LATE BREAKING

Abstract # 1300

CUSHING’S STORM SECONDARY TO ECTOPIC ACTH SekRETING METASTATIC BREAST CANCER

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Adrenal Disorders

Objective: Describe a rare ectopic acth secreting breast cancer causing cushings syndrome presenting with posterior reversible encephalopathy syndrome (PRES) from uncontrolled hypertension leading to psychosis along with opportunistic infection & liver function abnormalities posing unique management issues.

Case Presentation: A 31 year old female, with triple negative, high grade invasive right breast ductal carcinoma treated with chemotherapy, bilateral mastectomy & radiation presented with acute psychosis. She had no sleep for 4 days, was hyperenergetic, easily distracted & impulsive with racing thoughts, pressured speech & was paranoid that her husband was trying to hurt her. On exam she had round, ruddy hirsuite face with acne & BP 156/108. Labs showed K 1.7, random cortisol >70mcg/dL, 1mg & 8mg overnight dexamethasone showed cortisol >100mcg/dL. She had elevations in AST 103, ALT 237, ACTH 1173 pg/ml, Total testosterone 170ng/dL, DHEA-S 499mcg/dL, 17 OH progesterone 1780 ng/dL & 24 hr urine cortisol (UFC) 14766mcg. CT abdomen showed extensive hepatic metastatic disease & bilateral adrenal hyperplasia. Renin, aldosterone, plasma metanephrines, chromagranin A, corticotropin releasing hormone & gastrin levels were normal. Imaging was -ve for thyroid nodule, thymic neoplasm & bronchial carcinoma. Core liver biopsy revealed metastatic breast adenocarcinoma that was -ve for neuro endocrine markers CD56, synaptophysin, neuron specific enolase & chromogranin. CT head showed white matter disease consistent with PRES. She was psychotic & hypereensive despite using mifepristone with multiple antihypertensives including lisinopril, aldaetone & metoprolol targeting a systolic BP 110-130. Transaminitis did not allow mifepristone escalation > 600mg/day. Etomidate infusion at non sedating dose of 0.1mg/kg/min in ICU controlled her hypertension & cortisol levels to 20-30mcg/dL. UFC reduced to 820mcg. She was transitioned to metyrapone 1250mg PO Q 6h & spironolactone 100mg PO Q 6h. Dapsone used for PCP prophylaxis due to bactrim allergy did not prevent PCP pneumonia, which was treated primaquine. She was discharged from hospital on metyrapone with spironolactone & is undergoing chemotherapy.

Discussion: Ectopic ACTH cushings syndrome from breast cancer is extremely rare presenting with significant morbidity & mortality from opportunistic infections, psychoses, metabolic & coagulation derangements. This case, reports management of cushings psychosis resulting from PRES & hypertension with etomidate followed by metyrapone & spironolactone transition since mifepristone could not be increased in liver dysfunction.

Conclusion: Ectopic cushings syndrome presents unique diagnostic & management challenges.

Abstract # 1301

UNIQUE DIAGNOSTIC CHALLENGES OF CUSHINGS FROM LARGE BILATERAL ADRENAL ADENOMA AND MIFEPRISTONE UTILIZATION FOR HYPERCORTISOLISM PRIOR TO SURGERY.

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Adrenal Disorders

Objective: Describe evaluation & management of Cushings patient with bilateral (b/l) adrenal adenomas.

Case Presentation: A 47 year old female was found to have a 1.8 x 4.9 cm right adrenal nodule & 2.8 x 4.3 cm left adrenal nodule with calcification on CT during abdominal pain evaluation. She had hypokalemia of 6months, uncontrolled hypertension despite metoprolol & lisinopril treatment, glucose intolerance, stroke, peripheral vascular disease, CABG & chronic cellulitis at vein harvest site. She had high, urine free cortisol 89.2g/24hr, cortisol on 1mg & 8mg overnight dexamethasone testing was 18.4 & 20 μg/dL respectively, 11pm salivary cortisol X2, with random cortisol 17.3 μg/dL & ACTH < 5 pg/mL and normal DHEA-S, plasma metanephrine, androsteindione, renin & aldosterone levels. Since adenomas were > 4cms b/l adrenal surgery was indicated. Adrenal venous sampling (AVS) done with dexamethasone 2 mg Q6hours X 1 day, for nodule functional assessment, showed left cortisol 84.9, epinephrine 433 pg/mL, right cortisol 56.5, epinephrine 1911 and venacaval cortisol 11.7 and epinephrine 28. Adrenal vein epinephrine levels stepup >100pg/mL indicated succesful catheterization, b/l adrenal vein to vencaval cortisol ratio >4 was consistent with autonomous cortisol secretion & left to right adrenal cortisol ratio < 2 suggested b/l cushings based on literature from Mayo Clinic. First step of right adrenal surgery was deferred for her carotid artery surgery. Also her multiple co-morbidities, hypertension, poor functional status needing walker & cane, chronic cellulitis & glucose intolerance required medical management of hypercortisolism prior to...
surgery. Ketoconazole was contraindicated since she was on plavix & atorvastatin. Mifepristone was started after her carotid surgery. She had, 50lbs weight loss, normal BP, complete healing of chronic leg cellulitis of 10months on antibiotics & could ambulate independently by 4 months of Mifepristone. Her potassium, BP & liver functions tests were closely monitored. Her right adrenal adenoma resection on pathology had multiple adrenocortical nodules without adrenal carcinoma.

Discussion: This case had unique diagnostic & management dilemmas. Aldosterone in AVS, reportedly used in case reports, might have helped in diagnosis, given the subtly low right cortisol & low left epinephrine levels (likely from phrenic vein dilution) but based on size, b/l adrenal resection was indicated. Mifepristone, FDA approved for inoperable Cushing, facilitated pre surgery optimization by mitigating hypercortisolism.

Conclusion: Bilateral adrenal tumours in Cushings pose complex clinical problems. Mifepristone could potentially be utilized as a bridge when surgery needs to be deferred.

Abstract # 1302

INCIDENCE OF ADRENAL INSUFFICIENCY AND ITS RELATION TO MORTALITY IN PATIENTS WITH SEPTIC SHOCK

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Adrenal Disorders

Objective: To determine the incidence of adrenal insufficiency and its relation to mortality in patients with septic shock.

Methods: In patients of septic shock, APACHE II score was calculated and serum cortisol was measured at the time of admission and 1 hour after giving 250 μg ACTH. Hydrocortisone was added to inotropics in all patients after drawing 2nd blood sample for serum cortisol and was continued till 7 days or less. In our study, the patients with inadequate adrenal response were divided into two groups: 1) absolute adrenal insufficiency – baseline cortisol < 20 μg/dL and increment ≤ 9 μg/dL after the ACTH stimulation test; 2) relative adrenal insufficiency – patients with baseline cortisol>20 μg/dL and increment<9 μg/dL.

Results: The incidence of AI in septic shock was 42% (absolute 14%, relative 28%). The mortality rate was 48%, and it was higher in patients with AI than in patients without AI (P = 0.017). The APACHE II score > 25 carried higher mortality rate than a score of < 25 (P <= 0.001). Baseline serum cortisol > 1210 nmol/L had exceptionally high likelihood of mortality (OR 50, P <= 0.001). Among those who survived, inotropic support was required for longer period in relative as compared to absolute AI and to non-AI.

Discussion: We have observed that patients with AI required vasopressors support for longer time than who did not. Only the patients with absolute AI insufficiency and patients of non-AI with high level of serum cortisol (> 1210 nmol/L) had shown the response to vasopressor therapy. But, there was no response in patients with relative AI in septic shock. Though the study has one important limitation that it comprised of small number of subjects, we would like to conclude that AI was prevalent among patients with septic shock, and it was related to prolonged requirement of vasopressors and to prolonged duration of shock. In septic shock, despite treatment with hydrocortisone in patients with AI, these patients had higher mortality compared to those who did not have AI, and the higher mortality risk was most likely with absolute AI. The mortality risk was also high when baseline serum cortisol was more than 1210 nmol/L. We would like to recommend evaluating the adrenal status in patients with septic shock because it would entail prognosis.

Conclusion: AI is prevalent among patients with septic shock. We found that higher APACHE scores were associated with higher rates of adrenal failure and mortality in patients with septic shock. There also appears to be a bimodal distribution of mortality with adrenal status in patients with septic shock.

Abstract # 1303

PROGNOSTIC FACTORS IN PATIENTS HOSPITALISED WITH DIABETIC KETOACIDOSIS

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Diabetes/Prediabetes

Objective: 1. To evaluate the clinical and biochemical prognostic markers in diabetic ketoacidosis. 2. To correlate the various prognostic markers with mortality in diabetic ketoacidosis.

Methods: Two hundred and seventy patients hospitalized with diabetic ketoacidosis over a period of 1 year were evaluated clinically and by laboratory tests. Serial assays of serum electrolytes, glucose and blood pH, and clinical outcome of either discharge home or death were evaluated.
Results: The significant predictors of final outcome obtained were further regressed together and subjected with multivariate logistic regression (MLR) analysis. The MLR analysis further revealed that the male sex had 7.93 fold higher favorable outcome as compared to female sex (OR=7.93, 95% CI=3.99-13.51) while decrease in mean APACHE II score (14.83) and S. PO3-- (4.38) at presentation may lead 2.86 (OR=2.86, 95% CI=1.72-7.03) and 2.71 (OR=2.71, 95% CI=1.51-6.99) fold better favourable outcome respectively as compared to higher levels (APACHE II score: 25.00; S. PO3--: 6.04).

Discussion: Sex, baseline biochemical parameters like APACHE II Score, and phosphate level, were important predicators of mortality from DKA.

Conclusion: Diabetic ketoacidosis still remains a big threat to the emergency department. Prompt diagnosis and intervention can reduced the morbidity and mortality associated with diabetic ketoacidosis.

