34%) presented with fractures and significant bone disease. Nephrolithiasis in 18 (25.5%) and peptic ulcer in 9 (13.5%) patients were observed. The remaining 19 patients (27%) had asymptomatic PHPT. One patient declined surgery and followup data of another 6 patients was not available. Out of available follow up data of 60 patients, a total of 18 patients (30%) had persistently elevated PTH (PePTH) at 1 month. Patients with PePTH were older with higher baseline serum calcium, iPTH, alkaline phosphatase and lower serum phosphate and 25-hydroxy vitamin D3 levels. Creatinine clearance was significantly lower in patients with PePTH. Multiple linear regression analysis revealed that 25-OH D3 concentration, creatinine clearance and iPTH are the factors influencing re elevation of PTH levels. Significantly lower serum calcium and higher alkaline phosphatase levels were observed in PePTH patients with preoperative 25-OH D3 levels < 20 ng/ ml.Ten patients at 6months, 8 patients at 1 year, 6 patients at 2 years and 3 patients at 3 years had eucalcemic PTH elevation. Three out of 42 (7%) patient with normal initial postoperative calcium and iPTH levels developed PePTH, with none

culminating into recurrent hyperparathyroidism.
Discussion: Elevated PTH levels after successful parathyroidectomy may represent persistent/ recurrent hyperparathyroidism. Its incidence in our series was 20% at 1 week and 30% at 4 weeks after parathyroidectomy. Various factors have been proposed as etiology of such a commonly occurring phenomenon after surgery for primary hyperparathyroidism. They include bone hunger, vitamin D deficiency, inadequate calcium intake/ absorption, reduced peripheral sensitivity to parathyroid hormone, chronic kidney disease and renal leak of calcium. Adequate dosage of calcium and vitamin D supplementation is hypothesized to reduce such an occurrence.
Conclusion: Though the pathogenesis of such a phenomenon still remains to be elucidated, a multifactorial mechanism appears to play a role.

Abstract #641

ENHANCEMENT OF BONE GRAFT FUSION WITH TERIPARITIDE

Anupa Sharma, DO, Sara Lubitz, MD

Objective: To present the first case of Teriparatide (synthetic human PTH 1-34) use to enhance spinal fusion after repeated single level bone graft non-union.
Case Presentation: A 45-year-old Lebanese woman was undergoing neurosurgical treatment for cervical spondylosis causing neck pain radiating down her arms and progressive bilateral hand numbness. In August 2009, she underwent a C6-C7 anterior cervical discectomy and fusion using an allograft and DePuy Uniplate. Four months later she reported persistent radicular symptoms and a CT scan demonstrated lucency at the superior aspect of the graft endplate indicating that the fusion process had not been completed. Subsequently, in March 2010, she underwent a revision anterior cervical discectomy and fusion with use of autologous bone from the iliac crest. In September 2010, she again noted the return of neck pain and right hand numbness. Repeat CT scan showed non-union of the inferior aspect of the C6-C7 graft. Prior to a third attempt at fusion, the patient was evaluated by our Endocrinology service. The patient noted a history of traumatic hip fracture from a MVA in 1994 which healed normally and reported regular menses. She denied use of corticosteroids or anti-convulsants and personal or family history of metabolic bone disease. Lab evaluation was only significant for 25-OH Vitamin D of 20 ng/ml which was treated with Ergocalciferol 50,000 IU weekly for 8 weeks. The patient underwent a third neurosurgical intervention in December 2010 with anterior and posterior cervical fusion at C6-C7 using allografts. On post-operative day #2, Teriparatide 20 mcg SQ daily was started. After 11 weeks of therapy, repeat lab work revealed an expected increase in bone turnover markers and vitamin D repletion. Repeat CT scan demonstrated a solid fusion at C6-C7, and at that point Teriparatide was discontinued.
Discussion: Different mechanisms have been studied to determine what role Teriparatide plays in enhancing bone formation. Administration of intermittent PTH (1-34) can enhance fracture healing in animal and human models through quicker and stronger callus formation. Bone graft fusion relies on similar mechanisms of cartilage formation at the fusion site for union to occur. In limited animal models of fusion surgery, Teriparatide administration leads to increased quantity of fusion bone by increasing the number of osteoblasts and osteoclasts and enhancing callus formation and mineralization. This is the first case of Teriparatide use in a human to improve spinal fusion.
Conclusion: We propose Teriparatide as a treatment option in patients undergoing discectomy and fusion at high risk for non-union or undergoing revision surgery for failed fusion.

Abstract #642

FRACTURE BURDEN IN CHILDREN AND ADULTS WITH HYPOPHOSPHATASIA

Horacio Plotkin, MD, Michael Whyte, Cheryl Rockman-Greenberg, Priya Kishnani, Azziz Mhanni, Jill Mayhew, Nerissa Kreher, Alison Skrinar

Objective: Hypophosphatasia (HPP), an inherited rickets or osteomalacia, results from inactivating mutation(s) in the gene that encodes the tissue non-specific (bone) isoenzyme of alkaline phosphatase. Consequently, inorganic pyrophosphate accumulates extracellularly and inhibits skeleton and tooth mineralization. Fractures, including atypical hip fractures, are a complication of HPP that can cause significant morbidity, but their prevalence is not known. The objective was to determine the fracture prevalence and associated burden-of-illness for children and adults with HPP who either first manifested HPP in childhood or in adulthood, using data acquired from two patient-reported surveys.
Methods: Following informed written consent where appropriate, two surveys, one via the internet (The HPP Impact Patient Survey [HIPS], 9/2009-6/2011) and one via telephone interview (The HPP Outcomes Study Telephone [HOST], 12/2010—3/2011), explored the impact of HPP on children and adults (i.e., > 18 yr). If HPP symptoms and diagnosis first occurred > 18 yr, we diagnosed "adult-onset" HPP. Volunteer

and potential study subjects were self-selected (HIPS) or contacted by their medical centers (HOST). ***Results***: 184 HPP patients, 59 children and 125 adults, responded. 58% (106/184) reported at least one fracture. For the 125 adults, mean (SD) self-reported fractures were 13.9 (± 22.3, range: 1 - 100). For adult-onset HPP, 43/44 (98%) experienced at least one fracture and averaged 11 (± 9, range: 1 - 30). Approximately 75% were lower extremity fractures (thigh, leg, ankle, foot, or toe). 28/44 (64%) patients required surgical fixation. Importantly, 32% modified their home as a result of HPP, and 27% employed help for HPP-related difficulties.
Discussion: Although our study is likely skewed towards more severely affected HPP patients, regardless of the age of HPP onset, affected adults can have many fractures. Fracture location seems to condition the burden of illness. Even those with adult-onset HPP can have fractures, indicating that HPP can impose a significant disease burden at any time.
Conclusion: Even patients with adult-onset HPP can have fractures, indicating that HPP can impose a significant disease burden at any time.

Abstract #643

GOT PHOSPHORUS? - CASE OF TUMOR-INDUCED OSTEOMALACIA

Samineh Madani, MD, Alison Semrad, DO

Objective: Phosphate plays an important role in intracellular signaling, energy metabolism, and bone mineralization. Derangements in phosphate levels can be devastating to normal physiologic functioning, thus correct diagnosis and timely treatment are warranted. We present a case of hypophosphatemia due to tumor-induced osteomalacia.
Case Presentation: A 62 year old man with reactive arthritis and multiple spontaneous rib fractures presented to endocrinology clinic for evaluation of persistent hypophosphatemia. He reported of diffuse bone pains, muscle weakness and cramping, and decreased energy. Physical examination was unremarkable. Labs showed low phosphorus, normal calcium, elevated PTH, low 1,25-dihydroxy vitamin D, and elevated 24 hour urinary phosphate excretion levels. A DEXA scan revealed diffuse osteopenia. Further labs showed an elevated FGF-23 level and negative genetic testing for FGF-23 mutations. During this time, the patient was treated with calcitriol and phosphate supplements as the search for an underlying tumor via numerous imaging modalities remained unfruitful. Two years later, the patient began to complain of significant right foot pain. While X-rays were normal, an MRI of the right foot revealed a solid enhancing soft tissue mass in the medial plantar foot. An octreotide scan also showed increased uptake. A large, benign, fatty mesenchymal tumor was resected and tumor-induced osteomalacia was diagnosed. Subsequent to surgery, the FGF-23 level decreased and phosphate and PTH levels improved though did not completely remit as a small portion of the tumor was unresectable.
Discussion: Tumor-induced osteomalacia is a rare paraneoplastic syndrome characterized by elevated FGF-23 levels. FGF-23 is a hormone that works by reducing sodium-phosphate co-transporters in the kidney, decreasing 1-alpha-hydroxylase, and increasing 24-hydroxylase, leading to hypophosphatemia, renal phosphate wasting, and low 1,25-dihydroxy vitamin D levels. Typical tumors associated with this phenomenon include hemangiopericytoma, sarcoma, and osteoblastoma, which may difficult to locate. As in our patient, several years and multiple imaging modalities were used to finally identify the tumor's location. Octreotide scintigraphy improves detection given the high affinity for the somatostatin receptor. Treatment includes definitive treatment with surgical resection versus medical therapy with elemental phosphorus, calcitriol, and vitamin D if surgery is not an option.
Conclusion: The diagnosis of tumor-induced osteomalacia may be challenging due to the indolent course of non-specific complaints, albeit imperative given associated high morbidity and mortality without timely diagnosis and treatment.

Abstract #644

HUMORAL HYPERCALCEMIA OF MALIGNANCY: A CASE OF A PARATHYROID HORMONE- RELATED PEPTIDE (PTH-RP) - SECRETING UTERINE LEIOMYOSARCOMA

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Doctor of Medicine, Jennifer Swenski, Mikhail Signalov

Objective: To present a rare case of symptomatic hypercalcemia in a patient with elevated Parathyroid hormone-related peptide level (PTHrP) and otherwise clinically asymptomatic Uterine Leiomyosarcoma.
Case Presentation: A 56-year old woman presented for evaluation of symptomatic hypercalcemia of unknown etiology. She was hospitalized with new onset of dyspnea and was diagnosed with Hypercalcemia of 16.8 mg/dL (NL:8.3-10.5mg/dL) associated with suppressed intact Parathyroid Hormone (iPTH) of 8 pg/mL (NL:15-65 pg/mL). Subsequently requested PTHrP was 1.8 pg/mL. Chest CT, Bone scan and Mammogram were unremarkable. Renal US revealed no evidence of stones but suggested presence of Uterine Fibroid.

Pelvic US confirmed diffusely heterogeneous mass filling the endometrial stripe that was highly suspicious for malignancy. Her calcium level improved to 12.2 mg/d after a dose of intravenous bisphosphonate during the hospitalization. On follow up, labs showed normal 25OH-Vit D level at 31.8 ng/mL (NL:30-100 ng/mL) and iPTH remained suppressed at 7 pg/mL. PTHrP level was repeated via reference laboratory due to high index of suspicion for Humoral Hypercalcemia of Malignancy (HHM). PTHrP came back elevated at 60 pg/mL (NL:14-27 pg/mL). Dilatation and Curettage of the endometrial mass confirmed Leiomyosarcoma. Total Abdominal Hysterectomy and Bilateral Salpingo-oophorectomy was done with final pathology revealing High grade Uterine Leiomyosarcoma Stage 1B (T1BN0M0). Post-operative follow-up confirmed normalization of the calcium level and PTH-rP of 18 pg/mL.

Discussion: HHM accounts for up to 80% of patients with Hypercalcemia of Malignancy and is mostly associated with Squamous cell carcinoma (lung, head and neck), renal, bladder, breast or ovarian cancers. In 2003 Tang et.al reported a case of PTH-rP secreting metastatic epithelioid leiomyosarcoma in a woman with hepatic metastasis. To our knowledge, this is the first report demonstrating PTH-rP secreting Uterine Leiomyosarcoma with hypercalcemia in the absence of metastatic disease. Postsurgical normalization of the calcium and PTH-rP provided clinical evidence of suspected primary source of immunologically detectable and biologically active PTH-rP.

Conclusion: This case emphasizes the importance of high index of suspicion for PTH-rP secreting malignancy in the setting of hypercalcemia with suppressed iPTH level. Malignancies that are not historically associated with PTH-rP elevation should be etiologically considered and more extensive screening may be necessary.

Abstract #645

HYPERCALCEMIA IN A POSTMENOPAUSAL WOMAN WITH MULTIPLE MYELOMA, AMYLOIDOSIS AND HIGH PTHRP

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Objective: To describe a case of hypercalcemia in a postmenopausal woman in whom diagnosis of amyloidosis and multiple myeloma was made concurrently. To recognize that PTHrP may possibly contribute to hypercalcemia in multiple myeloma.

Case Presentation: A 64 y/o post-menopausal woman with osteoporosis (T-score -2.9 hip) was referred for evaluation of decreased range of motion of neck, shoulders, and upper and lower extremities; tingling, numbness of hands, and macroglossia. Symptoms started 5-6 years ago. She also reported difficulties in speaking and swallowing and unintentional 12 lb weight loss over past 6-8 weeks. Labs showed: serum calcium 12 (8.5-10.5) mg/dL, phosphorous 3.3 (2.5-4.5) mg/dL, PTH < 4 (10-60) pg/mL, 25 hydroxy Vitamin D 35 (31-80) ng/mL, 1,25 dihydroxy Vitamin D 57 (15-60) pg/mL, ALP 90 (40-150) U/L, 24 hour urine calcium 316 (100-300) mg, PTHrP 5.4 (n<2 pmol/L), serum free lambda light chains 513 (n < 26.3) mg/L, and positive free lambda light chains in urine. Positive Congo Red staining of focal amyloid accumulation from upper arm fat pad biopsy confirmed amyloidosis. Bone marrow biopsy showed 10% monoclonal plasma cells expressing lambda light chains. Diagnosis of multiple myeloma and AL amyloidosis was made. Chest Xray and heart ECHO were normal. Skeletal survey showed no lytic lesions. She became eucalcemic after treatment with borteozimib and zoledronic acid.

Discussion: There are several unique features about this patient's presentation. Firstly, while occult amyloidosis can occasionally be identified at the time of diagnosis of multiple myeloma, it is rare for a patient to present with classic manifestations of both diseases simultaneously. Secondly, hypercalcemia associated with multiple myeloma is commonly recognized as been driven by TNF-beta, IL-1 and IL-6 mediated osteoclastic bone resorption, while PTHrP is largely recognized as causative factor for humoral hypercalcemia of malignancy in solid tumors. Reports about expression of PTHrP in multile myeloma are limited to a few case studies.

Conclusion: Systemic amyloidosis may present simultaneously with multiple myeloma. PTHrP may contribute to the pathophysiology of hypercalcemia in multiple myeloma.

Abstract #646

INPATIENT SETTING HYPERCALCEMIA IN THE ELDERLY: MALIGNANCY?

Alvaro Puig Rodriguez, MD, Sanford Baim

Objective: Identify hypercalcemia due to vitamin D intoxication as a cause of life-threatening complications.

Methods: A case is described of severe hypercalcemia in an elderly man related to the ingestion of high doses of vitamin D prescribed by his physician.

Case Presentation: An 86 year old Ecuadorian male presented with a two week history of polyuria, lethargy, abdominal pain, and 10 pound weight loss. Initial laboratory testing revealed: serum calcium 14.9 mg/dL(8.5-10.5), albumin 3.2mg/dL(3.9-5), PTH intact 7.81pg/mL(10-65). SPEP, UPEP, and skeletal survey were normal. Additional

laboratory testing revealed: 25(OH)D >316 ng/mL(30-100), 1,25(OH)2D 74 pg/mL(18-72), PTHrP 11pg/mL(14-27). Initial treatment of hypercalcemia consisted of IV normal saline and pamidronate 60 mg IVx1. Due to onset of heart failure, IV fluids were held with worsening serum calcium. Endocrinology was consulted and after extensive questioning the patient admitted to have taken one 600,000 IU ampoule of vitamin D each week PO (Raquiferol by SpedrogCaillon Laboratories), as recommended by his physician in Ecuador for 6 weeks (total dose prior to being hospitalized was 3,600,000 IU). IV hydration, furosemide, and a second dose of pamidronate 90 mg x1 was initiated with volume status carefully monitored. The patient's mental status and physical condition progressively improved throughout the 1 month admission. Calcium levels were closely monitored twice per week in the outpatient setting.

Discussion: The majority of admissions for hypercalcemia are related with primary hyperparathyroidism or malignancy. Other causes include granulomatous diseases and side effects of medications. The institute of medicine considers Vitamin D intoxication of adults (70 years and older) as taking more than 60,000 IU per day. Several theories have been promoted to explain the hypercalcemia related to vitamin D toxicity: 1) increased concentration of 25(OH)D can bind to the vitamin D receptor (VDR) causing an increased effect on genetic expression, 2)25(OH)D may saturate the vitamin D binding protein with resulting increased tissue site availability, and 3) enhanced paracrine conversion of 25(OH)D to 1,25(OH)2D in local tissue. Differences in the pharmacokinetics between 25(OH)D and 1,25(OH)2D (potency, half life-15 days vs 15 hours, affinity to DBP, adipose tissue distribution, and slow turnover), may explain why the treatment of hypervitaminosis D induced hypercalcemia requires a longer than expected period of time to resolve.

Conclusion: Patients commonly self administer over-the-counter vitamin D preparations as well as high doses prescribed by their clinician thus emphasizing the importance of a thorough historical evaluation at the time of admission.

Abstract #647

MALE OSTEOPOROSIS SECONDARY TO HYPOGONADOTROPIC HYPOGONADISM

Andres Montanez-Flores, MD, Jose Garcia Mateo, MD

Objective: To present a case of marked osteoporosis from Kallmann Syndrome diagnosed in a young adult male with Slipped Capitis Femoral Epiphysis.

Case Presentation: A 24 years old male with slipped femoral capital epiphysis who referred exaggerated growth in last 3 years. On physical examination findings included high pitched voice, tall stature, thin with euchonoid body habitus, normal-sized thyroid, skin with lack of terminal hair growth on his face and chest, no gynecomastia or galactorrhea, firm testes, 4 mL bilaterally, micropenis, with Tanner stage G1, and anosmia. Laboratory tests showed total testosterone of 0.21 ng/dL(normal, 2.4-8.27 ng/dL), follicle-stimulating hormone(FSH) of 0.5 mIU/mL(normal, 1.4 - 18.1 mIU/mL), luteinizing hormone of 0.22 µIU/mL(normal, 1.5 - 9.3 mIU/mL), 25(oh)vit.D of 25.8 ng/mL(normal,30.0 - 100.0 ng/mL), and sex hormone binding globulin of 57mmol/L(normal, 10 - 50 mmol/L), the other tests were unremarkable. DXA scan showed a T-score of - 3.4 at L1-L4, and right hip T-score of - 1.8. MRI of brain and pituitary gland showed absent olfactory bulbs, otherwise unremarkable MRI of brain. Features were consistent with Osteoporosis in association with Kallman Syndrome. Patient was started with Testosterone Cypionate(100mg/mL)injection 1/2 IM every 2 weeks, Vit.D3 10,000 PO daily Mon-Fri for 4 weeks, and Calcium citrate + D (315-250) PO daily.

Discussion: Kallmann's Syndrome is a genetic disorder affecting 1 in 10.000 to 60.000 individuals characterized by gonadotropin deficiency associated with hyposmia or anosmia. Osteoporosis develops among these patients because the testosterone-mediated increase in bone density of adolescence fails to occur. Maintenance of bone integrity depends on the action of testosterone which promotes proliferation and differentiation of osteoblasts as well as inhibits osteoclast activity. However, the direct effect of testosterone is likely 25% of its effectiveness whereas its aromatization to estrogen contributes to the remaining 75%. Only a limited number of reports in the past implicated hypogonadism as the underlying cause of previously undiagnosed osteoporosis among young males. One of the primary treatment regimens for hypogonadism is testosterone replacement therapy, which helps not only to ameliorate the symptoms of hypogonadism, but to increase bone mineral density as well.

Conclusion: Osteoporosis is a frequently underestimated disease in men. Male hypogonadism is an important and treatable cause of osteoporosis. The present case emphasizes the importance of early diagnosis and management of this rare disease is essential to prevent severe osteoporosis.

Abstract #648

OSTEOPOROSIS IN NIGERIAN ADULTS: THE LOOMING EPIDEMIC

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Objective: General Objective- To determine the prevalence of osteoporosis in the study population. Specific Objectives- To measure the bone mineral density of the subjects and to determine any existing relationship between gender, age and T-score values.
Methods: Methods- This is an observational study in which 180 adult Nigerians were consequently screened using Dual X-ray Absorptiometry (DEXA) scan. The results were subjected to statistical analysis.
Results: Results- Of the 180 adults studied, 60 were males while 120were females. The mean age was 52.09 (SD 4) years (max.75, min. 30yrs), Mean weight was78.7(SD 3) kg, mean height was 161(SD8)cm while mean T-score was 1.7(SD 0.04). Of the men 27 (45%)were normal, 24 (40%) were osteopenic and 9(15%)were osteoporotic. Of the women 41 (34.2%) were normal, 44 (36.7%) were osteopenic while 35(29.2%) had osteoporosis. Overall 44 (24%) were osteoporotic, 68(38%) were osteopenic while 68(38%) were normal.
Discussion: It is postulated that after the age of 50 half of the women and a quarter of all men would have osteoporosis. Gradual decline in bone density mass probably starts from age 30. Up till now osteoporosis is erroneously thought to be non-existent in Nigeria due to available sunshine and rich diet in fruits and fish. Although there are studies with similar results as ours from some Asian and African countries where Purdah system is common, our study was carried out in Lagos where Purdah system is non-existent or minimal. Serum levels of Vit D or other biomarkers could have been cost effective but are unavailable in Nigeria. Our finding of high prevalence of osteoporosis of 24% in this study, has therefore painted a picture of a looming epidemic that should attract attention.
Conclusion: Screening of adult Nigerians and particularly the elderly, is therefore suggested to primary care physicians for early detection of osteoporosis by the most cost effective means available, in order to institute preventive therapy.

Abstract #649

SEVERE HYPOCALCEMIA AFTER INITIATING SUNITINIB THERAPY FOR METASTATIC RENAL CELL CARCINOMA

Benjamin Dennis, MD, Edward Chin, MD

Objective: To present a case of sunitinib associated hypocalcemia and discuss possible etiologies.
Case Presentation: A 55 year old woman with metastatic renal cell carcinoma presented to the emergency department with progressive fatigue, perioral and extremity tingling and carpopedal spasms over the preceding week. Sunitinib was started 17 days prior to presentation. On presentation total calcium was 6.1 mg/dL (8.7-10.4), albumin 4.0 g/dL, ionized calcium 2.8 mg/dL (4.5-5.3), parathyroid hormone 117.6 pg/L (11.1-79.5), magnesium 1.6 mg/dL (1.3-2.7), phosphorus 4. mg/dL 8 (2.4-5.1), 25-hydroxyvitamin D 35.6 ng/mL (25-80) and 1,25-dihydroxyvitamin D 72 pg/mL (18-78). She required intravenous calcium and magnesium followed by outpatient treatment with calcitriol and calcium which was decreased after discontinuation of sunitinib.
Discussion: Sunitinib, an oral small molecule receptor tyrosine kinase (RTK) inhibitor, is an ATP analogue that competes with ATP binding to the catalytic site of a number RTK's, platelet-derived growth factor receptors (PDGFRα and PDGFRβ), vascular endothelial growth factor receptors (VEGFR1, VEGFR2 and VEGFR3), stem cell factor receptor (KIT), Fms-like tyrosine kinase-3 (FLT3), colony stimulating factor receptor Type 1 (CSF-1R), and the glial cell-line derived neurotrophic factor receptor (RET). Sunitinib has also been shown to decrease activity of a downstream tyrosine kinase signaling molecule, SRC kinase. Src kinase is critical for activation of the human Transient Receptor Potential Vanilloid Type 6 (TRVP6) channel. TRVP6 is a calcium-selective channel critical for transcellular intestinal calcium absorption. Hypocalcemia was temporally associated with sunitinib initiation in our patient and improved with magnesium and calcitriol therapy prior to stopping sunitinib. Imitanib, an RTKi targeting BCR-ABL, PDGFR and KIT, is reported to cause a relative hypocalcemia with muscle cramps. Bone and calcium side effects of RTK inhibitor (RTKi) include increased PTH, secondary hyperparathyroidism, hypocalcemia, tetany, decreased bone turnover and renal magnesium wasting. In our patient hypocalcemia may be due to a relative magnesium deficiency as demonstrated by the modest PTH elevation considering the profoundly low ionized calcium and the lack PTH action. However, an effect of sunitinib on enteric calcium absorption cannot be excluded.

Conclusion: RTK's are widely used to successfully treat a variety of malignant diseases. Their nonselective action may result in severe endocrine related side effects and clinicians should be aware of the potential for development of severe hypocalcemia.

OBESITY

Abstract #700

RARE CASE OF HYPERCALCEMIC OBESITY - VITAMIN D DEFICIENCY IN MISSISSIPPI OBESE CHILDREN

George Moll, MD, PhD, Michael Torchinsky

Objective: We describe utility of a basic metabolic panel to diagnose a parathyroid adenoma in an obese adolescent male. Correction of hyperparathyroidism did not resolve his obesity but did halt his weight gain for several years. We hypothesize vitamin D deficiency occurs in a majority of obese Mississippi children.
Methods: We report clinical, laboratory and radiology results leading to diagnosis of a parathyroid adenoma in a 16-year-old obese (body mass index [BMI] 40kg/M2) male presenting with prolonged lethargy, polydypsia and polyuria. He noted improved obesity management following parathyroid adenoma resection. We searched our childhood obesity (BMI >95thpercentile) database for 25-hydroxy-vitamin D deficiency (<30ng/ml) over the past 2-years.
Case Presentation: Our obese male's test results revealed normal blood sugars, hemoglobin A1c 4.9% but hyper-calcemia 12.2mg/dl, hypo-phosphatemia 2.8mg/dl and elevated parathyroid hormone (IPTH) 139pg/ml. Radiology imaging located a parathyroid adenoma. Surgical removal of histology identified parathyroid adenoma returned his serum IPTH, calcium, phosphorous to normal. His weight stabilized at 308 lb for up to 4 years post-surgery. Our database search identified 112 childhood obesity referrals with 25-hydroxy-vitamin D levels: 70.5% had vitamin D deficiency consisting of 69% of 42 boys ages 5 to 18 years (BMI 34.8+3.9) and 71% of 70 girls ages 3 to 18 years (BMI 38.5+7.1).
Discussion: Obesity is a national health concern often starting in childhood. A basic metabolic profile on an obese adolescent male assisted our diagnosis of hypercalcemia associated with a parathyroid adenoma. Research has identified a mechanism for IPTH promotion of obesity through stimulating production of intracellular 1,25-di-hydroxy-vitamin D that stimulates intracellular lipogenesis and reduces thermogenesis. Through this mechanism, Vitamin D deficiency can contribute to obesity by promoting secondary hyperparathyroidism. As the only academic children's hospital in Mississippi, we surveyed our childhood obesity referrals for vitamin D deficiency as a treatable condition to assist their care.
Conclusion: Parathyroid adenoma is rarely reported in adolescence, and polyuria with fatigue and obesity are often due to diabetes mellitus. Attention to more than glucose can assist efficient management of obesity. We conclude a majority of obese adolescents can be found to have vitamin D deficiency as a treatable contributing factor through a described mechanism for primary or secondary hyperparathyroid contribution to obesity. Basic blood tests for obesity assessment can reveal rare diagnoses and contributing factors that when adequately treated improve long-term success with obesity management.

Abstract #701

PEDOMETER APPLICATIONS FOR THE IPHONE AND WEIGHT LOSS: MORE TECHNOLOGY THAN EVIDENCE?

Armand Krikorian, MD, Joumana Chaiban, MD

Objective: Several studies repeatedly showed that the use of pedometers is associated with increased physical activity and a decrease in BMI. There has been a recent explosion of pedometer applications that make use of the built-in accelerometer of the iPhone's built-in technology to promote weight loss. Our aim is to review current available iPhone pedometer (IP) applications and the evidence linking them to physical activity and weight loss.
Methods: We searched the iPhone App store using the keyword 'pedometer' and reviewed all available applications both free and paid. We excluded applications that were aimed solely at logging steps but had no built-in function to measure strides. In addition we conducted a Pubmed search using the keywords 'accelerometer' and 'iPhone'.
Case Presentation: A Total of 204 applications were found in the App store when searching for pedometer. About half of them were paid, with the free often being 'trial' or limited-use versions of paid ones. Most were making use of the iPhone's built-in accelerometer alone or in combination with GPS-tracking. Applications that relied only on GPS to estimate number of steps were deemed too inaccurate due to poor resolution of GPS when using 3G networks versus Wi-fi. The validity and accuracy of accelerometer based devices and applications was evaluated in small scale studies in the medical literature. Controversial results have been reported. Bergman et al found that IP apps were not accurate in counting steps when worn at pocket, waist, or arm. However, Fujiki showed that the IP has a good correlation with physical activity, the highest being when the phone is attached to the waist (for the waist, jacket side pocket, jacket top pocket, handbag, backpack and pants pocket, the correlation coefficients were respectively 0.8, 0.67, 0.63, 0.69, 0.66 and 0.74) and Manohar et al found that the IP was able to distinguish change in walking activity reliably with the following correlations: handbag: 0.91; Jacket side pocket: 0.89; Pants

pocket: 0.88; Jacket front pocket: 0.84, pants pocket: 0.88.
Discussion: There is a need to differentiate between GPS-based and accelerometer-based iPhone pedometers, as the latter appear to be more accurate and studied more extensively. While some studies appear to indicate that IPs can reliably detect movement and have been associated with physical activity, no studies have looked at IPs and weight loss. Moreover, all studies to date have been conducted in a laboratory setting and not in a 'real life' environment.
Conclusion: IP appear to hold promise for use in weight loss promotion. Further large scale studies are needed to be able to determine the real potential of these applications in weight management

Abstract #702

MULTIDISCIPLINARY APPROACH AS BEST PRACTICE FOR THE TREATMENT OF OBESITY

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Objective: Obesity is a gateway disease that contributes to many medical problems including: coronary heart disease, stroke, type 2 diabetes, cancers as well as mental disorders. In 2008, the overall medical care costs related to obesity in the US were $147 billion. Despite the availability of guidelines to treat obesity in the US, the prevalence of obesity is still rising, estimated to affect 165 million Americans by 2030. Traditional medical management of obesity patients is not enough and innovative strategies to treat obesity are critically needed. A multidisciplinary weight loss clinic, the Energy BALANCE (Behavior And Lifestyle Assessment with Nutrition Centered Education) was established at the University of Nevada, Reno in 2010. This clinic utilizes a team approach to evaluate, motivate and effectively treat patients with obesity. This program evaluation is to assess whether a multidisciplinary team is an effective way to treat obesity.
Methods: The Energy BALANCE clinic is led by a team of physicians, dieticians and an exercise specialist who evaluated and treated obesity patients in a 12 week pilot program. 119 overweight or obese patients who were referred to the clinic by PCPs were studied. The study's primary outcome was the change in patients' body weight and BMI before and after the 12 weeks treatment period. Fasting glucose, HgbA1C, and lipid panel were also evaluated. Data was collected and analyzed to assess the effect of the multidisciplinary weight loss program and compared to other studies.
Results: All patients (N=119) with 12 weeks follow up data were included in the study analysis. On average, body weight decreased by 13.1±8.3 lbs and BMI decreased by 5.27±2.83% ($p < 0.00001$) during the 12 week period with an average cost of $900/patient. Patients who visited the clinic more often lost more weight. Patients with baseline BMI<35 lost 7.8±5.2 lbs and patients with baseline BMI>35 lost 15.0±8.3 lbs. Fasting glucose significantly decreased from 103.2±39.1 to 95.2±22.8 ($p=0.004$). Patients with diabetes have significant decrease in HgbA1C from 7.89±1.46 to 7.05±1.04 ($p=0.006$, $n=15$). Significant decreases in TG, TC and LDL were seen at 9%, 15% and 8%, respectively.
Discussion: The program evaluation suggests that a multidisciplinary team is an effective approach to treat obesity. Significant decreases in body weight, BMI, HgbA1c, lipid parameters were seen in a relatively short period of time.
Conclusion: We anticipate a multidisciplinary team approach to be a best practice for obesity management.

Abstract #703

VIPOMA IN A PATIENT POST-GASTRIC BYPASS SURGERY

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Objective: We report a case of pancreatic neuroendocrine tumor secreting VIP and serotonin and also staining for somatostatin in a patient who underwent gastric bypass surgery.
Case Presentation: A 54year old female with history of Roux En Y surgery for weight loss in 2001, asthma, iron deficiency anemia, and gastric ulcers (diagnosed 6 years after gastric bypass) presented with watery diarrhea, abdominal pain, and weight loss. In 2009, when she underwent cholecystectomy for right upper quadrant pain, her potassium was 3.1mmol/L and potassium supplement was started. In 2010, she had nausea, vomiting, diarrhea, potassium 2.5mmol/L, and was started on spironolactone. CT of abdomen showed mass in pancreatic tail (biopsy: pancreatic neuroendocrine tumor) and liver lesions. Labs showed potassium 2.5mmol/L, chloride 116mmol/L, bicarbonate 9mmol/L, chromogranin A 44ng/mL (1.9-15ng/mL), VIP 368.5pg/mL (23-63pg/mL), somatostatin 16pg/mL (10-22pg/mL), 24 hour urine 5-HIAA35.5 mg (0-14mg), and gastrin 85pg/mL on proton pump inhibitor (0-100pg/mL). Octreotide scan showed pancreatic tail mass with metastatic liver lesions. Subcutaneous octreotide improved diarrhea. She underwent distal pancreatectomy, splenectomy, and liver wedge resection. Pathology was consistent with well-differentiated endocrine carcinoma, staining positive for VIP and weakly for somatostatin. Post surgery, potassium improved to 5.2mmol/L, chloride

98mmol/L, bicarbonate 35mmol/L, VIP 42.6pg/mL, and somatostatin 35pg/mL. Two months later, she underwent left lateral liver wedge resection, and open radiofrequency ablation to liver lesions.

Discussion: After surgical, radiofrequency ablative procedure, she had resolution of nausea, abdominal pain, diarrhea, with weight gain of 12 kgs over 7 months. Surveillance MRI of liver fused with octreotide scan in late 2011 showed new metastatic liver lesions. Octreotide LAR every month was started. She awaits re-evaluation for radiofrequency ablation to new liver lesions.

Conclusion: Gastric bypass surgery is associated with benefits as well as adverse outcomes. There have been case reports of pancreatic neuronendocrine tumors such as insulinoma and carcinoid syndrome after gastric bypass surgery. Our patient presented with Watery Diarrhea Hypokalemia Achlorhydria syndrome resulting from metastatic pancreatic neuroendocrine tumor staining for VIP. There was clinical, biochemical and histochemical evidence of increased serum VIP; biochemical evidence of carcinoid; and histochemical evidence of somatostatin. This is the first case report of VIP secreting tumor in a patient with previous gastric bypass surgery.

Abstract #704

SIGNIFICANT WEIGHT LOSS (WL) WITH CONTROLLED-RELEASE PHENTERMINE/ TOPIRAMATE (PHEN/TPM CR) IS ASSOCIATED WITH SIGNIFICANT REDUCTIONS IN CARDIOMETABOLIC PARAMETERS OVER 108 WEEKS

Nancy Bohannon, MD, FACP, Craig Peterson

Objective: Obesity is associated with chronic comorbidities, such as atherosclerosis and type 2 diabetes. PHEN/TPM CR has previously been shown to provide significant WL compared with placebo (PBO) through 56 weeks in the CONQUER study, a double-blind, Phase 3 trial of PHEN/ TPM CR in 2487 overweight/obese adults with ≥2 weight-related comorbidities. The long-term efficacy and safety of PHEN/TPM CR have been evaluated through 108 weeks in an extension study (SEQUEL) of CONQUER completers.

Methods: Subjects enrolled in this double-blind, PBO-controlled extension study continued with their original CONQUER randomized treatment for an additional 52 weeks: PBO (n=227), PHEN 7.5 mg /TPM CR 46 mg (7.5/46; n=153), or PHEN 15 mg/TPM CR 92 mg (15/92; n=295). Primary efficacy variables were least-squares (LS) mean percent WL and percentage of subjects with ≥5% WL from CONQUER baseline to Week 108. Additional variables included change in glycated hemoglobin A1c (HbA1c), fasting insulin (FI), homeostasis model assessment-insulin resistance (HOMA-IR), high-density lipoprotein (HDL), and triglycerides (TG) from CONQUER baseline to Week 108. Subanalyses were also conducted on subjects >65 years.

Results: At baseline, mean weight was 101.7 kg and mean body mass index was 36.1 kg/m2. After 108 weeks, LS mean percent WL for PBO, 7.5/46, and 15/92, respectively, was 1.8%, 9.3%, and 10.5% (P<.0001 vs PBO). Significantly more PHEN/TPM CR-treated subjects achieved ≥5% WL vs PBO (P<.0001 for all comparisons). At Week 108, LS mean change in HbA1c was 0.16%, 0.01%, and 0% for PBO, 7.5/46, and 15/92, respectively (P<.005 vs PBO for both doses); FI (μIU/ mL) was -2.6, -5.3, and -5.2 for PBO, 7.5/46, and 15/92, respectively (P≤.005 vs PBO for both doses); and HOMA-IR was -0.7, -1.5, and-1.6 for PBO, 7.5/46, and 15/92, respectively (P<.05 vs PBO). Significant improvements were also seen with PHEN/TPM CR in HDL (P<.0001 vs PBO for 15/92) and significant reductions in TG (P≤.0005 vs PBO for both doses). PHEN/TPM CR was generally well tolerated with 84% of subjects completing the study on drug. Common adverse events were paresthesia, dry mouth, and constipation. Whether studied in the overall population, as described here, or only in those >65 years, the magnitude and direction of efficacy and safety outcomes were not meaningfully different.

Discussion: PHEN/TPM CR produced significant WL and improvements in cardiometabolic risk factors through 108 weeks of treatment.

Conclusion: This indicates sustained efficacy in the treatment of obesity and potential for amelioration of comorbidities.

Abstract #705

DECREASED PROGRESSION TO TYPE 2 DIABETES MELLITUS (T2DM) AFTER 1 YEAR OF TREATMENT WITH CONTROLLED-RELEASE PHENTERMINE/TOPIRAMATE (PHEN/TPM CR) IN OBESE SUBJECTS WITH PREDIABETES

W. Timothy Garvey, MD, Wesley Day

Objective: Individuals with Prediabetes (impaired glucose tolerance [GT] or impaired fasting glucose [FG] that are below the levels required for T2DM diagnosis) are known to be at high risk of progression to T2DM. Weight loss (WL) is an important strategy for improving glycemic parameters and delaying progression to T2DM in overweight/obese individuals with Prediabetes. PHEN/TPM CR previously demonstrated significant WL in the 56-week CONQUER study of obese patients with comorbidities.

Methods: This was a post-hoc analysis of a prespecified population, subjects with Prediabetes at baseline in the CONQUER study (impaired FG ≥100 to ≤125 mg/dL or impaired GT ≥140 to ≤199 mg/dL measured by oral GT test [OGTT]). Subjects were randomized to placebo (n=994), PHEN 7.5 mg/TPM CR 46 mg (7.5/46; n=498), or PHEN 15 mg/TPM CR 92 mg (15/92; n=995) for 56 weeks. All subjects received lifestyle intervention for WL and were managed to standard of care throughout. Evaluations included change in weight, HbA1c, FG, fasting insulin (FI), and progression to T2DM, defined as 2 consecutive FG levels ≥126 mg/dL, or glucose levels ≥200 mg/dL (2-hour OGTT), or taking antidiabetic medications at endpoint. Further subgroup analyses were conducted on subjects >65 years.

Results: In total, 447 (45.0%), 238 (47.8%), and 434 (43.6%) subjects in the placebo, 7.5/46, and 15/92 groups, respectively, had Prediabetes at baseline. Baseline characteristics were similar between groups: mean age, 51.8 years; 67.7% female; mean body mass index, 36.7 kg/m2; mean HbA1c, 5.7%. At 56 weeks, in the placebo, 7.5/46, and 15/92 groups, respectively, change in weight was -2.2%, -8.9%, and -11.0% (*P*<.0001 vs placebo); change in HbA1c was 0.06%, -0.03%, and -0.04% (*P*<.0001 vs placebo); change in FG (mg/dL) was -1.5, -3.7 and -5.1 (*P*=.0055 for 7.5/46, *P*<.0001 for 15/92 vs placebo); and change in FI (μIU/mL) was 2.6, -4.0, and -4.5 (*P*=.0028 for 7.5/46, *P*=.0001 for 15/92 vs placebo). Annualized incidence rate of T2DM was 4.1%, 2.7%, and 2.2% for placebo, 7.5/46, and 15/92, respectively. PHEN/TPM CR was generally well tolerated, with 64% of subjects with Prediabetes completing the study on drug. Common adverse events were paresthesia, dry mouth, constipation. In general, the magnitude and direction of efficacy and safety outcomes were not meaningfully different between the overall population and subjects >65 years.

Discussion: In subjects with Prediabetes, PHEN/TPM CR led to significant WL and improvements in glycemic parameters vs placebo over 56 weeks and lower annualized incidence rates of T2DM in all populations assessed.

Conclusion: This indicates the potential for PHEN/TPM CR to decrease progression to T2DM.

Abstract #706

RELATIONSHIP OF EPICARDIAL FAT WITH OBESITY AND HYPERLIPIDEMIA

Ritu Madan, MBBS, Manu Kaushik, Venkata Alla, Alok Saurav, Aiman Smer, Shariq Khan, Mark Homberg, Syed Mohiuddin

Objective: Epicardial adipose tissue (EAT) is the metabolically active fat around the heart that is suggested to play a role in pathogenesis in cardiac disease and has been shown to be associated with the extent of coronary artery disease (CAD) and may play a role in development of atrial fibrillation.The objectives of this study were following- 1. To study the association of EAT thickness as assessed by 2D echocardiography with obesity. 2. To study the association of EAT thickness with lipid profile.

Methods: We retrospectively reviewed echoes on 632 patients to calculate Epicardial adipose tissue (EAT) thickness at pre specified locations over the right ventricle. Patients with satisfactory image quality were included to evaluate association of maximum EAT thickness with Body mass index(BMI) and lipid profile.

Results: 471 patients had satisfactory echocardiograms for calculation of Epicardial fat and data on BMI. There was a weak but significant correlation between BMI and maximal EAT thickness(Pearson r=0.232, p<0.001). Patients were then divided into three groups : normal weight (N=107,mean BMI 22.3±2.1kg/m2), overweight(N=167 ,mean BMI 27.4±1.4kg/m2) and obese(N=197 , mean BMI 36.1±5.9kg/m2). Between the groups ANOVA was consistent with significant differences in maximum EAT thickness between normal weight (mean: 0.64±0.26mm), overweight (mean: 0.72 ±0.24mm) and obese (mean: 0.79±0.26mm) subjects. Posthoc Tukey's test showed significant differences between normal weight and overweight (p= 0.04), overweight and obese (p=0.006), normal weight and obese(p<0.001) subjects. Clinical data and lipid profile were available in 270 patients. EAT thickness did not correlate significantly with presence or absence of hyperlipidemia (p=0.31). Similarly, no significant association was found between EAT thickness and total cholesterol levels or EAT thickness and triglyceride levels.

Discussion: The volume of epicardial fat as assessed by computerized tomography has been clearly shown to be associated with obesity parameters and metabolic syndrome.In this study, we used echocardiography to assess epicardial fat, a technique which is much cheaper and more commonly performed to assess cardiac disease. Lack of association with hyperlipidemia with EAT thickness may be the result of the fact that some of these patients were on statins. Alternatively it is possible that EAT thickness is not affected by lipid abnormalities.

Conclusion: EAT thickness by 2D echocardiography was found to correlate significantly with BMI. Maximal EAT thickness was found to differ between normal weight, overweight and obese subjects.

Abstract #707

EFFECT OF EARLY WEIGHT LOSS ON TYPE 2 DIABETES THROUGH SURGICAL INTERVENTION 2 YEARS AFTER GASTRIC BANDING

Ted Okerson, MD, Sunil Bhoyrul, Helmuth Billy, Christopher Cornell

Objective: Although weight loss can effect significant improvements in glycemic control, diet, exercise and/or pharmacotherapy are usually not effective at inducing substantial, durable weight loss. Laparoscopic adjustable gastric banding (LAGB) is an established bariatric surgical treatment option for Type 2 Diabetes (T2D) patients with a BMI > 30 mg/kg2. This study reports changes in T2D observed 2 years after LAGB, and the effect of the duration of T2D on likelihood of remission.

Methods: This is an interim analysis of subjects participating in the LAP-BAND AP® Experience (APEX) trial, an ongoing 5-year, prospective, multi-center, open-label, observational study. This analysis describes subjects who reported T2D requiring daily medical therapy at baseline and who have completed their 2 year post-operative visit. "Remission" is defined as elimination of hypoglycemic medication and improvement as reduction in hypoglycemic medication.

Results: There were 89/395 (22.5%) subjects with T2D at baseline, 66 of which had data sufficient to assess T2D status at 2 years. Remission and improvement of T2D was achieved by 48.5% (n=23) and 47.0% (n=24) of this population respectively, with 4.5% (n=3) experiencing no change. Subjects with remission or improvement had an average T2D duration of 4.0 and 6.7 years respectively (p=0.03 between groups). In contrast, those with no change averaged 8.9 years with T2D. Baseline BMI was 43.7, 44.4, and 41.0 in the remission, improvement and no change groups, with respective changes in BMI and % weight loss of -10.1/-22.8, -8.4/-18.5 and -8.0/-18.7 (no significant differences between the groups). Interestingly, percent excess weight loss (%EWL) at 2 years was significantly greater in the remitted (-55.7%) versus improved (42.9%) group (p=0.03) with %EWL being marginally correlated with change in T2D status (logistic regression; p=0.05). APEX subjects experienced resolution or improvement of other comorbidities of obesity: Hypertension (HTN) 90.8%, hyperlipidemia 76.9%, obstructive sleep apnea 86.0%, gastroesophageal reflux disease 91.0%, osteoarthritis 92.7%, and depression 75.4%.

Discussion: These data suggest that duration of T2D appears to affect the likelihood of improvement or remission through gastric banding, suggesting that early surgical intervention be considered in obese patients with T2D. The amount of weight loss also contributes to disease remission. Improvements were also observed in other cardiovascular risk factors such as HTN and hyperlipidemia.

Conclusion: Laparoscopic Adjustable Gastric Banding could be considered as a viable adjunctive treatment option for individuals with obesity and T2D.

OTHER

Abstract #800

COST CONSCIOUSNESS IN ENDOCRINOLOGY - LEVEL OF AWARENESS AMONG HEALTH CARE PROVIDERS AND NEED FOR EDUCATION

Nikhil Gupta, MD, MPH, Shane LeBeau

Objective: The rising cost of health care is an ever increasing concern. Cost awareness can influence diagnostic testing and treatment, and has been shown to lower health care costs. The aims of this study were to assess the awareness of health care costs among endocrine physicians and nurses at an academic medical center and to determine if the consideration for cost consciousness influences the number of tests ordered.
Methods: We conducted an online survey among attending physicians, fellows and nurses in the endocrine division at our institution. We asked them to estimate the hospital charges and CMS (Center for Medicare and Medicaid) reimbursements for common endocrine labs (basic metabolic panel, hemoglobin A1c, TSH, free T4, thyroid antibodies, 25-OH vitamin D, PTH, testosterone, cortisol and ACTH) and imaging tests (I-123 uptake/scan, CT abdomen, MRI pituitary and neck ultrasound). A hypothetical patient with symptoms of thyrotoxicosis was provided and physicians were asked to choose from a panel of tests as to what they would order to make a diagnosis. Physicians were then asked the same question keeping "cost-effectiveness" in mind. The survey was sent by email and responses were anonymous.
Results: Of the 30 respondents, 14 (47%) were attendings, 7 (23%) were fellows in training and 9 (30%) were nurses. The percentage of respondents who accurately estimated CMS reimbursement (75% to 125% of that posted on CMS website) ranged from 3%-33% for various lab tests, and 10%-27% for imaging studies. Accurate estimates were even less likely for hospital charges with mean estimates being markedly lower than actual charges. No difference in accuracy of estimates was seen between attendings, fellows and nurses. In the hypothetical patient scenario, the average number of tests selected by physicians to make a diagnosis was 4.3. This number decreased to 2.9 after being prompted to consider "cost-effectiveness" ($p<0.05$).
Discussion: The low level of awareness regarding costs among health care providers may be related to the difficulty in obtaining such information. Additionally, the significant difference in the amount charged and reimbursement received by health care institutions leads to a certain lack of transparency in terms of cost data. The low level of awareness may also be attributed to a degree of apathy among providers towards costs of care due to the perceived notion that everything is being covered by insurance.
Conclusion: Cost awareness among health care providers was low. Accuracy of cost estimates did not differ with level of training. Cost consciousness can influence the pattern of ordering tests which may ultimately help decrease health care costs.

Abstract #801

THE RACE TO STOP THE FALLS

Matilda Malm, MD, Tanu Pandey

Objective: Orthostatic hypotension is common and multifactorial in elderly patients, especially those institutionalized. We describe an elderly patient with orthostatic hypotension presumed to be from panhypopituitarism but later found to be a result of autonomic dysfunction due to multiple system atrophy (MSA).
Case Presentation: A 74 year old man complained of recurrent lightheadedness on standing for 3 weeks. Physical examination was significant for orthostatic hypotension and right hand tremors. Laboratory evaluation revealed evidence of panhypopituitarism with low morning cortisol, ACTH, prolactin and IGF, low TSH with normal free T4, very low FSH, LH and testosterone. A brain MRI revealed a 20 x 9 x 15 mm pituitary macroadenoma with no mass effect on the optic chiasm. A diagnosis of a non-functioning pituitary macroadenoma with subsequent panhypopituitarism was made and he was prescribed replacement therapy with levothyroxine, hydrocortisone and testosterone injections. Two months later he continued to have persistent lightheadedness and syncopal episodes with a new complaint of urinary frequency. Physical examination was remarkable for significant orthostatic hypotension and bradykinesia. These findings were attributed to non-compliance with medications. A month after strict compliance he remained symptomatic with worsening dizziness, syncope and urinary incontinence as well orthostatic hypotension, rigidity and bradykinesia on examination. Given the constellation of Parkinsonism, autonomic dysfunction and urinary symptoms, a diagnosis of MSA was considered. He was prescribed fludrocortisone and at 3 months follow up he denied further syncopal episodes.
Discussion: Orthostatic hypotension is encountered commonly and is most frequently observed in the elderly. Presence of extrapyramidal findings should prompt consideration of neurogenic etiologies like MSA. MSA is an adult onset, sporadic, progressive, neurodegenerative disease characterized by Parkinsonism, autonomic failure, cerebellar features and urogenital dysfunction. The definite diagnosis is based on post-mortem pathology. The defining neuropathology is an α-synucleinopathy.

Management is mainly symptomatic. Fludrocortisone can be used for management of orthostatic hypotension due to autonomic failure in MSA.
Conclusion: It is critical that clinicians recognize the underlying cause of orthostatic hypotension in the elderly and prescribe appropriate treatment to decrease the risk of complications like stroke, falls, and coronary events

Abstract #802

SEVERE HYPOCALCEMIA RELATED TO CHEMOTHERAPY IN BURKITT'S LYMPHOMA

Sarvpreet Ahluwalia, MD, Monica Agarwal, MD

Objective: To describe a rare case of hypocalcemia in Burkitt's lymphoma after receiving combination chemotherapy. The hypocalcemia was not attributable to tumor lysis syndrome.
Case Presentation: A 40 year old man was admitted to the hospital for weight loss and shortness of breath. He had spontaneous tumor lysis syndrome (TLS) on presentation. The biochemical evaluation revealed acute kidney injury (serum creatinine 5.4 mg/dL), hyperphosphatemia (12.3 mg/dL), hyperkalemia (5.5 mEq/L) and hyperuricemia (20.4 mg/dL). On admission the corrected calcium for albumin was 9.5 mg/dL (8.6-10.2 mg/dL). He was treated with rasburicase, allopurinol and hemodialysis for the management of spontaneous TLS. He was subsequently diagnosed with Burkitt's lymphoma on bone marrow biopsy. He received combination chemotherapy with Cyclophosphamide, Vincristine, Doxorubicin and Methotrexate(CODOX-M). The serum calcium started to decline four days after initiation of the chemotherapy. He developed severe hypocalcemia with total corrected calcium of 6.2 mg/dL and ionized calcium 0.71 mmol/L (1.15-1.33 mmol/L). He had been off hemodialysis for a few days with resolution of TLS and near full recovery of renal function. The intact parathyroid hormone(PTH) was 337 pg/mL (12-88 pg/mL), magnesium was 1.4 mg/dL (1.6 -2.6 mg/dL) and serum phosphorus was 2.3 mg/dL (2.5-4.5 mg/dL). The 25-hydroxyvitamin D level was 19 ng/ml (30-80 ng/mL) and 1, 25-hydroxyvitamin D level was 22 pg/ml (15-75pg/ml). The calcium was aggressively replaced with calcium gluconate infusion, oral calcium citrate and calcitriol. This intervention gradually restored normocalcemia. Seven weeks after the completion of chemotherapy, the calcium remained normal despite discontinuation of the calcium supplements. Six months later, patient was readmitted with lymphoma recurrence. He was deemed unsuitable for chemotherapy and died subsequently.
Discussion: The TLS did not account for the severe hypocalcemia in our patient as it had already resolved prior to the decline in the calcium level. The elevated PTH level along with hypocalcemia indicated a state of PTH resistance. This was caused by chemotherapeutic agents leading to hypocalcemia. This refractoriness of bone and renal tubules to PTH was probably potentiated by hypomagnesemia. Hypocalcemia in our patient was associated with administration of the chemotherapeutic agents in conjunction with the decline in the magnesium levels. Furthermore, the calcium returned to normal a few weeks after completion of chemotherapy.
Conclusion: Although malignancy related severe hypocalcemia is rare but it is important to recognize that hypocalcemia can potentially occur from certain chemotherapeutic agents, even in the absence of TLS.

Abstract #803

A MIND'S DISGUISE: THYROTROPH HYPERPLASIA MIMICKING PITUITARY ADENOMA

Shveta Gandhi, DO, Sunil Asnani

Objective: 1. To describe a case of pituitary thyrotroph hyperplasia mimicking a pituitary adenoma. 2. To discuss the changes seen in thyrotroph hyperplasia.
Case Presentation: A 60-year-old male with a history of post radioactive hypothyroidism was found to have 12mm x 12mm x 12mm pituitary macroadenoma on MRI brain ordered for a follow up for a history of CVA. Patient reported fatigue, dry skin, muscle aches and soreness along with a 20-pound weight gain. Directed questioning revealed a non compliance with levothyroxine for the last 18 months. Examination revealed no neurological deficits. Laboratory values: TSH 126.20 (0.4 and 5.0) µIU/mL, T4 1.6 (4.5 to12.5) µg/dL, and total T3 52 (80 to 200) ng/dL. Prolactin level was 21.5 (2.1 to 17.7) ng/mL. Serum cortisol, ACTH, GH, IGF-1, LH, FSH and testosterone were all within reference range. He was started on Levothyroxine 125 mcg daily. Repeat MRI of pituitary 3 months later showed a decrease in the size of the abnormality seen on prior imaging with a completed resolution of the abnormality at 10 months post thyroid hormone treatment reinitiation.
Discussion: Thyroid hyperplasia, a rare cause of pituitary enlargement is usually associated with long standing primary hypothyroidism. This hyperplasia is usually asymptomatic; however, symptoms of pituitary mass may be present. Duration of several years has been reported as the time required for pituitary enlargement; regression may take several months once thyroid hormone replacement is started. Thyrotrophs comprise approximately 5% of adenohypophysial cells. Increase in the secretion of TSH markedly affect the morphology and increase the number of

the thyrotrophs. Thyrotroph hyperplasia can be nodular or diffuse, but it is more commonly nodular. These large thyrotrophs have an abundant vacuolated acidophilic cytoplasm, eccentric nucleoli, prominent cytoplasmic processes and large, strongly PAS-positive lysosomes. Electron microscopy reveals dilatation and vesiculation of endoplasmic reticulum, a prominent Golgi complex, and normal mitochondria. Regression of the morphological changes seen in the pituitary is partly due to apoptosis and partly due to reversal of transdifferentiation.
Conclusion: Patients with primary hypothyroidism with pituitary mass should be given a sufficient trial with L-thyroxine before considering surgery. The so-called 'pituitary-mass' caused by thyrotroph hyperplasia is completely reversible in most instance with L-thyroxine replacement therapy.

Abstract #804

SUBCLINICAL ATHEROSCLEROSIS AND ARTERIAL STIFFNESS ARE ASSOCIATED WITH ENDOGENOUS TESTOSTERONE IN HEALTHY RECENTLY MENOPAUSAL WOMEN

Eleni Armeni, M.D., Kimon Stamatelopoulos, Maria Creatsa, Dimitrios Rizos, Georgios Georgiopoulos, Maria Kazani, Andreas Alexandrou, Christos Papamichael, Irene Lambrinoudaki

Objective: Although endogenous sex hormones may affect the cardiovascular risk profile in postmenopausal women, the results still remain controversial. We aimed to examine associations between circulating sex hormone levels and subclinical atherosclerosis by assessing surrogate markers of vascular function and structure.
Methods: This cross-sectional study recruited 120 healthy recently postmenopausal women, aged 41-60 years. Fasting venous blood samples were drawn for biochemical and hormonal evaluation, including levels of follicle-stimulating hormone, luteinizing hormone, estradiol, testosterone, sex hormone-binding globulin, dehydroepiandrosterone sulfate (DHEAS) and Δ4-androstenedione. We assessed blood pressure and anthropometric parameters, calculating the body mass index (BMI). Ultrasound evaluations included indices of arterial structure and function, namely carotid and femoral intima-media thickness (IMT), atheromatous plaques presence as well as flow-mediated dilation of the brachial artery, carotid-femoral pulse wave velocity (PWV) and augmentation index (AI). Possible associations between circulating sex hormones and surrogate markers of vascular function and structure were investigated.
Results: Total testosterone predicted significantly mean values of common carotid artery (CCA) IMT and combined IMT (β=0.376 and β=0.189, $p<0.001$ and p=0.035; respectively). Similarly, free androgen index predicted significantly mean levels of CCA-IMT (β=0.236, p=0.014) and PWV (β=0.254, p=0.027), after adjusting for age, smoking, BMI, HOMA-IR and lipids. Free estrogen index had a significant positive effect on PWV, but not independently of HOMA-IR and lipids. Age-adjusted levels of DHEAS exhibited a significant independent effect on measures of AI (β=-0.267, p=0.029) in a multivariate model that included age, BMI, current smoking, HOMA-IR, lipids and blood pressure. None of the remaining hormones associated with any of the vascular indices independently of traditional cardiovascular risk factors.
Discussion: Circulating testosterone is associated with subclinical atherosclerosis and arterial stiffness in healthy recently menopausal women. This association is independent of traditional cardiovascular risk factors or insulin resistance indicating a possible direct effect. On the contrary, serum DHEAS exhibits a negative association with arterial stiffness.
Conclusion: These findings are important in states of androgen excess, like the polycystic ovary syndrome. If the causality of endogenous testosterone and subclinical atherosclerosis is proven, the documentation of elevated androgens as a risk factor for cardiovascular disease will have important implications with regard to primary prevention policies.

Abstract #805

SUBCLINICAL ATHEROSCLEROSIS IS ASSOCIATED WITH MENOPAUSAL HOT FLUSHES IN HEALTHY YOUNG POSTMENOPAUSAL WOMEN

Eleni Armeni, M.D., Kimon Stamatelopoulos, Areti Augoulea, Demetrios Rizos, Andreas Alexandrou, Maria Creatsa, Irene Lambrinoudaki

Objective: Derived from a disturbance of the peripheral circulation, hot flushes are triggered by the withdrawal of estrogen at menopause. Furthermore, recent evidence supports the presence of an inverse association between vasomotor symptomatology and cardiovascular risk profile. The purpose of this study was to determine whether the presence and severity of climacteric symptoms is associated with changes in arterial structure and function in healthy, recently postmenopausal women.
Methods: This cross-sectional study recruited 110 postmenopausal women, aged 45 - 55 years. Menopausal symptoms were recorded using the Green Climacteric Scale. Fasting blood samples were obtained to estimate serum lipids, glucose, insulin as well as levels of sex- and thyroid

hormones, in each individual. Anthropometric measures and blood pressure were also assessed. Ultrasound evaluations included markers of arterial structure and function, namely intima-media thickness (IMT), flow mediated dilation and pulse wave velocity.

Results: Mean carotid IMT associated linearly with the severity of hot flushes (IMT for hot flushes, moderate to severe: 0.67±0.11mm, mild: 0.62±0.11mm, no: 0.61±0.08mm; p-value=0.034). This difference was independent of cardiovascular risk factors like age, menopausal age, smoking, blood pressure, adiposity, lipid levels and insulin resistance. Furthermore, the presence of severe hot flushes predicted significantly the mean common carotid artery IMT (R2=0.307, β-coefficient=0.264, p-value=0.015) in a multivariate model that included age, body mass index, systolic blood pressure, smoking and triglycerides. No significant association was observed between the severity of hot flushes and serum levels of sex and thyroid hormones or cardiovascular risk factors like obesity, arterial pressure, lipids, insulin resistance. Furthermore, no significant associations were observed with respect to psychological or psychosomatic symptoms.

Discussion: Women with hot flushes had increased carotid IMT compared to asymptomatic women, independently of traditional cardiovascular risk factors. On the contrary, no association was observed between indices of vascular function and menopausal symptoms.

Conclusion: Carotid IMT, a surrogate marker of subclinical atherosclerosis and cardiovascular risk, was found increased in healthy recently menopausal women with vasomotor symptoms as compared to asymptomatic women, independently of cardiovascular risk factors or endogenous hormone levels. It remains to be elucidated by larger prospective studies whether the presence of menopausal symptoms is an additional cardiovascular risk factor of developing vascular damage with advancing age.

Abstract #806

MODULATION OF ADIPONECTIN HOMEOSTASIS AS A POTENTIAL SUPPORT TO COMBINED VITAMIN D AND 1,25 DIHYDROXY VITAMIN D SUPPLEMENTATION IN VITAMIN D DEFICIENT CKD PATIENTS

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Objective: Presumed role of vit D deficiency in the development of metabolic and cardiovascular disorders have justified the use of vit D alone or in combination with its 1,25 $(OH)^2$ metabolite in patients with CKD. Proposed non-skeletal effects of vit D are primarily based on associations, rather than cause and effect. The present study was intended to assess the non-skeletal effect(s) of vit D compared to a 1,25 $(OH)^2$ vit D analog in patients with CKD.

Methods: This is a post-hoc analysis of a randomized controlled trial of secondary hyperparathyroidism therapy in 80 patients (age range 45-88 yrs, BMI 21-52 Kg/m^2) with stage 3 and 4 CKD and serum 25 OH vit D levels of <30 ng/mL who were randomly assigned 1:1 to receiving either ergocalciferol or paricalcitol [1,25 $(OH)^2$ vit D analog] for 16 weeks. The desired outcome for the ergocalciferol group was 25 OH vit D level of ≥30 ng/mL. Data was available for analysis in the present study in 38 patients (19 in each intervention arm). Paired t-tests were used to compare pre-and post-intervention levels of variables of interest.

Results: Subjects in the 2 groups were comparable in regard to GFR, age, BMI, albumin, corrected Ca, PTH, 25 OH vit D, testosterone, adiponectin, and high-sensitive (hs) CRP. After 16 weeks, serum adiponectin concentration (ng/mL) increased significantly in patients receiving ergocalciferol (9.9 ± 1.8 v 12.7 ± 2.1: P=0.01), but only showed a trend in the paricalcitol group (10.7 ± 1.5 v 12.3 ± 1.9: P=0.26). Changes in adiponectin levels in the ergocalciferol group corresponded with marked increases in circulating vit D (ng/mL) concentrations (17 ± 1.4 v 27 ± 1.9: P<0.0001). Alternatively, paricalcitol had preferential effect on bone metabolism with markedly decreased serum PTH (pg/mL) concentration (172 ± 18 v 128 ± 17: P=0.008). Intervention with either compound did not have significant effect on gonadal or inflammatory status, as respectively measured by testosterone (total, calculated free and bioavailable), and the hsCRP.

Discussion: While defying beneficial effect of vitamin D supplementation on gonadal function and inflammation in vitamin-D deficient CKD patients, the results are significant for a favorable effect on adiponectin.

Conclusion: Up-regulation of adiponectin could have significant clinical implications, considering its proclaimed role in rectifying peripheral insulin resistance, and as the result improving metabolic syndrome and related cardiovascular consequences. Future studies are warranted to assess long-term effect of ergocalciferol supplementation on adiponectin, and potentially related metabolic and cardiovascular correlates.

Abstract #807

PARATHYROID HORMONE IS PRESENT IN THE TISSUE OF THE NECK OUTSIDE OF THE PARATHYROID GLAND AND SURROUNDING THE THYROID

Raymon Grogan, MD, Karen Devon, MDCM, Peter Angelos, MD, PhD, Edwin Kaplan

Objective: When drawing intraoperative parathyroid hormone (IOPTH) samples from either internal jugular vein in the neck during parathyroidectomy (PTX) the results are often confusing. These results can be sporadically elevated and erroneous when compared to peripherally drawn samples. Some have suggested that this is due to manipulation of the gland during dissection, causing excess amounts of parathyroid hormone (PTH) in the internal jugular vein at the time of sampling. We questioned this hypothesis based on the sporadic nature of these findings, and sought to clarify this phenomenon.
Methods: In 40 patients undergoing PTX, peripheral blood was drawn and IOPTH determined in the operative suite. At varying times during PTX, fluid was obtained from the extrathyroidal space and examined for PTH concentrations and cytology. These samples were obtained unilaterally in patients undergoing minimally invasive PTX and bilaterally if a bilateral exploration was performed. Samples obtained during thyroidectomy were used as controls. The results were compared with simultaneously drawn peripheral blood samples for PTH.
Results: During dissection and following PTX, extrathyroidal samples of fluid exhibited PTH values as high as 75,000 pg/mL, far greater than serum values at 5 minutes post PTX (41.9 ± 34.9 pg/ml, mean ± SD) ($p<0.001$), and appeared to be related to the extent of operative dissection. This occurred both on the side of an adenoma and also on the contralateral side in which normal parathyroids were present. In thyroidectomy patients without primary hyperparathyroidism, these extrathyroidal fluid values were also elevated, as high as 9,100 pg/mL. No epithelial cells were seen in the extrathyroidal fluid on cytologic examination in PTX patients despite the high PTH levels. When as little as 5 microliters of extrathyroidal fluid were added to the blood samples used for IOPTH analysis, PTH values increased 20-fold (20 to 400 pg/mL).
Discussion: During PTX or thyroidectomy, high levels of PTH are found in fluid next to the thyroid. Accidental aspiration of miniscule amounts of this fluid may lead to a falsely elevated PTH value when blood from the internal jugular vein is used for IOPTH. Whether differences in extra capsular PTH levels can localize the side of the abnormal parathyroid gland is now being studied.
Conclusion: We report here the novel finding that PTH can be found outside of the parathyroid capsule in the tissues surrounding the thyroid gland and this can skew IOPTH results during surgery. We believe more work should be done to elucidate the physiologic significance of this finding, including studies on a possible link between hyperparathyroidism and thyroid nodular disease.

Abstract #808

PERSISTENT HYPOCALCEMIA AFTER DENOSUMAB

Hammad Hussain, MD

Case Presentation: 74 years old gentleman with history of metastatic prostate cancer was referred for evaluation of profound hypocalcemia. He was diagnosed with prostate cancer one year ago and a few months prior to presentation was found to have bone metastasis. He was started on Firmagon (GnRH receptor antagonist) monthly. In addition he also received one dose of XGEVA (Denosumab). Subsequently he became severely hypocalcemic (lowest calcium 3.5 mg/DL) which persisted for the next few months during which he had to be hospitalized multiple times for intravenous calcium replacement before he was seen in endocrinology clinic. 25 hydroxyvitamin D was within normal range. PTH was 127 pg/ML (with corresponding calcium 5 mg/DL). Magnesium, phosphorus and albumin were within normal range. Glomerular filtration rate was 42. He was then started on high dose oral calcium and calcitriol after his clinic visit. Over the next few months his serum calcium has normalized and calcium and calcitriol dose have been gradually decreased. Most recent calcium level is 9.4 mg/DL with PTH within normal range.
Discussion: Hypocalcemia was most likely related to denosumab use. Osteoclastic inhibition in the setting of renal insufficiency may have exacerbated hypocalcemia. Based on clinical trials using a lower dose of denosumab, patients with a creatinine clearance less than 30 mL/min or receiving dialysis are at greater risk of severe hypocalcemia compared to patients with normal renal function. Hypocalcemia after bisphosphonate use has been seen even 12 weeks after drug administration. However hypocalcemia persisting months after denosumab use, as seen in this case, has not been reported.
Conclusion: Care should be taken to monitor calcium level after denosumab use as severe hypocalcemia may occur in patients with renal insufficiency, which may persist for months, even with normal 25 hydroxyvitamin D level.

Abstract #809

BENIGN NON-FAMILIAL PHEOCHROMOCYTOMA PRESENTING WITH SYMPTOMATIC HYPERCALCEMIA FROM AN UNUSUAL ETIOLOGY

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Objective: To have physicians aware of an unusual etiology of hypercalcemia seen in patients with pheochromocytoma
Methods: We evaluated the etiology of a 57 year old man's hypercalcemia associated with pheochromocytoma while treating him in the hospital
Case Presentation: 52 year old African-American male was referred to the emergency room after his primary physician noted hematuria and hypercalcemia (12.6 mg/dl) on an annual physical in this patient with chronic hypertension. In the hospital BP was controlled with diltiazem 240 mg/day. Physical exam was otherwise unremarkable. His serum calcium was 13.1mg/dl, CBC, renal, and hepatic blood studies were normal. PTH was 8.7pg/ml(n:15-65), 25-hydroxyvitamin D and 1,25-dihydroxyvitamin D were WNL. TSH, free T4, serum and urine protein immunoelectrophoreses were WNL. A 7.1x4.9x7.3 cm mass of the right adrenal was noted on CAT scan. 24 hour urine revealed metanephrines=15143 mcg/24hours(N:224-832) and catecholamines=3494 mcg/24 hours(N:26-121). VMA=28.3 mg/24hours(N:<6). A PTH-r-P level was 21 (N:10-24). With hydration and parenteral calcitonin calcium levels were maintained around 11.0 mg/dl. After appropriate preparation for surgery the pheochromocytoma was resected and his hypercalcemia resolved. Postoperative PTH-r-P levels were undetectable.
Discussion: Several causes for hypercalcemia have been well described as etiologies associated with pheochromocytoma. However, the association with PTH-r-P has been described rarely and not recently in the literature. Those described had either elevated levels of serum PTH-r-P or very high tissue levels of PTH-r-P in the pheochromocytoma. Our case revealed high normal serum levels of PTH-r-P that became undetectable post-operatively at the same time that the patient's serum calcium returned to normal. Unfortunately there was no way available to us to measure tissue PTH-r-P of the tumor. Therefore we infer that this represents another rare case of PTH-r-P mediated hypercalcemia from a pheochromocytoma.
Conclusion: We describe an unusual etiology of hypercalcemia associated with pheochromocytoma that physicians need to be aware of in their differential of hypercalcemia in patients with pheochromocytoma, i.e. PTH-r-P secretion from pheochromocytoma.

Abstract #810

VALUE OF INTRAOPERATIVE PARATHYROID HORMONE MEASUREMENTS IN DETECTING UNSUSPECTED PARATHYROID ADENOMAS

Luis Ospina, MD, Aisha Afzal, Abdul Al-Kassab, Mario Villalba

Objective: Parathyroid adenoma is a benign tumor of the parathyroid gland, which can be easily cured with surgical resection. Sestamibi scan is useful for preoperative localization but it can fail to identify multi-glandular disease in 73% of cases. In this scenario, the success rate increases significantly when combined with the intraoperative PTH (ioPTH) measurements.
Methods: Two patients undergoing parathyroid adenoma resection had intraoperative PTH hormone levels measured prior to the surgery and 10 minutes after the removal of the adenoma , using a two sandwich chemiluminescent assay using a Siemens Centaur machine.
Case Presentation: Two female patients age between 50 to 60 years presented to the clinic with a history of hypercalcemia of approximately 5 years duration. They had no major complaints and their past medical history was otherwise unremarkable. The physical examination was also unremarkable but the laboratory results revealed elevated parathyroid and calcium levels. The first patient had PTH level of 148 pg/ml and serum Calcium of 11.1 mg/dl, while the second patient had a PTH level of 153 pg/ml and serum Calcium of 10.9 mg/dl. Tc-99m Sestamibi parathyroid scan revealed in the first an adenoma in the posterior aspect of the right paratracheoesophageal groove and in the second patient an adenoma in the superior aspect of the left lobe of the thyroid gland. Both patients went to the operating room for parathyroid adenoma excision. Intraoperatively following removal of the visualized adenoma the ioPTH did not decrease as expected. The levels dropped from 163 pg/dl to 110 pg/dl in the first patient and from 155 pg/dl to 150 pg/dl in the second patient. The lack of adequate drop in PTH level in both patients led to further exploration with removal of a second adenoma. The repeat hormone assay now showed a drop to 21 pg/dl in the first patient which was consistent with a biochemical cure. Similarly in the second patient the ioPTH levels dropped to 51 pg/dl after removal of the second tumor.
Discussion: These cases highlight that even though pre-operative localizing studies are valuable, ioPTH may improve detection of multi-glandular disease and improve the success rate of surgery for this condition. In our institution the test results were communicated within 15 minutes to the surgical team.
Conclusion: In summary these cases highlight a poten-

tially useful test to improve surgical outcomes in parathyroidectomy. Future studies should be done to validate the usefulness of this approach evident in these two interesting cases.

Abstract #811

AUTOIMMUNE POLYGLANDULAR SYNDROME IN A COMMUNITY BASED ENDOCRINE PRACTICE

Luz Prieto, MD, Victoria Rentas, Celeste Hart

Objective: We describe the frequency and characteristics of autoimmune polyglandular syndrome cases in a private clinical setting of a general endocrine practice.
Case Presentation: Review of Medical records of patients treated between 2002 and 2011 at North Florida Regional Thyroid Center (Tallahassee, Fl). We found 15 cases with different presentation of APS type 2, only 3 of them with classical presentation (AD, T1D, AITD). Nearly 62% of the patients have three or more manifestations associated with autoimmune polyglandular syndrome. Autoimmune thyroid disease is present in ten of the thirteen patients (nearly 77%). In addition, our study has eleven of the fifteen patients with autoimmune type 1 diabetes (nearly 77%) which varies significantly from the data presented in the literature, most of them with treatment failures on OHA until complete evaluation found with negative C-peptide and positive antibodies (GAD and Islet Cell Antibodies). Only three cases diagnosed with osteoporosis and fractures and one case had low PTHi levels. The average age of our autoimmune polyglandular patients at diagnosis is 44.08 ± 14.44 years. The male to female ratio is 3:12, highlighting a strong predominance towards females.
Discussion: Community based endocrinologists have the opportunity to evaluate patients with diabetes and thyroid disease presenting as the tip of iceberg of a complex autoimmune polyglandular syndrome. Most of cases with type 1 diabetes were referred as poorly controlled type 2 diabetes; some of them with diabetes complications.
Conclusion: Evaluation of subclinical endocrine abnormalities and/or specific antibodies would ensure early diagnosis assessment and treatment of these endocrine disorders with great benefit for the patient and prevention of severe complications.

Abstract #812

THE RELATION BETWEEN PLASMA LEPTIN LEVELS AND CAROTID INTIMA MEDIA THICKNESS WITH SEVERITY OF OBSTRUCTIVE SLEEP APNEA

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Objective: We compared basal plasma leptin levels and carotid intima-media thickness (CIMT) of mild, moderate and severe obstructive sleep apnea (OSA) patients.
Methods: Age, sex and BMI matched 78 eligible patients with OSA were divided into three groups according to apnea-hypopnea index consecutively as mild-OSA (5≤AHI<15), moderate-OSA (15≤AHI<30) and severe-OSA (AHI≥30). Any subjects with an acute/chronic diseases were not included. The CIMT was measured by B-mode ultrasound with 10-MHz linear transducer.
Results: Each of the study groups composed of 9 female, 17 male subjects with a mean age of 45.5±9.3 for mild-OSA (n=26, BMI=29.8), 47.1±9.5 for moderate-OSA (n=26, BMI=30.7) and 47.6±8.6 for severe-OSA (n=26, BMI=31.2). There was no difference in mean age and BMI values between groups (p=0.685 and p=0.322). Serum basal leptin levels (ng/ml) of mild-OSA (AHI≤15) patients were (13.9±10.9) significantly lower than moderate and severe OSA subjects (AHI>15) as 17.1±11.8 and 21.2 ± 11.3 respectively (p=0.06). The CIMT (mm) of patients increased significantly parallel to severity of OSA; as 5.96±1.1 for mild-OSA (AHI=7.8), 6.33±0.7 for moderate-OSA (AHI=21.2) and 7.01±1.07 for severe-OSA (AHI=64.1) (p<0.001).
Discussion: Hypoxia or sleep interruption, as occurs in OSA, was suggested to contribute to body fat composition and plasma leptin levels. Besides, nitric oxide-mediated vasodilator effect of leptin was shown to impair under inflammatory conditions. Patients with OSA were shown to present a low-grade systemic inflammation induced by intermittent hypoxia. We showed that severe OSA patients had higher CIMT and leptin levels independent of BMI.
Conclusion: According to those findings, impairment in leptin actions related to severity of OSA through inflammation and may be the cause of atherosclerotic changes and cardiovascular outcomes of OSA.

Abstract #813

STATUS OF VITAMIN D DEFICIENCY OF PATIENTS IN A TERTIARY CARE CENTRE AND ITS CO- MORBIDITIES

Hemant Thacker, MD, Aneesa Kapadia

Objective: Vitamin D deficiency is associated with a wide spectrum of diseases and dysfunctions which have a significant impact on the quality of life. In fact it often contributes to the mortality of the primary disease. However not much epidemiological data comparing age, sex, socioeconomic background as well as its correlation with various clinical conditions, is available. This study aims to provide precisely the same.
Methods: A hospital based retrospective analysis of randomly selected 162 patients, who were admitted between March 2010 and October 2011 for a wide range of clinical conditions. Their vitamin D levels were noted and a comparative analysis was carried out.
Case Presentation: 1) Prevalence: 75% of cases 2) AGE: Highest prevalence is noticed in the older age group (61-80 years) - 48%, but (18%), of younger people (<40 years), also deficient. 3) Sex: Males-58%, Females-42% 4) Socioeconomic Class (SEC): Equal. 5) Concomittant Diseases: Spectrum of conditions coexist: Infections (37%)-bacterial, viral & fungal, orthopedic conditions (16%), CAD-coronary artery disease (!12%), diabetes (11%), CNS disorders (10%), cancer (3%), others (10%) 6) Degree of Deficiency Of the 122 deficient: 15% were <5,30% were<10, 30% were<15. The remaining 25%were <30.
Discussion: Assuming deficiency as values less than 30, a majority of the patients were deficient. It is distributed across all age groups. Males more than females. SEC was a not a criteria, as even the higher income groups, were sufferers (43%). Though vitamin D deficiency was detected across the board with a wide spectrum of known conditions, remarkably, patients suffering from different infections (bacterial, viral, fungal etc) were found to have a higher association (37%). Whilst the incidence of diabetes and CAD was not surprising, neurological conditions(CerebroVascular Accidents, palsies etc), were a new entrant amongst the associates.
Conclusion: Vitamin D deficiency is no longer restricted to the older and less affluent groups but surpasses age and socioeconomic barriers. It also contributes to many apparently non musculoskeletal conditions and has serious implications on their pathogenesis and outcome. From the preventive aspect, a higher index of suspicion at the primary health care level may be beneficial in pre-empting the spectrum of illnesses it is seen to be associated with. In specific, its relationship to infections could be translated as a therapeutically treatable strategy to prevent morbidity amongst the community.

Abstract #814

MULTIPLE ENDOCRINE NEOPLASIA TYPE 2A. STUDY OF A FAMILY OF THREE GENERATIONS WITH PEDIGREE ANALYSIS OF THE RET PROTO-ONCOGENE

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Objective: Activating germline mutations of the RET proto-oncogene are found in more then 90% of families with MEN 2A. The majority of patients with these heredity tumors carry germline mutations that result in the substitution of one of five cysteine residues in exon 10 and 11. MEN 2A is characterized by medullary thyroid carcinoma (MTC), pheochromocytoma (pheo) and primary hyperparathyroidism (PHPT). Aims: Perform a genetic screening for RET gene mutations in a family with a proband with RET mutation.
Methods: We report a case of MEN 2A in a family in which the first patient had bilateral pheo associated with CMT. Molecular genetic testing of the RET exons confirmed the mutation at codon 634 TGC 634 TGG (Cys 634Trp) in RET exon 11. After identification of the proband we screened all her family members with genetic testing for the RET proto oncogene mutation. The subjects with the mutation were further assessed for pheo by measurement of the 24 - hour urinary metanephrrines and CT-scan. Three of them (the three daughters) were presented pheo, one of them bilateral pheo. At two daughters, high calcitonin levels and nodular goiter required the surgical treatment - total thyroidectomy. The CMT was confirmed by pathological analysis. The serum calcium, urinary calcium excretion and PIH secretion ware measured.
Case Presentation: From the nine family members screened, six had the RET proto oncogene mutation codon 634 TGC > TGC (Cys 634 Trp); four females: proband, three proband daughters and two children. A boy - two years old has normal level of calcitonin, his mother developed CMT during pregnancy. The second boy - ten years old has high level of calcitonin; his mother has CMT and bilateral pheo.
Discussion: MEN 2A may be suspected when MTC occurs at an early age or is bilateral or multifocal. It is widely recommended RET gene mutation testing be performed for all cases of MTC.
Conclusion: Genetic screening for germline RET proto-oncogene mutation in hereditary medullary thyroid cancer is accurate and allows for preventive total thyroidectomy to be performed early in patients who are gene carriers. RET proto-oncogene mutation given the almost 100% risk of developing CMT in MEN 2A, it is currently recommanded total thyroidectomy before the age of 5.

Abstract #815

DISPROPORTIONATE RISE IN SERUM DHT LEVELS FOLLOWING TRANSDERMAL TESTOSTERONE TREATMENT

Daniel Cosgrove, MD, Frank Stanczyk, Leandra Fraser

Objective: Although increasing serum testosterone (T) levels have not been associated with the development of prostate cancer, the role of dihydrotestosterone (DHT) is less clear. The current standard of practice for treating hypogonadism includes measuring T and PSA before initiating therapy, and monitoring these during therapy. However, measurement of DHT is not standard medical practice. DHT may accumulate in the body, especially in prostate tissue over time in men receiving T therapy. We sought to determine serum levels of T and DHT before and after therapy.
Methods: We measured T and DHT in serum from 26 men [average age 62 (42-84) years old] before initiating transdermal T therapy, and again after several weeks of therapy. T levels were measured by liquid chromatography-tandem mass spectrometry and DHT levels were determined using RIA at Quest Diagnostics Nichols Institute, San Juan Capistrano, CA.
Results: DHT rose disproportionately and dramatically. The median DHT/T ratio prior to therapy was 0.12 (12%), but rose to 0.18 (18%) after several weeks of therapy (median time 4 months). The mean rise in serum total testosterone and DHT following therapy was 308 ng/dl and 70 ng/dl, respectively. There was a trend toward greater DHT/T ratios following a longer duration of T therapy. T levels increased but remained within the reference range, whereas DHT nearly always increased to levels well above the reference range.
Discussion: Higher DHT levels may increase the risk of prostate cancer. Several studies suggest that inhibition of 5 alpha-reductase may lower prostate cancer incidence. Since the sole function of 5ARI is to decrease serum DHT relative to serum T levels, a disproportionate rise in DHT with transdermal T therapy is like prescribing "anti-5-AR" and may have adverse effects. Higher DHT levels increase the risk of BPH. Only 5-ARIs have demonstrated the potential for long-term reduction in prostate volume and need for prostate surgery (see graph). Conclusions about the role of DHT and 5ARIs in chemoprevention strategies of BPH should distinguish between men treated with exogenous T and those men never treated (producing their T serum levels naturally) because the levels of DHT are much greater in the former group. In men receiving transdermal T, we have shown that the proportion of DHT becomes 1.5 times as high.
Conclusion: We conclude that long term administration of T should include periodic measures of DHT, and that treatment with 5 alpha-reductase inhibitors (5ARI) in some of these patients must be considered.

Abstract #816

RESOLVING DEPRESSION AND NECROLYTIC MIGRATORY ERYTHEMA 2 WEEKS FOLLOWING GLUCAGONOMA RESECTION

Yousef Altowaireb, MD, Kamal Shoukri, Pooja Sherchan, MD, Sabapathy Sen

Case Presentation: 42 year-old female presented with depression, weight loss, and migratory, painful skin rash involving the extremities, trunk, and perineum for over 6 months. Initially, the rash was diagnosed as being eczematous changes; however, it persisted and remained resistant to standard topical steroid therapy. Biopsy showed spongiotic dermatitis with focal keratinocyte vacuolization and parakeratosis, features consistent with necrolytic migratory erythema (NME). Biochemical workup showed an elevated Glucagon level at 627 pg/ml (Normal: 40-130 pg/ml), hemoglobin A1C level of 5.2%, and a mean fasting blood glucose: 80 mg/dL corresponded to an Insulin level: 17 UIU/ml. CT of the abdomen demonstrated a 3.4×2.7×2.5 cm mass in the tail of pancreas. Octreotide scan confirmed the lesion and showed no evidence of metastatic disease. The patient underwent distal pancreatectomy. Pathology revealed a well-differentiated neuroendocrine tumor with positive immunohistochemical staining for glucagon. Post-operative Glucagon and Insulin levels had decreased to 37 pg/ml and 4.1 UIU/ml, respectively. 2 weeks following surgery, the patient had a near complete resolution of the skin rash and depression.
Discussion: Glucagonoma are rare tumors of the alpha pancreatic cells, classically occupy the tail of the pancreas. They are more common in females and typically present in the fifth decade. Reports of diabetes in glucagonoma vary from 75-95%. Our patient did not have diabetes; instead, she had a hyperinsulinemic state which may have represented a compensatory mechanism counteracting her elevated glucagon level. Our patient's disease was localized and was completely resected. Localizing imaging modalities did not show any evidence of metastatic disease, which is unusual. The prevalence of metastatic disease at the time of diagnosis range from 50-100%. The most common sites of metastasis are the liver, bones, adrenal glands, kidneys, and lungs. NME and depression usually present in 60-70% of the cases of glucagonoma. The exact mechanism by which glucagonoma causes these skin lesions and depression remains unclear. Cutaneous and CNS manifestation may resolve following successful treatment; however the ra-

pidity of this resolution as it was evident in our patient is unusual.