Abstract # 1304

PREVALENCE, CLINICAL PROFILE, AND GLYCEMIC VARIABILITY AND EFFECT OF GLUTEN FREE DIET ON WEIGHT AND GROWTH VELOCITY IN CELIAC DISEASE PATIENTS WITH TYPE 1 DIABETES MELLITUS IN WESTERN, U.P, INDIA

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Diabetes/Prediabetes

Objective: To study the prevalence, clinical profile and glycemic variability and the effect of gluten free diet on growth and diabetic control in celiac type 1 diabetes patients in a tertiary care referral centre in north India.

Methods: Total of two hundred and fifty six patients were screened (149 males and 107 females) during the study period of two years, patients were evaluated for the clinical signs, biochemical investigations and family history of celiac disease in tertiary care health center in western U.P.

Results: Twenty four (9.37%) patients were diagnosed to have celiac disease; the mean age at diagnosis of diabetes was 9.34 ± 7.3 years. Only 1/24 patients with celiac disease had been diagnosed before detection of diabetes mellitus. The common manifestations were normocytic normochromic anemia (66.6%) followed by diarrhoea (62.5%), abdominal pain/bloating sensation (58.3%), and short stature (58.3%). Weight SDS increased from −0.12+1.3 at the start of gluten free diet to 0.8+0.9 after 12 months later (p<0.05). Height SDS increased from −2.46+1.1 at the start of gluten free diet to −2.14+0.9 after 12 months later (p =0.087). Bone age SDS increased from 9.2+6.3 at the start of gluten free diet to 10.3+6.7 after 12 months later. Height velocity increased from 4.7+0.7 cm/year in the year before treatment to 5.1+1.2 during treatment (p= 0.05). The increased in Hemoglobin, serum calcium, and serum iron is statistically significant (p<0.05).

Discussion: There are very few studies which show the effect of gluten free diet on weight, height, height velocity, and other biochemical parameters in celiac type 1 diabetes patients. There is very limited data from the Indian subcontinent. Numerous studies showed the positive effect of Gluten-free diet on increased weight, height and growth velocity as well as on serum calcium, serum ferritin and hemoglobin. Moreover improvement of bone status in patients with type 1 diabetes mellitus and adherence to gluten-free diet has been reported.

Conclusion: Celiac disease was found to be significantly associated with type 1 diabetes, timely identification of these disorder are of paramount important for better glycemic control and to reduced the morbidity and mortality associated with the conditions.

Abstract # 1305

SENTARA HEALTH SYSTEM SEES GLYCEMIC IMPROVEMENTS FOR HOSPITAL BASAL BOLUS PATIENTS USING E-GLYCEMIC MANAGEMENT SYSTEM

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Diabetes/Prediabetes

Objective: Hospitalized patients who experience hypoglycemia, whether insulin induced or spontaneous, have been associated with increased mortality and hospital cost as compared with patients with an absence of hypoglycemia. Sentara’s successful experience with Glucomander®, (GM) a web based insulin algorithm integrated with the hospital EHR, at Virginia Beach Hospital in inpatient insulin protocols suggested that its expanded use across the Sentara Health System may improve glycemic control for Subcutaneously (SQ) treated patients with Basal Bolus Insulin (BBI) Orders. We studied the glycemic impact using the GM system for the treatment patients with hyperglycemia compared to BBI therapy alone.
Methods: This 30-day quality initiative is being conducted at Sentara Healthcare System in southeast Virginia across 2 Hospital sites. Patients needing SubQ insulin were placed on the GM system and we measured the first 30 days of use to the hypoglycemia baseline of the 30 days prior to GM at each site. Electronic BBI order sets were used to treat insulin patients during the baseline period. The glycemic targets being used for this project were % of blood glucose events <70 mg/dl and Glucose Profile Day 1 vs Day of Discharge for the 178 patients in this analysis.

Results/Case Presentation: Baseline hypoglycemia episodes (<70 mg/dl) were reduced by 56% with GM SQ compared to BBI. GM SQ had a Day1 to Day of Discharge delta of 70 mg/dl from 233 to 163 (mg/dl) for a 30% reduction (P=0.005 +/- 76). BBI fell from 215 to 184 (mg/dl) for a 15% reduction (P=0.05 NS +/- 117). GM SQ patients had an average of a 2-fold glucose reduction from baseline compared to patients treated with BBI (p=0.01). Day of Discharge glucose average for GM SQ was 163 directly at the midpoint of the target range of 140-180 (mg/dl).

Discussion: Use of the eGlycemic Management System GM SQ to treat hyperglycemic patients on SQ insulin showed dramatic reductions of up to 56% in hypoglycemia in the first 30 days of use and 2-fold improvements in hyperglycemia compared to use of BBI treatment orders.

Conclusion: Our results support that GM SQ is a safe and clinically effective tool for improving glycemic control for patients who need SQ insulin treatment while in the hospital.

Abstract # 1306

GLYTEC’S E-GLYCEMIC MANAGEMENT SYSTEM IN COMBINATION WITH REAL-TIME CLINICAL SURVEILLANCE REDUCES HYPOGLYCEMIA AND IMPROVES LENGTH OF STAY IN HOSPITALIZED PATIENTS WITH DIABETIC KETOACIDOSIS

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Diabetes/Prediabetes

Objective: Ongoing efforts for improving quality in the care of persons with diabetes frequently focus on avoiding unnecessary hospitalizations, decreasing length of stay and avoiding readmission to hospital following discharge. Additionally, as reimbursement shifts towards pay for performance; a greater emphasis will be placed on institutions to achieve nationally standardized glucometrics. Glucommander (GM) was previously proven to improve throughput effectiveness for the treatment of mild to moderate Diabetic Ketoacidosis (DKA) in the Emergency Department. This study is focused on safely improving length of stay (LOS) for the diabetic patients with DKA utilizing real-time clinical surveillance, GlucoSurveillanceTM.

Methods: We reviewed the hospital care of 168 patients with a diagnosis of DKA at 3 community hospitals. The patients were studied over a 3-month time period. Qualifying patients had a diagnosis code of DKA (ICD 150.10-250.13), elevated admission glucose (>250 mg/dl), elevated Anion Gap (AG>12 mEq/L), and were treated with Glucommander. Patients were divided into 2 groups: 116 patients were Appropriately Discontinued (AD) from GM once their AG resolved (<12 mEq/L) and 70 patients were Prematurely Discontinued (PD) from GM with an AG (>12). GlucoSurveillance™ was used to discover patients with controlled glucose (<180 mg/dl) and resolved AG (<12 mEq/L). There was no significant difference in glucose at discharge between the two Glucommander groups: PD group (187 mg/dl +/- 83) and AD group (178 mg/dl +/- 69). 100% of patients in both GM groups achieved target glucose. Hypoglycemia was low in both groups, but there was an increase in Hypoglycemia when GM was discontinued, with GM patients (0.1%) < 40 mg/dl compared with (0.9%) < 40 mg/dl for Post GM care. Length of stay was reduced by 2.4 days with AD patients at 5.33 LOS compared to PD patients at 7.7 LOS.

Discussion: This study shows the clinical impact of using the tools available in the eGlycemic Management System™, such as Glucommander™ and GlucoSurveillance™, to improve the efficacy, safety and efficiency of care provided to DKA patients in the hospital. Conclusion: Glucommander™ improved patient safety by reducing the incidence of hypoglycemia in patients with DKA, while bringing 100% of these patients into the glycemic target. Length of Stay was reduced by over 2 days for patients treated with Glucommander™ and appropriately discontinued.
**Abstract # 1307**

**EFFECT OF SITAGLIPTIN ON SHORT-TERM METABOLIC DYSREGULATION OF ORAL GLUCOCORTICOID THERAPY**

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**Diabetes/Prediabetes**

**Objective:** Steroid induced hyperglycemia, a complication of glucocorticoid therapy, occurs through mechanisms including impaired insulin secretion and insulin resistance. DPP-4 inhibitors (DPP4-i) represent attractive therapeutic agents to mitigate dysglycemia with glucocorticoids due to ease of dosing and side effect profile.

**Methods:** We are conducting a prospective, randomized, double-blind, placebo-controlled, crossover study to evaluate effects of DPP4-i therapy on metabolic dysregulation of oral glucocorticoid therapy in subjects with pre-diabetes. 6 subjects have completed the study and we present here preliminary results. Dosages and durations of therapy are 2.5 mg Dexamethasone (dex) daily plus either placebo or sitagliptin (sita) 100 mg daily for 7 days. Subjects were randomized to order of study drug: dex + placebo (pla) versus dex + sita. Each study period was followed by a wash out period (average 62±20 days) before crossover.

**Results:** Demographics for subjects enrolled to date are as follows: 4 men, 2 women; mean age 52±6 yrs; A1c 5.9±0.2%; BMI 30.9±2.9; fasting plasma glucose 96.3±10.1 mg/dL with pre-diabetes determined by A1c or 2 hr glucose after 75 g OGTT 140-199 mg/dL. There were no differences in baseline glucoses or insulin for IVGTT or MMT, suggesting adequate washout between study periods. We compared insulin area under the curve for the first 15 minutes (AUC0-15 mins) from IVGTT and found no difference between placebo and sita on dex (AUC0-15 mins insulin on placebo 1004±790 vs sita 967±732 µU/ml, p=0.87). We compared AUC0-30, AUC0-60 and AUC0-90 for glucose on dex during MMTs and found no difference between placebo and sita. While insulin values were not statistically different on study drug between placebo and sita, there was a trend towards lower insulin levels on sita compared to placebo in the first 30 minutes of MMT (AUC0-30 placebo 1748±630, sita 1338±932, p=0.10). This suggests DPP4-i therapy may enhance insulin sensitivity during MMT with glucocorticoid therapy, but results remain preliminary in this small cohort.

**Discussion:** While steroid-induced hyperglycemia will not be effectively treated in all patients with DPP4-i therapy, for those with mild dysglycemia, these well tolerated oral agents represent an attractive therapeutic option. Ease of dosing and safety profiles suggest they can be used without significant risk, and may ameliorate adverse metabolic effects of glucocorticoids.