Conclusion: Our patient is interesting in that her glucagonoma was localized and was completely resected, which is unusual for this disease. The rapid and marked improvement of her skin lesions and depression post-operatively was also unusual, and is indicative of the significant paracrine property of glucagon in the skin and the nervous system.

Abstract #817

COMPARISON OF OBESE AND LEAN PRIMARY HYPERPARATHYROID PATIENTS

Cindy Huang, MD, Dennis Han, Stanley Trooskin, Xiangbing Wang

Objective: Comparing metabolic characteristics in obese and lean patients with primary hyperparathyroidism (PHPT)

Methods: This was a retrospective chart review study. A total of 321 charts of patients with PHPT were reviewed. Lean patients were defined as having BMI of less than 27. Obese patients were defined as having BMI equal or greater than 27. The following characteristics were compared between the two groups, including age, BMI, intact PTH, serum calcium, systolic and diastolic blood pressures, fasting blood glucose, lipid profile, and 25-OH Vitamin D level.

Results: Of the 321 PHPT patients, 127 were lean (15 males, 109 females) and 197 were obese (51 males, 146 females). The average age of the study population was 59 years old. The average BMI of the lean group and the obese group was 23.6 and 33.2 respectively ($P < 0.001$). The iPTH levels were significantly lower in the lean group compared to the obese group (149 pg/ml versus 178 pg/ml, $P = 0.009$); while 25-OH Vitamin D levels were higher in the lean group compared to the obese group, 31.8 μg/ml versus 25.0 μg/ml ($P < 0.001$). The serum calcium levels were not significantly different between the two groups. However, the fasting blood glucose levels were significantly lower in the lean group compared to the obese group, 93.4 mg/dl versus 104 mg/dl ($P < 0.001$). Additionally, the systolic blood pressures were also lower in the lean group compared to the obese group (128 mm Hg versus 136 mmHg, $P < 0.001$). When lipid profiles were compared between the two groups, HDL was significantly lower in the obese group (52 mg/dl versus 62 mg/dl, $P < 0.001$) and triglycerides were significantly higher in the obese group (156 mg/dl versus 112 mg/dl, $P < 0.001$).

Discussion: Primary hyperparathyroidism is well known to be associated with metabolic syndrome. Specifically, PHPT has been known to be associated with an increased prevalence of insulin resistance, hypertension and dyslipidemia. The aim of this study is to investigate whether this association is directly related to primary hyperparathyroidism or differences in BMI. This study suggests that the association of primary hyperparathyroidism with parameters of metabolic syndrome might be related to obesity, relatively high iPTH levels, and relatively low 25-OH Vitamin D levels. Further studies are needed to define the temporal relationship between low Vitamin D levels, high iPTH and obesity.

Conclusion: Our study revealed that obese patients with PHPT are at a higher risk for metabolic syndrome compared to lean patients with PHPT. This increased risk might be related to the relatively high iPTH and low 25-OH Vitamin D levels observed in obese patients with PHPT.

Abstract #818

HYPERCALCEMIA IN A PATIENT WITH MEN 1 SYNDROME

Pooja Aggarwal, MD, Vishal Bhatia, MD

Objective: We report a case of a patient with MEN1 syndrome who presented with hypercalcemic crisis due to metastatic parathyroid cancer.

Case Presentation: A 42 year old male with known history of Multiple Endocrine Neoplasia type 1 (MEN 1) characterized by parathyroid adenoma with hyperparathyroidism, Pituitary adenoma (prolactinoma), non-functioning pancreatic and adrenal tumor underwent partial thyroidectomy with removal of three parathyroid glands at the time of his diagnosis. Prolactinoma was stable with 10 mg bromocriptine daily. Seven years later, he presented with recurrent hypercalcemia with serum calcium of 12.9 mg/dl. Parathyroid hormone (PTH) was elevated at 256 pg/ml. Ultrasound of the thyroid showed a left upper pole thyroid nodule. Fine needle aspiration (FNA) cytology was consistent with benign thyroid nodule. Computed tomography of the chest showed a 3.5 cm superior mediastinal mass attached to the inferior pole of the left lobe of thyroid gland (Fig 1). FNA of this lesion was hypocellular although showed very high PTH level of 636,017 pg/ml. Patient underwent complete surgical excision of this tumor. Pathology was consistent with parathyroid cancer. On follow up in two weeks, recurrent hypercalcemia was noted with calcium level at 11.3 mg/dl. Surprisingly, PTH level was still elevated at 202 pg/ml despite total parathyroidectomy. A pertechnate sestamibi scan showed increased uptake in the right lower pole of thyroid gland that persisted on delayed images (Fig 2). A total thyroidectomy and radical lymph node dissection was performed which showed metastatic parathyroid tissue within the thyroid

gland with no lymphatic involvement. On follow up, calcium was within normal limits with undetectable PTH.
Discussion: Parathyroid cancer is a rare cause of primary hyperparathyroidism accounting for less than one percent causes of primary hyperparathyroidism. Localized recurrences and metastasis are unfortunately frequent. The case represents the difficulty in localization of excessive parathyroid secretion with metastatic parathyroid cancer. One previous case of parathyroid cancer has been reported in conjunction with MEN1 syndrome.
Conclusion: Metastatic parathyroid cancer is a rare cause of primary hyperparathyroidism rarely seen in association with MEN1 syndrome.

Abstract #819

RELATION OF SERUM CALCIUM LEVEL WITH METABOLIC RISK FACTORS AND CORONARY ARTERY DISEASE IN AFRICAN AMERICAN

Swati Singh, MBBS, Anand Balachandran, Thomas John, Chirag Patel, Jacob Warman, Ciprian Nedelcu

Objective: Calcium has been shown to play an important role in the pathogenesis of atherosclerotic disease; either as an independent risk factor or as a possible component of metabolic syndrome. We propose to study the relationship between serum calcium, coronary artery disease (CAD) and metabolic risk factors (MRF) in the African American (AA) population which has not been studied extensively.
Methods: This study was a retrospective review of the medical records of all AA patients discharged with CAD during a one year period. A total of 325 charts were identified and reviewed for demographic, clinical, and laboratory details. The following variables were tabulated: age (35 to 85 years), gender, ethnicity, metabolic risk factors {Fasting blood sugar (FBS), HDL, triglycerides (TG), blood pressure, body mass index, albumin corrected serum calcium (ACC)}, and CAD identified via cardiac catheterization. Patients were included in the MRF group if they had three or more abnormal values out of the five factors (HDL, TG, FBS, BMI and BP). The abnormal values in this group were identified according to parameters that define metabolic syndrome. The criteria for CAD was to have 30% or more stenosis of any major coronary artery on catheterization.
Case Presentation: Complete records were available for 229 AA subjects . The majority of the cases (33%) were between 56 to 65 years with no relevant differences between the genders. 190 (82.9%) patients were diagnosed with CAD and 143 (75.2%) patients had metabolic risk factors. In 180 (94.7%) patients with CAD, the ACC level on admission was normal (8.4 to 10.2 mg/dl). 3% were found to have a high ACC and 2% a low ACC. Further analysis of values in normal range revealed that the number of subjects with ACC in the low normal (8.4 to 9.2 mg/dl) and high normal (9.3 to 10.2mg/dl) were also equal. In our study population, no statistical difference in ACC was noted between cases with or without metabolic risk factors ($p<0.05$). Also of note, no other statistically significant association was found between abnormal ACC with CAD in African Americans.
Discussion: Our study differed from the CoLus study which found strong association between serum calcium and MRF in Caucasian population. We did not find any large scale study correlating serum calcium with metabolic syndrome or CAD in African American population. Further prospective trials with larger patient pools are required to completely understand any relationship.
Conclusion: For the African American population, serum calcium level has no correlation with CAD or metabolic risk factor.

Abstract #820

MYSTERY TRAIL OF UNEXPLAINED HYPERCALCEMIA ENDS IN A FOOD SUPPLEMENT.

Nidhi Bansal, MBBS, Liviu Danescu

Case Presentation: Complementary medicine utilization continues to enjoy an impressive amount of attention and consumption in spite of grave concerns on their potential for toxicity. We present an interesting case of 59 year old caucasian lady with history of multiple sclerosis, osteoporosis, breast cancer status post lumpectomy, radiation and chemotherapy. She presented with c/o weakness, fatigue, headache, constipation, short term memory loss and palpitations. Her metabolic profile showed pre renal azotemia and hypercalcemia (16.2 mg %). She was started on aggressive intravenous hydration and intensive monitoring leading to rapid resolution of symptoms. A complete diagnostic work up for hypercalcemia was performed. PTH levels were suppressed at 5.6 pg/ ml arousing suspicion for hypercalcemia due to malignancy (prior history of breast cancer vs new primary). However her PTHrP levels were within normal limits (0.8 pmol/ l). TSH (0.623 IU/L) and free T4 (2.87 ng/dl) were also within normal limits. 25- hydroxy Vitamin D level was high at 96ng/ ml, while 1-25 hydroxy vitamin D was 28 pg/ ml (10-65 pg/ ml). Review of her medication list showed daily intake of 800 units of vitamin D and 500 mg of calcium. With limited sun exposure in Central New York, how our patient managed to achieve such a degree of symptomatic hypervitaminosis D became a mystery waiting to be resolved. We investigated her for granulomatous diseases and other causes of hypervitaminosis D. The patient denied over-

dosing on prescription vitamin D initially but later admitted that she was ingesting an expensive naturally derived gel preparation containing about 16,000 IU of vitamin D daily for last 10 months. This was one of several alternative medications she was prescribed online by practitioner of alternative medicine to cure her multiple sclerosis and osteoporosis. We counseled and provided information to the patient on potential side effects of excessive vitamin D supplementation and other alternative preparations and withheld the same at the time of discharge.

Discussion: 34 billion dollars are spent on complementary and alternative medicine (CAM) in the U.S.A. Surveys suggest that the potential for a negative drug - supplement interaction in users can be as high as 40%.The majority of the expense on CAM is directed towards "self-care," and is done without physician approval or guidance.

Conclusion: CAM is not regulated by the FDA , thus manufacturers tend to make unfounded health benefit claims while concealing the potential side effects as was the case with our patient.

Abstract #821

MYSTERY TRAIL OF RAISED SERUM HCG ENDS IN A LUNG MASS

Nidhi Bansal, MBBS, Sam Benjamin

Case Presentation: Choriocarcinoma is a malignant germ cell tumor characterized by a proliferation of syncytial cells and cytotrophoblasts, and secretion of β-human chorionic gonadotropin (β-hCG). It presents usually in gonads and less commonly in extra gonadal sites. We report an exceeding rare case of a 33 year old male with a past medical history of generalized anxiety disorder and tobacco abuse, who presented with left sided rib pain of 2 month duration. Physical exam was benign except for local tenderness of the left lower chest wall. Blood counts and metabolic profile were within normal limits. Chest X-ray revealed severe bullous disease in the apices and small bilateral pulmonary nodules. A CT thorax was then performed, which showed a low density mass in the left suprahilar region with bilateral pulmonary nodules and emphysema. A bronchoscopic guided biopsy of a lung nodule was done to unravel this medical puzzle. The histopathology showed a poorly differentiated malignant tumor present within lung parenchyma in sheets with severe pleomorphism, extensive necrosis and hemorrhage. Multinucleated giant cells resembling syncytiotrophoblast were focally present. The neoplastic cells were positive for keratin AE1/AE3 and B-hCG and focally for p63. They were negative for alpha-fetoprotein (AFP), PLAP and CK5/6. The presence of extensive necrosis and hemorrhage, syncytiotrophoblast-like cells and the strong beta HCG staining all supported a diagnosis of choriocarcinoma. Further staging did not reveal signs of malignancy elsewhere. β-hCG levels at time of diagnosis were 30039 mU/ml (normal 0-5 mU/ml). Patient was treated with chemotherapy as recommended by the oncology service with improvement in symptoms and reduction in β-hCG levels (25391 mU/ ml). At the end of the 4th chemo cycle, patient became asymptomatic with normalization of β-hCG levels and significant reduction in tumor burden in interval CT imaging.

Discussion: Primary pulmonary choriocarcinoma is an extremely uncommon entity, with only 23 cases reported in world literature. Treatment protocol is similar to that for non-small cell lung cancer. Chemotherapy is indicated irrespective of whether surgical resection performed or not. Measurement of β-hCG levels is useful for diagnosis, follow-up, and prognosis.

Conclusion: Normalization of β-hCG levels after therapy is indicative of clinical remission in primary pulmonary choriocarcinoma, as was the case in our patient.

Abstract #822

CASE REPORTS:UNUSUAL PRESENTATIONS OF GASTROINTESTINAL CARCINOID TUMORS

Monika Olchawa, MD, Janice Gilden, MD

Objective: To report two unusual presentations of carcinoid tumor in the gastrointestinal tract and highlight the necessity for timely detection and establishment of an early therapeutic plan. To emphasize the importance of management of neuroendocrine tumors by multispecialty groups.

Case Presentation: We present 2 cases of gastrointestinal carcinoid, and the major challenges physician face in early diagnosis, due to vague symptoms, often mistaken for other GI diseases. Our first patient presented with numerous GI complaints (abdominal pain, episodic nausea, vomiting, diarrhea) and facial flushing. He underwent extensive gastrointestinal and surgical evaluation, including endoscopy, liver ultrasound, UGI with small bowel follow-through, CT and MRI abdomen. The primary tumor site was localized intra-operatively. The second patient presented with poor appetite, failure to thrive, and chronic occult anemia. CT abdomen revealed a large right colonic mass with mesenteric retroperitoneal lymphadenopathy, mistaken for colon cancer. Upon further laboratory investigation, the two patients had elevated plasma chromogranin A levels, and 24-hour urinary excretion of 5-hydroxyindoleacetic acid. Both patients underwent surgical resection of the primary tumor. Gross pathology revealed multifocal tumors with firm nodules infiltrating the intestinal wall and adjacent mesentery. Histopathology was consistent with carcinoid, showing distinct nests of

tumors cells. Medical therapy involved management by multispecialty providers and treatment with somatostatin analogues. Due to the postponed diagnosis, both patients had advanced liver metastases.
Discussion: The diagnosis of carcinoid tumors is based upon biochemical markers, localization of the primary tumor with metastasis, and pathology. Some tumors are found incidentally during surgeries, endoscopies, or radiography. The management of gastrointestinal carcinoid depends upon the symptoms, type and size of the tumor, and the patient's functional status. Surgical approach is recommended for resection of the primary tumor and advanced disease, which improves prognosis and survival. Patients with tumors expressing somatostatin receptors should be treated with somatostatin analogues.
Conclusion: Neuroendocrine tumors of the gastrointestinal tract are challenging to diagnose. These are often mistaken for other GI diseases, such as gastritis, gastroenteritis, irritable bowel, inflammatory bowel disease, gluten or lactose intolerance, and colon cancer. A timely diagnosis is critical for proper multidisciplinary management. Supported by JAL FHCC.

Abstract #823

A LITTLE TOO MUCH CALCIUM IN MY VACATIONS: ACUTE SEVERE HYPERCALCEMIA IN A PREVIOUS ASYMPTOMATIC MAN

Ricardo Correa, MD, Jose Sandoval

Objective: Hypercalcemic crisis or parathyroid storm is a rare but life-threatening complication of primary hyperparathyroidism. Most cases are caused by a parathyroid adenoma and parathyroid gland hyperplasia, with parathyroid carcinoma being a very uncommon cause.
Case Presentation: This is a 66-year-old white male with no relevant past medical history that was transferred to our hospital, from a cruise ship secondary to respiratory arrest. Four days before the patient complained of loss of appetite associated with vomiting and right flank pain. He also referred progressive unsteady gait for the last 11 days. The patient was initially treated for possible gastroenteritis with ciprofloxacin and metroclopramide, with no improvement in symptoms. He consulted again to the cruise medical service because of the persistence of his symptoms associated to confusion, severe unsteady gait with bilateral leg weakness and severe left frontal headache. During the medical interview, he had a generalized tonic-clonic seizure and went into respiratory arrest. The patient was intubated, started on mechanical ventilation and transferred to our hospital. On the initial clinical assessment, the patient was comatose, intubated with stable vital signs, no cardiopulmonary abnormalities and no focal neurological deficits. Laboratory data include a high ionized calcium level (3.6) with high PTH (831), high sodium and evidence of acute kidney injury with a high serum creatinine and BUN. He was treated for his hypercalcemia with aggressive hydration with IV fluids, high doses of pamidronate and cinacalcet A thyroid US showed a large spiculated cystic structure in the inferior and mid aspect of the right lobe of the thyroid that the endocrinologist service consider to be a parathyroid carcinoma due to the acuity and severity of the clinical presentation.
Discussion: With the treatment given, the patient improved his altered mental status, the serum calcium and other electrolytes abnormalities were corrected and his renal function was normalized. He was discharge with the diagnosis of primary hyperparathyroidism due to possible parathyroid carcinoma. Surgical excision of the parathyroid mass was programmed as an outpatient.
Conclusion: Discussion and conclusion: Acute severe hypercalcemia should be suspected in acutely ill patients with altered mental status, muscular weakness, gastrointestinal and/or urinary symptoms. To reduce mortality, it is essential to provide appropriate emergency management correcting hypercalcemia and dehydration and at the same time, make an accurate diagnosis without delays to treat the patient with a possible curative procedure (i.e. parathyroidectomy).

Abstract #824

"I AM DIFFERENT FROM THE REST!" AN INTEREST CASE OF HORMONAL CONTROL

Ricardo Correa, MD, Melany Castillo, Ana Sandoval, Alejandro Ayala

Objective: Kallmann's syndrome is characterized by hypogonadotropic hypogonadism and 1 or more non-gonadal congenital abnormality, including anosmia, red-green color blindness, midline facial abnormalities such as cleft palate, urogenital tract abnormalities, synkinesis and neurosensory hearing loss.
Case Presentation: 28 yo gentleman who was first diagnosed with testosterone deficiency approximately at 12 yo in Cuba. He was treated with "a cycle of testosterone" but then it was discontinued by his doctors. At 17, he was told that one of his testicles had not descended into the scrotum while being examined for a military service. He then immigrated to US, where his PCP noted that he was hypogonadic and then referred to the endocrinology clinic. At his first visit, he denies any history of olfactory problems, although, upon further questioning, he clearly states that he has difficulty smelling dif-

ferent substances, which was clear during physical examination where he was unable to distinguish the smell of coffee, perfume or chocolate. He also mentioned that he has a 21 and a 4 year-old nephew that also appeared to be hypogonadic and have cryptorchidism and synkinesis. The patient had not developed secondary sexual male characteristics except for the mild presence of terminal hair in the pubic area and he had a fine voice with eunuchoid habitus. Imaging studies as well as test were unremarkable, except for low serum levels of FSH, LH, HCG, total testosterone and free testosterone indicating severe hypogonadism. The patient was stared in low dose of testosterone, with further counseling of fertility.

Discussion: Most cases of Kallmann's syndrome are sporadic, but familial occurrence also occurs. Inheritance is usually X-linked, as judged by the much greater number of cases in males than females. However, autosomal dominant or recessive transmission can occur. More recently, genetic testing has provided a more detailed understanding of the disease. Studies of patients with Kallmann's syndrome have demonstrated mutations of several genes that encode cell surface adhesion molecules or their receptors required for the migration of GnRH-secreting neurons from the olfactory placode into the brain and then into the hypothalamus. These genes include KAL1 , fibroblast growth factor receptor 1 (also called KAL2) , prokineticin-2 (PROK2) and its receptor (PROKR-2).

Conclusion: These mutations account for less than 50% of the cases. Therefore, identifying new patients with the disease that do not have previously described mutations may shed a new light into the pathophysiology of this rare disorder and bring upon further understanding of the mechanism that govern gonadal function.

Abstract #825

COINCIDENCE OR CAUSALITY? A RARE CASE OF SARCOIDOSIS AND MULTIPLE MYELOMA

Natalia Branis, MD, K. Chaudrey, V. Privman

Objective: Sarcoidosis is a chronic systemic condition of unknown etiology accompanied by a multilevel disruption of the immune system. Its incidence widely varies throughout the world with annual incidence among African-Americans being the highest at 35.5 cases per 100,000. However, only 15 cases of multiple myeloma in patients with sarcoidosis have been reported worldwide. Here, we describe a patient with a rare combination of sarcoidosis and multiple myeloma.

Case Presentation: A 39-year-old African-American female with h/o sarcoidosis, lung aspergilloma and asthma presented with subjective fever, productive cough and right-sided pleuritic chest pain of sudden onset 3 days ago. Physical exam was remarkable for decreased air entry to both lungs and right chest wall tenderness. ECG revealed short QT interval and chest x-ray demonstrated unchanged apical cavitation with scarring in the right lung. Patient was found to have severe hypercalcemia (serum Ca 17.3 mg/dL (8.8-10.3 mg/dL)), which later required bisphosphonate administration. She was successfully treated for acute bronchitis. New onset of hypercalcemia prompted further work-up that demonstrated PTH 6.02 pg/ml (10-60 pg/ml), PTH-rp less than 2.1 pmol/L, 25-Hydroxy-vitamin D 12 ng/mL (30-74 ng/mL), 1,25-Dihydroxy-vitamin D 11 pg/mL (15-75 pg/ml) and normal ACE levels. Serum protein electrophoresis revealed abnormal Ig G κ M-protein. She was noticed to have an abnormal band in γ-globulin region one year ago. A subsequent bone scan demonstrated multiple bilateral rib fractures. Diagnosis of multiple myeloma was confirmed by bone marrow biopsy (plasma cell population IgG/Kappa/CD45/CD56+). Patient was started on chemotherapy.

Discussion: Coexistence of sarcoidosis with various forms of malignancy has been noticed previously. A concept of 'sarcoidosis-lymphoma syndrome' was introduced by H.Brincker in 1986. Often malignancy is seen in chronic active form of sarcoidosis. However, sarcoidosis and multiple myeloma are rarely observed together. The causal connection between two disorders remains unknown. Possibly, an increase in CD4+ count and decrease in CD8+ T-cells seen in sarcoidosis facilitates continuous stimulation of B-cells. This leads to increased production of gammaglobulins, increased chances of mutation and development of multiple myeloma or other malignancies. Development of multiple myeloma is often preceded by monoclonal gammopathy of uncertain significance. It manifests itself as a premalignant condition with a 1% annual rate of progression towards multiple myeloma.

Conclusion: Collecting data on more patients with sarcoidosis and multiple myeloma is necessary for further investigation of connection between these two disorders.

Abstract #826

SECONDARY HYPERPARATHYROIDISM IN METASTATIC PROSTATE CANCER: A NEW SYNDROME?

Gaurav Shah, MD, Alan Peiris, MD, PhD, Bhavesh Barad

Objective: Secondary hyperparathyroidism is well recognized in renal insufficiency and vitamin D deficiency. Rarely, certain malignancies with metastatic involvement of bone such as breast and prostate cancer can be associated with hypocalcemia. We report a case of elevated parathyroid hormone (PTH) in the presence of a normal

ionized calcium, normal renal function and vitamin D in a patient with metastatic prostate cancer.
Case Presentation: A 77 year old Caucasian male was referred to endocrine clinic because of an elevated PTH level. On presentation he was feeling weak, nauseated and had a 29 lb weight loss over the last 2-3 months. He also had a history of colon cancer treated with a partial colectomy about 22 years ago, hyperlipidemia and Prostate cancer. His only medications were Finasteride and Simvastatin. His initial diagnosis of prostate adenocarcinoma was made in 1995. Various treatment modalities were tried including brachytherapy, radiation and chemotherapy. No abnormal physical findings were noted. Recent bone scan was strongly suspicious for widespread skeletal metastatic disease, likely related to prostate cancer. His PSA was 386.52(Normal 0-4.0] and alkaline phosphatase was 274 [Normal 38-126] which were consistent with metastatic prostate cancer. Treatment with calcium, Vitamin D followed by bisphosphonates resulted in a dramatic improvement in wellbeing, near normalization of PTH levels (64/71) and a reduction in alkaline phosphatase (236). However, PSA level increased (429).
Discussion: We believe that secondary hyperparathyroidism in prostate cancer is underestimated and likely has multiple etiologies including the presence of vitamin D deficiency and perhaps renal impairment. However, in our patient another etiology could well be the incorporation of calcium into osteoblastic metastases, resulting in PTH elevation secondary to the fall in ionized calcium. We believe that failure to recognize this syndrome may lead to hypocalcemia related to hungry bone syndrome. An elevated PTH value per se has been linked to a variety of adverse health outcomes including enhanced mortality.
Conclusion: Our patient responded very well to treatment with calcium and vitamin D followed by bisphosphonates. Initial treatment with calcium and vitamin D has the benefit of improving wellbeing and possibly reducing the side-effects of bisphosphonates. Providers managing patients with prostate cancer should be cognizant of this phenomenon and check PTH values especially in the presence of metastases either radiologically or in the presence of elevated alkaline phosphatase values.

Abstract #827

NOT YOUR ORDINARY NECK PAIN: CONCURRENT MULTIPLE MYELOMA AND HYPERPARATHYROIDISM

Zarah Lucas, MD, Aileen Cielo, Jowella Pineda

Objective: To recognize the coexistence of hyperparathyroidism and multiple myeloma.
Case Presentation: A 62-year old Caucasian male was admitted for neck pain of 3 months' duration. It was initially managed as osteoarthritis with intra-articular steroids, which provided no relief of the neck pain. MRI of the neck revealed multilevel degenerative disc disease, no cord compression and diffuse heterogenous bone marrow signal. Bone scan showed multiple foci of increased uptake in the ribs bilaterally. The patient's pain persisted despite opioids, thus he underwent bone marrow biopsy. After the procedure, the patient felt increased neck pain and he was then brought to the emergency department for evaluation. Physical exam revealed no fever and unremarkable systemic and neurologic examination. Initial work-up showed a normal CBC, mild renal insufficiency with creatinine of 1.29 and hypercalcemia 12.1 mg/dL. CT of the cervical spine revealed osteolytic lesions of all cervical vertebra and complete collapse of C3. A hard collar stabilized his cervical spine while awaiting surgery, and hypercalcemia was managed with aggressive saline hydration, calcitonin and pamidronate. Further work-up yielded an elevated PTH at 72.5pg/mL, low normal phosphorus 3.2mg/dL and normal PTHrP at 16pg/mL. Elevated free kappa light chains were noted on serum and urine immunofixation. Plasma cells involved 20% of the marrow cellularity and fluorescent-in situ-hybridization was positive for monosomy 13. After his cervical spine fusion, he received chemotherapy with bortezomib, thalidomide and dexamethasone as outpatient. His calcium levels have been within normal range with regular administration of zoledronic acid.
Discussion: The combination of multiple myeloma and primary hyperparathyroidism is rare but well documented. In a prospective study involving patients undergoing surgery for primary hyperparathyroidism, the prevalence of monoclonal gammopathy, including 2 cases of multiple myeloma, was 10%. On the other hand, the prevalence of monoclonal gammopathy in patients undergoing surgery for other diseases was 2-3%. On review of literature, there are 19 cases reporting the coexistence of multiple myeloma and hyperparathyroidism. The association may be explained by (1) simple chance; (2) monoclonal immunoglobulin may act as a growth factor for parathyroid cells; (3) parathyroid hormone stimulate osteoblasts to secrete IL-6, which drives myeloma proliferation; and (4) a common inherited or acquired gene may predispose to both conditions.
Conclusion: Consideration of both hyperparathyroidism and multiple myeloma in a patient with hypercalcemia is therefore important.

Abstract #828

AN INDEX OF CEREBRAL FLOW RESERVE CORRELATES WITH STROKE INCIDENCE IN MIDDLE-AGE (54+-15 YEARS) ENDOCRINE-METABOLIC PATIENTS WITH CEREBRAL NEUROLOGIC SYMPTOMS

Harold Pretorius, MD, PhD, John Idoine, Dennis Menke, Nichole Richards, Elizabeth Alexander

Objective: Report the relation between lifetime stroke incidence (S) and an index of cerebral flow reserve (FRi) from brain single photon emission computed tomography (SPECT) in endocrine-metabolic patients with cerebral symptoms.
Methods: Analysis of S and FRi included interrelated effects of blood pressure (BP), diabetes mellitus (DM), insulin resistance (IR), pituitary disease (PitD), brain trauma (TBI), thyroid (TD) and renal disease (RD), patient age (A) in years, Test Your Memory (TYM) scores, SPECT indices of brain metabolism (CMi) basal, perfusion (CPi) stimulated (e.g. acetazolamide 500 mg IV) and the ratio (GR) of calculated (from serum cystatin C or creatinine) to predicted glomerular filtration rate (GFR) in ml/min: GR = GFR/(160-A). GFR was considered high if GR > 1.15 and low if GR < 0.85.
Case Presentation: In 41 near normal patients, age was 51+-16 years, CMi (58.5+-4.6)%, CPi (66.3+-5.6)%, FRi (9.6+-3.1)% and TYM 47.3+-1.7. Similar middle-age patients with endocrine-metabolic disease and cerebral symptoms, most often memory loss, defined S = 13.9 -(1.4)(FRi), including 85 PitD with FRi -(7.98+-7.47)%, S 25%; 218 DM with FRi (0.75+-8.96)%, S 13.3%; 107 TD with FRi (0.44+-8.29)%, S 12%; 77 low GFR patients with FRi (0.47+-7.5)%, S 14% and 98 high GFR patients with FRi -(4.2+-5.9)%, S 19%. FRi in RD was corrected by subtracting {(3.22)[1-GR]} squared. In 94 patients age 79.8+-5.4 years, > 30% had atrial fibrillation or heart failure, > 60% had RD or IR, and FRi (2.0+-9.5)%, S 37% were >> expected in younger, middle-age patients. PitD was common (>40%) in TBI. Treatment trended to improve FRi and symptoms, e.g in 54 PitD patients, increasing FRi to (8.2+-6.6)% and TYM from 42.9+-5 to 46.3+-2, both near normal.
Discussion: FRi correlation with S suggests that physiologic response to pathologic vascular stress is critical to prevent stroke in middle-age endocrine-metabolic patients. Higher S in RD required FRi correction, here reduced to a single, nonlinear subtraction term for high or low GFR vs. separate, linear terms we reported before. This result suggests additional stroke pathophysiology, such as increased heart disease and likely increased inflammation and thromboembolism in RD. Perhaps progressive microangiopathy, common in longstanding IR or DM, also contributes to stroke in older patients, for whom S increases even beyond that predicted by the also high age-related incidence of RD.
Conclusion: Facilitating recognition of stroke risk with FRi and cognitive screening in potentially treatable, middle-age endocrine-metabolic patients may be important to reduce the overall burden of stroke-related long-term disability, the leading cause of long-term disability in most countries.

Abstract #829

POLYGLANDULAR AUTOIMMUNE SYNDROME TYPE 2 PRESENTING WITH VENTRICULAR TACHYCARDIA

Olusegun Sheyin, MD

Objective: To report a case of polyglandular autoimmune syndrome(PGA) type 2, presenting with sustained ventricular tachycardia to the emergency unit of a tertiary hospital in Lagos.
Case Presentation: A 36 year old female, known type 1 diabetic presented with a 3 week-history of heat intolerance and intermittent symptomatic palpitations. Examination findings after stabilization included a BMI of 15.2kg/m2, pallor, generalized hyperpigmentation, a 30g-sized goiter and an unrecordable pulse and blood pressure. Cardiac monitor revealed a sustained ventricular tachycardia at a rate of 186 b.p.m. Thyroid function tests revealed fT3 of 7.5pmol/L(reference 3.8-6.0), fT4 of 30pmol/L(7.2-16.4) and TSH of 0.01Miu/L(0.37-3.50) in keeping with thyrotoxicosis. Basal cortisol was low(3.5nmol/L), with 30- and 60-minute samples following co-syntropin administration of 92.9nmol/L and 105.5nmol/L respectively. Anti-thyroglobulin and anti-thyroid peroxidase antibodies were positive. Serum chemistry revealed hyponatremia(120mmol/L), potassium of 5.0mmol/L, normal anion gap metabolic acidosis of 19mmol/L and a fasting blood glucose of 136mg/dL. Hematology was significant for a macrocytic anemia (Hematocrit 26%). A diagnosis of sustained ventricular tachycardia in a patient with PGA type 2 with co-existent pernicious anemia was made. Treatment began with chemical cardioversion with intravenous lidocaine, transfusion with 2 units of packed cells and correction of electrolyte abnormalities. Hydrocortisone given intravenously initially and then orally, fludrocortisone together with divided daily doses of carbimazole and propanolol were also instituted. The patient reverted to sinus rhythm and was monitored in the intensive care unit. She was discharged following stabilization and is being followed up at the Endocrinology and Hematology clinics.
Discussion: Polyglandular autoimmune syndrome is an

immunoendocrinopathy which comprises of multiple endocrine gland insufficiency. Type 2 PGA syndrome is characterized by the obligatory occurrence of autoimmune Addison's disease with thyroid autoimmune disease (hypo or hyperthyroidism) and/or type 1 diabetes mellitus. Electrolyte abnormalities are frequently found in PGA type 2 and may predispose to arrhythmias on a background of thyroid dysfunction. To the best of my knowledge, no case of PGA type 2 presenting with ventricular tachycardia has been reported in the literature.

Conclusion: The presence of an immunoendocrinopathy warrants the search for other endocrine hypofunction. Early recognition and replacement therapy can be lifesaving. In situations where a life-threatening arrhythmia is present, urgent identification and appropriate treatment of the arrhythmia is top priority.

Abstract #830

EFFECT OF VITAMIN D SUPPLEMENTATION IN TYPE 2 DIABETES PATIENTS WITH PULMONARY TUBERCULOSIS

Sunil Kota, MD, Siva Kota, Svs Krishna, Lalit Meher, Kirtikumar Modi

Objective: We evaluated the effects of vitamin D supplementation on type 2 diabetes mellitus (T2DM) patients with pulmonary tuberculosis (PTB).

Methods: 45 subjects (M: F= 34: 11) were screened. Inclusion criteria were age > 15 years, newly diagnosed PTB cases with uncontrolled T2DM, serum vitamin D < 20 ng/ ml. The patients were randomly assigned to 2 groups. Group 1 subjects received oral cholecalceferol (60000 units/ week) and calcium carbonate (1 gm/day) along with anti tubercular treatment (ATT), while group 2 subjects did not. Sputum was checked at interval of 2 weeks for 12 weeks. Primary end point was time to achieve sputum smear conversion. Secondary end points were reduction in ESR and improvement in glycemic parameters.

Results: 15 patients with vitamin D > 20 ng/ml were excluded. So the prevalence of vitamin D deficiency in T2DM with PTB was 30/45 (66.66 %), with 25/34 males (73.5%) and 5/11 females (45.5%) were deficient in vitamin D levels. Mean age of patients (30) was 39.5 ± 18.9 years with FBS 230.5 ± 30.3 mg/dl, PLBS 320.5 ± 45.6 mg/dl, HbA1C 10.4 ± 4.4 % and 25 (OH) D 12.1 ± 4.3 ng/ ml. At the end of 12 weeks, group 1 patients had significantly higher levels of serum 25 (OH)D (25.4 ± 6.9 ng/ ml in group 1 versus 10.2 ± 0.9 ng/ ml In group 2). Sputum smear conversion was 6 weeks in group 1 versus 8 weeks in group 2 (p= 0.067). Reduction in ESR was significant in group 1 vs group 2 (39.6 ± 12.4 mm/ 1st hr vs 24.0 ± 14.9, p-0.004). Difference in the reduction in FBS, PLBS and HbA1C in the 2 groups did not attain statistical significance.

Discussion: Correlations exist between low vitamin D levels with PTB and low vitamin D levels with T2DM. Calcitriol modulates the host response to mycobacterial infection by induction of reactive nitrogen and oxygen intermediate, suppression of matrix metalloproteinase enzymes implicated in pulmonary cavitation, and induction of antimicrobial peptide cathelicidin. Calcitriol modulates immune responses by binding vitamin D receptors expressed by antigen-presenting cells and activated lymphocytes. Several case series have reported utility of 25000 IU to 100000 IU vitamin D in improving patients' response to ATT. Vitamin D supplementation also increases lymphocyte to monocyte ratio, a biomarker of disease resolution. We provided vitamin D at the dosage of 60000 IU / week, and report a 2 weeks reduction in sputum smear conversion. Though the sputum smear conversion was not significantly faster in group 1, it showed a trend towards earlier smear negativity.

Conclusion: Vitamin D may be the missing link between emerging epidemic of tuberculosis & diabetes. Vitamin D can serve as adjuvant treatment of tuberculosis in diabetics with vitamin D deficiency.

Abstract #831

AN OVERLOOKED CAUSE OF HYPERCORTISOLURIA

Mallory Carr, Alan Peiris, MD, PhD

Case Presentation: While endogenous Cushing's syndrome is a rare entity, the diagnostic workup can be challenging. We report a patient that presented with a marked excess in urine cortisol excretion. A 60 year old male veteran presented with diabetes mellitus, hypertension, obstructive sleep apnea, morbid obesity and hyperlipidemia. Family history was positive for type 2 diabetes. Medications included: atenolol, furosemide, gabapentin, sertraline, nifedipine, chlorthalidone, zolpidem, lisinopril, omeprazole, simvastatin, spironolactone, glyburide, glargine and regular insulin. Physical examination was pertinent for decreased sensation in lower extremities to monofilament and vibration consistent with diabetic neuropathy. Patient was obese (BMI of 44 kg/m) with a blood pressure of 140/70 mmHg. Other vital signs were normal. HbA1c was 9 %. A 24 hour urinary free cortisol was obtained on several occasions and values ranged from 108-181 mcg/24hr [normal, 0-50mcg/24hr], with urine volume ranging from 4500-5700 ml [normal, 600-1600ml], and normal urinary creatinine [1-2g/24hr]. Liver tests, renal function and electrolytes were normal. A serum midnight cortisol level measured 5 mcg/dL. A morning serum cortisol was

1.3mcg/dL after 1 mg dexamethasone.
Discussion: Given the normality of the overnight dexamethasone suppression test and midnight serum cortisol, we do not believe this patient had Cushing's syndrome. Patient had a long history of large fluid intakes for "health reasons" and could not comply when asked to decrease fluid intake. While there has been some controversy regarding the link of urinary free cortisol to urine volume, we believe that the high urinary cortisol reflects the increased urine flow.
Conclusion: Physicians should account for urine volume when evaluating urine cortisol levels.

Abstract #832

A RARE NEUROENDOCRINE TUMOR DECLARES ITSELF THROUGH FAILING HEART.

Nidhi Bansal, MBBS, Hani Kozman

Case Presentation: Neuroendocrine tumors account for <1% of all pancreatic neoplasms. We present a unique case of a 70 y/o old lady with past history of hypertension and osteoarthritis, who reported worsening pedal edema, dyspnea & abdominal discomfort. Physical exam revealed jugular venous distension, systolic murmurs over pericardium, hepatomegaly and B/L 3+ pedal edema. Metabolic profile and TSH were within normal limits. Chest X ray revealed moderate cardiomegaly. Echocardiogram showed aortic, tricuspid and pulmonary regurgitation with left ventricular hypertrophy. A diagnosis of decompensated diastolic heart failure was made and appropriate medical therapy was instituted. Lack of significant symptomatic improvement created a management dilemma for the primary team. Her hepatomegaly previously thought to be secondary to heart failure also persisted. This resulted in sonogram of the upper abdomen which surprisingly showed 2 large masses (>5 cm) in the right lobe of liver. Hepatitis viral serology and AFP tests were normal. CT scan of abdomen detected a new 3 cm mass in pancreas and other multiple hepatic lesions to further add to the mystery. A core biopsy of hepatic mass lesion showed complete replacement of liver by tumor cells with round nuclei and abundant cytoplasm containing eosinophilic granules. Immunostains showed marked positivity for synaptophysin and chromogranin, confirming their neuroendocrine origin. She subsequently underwent somatostatin receptor scintigraphy to reveal increased uptake in pancreas and multiple sites in liver. EUS of pancreas was done to delineate the tumor extent. Blood chromogranin levels were extremely high at 2885 ng/ ml (normal <50 ng/ ml). Urine 5- HIAA was 67 mg/ g creatinine (normal <14 mg/ g creat.). Thus a diagnosis of primary pancreatic carcinoid with hepatic metastases with carcinoid syndrome was made. The patient refused any aggressive interventions like radio frequency ablation or chemoembolization. She was initiated on sandostatin therapy with partial relief of her symptoms.
Discussion: Pancreas is an exceedingly rare location for foregut derived carcinoid tumors (< 0.6% of total cases). Prevalence of symptomatic pancreatic carcinoids are even rarer (estimated to be 1:10) and are usually associated with metastases or pancreatic duct compression. Most frequent associated symptoms are abdominal pain (66%) and diarrhea (52%). Our patient presented with abdominal pain and carcinoid heart disease secondary to infiltration of cardiac chambers and valves.
Conclusion: Pancreatic carcinoids are difficult to diagnose early due to subtle presentation. High index of suspicion is needed for timely diagnosis as metastatic disease carries a poor prognosis.

Abstract #833

DRAMATIC DIFFERENCES IN LONG-TERM CURE RATES FOLLOWING UNILATERAL VS. BILATERAL PARATHYROIDECTOMY: AN 18-YEAR, SINGLE-CENTER STUDY IN 15,500 PATIENTS

James Norman, MD, FACS, FACE, Douglas Politz, MD, Jose Lopez

Objective: To establish if unilateral parathyroidectomy for primary hyperparathyroidism (pHPT) provides equal long-term cure rates to those seen following bilateral surgery where all four glands are evaluated.
Methods: An 18 year, single center study of 15,500 patients undergoing parathyroidectomy for pHPT examined the number of glands removed and recurrence rates for unilateral vs. bilateral operations. To be included, all 15,500 patients were "cured", having normal calcium and PTH levels for a minimum of 2 months postop following removal of one or more abnormal parathyroid glands. 92% of failures underwent re-operation. Followup is 100% at 1 year, 97% at 5 years, 94% at 10 years, and 92% at 18 years. Mean followup is 6±4 years, range 0.5 to 18 years.
Case Presentation: Biochemical profiles and demographics were identical between groups. Unilateral operations were chosen based on localizing studies; 3,084 (19.9%) operations were unilateral while 12,416 (80.1%) were bilateral. More than one abnormal parathyroid gland was removed in 26% of bilateral, but only 3% of unilateral operations ($p<0.0001$). The recurrence rate (cumulative) for bilateral operations was 0.19% at one year, 0.27% at 10 years, and 0.31% at 18 years ($p=0.96$). In contrast, unilateral operations had recurrence rates of 3.9% at 1 year, 5.9% at 5 years, 8.1% at 10 years, and 10.1% at 18 years

(all $p<0.0001$), never reaching a plateau. Calcium and PTH levels at recurrence were lower than initially (10.9±0.2 vs. 11.4±0.3), and (102±32 vs. 115±44) respectively, (both $p<0.001$). At reoperation, unilateral patients had a second (or third) adenoma (not hyperplasia), which were smaller in size (19.6±7 vs. 24.3±9 mm) and weight (938±112 vs. 1142±130 mg) on average than the first removed adenoma (both $p<0.001$). Following a curative second operation, all patients remained cured long-term (mean followup 4±3 years, range 0.5 to 17 years).

Discussion: This study examines recurrence rates in patients believed to be cured after their first operation and does not take into account the initial 2-5% higher rate of initial operative failure seen with unilateral parathyroidectomy. This will be addative to the higher recurrence rates seen here.

Conclusion: Four-gland evaluation during parathyroidectomy for pHPT dramatically increases the number of abnormal glands removed resulting in cures that are durable for nearly two decades. In contrast, 8% of patients "cured" after a unilateral parathyroidectomy will recur by 10 years, recurring at a rate of 0.4% per year thereafter. Even with strict patient selection by highly experienced surgeons, unilateral parathyroidectomy is an inferior operation requiring closer followup.

Abstract #834

A PROJECT FOR ESTABLISHMENT A CENTER OF EXCELLENCE IN PEDIATRIC ENDOCRINOLOGY WITH DIABETES

Ali Al-Jumaili, MD

Objective: to describe a project aimed at establishment a center of excellence for the specialty of the pediatric endocrinology with diabetes.

Methods: 2-3 years project was designed to Introduce changed requirements for developing the existing diabetic clinic to a center of excellence for pediatric endocrinology and diabetes .Including the key drivers, sponsors, analysis of the existing clinic with identification problem areas, effective stakeholders, milestones of the change. New location and challenges including unfavourable security aspects related to terrorism in our country. Future vision for the health services. Timeline for implementation. Detailed planning took place between March 2007 and the January 2008. The programme of change began at March 2008 with agreement of Minister of Health, followed by new site location at January 2009. The next step was training staff, and then proceeded with organized phases beginning with opening the hormonal assay unit at June 2009 followed by opening pediatric endocrinology with diabetes inpatient ward late at 2009. During 2010, more medical trained staff with new laboratory equipments related to the center added. At the beginning of 2011, we substituted the growth hormone vials by multi doses pens in the country for scientific and economic reasons. Because the adverse security conditions, there is delay in finishing the center building

Results: The project was successfully applied. Improvement in the following occurred: Majority of investigations performed in our hospital under our supervision, good control and follow up of the inpatients and outpatients in comparison with the previous state. Majority of pediatric endocrine disorders and diabetic cases now referred to our center from different hospitals and specialists for management or consultation. Focus for training and teaching with better patient's outcome, participation in national and international scientific meetings

Discussion: When setting up a project you need to be taken into account the size of gap that exists between the rationality of the project design and the strategies of the high health authorities and the key drivers for the change and must be applicable and acceptable, leading to welfare of the community.

Conclusion: When setting up a project you need to be taken into account the size of gap that exists between the rationality of the project design and the strategies of the high health authorities and the key drivers for the change and must be applicable and acceptable, leading to welfare of the community.

Abstract #835

AGONISTIC AUTOANTIBODIES AS VASODILATORS IN ORTHOSTATIC HYPOTENSION: A NEW MECHANISM

Megan Vanderlinde-Wood, M.D., Muneer Khan, M.D., Allison Galloway, DO, Hongliang Li, Xichun Yu, David Kem, MD

Objective: Circulating agonistic autoantibodies serve as vasodilators and may cause or exacerbate orthostatic hypotension.

Methods: Patients were categorized into 10 idiopathic OH and 10 diabetics with OH. Purified IgG from all 20 patients and 10 healthy control subjects were examined in a receptor-transfected cell-based cAMP assay for β2 receptor activation and β-arrestin assay for M3 receptor activation.

Results: IgG from 3 OH patients (2 idiopathic OH and 1 diabetic OH) who were strongly ELISA positive for autoantibodies to β2AR and M3R were tested for their ability to activate β2AR and M3R in specific receptor-transfected cells. The idiopathic OH and diabetic OH groups both showed significantly increased β2AR activation when

compared to healthy controls (P=0.007 and P=0.014 respectively). The increases in M3R activation in the idiopathic OH and diabetic OH groups were even more significant (P=0.002 and P=0.003). This significant finding has been plotted with individual OH subject's bioactivity measured by the receptor activation assays compared with their ELISA OD values.

Discussion: OH is a condition that is associated with increased mortality causing falls and injury. It has been associated with nervous system disorders, diabetes, dehydration and heart conditions. Although patients with obvious central or peripheral neuropathies have reason to demonstrate significant orthostasis, many other subjects have either minimal or no evidence for such a severe autonomic deficiency yet present with clinically relevant symptoms and signs of OH. It has been demonstrated before that in diabetic hypoadrenergic orthostatic hypotension the basic pathophysiological defect is the lack of ability to increase vascular resistance, probably due to impaired sympathetic activity in the autonomic nerves innervating resistance vessels; cardiac output and plasma volume responses to standing are similar to those found in normal subjects and in diabetics without neuropathy (JCI. 1981 68(6): 1427-1434). There are multiple pathways involved in the pathophysiology of orthostasis. Our finding that vasodilatory autoantibodies to β2AR and M3R are present in a high proportion of OH patients with and without apparent autonomic dysfunction suggests that these autoantibodies may cause or exacerbate orthostasis by altering the compensatory postural vascular response.

Conclusion: These data support the concept that circulating agonistic autoantibodies serve as vasodilators and may cause or exacerbate OH. (Hypertension. February 2012)

Abstract #836

A CASE OF LOW TESTOSTERONE SECONDARY TO LONG-TERM STEROID THERAPY FOR UNCONTROLLED ASTHMA

Esti Charlap, MD, Patricia Dharapak

Objective: To report the occurrence of symptomatic low testosterone levels in a man on long-term steroid therapy secondary to uncontrolled asthma.

Case Presentation: A 54 year old man with severe asthma, with a history of multiple intubations in the past and frequent asthma-related hospitalizations, was admitted to the hospital with the complaint of one week of increasing shortness of breath. This was accompanied by a cough and wheezing. He had just finished a two-week Prednisone taper for an asthma exacerbation two days prior to admission, and had been on many similar steroid tapers in the past. He also had a history of diabetes and osteoporosis secondary to prolonged steroid use. In the hospital, he was started on antibiotics, nebulizers, and high dose steroids and his respiratory symptoms began to improve. On subsequent interviewing, he endorsed a history of excessive sweating and hot flashes, as well as fatigue, decreased energy, and depressed mood over the past few months. He denied a history of a decreased sex drive or erectile dysfunction. Blood work revealed a decreased total testosterone of 127 ng/dl (normal 181-758 ng/dl) and a free testosterone of 38 pg/ml (50-350 pg/ml). Estradiol was decreased at 7.3 pg/ml (11-41 pg/ml). Sex hormone binding globulin, FSH, and LH were normal. He was discharged home with an endocrinology appt. for further work-up and treatment for his decreased testosterone.

Discussion: Although testosterone levels decline steadily after age 40, the decline is relatively small, and only a small percentage of men have levels far below those considered normal for their age. The symptoms of low testosterone include low sex drive, erectile dysfunction, mood problems, fatigue, and sleep disturbances. At the lowest levels, men may experience hot flashes, much like those experienced by women during menopause. It has been reported that male patients taking high-dose steroids for a prolonged period of time have low testosterone levels but normal gonadotropin levels, suggesting a predominantly central mechanism for the decrease in testosterone. Symptoms improve with hormone replacement therapy aimed at normalizing testosterone levels, as well as those secondary hormones that are affected by testosterone levels. Exercise, dietary changes, and stress reduction are beneficial as well. Selective androgen receptor modulators have also been proposed as a possible treatment.

Conclusion: Prolonged steroid therapy has been shown to be associated with decreased testosterone levels in men. It is important to consider low testosterone in any male patient presenting with nonspecific complaints such as hot flashes, fatigue, decreased energy, or decreased mood, but especially in those at increased risk.

Abstract #837

EFFECT OF VITAMIN D REPLACEMENT ON INSULIN SENSITIVITY IN SUBJECTS WITH VITAMIN D DEFICIENCY

Vinaya Simha, MD, Muhammad Mahmood

Objective: Low Vitamin D levels have been shown to correlate with measures of insulin resistance and prevalence of diabetes mellitus. However, there is limited and conflicting data on changes in insulin resistance following Vitamin D replacement. The objective of the current study was to examine changes in insulin sensitivity in Vitamin D deficient subjects receiving replacement therapy with

Ergocaclciferol in comparison to placebo treated subjects.
Methods: Design: Randomized double-blind placebo controlled trial (Clinical trials.gov identifier: NCT01268111). Subjects: 12 healthy subjects with plasma 25-hydroxy Vitamin D (25 OHD) levels less than 20 ng/mL, randomly divided into two age (29 ± 3.6 y and 27.8 ± 2.6 y) and sex (4 male, 2 female) matched groups. The mean BMI was 23.6±2.9 and 24.7±1.3 respectively, and none of the subjects were taking any medications or other supplements. Primary outcome: Mean insulin stimulated glucose infusion rate (GIR) during the last 20 minutes of hyperinsulinemic-euglycemic glucose clamp studies conducted before and after the intervention. Insulin was infused at a rate of 40mU/M2.min during the clamp studies. Intervention: Treatment with Ergocalciferol 50,000 units once a week for 8 weeks or matching placebo
Results: Plasma 25 OHD D levels tended to increase from 13.3 ± 3.8 ng/mL to 18.8 ± 5 ng/mL in the Ergocaclciferol group (p=0.09), while it did not change in the placebo group (15.8 ± 2.4 to 13.1 ± 2.6 ng/mL, p=0.14). The post treatment levels were higher in the Ergocalciferol group (p=0.05). GIR during the last 20 minutes of the glucose clamp studies were similar between the two groups both at baseline and after 8 weeks of treatment. In the Ergocaclciferol group, the GIR increased non-significantly from 4.1 ± 1.6 mg/kg.min to 4.7 ± 1.4 mg/kg.min (p=0.4), while in the placebo group, the corresponding values were 5.6 ± 1.5 and 5.5 ± 1.7 mg/kg.min, respectively.
Discussion: Following 8 weeks of Vitamin D replacement therapy, we did not notice a significant improvement in insulin sensitivity. However, post treatment 25 OHD levels were still below 20 ng/mL in 4/6 subjects in the Ergocaclciferol group, and none of the subjects achieved levels greater than 30 ng/mL. A more aggressive replacement therapy may be required to restore 25 OHD levels to normal, and the effect of such therapy on insulin sensitivity needs to be studied. Given the small increase noted in this study, it is possible that more robust Vitamin D replacement may improve insulin sensitivity.
Conclusion: Administration of Ergocalciferol 50,000 units weekly for 8 weeks in subjects with low 25 OHD levels did not improve insulin sensitivity as measured by glucose clamp studies.

Abstract #838

EVIDENCE FOR NON-FUNCTIONAL PARATHYROID ADENOMAS

Lawrence Kim, MD, Carrie Hyde

Objective: Prior to the era of directed parathyroidectomy, the standard approach to parathyroidectomy was the removal of all grossly enlarged glands, the assumption being that enlarged glands were hyperfunctioning. In a previous report, we correlated gross findings with intraoperative parathyroid hormone (ioPTH) measurements and found that 4 of 17 (24%) patients with multigland disease as defined by gross inspection had an inappropriate decline in ioPTH and predicted a 6% failure rate for cases guided by ioPTH. However several reports of directed parathyroidectomy with ioPTH monitoring show a much lower failure rate than that prediction. One explanation for this discrepancy is the presence of non-functional parathyroid adenomas. In this report we present two cases which provide direct evidence for the existence of non-functional parathyroid adenomas.
Methods: Existing clinical records of two patients are presented.
Case Presentation: Patient A was a 26 year old man who presented to clinic for evaluation of recurrent papillary thyroid cancer. A hypoechoic nodule was found in the central compartment by ultrasound. FNA biopsy showed glandular cells that were atypical for thyroid tissue and the possibility of an enlarged parathyroid was considered. A serum calcium and PTH were obtained and were found to be normal (Calcium 9.6 mg/dl normal 8.6-10.2, PTH 39.5 pg/ml normal 12-88). At operation a 1.4 cm hypercellular parathyroid was found in addition to papillary thyroid cancer. Patient B was a 54 year old female with papillary thyroid cancer. Two preoperative serum calciums were obtained, 9.6 and 10.2 mg/dl. A preoperative serum PTH was not obtained. At the time of exploration, an enlarged parathyroid gland was encountered. Prior to disturbing the gland, an intraoperative calcium and PTH were obtained and were 8.6 mg/dl and 61.1 pg/ml respectively. Pathology revealed a hypercellular parathyroid, 19x7x7 mm, 509 mg.
Discussion: Both patients had no biochemical evidence of hyperparathyroidism yet clearly had parathyroid adenomas. The data for patient A are more complete, and show no evidence of hyperparathyroidism. The data for patient B are less complete because the only PTH value available is from a sample obtained after dissection had occurred in the vicinity of the gland. Taken together however, the evidence suggests that both patients demonstrated parathyroid adenomas in the absence of biochemical hyperfunction.
Conclusion: We conclude that non-functional parathyroid adenomas exist. Based on previous work and the literature these may be found in up to 6% of patients with hyperparathyroidism, and in an unknown proportion of normal individuals.

Abstract #839

ATYPICAL PRESENTATION OF HYPERCALCEMIA WITH PRIMARY HYPERPARATHYROIDISM AND FAMILIAL HYPOCALCIURIC HYPERCALCEMIA

Jason Jacob, MD, Ila Khanna, MD, Faryal Mirza, MD

Objective: Hypercalcemia is commonly encountered by clinicians in their practices. The differential for hypercalcemia and elevated parathyroid hormone (PTH) include primary hyperparathyroidism (PHPT) and familial hypocalciuric hypercalcemia (FHH) among many others. Typically these two disorders are seen as separate entities with their own set of criteria for diagnosis. There has been a growing number of case reports in the literature that illustrate a previously unidentified spectrum with both PHPT and FHH features.
Case Presentation: A 54 year old female of Jamaican descent with history of hypertension and hypercholesterolemia was referred to the endocrine clinic for evaluation of hypercalcemia and elevated parathyroid hormone levels. She was asymptomatic and denied a family history of kidney stones or known hypercalcemia. Physical exam was significant for a palpable left thyroid nodule. Subsequent work up confirmed above biochemical findings along with the presence of low urinary excretion of calcium despite hypercalcemia in the absence of thiazide diuretics, suggesting FHH. Thyroid ultrasound confirmed a 1.4cm nodule in the left lobe. The patient underwent fine needle aspiration biopsy twice, which revealed predominant microfollicular formation suspicious for microfollicular neoplasm, necessitating surgical intervention. A sestamibi scan was scheduled preemptive of thyroid surgery, which revealed a parathyroid adenoma in the lateral inferior pole of the left thyroid lobe. The patient underwent left thyroid lobectomy and simultaneous left inferior parathyroidectomy. Intraoperatively, parathyroid hormone declined from 150 pg/ml to 78 pg/ml with no change in calcium levels on the same day and on the first postoperative day. Pathology was consistent with multinodular goiter of the thyroid and confirmed the presence of a parathyroid adenoma with hypercellular PTH gland and compressed surrounding normal gland. The patient continues to have elevated levels of total calcium (10.5 mg/dl), ionized calcium (1.39 mmol/L), PTH (225pg/ml) and low urinary calcium excretion (0.037 gm/day) with low fractional excretion of calcium (<0.01 mmol/L).
Discussion: This case illustrates an atypical presentation of hypercalcemia with concomitant presence of PHPT and a likely diagnosis of FHH for which genetic studies are pending.
Conclusion: A review of the literature and genetic linkage studies have identified that the cytoplasmic tail of the calcium sensing receptor and the transmembrane domains may be implicated in the overlap between the two disorders. The role of vitamin D deficiency in accelerating parathyroid growth in FHH patients has also been hypothesized as an underlying pathology.

Abstract #840

VITAMIN D AND HUMAN SKELETAL HEALTH IN THE NEW MILLENNIUM

Sunil Wimalawansa, MD, PhD, MBA,FRCP,FACP

Objective: Several studies including a meta-analysis of eight randomized trials involving 2,426 older patients demonstrated that daily doses of vitamin D (700 to 1,000 IU) lowered fracture risk by 19%. Overall, the Cochrane Reviews suggest higher doses of vitamin D are more effective and provision of calcium with vitamin D is helpful.
Methods: Low vitamin D status is endemic, and most common among certain vulnerable groups and the elderly, predominantly due to inadequate sun-exposure. Most patients who need vitamin D supplements do not get them, or get them in inadequate doses. Moreover, long-term adherence to oral supplementation is poor.
Results: Better compliance seems to occur with 50,000 IU once or twice a month or 100,000 IU once a month. Such a maintenance dose can be commenced following a therapeutic loading dose of 50,000 IU of vitamin D given once or twice a week for a few weeks to obtain serum levels above 30 ng/mL.
Discussion: Low vitamin D status is endemic, and common among the vulnerable groups and the elderly. Whether widespread vitamin D deficiency is related to the increasing incidences of cancer, type 2 diabetes, obesity, etc. remains to be determined. Other vulnerable patients, such as the obese, those who have undergone bariatric surgery, and those with gastrointestinal malabsorption syndromes, may require much higher doses of vitamin D to maintain normal serum levels and be healthy
Conclusion: Whether widespread vitamin D deficiency is related to the increasing incidences of cancer, type 2 diabetes, obesity, and heart disease remains to be determined. Due to the high safety margin and the variability in measurements of serum 25(OH)D levels, and to assure adequate serum vitamin D levels, a maintenance value around 40 ng/ml would be useful. Clearly, vitamin D is directly correlated to one's quality of life. Endocrinologists are charged with using this information, efficaciously, helping their patients. Ref: Wimalawansa SJ. "Vitamin D: All you need to know." Karunaratne & sons, Homagama, Sri Lanka, 2012. Ross, A.C., et al., The 2011 report on dietary reference intakes for calcium and vitamin D from

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Abstract #841

CALCIUM SUPPLEMENTATION AND CARDIOVASCULAR EVENTS STUDY

Vaishali Patel, MD, James Vacek, Leland Graves, MD, Rajib Bhattacharya

Objective: Calcium supplements are commonly used in US to satisfy the daily dietary recommendations of calcium intake. Recent reports have suggested an association between calcium supplementation and increased rate of cardiovascular events. The goal of our retrospective study was to examine the association of calcium supplements with cardiovascular disease states and survival.
Methods: Data was collected for 5.7 years (1/1/2004 to 10/8/2009) from the electronic medical record of Mid America Cardiology, a large cardiovascular practice at the University of Kansas Medical Center. Patients > age 50 were included in our analysis. Diagnoses were derived from the patient problem list in the patients' electronic medical record based on International Classification of Diseases, 9th Revision (ICD-9) codes. Death was determined from the Social Security Death Index.
Results: The data set included 8060 subjects categorized as calcium supplement user vs. non-users. On multivariable logistic regression analysis, calcium supplementation was not an independent predictor of all-cause death, nor was calcium supplementation an independent predictor of the diagnosis of coronary artery disease. Survival analysis showed no survival differences for patients on calcium supplements (p=NS). Hazard ratios using the Cox proportional hazards model were calculated, showing that calcium supplementation was not an independent predictor of reduced survival. Hazard function analysis with additional predictive variables was then performed and again no overall significant impact of calcium supplementation on survival was evident.
Discussion: Our retrospective trial attempted to document important cardiovascular risk factors and other confounding variables which may have impacted the association of calcium supplementation with coronary artery disease and survival.
Conclusion: This study suggests that there was no association between calcium supplementation and coronary artery disease as well as overall survival in a large retrospective cohort of patients greater than 50 with high rates of significant cardiovascular risk factors.

PITUITARY DISORDERS

Abstract #900

THROMBOEMBOLISM IN A PATIENT WITH CRANIOPHARYNGIOMA: A SYMPHONY OF RISK FACTORS

Natalia Branis, MD, P. Park

Objective: Craniopharyngioma is a benign pituitary tumor which represents 6-13% of all pediatric brain tumor cases. Patients can present with non-endocrine symptoms such as headaches and visual disturbances, as well as signs of endocrine dysfunction. Management of craniopharyngioma includes surgery and radiation therapy. Post-operative period in these patients is challenging due to various degrees of hypopituitarism, high risk of thrombosis and neurocognitive dysfunction. Here we present a case of a young woman with craniopharyngioma, vitiligo and pulmonary embolism.
Case Presentation: An 18 year old female with vitiligo was evaluated for worsening headaches and intermittent vision loss. Her symptoms started 4 years ago. MRI brain with and without gadolinium contrast revealed 1.1*0.8*1 cm abnormal mass along the pituitary infundibulum, separate from the pituitary gland. Patient underwent surgical resection of the mass that proved to be benign craniopharyngioma. After surgery she developed hypopituitarism and was started on Desmopressin, Levothyroxine and Hydrocortisone; OCP was added 4 months post-operatively. Within 1 year after surgery patient gained 60 lbs. She complained of fatigue, excessive urination, shortness of breath, and bilateral leg weakness and swelling on exertion. Labs showed normal electrolytes, urine specific gravity >1.010, and thyroid hormones indicating adequate replacement of pituitary hormones. CT chest and pelvis with contrast revealed pulmonary embolism and IVC thrombosis. Patient was started on Enoxaparin and later transitioned to Warfarin; OCPs were discontinued upon diagnosis of the thrombosis.
Discussion: In the postoperative period following craniopharyngioma removal, thromboembolic complications, autoimmune phenomena and metabolic syndrome have been described. Dehydration and hemoconcentration secondary to diabetes insipidus, and Desmopressin therapy resulting in transient increase in von Willebrand factor level, are risk factors for developing thromboembolism. Excess weight is also considered to be a risk factor for venous thromboembolism. Following craniopharyngioma surgery, female patients are at greater risk of developing hypothalamic obesity as they suffer greater loss of appetite suppression compared with male patients. Various autoimmune conditions are associated with development of venous thrombosis.
Conclusion: In our patient with vitiligo, it is possible that venous thromboembolism was caused by a combination of autoimmune phenomenon with effects of Desmopressin and OCP therapy, as well as obesity. Patients with craniopharyngioma may benefit from early screening to evaluate their risk of thrombosis, and treatment with prophylaxis if indicated.

Abstract #901

RATHKE CLEFT CYST PRESENTING AS APOPLEXY.

Shalini Dabbadi Lakshmipathi, MD

Objective: 1. Present a rare case of Rathke Cleft Cyst (RCC) apoplexy. 2. Discuss presentation, imaging and histopathology of hemorrhagic RCC.
Case Presentation: A 61 year old gentleman presented with 1 week history of severe headaches, associated with nausea and blurring of vision. Neurological examination was normal. MRI of brain showed 11x12x11 mm rounded cystic mass within pituitary fossa with punctuate focus of T1 hyperintensity representing RCC with small amount of hemorrhage. Hormonal profile showed low TSH at 0.11(0.36 -5.6 uU/ml) and low triiodothyronine at 80 ng/dl(87-178 ng/dl) and free thyroxine was low normal at 0.83(0.6-1.89 ng/dl).There was also evidence of hypogonadotropic hypogonadism based on very low total and free testosterone at 55 ng/dl (300-720 ng/dl) and 9 pg/ml (47-244 pg/ml), respectively, with a concomitant low normal FSH 2.8 mIU/ml(1.27-19.26mIU/ml) and LH 1.84mIU/ml (1.24-8.62 mIU/ml). Other pituitary hormones showed cortisol at 12 ug/dl and ACTH at 28 pg/ml (7-69pg/ml), normal prolactin and IGF 1 values. Because of concern for apoplexy, patient was started on steroids and levothyroxine.He eventually had transphenoidal hypophysectomy which revealed xanthochromic fluid and predominantly necrotic material. Post operatively,headaches resolved.
Discussion: (RCC) are benign, epithelium-lined intrasellar cysts believed to originate from remnants of the rathke pouch. RCC often produce no symptoms and so are usually discovered incidentally. RCC presenting as pituitary apoplexy is very rare. Evaluation of symptomatic cases of RCC is done by CT and MRI studies. On CT scan images, these lesions appear as low density well circumscribed masses in the sella, with occasional rim like enhancement. The absence of calcification and homogenous enhancement of cyst on imaging strongly suggest RCCs rather than any other cystic pathologies like craniopharyngioma and arachnoid cysts. Hemorrhagic RCC needs surgical intervention, by drainage or excision of the cyst wall. Intraoperative findings are described

as proteinaceous, xanthochromic, yellowish to grayish creamy fluid and sometimes with necrotic tissue and blood. The presence of ciliated cuboidal epithelium on histopathology confirms the diagnosis.
Conclusion: Hemorrhage into an RCC is a rare event that mimics pituitary tumor apoplexy. In contrast to pituitary tumor apoplexy, hemorrhage into an RCC is more prevalent in females, and it is associated with a milder presentation with less significant visual symptoms, less severe pituitary dysfunction, and a smaller sellar mass. Transsphenoidal decompression is recommended in patients with visual compromise and those with pituitary dysfunction.

Abstract #902

OBSERVED MRI RESULTS IN MALES IDENTIFIED AS LOW TESOSTERONE WITH INAPPROPRIATE NORMAL RANGE LEUTINIZING HORMONE LEVELS

Edward Condon, MD

Objective: To identify the incidence of positive MRI findings in private practice when investigating males with secondary hypogonadism. There is an increase awareness of 'Low T' state in the media and the general public. General Practice and Urology do not always insist on Pituitary MRI, and Insurance Pre-authorization programs make MRI approval difficult.
Methods: During 2011 all males referred to Condon Medical P.C. for hypogonadism were investigated and if the average a.m. total testosterone was less than 300 ng/dl and the leutinizing hormone was not appropriately elevated the patients were sent for a contrast MRI of the pituitary. A retrospective review was carried out and pertinent history, blood results and MRI results were assessed.
Case Presentation: 166 patients were studied with the above criteria, one MRI request is still pending authorization. The 165 patients studied revealed the following results; pituitary tumor 36, empty sella 4, normal 100, pituitary cyst 12, brain tumor above the sella 4, multiple sclerosis 4, rathkes pouch cyst 5. Among the normal MRI patients, 63 were diabetic or dysmetabolic, 23 had histories of opiate use, 14 had prior anabolic steroid history, 3 had thyroid dysfunction and 24 had no obvious cause. Among the pituitary tumor group 12 were diabetic, 2 had prior opiate use, and 3 had prior anabolic steroid use. 3 Tumors were macro adenomas and required surgery, Amoung the brain tumor patients 1 had an astrocytoma superior to the pituitary and the patient did not survive, two were meningioma and 1 was not determined.
Discussion: There is an increasing awareness of males developing varying degrees of 'low T Syndrome' or hypogonadism. This small observation study illustrates the need for precise endocrine characterization of the cause of hypogonadism including anatomical assessment of the pituitary and surrounding region. Failure to do so may result in serious missed pathology.
Conclusion: There is a variable use of MRI in patients with low testosterone and inappropriately normal range Leutinizing hormone. Increasing diligence should be considered when investigating this group of patients in all medical specialities. Larger studies may be needed to provide convincing data.

Abstract #903

EFFICACY OF PITUITARY RADIOTHERAPY ON GROWTH HORMONE (GH) SECRETION IN PATIENTS WITH ACROMEGALY

Monica Gheorghiu, MD, PhD, Alexandra Nicolae, Mariana Purice, Corin Badiu, MD, PhD, Catalina Poiana, MD, PhD, Mihail Coculescu, MD, PhD, FRCP

Objective: Pituitary radiotherapy (RT) is reported to normalize GH secretion in about 50% of the patients with acromegaly after 10 years. The criteria for serum GH normalization have been progressively modified in the last 20 years. The aim of this study was to evaluate the cure rate of acromegaly after RT according to the 2010 - 2011 guidelines criteria.
Methods: We retrospectively assessed the efficacy of RT in patients with acromegaly evaluated during 1985 - 2010. The criteria for cure were: random GH or nadir GH during 75g-OGTT <1 ng/mL and (when available) age and sex - adjusted IGF-I. GH has been measured with RIA or IRMA assays, IGF-1 with different commercial assays. Only patients tested before or after at least 2 months withdrawal of specific medical therapy have been included.
Results: 135 patients with documented acromegaly underwent RT (88 women, 47 men, mean age 43.5 years, range 12 - 77, 97 macroadenomas, 28 microadenomas, 11 unknown tumor size), either postoperatively (n=60) or per primam (n=75): 34 had low-voltage conventional RT, 83 high-voltage RT, 30 had gamma-knife (15 patients had multiple RT). Median follow-up was 60 months (6 - 456). Cure criteria were met in 10/60 (16%) of postoperative patients and in 14/75 (18%) of primarily irradiated patients, median time to GH normalization 43 vs. 76 months, respectively, p = NS. Cumulative probability of GH normalization was 5.5% at 2 yr, 17.6% at 5 yr, 32.4% at 10 yr and 42.5% at 15 yr after RT.
Discussion: In patients treated with radiosurgery as compared to those treated with high-voltage RT the cumulative probability of GH normalization was

higher (log-rank test, $p < 0.05$), the median nadir OGTT- GH level was lower after 2 yr (2.5 ± 1.8 ng/mL vs. 4.8 ± 8.2 ng/mL, $p < 0.05$), while the rate of GH decrease was higher at 6 months (45 ± 20% vs 38 ± 59% respectively, $p < 0.05$), but not afterwards. During follow-up, cerebrovascular disease has been recorded in 7.8% of patients, new pituitary insufficiency in 31% of patients, another cerebral tumor in 1% of patients, with similar prevalence after primary or postoperative RT.
Conclusion: Pituitary radiotherapy, either as primary or postoperative therapy, resulted in normalization of serum GH (and IGF-1, when available) according to current criteria in about 30% of patients with acromegaly in 10 years after therapy, and was associated with non-negligible side-effects.

Abstract #904

A PROSPECTIVE STUDY OF CLINICAL AND RADIOLOGICAL IMPROVEMENT IN THYROTROPH PITUITARY HYPERPLASIA WITH TREATMENT OF PRIMARY HYPOTHYROIDISM

Satish Babu, MD, MRCP, CCST, Ravindra Kamble

Objective: Pituitary enlargement in patients with primary hypothyroidism has been established. Except for few case reports, to our knowledge, there are no studies prospectively analyzing the radiological regression of pituitary enlargemnet with treatment for primary hypothyroidism.Patients with hypothyroidism were evaluated with MR imaging to asses pituitary hyperplasia before and afterThyroxin treatment.
Methods: 7 Patients (6 females and 1 male), age ranging from 13 to 42 years, who presented with clinical and biochemical profound hypothyroidism (TSH >75µIU/ml) were studied prospectively with base line MR imaging before thyroxin treatment and further evaluated with repeat TFT and serial MR imaging. All patients were started on 100mcg of thyroxin at presentation after MR imaging. All came back for repeat imaging around two weeks (first follow up) and around 4 weeks(second follow up) after intiation of treatment, except for one patient, who had repeat scan at 4 weeks (first follow up) and 8 weeks(second follow up).The TFT was repeated on second follow to correlate clinically and up titrate the thyroxin dose. Routine MR imaging was done and pituitary gland measurements (height, AP diameter and width) were taken on T1W sagittal and T2W coronal sequences during baseline and follow up imaging using same parameters. The statistical analysis was done for obtaining p value.
Results: There was steady clinical improvement in all patients with near normalization of TFT in most patients at 4 weeks.The TSH normalization well correlated with regression of the pituitary enlargement on imaging at 4 weeks. The height of the pituitary gland significantly reduced in first and second follow up ($p<0.05$) and reduction in width on second follow up ($p<0.05$).
Discussion: Pituitary hyperplasia secoundary to primary hypothyroidism is well documented although poorly studied.These enlargements can be large enough to mimic as pituitary adenoma. Establishing a time bound radiological response with treatment will improve the critical decision making in these clinical situations, more so with current increased use of imaging as a diagnostic tool.
Conclusion: The pituitary enlargement secondary to thyrotrop hyperplasia in primary hypothyroidism regresses with steady normalization of TSH.There is a trend in reduction in height of pituitary gland within 2 weeks of initiation of treatment. We recommend to repeat MR Imaging within four weeks of intiation of thyroxine treatment in patients with pituitary enlargement who happen to have high TSH suggestive of primary hypothyroidism. It is also mandatory to check TFT when incidental homogenous pituitary enlargement is found on MR imaging.

Abstract #905

A CASE OF ACTH SECRETING PITUITARY CARCINOMA

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Ravindra Kamble, Vasudev Rao

Case Presentation: Pituitary tumors are mostly benign, pituitary carcinoma being very rare. We report a ACTH secreting pituitary adenoma which progressed later on to multiple liver and lung metastasis. A 42 year old male presented with headache and blurring of vision in the right eye.Evaluation of this led to pituitary macroadenoma with hemorrhage. He did not have much of clinical features of hormone excess, but the ACTH and serum Cortisol levels were high.Trans sphenoidal surgery was done, operative findings suggested tumor extending into sphenoidal sinus with evidence of pituitary apoplexy. The histology confirmed pituitary adenoma and immune-histochemistry (IHC) was indicative of ACTH positivity with Ki 67 of 10%.The tumour's aggressiveness led to institution of conventional radiotherapy for the remnant tissue. Repeat MRI scan revealed persisting residual tissue, for which cyber knife radio surgery was administered. Subsequent follow up showed normal ACTH and Cortisol levels with regression of the residual tissue on MRI. Year after,he presented with sudden onset of dyspnea and pedal

edema.On clinical examination signs of left consolidation were present. Evaluation of this with CT scan revealed, multiple lung and liver lesions.Hormone assay showed high levels of ACTH and Cortisol but, MRI of pituitary showed significant regression of residual tissue.CT guided biopsy of the liver lesion and IHC was consistent with metastatic ACTH secreting endocrine carcinoma. While awaiting chemotherapy, patient died at home.

Discussion: Pituitary carcinomas are very rare, incidence being less than 0.5% of Pituitary tumors. Approximately until 2004, there were 140 published cases of Pituitary carcinoma in English literature.Our patient had pituitary adenoma,which was invasive along with atypical features on histology in the form of high Ki67 score. On further follow up, patient progressed rapidly to develop multiple liver and lung metastases which confirmed the diagnosis of ACTH secreting Pituitary carcinoma.

Conclusion: Even though Pituitary Carcinomas are rare, our case illustrates high index of suspicion by the treating physician to enable early diagnosis. The features suggestive of neoplasm in our case were, local invasiveness and high proliferative index in the form of Ki67.

Abstract #906

THE 'PIT-VIPER' CONNECTION - A CASE REPORT OF HYPOPITUITARISM FOLLOWING VIPER ENVENOMATION

Satish Babu, MD, MRCP, CCST, N. Padmanabhan, Anil Kumar, Madhu Muddaiha

Objective: Russell's Vipers are found in the Indian subcontinent and in south east asian countries. Features of envenomation by these snakes include coagulopathy, shock, acute kidney injury, neurotoxicity and local tissue damage. This leads to morbidity and mortality. Hypopituitarism following envenomation by these vipers is a rare complication. We describe a case of hypopituitarism following viper's envenomation.

Case Presentation: A 34 year old single unemployed male was bitten by a viper in August 2009. He developed severe intravascular haemolysis and acute kidney injury. He recieved anti-snake venom, multiple transfusions and haemodialysis. He recovered from the acute effects completely. Two months later he developed generalised weakness, anoerexia, loss of weight and was getting withdrawn and less communicative. He was referred to us for suspected chronic kidney disease. Clinical examination revealed persistant Hypotension, which led to suspicion of hypocortisolaemia. Complete hormone profile confirmed deficiencies of gondal, thyroid and steroid axes due to hypopitutarism. He showed marked improvement after supplementation of anterior pituitary hormones.

Discussion: Russell's Viper envenomation is known to produce both acute and chronic hypopitutarism. The exact pathogenesis is still speculative. Vast majority of the reported cases of hypopitutarism following viper envenomation had acute kidney injury immediately after the bite. While unrecognised acute hypopitutarism is potentially fatal,chronic hypopituitarism can be debilitating.

Conclusion: Physician awareness about this complication will lead to early detection and facilitate essential hormone replacement in this setting.

Abstract #907

NOCTURNAL PROFILES OF SERUM GROWTH HORMONE AND INSULIN IN ACROMEGALIC PATIENTS WITH OR WITHOUT SLEEP APNEA

Dan Niculescu, MD, Andra Caragheorgheopol, Teodora Parvu, Mihail Coculescu, MD, PhD, FRCP

Objective: In obese subjects sleep apnea syndrome (SAS) is associated with hyperinsulinemia and absence of growth hormone (GH) bursts of secretion linked to slow wave sleep (SWS). In acromegaly SAS is highly prevalent but its effects on night plasma insulin and GH profiles is unknown. The aim of our study was to characterize SAS effects on nocturnal profiles of serum GH and insulin in patients with acromegaly.

Methods: Five patients with active acromegaly (3 before any treatment, 1 after sfenoidal surgery and 1 after radiotherapy) and 2 controls were polysomnographicaly recorded between 10 p.m. and 6 a.m. Last meal was served at 18:00 o'clock. No patient had pituitary insufficiency. At 30 minutes interval blood was drawn through a i.v. canula and a tubing system from an adjacent room without disturbing the patient. Sleep stages and respiratory events were scored using accepted criteria. Serum glucose, insulin, GH and cortisol were measured in each blood sample.

Results: Three patients with acromegaly proved to have SAS (apnea-hypopnea index [AHI] of 25, 20 and 11.6 events/h respectively). The other 2 acromegalic patients and the 2 control did not present SAS (AHI<5 events/h). In both patients and controls serum cortisol was low at the beginning of the night (4.1±1.8 μg/dL) and rose toward the morning (15±4.2 μg/dL) thus validating the method. Serum GH profiles were flat in acromegalic patients (~150% variation between minimum and maximum nocturnal values) but not in controls (>5000% variation). No patient with acromegaly had characteristic GH secretion peaks associated with SWS. There were no differences in GH profiles of acromegalic patients with or without SAS and with central or obstructive type of apnea. Serum glucose and insulin profiles were flat in both patients and controls (less than 100% variation), irrespective of SAS.

Discussion: In normal subjects the nocturnal GH bursts of secretion are initiated by SWS episodes. As SAS impairs deep sleep this translates in flat GH profiles. In acromegaly the autonomous GH secretion probably overrides the SWS-induce bursts and the GH serum levels are constantly high. Because in acromegaly the GH serum levels are not dependent on SWS the SAS does not influence nocturnal GH serum levels. Glucose and insulin levels are constant during the night in both normal and acromegalic subjects and do not interfere with GH secretion in the short term.
Conclusion: In patients with acromegaly nocturnal serum GH and insulin levels are elevated and are not dependent on sleep stages or sleep apnea.

Abstract #908

EMPTY SELLA SYNDROME MASKED BY CHRONIC STEROID EXPOSURE

Scheherezada Urban, MD, C. Jonkam, M. Sha, H. Taher, I. Orija

Objective: To report a case of adrenal insufficiency in a patient with empty sella syndrome (ESS).
Case Presentation: An 80-year-old African American female became bedridden due to severe fatigue, back and knee pain for 2 weeks. She had a longstanding history of hypothyroidism, generalized DJD and heart failure. She received intra-articular steroid injections every 1-3 months for 2 years with increasing doses over the past several months due to worsening pain. On presentation she was in severe painful distress, blood pressure was 157/80 mmHg and other vital signs were stable. Physical exam was remarkable for significant bilateral lower extremity pitting edema. Further evaluation showed a random cortisol level of 1.0 mcg/dL, TSH was 0.867 mUI/mL and free T4 was 0.52 mcg/dL. ACTH stimulation test revealed a cortisol of 2.0, 4.5 and 6.0 mcg/dL at 0, 30 and 60 minutes respectively, confirming adrenal insufficiency. ACTH was 15.1 pg/mL (10-60) and prolactin 38.2 ng/mL (normal <20); IGF1, FSH, LH, and GH were low suggestive of panhypopituitarism. A pituitary MRI revealed an empty sella with no tumor. During hospitalization she developed severe hypotension which improved with IV hydrocortisone. There was also a dramatic improvement in her symptoms. She was subsequently discharged on prednisone. At 6 weeks follow up she remains symptom free.
Discussion: Empty sella (ES) is an anatomical entity in which the pituitary fossa is completely or partially filled with CSF, while the pituitary gland is compressed against the fossa. ES can be either primary or secondary. Primary ES (PES) is caused by a weakness of the sellar diaphragm and/or an increase in intracranial pressure with herniation of arachnoid into the pituitary fossa. Secondary ES may be caused by pituitary adenomas undergoing spontaneous necrosis, infective, vascular, autoimmune, and traumatic causes or by surgery and radiotherapy. PES may be associated with pituitary insufficiency. PES related endocrine dysfunction is secondary to the compression of the pituitary gland and stretching of the pituitary stalk leading to mild hyperprolactinemia and hypopituitarism. Panhypopituitarism has been reported in 2% of cases with PES. Isolated or multiple pituitary hormone deficiencies are described with GH deficiency as the most frequent (30-60% prevalence), LH/FSH (6%), ACTH, TSH and ADH (all about 1%). Serum prolactin levels are moderately increased (10-12%). Our patient had biochemical evidence of adrenal insufficiency and hypothyroidism secondary to ES.
Conclusion: ACTH deficiency is an uncommon feature of ESS and highlights the importance of a thorough clinical evaluation. A high level of suspicion is key for a timely diagnosis and prompt treatment.