**Conclusion:** This suggests DPP4-i therapy may enhance insulin sensitivity during MMT with glucocorticoid therapy, but results remain preliminary in this small cohort.

**Abstract # 1308**

**EARLY COMBINATION TREATMENT IMPROVES GLYCEMIC GOAL ATTAINMENT IN TYPE 2 DIABETIC PATIENTS FAILING METFORMIN MONOTHERAPY: A META-ANALYSIS**

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**Diabetes/Prediabetes**

**Objective:** Type 2 diabetic patients on metformin often need treatment intensification with an add-on drug to reach glycemic targets. However, the benefit of appropriate intensification in terms of glycemic goal attainment has not been adequately quantified. We conducted a meta-analysis of existing data from randomized controlled trials (RCTs) evaluating the impact of non-insulin antidiabetic drugs added to metformin on glycemic goal attainment among patients uncontrolled on metformin monotherapy.

**Methods:** Systematic literature search was first conducted using in PubMed, Embase, and Cochrane Central through 02/2013. Random effects meta-analysis yielded relative risk (RR) and 95% confidence interval (CI) for attainment of A1c goal <7%. Subgroup analysis was conducted based on duration of follow-up.

**Results:** Overall, 24 RCTs (n=9,485, median follow-up 24 weeks; range 12-104 weeks) were included comparing any add-on drug to metformin vs. continuing metformin monotherapy. Various classes of antidiabetic drugs included SU (1 RCT), glinide (1 RCT), TZD (3 RCTs), AGI (1 RCT), DPP4 inhibitor (14 RCTs), GLP1 agonist (3 RCTs), and SGLT2 inhibitor (1 RCT). Goal attainment was defined as A1c <7%. Upon meta-analysis, treatment intensification was associated with significantly increased likelihood of glycemic goal attainment (41% vs 25%, RR 2.14, 95%CI 1.81-2.54) vs. metformin monotherapy.
Discussion: Benefit of adding add-on drug to metformin was evident in both short term and medium term durations, as seen in subgroup analysis, with 12-18 week follow-up (7 RCTs, 44% vs 27%, RR 2.51, 95%CI 1.65-3.83) and 24-36 week follow-up (15 RCTs, 42% vs 20%, RR 2.02, 95%CI 1.67-2.45) providing significant findings.

Conclusion: The use of an add-on agent to inadequately performing metformin monotherapy is associated with significant improvement in glycemic goal attainment.

Abstract # 1309

MEASURING SMALL FIBER FUNCTION AS A MEANS TO ASSESS CLINICAL RESPONSE IN THE TREATMENT OF CIDP

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Diabetes/Prediabetes

Objective: Chronic inflammatory demyelinating polyneuropathy (CIDP) is 11X more common among people with diabetes than the general population and is treatable with appropriate immunotherapy. There is no agreement on criteria to evaluate treatment response and no objective measure of sensory or autonomic small fiber function. We present the case of a patient with type 2 diabetes (T2D) and CIDP whose treatment response was measurable with the Sudoscan sudomotor function test. This test may represent a new objective evaluation of the treatment of CIDP.

Case Presentation: The patient is a 60 year-old male initially referred to our center in August 2012 with a diagnosis of CIDP based on AAN electrodiagnostic criteria (NCS). He complained of burning, numbness, shooting pains, and gait impairment. Autonomic functions were significant for low heart rate variability response to expiration/inspiration (E/I), Valsalva maneuver and the ratio of the RR interval for the 30th to the 15th beat upon standing (1.08, 1.12, 1.05 respectively), and frequency analysis of the total spectral power, the standard deviation of the normal RR intervals (sdNN) and their root mean squared (rmsSD). Sudoscan electrochemical skin conductances (ESC), measuring small nerve fiber function on the palms and soles, were very low: 23μS in the feet and 32μS in the hands. After one cycle of intravenous immunoglobulin (IVIG – 6 doses total, 75g each) the patient had no change in symptoms or Sudoscan scores for feet or hands (23μS and 32μS.) However, E/I, Valsalva, and 30:15 ratios (1.19, 1.36, 1.39 respectively) were improved. In March 2013, the patient ’s autonomic functions worsened (E/I, Valsalva, and 30:15 ratios 1.1, 1.07, 1.12 respectively) but feet and hand ESC started to improve (35μS and 52μS respectively). Azathioprine was started. Eight days after a second cycle of IVIG in January 2014, the patient reported for the first time less burning, shooting pains and tingling. E/I, Valsalva, and 30:15 ratios remained low (1.03, 1.07, and not analyzable, respectively), while foot and hand ESC scores continued to improve (43μS and 55μS respectively). NCS never showed significant measurable change.

Discussion: Goals of CIDP treatment are to reduce symptoms, improve functional status, and obtain long-term remission. We found that NCS and autonomic function tests did not correlate well with clinical status while numerical Sudoscan scores matched closely symptomatic changes. ESC have been found to correlate well with peripheral small fiber function and neuropathic symptoms in DPN.

Conclusion: The findings in this patient warrant further investigation of the use of Sudoscan to monitor CIDP response to therapy.

Abstract # 1310

METFORMIN AS ADJUNCT THERAPY TO INSULIN TREATMENT IN PATIENTS WITH TYPE 1 DIABETES DOES NOT SIGNIFICANTLY IMPROVE GLYCEMIC OUTCOMES: SYSTEMATIC REVIEW AND META-ANALYSIS

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Diabetes/Prediabetes

Objective: This study was intended to evaluate whether metformin therapy can show benefits in patients with T1DM by improving A1C, fasting plasma glucose (FPG) and reducing daily insulin requirements.

Methods: A systematic literature search of PubMed, EMBASE and the Cochrane Library (through 09/2013) sought randomized controlled trials (RCTs) evaluating metformin compared to control in patients with T1DM reporting A1C, FPG or daily insulin requirements. Two investigators independently determined study selection and data extraction with discrepancies resolved by discussion. The weighted mean differences (WMD) of change from baseline (with 95% CIs) were calculated using a random-effects model while I2 statistic was used to determine heterogeneity.

Results: A total of 10 studies consisting of RCTs (n = 450
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pts, age range 16-48 yrs, follow-up range 3-24 wks) met inclusion criteria and were meta-analyzed. The combined results showed that metformin as adjunct to insulin therapy did not significantly lower A1C -0.22% (95% CI = -0.51 to 0.068, I² = 42%, n = 6 study) or FPG -17.63 mg/dL (95% CI = -95.7 to 60.4, I² = 91%, n = 3 studies) compared to insulin therapy alone. There was however, a statistically significant reduction in total daily insulin requirements measured by either -0.10 units/kg/day (95% CI = -0.17 to -0.029, I² = 0%, n = 3 studies) or -4.79 units/day (95% CI = -9.32 to -0.25, I² = 0%, n = 3 studies).

Discussion: Although it has been suggested that metformin promotes glucose control in patients with T1DM, there is insufficient evidence in the literature to make a definitive conclusion about the effects of metformin in patients with T1DM.

Conclusion: Results suggest that metformin as an adjunct agent in patients with T1DM may help to reduce the daily insulin requirements in patients with T1DM compared to placebo control. There was no statistically significant effect on either A1C or FPG with metformin as an adjunct to insulin therapy in patients with T1DM.

Abstract # 1311

CASE REPORT AND CLINICAL ANALYSIS: TYPE 2 DIABETES BEFORE, DURING AND AFTER THE PREGNANCY

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Diabetes/Prediabetes

Objective: Obstetricians more often than endocrinologists see the consequences of uncontrolled diabetes in women - infertility, miscarriage, preeclampsia, birth defects, birth injuries to mother and child are well known complications of diabetes in pregnancy. Gestational diabetes alone complicates 18% of pregnancies in the US and 10.8% of all women aged 20 or older have diabetes. This case report describes what has now become a frequent scenario and presents an opportunity to discuss pathophysiology and therapeutic approach to T2DM in pregnancy.

Case Presentation: Our patient is a 30 year old Hispanic female with well controlled hypothyroidism prior and during pregnancy and uncontrolled T2DM diagnosed 3 years earlier. Patient’s initial visit to clinic was after a missed abortion a year prior, her HbA1c at the time was 10.7%. Lost to follow up, she was able to conceive again with a HbA1c of 9.1% and was referred back to our clinic one year later. Patient was started on insulin therapy on her first visit and within the first month she was able to reduce the HbA1c from 9.1% to 6.9% (gestational weeks 9 through 13). She received intensive diabetes education and was encouraged to make dietary changes and exercise regularly. Patient was seen weekly for HbA1c monitoring, blood sugars and diet log review and insulin dose adjustments.

Discussion: It is well documented that insulin requirements increase gradually during the pregnancy. The “amount” of insulin resistance can be best observed by nighttime insulin requirements that are needed to achieve morning euglycemia. The nighttime is the only time that is not affected by daily activities such as diet, lifestyle, exercise. Our patient was able to maintain euglycemia during the day without long-acting insulin, however at bedtime she did require 50 units of NPH insulin by the 36th week (almost 50% greater than pregnant women with T1DM. This large dose of NPH is the true representation of the severity of insulin resistance at the end of her pregnancy. Her prandial blood sugars were well controlled with 20-30 units of rapid-acting insulin by the 36th week. During the third trimester, when insulin resistance reaches it’s peak, our patient achieved a HbA1c of 5.5 - 6.1%. Patient delivered early at 36 weeks a healthy 6lb 12oz (3060g) baby with APGAR 8 and 9 on minutes 1 and 5 accordingly. Her insulin requirement decreased from 130 units TDI at the end of the pregnancy to 60 units postpartum. During breastfeeding, bedtime NPH requirement decreased from 40 to 10 units nightly.

Conclusion: It is important to individualize insulin therapy in pregnancy with the notion of evolving insulin resistance to prevent dreaded complications to the mother and child.
Abstract # 1312

SHORT TERM CONTINUOUS SUBCUTANEOUS INSULIN INFUSION THERAPY (CSII) SIGNIFICANTLY IMPROVES ERECTILE DYSFUNCTIONS IN PATIENTS WITH TYPE-2 DIABETES MELLITUS

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Diabetes/Prediabetes

Objective: To determine the efficacy of 12 weeks CSII Vs basal bolus MDI among uncontrolled T2DM patients with ED.

Methods: This is a comparative study over a period of 12 weeks from two endocrine centers on 46 T2DM patients (age 32 to 60 y, mean age 43.8 y) who presented with uncontrolled hyperglycemia (FPG > 200 mg/dl, HbA1c>10.0 %) & having ED and were on multiple oral hypoglycemics. All of these patients had ED as graded on the Erectile Hardness Grading Scale 1 to 4 (EHGS) [European Association of Urology]. EHGS grade 4 indicates completely hard and fully rigid erections, Grade 3 enough for penetration only, grade 2 means the penis is hard, but not hard enough for penetration and Grade 1 indicates that the penis is larger than normal, but not hard. All the study patients had EHGS grading of 1 to 2 only. The patients were randomized into 2 treatment groups, 23 patients were put on MDI while on another 23 CSII was initiated to manage severe hyperglycemia. Patients in both the groups were matched in terms of duration of diabetes & complications, BMI, HbA1c, creatinine clearance, S testosterone, FT4 TSH & prolactin levels. Stamp test & International Index of Erectile Function (IIEF-5) were recorded. EHGS scale was recorded by patients in their diary at -7 day, baseline & weekly till the end of study. Lispro was used in CSII and for bolus in MDI group along with glargine. All the study patients gave informed consent for not using drug or device for ED.

Results: Baseline HbA1c in CSII group was 11.2% and 8.7% at the end of study while in MDI group it was 11.1% and 9.1% respectively. In CSII group, 4 patients achieved Grade 4 erections, 5 achieved Grade 3 erections (response rate 39.1%) while in MDI,one achieved Grade 4 & three recorded Grade 3 response (17.3%).

Discussion: Usage of CSII is increasing in T2D for better control, though its effects on ED (a commonly associated complication) have not been studied. Diabetes, characterized by endothelial dysfunctions and defective insulin mediated vasodilation may explain ED. CSII by improving endothelial dysfunctions may explain improvement of vasodilatory functions of Carpora Cavernosa. Our study,first time observed that CSII is clinically significant & effective for improvement of ED.

Conclusion: 1)Short Term CSII therapy significantly improved ED as achievement of Grade 4 erections was four times higher in this group (4/23 patients) compared to MDI (1/23) as well as the overall response rate (Grade 3 and 4 erections) was 2.25 times higher in CSII group.2) Both the groups showed significant reduction in HbA1c, more marked in CSII.3)We recommend a large scale study for CSII usage in ED.

Abstract # 1313

INSIGHT INTO THE MOLECULAR MECHANISM OF ACTION OF BTI320, A NON -SYSTEMIC NOVEL DRUG TO CONTROL SERUM GLUCOSE LEVELS IN INDIVIDUALS WITH DIABETES.

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Diabetes/Prediabetes

Objective: Starch is an α(1-4)-linked polymer of glucose, which is enzymatically digested or hydrolyzed down to smaller polysaccharides (e.g. dextrin) and then onto smaller sugars like maltotriose and maltose, and eventually to glucose. The key enzymes responsible for the breakdown of starch are generally called α-glycosidases, with α-amylase being primary among them. BTI320 is a novel, non-systemic therapy that safely reduces postprandial glucose excursions with reduced side effects compared with acarbose. Acarbose is a natural microbial pseudo-tetrasaccharide that binds reversibly and competitively to the oligosaccharide binding site of α-glucosidases. BTI320 is composed of non-glucose-containing polysaccharides. It is essentially a composite of two modified galactomannans: GM-α (1-1) linked polymer, with α-amylase being primary among them. BTI320 is a novel, non-systemic therapy that safely reduces postprandial glucose excursions with reduced side effects compared with acarbose. Acarbose is a natural microbial pseudo-tetrasaccharide that binds reversibly and competitively to the oligosaccharide binding site of α-glucosidases. BTI320 is composed of non-glucose-containing polysaccharides. It is essentially a composite of two modified galactomannans: GM-α (1-1) linked polymer, and GM-β (1-4) linked polymer. We believe that BTI320 functions by targeting several polysaccharide hydrolyzing enzymes and that the active ingredient is GM-α. The present study was focused on assessing the molecular mechanism of action of BTI320 in relationship to α-amylase.
Methods: We used 1H and 13C nuclear magnetic resonance (NMR) spectroscopy to investigate interactions between BTI320 components, GM-α and GM-β, and the enzyme α-amylase, as well as the effects that GM-α and GM-β have on the rates of amylase-mediated starch hydrolysis towards glucose. The amylose iodine assay was also used to assess starch hydrolysis. Results are compared with those on acarbose.

Results: Chemical shift changes in NMR spectra of α-amylase demonstrate that GM-α interacts with the enzyme, possibly at or near its active site; GM-β appears to have no effect on α-amylase. GM-α and GM-β both interact with starch and apparently change the amylose structure, thus affecting how amylase hydrolyses the starch. Under certain conditions, the rate constants for starch (1 mg/ml) hydrolysis with α-amylase goes from 22.5 s⁻¹ in the absence of GM-α to 2.7 s⁻¹ in the presence of 4 mg/ml GM-α (p<0.005). The effect on the rate of starch hydrolysis with acarbose is similar, but at comparatively lower acarbose concentrations. In addition, comparison of NMR data on α-amylase with GM-α and acarbose suggest that GM-α binds at or near the same site on the enzyme as acarbose.

Discussion: GM-α acts as an inhibitor, possibly competitive, of α-amylase function, and appears to be the active ingredient in BTI320.

Conclusion: Our findings provide insight into how BTI320 may function in vivo and support the potential viability of BTI320 as an alternative to acarbose therapy for glycemic control.

Abstract # 1314

CANAGLIFLOZIN MONOTHERAPY PROVIDES REDUCTIONS IN BOTH A1C AND BODY WEIGHT IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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Diabetes/Prediabetes

Objective: Canagliflozin (CANA) is a sodium glucose co-transporter 2 inhibitor approved for the treatment of adults with type 2 diabetes mellitus (T2DM). CANA treatment has been associated with significant reductions in A1C and body weight across Phase 3 studies in a broad range of patients with T2DM. This analysis explored the impact of canagliflozin on A1C and body weight reduction in patients with T2DM enrolled in a placebo (PBO)-controlled study of CANA monotherapy.

Methods: In this 26-week, randomized, double-blind, PBO-controlled, Phase 3 study, patients with T2DM inadequately controlled with diet and exercise (N = 584; mean age, 55.4 y; A1C, 8.0%; body weight, 86.8 kg) received CANA 100 or 300 mg or PBO once daily. The distribution of change from baseline in A1C and body weight at Week 26 in individual patients was analyzed as a composite endpoint.

Results: At Week 26, significant reductions in A1C (−0.77%, −1.03%, and 0.14%) and body weight (−2.5 kg [−2.8%], −3.4 kg [−3.9%], and −0.5 kg [−0.6%]) were observed with CANA 100 and 300 mg compared with PBO, respectively. A greater proportion of patients treated with CANA 100 and 300 mg compared with PBO had reductions in both A1C and body weight (70.7%, 84.0%, and 27.5%, respectively); PBO-subtracted differences (95% confidence interval) were 43.2% (33.6, 52.8) and 56.5% (47.8, 65.2) with CANA 100 and 300 mg, respectively. CANA was generally well tolerated and was associated with an increased incidence of genital mycotic infections, urinary tract infections, and osmotic diuresis-related adverse events compared with PBO and a low incidence of hypoglycemia.

Conclusion: The majority of patients with T2DM treated with CANA monotherapy achieved reductions in both A1C and body weight at 26 weeks.

Abstract # 1315

IMPROVED INSULIN SENSITIVITY IN PRADER-WILLI SYNDROME ON CANAGLIFLOZIN

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Diabetes/Prediabetes

Objective: Is to report on a female patient with Prader-Wili syndrome (PWS) and her response to Canagliflozin (CAGZ) for insulin sensitivity, food intake, and glycemic control. PWS is associated with obesity and diabetes with insulin resistinace. Bariatric surgery and topiramate have been unsuccessful. Metformin improves the sensation of satiety in some (30%) of PWS patients. GLP-1 receptor agonists have been effective. CAGZ is a sodium glucose co-transporter inhibitor that lowers renal glucose threshold thereby increasing glucose excretion. Hyperglycemia causes increased insulin resistance and removal of glucose toxicity after CAGZ treatment would hypothetically improve insulin sensitivity.

Methods: Thirty-two-year old female with PWS (BMI
of 50 kg/m2 and HbA1c 11.2%, daily Lantus insulin and exenatide XR weekly) underwent 2-h OGTT before and at 2 wks and 8 wks after taking 300mg of CAGZ daily. C-Peptide and glucose levels were measured at time 0, 1h and 2 h after 75 g of glucose. In addition, she underwent a cereal breakfast meal test. The study counted the number of boxes of Raisin Brain cereal (120 kcal/box mixed with 4 oz of 2% milk). She consumed in 3 consecutive 10-min eating periods with a 5-min resting interval between eating periods.

**Results:** C-PEPTIDE (pM) and GLUCOSE (mM) data during 2-h OGTT (at time 0,1h and 2h) before and at 2 and 8 wk after CAGZ. At time 0 on OGTT, glucose declined from 11.7---9.7 (2 wk) --- 7.8 (8 wk). At time 1h on OGTT, glucose fell from 20---17.8 (2 wk) ---14.8 (8 wk). At time 2h on OGTT, glucose levels were reduced from 16.7---16.1 (2 wk)---11.7 (8 wk).

C-PEPTIDE levels at time 0 were 3.0---3.1 (2 wk) --- 2.4 (8 wk). C-PEPTIDE levels at 1h were 4.6---4.9 (2 wk)--- 4.2 (8 wk). C-PEPTIDE levels at 2 h were 5.3---5.8 (2 wk)--- 4.5 (8 wk). CALCULATED C-PEPTIDE areas under the curve (pMx min) were 525 before CAGZ and 538 (2 wk) and 453 (8 wk) after CAGZ. Fasting insulin fell from 39--18.9 (2 wk)---12.8 (8 wk).