Abstract #909

AN INCIDENTAL PITUITARY ADENOMA WITH INTACT LH HYPERSECRETION

Shamsuddin Shaik, MD, Haseeb Kazi, MD, Bankim Bhatt, MD, Mohammad Arastu

Objective: We present a relatively uncommon case of gonadotroph cell adenoma with elevated FSH and intact LH resulting in supranormal free and total testosterone.
Case Presentation: A 52 year old Caucasian male with a history of hypertension, diabetes mellitus, normal pubertal and fertility history presented with head trauma due to assault by his son. He did not have any focal neurological deficit on initial presentation. CT of the head incidentally showed a large rounded mass measuring 4.1 x 4.3 x 6.3 cm. Brain MRI showed a large sellar and suprasellar mass extending to the right anterior cranial fossa with surrounding vasogenic edema. Imaging was most suspicious for a pituitary macroadenoma. His initial labs were FSH 58.3 mIU/mL (0.9-15), LH 9.1 mIU/mL (1.5-9.3), prolactin 25.3 ng/mL (1.8-14.4), free testosterone 29 ng/dL (7.2-24) and total testosterone 837 ng/dL (193-740). LH was confirmed to be intact by immunoassay. TSH and ACTH were normal, but GH was 0.1 ng/mL (0-6.0) and IGF-1 was 59 ng/mL (87-238). The patient underwent image guided transnasal, transsphenoidal resection of the tumor, right frontotemporal craniotomy and orbital osteotomy for resection of the supratentorial component of the tumor. Pathology returned as pituitary adenoma with expression of only FSH and LH. His postoperative course was complicated by intracranial hemorrhage

and Enterobacter encephalitis, eventually requiring tracheostomy and PEG tube placement. Post resection labs showed LH <0.1 mIU/mL, FSH 2.2 mIU/mL, testosterone <3 ng/dL, prolactin 7.4 ng/mL, TSH 0.009 uIU/ML, free T3 0.2 pg/mL, and free T4 0.07 ng/dL. His pituitary insufficiency was treated with methylprednisolone and levothyroxine. Secondary hypogonadism was treated with testosterone gel. The most recent free T4 was 0.7 ng/dL, free T3 1.7 pg/mL and testosterone 139 ng/dL. His neurological status gradually improved and was discharged to a long term rehabilitation center. ***Discussion***: Many patients who have gonadotroph cell adenomas are often middle aged males with normal pubertal development and fertility history. They are brought to medical attention because of visual impairment due to the size of the adenoma. The most common hormonal characteristic of gonadotroph cell adenomas is hypersecretion of FSH and less often by hypersecretion of intact LH. Our patient had an adenoma hypersecreting intact LH resulting in supranormal testosterone levels. ***Conclusion***: This unique case describes an incidental pituitary adenoma with intact LH hypersecretion, whereas the vast majority of pituitary adenomas have impaired LH secretion.

Abstract #910

APOPLEXY IN A PLURIPOTENT PITUITARY TUMOR WITH ACROMEGALY, HYPERPROLACTINEMIA, AND HYPOGONADISM

Shahid Aziz, MD, Harikrashna Bhatt, MD, Ali Rizvi, MD

Objective: To describe a functional pituitary somatotropinoma and macroprolactinoma presenting with acute apoplectic change.
Methods: A 31-year-old male presented to the ER with acute onset of severe headache and left monocular vision loss. He gave a 2-year history of headaches, gradual fatigue, loss of libido, erectile dysfunction, and his "wedding ring no longer fit." He denied breast discharge or cold and heat intolerance. Visual acuity was 20/30 in the right eye and no light perception in left eye. There was no thyromegaly or expressible galactorrhea, Large, fleshy hands and mild prognathism was noted. Labs showed prolactin 1351 ng/mL (2.1-17.7), IGF-1 608 ng/mL (115-307), GH 6.46 ng/mL (0.01-1.0), total testosterone 54 ng/dl (241-827), TSH 2.15 ulU/mL (0.35-4.5), free T4 0.93 ng/dL (0.89-1.76), LH 6.5 mlU/mL (1.5-9.3), and FSH 4.86 ml U/mL (1.4-18.1). MRI revealed a 3.8 x 5.3 x 4.3 cm sellar expansile mass with bilateral cavernous and sphenoid sinus infiltration, carotid encasement, displacement of anterior-middle cerebral arteries, and severe mass effect on optic chiasm and hypothalamus.
Case Presentation: A 0.8 x 0.6 x 0.3 cm mass was removed on transphenoidal tumor resection. Microscopy showed hemorrhagic fragments with degenerative change and large monomorphic cells with eosinophilic cytoplasm. Vision improved and there was no postsurgical diabetes insipidus. Glucocorticoids were tapered and bromocriptine was initiated. At follow-up 1 month later some residual vision loss persisted, there was marked improvement in headaches, GH was 2.2 ng/mL, IGF-1 346 ng/mL, prolactin 577 ng/mL, and free T4 0.66 ng/dL. Steroids were continued, bromocriptine was titrated, and levothyroxine started. Radiation therapy and somatostatin receptor antagonist treatment were considered. ***Discussion***: Pluripotent pituitary lesions are uncommon but can manifest as clinical and biochemical acromegaly and hyperproloactinemia. This case came to light as a result of apoplexy and acute neurologic deficits requiring surgical decompression. GH and prolactin excess persisted and required long-term pharmacologic therapy, with anterior pituitary hormone replacement. The degree of prolactin elevation was unlikely from "stalk effect." Such tumors may occur more commonly in MEN 1; however, no other features of endocrine neoplasia were evident in the patient and his family history was unremarkable. ***Conclusion***: Apoplexy in a pituitary neoplasm can manifest with a combination of rapid neurologic deficits in the setting of multiple endocrinopathies (both hyper- and hypofunction). These presentations should be borne in mind when evaluating pituitary lesions.

Abstract #911

AN UNUSUAL CASE OF RECURRENT UTI IN A MALE PATIENT WITH ACROMEGALY

Dongning Chen, MD, Maurita Carrejo, MS, Robert Tan

Case Presentation: Acromegaly is associated with excessive growth, leading to disfigurement. Sometimes this is purely cosmetic; at other times it leads to unexplained symptoms, as we report in this case. A 61yo acromegalic male presented with recurrent dysuria, postvoidal dribbling, frequency and urgency. He was also uroseptic on one occasion, leading to further investigation, which elucidated the etiology of these episodes. He was a Vietnam veteran and was first suspected to be acromegalic based on his frequent need to increase boot size. He had no history of genitopelvic injury or STD. Possible exposure to Agent Orange is unknown. Diagnosis included confirmed serial elevations of IGF-1. Neuroimaging showed a pituitary macroadenoma, treated with transphenoidal hypophysectomy, resulting in lifelong hypopanpituitarism. PE: BP: 109-133/53-76 mmHg; acromegaly features:

acral enlargement, gigantism, prognathism, jaw malocclusion; genitourinary: cord-like hardening of urethra, bilateral testicle atrophy, hardly palpable prostate. Labs:Multiple UAs: WBC in clumps; large leukocyte esterase; glucose: negative; blood: moderate; pH: 7.5-8.5; bacteria: many. Urine ctx: MRSA; E.coli; enterococcus; citrobacter; p. mirabilis. Blood ctx: citrobacter. Endocrinology: IGF-1(serial): 308, 381, 47, 92 ng/ml (81-225); GHRH: 6 pg/ml (5-18); ACTH: 5pg/ml (6-48); GH: 0.4-1.0 ng/ml (0-6); TSH: 0.1 to 0.3 uiu/ml (0.5-4.5); cortisol (am): 0.5-5mcg/dl(5.7-16.6); testosterone: total: <1.75ng/dl (1.75-7.81); free testosterone: 9.9% (9-46); aldosterone: 11.5ng/dl (1-16 supine). Imaging: CT of abd/pelvis: focal scarring of L kidney, stones; fluid-filled cysts, R/L kidneys; diverticulum on lateral surface of urinary bladder; IV urogram: submucosal irregularity of bladder. Head MRI: clip for aneurysm of left ICA at origin of PCA. Cystoscopy: hardening and thickening of epithelium of urethra and bladder, c/ numerous fibrotic structures; several diverticula on one side of bladder; urethra meatal stenosis. Treatment: IV fluids, abx, urethral dilation resulting in symptom improvement. He continues to have milder and less frequent relapses.

Discussion: Despite hypophysectomy, IGF-1 was persistently high in the presence of low GHRH-GH. This may play a pivotal role in the formation of diverticula, as a result of overstimulation by IGF-1. We suggest that over-growth of epithelial and submucosal muscular tissue narrowed the lower urinary tract, leading to partial obstruction of the outlet, and increased intravesical pressure led to diverticula in the lower urinary tract, causing the multiple UTIs.

Conclusion: The unusual growth seen in acromegaly can have significant and difficult-to-diagnose medical consequences.

Abstract #912

CASE OF CONGENITAL HYPOPITUITARISM WITH ECTOPIC POSTERIOR PITUITARY AND PULMONIC STENOSIS; HORMONAL AND RADIOLOGIC FOLLOW UP INTO ADULTHOOD

Cherie Lisa Vaz, MD, Sandeep Dhindsa

Objective: We describe the course of a now 24 y/o M born with congenital hypopituitarism, severe micropenis, cryptorchidism, hypoplastic testes, hypospadiasis and pulmonic stenosis

Case Presentation: The baby was born at 42 wks, birth wt 6 lbs, pregnancy complicated by maternal bleeding during first 15 weeks. He presented with neonatal hypoglycemia and cryptorchidism. MRI showed small anterior pituitary & ectopic posterior pituitary near tuberous cinereum. Pulmonic stenosis detected after auscultation of murmur was mild grade on echo. Free T4 1ng/dl (1-2.5), TSH 6.7 uU/ml (0.2-6). Glucagon stimulation for GH & cortisol was blunted at 2.9ng/ml & <1 respectively. Prolactin 3.3ng/ml(3-14.7). He received levothyroxine & hydrocortisone from infancy. Total testosterone & gondotropin concentrations were undetectable (<10ng/dl & <3mIU/l respectively). He received intermittent testosterone to augment penis size. Inspite of testosterone level between 315 to 921 ng/dl(241-827), he had subnormal penis size. Height increased at a constant rate until age 4 when it fell 2.2 SD units below mean. GH deficiency was confirmed with flat response to arginine & dopamine with peak GH level of 1.8ng/ml. He was started on GH at age 4 & attained nl height. Panhypopituitarism persists into adulthood with gradual diminution of pituitary size. MRI age 24 revealed small sella which only contained infundibulum of the pituitary. Gland itself is not visualized. Adult hormone levels are undetecetable LH & FSH, IGF 92ng/ml(83-456), ACTH 6pg/ml (6-50), am cortisol <0.5 Prolactin 4.7ng/ml(2-18), TSH <0.01mIU/l(0.4-4.5) Free T4 1.6ng/dl(0.8-1.8) Testosteron 280ng/dl(25-1100), Free testosterone 30.8pg/ml(35-155). He remains on thyroxine, hydrocortisone, testosterone & adult GH.

Discussion: Previously reported associations of multiple pituitary hormone deficiency were with septo-optic dysplasia, ophthalmologic, cranial nerve & cerebral midline defects. This is the first case demonstrating the association of congenital hypopituitarism & cardiac anomaly specifically pulmonic stenosis. Congenitally low gonadotrophins tend to improve with age. Contrary to this, FSH & LH levels remained low such that the patient required testosterone during childhood to treat micropenis but achieved subnormal response. This case illustrates that pituitary size & function although subnormal at birth, gradually deteriorates.

Conclusion: One should consider screening patients with congenital hypopituitarism for cardiac anomalies at birth. Congenital hypopituitarism presenting in adults is reported. The gradual diminution of our patient's pituitary size indicates that patients need close follow up when no or partial hormonal abnormalities are found on initial testing.

Abstract #913

THYROID CANCER IN A PATIENT WITH ACROMEGALY

Amanjot Lehil, MBBS, Rajib Bhattacharya, MD

Objective: Acromegaly is a chronic disease caused by increased GH secretion, most commonly from a growth

hormone secreting adenoma of anterior pituitary. Several studies have shown association between acromegaly and cancers of colon, brain, breast, prostate, and cancers of hematological system. Recent studies have demonstrated that overall mortality rates in patients with acromegaly were comparable to population-based controls in patients who achieved biochemical remission, and were elevated in patients with persistent disease. A few recent retrospective studies have shown an association between acromegaly and thyroid cancer. In this case report we describe a 32 y/o M with acromegaly diagnosed with thyroid cancer.

Case Presentation: 32 y/o M was diagnosed with Acromegaly in 2005, shortly afterwards he underwent transphenoidal resection of pituitary macroadenoma. He was lost to follow up following surgery, he returned to clinic in 2010, when MRI showed recurrence of pituitary macroadenoma and his IGF-1 level was 998 ng/mL. He was started on octreotide and underwent resection of the macroadenoma in Nov 2010. Pathology showed patchy staining with growth hormone, and cytological features were compromised due to compression by presence of dense fibrous tissue. Following the repeat surgery his IGF-1 level, though still elevated, went down to 460 ng/mL. Octreotide LAR dose was increased to 40mg every month, and he underwent radiation to pituitary area for one month. His most recent IGF-1 level was 309 ng/mL in June 2011, at this visit thyroid nodules were felt on physical examination. Thyroid ultrasound showed several bilateral complex thyroid nodules, the largest nodule measured 4.7 x 3.2 x 3.5 cm. FNAC showed follicular cells with atypical nuclear features, suspicious for papillary carcinoma. He underwent total thyroidectomy in June 2011, biopsy showed papillary carcinoma.

Discussion: In this case, patient had persistently elevated IGF-1 levels over the past several years. Elevated IGF-1 is believed to be responsible for increased risk of cancer in acromegalic patients, by exerting its proliferative and anti-apoptotic actions. A meta-analysis of 3 large epidemiological studies showed a relative risk of 3.64 [95% CI: 1.63 - 8.11] for developing thyroid cancer in acromegalic patients compared with the general population. More studies are needed to confirm this association.

Conclusion: Current level of evidence suggests that acromegaly may be a risk factor for thyroid cancer, thus the need for careful and thorough work up of acromegalic patients who develop thyroid nodule and potentially using thyroid ultrasonography for screening.

Abstract #914

DIABETES INSIPIDUS ASSOCIATED WITH BURN INJURY

Arinola Ipadeola, MBBS, Oluwakayode Iyun, Olayinka Olawoye, Jokotade Adeleye, MBBS

Objective: To report the case of a patient admitted with burn injury that developed diabetes insipidus.

Case Presentation: 35year old electrician admitted by the plastic surgical team on account of 31% superficial deep dermal flame burn and inhalational injury following petrol explosion in a generator house. On admission, he had tachycardia with extensive burn injury involving head & neck, anterior and posterior trunk, upper and lower limbs. Electrolytes (E&U,Cr) and packed cell volume were normal. Chest -X-ray was essentially normal. Urinalysis showed haematuria and specific gravity (SG) was 1.020. He was rehydrated and managed on antibiotics and high calorie diet. He developed fever and became polyuric on the 2nd and 3rd day of admission respectively (approx. 2.7mls/kg). He also had stress induced hyperglycaemia which resolved spontaneously. SG reduced to 1.015 then 1.005 and Input/output ranged between 5150/3355 to 8050/16387mls. A diagnosis of Diabetes insipidus (DI) was made. He was then commenced on desmopressin (DDAVP) but only vasopressin was available and administered at 30iu/day, later 42iu/day. Blood Pressure was elevated and controlled on oral nifedipine 20mg b.d. Polyuria resolved with DDAVP(on 6mcg per day & gradually tailed off). He had persistent hyponatraemia and pedal odema likely due to high dose of DDAVP required for resolution of symptoms. DI resolved on the 32nd day of admission and input output remained normal without medication.

Discussion: A few cases of DI following traumatic injury other than brain injury have been reported. Burn injury has been reported in literature to be associated with DI within a few days after the injury as in this case. The emergence of electrolyte imbalance and increased urinary loss of fluid may lead to an increase in mortality in such cases. DI following burn injury is thought to be due pituitary hypoxic injury leading to local brain tissue oedema, and sometimes associated kidney tissue damage. Prompt treatment has been associated with good recovery.

Conclusion: Burn Injury is an uncommon cause of DI. Fluid and electrolyte balance is crucial in all cases for early diagnosis and management, thus improving survival.

Abstract #915

PARTIAL HYPOPITUITARISM: AN UNCOMMON COMPLICATION OF STURGE-WEBER SYNDROME

Sunil Kota, MD, Siva Kota, Svs Krishna, Lalit Meher, Kirtikumar Modi

Objective: To report the presentation of partial hypopituitarism in a patient with sturge weber syndrome (SWS).
Methods: Clinical, laboratory, and imaging data are reported on a known case of SWS who was referred for evaluation of short stature.
Case Presentation: A 19 year old male diagnosed to have SWS was evaluated of short stature. There was port wine stain (PWS) involving left half of face extending variably to right half, with hyperplastic gum, lips and nonblanchable erythema extending to left upper limb. With < 3rd percentile height (155 cm, height age- 13 years and height SDS -2.8) for age and Tanner's puberty stage I (testes bilateral 3ml), dry skin and delayed relaxation of ankle reflex, his laboratory parameters revealed central hypothyroidism (T4- 3.2 µg/ dl, TSH- 1.2 µIU/ml) and hypogonadotropic hypogonadism (8.00 AM testosterone- 1.6 ng/ ml, FSH-1.1 IU/l & LH -0.3IU/l). Computed tomography of the brain revealed serpiginous gyriform cortical calcification with atrophic right atmosphere. Contrast study revealed choroid plexus hypertrophy and leptomeningeal enhancement. After confirming cortisol sufficiency (8.00 AM serum cortisol- 12 µg/ dl, ACTH- 30 pg/ml, stimulated peak serum cortisol level after 5U insulin was 24 µg/dl at blood glucose nadir of 35 mg/dl), patient was prescribed oral thyroxine replacement (50µg), testosterone enanthate 250 mg intramuscularly every 3 weekly. After achievement of euthyroidism, growth hormone stimulation test with insulin (0.1 U/kg, i.e. 5U) and clonidine (0.15 mg/m2) revealed growth hormone deficiency (4 ng/ml at blood glucose nadir of 38 mg/dl with insulin and 3.4 ng/ml with clonidine respectively). The patient was simultaneously prescribed recombinant growth hormone 0.3 mg/ kg/ week in 7 divided subcutaneously daily at night.
Discussion: In view of the combined picture of central hypothyroidism, hypogonadotropic hypogonadism, growth hormone deficiency in presence of an organic brain lesion, the diagnosis of partial hypopituitarism was established. The possible reasons attributed for hypopituitarism are 1) structural impairment of hypothalamic pituitary axis (HPA), 2) Functional deficiency due to injury to hypothalamus. Hyperprolactinemia due to interruption of the inhibitory dopaminergic fibres from the arcuate nucleus in hypothalamus can additionally explain hypogonadotropic hypogonadism. Other factors implicated in causation of central hypothyroidism include usage of anticonvulsant therapy, decrease in expression of type 2 deiodinase II messenger RNA in the cerebral cortex.
Conclusion: HPA is at risk for impairment in SWS. It calls for need to determine the prevalence of hypopituitarism and other associated endocrine illnesses in SWS.

Abstract #916

ACUTE HYPOKALEMIA AS THE FIRST MANIFESTATION OF CUSHING'S DISEASE DURING PREGNANCY

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Objective: To report the case of a woman with Cushing's disease who presented with hypokalemia during pregnancy, and the importance of appropriate diagnostic testing.
Case Presentation: 28 y/o woman, with recurrent hypokalemia during her second pregnancy. After giving birth she presented general weakness, progressive central obesity, acne, hirsutism, irregular menses, depression and hypertension. Use of current or previous glucocorticoid therapy was denied. Physical exam revealed moon facies, facial acne, supraclavicular fat pads, buffalo hump, increased abdominal girth and large purple striae. Laboratories with Cortisol after 1mg Dexamethasone suppression test (DST) =2.51mcg/dL, ACTH= 204 pg/mL, Prolactin= 9.8 ng/mL, Sodium=146 , Potassium=2.6, fasting glucose=92, 24 hour urinary free cortisol (UFC)> 1342 ug, 24 hour UFC after 2 day high dose dexamethasone (HDDST) >1425 ug, 2 hour oral glucose tolerance test=124 mg/dL and Hb A1c=5.1%.Brain MRI with a 6mm pituitary microadenoma. Inferior petrosal sinus sampling (IPSS) was done with a right central:peripheral ACTH ratio of 14.6 at baseline, 57.7 at 2min, 32.1 at 5min and 16.8 at 10min of CRH administration, with a central:peripheral Prolactin ratio of 2.85 (right) and 1.43 (left). Biochemical evidence of pituitary source of ACTH suggests Cushing's disease. Patient was referred to Neurosurgery clinics for transphenoidal surgery.
Discussion: Establishing the etiology of Cushing's syndrome is difficult. Hypercortisolemia must be confirmed by the overnight 1mg DST, 24 hour UFC or 11pm salivary cortisol levels. If ACTH dependant, the most common site of secretion is a pituitary tumor, for which pituitary MRI should be ordered, however a small microadenoma, as in this patient, could be nonspecific. Ectopic vs central secretion must be distinguished with bilateral IPSS, known to be more accurate than HDDST, which did not suppressed in this patient. A baseline central-peripheral ACTH ratio >/= 2 or >/= 3 after CRH

administration is diagnostic of pituitary ACTH secretion. False negative results can be seen during this procedure. A recent study published by Cleveland Clinic proved that a central:peripheral Prolactin ratio > 1.3 suggests successful petrosal sinus catheterization. Hypokalemia and marked elevation of ACTH are common in ectopic ACTH syndrome, which was highly suspected in this patient . Using the appropriate diagnostic testing, as the IPSS, confirmed pituitary source of ACTH, avoiding further imaging studies.

Conclusion: Cushing's disease may present in different ways. A pituitary adenoma may present as ectopic ACTH syndrome. It is important to follow diagnostic testing adequately and avoid unnecessary imaging and laboratory studies.

Abstract #917

CASE OF EMPTY SELLA TURCICA PRESENTING AS PARTIAL HYPOPITUITARISM

Richard Pinsker, MD, Pinkesh Prajapati, Narinder Kukar, Hineshkumar Upadhyay, Kelly Cervellione

Objective: To report an unusual case of empty sella turcica presenting as partial hypopituitarism (with selectively low ACTH and GH).

Case Presentation: A 24-year-old female with no significant past medical history was brought to the ER after being found unresponsive in bed. At that time, patient was found sweaty and had cold and clammy extremities. EMS noted that the patient was hypoglycemic and gave IV dextrose and glucagon. Upon arrival at hospital, patient was relatively asymptomatic except for hypotension which responded poorly to IV fluid challenge. She reported generalized weakness for the last several weeks and significant weight loss over the past 3 years. She also reported heavy but regular menstrual cycles since puberty. Cosyntropin test showed very low baseline cortisol (0.2, N=4-22ug/dL) that didn't respond to cosyntropin stimulation test (post cosyntropin cortisol level=0.4ug/dL). ACTH was also low (<5, N=5-27 pg/mL). MRI of brain revealed empty sella turcica with small asymmetric pituitary gland. Further labs revealed hypothyroidism (TSH=6.50, N=0.47-4.7uIU/ml, freeT4=0.66, N=0.8-2.2ng/dl) with GH deficiency (GH=0.2 ng/mL with IGF-I=21, N=83-456ng/mL) but normal FSH, LH and prolactin levels. Adrenal and thyroid autoantibodies were negative. Patient was discharged home on hydrocortisone 20 mg orally at 7 AM and 10 mg at 5 PM along with fludrocortisone 0.05 mg and levothyroxine 50 mcg orally once a day.

Discussion: Primary empty sella is caused by a hole in the membrane surrounding the pituitary, which allows the fluid in and compresses the pituitary. Secondary empty sella can be caused by damage to the pituitary gland from a tumor, radiation therapy or surgery. In some cases the cause is unknown, as in our patient. Empty sella turcica is present in 5.5-23% of autopsies. In most patients, it is found incidentally. Endocrinological dysfunction is found in 20-50% of patients with empty sella, of which partial hypopituitarism has been described in 5%, panhypopituitarism in 25% and hyperprolactinemia in 10%. Treatment consists of replacement of the hormones that are no longer synthesized by organs under the control of the pituitary gland. These may include corticosteroids (cortisol), GH, sex hormones (testosterone for men and estrogen for women), thyroid hormone. With adequate treatment, prognosis is in patient with this disorder.

Conclusion: Endocrinological dysfunction is very common in patients with empty sella turcica. In the majority of patients with this disorder, the secretion of gonadotropins and GH is more likely to be affected than ACTH and TSH. In our case, the patient presented with selectively low ACTH and low GH with normal gonadotropins (selective partial hypopituitarism).

Abstract #918

CABERGOLINE THERAPY FOR MACROPROLACTINOMA DURING PREGNANCY

Aisha Sheikh, MBBS, FCPS

Objective: We are reporting this case in order to contribute to the relatively meager data available to advocate the safety of cabergoline(CAB) therapy in pregnant patients with macroprolactinoma.

Case Presentation: A 31 year old lady, mother of three children, presented with an eight year history of macroprolactinoma. She was non-compliant with bromocriptine(BRC) due to tolerance issues. She had expressible galactorrhea and her Prolactin (PRL) was 1300ng/dl (1.9 - 25 ng/ml) at presentation. Magnetic Resonance Imaging (MRI) showed a Pituitary Macroadenoma measuring 2.2cmx2cmx1.3 cm with minimal suprasellar extension, involving the right cavernous sinus with encasement of internal carotid artery and extending into the optic canal abutting the optic chiasm superiorly. Due to intolerance to BRC; CAB was started at a low dose of 0.25 mg once a week and increased to 0.5mg twice weekly. PRL dropped to 40ng/dl after eight months. Her menstrual cycles returned to normal. A repeat MRI demonstrated reduction in tumor size. Twenty months after her initial presentation, the patient conceived. Because of the macroprolactinoma, she was advised to continue CAB at

0.5mg once weekly. Patient was referred for antenatal care. Fetal Nuchal thickness ultrasound performed at 13 weeks to screen for major chromosomal abnormalities was normal. MRI without contrast during the 2nd trimester demonstrated further reduction in the tumor size. It was decided to continue CAB throughout pregnancy to ensure further reduction in tumor size until delivery and to hold CAB during postpartum period to allow for an adequate interval of breastfeeding. Perimetry remained normal in each trimester. At 37+ weeks; she delivered a healthy baby boy. To allow breast feeding; her CAB was stopped.

Discussion: Once ovulation and fertility is reinstated in women with macroprolactinoma, there are two main issues ; firstly, the effects of dopamine agonists on early fetal development and the outcome of pregnancy, and secondly, the effect of hormonal milieu on the size of prolactinoma. CAB studies demonstrated a frequency of 2.2% congenital malformations which is still lower than the incidence (3.0%) found in the general population. Pregnancy itself and stopping dopamine agonistic therapy during pregnancy can cause further growth in the size of macroprolactinoma. By continuing therapy, we could diminish the size of tumor further such that after delivery, we could hold CAB while she breastfed.

Conclusion: CAB can be used safely and effectively if required to treat macroprolactinoma during pregnancy. It is important to discuss with the parents about the limited data available on the use of CAB during pregnancy.

Abstract #919

HYPONATREMIA AS THE FIRST PRESENTATION OFA PITUITARY ADENOMA

Khalil Alsoutary, MD

Objective: To report an unusual presentation of a patient with pituitary adenoma. A 61 yr old male, patient with no significant past medical history,presented to the Emergency room at Prince Hamza hospital,Amman with recurrent vomiting for the last 5 days, associated with generalised headache, dizziness on walking and standing, not associated with fever, abdominal pain or visual disturbance. This patient also has got sexual dysfunction,and secondary hypogonadism.

Methods: Physical examination: Elderly male, looks pale, with continuous vomiting,BP standing 100/70 mmHg. Temp:36.5C; Pulse:88/min; Head and neck: no palpable Lymph nodes, no thyroid swelling; Chest exam: clear; Heart exam : normal S1,S2; No murmers; Abdomen: soft, non tender,no palpble liver or spleen; LL: No edema, Absent body hair, small testis around 15 ml, soft .

Case Presentation: Lab data-Chronological order: 9/14/2011 (Na 105mmol/l,K 4.2 mmol/l,Cl 76 mmol/l, creat 64umol/l,CPK 1217u/l) 9/15/2011(Na105 mmol/l,K3.8 mmol/l,Cl72,creat65umol/l, CPK1071u/l,prolactin295ng/ml-normal up to 20)TSH1.11uIU/l-Normal0.34-4.2, free T4 0.66ng/ml, AM.cortisol 5.4nmol/l(Norm76-520), Total testosterone1.2nmol/l-Norm9-27. MRI Pituitary showed large pituitary tumor 3x2.5 cm compressing the optic chiasm.

Discussion: In patients with severe hyponatremia,the physician should determine pituitary hormones,cortisol, TSH,prolactin,LH,FSH and if abnormal MRI is needed to exclude the rare possibility of pituitary tumor causing panhypopituitarism or partial hypopituitaism .,however, other more common causes to be kept in mind like diuretic use,heart failure,and syndrome of Inappropriate diuretic hormone(SIADH).

Conclusion: Pituitary adenoma is a rare cause of hyponatremia,but it is important to keep in mind so proper management and treatment of other hormonal deficiencies can be done.

Abstract #920

DELAYED EMERGENCE OF GROWTH HORMONE (GH) SECRETION FROM A LARGE, CYSTIC MACROPROLACTINOMA: A RARE TRANSFORMATION IN PITUITARY DISEASE

Ranee Angeli Lleva, MD, Susan Brian, MD, Silvio Inzucchi, MD

Case Presentation: A 42 year old man presented with bitemporal hemianopia and decreased libido, but no headaches, other neurologic deficits, or galactorrhea. There was no family history of pituitary disease. Notable exam findings included a mildly hypogonadal appearance, low testicular volume, bilateral gynecomastia without galactorrhea, bitemporal hemianopia confirmed on formal perimetry testing, and no acromegalic features. MRI demonstrated a 3.5 cm bilobed, cystic pituitary mass; solid tumor appeared to fill the sella and invaded the right cavernous sinus. The optic chiasm was stretched by the mass. Prolactin (PRL) was 27,000 (normal: 2-18) ng/mL; total testosterone (T) 0.3 (2.0-8.0) ng/mL; free T 0.15 (1.0-5.2) ng/mL; TSH 3.77 (0.3-4.2) uIU/mL; free thyroxine 1.3 (1-2.2) ng/dL; cortisol 12 mcg/dL; LH 0.3 mIU/mL; FSH 1.2 mIU/mL; IGF-1 137 (121-237) ng/mL; GH 0.48 (<5) ng/mL. Bromocriptine 5 mg BID was initiated. After 1 week, the PRL had decreased to 257 ng/mL; after 2 weeks it was within the normal range. At 1 month, repeat visual field testing showed complete resolution of deficits. Repeat MRI at 3 months revealed marked decrease in size of the mass, specifically in the cystic component, with decompression of the chiasm.

Subsequent MRIs have shown minimal further shrinkage. After 1 year, bromocriptine was decreased to 5 mg QD, with PRL remaining normal. However, the IGF-1, originally being tracked to detect GH deficiency, increased to 305 ng/mL and continued to climb to 535 ng/mL over the next 2 years despite increasing bromocriptine back to 10 mg QD. An oral glucose GH suppression test was abnormal (nadir GH=11.3 ng/mL). Follow-up MRI demonstrated no evidence of a second pituitary neoplasm. The patient was switched to cabergoline 1mg Q2 days, with minimal lowering of IGF-1. Other than some new skin tags, he has developed no physical signs of acromegaly. Octreotide therapy is now being considered.

Discussion: Pituitary adenomas are occasionally plurihormonal in their secretory patterns, with prolactin-GH being a common pairing. However, the apparent transformation from an isolated prolactinoma to one which additionally secretes GH is extraordinarily rare. We found a single case report involving a GNAS mutation (gsp oncogene) in a GH-secreting tumor that appeared to evolve from a well-documented prolactinoma.

Conclusion: Our patient would appear to represent a second similar case - although the underlying pathogenesis and the precise temporal sequence of any contributing genomic events remains conjectural.

Abstract #921

COMPLEX MANAGEMENT OF DDAVP RESISTANT DIABETES INSIPIDUS AFTER REMOVAL OF A THIRD VENTRICULAR TUMOR: IMPORTANCE OF CORRECTING ANTERIOR HYPOPITUITARISM

Uzma Shafqat, MBBS, Maya Raghuwanshi, MD, David Bleich, MD, James Liu, Norman Ertel, MD, MACP

Objective: We present a challenging case of postoperative diabetes insipidus (DI) that was resistant to conventional DDAVP and then DDAVP therapy combined with chlorpropamide, which subsequently responded to treatment once unsuspected anterior pituitary insufficiency was corrected.

Methods: This 20 year old male presented with worsening vision, unsteady gait, and headaches for 6 months. Ophthalmological evaluation revealed 20/30 in the right eye, 20/100 in the left eye, bitemporal hemianopsia, and chronic papilledema. MRI of the brain showed a large 3.8 cm tumor within the third ventricle compressing the optic chiasm resulting in obstructive hydrocephalus. A partially empty sella was also noted. Laboratory work-up revealed normal pituitary function preoperatively. The patient underwent a craniotomy for near-total resection of a pathologically confirmed central neurocytoma.

Case Presentation: Postoperatively, the patient developed DI that was controlled on a vasopressin drip. Conversion to IV and oral DDAVP was unsuccessful resulting in DDAVP resistant DI. Therefore, the vasopressin drip was restarted with initiation of chlorpropamide 250 mg twice a day for 48 hours. Conversion to IV DDAVP (12 mcg/24 hrs) resulted in a favorable response, however, the patient did not respond to oral DDAVP. Recurrent DI ensued along with tachycardia, hypotension and fever. Bacterial cultures were negative and laboratory studies suggested anterior pituitary insufficiency. The vasopressin drip and chlorpropamide were started again along with hydrocortisone and thyroxine. After 72 hours, the vasopressin drip was successfully converted to IV and later PO DDAVP. Chlorpropamide was stopped and the DI was controlled on 0.4 mg PO DDAVP three times a day.

Discussion: DI is a common after surgery on parasellar tumors near the hypothalamus and can be readily controlled with DDAVP. DDAVP resistant DI is exceedingly rare and can be successfully treated with a short course of chlorpropamide to sensitize a favorable response to DDAVP. In this case, panhypopituitarism developed postoperatively due to the tumor involvement near the hypothalamus, and may have contributed to DDAVP resistant DI despite initial chlorpropamide therapy. Hormone replacement of anterior hypopituitarism resulted in a favorable response to chlorpropamide therapy and eventual response to DDAVP.

Conclusion: This highlights that both anterior and posterior pituitary hormones work synergistically to preserve normal serum osmolality.

Abstract #922

IPILIMUMAB THERAPY RELATED ENDOCRINOPATHIES

Archana Jarathi, MD, Monica Agarwal, MD, Fred Faas

Objective: To describe 3 patients with advanced melanoma who developed immune mediated endocrinopathies with Ipilimumab. It is a human monoclonal antibody that blocks cytotoxic T-lymphocyte antigen 4 (CTLA4), causing disruption of immune tolerance to antigens located on tumor cells. It is increasingly been used for treatment of metastatic melanoma.

Case Presentation: A 70 year old man presented with extreme weakness and confusion. The symptoms started 12 weeks after completion of Ipilimumab therapy. The TSH was 72.7 uIU/ml (0.34-5.6) and FT4 was 0.25 ng/dL (0.58-1.64) diagnostic of primary hypothyroidism. He was started on prednisone and levothyroxine. There was marked improvement in the symptoms. He died in

2 weeks from complications of melanoma. A 71 year old man presented with dizziness. He was on Ipilimumab therapy. The serum sodium was 121 meq/L (135-145), cortisol 0.8 ug/dl, ACTH 2 pg/ml (7-69), FT4 0.47 ng/dL (0.58-1.64), TSH 0.28 uIU/ml (0.34-5.6), testosterone was less than 10 ng/dL (260-1000) and LH 0.87 mIU/mL (1.24-8.6) characteristic of hypopituitarism. He failed Cortrosyn stimulation test. The MRI of the sella was suggestive of pituitary hypophysitis. He was treated with prednisone and levothyroxine with alleviation of symptoms. The follow up MRI in 2 months showed improvement of the pituitary lesion. He died a few weeks later from complications of melanoma. A 49 year old man presented with extreme weakness. The symptoms started after 3 weeks of Ipilimumab therapy. The cortisol was less than 0.4 ug/dL, testosterone was less than 10.0 ng/dL, FSH 1.07 mIU/mL (1.27-19.26), LH 0.78 mIU/mL (1.24-8.62), FT4 0.32 ng/dL (0.58-1.64) and TSH 0.11 uIU/mL (0.34-5.60) characteristic of hypopituitarism. The MRI of the sella showed 1 x 0.5 x 0.6 cm pituitary lesion which was not present 4 months previously. He was treated with prednisone and levothyroxine with improvement in the symptoms.
Discussion: The presentation in all patients was non-specific and could also be attributed to the progression of malignancy. Ipilimumab related endocrinopathies may occur during or after completion of therapy. Treatment with prednisone and hormone replacement resulted in prompt improvement in all 3 cases. It is not clear if the functionality of the gland recovers after treatment with glucocorticoids.
Conclusion: Ipilimumab can cause a variety of immune related adverse events. Endocrinologists should be familiar with the potential endocrinopathies like secondary adrenal insufficiency and hypothyroidism associated with Ipilimumab and it is important to screen these patients periodically. Early recognition and treatment is important to prevent life threatening consequences from a readily treatable endocrinopathy.

Abstract #923

SQUAMOUS CELL CARCINOMA OF THE PITUITARY MIMICKING RATHKE'S CLEFT CYST

Brian O'Neill, MD, PhD, Johanna Pallotta, MD

Objective: To present a case of squamous cell carcinoma (SCC) of the pituitary initially diagnosed as hemorrhage into a Rathke's Cleft Cyst (RCC).
Methods: We describe a 49-year-old man who presented with a 3-month history of progressive fronto-temporal headaches and was found to have a 1.2 cm cystic pituitary mass consistent with hemorrhagic RCC. Biochemical, radiographic, and pathologic findings are summarized.
Case Presentation: The patient presented with severe headache, but without hypo-pituitary symptoms, acute visual changes, or pituitary hormone abnormality other than hypogonadotrophic hypogonadism in the setting of narcotic use for headache control. MRI revealed a 1.2 cm cystic pituitary mass consistent with RCC versus hemorrhagic adenoma. Over the next 3 months he had ongoing, unremitting headaches, and a repeat MRI confirming unchanged RCC with proteinaceous contents. He subsequently underwent trans-sphenoidal resection of the pituitary cyst. Pathologic analysis revealed atypical squamous epithelium with positive immuno-histochemical staining of p63 and cytokeritin cocktail confirming the diagnosis of SCC lining the cyst. MRI of the brain and spine, PET scan of the head and torso, and a complete nasopharyngeal exam revealed no primary source for metastasis. The patient's headache resolved post-surgically, and he was referred to neuro-oncology and radiation-oncology who recommended a 6-week course of stereotactic radiation therapy to the pituitary. Follow-up MRIs at 3 and 6 months showed no evidence of recurrence. His hypogonadotrophic hypogonadism resolved off narcotic therapy and he remains hormonally intact.
Discussion: Intracranial and sellar SCC can originate from metastases from external sites, direct invasion from the naso-pharynx, or least commonly from metastatic degeneration of an epithelial cyst. There are several case reports of malignant degeneration of a known epithelial cyst, such as a craniopharyngioma or epidermoid cysts, causing SCC and other intracranial malignancies, often at the cerebello-pontine angle. To our knowledge, this is the first case of a SCC of the pituitary mimicking a Rathke's cleft type cyst by MRI characteristics.
Conclusion: We report a rare case of SCC of the pituitary lining a RCC. Literature review reveals that non-metastatic SCC of the pituitary is exceedingly rare and may arise from malignant degeneration of cystic epithelial lesions which include craniopharyngioma and Rathke's cleft cysts.

Abstract #924

RESISTANT PROLACTINOMA: AN UNUSUAL CASE OF PROLACTINOMA UNRESPONSIVE TO MEDICAL TREATMENT WITH HIGH DOSE DOPAMINE AGONISTS

Mahshid Mohseni, MD, Tipu Faiz Saleem, MD

Objective: Prolactinoma is the most common functional pituitary tumor. It is treated medically with dopamine agonists.Resistance to medical therapy is rare and is

defined as failure to achieve normal prolactin level or inability to induce 50% tumor shrinkage on standard dose of dopamine agonist . We are presenting a case of dopamine agonist resistant prolactinoma (DARP).

Case Presentation: A 45 year old lady , with a history of hypertension and diabetes, presented with amenorrhea for 2 years and recent visual disturbance. Physical examination showed bitemporal hemianopea superiorly without optic nerve atrophy. Initial serum prolactin level was over 600ng/ml (Normal 2.8-29.2ng/ml) with MRI showing a 1.9 x 2.4 cm enhancing suprasellar mass. On initial evaluation, patient also had initial high gastrin and blood calcium levels with normal PTH level. Follow up gastrin levels were normal and CT scan of the abdomen and pelvis was negative for any tumors .Genetic testing for Multiple Endocrine Neoplasia type 1(MEN-1) syndrome was negative. She was initially started on Parlodel but later on switched to cabergoline due to lack of complete response to Parlodel. Patient's serum prolactin level responded partially to Carbergoline and decreased to 173ng/ml in few weeks.Follow up MRI in 3 months showed a macroadenoma 2.1 x 2.7 cm impinging on optic chiasm and encasing carotid arteries without any significant change from previous study. Due to improvement in neurologic symptoms and partial decline of prolactin levels, continuation of medical therapy was recommended by the consulting neurosurgeon. . Carbergoline dose was slowly titrated up to 8mg per week with no further decrease in plasma prolactin level. Due to lack of complete response, higher cost and risk of side effects of long term high dose dopaminergic therapy , patient was reffered for surgical debulking of tumor.

Discussion: Prolactinomas are successfully treated with dopamine agonists 90-95% of the time. Resistance to medical treatment in these tumors is associated with reduced density of Dopamine Receptor D2 and or altered Neuron Growth Factor Beta Receptor gene expression. Resistant tumors are more invasive with higher mitotic index and usually associated with cavernous sinus invasion. Initial resistance can be overcome by increasing the dose (up to 11 mg/wk) or switching from one to the other dopaminergic agent. Due to the invasive nature of these DARP, treatment with surgical resection or radiation therapy is needed.

Conclusion: Management of DARPs can be challenging. Failure to achieve normal serum prolactin levels and lack of tumor shrinkage on MRI with optimal medical treatment may indicate the need for surgical resection or radiation therapy.

Abstract #925

HYPOPITUITARISM SECONDARY TO ASPERGILLUS SELLAR ABSCESS

Sarvpreet Ahluwalia, MD, Jennifer Mccarty, Debra Simmons, MD, FACP, Petpring Prajuabpansri, MD

Objective: To describe a rare case of aspergillus sellar abscess causing hypopituitarism in a patient with multiple myeloma.

Case Presentation: A 56 year old woman currently undergoing chemotherapy for multiple myeloma was admitted with complaints of severe headache and diplopia. She had maxillary sinus tenderness and right eye ptosis on examination. CT sinuses showed mucosal disease in bilateral maxillary, sphenoid, ethmoid and frontal sinuses. Aspergillus Index was positive at 7.3. She underwent Functional Endoscopic Sinus Surgery with intra-operative findings of invasive aspergillosis. Sinus mucosa biopsy confirmed aspergillus with evidence of angio-invasion. MRI brain on admission was unremarkable but repeat imaging done several weeks later showed right temporal lobe abscess and a 2 cm ring-enhancing cystic mass lesion adjacent to planum sphenoidale extending above the pituitary gland. Hormonal evaluation was significant for hypogonadotrophic hypogonadism with low levels of LH 1.5 mIU/ml (10.8-58.6), FSH 3.7 mIU/ml (16.7-113.5) and Estradiol <20 pg/ml. Thyroid stimulating hormone was inappropriately low at 0.95 uIU/ml (0.34-5.60) with low free T4 of 0.40 ng/dL (0.58-1.64). Serum prolactin level was 31.42 ng/mL (2.7-19.6). There was also evidence of HPA axis dysfunction with low cortisol (2.9 ug/dL) and inappropriately low ACTH level of 9 pg/mL (6-58). She was started on steroid replacement and levothyroxine. Invasive aspergillosis was aggressively treated with intravenous antifungals consisting of Liposomal Amphotericin-B and Voriconazole. She was deemed unsuitable for neurosurgical intervention. Repeat imaging several weeks later showed stable temporal and sellar abscesses but an interval development of a new mycotic aneurysm of right carotid artery. She remains on antifungal treatment.

Discussion: Sellar abscess from CNS aspergillosis is an extremely rare entity with only few hundred reported cases. Direct extension from a contiguous extacranial location such as paranasal sinuses is usually the most common route of spread of infection to CNS. Diagnostic features suggestive of a sellar abscess consist of diabetes insipidus (though not seen in our patient), hypopituitarism and the presence of a sellar cystic mass with an enhanced rim on imaging. Transsphenoidal evacuation followed by antifungal therapy is generally recommended. Since our patient was noted to have multiple abscesses and

widespread CNS infection she was deemed unsuitable for neurosurgical intervention.
Conclusion: Sellar abscess from invasive aspergillosis though a rare entity can lead to hypopituitarism and should be considered in the differential especially when imaging shows the presence of a ring-enhancing sellar cystic mass.

Abstract #926

EFFECTS OF GROWTH HORMONE THERAPY ON IGF-1 LEVELS IN GROWTH HORMONE-DEFICIENT INDIAN CHILDREN

Sunil Kota, MD, Siva Kota, Svs Krishna, Lalit Meher, Kirtikumar Modi

Objective: We evaluated the impact of GH treatment on auxological and biochemical parameters (serum IGF-1) in Indian children with growth hormone deficiency (GHD).
Methods: Patients with short stature were evaluated. The inclusion criteria were a) Height < 3rd percentile or 2 standard deviation score (SDS) below national mean b) growth velocity (GV) below the 25th percentile on the velocity chart, c) Serum GH concentration < 10 ng/ml during 2 provocation tests. The exclusion criteria were a) Presence of active tumor, systemic disease, chromosomal abnormality, or syndromic illness b) chronic medication other than thyroid or cortisol replacement. Patients were followed up at 3 months interval with auxology evaluation, pubertal staging, bone age determination and IGF-1 assay, HbA1C and fasting blood sugar (FBS) measurement. All patients received 0.3 mg/kg/week GH in 7 divided doses subcutaneously daily at night. Patients with multiple pituitary hormone deficiencies received additional substitution therapy. Analysis of variance and unpaired t test were used to calculate differences among groups. P value < 0.05 was considered significant.
Results: 25 prepubescent children (M: F= 14:11) with mean age 8.6 ± 2.9 years were enrolled. Maximum height was attained in the first year (110.7 ± 16. 1 cm). The GV SDS increased significantly to 3.3 ± 1.9 and 2.1 ± 1.3 at 12 and 24 months respectively, compared to -2.1 ± 1.1 at baseline ($p< 0.05$). Serum IGF-1 levels increased to 121.2 ± 113.8 and 195.9 ± 147.1 ng/ ml respectively at 12 and 24 months, both showing significant increment compared to 29.2 ± 17.3 ng/ ml at baseline ($p< 0.05$). The height SDS at baseline, 1 year, and 2 years was -5.38 ± 1.4, -4.10 ± 1.4, and -3.6 ± 1.3, respectively ($P < 0.005$) whereas the IGF-1 SDS at corresponding intervals was -3.40 ± 0.8, -1.74 ± 1.2 and, -1.54 ± 1.7, respectively ($P > 0.1$). Height SDS in children with normal IGF-1 SDS did not improve significantly in comparison to children with IGF-1 SDS < -2 at 2 years. Bone age advancement, the occurrence of puberty, and FBS, HbA1C did not change significantly during therapy. One girl at 12 months and 2 girls at 24 months entered puberty.
Discussion: IGF-1 is measured to monitor GHD patients. Significant correlations between IGF-1 concentration and height SDS are reported in patients receiving GH. Though serum IGF1 levels and height SDS attained statistical significance, IGF-1 SDS failed to achieve same in our patients. Similarly no significant differences in height SDS and GV SDS were observed at 24 months between children with normal IGF-1 SDS versus those with IGF-1 SDS < -2.
Conclusion: Changes in serum IGF-1 SDS may not be a reliable marker for responsiveness to GH therapy in Indian children.

Abstract #927

EFFICACY OF RAPID ESCALATION OF CABERGOLINE IN COMPARISON TO CONVENTIONAL DOSING IN PROLACTIN SECRETING MACROADENOMA

Ashu Rastogi, MD, Anil Bhansali, Rama Walia, Pinaki Dutta

Objective: To study the efficacy of rapid escalation of Cabergoline dose as compared to conventional escalation in patients with prolactin secreting macroadenomas with relation to: - Duration to achieve normoprolactinemia and > 50% reduction in tumor volume (end point) - Cumulative dose of cabergoline required
Methods: Patients with drug naive prolactin secreting macroadenoma (maximum tumor diameter >10 mm)(male and female)were randomized to receive cabergoline, with conventional monthly (Group A) or weekly dose escalation till end point(Group B). Patients were followed with weekly assessment of prolactin, visual field and 4 weekly tumor volume with 3 tesla magnetic resonance imaging (MRI).
Results: Twenty seven subjects (13 and 14 in group A and B respectively) completed a minimum follow up of 24 weeks. The baseline prolactin (3301±4502.3 ng/ml and 7490±10918 ng/ml; p=0.28), and tumor volume (11637±14795 mm3 and 20212±25175 mm3; p=0.29) were not significantly different. Duration to achieve normoprolactinemia was 10.9±10.5 weeks (2-36 weeks) in Group A and 7.6±6.8 weeks (1-24) in group B (P=0.37). The overall tumor reduction was 70.7±20.8% and 84.6±16.3% (p=0.30) in group A and B respectively. The dose of cabergoline for achieving the above end points was higher in group B compared with Group A (4.3 mg/week and 1.3 mg/week respectively; p=0.01). There was a significant positive correlation between prolactin and tumor reduction at 4 weeks with subsequent response by 24

weeks (r=0.79;p<0.000 and r=0.62;p=0.001 respectively).
Discussion: The mean prolactin reduction was 93.6%, 94.2% and 96.9% at 4, 12 and 24 wk respectively and tumor volume reduction of 76.2% at 24 weeks in the whole cohort which is significantly greater than the previous studies. The mean prolactin and tumor reduction were 98.2% versus 96.9% and 72.2% versus 80.3% in rapid and conventional escalation group which were not significantly different (p=0.39), suggesting an equal efficacy using either of the dosing regimens of cabergoline for macroprolactinoma. However, the incidence of side effects were similar in the two groups and rapid dose escalation was well tolerated by the subjects.
Conclusion: Rapid escalation of cabergoline dosing is not superior to conventional escalation regarding, duration to achieve normoprolactinemia and reduction in tumor volume. The cumulative or mean dose of cabergoline required is higher in the rapid escalation.

Abstract #928

APOPLEXY MIMICKING MENINGITIS: A DIAGNOSTIC INTRICACY

Margie Banzuelo-Rio, MD, Akash Patel, Sunil Asnani, MD, FACE, Marnie Rosenthal

Objective: To discuss a case of pituitary apoplexy presenting as chemical meningitis.
Methods: A case report and review of the literature
Case Presentation: A 25-year old male with no history presented with 4 days of flu-like illness consisting of generalized weakness and runny nose, nausea, vomiting, headache, stiff neck and fevers to 103oF followed by progressive confusion and combative behavior. Physical exam revealed a left lateral visual field defect and nuchal rigidity. Cerebrospinal fluid (CSF) studies showed an elevated protein 214 mg/dl; Normal (N) 14-45), white blood cell count (WBC) 482 /cu mm (N:5-8) with 69% neutrophils and 15% lymphocytes, normal CSF glucose, many RBC (N:0/uL) and mild xanthochromia. Vancomycin and Ceftriaxone were initiated in addition to Dexamethasone. A non-contrast CT and MRI demonstrated a 3x2x2.5 cm intrasellar mass compressing the optic nerve and chiasm consistent with a pituitary macroadenoma plus an incidental finding of sphenoid sinus disease. Laboratory studies revealed a TSH of 0.26 µIU/mL (N: 0.34-5.6), Free T4 0.81 ng/dl (N: 0.5-1.26), FSH 2.5 µIU/mL (N: 1.4-18.1), LH 1.4 µIU/mL (N: 1.5-9.3), and prolactin 0.9 ng/mL (N: 2.1-17.1). Levothyroxine was added. After transphenoidal resection of the pituitary mass, the patient's visual deficits vastly improved. Histopathology revealed a pituitary adenoma with hemorrhage and necrosis consistent with apoplexy. ACTH and prolactin stains of the tumor were negative. Blood and CSF cultures were negative. Antibiotics covering sinus microorganisms were continued post-operatively based on the MRI findings of chronic sinusitis given a transphenoidal surgical approach. The patient's clinical status improved and he was then discharged on hydrocortisone and levothyroxine.
Discussion: Pituitary apoplexy is a rare and potentially life-threatening condition. Meningismus may be seen in patients with pituitary apoplexy, caused by either subarachnoid hemorrhage or sterile chemical meningitis.
Conclusion: It is important to keep pituitary apoplexy in the differential while treating meningitis/meningoencephalitis and addressing the underlying endocrinopathy once confirmed, from both a surgical and physiologic perspective by hormone replacement and resection, to improve outcomes and decrease morbidity and mortality.

Abstract #929

MISSED CASE OF MYXEDEMA DUE TO CONCOMITANT EMPTY SELLA

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Objective: Severe hypothyroidism if undiagnosed can lead to myxedema coma, which has a high mortality rate. We describe a case of severe hypothyroidism in a patient with primary hypothyroidism whose diagnosis was delayed as she had a normal TSH. She was later found to have a low Free T4 and an Empty Sella on MRI
Methods: Case Presentation.
Case Presentation: A 43-year-old female was admitted to the hospital with shortness of breath. She was diagnosed with hyperthyroidism 20 years earlier and received RAI treatment. She was lost to follow up and was not on replacement Levothyroxine (LT4) for a period of years. Her TSH at admission was 4.39 uIU/ml (0.35-5.50). At presentation she was slow to respond. Her face was puffy and had a waxy appearance. Her skin was dry and thick and her thyroid was not palpable. She had a delayed relaxation phase on testing her deep tendon reflexes. Her CXR showed bilateral pleural effusion for which she underwent thoracenthesis. Her Echocardiogram showed large pleural effusion with no tamponade physiology. Her pleural effusion recurred again during her admission and she had no improvement in her symptoms. . As she had a normal TSH hypothyroidism was not brought up in the differential diagnosis until an astute physician checked her free T4, which <0.10 ng/dl (0.8-1.80). Free T3 was <0.2 pg/ml (2.3-4.2). She was started on LT4 100 mcg daily and IV hydrocortisone and in a few days her pleural effusion improved and a follow up echocardiogram showed

resolution of her effusion. Her other labs were IGF -1 <25 ng/ml (98-261), GH 0.01 ng/ml (0.01-3.61), LH 4.2 mIU/ml, FSH 17.8 mIU/ml, Estradiol 62 pg/ml, Prolactin 5.7 ng/ml (1.8-20.3), ACTH <5.0 pg/ml (10-66). She was discharged on oral hydrocortisone and LT4 and continues to do well An outpatient Pituitary MRI showed partially empty sella with majority of residual pituitary tissue displaced inferiorly and along the floor of sella

Discussion: Central hypothyroidism is an uncommon condition and coexisting primary and secondary hypothyroidism has been rarely reported. We could only find one other case in the literature with a patient with both primary and central hypothyroidism

Conclusion: Correct diagnosis of patients suspected of having clinical signs and symptoms of hypothyroidism is of great importance. When applicable, clinicians must also check for free T4 levels which can help diagnose central hypothyroidism and provide a clue for other pituitary hormone deficiencies especially that of cortisol as well as prevent severe hypothyroidism to progress to Myxedema coma

Abstract #930

FATAL METASTATIC PITUITARY CARCINOMA 17 YEARS AFTER INITIAL PRESENTATION OF LARGE PITUITARY TUMOR

Ivica Boban, MD, James Evans, Jeffrey Miller, MD

Objective: We present a case of pituitary carcinoma which developed 17 years after endoscopic transsphenoidal hypophysectomy (TSHx) for a large non-functional pituitary macroadenoma.

Case Presentation: A 45 years old male underwent TSHx in 1993 for a non-functioning pituitary macroadenoma. His past medical and family history were unremarkable. There was no evidence of recurrent disease until 2006 when he experienced sudden onset of diplopia. MRI of the pituitary revealed tumor recurrence with chiasmal compression, for which he underwent a second TSHx. Histopathology revealed partially necrotic pituitary adenoma, with increased pleomorphism, but no increased mitotic activity. Following surgery he had normal visual fields, a normal pituitary panel and no evidence of residual or recurrent tumor on follow-up imaging. In 2009 he experienced blurred vision and polyuria. MRI confirmed recurrent pituitary macroadenoma with a mass effect on the optic chiasm again. Biochemical work-up was consistent with panhypopituitarism and central diabetes insipidus, requiring replacement therapy. Another TSHx was performed and histopathology was unchanged from prior. Two months after the third TSHx, imaging again showed persistent pituitary mass for which stereotaxic radiotherapy (XRT) was administered. In 01/2011, a year after XRT, MRI showed further pituitary tumor growth. Pituitary biopsy was consistent with necrotic pituitary adenoma, with infiltration of fibrous tissue, and immunohistochemical stains were negative for all pituitary hormones, but strongly positive for synaptophysin and a very high cellular marker proliferation index (Ki67) of 96%. Body CT scans were without demonstrable metastatic disease. Patient underwent second salvage XRT of the pituitary mass. Three months after XRT he developed low extremity weakness. Work up showed metastatic lesions in the thoracic spine, visceral organs, muscle and skin. Metastatic pituitary carcinoma was confirmed based on biopsy of hepatic mass. Despite aggressive treatment with chemotherapy and palliative XRT, patient's condition continued to deteriorate and he expired 3 months later.

Discussion: While pituitary adenomas are common, pituitary carcinoma are extremely rare. Diagnosis of pituitary carcinoma can be very challenging as the presence of metastasis is the criterion for malignancy. Usually pituitary carcinomas develop from relapsing and previously operated or irradiated invasive adenomas.

Conclusion: This case demonstrates the typical natural history and aggressive course of pituitary carcinoma. A high Ki-67 index appears to predict rapid disease progression.

Abstract #931

AN EXTREMELY DELAYED PRESENTATION OF SIADH(SYNDROME OF INAPPROPRIATE ANTI DIURETIC HORMONE SECRETION) AFTER TSS (TRANSSPHENOIDAL SURGERY)

Niharika Singh, MD, Farah Hasan, MD,
Tahira Yasmeen, MD

Objective: To present a case of extremely delayed development of SIADH after TSS for prolactinoma.

Case Presentation: A 43year old male with history of schizophrenia presented with a month long history of blurred vision and headache. Head CT scan showed a large sellar mass with suprasellar extension and skull base erosion. On laboratory testing he was found to have normal electrolytes, renal function and thyroid function but prolactin was elevated at 1,379ng/ml. He was seen by a neurosurgeon and underwent TSS. The pathology report was consistent with pituitary adenoma. Patient did not have any significant postoperative complications and was discharged home. He remained asymptomatic until 15weeks after the surgery when he presented with a two day history of headache. A brain MRI showed residual burden of the tumor in sellar/suprasellar location. Significant laboratory values were- prolactin

880ng/ml, serum sodium 119mmol/L, serum osmolality 249mosm/kg, urine osmolality 437mosm/kg, urine sodium 131mmol/L, BUN 4md/dl, creatinine 0.71mg/dl, TSH 0.477mcunit/ml and random cortisol 22mcg/dl. He was clinically euvolemic. He was put on 500ml/day water restriction but serum sodium level dropped to 117mmol/L within a few hours and he was started on 3% saline. His haloperidol was switched to aripiprazole. With these interventions the serum sodium gradually improved.
Discussion: SIADH occurs in about 9-35%patients postoperatively after TSS. It is usually detected upto about 2weeks in the postoperative period, with sodium level nadir occurring around the seventh day. It can be symptomatic or asymptomatic depending on the sodium level and the rapidity of its development. This patient developed SIADH about 15weeks after the transsphenoidal pituitary surgery. His TSH and cortisol levels were normal, ruling out the possibility of hypothyroidism and adrenal insufficiency respectively as the cause of hyponatremia. The only medications that the patient was on were cabergoline and haloperidol. Cabergoline is not associated with hyponatremia. Haloperidol is an extremely rare cause of SIADH, due to this and the fact that the patient was on haloperidol for years before presentation, haloperidol was considered an unlikely cause. Even though prolactinoma itself is a rare cause of SIADH residual tumor was seen on MRI and could have been the cause of SIADH. The development of SIADH was therefore thought be related to the either TSS or the residual prolactinoma.
Conclusion: SIADH is a fairly common complication of TSS and usually occurs upto second week in the postoperative period. However, if SIADH is encountered in clinical practice even beyond this time frame, TSS and residual tumor should be considered in the differential.

Abstract #932

RESPONSIVENESS OF A GIANT PROLACTINOMA TO MEDICAL MANAGEMENT

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Yousef Altowaireb, MD,

Case Presentation: A 24-year old healthy male was hospitalized with a 1-month history of bitemporal headache, behavioral change, sexual dysfunction and an episode of seizure. MRI of the brain showed an irregular and heterogeneous mass measuring 6 x 4.3 x 5.8 cm in the sellar and suprasellar area with involvement of optic chiasm and significant extension into the left middle cranial fossa and bilateral cavernous sinuses. The left superior temporal lobe was also compressed and displaced laterally. Biochemical work-up revealed a prolactin level of 13,458 ng/ml (4.0-15.2 ng/ml), low normal gonadotropins with an FSH of 2.2 mIU/ml (1.5-12.4 mIU/ml), LH of 4.8 mIU/ml (1.7-8.6 mIU/ml) and a low free testosterone of 2.41 ng/dl (3.7-14.7 ng/dl). Somatomedin C was within normal limits. Pituitary thyroidal and adrenal axes were intact. Initial visual field testing showed a small left nasal defect. He was started on Cabergoline at a dose of 0.5 mg twice weekly. Six months later, MRI of the pituitary showed a decrease in the size of the prolactinoma to 3.0 x 3.8 x 3.1 cm. Serum prolactin level had decreased to 303 ng/ml. Symptoms including personality changes and sexual dysfunction had completely resolved, with no recurrence of seizures.
Discussion: Giant prolactinomas are a rare subset of pituitary macroadenomas, characterized by a size greater than 3 cm. They may be aggressive with massive extrasellar involvement and are usually associated with prolactin levels greater than 1000 ng/ml. In our patient, initial presentation of seizures was likely related to compression of temporal lobe which is known to be highly epileptogenic. Lactotroph adenomas are usually responsive to medical treatment. Studies have shown higher efficacy with Cabergoline compared to Bromocriptine, in terms of normalization of prolactin level as well as reduction in tumor size. The latter generally occurs in 6 weeks to 6 months. Being D2 specific, Cabergoline also has a favorable side effect profile. The efficacy of Cabergoline has been well documented in treatment of giant prolactinomas, even in the presence of neurological sequelae. Therefore, surgery should only be considered if medical therapy is contraindicated or has failed or in a woman with a large prolactinoma who wishes to become pregnant. Rarely, resistant prolactinomas (particularly if familial) are associated with mutations in the AIP gene.
Conclusion: This case report demonstrates that Cabergoline is a safe and effective treatment option for giant prolactinomas, even in the presence of neurological compromise.

Abstract #933

DIABETES INSIPIDUS WITH AN AUTONOMIC VARIANT OF GUILLAIN BARRE SYNDROME

Hussein Rajab, MD, Julius Sagel, Soonho Kwon, MD

Objective: To report a case of DI in a patient with an autonomic variant of Guillain Barre Syndrome, (GBS).
Case Presentation: A 31 year-old male with past medical Hx of chronic back pain, was admitted to the ER for severe abdominal pain, he had a normal CT of the abdomen, pelvis, and a normal EGD. During his hospitalization, he developed an episode of hives, hypotension, and pulseless electrical activities (PEA). Anaphylactic reaction was suspected; after resuscitation,

he was transferred to the ICU for further care. He was quickly extubated, but he soon developed peripheral neuropathy and burning pain in his extremities and face. He began to have difficulty swallowing. MRI of the brain was normal and a lumbar puncture showed an elevated protein and albuminocytologic dissociation. The working diagnosis was an atypical variant of GBS. The patient was treated with plasmapharesis followed by IVIG. He had a very labile BP, hyporeflexia, a neurogenic bladder requiring intermittent catheterization. After the patient was stabilized and transferred to the floor, he started to complain of polydipsia, and polyuria. A water deprivation test was done. Prior to the test, the serum Na was 139, serum osm was 287, urine osm was 178, after 2 hours of water deprivation, plasma Na remained 139, serum osm has increased to 306, that time, urine osm increased to 372. After ADH, serum Na decreased to 136, serum osm decreased to 287, and urine osm increased to 529. These results were consistent with DI, possibly partial central since the increase in urine osmolality was less than 100 %, but about 30 % only after ADH administration. The anterior pituitary hormones were normal. DDAVP started at night and the symptoms related to DI have improved.

Discussion: Guillain-Barre syndrome is an acute immune-mediated polyneuropathy. Dysautonomia occurs in 70 % of patients and manifests as symptoms that include tachycardia, urinary retention, hypertension alternating with hypotension, orthostatic hypotension, and bradycardia. Autonomic dysfunction in GBS has been postulated as being a result of sympathetic and parasympathetic failure or over activity.

Conclusion: While DI is apparently rare, the reverse situation, namely excessive ADH secretion, has been reported with GBS. Hyponatremia due to SIADH, and Salt Wasting Syndrome, has been frequently reported with GBS. To the best of our knowledge, only two reports, from 1968, and 1972, documented GB with DI. It is important for the clinician to be aware that this dysfunction can complicate GB syndrome. Further pathological studies of the CNS including the hypothalamic-posterior pituitary area would assist and clarify the mechanism responsible for this association.

Abstract #934

ONCOLOGIC PROFILE IN A SERIES OF CASES DIAGNOSED WITH INSIPIDUS DIABETES

Mara Carsote, MD, Cristina Ene, Dana Terzea, Dan Hortopan, Madalina Musat, Raluca Trifanescu, MD, Catalina Poiana, MD, PhD

Objective: We present a series of 3 cases with tumor associated insipidus diabetes (ID).

Case Presentation: .F. 61 - year old female who has been diagnosed with ID 8 months ago. At diagnosis, the cerebral and pituitary-hypothalamic CT showed no tumor, except for a maxillary cyst of 18 by 12mm. 6 months later, while the symptoms were controlled by ADH therapy, the MRI showed an enlargement of the pituitary stalk of 5 by 5 mm and, at the level of the left occipital diploe, a tumor of 21 by 11 by 19 mm was diagnosed. Histiocytosis was suspected and then confirmed and chemotherapy was started. N.N. 60 -year old male was diagnosed in July 2011 with idiopathic ID and had a good response to ADH therapy. After 6 months, he was re-evaluated, but the MRI showed changes, respective a tumoral stalk 5.5 by 6 by 5 mm, probably related to a hypothalamic tumor that cannot be described at the moment. The neurosurgical consult indicated close follow up by serial MRI every 3-6 months. M.N., 55 - year female patient is treated for ID since 2006. She was yearly evaluated, but no evident cause was found until August 2010 when the MRI revealed a left medio-lateral tumor at the level of Willis polygon of 8.5 by 7.4mm in contact with the medial cerebral artery and the pituitary stalk. She also presented asymptomatic non-PEG suppressible hyperprolactinemia of 108.6 ng/mL (normal 2.8-29.2). A hypothalamic hamartoma was diagnosed, but no neurosurgical approach was recommended. Therapy with cabergoline (0.5 mg weekly) was started. In September 2011, the MRI scan showed increased tumor dimensions to 15.3 by 10.6 mm.

Discussion: Adult idiopathic ID is sometimes an epiphenomenon of a cancer and it might be considered in selected cases the first sign of the oncologic disease. It might take some time in order to diagnose the causing disease, thus close follow up is necessary.

Conclusion: Practitioners should be aware of the possibility that acute onset of the polyuria, polydipsia syndrome might hide a severe oncologic disease.

Abstract #935

CORRELATION OF PROLACTIN CONCENTRATIONS WITH CLINICAL ACTIVITY IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

Kaye-Anne Newton, MD

Objective: To investigate the relationship between hyperprolactinemia and systemic lupus erythematosus (SLE).

Case Presentation A 26 year old African-American woman presented with amenorrhea of one year duration. She had no significant past medical history. Her home medications included fluticasone nasal spray and fexofenadine and her

initial physical examination was significant only for an overweight female. Her initial evaluation showed negative serum βhCG, elevated prolactin (PRL) of 75 ng/mL and MRI of the brain was negative for a prolactinoma. Patient denied nipple discharge, headache or visual disturbance. Work up for the evaluation of hypothyroidism, chronic renal failure, cirrhosis, and adrenal insufficiency as the etiology of hyperprolactinemia was negative. At a subsequent endocrine evaluation, patient presented with diffuse joint swelling with tenderness, increased warmth and decreased range of movement. Blood work showed positive ANA, elevated ESR at 83 mm/hr, elevated C-reactive protein at 7.8, anti-dsDNA >300 IU/mL, and her prolactin level had increased to 129ng/mL. Her WBC also decreased from 3 to 1.7, hemoglobin decreased from 11.8 to 9.7 g/dL and creatinine increased to 1.59 mg/dL. Patient met the American College of Rheumatology criteria for SLE and was started on hydroxychloroquine and prednisone. MRI of the sella turcica showed a 6mm microadenoma.
Discussion: Prolactin is a versatile hormone with many functions. It is produced by the anterior pituitary and various other extrapituitary sites including endothelial cells and immune cells. Its effect on the immune response has been proved in both animal experiments and in humans. Elevated prolactin levels have been reported consistently in SLE patients and occur in 15-31% of SLE patients. Recent evidence suggests that PRL plays a part in the pathogenesis of systemic lupus erythematosus. rolactin functions similarly to a cytokine. It has been shown to stimulate the immunoglobulin and autoantibody production of human peripheral blood mononuclear cells (PBMC) from healthy subjects and patients with SLE. In, addition, PRL is secreted by PBMC from patients with SLE in larger amounts than from healthy subjects. This may be the result of a single nucleotide polymorphism across the PRL extrapituitary promoter. Several studies have showed that treatment with bromocriptine in SLE patients with elevated free prolactin showed improvement in disease activity and survival.
Conclusion: Autoimmune diseases should be considered in the workup of hyperprolactinemia. Treatment of elevated prolactin may improve the course and outcome of autoimmune disease.

Abstract #936

CENTRAL RETINAL VEIN OCCLUSION (CRVO) IN A PATIENT DIAGNOSED WITH FACTOR V LEIDEN MUTATION (FVL) & UNDERLYING CUSHING'S DISEASE (CD)

Rod Marianne Arceo-Mendoza, MD, John Kennedy

Objective: To describe a unique case of a younger woman with sudden unilateral decrease in vision, diagnosed with CRVO. Diagnostic workup revealed Factor V Leiden mutation & Cushing's Disease.
Case Presentation: A 43 year old woman with obesity, hypertension & hyperlipidemia presented to Ophthalmology complaining of sudden, painless decreased right eye vision (acuity = 20/100). She was diagnosed with Non Ischemic CRVO. Hypercoagulable work up revealed heterozygous FVL mutation. One month later, a brain MRI for right otalgia noted an incidental 5 x 4 mm hypoenhancing left microadenoma within the pituitary gland. An elevated 24-hour urine free cortisol of 124.7 mcg (NL: 4-50 mcg/24hour) established cortisol excess with incompletely suppressed 8 AM serum cortisol level = 7.3 mcg/dL (NL: <1.8) after a low dose 1 mg overnight dexamethasone suppression test (ODST). An elevated ACTH = 47 pg/mL (NL: 0-46) & suppressed 8 AM cortisol level of 0.9 mcg/dL by high dose 8 mg ODST provided evidence that the pituitary microadenoma was ACTH producing consistent with Cushing's Disease. Pathology at transphenoidal endoscopic resection immunostained positive for ACTH. Post-operative biochemical & radiologic studies were consistent with complete resection.
Discussion: CRVO results from thrombosis of the central retinal vein at the level of the lamina cribrosa & according to Virchow's triad for thrombogenesis, requires vessel damage, stasis and hypercoagulability. CRVO in younger patients is an uncommon finding. In our patient, the younger age led to the initiation of a hypercoagulable work up with subsequent finding of heterozygous FVL mutation, which renders Factor V partially resistant to degradation by Activated Protein C (APC). The unimpeded Factor V protein retains its pro-coagulant activity and predisposes to thrombosis. FVL and APC resistance have been described in association with CRVO. This hypercoagulable state can be exacerbated when a patient who is heterozygous for FVL has one or more additional causes for hypercoagulability. In our patient, the underlying Cushing's Disease may have contributed to the hypercoaguable milieu of her Factor V Leiden mutation, with the resultant clinical expression of a CRVO in a younger woman. Reports linking Cushing's syndrome to an increased incidence of thromboembolic complications more commonly report clots in larger

veins such as deep vein thrombosis of legs or pulmonary embolism. The mechanism is felt to be due to an increase in plasma clotting factors & an impairment of fibrinolytic capacity.
Conclusion: To our knowledge, this is the first report demonstrating a temporal association of a CRVO in a patient with Factor V Leiden Mutation and underlying Cushing's disease.

Abstract #937

RESPONSES OF PATIENTS WITH ADULT GROWTH HORMONE DEFICIENCY TREATED WITH GROWTH HORMONE OVER 3 YEARS: ANALYSIS OF RESULTS FROM THE ANSWER PROGRAM®

Murray Gordon, MD, Richard Levy, MD, Jeff Goldstein, Robert Gut, John Germak, MD

Objective: Data on adult patients with growth hormone deficiency (AGHD) have been collected through the American Norditropin Studies: Web-Enabled Research (ANSWER®) Program/NovoNet® registry on patients enrolled by participating physicians. The current analysis focuses on the outcomes of AGHD patients with either isolated growth hormone deficiency (IGHD) confirmed with a growth hormone stimulation test (GHST) or multiple pituitary hormone deficiency (MPHD) treated with growth hormone (GH) over 3 years.
Methods: Data from 425 patients enrolled as of October 2011 with IGHD or MPHD were analyzed for age (years), body weight, body mass index (BMI), GH dose, serum IGF-1 levels and IGF-1 SDS at baseline and at years 1, 2, and 3 of GH treatment (GHT). The frequency and type of GHST were also determined. Data are expressed as mean ± standard deviation.
Results: Baseline characteristics were similar among 425 patients with IGHD (46%; 68M, 128F) and MPHD (54%; 110M, 119F), except for sex (F>M) and a lower serum IGF-1 level (ng/mL) in IGHD subjects (120.4±93.5) compared with MPHD subjects (146.2±113.2; P<0.03). Of MPHD patients, 44% (101/229) underwent 1 or more GHSTs and among those with reported hormonal deficiencies, 24.7% (43/174) had 3 or more hormonal deficiencies. The most common GHST agents used in IGHD patients were arginine/L-Dopa (42.9%) and glucagon (21.9%), and for MPHD arginine (29.7%) and glucagon (22.8%). Use of arginine/GH-releasing hormone (GHRH) decreased since discontinuation of GHRH in the United States in 2008, while use of the glucagon test gradually increased. Cross-sectional data analysis showed that mean serum IGF-1 concentrations increased during GHT; IGF-1 SDS levels also increased over time (IGHD SDS: -0.7±1.22 at baseline, 0.4±1.42 at year 3; MPHD SDS: -0.5±1.92 at baseline, 0.6±1.47 at year 3). Change from baseline in body weight and BMI were not significantly different between IGHD and MPHD groups during GHT. In comparison with older patient age groups (35-60 years and >60 years), mean GH dosages were higher in younger subjects (18-35 years: IGHD, 0.45 mg/d [7.0 mcg/kg/d]; MPHD, 0.95 mg/d [11.0 mcg/kg/d]).
Discussion: While all patients diagnosed with IGHD in this analysis underwent GHST, not all with MPHD had a GHST.
Conclusion: Discontinuation of arginine/GHRH apparently led to increased use of the glucagon GHST, which reflects current guidelines and literature recommending its use for the diagnosis of AGHD. Mean IGF-1 SDS levels increased appropriately during GHT. Mean GH doses were higher in younger than in older patients, consistent with some younger patients receiving treatment during the transition phase from childhood GHD.

Abstract #938

FROM EMPTINESS TO A TOTAL ECLIPSE OF THE HEART

Willy Marcos Valencia Rodriguez, MD,
Ricardo Correa, MD, Jerson Munoz Mendoza, Atil Kargi

Case Presentation: A 39-year-old white Hispanic female presented to the emergency room with progressively worsening shortness of breath, pleuritic chest pain and fatigue. The information was provided by her family, who described a 30-lb-weight lost over 6 month, denied fever, cough or sick contacts. The patient had hypothyroidism and familial hypokalemic periodic paralysis, with four female relatives affected by the latter. She was in acute respiratory distress. Her skin was dry and cold skin, without lesions or hyperpigmentation, absent pubic and axillary hair. Her pulse was 110 bpm and blood pressure was 50 mmHg. She had a 7-cm jugular vein distention. The thyroid was not palpable. The lungs were clear to auscultation bilaterally. The heart sounds were distant without murmurs, rub or gallops. She had a 1+ bilateral pitting edema below the knees. Her blood tests showed normocytic, normochromic anemia, leukocytosis and normal platelet count, hypokalemia, non-anion gap metabolic acidosis with increased lactic acid, and elevated creatinine. Liver tests were within normal limits. Thyroid stimulating hormone was normal (0.84 μIU/mL). The electrocardiogram showed low voltage QRS complexes. A chest x-ray showed interstitial pulmonary edema. A transthoracic echocardiogram revealed pericardial effusion, signs of diastolic collapse of the right atrium, and normal ventricular function. Emergent

pericardiocentesis removed 100 mL of straw-colored fluid, yet the patient remained hemodinamically unstable, requiring increasing doses of vasopressors. Infectious and rheumatologic causes were ruled out. A stimulation test with 250 µg of cosyntropin showed serum cortisol of 1.0 and 2.8 µg/dL, and IV steroids were started. The pressors were decreased but could not be weaned off. A hormonal panel was obtained, with low levels of prolactin, corticotropin, gonadotropins and estradiol, while free thyroxine (0.15 ng/ml), free triiodothyronine (0.5 pg/ml) and reverse triiodothyronine (12 ng/ml) were also low. Levothyroxine and then liothyronine were added, and slow clinical improvement followed. A Magnetic Resonance Imaging of the brain found an empty sella turcica with residual pituitary tissue. After thirty days, the patient was discharged with a regimen of levothyroxine and dexamethasone daily, follow up instructions and bracelet alert for adrenal insufficiency and hypothyroidism.

Discussion: To our knowledge, we report the first case of empty sella syndrome manifesting with panhypopituitarism leading to cardiac tamponade.

Conclusion: Prompt recognition of this association, pericardiocentesis and proper hormonal replacement are paramount to effective therapy of this life-threatening condition.

Abstract #939

FSH SECRETING GONADTROPH ADENOMA OF PITUITARY GLAND

Prasuna Madhavaram, MD, Murat Gokden, Monica Agarwal, MD

Objective: To report a rare case of gonadotroph adenoma with circulating FSH as a tumor marker.

Case Presentation: A 48 year old man presented with sudden vision loss. The MRI of the sella showed a pituitary macroadenoma with mass effect and hemorrhagic changes requiring emergent surgery. Post surgery, FSH was 23.84 mIU/ml (1.27-19.26), LH was 0.46 mIU/ml (1.24-8.62) and testosterone was less than10 ng/dl (260-1000). The pathology showed an oncocytic adenoma with low mitotic activity, Ki-67 proliferation index of 5%, p53 over expression, diffuse LH positivity with variable intensity and histologic evidence of invasion. FSH and other pituitary hormones were negative. The circulating FSH level gradually increased to 63.2 mIU/ml correlating with increase in tumor size on imaging. He subsequently required two pituitary surgeries in two years. Post resection, FSH was 5.59 mIU/ml. The adenoma in second resection showed similar features and more obvious invasion. Mitotic activity was brisk. The adenoma in the third resection had widespread invasion of the cranial nerves, blood vessels and soft tissues. Proliferative index and mitotic activity were low; p53 was negative and LH was weakly positive. Both the second and third specimens were also positive for FSH. Furthermore, the most recent FSH level was 39 mIU/ml with tumor recurrence requiring radiation therapy. The testicles were not enlarged on ultrasound suggesting nonfunctional FSH tumor.

Discussion: The prevalence of gonadotroph adenoma is 15-20 per 100,000 of the population. Most of these tumors are inefficient producers and secretors of gonadotroph hormones. They may secrete LH or FSH or their subunits, but seldom elevate serum gonadotropin levels. These tumors are nonfunctional but rarely FSH secreting tumor may present with testicular enlargement in men and ovarian hyperstimulation in women. Isolated hypersecretion of LH is rare and may present with elevated testosterone level. The high gonadotropin levels associated with testicular failure or menopause can complicate interpretation of gonadotropin levels. In our case, the nonfunctional FSH producing adenoma had atypical features with aggressive potential for future malignant transformation. There was an imbalanced secretion of serum gonadotropins in favor of FSH to LH. Even though the immunohistochemistry was negative for FSH and positive for LH in the first resection, in subsequent resections it became positive for both FSH and LH. Circulating FSH level has correlated with tumor growth and response to treatment.

Conclusion: It is important to recognize that gonadotroph adenomas can elevate serum gonadotrophin levels. Serum FSH levels could predict tumor burden, assess treatment response and recurrence.

Abstract #940

PITUITARY APOPLEXY DURING PREGNANCY DUE TO HEMORRHAGE IN A NON-NEOPLASTIC INTRASELLAR CYST

Manash Baruah, MD,DM, Roopjyoti Hazarika

Objective: We present an interesting case of intrasellar cyst, non-neoplastic in nature, presenting during early 1st trimester of pregnancy with presenting features of acute pituitary apoplexy.

Case Presentation: A 26y old female presented with acute onset headache, vomiting, and diplopia during 7th week of her gestation. MRI revealed a T2 hyperintense cystic lesion in the pituitary fossa with suprasellar extension abutting the optic chiasma. Hormone analysis revealed panhypopituitarism. Transnasal transsphenoidal surgery was done for debulking. Histopathological examination revealed fragment of fibrocollagenous tissuealong with areas of hemorrhage and acute inflammatory exudates.

There was no evidence o tumor. Post operatively patient required multiple pituitary hormone replacement. Post operative transient diabetes insipidus was treated with vasopressin.

Discussion: Intrasellar cysts are broadly classified as neoplastic or non-neoplastic; the latter may be primary lesions of the pituitary fossa or they may arise from the parasellar region and invade into the sella. The later group is mainly constituted of craniopharyngiomas and benign cysts. These developmental sellar and/or suprasellar cystic lesion may rarely become acutely symptomatic due to hemorrhage. Presenttation during pregnancy is extremely rare.

Conclusion: Non-neoplastic intrasellar cyst during MRI are not uncommon. However it is important to consider such a pathology in the presence of apoplectic symptoms, especially in a patient with high risk background such as pregnancy.

Abstract #941

AN UNCOMMON TYPE OF PITUITARY ADENOMA IN A MALE PATIENT WITH HYPOGONADISM: A CASE REPORT

Leticia Hernandez-Davila, MD, Myriam Allende-Vigo, MD, MBA, FACP, Margarita Ramirez-Vick, MD, Meliza Martinez-Rodriguez, Marielba Agosto, Roman Velez-Rosario, William Gonzalez-Marquez

Objective: We present a patient with visual disturbances and hypogonadism, initially diagnosed with a non-functional pituitary macroadenoma.

Case Presentation: A 58-year-old male patient, presented with headaches, visual disturbances (blurry vision and diplopia), decreased libido and impotence of one year of evolution. There was no other symptom consistent with pituitary hormone over or underproduction. Physical examination revealed impairment in lateral visual fields, conjunctival injection and periorbital edema with no other abnormal physical findings. Laboratory workup revealed a total prolactin [7.0 ng/mL], monomeric prolactin [6.6 ng/mL], a morning salivary cortisol [0.28 mcg/dL], Insulin-like growth factor-1 (IGF-1) [100 ng/mL], thyroid stimulating hormone (TSH) [0.92 uIU/mL], low free thyroxine [0.64 ng/mL], follicle stimulating hormone (FSH) [5.43 mIU/mL], luteneizing hormone (LH) [1.4 mIU/mL], a low free testosterone (4.50 pg/mL), low total testosterone of 1.16 ng/mL, and low 0.39% of free testosterone. A brain MRI revealed an enhancing sellar/suprasellar mass of 3.0 x 3.2 x 3.2 cm with optic chiasm compression. Visual field analysis revealed bilateral hemianopsia. At this point, based on biochemical tests a diagnosis of non-functioning pituitary adenoma was made. Patient was referred for transsphenoidal tumor resection. Immunostains were highly positive for FSH, consistent with a FSH producing pituitary macroadenoma despite normal serum gonadotropin levels.

Discussion: Gonadotroph adenomas are hard to recognize preoperatively. These type of tumors may secrete FSH, LH or alpha subunits, but mostly occur as non-functioning tumors with normal serum gonadotropins, but amenable to detection by immunostain analysis. In contrast to other types of pituitary adenomas, rarely a recognizable clinical syndrome occur resulting from a gonadotroph adenoma. As a result, they are mostly clinically unrecognizable until they reach a considerable size capable of producing neurologic symptoms, hormonal deficiencies due to mass effect, or detected as incidental finding in neuroimaging studies. Rarely, this tumors may present hormonal abnormalities such as: ovarian hyperstimulation syndrome (very high estradiol levels and multiple ovarian cysts), elevated LH resulting increased serum testosterone in a men, or as a component of the multiple endocrine neoplasia type-1.

Conclusion: Gonadotroph adenomas, due to their clinical and biochemical presentation, may be misdiagnosed as a non-secreting pituitary adenoma. Thus, further evaluation with measurement of alpha subunit or thyroid hormone-releasing hormone stimulation tests should be considered.

Abstract #942

“SKIN CANCER WONDER DRUG” IPILIMUMAB INDUCED HYPOPHYSITIS.

Toyiba Syed, MBBS, Soamsiri Niwattisaiwong, MD, Namratha Mapakshi, David Baldwin

Objective: Metastatic melanoma is the 6th most common cancer in US with a median survival reported to range from 6 to 9 months. Immune system is a powerful natural agent against cancer. Anti-cytotoxic T-lymphocyte antigen-4 (CTLA-4) therapies represent a novel approach to cancer treatment via disruption of immune tolerance to antigens located on tumor cells. Ipilimumab, a monoclonal antibody has been shown to improve overall survival in patients with metastatic melanoma. Ipilimumab overcomes CTLA-4 mediated T cell suppression to enhance immune response against tumors.