**Discussion:** The data showed (1) Eight wks of CAGZ resulted in a fall in fasting glucose from 11.7 to 7.8 mM with concurrent lowering in HbA1C from 11.2 to 8.9% (2) Improved glycemic control without a change or increase in stimulated c-peptide and c-peptide area under curve(AUC) indicated increased insulin sensitivity after CAGZ (3) Before CAGZ she ate the same amount (380 kcal) in each of 3 eating periods(1140 kcal) without a decline in the 2nd and 3rd intervals implicating loss of normal sensory specific satiety in PWS and (4) after CAGZ, no was no decrease in food intake detected.

**Conclusion:** CAGZ restored insulin sensitivity in PWS that was mediated by removal of glucose toxicity.

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**Abstract # 1316**

**THE EFFECT OF TUMOR NECROSIS FACTOR-ALPHA ANTAGONISTS ON FASTING BLOOD GLUCOSE LEVELS AND HEMOGLOBIN A1C IN PATIENTS WITH RHEUMATOID ARTHRITIS**

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**Diabetes/Prediabetes**

**Objective:** This study aims to evaluate the effect of TNF-α antagonists on fasting blood glucose (FBG) levels and hemoglobin A1c (HbA1c) in patients with Rheumatoid Arthritis (RA).

**Methods:** FBG, HbA1c values and type of RA therapy were reviewed in 284 patients with RA. Patients were divided in four main groups depending on type of therapy (TNF-α inhibitors versus traditional DMARDs) and diabetes status. FBG and HbA1c values were compared before and after treatment with TNF-α antagonists in diabetic and non-diabetic patients respectively. Additionally same values were compared between patients on TNF-α antagonists therapy and patients on DMARD therapy only (control group). Incidence of diabetes was reviewed.

**Results:** No statistically significant difference was seen when comparing FBG and HbA1c values before and after anti-TNF-α therapy in non-diabetic and diabetic patients respectively. Likewise no difference was seen when comparing the same values between patients on TNF-α antagonists with control group (on traditional DMARDs). The difference in diabetes incidence between patients receiving DMARDS and patients receiving TNF-α antagonists was not statistically significant.

**Discussion:** TNF-α is an important mediator of insulin resistance in obesity and diabetes through its ability to decrease insulin responsiveness at the cellular level. The effect of TNF-α inhibitors on glucose homeostasis remains controversial. Few studies suggested a possible improvement in insulin resistance with TNF-α antagonists use in patients with RA. Few case reports describe
patients treated with TNF-α antagonists who developed hyperglycemia. Finally a pilot study in 9 RA and 9 healthy subjects, despite showing reduction of CRP and IL-6 levels with adalimumab therapy, did not show difference in insulin sensitivity. Despite the limitations of a retrospective study, the results obtained in our analysis suggest that TNF-α antagonist’s influence on glucose homeostasis is not substantial. Patients receiving anti-TNF-α therapy had similar fasting blood glucose values and HbA1c values as patients who were treated with traditional DMARDs. These values did not differ even when compared in the same patients before and after the initiation of therapy. Moreover the incidence rates of diabetes were similar between the two medication groups.

Conclusion: While some studies point at a possible improvement in insulin resistance with use of TNF-α antagonists others describe development of either hyperglycemia or hypoglycemia. On the contrary our study results suggest that TNF-α antagonist’s effect on glucose homeostasis in patients with RA is not significant.

Abstract # 1317

LIRAGLUTIDE AS ADDITIONAL TREATMENT TO INSULIN IN PATIENTS WITH TYPE 1 DIABETES MELLITUS : A RANDOMIZED CLINICAL TRIAL

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Diabetes/Prediabetes

Objective: We have previously demonstrated that the addition of liraglutide to insulin therapy in patients with type 1 diabetes (T1D) results in improvement in glycemic control, weight loss and a reduction in systolic blood pressure (SBP). We have now conducted the first prospectively randomized study investigating effects of liraglutide in T1D.

Methods: Seventy two patients (Placebo=18; liraglutide = 54) with T1D for at least one year, on insulin therapy and had no detectable c-peptide in plasma (mean BMI: 30±1; mean body weight: 184±5 lbs; mean HbA1c: 7.57±0.99%; mean age: 44±2 years; mean age of T1D diagnosis: 20±1 years) were randomized to receive placebo, 0.6, 1.2 and 1.8mg of liraglutide daily for 12 weeks.

Results: In 1.2 mg and 1.8 mg groups, mean change in average blood glucose was -10±2 and -10.0±1mg/dl, respectively, (p<0.0001 vs. placebo). In 1.2mg, HbA1c fell by 0.78% from 7.84±0.17% to 7.06±0.15% (p<0.0001, p<0.01 vs. placebo) and in 1.8mg group fell by 0.42% from 7.41±0.15 to 6.99±0.15 (p=0.001, p=0.39 vs placebo). Percent time spent between 70 to 160 mg/dl increased by 5±1% (p<0.05 vs placebo) and that between 160-400 mg/dl decreased by 7±1% (p<0.01 vs placebo) in 1.8mg group with no additional hypoglycemia. During the 12 weeks period, the average total daily dose of insulin in 1.2mg group fell by 12.4±3.9 units and by 10.0±2.3 units in 1.8mg group (p<0.05 vs baseline and placebo). There was a reduction in body weight (from 210±9 to 199±9lbs, p<0.001, in 1.2 group and from 182±10 to 171±10, p<0.0001, in 1.8mg group, and from 176±8 to 170±9, p<0.01 in 0.6 mg group) and in daily carbohydrate intake (171±17g vs. 127±18g; p<0.01 and 153±18g vs 115±16g, p<0.01 in 1.2mg and 1.8 mg groups, respectively) over 12 weeks. SBP in 1.8mg group fell by 9mmHg (120±2 to 111±3mmHg, p=0.01). CRP fell by 15±6 and 19±8% in 1.2 and 1.8mg groups, respectively (p<0.05). The quality of life in 1.2 mg and 1.8 mg groups significantly improved compared to baseline and placebo. There was no change in any of these indices in patients treated with the placebo and 0.6 mg liraglutide.

Discussion: This is the first randomized clinical trial to show that the addition of liraglutide 1.2 and 1.8mg to insulin significantly reduces HbA1c, mean blood glucose, body weight, carbohydrate intake and CRP in subjects with type 1 diabetes over a period of 12 weeks. Our findings have significant implications for the future treatment of T1D.

Conclusion: We conclude that the addition of 1.2mg and 1.8mg of liraglutide to insulin significantly reduced HbA1c, mean blood glucose, insulin dose, body weight, carbohydrate intake and CRP; significantly improved quality of life and reduced SBP in 1.8mg group in patients with type 1 diabetes.
Abstract # 1318

NEW INTERFACES FOR EGLYCEMIC MANAGEMENT SYSTEM SAVES NURSING TIME AND IMPROVES PATIENT OUTCOMES: TIME AND MOTION NURSING STUDY

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Diabetes/Prediabetes

Objective: Hospitalized patients with critical illness require intense monitoring and dose adjustments of intravenous (IV) insulin to maintain glycemic control. Numerous organizations (including the AACE, ADA, AATS, SCCM, and STS) support the need for blood glucose control to optimize patient outcomes, particularly related to post-operative infections. With CMS’ Surgical Care Improvement Project item SCIP-Inf-4, hospitals continue to place emphasis on glycemic management. IV Insulin therapy can place time stress on nursing staff managing glucose tests, IV drip rate changes, and documentation. We studied the effect a deeper integration (ADT, LIS, Cerner feeds) of eGlycemic Management System Glucommander (GM) by Glytec has on: nursing time, patient glycemic outcomes, and nursing satisfaction.

Methods: The study examined nursing time needed for patients requiring IV insulin treatment in critical care units of a 635-bed tertiary care hospital in rural west Tennessee. Group1 (G1) used a non-integrated IV insulin management system and Group2 (G2) used a fully integrated GM system. Time was measured between the two groups by an observer in the unit. Study outcomes included: time to start patients on IV infusion, time to adjust IV drip patients, hypo and hyperglycemic rates, and nursing satisfaction using GM.

Results: The time to start IV infusion for G1 was 4:07 compared to 52 seconds for G2. 12 nurses, caring for 8 unique patients were timed in both groups, totaling 118 Point of Care glucose test to IV change engagements. Group1 had an average time of 3:32/patient IV encounter compared with 35 seconds for G2. Glycemic control was maintained in both groups: Hypoglycemia rates: <40% = 0%, <70% = 1%, Hyperglycemia greater than 180 mg/d was <20%; all improvements with GM. Nursing satisfaction scores were 90-100% using GM. Nurses found GM integration with the EHR easy to locate, easy to use, less likely to have insulin/math errors, decreased physician calls, and decreased shift workload.

Discussion: Use of the deeper integrations reduced valuable nursing time in the Post Cardiac, Critical Care, and Surgical Care units. Up to 72 minutes/nurse/patient were reduced with GM for patients using IV insulin. An additional 3 minutes was saved per patient starting IV insulin drips. Nursing satisfaction was extremely high using the GM integration in their EHR. Finally glycemic control was well maintained, exceeding expectations for previous (at time of study) and current CMS measures without increasing rates of hypoglycemia.

Conclusion: Our results suggest using an interfaced GM product will save nursing time and improve satisfaction while treating patients with IV insulin and maintaining blood glucose control.

Abstract # 1319

RACIAL DISPARITIES IN GLYCEMIC CONTROL AND RELATED COMPICATIONS IN THE INPATIENT SETTING

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Diabetes/Prediabetes

Objective: Racial disparities have been reported in the prevalence of diabetes-related complications and mortality in minority groups. It is not known, however, if there are racial differences in inpatient complications and mortality. Accordingly, we aimed to compare the prevalence of inpatient hyperglycemia and diabetes (DM) and hospital outcomes among Black and White patients.