Methods: 46 year old female with past medical history of meningioma status post resection was diagnosed with metastatic malignant melanoma and started on Ipilimumab. After completing her third treatment she developed retro-orbital headache, nausea, vomiting and cold intolerance. Endocrine workup revealed an elevated TSH and Prolactin. An MRI was done which showed homogeneously enlarged pituitary. She was diagnosed with hypophysitis and started on decadron. Her symptoms improved and the endocrine

labs returned to normal. The plan was to continue tapering doses of steroids for total of four weeks.
Case Presentation: The patient prematurely stopped taking steroids. Next day she developed altered mental status and was admitted to a hospital where she was found to be hypotensive. Repeat MRI showed internal enlargement of pituitary with homogeneous enhancements. Stress dose of steroids was started which resulted in marked improvement in the symptoms. The steroids were gradually tapered and patient returned back to her baseline.
Discussion: Disruption of immune tolerance, as caused by ipilimumab, occurs at a cost. A host of immune related adverse events (IRAEs) are associated with anti-CTLA-4 therapy. Of all the adverse effects associated with ipilimumab, hypophysitis is the possibly the hardest to recognize.
Conclusion: Clinical manifestations of hypophysitis are probably dependent on the rapidity of onset , severity and relative suppression of endocrine axes. Enlargement of the pituitary gland on imaging has been reported as earliest sign. Unlike most other IRAE where treatment with corticosteroids usually leads to resolution of symptoms, endocrine dysfunction seems to have a protracted course and is irreversible in many cases.

Abstract #943

ISOLATED AND PERMANENT CENTRAL DIABETES INSIPIDUS AFTER SEVERE TRAUMATIC BRAIN INJURY

Deepika Nallala, MBBS, MD, Michael Jakoby, IV, MD

Objective: Disorders of water balance are well recognized after moderate or severe traumatic brain injury (TBI) and are a significant risk for morbidity and death. Post-traumatic central diabetes insipidus (CDI) occurs in up to 20 percent of TBI patients and is usually transient. There is growing awareness of long term posterior pituitary dysfunction after TBI, but little is published regarding posterior pituitary injury without loss of anterior pituitary function. We present a case of isolated, permanent CDI after severe TBI with favorable outcome.
Case Presentation: A previously healthy 20-year-old woman was admitted to intensive care after a motor vehicle rollover accident. Glasgow Coma Scale score (7) was indicative of severe brain injury, and the patient was noted to suffer skull, facial, and clavicular fractures. Computed tomography (CT) imaging of the brain was notable for pneumocephalus extending into the sella turcica, subdural hematoma of the right temporal fossa, extensive petechial hemorrhages of the right temporal lobe, right hemispheric cerebral edema, and 3 mm midline shift to the left. Craniotomy, evacuation of hematoma, and placement of an intracranial pressure monitor were performed. After surgery, polyuria (3.5 L of urine in 8 hr) and hypernatremia (serum sodium 164 mM) occurred and responded well to therapy with parenteral desmopressin. Gonadotropins were low, but other measures of anterior pituitary function including 8:00 AM cortisol and ACTH, TSH, free thyroxine, and IGF-1 level were unremarkable. Thirst response was intact, though the patient required therapy with desmopressin to control polyuria. After a four week hospital stay, the patient was discharged home on 0.1 mg desmopressin twice daily. Three months after hospital discharge, spontaneous monthly menses recurred. Biochemical evaluation of anterior pituitary function six months after discharge was unremarkable.
Discussion: CDI is a typically an early and transient complication of severe TBI associated with high rates of mortality. In survivors of TBI, permanent CDI is usually reported in association with anterior pituitary hormone deficiencies. Although previously regarded as rare, recent patient series document a prevalence of long term partial or complete CDI after head injury ranging from two to seven percent. In addition to screening for evidence of late onset or persistent anterior pituitary dysfunction, survivors of moderate or severe TBI should be monitored for persistence of disordered water balance. Screening for CDI by water deprivation testing should be considered in TBI survivors with more than 3 L per day urine output.
Conclusion: Isolated, permanent CDI may be the sole pituitary dysfunction in TBI survivors.

Abstract #944

A RARE CASE OF INTRACRANIAL GERM CELL TUMOR PRESENTING WITH DIABETES INSIPIDUS [DI] AND NORMAL URINE SPECIFIC GRAVITY

Issac Sachmechi, MD, FACP, FACE, Narmada Movva, Sethu Muralidharan, Taisiya Tumarinson

Objective: To describe a case of suprasellar germ cell tumor with Diabetes Insipidus with misleading normal urine specific gravity due to new onset Diabetes mellitus.
Methods: Germ cell tumors are primarily divided into seminomas and non seminomas. Intracranial germ cell tumors vary in their geographic incidence with 0.4% of primary CNS tumors. Suprasellar germinomas usually present with evidence of hypothalamic and pituitary dysfunction which most commonly includes Diabetes insipidus and hypopitutarism.
Case Presentation: A 25 year old Asian male with bilateral thigh pain and difficulty walking for few days prior to admission. He had a history of drinking 5 to 6 gallons of liquids per day increased urination, hyperphagia,

decreased sexual drive and intermittent headaches. Physical exam was normal except for bitemporal hemianopsia and decreased muscle tone. Laboratory Test: Na 151, high serum osmolality, 325 mosm/kg, urine osmolality 353 mosm/kg but normal urine specific gravity 1.006, blood glucose level when presented was in above 300mg/dl. Thyroid function tests showed 'normal' TSH 2.33miu/ml, low Total T4 2.31mcg/dl [6.09-12.2mcg/dl] and free T4 0.30ng/dl [0.58-1.64ng/dl] suggesting central hypothyroidism. AM cortisol levels showed low normal results 0.7Ug/dl [0.7-22.4Ug/dl]. CAT scan of the head revealed a suprasellar mass. MRI revealed also compression of the optic chiasm. Patient was treated with Hydrocortisone, Synthroid, DDAVP and insulin. This resulted in significant improvement of his symptoms. He had elevated serum and CSF beta HCG and normal Alpha feto-protein [AFP]. Transphenoidal biopsy of the suprasellar mass was diagnostic for Seminoma and chemotherapy followed by radiation treatment was adviced.

Discussion: Our patient characteristically presented with symptoms of anterior and posterior pituitary hormone deficiency but had normal urine specific gravity. The patient also had new onset Diabetes mellitus with glucosuria. This explains the normal urine specific gravity seen in the patient, despite having Diabetes Insipidus. The diagnosis of Diabetes Insipidus could have been missed due to normal urine specific gravity. There is no known association between development of new onset DM and germ cell tumors. Radiation therapy is the main treatment for this type of tumor although recently chemotherapy is being integrated in the management of such cases.

Conclusion: The clinician should be aware that normal urine specific gravity does not rule out Diabetes Insipidus. In patients with a strong clinical suspicion for Diabetes Insipidus, confounding explanations for having a normal urine specific gravity should be sought.

REPRODUCTIVE ENDOCRINOLOGY

Abstract #1000

AN UNUSAL CASE OF PURE XY GONADAL DYSGENESIS

Jennifer Cheng, DO, Janice Gilden, Theresa Kepic

Objective: To describe an unusual presentation of pure XY gonadal dysgenesis.
Case Presentation: A 39 year old Asian phenotypical female (with male partner) presenting with past medical history of primary amenorrhea, is referred for hot flashes and early menopause. She was born in Japan, where at age 16, was treated with "hormonal therapy" to induce pubertal development and menses. She continued this treatment with "regular menses", until coming to U.S. at age 28, when therapy was changed. By age 30, she began to have menses every other month and progressively prolonged intervals , until complete amenorrhea at age 38. For the last 5 years, she has been taking Yaz™(drospirenone/ ethinyl estradiol). For the past 6 months, she has hot flashes (one time per week). There is no desire for pregnancy. Family history-identical twin sister living in Japan, known to have had primary amenorrhea, requiring "hormone replacement therapy" for inducing puberty and menses, and osteoporosis/fragility fractures of wrist and spine. A half sister (same father), with 2 children, is not known to have medical problems. Mother had puberty at "normal age" and had hysterectomy for fibroids. Physical Examination: obese tall female with stocky upper body muscular development, normal female external genitalia (no cliteromegaly, narrow introitus and vaginal vault approximately 4 cm in length) , breasts -Tanner stage IV, and sparse axillary and pubic hair. Laboratory data: Yaz ™therapy: Progesterone <0.5 ng/mL, Free Testosterone 0.9 pg/mL(0.1- 6.4), Testosterone 18 ng/dl (14-76), DHEA S 154 mcg/dL , 17OH Progesterone 10 ng/dl (0-10), Estradiol <11.8 pg/mL , SHBG 106 nmol/L(18 -144), LH 16 mIU/ml(1 -9), FSH 30 mIU/ml(1-18) Prolactin 7 ng/mL (2-18). FISH for SRY gene: positive, Karotype: 46 XY no mosaicism. Imaging: Ultrasound-small uterus without masses , no visualization of ovaries; MRI-small uterus, no ovaries ; Bone Mineral Density on Dexa scan-femoral osteopenia with T score of -1.4.
Discussion: This case represents pure 46 XY gonadal dysgenesis associated with female internal genital tract and bilateral streak gonads in a phenotypical female, also known as Swyer's syndrome.
Conclusion: This case also highlights the need to perform a detailed history in patients referred for primary amenorrhea and/or early menopause. Karotype should be performed. They should be evaluated for osteopenia /osteoporosis, since there are no endogenous androgens or estrogens. Imaging is required to determine whether there are gonadal streaks, which require surgical removal, and carry a 20-30% chance of dysgerminoma. Psychological counseling is recommended. Supported by FHCC.

Abstract #1001

VIRILIZATION DURING AN IVF PREGNANCY AND DELIVERY OF FEMALE TWINS WITH AMBIGUOUS GENITALIA

Yousef Altowaireb, MD, Kamal Shoukri

Case Presentation: 40-year-old Caucasian female reported a gradual deepening in her voice, acne and facial hair growth starting at 16 weeks of gestation. The pregnancy was conceived following in vitro fertilization (IVF) and was complicated by pre-eclampsia requiring C-section at 33 weeks of gestation. The newborn twins had ambiguous genitalia and were confirmed to be genetically females on Karyotype. There was no history of virilization, hirsutism or systemic steroid use prior to pregnancy. Her biochemical workup showed a significantly elevated total testosterone level one day prior to delivery (1981; normal 10-55 ng/dl), a 20-fold elevation in free testosterone level (20.46; normal 0.07-0.99 ng/dl), an inappropriately low normal sex hormone binding globulin (SHBG) level (42.2; normal 18-144 nmol/l) and a declining estradiol level (1291 pg/ml in 1st trimester; 206 pg/ml in 3rd trimester). Abdomen and pelvis MRI showed uniformly enlarged ovaries without discrete lesions. There were no adrenal lesions seen. 3 weeks following delivery, she had some improvement in her virilization. Her total and free testosterone levels had normalized to 24 ng/dl and 0.35 ng/dl, respectively. The twins were referred for surgical correction of the ambiguous genitalia.
Discussion: The presentation of our patient is suggestive of luteomas of pregnancy. These lesions represent hyperplastic masses of large lutein cells, which usually regress after delivery. They are usually bilateral and are more frequent in black females. Exogenous gonadotropin use during assisted reproduction may play a role in the pathogenesis of luteomas as human chorionic gonadotropin may either initiate or perpetuate hormone production by these lesions. The incidence of these lesions is greater than what is reported, as most of them are not associated with virilization. This is due to blunting of androgen action by placental aromatization of excess androgen to estrogen, which in turn results in increased hepatic SHBG

production that bind the free circulating androgens. Our patient on the other hand had an inappropriately declining estradiol and low normal SHBG levels. This indicates that she had a defective placental aromatization, which may have resulted from placental insufficiency in the setting of pre-eclampsia.
Conclusion: Our patient is interesting in that her pregnancy was assisted by the use of exogenous gonadotropins, which may have contributed to the development of her luteomas. In addition, her luteomas were clinically obvious, indicating a defect in protective mechanisms against virilization during pregnancy, which was evident in her case.

Abstract #1002

EFFECT OF TIBOLONE AND RALOXIFENE ON SERUM MARKERS OF APOPTOSIS IN HEALTHY POSTMENOPAUSAL WOMEN

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Objective: To investigate the effect of tibolone and raloxifene on serum markers of apoptosis.
Methods: Design: Randomized, open-label, prospective study. Setting: University Menopause Clinic. Patients: Ninety-nine healthy, naturally menopaused women. Interventions: Patients were randomized to tibolone (n=30), raloxifene (n=29) or no treatment (n=30) for 6 months. Main outcome measures: Serum soluble Fas (sFas), soluble Fas Ligand (sFasL) and cytochrome-c (cyt-c) at baseline and at 6 months.
Results: Serum sFasL decreased significantly in women receiving tibolone (baseline: 53.8 ± 28.3 pg/mL, 6 months: 40.45 ± 19.2 pg/mL, p=0.001), whilst sFas levels did not significantly change in this group. Serum sFas or sFasL did not change either in the raloxifene or in the control group. Serum cyt-c concentrations were under the detection limit of the assay in all women assessed.
Discussion: Intensive investigations have focused on the identification of novel, easily accessible serum biomarkers, which may aid in the diagnosis and follow-up of diseases whose pathophysiology involves apoptosis. In our study, tibolone use resulted in a significant decrease in serum sFasL, but not in serum sFas. Tibolone's hydroxy-metabolites exert estrogenic effects on bone, brain and urogenital system, whilst its Δ-4- metabolite exerts progestogenic action on endometrium and androgenic action on brain, thus inhibiting estrogenic stimulation of the endometrium and increasing libido. It could be suggested that tibolone decreases sFasL levels via its estrogenic action on downregulation of FasL, however we can not specify the cell type it comes from, as soluble FasL lacks tissue-specificity. Raloxifene had no effect on either sFas or sFasL. Finally, cyt-c levels were undetectable in our study. It is not clear if this result suggests that mitochondrial apoptosis was not activated at baseline and therefore none of the investigated regimens exerted an effect on mitochondrial apoptosis, or if cyt-c binding proteins have possibly interfered with the ELISA, thus resulting in misleading results.
Conclusion: Tibolone use resulted in a significant decrease in serum sFasL, but not in serum sFas. These results may indicate that tibolone use is associated with a decrease in receptor - mediated apoptosis. Further studies are necessary to elucidate the effect of tibolone and raloxifene on apoptotic products.

Abstract #1003

PATIENTS' PERCEPTION FOR CARDIOVASCULAR RISK FACTORS IN PCOS AND/OR OBESITY

Zdravko Kamenov, MD, PhD, DMedSc, Antoaneta Gateva

Objective: The aim of the present study is to investigate the perception for the presence of cardiovascular risk factors in Bulgarian patients with polycystic ovarian syndrome (PCOS) and/or obesity.
Methods: One hundred women (30 obese, 50 lean PCOS and 20 obese PCOS) aged 18-45 years were included in the study. They were asked to fill a questionnaire, containing questions about common and popular cardiovascular risk factors like obesity, arterial hypertension, dyslipidemia, diabetes, stress. Then their answers were compared to the results form the anthropometric measurements and laboratory tests, performed during the hospital stay.
Results: Almost all of the patients with body mass index ≥30 kg/m2 stated that they have obesity and do not find their weight appropriate. Only 33,3% of the obese PCOS patients however were able to point their present weight correctly, compared to 59,3% in obese and 67,3% in lean PCOS patients group. 56,7% of obese and 65% of obese PCOS patients stated that they are treated for obesity (diet, physical activity or drugs). 59,3% of obese patients had dyslipidemia without knowing that while the actual rate of dyslipidemia in this group was 70%. The highest matching between the answers of the patients and the test results was observed for arterial hypertension - 82,1%, 96,0% and 84,2% for obese, lean PCOS and obese PCOS groups respectively. The data about the presence of stress at home or at work showed that women with PCOS have generally higher rates of stress compared to obese women without PCOS.

Discussion: PCOS is a common endocrine disturbance in women of reproductive age. It is thought to be linked to increased risk for cardiovascular disease in older age. Any prevention strategy is highly dependent on patient's compliance and motivation for lifestyle modification and treatment. Women with PCOS and/or obesity in our study population have relatively correct perception for the existing cardiovascular risk factors except from dyslipidemia. The reason for the low rate of patients that report their real weight correctly in the obese PCOS group is probably the fact tat they have a higher inclination to hide their real weight or do not follow their weight regularly. The high percentage of unsuspected dyslipidemia is not surprising as these are young women that are not routinely tested for lipid and glucose levels. Because diabetes was an exclusion criterion in the present study, there are no patients reporting elevated blood glucose levels.
Conclusion: Patients with PCOS and/or obesity are well informed about the presence of the common cardiovascular risk factors.

Abstract #1004

THE EFFECT OF DIFFERENT DOSES OF VITAMIN D SUPPLEMENTATION ON INSULIN RESISTANCE DURING PREGNANCY

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Objective: Low serum of vitamin D is correlated with insulin resistance during pregnancy. Data about the appropriate dose of vitamin D for improving insulin resistance is scant. This study was done to assess the effect of different doses of vitamin D on insulin resistance during pregnancy
Methods: A randomized clinical trial was done on 120 women with gestational age less than 12 weeks. These subjects were divided into three groups randomly.Group A received 200 IU vitamin D daily , group B 50,000 IU monthly and group C 50,000 IU vitamin D every 2 weeks from 12 weeks of pregnancy until delivery. The serum level of fasting blood glucose , insulin and 25-hydroxyvitamin D were measured before and after intervention. We used homeostatic model assessment- insulin resistance HOMA-IR as a surrogate measure of the insulin resistance.
Results: The median (interquartile range) of serum 25-hydroxyvitamin D increased in group C from 5.38 (6.5) at the baseline to 33.25 (11.6) ng/ml and in the group B increased from 5.32 (5.4) to 28.7 (11.9) ng/ml, but the level of Vitamin D in the control group increased from 5.24 (6.7) to 19.3 (15.3) ng/ml (P=0.00). Our study showed with supplementation of pregnant women with 50,000 IU every 2 weeks vitamin D, insulin resistance significantly improved. The mean difference of insulin and HOMA-IR before and after intervention between the groups A and C were significant (P=0.00) but these differences were not significant between groups A and B and between groups B and C.
Discussion: Vitamin D deficiency may play a role in gestational diabetes. The appropriate dose of vitamin D supplementation during pregnancy is unknown but it is to be more than 200 - 400 I U daily. Our study showed consumption of 50,000 I U vitamin D orally every 2 weeks for 5 months could raise serum vitamin D above 30 ng/ml in 64% of pregnant women. This study also showed with consumption of 50,000 I U vitamin D every 2 weeks the insulin resistance decreased. Some evidence showed the role of vitamin D in insulin secretion, such as the presence of vitamin D receptor in β cells and the vitamin D binding protein in pancreatic tissue.Vitamin D increases cellular glucose absorption either directly or by increasing insulin sensitivity. Vitamin D may directly or indirectly modify β cell function and secretion by binding its circulating form 1, 25-dihydroxyvitamin D to β cell vitamin D receptors.
Conclusion: Supplementation with 50,000 I U every 2 weeks increased serum Vitamin D above 30 ng /ml and improved insulin resistance during pregnancy.

Abstract #1005

HEART TYPE FATTY ACID BINDING PROTEIN LEVELS IN POLYCYSTIC OVARY SYNDROME PATIENTS

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Objective: Women with polycystic ovary syndrome (PCOS) have higher risk for cardiovascular disease. Heart type fatty acid binding protein (HFABP) has been found to be predictive for myocardial ischemia. We tested whether HFABP is the predictor for cardiovascular diseases in PCOS patients, who have an increased risk of cardiovascular disease, or not.
Methods: This was a prospective, cross sectional controlled study. The study population consisted of 46 PCOS women and 28 control women in reproductive-age. We evaluated anthropometric and metabolic parameters, carotid intima media thickness (CIMT) and HFABP levels in both PCOS patients and control group.
Results: Mean fasting insulin, homeostasis model assessment insulin resistance index (HOMA-IR), triglyceride, total cholesterol, low density lipoprotein cholesterol, free testosterone, total testosterone levels and CIMT measurements were significantly higher in PCOS patients. Although HFABP levels were higher in PCOS

patients, the difference did not reach statistically significant in early age groups.
Discussion: After adjustment for age and body mass index, HFABP level was positively correlated with hsCRP, free testosterone levels, CIMT and HOMA-IR measurements.
Conclusion: HFABP appeared to have an important role in metabolic response and subsequent development of atherosclerosis in insulin resistant, hyperandrogenemic PCOS patients.

Abstract #1006

THE USE OF OVARIAN VEIN SAMPLING TO FIND A RARE OVARIAN TUMOR

Vijayaratna Chockalingam, M.B.B.S, Wendell Malalis, MD, Alan Burshell, MD

Objective: Leydig cell tumors are rare small tumors in the ovary that may secrete both testosterone and estrogen. Because of size, it can be difficult to find on imaging studies, making a diagnostic and therapeutic plan difficult. The following case of hirsuitism illustrates this diagnostic dilemma and provides another diagnostic option before surgical intervention.
Case Presentation: 41 year old female was referred to the endocrinology clinic from her gynecologist for excessive hair growth for about four months. Hair growth is mostly on her chin and she needs to shave once a week. She has also noticed hair on her chest and abdomen. Other concerning symptoms include vague abdominal pain and vaginal spotting. Prior to this time, she was having normal menses. She has no children and wants to get pregnant in the future. Past medical history includes hypertension, GERD, sarcoidosis of the lung, and ovarian cysts. She denies any family history of adrenal, ovarian, uterine, breast, or colon cancer. On physical exam, she is an obese female with acanthosis nigricans with marked terminal hair on the beard and chin area as well as marked excess in the epigastric region and chest. Initial diagnostic studies reveal elevated total testosterone 321ng/dL (normal 8-60) and estradiol 92pg/ml (normal 10-50). Normal dehydroepiandrosterone sulfate (DHEAS), insulin, cortisol, and 17 hydroxy-progesterone. A CT scan of the abdomen and pelvis showed mild fatty change of the liver, but all other organs including the adrenal gland were normal. Transvaginal ultrasound showed multiple uterine fibroids with normal ovaries. With an elevated testosterone and estrogen levels and normal DHEA-S, the excess hormone production was suspected to be from the ovaries. However, radiological evidence was lacking and the patient's wish to preserve fertility limited treatment options. Selective ovarian vein catheterization and hormonal sampling was used to possibly show lateralization of testosterone and estrogen production in hopes to proceed with a unilateral oopherectomy. The results showed lateralization of estradiol and testosterone to the right ovary. Subsequently, she underwent robotic assisted right salpingo-oopherectomy. Final pathological diagnosis was a 1cm leydig cell tumor in the right ovary. Hormone levels normalized and symptoms improved rapidly.
Discussion: Selective ovarian vein catheterization and hormonal sampling (SOVHS) was effective in this challenging diagnostic case.
Conclusion: SOVHS helped localize the tumor and guided a less invasive approach to surgery. Consequently, the patient preserved fertility in hopes to become pregnant.

Abstract #1007

LEVELS OF CIRCULATING OSTEOPROTEGERIN AND SOLUBLE RANKL IN PREGNANCIES COMPLICATED WITH PREECLAMPSIA

Eleni Armeni, M.D., Nikolaos Vitoratos, Demetrios Rizos, Andreas Alexandou, George Creatsas, Irene Lambrinoudaki

Objective: In the present cross-sectional study, we aimed to evaluate alterations in the concentrations of osteoprotegerin (OPG), RANKL and the OPG/RANKL ratio in preeclamptic women during the puerperium, trying to assess the effect of preeclampsia on osteoclastic activity.
Methods: Fifteen pregnant women with severe preeclampsia and 15 matched controls, which remained normotensive throughout their pregnancy but with premature rupture of the membranes, were recruited. Women with chronic hypertension, chronic renal disease, autoimmune disorders, gestational diabetes mellitus or inflammatory conditions, a history of osteoporosis as well as smokers or women on any form of medication pre-pregnancy known to interfere with bone metabolism were excluded from the study. Fasting blood samples were obtained antepartum, immediately after diagnosing preeclampsia (median: 32nd gestational week), and during the 3rd-6th day postpartum, to estimate levels of circulating OPG and RANKL as well as the OPG/RANKL ratio. The anthropometric parameters evaluated included body mass index and blood pressure. Patients with preeclamptic pregnancies were treated with hydralazine hydrochloride and methyldopa.
Results: Mean levels of circulating OPG decreased significantly in both groups after delivery (preeclamptic group: 72.3±49.9 vs 49.7±40.9 ng/ml, p = 0.002; controls: 43.7±19.1 ng/ml vs 22.9±9.1 ng/ml, p =

0.008). The prepartum OPG/RANKL ratio was not significantly elevated in hypertensive pregnancies compared to normotensive pregnancies (2.41±1.72 vs. 1.45±0.63, p = 0.1). The OPG/RANKL ratio decreased in the control group following delivery, while it remained unchanged in the preeclamptic group. Consequently, the postpartum OPG/RANKL ratio was significantly higher in the preeclamptic group compared to control women (1.63±1.40 vs 0.76±0.30, p = 0.01). Levels of RANKL demonstrated no significant alterations during puerperium in both cases.

Discussion: Women who experienced a preeclamptic pregnancy had higher levels of OPG and a higher OPG/RANKL ratio during the whole peripartum period, when compared with controls with the difference becoming significant after delivery. Pregnancies complicated by preeclampsia are associated with a systemic inflammatory process, with cytokines being dominant. On the other hand, proinflammatory cytokines have been demonstrated to enhance the expression of OPG and thus regulating the OPG/RANKL ratio.

Conclusion: Pregnancies complicated with preeclampsia might be associated with lower bone turnover, indicative of a possible positive effect on the pregnancy-related bone loss. The significance of this finding as a predictor of pregnancy-associated bone loss remains to be elucidated in larger studies.

Abstract #1008

THE IRONY OF IRON: CAN MAKE YOU STRONGER, OR CAN LOWER YOUR TESTOSTERONE!

Adam Maghrabi, MD, Hotiana Mateen, John Leidy

Objective: Hereditary hemochromatosis is an autosomal recessive disease characterized by increased absorption of iron from the GI tract. The major gene responsible for most inherited cases of hemochromatosis is the HFE gene (90%), and the two most common mutations associated with hemochromatosis are C282Y and H63D. We are presenting a puzzling case of hypogonadism secondary to hemochromatosis.

Case Presentation: A 63 year old white male with past medical history significant for type 2 diabetes mellitus, pulmonary sarcoidosis (inactive), hypertension and hyperlipidemia presented to his primary care physician complaining of erectile dysfunction. Initial laboratory evaluation showed normal total testosterone. No intervention was done at that time. One year later his erectile dysfunction did not improve. A repeat total testosterone test revealed a low level of 186 ng/dl. He was started on Verdanafil. He was referred to Endocrinology for further evaluation. His prolactin and TSH were normal. His FSH was 2.1mIU/ml and LH was 4.1 mIU/ml which were inappropriately normal given his low testosterone level. IGF-1 (Insulin-like Growth Factor) level was also low. MRI of the brain demonstrated a normal pituitary gland. The patient was started on testosterone replacement. Further questioning revealed a family history of a "sister's blood illness requiring blood transfusions" as he described it. Iron studies revealed high ferritin and Iron levels. DNA testing for hemochromatosis was positive for C282Y double. He was scheduled for regular phlebotomy in addition to family counseling.

Discussion: Excess iron deposition in pituitary cells may lead secondary hypogonadism, the most common endocrine abnormality in hemochromatosis. Decreased libido and impotence in men are among the presenting signs and symptoms of this disorder. The evaluation of hemochromatosis starts with measurement of serum transferrin saturation. Genetic testing is used to confirm the diagnosis of hemochromatosis and also for family screening. Treatment includes scheduled weekly or biweekly phlebotomy until the patient's ferritin level is less than 50 ng/ml, transferrin saturation is less than 50% and/or iron deficiency anemia develops. Maintenance phlebotomy every 2-4 months is recommended.

Conclusion: Hemochromatosis is a common disorder that may cause secondary hypogonadism and erectile dysfunction. It is important to consider hemochromatosis in any patient with signs and symptoms of hypogonadism. Treatment of hemochromatosis can lead to improvement in symptoms of secondary hypogonadism.

Abstract #1009

A REVIEW OF THE ADVERSE PREGNANCY OUTCOMES IN PATIENTS WITH GLUCOSE INTOLERANCE AND DIABETES MELLITUS SEEN IN A NIGERIAN HOSPITAL.

Rosemary Ikem, MD, David Ajani, Ade Adeyemi

Objective: Maternal hyperglycemia is a known risk factor for adverse pregnancy outcomes. The aim of this study is to describe the pattern of the adverse pregnancy outcomes in patient with impaired glucose tolerance (IGT) and DM in our hospital.

Methods: Sixteen patients managed for IGT and DM during pregnancy were reviewed.

Results: The mean age of these subjects was 34.2 ±5.2 years with their mean fasting plasma glucose (FPG) and two hours postprandial plasma glucose (2HPP) results being 8.5 ± 4.1 mmol/L and 14.0±5.9 mmol /L respectively. Majority of the subjects 12 (75.0%) had gestational diabetes (GDM) while the rest had type 2 DM.

Six (37.5%) had preterm delivery and 10 (62.50%) had full term delivery. Delivery was through caeserian section in 2 (33.0%) of the preterm and in 8 (80.0%) of full term babies while delivery was by spontaneous vaginal delivery (SVD) in 4(66.7%) of the preterm and in 2 (20.0%) of the full term babies. Three (18.7%) were of dead fetuses. The birth weight of the babies recorded were, low birth weight in 3 (20.0%) mean birth weight of 2.02±0.41Kg and 5 (33.3%) had high birth weight mean birth weight of 4.3±0.2Kg. Associated complications in mothers include; Gestational hypertension, Preeclampsia and Eclampsia and, Hyperosmolar hyperglycemic state. There was no case of maternal mortality.

Discussion: The pattern of the adverse outcomes observed in this study showed that babies are more affected than mothers with higher rate of birth weight abnormalities, preterm delivery, perinatal deaths. There is also a high number of caeserian delivery with few hyperglycemic emergency, pre-eclampsia, eclampsia and no maternal death among mothers.

Conclusion: The pattern of adverse pregnancy outcomes reported is an obvious indication of a need for improvement in the existing standard of diabetic care and prevention for women.

Abstract #1010

CHROMOSOMAL ABERRATIONS & HYPERGONADOTROPHIC HYPOGONADISM; CLINICAL HETEROGENEITY AND IMPLICATIONS FOR THE DIAGNOSIS OF ANDROPAUSE: A BRIEF REPORT

Tarik Elhadd, MD, FRCP, Charlotte Wilson, Laura McCreight

Objective: To highlight the clinical heterogeneity related to rare chromosoamal aberrations in adult patients picked up to have hypergondotrophic hypogondism.

Case Presentation: Three male patients were found to have hypergonadotrophic hypogonadism. The first two cases were elderly (aged 74 and 87 years) and were picked up in the process of investigation for a possible endocrinopathy. The first was refered for hyponatraemia; the second was noted to have gross gynaecomastia and features suggestive of hypogonadism during investigation for renal impairment and anaemia. Both were found to have rare unbalanced translocation. The first had 46 XY, t(4; 7) (q21.3 ; p15.3) karyotype, and the second had a male karyotype with one normal X chromosome and an isochrome of the Y chromosome 46,X, i(Y)(p10) resulting in an unbalanced karyotype. The third case was a young male who presented with persistent unilateral gynaecomastia following use of Finasteride (a 5-alpha reductase inhibitor) for scalp hair thinning. Questioning revealed low libido but normal sexual potency, and examination showed small penis and small rather atrophic testicles. He had elevated gonadotrophins with low testosterone for age, and chromosomal analysis showed 46 XX, ish der (x)t(X;Y) (p22.3;p11.3) (SRY+,CEPX+) which on FISH (fluorescence in-situ hyperidization) confirmed unbalanced translocation between the X and Y short arms resulting in a derivative of X chromosome containing the SRY locus at p22.3.

Discussion: These cases highlight that hypergonadotrophic hypogonadism in adult patients may have, in many cases, an underlying chromosomal aberration, which could result in heterogeneous and diverse clinical phenotypes. Vigilance must be exercised when seeing cases with subtle clinical findings and consideration of unusual pathology should be made when assessing cases who would have otherwise been diagnosed as suffering from 'andropause'.

Conclusion: Hypergonadotrophic hypogonadism may have unusual chromosomal aberrations. Furthermore, our report underlines the need for obtaining chromosomal analysis in arguably all cases of hypergonadotrophic hypogonadism picked up in adult patients who would otherwise have been passed as cases of andropause

Abstract #1011

A CASE OF MAYER-ROKITANSKY- KUSTER-HAUSER SYNDROME WITH LATENT AUTOIMMUNE DIABETES OF ADULT.

Ankit Shrivastav, MD, Indira Maisnam, Deep Dutta, Pradip Mukhopadhyay, Satinath Mukhopadhyay, Subhankar Chowdhury

Objective: Mayer-Rokitansky- Kuster- Hauser (MRKH) syndrome is a rare but widely discussed developmental disorder of part or whole of the Mullerian duct. It affects about 1 out of 4500 - 5000 women. MRKH may be isolated (type I) but it is more frequently associated with renal, vertebral, and, to a lesser extent, auditory and cardiac defects (MRKH type II).Its association with endocrine anomalies is exceedingly rare.

Case Presentation: A 20 year old female presented with failure to menstruate in presence of normal secondary sexual characters. She was recently detected to have diabetes. There was no family history of Diabetes or menstrual abnormalities. She was 1.42 m tall and weighed 26 kg. There were no skeletal or spinal abnormalities. On examination, pubic hair was P 2 and breasts were B 4 (Tanner). There was no clitoromegaly and external genetalia was unambiguously female. LH and FSH were 1.16 and 5.83 respectively. Karyotype was 46 XX .Ultrasound showed the absence of uterus and vagina

with normal bilateral ovaries. MRI confirmed the same leading to diagnosis of MRKH syndrome. There was no renal anomaly on imaging and renal function was normal. Spinal X-rays and Audiometry were normal. Fasting Serum C peptide was 2.02 ng/ml. Anti GAD 65 Antibody was positive and Anti IA2b was negative leading to presumptive diagnosis of Latent Autoimmune Diabetes of Adults. MODY 5 was ruled out due to absent family history, normal pancreas on USG and presence of Autoantibody positivity. Genetic analysis for MODY turned out to be negative

Discussion: MRKH syndrome associated with Diabetes (MODY 5) has been reported earlier. To the best of our knowledge, this is first reported case of MRKH syndrome with LADA. In patients of MRKH with MODY type Diabetes, mutations within the hepatocyte nuclear factor (HNF)-1β gene were suspected to account for both Diabetes and MRKH-like phenotype. This case highlights the fact that MRKH may be associated with autoimmune diabetes also making the HNF-1 mutation unlikely to be the only cause of diabetes in patients with MRKH syndrome.

Conclusion: To the best of our knowledge, this is first reported case of MRKH syndrome with LADA.

Abstract #1012

SECONDARY AMENORRHEA IN A PATIENT WITH THALASSEMIA MAJOR: THE EFFECT OF IRON OVERLOAD ON THE PITUITARY GLAND

Huiling Liew, MBBS, MRCP, MMED, Rinkoo Dalan, MBBS, MRCP, FRCP (Edin), FAMS (Endocrinology)

Objective: To describe a case of pituitary haemosiderosis secondary to multiple blood transfusions for thalassemia major.

Case Presentation: A 27-year-old Malay lady, diagnosed to have thalassemia major when she was 2 years old, had received multiple transfusions throughout her lifetime. She had splenectomy and cholecystectomy 5 years ago. She presented to the endocrinology service for secondary amenorrhea for 2 years. Her menarche started at age 12 years and her menstrual cycle had been regular until 5 years ago when she had many missed periods with complete amenorrhea for 2 years. On examination, she was 154 cm tall (achieved mid parental height). There was marked generalized hyperpigmentation. Her secondary sexual characteristics were fully developed (Tanner stage 5). Biochemical investigations: Luteinising hormone, LH: <2 IU/L; Follicular stimulating hormone, FSH <2IU/L; Estradiol <37 pmol/L; and Insulin-growth factor 1, IGF-1 :38 ug/L (RI: 117-329) were consistent with hypogonadotrophic hypogonadism and low IGF-1 concentrations. The cortisol concentrations and thyroid function test were normal. A magnetic resonance imaging (MRI) of her pituitary gland showed markedly hypointense anterior pituitary gland on both T1 and T2 weighted images characteristic of pituitary hemosiderosis. Progyluton was started to regulate her menstrual cycle. She also has cardiac and liver hemosiderosis and is on regular iron chelation therapy. Other options of treatment include pulsatile gonadotophin releasing hormone.

Discussion: In cross sectional studies, it has been seen that up to 24% of patients with thalassemia may have pituitary hemosiderosis. The characteristic feature on MRI is the reduced signal intensity of the pituitary gland in both T1 and T2 weighted image. The degree of signal intensity reduction correlates with the severity of pituitary dysfunction and the severity of iron overload. It has been observed that excess iron deposition affects pituitary gonadotroph cells selectively and the excess iron is preferentially distributed in these cells. Findings of a histopathologic study showed excess iron deposition causes degranulation of the adenohypophyseal cells and decreased hormone storage within such cells. The IGF-1 levels may be low because of decreased conversion of GH to IGF-1 as a result of liver hemosiderosis or because of growth hormone (GH) secretory defects in the pituitary gland. The GH axis is the second common axis to be affected in pituitary gland hemosiderosis.

Conclusion: Physicians should be aware of the common pituitary function abnormalities in patients with thalassemia as clinical suspicion is the key to rapid diagnosis and treatment.

Abstract #1013

A REVIEW OF THE ADVERSE PREGNANCY OUTCOMES IN PATIENTS WITH GLUCOSE INTOLERANCE AND DIABETES MELLITUS SEEN IN A NIGERIAN HOSPITAL.

Rosemary Ikem, MD, David Ajani, Ade Adeyemi

Objective: Maternal hyperglycemia is a known risk factor for adverse pregnancy outcomes. The aim of this study is to describe the pattern of the adverse pregnancy outcomes in patient with impaired glucose tolerance (IGT) and DM in our hospital.

Methods: Patients managed for IGT and DM during pregnancy were reviewed. Types of DM and the presence or absence of various adverse pregnancy outcomes were identified.

Results: The mean age of these subjects was 34.2 ±5.2 years with their mean fasting plasma glucose (FPG) and two hours postprandial plasma glucose (2HPP) results being 8.5 ± 4.1 mmol/L and 14.0±5.9 mmol /L

respectively. Majority of the subjects 12 (75.0%) had gestational diabetes (GDM) while the rest had type 2 DM . Six (37.5%) had preterm delivery and 10 (62.50%) had full term delivery. Delivery was through caeserian section in 2 (33.0%) of the preterm and in 8 (80.0%) of full term babies while delivery was by spontaneous vaginal delivery (SVD) in 4(66.7%) of the preterm and in 2 (20.0%) of the full term babies. Three (18.7%) were of dead fetuses. The birth weight of the babies recorded were, low birth weight in 3 (20.0%) mean birth weight of 2.02±0.41Kg and 5 (33.3%) had high birth weight mean birth weight of 4.3±0.2Kg. Associated complications in mothers include; Gestational hypertension, Preeclampsia and Eclampsia and, Hyperosmolar hyperglycemic state. There was no case of maternal mortality.

Discussion: This study revealed that the most common cause of hyperglycemia in pregnant women is GDM. The study also showed a high level of maternal and perinatal adverse outcome among GDM subjects. The pattern of the adverse outcomes observed in this study showed that babies are more affected than mothers with higher rate of birth weight abnormalities, preterm delivery, perinatal deaths. No maternal death among mothers.

Conclusion: The pattern of adverse pregnancy outcomes reported is an obvious indication of a need for improvement in the existing standard of diabetic care and prevention for women.

Abstract #1014

ANTIFERTILITY EFFECTS OFCARICA PAPAYA BARK EXTRACT ON THE MORPHOLOGY OF ACCESSORY REPRODUCTIVE TISSUES AND EXTRA-GONADAL TESTOSTERONE MILLIEU OF MALE RATS .

Taiwo Kusemiju, PhD, Oshiozokhai Yama

Objective: This research attempts at studying the histological perturbation/ cytomorphologic differences in the major tissues connected with production of semen in rats treated with contraceptive doses of Carica papaya extract and how it relates to testosterone ratio.

Methods: Methods: Sixty mature 6 weeks old male Sprague-Dawley rats randomized into 3 main groups; each further subdivided into 2 groups A and B; 10 rats/ subgroup. Group 1: The control fed distilled water (2-4 ml.day-1). Group 2: administered CP (50 mg .ml-1.kg-1. day-1), while Group 3: treated orally at a higher dose of 100 mg .ml-1.kg-1.day-1. Doses were administered once a day to the rats for 4 and 8 weeks. Body weights (initial and final) were evaluated. At the end of the experimental period, rats were sacrificed. Venous blood obtained for testosterone assay, testes and accessory organs were harvested for microscopic study. Testicular and accesssory organ weights were also determined. A probability of p value less than 0.05 was considered to be significant.

Results: Carica papaya significantly decreased peripheral testosterone concentration in both groups. However, the reduction was more pronounced with the high than the low dose groups in all cases. There was a significant reversal in the testosterone concentration of the rats in the group administered with CP 50 mg .ml-1.kg-1.day-1 than in those treated with CP 100 mg .ml-1.kg-1.day-1, this suggests injury inflicted in the low dose group were nominal. The administration of Sprague-Dawley rats with Carica papaya bark extract for 4 and 8 weeks showed significant alteration in the histology of the testis, prostate, epididymis, and seminal vesicle. There was a dose dependent alteration in the cytoarchitecture of the reproductive organs.This feature supports values from testosterone concentration.

Discussion: Deductively a heavy consumption of Carica papaya bark could cause impaired seminal and prostate function as revealed by histological analysis. The cytoarchitectural perturbation of the accessory sex organs caused by the extract administration is probably the result of the decreased secretory activity supported by histological analysis. This strong inhibitory effect probably results in reduced availability of androgens. Findings from this study have further reiterated our prior qualitative proofs of the bark of Carica papaya as a potential male contraceptive agent and that its effect is reversible when used at a lower dose.

Conclusion: This study has demonstrated a time dependent reversible alterations in the morphologies of testes and accessory organs treated with contraceptive doses of CP. The reversal outcome suggests that it is safe to exploit its contraceptive use.

Abstract #1015

A "HEARTY" COST FOR MUSCLE MASS

Sumi Ittan, MBBS, Joseph Aloi, MD

Case Presentation: 44 yo previously healthy male was admitted with heart failure/hemoptysis. He was using Sustanan, Deca-Durabolin IM injections twice a week for 2 yrs and stopped 2 months ago.Physical exam was remarkable for a muscular built person, JVD, heart: S3/S4 and markedly reduced testicular size.Lab work: RBC: 6.86 M/uL,H/H:16.4/ 56.8,Cholesterol 102,LDL:68,HDL:17,total testosterone 2645 ng/dl (241-827),free testosterone 54.64 ng/dl (5-21) estradiol 80 ng/l (8-43),FSH: <0.100,LH <0.100,somatostatin 95 ng/ mL,SHBG 9.6 ,TSH 2.49,free T4 1.2,PSA: 2.2.ECHO/ cardiac MRI: severe dilation of 4 chambers of the

heart, EF: 19%;LV thrombus. RHC: elevated filling pressures,normal cardiac output.Endomyocardial biopsy: myocyte hypertrophy and myocardial fibrosis which was consistent with but not diagnostic of cardiac non-compaction.CT scan chest: wedge shaped consolidation with central lucencies.Open lung biopsy: pulmonary infarction. He was started on medications for heart failure. Repeat ECHO: improved LV dilatation, LVEF 25% and normal repeat RHC. He is currently listed for cardiac transplant.The hypercoagulable work up was negative. He was anticoagulated with leipuridin due to heparin resistance, which was assumed to be from anabolic steroid abuse, it responded to a heparin challenge 6 months later. Meanwhile the testosterone levels gradually declined and was on replacement to mitigate hormone withdrawal symptoms.

Discussion: Anabolic androgen abuse (AAA) can cause several deleterious outcomes. Cardiac effects include cardiomyopathy with systolic/diastolic dysfunction, which can reversible or persist, occasionally MI or sudden cardiac death are seen. AAA confers an enhanced pro-thrombotic state which can lead to atherosclerosis/ thromboembolic disease, a reversible heparin resistance as seen in this case. These haemostatic effects are likely secondary to estrogen from testosterone aromatization. Excess exogenous testosterone exerts negative feedback on the hypothalamic-pituitary testicular axis subsequently reducing FSH/LH and intratesticular testosterone conc. This effect may persist for 6 months to 3 yrs or lifelong after cessation. It can also cause reversible effects like adrenal gland suppression, BPH with elevated PSA. AAA can increase LDL by >20%,decrease HDL by 20%-70% which is generally reversible. Our patient exemplifies the pro-thrombotic effects of supra-physiologic androgens. Currently, his heart failure is improving though his prognosis remains guarded.

Conclusion: Anabolic steroids are generally abused to enhance muscle mass.It has its own share of adverse effects; some are reversible while some lead to major health impairments.

Abstract #1016

POLYCYSTIC OVARIAN SYNDROME AND FUNCTIONAL HYPERANDROGENISM

Adedayo Adegite, MD,

Objective: Polycystic ovarian syndrome (PCOS) has been associated with functional hyperrandrogenism. This may result from generalized (ovarian and adrenal) dysregulation of steroid biosynthesis and metabolism. We describe two cases of PCOS associated with functional adrenal hyperandrogenism.

Case Presentation: CASE1 A 22 year old lady with excessive weight gain, hirsutism and severe acne since childhood was referred for endocrine assessment. She also had been oligomenorrhiec for about 6 years. Clinically she was obese(BMI 39) and hirsute. Biochemical assessment showed, fasting insulin 66.3mU/L(2.6-24.9), FBS 5.7mmol/L, Free testosterone 5.1nmol/L(0.2-2.9), Free androgen index(FAI) 21.7%, DHEAS 2.6nmol/L(2.7-9.2), Androstenedione 16.6, 17-OH Progesterone 6.2nmol/L(06-5.5). 17-OH Progesterone level rose to 18.7 from the baseline 60 minutes after ACTH stimulation test but the androgen levels were unchanged. The plasma cortisol and androgen levels were all suppressed following dexamethasone suppression test. Three weeks following administration of estrogen containing pills, the Free testosterone and DHEAS levels were minimally reduced but there was marked reduction in the Androstendione level. Pelvic USS also showed features consistent with polycystic ovarian morphology. CASE 2 A 23 year old lady with primary infertility was referred for endocrine workup. She had developed oligomenorrhea since age 17 and gained 30kg of weight over 3 years prior to presentation. She also complained of hirsutism acne. Clinically, she was obese(BMI 32). Baseline blood revealed, Fasting Insulin 17.4mU/L, FBS 4.5mmol/L, Free testosterone 4.4nmol/L, FAI 16.5%, DHEAS 15.2nmol/L, 17-OH Progesterone 6.7nmol/L .Sixty minutes after ACTH stimulation test, 17-OH Progesterone went up to 17.2, Free testosterone 3.9 and DHEAS 12.2. After three weeks on estrogen containing pills, the androgen levels were unchanged but the levels were suppressed following dexamethasone suppression test.Pelvic USS and CT abdomen were in keeping with polycystic ovarian morphology. Both adrenal glands were of normal size without any focal abnormalities.

Discussion: A primary defect in sex steroid synthesis and metabolism in the ovaries and adrenals may result in exaggeration of androgen secretion among women with PCOS. This phenomenon was suggested in Case 1 where androgens were presumably produced in both ovaries and adrenal however Case 2 would suggest that most of the excess androgens were of adrenal source.

Conclusion: Hyperandrogenism seen in women with PCOS may be of ovarian and adrenal origin and this may assist in management of this condition.

THYROID DISEASE

Abstract #1100

ON-SITE CYTOLOGY QUALITY ASSESSMENT OF THYROID NODULES BY THE ENDOCRINOLOGIST WITHOUT A PATHOLOGIST LOWERS COSTS,SAVES REPEATS AND CAN ADD ADDITIONAL MOLECULAR STUDIES

Richard Guttler, MD, FACP,FACE,ECNU

Objective: The value of on-site quality assessment has been reported in several large studies done by pathologists present during the FNA procedure. 20% inadequate rates were decreased to 1% in a series of 6000 FNA procedures. The cost savings at this institution was $500,000 over 5 years. This report will show how endocrinologist performing on-site QA can improve patient care.
Methods: A Microscope and digital camera is used to assess follicular cell adequacy. The failure to obtain follicular cells on the first pass can help the FNA physician modify the technique. Once cells are seen then the biopsy will be done with the method that worked to obtain cells.The first smear was air dried, and put in 3 solutions in a Kit from Fischer scientific call Dif Quik. Photomicrographs are taken of each QA pass. There is no need to have a pathologist present, as endocrinologists can do QA without a CLIA permit.
Case Presentation: There are several effects seen with the use of QA by the endocrinologists. Less expense by not having a pathologist on-site, and not having to rebiopsy due to inadequate sample. 2. A QA first look slide with obvious papillary thyroid cancer can alert the endocrinologist to do a molecular marker test for BRAF during the actual biopsy from the needle washout of the syringe. The presence of micro-follicle groups can tip offer the potential of a micro-follicular lesion of undetermined significance. A possible indeterminate biopsy result can be helped if a needle washout was obtained and held for molecular markers, and discarded if a definitive diagnosis is made.
Discussion: The value of On-Site QA of thyroid cytology has been studied and does save money,and improves patient care by not asking the patient to return because of inadequate result. Obtaining molecular markers during the first biopsy will save the patient from a rebiopsy to obtain markers later. The morbitity of re-biopsy is traumatic and unnecessary if QA is done by all FNA physicians. When done in the office of an endocrinologist the added expense of having a pathologist present is now unnecessary. However, in order to function as a QA "thyroid cytologist, endocrinologists must learn to use the microscope, the technique of staining the QA slides and interpreting the present or absence of follicular cells. Adequate smears will decrease the possibility that the pathologists will over-disagnose from bloody inadequate material sent to them.
Conclusion: Endocrinologists acting as FNA physicians, should consider learning ON-Site Method. Finally, a coding, and CLA breakthrough allows payment for all the extra work to provide your patients with maximum patient care.

Abstract #1101

ELEVATED THYROID HORMONES WITH NON SUPPRESSED THYROID STIMULATING HORMONE AND ANTERIOR PITUITARY ADENOMA: CAN IT STILL BE RESISTANCE TO THYROID HORMONE SYNDROME (RTH)?

Deepika Pradhan Shrestha, MD, Karen Smith

Objective: RTH is an inherited syndrome characterized by reduced end organ responsiveness to thyroid hormone (TH) resulting from mutation in thyroid hormone receptor gene. Patient usually has family history of RTH, goiter, persistently elevated TH levels and non suppressed TSH. These findings are similar to those found in TSH secreting pituitary adenomas (TSH-omas) which are important diagnosis to exclude especially in the presence of anterior pituitary adenoma. Failure to differentiate these conditions may result in erroneous treatment such as improper thyroid ablation or unnecessary pituitary surgery in RTH patients. We describe a patient with RTH and anterior pituitary microadenoma.
Case Presentation: 41 year old female presented for follow up of abnormal thyroid function. Her recent thyroid function showed elevated TSH of 6.8UIU/ml (0.3 -5 UIU/ml) and FT4 of 3.36 ng/dl (0.5-1.6 ng/dl) and her previous TSH and TH within last 5 years were both consistently elevated. She had a past medical history of goiter at 5 years of age erroneously thought to be Grave's disease for which she underwent partial thyroidectomy and radioactive iodine treatments with the aim to normalize TH. She was started on replacement therapy but was non complaint and lost follow up for years only to present with depression and suicidal thoughts and elevated TSH of 200 UIU/ml and FT4 of 3.96 ng/dl in the emergency. MRI of brain showed anterior pituitary microadenoma measuring 7mmX7mm raising the possibility of TSH-oma. A detailed history revealed family history of goiter in grandmother. She did not have any clinical or laboratory evidence of concomitant hyper secretion of other pituitary hormones (Prolactin 15.6 ng/ml, ACTH 39pg/ml). Further work up showed normal alpha TSH subunit 3.1ng/ml (0.9-3.3 ng /

ml), alpha subunit to TSH molar ratio of <1 (TSH 40.06 UIU/ml) and low level of sex hormone binding globulin of 11nmol/ L (20-130nmol/L). Based on family history and lab work , diagnosis of RTH was made with co existing non functional pituitary adenoma.
Discussion: RTH and TSH-oma are two important differentials in patient with elevated TH levels and nonsuppressed TSH. Pituitary incidentaloma is reported to be as high as 23% in patient with RTH. Even in the presence of pituitary adenoma, a complete work up is must to avoid misdiagnosis and unnecessary pituitary surgery.
Conclusion: A positive family history, non elevated alpha TSH subunit and low alpha subunit to TSH molar ratio (<1) along with negative pituitary work up helps to differentiate RTH from TSH-oma.

Abstract #1102

INVASIVE PULMONARY ASPERGILLOSIS SECONDARY TO METHIMAZOLE-INDUCED NEUTROPENIA

Miguel Pinto, MD, FACE, Claudia Banda, Carlos Seas

Objective: To describe a case of pulmonary aspergillosis in a patient with methimazole-induced neutropenia.
Methods: We abstracted the clinical chart and reviewed the pertinent medical literature.
Case Presentation: A 48-year-old woman was admitted because of fever and sore throat. She was diagnosed with Graves' disease and medicated with methimazole. On admission, her white blood cell count was 1100 per mm3 (110 neutrophils per mm3). She was started with filgastrim. After 10 days of treatment, her leucocyte count rose to 82600 per mm3 (79200 neutrophils per mm3). She continued with fever, cough and dyspnea. A pulmonary CT scan showed a lung cavity on the right upper lobe and scattered nodules in both lungs. Cultures from brochoalveolar lavage were positive for Aspergillus. Amphotericin B was empirically started but was switched to voriconazole upon confirmation of the pathogens. The patient did not respond to therapy and died of multi-organ failure few days after.
Discussion: Invasive pulmonary aspergillosis (IPA) secondary to methimazole-induced neutropenia. Aspergillus spp are ubiquitous filamentous fungi capable of inducing a wide array of clinical syndromes, ranging from allergic conditions to life-threatening presentations. IPA is one of the most severe clinical presentations and it is almost exclusively seen in severely immunosuppressed patients, usually in the setting of profound neutropenia. Major risk factors for IPA in addition to neutropenia are corticosteroid therapy, transplantation, cytotoxic therapy, advanced AIDS, and chronic granulomatous disease. The risk of developing IPA is strongly associated with the duration and degree of neutropenia. Antithyroid drugs are associated with a variety of minor side effects. Side effects associated with methimazole use are dose-related, and occur usually during the first few months of treatment. Agranulocytosis is the most feared side effect of antithyroid drugs, occurring in 0.35% of patients, and it is thought to be immunue mediated.
Conclusion: Invasive pulmonary aspergillosis is a life-threatening condition, it is almost exclusively seen in severely immunosuppressed patients, usually in the setting of profound neutropenia. Methimazole-induced neutropenia was the underlying process that predisposed our patient to pulmonary aspergillosis.

Abstract #1103

THE ROLE OF I-131 RETENTION FOLLOWING REMNANT ABLATION IN DISEASE RECURRENCE IN DIFFERENTIATED THYROID CANCER

Rene Joukhadar, MD, Behrouz Salehian, Ken Chiu

Objective: To study whether increased retention of I-131 after 24 hours from remnant ablation is associated with a higher or lower risk of disease recurrence in differentiated thyroid cancer.
Methods: We retrospectively studied forty patients who received I-131 for differentiated thyroid cancer. The I-131 dose ranged from 100 to 200mCi. Radiation levels where measured at initial exposure and then 24 hours afterwards. A 7 day post treatment scan was done for each patient and demonstrated uptake at least in the thyroid bed. A one year post treatment diagnostic scan was also done for each patient and these scans along with additional data were used to evaluate disease remission versus persistence/ recurrence. The 24 hour post treatment radiation level was measured at 1 meter from each patient in mRad/ hour and was used to compare I-131 retention among patients. An association between I-131 retention and the outcome after 1 year from treatment was investigated.
Results: The radiation levels after 24 hours from treatment with I-131 for all patients were compared with regard to outcome set as either remission or persistence/recurrence of thyroid cancer. Using a two-sample t-test no statistically significant relationship was found correlating radiation level to a particular outcome (p value 0.33). This did not change significantly when adjustment for dose, age and gender was made.
Discussion: Our study shows that differences in I-131 retention post treatment for thyroid cancer is not correlated with a particular outcome. A reasonable explanation for these results is the following: patients who have increased

retention of I-131 may have a higher tumor burden, either locally in the thyroid bed or metastatic elsewhere and thus can have a worse outcome but on the other hand increased retention of I-131 may reflect an increased sodium iodide symporter expression or activity which increases intracellular I-131 and decreases its rapid extracellular elimination from the body and thus exposes cancer cells to more radiation and result in a better outcome.
Conclusion: In patients with differentiated thyroid cancer (average age 50 years, 75 percent of which were females) who received I-131 remnant ablation with doses ranging between 100 and 200mCi (average 158mCi), the degree of I-131 retention after 24 hours of treatment was not a good predictor of outcome after one year from treatment.

Abstract #1104

RECOGNITION FOR THE NEED OF PARENTERAL LEVOTHYROXINE IN SEVERE ORAL MALABSORPTION

Jordan Brodsky, M.D.

Objective: Recognition for the need of parenteral levothyroxine in severe oral malabsorption non-responsive to increasing oral doses.
Methods: Presentation, diagnosis and successful therapy in a case of levothyroxine malabsorption following pancreaticoduodenectomy (Whipple procedure) for pancreatic cancer.
Case Presentation: A 57 year old woman with hypothyroidism and pancreatic cancer status-post pancreaticoduodenectomy was transferred to inpatient care for altered mental status and diarrhea. TSH level was elevated to 8.0 IU/L (normal 0.55-4.78 IU/L) and increased acutely over three days to 51 IU/L. Her medications were reconciled and no evidence of interactions that hinder the absorption of levothyroxine was noted. She was placed on increasing doses of oral levothyroxine over six days to a maximum of 225 mcg daily with persistently low levels of free T4 and T3. Oral levothyroxine was changed to 100 mcg parenteral levothyroxine, with a rapid TSH drop to normal two days later. After her diarrhea resolved she was restarted on oral levothyroxine. Two months later her TSH was again elevated to 69.79 IU/L with a free T4 of 0.3 ng/dl (normal 0.7-1.7 ng/dl). Levothyroxine dose was increased to 150 mcg daily. A few weeks later TSH had risen further to 95.88 IU/L with a free T4 0.5 ng/L. She was restarted on 80 mcg parenteral levothyroxine and TSH once again returned to normal.
Discussion: Levothyroxine is one of the most prescribed medications in the United States and is indicated for treatment of hypothyroidism. Elevated TSH levels are a common clinical problem. Malabsorption of levothyroxine may be suspected in patients for whom levothyroxine dose requirements far exceed typical doses. The precise mechanisms of levothyroxine absorption in the gastrointestinal track are unknown. Studies have shown that hormone absorption occurs throughout the intestine; duodenum 15%, upper jejunoileum 29%, and lower jejunoileum 24%. Clinicians need to be aware of the potential for levothyroxine malabsorption while understanding the importance of the integrity of the bowel wall to the absorption process and be prepared to adjust therapy appropriately. Further diagnostic testing and workup may be appropriate to yield evidence of gastrointestinal disease or drug interaction.
Conclusion: Altered absorption of orally administered levothyroxine may affect the efficacy of therapy, and increase the risks of complications related to inadequate or excessive dosing. If malabsorption is documented and increased doses of levothyroxine are needed and are unsuccessful at normalizing TSH, parenteral levothyroxine should be considered.

Abstract #1105

FAMILIAL DYSALBUMINEMIC HYPERTHYROXINEMIA- A RARE CAUSE OF EUTHYROID HYPERTHYROXINEMIA

Shalini Dabbadi Lakshmipathi

Objective: What is Euthyroid Hyperthyroxinemia? Recognize Familial Dysalbuminemic hyperthyroxinemia as a rare cause of euthyroid hyperthyroxinemia.
Case Presentation: A 66-year-old Hispanic gentleman, who underwent craniotomy in 1960 secondary to the gunshot wound, was referred to us for abnormal thyroid function tests. The patient had routine screening in November 2010, when he was found to have elevated total T4 levels.He was started on methimazole 5 mg/day by his primary care physician.However, because of the persistently elevated total T4 levels, patient was started on propylthiouracil and methimazole was discontinued. In May 2011, he developed generalized desquamating rash and was hospitalized for Stevens-Johnson syndrome. The rash was thought to be a drug reaction from propylthiouracil and it was discontinued. He was then referred to outpatient clinic. When we evaluated him in July 2011, he denied any symptoms of thyroid disease, and was clinically euthyroid. There was no exophthalmos or thyroid enlargement. He denied any family history of thyroid disease. We performed extensive work up for hyperthyroxinemia. All labs including ,TSH, free T4, total T3, free T3, prolactin, thyroglobulin, TBG, anti T4 antibody, Alpha subunit of growth hormone were within normal limits. We concluded that Familial dysalbuminemic hyperthyroxinemia was the most likely cause of his hyperthyroxinemia which requires no treatment.

Discussion: Euthyroid hyperthyroxinemia can be due to Familial Dysalbuminemic Hyperthyroxinemia (FDH), thyroid binding globulin excess, thyroid hormone resistance, acute non thyroidal illness, TSH producing pituitary adenoma. In addition to the laboratory finding of elevated serum thyroxine levels, many of these syndromes are also accompanied by abnormalities in triiodothyronine and free thyroid hormone levels, as well as unresponsiveness of thyroid-stimulating hormone to thyrotropin-releasing hormone, all of which further erroneously indicate a diagnosis of thyrotoxicosis. FDH is a very rare genetic, autosomal dominant disorder, occurring mostly in Hispanics. It is due to the production of mutant albumin molecule that have low affinity and high capacity for T4 , but not T3.They usually present with high serum total T4, and normal free T4, total and free T3 and TSH . Diagnosis is mainly by excluding other causes of hyperthyroxinemia, however serum electrophoresis, measuring T4 in relatives and gene testing can be confirmatory.
Conclusion: Typically, FDH is detected incidentally. But unfortunately it may be confused with hyperthyroidism or thyroid hormone resistance syndromes, prompting repeated unnecessary laboratory testing and inappropriate treatment such as in our patient.

Abstract #1106

FINE NEEDLE ASPIRATION BIOPSY AND FINAL OPERATIVE HISTOPATHOLOGY: A THREE-YEAR REVIEW OF UTILIZATION OF THE BETHESDA CLASSIFICATION SYSTEM

Amanda Laird, MD, Xin Jing, Paul Gauger, MD, Barbra Miller, MD, Gerard Doherty, MD

Objective: Fine needle aspiration biopsy (FNAB) is essential in the diagnostic workup of thyroid nodules. Results are used to inform recommendations for observation or operative management. Our hypothesis is that utilization of the Bethesda system for thyroid nodule FNAB classification as a guideline would increase the likelihood of malignancy in surgically treated patients.
Methods: Records of patients undergoing hemithyroidectomy or total thyroidectomy at a single institution from April 2008 to May 2011 were reviewed. Clinical data, FNAB results, and final operative histopathology are compared. All patients had original or second-opinion FNAB interpretation by a dedicated cytopathology group. A single pathologist re-reviewed FNAB data to categorize results according to the Bethesda classification.
Results: Four hundred twenty-eight patients had operation for 543 biopsied nodules. The population included 69 men, 359 women, and had a median age of 53.7y (10.9-90.7). Median nodule size by ultrasound was 2.3cm (0.4-10.0). FNAB results were re-categorized according to the Bethesda system; these categories were used in 97% of original readings. FNAB results included 114 (21%) malignant, 147 (27%) benign, 132 (24%) atypia of undetermined significance/follicular lesions of undetermined significance (AUS/FLUS), 54 (10%) follicular neoplasms (FN), 38 (7%) suspicious for malignancy, and 58 (11%) nondiagnostic aspirations. The rates of malignancy of nodules selected for operation were 15% of AUS/FLUS, 35% of FN, and 61% of suspicious for malignancy. 10% of benign FNAB, 9% of nondiagnostic FNAB, and 97% of malignant FNAB were malignant on final histopathology. The overall rate of malignancy for patients selected for operation was 35%.
Discussion: The rates of malignancy for patients selected for operation within the subcategories of indeterminate nodules and for benign nodules are higher than expected in this surgical series. FNAB helps to clarify the decision for operation in patients with thyroid nodules.
Conclusion: FNAB helps to clarify the decision for operation in patients with thyroid nodules. Additional clinical features affecting this decision likely increase the rate of malignancy within each category compared to the standard risk estimates and should be considered in clinical care.

Abstract #1107

LACK OF SIGNIFICANT INCREASED RATE OF THYROID CANCER DETECTION IN UNIVERSITY OF PISA HOSPITAL DEPARTMENTS FROM 1980 - 2010.

Angelo Carpi, MD, Giuseppe Rossi, Rosanna Romani, Andrea Nicolini, Jeffrey Mechanick, MD, FACP

Objective: The number of newly discovered thyroid nodules (TN) and incidence of thyroid cancer are reportedly increasing (1). These results have been generalized into a conceptualized framework that the incidence of thyroid cancer is increasing, due to underlying pathogenetic mechanisms, risk factors, detection thresholds, or any combination thereof. To evaluate this concept locally, we conducted a retrospective long term monitoring of the proportion of thyroid cancer within all the clinically detected TN in two University of Pisa Hospital Departments.
Methods: All preoperative cytological and postoperative histological data for palpable and nonpalpable TN were reviewed at the Institute of Pathology of the University of Pisa from 1980 to 2010. The data were categorized based on the University Hospital Departments (A or B) and study period (1 [1980-1992]; 2 [1993 - 2010]; 3 [1997 - 2004]).
Results: The proportion of postoperative thyroid carcinoma diagnoses (number of TN patients) are given

as: A1) 2.8% (5403) (2) A2) 3.1% (1568) (3) B3) 3.1% (34266) (4) The proportion of malignant diagnoses in the later period (B3+A2; 3.1%, 95% C.I.=2.9-3.3%) was not significantly higher than that in the earlier period (A1; 2.8%, 95% C.I.= 2.3-3.2%) (Rate of proportions: 1.12, 95% C.I.=0.94-1.32, p=0.201; NS).
Discussion: Within two University of Pisa Hospital Departments , the proportion of the postoperativelly detected thyroid carcinomas among the TN patients remained relatively constant over the last 30 years.
Conclusion: The reported increased incidence of thyroid cancer does not seem to be due to the increased risk of cancer among the TN patients.

Abstract #1108

THE IMPACT OF BENIGN GENE EXPRESSION CLASSIFIER TEST RESULTS ON THE PHYSICIAN DECISION-TO-OPERATE IN PATIENTS WITH THYROID NODULES WITH INDETERMINATE FNA CYTOLOGY

Daniel Duick, MD, FACP, Joshua Klopper, MD, James Diggans, Lyssa Friedman, Giulia Kennedy, Richard Lanman, MD, Jonathan Romanowsky, Bryan McIver, MD, PhD

Objective: Thyroid nodules are common but over 20% of thyroid fine needle aspiration (FNA) cytology results are read as indeterminate. Current guidelines recommend that most patients with indeterminate thyroid nodules undergo thyroidectomy, though most of these nodules ultimately prove benign histologically. The Afirma® Gene Expression Classifier (GEC) (Veracyte, Inc.) can be used pre-operatively to identify benign nodules in these indeterminate thyroid nodules. The cancer risk of a GEC benign result is reported to be similar to an operated benign cytology nodule, so observation or ultrasound follow-up may be recommended in lieu of diagnostic thyroid surgery, which would improve quality of life and may reduce healthcare costs. The physician decision-to-operate in patients with indeterminate cytology and benign GEC thyroid nodules has not been quantitatively assessed in clinical practice.
Methods: A total of 27 physicians at 11 sites with 3 or more benign GEC patient results on 1cm or larger nodules with indeterminate FNA cytology contributed data in this IRB-approved study. Patients with suspicious GEC results were excluded. Demographic data, nodule size and location, decision to operate, surgery type (hemithyroidectomy (HT) or total thyroidectomy (TT)) and reason for recommending surgery were reported.
Results: We analyzed data on 138 patients (158 nodules). Most were female (110, 80%) with median age 56 (range 25-86) and median nodule size 2.0cm (range 1.0-7.0cm). Non-surgical management was recommended for most patients (128, 93%, [88.0%-100%] 95% confidence interval (CI), p-value <0.0001 when compared to the 26% rate previously reported); surgery was recommended for those with large (3, 2%), rapidly growing (3, 2%) and symptomatic (3, 2%) nodules. One patient (1%) elected surgery despite the benign GEC result. Both HT (6, 4%) and TT (4, 3%) were recommended (1 unknown). The rate of ultrasound observation in lieu of surgery (93%) was not significantly different from that previously reported (91%) for cytology benign FNAs (p = 0.55, [87.1%-96.5%] 95% CI).
Discussion: Prior to commercial availability of the GEC, approximately 74% of patients with indeterminate cytology underwent surgery, as did 9% of patients with benign cytology. This multicenter, retrospective analysis found an operative rate in cytology indeterminate/GEC benign thyroid nodules (7%) comparable to that of cytology benign nodules. Cytology indeterminate/GEC benign nodules are resected for reasons similar to cytology benign nodules: large size, recent growth or clinical symptoms.
Conclusion: A benign GEC leads to a 90% reduction in the rate of diagnostic surgeries on cytologically indeterminate thyroid nodules in clinical practice.

Abstract #1109

ESTIMATED PREVALENCE OF HYPOPARATHYROIDISM IN THE UNITED STATES USING A LARGE CLAIMS DATABASE AND DISEASE SEVERITY FROM PRIMARY MARKET RESEARCH

Hjalmar Lagast, MD, Karen Joy, Aimee Ruscio, Julie Powers

Objective: Hypoparathyroidism (HypoPARA) is an endocrine disorder in which the parathyroid glands produce insufficient parathyroid hormone, causing hypocalcemia and hyperphosphatemia. There are limited prevalence estimates of HypoPARA. This study aimed to estimate the number of insured HypoPARA patients in US using two epidemiologic approaches and obtain physician assessment of disease symptoms and severity.
Methods: Prevalence was estimated through diagnoses of HypoPARA in the IMS LifeLink Health Plan Claims Database, with 60 million unique patients, over a 12-month period (Oct 2007-Sept 2008). Incidence was calculated by counting the total number of parathyroidectomy, thyroidectomy, and neck dissection surgeries in the same database. Surgeries resulting in either transient or chronic (>6 months) HypoPARA were also counted to determine proportion of surgeries resulting in HypoPARA. A

physician market research study was conducted to determine percentage of new patients due to non-surgical causes. Incidence was entered into a model to derive estimated prevalence. Physicians also reported on disease symptoms and severity.

Results: Using the prevalence approach, 48,674 patients had a HypoPARA diagnosis and 16,651 had a hypocalcemia diagnosis with concomitant diagnosis codes that reasonably suggest HypoPARA. The prevalence approach yields an estimate of 65,325 insured HypoPARA patients, of which 90% are chronic. The incidence approach yields 117,342 relevant surgeries resulting in 8,901 cases over a 12 month period. Overall, 7.6% of surgeries resulted in HypoPARA (75% transient, 25% chronic). When entered into the model, the estimated prevalence of insured patients is 65,389. The physician market research found that among all thyroidectomies, parathyroidectomies and neck dissections performed in a year, 26% resulted in transient HypoPARA and 5% become chronic. Of the HypoPARA patients treated over the previous 12 months, 75% were due to surgery (total thyroidectomies, 38%; parathyroidectomies, 21%; partial thyroidectomies, 9%; neck dissections, 5%). Other notable causes were idiopathic (7%) or autoimmune related (5%). Physicians categorized their patients as mild (43%), moderate (39%), and severe (18%).Common symptoms were depression (9.3%), numbness (15.8%), muscle cramping (17.8%), tingling (19.1%) and fatigue (19%).

Discussion: The two methods yielded similar epidemiology estimates. The results do not include the uninsured population and HypoPARA may not always be coded correctly in claims.

Conclusion: Two methods yielded similar estimates of the number of insured HypoPARA patients in the US (65,000). When extrapolated to the entire US population, the number of HypoPARA patients is estimated at 78,000.

Abstract #1110

A HITHERTO UNDESCRIBED CASE OF CEREBELLAR ATAXIA AS THE SOLE PRESENTATION OF THYROTOXICOSIS IN A YOUNG MAN. A PLAUSIBLE ASSOCIATION

Tarik Elhadd, MD, FRCP, K. Linton, Caoihme McCoy, Subrata Saha, Roger Holden

Objective: To highlight a possible causal relationship between autoimmune thyrotoxicosis and cerebellar syndrome, a hitherto undescribed association.

Methods: Clinical case description

Case Presentation: A 16 year old keen footballer, presented to the emergency department following an episode of unusual behaviour on the football pitch, during which time he was witnessed to be grossly ataxic, by his team mates. An ambulance was called and the boy was taken to our hospital, where he displayed little recollection of the prior events, however demonstrated marked cerebellar signs on examination but no other neurological deficit. This followed a background of recent referral to the medical outpatient clinic, prompted by a 4 week history of 'unsteadiness.' Investigation was initiated as an outpatient, and showed evidence of biochemical thyrotoxicosis with free T4 AT 37 pmol/l (NRR: 11-27) and TSH <0.003 mU/L. Following admission, full investigations including CT brain scan with contrast, lumbar puncture with CSF examination, MRI and MRA didn't reveal abnormalities. He was initiated on carbimazole 40mg q.d. Thyroid ultrasonography revealed a goitre with increased blood flow and his thyroid antibodies showed positive anti-TPO but negative TSH Receptor Antibodies. EEG did not reveal any abnormalities. His neurological disability resolved completely after his thyroid function normalized.

Discussion: The association of cerebellar syndromes is well described with hypothyroidism, however, it is hitherto undescribed with thyrotoxicosis. The causal relationship is plausible as alternative aetiologies were excluded and normalization of thyroid function with treatment was coupled with complete resolution of the neurological syndrome. It was only intriguing that our patient didn't have any other physical signs of thyroid hormone excess and there was no family history of thyroid dysfunction. The exact pathophysiological mechanisms, however, only remain speculative, and may involve autoimmune and or microvascular perturbation.

Conclusion: Cerebellar syndromes may well be one of presenting feature of thyrotoxicosis and this should be in the list of its differential diagnosis.

Abstract #1111

A RARE CASE OF MYXEDEMA COMA CAUSING REVERSIBLE HIGH-DEGREE ATRIOVENTRICULAR BLOCK

Marc Laufgraben, MD, MBA, FACE, FACP, Michael Schaefer, MD

Objective: Although bradycardia is a common manifestation of myxedema coma, heart block is exceedingly rare. We describe an 81 year-old woman with myxedema and high-degree atrioventicular (AV) block that resolved after 10 days of levothyroxine (LT4) therapy.

Case Presentation: An 81 year- old female with a history of hypothyroidism and type 2 diabetes was found to be barely responsive by family members. Her blood glucose was 44mg/dl; her mental status did not improve after correction of hypoglycemia with glucagon. In the

emergency department she became unresponsive. Her core body temperature was 84.6 degrees F. Her heart rate was 36 and irregularly irregular and her respiratory rate was 10. She had dry, doughy skin and periorbital edema. Her thyroid was difficult to palpate due to body habitus. Venous blood gas confirmed hypercarbic respiratory failure with a PC02 of 126 (nl 41-55 mmHg) and she was emergently intubated. Despite the use of 1mg of Atropine for bradycardia, her blood pressure dropped to 80/50 requiring the initiation of a dopamine infusion. Initial EKGs showed low-voltage atrial fibrillation with high-degree AV block and intermittent accelerated idioventricular rhythm. TSH was 54.57 (nl 0.35-5.5 uIU/ml) and Free T4 0.71 (nl 0.8-1.8 ng/dl). An empiric dose of Hydrocortisone 100mg IV was administered followed by a total of 300mcg IV LT4. On the second hospital day, she was placed on LT4 75mcg IV daily. She continued to require dopamine. A FT4 drawn on hospital day nine was still subnormal at 0.71 ng/dl and an additional bolus of LT4 200mcg IV was administered. The next morning she was successfully weaned from dopamine with no further evidence of heart block or bradycardia. She later expired from respiratory and renal failure.

Discussion: The EKG changes in hypothyroidism are well known, including sinus bradycardia, low voltage, delayed intraventricular conduction, and flattened P or T waves. Surprisingly, there have been few cases described with heart block. The severity of hypothyroidism in these cases ranged from subclininical disease to, as in our patient, frank myxedema. Heart block resolves in nearly all cases with thyroid hormone treatment. A proposed mechanism is direct compression of the conduction system by myocardial edema. Decreased chronotropy and inotropy may also play a role.

Conclusion: Although rarely reported, hypothyroidism can precipitate high-degree AV block that usually reverses with thyroid hormone therapy. While the signs of myxedema coma are easily identified in our case, it is important to consider that even mild forms of hypothyroidism may be a reversible cause of new-onset heart block.

Abstract #1112

MALIGNANT PLEURAL EFFUSION: A RARE COMPLICATION OF PAPILLARY THYROID CANCER

Marc Laufgraben, MD, MBA, FACE, FACP,
Megan Baumgart

Objective: Malignant pleural effusion is a rare complication of papillary thyroid cancer (PTC). We describe a 60 year-old man with a 30-year history of PTC admitted with increasing dyspnea and found to have a malignant pleural effusion with cytological evidence of metastatic thyroid cancer.

Case Presentation: A 60 year-old man with PTC was admitted with increasing dyspnea. He was initially diagnosed in Ghana at age 29 and treated with surgical resection prior to immigration to the United States. He had no further care until he presented at age 54 with a neck mass. He underwent thyroidectomy and modified radical neck dissection followed by 150 mCi of radioactive iodine (RAI). Post-treatment imaging showed foci of disease in the thyroid bed, lungs and liver. He was lost to follow-up until age 58, then returned with metastases to the sternum and clavicle with innumerable lung nodules. He was treated with radiofrequency ablation of bony lesions and 350 mCi RAI. His disease showed minimal response to treatment with disease progression over the next two years. At his present admission, imaging revealed a right-sided pleural effusion in addition to the innumerable pulmonary nodules. One liter of fluid was removed by thoracentesis. Pleural fluid analysis demonstrated an exudative effusion with pH 7.52, LDH 137 IU/L, and protein 4.1 g/dl (with serum protein of 7.4 g/dl). Cytology showed cells staining positive for TTF-1, thyroglobulin and Cytokeratin 19, consistent with metastatic thyroid cancer. Shortly thereafter, the patient expired.

Discussion: Malignant pleural effusions complicate less than 1% of cases of PTC and are associated with a grave prognosis. Mean survival is 13.5 months. There is poor penetration of RAI into pleural fluid which likely contributes to the poor prognosis. Small studies have shown no survival benefit with systemic chemotherapy. Drainage of the pleural fluid provides minimal relief as 90% of patients have recurrent effusions within thirty days, though pleurodesis may have palliative benefit.

Conclusion: PTC complicated by malignant pleural effusion portends a very poor prognosis. To date, no interventions have been shown to improve survival. Treatment should focus on symptomatic relief with consideration of referral to a clinical trial.

Abstract #1113

ISOLATED CENTRAL HYPOTHYROIDISM: AN UNUSUAL PRESENTATION OF PRIMARY EMPTY SELLA SYNDROME

Jagriti Upadhyay, MD, David Lavoie,
Gayatri Kuraganti, Nitin Trivedi, MD

Objective: Primary empty sella (PES) is a common entity, with an estimated prevalence of 5-25% in the general population. Presenting symptoms may include neurologic, ophthalmic, or endocrine dysfunction. Current literature suggests that hypopituitarism may be present in up to 25% of cases of PES. While abnormalities of growth hormone

(GH), follicle stimulating hormone (FSH), luteinizing hormone (LH) and prolactin are commonly reported, involvement of the hypothalamic-pituitary-thyroid axis is very rare and is scantily described in the literature. We report a case of isolated central hypothyroidism associated with PES, which, to our knowledge, has never previously been observed.

Case Presentation: A 45 year old woman presented with fatigue and myalgias of gradual onset. She denied any headaches, rhinorrhea, visual changes, or head trauma. On exam she had stable vital signs and appeared obese with BMI of 40.16. Thyroid exam revealed a normal gland without enlargement or nodules. She had no chemosis, proptosis, or visual abnormalities. Initial lab work showed a deficiency of TSH (<0.03 IU/ml) with normal total T3 (133 ng/dl) and free T4 (1.1 ng/dl). Repeat labs 2 months later showed a persistently low TSH (0.17 uIU/ml) as well as low free T4 (0.55 ng/dl) levels. Radioactive iodine thyroid scan showed normal uptake. Central hypothyroidism was suspected, and an MRI of the brain revealed an empty sella. Only a thin rim of pituitary glandular tissue was seen with a midline stalk. No tumor or other source of mass effect was observed. Other pituitary hormone levels were found to be normal (FSH 3.10mIU/ml, LH 6.80mIU/ml, and prolactin 13.4 ng/mL). Her insulin like growth factor 1 (IGF-1) (147ng/ml) and estradiol (95.79 pg/ml) levels were also normal. A morning cortisol level was within normal limits. The patient was started on thyroid replacement therapy, and 6 months later her symptoms were improved and free T4 was normalized.

Discussion: While hypopituitarism is a known feature of PES, central hypothyroidism is rarely observed. To our knowledge, only two cases of clinical central hypothyroidism resulting from PES have been reported in the literature. In both of these cases, TSH deficiency was associated with defects of other hormones of anterior pituitary axes. Isolated central hypothyroidism has never been reported in PES.

Conclusion: Our case emphasizes the need to further explore the association between PES and central hypothyroidism, as well as its pathogenesis and associated hormonal abnormalities.

Abstract #1114

THYROTOXICOSIS FOLLOWING GAMMA KNIFE PARATHYROIDECTOMY

Jagriti Upadhyay, MD, Sandhya Ventrapragada, Suneel Dhand, Nitin Trivedi, MD

Case Presentation: Radioguided Gamma Knife parathyroidectomy is a preferred surgical intervention for removal of a hyperactive parathyroid gland. This minimally invasive procedure avoids stimulation of the adjacent thyroid gland commonly seen in classical surgery. We report the first case of post parathyroidectomy transient thyrotoxicosis (PTT) in a patient who underwent radioguided Gamma Knife parathyroidectomy. A 70-year-old lady with primary hyperparathyroidism was treated with radioguided Gamma Knife parathyroidectomy for a right superior parathyroid adenoma. There were no post-operative complications. However after three weeks she presented to the hospital with asthenia, fatigue, episodic dizziness, palpitations and a six pound weight loss. She denied any neck pain, diaphoresis, headache, visual changes or fever. She denied exposure to radio contrast agents. She had no personal or family history of thyroid disease. Her past medical history was significant for primary hyperparathyroidism and associated hypercalcemia, osteoporosis and supraventricular tachycardia (SVT). Despite the use of alendronate, bone scan showed a decrease in hip bone density prompting the decision for parathyroidectomy. On examination her skin was dry and warm. Heart rate was 114/minute. . She had significant lid lag but no proptosis, tremors or myxedema. Neck examination was negative for thyromegaly, palpable nodules or tenderness. Lab investigations revealed a TSH of 0.014uU/l, free T4 of 2.1ng/dl, T3-167ng/dl; consistent with increased thyroid activity. Anti-TPO antibodies were absent and thyroglobulin level was normal (45.3ng/ml). Thyroid scan showed low uptake of radioactive iodine, ruling out toxic nodule and Graves' as a cause of thyrotoxicosis. The patient responded well to treatment with a beta blocker. A repeat evaluation of thyroid, 10 days after discharge showed improvement (TSH of 0.05uU/l and free T4 of 1.1ng/dl). One month after discharge, the repeat TSH was normal (1.66uU/l) and patient was symptom free.

Discussion: PTT is usually a self-limiting, asymptomatic condition seen mostly after traditional parathyroidectomy. Cases of symptomatic thyrotoxicosis have been reported after this surgery, but not in patients who undergo Gamma Knife procedure. In the absence of a pre-existing thyroid condition or any thyroid nodules, thyrotoxicosis seen in this patient three weeks later has occurred as an operative complication.

Conclusion: Patients undergoing radioguided Gamma Knife parathyroidectomy should also be advised to look for and report any thyroid symptoms. They should also be considered for thyroid function testing if clinically indicated.

Abstract #1115

INTRAMUSCULAR LEVOTHYROXINE: AN EFFECTIVE ALTERNATIVE FOR TWO PATIENTS WITH PROFOUND LEVOTHYROXINE MALABSOPRTION

Bhavika Bhan, MD, Hammad Hussain, MD, Leland Graves, MD

Objective: To describe two cases of profound levothyroxine (L-T4) malabsorption successfully treated with intramuscular (IM) L-T4.
Case Presentation: A 35-year-old female with Crohn's disease underwent thyroidectomy for multinodular goiter. L-T4 replacement was started at 1.6 mcg/kg/day and escalated to a dose of 600 mcg daily with persistent hypothyroidism. Appropriate adherence to oral administration and avoidance of interfering medications was confirmed. An oral L-T4 absorption study was less than 20% of expected. IM L-T4 was started with an initial dose of 0.3 mcg/kg/day divided twice weekly. This was increased to a final dose of 1.2 mcg/kg/day given as 200 mcg IM twice weekly with normalization of TSH and resolution of clinical hypothyroidism. There were no adverse effects of IM L-T4 recognized. The second case, a 43-year-old female with primary hypothyroidism was initially treated with oral L-T4 with doses escalated to 900 mcg daily. Hospitalization was required for profound hypothyroidism with pericardial effusion and mental status changes. An oral L-T4 absorption study was less than 1% of expected. IM L-T4 was started at a dose of 0.3 mcg/kg/day divided into two injections weekly. This was titrated to a final dose of 1.0 mcg/kg/day given as 200 mcg three times weekly. This resulted in normalization of TSH and resolution of hypothyroid symptoms.
Discussion: Most hypothyroid patients are managed within a fairly narrow dose window of L-T4, averaging 1.6-1.8 mcg/kg/d. Doses exceeding the usual requirements may result from impaired absorption caused by diseases such as inflammatory bowel disease or celiac disease. Absorption may be impaired by medications such as ferrous sulfate, sucralfate, calcium supplements and proton pump inhibitors. Taking L-T4 with food high in fiber, soy protein or coffee may reduce absorption. Lack of adherence always remains a concerning possibility. There have been previously described ultimately unresolved cases of hypothyroidism refractory to oral therapy. Almost all have been described in women, mostly aged 40-50 and with history of papillary thyroid cancer. An improvement in thyroid absorption has been demonstrated with intermittent parenteral L-T4 suggesting that severe hypothyroidism itself may impair absorption, possibly due to edema of the small bowel mucosa.
Conclusion: In patients with hypothyroidism despite large doses of L-T4, thyroid malabsorption should be considered after non-compliance and interfering medications have been excluded. If adequate replacement with oral preparation is ineffective, IM replacement may be an acceptable alternative.