Methods: We analyzed blood glucose (BG) on admission and during hospitalization, history of DM, insulin treatment, and hospital complications in medicine and surgery patients admitted to two academic medical centres between 01/2012 and 12/2013. Hospital complications included a composite of pneumonia, myocardial infarction, respiratory failure, acute kidney injury (AKI) and bacteremia. Hyperglycemia was defined as a BG >140 mg/dl.

Results: Among 55,530 patients (male 49%, age 59±17 years), there were 29,983 Whites (57.3%), 21,483 Blacks (41.1%), and 1662 of other racial groups (3.4%). Of them,
55.4% were admitted to medicine and 44.6% to surgical services. Compared to Whites, Blacks had an increased prevalence of DM (38.3% vs 26.4%, p<0.0001) and hyperglycemia among non-DM patients (38% vs 32.6%, p<0.0001). Blacks with DM also had a higher admission BG (167±68 vs 162±58 mg/dl, p=0.0001) and higher 48-hour maximal BG (239±119 vs 221±94 mg/dl, p=0.0001) compared to Whites. Among DM and non-DM patients, Blacks had increased rates of complications compared to Whites (DM: 27.1 vs 24.1%, p<0.0001; non-DM: 21.3% vs 18.1%, p<0.0001). This difference persisted even when adjusted for admission BG, age, gender and BMI [DM: OR 1.14 (95% CI: 1.05 - 1.24); non-DM: OR 1.21 (95% CI: 1.13-1.29)]. Among DM patients, Blacks were more likely to receive insulin treatment than Whites (73% vs. 69.6% p=<0.0001); however, in non-DM with hyperglycemia, Blacks were less likely to receive insulin (38.4% vs 40.5%, p=0.0193). Mortality was similar among Whites and Blacks with DM (1.9% vs 2.9%, p=NS); however, mortality was higher in non-DM Blacks with hyperglycemia (2.1% vs 1.7%, p=0.02).

**Discussion:** In summary, Blacks have higher prevalence of diabetes, worse inpatient glycemic control, and increased rates of hospital complications compared to Whites.

**Conclusion:** Our investigation demonstrated the presence of racial disparities in hospitalized patient with hyperglycemia. Prospective studies are needed to determine underlying factors contributing to these differences.

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**Abstract # 1320**

**HIGH RATES OF UNCONTROLLED DIABETES OVERALL, AND OF RENAL IMPAIRMENT IN PATIENTS WITH DIABETES OVER AGE 50: RESULTS FROM A NATIONWIDE DATABASE**

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**Diabetes/Prediabetes**

**Objective:** To use a lab results database (Medivo Lab Value Exchange (LVX™), Medivo Inc., New York, NY) to assess population-level data for rates of uncontrolled diabetes and renal impairment in a nationwide population of patients with diabetes.

**Methods:** We analyzed the results of over 230,000 patients with diabetes in the LVX who had glycosylated hemoglobin (HbA1c) testing conducted between Jan 1, 2012 - Dec 31, 2013. We also analyzed the results of over 250,000 patients with diabetes who had their renal function tested during the same time period using serum creatinine and/or estimated glomerular filtration rate (eGFR).

**Results:** Of the 234,611 patients with diabetes tested for HbA1c levels, 57.8% had a result of 6.5% or more, and 41% had a result of 7% or more. Of the 253,587 patients with diabetes with renal function results, 24% had a result that indicated impaired renal function (eGFR < 60 or creatinine > 1.3 mg/dL). Our analysis also found that 5% of these patients had an eGFR < 30. The rate of patients with a low eGFR (<60) was higher than the rate of patients with a high serum creatinine (25.6% vs. 14.2%). There was a higher rate of lab test results signifying renal impairment in patients over age 50 compared with younger patients (29% vs. 5%). Analysis showed that, compared with patients aged 49 and younger, patients with diabetes ≥ 50 years were 4.5 times more likely to have an eGFR <60 (p<0.001) and 4.11 times more likely to have a serum creatinine >1.3 mg/dL (p<0.001).

**Discussion:** More than half of patients had uncontrolled diabetes, as indicated by HbA1c testing. This indicates that more aggressive treatment of diabetes is imperative. The much higher rate of renal impairment in patients with diabetes over 50 compared with younger patients as indicated by lab testing suggests that regular monitoring of renal function is key in older patients.

**Conclusion:** Research based on a nationwide laboratory results database can provide valuable insights on control rates and renal function in patients with diabetes.

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**Abstract # 1321**

**INSULINOMA IN A WOMAN WITH DEPRESSION WITHOUT HYPERINSULINEMIA IN FASTING TEST AND HYPOGLYCEMIA IN OGTT**

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**Hypoglycemia**

**Objective:** Describe the characteristics of a patient with insulinoma without hyperinsulinemia in Fasting Test.

**Methods:** We present clinical features and analysis of a patient with insulinoma characterized by hypoglycemia without hyperinsulinemia in fasting test and hypoglycemia in OGTT

**Case Presentation:** Female, 45 yo, father with pharyngeal cancer, mother with ovarian cancer. HTN, subclinical hypothyroidism receiving LT4 50 mcg/d. She take sertraline and alprazolam for depressive syndrome. Hospitalized in May 2012 by episodes of symptomatic hypoglycemia, fasting test was done
ABSTRACTS – Late Breaking

presenting asymptomatic hypoglycemia at 18 hours (G:59mg%) without hyperinsulinemia (insulin <2uUI/ml, PeptidoC:1.65ng/mL), the test was discontinued at 26 hours with symptoms (G:46mg%, Ins<2, PpC:1.6); she had symptomatic hypoglycemia at the 3° hour of OGTT (G:58mg%, Ins:14.1, PpC:4.1). Pro-insulin not available. CT pancreatic normal, was discharged with a diagnosis of reactive hypoglycemia in ambulatory monitoring. She continues with episodes of loss of consciousness without apparent cause, preceded by occipital headache of moderate intensity, dizziness and drooling at night with spontaneous remission; blurred vision, which refer to the intake of ‘sweet’. In Oct2012 she re-entered emergency with sensory disorder associated with hypoglycemia (27mg/dL). fasting glycemia: 50-62mg/dL, fasting insulin:7.39-9.82μUI/mL, fasting C-peptide:2.67-3.72ng/mL, Ab anti-insulin(-), SU in urine not available. TSH:0.66μUI/mL, FT4:1.36ng/dL, F:26ug/dL, PTHi:27pg/mL. MRI pancreatic inconclusive; Selective intra-arterial injection of the pancreatic arteries with calcium:Hypersecretion of insulin in the region of the splenic artery. She underwent distal pancreatectomy+splenectomy: tumor in the posterior-inferior of the pancreatic body 1x1cm . Histopathology: well-differentiated neuroendocrine carcinoma of low-grade pancreatic tail, unique, 16x12mm; Ki67 at 1%. She was discharged with fasting glucose:100-150mg/dL, Insulin:2.94μUI/mL and C-peptide:1.59ng/mL. Discussion: Insulinomas are rare, They generally present with fasting hypoglycemia (73%), however there are reports of hypoglycemia in functioning insulinomas with postprandial period (6%), as well as fasting and hypoglycemia in the postprandial period (21%).

Conclusion: Insulinomas are usually benign neuroendocrine tumors, approximately 20% of patients are diagnosed with psychiatric or neurological disorders.

Abstract # 1322

IMPROVING AMBULATORY HYPERTENSION DIAGNOSTICS: SERIAL BLOOD PRESSURE MEASUREMENTS

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Lipid/Cardiovascular Disorders/Hypertension

Objective: 1 in 3 Americans suffer from hypertension (HTN). NHANES data showed that control of the disease is inadequate. The reasons for poor control are numerous including poor access to health care and poor adherence to long-term treatment regimens. Additionally, it is estimated that approximately 20-25 percent of patients with stage 1 office HTN have “white coat” syndrome. One way to minimize this effect is to perform serial BP measurements. The aim of this study is to compare and trend serial automated BP readings to determine if initial readings are falsely elevated. The study was designed to introduce a practical technique that can be implemented in the office to significantly improve diagnostic accuracy and treatment of HTN.

Methods: A study population of 465 consecutive patients from Jan/2013 to Jan/2014 was evaluated in an outpatient clinic. All patients who tolerated BP monitoring were included. Prior to physician encounter, BP measurements were taken using a BpTRU BPM-200. Three serial recordings were taken subsequently over a time frame of 10 minutes. Data was compiled and comparisons were made using t-test analysis.

Results: A significant reduction in BP was demonstrated over serial measurements in the studied group. From first to second and third readings there was a mean systolic reduction of 3 mmHg (P = 0.026) and 5.9 mmHg (P = 0.00002), respectively. A significant reduction was seen when comparing second to third readings with a mean reduction of 2.8 mmHg systolic blood pressure (P = 0.038). A mean reduction in diastolic pressure was also demonstrated, significant when comparing initial to third measurements with a mean reduction of 2.8 mmHg (P = 0.0007). No difference in heart rates were noted. The average of all systolic blood pressures was 127.4 mmHg. This was most closely represented by the average of the second systolic reading, 127.3 mmHg.

Discussion: A significant reduction in blood pressure was demonstrated over serial measurements in the population group. The highest BP value was the first reading. BP values declined with subsequent measurements with the second reading the closet to the mean.

Conclusion: Serial blood pressure monitoring is a practical office-based technique that can help improve diagnostic accuracy and promote cost savings by avoiding unnecessary pharmacologic treatment in patients with white coat syndrome. The study shows that the second BP measurement most closely mirrored the average of all three readings. It can be argued that patients should receive at least two BP measurements in the office with the second reading being used to base clinical decision making and treatment.
Abstract # 1323

A CASE OF HYPERTRIGLYCERIDEMIA WITH APPENDICITIS

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Lipid/Cardiovascular Disorders/Hypertension

Objective: Severe hypertriglyceridemia (HTG) is an emergency which requires rapid institution of therapy to prevent pancreatitis. Here, we present a case of severe HTG with incipient pancreatitis who was managed with i.v. insulin, fibrates, n-3 fatty acid rich fish oil & a 48 hour fast.