Abstract #1116

PAPILLARY CARCINOMA OF AN OVARIAN TERATOMA DISCOVERED AFTER RADIOIODINE TREATMENT FOR PAPILLARY THYROID CARCINOMA

Brian O'Neill, MD, PhD, Johanna Pallotta, MD

Objective: To present a case of papillary carcinoma of an ovarian teratoma (or struma ovarii) discovered after radioactive iodine treatment in a patient with a history of papillary thyroid carcinoma and a recently rising thyroglobulin level one year after a completion thyroidectomy.
Methods: We describe a 59 year-old woman who presented for radioiodine therapy and was found to have a markedly elevated stimulated thyroglobulin level with significant I-131 uptake in an adnexal mass. Biochemical, radiographic, and pathologic findings are summarized.
Case Presentation: The patient presented with a thyroid nodule and underwent a hemi-thyroidectomy for a 1.2 cm encapsulated papillary thyroid carcinoma, follicular variant. It was recommended that the patient have a completion thyroidectomy and radioactive iodine ablation which she was reluctant to do. After two years, and two acellular biopsies of subcenimeter nodules in the residual lobe, the patient underwent completion thyroidectomy revealing nodular hyperplasia, but no lymph nodes were sampled. The patient was again reluctant to undergo radioactive iodine remnant ablation and her thyroglobulin reached a nadir of 35 six months after completion thyroidectomy, then rose to 57 one year post-surgery with a TSH of 3.7. Upon levothyroxine withdrawal, thyroglobulin was measured at 3490 with a TSH of >100. I-123 whole body scan (WBS) showed increased uptake in the pelvis which was thought to be bladder accumulation. She was treated with 150 mCi of I-131 and post-treatment WBS showed significant uptake in an adnexal mass localized by SPECT. She subsequently underwent resection of this ovarian mass and pathological examination revealed a 2.5 cm papillary thyroid carcinoma arising within a mature cystic teratoma. Post-surgically, her thyroglobulin level was undetectable with a TSH of 3.0 on thyroid hormone replacement.
Discussion: We report a rare case of papillary carcinoma arising in an ovarian teratoma, which was discovered after

radioactive iodine treatment for rising thyroglobulin in a patient with a history of papillary thyroid carcinoma. Although discovery of a benign struma ovarii by radioactive iodine WBS has been described, this is the first case to our knowledge of I-131 WBS revealing an ovarian teratoma containing papillary carcinoma.
Conclusion: Struma ovarii with papillary carcinoma is an unexpected, but possible explanation for elevated thyroglobulin in a post-thyroidectomy patient.

Abstract #1117

HYPOTHYROID MYOPATHY

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Case Presentation: A 64 yo male presented to the ER with complaints of weakness, fatigue, muscle tightness and cramps for 2 weeks. He was found to have a creatine kinase (CK) level of 3587 U/L (35-232). This was thought to be secondary to rosuvastatin, and he discontinued it, and fenofibrate. He again presented one week later with a CK level of 4886 U/L. He presented to the ER 3 days later with the same complaints, but also shortness of breath, swelling of hands, thickness of his tongue, and constipation. CK level was 5563 U/L. His family history was significant for rheumatoid arthritis in his mother and lupus in his sister and daughter. He was admitted to the hospital. His labs on admission were significant for TSH 77.7 mcu/ml (0.35-5.0), free T4 0.2 ng/dl (0.6-1.6), creatinine 2.27 mg/dl (0.4-1.24), and erythrocyte sedimentation rate of 0.5 mm/hr (0-20). Urine color was straw. An extensive panel of rheumatology labs was normal, including myositis panel. Upon questioning, the patient endorsed a history of hypothyroidism, and stated that he was told by a physician to stop his levothyroxine (LT4) 5 months prior. Physical exam was significant for peri-orbital edema, bradycardia, muscle strength 4/5 in bilateral shoulders and thighs, thickened skin on arms and lower legs, and delayed relaxation phase tendon reflexes. There was no thyromegaly or nodularity. His previous dose of LT4 was 150 mcg/day. He was treated with LT4 50 mcg/day for 2 weeks, then titrated to his previous dose. He still complains of fatigue, but has had resolution of his presenting symptoms.
Discussion: Complaints of muscle weakness and myalgias are common in patients with hypothyroidism, though rarely the chief complaint. Occasionally, hypothyroid myopathy is more severe, with marked elevation in CK, and a course that clinically resembles polymyositis. The pathogenesis of hypothyroid myopathy is not understood. Thyroxine, T4, promotes expression of the glucose transporter GLUT-4. Levels of GLUT-4 are reduced in hypothyroidism and reduces glucose disposal in skeletal muscle. Reduced T4 is also associated with impaired mitochondrial oxidative metabolism, and reduced muscle function. Thyroid replacement usually leads to resolution of laboratory abnormalities and symptoms over weeks to months.
Conclusion: The case presented above did not involve rhabdomyolysis, though he did present with acute kidney insufficiency. Severe hypothyroidism causing severe myopathy was missed twice by the ER physician and by the patient's PCP. We want to emphasize that hypothyroidism is a cause of CK elevation. An increased awareness could lead to earlier diagnosis and treatment.

Abstract #1118

A SURVEY ON INITIAL MANAGEMENT OF THYROID NODULES AMONG PRIMARY CARE PROVIDERS AND INTERNAL MEDICINE RESIDENTS

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Objective: The American Association of Clinical Endocrinologist (AACE) and the American Thyroid Association (ATA) revised their guidelines on thyroid nodule management. The study aims to examine the knowledge and practice patterns of primary care providers (PCP) and internal medicine (IM) residents in the initial evaluation of thyroid nodules and to compare the results to the evidence-based guidelines.
Methods: A questionnaire on initial evaluation of thyroid nodule was distributed to primary care providers and IM residents at a community hospital. Data was entered into Microsoft Excel and analyzed using Fisher's exact test.
Results: The total response rate is 70% (47/67) for the PCP (16/34) and residents (31/33). Most responders (96%) will always obtain a TSH. All PCP (100%) and most IM residents (94%) will obtain a TSH and of these, 21% of PCP and 25% of residents will obtain a TSH without any other laboratory work-up. Fifty percent of the physicians (PCP, 75%; resident, 39%) will always obtain a thyroid ultrasound (US) ($p = 0.043$). Most physicians (97%) will refer for a fine-needle aspiration (FNA) biopsy of a nodule >1cm. Many (76%) will refer for US-guided FNA biopsy and a small number (20%) will refer for FNA by palpation. Sixty-two percent of the physicians will not put a euthyroid patient on levothyroxine suppression therapy. Many physicians (48%) are not aware of the AACE and ATA thyroid nodule guidelines. Most physicians (65%) have not read the guidelines.
Discussion: Although many physicians were not aware of the guidelines, and a small number of physicians have read them, many PCP and residents responded in concordance

with the guidelines in obtaining TSH, an ultrasound, performing FNA biopsy, and not providing levothyroxine suppressive therapy in euthyroid patients. No differences were found between the responses of PCP and residents except for obtaining an ultrasound. Limitations include low response rate among PCP and that results are from one community hospital.
Conclusion: Based on this survey, many physicians in this community hospital are not aware of the thyroid nodule management guidelines; however, most responded in agreement with the AACE and ATA guidelines.

Abstract #1119

CLINICAL RESPONSE TO INTRAVENOUS L-THYROXINE IN MYXEDEMA COMA

David Cohen, MD

Objective: Myxedema coma is decompensated hypothyroidism with high mortality. The study aim was to describe the clinical response of patients with myxedema coma given intravenous levothyroxine (LT4).
Methods: An ongoing retrospective chart review identifying over 40 patients at LAC+USC Medical Center in Los Angeles from 2002 through 2011 with myxedema coma treated with IV LT4. Myxedema coma was defined by symptoms and signs of hypothyroidism, altered mentation, low serum FT4 and/or high TSH values.
Case Presentation: Preliminary data includes 20 patients' ages ranged from 38 to 97. Nine patients were male, and nine presented between November and March. The most common precipitating factors were non-compliance with environmental exposure, and infection. Six patients had known hypothyroidism. Average time to diagnosis was 24 hours (12 hrs for those with known hypothyroidism, and 42 hrs for those without). All patients received IV LT4 and survived to hospital discharge. Mean systolic blood pressure (MSBP) 16 hours after treatment (127.9 +/-4.4) was significantly higher than pre-treatment MSBP (118.9 +/-5.4; $p<0.05$). An upward trend in mean arterial blood pressure was observed 16 hours after treatment versus pre-treatment (86 +/- 2.8 vs. 80.6 +/-2.8) as was an upward trend in diastolic blood pressure (68.8 +/-5.5 vs. 62.5 +/-5.5). Mean temperature 48 hours after treatment (98.0 F +/-0.6) was significantly higher than pre-treatment mean temperature (97.0 F +/-0.4; $p<0.05$). Mental status normalized within 48 hours of treatment.
Discussion: Patients showed an early clinical response to IV LT4 with increased MSBP 12-16 hours after treatment. 48 hours after treatment mean temperature increased and the mental status of all patients normalized.
Conclusion: Patients with myxedema coma respond rapidly to IV LT4. The early rise in blood pressure may be due to a direct effect of the administered T4 on the cardiovascular system while the rise in mean temperature and normalization in mental status 48 hours after treatment likely coincided with T3 production from T4 in the brain.

Abstract #1120

A UNIQUE CASE OF AUTOIMMUNE THYROID DISEASE IN A PREGNANT PATIENT

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Objective: Autoimmune (AI) thyroid disease may be seen as a continuum with dynamic TSH receptor antibodies (TSHR Abs). In pregnancy immunity is suppressed and AI hyperthyroidism tends to become quiescent requiring lower doses of antithyroid agents, usually during the second and third trimesters. Conversely, AI hypothyroidism requires upscaling of hormone replacement therapy (HRT) due to pregnancy-related demands. We present a case of AI hypothyroidism that progressed early in pregnancy to AI hyperthyroidism, which persisted for six months postpartum, requiring complete cessation of HRT.
Case Presentation: The patient is a 33 yo female who was diagnosed with hypothyroidism after her first pregnancy and was started on daily levothyroxine (LT4) 100 µg. She remained euthyroid, and was switched to Synthroid 125 µg daily when she became pregnant two years later. In the first trimester, at wk 11, TSH was <0.01 µU/ml (normal 0.35-5 µU/ml), fT4 2.0 ng/dL (normal 0.8-1.5 ng/dL), and Synthroid was decreased to 112 µg. During the second trimester, TSH remained <0.01 µU/ml, with fT4 2.3-1.7 ng/dL, despite HRT decrease to 88 µg. During the last trimester, TSH stayed <0.01 µU/ml, but fT4 normalized on Synthroid 75 µg. As the hyperthyroid picture continued to worsen after delivery, HRT was discontinued at 3 mo postpartum. Between 5-7 mo postpartum, TSH was still suppressed <0.007 µU/ml but fT4 normalized again. Between 7-12 mo postpartum, the patient was clinically euthyroid, with TSH fluctuating from 2.275 to 8.107, and 3.816 µU/ml, respectively. TSHR Abs were 0.98 IU/L (normal < 1.75), while thyroid stimulating immunoglobulins (TSI) were elevated at 289% (normal 1-129%).
Discussion: This is a pregnant patient with known AI hypothyroidism who showed decreasing requirements for LT4 during pregnancy and eventual LT4 discontinuation in postpartum. This may be explained by the presence of stimulating TSHR Abs during pregnancy. Postpartum TSH rose to 8.107 µU/ml and this may be due to coexistence of TSH blocking Abs.
Conclusion: To our knowledge this is the first case of a patient with AI hypothyroidism who developed AI

hyperthyroidism early in pregnancy and maintained it well into postpartum, requiring cessation of HRT. Although stimulating TSHR Abs were not measured during the pregnancy, the early signs of hyperthyroidism and the presence of high titers of TSI in postpartum support our hypothesis that this is a case of coexisting stimulating and blocking TSHR Abs. The fact that the hypothyroidism in postpartum was associated with fluctuating TSH in the absence of treatment, suggests the presence of a continuous changing balance between blocking and stimulating Abs.

Abstract #1121

SIGNET RING CELL FOLLICULAR ADENOMA OF THE THYROID

Rod Marianne Arceo-Mendoza, MD,
Mikhail Signalov

Objective: To present a rare case of Signet Ring Cell Follicular Adenoma of the Thyroid in a 27-year-old male.
Case Presentation: A 27-year-old male presented with a history of Right thyroid lobe nodule diagnosed on physical examination three years ago. He denied any compressive symptomatology. Biochemical evaluation revealed subclinical hypothyroidism and elevated Thyroid Peroxidase antibodies. Thyroid Ultrasound confirmed a 3.4 x 1.8 x 3.4 cm complex nodule occupying the Right thyroid lobe and thyroid tissue changes suggestive of Chronic Lymphocytic Thyroiditis. Ultrasound guided Fine needle aspiration biopsy revealed hypocellular specimen consistent with benign colloid cyst. He subsequently underwent right hemithyroidectomy. Final pathology revealed follicular adenoma with the majority of the follicular cells demonstrating a signet ring like morphology. There was no evidence of capsular or lymphovascular invasion. A panel of immunohistochemical stains confirmed the thyroid origin with positive intracytoplasmic thyroglobulin and TTF-1. CK19, Mucicarmine and Calcitonin were negative. The MIB-1 proliferative index was low (1%). The surrounding thyroid tissue showed Hürthle cell changes associated with lymphoid infiltration. Postoperatively, patient was started on Levothyroxine and remained clinically stable without any evidence of new nodules in the remaining thyroid lobe at one year follow up.
Discussion: Signet ring cell follicular adenoma of the thyroid is a very rare pathological entity. Diagnosis of Signet ring cell in the thyroid usually raises a concern regarding possible metastatic origin of the lesion and requires an additional investigation. However, evidence of Signet ring cell in the thyroid may not represent a significant warning sign in terms of the aggressive behavior as it does in the gastrointestinal tract. Rare cases of Signet ring cell carcinoma have been described based on the presence of capsular or vascular invasion as in the case of Follicular Carcinoma. Immunohistochemical staining for thyroglobulin is a useful marker in distinguishing Signet ring cell of the thyroid origin from metastatic Signet ring cell carcinoma.
Conclusion: Signet ring cell follicular adenoma of the thyroid is a very rare pathological entity with few clinical cases described. Physicians should be aware about this rare thyroid tumor that requires additional immunohistochemical investigation. The etiology and clinical behavior of this lesion will require further investigation and data collection due to limited number of cases reported in the literature.

Abstract #1122

A CASE OF HYPERCALCEMIA IN METASTATIC FOLLICULAR THYROID CANCER

Kavya Chitra Mekala, M.D, Julie Chan

Objective: To report a case of hypercalcemia associated with metastatic follicular thyroid cancer (FTC).
Case Presentation: An 86 year old female presented to PCP's office with complaints of 3 weeks of decreased appetite, generalized weakness and altered taste in mouth. Routine blood work revealed serum Ca 13.6 mg/dL (8.4-10.4), BUN 47 mg/dL (7-23), Cr 3.8 mg/dL (0.5-1.1). Patient was admitted and aggressively fluid resuscitated. Additional labs were remarkable for intact PTH 10 pg/ml (12-88), PTHrP <2.4 pmol/L (0-4), 25OH Vitamin D 68 ng/ml (20-60) and Calcitriol 118 pg/ml (15-75). SPEP and UPEP were negative. ACE level was 62 U/L (9-67). Her PMH was significant for history of FTC diagnosed in 1996 after right thyroid lobectomy for a 3.5 cm nodule. Patient had subsequently undergone partial left lobectomy and I131 radioiodine remnant ablation. She later required tracheostomy for bilateral vocal cord paralysis in 2005. Her previous care had been at outside facilities, and we were unable to obtain records of endocrine follow-up. Thyroglobulin (TG) level came back elevated at 1140 ng/ml (1.3-31.8) with TG antibody <20 IU/ml (0-20). Thyroid USG revealed complex cystic nodules in the thyroid bed as well as bilateral carotid lymphadenopathy. Whole body CT scan confirmed a right neck mass and revealed a large expansile destructive lesion of the right iliac bone. Bone scan showed uptake only in the right iliac bone with no skeletal uptake elsewhere. FNA biopsy of this mass was strongly positive on immunostaining for TG and TTF-1 consistent with metastatic FTC. RhTSH stimulated I123 radioiodine whole body scan showed uptake in iliac mass, no uptake in thyroid bed. Patient was treated with calcitonin for 3 days followed

by renally dosed zoledronic acid with improvement in serum Ca to 9. She also received palliative XRT to the iliac mass. Unfortunately, patient's clinical status deteriorated rapidly and she was made comfort care, dying within 2 weeks of initial presentation.
Discussion: Hypercalcemia reportedly occurs in up to 20-30% of all cancer patients and signifies poor prognosis with about 50% dying within 30 days. In a retrospective analysis of 44 patients with thyroid cancer with bone metastasis, 12 patients had hypercalcemia, and this was a significant prognostic factor for survival time. Hypercalcemia of malignancy can result from local osteolytic (via cytokines), humoral mediated (via PTHrP), ectopic PTH or calcitriol mediated mechanisms. This patient had elevated calcitriol and 25OHD levels suggesting a calcitriol mediated hypercalcemia from metastatic FTC.
Conclusion: This case highlights the significance of hypercalcemia in FTC as a poor prognostic variable.

Abstract #1123

HYPOTHYROIDISM-INDUCED RHABDOMYOLYSIS

Kristine Nicolas, MD, Sabrina Raroque, MD, Joumana Chaiban, MD

Objective: Myopathy in hypothyroidism is common. Symptoms include myalgia, weakness, and cramps. However, rhabdomyolysis secondary to hypothyroidism can also occur. It is usually associated with mild to moderate increase in serum creatinine kinase (CK) levels. Overt rhabdomyolysis has been reported to occur in the presence of additional precipitating factor. We are reporting a rare case of hypothyroidism-induced rhabdomyolysis with no other apparent precipitating factor.
Case Presentation: A 34-year-old male presented to the emergency room with a 3-month history of bilateral lower extremity swelling. He also reported cold intolerance as well as fatigue and pain on both thigh muscles upon climbing stairs. He did not have any other medical conditions or any history of trauma or recent infection and was not on any medications. Physical examination revealed pale skin, nonpitting edema around the ankles and dorsum of both feet. Muscle strength was 5/5 in all major muscle groups. . Otherwise the physical exam was unremarkable. Laboratory studies were remarkable for potassium of 3.4 mmol/L (N: 3.5 - 5.1 mmol/L), CK level of >10,000 U/L (N: 39 - 308 U/L), CK-MB of 118.7 ng/mL (N: 0.5 - 3.6 ng/mL), and serum creatinine of 1.2 mg/dL (N: 0.5 - 1.5 mg/dL). Urinalysis was normal and urine toxicology was negative for any illicit substance. His TSH was elevated at 247.2 uIU/mL (N: 0.358 - 3.74 uIU/mL) with a concomitantly low Free T4 at less than 0.2 ng/dL (N: 0.76 - 1.46 ng/dL).Thyroglobulin antibodies were 27 IU/mL (0 - 40 IU/mL). He was started on intravenous fluid hydration and levothyroxine therapy was initiated with gradual improvement in the creatine kinase levels. He was discharged in a stable condition.
Discussion: The cause of rhabdomyolysis in patients with hypothyroidism is not completely clear. Several theories include abnormalities in glycogenolysis, damage in mitochondrial structure and function, and low ATP turnover in skeletal muscles. These processes cause selective atrophy of Type II muscle fibers. In a few case reports, additional precipitating factors including exercise, statin use, alcohol abuse or renal failure have been identified. Institution of thyroxine therapy leads to normalization of laboratory abnormalities as well as resolution of symptoms. In our patient no additional precipitating factor was identified.
Conclusion: Hypothyroidism, a readily treatable condition, should be considered in patients presenting with rhabdomyolysis.

Abstract #1124

A CASE OF APATHETIC THYROID STORM WITH RESULTANT HYPERTHYROIDISM INDUCED HYPERCALCEMIA

Kristine Parker, MBBS, Aundrea Loftley, Carmina Charles, Kathie Hermayer

Objective: To present a case of apathetic thyroid storm and hyperthyroidism induced hypercalcemia.
Case Presentation: A 63 year old woman with untreated hyperthyroidism presented with 1 month of decreased responsiveness, anorexia, weight loss of 10 lbs, nausea, emesis and weakness. She was in A. Fib at presentation with a pulse of 110. TSH was 0.03 mIU/L (0.55-4.78) and FT4 was 5.12 ng/dL (0.80-1.90). She was transferred to our MICU for further management of thyrotoxocisis. O/E she was unresponsive with occasional eye opening, normal reflexes, dry skin, coarse breath sounds and no exophthalmos or thyromegaly. Vitals included: temp 38.2 C, pulse 98, RR 28 , BP 152/81 and O2 Sat 98%. Admission labs were TSH <0.01, FT4 4.44 ng/dl, FT3 9.1 pg/mL (2.3-4.2), TSI 318 (<110), Ca 11.3 mg/dL (8.4-10.2), corrected Ca 11.94 mg/dL, albumin 3.2 G/dL (3.5-4.8), iPTH 18.2 pg/mL (14.0-72.0), PTHrp <0.2 (<0.2), Phos 3.0 mg/dL (2.4-4.7) , 25-OH vit. D 56 ng/dL (25-80) and creatinine 0.9mg/dL (0.4-1.0). CXR showed left pleural effusion and atelectasis. She was intubated and an NG tube was placed. Thyroid storm was diagnosed and she was started on MMI 20 mg PO q 8 hours (h), hydrocortisone 100 mg IV q8h, SSKI 250mg (5 drops) PO q6h and metoprolol 100mg

PO q12 h. Hypercalcemia was treated with IV fluids and calcitonin 75 units q8h for 2 days. Calcium peaked at 13.28 on day 5. FT4 and FT3 normalized at day 7 and calcium at day 9 and she was given zonedronic acid. LP was negative and she was treated for aspiration pneumonia which likely caused her thyroid storm. At extubation she was verbal, appropriate,followed commands and was discharged on day 25 . At her 3 week follow up she was euthyroid and normocalcemic on MMI 20mg PO BID.

Discussion: Thyroid storm can present in an apathetic manner without the typical features of hyperthyroidism and can also be associated with hypercalcemia in the absence of primary hyperparthyroidsim or malignancy. Early recognition and treatment of thyroid storm is critical to decrease mortality. Factors contributing to hypercalcemia in this setting include increased osteoclast activity, decreased intestinal and renal absorption of calcium and increased differentiation of monocytic precursors to osteoclasts. Symptoms of hypercalcemia and apathetic thyroid storm can overlap causing a diagnostic challenge.

Conclusion: Thyroid storm can present in an apathetic manner and diagnostic criteria are useful in making the diagnosis. While hypercalcemia is known to occur in hyperthyroidism this case highlights that apathetic thyroid storm can also be complicated by hypercalcemia both of which must be diagnosed and treated and the hypercalcemia usually resolves when the patient becomes euthyroid.

Abstract #1125

PREOPERATIVE ULTRASONOGRAPHIC THYROIDITIS HELPS PREDICT THE NEED FOR THYROID HORMONE REPLACEMENT (THR) AFTER THYROID LOBECTOMY

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Objective: Preoperative criteria to predict the need for thyroid hormone replacement (THR) after lobectomy are critical to patient care decision-making. We hypothesized that preoperative ultrasonographic (US) characteristics could help predict the need for THR after thyroid lobectomy.

Methods: Data from patients who underwent thyroid lobectomy between 6/06-6/11, were not taking THR preoperatively, and had at least 1 month follow up were reviewed retrospectively. THR was prescribed at the physician's discretion based on elevated TSH and symptoms of hypothyroidism. The percentages of patients who required THR at 6, 12, and 18 months postoperatively were estimated using the Kaplan Meier method, and univariate and multivariate Cox proportional hazards models evaluated prognostic factors for requiring postoperative THR. Demographic and clinical factors included in modeling were patient gender, age at surgery, race, preoperative TSH, US evidence of thyroiditis, pathological confirmation of thyroiditis, and preoperative thyroid function (hyperthyroid, euthyroid).

Results: We identified 98 consecutive patients who underwent thyroid lobectomy during the study period and did not require THR preoperatively. During follow-up (median = 11.9 months, range 1-53 months), 45 patients (46%) required THR. Time to start of THR ranged from 1 to 33 months following surgery. 22% of patients were taking THR at 6 months postoperatively (95% CI 15.1 to 32.0%), which increased to 46% at 12 months (95% CI 35.6 to 57.3%), and 55% at 18 months (95% CI 43.2 to 67.6%). Univariate analysis indicated that significant prognostic factors for postoperative THR included preoperative TSH level > 2.5 uIU/mL (HR 2.8, 95% CI 1.4 to 5.5, P = 0.004) and pathologically confirmed thyroiditis (HR 2.4, 95% CI 1.3 to 4.3, p = 0.005). Patients with both preoperative TSH > 2.5 and US findings of thyroiditis have a 4.6-fold increased risk of requiring postoperative THR (95% CI 1.7 to 12.2, p = 0.002) compared to patients with TSH < 2.5 and no US evidence of thyroiditis.

Discussion: Preoperative decision-making regarding timing and extent of thyroid surgery is often dependent on whether patients will require THR postoperatively. The long-term follow up, high incidence of pathologic thyroiditis (75%), and analysis of postoperative THR requirement instead of biochemical markers of hypothyroidism may account for the long-term postoperative THR requirement in over half of our patients.

Conclusion: The combined preoperative prognostic factors for requiring THR postoperatively, including TSH > 2.5 and US evidence of thyroiditis (4.6-fold increased risk), can guide preoperative patient counseling and surgical decision-making.

Abstract #1126

A NEW KINDRED WITH RESISTANT THYROID HORMONE SYNDROME: CHALLENGE IN MANAGEMENT

Liliana Garcia, MD, Mariana Garcia Touza, MD, Uzma Khan, MD

Case Presentation: 20 year old male presented at age of 11 with attention deficit disorder (ADD) and hearing impairment. Physical examination revealed palpable goiter. Antropometric measurements were appropriate for age. TSH was 1.31mIU/ml, free T4(FT4) 2.91ng/ml, T3 258 ng/dl and thyroperoxidase antibodies were negative.

At that time the diagnosis was highly suggestive of RTH. At age 15, his mother and 2 other siblings were diagnosed with RTH based on genetic testing. Methimazole was discontinued after the diagnosis was confirmed on him. Currently, his ADD is considered resistant to medications and he continues to have difficulties with social interactions. Mother of the patient was initially diagnosed with Graves's disease; underwent I-131 radioablation therapy and was placed on levothyroxine therapy. She presented to our clinic years later with complaints of palpitatations, anxiety, weight gain and fatigue. TSH was 38mIU/ml and FT4 was 1.79 ng/ml. Mutational analysis revealed a heterozygous mutation located in p.A317T of the THRβ gene. Her current dose of T4 is 88 mcg. In addition, she uses propanolol to control palpitations and anxiety. Two sisters, 12 and 10 years old respectively, were diagnosed also with the disorder. The older is asymptomatic. Her TSH is 4.95mIU/ml, FT4: 3.18ng/ml. The younger sister, who has a small goiter, was recently diagnosed with ADD. Her TSH is 4.49mIU/ml, T4: 22 mcg/dl and FT4 is 3.48 ng/dl. They are not taking medications.

Discussion: It is common to find patients with RTH being misdiagnosed with Grave's disease and treated as such. This issue brings up a discussion about the implications of these interventions. For example, one of our cases received methimazole. The use of this medication in children with this condition may induce hypothyroidism during an essential phase in brain development which is dependent of thyroid hormone. Also ADD which is highly prevalent in this condition can result in a more resistant form of ADD. As a consequence, these children can have a detrimental effect in their neurodevelopment and social interactions. Currently there is not therapy for RHT. The tendency is to let the patient self- compensate. LT4 therapy is indicated in cases that has undergone radioablation or has underline thyroid disease. The aim is to bring the TSH close to normal but many times this is difficult to achieve because the counteractive thyroid effect in the heart and central nervous system.

Conclusion: Resistant Thyroid Hormone Syndrome (RTH) is a rare condition frequently confused with hyperthyroidism. Early diagnosis in suspicious cases is essential in order to reduce the risk of mistreatment.

Abstract #1127

THROMBOCYTOSIS AND THYROID CANCERINOMA

Tadele Desalew, MD

Objective: Thrombocytosis associated with malignant disease has been described in some malignant tumors like; lung cancer, pleural mesothelioma, GI tract carcinoma, lymphoma and acute leukemia; however, thrombocytosis is not commonly seen with thyroid cancer. This is probably the second case associated with thyroid carcinoma.

Case Presentation: A 31 year old man presented with left arm paresis. He was hospitalized and work up for presumptive stroke revealed an acute right sided middle cerebral artery infarct. Carotid Doppler ultrasound (US) done revealed patent carotid vessels with an incidental finding of a left thyroid nodule. Thyroid US showed 1.7x 2.9x 2cm left thyroid nodule. Fine needle aspiration of this nodule revealed follicular thyroid neoplasm. Patient was scheduled for thyroid surgery. Physical Examination: Vital signs remained stable throughout hospitalization. His neck was supple without lymphadenopathy and thyromegaly. Abdomen was soft and no hepatosplenomagaly. Neurologically he was alert and oriented x 3, with left arm weakness, impaired sensation and twitching of the left hand every 10-15 minutes. Labs: Lab work was significant for elevated platelet count of 807,000 mm3, normal hemoglobin, iron, ferritin, and coagulation profile. Thyroid function tests were normal.. Peripheral smear showed normocytic normochromic cells, giant platelets without evidence of schistocytes. Hypercoagulable work up was negative. Bone marrow biopsy revealed myeloid megakaryocytes hyperplasia, but JAK 2, Bcr-ABL and flow cytometery were normal.

Discussion: Thrombocytosis may be caused by either a cytokine-driven (reactive) mechanism; (infection, surgery, trauma, inflammation, iron deficiency anemia or malignancy) or may be the result of growth factor-independent (autonomous) overproduction of platelets by clonal/neoplastic megakaryocytes. The former is more frequent and usually results in the platelet count of >600,000/mm3. Thrombocytosis of the myeloproliferative disorder is the best example of the later. Although vasomotor, hemorrhagic and thrombotic episodes are characteristic of autonomous thombocytosis, they can also occur in patients with reactive thrombocytosis due to other neoplasm. In these patients, platelet production probably occurs in the tumor itself or in bone marrow by proteins produced by the neoplasm called thrombocyte stimulating factor. In our case, other causes for thrombocytosis were ruled out except for the possibility of follicular thyroid cancer.

Conclusion: We suggest that clinicians should be aware of this rare association of thrmbocytosis with thyroid malignancy.

Abstract #1128

A RARE CASE OF ACUTE PARALYSIS PRESENTING IN A YOUNG MALE FROM HYPOKALEMIA INDUCED BY THYROTOXICOSIS

Manoj Mathew, MD, Douglas Clarke

Objective: Episodic motor paralysis present in conditions like hypoglycemia, periodic paralysis from electrolyte abnormalities ,hypervolemia, Todd's paralysis, brainstem stroke, subarachnoid hemorrhage etc. We present a case of paralysis induced by hypokalemia occurring in a young healthy patient associated with a thyrotoxic state.
Case Presentation: 32 yr-old male presented to the ER for acute onset of weakness, severe leg cramping, paralysis, and a resting tremor. Serum potassium was 2.1 mEq/L .A thyroid panel showed hyperthyroidism with elevated T3 and FT4 with an undetectable TSH. T3 = 4.29 ng/dL T4 = 4.96 ng/dL), and TSH < 0.015 mIU/L .An EKG showed U-waves and QT prolongation with tachycardia. The patient denied any evident causes for hypokalemia like recent diarrhea, binge drinking, polyuria, or use of potassium losing medications. He denied any family history of hypokalemia like periodic paralysis, thyroid disease, or kidney disease. The patient was treated with potassium and propranolol. After treatment, all symptoms resolved and potassium returned to normal (4.1mmol/L). The patient followed-up in endocrinology to evaluate for causes of hyperthyroidism. Serum thyroid stimulating immunoglobulins, TSH receptor antibodies, thyroid function tests, thyroid ultrasound, and nuclear thyroid scan/uptake were consistent with Graves' disease. Our diagnosis was Hyperthyroid Periodic Paralysis (HPP)
Discussion: HPP is a rare complication of thyrotoxicosis resulting in severe episodic flaccid paralysis. It typically presents with nocturnal onset of proximal muscles weakness. Hyperthyroid induced hyper-adrenergic states up-regulate Na/K-ATPase activity, causing an intracellular shift of potassium. This results in acute hypokalemia and an associated motor paralysis. Thyroid hormone, epinephrine, and insulin can also up-regulate Na/K-ATPase activity and induce hypokalemia. Precipitating factors include high carbohydrate or alcohol ingestion, strenuous activity, and physical or emotional stress. One must exercise caution with repletion of potassium to avoid rebound hyperkalemia. Treatment of the hyper-adrenergic state is with nonselective beta-blockers .Treatment of the hyperthyroid state is with anti-thyroid drugs thyroid ablation with radioactive iodine or surgery
Conclusion: HPP is a self-limited disorder if a euthyroid state is attained and maintained. It resolves spontaneously as electrolytes return to the extracellular space. Potential complications include cardiac arrhythmias, respiratory failure, and rebound hyperkalemia and hyperphosphatemia. It is important to diagnose and treat this condition appropriately as it can be potentially life threatening, particularly in austere operational environments

Abstract #1129

A CASE OF FALSELY ELEVATED TSH LEVEL DUE TO HETEROPHILE ANTIBODY INTERFERING WITH THYROTROPIN IMMUNOASSAY

Manoj Mathew, MD, Huong Nguyen

Objective: Subclinical hypothyroidism is defined as elevated serum thyrotropin (TSH) and normal serum free thyroxine (FT4) levels, is common clinical in practice. Patients with subclinical hypothyroidism and TSH concentration above 10 mIU/L are usually treated with thyroid hormone supplement. We hereby present a case of heterophile antibodies interfere with the TSH assay, leading to a falsely elevated TSH level and unnecessary thyroid hormone therapy.
Case Presentation: 41 yr-old female presented to her primary care physician for annual physical examination. She reported no symptoms. The thyroid gland was normal. A screening thyroid panel showed serum TSH level 19.6mIU/L and FT4 level 1.46ng/dL). She was diagnosed with subclinical hypothyroidism and started on levothyroxine. Despite of medication TSH level remained elevated. Gradually increased the levothyroxine to 300mcg. She subsequently developed palpitations and tremor and was referred to endocrinology. Repeated lab study showed FT4 2.91ng/dL, FT3 1.7ng/mL and TSH 17.7mIU/L. A Cosyntropin stimulation test showed the peaked cortisol level of 26.6mcg/dL .TSH heterophile antibody was present. We tapered and eventually discontinued the levothyroxine treatment. The symptoms were resolved. Subsequent study showed TSH 22.9mIU/, FT3 1.11ng/mL, and FT4 0.80ng/mL. The TSH dilution test showed serum TSH levels 15.2 mIU/L initially and 5.2 mIU/L after a five-fold dilution. With mouse serum pretreatment, the TSH level was 1.19 mIU/L.
Discussion: The differential diagnosis for elevated serum TSH with normal FT4 and FT3 includes subclinical hypothyroidism, resistance to TSH, TSH-secreting pituitary adenoma, untreated adrenal insufficiency, and interfering substances. This case subclinical hypothyroidism is unlikely given that the TSH level never normalizes. Resistance to thyroid hormone is unlikely given the normal TSH level in 2007 and absence of family history of thyroid disease. A TSH secreting pituitary adenoma is also unlikely given the normal FT4 and FT3 levels and absence of a thyroid goiter. Normal Cosyntropin stimulation makes adrenal insufficiency less likely. The elevated TSH level in this patient

is caused by human anti-mouse monoclonal antibodies (HAMA). The TSH value normalizes when the serum is pretreated with mouse serum. HAMA can interfere with TSH immunoassays, causing falsely elevated TSH values. Many laboratories have HAMA-blocking reagents that can eliminate the interference of these heterophile antibodies. ***Conclusion***: Heterophile antibody interference with TSH immunoassay should be on the differential diagnosis of asymptomatic patients with elevated TSH concentration. Evaluation for this condition may prevent unnecessary thyroid hormone treatment.

Abstract #1130

REGIONAL DIFFERENCES IN STAGE OF PRESENTATION AND SURVIVAL OF INDIVIDUALS WITH THYROID CANCER

Marlon Guerrero, MD, Jessica Rose, Betsy Wertheim

Objective: The incidence of thyroid cancer has been steadily increasing. Several studies have identified differences in the incidence and prognosis of thyroid cancer according to gender and racial/ethnic differences. In this study, we sought to determine whether differences exist in the stage of presentation and survival in patients with thyroid cancer according to geographic distribution in the United States.
Methods: Using the Surveillance Epidemiology and End Results (SEER) database we identified 87,308 individuals diagnosed with thyroid cancer between 1973 and 2008. We then assessed historical stage of diagnosis and cancer-free survival (CFS) according to geographic region and race/ethnicity. Stages of diagnosis were compared using multinomial logistic regression, and survival rates were compared using Cox proportional hazards regression. Multivariate models were adjusted for age, year of diagnosis, cancer type, and registry site.
Results: Of those individuals analyzed, 47,958 (54.9%) were from the West, 15,802 (18.1%) from the East, 13,386 (15.3%) from the Midwest, and 10,162 (11.6%) from the South. Overall, 60% presented with localized disease, 34% with regional metastasis, and 5.6% distant metastasis. Those from the West had a higher risk of presenting with regional and distant metastases compared with the other regions. Individuals with thyroid cancer from the South had lower CFS than those from any other region. There was no significant difference in CFS by geographic region for non-Hispanic white or Hispanic white individuals with thyroid cancer. However, blacks from the West had significantly better CFS than blacks from the South. When double-stratifying by cancer subtype and race/ethnicity, we found no significant associations between geographic region and CFS.
Discussion: This study demonstrates that geographic differences exist in stage of presentation in patients with thyroid cancer. The reason for this is multifactorial, but differences in socioeconomic factors, access to health care, and cultural beliefs are contributing factors. ***Conclusion***: There are differences in the stage of presentation and survival of individuals with thyroid cancer according to geographic region.

Abstract #1131

DIAGNOSIS OF SUBACUTE THYROIDITIES IS REALLY A PROBLEM!

MD Uddin, DEM, MD, M. Hossain, A. M Abdullah, M. Badiuzzaman, R. Haider, M. Hasanat

Objective: Su bacute thyroiditis is an important cause of thyrotoxicosis, often misdiagnosed as Graves' disease and given anti-thyroid drugs. Etiological diagnosis can yield proper guideline of management and a good result.
Methods: A total of 45 cases [36 females, 9 males; age (mean ± SD): 33 ± 4.7 yr) were recruited from Endocrine out-patient department of BSMMU to see the clinical and biochemical profile of subacute thyroiditis.
Results: Most of the patients had a history of sore throat which was less common among elderly ones. Only 7 cases were below age 20 years. Frequency of thyroiditis was higher among young adults. Elderly people presented with less features of thyrotoxicosis; rather they presented more with constitutional symptoms. All patients had painful thyroid gland (100%) with or without dysphagia, palpitation (93%), fever (91%), sweating and heat intolerance (80%) and IBS like symptoms was 46%. Among 36 females, 16 patients (44.4%) presented with oligomenorrhea / amenorrhea. Clinically palpable thyromegaly was present in 33 cases (73%), which was diffuse in 24 patients (73%), resting tachycardia in 35 (78%) while anemia in 23 (51%). A few cases (13) had raised blood pressure. Elevation of thyroid hormone was found in 44 (97.7%), but in most it was mild. Similarly mild reduction of TSH (0.1-0.3 mIU/L) was present in 43 (95%). Characteristically low radio-iodine uptake (<5%) in 2 hrs and 24 hrs was found in 100% and thyroid scan revealed uniform uptake. ESR was raised in 100% cases and leukocytosis was present in (20%).
Discussion: Sub acute thyroiditis is not an uncommon cause of thyrotoxicosis but it is a self limited thyroid disease. It can be diagnosed easily on careful clinical examination & is supported by low radioiodine uptake by thyroid.
Conclusion: Sub acute thyroiditis is a self-limiting disease and does not usually need any anti-thyroid medication. Therefore, subacute thyroiditis should be excluded in thyrotoxic patients before initiating anti-thyroid drugs.

Abstract #1132

THE FREQUENCY AND BIOLOGIC SIGNIFICANCE OF BRAF V600E MUTATIONS IN PATIENTS WITH PAPILLARY THYROID CANCER.

Michael Demeure, MD, MBA, Philip Gafford, MD, Michael Demeure, MD, MBA

Objective: The V600E activating mutation in BRAF mutation has been reported in approximately 45 % of papillary thyroid cancers (PTC). Some studies have suggested that tumors harboring a BRAF mutation are more aggressive cancers. We sought to determine the frequency and biologic significance in our patient population with PTC.
Methods: We conducted a retrospective analysis of all patients who underwent surgery for PTC from February 2009 through August 2011. The dominant tumor mass was tested for the presence of a V600E BRAF mutation by a commercial CLIA certified laboratory. Statistical analysis was done using Fishers' exact test or by unpaired Student's t-test.
Results: We identified 53 patients. BRAF analysis was available for 50 (36 women and 14 men). Of these, 32/50 (64 %) exhibited a V600E mutation in BRAF. There was no association of BRAF mutation with gender, age, tumor size, multicentricity, extrathyroidal extension or likelihood of lymph node metastasis.
Discussion: We found a V600E BRAF mutation in 64% of PTC cases, which is a higher rate than previously reported in other U.S. series. The presence of BRAF mutation is not associated with clinical parameters associated with more advanced tumors including age over 45, male gender, extrathyroidal extension, or lymph node metastases.
Conclusion: Although presence of a BRAF mutation may offer a therapeutic target if PTC recurs, we find no indication that BRAF mutant tumors are more aggressive and therefore no need to alter initial clinical or surgical treatment based on analysis of BRAF.

Abstract #1133

METHIMAZOLE-INDUCED AGRANULOCYTOSIS. A CASE SERIES

Miguel Pinto, MD, FACE, Helard Manrique

Objective: To describe a case series of agranulocytosis due to methimazole in a general hospital from Lima, Peru.
Methods: We abstracted the clinical charts and describe the clinical and laboratory details at presentation.
Case Presentation: We report 30 cases of agranulocytosis (86.6% female, mean age 36.9 years) from the period 2002 to 2008 (frequency of 0.58%). In all cases, methimazole was prescribed because of Graves' disease. Fifth-three percent of cases were medicated with methimazole 30 mg/day, 20% with 40 mg/day, and 16.6% with 60 mg/day. At presentation, 96.7% presented with fever, and 90% with sore throat. The mean time from starting therapy to agranulocytosis was 13 weeks. The mean time to recovery from agranulocytosis was 10 days. All patients were treated with reverse isolation, and broad spectrum antibiotics. In 9/30 (30.4%) lithium was added, and in 12/30 (40%) GM-CSF was necessary due to severe neutropenia. In all cases, radioactive iodine was the definitive treatment for the hyperthyroidism. Four patients (13.3%) died because of septic shock.
Discussion: Agranulocytosis is the most feared side-effect of thionamides use for hyperthyroidism. Its prevalence is 0.35%. Most cases occurred in the 90 days of treatment, but this complication can occur at any moment after starting therapy. Agranulocytosis is thought to be autoimmune-mediated, and anti-neutrophil cytoplasmic antibodies may play a role.
Conclusion: Agranulocytosis is major side effect of thionamides. In our hospital, its prevalence is 0.58%. Most patients recovered without complications. In these patients, radioactive iodine is the treatment of choice.

Abstract #1134

ELEVATED C-REACTIVE PROTEIN IS NOT A GOOD PREDICTOR OF CARDIOVASCULAR RISK IN THYROID DISORDERS.

Olufunmilayo Adeleye, MD, Okeoghene Ogbera, O. Dada, Ayotunde Ale, Femi Abatan, Aramide Adediran

Objective: Elevated C-reactive protein(c-rp) is an indicator of low grade inflammation associated with increased cardiovascular risk across various medical conditions. The significant prevalence of thyroid dysfunction in our population and the often documented associated cardiovascular complications confers the need to determine the association between c-rp and cardiovascular risk in various thyroid disorders.
Methods: A descriptive study involving 171 subjects with the following(ff) thyroid conditions: Graves disease(GD), Primary hypothyroidism, Iatrogenic hypothyroidism ff thyroid surgery (IH ff surg), Iatrogenic hypothyroidism ff Radioiodine(IH ff RAI), Iatrogenic hypothyroidism following thionamides (IH ff thionamide), Non toxic goiter(NTG), subclinical hyperthyroidism, subclinical hypothyroidism, Toxic goiter(TG). Anthropometrics were obtained using standard methods. High sensitivity c-rp (hs-CRP), TSH,T3, T4, fasting lipids and blood glucose were also determined.

Results: The Mean age (SD) of study subjects was 44.7(13.2) and ages ranged from 12 to 75 years. 86% were females and 24% were males. 53% (n=91) had GD, 3.5% (n=6) had primary hypothyroidism, 3.5% (n=6) had IH ff thyroid surgery, 2.9%(n=5)had IH ff RAI, 1.8%(n=3) had IH ff thionamides, 15.2%(n=26) had NTG, 16.4%(n=28) had TG, 2.3%(n=4) had subclinical hypothyroidism, 1.2%(n=2) had subclinical hyperthyroidism. 15 subjects making up 8.7% of the Study population had elevated hs- CRP (13 females, 2 males). Majority of the subjects with elevated hs-CRP (n=9) had GD. 60% of subjects with elevated hs-CRP were overweight/obese. Elevated hs- CRP was also associated with hypertension. A higher proportion of subjects with elevated hs-CRP had hypertension compared with those with normal hs-CRP (p=0.06). Elevated hs-crp did not correlate significantly with dyslipidemia after correcting for duration of treatment with thionamides and lipid lowering agents. SPSS was used for data analysis.
Discussion: A significant proportion of subjects with thyroid disorders in this study were females and GD was most prevalent. Although previous reports have indicated increased CRP values with progressive thyroid failure, the hs-CRP levels did not correlate with hypothyroidism in this study.
Conclusion: C-RP may not be a useful marker of cardiovascular risk in patients with thyroid disorders.

Abstract #1135

INTEGRATIVE ENDOCRINE SURGERY PRACTICE IMPROVES EFFICIENCY IN THE DELIVERY OF ENDOCRINE SURGICAL CARE

R. Harrell, MD, FACP, FACE, ECNU,
David Bimston, MD

Objective: To compare the surgical completion rate in an integrative endocrine surgery practice over the first four months of service consolidation in 2011 with the surgical completion rate for the same endocrinologist and endocrine surgeon team when they were in separate, non-dedicated endocrine and endocrine surgery practices in 2009 and 2010 and over the same summer months (5/1-8/31).
Methods: A database of surgical referral dates and surgical completion dates was interrogated for the 4 month period from 5/1-8/31 for the years 2009, 2010 and 2011. Interrogation was performed on 12/30/2011, so that a full 4 months of time were elapsed between the last referral in 2011 and the analysis. Surgical completion rate was expressed as a ratio of the number of surgeries requested by the endocrinologist/ the number of surgeries on requested patients completed by the endocrine surgeon. Data for 2009, 2010 and 2011 are analyzed.
Results: Over the summer time interval from 5/1-8/31, while the authors were practicing endocrinology and endocrine surgery in separate but physically adjacent practices, their surgical completion rate for patients with thyroid, parathyroid and adrenal masses was 75% in 2009 and 81% in 2010. Over the same time frame in 2011, after joining forces in a single dedicated endocrine surgery practice, the surgical completion rate rose to 94.3%. After consolidation, referral volume over the 4 month period rose by 35%.
Discussion: In May of 2011, the authors formed the Memorial Center for Integrative Endocrine Surgery in Hollywood, Florida. In this paper, we demonstrate that this practice model generates a dramatic improvement in the efficiency of surgical thoughput. In 2009 and 2010, in spite of our adjacent endocrine and endocrine surgery office locations, 25 and 19% of patients, respectively, referred for surgical treatment of endocrine disease did not get the surgery they needed from the preferred surgeon. With the consolidation of all imaging, endocrine and endocrine surgical services under one roof, the loss rate was reduced to 6.7%.
Conclusion: Many endocrinologists and endocrine surgery trained surgeons fear that they would be unable to survive financially in private or hospital-based practices devoted exclusively to endocrine surgery. We believe that this fear is unfounded. Our data suggests that a consolidated imaging/endocrinology/endocrine surgery practice actually improves the throughput of endocrine surgery patients by improving the surgical completion rate. In addition, we have found that in spite of opening our practice in a new location, we have generated more referrals as a dedicated endocrine surgery practice than we ever did as separate practitioners.

Abstract #1136

ACUTE THYROTOXIC PERIODIC PARALYSIS BEYOND THE ASIAN POPULATION: A DIAGNOSTIC CHALLENGE

Reshmi Srinath, MD, Beatrice Hong

Objective: To describe a case of a young Hispanic male presenting with acute thyrotoxic periodic paralysis without signs of hyperthyroidism.
Case Presentation: A 29 year old Hispanic male with no prior medical history presents with diffuse weakness upon waking from sleep at 4 AM. He complains of nausea and shortness of breath. No noted ingestions, illicit substance use, or herbal supplements. He works as a mechanic in an auto body shop outdoors. On examination he is tachycardic with a heart rate 100; but otherwise afebrile, normotensive, and breathing comfortably on room air. He is unable to

transfer or bear weight. Strength is 3/5 bilaterally in his shoulders, triceps and biceps, with 4/5 grip strength. Both hips and quadriceps are noted to have 2/5 strength with 4/5 strength on dorsiflexion and plantarflexion of the feet. Labs show an initial K 1.4, Na 141, glucose 136 with normal calcium and magnesium levels. Creatinine kinase level is >2000. EKG demonstrates sinus tachycardia and prolonged QT interval. He is initiated on intravenous fluids with potassium and magnesium supplementation and transferred to the intensive care unit. TSH is .01 uIU/mL with a free T4 4.18 ng/dL, and free T3 of 8.3 pg/mL. Methimazole and propanolol are administered. Thyroid stimulating immunoglobulin level returns positive with normal sedimentation rate. The patient's weakness resolves over the next twenty four hours with correction of hypokalemia. On discharge 2 days later strength is 5/5 throughout and he is tolerating medications. Repeat TSH is .02 uIU/mL , free T3 9.36 pg/mL with normal potassium and creatinine kinase levels.

Discussion: Acute thyrotoxic periodic paralysis is a rare complication of thyrotoxicosis which initially was felt to predominantly occur in Japanese and Chinese males, but is now being seen in young males of all ethnicities in Westernized countries. Incidence in the U.S is currently unknown, but has been estimated at .1-.2%. Risk factors include recent exertion, stress, alcohol intake and high carbohydrate load. Severity of hypokalemia is due to thyroid hormone, catecholamines,and elevated insulin levels driving Na-K ATPase activity and thus potassium uptake into muscle. While paralysis correlates with the degree of hypokalemia, most patients demonstrate minimal signs of thyrotoxicosis.

Conclusion: Acute onset muscle weakness in a young male of any ethnicity should raise suspicion for thyrotoxic periodic paralysis, even without clinical signs of thyrotoxicosis. Prompt testing of thyroid function and electrolytes should be pursued to direct management.

Abstract #1137

THE PREVALENCE, CLINICAL AND BIOCHEMICAL CHARACTERISTICS OF BONE DISEASE IN HYPERTHYROID PATIENTS

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Anthonia Ogbera, MBBS, FMCP

Objective: The occurrence of bone disease in hyperthyroidism is an understudied and poorly reported aspect of thyrotoxicosis especially in Sub-Saharan Africa. This study is set out to determine the prevalence, possible predictors and characteristics of bone disease in hyperthyroid patients.

Methods: This is a cross-sectional study in which 40 hyperthyroid patients whose ages ranged from 21-49years with active thyrotoxicosis were randomly selected and 20 healthy age and sex matched who met the inclusion and exclusion criteria were recruited. Interviewer-administered questionnaire was administered. Their fasting venous blood and early morning urine were analyzed for biochemical and hormonal indices. Peripheral DXA Scan was done using the left forearm as a reference for bone densitometry. Statistical analysis using SPSS 15 package was carried out. p value < 0.05 is significant.

Results: The prevalence of bone loss was 77.5%.Using the ISCD criteria, osteoporotic rate was 45 % (18) Z-Score < -2.However using the WHO criteria 47.5%(19) had osteopenia with fracture rate of 7.5 %(3) from trivial trauma. Only 2.5 %(1) met the criteria for osteomalacia. The anthropometric indices were not significantly different between hyperthyroid and controls p>0.05. Proximal myopathy had significant association with bone disease, p=0.04.The bone markers-osteocalcin,Total alkaline phosphatase and urinary calcium/creatinine ratio were significantly elevated in persons with thyrotoxicosis compared with the control ,p= 0.000, p=0.000 and p=0.02 respectively.Osteocalcin levels correlated significantly with FT4 and FT3(r=0.3,p=0.01 &r=0.5,p=0.000) but negatively with TSH(r= -0.3,p=0.002) while urinary calcium/creatinine did not correlate p>0.05.The mean serum levels of Ca,P and urinary Phosphorus were slightly elevated but these were all statistically insignificant while 25-Hydroxylvitamin D was significantly reduced in thyrotoxicosis patients with bone disease p=0.05. The BMD (g/cm2) was significantly reduced in hyperthyroid patients compared to control (0.392 + 0.07 vs. 0.537 +0.07, p=0.000), negatively correlate with osteocalcin, FT3, FT4 p<0.05 and positively correlate with TSH, r= 0.5, p=0.000.

Discussion: This study showed that hyperthyroidism is associated with high bone turnover which is a significant risk factor for osteoporosis. The lack of correlation between bone formation and resorption markers may reflect the imbalance in bone turnover and the lag phase between resorption and formation.

Conclusion: The prevalence of bone disease was very high and strongly associated with proximal myopathy, severity of thyrotoxicosis,the degree of elevation of the bone markers and reduced levels of 25-Hydroxylvitamin -D.

Abstract #1138

AN UNUSUAL CASE OF LEVOTHYROXINE ALLERGY

Issac Sachmechi, MD, FACP, FACE, Aileen Wang, Barbara Hirsh

Objective: Allergic reaction to levothyroxine preparation is presumed to be attributed to the dye ingredients in the tablets. These patients will usually not develop allergic reaction to dye free levothyroxine, which comes in 50 microgram tablet. We report a case of hypothyroid patient who developed allergic reaction also to the dye free levothyroxine preparation and liothyronine but not to new gel capsule preparation of levothyroxine (tirosint).
Case Presentation: A 54 year old female with Hashimoto's thyroiditis was started on Levothyroxine sodium (synthroid), developed a generalized skin rash. Switching her to a dose equivalent alternative preparation (Levoxyl 112 micrograms), she again developed rash. Subsequently was put on dye free preparation (2.5 tablets of Levoxyl 50 micrograms) but the rash recurred. The patient also developed similar reaction to other liothyoronine preparation (cytomel). Assuming that the allergic reaction is to the base powder in the tablet form, she was switched to gel capsule tirosint and she did not developed a rash. She remained stable with her hypothyroidism controlled on this preparation.
Discussion: There are two brands of levothyroxine (levoxyl and unithroid), which are both free of acacia and lactose. Our patient had allergic reaction to synthroid, levoxyl and unithroid, and subsequently to liothyronine (cytomel). Allergic reaction to levothyroxine is thought to be related to its inactive base ingredients; usually to color additives or to acacia, talc, lactose, magnesium stearate, povidone, gluten, confectioner's sugar/corn starch, alcohol and similarly the liothyronine inactive base ingredients include calcium sulphate, gelatin, starch, stearic acid, sucrose and talc. Switching to color free tablets (e.g. levoxyl 50mcg) usually resolved this problem. Failure to resolve with this switch may implicate other base components, such as acacia or lactulose, as possible allergens. The other alternative preparations in case of allergy is the capsule formulation (e.g tirosint) which only has the active ingredient, water, glycerine and gelatin. Other plausible alternatives are natural desiccated thyroid drug like armor thyroid or hypoallergic natural thyroid which are also free of acacia and lactose.
Conclusion: In case allergic reaction to Levothyroxine tab that not resolved by switching to dye free preparation (50mg Levothyroxine tablets); one should switch then to the gel capsule formulation: Tirosint.

Abstract #1139

EXACERBATION OF GRAVES' DISEASE WITH THE ACUTE ADMINISTRATION AND INCREASED DOSE OF EPOPROSTENOL FOR PULMONARY HYPERTENSION: A CASE REPORT AND REVIEW OF THE LITERATURE

Diep Nguyen, DO, Donald Richardson, MD, FACE, FACP, Romesh Khardori

Objective: To report a case of Graves' disease exacerbated by acute administration and up titration of epoprostenol in a patient with idiopathic pulmonary hypertension.
Case Presentation: 58 year-old African American male with idiopathic pulmonary arterial HTN since 2004, was admitted for progressive worsening dyspnea on exertion, shortness of breath, fatigue, lower extremity edema, exercise intolerance, and postural dizziness, despite being on "triple" therapy for PAH - bosentan, tadalafil, and treprostinil at high dose. He also reported a 20 lbs weight loss, muscle weakness, anxiety, sleep disturbance, and hyper-defecation. Right heart catheterization showed increase in PAP 100/44mmHg, mean 66mmHg, RVSP 100mmHg, and cardiac index of 2.5L/min/m2. He did not respond to IV diuresis; treprostinil was changed to epoprostenol 51ng/kg/min. TSH was suppressed at <0.005 and elevated FT4 of 4.3, FT3 of 6.6 and TSI of 522. Thyroid ultrasound was consistent with Graves' disease. Patient was started on MMI and Questran. Coreg at low dose was attempted but resulted in severe bradycardia with hypotension. One week later, his repeated FT4 was 1.8 and symptoms improved, thus MMI was decreased. Repeated RHC showed mild improvement; epoprostenol was increased. A few days after increasing the dose of epoprostenol, patient's HR went up to 120 beats/min, O2 requirement increased. Patient was transferred back to ICU. Epoprostenol dose was increased further to 62ng/kg/min. Within days FT4 went back up to 2.5 with FT3 of 5.7. MMI was increased again. Solumedrol was given for unrelated reasons (thrombocytopenia). He was discharged home on epoprostenol 60ng/kg/min, sildenafil, bosentan, and MMI 30mg BID. On the day of discharge, his serum FT4 was 2.3, FT3 6.3, NT-proBNP 102. He got readmitted 2 days after discharge for fever of 102 with distress. Infection and vasculitis workup were negative. FT4 2.2, FT3 4.6, NT-proBNP 1552. We felt his presentation was due to exacerbation of his thyrotoxicosis. Epoprostenol was discontinued and switched back to IV tresprostinil, and his symptoms and thyroid function improved once epoprostenol was stopped.
Discussion: A 6.7% prevalence of thyrotoxicosis has been reported in thyroid stimulating immunoglobulin (TSI) negative patients treated with epoprostenol. (Chadha C

et al. Endo Practice 2009 Mar;15(2):116-21). Our patient was also at greater risk and predisposed to relapse due to presence of TSI.
Conclusion: Considering the temporal relation of epoprostenol use, the data strongly suggests the drastic increase in dose of epoprostenol worsened our patient's Graves's disease. Discontinuation of this drug led to dramatic improvement.

Abstract #1140

ARTERIAL STIFFNESS IS ASSOCIATED WITH HIGH-NORMAL TSH LEVELS IN HEALTHY POSTMENOPAUSAL WOMEN

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Objective: Beyond clinical or subclinical thyroid dysfunction, fluctuation of thyroid hormones within the reference range has been associated with significant effects on the cardiovascular system. The present study aimed to assess the effect of thyroid hormones on surrogate markers of early cardiovascular disease in a sample of euthyroid postmenopausal women.
Methods: This cross-sectional study recruited 106 healthy postmenopausal women, aged 41-68 years, with thyroid stimulating hormone (TSH) levels within the laboratory reference range (0.4-4.5μIU/mL). Fasting venous blood samples were drawn for biochemical and hormonal evaluation, which included free triiodothyronine, free thyroxin, as well as serum thyroid peroxidase and thyroglobulin autoantibodies. Ultrasound evaluations included indices of arterial structure and function, namely intima-media thickness, flow-mediated dilation of the brachial artery, carotid-femoral pulse wave velocity (PWV) and augmentation index. Blood pressure and anthropometric measures were also determined in each individual. We evaluated the associations between arterial markers and serum levels of thyroid hormones and autoantibodies.
Results: Mean values of PWV increased linearly across increasing TSH quartiles (8.23±1.80m/s vs. 9.45±2.18m/s in quartiles Q1 and Q4 respectively, p-value=0.014). Furthermore, TSH levels correlated positively with measures of PWV (r=0.2, p=0.047). Postmenopausal women with serum TSH>2.5μIU/mL had higher values of PWV when compared with subjects with lower TSH levels (9.68±1.97m/s vs. 8.54±1.83m/s; p-value=0.030 in the univariate analysis). Linear regression analysis revealed that levels of TSH>2.5μIU/mL, age and insulin resistance were the only significant predictors of PWV (TSH, β-coefficient=0.222; p-value=0.014). In contrast, TSH as a continuous variable did not independently correlate with PWV in multivariate analysis, possibly indicating a non-linear type of correlation between these two parameters with a "step-up" biologic effect of TSH above the cutoff value of 2.5μIU/mL in this population. No associations were found between the remaining markers and levels of thyroid hormones, whereas thyroid antibodies were not associated with any of the arterial markers.
Discussion: Women with TSH levels in the upper reference range have increased arterial stiffness compared to women with lower levels of TSH.
Conclusion: Serum TSH is an important predictor of arterial stiffness in euthyroid postmenopausal women. These results are supportive of the need of redefining the upper normal TSH range in postmenopausal women, with respect to the effects on the vasculature.

Abstract #1141

ZENKER DIVERTICULUM PRESENTING AS THYROID NODULE: 2 CASE REPORTS

Erjola Balliu, MD, Nirali Shah, M.D, Marina Charitou, Steven Weitzman

Objective: The incidental discovery of a thyroid nodule by ultrasound (US) occurs in 19-67% of the general population. Occasionally, a Zenker diverticulum may be mistaken for a left-sided thyroid nodule on US. We present two case reports of Zenker diverticulum that mimicked a thyroid nodule on US.
Case Presentation: A 54 year old woman was referred to the endocrinology clinic for an incidental left sided 1.3 cm mid lobe thyroid nodule with internal calcifications incidentally seen on cervical spine MRI. Given concern for thyroid malignancy she underwent a fine needle aspiration (FNA). FNA revealed abundant mature squamous cells, few clusters of atypical cells and vegetable material in the background of debris and bacteria. No follicular cells or thyroid parenchyma were visualized. A repeat FNA under US guidance was performed given above concerning findings. This time the esophagus was noted to be intimately adjacent to the thyroid lesion and a Zenker diverticulum was suspected. Cytology revealed similar findings to the prior one. Further evaluation with a CT scan with contrast of the soft tissue of neck revealed an ovoid debris-containing diverticulum posterior to the left thyroid lobe consistent with a Zenker diverticulum. The second case is a 47-year-old woman with hypothyroidism and celiac disease who was incidentally found to have a left sided thyroid nodule measuring 1.2 cm with micro calcifications on MRI of the cervical

spine. Initial FNA was non-diagnostic due to marked hypocellularity. She underwent a repeat US and FNA where a Zenker diverticulum was suspected based on cytology and sonographic features. Cytology showed numerous benign reactive squamous cells in a background of fungus, yeast, bacteria and debris in the absence of follicular or colloid cells. Further evaluation was recommended and the patient underwent an EGD, however a diverticulum was difficult to demonstrate and the patient refused further work up.

Discussion: These cases illustrate the importance of being aware of the unique sonographic and cytopathological findings of a Zenker diverticulum in order to prevent unnecessary procedures. Some of these typical sonographic features include a change in shape during swallowing, connection to the esophagus, a peripheral echogenic line, internal echogenic foci and a boundary hypoechoic zone.

Conclusion: It is important to be able to identify these findings to differentiate Zenker diverticulum from thyroid nodules and possible cancerous lesions.

Abstract #1142

A RARE CASE OF EARLY ONSET THYROTOXIC PERIODIC PARALYSIS

Grace Chang, MD, Akshay Jain, MD,
Ronald Swerdloff, MD

Objective: To present a unique case of early onset thyrotoxic periodic paralysis (TPP) in a patient of Hispanic descent and discuss its pathophysiology and management.

Case Presentation: A 22 year old Hispanic male presented with a 16-hour history of worsening paraparesis. He had consumed bean burritos the night before and woke up with weakness in his legs that gradually progressed to complete paralysis of bilateral lower extremities. On examination, the patient's pulse was 110/min and blood pressure 144/91mmHg. The thyroid gland was smooth with no enlargement. Motor function was 1/5 in bilateral lower extremities. Labs were significant for potassium 1.9mmol/L (3.5-5mmol/L), TSH 0.02mIU/dL (0.34-5.6mIU/dL) and FT4 3.3ng/dL (0.7-1.48ng/dL). EKG showed Uwaves in precordial leads and prolonged QT interval of 461msec. Patient received intravenous potassium supplementation with symptomatic improvement and completely regained motor function in 5 hours. It was later elucidated that the patient's past medical history was positive for similar episodes that first began at age 13. He had 3 more episodes before he was also diagnosed with hyperthyroidism and started on propylthiouracil (PTU), following which he had no recurrence of symptoms. He stopped taking PTU 6 months prior to presentation.

Discussion: TPP is a rare myopathy seen in Graves' disease. In North America, TPP has a prevalence of 0.1% to 0.2% of all cases of hyperthyroidism, of which 90% occur in people of Asian descent. The age of onset is in the third decade of life. Its pathophysiology includes a combination of thyrotoxicosis, genetic predisposition with Kir 2.6 potassium channel mutation, and precipitating factors, including high carbohydrate diet, glucocorticoids, and intense exercise. The compounding effect of elevated levels of thyroid hormones, insulin, and catecholamines over-activates the Na/K ATPase pump, which leads to hypokalemia with resultant skeletal muscle paralysis. Treatment includes potassium replacement therapy to enable the patient to recover from paralysis and to prevent cardiac arrhythmia. Nonselective β adrenergic blockers, such as propranolol, are also used to inhibit adrenergic overstimulation of the Na/K ATPase pump. Definitive treatment of TPP is the management of thyrotoxicosis by medical therapy using methimazole or PTU, radioactive iodine therapy, or surgery.

Conclusion: Severe TPP is known to cause significant paralysis and arrhythmias. Although generally seen in Asians and in early adulthood, it can occur in other ethnicities and in childhood. When diagnosed in a timely manner and treated appropriately, it has potential to resolve completely without sequelae.

Abstract #1143

HYPOTHYROIDISM AS A CAUSE OF HYPONATREMIA: FACT OR FICTION?

Grace Sun, D.O., Kevin Pantalone, DO,
Betul Hatipoglu, MD

Objective: Hypothyroidism, a long-recognized etiology of hyponatremia, is often considered in the differential diagnosis of low serum sodium in the euvolemic patient. To identify the correct etiology of hyponatremia, accurate assessment of volume status and evaluation for other causes of euvolemic hyponatremia (syndrome of inappropriate anti-diuretic hormone-SIADH or adrenal insufficiency) are critical. Our aim is to illustrate that severe primary hypothyroidism alone may not be enough to cause hyponatremia in the otherwise healthy ambulatory patient. We present 10 patients with thyroid stimulating hormone (TSH) levels >95 μU/mL without concomitant hyponatremia.

Methods: Over 6 months, the first 10 consecutive patients with primary hypothyroidism who were ordered a levothyroxine oral challenge test were entered into our case series if they met the following inclusion criteria: TSH level >95 μU/mL and a same-day sodium level. A

retrospective chart review was conducted using electronic health records. All lab tests were collected on an outpatient basis. Same-day TSH, sodium, and glomerular filtration rates (GFRs) were obtained; same-day free triiodothyronine (FT3) or free thyroxine (FT4) was also recorded if tested.

Case Presentation: The 10 subjects (2 men, 8 women) were ages 19-97 years (median-46.5). TSH ranged from 96.9-515.6, (median 169; normal 0.40-5.50 μU/mL) with sodium levels of 136-139 (median 138; normal 135-146 mmol/L). The lowest sodium was 136 with concurrent TSH of 469.7, FT3 of 1.0 (normal: 1.8-4.6 pg/mL) and FT4 of 0.2 (normal: 0.7-1.8 ng/dL). GFR ranged from 44-114 mL/min/1.73m2 (median 67.5; normal: 90-120).

Discussion: In our small series of patients with extreme TSH elevations, none had a serum sodium level below normal (<135 mmol/L), even in the presence of low GFR. The results of this otherwise healthy ambulatory population suggest that hypothyroidism alone is unlikely to result in hyponatremia. Primary hypothyroidism, which has been reported to reduce free water clearance, may require the presence of other trigger(s) to result in concurrent hyponatremia. We hypothesize that in the absence of other conditions which may alter the kidney's ability to clear free water, hypothyroidism is unlikely to cause low sodium. Many cases of "hypothyroid hyponatremia" reported in the medical literature may merely represent SIADH in patients who concurrently have an elevated serum TSH.

Conclusion: Hyponatremia can be a common occurrence in the hospitalized and/or chronically ill. However, in an otherwise relatively healthy ambulatory population, hypothyroidism may be a much less clinically relevant cause of hyponatremia.

Abstract #1144

FORTY-SEVEN YEARS OF FOLLOW-UP OF A PATIENT WITH TALL CELL VARIANT PAPILLARY THYROID CANCER

Ivica Boban, MD, Jeffrey Miller, MD

Objective: Tall cell variant (TCV) of papillary thyroid cancer (PTC) represents an aggressive form of differentiated thyroid cancer. We present a case of TCV for over 47 years with good clinical outcomes despite locally recurrent disease.

Methods: Case report of long living tall cell variant of papillary thyroid carcinoma.

Case Presentation: Our patient was diagnosed in 1964 at the age of 20 with micropapillary thyroid cancer, for which he initially underwent left lobectomy. He had neither significant past medical history nor risk factors for thyroid malignancy. After the lobectomy, the patient was lost to follow up until age 45 when metastasis was discovered in cervical lymph nodes. He then underwent completion thyroidectomy and neck dissection. Histopathology revealed TCV, which on review had also been present on the initial pathology from the lobectomy. Subsequently he had his first RAI. Fifteen years later at age 60, he had a second episode of locally recurrent disease treated with a second neck dissection and subsequent RAI. Post-ablative whole body scan was negative. Four years later, at age 64, he had mildly elevated thyroglobulin levels with locally recurrent disease confirmed on FNA requiring a third neck dissection followed by another dose of RAI. Repeat whole body post-ablative scan was again negative. Three years later, at the age of 67, recurrent disease was found in cervical lymph nodes. Patient underwent a fourth neck dissection for recurrent disease. Six months after the last neck dissection, the patient's thyroglobulin level was 0.2, with a mildly suppressed TSH of 0.3. Recent PET-CT whole body scan showed mild persistent metabolic activity in the right neck lymph nodes. The patient is currently symptom-free. As he has previously undergone extensive neck dissection, received high cumulative dose of RAI, and currently has only mildly persistent thyroglobulin levels in the setting of probable non-iodine-avid local disease, he has opted to defer additional therapy at this time.

Discussion: PTC is the most common endocrine malignancy with the best overall prognosis. Only a minority of patients have a more aggressive course of disease requiring additional surgery and RAI. Compared to classical PTC, TCV is a poor prognostic indicator with a higher recurrence rate, higher resistance to RAI, and overall significantly increased mortality.

Conclusion: Although TCV PTC caries a worse prognosis, our patient had good clinical outcome with only locally recurrent disease over a 47 year time period.

Abstract #1145

SUPPURATIVE THYROIDITIS DUE TO NOCARDIA ASTEROIDES IN AN IMMUNUSUPPRESSED PATIENT

Sayed Aamir, MD, Ali Rizvi, MD

Objective: To describe the presentation of thyroid abscess and suppurative thyroiditis caused by Nocardia Asteroides in the setting of renal transplant and chronic immunosuppression

Methods: A 64 year-old male with hypertension s/p renal transplantion was hospitalized with 2 weeks of fever, sore throat, and neck pain. Immunosuppressive medications included prednisone, mycophenolate, and tacrolimus. Physical examination revealed a toxic-appearing, febrile

person with tachycardia and BP of 128/80 mm Hg. The anterior neck and thyroid was tender, with the right lobe being larger and firm. There were generalized shivers but no lymphadenopathy, exophthalmos, or tremor. WBC count was 13,000 with 45% bands. No usual sources of sepsis were found, blood cultures showed no growth, and broad spectrum antibiotics were started. TSH was <0.004 and free T4 3.66 ng/dl (0.86-1.76). Neck MRI pointed to thyroid nodularity and increased signal in the right lobe suggesting fluid attenuation on T2-weighted images. The possibility of an abscess was raised. Ultrasound showed a multinodular thyroid gland with a 2.3 cm heterogeneous, slightly hyperechoic area on the right. Peroxidase antibody titer was <10, and TSI activity was 93% (normal <127). I-123 uptake was profoundly low at 1.5% and 0.8% at 6 and 24 hours respectively, and thyroid scan showed markedly reduced activity.

Case Presentation: The patient experienced continued fever and neck discomfort. An US-guided aspiration with a 22-guage needle of the right thyroid lesion was performed, revealing frank purulent material and branching, gram-variable rods on gram stain. Culture grew branching partially acid-fast bacilli consistent with the organism nocardia asteroides. A 2.6 cm cavitary lesion in the left lung was noted on chest CT, a biopsy of which however, failed to show any pathogen on smear or culture. The patient responded well to drainage of the thyroid abscess and meropenem antibiotic therapy.

Discussion: Nocardia asteroides is a gram-positive, partially acid-fast, soil-borne aerobic actinomycete bacterium that causes both localized and disseminated infection in immunocompromised patients. Very few cases of thyroid involvement by Nocardia have been reported. Our case reinforces that an opportunistic infection of the thyroid gland by nocardia and lead to inflammatory thyroiditis in immunocompromized individuals that can be life-threatening.

Conclusion: Thyroid nocardiosis can occur in the setting of immune suppression, mimic generalized sepsis, and lead to diagnostic confusion. Management consists of aspiration biopsy to establish the cause, drainage, surgical intervention if indicated, antibiotic therapy, and symptomatic treatment of thyrotoxicosis.

Abstract #1146

RARE LIFE THREATENING COMPLICATIONS IN A COMMONLY USED DRUG

Ibrahim Ibrahim, MD, FRCP, Elzubier Elzubier

Case Presentation: 17 Years old girl diagnosed with grave's disease 2 years ago. She was treated with propylthiouracil (PTU) and maintained on 150 mg of PTU. After being on treatment for 22 months, she presented with worsening symptoms of thyrotoxicosis, large goitre and thyroid eye disease. The dose of PTU was increased and propranolol was added. Few days later she represented with symptoms of sore throat and fever. Routine investigation showed leucopenia. PTU was reduced and she was referred for total thyroidectomy. On examination she was febrile, tachycardic, had large goitre and exophthalmos. Blood tests showed free T3 of 8.5 (2.8-7.1 pmol/l), free T4 33.8 (12-22 pmol/l), WBC 1.44 (4-11 K/UL), neutrophil count 0.44 K/UL (2-6.9 K/UL), Hb 10.9 g/dl. platelet of 251 KU/l. PTU was stopped and she was commenced on Luogol's solution and propranolol. Full septic screen identified no organism.However her neutrophils count continued to drop down to 0.1 K/UL. She was commenced on Granulocyte Stimulating Factor (GCSF). Her WBC and neutrophils responded well to GCSF, but she developed severe extensive vasculitic rash all over her body, mouth ulcers,and fever. Her chest x ray was normal. Renal and Liver profile within normal ranges. Coagulation screen normal. Further investigations showed, ANCA strongly positive. PR3 (73.65) and MPO (26.3). ANA and ENA were negative.She was commenced on high dose of Prednisolone. She responded very well to steroid which was discontinued after 10 days. She underwent total thyroidectomy with uneventful post operative course. She was discharged home on Thyroxine 75 ugm. Over all her presentation in keeping with diagnosis of PTU induced ANCA positive vasculitis and Agranulocytosis.

Discussion: Antineutrophil antibodies (ANCA) are thought to be involved in both drug-induced neutropenia, and vasculitis in patients on PTU. It is proposed that neutropenia is caused through a complement-mediated mechanism. On the other hand neutrophils, perhaps activated by a viral infection, release MPO from their granules, which converts PTU into cytotoxic by product that mediate the vascular injury leading to vasulitis. Immunological reactions including neutrophil cytotoxicity and agglutination by ANCA can be a plausable mechanism underlying both neurtropenia and vasculitis in this case.

Conclusion: As PTU is a commonly used drug, physicians should have a high index of suspicion when patients receiving PTU develop systemic disease consistent with vasculitis and or neutropenia, regardless of the duration of the drug therapy.This case also support the evidence that ANCA induced by PTU contribute to both leucopenia and vasculitis.

Abstract #1147

CONCOMITANT UNILATERAL PAPILLARY THYROID CARCINOMA AND PRIMARY HYPERPARATHYROIDISM - A CASE REPORT

Suneetha Vysetti, MD, Preethi Sridhar, MD, Boby Theckedath, Janice Gilden, MD

Objective: Most clinicians are well aware of the coexistence of medullary thyroid cancer and hyperparathyroidism in hereditary and sporadic multiple endocrine neoplasia syndromes. However the association of nonmedullary thyroid carcinoma and hyperparathyroidism is rare and comprises only 3% of the patients treated for hyperparathyroidism. We report a case of a 53 year old asymptomatic female patient with a concomitant parathyroid adenoma and unilateral papillary thyroid carcinoma with its clinical, biochemical, scintigraphic, and histologic features.
Case Presentation: A 53 year old female patient with no significant personal or family history, was referred to the Endocrine clinic for evaluation and management of thyroid nodules, discovered on clinical exam. She was clinically and biochemically euthyroid. Thyroid ultrasound showed multiple nodules bilaterally. Pertinent serum biochemistry results included a high serum calcium of 10.8 mg/dl (normal: 8.5 - 10.1) with high PTH of 181.5 pg/ml (normal :14-72), consistent with primary hyperparathyroidism. The Technetium sestamibi scan revealed a functioning left parathyroid adenoma and an area in the left thyroid lobe with persistent uptake on delayed images. FNA of the dominant right sided nodule and two left sided nodules was performed. The capsule of the left inferior nodule was thick, making the FNA difficult. The cytology from the superior nodule on the left lobe was consistent with papillary thyroid carcinoma, and the right nodule was benign. The patient underwent a total thyroidectomy and surgical excision of the left parathyroid adenoma. Surgical pathology showed papillary thyroid carcinoma of left thyroid lobe with well differentiated classical papillary and follicular variants. The tumour size was noted to be more than 2 cm , with no extrathyroidal or lymphovascular extension. Surgical specimen of the left parathyroid specimen revealed a 5 gm adenoma measuring more than 2 cm.The thyroid ultrasound report suggested a possible left inferior nodule, but was later found to be the parathyroid adenoma.
Discussion: Technitium (99mTc) scanning is known to show more rapid washout from a normal thyroid than that from an abnormal parathyroid tissue. But in practice, confusion could occur from a coexisting thyroid nodule, as in our patient. Furthermore, the isotope can accumulate in thyroid nodules, thus decreasing the specificity of parathyroid scans.
Conclusion: Our case highlights the need to be aware of concomitant thyroid and parathyroid pathology, which is rare. These type of situations often complicate diagnostic imaging and management. It is optimal to deal with both problems in one surgical procedure. Supported by JAL FHCC.

Abstract #1148

A SERIES OF UNFORTUNATE EVENTS: DILATED THYROTOXIC CARDIOMYOPATHY RESULTING IN CARDIOEMBOLIC STROKE IN A YOUNG MAN WITH UNRECOGNIZED GRAVE'S DISEASE

Soamsiri Niwattisaiwong, MD, Mana Dissadee, Toyiba Syed, MBBS, Jennifer Bernard, MD

Case Presentation: An 18-year-old man without known cardiac disease presented with palpitation and worsening shortness of breath for 10 days. On examination, the patient had a heart rate of 140 beats/min, a respiratory rate of 38 breaths/min, and diffuse thyroid enlargement. Thyroid function tests revealed the following: TSH < 0.03 μU/ml (0.34-5.60 μU/ml), free T4 3.18 ng/dL (0.58-1.64 ng/dL), and total T3 2.13 ng/dL (0.87-1.78 ng/dL). Thyroid stimulating immunoglobulin was 389%. Echocardiogram showed left ventricular (LV) ejection fraction of less than 20% and enlargement of all cardiac chambers. No structural valve abnormalities or intracardiac thrombi were visualized. The patient was diagnosed with Grave's thyrotoxicosis and congestive heart failure (CHF). Treatment with propranolol, PTU, potassium iodide, along with hemodynamic support was started. However, the patient developed altered mental status in the next few days. Emergency CT brain revealed a large infarct in the left frontoparietal lobe with midline shift. Repeated echocardiogram with bubble study was negative for intracardiac shunting. Retrospective review of telemetry strip showed persistent sinus tachycardia without atrial fibrillation (AF). Decompressive craniectomy was performed. The patient underwent a complicated hospital course and was discharged with permanent neurodeficit.
Discussion: The hallmark of hyperthyroidism is hyperdynamic circulatory state with increased cardiac output. Despite increased cardiac performance, approximately 6% of hyperthyroid patients paradoxically present with CHF. Less than 1% develop dilated thyrotoxic cardiomyopathy (DTC). Persistent tachycardia in hyperthyroidism results in myocardial energy depletion and impaired myocyte calcium handling, leading to dilated cardiomyopathy. Thyroid hormone may also alter the expression of certain cardiac proteins essential for myocardial contraction. Treatment of hyperthyroidism

usually results in partial or complete resolution of DTC. Cardioembolic stroke is a rare but devastating complication usually associated with thyrotoxic AF. Our patient developed a stroke with no evidence of AF. We suspect an LV thrombus formation in the setting of severe DTC. Excess thyroid hormone is also known to modify the coagulation-fibrinolytic balance, possibly making the patient more prone to thrombus formation. ***Conclusion***: DTC is a rare presentation of hyperthyroidism and is associated with increased cardiovascular morbidity and mortality, mainly due to CHF and cardioembolism. Awareness of this uncommon presentation of hyperthyroidism is essential to identify patients with potentially reversible dilated cardiomyopathy.

Abstract #1149

A PATIENT WITH PITUITARY RESISTANCE TO THYROID HORMONE (PRTH)AND FAMILIAL HYPOCALCIURIC HYPERCALCEMIA(FHH)

Gauri Dhir, MD

Objective: Pituitary resistance to thyroid hormone (PRTH) is a rare disorder in which the pituitary thyrotropes are resistant to the inhibitory effect of thyroid hormone, resulting in peripheral hyperthyroidism. We present a case of PRTH found in conjunction with inactivating mutation of calcium sensing receptors,the syndrome of familial hypocalciuric hypercalcemia (FHH).
Case Presentation: A 79 year old white woman with a history of thyroid ablation several years ago for presumed hyperthyroidism, presented to our clinic with weight loss, sweating,palpitations and nervousness. Tests showed a normal TSH of 0.76 mU/L, increased total T4 (>20 mcg/dl), elevated T3 resin uptake (44.6%), and an elevated calculated FT4 index of 4.41, while she took LT4 300 mcg daily.The clinical hyperthyroidism, with a normal TSH, and an elevated T3 resin uptake and total T4, on a high dose of LT4, suggested either a TSH-secreting pituitary adenoma or PRTH. An MRI showed a normal pituitary gland. She was clinically euthyroid only when a dose of LT4 was given that resulted in normal FT3 and FT4 and very high levels of TSH (60-100 mU/L). The likely diagnosis is thought to be PRTH. She is now on LT4 100mcg daily, clinically euthyroid, with a TSH of 58.6 mU/L, FT4 1.7 ng/dl, and FT3 221 ng/dl. This patient also had moderately elevated serum calcium levels of 10.6 to 11.8 mg/dl, with intact PTH of 36-70 pg/ml, normal creatinine of 1.1mg/dl and low 24-hr urinary calcium excretion of 65mg. She had a normal vitamin D levels of 31ng/ml & no complications of hypercalcemia. We believe she had FHH in addition to PRTH.
Discussion: PRTH is characterized by clinical hyperthyroidism with inappropriately nonsuppressed TSH. This syndrome differs from generalized resistance to thyroid hormone, in which clinical manifestations of hyperthyroidism may be absent (despite elevated FT3 and FT4). Our patient had received thyroid ablation, so euthyroidism had to be achieved by finding a dose of LT4 that resulted in normal FT3 and FT4 levels, while ignoring the resulting elevation in TSH. A mutation in the thyroid hormone receptor β (TRβ) gene is the most common cause of PRTH. Similar findings may be caused by a TSH-producing pituitary adenoma; this may be ruled out with MRI of the pituitary, and a normal response of TSH to thyrotropin-releasing hormone (TRH), typically blunted in a TSH-producing adenoma. More than 1,000 subjects with PRTH have been identified. Our patient also has FHH. Both PRTH and FHH involve mutations in the genes located on chromosome 3.
Conclusion: Elevated TSH with high FT4 and FT3 does not always indicate a TSH-secreting pituitary adenoma but should prompt us to look into other causes such as PRTH.

Abstract #1150

ANAPLASTIC THYROID CARCINOMA WITH CUTANEOUS EXTENSION

Pratima Kumar, MD, Shwetha Thukuntla, MD

Objective: To describe a case of rapidly progressive anaplastic thyroid cancer.
Case Presentation: A 76-year-old woman presented with an 8 month history of a rapidly enlarging right neck mass. She denied hoarseness of voice, dysphagia, dyspnea, or stridor; she did report a 10 pound weight loss. Serum thyrotropin was 1.24 μIU per milliliter. Computed tomography of the neck showed a 7.9 cm solid and cystic mass with calcifications. Fine needle aspiration revealed papillary thyroid cancer with a poorly differentiated component. Within a month, the mass had markedly increased in size and was eroding through the skin. The patient underwent a total thyroidectomy, radical wide excision of the neck and soft tissue, central compartment dissection, and right modified neck dissection.Pathology showed anaplastic thyroid carcinoma with direct extension into the skin and sternocleidomastoid muscle with lymph node metastasis. There was no evidence of distant metastasis. The patient declined further therapy.
Discussion: Anaplastic thyroid cancer is an uncommon, typically lethal malignancy with a mean survival time usually less than 6 months from the time of diagnosis. It can arise de novo or more commonly from a pre-existing

well differentiated thyroid cancer and can present as a rapidly enlarging neck mass and the diagnosis of anaplastic carcinoma should be considered when there is rapid growth in a preexisting thyroid mass. Despite an extremely poor prognosis in patients with anaplastic thyroid carcinoma, early diagnosis in patients with intrathyroidal tumors and surgical resection with external beam radiotherapy was associated with lower cause-specific mortality.
Conclusion: The diagnosis of anaplastic carcinoma should be considered when there is rapid growth in a preexisting thyroid mass.

Abstract #1151

UNMASKING HYPERTHYROIDISM IN LITHIUM-INDUCED THYROIDITIS

Kristine Nicolas, MD, Chitra Manickam, Ahmed Ibrahim, Osama Amro, Sabrina Raroque, MD, Joumana Chaiban, MD

Objective: Lithium has known impacts on thyroid function, most notable of which is its inhibition of thyroid hormone release, thus causing either goiter or hypothyroidism in certain patients. In rare occasions, lithium might induce silent thyroiditis. We are reporting a case with unusual presentation of lithium-induced silent thyroiditis
Case Presentation: A 54-year-old male with past medical history of hypertension, diabetes mellitus, and bipolar disorder presented with chest pain and shortness of breath. He was also complaining of tremors and drenching sweats. He has been on lithium for his bipolar disorder, however, this was recently discontinued after he developed lithium toxicity. His medications included lisinopril, metformin, furosemide, and trifluoperazine. Physical examination revealed tachycardia and an irregularly irregular heart rhythm. Electrocardiogram revealed atrial fibrillation with rapid ventricular response. Diltiazem drip was started along with heparin and warfarin. As part of the work-up of atrial fibrillation, a serum TSH was done which showed a level of <0.0001. Serum free T4 level was >8 (N: 0.76 - 1.46). Thyroid peroxidase antibody was elevated at 55 (N: 0 - 34). He was then started on propylthiouracil and metoprolol. He was discharged in a stable condition.
Discussion: Patients with bipolar disorder are usually found to have pre-existing thyroid antibodies. Lithium therapy has been associated with an increase in antibody titers in patients who already have pre-existing antibodies in their system. This can then lead to the development of thyroiditis. However, due to the "hypothyroid" effect of lithium, it likely suppresses the hyperthyroid state of such patients. Upon discontinuing lithium, only then will thyrotoxicosis develop.
Conclusion: The case we are reporting emphasizes the importance of monitoring thyroid function test in patients taking lithium and highlights the importance of understanding the pathophysiology of lithium-induced thyroid dysfunction.

Abstract #1152

PTU: SUPPRESSING MORE THAN THYROID HORMONE

Thomas Jensen, MD, Jerald Marifke, MD

Objective: To describe a rare case of PTU-induced agranulocytosis in a patient with relapse of Graves' hypothyroidism, who previously tolerated the medication.
Case Presentation: 56 year-old woman with a past history of Graves' disease presented with a chief complaint of fever, chills, nonproductive cough, and general malaise for 4 weeks. Her symptoms began immediately after initiation of propylthiouracil (PTU) for recurrence of hyperthyroidism, though she failed to report this until a day before admission. She was diagnosed with Graves' hypothyroidism in 1996 and initially treated with methimazole. However, she did not tolerate this and was switched to PTU. She received this therapy for two years, at which time it was stopped due to remission of her disease. At this presentation the patient had a temperature of 100.3F, rigors, exudative tonsillar lesions, and an erythematous pharynx. She had a normocytic anemia (hgb of 9.2g/dL), and thrombocytopenia (123e3/uL), but more substantially her white cell count (WBC) was 1.3x10^9/L, and an absolute neutrophil count of 0. Her TSH was 0.015µU/mL. PTU was held and hematology concurred with a diagnosis of PTU-induced agranulocytosis. Granulocyte colony stimulating factor (GCSF) was not started since evidence was inconclusive as to it being a benefit in PTU-induced agranulocytosis. Her neutrophil count recovered on hospital day 10 to 1.8x10^9 and remained above 1.5 x10^9/L during the remainder of the hospital stay. She had a WBC of 4.8 x10^9/L, hemoglobin 11.8g/dL, and platelet count of 265e3/uL on discharge. She underwent radioactive iodine treatment for definitive therapy.
Discussion: Agranulocytosis is a rare, serious complication of thionamides occurring in 0.3% of patients. Of interest, there are very few case reports of PTU-induced agranulocytosis on second time exposure. Therefore, physicians and patients must be aware of symptoms of agranulocytosis even if it was previously tolerated, discontinue the thionamide, and perform further investigation immediately. Routine monitoring is not recommended since agranulocytosis develops

suddenly, though typically within the first three months of initiation of therapy. The median time to resolution of agranulocytosis is 10-14 days. Conflicting data exists from retrospective and small randomized control studies as to whether GCSF improves recovery time. Management includes discontinuation of the thionamide and supportive treatment with antibiotics for neutropenic fever is recommended. Either RAI or thyroidectomy should then be pursued for definitive treatment of Graves' disease. ***Conclusion***: Agranulocytosis is a serious complication of PTU that can occur even after prior tolerance to the medication.

Abstract #1153

A RARE OCCURRENCE OF PAPILLARY THYROID CARCINOMA WITHIN AN AUTONOMOUS HOT NODULE.

Karla Arce, MD, Jose Cabral, MD

Objective: Autonomous hot nodules in association with thyroid cancer represent a very rare condition. Discovery of a hot nodule usually implies a benign entity and deters one from performing a biopsy. We present a rare case of a hyperfunctioning hot nodule harboring a papillary thyroid carcinoma.
Case Presentation: This is a 29 year old female with past medical history of ulcerative colitis and migraines who presented to our clinic with complaint of palpitations and a neck mass. At an outside institution the patient incidentally was found to have a lump in her right thyroid in 2008 consequently thyroid ultrasound was performed and showed a mass in the right lobe measuring 1.7 x 1.3 x 1.5 cm. She underwent ultrasound guided fine needle aspiration and was told that cytology was benign. One year later the patient came to our clinic with complaint of palpitations, 5 lbs weight loss in two weeks and an enlarging neck mass. Physical examination revealed a 2 cm mobile, non-tender, right thyroid nodule. Thyroid function tests were consistent with low TSH (0.07mIU/L) and elevated FT4 (1.9 ng/dL). Thyroid ultrasound revealed a 2.6 cm nodule in the right lobe and thyroid uptake and scan showed a large hyperfunctioning nodule in the lower portion of the right lobe suppressing the rest of the gland. The patient underwent right hemithyroidectomy and final pathology confirmed a 1.9 cm papillary carcinoma within the nodule without capsular or vascular invasion. the patient was then referred for completion thyroidectomy and the pathology of the left lobe was negative for malignancy.
Discussion: Autonomous or hot nodules are defined as a nodular region of the thyroid gland that takes up large concentrations of radioactive iodine relative to the rest of the thyroid gland. In the case of hyperfunctioning "hot" nodules, current guidelines do not recommend FNA since generally hot nodules are rarely cancerous. However, reports of the occurrence of malignancy of hot nodules show a varied prevalence of 0.9% to 9% in adults and approximately 25 such cases have been reported until 2005. When it comes to non-metastatic hyperfunctioning thyroid carcinomas it is difficult to predict malignancy within a hot nodule.
Conclusion: Although hot nodules rarely harbor malignancy, in the case of rapidly growing hot nodules we suggest performing fine needle aspiration so that thyroid malignancies are not missed.

Abstract #1154

IODINE DEFICIENCY: STILL A PROBLEM IN AN ENDOCRINE PRACTICE

Caroline Davidge-Pitts, MBBCh, Marius Stan, MD

Objective: Iodine is essential in the synthesis of thyroid hormones therefore its deficiency may lead to thyroid dysfunction. Iodine deficiency is common in developing countries but it appears that it may be re surging in the United States, in certain populations. The best method of evaluating and diagnosing these patients remains a matter of debate while at a population level, urinary iodine measurement remains the gold standard. We are reporting a series of five patients evaluated for thyroid disease who were subsequently found to have low urinary iodine levels.
Case Presentation: All patients were women, and the median age was 47 years (IQR 33-60). Patients were referred, or seeking second opinion for goiter (two patients), abnormal thyroid function tests (two patients) and increased thyroid uptake of 34.7% at four hours, normal range 3-16% (one patient). Prior iodine evaluation was performed in one patient. Three patients were found to be euthyroid and two patients had established hypothyroidism on presentation. Three patients had dietary restrictions or deviations including a) diarrhea with intolerance to dairy products b) well water drinking and c) consumption of predominantly Vietnamese cuisine. 24 hour urinary iodine collections were performed in all patients, with a mean of 59 mcg/24 hours, SD 28 (normal range 100-460 mcg/24 hours). Planned tests and therapies prior to diagnosis here included pituitary MRI (one patient) and radioactive iodine (one patient); both were avoided after the low urinary iodine was discovered and treated. Therapy with iodine led to resolution of diarrhea in one patient, and improvement of non specific symptoms, including anxiety, in 2 patients. Four hour thyroid uptake normalized (decreased to 14.4%) after iodine replacement in one patient. Two patients showed

improvement in prior abnormal thyroid function.
Discussion: Inadequate iodine intake is still present in the Midwest in certain populations. 24 hour urinary iodine is an established screening tool for iodine deficiency in a population; however optimal method for diagnosis in individual patients is still a matter of debate. Consideration can be made to measure urinary iodine levels in patients who present with thyroid disease in the setting of dietary modifications for medical or cultural reasons as iodine replacement may lead to correction of thyroid function abnormalities, as well as improvement of symptoms. Failure to identify this problem may lead to unnecessary studies or treatments.
Conclusion: Inadequate iodine intake is still be a problem in certain populations of the United States, and should be considered during the evaluation of patients with thyroid disease.

Abstract #1155

A DIAGNOSTIC PITFALL: AN UNUSUAL INTERFERING FACTOR TO THE ROCHE ECLIA TSH ASSAY RESULTING IN MISDIAGNOSIS OF HYPOTHYROIDISM DURING PREGNANCY

Tarik Elhadd, MD, FRCP, Laura McCreight, Julia Anderson

Methods: Measurement of TSH in a reference lab with clinical assessment.
Case Presentation: A 21 week pregnant 36 year old Caucasian lady was seen in Medical Ante-natal Clinic in with a suspected primary hypothyroidism. She had a TSH level of 26 mU/L (0.3-5 mU/L) and a fT4 level of 10 pmol/L(11-27pmol/L) measured by an electrochemiluminescence immunoassay (ECLIA) on the Roche E170-module. This was confirmed on repeat testing. The patient symptom was tiredness but no family history of goitre, thyroid disease or neck irradiation. She was clinically euthyroid, no palpable goitre. The patient was started on thyroxine in a dose of 100 µg/ day and was regularly thereafter, when repeat TSH level showed very little change, despite regular increments of the dose with the appropriate rise of her fT4 level. Antibody interference in the TSH assay was suspected and a sample was sent to a reference laboratory which showed a suppressed TSH confirming assay interference. The thyroxine was withdrawn but following delivery and on repeat testing up to 6 month postnatal the TSH level measured by the ECLIA failed to normalize. The interference was found to be due to the presence of an interfering factor to the F(ab')2 fragment of the assay antibody in the patient serum. The interference was also found to be specific for the Elecsys TSH assay
Discussion: Several interfering substances, usually heterophile antibodies, were reported before to interfere with either TSH or fT4 assays and this may cause confusion or diagnostic pitfalls. The diagnostic suspicion is usually brought up when there is a disparity between the clinical picture and the biochemical profile. In our case the patient was pregnant and that added a difficult dimension to the interpretation of the tests specially at the begining. Failure of TSH level to normalise with gradual increase in the dosage of thyroxine gave the clue to the presence of the interference.
Conclusion: The type of TSH intereference detected in our case is rather unusual and not been reported before to the best of our knowledge. Given the complexity of the interpretation of thyroid function tests during pregnancy together with the foetal and maternal implications of thyroid dysfunction in pregnancy awareness of this finding is imperative for both clinicians and scientific staff.

Abstract #1156

THYROID CANCER ; FOLLICULAR CANCER WITH ANAPLASTIC TRANSFORMATION

Omar Akhtar, MBBS, Omolola Olajide, MBBS

Case Presentation: 55 y/o white male, was seen in our endocrine clinic for management of thyroid cancer. He had a history of a neck mass for 20 years. It had been rapidly enlarging over the last 1-2 years and this made him seek medical attention. He was admitted to an outside hospital nine months prior with paraplegia and was found to have a T3 epidural mass. A CT scan of the neck showed a thyroid mass that was 10.2cm x 15.5cm x 9.1cm in size. He also had a FNA biopsy of the thyroid which showed features of a follicular neoplasm. Biopsy of the epidural mass done during his stay showed features of metastatic follicular thyroid cancer . Thyroglobulin level was > 40,000.The diagnosis at the time was follicular thyroid cancer metastatic to the spine. He was then referred to a tertiary center for a radical neck dissection and resection of the epidural mass. Patient refused surgery but was subsequently treated with palliative radiation of the neck and spine. He finally agreed to have a resection of the metastatic site four months later ,with improvement in his leg weakness. Pathology of the resected tumour was consistent with metastatic follicular thyroid cancer. At his initial visit in our clinic, he complained of increasing dysphagia and size of the thyroid mass over the last 2 weeks, so we referred him to a surgeon for debulking surgery. A week after our evaluation, he was admitted to the hospital with worsening dysphagia , shortness of breath and marked leukocytosis (WBC 50,000).The impression was

that he had a leukamoid reaction. He was then transferred to a tertiary center (OSU) for emergent surgery. He was intubated on arrival to the hospital because of respiratory distress.They repeated the Thyroglobulin level which was 230.They also repeated a FNA biopsy of the thyroid which showed anaplastic thyroid cancer. The final diagnosis was follicular cancer with anaplastic transformation. The diagnosis and prognosis was discussed with the family who decided to withdraw care.

Discussion: Anaplastic cancer ranks among the most lethal of all human malignancies(1). Approximately 20 percent of patients with anaplastic thyroid cancer have a history of differentiated thyroid cancer,and 20 to 30 percent have a coexisting differentiated thyroid cancer(2,4). The majority of synchronous thyroid tumors are papillary cancers, but coexisting follicular cancers have also been reported. Long standing history of differentiated thyroid cancer and radiation exposure may be some risk factors for anaplastic transformation(3,4).

Conclusion: Anaplastic transformation should always be considered in follicular thyroid cancer with rapid enlargement.

Abstract #1157

A CASE OF SUDDEN HYPERTHYROIDISM IN A PATIENT ON LITHIUM.

Fadi Siyam, MBBS, Sanaa Deshmukh, Mariana Garcia Touza, MD

Objective: While hypothyroidism is a common event in patient on Lithium, hyperthyroidism is a rare one with an estimated incidence of 0.1 %. For the hyperthyroid state to resolve is even a rarer finding.

Case Presentation: We report the case of a 27- year-old male with Schizoaffective disorder who was admitted with a history of 3 weeks of agitation and auditory hallucinations despite adherence with treatment that included Lithium at a total dose of 1,350 mg daily for the last 3 years. On examination, he was agitated and tachycardic with a heart rate ranging between 90 and 120 /min. He was oriented to time, place and person though he was actively having hallucinations. No goiter, tremor, brisk reflexes or abnormal eye manifestations were noted. His complete blood count and metabolic profile were normal. Thyroid stimulating hormone (TSH) was 0.006mcunit/mL (0.27-0.42) with normal Total T4, free T4 and Total T3 levels. The laboratory work up was repeated and confirmed the findings. Upon review of his medical record he had a normal thyroid panel over the last 3 years since on Lithium. ESR and thyroid antibodies were negative. A Thyroid uptake scan showed a reduced uptake of 1 % at both the 6-hour and 24 hour readings. He had an elevated thyroglobulin at 80.7ng/dl (1.3-31.8) compatible with thyroiditis and ruling out factitious thyrotoxicosis. A diagnosis of thyroiditis related to Lithium was made. Lithium was discontinued. At a two month follow up appointment he was completely asymptomatic with normalization of TSH at 1.15mcunit/ml, but a thyroglobulin level of 116.8ng/ml. A repeat thyroid uptake scan showed normalization with readings of 22 % and 43 % at 6 and 24 hours, respectively.

Discussion: The etiology of Lithium induced hyperthyroidism is not certain: It is hypothesized to be related to thyroiditis secondary to increased autoimmunity evidenced by elevated antithyroid antibodies and increased B-cell activity in some of the cases. Other studies, however, have failed to detect any difference in the prevalence of autoimmunity and the probable mechanism is thought to be a direct toxic effect of lithium on the thyroid gland similar to Amiodarone with non-inflammatory ultra-structural lysosomal and mitochondrial damage. Treatment is mainly symptomatic. In our case we decided to stop Lithium and the patient had a full recovery of his thyroid function with normalization of thyroid uptake

Conclusion: Management of Lithium induced hyperthyroidism depends on whether it is immune mediated or not. In the latter case, we can conclude that stopping Lithium should be recommended.

Abstract #1158

THYROID DYSFUNCTION WITH ANTI CANCER DRUGS

Radha Devi, MD, Parakkal Deepak

Objective: Ipilimumab is a monoclonal antibody against cytotoxic T lymphocyte antigen 4 (CTLA-4) approved for use in metastatic inoperable melanoma. Bevacizumab is a monoclonal antibody that inhibits vascular endothelial growth factor A (VEFG-A). It has been used in combination with Ipilimumab for metastatic inoperable melanoma. There have been case reports of thyroiditis and Graves' ophthalmopathy with Ipilimumab and hypothyroidism with Bevacizumab. We examined the US food and drug administration's (FDA) Adverse Event Reporting System (AERS) database to identify cases of thyroid dysfunction with Bevacizumab and Ipilimumab.

Methods: The FDA (AERS) is a publicly available computerized database which utilizes voluntary reports by consumers and health professionals, to support post marketing surveillance for all approved drugs. We performed a search of the FDA AERS database from January 2004 - June 2011 using the drug search terms "Avastin" ,"Bevacizumab" ,"Yervoy" and "Ipilimumab" to search for thyroid dysfunction as an adverse event using wild cards to obtain maximum results.

Results: A total of 27 cases of thyroid dysfunction were obtained of which one was reported to be secondary to Ipilimumab and 26 cases were reported to be secondary to Bevacizumab. There were 3 cases (11%) of autoimmune thyroiditis, 12 (44%) of hypothyroidism, 3 (11%) of hyperthyroidism and 9 (33%) were unspecified (abnormal thyroid function tests and disorders). The mean age of the patients' was 63 years (28-80 yrs), and the mean weight 80.5 kg. Cases for which gender data were present revealed a gender distribution of 12 men and 11 women. Bevacizumab being in use for a longer period may account for the higher number of cases with this agent.
Discussion: Graves' disease is an autoimmune thyroid disease. Multiple genetic loci have been postulated in the pathogenesis of Graves including the CTLA4 gene that inhibits proliferation of activated T cells. Blocking this receptor through Ipilimumab may lead to increased T cell activation. Bevacizumab may cause impairment of thyroid function by regression of the capillaries around the thyroid follicles. However, AERS cannot be used to establish causal association.
Conclusion: Our study highlights the importance of having a high clinical suspicion for thyroid dysfunction when patients are on these anticancer drugs. Thyroid dysfunction may not only cause symptoms for the patient but can also alter drug metabolism. All patients must have thyroid function tests evaluated prior to initiating treatment with these agents and clinicians must monitor patients for signs and symptoms of hypo or hyperthyroidism.

Abstract #1159

RIEDEL THYROIDITIS ASSOCIATED WITH INTENSE CERVICAL PAIN -CASE PRESENTATION

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Objective: Introduction. Riedel thyroiditis is a very rare form of thyroid disease of unclear etiology, characterized by a firm painless goiter due to extensive fibrosis of the thyroid gland. Aim. We describe the case of a patient with Riedel thyroiditis causing extreme cervical pain.
Case Presentation: Case presentation. At initial presentation the patient (a 30 years old woman) presented with a large asymmetrical goiter (predominant development of the left lobe) with very rapid progression (in the last month before admission) and very intense cervical pain. Initial investigation disclosed a large, very firm but mobile asymmetric goiter, associated with severe hypothyroidism, high ESR (80 mm/h) and very low thyroid iodine uptake (1% at 24h). No hoarseness or dysphagia, no local inflammatory signs were noticed. The thyroid fine needle aspiration biopsy revealed an intense chronic scleroatrophic inflammatory reaction. A diagnosis of subacute thyroiditis episode on the background of chronic thyoiditis (supported by high ATPO titer and ultrasonographical appearance) was done and oral corticotherapy as well as levothyroxine therapy were initiated with good clinical response. 2 months later the patient was euthyroid, ESR became normal but mild cervical pain persisted under high doses of corticotherapy. One year after the initial presentation, because recurrence of intense pain prevented any attempt to decrease the dose of prednisone, surgical therapy was indicated and total thyroidectomy was performed. The pathological examination diagnosed Riedel thyroiditis. After surgery, cervical pain disappeared and severe hypoparathyroidism was diagnosed (calcium level 4.4 mg/dl, phosphate7.7 mg/dl). For the adequate correction of hypoparathyroidism high doses of calcitriol (2 micrograms daily) and calcium (4g daily) were needed.
Discussion: Fine-needle biopsy in patients with Riedel thyroiditis describes fibrotic changes in the thyroid gland that cannot always be reliably distinguished from those associated with other thyroid diseases. Therefore, histological confirmation is essential for establishing the correct diagnosis.
Conclusion: The presence of intense cervical pain can occur in patients with Riedel thyroiditis and, although it is alleviated by corticosteroids, it can represent an indication for surgical management. Due to the invasive fibrotic nature of the disease local complication of thyroid surgery are more likely to occur.

Abstract #1160

THYROTOXICOSIS NECESSITATING TOTAL PLASMA EXCHANGE (TPE), A NOVEL THERAPY DUE TO FAILURE OF STANDARD TREATMENT: 2 CASES.

Lakshmi Goudar, MD, Donald Richardson, MD, FACE, FACP, M. Mason, MD

Objective: In patients presenting with thyrotoxicosis, medical management typically consists of thionamides, beta-blockers, steroids and "cold iodine", depending on the etiology of the presentation. However at times, patients are so acutely ill and standard treatment is not sufficient in reducing their free hormone levels or symptoms. We present 2 cases of patients who presented in thyrotoxicosis where the above treatments were insufficient and therefore total plasma exchange (TPE) was instituted to reduce their hormone burden.
Case Presentation: Patient EW initially presented with an undetectable TSH, free T4 of >7.7 and free T3 of 27.1.

After initiation of medical therapy including methimazole, steroids, beta-blocker and cholestyramine, she neither improved clinically nor based on lab values. Based on her clinical history and her radiographic findings, it was thought she had amiodarone induced thyrotoxicosis in the setting of multinodular goiter. Due to her lack of response to medical therapy, and high endogenous iodine concentration due to amiodarone precluding I131 therapy, thyroidectomy was thought to represent the best therapy for her. However, given her hyperthyroid state with resultant difficult to control hypertension and hyperglycemia, this was not considered safe, and therefore TPE was instituted to bridge her to thyroidectomy. Her free hormone levels decreased and she underwent surgery without incident. Patient CP initially presented with an undetectable TSH, free T4 of 4.2 and free T3 of 9.3. After initiation of medical therapy including methimazole, steroids, beta-blocker, and cold iodine, she also failed to improve clinically or based on lab values. Based on her clinical history, positive antibodies and radiographic findings, it was thought she had Grave's disease. Due to her lack of response to therapy, and her resultant heart failure, thyroidectomy was thought to be the best therapy for her. Complicating her case was liver failure that obligated the withdrawal of methimazole. In order to get her euthyroid for surgery, TPE was instituted and she completely normalized her thyroid hormone levels and symptoms improved. Unfortunately, she developed multi-organ system failure before surgery and died.

Discussion: In difficult to control thyrotoxicosis, there are unusual therapies which can efficiently reduce thyroid hormone levels when the standard modalities fail. TPE was demonstrated in both cases above to reduce thyroid hormone levels and symptoms. TPE is discussed, as well as other less common agents, such as cholestyramine and lithium.

Conclusion: TPE should be considered as a second-line treatment as a bridge to thyroidectomy when conventional therapy is not effective.

Abstract #1161

SUBCLINICAL HYPOTHYROIDISM AND METABOLIC SYNDROME IN WORKERS OF AN URBAN HOSPITAL IN PERU

Juan Lizarzaburu, Victor Cornetero

Objective: To determine the prevalence of subclinical hypothyroidism and metabolic syndrome in workers of a hospital in Lima - Perù, and the prevalence of subclinical hypothyroidism in the group with metabolic syndrome. To determine the relation between subclnical hypothyroidism with metabolic syndrome components and body mass index.

Methods: A total of 69 workers of the Solidaridad Comas Hospital in Lima -Perú with no past history of thyroid disease, diabetes mellitus diagnosis, cardiovascular disease, high blood pressure and stroke were included in the study. Exclusion criteria: pregnancy and subjects who did not accept to participate in the study. Variables: Thyroid stimulating hormone (TSH), free thyroxine (FT4), body mass index (BMI), metabolic syndrome (MS) based on the International Diabetes Federation (IDF) definition. Subclinical hypothyroidism (SHC) was defined as a condition with high TSH and normal FT4 levels.

Results: The mean age of the participants was 32, 32 (SD ± 11,67) years, 21 (30,43%) males and 48 (81,4%) females. The prevalence of SHC was 14,5% (10 patients , 80% were females), and metabolic syndrome 34,78% (24 patients, 83.3% were females) The prevalence of subclinical hypothyroidism in the group with metabolic syndrome was 14,28 % (3 patients) and 15,55% (7 patients) in the group without MS. In the participans with MS we did not find significant differences in the mean of metabolic syndrome components: HDL cholesterol (P=0,569), triglycerides (P=0.891), fasting glucose (P=0.572), systolic blood pressure (P= 0.780), diastolic blood pressure (P = 0.820), abdominal perimeter (P=0.904), and BMI (P= 0,43) in the group with and without SCH.

Discussion: Subclinical hypotiroidsm is prevalent condition in adult population and more frequent in women. We found a higher prevalence compare with the literature's. There are some reports that suggest association between subclinical hypotirodism and metabolic syndrome and other an association with BMI. In our study we did not find this association.

Conclusion: The prevalence of subclinical hypothyroidism and metabolic syndrome was high. The prevalence of subclinical hypotiroidism was lower in the group with metabolic syndrome in the population study. There is no association between subclinical hypothyroidism and components of metabolic syndrome in the group with metabolic syndrome.

Abstract #1162

A CHANGE IN THYROGLOBULIN ANTIBODY (TG-AB) ASSAY RECLASSIFIES SOME PATIENTS FROM TG-AB NEGATIVE TO TG-AB POSITIVE AND OTHERS FROM POSITIVE TO NEGATIVE, WITH POSSIBLE CLINICAL CONSEQUENCES

Bryan McIver, MD, PhD, Diane Donegan, Stefan Grebe, Alicia Algeciras-Schimnich

Objective: To assess the impact of a change in thyroglobulin antibody (Tg-Ab) assay on apparent disease status of patients with low-risk thyroid cancer.

Methods: We assessed disease status in 812 thyroid cancer patients whose Tg and Tg-Ab status were measured during the 6 months after introduction of a replacement Tg-Ab assay (Roche, Indianapolis, IN), necessitated by withdrawal from the market of the previously used assay (Beckman Coulter Inc, Chaska, MN). The replacement assay was selected because of excellent agreement between the two assays in a study of 130 patients, with 95% positive agreement and 96% negative agreement, based on the laboratory established cut-off for antibody positivity.
Case Presentation: Amongst 812 patients, 751 patients remained concordant following the assay change (unchanged Tg-Ab status). However, in 6 (0.7%) patients a previously positive Tg-Ab became negative with the new assay, with no change in the patients' disease status. In 55 (6.8%) patients, a previously negative Tg-Ab became positive with the new assay. Once again, however, there was no evidence for change in any of these patients' disease status.
Discussion: American Thyroid Association (ATA) guidelines for thyroid cancer management recommend annual clinical evaluation and thyroglobulin (Tg) measurement as the sole means for long-term surveillance of low-risk patients. Because Tg-Ab interfere with the accuracy of Tg measurements, lowering the measured Tg concentration in most immunometric assays, the ATA recommends the use of neck ultrasound in patients with positive Tg-Ab. As a result of a change in Tg-Ab assay 61 of 812 patients (7.5%) exhibited a change in antibody status with no other evidence of altered disease status. Most of these patients, previously classified as being in remission, would be reclassified as having possible residual disease according to ATA criteria. In none of these cases did imaging document residual or recurrent disease. Nevertheless, these newly antibody-positive patients may now be subjected to a different recommended follow-up protocol.
Conclusion: Thyroglobulin antibodies measured by any of the available assays represent a subset of the Tg-Ab spectrum and no assay is capable of detecting all antibodies. In our study, 7.5% of patients experienced a Tg-Ab status change with a change in assay. Impaired Tg assay sensitivity occurs even with these undetected antibodies. The use of Tg / Tg-Ab as the sole determinant of these patients' disease status is inappropriate, and would offer false reassurance and delay the diagnosis of recurrent disease, with possible serious consequences. The use of neck ultrasound is appropriate, even in patients with undetectable Tg and apparently negative Tg-Ab.

Abstract #1163

GRAVES DISEASE: EFFECTIVE DOSING OF CARBIMAZOLE (CMZ) AT DIAGNOSIS OF HYPERTHYROIDISM AND OPTIMAL SUBSEQUENT FOLLOW UP INTERVAL POST-CMZ INITIATION

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Objective: Background and Aims: Graves' disease is the commonest cause of hyperthyroidism. The initial starting dose of carbimazole (CMZ) is often estimated arbitrarily by the physician based on a combination of clinical experience and a number of factors tenuously backed evidence. Very often upon subsequent review after a variable period, patients either remain hyperthyroid or become hypothyroid from overtreatment. This retrospective study was undertaken to look at optimal effective CMZ doses at initiation, time interval to review post-initiation of CMZ and possible predictive factors for an early response to CMZ.
Methods: Retrospective records of 71 patients managed by a two endocrinologists were scrutinized with respect to free T4, TSH, TRAb, sex, age, weight, ethnicity, initial clinically estimated goiter size, CMZ initiation dose, and follow up free T4 levels at 6 weeks, 3 months and at 6 months. ANCOVA analysis was done to look at the relationship of change of free T4 at 6 weeks, 3 months and 6 months from baseline with initial goiter size, TRAb, initial free T4, age and weight after adjustment for the initial CMZ dose.
Results: A statistically significant association was seen at 3 months and at 6 months with the initial free T4 concentrations ($p<0.005$) even after adjusting for all the variables. A statistically significant association was also seen with large goiter size at 3 and 6 months suggesting a poorer response in patients with large goiters.
Discussion: From this study, two crucial factors -initial Free T4 and the goiter size have been identified which can help in predicting the free T4 levels with a given carbimazole dose at 3 and 6 months. The initial free T4 maybe suggestive of the actual activity of the thyroid gland. Unfortunately, we didnt measure free T3 levels which may also indicate the activity of the gland. The initial goiter size is also predictive, hence a small thyroid gland may indicate an early response and a large gland a more resistant response.
Conclusion: This suggests that a crucial factor in the consideration of the initial CMZ dose and review interval post-initiation in patients with Graves' disease is the goiter size and the initial free T4 concentrations. Patients whose thyroid glands are small and who have relatively low free

T4 levels are best reviewed early or given smaller doses of CMZ. Prospective trials with more objective assessment of thyroid volume by ultrasonography, and free T3 levels may clarify our preliminary findings.

Abstract #1164

PENDRED SYNDROME: AN INTRIGUING DEVELOPMENTAL ANOMALY COMBINING GOITER, HYPOTHYROIDISM AND CONGENITAL DEAFNESS

Saleh Aldasouqi, MD, Tyler An, Ala Elayyan, Vincent Cracolici, Deepthi Rao, MD, Sameer Ansar, MD, Bhavini Bhavsar, MBBS, M.D

Objective: Over 2/3 of patients with Pendred syndrome (PDS) have goiters, and about ½ have hypothyroidism. Since PDS is the principal cause of genetic hearing-speech disorders (70%), it is prudent to check the thyroid function test (TFT's) in all patients with hearing-speech disorders, especially children and young adults. We present a case of PDS to underscore the close association with the thyroid.
Methods: Case Presentation and review of the literature.
Case Presentation: A 20 year old man was referred to the endocrinology clinic for a large goiter. The family were not certain about the duration of the goiter. TFT's had demonstrated primary hypothyroidism, 2 years prior, and he had been taking levothyroxine. The patient was deaf-mute. He had no other features to suggest specific genetic or congenital disorders. His goiter was quite large, causing compression and dysphagia, warranting surgical resection in view of no response to thyroid replacement. No diagnosis was known to the family, in regards to his deafness. In retrospect, a paternal grandfather (deceased) was reported to be deaf-mute. A presumed diagnosis of PDS was made, and confirmed by genetic testing. The family appreciated the knowledge about the diagnosis, being a genetic disorder, and planned to share this knowledge with all family members.
Discussion: PDS is the commonest cause of congenital deafness. It is inherited as an autosomal recessive trait; the gene for the disease (PDS gene) was localized on chromosome 7. The thyroid connection is believed to be via a partial defect of organification, which can be diagnosed by the classical Perchlorate test. This defect may result in goiter (73%) of variable size, and hypothyroidism (47%) of variable degrees. TFTs may be normal at birth, and hence the importance of monitoring of TFTs in all patients with hearing or speech disorders, especially in children, in order to diagnose subtle or subclinical hypothyroidism which may affect growth and development, as well as cognitive function and school performance. Similarly, meticulous examination of the thyroid (with sonography if indicated) is recommended, in order to detect goiters that can be compressive such as the case in our patient. Diagnosis of PDS is prudent to help adults in pre-marital screening and family planning, especially in communities where consanguinity is common.
Conclusion: PDS is intriguely associated with goiter and hypothyroidism. Since PDS is the commonest cause of congenital deafness-mutism, all patients with congenital auditory-speech disorders, should undergo meticulous examination of the thyroid anatomy and function, especially in early life. This is prudent to detect and treat subtle hypothyroidism and goiter, and for family planning.

Abstract #1165

A UNIQUE COMBINATION OF HASHIMOTO'S DISEASE, GRAVES'DISEASE AND VITILIGO IN A PATIENT WITH VOGT-KOYANAGI-HARADA SYNDROME

Saleh Aldasouqi, MD, Bhavini Bhavsar, MBBS, M.D, Sameer Ansar, MD, Satish Chandolu, Tyler An, Shaza Khan, MD, Deepthi Rao, MD

Objective: To report a rare case of Vogt-Koyanagi-Harada (VKH) Syndrome in a patient with Hashimoto's disease (HD), Graves' disease (GD), Graves' ophthalmopathy (GO), and vitiligo, to increase awareness about possible retinal involvement in patients with multiple autoimmune disorders, especially in patients with GD with visual symptoms.
Methods: Case presentation and literature review.
Case Presentation: This is a 33 year old woman from Taiwan was diagnosed with GD in 2006, who also had positive thyroperoxidase antibodies (HD) and vitiligo. Her hyperthyroidism was mild and controlled with antithyroid medications. She also had GO, which required plastic corrective surgery. She recently developed diplopia and partial visual loss. MRI of the orbit revealed findings suspicious for retinal detachment. She was seen by a neuro-ophthalmologist who diagnosed a retinal inflammatory disorder, consistent with VKH syndrome, which was confirmed later with a positive HLA-DR4 testing. She was started on oral steroids, on which she noticed improvement in vision.
Discussion: VKH syndrome is a rare systemic autoimmune disorder affecting melanocyte containing tissues in the ocular, auditory, integumentary and central nervous systems. It is more common in Asians, Latin Americans, Native Americans and African Americans. The pathogenesis of VKH is thought to be related to T-cell mediated autoimmune reaction against antigen components shared by uveal, dermal and meningeal melanocyte. It has been linked to HLA-DR4 and HLA-

Dw53. Clinical menifestations include a prodromal phase similar to a viral infection, ocular symptoms due to uveitits, retinal detachment and cutaneous symptoms eg, hair loss, poliosis, vitiligo. Differential diagnoses of the ocular manifestations include sympathetic ophthalmia, sarcoidosis, primary intraocular B-cell lymphoma, posterior scleritis, and uveal effusion syndrome. Treatment of inflammatory ocular manifestations is with oral steroids. Patients may require immunosuppressive therapy if they fail to respond to steroids. VKH syndrome has rarely been described in patients with type 1 diabetes mellitus, and other autoimmune disorders. It has also been described in GD and HD. Our patient has GD, HD, vitiligo and VKH syndrome, an extremely rare combination of autoimmune disorders.

Conclusion: VKH is a rare autoimmune syndrome which can be associated with other endocrine autoimmune disorders such as autoimmune polyglandular syndrome, autoimmune thyroid disorders and diabetes mellitus. In patients with GD who develop ophthalmic manifestations, in addition to GO as the usual differential diagnosis (etiology), it is prudent to keep VKH syndrome in mind, as a rare autoimmune manifestation, in view of potential vision loss.

Abstract #1166

A UNIQUE CASE OF UNFOLDING GRAVES' DISEASE PROVIDES A SIMPLE MODEL FOR A BETTER UNDERSTANDING OF THE PATHOGENESIS OF THYROID AUTOIMMUNITY

Saleh Aldasouqi, MD, Bhavini Bhavsar, MBBS, M.D, Sameer Ansar, MD, Deepthi Rao, MD, Ala Elayyan, Satish Chandolu, Tyler An

Objective: Spontaneous Graves' disease (GD) is typically diagnosed by incidental or targeted testing, and no case has been described as unfolding with real-time observation. The study of the pathogenesis of GD remains retrospective, based on historical and laboratory correlation. We report a case of GD that was meticulously recorded as it unfolded, which confirmed the speculated mechanisms of thyroid autoimmunity.

Methods: Case Presentation.

Case Presentation: A 32 year old female was referred for painful neck swelling, and abrupt onset of hyperthyroid symptoms (February). She was euthyroid clinically, with a firm, enlarged and tender thyroid. Her thyroid function tests (TFT's) showed: TSH 0.05 mIU/ml; free T4 3.7 ng/dl; free T3 13.3 pg/ml (normal ranges: 0.4-5.1; 0.8-1.8; 2.3-4.2, respectively). TRAB and TSI were undetectable (<1.00 IU/L and <1.0, with reference ranges of 0-1.75 IU/L and TSI index <=1.3, respectively). ESR was 91 mm/hr. Two weeks prior, she had a viral upper respiratory infection (URI), with a normal TSH (1.00). A diagnosis of De Quervain thyroiditis (DQT) was made, which improved with ibuprofen. In April, she felt tired, and her TSH became 69.8, with low free T4 and T3 (0.63, 2.2), and she was started on levothyroxine, 75 mcg daily. In June, and while euthyroid clinically, her TSH became suppressed (<0.01), with elevated free T4, T3 (2.47, 7.1). It was unclear if this was iatrogenic (patient was very thin), or ensuing de-novo hyperthyroidism. Levothyroxine was stopped, and she was lost for follow up. In October (4 months off thyroxine), her TSH was suppressed, but with normal T3 and T4 (1.2, 2.7), suggesting endogenous, spontaneous hyperthyroidism due to GD, in view of seroconversion of TRAB and TSI, which were then elevated (2.23 and 4.0, respectively). She was not treated, and is being monitored clinically and biochemically.

Discussion: TRAB and TSI are believed to cause hyperthyroidism, but the triggers for their formation are still elusive, believed to be the result of an interaction of genetic and environmental factors, including viral infections. However, these postulations were based on retrospective evaluation. Our patient developed an URI consistent with a viral illness, with normal TSH, followed by painful thyroiditis (DQT) with hyperthyroidism, with negative antibodies (TRAB, TSI), and then during the recovery stage, she developed hyperthyroidism with seroconversion of TRAB and TSI.

Conclusion: This case supports the speculation that a viral infection affecting the thyroid gland directly (in view of the rapid onset of hyperthyroidism), exposes the thyroid antigens (severe inflammation) to the immune system, which then will initiate the auto-antibodies in genetically vulnerable subjects.

Abstract #1167

A PROPOSAL FOR ROUTINE ULTRASOUND SCREENING OF ALL PATIENTS WITH GRAVES' DISEASE IN VIEW OF INCREASED RISK AND AGGRESSIVENESS OF ASSOCIATED PAPILLARY THYROID CANCER: A CASE SERIES AND REVIEW OF THE LITERATURE

Saleh Aldasouqi, MD, Bhavini Bhavsar, MBBS, M.D, Sameer Ansar, MD, Mamata Ojha, Shaza Khan, MD, Tyler An, Satish Chandolu

Objective: In patients with Graves' disease (GD), the prevalence of palpable thyroid nodules is approximately threefold higher than in the general population. Several studies indicated that there is an increase risk and aggressiveness of papillary thyroid cancer (PTC) in patients with GD. The objective of this case series is to review

the relevant literature and point out to the importance of routine ultrasound (US) screening in patients with GD, who are usually evaluated with radioactive iodine (RAI) scans, which can miss smaller nodules embedded in big goiters, typically seen in GD.

Methods: Case Series Study: We report 7 cases of GD associated with PTC, which were recently seen in a single endocrine clinic.

Case Presentation: All patients were women aged 27 to 65 years, who all had elevated serum thyroid stimulating immunoglobulin levels. Only one patient had history of thyroid cancer in her grandmother, and none had history of radiation exposure to the head or neck. RAI scanning, which is the standard imaging method in patients with GD, done in 5 patients, identified a cold nodule in 2 cases only, while US identified all suspicious nodules. Five patients underwent US-guided fine needle aspiration (FNA) which showed findings suggestive/consistent with PTC and 2 patients skipped FNA. All 7 cases were confirmed surgically. Tumor size ranged from 0.8 to 1.6 cm, but below 1 cm in 5 patients. The cancer was multifocal in two patients, and was locally metastatic in 1 patient and distally metastatic in another patient (questionable miliary pulmonary uptake on RAI scan). Three patients required 2 doses of RAI treatments.

Discussion: Malignant thyroid nodules are present in 1.7-2.5% of patients with GD but only 0.25% of general population, i.e. 6.8-10 times more in patients with GD. RAI scan which is the standard radiologic procedure in GD can miss small thyroid nodules and cancers. Also GD patients usually have moderate or large goiters and smaller embedded nodules can escape physical exam; studies have shown inaccuracy of clinical exam of the thyroid in over 50% of cases. Furthermore, US is not routinely used in GD. Concomitant GD is considered to be a negative prognostic factor in a patient with differentiated thyroid cancer. High levels of thyroid receptor antibodies might stimulate thyroid cancer growth and promote early metastatic spread, thus negatively affecting patient outcome.

Conclusion: This study demonstrates the importance of routine US scanning in all patients with GD, in view of the documented increased risk and aggressiveness of differentiated thyroid cancer in patients with GD. Smaller nodules embedded in large goiters can escape physical exam and RAI imaging, the standard imaging modality in GD.

Abstract #1168

PROGNOSTIC IMPLICATIONS OF BRAF MUTATION AND PAPILLARY THYROID CANCER

Sandra Weber, MD, Steven Trocha, Stanley Von Hofe, MD, Danniel Nichols, Pressley Merchant

Objective: To determine the utility of BRAF mutation determination in the analysis of papillary thyroid cancer in an active endocrinology practice.

Methods: Review of all BRAF mutation results beginning with the first sample sent in August 2009 and chart review of all cases of papillary thyroid cancer during the corresponding time period.

Case Presentation: From August 2009 through June 2011, 92 patients with pathologically determined papillary thyroid cancer underwent thyroidectomy at our institution. The age ranged from 21-95 years, 79 (85.9%) female and 13 (14.1%) male. The ethnicity was 79 Caucasian, 2 African American, 3 Hispanic, 1 Asian, and 7 Unknown. Of the 92 patients, 1 of 13 (7.7%) in 2009, 11 of 51 (21.6%) in 2010 and 17 of 28 (60.7%) in 2011 had BRAF analysis. Of the 29 specimens were sent for analysis, the first sample sent 8/14/09. BRAF was positive in 12 (41.4%), negative in 7 (24.1%) and unknown or unclear in 9 (31%). The reports for the unknown or unclear reports were as follows: Can not be excluded (2); Tumor component small in sample, could be false negative; Could not sample (2); No mutations in 12/15, unable to obtain sample in 11; No mutation at codon 600 of exon 15, other sequences unreadable; No PCR amplified product obtained after repeated attempts; Insufficient or degradation of nucleic acid. Tumor analysis was available in 87 of the 92 specimens. Forty eight were 1.0 cm or less, 33 were 1.1-4.0 cm, 6 were greater than 4.0 cm; 6 were multifocal. Of the 12 BRAF positives cancers: 1 was 1.0 cm or less, 9 were 1.1-4.0 cm, 2 were greater than 4.0 cm; 1 was multifocal. The cost of a thyroid BRAF varied depending on whether the surgery was done as an inpatient, at a cost of $1461.11, or as an outpatient, at a cost of $895.90.

Discussion: BRAF positive mutations were found in all tumor size categories, single and multifocal tumors, and throughout the age range. The yield for BRAF mutation analysis was not ideal with 31% having unknown or unclear results. The cost of BRAF mutation analysis is high.

Conclusion: BRAF mutation analysis may have future implications in the determination of best therapy for patients with papillary thyroid carcinoma. Future studies will need to address how to apply information obtained as well as determine cost effectiveness.

Abstract #1169

HUMERUS METASTASIS AS THE FIRST CLINICAL SIGN OF FOLLICULAR THYROID CARCINOMA

Simona Fica, MD, PhD, Sorina Martin, Ramona Olaru, Dana Terzea, Dumitru Ioachim, Mircea Ghemigean

Case Presentation: We present the case of a 67 years old men, known with high blood pressure, diabetes mellitus and dyslipidemia, with no personal or family history of thyroid disorders, who first presented to the general practitioner for pain in the right humeral region. Right shoulder X ray, MRI and whole body bone scan showed 50/35/50 mm osteolytic tumor of the right humeral head. CT scan revealed no lung, pelvis or abdominal metastasis. The histological exam and IHC after the bone biopsy revealed that the origin of tumor is in thyroid follicular epithelium. The patient was referred to our clinic for further investigation. Thyroid ultrasound: 12/8mm left lobe hypoechoic nodule with irregular borders, without peripheral halo and increased intranodular vascularity. TSH, calcitonin, antitireoglobulin and TPO antibodies levels were normal. Total thyroidectomy and lymph nodes neck dissection was performed. The histological exam showed follicular type thyroid carcinoma with capsular and vascular invasion. The patient was referred to the oncology and nuclear departments for subsequent treatment (radiotherapy/ radioiodotherapy) and follow up.
Discussion: Follicular carcinomas are the second most common thyroid cancer (10%). Vascular invasion is characteristic for follicular carcinomas and therefore distant metastasis are more common (11-25%) than in other types of thyroidian cancer with lung, bone, brain, liver and skin being the potential sites of distant spread.
Conclusion: Even if distant spread to bones is an uncommon first clinical sign in thyroid follicular carcinoma, we should consider thyroid gland exam in the setting of a single bone metastasis of unknown origin.

Abstract #1170

A FAKE THYROID NODULE: AN UNUSUALLY LONG AND OBLIQUELY LOCATED PYRAMIDAL LOBE MASQUQRADING AS AN ISTHMIC THYROID NODULE

Saleh Aldasouqi, MD, Bhavini Bhavsar, MBBS, M.D, Sameer Ansar, MD, Deepthi Rao, Tyler An, Shaza Khan, MD, Satish Chandolu

Objective: Ultrasonography (US) is a very helpful diagnostic tool in clinical practice, but unlike all other imaging modalities, US is entirely operator-dependent. Unless, a clinician or radiologist is directly supervising an US study, the ultrasonographer is the ultimate decider of the study findings, and by manipulation of the US transducer, these findings can be tricky. Experience of sonographers is thus of paramount importance for accurate US imaging. In the case of thyroid US, the distinction between real nodules and "fake" nodules can be difficult, and challenging. We present such a difficult case.
Case Presentation: A 64 year old woman was referred for US-guided fine needle aspiration (FNA) of an incidentally discovered 1.8 cm nodule in the isthmus. The patient had hyperthyroidism, treated with methimazole. Review of the prior US images confirmed the presence of a well-defined 1.76 x 1.26 x 0.85 cm nodule in the region of the isthmus (see images). During the pre-FNA real-time scanning, the sonographer initially had confirmed the prior findings, but upon more meticulous scanning with more fine adjustment of the transducer, the sonographer discovered that this was not a real nodule. In both the transverse and longitudinal applications, the lesion would maintain its integrity as a nodule, but as the transducer is manipulated further, obliquely, the lesion turns into an elongated structure, traversing from the medial end of the right lobe medially and superiorly crossing the midline and ending in the sub-hyoid region: This lesion turned out to be a pyramidal lobe. It was unusually long and obliquely oriented, and thus it was easily mistakable for a real nodule in both transverse and longitudinal orientations. The FNA was cancelled.
Discussion: Thyroid pyramidal lobe is a normal thyroid tissue that buds out of the upper end of either lobe or isthmus as an elongated structure, and is present in ~ 80% of the population, but with variable length and orientation. It represents a remnant of thyroidal tissue during thyroid decent during embryogenesis. Although common, they are rarely reported on US imaging, as we observed over the years. If a pyramidal lobe has an unusual location or orientation, it may present diagnostic difficulties, and it can easily be misdiagnosed as a thyroid nodule, leading to unnecessary FNA. This case illustrates this diagnostic difficulty. The experience of a sonographer is of utmost importance in avoiding such mis-diagnosis.
Conclusion: Thyroid pyramidal lobes may be misdiagnosed as thyroid nodules, requiring meticulousness and vast experience of sonographers to avoid such circumstances.

Abstract #1171

A RARE CASE OF REVERSIBLE CARDIOMYOPATHY MANAGED SUCCESSFULLY.

Nidhi Bansal, MBBS, Luke Yuhico, Barbara Krenzer

Case Presentation: Dilated cardiomyopathy is responsible for approximately 10,000 deaths and 46,000 hospitalizations annually in the United States. Only a small proportion of these cases are reversible. A heterogeneous group of metabolic, toxic and infectious etiology are responsible for reversible cardiomyopathy. We present a unique case of a 48-year old African American female with past history of hypertension and bronchial asthma, who was admitted with gradually progressive exertional dyspnea of 4 month duration. There was no history of orthopnea, recent pregnancy, viral illnesses, alcohol or drug abuse. Physical examination revealed tachycardia, jugular vein distention, positive hepatojugular reflex, no thyromegaly, S3 gallop, diminished right basilar air entry and bibasilar crackles. Preliminary labs including complete blood counts and metabolic profile were unremarkable. Chest X-ray showed cardiomegaly with right pleural effusion and prominent pulmonary vascular markings. Echocardiogram showed an ejection fraction of 25%-30% with global hypokinesis of the left ventricle consistent with cardiomyopathy. Extensive work up for cardiomyopathy including cardiac enzymes, iron studies, hemoglobin A1c, alcohol level and urine drug screen were also noncontributory. Endocrine work up revealed very low TSH at <0.030 uU/ml (0.270-4.200 uU/ml), free T3 577.10 ng/dl ((80.00-200.00 ng/dL) and free T4 > 4.50 ng/dl (0.90-1.70 ng/dl). She was initiated on methimazole, carvedilol, diuretics and supportive therapy with rapid resolution of symptoms. Follow up after 6 weeks showed remarkable improvement in her cardiac function,thyroid function profile with restoration of her baseline functional status. ***Discussion***: Heart failure can be the initial presentation in approximately 6 % of patients with hyperthyroidism. Exacerbation of pre-existing clinically active or silent heart failure due to hyperthyroidism accounts for most of these cases. Rarely, hyperthyroidism can manifest primarily as decompensation of a structurally normal heart as was the case in our patient. This entity is known as hyperthyroid cardiomyopathy. The underlying pathophysiology of hyperthyroid cardiomyopathy remains unclear. In most cases, it is associated with either persistent sinus tachycardia/atrial fibrillation with uncontrolled ventricular response or an alteration in gene expression in cardiac cells leading to rate-related heart failure. ***Conclusion***: Given that this is a potentially reversible cause, hyperthyroidism should be considered in the differential diagnosis of dilated cardiomyopathy, even in the absence of other stigmata of thyrotoxicosis.

Abstract #1172

A RARE CAUSE OF COUGH: METASTATIC THYROID DISEASE FROM RENAL CELL CARCINOMA

Paulina Cruz, MD, Farah Hasan, MD, Tahira Yasmeen, MD

Objective: Metastatic disease to the thyroid gland is rare. Renal cell carcinoma (RCC) accounts for approximately 3% of adult malignancies. We present a case of metastatic renal cell carcinoma to the thyroid presenting as a goiter.
Case Presentation: A 63-year-old woman was admitted with complaints of cough and an enlarging goiter over the past 2 years. Her cough was constant and occasionally had shortness of breath. She had a positive history of RCC status post left nephrectomy 11 years prior to presentation. Other history was significant for breast cancer status post left breast lumpectomy and remote history of smoking. Patient did not report any significant weight loss. There were no clinical manifestations of hypo or hyperthyroidism. Examination revealed an overweight woman in no acute distress. Her pulse was 80, respirations 8 and blood pressure 140/70. Oropharynx was clear, thyroid was diffusely enlarged and nodular but without a dominant palpable nodule. No stridor or voice changes. Rest of the examination was unremarkable. A prior FNA showed benign disease, however due to history of malignancies and recent onset of compressive symptoms patient underwent total thyroidectomy without complications. Baseline TSH was 0.7. Pathology showed metastatic renal cell carcinoma grade 2, measuring 5.5 cm in the right thyroid lobe and isthmus with surgical margins free of tumor. Patient was started on levothyroxine and sunitib, the latter had to be discontinued 8 weeks later due to pancreatitis.
Discussion: After nephrectomy for RCC, up to 50% of patients develop recurrent or metastatic disease. RCC most frequent sites for metastases are the lung, contralateral kidney, bone, liver and retroperitoneum. In the head and neck the thyroid is the most common site. Metastatic disease can occur several years after nephrectomy, with the average interval being 7.5 years. Only 2% of all thyroid malignancies are metastases. All thyroid nodules should be followed and growing nodules should be reevaluated with biopsy or surgery depending on symptoms and concerns. Therapy with sunitinib or sorafenib, both VEGF inhibitors, is recommended for metastatic RCC. When partial thyroidectomy is done or when the thyroid is not involved, TFTs should be performed, as both medications are associated with hypothyroidism. Surgical treatment is recommended and has good prognosis when the disease is a single nodule. In disseminated disease, surgery should only

be palliative in the presence of compressive symptoms.
Conclusion: Thyroid metastases, even if rare in clinical practice, should be considered as a differential diagnosis of a thyroid nodule or enlarging goiter, particularly in patients with history of RRC.

Abstract #1173

IMPENDING CARDIAC TAMPONADE WITH HYPERTENSIVE EMERGENCY AS PRIMARY PRESENTATION OF HYPOTHYROIDISM

Quang Ton, MD, Maria Molineros, Hyesoo Lowe-Shin, Andrew Weissman

Objective: To report a rare case of primary hypothyroidism presenting as massive pericardial effusion and tamponade with hypertensive emergency.
Case Presentation: A 53-year old female with no past medical history presented with dyspnea and lower extremity edema. Patient has not had medical evaluation for many years. One year prior to presentation patient began to develop lower extremity edema that became refractory over next six months. She eventually developed shortness of breath that became quite severe. On review of systems patient does endorse cold intolerance, fatigue, lethargy, and short-term memory deficits. Family history is positive for thyroid disease in mother. On examination blood pressure was 221/126 with heart rate of 78 bpm. Small goiter was palpated and mild periorbital edema noted. Cardiovascular examination revealed sinus rhythm with distant heart sounds and no jugular venous distention. Lungs had decreased breath sounds with dullness to percussion. Lower extremity edema bilaterally noted to be 3+. Remaining physical exam was unremarkable. Significant laboratory results revealed TSH 116.5 uiu/ml, free T4 less than 0.1 ng/dl, white count 3.3 k/ul, hemoglobin 9.9 g/dl, hematocrit 29.9%, total creatinine kinase 1008 u/l, creatinine kinase-MB 17.5 ng/ml, and troponin T 0.05 ng/ml. Chest X-ray showed marked cardiomegaly with left pleural effusion. CT of chest showed massive pericardial effusion. Bedside 2d echocardiogram revealed large circumferential pericardial effusion, respiratory variation of mitral valve inflow, right atrial collapse, and right ventricle collapse suggesting tamponade physiology. Patient underwent urgent subxyphoid pericardial window with 2000 cc straw colored fluid drained. Pathology reported pericardium specimen as chronic fibrous pericarditis. Additional laboratory results showed Anti-TPO 333 iu/ml, Anti-TG greater than 4000 iu/ml, and total T3 less than 25 ng/dl. Patient post-operatively was admitted to cardio-thoracic intensive care unit and started on levothyroxine 100 mcg IV and liothyronine 10 mcg oral daily. After three days of hospitalization, patient improved clinically and was discharged.
Discussion: Cardiac tamponade is a life threatening spectrum of cardiac compression severity. Hypothyroidism is a rare cause of pericardial effusions through increased capillary permeability and decreased lymphatic drainage with only about twenty cases presenting as tamponade being reported in literature. For the patient described in our report, the subsequent hypertensive emergency necessitated urgent surgical intervention.
Conclusion: Clinicians should include hypothyroidism in the differential diagnosis of pericardial effusions and not hesitate with prompt intervention.

Abstract #1174

A CURIOUS CASE OF DYSHORMONOGENETIC GOITER

Ram Jhingan, MD, Gauri Dhir, MD, Daniel Rubin, MD

Objective: To report an unusual case of a 32-year old with Dyshormonogenetic Goiter and Airway Obstruction
Case Presentation: A 32 year-old female presented to Emergency Room with acute respiratory distress secondary to a large goiter with tracheal compression and pneumonia. Her medical history was significant for hypothyroidism since 6 months of age being treated with levothyroxine. The patient had developed a progressively enlarging multinodular goiter as a teenager, but had declined surgery in the past. A CT of the neck confirmed the presence of a large goiter with marked tracheal and esophageal narrowing. Laboratory evaluation revealed TSH of 213 MIU/ml, a FT4 of 0.4ng/dl, (with absent TPO antibodies), and the patient was started on intravenous Levothyroxine. Failed extubation attempts prompted total thyroidectomy, after which she was successfully extubated and discharged home. Pathology reported a 297 gram multinodular thyroid. Histology revealed an adenomatous gland with some nodules containing papillary hyperplasia and others with oncocytic change. There were also hyperplastic solid nodules with nuclear pleomorphism and rare mitotic figures. These findings were consistent with Dyshormonogenetic Goiter(DG).
Discussion: Dyshormonogenesis of the thyroid accounts for 10-20% of all cases of congenital hypothyroidism and a majority of neonates present with a large goiter. The two most common causes for DG are the defects in organification of iodine, frequently due to TPO gene mutations, and the defects in thyroglobulin synthesis and secretion. Defective hormone synthesis results in increased TSH secretion that causes hyperplasia and hypertrophy of the follicles and subsequent goiter. Although biochemical confirmation of DG is not always possible, clinical features such as goiter presenting in infancy, family history of goiter in a nonendemic area,

and a combination of goiter and deafness can be highly suggestive. Our case was diagnosed at 6 months of age due to delayed milestones and started on treatment but developed goiter much later in life which worsened in size due to medication noncompliance.

Conclusion: To our knowledge there are only 3 other reported cases of DG presenting with airway obstruction. DG typically presents during first year of life but may present later as our patient did. It is associated with metabolic block of thyroid hormone synthesis and not with autoimmunity. Maintaining euthyroidism is extremely important in preventing goiter growth but total thyroidectomy is often required for cosmesis or compressive symptoms. Patients with DG should seriously consider prophylactic thyroidectomy to avoid future complications.

Abstract #1175

HOT NODULES AND THYROID CANCER

Hagop Kojanian, MD

Objective: It is believed that the presence of hot nodule on a radionuclide scan can almost always rule out malignancy in it, but in few cases hot nodules contain malignancy, therefore the risk of malignancy in autonomously functioning nodules is continuously underestimated in clinical practice. We present a case of 19 year old male, who had hyperthyroidism and hyperfuctioning left thyroid Lobe, which, after lobectomy was found to be papillary thyroid cancer.

Case Presentation: 19 year old male presented for evaluation of hyperthyroidism, with complains of nervousness and heat intolerance, he had a Free T4: 1.65 (N: 0.8-1.5 ng/dl), Free T3: 5.91(N: 0.2-0.5 ng/dl) and TSH: 0.01 (N: 0.52-4.89 mu/l). He denied any history of radiation exposure, weight change, hair loss, palpitation, visual change or family history of thyroid cancer. He had normal vital signs, and mild enlargement of the left thyroid lobe, otherwise unremarkable exam. His thyroid ultrasound showed a mildly enlarged left lobe with lobulated contour and diffuse punctuate areas of increased echogenicity, replacing much of the left lobe, and a normal appearing right lobe. His (I-123) Uptake and scan showed a hot nodule replacing the whole left lobe, no discrete cold nodules, and increased uptake: 41.3% at 4 hours (N: 5-15%), and 71.4 % at 24 hours (N: 15-35%), only on the left. He decided to have left lobectomy as treatment, and the pathology of the removed lobe showed: Unencapsulated Papillary thyroid carcinoma with lymphovascular invasion, so he had completion of thyroidectomy, and the pathology of the right thyroid and isthmus showed no malignancy.

Discussion: Autonomous nodules account for only 5-10% of palpable nodules. But few patients with autonomous nodules have been found to have thyroid cancer. Our patient presented with hyperthyroidism and increased I-123 uptake on the scan of the left lobe with a toxic nodule occupying the whole left lobe and supressing the right lobe. He had left lobectomy which showed a papillary cancer, followed by completion of thyroidectomy which showed no malignancy in the isthmus and the right lobe. After reviewing the literature, we found that incidental thyroid carcinoma in patients who had thyroidectomy for hyper functioning nodules are not a rare event. Major endocrine societies do not recommend biopsies of hot nodules. Radioactive iodine would have been an option to treat his toxic nodule; however we would have missed detecting the papillary thyroid cancer.

Conclusion: Since the thyroid Uptake and Scan used in the differential diagnosis of hyperthyroidism, cannot exclude malignancy within a hyperfunctioning nodule, clinicians should not ignore the risk of malignancy in those nodules.

Abstract #1176

OCCULT THYROID CANCER WITH DISTANT METASTASIS

Tulsi Sharma, MBBS, Pankaj Mehta, Roberto Izquierdo

Objective: Papillary thyroid carcinoma (PTC) generally carries a good prognosis since it usually remains intrathyroidal and tends to metastasize locally to regional lymph nodes alone. Distant metastases occur to the lungs and bone, and these are very rare especially from an occult primary.

Case Presentation: A 21-year-old lady presented to an outside facility in October 2008 after a motor vehicle accident. CT thorax was negative for any traumatic injuries; however, it revealed a diffuse reticulonodular pulmonary process. Sputum for AFB and PPD were negative and she was asymptomatic. She did not return for her follow-up appointments and returned to her PCP in November 2009 for an annual evaluation. Repeat CT thorax revealed a stable nodular pattern. She was referred to Upstate Medical University in early 2010 with possible diagnosis of sarcoidosis. She still denied any constitutional symptoms and clinical exam was normal. Repeat CT-thorax revealed persistence of the miliary pattern. Blood work, ACE-level and PPD were negative. What would cause these numerous nodules which have been stable for 2 yrs and without any clinical manifestations? After a detailed discussion the patient agreed for a bronchoscopy. Bronchoscopy with transbronchial biopsy revealed metastatic well-differentiated PTC! Thyroid sonography revealed a small solid nodule in the left thyroid lobe with internal and peripheral vascularity.

The patient underwent a near-total thyroidectomy and a limited central compartment neck dissection. The patient has since undergone radioactive iodine therapy and is on thyroid replacement therapy. Her follow up hypothyroid I131 whole body scan showed a significant decrease in uptake in the lungs.

Discussion: PTC has the best prognosis of the thyroid malignancies with a 90% 10-year survival. Even in the presence of metastatic spread, survival periods may exceed 20 years, especially in the young. The lung is the most common site for distant spread. Diffuse lung metastatic disease from an occult thyroid cancer is however extremely rare. The growth rate of pulmonary nodules is often used to help differentiate benign from malignant disease. Pulmonary nodules that exhibit lack of growth for more than two years are generally considered benign. However, nodular lung metastases from PTC are an exception and may demonstrate lack of significant growth over many years. The cause of this growth arrest is unknown but is hypothesized to occur as a result of an immune response to the cancer cells.

Conclusion: 1.Distant metastatic disease is extremely rare from an occult PTC 2.Pulmonary metastases from PTC may have prolonged periods of growth arrest.

Abstract #1177

ACTIVATING AUTOANTIBODIES TO THE β1 ADRENERGIC AND M2 MUSCARINIC RECEPTORS FACILITATE ATRIAL FIBRILLATION IN PATIENTS WITH GRAVES' HYPERTHYROIDISM

Megan Vanderlinde-Wood, M.D., Muneer Khan, M.D., Allison Galloway, DO, Hongliang Li, Xichun Yu, David Kem, MD

Objective: To expand a study which previously revealed activating autoantibodies may facilitate the initiation and maintenance of atrial fibrillation in Graves' hyperthyroidism.

Methods: Patients were categorized into 31 patients with Graves' hyperthyroidism and known atrial fibrillation, 36 patients with graves hyperthyroidism and no known atrial fibrillation, and 9 patients with toxic multinodular goiter including one with atrial fibrillation. Peptides corresponding to amino acid sequence of the second extracellular loop of human β1AR and M2R were synthesized and used to coat ELISA plates.

Results: This study confirms the increased concordance of autoantibodies to these receptors in Graves' patients with atrial fibrillation. Nearly 1/3 of patients with Graves' hyperthyroidism along with active atrial fibrillation were found to have positive autoantibodies to the second extracellular loop of the β1AR receptor and nearly 2/3 of patients demonstrated positive autoantibodies to the M2 receptor. By contrast, patients with TMNG had a low prevalence of autoantibodies directed toward these autonomic receptors. The single patient with toxic multinodular goiter with atrial fibrillation had normal autoantibody titers to β1AR and M2 receptors.

Discussion: We previously demonstrated activating autoantibodies to β1 and M2 autonomic receptors, whose activation is known to facilitate atrial tachyarrhythmias, were markedly elevated in Graves' hyperthyroidism with concurrent atrial fibrillation (Stavrakis et al. JACC 2009). Hyperthyroidism has been associated with tachyarrhythmias with sustained atrial fibrillation in 20-30% of patients even after treatment. The prevalence of AF in patients with Graves' disease as in all other forms of hyperthyroidism increases with age (1). These autoantibodies to the beta-1 adrenergic receptors and to the M2 muscarinic receptors have been demonstrated previously to increase intracellular Ca2+, shorten the action potential and hyperpolarize atrial cells. This combined sympathetic and parasympathetic stimulation generates early afterdepolarizations and rapid triggered firing in the pulmonary veins which in turn induces AF (1). Patients with TMNG in contrast to those with Graves' hyperthyroidism have a relatively low prevalence of these activating autoantibodies compatible with the non-autoimmune pathogenesis of their hyperthyroidism.

Conclusion: This study supports and expands our previous clinical evidence that activating autoantibodies to β1AR and M2R in association with hyperthyroidism facilitates atrial fibrillation.

Abstract #1178

ERLOTINIB ASSOCIATED EXACERBATION OF HYPOTHYROIDISM WITH PERICARDIAL TAMPONADE

Naga Nalini Tirumalasetty, MBBS, Tony Kastoon, MD, Craig Stump, MD, PhD, Stephen Thomson, Hussein Yassine

Objective: To report an association between hypothyroidism and Erlotinib use

Methods: We present a case report of Erlotinib associated exacerbation of hypothyroidism in one of our patients and review the relevant literature

Case Presentation: After 3 month of using Erlotinib 150 mg daily for advanced non-small-cell lung cancer, we report changes in thyroid function tests in a patient from a baseline of TSH of 8.7 mIU/L (normal 0.45-4.5) to a TSH of 260.9 mIU/L (normal 0.45-4.5). The case was complicated by cardiac tamponade. We review the literature of

tyrosine kinase inhibitor use and thyroid dysfunction.
Discussion: This is the first case report linking Erlotinib use and thyroid disease. Thyroid function test abnormalities have been reported for different TKI, most notably Sunitinib where the incidence of Sunitinib-induce hypothyroidism ranges from 36% of patients in one report (15 out 42 patients) to 52% in another report (21 out of 40 patients), the risk increased with longer duration of therapy. The exact mechanism of how Sunitinib causes thyroid dysfunction is yet to be determined. There are several proposed mechanisms that include inhibition of peroxidase activity leading to decrease in both iodination and thyroid hormone synthesis. Sunitinib has antiperoxidase activity that was about one fourth the potency of Propyllthiouracil. This observation has not yet been confirmed in vivo. Another study suggested Sunitinib induces transient hypothyroidism by blocking iodine uptake through a direct effect on sodium iodide symporter (NIS), however, an in vitro study with rat thyroid cells argued against the previous conclusion. Another potential mechanism is a destructive thyroiditis picture characterized by transient thyrotoxicosis, with increased Free T4, low TSH, increased thyroglobulin with low radioiodine uptake (as opposed to Graves' disease with increased uptake) followed by hypothyroidism.Sunitinib inhibits vascular endothelial growth factor receptors (VEGFR) as a principal mechanism of its action against tumor, however this shouldn't be the case for erlotinib where it's major target is epidermal growth factor receptors (EGFR) rather than VEGFR. In vitro data suggests that Erlotinib is a potent inhibitor of CYP1A1 and UGT1A1, however the relevance of these data has not been elucidated. Another differential diagnosis.
Conclusion: This is the first case report to describe Erlotinib associated exacerbation of hypothyroidism.
his may highlight the need for screening and monitoring of thyroid function in patients starting Erlotinib chemotherapy.

Abstract #1179

PERSISTENT AMIODARONE-INDUCED THYROIDTOXICOSIS (AIT)

Rajib Bhattacharya, MD, Kerstin Stephens

Objective: To describe an unusual case of glucocorticoid-resistant amiodarone thyrotoxicosis.
Case Presentation: This patient is a 56 year-old male who initially presented with atrial fibrillation. He had a history of paroxysmal atrial fibrillation in 2005-2006 he was initiated on amiodarone. Over five years, he was completely asymptomatic and remained on amiodarone without follow up. The patient had then discontinued the therapy. Later, the patient presented to cardiology and reinitiated on amiodarone. The patient did have a TSH suppressed to 0.373 at that time. Six weeks later, the patient presented to cardiology again with complaints of palpitations. During the follow up the TSH was < 0.005. On presentation in endocrinology clinic, he had no significant symptoms. He had a thyroid uptake scan that showed low uptake, 4.0% at 23 hours, so radioactive iodine ablation was not considered. Initial diagnosis was type 2 amiodarone toxicity, and discontinuation of the amiodarone was recommended. He was placed on a prednisone 20 mg daily. Later a cath revealed RCA blockage, and he underwent double stent placement. He was later readmitted to the hospital for recurrent atrial fib. Repeat thyroid labs revealed TSH 0.02, total T3 188 ng/dL, and free T4 2.8 ng/dL. TSI index was 6,6. The patient was placed on methimazole 20 mg twice daily. Post hospital discharge, he continued to complain of intermittent palpitations and shortness of breath. Due to his persistent symptoms, the methimazole was increased to 20 mg three times daily, and the prednisone continued. Thyroidectomy was recommended because the patient was recalcitrant to medical therapy. However, cardiology determined he was not a good surgical candidate currently. Thyroid uptake was repeated. It demonstrated uptake of 23.6 % at 23 hours, consistent with underlying Graves'. Therefore, an ablation was done.
Discussion: AIT is a very common presentation seen by endocrinologists. However the diagnosis and management is still perplexing. A recent article described the average amount of time to a euthyroid state in individuals after discontinuation of amiodarone therapy was 47 days. This patient had persistently uncontrolled hyperthyroidism several months off of amiodarone and concurrently on methimazole. The repeat thyroid uptake and scan helped us diagnose the Graves's disease which persisted.
Conclusion: Individuals who are recalcitrant to steroid and thioamide therapy with amiodarone-induced thyrotoxicosis may need a repeat thyroid uptake and scan to reassess the underlying thyroid disorder.

Abstract #1180

DILEMMA IN MANAGEMENT OF WELL-DIFFERENTIATED THYROID CANCER IN PATIENTS ON HEMODIALYSIS

Mallika Bhat, MD, Matty Mozzor, Guy Valiquette, Monica Schwarcz

Objective: To discuss, in the absence of clear guidelines, the management of well-differentiated thyroid cancer in patients on hemodialysis, including the dose of radioiodine and the timing of hemodialysis.
Case Presentation: A 49-year-old female with a history of goiter, hypertension and end-stage renal disease, presented with obstructive symptoms. Ten years earlier, she had

undergone a left hemithyroidectomy. Examination now revealed an enlarged right thyroid. The patient underwent a completion thyroidectomy, and histopathology revealed a follicular variant of classical papillary thyroid cancer, 5.5 X 4 cm, with no lymphovascular or extrathyroidal invasion. The patient was then treated with 131I at a dose of 50 mCi. Post-treatment, she received 3 inpatient hemodialysis sessions at 12, 24 and 36 hours. She was discharged after ensuring that radiation levels met regulatory requirements. She received a total-body scan 7 days later. This showed residual radiotracer uptake in the thyroid bed with a focus of uptake in the left superior thyroid bed, confirming post treatment 131I uptake. All subsequent thyroglobulin/antithyroglobulin antibody tests have been negative, thus showing that the dose was effective. The total effective extrathyroidal dose for this patient was approximately 2030 mrem (20.3 mSv), 40% more than what a patient with normal renal function would have received for management of identical disease. This is still within acceptable limits, showing that the dose was safe.

Discussion: The most effective adjuvant treatment, after thyroidectomy, for certain well-differentiated thyroid cancers is radioiodine ablation. The American Thyroid Association recommends that the dose delivered should be the minimum dose necessary to achieve successful remnant ablation, especially for low risk patients. This is most often achieved with 30-100 mCi. Higher doses of 100-200 mCi may be used if residual microscopic disease is suspected or documented or for more aggressive histology. However, this dosing applies to individuals with normal renal function. Since iodine is primarily cleared by the kidneys, the dose of 131I and timing of dialysis need to be adjusted while treating patients with compromised renal function. Dialysis should not be too often or too early, because it can reduce the efficacy of the treatment. It must not be delayed, because it can increase the total-body dose to the patient.

Conclusion: Current literature has varying recommendations on dosage of radioiodine and timing of hemodialysis. In our patient, the administered activity was reduced by 50% and dialysis performed 12, 24 and 36 hours post radioiodine ablation. The treatment was both safe and effective.

Abstract #1181

THYROID DYSFUNCTIONS IN PATIENTS WITH CHRONIC HEPATITIS C VIRUS INFECTION WITH AND WITHOUT INTERFERON THERAPY

Aziza Hammad, MD Endocrinology, Fatma Hamad, Olfat Fawzy, Zeinab Hassan, Dina Abaza, Sumayia El shazly, Ihab El tayeb

Objective: to assess the frequency and pattern of thyroid dysfunctions (TD) in Egyptian patients with chronic hepatitis C virus infection (HCV) with and without interferon alpha (IFN) therapy.

Methods: thyroid function as well as thyroid peroxidase antibodies (TPO Ab) were assessed in 40 untreated Chronic HCV patients (HCV group), 30 HCV patients under IFN alpha therapy, for more than three months (IFN group) and 50 healthy age and sex matched controls.

Case Presentation: TD, either overt or subclinical, was detected in 12.5% of HCV group, 33.4% of IFN group and 2% of controls. Among HCV group, 5% had overt hypothyroidism, 7.5% subclinical hypothyroidism and no one had hyperthyroidism. While overt hypothyroidism was detected in 10% of IFN group, subclinical hypothyroidism observed in 16.7% and hyperthyroidism reported in 6.7 %. Among controls, 2 % had subclinical hypothyroidism whereas no one had overt hypothyroidism or hyperthyroidism. TD was more often detected in females (47.8%) compared to males (5.1%). Higher levels of TPO Ab were observed among IFN group compared to HCV group and controls ($P<0.01$&$P<0.01$, respectively). The study showed significant association between thyroid dysfunctions and TPO Ab positivity ($P=0.027$).

Discussion: TD among Egyptian HCV patients, especially after treatment with IFN, was more frequent than usually reported. This could be attributed either to different virus genotype distribution among countries (genotype 4 is the most prevalent in Egypt), which might lead to different influence on thyroid function, or to ethnic variation causing diverse genetic predisposition to thyroid autoimmune disease. It is likely that HCV and IFN act in synergism to trigger TD in patients.

Conclusion: TD is a common clinical problem in Egyptian HCV patients, especially those treated with IFN. Women appear to be more vulnerable to TD than men. The predominant TD is hypothyroidism. In view of high frequency of TD, routine screening and surveillance of HCV patients, especially after receiving IFN, is recommended.

Abstract #1182

DON'T BE FOOLED BY THE TSH: PERICARDIAL EFFUSION IN A PATIENT WITH CONCURRENT PRIMARY AND SECONDARY HYPOTHYROIDISM

Daniel Rosenbaum, MD, Bert Bieler, Marc Laufgraben, MD, MBA, FACE, FACP

Objective: TSH is widely regarded as the single best test of thyroid function, though TSH will not be valid in all situations. We report the case of a 46 year old woman who was profoundly hypothyroid with a pericardial effusion though her TSH was "only" 6.65 mIU/ml

Case Presentation: A 46 year old female with no known cardiovascular disese presented with chest discomfort. Five years prior to admission, she had a nonfunctioning pituitary adenoma resected surgically. Two years later, she developed Graves Disease and was treated with radioactive iodine. She was then started on Levothyroxine but only took this transiently. Several months prior to the onset of chest pain, her TSH had been checked and was only mildly elevated to 7.2. She reported amenorrhea since her pituitary surgery, and also complained of cold intolerance, dry skin, and poor energy. Evaluation of her chest pain included an echocardiogram demonstrating a large pericardial effusion without tamponade. Lab testing demonstrated TSH 6.65 mIU/ml (nl 0.35-5.55) and free T4 0.2 ng/dl (nl 0.89-1.76). She was treated with Levothyroxine 100 mcg daily. Three months later, her free T4 was within the normal range, her symptoms had improved, and the pericardial effusion had resolved.

Discussion: The patient's severe hypothyroidism was not diagnosed for several years because of the primary physician's reliance on TSH levels alone. Although TSH is the best marker of thyroid status in the majority of patients, it cannot be relied upon in patients with pituitary disease. Our patient was presumed to have hypothyroidism due to radioiodine ablation for her Grave's Disease, which is traditionally monitored via TSH, but she also had a history of pituitary surgery, which should have raised concern about TSH insufficiency. A patient with normal pituitary function and a free T4 of 0.2 would be anticipated to have a TSH > 5-10 times the upper normal limit, not a minimal elevation to 6.65. Patients with TSH insufficiency should be treated with Levothyroxine with a goal of maintaining their free T4 in the upper half of the normal range.

Conclusion: As demonstrated by this case, in patients with pituitary disease and TSH insufficiency, monitoring the TSH level is unhelpful and can be frankly misleading.

Abstract #1183

THYROTOXICOSIS AND SEVERE CORONARY VASOSPASM

Pankaj Sharda, MBBS, MD, Sowjanya Bhagavatula

Objective: Acute myocardial ischemia is rare and potentially life-threatening manifestation of hyperthyroidism; the exact mechanism has not been well-defined. An unusual case is reported of myocardial infarction in a patient with thyrotoxicosis causing severe coronary vasospasm. Possible mechanisms of coronary vasospasm in thyrotoxicosis are discussed.

Case Presentation: A 32-year old woman presented with chest pain and palpitations for 4 days. Her medical problems included hyperthyroidism, coronary artery disease status-post drug eluting stent eight months prior. Medications included carvedilol, clopidogrel, simvastatin and propylthiouracil but she was noncompliant. Physical examination revealed pulse of 130, BP 137/60 and oxygen saturation 98%. Electrocardiogram showed sinus tachycardia, ST depression in inferior and V4-V6 leads, T wave inversion V2-V6. Diagnostics showed high values of free T4 7.6, free T3 22.34, total T4 22.8, troponin-I 10.2, and low TSH <0.01. Aspirin, clopidogrel, eptifibatide, propylthiouracil, Lugol's iodine, propranolol and hydrocortisone were started. She deteriorated and had emergent cardiac catheterization on day two which revealed patent stent but diffuse coronary vasospasm worsening on dye injections. She had multiple cardiac arrests requiring pressor support and anti-arrhythmics but could not be revived.

Discussion: Despite the fact that almost 20% of the patients with hyperthyroidism develop angina, the documentation of coronary spasm is rare and there are no published series describing the underlying pathophysiology. Of the many causes, atherosclerosis and coronary vasospasm are two proposed mechanisms and our case further implicates spasm as a mechanism. One proposed mechanism of coronary spasm involves imbalance of cardiac autonomic innervation. Hyperthyroid state is associated with enhanced sympatho-adrenal activity due to increased adrenal receptor sensitivity and receptor numbers. Consequently, activation of α-adrenergic receptors in the coronary arteries, either by adrenergic or cholinergic means, leads to coronary vasospasm. Another proposed mechanism of coronary vasospasm involves changes in prostaglandin levels, especially TxA2 and prostacyclin. This case strengthens the hypothesis concerning causality in the association between hyperthyroidism and vasospastic angina which can provoke myocardial infarction.

Conclusion: Hyperthyroidism should be considered as a cause of life-threatening myocardial ischemia especially

in patients with normal coronary arteries and without conventional risk factors of atherosclerosis. The relationship between thyroid hormone, coronary artery spasm and ischemia is complex and poorly defined; and requires further investigation.

Abstract #1184

APATHETIC THYROTOXICOSIS PRESENTING WITH HEMOPTYSIS

Olusegun Sheyin, MD

Objective: To report a case of apathetic thyrotoxicosis in an elderly Nigerian female presenting to the emergency unit of a tertiary hospital in Lagos with hemoptysis.
Case Presentation: An 85 year old female presented with a 3 month-history of weight loss and a 2 week-history of cough, hemoptysis and progressively worsening breathlessness. Examination findings included wasting, dyspnea, pallor, an 80g goiter, an irregularly irregular pulse with a heart rate of 106 beats per minute, blood pressure of 126/80mmHg, jugular venous distension, cardiomegaly and a biventricular 3rd heart sound. Other findings were tachypnea (RR 36cpm), bibasal rales and a tender hepatomegaly. Thyroid function tests revealed free T3 of 6.2pmol/L(reference 3.8-6.0), free T4 of 23.9pmol/L(7.2-16.4) and TSH of 1.06 mIU/L(0.37-3.50). Anti-thyroglobulin and anti-thyroid peroxidase antibodies were both positive. ECG revealed atrial fibrillation with a ventricular rate of 112 b.p.m. Chest X ray findings were cardiomegaly, unfolded aorta and features of pulmonary edema. Dilated left atrium and grade 1 diastolic dysfunction were found on echocardiography. Complete blood count, sputum culture and staining for acid fast bacilli, coagulation profile, chemistry and upper gastrointestinal endoscopy revealed no abnormalities. Thyroid ultrasound revealed an enlarged right lobe with homogenous echotexture and absent pressure effect. A diagnosis of apathetic throtoxicosis in biventricular failure precipitated by atrial fibrillation was made. Intravenous furosemide 20mg 12 hourly, subcutaneous enoxaparin 40mg daily and per oral carbimazole 5mg 8hourly, spironolactone 25mg daily, lisinopril 5mg daily and digoxin 0.125mg daily were instituted. The patient reverted to sinus rhythm with resolution of hemoptysis and heart failure symptoms within a week of admission. She was discharged and is being followed up at the Endocrinology and Cardiology clinics.
Discussion: Apathetic thyrotoxicosis is an atypical manifestation of hyperthyroidism more commonly found in the elderly. It presents with cardiac disease, wasting or depression with only a few of the more typical clinical manifestations of thyrotoxicosis. Hemoptysis may occur as a consequence of pulmonary edema from left ventricular failure. Only a few cases of apathetic thyrotoxicosis presenting with hemoptysis have been reported in the literature.
Conclusion: A diagnosis of apathetic thyrotoxicosis should be considered in the elderly patient presenting with heart failure in the absence of the usual risk factors for heart failure. Early recognition and treatment can reduce the morbidity and mortality associated with this condition.