Case Presentation: A 38 year old man was admitted for pain abdomen, later diagnosed to be due to sub-acute appendicitis. He is a known case of Type 2 diabetes since 7 years and has not been on treatment. He consumes 100-150 ml of alcohol daily and takes a fat rich diet. His family history is remarkable for cardiovascular events in his father and brother. On examination, he had a BMI of 31 and a waist circumference of 103 cm and tenderness in the right iliac fossa. Blood sample was markedly lipemic, and total cholesterol was 39 mg/dl, with HDL 55 mg/dl and triglycerides (Tg) of 4619 mg/dl. HbA1C was 9.96%. Serum lipase was 150 u/lt and amylase was 90 u/lt, suggesting impending pancreatitis. Blood glucose was 252 mg/dl and liver function tests showed elevated enzymes (SGPT-130 U/l, SGOT -146 U/l) and total bilirubin 2.34 mg/dl. Ultrasound abdomen showed Grade 1 fatty liver and fluid in right iliac fossa. He was shifted to MICU and started on i.v normal saline and insulin infusion, nil per oral, fenofibrate 160 mg and 4 grams fish oil per day. Heparin was not started in view of the possibility of patient being taken up for appendicectomy. The surgeon decided to adopt a conservative approach for appendicitis. At the end of 48 hours, RBS was 122mg/dl and total cholesterol was 240 mg/dl, HDL was 48 mg/dl, LDL was 114 mg/dl and Tg 392 mg/dl. He was discharged after 2 days, on glargine, glimepiride and metformin for diabetes and fenofibrate and n-3 fatty acids for HTG, along with advice about lifestyle modification. Appendicitis had subsided and the surgeon opted for conservative management.

Discussion: Tg >1000 mg/dl carries an exponential risk of pancreatitis. Acute treatments include insulin and or heparin infusions, plasmapheresis and purified apo CII infusion. Plasmapheresis and purified apo CII are unavailable in most centres. While heparin is used because it causes an acute release of lipoprotein lipase (LPL), use beyond 48-72 hours causes depletion of LPL and reduced peak LPL levels. On the contrary, insulin acts by increasing production of LPL and does not cause depletion of LPL. It is also useful for a rapid reduction of blood glucose, which can further help in reduction of Tg, mediated through Randle cycle.

Conclusion: In severe HTG, insulin infusion alone, without resorting to heparin infusion might be sufficient to reduce HTG, especially when the patient is put on a 48 hour fast under observation.

Abstract # 1324

EXENATIDE INDUCES AN INCREASE IN VASODILATORY MEDIATORS

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Lipid/Cardiovascular Disorders/Hypertension

Objective: Following our initial demonstration that treatment with exenatide results in reduction of systolic blood pressure (1), and the confirmatory data from subsequent studies on GLP-1 receptor agonists, we have now hypothesized that exenatide induces an increase in vasodilatory factors.

Methods: A series of 12 patients with type 2 diabetes on treatment with OHA and insulin were started on exenatide 5 µg b.i.d; the dose was increased to 10µg b.i.d a week later for a period of 12 weeks. Another 12 patients were treated with placebo. Blood samples were obtained prior to and at 2, 4 and 6 hours after the first injection and then at 12 weeks. Another 12 patients were treated with placebo. Blood samples were obtained prior to and at 2, 4 and 6 hours after the first injection and then at 12 weeks.

Results: Systolic blood pressure decreased from 134±6 to 127±5 mmHg (NS) and diastolic blood pressure was reduced from 82±2 to 77±3 mmHg (NS). Plasma concentrations of cGMP increased by 47±19% at 2hr of a single dose and by 72±21% and at 12 weeks of treatment with exenatide (p<0.05). There was a significant increases in concentrations of cAMP by 28±9% and ANP by 26±11% while those of b-NP did not alter at 12 weeks of exenatide. In addition, plasma concentrations of angiotensinogen were reduced by 19±5% at 2hr of a single dose and by 28±7% at 12 weeks of treatment with exenatide (p<0.05). Plasma concentrations of renin and angiotensin II also fell significantly by 18±7% and 25±8% (p<0.05), respectively following a single dose while it did not change at 12 weeks. In contrast, there was no change in plasma endothelin-1 concentrations.
**Discussion:** These findings have significant implications for the future treatment of patients with diabetes and or hypertension and in reducing cardiovascular risk.

**Conclusion:** These data demonstrate that exenatide treatment induces an increase in a series of vasodilators, cGMP, cAMP and ANP while suppressing the renin-angiotensin system as reflected in the plasma concentrations of renin, angiotensinogen and angiotensin II. These actions would contribute to the anti-hypertensive actions of exenatide in patients with type 2 diabetes on treatment with OHAs and insulin.

**Abstract # 1325**

**PHOSPHATURIC TUMOR IN A WOMAN OF 20 YEARS. FIRST CASE DOCUMENTED IN PERU**

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**Metabolic Bone Disease**

**Objective:** To present the first documented case of tumor-induced osteomalacia in our country.

**Methods:** Clinical, biochemical, and histopathological imaging of a patient with a mesenchymal phosphaturic tumor are presented.

**Case Presentation:** Female, 20 yo, nursing student, was on the track team at his school. 4 years disease duration, insidious onset and progressive course, characterized by decreased proximal lower limb strength, neuralgia and polyneuropathy later pathological fractures in metatarsals of both feet; 1 year before his admission decreased strength girdle and more difficult to roam. On examination: crutches, sitting up with support, poor dental status (caries and fractures), dorsal kyphosis, limited hip flexion by pain, hyporeflexia, proximal quadriplegia, atrophy and scapular deformity. Analysis: Serum iron and transferrin saturation low; HTLV1/HIV/ANA/ANCA(-); 25OHVitD:5.9ng/ml, 1.25OH2VitD (not available) and BMD: Z-score lumbar spine and femoral neck: -4.5 to -5.5, received monthly ergocalciferol and calcitriol bid with subsequent 25OHVitD of 56ng/ml, without clinical improvement. Corrected CaS:9.26mg/dL, PO4S:1.7mg/dL, CaU24h:149mg, UPO4: 1.157g/24h, CrS:0.38mg/dL, CrU 24h:25mg/dL,%TRP:98.6%, TmP/TFG: 2.38mg/dl (3.18-6.41 in women 16-25 years). FePO4:1016.2mg/24hs.

Rx pelvis: decrease in bone density and prevalence of bilateral femur ischio-pubic rami. PET-CT(-); Octreoscan with Tc99m: increased metabolic activity in right fibula head; Angio-MRI: Training 'balled' from 20x14mm in diameter at the level of the proximal right leg, part of the anterior tibial artery. In May 2013 underwent resection of well vascularized solid tumor. Histopathology: mesenchymal tumor type mixed connective tissue tumor. Subsequently improvement of phosphorus metabolism evident from the first postoperative week, a month of surgery: marked decrease in polymyalgia and moderate increase in muscle strength, patient remains in rehabilitation and physiotherapy.

**Discussion:** Phosphaturic mesenchymal tumors are usually benign, of mesenchymal origin call causing tumor-induced osteomalacia. Are usually small, are located in the soft tissues or axial skeleton, heterogeneous, difficult to locate histological characters sometimes in unusual places (distal ends of fingers) and therefore discovered late.

**Conclusion:** These tumors produce FGF-23, a hormone that induces hyperphosphaturia and produce a rare disease characterized by a clinical-pathological syndrome consisting of hypophosphatemia, low plasma levels of 1,25-OH2VitD and normal levels of serum calcium, PTH and 25OHVitD. After resection the biochemical improvement precedes clinical improvement which may take several weeks depending on weather condition.

**Abstract # 1326**

**RESPONSE TO DENOSUMAB THERAPY IN A PATIENT WITH PRIMARY HYPERPARATHYROIDISM-RELATED OSTEOPOROSIS**

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**Metabolic Bone Disease**

**Objective:** The only currently available treatment of PHPT (primary hyperparathyroidism)-related osteoporosis is surgery; anti-resorptive drugs are generally ineffective. PTH has both anabolic and catabolic effects in bone. The use of denosumab, a RANKL inhibitor, may shift PTH action to its anabolic effect, potentially improving bone mineral density (BMD).

**Case Presentation:** A 79-year old woman was being reevaluated for osteoporosis related to PHPT. She had refused surgical treatment. DXA scan showed progressive BMD loss in spite of annual infusion of zoledronic acid 5 mg. The report of a 2-year follow-up DXA scan showed progressive BMD loss in spite of annual infusion of zoledronic acid 5 mg. The report of a 2-year follow-up DXA scan showed progressive BMD loss. The report of a 2-year follow-up DXA scan showed progressive BMD loss in spite of annual infusion of zoledronic acid 5 mg. The report of a 2-year follow-up DXA scan showed progressive BMD loss. The report of a 2-year follow-up DXA scan showed progressive BMD loss in spite of annual infusion of zoledronic acid 5 mg.
Abstract # 1327

BRACHYDACTYLY-MENTAL RETARDATION SYNDROME DIAGNOSED IN ADULTHOOD

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Metabolic Bone Disease

Objective: To report a case of Brachydactyly-mental retardation syndrome (BDMR) diagnosed in an adult patient initially thought to have pseudopseudohypoparathyroidism (PPHP).