Abstract #1185

THYROID STORM AFTER RAI THERAPY WITH CONCOMITANT DIABETIC KETOACIDOSIS: A CASE REPORT

Odessa Wilson, MD, Augusto Litonjua, MD

Case Presentation: A 25 year-old female, admitted due to abdominal pain associated with nausea, poor appetite, palpitation, heat intolerance, hyperdefecation and lightheadedness. She is a diagnosed case of DM Type 1, on insulin, but was unable to inject for 2 days, and Graves' disease, on methimazole. But despite good compliance, she was never biochemically euthyroid, hence, underwent RAI treatment (15 mci). Physical examination showed a restless and tachycardic patient. The thyroid was diffusely enlarged, about 60-80 grams, with bruit but nontender. She had bilateral exophthalmos. There was direct epigastric tenderness. Pulses were bounding and she had grade 1 pedal edema. Initial blood sugar was 435 mg/dl. Serum ketone was high (4.0). Arterial blood gas showed metabolic acidosis with adequate oxygenation with an anion gap of 13.2 mEq/L. Serum electrolytes and creatinine were normal. She was initially diagnosed and managed as diabetic ketoacidosis (DKA). However, TSH was noted to be low (0.01) with a high FT4 (40.66). The patient scored 55 on the Burch and Wartofsky scale suggesting thyroid storm. She was then managed as a case of thyroid storm with concomitant DKA.
Discussion: Thyroid storm and diabetic ketoacidosis (DKA) both can be life threatening if not diagnosed promptly and managed appropriately. Their co-existence is rare. The incidence of glucose intolerance is increased under the hyperthyroid state [2]. Severe hyperthyroidism worsens glycemic control in diabetic patients through several mechanisms. Two scenarios may explain the concomitant occurrence of thyroid storm and diabetic ketoacidosis in this patient. First, the hyperthyroid state may have resulted to an enhanced basal hepatic glucose production and decreased peripheral glucose utilization. Together with the patient's omission to inject insulin because of nausea and poor appetite, the patient developed

diabetic ketoacidosis which could have triggered the thyroid storm. On the other hand, the patient may have developed thyrotoxicosis, or even so, thyroid storm, from a probable radiation thyroiditis or from discontinuation of the anti-thyroid drugs prior to the RAI which may have caused a relative insulin deficiency that was confounded by the patient's omission to inject insulin triggering the DKA.
Conclusion: The concomitant occurrence of these two endocrine emergencies can present in a manner masking the other but may be fulminant and be potentially fatal. This case emphasizes that recognizing the coexistence of these two conditions in a patient is very crucial inorder to provide correct, adequate and prompt management.

Abstract #1186

RHABDOMYOLYSIS DUE TO THE ADDITIVE EFFECTS OF UNTREATED HYPOTHYROIDISM AND ALCOHOL ABUSE

Esti Charlap, MD, Supreeti Behuria, Satcha Borgella, Yamin Shwe, Joseph Arcuri

Objective: To report a case of severe rhabdomyolysis caused by non-compliance with hypothyroidism medications and compounded by alcohol abuse.
Case Presentation: A 51 year old man with a history of hypothyroidism and depression was brought to the hospital by EMS after his neighbor called 911 to report water leaking from the apartment above hers. On arrival, EMS reported finding the patient in the bathtub with his clothes on, groaning. In the emergency room, he could not recall what happened or how he ended up in the bathtub. He only verbalized that he "drank a lot of alcohol". He was only oriented to person. On admission, his white count was elevated to 20.4 k/ul (normal 4.5-10.8 k/ul). His creatinine was normal at 0.87mg/dl (normal 0.66-1.25 mg/dl) and his electrolytes were normal. He had an elevated AST of 361 u/l (normal 15-46 u/l). His CPK was 1,482 u/l (normal 55-170 u/l). His troponins were negative and EKG did not show any ischemic changes. His urinalysis showed a moderate amount of blood but only 7 RBCs. He was started on aggressive IV hydration with normal saline but his CPKs continued to trend upwards. IV hydration was increased but CPK continued to increase, peaking at 96,907 u/l. The patient's cognitive function rapidly improved by day 2 of hospitalization, at which time he admitted that he had not been taking his levothyroxine for several months. At that point, a TSH and free T4 were checked. The TSH was found to be profoundly elevated at 135.79 mu/l (normal 0.55-4.78 mu/l). Free T4 was low at 0.3 ng/dl (normal 0.7-1.7 ng/dl) and T3 was 2.5 ng/dl (normal 5.3-10.5 ng/dl). He was started on 50mcg of Synthroid. The CPK continued to trend up. At that point, the Synthroid dose was increased to 112mcg and the CPK began to rapidly trend down. By hospital day 9 the CPK was 7,089 u/l.
Discussion: Although hypothyroidism can cause muscle abnormalities such as myalgias, muscle stiffness, and elevated muscle enzymes, rhabdomyolysis is uncommon. The mechanism is unknown but hypotheses include impaired glycogenolysis and impaired mitochondrial oxidative metabolism. Usually an aggravating factor such as excessive alcohol consumption, use of a medication, or vigorous exercise is identified. Thyroid hormone replacement therapy improves thyroid function and reverses rhabdomyolysis. The CPK elevation in our patient was profound, likely secondary to the additive effect of alcohol abuse in the setting of severe untreated hypothyroidism.
Conclusion: Though rare, hypothyroidism can be associated with rhabdomyolysis. This report highlights the importance of always including hypothyroidism in the differential diagnosis of any patient presenting with rhabdomyolysis, even when other possible causes are present.

Abstract #1187

ISOLATED ACTH DEFICIENCY ASSOCIATED WITH PAINLESS THYROIDITIS: THE POSSIBLE EFFECT OF ACUTE ADRENAL INSUFFICIENCY ON THYROID AUTOIMMUNITY

Tetsuya Mizokami, MD, Youhei Itoh, Yuichi Sato, Ken Okamura, Kiyohide Nunoi

Objective: Although isolated ACTH deficiency (IAD) is a heterogeneous disorder with several etiologies, the majority of acquired cases are most likely due to an autoimmune mechanism. Painless thyroiditis is considered a variant of Hashimoto's thyroiditis. Although acquired IAD is not uncommonly associated with other autoimmune endocrine diseases especially Hashimoto's thyroiditis, reported cases of IAD associated with painless thyroiditis are scarce.
Case Presentation: A 53-year-old Japanese man was referred with a 3-months history of transient headache followed by general malaise, myalgia and weight loss. He had a history of ocular myasthenia gravis which had been in remission following thymectomy 30 years ago. On physical examination, there was a small diffuse goiter without tenderness. Laboratory examinations revealed mild thyrotoxicosis with TSH of 0.03 μIU/mL, free T4 of 2.6 ng/dL, positive anti-thyroid peroxidase and anti-thyroglobulin antibodies, negative anti-TSH receptor antibody, and low thyroid radioactive iodine uptake. Since malaise was so remarkable and he had slight eosinophilia,

adrenal insufficiency was suspected. Both plasma ACTH and serum cortisol were undetectable. ACTH exhibited no response to CRH stimulation or insulin-induced hypoglycemia, and the other pituitary hormones except for TSH responded normally to the intravenous injection of TRH, GHRH and LHRH. Serum cortisol showed a subnormal response to the rapid ACTH stimulation test. Magnetic resonance imaging of the pituitary gland did not show any abnormalities. Anti-pituitary antibodies were negative. The patient was diagnosed as having IAD associated with painless thyroiditis. Treatment with cortisone acetate quickly relieved his symptoms of malaise and myalgia. Thyrotoxicosis was followed by an episode of hypothyroidism, and he became spontaneously euthyroid 3-months after the first visit.

Discussion: Based on the clinical course, painless thyroiditis is presumed to have developed soon after the onset of adrenal insufficiency in this patient. Patients with Hashimoto's thyroiditis sometimes develop painless thyroiditis after cessation of glucocorticoid therapy for rheumatoid arthritis or allergic rhinitis, after adrenalectomy for Cushing's syndrome, and following hypopituitarism due to pituitary apoplexy. Moreover, the simultaneous occurrence of autoimmune Addison's disease and painless thyroiditis has been reported. The immunological changes associated with acute adrenal insufficiency are therefore suggested to trigger the development of painless thyroiditis.

Conclusion: Hypoadrenalism-induced immunological changes may be one of the triggers of painless thyroiditis in patients with Hashimoto's thyroiditis.

Abstract #1188

RARE THYROID TUMOR IN AN ELDERLY FEMALE

Ricardo Correa, MD, Mario Ponce, Sheyla Zelaya, Silvia Cuadra, Melany Castillo, Fernando Calmet, Bernice Acevedo, Alejandro Ayala

Objective: Thyroid paraganglioma is a rare entity.Almost all cases have been described in women. This tumor is usually difficult to diagnose because it is rare and potentially malignant. This tumor is rarely functional, like in this case that found incidentally.

Case Presentation: 69 yo female with PMH of cervical cancer and benign goiter 44 yrs ago, for which she received radiation + hysterectomy and left partial thyroidectomy. 1 year before, patient presented with weight loss, anemia and syncope. A colonoscopy found a poorly differentiated mucinous colonic carcinoma. PET Scan revealed 4.2cm left adrenal lesion suspicious for malignancy with ring-like hypermetabolism. The biopsy showed crowded cells with hyperchromatic nuclei with cells in a more fibrous-appearing background and it was positive for synaptophysin and negative for cytokeratin and TTF-1. When the colon cancer was diagnosed, a right thyroid nodule was diagnosed. The biopsy of the thyroid revealed a neuroendocrine tumor consisting of tumor cells with predominantly isolated single nuclei, which occasionally forms small groups with focal nuclear molding. The inmunochemistry showed synaptophysin in most tumor cells and chromogranin was weakly to moderately positive. The tumor did not stain for epithelial markers. A rare cohesive group of cells of less than 1% exhibited expression of keratins and cytokeratin AE1-AE3, CAM 5.2 and CK7. The proliferation index of Ki67 was increased at 40%. Staining demonstrate a rare group of cells that stain positive for TTF-1. CDX-2 was negative. LCA, CD3 and CD20 all were negative. Physical examination was positive for right thyroid lobe enlarged at 2-3cm, firm and not very mobile.

Discussion: This patient had an adrenal tumor with similar characteristics, but would be very unusual for this tumor to metastasize. The patient has a history of multiple neoplasia, but this tumor has not been associated with any multiple endocrine neoplasia syndrome. In the differential there are 2 other tumors: medullary carcinoma of thyroid and hyalinizing trabecular adenoma of the thyroid . Morphologically these tumors are similar, so immunochemistry is very important to differentiate them. It is important to distinguish between them because of the prognosis and management.

Conclusion: Thyroid paragangliomas are keratin, cytokeratin markers negative, like in our case that was present in only 1% of cells. The string stain for synaptophysin marker is another characteristic of thyroid paragangliomas. MCT stains positive for TTF-1, compared to thyroid paragangliomas that are negative for this marker. The absences of CD markers in this particular case were helpful to rule out lymphomas. The treatment is surgery.

Abstract #1189

HIRSCHPRUNG'S DISEASE WITH CONGENITAL HYPOTHYROIDISM

Sunil Kota, MD, Siva Kota, Svs Krishna, Lalit Meher, Kirtikumar Modi

Objective: To report a case of congenital hypothyroidism (CH) coexisting with hirschprung's disease (HD) and to discuss possible theories for such an uncommon association.

Methods: Clinical and laboratory data are reported on a neonate presenting with large bowel obstruction and diagnosed to harbor CH.

Case Presentation: A 21-day old female infant presented with vomiting, abdominal distension and passage of

small quantity of liquid stool. There was history of poor feeding and excessive cry. Examination revealed facial puffiness, open anterior and posterior fontanelles, rough dry skin and cold peripheries and prominent abdominal veins with visible peristalsis. There was no goiter or umbilical hernia. Barium enema demonstrated dilated proximal colon, empty rectum, funnel like transition zone between proximal dilated and distal constricted bowel. High TSH (>150 μIU/mL) with athyrosis in scintigraphy confirmed CH. Colonoscopic biopsy revealed aganglionic segment, confirming the diagnosis of HD. The patient's genetic analysis revealed 46XX karyotype without any chromosomal abnormality or any mutations. She was discharged on oral thyroxine replacement.

Discussion: HD results from the failure of neural crest cell precursors to colonize the gut resulting in absence of myenteric and submucosal plexus. There are eight genomes associated with this disorder, most common being RET mutation. Our patient did not reveal any mutations. There are 3 theories linking congenital hypothyroidism with HD. The most commonly accepted theory is that there is a defective cranio-caudal migration of neuroblasts due to thyroid hormone deficiency which is necessary for appropriate neuronal migration and lamination during brain development. The other two theories include: defects in the differentiation of neuroblasts into ganglion cells and accelerated ganglion cell destruction within the intestine. Cranio-caudal migration of neuroblasts originating from the neural crest occurs between 5-12 weeks of gestation and synthesis and secretion of T4 and T3 starts at approximately 12 weeks of gestation while the maturation of the hypothalamic-pituitary-thyroid axis occurs during the second half of gestation. If we consider the other two theories about HD, fetal thyroid hormone levels seem more important than the mother's. In our case, fetal hypothyroidism may have led to HD by affecting cellular differentiation or apoptosis.

Conclusion: Although hypothyroidism impairs the colonic motility and function, the effect of thyroid hormone on neural crest cell migration through the bowel have not been studied. Our case highlights the possible role of thyroid hormones in the development of HD.

Abstract #1190

APATHETIC THYROTOXICOSIS SECONDARY TO ATYPICAL SUBACUTE THYROIDITIS AFTER INFLUENZA VACCINATION

Alessia Carluccio, MD, Nina Sundaram, Robert Yanagisawa, MD, Yaron Tomer, MD, FACP, FACE

Objective: Most cases of apathetic thyrotoxicosis have been liked to hyperthyroidism from Graves' disease. There are few documented reports of apathy as a manifestation of subacute thyroiditis.

Methods: We report the case of an 85-year-old female with no past medical history of thyroid disease who presented with severe obtundation and altered mental status a few days after receiving an influenza vaccination.

Case Presentation: The patient had been in her usual state of heath until her family noticed some difficulty with memory 9 days prior to presentation. Over the ensuing days, her mental status rapidly declined and by the time she presented to the hospital, she was obtunded with a Glascow coma score of 8. Her recent medical history included an influenza vaccination 2 days prior to the onset of symptoms. Non-contrast head CT was negative for acute pathology. Blood work was significant for elevated free thyroxine (FT4) and triiodothyronine (FT3) with suppressed thyroid stimulating hormone (TSH). Her erythocyte sedimentation rate (ESR) was also elevated. Thyroid antibodies were negative. A radioactive iodine study demonstrated severely diminished uptake, suggestive of thyroiditis. After a short course of steroids, the patient's mental status returned to baseline, and follow up labwork revealed normalizing thyroid function tests and ESR.

Discussion: Thyroiditis occurs after the proteolysis of preformed thyroglobulin molecules in follicular cells, resulting in a sudden increase in thyroid hormone concentrations in the peripheral circulation. While the inciting event for these episodes is often difficult to identify, it has been linked to release of inflammatory cytokines. We suggest that administration of the influenza vaccine may have been the trigger for thyroiditis in our patient.

Conclusion: Thyroid dysfunction should be included in the differential diagnosis of altered mental status, particularly in the elderly, even in the absence of a history of thyroid disease. Events potentiating the release of inflammatory mediators should be considered as inciting triggers for episodes of thyroiditis.

Abstract #1191

PATIENT EDUCATION IMPROVES PROPER T4 ADMINISTRATION PRACTICES AND MAINTENANCE OF TSH WITHIN A TARGET RANGE

Stephanie Lee, MD, PhD, Ji Youn Lee

Objective: Prior studies show patients taking T4 often have abnormal TSH levels. Multiple factors contribute including non-compliance, interference of T4 absorption by food, calcium, and iron, and refills of T4 from different manufacturers. We instruct patients at every visit about the appropriate method to take T4. The instructions include

taking T4 with an empty stomach, avoiding calcium, iron and other medications for 3-4 hours and doubling the daily dose for missed doses of T4. We performed an IRB approved study to test the hypothesis that compliance with these instructions improves achieving target TSH levels.

Methods: We prospectively recruited 100 consecutive hypothyroid patients taking T4 seen in our clinic between June 27 and August 5, 2011. We excluded pregnant patients and patients whose T4 was being adjusted after surgery or radioiodine therapy. We administered an IRB approved questionnaire to determine the number of patients who recalled receiving instructions how to properly take T4 and their compliance with these instructions. A TSH for each subject was obtained on the same day.

Results: The TSH targets were narrower than the reference range (0.3 - 5 uU/mL). The TSH goal for T4 replacement therapy was 0.3 - 3.5 uU/mL and for thyroid cancer the goal was <0.05 - 2 uU/mL. Of the 100 patients, 88 (88%) were at their TSH target. Eighty-nine percent of the patients acknowledged being instructed how to properly take T4. Of these patients, 97% (86/89) were actually taking T4 correctly. For the 11 patients who declared they were not instructed on the proper methods, 54% (6/11) were taking T4 correctly. When the patients were segregated according to whether they were taking T4 correctly or incorrectly, the TSH was at goal 89% (82/92) and 75% (6/8) of the time, respectively.

Discussion: In our clinic 12% of hypothyroid patients had TSH values outside the target range. Prior publications showed 32 - 48% of hypothyroid patients taking T4 had abnormal TSH levels. This data suggests that reminders for the proper methods to take T4 result in improved patient compliance. Furthermore, patients who take T4 properly are more likely to be at their target TSH level. A limitation of this prospective, non-randomized study is the small sample size.

Conclusion: Educational reminders at every visit on the proper method of taking T4 may have a significant impact on the patient compliance and their ability to maintain a TSH within the target range while reducing the clinician's time required to adjust the T4 dose and retest thyroid levels.

Abstract #1192

FOCAL 18F-FDG UPTAKE MIMICKING THYROID CANCER IN A PATIENT WITH HASHIMOTO'S THYROIDITIS

Michael Gonzales, MD,
Donald Richardson, MD, FACE, FACP,
John O'Brian, MD

Objective: To describe a case of incidental focal uptake of fluorine 18 flourodeoxyglucose in a euthyroid patient possessing anti-thyroid antibodies.

Methods: Clinical findings and laboratory data are presented followed by a review of pertinent literature.

Case Presentation: A 60 year-old woman was seen for evaluation for possible thyroid cancer with pulmonary metastases. She had 3 month history of exertional dyspnea associated with productive cough and was found to have non-calcified pulmonary nodules on chest x-ray. Chest CT was read as metastatic pulmonary disease. A PET scan showed focal uptake in the left thyroid lobe with a standard uptake value (SUV) of 5.49. She had no prior history thyroid disease, neck irradiation and no family history of thyroid disease or thyroid cancer. She was an obese female with a firm, normal-sized gland with no palpable nodules. Laboratory testing revealed a TSH of 3.08 mIU/L, anti-thyroid peroxidase of 71 IU/ml and undetectable antithyroglobulin antibodies. Thyroid ultrasound done prior to her PET scan showed a slightly enlarged heterogenous gland with no nodules but upon review two discrete lesions were identified: a <1 cm hypoechoic nodule in the isthmus and a 2.0 cm isoechoic nodule on the midpole of the left lobe which corresponded to the thyroid nodule identified on the PET/CT. FNA of the left lobe nodule showed sheets of follicular epithelium with atypia consistent with a thyroid neoplasm. She underwent a total thyroidectomy with central and lateral neck dissection and postoperatively she developed hypocalcemia due to the inadvertent removal of 3 parathyroid glands. Pathology however showed no evidence of thyroid malignancy and only a few pleomorphic follicular cells consistent with hashimoto's thyroiditis, the nodule detected on US was an adenomatoid nodule. She was started on thyroid hormone replacement and calcium supplementation.

Discussion: Whole body PET has a role for the evaluation of metastatic disease but its role is also expanding in screening for malignancy for patients at risk. Incidentally detected thyroid nodules with increased FDG uptake are seen in 1-4% of healthy subjects with lesion having focal uptake having a risk of malignancy. Autoimmune thyroid disease has been associated with diffuse uptake or diffuse plus focal uptake throughout the gland but only a minority (9%) of patients with Hashimoto's thyroiditis present with PET activity. Focal uptake with this condition however has not been reported. The etiology of PET of PET activity in autoimmune thyroid disease is unknown.

Conclusion: Clinicians should be aware that PET activity in autoimmune thyroid disease may vary significantly and that it may not represent malignancy.

Abstract #1193

RESISTANCE TO THYROID HORMONE (RTH) IN A 63 YEAR OLD FEMALE WITH MULTINODULAR GOITER AND MALIGNANT NEOPLASMS OF THE COLON, BREAST, AND SKIN

So-Young Kim, MD, Yun Feng, MD, Alina Goulller

Objective: To present a case of a patient with RTH and malignant neoplasms of the colon, breast and skin.
Case Presentation: A 63yo woman with a history of multinodular goiter (MNG), hepatitis C, colon cancer, breast cancer, and melanoma presented to endocrine clinic in 2008 with abnormal thyroid function tests (TFTs). She was clinically euthyroid and denied any family history of thyroid disease. Laboratory analysis revealed elevated free thyroxine (FT4) 1.8 ng/dL (0.7-1.7 ng/dL), free triiodothyronine (FT3) 4.8 pg/mL (2.3-4.2 pg/mL) and normal thyrotropin (TSH) 3 mU/L (0.55-4.78 mU/L). Thyroid antibodies (Ab) (thyroglobulin Ab <20 IU/mL; anti-TPO Ab <10 IU/mL) were negative. A thyroid ultrasound (US) and biopsy of thyroid nodules showed benign nodular goiter. Subsequently, she was lost to follow-up. In 2011, the patient returned to the endocrine clinic with an elevated FT4 2 ng/dL, FT3 6 pg/mL, and a normal TSH 3.53 mU/L. Free T4 and free T3 were repeated using equilibrium dialysis (to rule out immunoassay interference), with similar results: elevated FT4 3.3 ng/dL (1.1-2.4 ng/dL); upper normal FT3 6.4 pg/mL (3.2-6.6 pg/mL). Again, the patient was clinically euthyroid. The differential diagnoses included RTH or a TSH-producing pituitary adenoma. The alpha subunit glycoprotein was normal 1.2 ng/mL (0.9-3.3 ng/mL) and a head CT was negative. Therefore, pituitary adenoma was unlikely. Patient's serum was checked for heterophile Ab, given inaccuracy of free thyroxine assay, and was negative. Thyroid US showed stable MNG. The patient continues to be monitored with no medications and remains asymptomatic. She refused genetic testing.
Discussion: RTH is an inherited syndrome of tissue hyposensitivity to thyroid hormone. It presents with elevated FT4, FT3, and normal or elevated TSH. RTH has been detected in 1 of 40,000 life birth. Clinical features include goiter, hyperactivity, and tachycardia. In 85% of cases, RTH is caused by mutation in the TH receptor beta gene. 15% are caused by undetermined genetic mutation. Our patient was clinically euthyroid with elevated FT4 and FT3 but normal TSH, which is consistent with RTH. The diagnosis could not be confirmed by genetic analysis as the patient refused further testing. It is not clear whether there is a relationship between RTH and patient's prior history of multiple neoplasms.
Conclusion: Prior case reports described RTH with negative genetic testing, most likely from mutations yet-to-be discovered. It is important to correlate biochemical workup with clinical findings and genetic analysis to prevent misdiagnosis of RTH. It would be helpful to determine whether there is a genetic link between RTH and the patient's other malignant neoplasms.

Abstract #1194

PATTERN OF PRESENTATION AND OUTCOMES OF MEDICAL MANAGEMENT OF THYROTOXICOSIS

Andrew Edo, MBBS, FMCP, Steve Obanor, Gloria Edo, Aihanuwa Eregie

Objective: Thyrotoxicosis is a common endocrine disorder. Treatment options include medical, surgical and radioiodine therapy. Many patients prefer only medical treatment with antithyroid drugs. They are reluctant to accept thyroidectomy when euthyroid hoping that the goitre will melt away with antithyroid drug administration. Radioiodine therapy is not readily available in our practice locale. This study is to document the pattern of presentation of thyrotoxicosis and the outcomes of medical treatment.
Methods: This is a retrospective study of all patients with thyrotoxicosis managed in a tertiary care center over a 3 year period. Their medical records were retrieved. Data extracted included age, gender, presenting complaints, weight, blood pressure, co-morbid disorders, and thyroid hormone profiles. The data was analyzed using SPSS version 16. Significant level was set at $p < 0.05$.
Results: Thirty five patients (32 females, 3 males) with thyrotoxicosis were seen during the study period giving a female to male ratio of 8:1. Mean age of the patients was 44.3±13.9 years (females 43.8 ±13.1, males 40.0±19. 1 year). The peak age range at presentation was 40-49 years. Mean systolic blood pressure was 136.9±18.2mmHg, diastolic blood pressure 82.2±12.8 mmHg. Pulse 100.6±22.4 beats/ min. T3 4.3 ±3.1, T4 18.5±9.3, TSH 1.5±3.5. Thirteen (37.1%) had hypertension and 11.4% had diabetes mellitus. The modes of presentation were as follows: thyrotoxicosis 82.86%, Graves'disease 20%, Graves'ophthalmolpathy 5.71%, thyrocardiac heart disease 5.71%. The frequency of presenting symptoms were anterior neck mass 68.57%, weight loss 60%, palpitation 60%, heat intolerance 37.14%, bulging eyes 31.43%, hyperdefaecation 31.45%, tremors of the hands 19.99%, menstrual irregularities 17.14%, excessive sweating 17.14%, increased appetite 14.29%, sleeplessness 11.43%,weakness 11.43%, hyperpigmentation 8.57%, and pruritus 5.71%. Goitre was found in 77.14%, proptosis 45.71% and oncholysis 8.57%. Twenty percent

of the patients remained hyperthyroid, 5.71% became hypothyroid. The mean weight gain after 12-24 months of treatment with anti-thyroid drug (carbimazole) was 8.5±6.4 kg. Goitre persisted in all the cases that had goitre prior to commencement of treatment

Discussion: The challenges of management of thyrotoxicosis in resource-constraint environment will be discussed.

Conclusion: Thyrotoxic patients commonly present with goitre, weight loss, and palpitation. There was resolution of the symptoms (except for the goitre) with medical treatment. Therefore, patients seeking resolution of the goitre should be encouraged to opt for either surgery or radioiodine therapy.

Abstract #1195

SECONDARY ADRENAL INSUFFICIENCY AND THYROIDITIS FROM IPILIMUMAB - CASE REPORT AND LITERATURE REVIEW.

Bhavika Bhan, MD, Rudruidee Karnchanasorn, Vaishali Patel, MD

Objective: Anti CTL-4 agents like Ipilimumab are a novel approach to cancer treatment. Ipilimumab has been used for the treatment of metastatic melanoma and RCC. A host of immune related adverse events are associated with anti-CTLA-4 therapy including endocrine AE's like hypopituitarism, hypothyroidism, hypophyistitis and adrenal insufficiency. We report a case of secondary adrenal insufficiency and hyperthyroidism from Ipilimumab in a patient with metastatic melanoma.

Case Presentation: 77 year old male with h/o scalp melanoma diagnosed in 2007, s/p resection was found to have lung and liver metastases for which he was started on Ipilimumab in June 2011.He was admitted in October 2011 with decreased appetite, fatigue, nausea without vomiting, weight loss of20 lbs over 4 months, occasional light-headedness and diarrhea. On admit he was hypotensive BP of 96/44 mm Hg. Lab was significant for Na of 123 mEq/L,creatinine 1.75 mg/dl,low random cortisol low 1.3 mcg/dl. Anterior pituitary functions showed FSH 18 miu/ml, LH 10 miu/ml, prolactin 9 ng/ml, IGF1 157 ng/mL, ACTH <5pg/mL. Patient had a subnormal reponse to ACTH stim test.TSH was low at 0.049 mcu/ml with high fT4 2.0 ng/dl, total T3 117ng/dl on admit .TSH previously was normal. TPO Ab 134 IU/ml. TSI Ab <1. MRI head was without evidence of metastases. Thyroid uptake and scan could not be done as the patient had received iodinated contrast on admit. Patient was started on steroids and methimazole and had symptomatic improvement. Na improved to 131mEg/L in 2 days.

Discussion: Ipilimumab, is a new anticancer drug with proven clinical efficacy in the treatment of metastatic melanoma and RCC. The association of anti- CTLA-4 agents and endocrine abnormalities especially adrenal insufficiency and thyroid dysfunction suggests that endocrinologists should be aware of such potential endocrine toxicities. To our knowledge this is the first report of hyperthyroidism/thyroiditis caused by Ipilumamab. Hence we recommend baseline thyroid function , thyroid antibody and anterior pituitary function tests prior to starting treatment and than periodic monitoring.

Conclusion: Hypothyroidism has been reported with Ipliumamab therapy. However, this drug may also induce hyperthyroidism/thyroiditis. Normal thyroid functions prior to treatment suggest that hyperthyroidism was a result of Ipilimumab therapy. Normal TSI, +TPOAb suggest that the most likely cause of hyperthyroidism was thyroiditis. RAI could not be done to confirm our findings. Secondary adrenal insufficiency in absence of pituitary metastases and the temporal relationship with medication suggest that Ipilimumab had a causative role.

Abstract #1196

SUCCESSFUL TREATMENT OF GRAVES' HYPERTHYROIDISM WITH CHEMOTHERAPY FOR ACUTE LYMPHOBLASTIC LEUKEMIA

Katrina Abadilla, MD, Henry Fein, MD

Case Presentation: Autoimmune thyroid disease has been reported to have a strong association with acute leukemia. A 37-year-old Filipino man was diagnosed with Graves' Hyperthyroidism after presenting with fatigue, palpitations and weight loss. He had a suppressed TSH, elevated fT4 and T3 and increased RAI uptake. CBC, BMP and lipid profile were normal. He had bilateral conjunctival injection with restriction of upward gaze of the left eye and minimal diplopia on upward lateral gaze. Hertel exophthalmometry: left eye: 21 mm, right eye: 19 mm (upper normal for Asians: 18 mm). The thyroid was 35 gm and was diffusely moderately firm. The right lobe was larger than the left. He was begun on propylthiouracil (unknown dose) and then switched to methimazole 10 mg after one year. Seven years later, his Graves' Hyperthyroidism was still present and he remained on methimazole 20 mg. He had bilateral proptosis with Hertel measurements of right: 22 mm, left: 23 mm. Radioactive iodine treatment was considered but deferred due to his active smoking. Shortly thereafter, he was found to have a leukocytosis of 60,000/mm2. He was diagnosed with Philadelphia-chromosome negative pre-cursor B-cell acute lymphoblastic leukemia and found to have high risk (deletion 6q) after a bone marrow biopsy. His oncologist requested discontinuation of methimazole, due to possible drug interactions, prior to initiating chemotherapy. He then

completed 8 cycles of Hyper-CVAD/MTX-Cytarabine chemotherapy (alternating cycles of cyclophosphamide/vincristine/doxorubicin/dexamethasone and methotrexate/cytarabine) maintenance therapy, and allogenic unrelated stem cell transplantation. All thyroid hormone and TSH levels were normal throughout these therapies. Two years later, he remains in complete remission of both his leukemia and Graves' Hyperthyroidism without resumption of methimazole. Proptosis and diplopia have cleared although his globes remain moderately firm.

Discussion: Review of the literature suggests an association between autoimmune thyroid disease and acute leukemia and further suggests that patients with this association have a more favorable prognosis for their leukemia. There is no clear hypothesis to this association, however, the role of thyroid hormones in regulating hematopoiesis and utilizing receptors may play a role. Other links point toward hematopoietic damage caused by antithyroid drugs.

Conclusion: To our knowledge, this is the first case report of remission of difficult-to-control Graves' Hyperthyroidism by induction chemotherapy of acute leukemia.

Abstract #1197

FINE NEEDLE ASPIRATION (FNA) OF THYROID NODULES OUR EXPERIENCE WITH THYROID CYTOLOGY

Barbara Gredysa, MD, Ayse Bag Ozbek, MD, Marina Charitou

Objective: To share our experience with fine needle aspirations of the thyroid.

Thyroid nodules are a common clinical finding with prevalence ranging from 4-67 % depending on the mode of detection. High-resolution thyroid ultrasound (US) is the most sensitive test available to evaluate thyroid nodules. Fine-needle aspiration (FNA) of the thyroid has been accepted as a first-line screening test for patients with thyroid nodules.

Methods: The study population consists of 576 FNA samples collected in our institution between April of 2008 and February 2011. All FNAs were performed by the same endocrinologist with US guidance with or without a pathologist's on-site adequacy evaluation. Diagnostic criteria were reviewed with the Department of Pathology and were consistent with diagnoses proposed by The Papanicolaou Society of Cytopathology. The samples were categorized as: unsatisfactory, benign, atypical lesion, follicular neoplasm (FN), suspicious for malignancy, and positive for malignancy.

Results: Using 6 diagnostic categories our experience with thyroid FNAs was studied. The results yielded 60.59% benign FNAs (349 samples), 18.4% atypical lesions (106 samples) and 7.12% follicular neoplasms (41 samples); 1.39% FNAs was suspicious for malignancy (8 samples), 5.38 yielded malignant results (31 samples) and 6.42% was unsatisfactory(37 samples). Three FNA samples could not be classified with these criteria (lipoma, esophageal origin, parathyroid).

Discussion: The results were compared to another large study and yielded similar results in the benign category (60.59% in our study vs 64.6%). The rate of malignant results, FN and unsatisfactory specimens was lower in our study (5.38% vs 7.6%; 7.12 % vs 11.6%, 6.42 % vs 10.4% respectively). Atypia was diagnosed more frequently (18.4% vs 3.2%).

Conclusion: Our experience demonstrated a lower incidence of malignancy based on FNA compared to other studies. Indeterminate FNA diagnoses such as atypia and FN have ranged from 5 to 42% in the published literature and were 25.52% in our study. Histopathology correlation is needed for evaluation of sensitivity and specificity of FNA in diagnosing thyroid malignancy and should be further studied.

Abstract #1198

A CASE OF NEGATIVE RHTSH WHOLE BODY SCAN CONVERTING TO POSITIVE FOLLOWING WITHDRAWAL OF LEVOTHYROXINE

Richard Pinsker, MD, Pinkesh Prajapati, Narinder Kukar, Jose Cervantes, Eduardo Andre, Sandeep Ponniah, Mahboob Ali, Kelly Cervellione

Objective: To describe a case of recombinant human TSH (rhTSH) whole body scan (WBS) that was negative for metastatic papillary thyroid carcinoma, which became positive following levothyroxine withdrawal.

Case Presentation: A 62-year-old female presented with a neck mass. Upon physical exam, the patient was euthyroid both clinically and biochemically. Sonogram revealed an enlarged thyroid. Fine needle aspiration biopsy showed a nodular goiter, however follicular neoplasm could not be excluded. CXR was showed a mediastinal mass. One year later, repeat CXR and CT showed new bilateral non-calcified lung nodules. Patient underwent VATS lung wedge biopsy, which confirmed metastatic papillary thyroid carcinoma. A total thyroidectomy and radioactive iodine (RAI) ablation was performed and levothyroxine was started. CXR and sonogram on follow up revealed no masses or nodules. Thyroglobulin levels were monitored regularly. One year later, repeat CXR revealed bilateral nodules along with significantly high thyroglobulin (peak=2420). Despite positive PET scan for pulmonary metastases, rhTSH WBS was negative. Levothyroxine

was discontinued. After elevation of TSH, repeat WBS was positive for gross lung metastases. Patient was successfully treated with second RAI ablation.
Discussion: rhTSH WBS is used to detect recurrence or metastasis of thyroid cancer. However, at times it can give rise to false negative result. In order to increase the sensitivity of the test, rhTSH stimulation could be accompanied by levothyroxine withdrawal to allow the RAI uptake by the metastatic thyroid cancer.
Conclusion: The current case illustrates a false-negative rhTSH WBS that converted to a positive result after discontinuation of levothyroxine treatment. We are not clear about the etiology for the initial negative WBS, but multiple previous cases have noted conversion from negative to positive after both levothyroxine withdrawal and rhTSH stimulation.

Abstract #1199

INSULAR CARCINOMA : A NEW FACE OF THYROID CANCER

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Objective: Thyroid carcinomas range from well differentiated to anaplastic carcinomas. Insular carcinoma is a histologically distinct subset of thyroid carcinoma which falls between well-differentiated and anaplastic carcinomas. Although the differentiation of insular carcinoma from other thyroid carcinomas has important prognostic and therapeutic significance, relatively little about insular carcinoma has been published . We describe a rare case of insular carcinoma and discuss the it's findings.
Case Presentation: We present a rare case of poorly differentiated thyroid carcinoma. Patient is a 79 year old male who presented for follow up of aortic aneurysm. A CT scan was performed which showed a large liver lesion. Biopsy of liver lesion revealed metastatic thyroid carcinoma. Ultrasound of thyroid gland did show a left side nodule. PET scan revealed activity in thyroid, liver as well as mediatinum. Patient was clinically euthyroid and was asymptomatic. He underwent total throidectomy with wedge resection of liver and radiofrequency ablation. Histology of thyroid lesion revealed mixed insular and papillary carcinoma. Following surgery he also underwent radioiodine ablation. Patient is presently doing well. He is under close observation for recurrence of disease and is followed up with scans and thyroglobulin. Recent scan has not identified any recurrence of lesions or progressive disease.
Discussion: Insular carcinoma is a subtype of thyroid carcinoma whose criteria for diagnosis is not uniform among pathologists. It exhibits increased aggressiveness, propensity to both local recurrence and distant metastases and increased mortality. The exact incidence of insular carcinoma remains unknown. The most common initial signs are enlarging neck mass, dysphagia or dyspnea. Most patients are clinically euthyroid. Both insular and anaplastic carcinomas are metastatic at presentation. However they do have few differences. Most important difference is in there clinical course, management and prognosis. In anaplastic carcinoma, a significant proportion of the patients have an acute deterioration in general condition and die shortly after diagnosis. Insular carcinoma is intermediate between and anaplastic cancers in terms of prognosis. It represents a disease where appropriate administration of aggressive treatment not effective for anaplastic disease may uniquely result in substantial benefit as was seen in our case.
Conclusion: It is important to identify insular thyroid carcinoma as it warrants aggressive management with total thyroidectomy followed by radioactive iodine ablation of any remaining thyroid tissue which leads to better outcomes.

Abstract #1200

METASTATIC PAPILLARY THYROID CARCINOMA PRESENTING WITH TOXIC SYMPTOMS: A CASE REPORT

Innocent Okpe, MBBS, FMCP, Yaqub Lawal

Objective: To report a rare case of metastatic papillary carcinoma presenting with thyrotoxicosis.
Methods: Full history, examination and appropriate investigations were carried out. Various literatures were reviewed to ascertain the rarity of this kind of presentation.
Case Presentation: E.R. is a 56 year old woman presenting with a one and a half year history of anterior neck swelling, three month history of low back pain, three weeks history of progressive weakness of both lower limbs and cough. There is associated heat intolerance, weight loss, sweating, rapid paroxysmal palpitation. She was diagnosed diabetic one year earlier. Clinical, biochemical, radiological and pathological diagnosis of metastatic papillary thyroid cancer and thyrotoxicosis were obtained.
Discussion: This patient with papillary thyroid carcinoma presented with toxic symptom quite a rare occurrence. Thyrotoxicosis generally is not common in all forms of thyroid malignancies but when they do occur, follicular thyroid carcinoma is the likely histological type but this case of papillary cancer presented with toxic symptoms and signs. Thyroid carcinoma is usually first seen clinically as a thyroid mass, sometimes with an enlarged cervical lymph node as seen in this case. Papillary thyroid carcinomas are also discovered when a hard nodule is found in multinodular goiter, when enlarged cervical lymph nodes

are detected, or when there are unidentified metastatic lesions elsewhere in the body as found in this case. Expanding lesions found in the thyroid gland, especially if they are painful, should be examined as they may indicate the presence of papillary thyroid carcinoma. Pain was however absent in this case. Other clinical signs that could indicate papillary thyroid are fixation to the trachea, stony hardness which were all found in this this patient. Damage to recurrent laryngeal or cervical sympathetic nerves which are features of local invasiveness were however not clinically obvious in this patient. However the presence of distant metastasis in this middle-aged patient portrays the fact of her late presentation thereby limiting her options of treatment including curative surgical non-eligibility.

Conclusion: Papillary Thyroid carcinoma can present with toxic symptoms and may easily be mistaken for a benign toxic adenoma of the thyroid. A high index of suspicion is required for the early detection of this treatable cancer

Abstract #1201

UNTREATED GRAVES DISEASE WITH AUTOIMMUNE CLUSTER

Aasia Khan, MD, Karla Wyatt, Jolene Lowery

Case Presentation: 37 y/o female presented with 2-week history of shortness of breath, severe jaundice, fatigue, abdominal and leg swelling associated with difficulty concentrating, palpitations, decreased appetite, diarrhea, 100 pound weight loss, hair thinning, irregular menstrual cycles and heat intolerance. Six months earlier she was diagnosed with thyrotoxicosis and heart failure, but she pursued no treatment. Her family history was significant for unspecified thyroid disorder in her mother, father, grandmother and niece. Physical exam revealed normal heart rate and blood pressure, jaundice, proptosis, thyromegaly with thyroid bruit, pitting edema from the feet to the abdomen, and bilateral rales to apices. The laboratory data was remarkable for normocytic anemia with elevated total and direct bilirubin at 9.6 mg/dl and 5.9 9mg/dl respectively. TSH was <0.01 mIU/L with total T3 at 471 ng/dl, free T4 at 4.55 ng/dl, and TSI was 431. The BNP was 2436 pg/ml. Direct coombs' test was positive. The ANA, Anti-RNP, Anti-Smith and anti-centromere were positive. Hepatitis panel and HIV tests were negative. The transaminases, LDH, haptoglobin and GGT were all normal. Chest X-ray showed pleural effusions with cardiomegaly. The echocardiogram showed an ejection fraction of 45-50%. She was treated with diuretic and beta blocker therapy. She also received a blood transfusion. She was unable to obtain a thyroid uptake scan because she had received IV contrast for a CT scan. Methimazole was started to treat the hyperthyroidism. Her symptoms gradually improved over a week with the treatment.

Discussion: This clinical case illustrates a presentation of autoimmune thyroid disease associated with several other autoimmune diseases manifesting with pancytopenia and cholestatic jaundice. In addition, the positive ANA, direct Coombs' test, anti-Smith and anti-RNP is consistent with a diagnosis of Lupus.

Conclusion: Untreated Graves disease can be associated with several rare complications as seen in this case ,however it is recommended to delay interpretation of autoimmune testing until hyperthyroidism is in control.

Abstract #1202

SPONTANEOUS CERVICAL HEMATOMA FROM A BENIGN THYROID NODULE

Karen Devon, MDCM, Raymon Grogan, MD, Peter Angelos, MD, PhD, Edwin Kaplan

Case Presentation: A 64 year old woman developed a sudden severe neck pain and swelling lasting 5 days. An ecchymosis appeared on her neck and chest (Fig 1) and she had a breathy voice. Her history was negative for trauma, surgery, a bleeding disorder or medications. An ultrasound showed an enlarged left thyroid lobe (6.1 x 2.9 x 2.6 cm) that was thought to contain a heterogeneous hypoechoic nodule(Fig 2). A CT scan (Fig 3) revealed a 4.3 x 3.5 x 2.6 cm mass posterior to the left lobe of the thyroid with foci of low density, likely representing necrosis. This mass was thought to be extrinsic to the thyroid gland, displacing the trachea and esophagus. A possible parathyroid neoplasm or other tumors were suggested in the differential diagnosis. The remaining structures appeared normal. Serum calcium and PTH were normal. A fine needle aspiration showed no epithelial cells. Several months later we performed subtotal thyroidectomy. Significant fibrosis was encountered. There was no evidence of any mass extrinsic to the thyroid. The left lobe contained a 1 cm benign-appearing nodule. Her post-operative course was unremarkable. Final pathologic examination confirmed colloid nodular disease with a dominant 1 cm nodule on the left. Within the nodule histologic examination revealed interstitial and intrafollicular hemorrhage as well as some hemosiderin pigmentation within a few follicles. The interstitial hemorrhage extended into the adjacent soft tissue.

Discussion: Spontaneous cervical and mediastinal hematomas have usually been associated with direct and indirect trauma, thyroid artery aneurysms, or ruptured parathyroid adenomas. Less than 5 of cases of spontaneous extracapsular rupture of a thyroid nodule have been described. Most bleeding due to trauma (usually exertional)

to the thyroid gland occurs intracapsularly, into a goiter or a nodule. Extracapsular hemorrhage occurs more often with parathyroid adenomas- even occur with normal calcium and parathyroid hormone levels. Some of the most severe presentations of spontaneous cervical hemorrhage have been due to a rupture of an inferior thyroid artery aneurysm. In our patient, no previous history of hypertension, diabetes, or hypercholesterolemia was present. At operation, 3 months after the acute event, an enlarged parathyroid gland or aneurysm was not found. Fibrosis around the left thyroid lobe and a colloid nodule containing hemosiderin laden macrophages, suggesting previous bleeding.

Conclusion: In our case hematoma likely occurred from spontaneous rupture of a benign thyroid nodule. We recommend that similar patients have immediate imaging, admission to hospital for management of bleeding, and elective treatment of their disease.

Abstract #1203

A PROMISING ROLE OF ULTRASOUND IN THE LOCALIZATION OF PARATHYROID ADENOMAS

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Objective: Sestamibi (MIBI) scan is the standard imaging modality for localization of parathyroid adenomas (PA). However, the sensitivity of MIBI scan is (54-93%), so some adenomas may be missed. Ultrasound (US) has emerged recently as an alternative imaging modality, but has not been widely used in the localization of PA. We present a case of primary hyperparathyroidism (PHPT) which highlights the effectiveness of US as an alternative, or adjunct imaging modality in PHPT

Case Presentation: A 64 year old man was referred to the endocrinology clinic for evaluation of multinodular goiter. A fine needle aspiration of a nodule in 2005 showed a follicular neoplasm. Surgery was recommended but he refused. Laboratory data revealed hypercalcemia, which had been gradually increasing to a maximum of 11.75 mg/dl (ref:8.00-10.5), with hypercalciuria (633 mg/24 hour-ref:100-300). His parathyroid hormone (PTH) was 150. 5 pg/ml (ref:14.0-72.0). MIBI scan findings were consistent with a PA. He was recommended surgery but he again declined. Therefore, he was started on cinacalcet, which normalized his calcium and PTH. In retrospect, US images showed a left inferior posterior lesion which had grown in size (8, 10 and 22 mm in 2004, 2006 and 2012, respectively). This lesion was hypoechoic and oval in shape, suggesting a parathyroid lesion, corresponding to the PA suggested on the MIBI scan. The size of the lesion chronologically correlated with increasing serum calcium.

Discussion: With the advancements in US over the last few years and its increasing use in the endocrinologist office, US can also be used in localization of PA's. US is noninvasive, time saving and is inexpensive, compared to MIBI scan and is also useful for the characterization and evaluation of any concomitant thyroid pathology, facilitating operative planning. However, there is little awareness about the utility of US in identifying PA's in clinical practice. When the diagnosis of PHPT is confirmed biochemically, we propose to begin with US as the principal localizing imaging study. If US confirms an unequivocal parathyroid lesion, then no further imaging is needed. However, if US is non-conclusive, then MIBI scan is recommended for localization. Finally, it is prudent to point out that there are rare cases where MIBI scans may be superior to US, namely in cases of multiple parathyroid adenomas as well as adenomas located outside the thyroid area.

Conclusion: Several studies have highlighted the usefullness of US in localization of PA's. We suggest that US be considered as an initial or adjunct imaging modality in the workup of PHPT.

Abstract #1204

NEWLY DIAGNOSED GRAVES' DISEASE IN A PATIENT WITH ACUTE INTERMITTENT PORPHYRIA (AIP) AND ASSOCIATED SYNDROME OF INAPPROPRIATE ADH SECRETION (SIADH)

Geeti Mahajan, MD, Natia Potter, MD, Arathy Vamadaven, Nathaniel Winer

Objective: A case of known AIP and associated SIADH is presented to alert the endocrinologist to the possibility of underlying Graves' disease in patients with AIP and to illustrate the most appropriate modality of therapy for Graves' disease in AIP.

Case Presentation: A 29 year-old African-American woman with no significant past medical history presented with sinus tachycardia, severe abdominal pain, nausea and vomiting in 2009. During this hospitalization the patient was found to have serum sodium of 112 mEq, and suffered a generalized seizure, leading to the diagnosis of SIADH. The combination of SIADH and abdominal pain suggested the diagnosis of AIP, which was confirmed by elevated urinary and red blood cell porphyrobilinogen levels. In October 2011 she presented with similar symptoms, along with unintentional weight loss and proptosis. Patient was then diagnosed with Graves' disease on further work up. She was started on propylthiouracil, steroids and propanolol, and discharged after radioiodine ablation. During hospitalization her AIP stabilized after Hemin

infusion and improvement of hyperthyroidism.
Discussion: AIP occurs secondary to an autosomally-inherited deficiency in porphobilinogen deaminase. There is a recognized association between AIP and SIADH. In patients who have died of AIP complicated by SIADH, neuronal loss were seen in the supraoptic and paraventricular nuclei. However, there is no clear association between hyperthyroidism and AIP. Theoretically, the hypermetabolic state of thyrotoxicosis may increase hepatic delta-aminolevulinate synthase (ALAS1) activity, thereby accelerating the depletion of heme products and causing an AIP exacerbation. Medications can cause AIP exacerbations, especially those that inhibit ALAS1 and cytochromes. During an attack of AIP, the regulatory heme pool in the liver is depleted. Inhibitors of Cytochrome P450 enzymes, such as methimazole, may result in compensatory acceleration of heme synthesis, which may overload the deficient porphobilinogen deaminase-step.
Conclusion: A woman with known AIP and a history of SIADH was diagnosed with Graves' hyperthyroidism. PTU was chosen as the treatment modality rather than methimazole, since methimazole inhibits several Cytochrome p450 enzymes which would theoretically exacerbate AIP. Even though previous studies have described the use of methimazole without complications, the American Porhyria Foundation recommends PTU as the safest treatment modality. Also, given the similar symptomatology, thyroid function should be evaluated during the assessment of AIP, since hyperthyroidism can precipitate an acute episode of AIP and may worsen the disease severity.

Abstract #1205

A CASE OF METASTATIC PAPILLARY THYROID CARCINOMA WITH CYSTIC DEGENERATION, MASQUERADING AS A BENIGN-APPEARING THYROID CYST WITH BENIGN FINDINGS ON FINE NEEDLE ASPIRATION

Saleh Aldasouqi, MD, Michael McLeod, MD, FACS, Ala Eayyan, Bhavini Bhavsar, MBBS, M.D, Rueben Cuison, Sameer Ansar, MD, Deepthi Rao, MD

Objective: A common Ultrasound (US) feature of papillary thyroid carcinoma (PTC) is the appearance of a solid lesion. PTC can degenerate and masquerade as a cystic lesion. We present a challenging case of cystic PTC presented as thyroid cyst.
Case Presentation: A 29 year old man was referred for a recently discovered neck mass, which interfered with swallowing. He had no family history of thyroid cancer or personal history of radiation exposure. He was clinically and biochemically euthyroid. US showed a 1.1 cm cyst located in the isthmus. US-guided fine needle aspiration (FNA) was performed for the purpose of fluid aspiration to relieve the swallowing difficulty. 1 cc of dark greenish fluid was aspirated, and pathology was consistent with a benign cystic nodule. Few days later, the cyst refilled, and the patient's swallowing difficulties recurred, and he requested surgical removal of the cyst. He underwent partial isthmectomy (the portion encompassing the cyst), which surprisingly revealed that the "cyst" was in fact a degenerative PTC, measuring 7 mm, with 2 solid foci, measuring 5 and 2 mm, attached to the cystic portion, with multiple regional lymph node (LN) metastasis documented in the incidentally removed LN's (4/4). The patient had a completion thyroidectomy which revealed remnant PTC in the residual part of isthmus as well as in an additional pretracheal LN. He also underwent radioactive iodine treatment.18 months later, the patient is now cancer-free, healthy, and is clinically euthyroid on levothyroxine, at target TSH suppression.
Discussion: PTC is typically described as a hypoechoic solid lesion on US. Suspicious features include microcalcifications, hypervascularity and irregular borders. The vast majority of smooth cystic lesions with posterior enhancement and thin walls are considered benign. However, PTC is well-known to undergo cystic degeneration, and may thus present as a cystic lesion. This case was deceiving, because the lesion was perceived as a benign cyst on US imaging, with benign FNA findings consistent with a thyroid cyst. The location of the lesion in the isthmus, and the exaggerated posterior enhancement may have contributed to the deceiving attitude of this malignant lesion. The literature on thyroid nodules states that pure thyroid cysts are exclusively benign, but that entirely purely cystic lesions are rare. Main features that may help to distinguish pure cysts from other cystic lesions (e.g., cystic PTC) include smooth and thin wall and absence of solid echogenic structures.
Conclusion: PTC can masquerade as cystic lesions, and these can be deceiving, both upon US imaging and FNA. Clinicians should exercise caution before declaring a cyst as entirely benign.

Abstract #1206

OLD, COLD AND COMATOSE: WE WERE TOLD!

Nisha Acharya, MD, Suman Jana, Leann Olansky, MD

Case Presentation: We present a case of a 97- year- old male with past medical history significant for hypertension, bladder cancer and recently diagnosed hypothyroidism who presented to the emergency department with altered mental status. He was diagnosed with hypothyroidism two days prior to the presentation, and had been prescribed levothyroxine 25 mcg daily . Physical exam revealed a minimally responsive elderly gentleman with bradycardia (heart rate 42/min), hypothermia (temperature 31.4C),

hypotension (BP 70/30 mm Hg) and 1+ bilateral lower extremity edema. Rest of the physical exam was unremarkable. Lab work revealed urinary tract infection and TSH of 36.8 uU/mL (normal 0.400-5.500 uU/mL) and low free T3 of 1.5 pg/mL (normal 1.8-4.6 pg/mL). EKG showed sinus bradycardia with 1st degree heart block. Comprehensive metabolic panel showed normal glucose, normal sodium and acute kidney injury with elevated BUN and creatinine. Chest X-ray was not suggestive of pericardial effusion. CT-head was negative for any acute abnormality. A diagnosis of myxedema coma was made. Treatment was initiated with 100 mg IV hydrocortisone followed by 200 mcg IV levothyroxine. Supportive treatment included warming blanket, IV fluids, IV antibiotics, respiratory and inotropic support. Thyroid hormone replacement was continued with 50 mcg IV levothyroxine the next day, followed by PO levothyroxine 25 mcg daily. His clinical status improved rapidly and TSH normalized within 2 days. Blood cultures were negative. ACTH stimulation test ruled out adrenal insufficiency. Microsomal antibody was negative. He was later discharged under stable conditions.
Discussion: Myxedema coma results from severe, decompensated hypothyroidism leading to a depressed mental status, hypotension, and hypothermia. It is a serious, but rare medical emergency that carries a high mortality rate 30-60%. Elderly patients and those with cardiac disease are particularly at risk. Myxedema coma can occur insidiously as the result of severe long-standing hypothyroidism, or it can be precipitated by an acute event such as infection, myocardial infarction, cold exposure, or certain medications. In this case infection was the most likely trigger.
Conclusion: Myxedema coma is extreme form of hypothyroidism, which can lead to severe decompensated metabolic state. Early diagnosis and treatment can significantly curtail the mortality. Our case illustrates an example of multisystem manifestations of myxedema coma in extreme age group patients. To our knowledge, this is the oldest patient with myxedema coma that has been reported.

Abstract #1207

THE INTERNIST'S TUMOR - A CASE OF RENAL CELL CARCINOMA TO THE THYROID GLAND 12 YEARS AFTER RADICAL NEPHRECTOMY

Shaina Rozell, Diana Dean, MD

Case Presentation: A 77-year-old male presented for evaluation of a right thyroid nodule. Past medical history is significant for left radical nephrectomy with pelvic lymph node dissection for RCC stage III, 12 years prior, melanoma and right hypoechoic thyroid nodule (2006) managed annually with clinical exams. In 2010, CT of the chest revealed an increase in the size of the right thyroid nodule. Subsequent ultrasound demonstrated a 2.3 x 2 x 2.5 cm solid nodule in the mid right lobe of the thyroid. Thyroid function tests were unremarkable. Ultrasound-guided Fine Needle Aspiration (FNA) was performed and positive for metastatic RCC stage III, clear cell type. He underwent right thyroid lobectomy and isthmectomy with an uneventful postoperative course. He remains disease free with no further evidence of metastasis or local recurrence.
Discussion: The most common sites for RCC metastases are the lung, liver, adrenal gland, bone, brain, skin, contralateral kidney, and thyroid gland. Metastasis to the thyroid is exceedingly rare, accounting for less than 1% of all thyroid malignancies. In contrast to clinical practice, 24% of metastasis is discovered at autopsy, suggesting that unrecognized metastasis is more common than clinically recognized disease. Metastases may appear beyond 10 years after nephrectomy - making detection extremely difficult. Latent intervals up to 20 years have been reported with average time interval being 7.5 years. Factors accounting to the slow development of thyroid metastases include the high oxygen and iodine concentration of the thyroid, the filtering capacity of the lung, and the local cytoregulating effects of thyroid hormones. Radiologically, it is difficult to distinguish metastatic from primary malignant neoplasms as there are no specific findings differentiating the two, thus making diagnosis difficult. Preoperative FNA has been an effective way to distinguish primary and secondary thyroid carcinomas. Once diagnosis of metastatic disease has been confirmed; the patient should undergo metastatic work-up to rule out other distant metastasis. Thyroidectomy is recommended for local control and is considered the best effective treatment for metastatic thyroid tumor especially with no evidence of recurrence elsewhere. Chen et al. reported that 60% of patients with solitary thyroid metastasis were still alive after thyroidectomy during a median follow-up period of 5.2 years. In those with disseminated disease, surgical treatment is considered palliative.
Conclusion: Thyroid metastases from RCC should be considered in the differential diagnosis of a thyroid nodule, particularly in patients previously with history of nonthyroid primary malignancies.

Abstract #1208

INAPPROPRIATE TSH SYNDROME IN CLINICAL PRACTICE - NEED FOR A REVISIT

Adedayo Adegite, MD,

Objective: Inappropriate TSH syndrome represents a biochemical condition where raised levels of Free

T3 orT4 is accompanied by inappropriately normal or elevated TSH levels.The clinical scenarios where these biochemical pictures are observed are many and diverse. These biochemical pictures could be misinterpreted and confused by even endocrinologist.We seek to report and explore the different inappropriate TSH syndrome cases that were seen in our clinical practice.

Case Presentation: Inappropriate TSH syndrome is seen commonly among patients on thyroid hormone replacement therapy. Erratic intake of pills or blood taken for assay shortly after intake of pills are two major explanations. Antibody interference to either TSH or thyroid hormone represents another important cause of inappropriate TSH syndrome. These individuals are clinically euthyroid and are often diagnosed during routine thyroid function tests for some other reasons. A rare but perhaps very fascinating cause is acute psychosis.The thyroid hormone level in these patients usually normalizes after a few days. Inappropriate TSH syndrome could also be observed in the recovery phase of the patients with sick euthyroid syndrome. It could also be encountered frequently among patients who had been on amiodarone for some time. Other potential causes are Amphetamine and heparin use. Thyroid hormone resistance and TSH secreting pituitary tumor(TSHoma) are 2 rare but clinically important causes of Inappropriate TSH syndrome. A good history, antibody screening and further laboratory investigations are useful in identifying these two conditions. From the young lady with antibody interference,the patients on thyroid hormone replacement, the old lady on amiodarone therapy,the young man with acute psychosis, we explore the two most clinically important causes of inappropriate TSH syndrome.

Discussion: Inappropriate TSH syndrome is seen in a wide range of clinical conditions but a step wise screening approach helps in identifying the more clinically important cases.

Conclusion: Inappropriate TSH syndrome represent an uncommon but clinically important thyroid function picture which are often confused and misinterpreted in many quarters. A good knowledge of the differentials help in excluding the less clinically important causes.

Abstract #1209

UNILATERAL GRAVES DISEASE PRESENTING AS THYROID STORM

Swapna Kolukula, MD, Bilal Rizvi, Kelsi Lacock, Robert Anderson, MD, FACP

Case Presentation: A 62 year old woman with a recent diagnosis of hyperthyroidism presented with acute onset of diaphoresis, dyspnea, near-syncope and was admitted with a working diagnosis of NSTEMI and thyrotoxicosis. Later she was transferred to the ICU for atrial fibrillation with rapid ventricular rate, hypertensive urgency and acute pulmonary edema. Vitals: Blood pressure 180/115 mm Hg, heart rate 170 per min, respiratory rate 52 per min, temperature 36.8°C, pulse oximetry low 80s. She was anxious, confused, and diaphoretic with peripheral cyanosis. Transthoracic Echocardiogram: Ejection fraction of 20%, Right ventricular systolic pressure 60mmHg [15-30mmHg]. She stabilized with IV metoprolol and BIPAP. Contrast chest CT scan was negative for pulmonary embolism, but showed an enlarged left thyroid lobe with tracheal deviation. She was started on IV heparin drip, bolus IV lasix, IV esmolol drip, IV hydrocortisone, oral propylthiouracil. TSH-0.01 microIU/mL (0.34-5.60), FT4-4.86 ng/dl (0.6-1.60), FT3-5.6 pg/ml (2.5-3.9); anti-thyroid peroxidase antibodies and thyroid stimulating immunoglobulin were positive. Thyroid imaging studies one week prior to admission showed a diffusely heterogeneous and hypervascular enlarged left lobe (7.9x4.5x3.8 cm) and a smaller right lobe (4.0x1.3x1.1 cm) with similar blood flow and heterogeneity on ultrasound. Thyroid scan (123I) revealed an enlarged left thyroid lobe with a vague photopenic region, a suppressed right lobe, and 4 and 24 hour uptakes of 56.5% and 66.3%, respectively. Her cardiopulmonary status slightly improved, but her Thyroid Storm Score (Burch & Wartofsky, EMCNA 1993) of 60 was highly suggestive of thyroid storm. Steroids were switched to dexamethasone and she was treated with high doses of propylthiouracil, potassium iodide, and metoprolol. Upon discharge propylthiouracil was tapered and switched to methimazole with a plan for radioiodine ablation.

Discussion: Unilateral Graves disease as an autoimmune clinical entity might co-exist in a nodular goiter or be confused with a toxic nodular goiter in patients with unilateral thyroid enlargement. This diagnostic dilemma can occur even in the presence of sophisticated diagnostic imaging and laboratory tests. The Thyroid Storm Score confirmed the need for early aggressive management of life-threatening hyperthyroidism, and placed this patient in the category of thyroid storm even in the absence of fever as a classical criterion.

Conclusion: Unilateral Graves disease is a challenging clinical entity. Early diagnosis and treatment are crucial for prevention of the life-threatening syndrome of thyroid storm.

Abstract #1210

A RARE CASE OF AMIODARONE INDUCED THYROTOXICOSIS AND THIONAMIDE AGRANULOCYTOSIS

Sudeep Dhillon, MD, Alexander Shifrin, MD, FACS, Fransisco Cruz, Danielle Lann, MD

Objective: To present a rare case of amiodarone induced thyrotoxicosis (AIT) and thionamide agranulocytosis and to discuss therapeutic options.
Case Presentation: 61 year old man with a 10 year history of Amiodarone therapy for atrial fibrillation, presented with thyrotoxicosis. TSH was <0.01 uIU/mL, FT4 was 2.31 ng/dL, FT3 291 pg/dL. IL-6 was 5.88 pg/dL. Thyroid uptake scan showed significantly decreased uptake: 0.6% - 5 hr; 0.5% - 24 hr. Thyroid ultrasound showed a subcentimeter right lower pole nodule with normal vascularity. 24 hour urinary iodide collection was >2000 mcg. Amiodarone was discontinued, while methimazole and high-dose steroids were instituted for what appeared to be a mixed type 1 and 2 AIT picture. Hyperthyroidism was refractory to treatment despite escalating methimazole doses (up to 60 mg daily in divided doses). After three months, the patient developed weakness, fatigue and fever 103 oF, WBC 0.3 k/uL. Neutropenic fever was diagnosed. Methimazole was discontinued and WBC count improved to 6.9 k/uL over the next week and remained stable. However, the patient remained thyrotoxic despite continued high dose steroids (up to 60 mg prednisone daily). Thyroidectomy was ultimately performed. Surgery was well tolerated, and the patient successfully achieved euthyroidism with thyroid hormone replacement.
Discussion: AIT is a well recognized entity with a prevalence of 3% in the United States. Due to lack of standardized control trials, AIT remains a therapeutic challenge in many cases. Withdrawal of amiodarone and standard medical management with thionamide and/or corticosteroids is first-line treatment. Thionamides and corticosteroids are often coadministered for AIT in cases where it is difficult to distinguish between type 1 and type 2 thyrotoxicosis. Often, high doses of thionamide therapy are required for a type 1 or mixed AIT picture as a result of elevated iodine stores within the thyroid gland inhibiting the action of these drugs on thyroid hormonogenesis. Agranulocytosis, occurring in approximately 0.2 to 0.5% of cases in the US, appears to be dose-related with methimazole. In cases of refractory thyrotoxicosis or thionamide toxicity, surgical treatment is warranted.
Conclusion: This case illustrates a combination of two rare complications and confirms surgical treatment as a viable option in hyperthyroidism resistant to standard medical therapy.

Abstract #1211

TRAUMATIC THYROIDITIS: A UNIQUE PRESENTATION

Margie Banzuelo-Rio, MD, Sunil Asnani, MD, FACE

Objective: To discuss a case of Thyroid Storm after parathyroidectomy for primary hyperparathyroidism.
Methods: Clinical and laboratory findings of a case are presented, and the relevant literature is reviewed.
Case Presentation: A 70-year-old woman presented to the hospital with worsening lower extremity swelling over the last few days. She was found to have a fever of 103*F and tachycardia of 137/min. Relevant history included a neck exploration and parathyroidectomy for primary hyperparathyroidism 1 week prior to admission and a contrast CT of the neck a few weeks prior to surgery. Physical examination revealed a healing scar, sinus tachycardia, bibasilar crackles and pedal edema. Laboratory values: TSH 0.08 (0.34-5.60) µIU/mL, Free T4 2.66 (0.50-1.26) ng/dL, Thyroglobulin level 25.9 IU/mL, Thyroid Stimulating Immunoglobulin 101% (<110%) and Sr. Calcium 8.9 mg/dL. She was admitted to the ICU with the diagnosis of thyroid storm and aggressively treated with glucocorticoids, beta blockers, methimazole and cholestyramine. Patient recovered over the next few days and was discharged to rehabilitation.
Discussion: Parathyroidectomy-induced thyroiditis is under-recognized. Upwards of 30% patients may experience transient hyperthyroidism after surgery. This seems to be a self-limiting phenomenon and resolves within 1-2 weeks. Disturbance of the microcirculation in the thyroid gland by manipulation during operation might be a cause of the transient hyperthyroidism after parathyroidectomy. Patient with clinically significant renal disease and tertiary hyperparathyroidism are obviously at higher risk due to the extent of the surgery. The majority of patients are asymptomatic. Our patient clearly was at a disadvantage given her advanced age and contrast exposure in addition to her thyroid manipulation. The presentation of our patient with thyroid storm after parathyroidectomy for primary hyperparthyroidism makes it a unique presentation.
Conclusion: Surgery induced 'traumatic' thyroiditis can pose significant clinical problems. Candidates for parathyroidectomy should be informed of this potential complication, and thyroid function should be assessed if clinically indicated.

Abstract #1212

UNUSUAL CASE OF FULMINANT HEPATOCELLULAR INJURY DUE TO METHIMAZOLE

Richard Pinsker, MD, Bhaveshkumar Vekariya, Mayur Gohel, Mahendra Patel, Asit Mehta, Kelly Cervellione

Case Presentation: A 51 year old female presented to the ED with altered mental status, combative behavior and respiratory distress. She ultimately required intubation. She had been taking methimazole for hyperthyroidism for the last three months. Labs revealed glucose 21, high anion gap metabolic acidosis, alk phos 439, AST 5,337, ALT 2,025, biliirubin 1.7, ammonia 76, and INR 4.6. LFT's were normal before methimazole treatment. All other causes of fulminant liver failure including shock liver, viral hepatitis, acetaminophen toxicity, and HELLP syndrome were ruled out. Methimazole was discontinued. LFT's returned to normal in 15 days. The patient was extubated and slowly recovered. Hyperthyroidism was treated with beta blockers. Radioactive iodine treatment was given a month later.
Discussion: Liver injury is a serious complication of anti-thyroid drugs. The incidence is higher with propylthiouracil as compared to methimazole. Cholestasis is often predominant. Our case showed predominant hepatocellular damage. This patient improved with discontinuation of methimazole thus implicating this drug as the proximate cause of the liver damage.
Conclusion: Liver damage is one of the rare side effects of anti-thyroid medications. The exact mechanism is not known. Most patients respond to early withdrawal of anti-thyroid medication resulting in a good long term prognosis and do not require an invasive workup. If untreated, fulminant liver failure and death may result. Early recognition and withdrawal of the offending agent are key.

Abstract #1213

MICRONODULAR RADIOGRAPHIC PULMONARY PATTERN IN METASTATIC MEDULLARY THYROID CARCINOMA - A CASE REPORT

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Objective: Medullary carcinoma of the thyroid is a rare form of all thyroid malignancies, thereby limiting the clinical nature and the ability to optimize diagnostic tools. Here we present a case of a micronodular radiographic pulmonary pattern in metastatic medullary thyroid cancer to enhance awareness of the disease process.
Case Presentation: A young man of 26 years Bakery worker referred to Endocrine unit of BSMMU, Dhaka, with the complaints of persistent multiple swelling in the neck for 6 years and frequent loose motion for same duration. The swelling had progressively increased in size in the last two years. Patient also gave history of irregular fever for last two months. There was no such family history. His cervical lymph node of all groups were palpable, firm to hard in consistency, nontender, some are matted but not fixed with underlying structure and no discharging sinus. He had multinodular goiter with dominant right sided non tender nodule. He gave history of 5 kg weight loss in last 2 months. Chest X- ray showed multiple milliary (micronodular) mottling in both lung fields. His ESR was normal, MT and sputum for AFB were negative. On the basis of above findings patient receive anti TB, Category - 1 for two months from local DOT centre, but there was no improvement. Patient came to us for evaluation of thyroid swelling. His thyroid function tests and thyroid scan were normal. FNAC of thyroid nodule revealed few follicular and Hurthle cells. Lymph node biopsy showed metastatic medullary carcinoma of thyroid. S.calcitonin level was > 2000 pg/dl. Patient was transferred to Surgery department for total thyroidectomy with bilateral neck dissection. After a successful surgery patient died on 2nd postoperative day.
Discussion: metastatic medullary carcinoma may present with bilateral cervical lymphadenopathy & milliary mottling in the lungs. Early detection of medullary carcinoma is sometimes very difficult but extremely important for overall survival. High index of suspicion is important to exclude medullary carcinoma.
Conclusion: Metastetic medullary carcinoma may mimic disseminated tuberculosis.

Abstract #1214

MALTOMA OF THE THYROID: A CASE REPORT

Aarti Manchanda, MD, Yannis Guerra, Rafid Kouz, Paula Kovarik, Leon Fogelfeld

Case Presentation: A 58 year old male presented to the hospital with an eight month history of painless cervical "lumps" in the neck, dysphagia to solids and weight loss of twenty pounds. On exam he had diffuse, firm, painless thyromegaly with bilateral cervical and axillary lymphadenopathy. Laboratory revealed microcytic anemia, TSH of 61.44uIU/ml (0.27-4.20 uIU/ml) and free T4 of 0.81ng/dl (0.93- 1.70 ng/dl). Anti microsomal and anti thyroglobulin antibodies were present. Computerized

tomography confirmed exam findings. Incisional biopsy of right thyroid lobe showed replacement of the parenchyma by sheets of intermediate-sized, irregular lymphoid cells. Immunohistochemical stains were positive for CD5, CD20, and cyclin D1. Flow cytometry showed a monoclonal B-cell population with expression of CD5, CD 19, CD20k, CD45, HLA-Dr and kappa immunoglobulin light chain consistent with a diagnosis of MALT(mucosa associated lymphoid tissue) lymphoma of the thyroid. Colonoscopy and endoscopy revealed submucosal masses in the rectum, ascending colon, antrum and duodenum. Their biopsy and immunohistochemical profile confirmed the presence of MALToma. Chemotherapy was started with complete resolution of the goiter. After six cycles, the patient decided to stop treatment.

Discussion: Primary thyroid lymphomas constitute upto 5% of all thyroid malignancies and are divided into non hodgkin lymphomas of B and C cell type. MALTomas represent 7 to 8% of B cell lymphomas. Thyroid involvement is rare. Although thyroid is devoid of lymphocytic tissue, MALTomas may arise as a result of a chronic inflammatory or autoimmune process. Chronic autoimmune thyroiditis is associated with an increased risk. Lymphoepithelial lesions on histology are characteristic but not pathognomic for MALTomas. Also, coexistence of reactive and neoplastic processes may cause difficulty in making a cytologic or histologic diagnosis. Immunocytochemistry and flow cytometry are used to confirm or exclude the diagnosis. Immunocytochemistry shows expression of pan B antigens including CD19, CD20, CD22. CD5 expression is associated with aggressive disease Treatment includes radiotherapy for localized and chemotherapy for disseminated disease. These tumors can recur at any other mucosal surface. Hence long term follow up is necessary.

Conclusion: We present a rare case of thyroid MALToma. This lymphoma usually arises in the setting of chronic autoimmune thyroiditis. Cytology and histology may not distinguish between reactive or low grade lymphomatous thyroid processes and immunophenotypic and molecular techniques may be required to diagnose MALToma. Given its rarity, the nature of optimal treatment and long term follow up remains controversial.

Abstract #1215

UNMASKING OF ADRENAL INSUFFICIENCY BY THYROXINE REPLACEMENT

Prathima Jasti, MD, Ioannis Papagiannis, MD

Objective: To reiterate the need to monitor for signs of adrenal insufficiency in patients with hypothyroidism who do not respond to appropriate levothyroxine therapy.

Methods: We present a case report of a patient with hypothyroidism and worsening symptoms after treatment.

Case Presentation: A 51 year old woman with no significant past medical history was started on levothyroxine by her primary care physician for symptoms of fatigue and TSH of 114 U/mL. She then presented to us 4 months later with worsening symptoms of fatigue, dizziness, orthostatic hypotension, weight loss and hyperpigmentation despite normalization of TSH. She failed to respond to high dose cortrosyn stimulation test with cortisol levels of 1mcg/dL and 1.2 mcg/dl before and after administration of cortrosyn respectively with elevated ACTH of 1088pg/ml , which confirmed primary adrenal insufficiency. Replacement of hydrocortisone and fludrocortisone immediately improved her symptoms.

Discussion: Hypothyroidism is a common endocrine disorder diagnosed and managed by primary care physicians as well as endocrinologists worldwide. Addison's disease is much rarer with reported prevalence of about 120 cases per million population. Autoimmune adrenalitis is the most common cause of adrenal insufficiency, affecting almost 80 % of cases. Patients with hypothyroidism may have other autoimmune disorders like type 1 diabetes, Addison's disease or pernicious anemia as part of autoimmune polyglandular syndrome type II. The coexistence of thyroid and adrenal insufficiency is greater than expected and often undiagnosed. The symptoms of both these disorders are overlapping and often insidious and ambiguous which makes the diagnosis challenging. Elevated TSH with normal or low free T4 has been reported in cortisol deficiency, which makes it even more confusing. Chronic glucocorticoid deficiency can stimulate TSH secretion directly at the level of the pituitary. Hypothyroidism is associated with low cortisol secretion as a response to decreased metabolic rate. Replacement of thyroxine increases the metabolic rate demanding more cortisol, without which patients may present with adrenal insufficiency.

Conclusion: Patients treated for hypothyroidism usually report improvement of symptoms in 2-3 weeks. If not so, clinicians should have a low threshold for suspicion of adrenal failure as prompt diagnosis and treatment will avoid fatal adrenal crisis. Patients with hypothyroidism who show stigmata of autoimmune disorders like vitiligo, alopecia or family history of auto immune disorders should be tested for adrenal insufficiency before initiating thyroxine therapy.

Abstract #1216

A META-ANALYSIS ON THE EFFECTIVENESS OF RADIOACTIVE IODINE VERSUS THYROIDECTOMY IN THE TREATMENT OF GRAVES' DISEASE

Shamsa Ali, MBBS, Salem Noureldine, Bradly Genovese, Elizabeth Gleeson, Ralph Tufano, Ajaz Banka, MBBS, Emad Kandil, MD

Methods: A Pubmed search was conducted from January 2001 to May 2011. Outcomes of interest included post-operative hypothyroidism, euthyroidism and persistent/ recurrent hyperthyroidism. Patients were stratified into two groups based on their treatment modality; radioactive iodine and surgery. Success was defined as postoperative euthyroidism or hypothyroidism
Case Presentation: Of the 14,245 patients, 4,546 underwent surgery and 9,699 had radioactive iodine. 3,158 patients had subtotal thyroidectomy and 1,388 had total-thyroidectomy. Surgery was found to be 3.44 times more likely to be successful than radioactive iodine ($p<0.001$). Subtotal and total thyroidectomy were found to be 2.33 and 94.45 times more likely to be successful than radioactive iodine ($p<0.001$), respectively
Discussion: Recent American Thyroid Association guidelines do not favor a particular treatment for Graves' disease (GD). In the US, radioactive iodine has been the preferred option. We believe this is largely influenced by the historical fixed practice. No in depth analysis has been performed comparing the treatment options. We used meta-analysis to compare the treatment options for GD
Conclusion: Surgery should be considered as the mainstay approach in the treatment of Graves' disease, with total-thyroidectomy being the preferred option

Abstract #1217

THYROID STROM COMPLICATED WITH FULMINANT HEPATIC FAILURE (FHF)

Shamsa Ali, MBBS, Catherine Hambleton, Sima Mistry, Salem Noureldine, Nicholas Avitabile, MD, Ajaz Banka, MBBS, Emad Kandil, MD

Case Presentation: A twenty-two year-old woman with a history of pregnancy-related Graves' disease presented with thyroid storm. The patient underwent medical treatment with antithyroid medications during her pregnancy but she was advised to discontinue these antithyroid medications when she delivered her baby, 5 months prior to her presentation. Physical examination findings included mild diffuse thyromegaly, a thyroid bruit ,heart rate between 130-140 beats per minute and abdominal exam was significant for mild tenderness on palpation of the right upper quadrant. Her liver function tests included a total bilirubin of 27mg/dL (normal: <1.1mg/dL) ,INR of 1.6 (0.8-1.2), AST was 1400 U/L (normal : <35 U/L), ALT was 700 U/L (normal : 30-65 U/L), alkaline phosphatase 337 U/L (normal: 40-120 U/L), TSH 0.006 mlU/L and free T4 > 8.0 ng/dL (normal range: 0.76-1.46ng/dL). During her hospitalization her AST peaked at 1803U/L, ALT 1218U/L and alkaline phosphatase 327, total bilirubin 28.0 t, and INR peaked at 2.00 . Inpatient medications included antithyroid drugs, propranolol, hydrocortisone, and SSKI. The patient underwent total thyroidectomy and liver transplantation from a cadaveric donor twelve days post thyroidectomy. Free T4 had diminished significantly to 1.36 ng/dL, and 17 days post liver transplantation, AST was 25 U/L, ALT was 83 U/L, alkaline phosphatase was 248 U/L
Discussion: FHF associated with thyroid storm is extremely rare however liver dysfunction in conjunction with hyper-thyroidism is a well-demonstrated phenomenon. The liver contributes to thyroid hormone metabolism. Liver function tests are elevated in 27-37% of hyperthyroid patients, even though most of these patients showed no clinical signs or symptoms of liver disease. Fong et al report elevated serum bilirubin in 50% of uncomplicated hyperthyroid patients and 79% of patients with hyperthyroidism-induced congestive heart failure. Hepatic injury in hyperthyroid patients has been most often attributed to relative ischemia. Due to the hypercatabolic state of hyperthyroid patients, the liver requires more oxygen but does not receive greater blood flow, creating a relative ischemia. Other causes of liver dysfunction in the setting of hyperthyroidism include malnutrition, heart failure, and direct effects of thyroid hormones
Conclusion: Fulminant hepatic failure is a rare but serious complication of thyroid storm. Rapidly progressing thyroid storm and FHF should be considered for thyroidectomy and liver transplantation

LATE BREAKING

Abstract #1300

PAPILLEDEMA AND INCREASED RADIOACTIVE IODINE UPTAKE IN THE EYE: A RARE COMPLICATION AND A UNIQUE PRESENTATION IN A PATIENT WITH THYROID CANCER

Nesreen BenHamed, MD, Vishnu Garla, MBBS, Omolola Olajide, MBBS, Michael Krasnow, Abid Yaqub, MBBS

Case Presentation: A 40 year old male patient noticed a neck mass while shaving. CT neck revealed a 2 cm mass on the superior pole of the left thyroid lobe and a 4 cm calcified mass inferior to this. FNA biopsy of the thyroid nodule was suspicious for papillary thyroid cancer. He subsequently had a total thyroidectomy and modified left radical neck dissection. Pathology revealed a 1.9 cm classical papillary thyroid cancer with extra thyroidal extension and extensive cervical lymph node metastasis. Following surgery, Thyroid hormone withdrawal protocol was initiated in preparation for radioactive iodine ablation. He received 148 mci of RAI-131. Post therapy I-131 WBS performed 8 days following RA-131 therapy showed intense uptake in the right eye. Patient stated he had increased lacrimation and photophobia in the right eye and was referred to ophthalmology. Fundoscopy revealed bilateral papilledema with greater severity on the right. MRI of the brain and orbits was normal. Ocular optical tomography (OCT) did not reveal any evidence of intraocular metastasis. A Lumbar Puncture was performed which revealed a high opening pressure but otherwise normal CSF fluid analysis with no abnormal cells. A diagnosis of Pseudo tumor cerebri was made. Patient's ocular symptoms and papilledema improved over several weeks with conservative management.
Discussion: Papilledema resulting from increased intracranial hypertension has been reported both in patients with hypothyroidism as well as those who underwent neck dissection. Hypercoaguability and increase in CSF protein are some of the mechanisms implicated in the pathogenesis. Radioactive whole body scans can be useful in the diagnosis of occult thyroid metastasis, however false positives may occur. We think that our patient developed intracranial hypertension as a combination of neck dissection, radiotherapy as well as iatrogenic hypothyroidism developed as a result of the thyroid hormone withdrawal protocol. The increased uptake is probably due to underlying inflammation of the optic disc.
Conclusion: To our knowledge this is the first case report describing the occurrence of papilledema and increased radioactive iodine dye uptake following radioactive iodine ablation

Abstract #1301

TRANSCRIPTIONAL PROFILING OF EARLY ADRENOCORTICAL CARCINOMA DEMONSTRATES THAT PTTG1 OVER-EXPRESSION IS ASSOCIATED WITH POOR SURVIVAL AND IDENTIFIES PERTURBATIONS OF THE G2/M TRANSITION AS THERAPEUTIC TARGETS

Kathryn Coan, MD, Elizabeth Stephan, Shripad Sinari, Michael Barrett, Kathleen Delgiorno, Clive Grant, MD, Richard Komorowski, Paul Gonzales, David Mount, Kimberly Bussey, Michael Demeure, MD, MBA

Objective: Adrenocortical carcinoma (ACC) is associated with poor survival rates due to aggressive biology and a lack of effective systemic therapy. An understanding of the molecular pathogenesis is necessary in order to develop novel treatments.
Methods: RNA from 19 ACC tumors and 4 normal adrenal glands was profiled on Affymetrix U133 Plus 2 expression microarrays. Pathway analysis was performed with Gene Set Enrichment Analysis (GSEA). Pathway enrichment and transcriptional regulation were analyzed with GeneGo. Western blots were used to assess protein levels. Drug dose response curves were generated to assess drug toxicity.
Results: GSEA identified marked dysregulation of the cell cycle control, focused on sister chromatid adhesion and cytokinesis in the G2/M transition. GeneGo identified potential alterations in p53 activity as genes that are normally repressed by p53 were over expressed and those normally promoted had decreased expression. Over-expression of PTTG1, which encodes securin, a negative regulator of p53 and a protein involved the G2M transition, was identified as a marker of poor survival. Median survival for patients with tumors expressing high PTTG1 levels (log2 value > 8.5) was 1.2 years compared to 9.8 years if tumors expressed lower levels (P=0.002). Treatment of two ACC cell lines with a histone deacytlase inhibitor, vorinostat, decreased securin levels in both cell lines and inhibited cell growth with IC50 values of 1.69 uM and 0.891 uM, for SW-13 and H295R, respectively.
Discussion: Our expression profiling analyses in ACC have identified the G2/M transition and in particular, sister chromatid adhesion and cytokinesis, as perturbed in ACC. Notably, the chemotherapy regimen for which the most promising phase II results are available in ACC includes doxorubicin, etoposide, cisplatin and mitotane. Doxorubicin and etoposide both have mechanisms of action that involve proteins in the G2/M transition. Over expression of PTTG1, which plays a role in both sister chromatid adhesion and p53 regulation, was associated with poor prognosis in our samples. Treatment

with vorinostat decreased securin levels and inhibited cell growth in two ACC cell line may prove to have potential therapeutic benefit.
Conclusion: Over-expression of PTTG1 correlates with poor survival. Further investigation into the role PTTG1 over-expression in the pathogenesis of ACC and validation of PTTG1/securin as a prognostic marker or potential therapeutic target is warranted.

Abstract #1302

SCREENING FOR CARDIOVASCULAR AUTONOMIC NEUROPATHY AMONG TYPE II DIABETIC PATIENTS FORM GORGAN, NORTHERN IRAN

Hamid Bazrafshan, MD, Seyed Mehran Hosseini, Ali Reza Maleki, Mohsen Jamshir

Objective: Cardiovascular autonomic neuropathy (CAN) is one of the most prevalent and most important diabetic autonomic neuropathies. Silent myocardial infarction, respiratory insufficiency and increased risk of mortality are some consequences of CAN. Prevalence of CAN in northern Iran has not been studied yet. Present study aimed to evaluate the prevalence of CAN in diabetic patients from Gorgan, Iran, using standard methods.
Methods: This cross-sectional study has conducted in Diabetes Clinic of 5th Azar Hospital, Gorgan. Volunteering patients who have fulfilled the inclusion criteria recruited in the study. We used the resting heart rate (RHR), heart rate (Ratio30:15), systolic blood pressure (ΔSBP) and diastolic blood pressure change (ΔDBP) after standing and corrected QT interval (QTc) for diagnosis of CAN in our patients. The results of each test were classified into two separate group and scored as follow; 0, for normal findings and 1, for abnormal findings. Then, total CAN score was calculated by adding points from these five tests.
Results: Totally 70 patients with mean age of 55.19 ± 9.79 included in this study. 48 (68.6%) were female. After summing of the final score for each patient, 10 (14.3%) had 0 point (CAN negative), 35 (50%) had 1 point (borderline) and 25 (35.7%) had ≥ 2 points (CAN positive). Patients with duration of diabetes more than 10 years had significantly higher CAN score compared to patients with less duration of diabetes (P=0.041). BMI over 30 kg/m2 resulted in QTc prolongation in a significant number of patients with type 2 diabetes (P=0.02). Systolic blood pressure change after standing (ΔSBP) had highest correlation with CAN severity (score) among other tests (r=0.509).
Discussion: The reported prevalence of CAN varies, depending on whether studies have been carried out in the community, clinic, or tertiary referral center. The variance among prevalence studies also reflects the type and number of tests performed and the presence or absence of signs and symptoms of cardiac autonomic neuropathy.
Conclusion: Prevalence of CAN among these patients was remarkably high when considering the patients with borderline CAN (score 1) in addition to CAN positive patients. Duration of diabetes more than 10 years was the most important risk factor among this population. Application of simple test of ΔSBP is recommended for routine outpatient evaluation of diabetic CAN.

Abstract #1303

UTILITY OF ESTIMATED TOTAL BODY COMPOSITION (AS ASSESSED BY DXA SCANNING) IN THE DIAGNOSIS OF OBESITY IN POST MENOPAUSAL WOMEN IN COMPARISON TO CONVENTIONAL BMI METHOD

Adam Maghrabi, MD, Abid Yaqub, MBBS

Objective: 1) To study the utility of estimated total body composition in diagnosis of obesity in post menopausal women in comparison to the conventional BMI method,
2) To assess the correlation of Fat Mass Index (FMI), Body Mass Index (BMI) and Android to Gynoid (A/G) ratio with various components of metabolic syndrome, and
3) To study the correlation between FMI, BMI and Percentage Body Fat (PBF).
Methods: We studied the charts and DXA scans of 99 post menopausal women being followed at the department of medicine clinics. We calculated FMI, BMI, A/G ratio and percentage body fat (PBF) from their DXA scans and studied the correlation between FMI, BMI and PBF as well as the correlation between FMI, BMI and A/G ratio with various components of metabolic syndrome.
Results: Misclassification of the weight categories between BMI and FMI was found in 30% of the studied population. 27% of the patients were upgraded to a higher weight class by FMI. 8%, 14%, 4% and 2% of patients, classified as Normal, Overweight, Obese class 1 and Obese class 2 by BMI criteria, respectively, were classified as Excess Fat, Obese class 1, Obese class 2, and Obese class 3, respectively, by the FMI criteria. 80% of women with normal BMI were found to have PBF > 34% (which is considered excessive fat for their age group). Both BMI and FMI correlated significantly with Diabetes Mellitus, hyperlipidemia and sleep apnea but not with hypertension and impaired fasting glucose. A/G ratio did not correlate significantly with any of the above mentioned metabolic syndrome variables. BMI, FMI and PBF were all significantly correlated with each other.
Conclusion: Our study highlights the limitations of conventional BMI criteria in diagnosing and classifying obesity in post menopausal when compared to methods

utilizing estimated whole body composition as assessed by DXA scans. Further studies are needed with larger and more diverse population samples to further confirm our findings.

Abstract #1304

COLESEVELAM HCL: GLYCEMIC AND LIPID PARAMETER EFFECTS IN PATIENTS WITH TYPE 2 DIABETES MELLITUS (T2DM) TREATED WITH METFORMIN-BASED THERAPY AND A STATIN

Michael Jones, Ph.D., Harold Bays, MD, FACP

Objective: Effective glycemic and lipid control are cornerstones of the treatment of T2DM. In a previously-reported 26-week study in adults with T2DM who had A1C values of 7.5%-9.5% on metformin-based therapy, subjects were randomly administered colesevelam HCl (COL) 3.75 g/day (N=159) or matching placebo (PBO; N=157). This was in addition to their pre-study background therapies. In the total study population, COL had a mean treatment difference in A1C of −0.54% (p<0.001) and LDL-C of −15.9% (p<0.001). Sixty-two COL recipients (39%) and 75 PBO recipients (42%) were treated with pre-study statin therapy, which continued throughout the trial. Statins may mildly increase glucose levels, which might conceivably affect the glucose and lipid efficacy of metabolic drug therapies.
Methods: This post-hoc analysis of COL versus PBO evaluated the subgroup of statin users. The analysis included the least-squares mean/median percent changes from baseline in glycemic and lipid metabolic parameters.
Results: Statin users receiving COL or PBO had similar demographic characteristics and similar baseline A1C compared to the overall study population (8.1%). Regarding results, COL reduced A1C more than placebo in both statin users (mean treatment difference −0.63% [95% CI −0.97%, −0.30%]; p=0.0003) and statin non-users (−0.49% [−0.77%, −0.20%]; p=0.001). COL also reduced LDL-C levels more than PBO in both statin users (mean treatment difference −16.4% [−26.9%, −6.0%]; p=0.0024) and statin non-users (−15.8% [−22.3%, −9.3%]; p<0.0001). Among statin users, compared to PBO, COL reduced non-HDL-C (mean treatment difference −11.7%, p=0.021), reduced total cholesterol (−8.5%, p=0.025), and reduced apolipoprotein (apo) B/apo A1 (−0.1%, p=0.014), with no change in HDL-C and apo A1 levels. Also in statin users, compared to PBO, COL tended to reduce apo B (−6.9%, p=0.090), but the difference was not statistically significant. COL did not significantly alter triglyceride levels compared with PBO in statin users (median treatment difference −1.6% [Interquartile Range −15.2, 11.2%]; p=0.885).
Discussion: In this post-hoc analysis compared with PBO, COL significantly reduced A1C and LDL-C levels in adults with T2DM, whether subjects were statin users or statin non-users. In fact, the reduction in A1C with COL was numerically greater in statin users, compared with statin non-users.
Conclusion: To whatever extent statins may affect glycemic parameters, this post-hoc analysis did not support any attenuation of COL efficacy depending upon statin use, or lack of statin use.

Abstract #1305

CORRELATIONS BETWEEN FOOD INSECURITY, DIETARY HABITS, AND CONTROL OF TYPE 2 DIABETES IN AN APPALACHIAN AREA

Omar Akhtar, MBBS, Abid Yaqub, MBBS

Objective: Several studies have suggested a relationship between food insecurity and diabetes. We designed this study to assess the prevalence of food insecurity in a cohort of DM-2 patients from Appalachia and to study the correlation between food insecurity, life style habits, medication compliance, self-perception of body image and glycemic control.
Methods: Our study population included DM-2 patients treated at the Department of Medicine outpatient clinics. A total of 100 patients was surveyed. We devised various questionnaires to obtain information pertaining to food insecurity, dietary habits, physical activity, body image perception and medication adherence. Morisky 8 Medication Adherence Questionnaire was used to assess medication adherence. International Physical Activity Questionnaire was used to assess activity level. Food insecurity was assessed by a questionnaire from USDA Community Food Security Assessment Tool, and a scale was devised to categorize patients into food secure, insecure and those experiencing hunger. BMI-based pictorial instruments were used to analyze self perception of body image. Other variables studied were obtained by chart review from the electronic medical records and included information regarding glycemic control, co-morbid conditions, BMI and patient's medical insurance.
Results: Statistical Analysis was performed using R v 2.13. Food insecure group was found to have higher Mean A1c as compared to food secure group (8.3 % vs. 7.2 % respectively; p-value <0.05). Food secure group was also found to be more medication adherent as compared to food insecure group (p-value <0.05). Results from dietary survey showed increased protein intake in the food secure as compared to food insecure (p<0.05)group, but there

was no difference in their fat or carbohydrate intake. There was no statistical difference between exercise activity level and prevalence of depression between these groups. Obese patient were found to have more accurate body image perception as compared to non-obese patients ($p < 0.05$). Analysis did not show any correlation of A1c with exercise level, type of insurance or medication adherence.
Discussion: Patients with food insecurity had worse glycemic control and poorer medication adherence as compared to their food secure counterparts in our study population. Patients with DM-2 can be screened for food security because of potentially negative implications on diabetic care. Further studies in larger and more diverse patient populations are recommended to study this association furthur.
Conclusion: Screening patients with diabetes for food insecurity should be considered while treating these patients.

Abstract #1306

ENDOCRINOLOGIST AS QUALITY CONTROLER OF THE RESULTS OF THYROID NODULE SURGICAL PATHOLOGY. A CASE OF WARTHINS VARIANT OF PAPILLARY CARCINOMA PTC MISDIAGNOSED AS BENIGN PAPILLARY HYERPLASIA IN HASHIMOTOS THYROIDITIS CHT.

Richard Guttler, MD, FACP,FACE,ECNU

Objective: The patient with a thyroid nodule who has an endocrinolgist deserves to have a careful follow up of the results of the surgery. This requires the endocrinologist, to obtain and read the pathology report,and talk with the pathologist about the diagnosis before beginning the post surgery therapy.
Methods: A case will show how an endocrinologist doing on-site adequacy assessment AA, and biopsy, can take control of their patients cytology and surgical pathology results.
Case Presentation: 27 Y/O female with a CHT goiter with nodules.An ultrasound found a suspicious 1 cm nodule in a background of CHT.. During FNA the endocrinologist collected a first pass for on-site AA. The technique was adequate, but cells seen suggesting PTC. “On-the fly” a needle washout for miR Inform markers was obtained. Cytology DX = 75% risk of PTC. After a total thyroidectomy, the pathologists at a major medical center did not find PTC, but just CHT with the nodule having papillary “features”. They saw rare inclusions , but no other features to diagnose PTC. The endocrinologist called the pathologist and told of a BRAF mutation, highly suspicious cytology. Outside expert second opinion confirmed it was cancer, but that it was an invasive Warthin’s-like variant of PTC. The expert tried to discuss the case with the pathologist and was told she was unable to discuss the case but faxed over revised report. The new report was also a problem. Not a classic PTC, and failed to mention the invasive nature of the lesion. This was not classic PTC but a Warthin-like variant. The pathologist was a research dermatopathologist, and was doing her surgical pathology on call day.
Discussion: This case was a misdiagnosis of not only the cancer but the type of cancer and the severity of the cancer with invasion and a BRAF marker. The presence of BRAF marker and suspicious cytology played a part in the decision to get a second opinion. If the original diagnosis was not challenged and the patient was considered to not have cancer, she would have surely been seen years later with a larger and more invasive BRAF positive Warthins-like PTC.
Conclusion: The endocrinologist has a responsibility to the patient with a nodule to make sure the needle biopsy result is correct. Also to review all surgical pathology reports when the patient goes to surgery.This is the way an endocrinologist can exert influence in correcting a misdiagnosis.

Abstract #1307

LONG TERM FOLLOW UP OF PATIENTS WITH TYPE 1 DIABETES ON LIRAGLUTIDE AND THE EFFECT OF LIRAGLUTIDE AS ADDITIONAL TREATMENT IN OBESE PATIENTS WITH TYPE 1 DIABETES

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Results: We have recently shown that the addition of Liraglutide to insulin in the treatment of well controlled, non-obese patients with type 1 diabetes leads to a significant further rapid reduction in glycemia, glycemic excursions, HbA1c, insulin requirements and body weight within days. We now present data of 8 patients with type 1 diabetes (mean age: 43±8 yrs; mean duration of diabetes: 29±11 yrs) on Liraglutide treatment (mean dose: 1.72±0.67mg) for 1.24± 0.21 yrs. Percent time spent in hyperglycemia calculated by using 3 glycemic thresholds (>150,200 and 250 mg/dl) fell by 45.35%($p = 0.01$), 45.19%($p = 0.03$) & 61.92%($p = 0.001$) respectively. There was a reduction in the bolus, basal and total daily doses of insulin by 26%($p = 0.009$), 19%($p = 0.01$) and 20%($p = 0.008$) respectively. The body weight fell from 85.3± 12.6 to 78.7±15.9 Kg ($p = 0.01$) and the BMI from 30.3 ± 3.9 to 27.8±4.9Kg/m2 ($p = 0.01$). Fasting C-peptide concentrations were non-

detectable at the outset and at the end of the study. We have now also investigated the effect of the addition of 1.8 mg of Liraglutide in 15 obese patients with type 1 diabetes (10 females, 5 males, 14 Caucasian, 1 African American; mean age: 47 ±13.81 yrs; mean duration of diabetes: 20.06 ± 10.18 yrs) who were not well controlled (mean HbA1c: 7.8±0.82). Over a period of 6 months,HbA1c fell to 7.39± 0.77%(p = 0.05); the bolus dose of insulin decreased significantly from 35.92 ± 19.69 to 29.52 ± 17.10 units (p = 0.03) while the basal dose did not change; the body weight fell from 100.63 ± 18.31 Kg to 95.96±19.22 kg (p =0.008) and the BMI from 34.06±7.36 to 32.33±6.85 Kg/m2 (p = 0.01). The systolic BP decreased from 137.53± 18.86 to 121.86±13.53(p = 0.003).

Discussion: Large, prospectively randomized studies are required to establish the use of Liraglutide in type 1 diabetes. Mechanistic studies to elucidate the mode of action of Liraglutide in these patients are also required since they do not have any beta-cell reserve.

Conclusion: We conclude that Liraglutide treatment in patients with type 1 diabetes has a rapid, sustained and durable effect on glycemia, body weight, insulin dose and systolic blood pressure. In addition, poorly controlled, obese patients with type 1 diabetes also benefit from this treatment.

Abstract #1308

CONTINUOUS GLUCOSE MONITORING SENSOR DATA IN A PATIENT WITH TYPE 2 DIABETES DEMONSTRATE THAT RAPID RELEASE FORM OF BROMOCRIPTINE THERAPY IMPROVES GLYCEMIC VARIABILITY AND POST-PRANDIAL HYPERGLYCEMIA

Ayse Bag Ozbek, MD, Michael Shanik, MD

Objective: To show glycemic benefits of bromocriptine mesylate via continuous glucose monitoring sensor (CGMS).

Case Presentation: A 68 year old woman type 2 diabetes on basal-bolus insulin therapy was evaluated with CGMS. Bromocriptine mesylate was added (0.8 mg daily, titrated to 3.2 mg daily over 4 weeks) while insulin dosing remained the same. After 2 months of therapy CGMS was utilized again. The data showed significant improvement of glycemic variability and post-prandial hyperglycemia.

Discussion: Administration of bromocriptine mesylate either as monotherapy or as an adjunct to metformin, sulfonylurea, or insulin reduces HbA1c levels relative to placebo by 0.55-1.2 %. It is unclear as to the exact mechanism of action of bromocriptine mesylate. Animal studies indicate that reduced hypothalamic dopaminergic tone is associated with the insulin resistant state, and appropriately timed central nervous system delivery of bromocriptine reduces insulin resistance and glucose intolerance. These data show improved glycemic variability and post-prandial hyperglycemia. To the best of our knowledge, this is the first reported case of the glycemic benefits of bromocriptine mesylate via CGMS.

Conclusion: Patients with T2D may have better glycemic control with bromocriptine mesylate.

Abstract #1309

A PATIENT WHO HAD TYPE 2 DIABETES BUT WANTED TO LIVE FOREVER ANYWAY

Scott Ahl, D.O., Lois Jovanovic, MD

Case Presentation: A chart review was conducted on a 92 year old who was diagnosed with type 2 diabetes mellitus around the age of 60. This is an accomplished man whose ambition in life parallels his desire to manage his medical problems. Upon being referred to a diabetes specialist at age 79 after having a stroke, his glycosylated hemoglobin (A1C) was 6.8% (Point of Care DCA 2000, normal <5.7%) on first encounter. Despite his initial A1C value, his diabetes control had been described as "good" in his records prior to initial consultation. His past medical history at this time also included sensory neuropathy, proteinuria and complete blindness in one eye. Through diligent efforts to control glucose with diet, exercise, weight control and medication, his A1C levels have averaged 5.5% over the 14 years since having an endocrinologist. The main oral medicine used was glimepiride, a drug with the propensity to cause hypoglycemia. However, he managed to control his glucose stringently without having any episode of hypoglycemia. His kidney disease and neuropathy have remained stable. He has not had any recurrent stroke. The patient's primary care doctor does not think that the patient has diabetes and has recommended stopping treatment at times. When treatment for diabetes was scaled back, the patient's A1C would subsequently increase indeed. In addition to good fortune, our patient continues on towards his goal of living forever because of his meticulous control of diabetes. Perhaps his stroke in 1998 may have been prevented had his diabetes been better controlled since his age of 60.

Discussion: Prior studies have shown that the benefits of tight glucose control in elderly persons with type 2 diabetes mellitus does not have the same benefits as in the younger age cohorts in terms of preventing long-term complications of diabetes due to microvascular and macrovascular disease. The risk of hypoglycemia is paramount and trumps the benefits of tight glycemic control. This case report lends credence to the contrary and serves as a platform for a discussion of the lifelong

treatment of type 2 diabetes, regardless of age, and its resultant benefits.
Conclusion: Treating diabetes holistically with exercise, diet, weight control and medication should continue into old age. The risks of hyperglycemia are lifelong. With a dynamic physician-patient relationship and frequent monitoring by the physician, hypoglycemia can be anticipated and prevented, while achieving excellent glycemic control.

Abstract #1310

LOWER RATES IN OVERALL AND NOCTURNAL HYPOGLYCEMIA OF INSULIN DEGLUDEC VS. INSULIN GLARGINE WITH TREATMENT INTENSIFICATION IN TYPE 2 DIABETES PATIENTS IN MODERATELY GOOD CONTROL (A1C 7.5-8.5%) AT BASELINE: A META-ANALYSIS

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Søren Rasmussen, Nathan Lassota, Stephen Gough

Objective: Current basal insulin treatments increase the risk of hypoglycemia when A1c levels approach normoglycemia. Insulin degludec (IDeg) is a new basal insulin that forms soluble multi-hexamers upon subcutaneous injection, resulting in an ultra-long action profile with a low day-to-day variability.
Methods: We analyzed whether these characteristics of IDeg would improve glycemic control and result in lower rates of hypoglycemia compared to insulin glargine (IGlar) in patients with type 2 diabetes (T2D) in moderately good control at baseline (A1c of 7.5-8.5%). Changes in A1c and fasting plasma glucose (FPG) were analyzed with linear models, and rates of hypoglycemia with a negative binomial regression model. Hypoglycemia was defined as self-reported confirmed hypoglycemia (PG <56 mg/dL; 3.1 mmol/L) or severe episodes requiring assistance; nocturnal confirmed hypoglycemia was defined if onset occurred between 00:01 and 05:59 inclusive. Analysis included patient-level data from all five open-label randomized treat-to-target phase 3a trials in T2D (26 or 52 weeks) in which IDeg (n=930) or IGlar (n=446) was given once daily.
Results: A1c decreased from 8.0% at baseline in both groups to 7.0% versus 6.9% at end of trial for IDeg versus IGlar, respectively (treatment difference: 0.08%-points [95% CI: -0.01; 0.18]; p=0.08). Observed FPG decreased from 162 to 109 mg/dL for IDeg and from 161 to 113 mg/dL for IGlar, achieving a statistically significant estimated treatment difference of -6.4 mg/dL [95% CI: -10.8; -2.0]; p<0.01. The rate ratios (IDeg:IGlar) of confirmed hypoglycemia (0.80 [95% CI: 0.67; 0.96]; p=0.02) and nocturnal hypoglycemia (0.69 [95% CI: 0.51; 0.92]; p=0.01) were both statistically significantly lower for IDeg versus IGlar.
Discussion: Patients with T2D with baseline A1c of 7.5-8.5% treated with IDeg showed comparable improvement in A1c, significantly greater reduction in FPG, and significant reduction (20%, 31%) in rates of overall and nocturnal hypoglycemia, respectively, compared to IGlar.
Conclusion: Treatment intensification in T2D patients in moderately good control is achieved with lower rates of both overall and nocturnal hypoglycemia with insulin degludec compared to insulin glargine

Abstract #1311

SHEEHAN'S SYNDROME PRESENTING AS SEVERE HYPONATREMIA - 25 YEARS AFTER INCITING EVENT

Swapna Bemalgi, MD, Yugandhar Manda,
Raghava Reddy Levakaveera, Laura Fitzpatrick

Objective: To report a rare cause of hyponatremia and late presentation of Partial Sheehan's Syndrome 25yrs after the inciting event
Case Presentation: HPI: 45 y/o Brazilian female presented to the ER with c/o nausea, epigastric pain, lethargy, confusion for 3 days. ROS was negative except for chronic fatigue. PMH: anemia and hypotension. Social h/o: Migrated to USA 5 yrs ago. No smoking, alcohol intake, illicit drug or medication use. She was married, had 3 kids. PE: Vital signs: BP: 70/50 mm hg, PR: 64/min, RR: 18/min,O2 sat: 98% on RA. Exam was unremarkable except confusion, lethargy and epigastric tenderness. Labs: BMP: Glu 83, BUN 8, Cr 0.59, Na 108, K 4, Cl 84, CO2 17. LFT's, CBC, lipase- normal. Serum osm: 238 (mosm/kg), Urine osm: 534, Urine lytes: Na 197, K 23, Cl 171. CXR,US abdomen- normal. AM Cortisol- 4.8mcg/dl. Cosyntropin Stim test- Cortisol levels of 4.8 at baseline, 13.9 at 30 min, 15.9 at 60 min. Additional history at this time revealed h/o Post Partum Hemorrhage during her last delivery 25 yrs ago in Brazil requiring blood transfusions. She was unable to lactate after that and had oligomenorrhea. Further labs: TSH: 3nl (0.5 -5.5), Free T4: 0.4 (0.7-1.9ng/dl), Prolactin: 3.8 ng/ml (5.2 -26.5), FSH: 7.8(MIU/ml), LH: 4.9 (MIU/ml), ACTH: <5 pg/ml (6-50). MRI Brain: Empty sella. These findings were s/o partial Sheehan's Syndrome and patient was started on stress dose steroids. Her BP and hyponatremia improved after that. Steroids were weaned to 20mg (am) -10 mg (pm) of hydrocortisone and patient was started on 75mcg of Levothyroxine.
Discussion: This is a case of late presentation of

Sheehan's Syndrome 25yrs after the inciting event. Pituitary enlarges during pregnancy and is hence sensitive to decreased blood flow caused by massive hemorrhage and hypovolemia which leads to postpartum necrosis. The degree of hypopituitarism may vary from panhypopituitarism to selective hormone deficiencies. Sometimes diagnosis is not made until years later when they present with secondary hypothyroidism or adrenal crisis. Hypothyroidism and glucocorticoid deficiency cause decreased free water clearance leading to hyponatremia. Hypopituitarism can itself stimulate vasopressin secretion causing hyponatremia.

Conclusion: In retrospect our patient presented with symptoms of severe hyponatremia and an important clue to the diagnosis was the h/o PPH along with symptoms of inability to lactate and oligomenorrhea since her last delivery. Workup revealed low cortisol, partial adrenal insufficiency, low ACTH, low PRL, low Fr T4 which goes in favor of partial hypopituitarism. Her TSH although normal, was inappropriately low for the level of free T4. The diagnosis was supported by MRI showing empty sella.

Abstract #1312

HASHIMOTO'S HEART DISEASE: A RARE CASE OF PRIMARY HYPOTHYROIDISM PRESENTING AS MASSIVE PERICARDIAL EFFUSION

Pankaj Sharda, MBBS, MD, Mirela Feurdean

Objective: The incidence of massive pericardial effusion in primary hypothyroidism is only about 3%, an extremely infrequent occurrence which appears to be dependent on the severity of the disease. This is the case of a young Hispanic woman who presented with symptomatic, massive pericardial effusion secondary to Hashimoto's disease. The pathophysiology and treatment approach to management of hypothyroidism induced pericardial effusion has been reviewed.

Case Presentation: A 24-year old obese woman presented with worsening shortness of breath and chest pressure for one month. She also had easy fatigability, orthopnea , multiple near-syncope events. Other complaints were constipation, cold intolerance, hair loss and menorrhagia. She denied family thyroid disorders and took no medications. Vitals were normal except for BP 90/57. There was no pulsus paradoxus or JVD. Thyroid was not palpable. Distant heart sounds, decreased reflexes and alopecia were appreciated. Her significant labs were hemoglobin 7.4, TSH 352 and FT4 <0.20, TPO Ab 6101, thyroglobulin Ab 3128. EKG showed normal sinus rhythm with low voltage QRS. Echocardiography confirmed massive pericardial effusion with normal systolic function and no cardiac tamponade. Levothyroxine was started and she underwent pericardiocentesis with 450ml of exudative fluid drainage and pericardial window. She was discharged home on levothyroxine and iron supplements.

Discussion: Pericardial effusion is seldom the initial presentation of hypothyroidism, with no correlation between the severity of effusion and TSH levels. Although not fully understood, the proposed pathogenesis is generalized exudative polyserousopathy due to increased permeability of capillaries to protein and increased proportion and quantity of exchangeable albumin localized to the extra vascular space. Accumulation of hygroscopic mucopolysaccharides and decreased lymph flow are other demonstrated mechanisms. The effusion rarely causes tamponade because it accumulates slowly and allows time for the pericardial sac to distend. Controversy exists regarding the timing and drainage of massive pericardial effusion. Pericardiocentesis or immediate pericardial window are common choices favored in hemodynamically compromised cases. Thyroid replacement therapy prevents recurrence and promotes absorption of effusion.

Conclusion: Massive pericardial effusion is a very rare initial presentation of hypothyroidism, and especially so in young age. The treatment is simple and gratifying, with very rare recurrence rates upon adequate thyroid replacement. The pathophysiology and treatment modalities reviewed here enhance the practitioner's understanding of thyroid disease management.

Abstract #1313

IMPACT ON DIABETES BEHAVIORS: KNOWING VERSUS GUESSING BLOOD GLUCOSE VALUES

Jeremy Pettus, MD, Jimm Greer, Patricia Stenger, Holly Schachner, MD, Nancy Dunne, Joan Lee Parkes, Scott Pardo, Steven Edelman, MD

Objective: Research suggests that some people with diabetes use perceptions of their blood glucose (BG) levels rather than BG testing to make decisions about their diabetes management. This study was conducted to assess the difference between self-reported, estimated BG values and BG values as measured on a BG meter. Another objective of the study was to obtain information on the perceptions that people with diabetes have about BG testing and the impact of knowing their BG value on their diabetes management.

Methods: Subjects aged ≥18 years with type 2 diabetes (N = 297) attending 1 of 2 Taking Control of Your Diabetes (TCOYD; 501c3) conferences were asked to take a pre-fingerstick questionnaire about their diabetes management. Subjects were then asked, "What do you think your blood sugar level is now?" Study staff then performed a

fingerstick to measure the subject's BG value on a BG meter. Subjects were advised of their BG value and were asked to respond to additional statements related to their BG testing practices on a post-fingerstick questionnaire.

Results: On the pre-fingerstick questionnaire, the majority of subjects either strongly agreed, agreed, or neither agreed/disagreed with the statement, "My body tells me without testing if my blood sugar is low or high" (77%) and made decisions about their diabetes, such as insulin dosing, without testing on a BG meter (71%). However, nearly half (46%) of subjects estimated BG values that were outside current ISO accuracy guidelines (ie, more than ±15 mg/dL or ±20% of meter glucose values <75 and ≥75 mg/dL, respectively); 58% estimated BG values that were outside proposed more stringent accuracy guidelines (ie, more than ±15 mg/dL or ±15% of meter glucose values <100 and ≥100 mg/dL, respectively). On the post-fingerstick questionnaire, nearly all subjects reported that knowing their blood sugar level by checking could help them make different diabetes decisions (99%), give them more confidence in their ability to manage their diabetes (98%), help them prevent low BG (98%), help them recognize and treat low BG (98%), and give them a better understanding of how food affects their BG level (98%). In addition, 99% of subjects responded that they would make different decisions about their meals/snacks if they knew their BG by checking on a BG meter. Among subjects taking insulin (n = 86), 98% felt that checking their BG on a meter could give them more confidence in adjusting their daily insulin dose.

Conclusion: These findings suggest that regular self-monitoring of BG versus guessing can contribute a significant impact on diabetes management behaviors in people with diabetes.

Abstract #1314

INDIVIDUALIZATION THROUGH STANDARDIZATION -- ELECTRONIC ADMINISTRATION INSTRUCTIONS FOR SUBCUTANEOUS INSULIN ORDERS IN THE HOSPITAL

Susan Braithwaite, MD, FACP, Mary Kennihan, Tatheer Zohra, Radha Devi, MD, Chitra Srinivasan, Josefina Diaz, Bradley Howard

Objective: Variances between individual hospitalized patients, differences of expression between providers, and complexity itself may lead to misunderstanding of written orders for subcutaneous insulin, jeopardizing patient safety. The objective was to design electronic order sets appropriate to patient carbohydrate exposure, offering standardized choices that would promote safe, effective, and individualized insulin order entry.

Methods: Saint Francis Hospital, a community teaching hospital, was selected as the pilot site for 6 hospitals in the Resurrection Health Care System to introduce use of an electronic medical record. Three subcutaneous insulin order sets were designed, designated in short: "patients eating," "patients not eating," and "patients receiving overnight enteral feedings."

Results: Under each order set, the user chooses orders for glucose monitoring and insulin from menus appropriate to the pattern of carbohydrate exposure associated with the order set. Each insulin order specifies the insulin name, route (subcutaneous), priority and frequency. The user enters insulin doses, start time, and duration. Programmed menus of administration instructions expressed in standardized language are associated with each specific scheduled insulin order. During 4 weeks of availability in the 1st calendar month after initiation, there were 103 orders given during 19 admissions under the order set for "patients eating" and 46 orders given during 13 admissions under the order set for "patients not eating. The combination of glargine, scheduled lispro, and correction dose lispro under the order set for "patients eating" was ordered on 6 admissions. The combination of NPH and scheduled regular insulin with correction dose regular insulin under the order set for "patients not eating" was ordered on 3 admissions.

Discussion: To implement published guidelines on writing subcutaneous insulin orders, a mechanism is required to convey administration instructions to pharmacy and nursing staff. Feasibility of use has been shown for order sets differentiated according to patient carbohydrate exposure that offer menus of standardized insulin administration instructions. In the first month of use, not all but some entries made under the order sets employed the specific combinations of insulin for which the order sets were designed. Preliminary data will assist in the planning of in-servicing and future studies.

Conclusion: The long-range goal is that with utilization, under each order set, subcutaneous insulin administration instructions will become familiar to pharmacy and nursing staff and will facilitate the individualization of care, attainment of glycemic targets, and protection of patient safety.

Abstract #1315

CHANGES IN BODY COMPOSITION FOLLOWING GASTRIC BYPASS OR GASTRIC BANDING

Helmuth Billy, Ted Okerson, MD, FACP

Objective: There are limited data available as to the effects of rapid versus more gradual weight loss after bariatric

surgery. The aims of this study are to assess the body mass index (BMI) and percent excess weight loss after Roux-en-Y gastric bypass (RYGB) or laparoscopic adjustable gastric banding (LAGB) and to determine the effect of the rate of weight change on body composition.

Methods: Patients were selected from Ventura Advanced Surgical Associates database if they had either RYGB or LAGB between January, 01, 2007 and December 31, 2009. Lean body mass (LBM), dry lean mass (DLM), percent body fat, basal metabolic rate (BMR), and BMI were recorded at baseline; follow-up assessments were completed 3, 6, 9, and 12 months post surgery.

Results: Baseline measures were available for 480 patients: 188 in the RYGB and 292 in the LAGB groups. At baseline, BMI and % body fat were 46.7 and 52.5% respectively for RYGB, and 42.5 and 49.9% respectively for LAGB. There was no difference in LBM, %LBM or BMR. One year after surgery, patients who had undergone RYGB had a greater reduction in BMI (-17.10 vs -7.69 kg/m2) and excess weight loss (78.35% vs 48.04%) compared with the LAGB group. The rate of BMI decline for both RYGB and LAGB patients was greatest during the first 3 months post surgery compared with months 3 to 12 (RYGB: 2.65 vs 1.37 kg/m2; LAGB: 1.02 vs 0.40 kg/m2). Lean Body Mass loss was larger for RYGB patients than LAGB patients (19.62 vs 12.41 lb after 1 year), with the greatest reduction in LBM occurring during the first 3 months after surgery (10.15% vs 4.95% of baseline LBM). LBM loss was tightly correlated with change in BMR; at 1 year post surgery, BMR declined by a mean of 200.21 kcal/d and 108.53 kcal/d in the RYGB and LAGB patients, respectively.

Discussion: Although both types of bariatric surgery led to a loss of both fat and LBM, RYGB patients lost 52% more LBM during the first 3 months, and 37% more in the first year. This may prove clinically meaningful in patients' ability to maintain weight loss over time. It is not known whether regular strength training and aerobic exercise could mitigate some of this LBM loss, given the degree of caloric restriction in RYGB patients, particularly in the first 3 months.

Conclusion: RYGB patients experienced a faster and greater overall weight loss compared with LAGB patients, but at the expense of greater loss in LBM and BMR. The more gradual weight loss attained through LAGB maintained more muscle mass and preserved a greater degree of BMR. This may improve the ability to maintain weight loss over the long-term; further follow-up and study is planned.

Abstract #1316

ULTRA-LONG PHARMACOKINETIC PROPERTIES OF INSULIN DEGLUDEC IN YOUNGER ADULTS ARE PRESERVED IN GERIATRIC SUBJECTS WITH TYPE 1 DIABETES

Stefan Korsatko, Sigrid Deller, Julia Mader, Katharina Glettler, Gerd Köhler, Gerlies Bock, Martina Urschitz, Michael Wolf, Hanne Hastrup-Nielsen, Flemming Søndergaard, Hanne Haahr, Thomas Pieber

Objective: Insulin degludec (IDeg) is an ultra-long-acting basal insulin that forms soluble multi-hexamers upon subcutaneous injection resulting in a depot from which IDeg is continuously and slowly absorbed into the circulation leading to a flat and stable glucose-lowering effect. This study investigated the pharmacokinetic and pharmacodynamic properties of IDeg in geriatric (≥65 years) versus younger adult (18-35 years) subjects with type 1 diabetes (C-peptide <0.3 nmol/L).

Methods: This was a randomized, double-blind, two-period crossover, multiple-dose study with 6 days of once-daily administration of 0.4 U/kg IDeg or 0.4 U/kg insulin glargine. Data are shown for IDeg only. Fourteen geriatric (mean age: 67.8 years; baseline HbA1c: 7.7%; body mass index [BMI]: 26.2 kg/m2) and 13 younger adult (mean age: 27.1 years; baseline HbA1c: 7.8%; BMI: 24.4 kg/m2) subjects participated.

Results: The mean IDeg concentration-time profile at steady state was similar in geriatric and younger adult subjects. There was no statistically significant difference between age groups in total exposure (area under the curve [AUCIDeg,τ,SS]; least square mean 85 673 pmol*h/L in geriatric and 82 727 pmol*h/L in younger adults) or maximum concentration (*C*max,IDeg,SS) of IDeg, mean ratio geriatric/younger adults [95% CI] AUCIDeg,τ,SS: 1.04 [0.73; 1.47], *C*max,IDeg,SS: 1.02 [0.74; 1.39]. The ultra-long properties of IDeg were preserved in geriatric subjects; the estimated harmonic mean terminal half-life was 25 hours. There was no statistically significant difference in total glucose-lowering effect of IDeg (AUCGIR,τ,SS) between geriatric (least square mean 1923 mg/kg) and younger adults (least square mean 2457 mg/kg), mean ratio geriatric/younger adults [95% CI] 0.78 [0.47; 1.31].

Discussion: The pharmacokinetic properties of IDeg in younger adults were seen also in geriatric subjects. Total exposure was similar and there were no differences in the glucose-lowering effect of IDeg between geriatric and younger adult subjects.

Conclusion: The ultra-long pharmacokinetic properties of IDeg observed in younger adults were preserved in geriatric subjects with type 1 diabetes.

Abstract #1317

AMYLOID GOITER: REPORT OF THREE CASES

Jose Paz-Ibarra, MD

Objective: In some cases of primary or secondary amyloidosis has been reported the involvement of the thyroid gland. We report three cases of thyroid amyloidosis.
Methods: We report the clinical and paraclinical characteristics of patients.
Case Presentation: Case 1: Male, 28 years, history of pulmonary tuberculosis (1995), bronchiectasis and ESRD by renal amyloidosis 9 years of evolution, giant goiter presented with 3 years of evolution, associated with dyspnea, dysphagia and dysphonia. FNAB: adenomatous goiter, TSH: 1.2uUI/mL, AntiTPO (-), albumin: 4.95g / L, globulin: 4.64g / L, Hb: 8.5g% underwent total thyroidectomy with AP: Thyroid amyloidosis, Congo red (+). Case 2: Female, 43 years, history of diffuse pulmonary interstitial disease (1999), bronchiectasis, aspergillosis and respiratory failure, was admitted with nephrotic syndrome for renal amyloidosis, in addition to neck pain, dyspnea, and dysphonia; giant multinodular goiter was found. FNAB: Amyloid goiter in the cell block, Congo red (+), TSH: 0.64uUI/mL, AntiTPO (-), albumin: 3.1g / L, globulin: 3.8 g / L, Hb: 10.2 g%, not performed thyroidectomy for high surgical risk. Case 3: Female, 65 years, history of RA (1992), Sjögren's syndrome, ESRD by nefroangiesclerosis and polycystic kidney disease, had multinodular and giant goiter of 6 years of evolution, also tightness and dysphagia. FNAB: Amyloid goiter in the cell block: TSH: 2.16uUI/mL, AntiTPO (+), albumin: 3.6g / L, globulin: 3.5 g / L, Hb: 10.5 g%, subjected to total thyroidectomy with AP: Amyloidosis thyroid , Congo red (+).
Discussion: Secondary amyloidosis usually neoplastic in origin or secondary to chronic inflammatory diseases.
Conclusion: The development of goitre giant compressive symptoms is the most common presentation and represents amyloid infiltration of the gland.

Abstract #1318

PAPILLARY THYROID CANCER IN A THYROGLOSSAL DUCT CYST

Jose Paz-Ibarra, MD

Objective: To present a case of papillary thyroid cancer in a thyroglossal duct cyst.
Methods: We describe the clinical and paraclinical characteristics of the patient.
Case Presentation: Women, 47 yeras, 3 years ago underwent left hemithyroidectomy nodule 3 cm tumor compatible with follicular cytology, whose pathology was: colloid goiter. Go to endocrinology for thyroid evaluation. Cervical tumor was detected in 2.5-cm midline of increased consistency and mobile with swallowing. TSH: 4.01uUI/mL, FT4: 1.26ng/dL, thyroid ultrasonography: RTL 33x10x9mm without focal lesions; 2mm isthmus, LTL absent. In the midline suprahyoid region was observed 21x14x19 mm solid nodule, the Doppler central and peripheral vascularity presented. FNAB: papillary cancer. Spiral CT: oval mass lesion precartilaginosa midline 18 mm. She underwent Sistrunk operation, nodule found inside which showed cystic cavity occupied by brown tumor whose analysis was: 2.5x2 cm papillary cancer, classic variety (20%) and follicular (80%) with infiltration of the wall and stroke lymphovascular. At present the patient expects to complete thyroidectomy, TSH presents: 5.92uUI/mL, FT4: 0.95ng/dL; Tg: 27.5ng/mL; ABTG / AbTPO (-), has initiated LT4 suppressive therapy.
Discussion: The development of differentiated thyroid carcinoma in a thyroglossal duct cyst is rare, less than 1%, about 200 cases have been reported, the most frequent papillary.
Conclusion: In cases of differentiated thyroid carcinoma in a thyroglossal duct cyst treatment should be individualized and selective complete surgical resection of the cyst together with the hyoid bone is curative in almost all cases, with some specific situations where it is recommended total thyroidectomy and radioiodine to decrease recurrence and metastasis.

Abstract #1319

COEXISTING MEDULLARY THYROID CANCER WITH GRAVES' DISEASE

Jose Paz-Ibarra, MD

Objective: To present a case of coexistence of medullary thyroid cancer and Graves' disease.
Methods: We describe the case from the standpoint of clinical and paraclinical.
Case Presentation: Male, 54 years, a native of Huancavelica, from Lima. History of Gilbert's syndrome treated with phenobarbital, allergic to salicylate. Admitted with a time of disease of 10 months characterized by low weight, distal tremor, palpitations, hiperdefecación, diarrhea and decreased muscle strength predominantly proximal lower limbs. On examination: Weight: 65 kg, BMI: 21.9 kg/m2, PA: 100/70 mmHg, HR: 100 x '. Diffuse goiter with nodule 1.5 cm in the lower pole of RTL; Eyes: ACS = 0 points, eyelid retraction. Analysis: TSH: 0.054 uUI / mL, FT4: 5.1 ng / dL; Uptake of Iodine: 29% (2 hours) and 45% (24 hours),

scintigraphy with Tc99m: diffuse goiter. Ultrasonography: hypervascularized diffuse goiter, hypoechoic nodule in RTL presents of 15x12 mm with microcalcifications. FNAB: positive for malignant tumor cells, consistent with medullary thyroid cancer; Calcitonin 594.45pgml (VN 0 to 50). After the pre-surgical preparation with triple therapy and after ruling out possibility of MEN2 underwent total thyroidectomy with neck dissection central pathological study reported: intraglandular medullary thyroid cancer of 1.1 cm solid, whitish, not encapsulated in RTL; nodes (-). Rest of the parenchyma with signs of hyperfunction and lymphocytic infiltrate. Subsequent evaluation to date reveals no recurrence of medullary thyroid cancer, taking levothyroxine 150 ug / day; TSH: 3.2uUI/mL, Tc <2.0, TBS with MIBG (-).
Discussion: Between 2 and 10% of patients with Graves' disease has an associated cancer. As the papillary the most common. Rare is the coexistence of medullary thyroid cancer and Graves' disease.
Conclusion: The cases reported in the literature of medullary thyroid cancer associated with Graves' disease are rare, reaching less than fifty worldwide.

Abstract #1320

COEXISTENCE OF MEDULLARY THYROID CANCER WITH HASHIMOTO'S THYROIDITIS IN A PATIENT

Jose Paz-Ibarra, MD

Objective: To report a case of coexistence of medullary thyroid cancer and Hashimoto's chronic thyroiditis.
Methods: We describe the case from the point of clinical and paraclinical.
Case Presentation: Female, 45 years old, born in Chiclayo from Lima, attended the service with a disease duration of five months for cervical volume slightly increased and ultrasound finding of thyroid nodule 6 mm in right thyroid lobe (RTL). BMI: 24.6 kg/m2; goiter 1b, no lymphadenopathy. Analysis: TSH: 9.84uUI/mL, FT4: 0.96ng/dL; AbTPO> 1000 IU / mL; ABTG: 277 UI / mL. Ultrasonography: RTL of 50x19x18mm, heterogeneous parenchyma with nodule isoechoic of 6.9x5mm with defined borders, hypoechoic halo; isthmus 4 mm, left thyroid lobe 41x13x18mm. Echo-guided FNAB: positive for malignant tumor cells, consistent with medullary thyroid cancer; Calcitonin 245pg/mL (VN: 0-18.3), CEA: 3.2ng/mL (VN: 0-3.0). When disposing MEN2 underwent total thyroidectomy with neck dissection central; pathological study reported: intraglandular medullary thyroid cancer of 0.7 cm unencapsulated in RTL; nodes (-). Rest of the tissue with Hashimoto's chronic thyroiditis. Subsequent evaluation to date reveals no recurrence of medullary thyroid cancer, taking levothyroxine 100 ug / day; TSH: 2.2uUI/mL, Tc <2.0, TBS MIBG (-).
Discussion: Hashimoto's thyroiditis is a chronic disease that affects 5% of the population. In cases of goiter or differentiated carcinoma surgical resection is recommended. The coexistence of medullary thyroid cancer and Hashimoto's chronic thyroiditis is uncommon as evidenced in the literature also is under debate whether the lymphocytic infiltration may predispose to the beginning of medullary thyroid cancer or whether this would be a defense mechanism against tumor.
Conclusion: It is rare coexistence of medullary thyroid cancer and autoimmune thyroid disease.

Abstract #1321

PITUITARY TUMOR IN A PATIENT WITH AUTOIMMUNE HYPOTHYROIDISM

Jose Paz-Ibarra, MD

Objective: Report a case of thyrotroph hyperplasia in a patient with autoimmune.
Methods: We describe the case from the standpoint of clinical and paraclinical.
Case Presentation: Female, 43 years old, born in Lima, G1P1, 8 year old daughter, catamenial regime: 4/30 days, menorrhagia. History of head trauma in October 2010, no family history of thyroid diseases. In December 2010, his neurosurgeon underwent MRI for persistent headache, showing expansive process of intra-and suprasellar of 15x10mm. In January 2011, went to preoperative endocrinology, being manifestations of myxedema of approximately 1 year earlier. FC: 60x '; Weight: 52kg. BMI: 23.1Kg/m2; goiter 1b irregular. CBC: WBC 5590, Hb: 7.8g%, MCV: 83, G: 66mg%, Cr: 0.83mg%, Albumin: 4.66g / L; Globulins: 3.28g / L, SGOT / SGPT: 103/68U/L ; LDH: 1235U / L, CK: 1510 U / L; Cholesterol: 342mg%, Triglycerides: 84mg%, Ca: 8.82mg% Na: 138mEq / L. TSH: 1381uUI/mL, FT4 <0.3ng/dL, AbTPO: 128.5UI/mL, ABTG> 3000UI/mL; PRL: 68.5ng/mL; FSH: 4.3mUI/mL; E2: 88pg/mL, GH: 0.68 ng / mL; IGF1 <25ng/mL. ACTH: 16.1pg/mL, Cortisol: 4.64ug/dL. Visual field tests: normal. Started replacement therapy with prednisone 10 mg / d for 5 days levothyroxine 200 ug / d, then 150 ug / d to achieve a maintenance dose of 100 ug / d. Ten weeks after MRI: Normal pituitary, TSH: 0.05uUI/mL, FT4: 1.4ng/dL, FT3: 3.07pg/mL; PRL 30.6ng/mL.
Discussion: When evidence of a pituitary expansive process in the context of primary hypothyroidism should consider the existence of thyrotroph hyperplasia that may resolve with replacement therapy without surgery.
Conclusion: There have been reports of thyrotroph hyperplasia in patients with untreated chronic primary hypothyroidism.

Abstract #1322

LINGUAL THYROID IN A WOMAN OF 47 YEARS OLD

Jose Paz-Ibarra, MD

Objective: To present a case of symptomatic lingual thyroid.
Methods: We describe the case from the standpoint of clinical and paraclinical.
Case Presentation: Female, 47 years old, born in Ica, clerk, single, a history of nephrolithiasis and cervical cancer with conization successfull. Families with uterine cancer, type 2 diabetes and goiter. Go to endocrinology with a history of 1 year of mild dysphagia and cough associated with finding a tumor in the base of tongue. Physical examination evidence a pink tumor in base of the tongue, no bleeding or ulceration of about 3x2cm and neck level of not palpable thyroid gland or lymphadenopathy. Cervical ultrasonography: lack of thyroid usual location, no regional lymphadenopathy. Cervical CT without contrast: blade tissue, lobulated contours on base of tongue with midline of 35x23x20mm (height, anteroposterior and transverse diameter). Scintigraphy with Tc99m: Uptake in base of tongue, absent at cervical level. TSH: 9.3uUI/mL, FT4: 0.8ng/dL; AbTPO / ABTG (-), Tg: 87ng/mL. Hb 12.9g% Sodium: 142mEq / L, CPK: 91U / L, cholesterol: 234mg/dl, Triglycerides: 231mg/dl. FNAB: lingual thyroid with features of colloid goiter. The patient refused surgery so radioiodine was noted at doses of 40 mCi. and the third day post therapy initiated levothyroxine 150 ug / day.
Discussion: Symptomatic lingual thyroid is a rare disorder, predominantly women, affected individuals usually have no other thyroid tissue and manifestations depend on the patient's age, size of the mass and thyroid function. For patients with symptomatic disease, treatment options include suppression, surgery and radioablation.
Conclusion: This case shows the importance of considering the presence of lingual thyroid in the differential diagnosis of mass base of the tongue as a cause of dysphagia.

Abstract #1323

THYROTOXIC VOMITING

Jose Paz-Ibarra, MD

Objective: Report a case of thyrotoxic hyperemesis.
Methods: We describe the case from the point of clinical and paraclinical.
Case Presentation: Woman, 38 years old, from Iquitos, teacher; with a history of bronchitis. She was diagnosed with hyperthyroidism four years before admission received methimazole for 1 year, suspended in late pregnancy. Three months before admission resubmitted clinical picture of thyrotoxicosis, was hospitalized in Iquitos for epigastric pain and nausea and referred to our hospital with vomiting and oral intolerance. She showed tachycardia, goiter 2.5 N, slightly increased consistency. TSH <0.004uUI/mL, FT4> 6ug/dL, FT3: 19.2pg/ml; AntiTPO> 1000UI/mL; uptake of I-131: 62% (2 hrs) / 68% (24h), ultrasonography of neck: diffuse and hypervascularized goiter. FNAB: hyperfunctioning goiter with lymphocytic infiltrate. Amylase and lipase: normal, blood, urine, markers of viral hepatitis and agglutinations: negative. Abdominal ultrasound: normal. Upper endoscopy: antritis mild, H. pylori (-). We indicated her: NPO, propranolol 160 mg / d, parenteral antiemetics, after 10 days, she received radioiodine (20mCi) and the 3rd day started methimazole 45 mg / d with complete remission of abdominal pain, nausea and emesis after 2 weeks. At 3 months he developed hypothyroidism and currently receives 125 ug of levothyroxine without resubmitting emetic box.
Discussion: The gastrointestinal manifestations of hyperthyroidism are generally characterized by hyperphagia and hiperdefecación sometimes intractable hyperemesis is a rare symptom.
Conclusion: Persistent vomiting and epigastric pain may be symptoms of thyrotoxicosis, symptoms resolved rapidly and completely to treatment with beta blockers and thyroid.

Abstract #1324

PERFORMANCE VARIABILITY OF SOME COMMONLY USED SMBG SYSTEMS: CLINICAL CONSIDERATIONS FOR PATIENTS AND PROVIDERS

Ronald Brazg, MD, Leslie Klaff, MBBCh, PhD, Christopher Parkin

Objective: Blood glucose data are frequently used in clinical decision-making, thus, it is critical that self-monitoring of blood glucose (SMBG) systems consistently provide accurate results. Although several variables (e.g. user technique) can impact the accuracy of SMBG results, the inherent accuracy of the SMBG system, itself, is often not considered in clinical practice. It is assumed that because SMBG systems marketed in the US have met the international standard ISO 15197:2003 (ISO), they must be accurate. Concerns about SMBG accuracy have prompted the development of a newly proposed ISO standard, whereby the acceptance criteria for accuracy is that ≥95% of the individual glucose results shall fall within ±15 mg/dL of the results of the manufacturer's

reference procedure at glucose concentrations <100 mg/dL and within ±15% for values ≥100 mg/dL. We evaluated 7 marketed systems against the current and proposed ISO criteria.

Methods: Capillary blood samples were collected from 162 subjects by deep finger puncture and tested on 7 systems: ACCU-CHEK® Aviva Plus; AdvocateTM Redi-Code; ElementTM; EmbraceTM; Prodigy® Voice; TRUEbalanceTM; and WaveSense PrestoTM. Results from these systems were compared to the manufacturer's documented reference system, YSI or PCA-HK; 3 different strip lots from each system were tested on each subject, in duplicate.

Results: Only the ACCU-CHEK Aviva Plus met the proposed ISO criteria in all 3 lots. The other 6 systems failed to meet the criteria in at least 2 of the 3 lots, showing lot-to-lot variability, high/low bias and variations due to hematocrit. Compared against the current ISO standard (≥95% within ±15 mg/dL for values <75 mg/dL and ±20% for values ≥75 mg/dL) only the ACCU-CHEK Aviva Plus, Element, and WaveSense Presto systems met accuracy criteria. Whilst only 3 of 7 systems tested met the current ISO accuracy standard, only the ACCU-CHEK Aviva Plus met the newly proposed accuracy criteria.

Discussion: Because SMBG data are frequently used to make therapeutic decisions, inaccurate glucose readings can potentially adversely impact clinical outcomes in people with diabetes. SMBG accuracy is particularly important in the elderly (who are often more susceptible to hypoglycemia) and those with diabetic nephropathy (who are often anemic).

Conclusion: Although there are many factors that should be addressed through patient education and training, clinicians can reduce controllable variables, by prescribing accurate SMBG systems. The proposed ISO criteria should enhance patient safety by improving the accuracy of available SMBG systems.

Abstract #1325

NON-PREGNANCY GLP-1 LEVELS AMONG WOMEN WITH HISTORY OF GESTATIONAL DIABETES MAY PREDICT RISK OF TYPE 2 DIABETES

Joan Sullivan, BS, Angelina Trujillo, MD, Lois Jovanovic, MD

Objective: To determine whether the third trimester pregnancy and postpartum GLP-1 levels in women diagnosed with gestational diabetes mellitus (GDM) may be a marker of risk for developing Type 2 diabetes mellitus (T2DM) compared to GLP-1 levels among women with history of normal pregnancy (NL).

Methods: We studied 13 women with diet treated GDM (DGDM), 8 women with insulin treated GDM (IDGM), 3 pregnant T2DM women and 10 pregnant normal women. GLP-1 levels were measured fasting and at 15, 30, 60 and 120 minutes following the start of a mixed meal. Women were initially studied during the third trimester of pregnancy and followed up after delivery, when lactation had been discontinued.

Results: GLP-1 levels during pregnancy were reduced in all subjects compared to non-pregnancy levels. During pregnancy, DGDM and NL reached peak GLP-1 levels (DGDM 15 pM, NL 15 pM) at 30 minutes postprandial. In contrast, IGDM and T2DM had delayed GLP-1 responses and achieved peak GLP-1 levels at 60 minutes postprandial (IGDM 14 pM, T2DM 8 pM). At follow up visit, non-pregnant DGDM and NL reached peak GLP-1 levels (DGDM 17 pM, NL 20 pM) at 30 minutes postprandial while non-pregnant IGDM and T2DM again displayed delayed GLP-1 responses achieving peak levels at 60 minutes postprandial (IGDM 16 pM, T2DM 11 pM).

Discussion: GDM is a high risk for the development of T2DM and approximately 50% of women with a history of GDM will progress to T2DM within 5 years. Predicting which women are at greatest risk of developing T2DM has not been well defined. In published literature, it has been reported that the release of the incretin hormone GLP-1 is attenuated in T2DM. Our observation of a postprandial delay and attenuation of the GLP-1 responses in women with GDM identifies those women who will subsequently develop T2DM.

Conclusion: Our data among women with a history of GDM compared to women with normal pregnancy suggests that GDM women who require insulin treatment have a reduction of GLP-1 levels similar to T2DM. This attenuated response, compared to normal and diet treated GDM, persists post delivery and may be predictive of the risk for developing T2DM.

Abstract #1326

INITIAL COMBINATION OF LINAGLIPTIN AND METFORMIN IN PATIENTS WITH TYPE 2 DIABETES MELLITUS: EFFICACY AND SAFETY IN A 1-YEAR, RANDOMISED, DOUBLE-BLIND EXTENSION STUDY

Maximilian von Eynatten, MD, Thomas Haak, Thomas Meinicke, Russell Jones, Sonja-Anna Weber-Born, Hans-Juergen Woerle

Objective: To determine the long-term efficacy and safety of linagliptin in initial combination with low- or high-dose metformin in patients with type 2 diabetes mellitus (T2DM).

Methods: We report the 1-year randomized, double-blind

extension study (NCT00915772) of a previously reported 6-month randomized controlled trial (NCT00798161). In the 6-month study, adults with T2DM were randomised to 1 of 6 combination or monotherapy regimens (lina 2.5 mg + met 500 mg BID, lina 2.5 + met 1000 BID, met 1000 BID, met 500 BID, lina 5 QD, or placebo). All patients completing the 6-month trial without rescue therapy were eligible for the 1-year extension: patients in the first 3 treatment groups continued their regimen (nonswitched group, n=333) while patients in the met 500 BID, lina 5 QD, and placebo groups were randomised to 1 of the 3 continuing BID regimens (switched group, n=233). Descriptive statistics were used to summarize all safety and efficacy data.

Results: For the nonswitched patients, all 3 treatment groups maintained the HbA1c reductions achieved during the 6-month study, with the linagliptin + metformin combination therapy groups showing greater HbA1c decreases versus metformin alone. The mean (±SD) changes in HbA1c across the combined 1.5-year period were -1.63±1.05% in the lina 2.5 + met 1000 BID group, -1.32±1.06% in the lina 2.5 + met 500 BID group, and -1.25±0.91% in the met 1000 BID group. For switched patients, mean HbA1c changes in the 1-year extension were more marked in the lina 2.5 + met 1000 BID group (-0.96±1.05%) than with lina 2.5 + met 500 BID (-0.63±0.83%) and met 1000 BID (-0.42±0.76%). During the extension, use of rescue medication was lower for the lina 2.5 + met 1000 BID group (14.0%) than for lina 2.5 + met 500 BID (27.6%) and met 1000 BID (24.7%) groups. There were no clinically meaningful changes in body weight or waist circumference in any of the groups. Adverse event (AE) rates were similar between groups, with most AEs being mild or moderate, and considered unrelated to study drugs. The incidence of investigator-defined hypoglycaemia was 4.9%, 6.4% and 2.9% for the 3 regimens; respectively, with none of the events classified as severe.

Discussion: Initial combination of linagliptin and metformin was well tolerated over the long-term with low risk of hypoglycaemia, and improved glycemic control versus metformin alone.

Conclusion: The initial combination of linagliptin and metformin appears to provide a useful treatment option in patients whose blood glucose levels are increased to an extent that metformin monotherapy alone may not achieve treatment targets.

Abstract #1327

HOME MEDICAL MONITORING SYSTEM(HMMS): A BRIEF CASE FOR COMPUTER NAIVE PATIENTS AND PROVIDERS

R. A. Ramanujan, MD

Objective: Design a clinical tool for the direct integration the of patients home blood pressure(BP), pulse(P), and blood sugar(BS) all in one with direct connectivity to the Electronic Medical Record(EMR) by cloud computing.

Methods: This is a self-contained, compact wireless based, tablet size portable unit used to compute daily values or aggregates and integrate in real-time the patients medications and export them directly into the patient's office based EMR. Progress notes can be generated on a daily or weekly basis. Clinic based interaction, medication titration and diagnostic tests are interposed in real-time to maintain linearity in care.

Case Presentation: Case 1: Hypertensive female with diabetes and hypertension seen for the first time. Treatment stategy, the patient wanted to go off insulin and get her BP under control. Case 2: 50 year old male who opted to make a change in his diet and life habits. He had been on medication for diabetes and hypertension. Case 3: Masked hypertension in an 85 year old male managed using HMMS. Please visit poster session for details on flow schematics.

Discussion: Patients with DM, HTN, chronic kidney disease(CKD), and coronary artery disease(CAD) are managed using home BP and BS for day to day care. HTN like DM is more based on aggregate home BP readings. In our contemporary practice integrating this type of information for medication titration is labor intensive. Computer naïve patients depend on several schemes to report for achieving target goals and maintain their follow up care. Compiling and integrating this data with medicine titration, and diagnostic tests in a linear flow is arduous. Computer based technology via the internet or other means is expensive and far from being patient friendly. Ascot Technology has devised a wireless system that is "TABLET SIZE" with the unique ability to directly log in the patient's test results(BP,BS and P) thus, there is no manual entry of data. Patients can export their data directly to their chart portal at the clinic using the friendly touch key SEND. Medical clinic interaction is displayed in real-time with a similar friendly format at the patient's site.

Conclusion: We believe patients with chronic illnesses like DM, HTN, CKD, and CAD can be managed with greater intensity using the Ascot Technology's HMMS. Furthermore, we have for the first time, a simple briefcase size tool for use by patient's family or any care giver without having to depend on internet based home monitoring equipment or frequent office visits.

Abstract #1328

IN T2D PATIENTS WITH BASELINE A1C <8.0%, LIRAGLUTIDE ACHIEVES A1C TARGETS MORE OFTEN THAN SITAGLIPTIN OR EXENATIDE

Allen King, MD, FACP, E. Montanya, R. Pratley, Lawrence Blonde, MD, FACP, FACE, Claus Svendsen, Morten Donsmark, Giorgio Sesti

Objective: Limited data are available to clinicians on the efficacy of incretin therapies in type 2 diabetes (T2D) patients who are within 1% of glycemic target (≤7.0%).
Methods: Our post-hoc analysis compared the efficacy of liraglutide 1.8 mg once-daily (OD) to exenatide 10 µg twice daily (BID) (LEAD-6) and sitagliptin 100 mg OD (LIRA-DPP-4) after 26 weeks' treatment; only patients treated as true add-on to metformin with a baseline A1c <8% were included. Patient baseline data were similar in each study (mean A1c 7.3-7.6%) except a shorter mean disease duration in LEAD-6 for exenatide vs. liraglutide (3.9 vs. 6.9 years). Change in A1c and body weight were analyzed using an analysis of covariance (ANCOVA) model based on the intention to treat (ITT) population, last observation carried forward (LOCF). Logistic regression analysis was performed on ITT population, LOCF to compare the proportion of patients achieving glycaemic targets (≤6.5% and ≤7.0%).
Results: In LEAD-6, liraglutide produced a numerically greater mean A1c reduction vs. exenatide (-0.86% vs. -0.61%; estimated treatment difference (ETD) -0.27%, p=0.05), reflected in a higher proportion of patients achieving A1c ≤7.0% (84% vs. 73%; p=NS) and around twice as many reaching A1c ≤6.5% (61% vs. 37%; p<0.05). In LIRA-DPP-4, liraglutide produced a significantly greater reduction in A1c (-1.00% vs. -0.49%; ETD -0.53%, p<0.0001) and higher proportion of patients achieving both A1c ≤7.0% and A1c ≤6.5% vs. sitagliptin (82% vs. 46%; p<0.0001 and 51% vs. 20%; p<0.005). Weight loss with liraglutide was greater vs. exenatide (-3.67 kg vs. -2.63 kg; ETD -1.06 kg) but did not reach statistical significance, whereas the difference was significant vs. sitagliptin (-3.39 kg vs. -0.58 kg; ETD -2.96 kg, p<0.0001). Few patients (8-10%) experienced minor hypoglycemia with all therapies.
Discussion: In patients already close to target A1c, liraglutide 1.8 mg brings more patients to target with more weight loss than exenatide or sitagliptin.
Conclusion: In contrast to liraglutide, sitagliptin and exenatide are unlikely to reduce A1c by approximately 1% in this baseline A1c range and this should be considered when choosing an add-on to metformin in patients close to target.

Abstract #1329

A RARE CAUSE OF FRACTURES: CARBONIC ANHYDRASE II ENZYME DEFICIENCY

Swapna Bemalgi, MD, Ram Jhingan, MD, Yugandhar Manda, Daniel Rubin, MD

Objective: To report a rare cause of recurrent fractures.
Case Presentation: A 22 year-old pregnant lady presented with lower extremity pain and weakness. She was diagnosed with Osteopetrosis and Renal Tubular Acidosis(RTA type-1) at the age of 2 years in Puerto Rico after she presented with a fracture. She was later found to have Carbonic Anhydrase II (CA II) deficiency as the cause of her RTA. She now reported progressively worsening leg pains and weakness ongoing for months since she stopped taking her Bicarbonate pills. Examination revealed a short statured female with multiple bone deformities and mild mental retardation. Her labs revealed -K 1.7mmol/L , Bicarbonate 16mmol/L , serum anion Gap 9mmol/l ,positive urine anion gap(U.Na 87, K 27, Cl 89). Other labs were, Cl 116mmol/L, Ca 8.6mg/dl , albumin 3.2gm/dl, Phosphate 3.9 mg/dl, ALP - 52 IU/L, 25-OH Vit D 11ng/ml, 1,25-OH Vitamin D 109pg/ml , PTH 48pg/ml, S.osm 278mosm/kg, Urine studies -pH 7. These labs confirmed Severe Hypokalemia with Non Anion gap Metabolic Acidosis secondary to RTA type-1. She was treated with IV sodium bicarbonate and IV potassium and her acidosis and symptoms improved dramatically. She was discharged on potassium chloride, PO Bicarbonate and ergocalciferol.
Discussion: CA II deficiency is a rare Autosomal Recessive disorder leading to development of Osteopetrosis, RTA and mental retardation. This enzyme is found in bone, kidney, and erythrocytes. CA2 is involved in proximal tubular bicarbonate reabsorption and distal tubular acidification; hence deficiency causes Proximal and/or Distal RTA. CA II deficiency impairs osteoclast function which inhibits bone resorption. As the balance between osteoclast and osteoblast activity is disrupted, the excessive accumulation of brittle bone leads to Osteopetrosis and increased fractures risk. These patients have normal levels of calcium and phosphorus and diagnosis is established by quantifying carbonic anhydrase activity in erythrocytes. Early institution of alkali supplementation is the key in the management of RTA as chronic RTA causes growth retardation and metabolic bone disease.
Conclusion: Our patient had the triad of symptoms of CA2 deficiency. Her noncompliance with treatment resulted in severe bone disease with recurrent limb fractures with poor healing. CA II deficiency should be considered in evaluation of young adults with osteopetrosis associated with metabolic acidosis.

Abstract #1330

RAPID RESOLUTION OF DIABETES-RELATED RISK MARKERS AND HYPERTENSION IN MORBIDLY OBESE INDIVIDUALS WITH AN EXERCISE-CENTRIC INTENSE LIFESTYLE INTERVENTION

Robert Huizenga, MD, Mickey Urdea, Juan Frias

Objective: To assess the metabolic and blood pressure (BP) response to a regimen of intense exercise and moderate caloric restriction in morbidly obese television contestants.
Methods: "Biggest Loser" participants (17M/18F, 40±14yo, 28 Cauc./4 AA/3 Latino, weight 143±30kg, BMI 46±6Kg/m2, A1C 5.6±0.8%, mean±SD) were studied retrospectively between May, 2011 and March, 2012. The weight loss intervention consisted of ~4hrs of daily exercise (1hr intense resistance, 1hr intense aerobic, 2hrs moderate aerobic activity) with a caloric intake of at least 70% of estimated resting daily energy expenditure. Based on FPG, A1C and/or 2-hr OGTT glucose, 17, 12, and 6 participants had normal glucose tolerance, prediabetes, and T2DM (3 taking metformin) (ADA definition), respectively. HTN was common at the initial exam (n=30), with participants taking a total of 24 separate antihypertensives. We assessed body weight, BMI, % body fat (DEXA), BP, and PreDx® Diabetes Risk Score (DRS) (a laboratory developed diagnostic consisting of fasting glucose, A1C, insulin, adiponectin, CRP, ferritin and IL-2 receptor alpha and an algorithm to predict the 5-yr risk of T2DM; Tethys Bioscience, Emeryville, CA), at baseline and at approximately 1, 5 and 24 wks.
Results: The mean (±SD) % weight loss at Wks 1, 5 and 24 was 3.7±1.2, 14.3±3.5, and 31.9±8.0, respectively (all $p<0.0001$). % body fat decreased from 48.9±4.5 to 30.4±8.4 at Wk 24 ($p<0.0001$). BP declined significantly by Wk5 (SBP 138±19 to 123±11, $p=0.005$; DBP 90±10 to 76±8 mmHg, $p<0.0001$) and remained stable throughout the assessment period. Significant improvements in fasting glucose, insulin, and adiponectin were seen at Wk1 and persisted throughout the assessment period (Baseline vs Wk 1, fasting glucose 91.1±22.7 vs 75.1±11.7mg/dL, fasting insulin 14.1±10.1 vs 5.5±3.3µIU/mL, adiponectin 8.6±3.2 vs 10.5±3.9µg/mL, mean±SD, all $p<0.0005$). A1C decreased progressively throughout the assessment period, with an absolute reduction of 0.53% from baseline by Wk24 ($p=0.0003$). By Wk5, all diagnostic criteria for prediabetes, diabetes and HTN were absent in each participant (despite discontinuation of all DM and HTN meds), and the DRS had decreased significantly ($p=0.005$).
Discussion: An intensive exercise-centric weight loss program resulted in improvements in a broad range of health measures beginning at ~1wk, when mean % weight loss was 3.7%. The weight loss regime quickly "reversed" prediabetes, T2DM and HTN. Additionally, the risk of progressing to T2DM, as assessed by the PreDx DRS, was significantly reduced.
Conclusion: This analysis demonstrates that an intensive exercise-based weight loss program can have rapid and favorable metabolic effects in morbidly obese individuals.

Abstract #1331

PATIENTS ARE MORE LIKELY TO REACH A1C TARGET AT ANY GIVEN TIME DURING 26 WEEKS' TREATMENT WITH LIRAGLUTIDE COMPARED WITH BOTH SITAGLIPTIN AND EXENATIDE

Robert Ratner, MD, Claus Svendsen, Monet Sifford-Wilson, E. Montanya

Objective: Timely attainment of target A1c levels improves patient adherence during intensification of type 2 diabetes (T2D) therapy.
Methods: In a post-hoc analysis, we analyzed proportions of patients reaching ADA target A1c <7.0% at 12, 20, and 26 weeks in two Phase 3b trials: liraglutide (n=233) vs. exenatide BID (n=231) (LEAD-6) and liraglutide OD (1.2 mg: n=221 and 1.8 mg: n=218) vs. sitagliptin OD (n=219) (LIRA-DPP-4). The "time to A1c target" was further analyzed by a Cox proportional hazards model with treatment and previous OAD treatment as fixed effects, and baseline A1c as covariate.
Results: First time to A1c target data demonstrated that a greater proportion of patients achieved glycemic target with liraglutide than with exenatide BID or sitagliptin. In LEAD-6, the estimated odds ratio was 1.50 [95% CI: 1.17; 1.92]; estimated chance of reaching target during the treatment period is 50% higher with liraglutide 1.8 mg than with exenatide BID ($p<0.0068$). In LIRA-DPP-4, odds ratios were 1.76 [95% CI: 1.32; 2.34] and 2.13 [95% CI: 1.62; 2.82] for liraglutide 1.2 and 1.8 mg compared with sitagliptin, respectively ($p<0.0030$; $p<0.0001$).
Discussion: Treatment adherence with liraglutide may be greater compared with sitagliptin or exenatide BID when used to intensify T2D therapy due to the greater likelihood of achieving A1c target.
Conclusion: We demonstrate that the timely achievement of target A1c levels is more likely with liraglutide compared with sitagliptin or exenatide BID.

Abstract #1332

NOVEL GENE EXPRESSION CLASSIFIER RAISES PRE-OPERATIVE SUSPICION OF MEDULLARY THYROID CANCER

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Objective: Medullary thyroid carcinoma (MTC) often mimics non-medullary thyroid follicular neoplasms on fine needle aspiration (FNA) cytology. The Afirma® Gene Expression Classifier (GEC) measures RNA transcript signals from 142 genes and applies an algorithm to classify nodules with indeterminate FNA cytopathology as either "Benign" or "Suspicious". Twenty-five additional genes on the array are used to detect rare neoplasms, including MTC.

Methods: The MTC signature was trained on 22 banked snap-frozen MTC tissue samples, and subsequently locked. Thyroid FNA samples were collected prospectively from several studies: a multicenter study of 49 practice sites (clinic FNAs N=4812), a single academic center study (pre-operative FNAs N=155), a single academic center media study (ex-vivo FNAs N=172) and de-identified consecutive FNAs from 248 physicians in the Veracyte CLIA laboratory (CLIA FNAs N=6,378). After exclusion of samples with inadequate RNA, the GEC was run prospectively and pre-operatively on all cytologically indeterminate samples. A small set of study FNAs with malignant cytology, later confirmed by surgical pathology to be MTC, were run retrospectively on the GEC.

Case Presentation: The GEC identified 13 FNA samples as suspicious for MTC out of a total of 11,345 FNAs collected. Thus far, 11 have been confirmed as MTC on histopathology, and two cases are pending surgery. When surgical pathology was negative for MTC, the GEC did not identify any as suspicious for MTC.

Discussion: This study of over 10,000 biopsied thyroid nodules has permitted prospective analysis of a pre-operative case series of cytologically indeterminate FNAs, and a small set of cytologically malignant FNAs. In both cytologically indeterminate and malignant FNAs, the specific diagnosis of MTC is often not suspected pre-operatively. In this series, the GEC raised suspicion for this uncommon but aggressive neoplasm in 13 samples, with 11 cases confirmed so far. No false positive cases occurred.

Conclusion: The GEC can raise suspicion for MTC in cytologically indeterminate and malignant FNA samples. A pre-operative diagnosis of MTC impacts the pre-operative evaluation of patients, including evaluation for MEN2 and concomitant pheochromocytoma. Additionally, surgical management is altered to include a minimum of total thyroidectomy and central neck dissection. Finally, pre-operative RET proto-oncogene status alters management of unintentionally devascularized parathyroid glands. Larger numbers of MTC FNA samples are required to further validate this novel diagnostic approach.

Abstract #1333

MULTICENTER VALIDATION OF A NOVEL GENE EXPRESSION CLASSIFIER TO PREOPERATIVELY IDENTIFY BENIGN THYROID NODULES WITH INDETERMINATE FNA CYTOLOGY

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Objective: Following fine needle aspiration (FNA), 15-30% of thyroid nodules are not clearly benign or malignant. These cytologically indeterminate nodules are often referred for diagnostic surgery, though most prove benign. A novel gene expression classifier (GEC) was developed to identify benign nodules from cytologically indeterminate aspirates and reported to have high sensitivity and negative predictive value (NPV). After further optimization of the assay, we analyzed diagnostic performance of this novel gene expression classifier in a large prospective, multicenter validation study.

Methods: Thyroid FNA samples were collected from 49 clinical sites, enrolling 3,789 patients with 4,812 thyroid nodules >1cm requiring evaluation. From this, 414 cytologically indeterminate aspirates were obtained, all of which were surgically removed allowing for expert endocrine pathologist assessment of excised lesions. The central histopathologic diagnosis served as the reference standard. Following exclusions for pre-specified criteria (inadequate or degraded RNA (51), excessive study site storage time (36), unavailable reference standard (16), etc.), 265 operated indeterminate nodules remained.

Results: 85 of 265 indeterminate nodules were malignant (32%). The gene expression classifier correctly identified 78 of 85 as 'suspicious' (92% sensitivity, [84%-97%] 95% CI). Specificity was 52%, [44%-59%]. A recent large

study reported a 24% prevalence of malignancy in clinical practice, and when applied to this study, results in an increase in overall NPV from 93% to 95%. Sensitivity was high for each subcategory of cytology diagnosis of atypia/FLUS, follicular neoplasm, or suspicious for malignancy at 90%, 90% and 94% respectively. NPV was 95%, 94%, and 85% for each subcategory respectively, reflecting the study prevalence of malignancy in each subgroup. Prevalence of malignancy in clinical practice may differ from that reported in this study, thereby affecting estimates of NPV in other clinical settings. Analysis of the 7 false negative cases revealed 6 with a paucity of thyroid follicular cells, suggesting that insufficient sampling of the nodule may have occurred.

Discussion: A novel GEC with high NPV, when applied to cytologically indeterminate thyroid nodule aspirates, can modify preoperative assessment of thyroid cancer risk. When applied to nodules with cytology 'atypia/FLUS' or 'follicular neoplasm' the GEC results in a risk estimate comparable to cytologically benign nodules.

Conclusion: These data support consideration of a conservative observation approach for many patients with indeterminate FNA cytology and a benign GEC result.

Abstract #1334

IMPROVEMENT IN INPATIENT GLUCOMETRICS AND REDUCTION IN HYPOGLYCEMIC EVENTS WITH TRANSITION TO BASAL-BOLUS THERAPY AND INTENSIVE DIABETES EDUCATION OF HEALTH CARE PROVIDERS

Michael Gonzales, MD, Christopher Mulla, MD, Joseph Aloi, MD, Paul Chidester

Objective: Glycemic control has been shown to reduce complications, length of hospital stay and improve mortality in some hospitalized patients. There is also a thrust to improve and standardize the quality of inpatient glycemic control. Additionally, as reimbursement shifts towards pay for performance; a greater emphasis will be placed on institutions to achieve nationally standardized glucometrics. Sliding scale insulin is suboptimal for inpatient glucose control and is slowly being replaced by basal bolus therapy. In this study we demonstrate improvement in inpatient glucometrics during the transition from sliding scale insulin (SSI) usage to protocol driven weight-based basal-bolus therapy (BBT) coupled with diabetes education of health care professionals (HCPs).

Methods: This study was conducted in an integrated healthcare system in southeast Virginia that includes 7 hospitals with approximately 1800 beds. A diabetes education program was implemented for all doctors, nurses, pharmacists and other HCPs and their knowledge was assessed before and after the transition from sliding scale insulin to the full implementation of basal bolus order-sets. The use of basal bolus order-sets was introduced via an electronic health care system. Length of hospital stay, outcomes, and percentages of hypoglycemia (<60 mg/dl, goal <2%) and hyperglycemia (>180 mg/dl, goal <20%) before and after the transition period were measured.

Results: Education scores on inpatient diabetes management improved from 51% to 92%. Utilization of SSI was reduced from 87% to 15% while utilization of BBT increased from 13% to 85%. The percent of hypoglycemic events decreased from 1.47% to 1.25%, as did hyperglycemia (22.28% to 17.8%). The length of stay for patients with a secondary diagnosis of diabetes was reduced from 12.98 days to 11.02 days. Despite the decrease in hyperglycemic events there was no concurrent increase in hypoglycemia.

Discussion: Hospital hyperglycemia is associated with increased morbidity, as well as increased length of stay and costs. One barrier to improvement of hyperglycemia is fear of hypoglycemia and inexperience with basal bolus therapy. This study shows that the effective use of an education tool can result in decrease in hyperglycemia without increasing hypoglycemia. A multidisciplinary approach to caring for the hyperglycemic hospitalized patient and using validated treatment protocols is the best approach.

Conclusion: This study details the benefits of educating staff and transitioning treatment protocols from SSI to BBT across seven hospitals. The hyperglycemia improvement program led to improved knowledge and glucometrics and shortened length of stay without increasing hypoglycemic events.

Abstract #1335

PRIMARY CARE DETECTION OF CKD IN ADULTS WITH TYPE-2 DIABETES IN THE ADD-CKD STUDY

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Results: Approximately 10-15% of the US population has chronic kidney disease (CKD). Diabetes is the leading cause of CKD. Early detection will encourage clinicians and patients to address factors that can improve outcomes. ADD-CKD was a US, multicenter, observational study that assessed the prevalence of CKD in adult patients with type-2 diabetes (T2DM) and assessed and characterized the proportion of patients with detected and undiagnosed CKD in the primary care setting using the following: a clinician survey; a patient physical exam and medical history; a single patient blood draw for eGFR and glycosolated hemoglobin (HbA1c); a urine dipstick for

protein; an albumin-creatinine ratio; two patient quality of life questionnaires; and a 15-month patient medical record review. The study consisted of 9339 adult patients with T2DM and 466 investigator sites. Of the 9339 enrolled, 9307 could be assessed using tests for urine protein and serum creatinine to establish the presence of CKD and to assess the sensitivity of the clinician of identifying the presence of CKD.

Discussion: Of the 9307 patients, 5036 (54.1%) had Stage 1-5 CKD based on eGFR and albuminuria; however, only 607 (12.1%) of those patients were identified as having CKD. Clinicians were more successful in diagnosing patients with Stage 3-5 CKD (517/2396 or 21.6%) than Stages 1 and 2 (90/2640 or 3.4%). Of the 445 clinicians who enrolled at least 10 patients, 19 (4.3%) had a ≥50% likelihood of identifying patients with CKD, 217 (48.8%) had a likelihood of <50%, and 209 (47.0%) didn't identify any of their patients as having CKD. The majority of clinicians recognized the eGFR definition of CKD. Of the 445 total clinicians, 382 (85.8%) considered an eGFR value of <60 mL/min/1.73 m2 an indicator of CKD. Most clinicians also recognized low level proteinuria as an indicator of CKD with 61.3% considering trace or +1 proteinuria as the lowest diagnostic value. Despite demonstrated awareness in eGFR and proteinuria test results that indicate CKD, during the 15 months prior to the Study Visit, 51.4% of patients had no protein urine dipstick test, 52.9% of patients did not have urinary albumin-creatinine test, and 15.2% of patients did not have an eGFR test performed.

Conclusion: CKD is significantly under-diagnosed in the T2DM population. Simple and cost-effective testing for the presence of protein is essential in diagnosing CKD in its early stages. Early recognition may reduce the burden of end-stage renal disease and high rates of death and cardiovascular disease associated with CKD. Additional analyses will explore the impact of CKD detection on the implementation of evidence-based CKD interventions and outcomes.

Abstract #1336

ACCURACY AND ACCEPTABILITY OF THE 6-DAY ENLITE GLUCOSE SENSOR

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Objective: The Enlite glucose sensor was previously evaluated in adults and children and shown to be accurate for 6 days, durable, and acceptable to patients and parents/caregivers. We evaluated its accuracy using abdominal insertion sites at a wide range of glucose concentrations and at different rates of glucose concentration change.

Methods: A pivotal 6-day trial in adults was conducted at 7 US investigational centers. Sensors were self-inserted and taped. Each patient wore 1 or 2 sensors on the abdomen and these sensors were calibrated 3-4 times per day throughout the study. Accuracy was evaluated vs frequently-sampled YSI plasma glucose values. The frequent sampling tests lasted for 12 hours on days 1, 3, and 6. Hypoglycemia (to ≤75 mg/dL) and hyperglycemia (to ≥180 mg/dL) were induced on day 1 (beginning immediately after initial calibration), day 3, and day 6. Accuracy was assessed on different days (1, 3, and 6) and at different glucose concentration ranges (<75 mg/dL, 75-180 mg/dL, and >180 mg/dL). Accuracy of calibration was also assessed at rapid, moderate, and slow absolute rates of change (|ROC|) (>2, 1-2, <1 mg/dL/min, respectively). Patient satisfaction with Enlite was evaluated with a 7-point Likert-type questionnaire. Adults with type 1 (n=65) or type 2 (n=25) diabetes (mean age 44, range 18-71) participated.

Results: Mean self-reported survey responses were 5.9/7 for "ease of use," 6/7 for "comfort," 5.9/7 for "ease of insertion," and 5.8/7 for "would recommend." The overall mean (median) absolute relative differences (ARD) were 13.6% (10.1%). The mean ARD was highest on Day 1 (15.9%) and lowest on Day 3 (11.8%). At sensor glucose (SG) <75 mg/dL, the mean (median) absolute differences between sensor and YSI values was 10.8 mg/dL (8.5 mg/dL). At SG >180 mg/dL, the mean (median) ARD were 12.0% (9.0%). At rapid ROC the mean (median) ARD were 16.3% (12.9%), at moderate ROC were 12.9% (9.6%), and at slow ROC were 13.6% (10.1%). There were no device-related adverse events.

Discussion: The Enlite sensor is accurate, durable, comfortable, safe, and easy to use. It is accurate during periods of stable or changing glucose concentrations and met predefined success criteria for agreement with YSI values at all glucose ranges.

Conclusion: Improvements in continuous glucose sensing should expedite development of semiautomated insulin delivery features in modern pumps.

Abstract #1337

PSEUDOPAPILLARY ADRENAL TUMOR: CASE REPORT

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Methods: We report an extremely rare case of an ectopic solid pseudopapillary tumor of the pancreas in a 27 year old woman. She had the diagnosis of familial

adenomatous polyposis (FAP). Genetics department evaluated the patient and considered it a unique case of familial adenomatous polyposis (since no other family member is affected).

Case Presentation: An abdominal CT scan for postoperative follow-up reported a left adrenal heterogeneous tumor, 4.6 x 3.8cm, with irregular borders, hypodense areas, irregular coarse calcifications and areas of solid tissue with striation marks, and adjacent fat with 90 Hounsfield Units. The tumor was explicitly separated from the pancreas tail and spleen, but no interface between the kidney and adrenal was identified, increasing the likelihood of malignancy. An endocrinologic work-up excluded a functional adenoma: urinary metanephrines 119µg/24hrs, 491µg/24hrs urinary normetanefrines, urinary total metanephrines 610µg/24hrs, cortisol 12mcg/dl and 0.6mg/ml post 1 mg dexamethasone, ACTH 36pg/ml, plasma renin activity 9.49ng/ml/h, serum aldosterone 106ng/ml and PAC/PRA ratio was 11.2. Left adrenalectomy was performed and the histopathological report was solid pseudopapillary tumor.

Discussion: This is a 27 year old patient diagnosed with FAP and solid pseudopapillary tumor in an adrenal incidentaloma. Multiple studies have reported a prevalence of 13% of adrenal incidentalomas in FAP patients. Biallelic inactivation of APC may result in adrenal adenomas. Up to 74% of adrenal lesions are non-secretory, 14.8% are functional and 4% are carcinomas. A possibility in FAP patients with adrenal lesions is the presence of metastasis from another cancer, especially those with previous diagnosis of colon cancer. In these cases it may be helpful to perform a fine needle biopsy. The solid pseudopapillary tumor is a rare neoplasm with low malignancy, which usually begins in the pancreas. Its extrapancreatic location is rare worldwide. These tumors usually occur in women <35 years of age and may also arise in the omentum, mesentery, peritoneum or liver. Tomographically these lesions are well demarcated, solid and cystic and have calcifications. Complete surgical resection is curative and the prognosis is favorable, although 15% of these patients develop metastases, and therefore need close monitoring (CT scan each year). Local recurrence is rare.

Conclusion: In this case, the tumor was separated from the pancreas and pancreatic tissue was not reported as ectopic. Only 6 cases have been reported of extrapancreatic solid pseudopapillary tumors; 2 were due to ectopic pancreatic tissue. Currently only one reported case was in the retroperitoneum.