Case Presentation: A 45 year old female presented to Endocrinology clinic for evaluation of weight gain. During interview she reported polyphagia and polydipsia. Her past medical history was significant for hypertension, hyperlipidemia, obesity, sleep apnea, peripheral vascular disease and mood disorder. She also was diagnosed with mild mental retardation as a child. She experienced her menarche at the age of 13 and had had regular periods. She was living in a group home and was not married or had kids. Patient’s height was 4 feet 11 inches and she had a BMI of 40. Physical examination was significant for short stature, facial dysmorphism with prominent forehead, upslanted eyes, flat nasal bridge and a thin upper lip. Extremity examination revealed short 4th and 5th metacarpal and metatarsal bones bilaterally. Secondary causes of obesity and Albright hereditary osteodystrophy (OHA) were considered in differential diagnosis. On blood testing patient’s calcium, phosphate and PTH levels were normal. Cortisol, TSH and free T4, FSH and LH levels were all normal. Patient was sent for genetic testing with a presumed diagnosis of PPHP. Karyotype test showed terminal deletion of the long q-arm of one chromosome 2 in all analyzed cells-46, XX, del(2)(q37.1), consistent with BDMR.

Discussion: BDMR also known as AHO-like syndrome and 2q37 deletion syndrome is a rare disease that clinically resembles AHO and PPHP. BDMR has significant variability in clinical presentation, but almost all patients have some degree of mental retardation, facial dysmorphism, short stature and obesity. Brachymetaphalangism has been reported in approximately 50% of cases. Congenital heart anomalies are present in around 20% of patients. The main candidate gene for the brachymetaphalangism seen in BDMR is histone deacetylase 4 (HDAC4) on chromosome 2q37.2. BDMR presents with a phenotype resembling the physical anomalies found in AHO but has a normal calcium metabolism therefore can be easily misdiagnosed as PPHP. Diagnosis of BDMR is based on the detection of the deletion on the long arm of chromosome 2 and can usually be made from the karyotype. Neurodevelopmental and behavioral evaluations, skeletal survey, endocrine evaluation, and an echocardiogram are indicated.

Conclusion: BDMR clinically resembles AHO and can be easily misdiagnosed as PPHP. Most cases are recognized during childhood, however diagnosis should be considered in adults with AHO like phenotype and normal calcium metabolism.
Abstract # 1328

WHY OBESE PATIENTS MAY HAVE NORMAL THYROID TESTS DESPITE ‘THYROID SYMPTOMS’

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Obesity

Objective: Every endocrinologist is faced with referred patients who have “thyroid symptoms” with normal or low TSH. Some hypothyroid patients continue to be dissatisfied with being overweight despite adequate replacement. We hypothesized these symptoms relate to hypothalamic dysfunction but there are no studies documenting how common is Hypothalamic Obesity Disorder in patients referred for thyroid disease?

Methods: We prospectively tabulated complaints of 50 consecutive patients referred for evaluation of ‘thyroid symptoms”. Complaints were extracted from the patient ROS in the EHR. The prevalence of the seven groups of hypothalamic symptoms were quantified. The diagnosis of hypothalamic dysfunction was likely if symptoms from 3 different systems were present, definite if present from four or more. Thyroid disease was diagnosed by standards thyroid tests.

Results: Female were 38(%76), age (18-68), the majority are 20-60. Thyroid tests are not presented here for space. Reasons for referral were often proven not to be present: of 50 patients referred 14 were proven to have thyroid disorder, the rest 36/50 (%72) had normal thyroid. Frequency of hypothalamic symptoms: 1) Energy/fatigue %76; 2) Temperature dysreg %68; 3) Appetite/weight change % 88; 4) sleep/diurnal change %70; 5) Pain, spasm/ fibromyalgia %72; 6) Mood disorders %80; 7) Libido/ Reproduction %38, 8) sympathetic/ parasympathetic %64. More than 12/18 Trigger point’s tenderness was documented in %68. Frequency of Hypothalamic Dysfunction diagnosis in the total 50 referred population was either: 1) Likely (3 systems) 11/50 (% 22); or 2) definite (4 or more systems) 34/50 (%68).

Discussion: Patients who insist they have “thyroid disease“ causing their weight problem are frequent. Some try thyroid medications, yet they feel worse and don’t lose weight. Some has been named malingering, histrionic, borderline personality or “uncharacterized disorders’ like “Wilson’s Temperature syndrome” “yeast /monilia syndrome”, ”adrenal exhaustion/depletion” and others, when hypothalamic dysfunction would explain most. An important physical finding is the presence of trigger points tenderness in%68. These patients would only benefit from therapy to their Hypothalamic Dysfunction.

Conclusion: Hypothalamic Obesity Disorder, an easy diagnosis to make on clinical ground, is the most common disease (%68) documented in patients with “thyroid symptoms” and normal thyroid tests. Treating the thyroid in these patients often fails. They need specific therapy for Hypothalamic Dysfunction based on stress/reward/ behavioral therapy and dual pharmacologic therapy for the dual hypothalamic nuclei pathways affecting Satiety/ Hunger and Weight/Metabolism.

Abstract # 1329

SUCCESSFUL ALGORITHM TO TREAT HYPOTHALAMIC OBESITY DISORDER

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Obesity

Objective: Lifestyle change and medications used separately often fails since the many pathways effecting satiety, energy, and weight need combination therapy. Since Hypothalamic Obesity is a symptom of Hypothalamic Dysfunction it requires treating all disturbed hypothalamic functions to succeed.

Methods: 108 consecutive obese patients (Average weight: 226 Lbs, BMI: 38),were included. Hypothalamic Obesity Treatment Algorithm includes: 1) Obesity standards of care recommended by AACE and SBP(2013). 2) A prescription for daily exercise, East Mediterranean diet, treatment for anxiety, depression, OCD. The diagnosis is considered ‘likely’ if symptoms from 3 different hypothalamic systems were present,’definite’ if four or more. Pharmacologic treatment was started with Fluoxetine/Spironolactone followed by Topiramate/Phentermine, anxiety with Buspiron, sleep/ fibromyalgia with Trazodone, OCD with high dose Fluoxetine or Bupropion, hypogonadism in males with testosterone, and hyperprolactinemia with Bromocriptine, as well as, GERD, constipation. Measurements were made before and after intervention.

Results: Hypothalamic Dysfunction diagnosis: 1)’Likely’ %30; 2) ‘definite’ %55.4. In 12 weeks: Mean Weight loss was 13 Lbs (range 6-24), total BMI drop was 2. Patients lost 1.1 Lbs/week. Blood pressure was within target in %88; %60 of patients lost more than %5 of weight, %28 lost more than %7, and %12 lost more than %10. Combinations has variable weight loss effect: Topiramate/ Phentermine: 14.8 lbs, Fluoxetine/ spironolactone: 11.2 Lbs, and all four combined 17.6 Lbs. Diabetic patients on GLP1 lost 12 Lbs. 4 patients who were treated with Cymbalta for fibromyalgia gained 4 Lbs.
Discussion: Since the majority of clinic obesity is hypothalamic in nature, many other symptoms need to be addressed as well to treat the etiology as well as the weight problem itself. Treatment for anxiety, depression, compulsive behavior, sleep disorders, and fibromyalgia, all of which often cause the behavioral pattern leading to obesity, by weight neutral medications is intuitive but rarely reported as we did in this study.

Conclusion: Without treating hypothalamic dysfunction in its entire spectrum (anxiety, depression, OCD, sleep, and fibromyalgia) by stress/behavior/reward management and appropriate dual pharmacology the treatment of obesity often fails. Synergy between Spironolactone and Fluoxetine is very promising, inexpensive safe combination for weight reduction, which could be a useful step before using GLP1 or topiramate/phentermine. There is a need for large scale efficacy comparative study for future practice guidelines.

Abstract # 1330

SERUM 25-HYDROXYVITAMIN D LEVELS AFTER SUPPLEMENTATION WITH VITAMIN D2 OR VITAMIN D3 IN AN OBESE POPULATION.

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Obesity

Objective: People worldwide suffer from vitamin D deficiency, which may result from limited exposure to sunlight, use of sunscreen, low consumption of food containing ergocalciferol, and malabsorption syndrome. The vitamin D receptors (VDR) and the 1α-hydroxylase enzyme, which catalyzes the conversion of calcidiol to calcitriol, are found in more than 40 human cell types, indicating its potential role in the regulation of numerous metabolic processes. According to recent data, there may be a connection between vitamin D levels and metabolic diseases such as obesity, diabetes mellitus type 2, hypertension, and dyslipidemia. Although the mechanisms are still unclear, vitamin D deficiency is associated with a greater risk of these pathological conditions. Furthermore, an increased body fat and obesity is associated with low circulating 25(OH)D levels. The presence of VDR in adipocytes suggests that vitamin D plays a role in lipogenesis and lipolysis regulation. It was shown that the active form 1,25(OH)2D - could regulate adipocyte death and decrease fat mass. The purpose of this study is to determine if different forms of vitamin D (ergocalciferol and cholecalciferol) work equally well in treating vitamin D deficiency in obese patients.

Methods: 40 consecutive patients with vit D < 30 ng/ml were recruited from an obesity clinic. All patients received similar treatment including dietary counseling and exercise interventions. Patients were then randomized to either 50,000 IU vitamin D2 once/week vs. 50,000 IU vitamin D3 once/week. Data was analyzed after 12 weeks of treatment.

Results: Vitamin D was replaced successfully in all patients. The D2 and D3 group saw increased from a vitamin D mean of 18.96 ng/dl to 34.1 ng/dl (P<0.01) and 20.14 ng/dl to 56.82 ng/dl (P<0.01), respectively. Increased Ca2+/ PO4 and decreased PTH were noticed more in D2 group, than D3 group. Decreased cholesterol and LDL levels were noticed after D3 treatment, but not in D2 group. No significant changes were seen in other tests. (Cr, BUN, hsCRP, HDL)

Discussion: Prior to the initiation of this study there has been no published studies that addressed supplementation of vitamin D in an obese population. Due to the close connection between obesity and vitamin D it is important to investigate different forms of supplementation in this population. We found that supplementing with Vitamin D3 yielded higher 25-OH Vit D levels compared to Vitamin D2.

Conclusion: Vitamin D3 50,000 IU weekly is an effective and plausible replacement method for vitamin D deficiency in an obese population.

Abstract # 1331

ABSTRACT WITHDRAWN
Abstract # 1332

ASSOCIATION OF ANTHROPOMETRIC AND BIOCHEMICAL MARKERS OF OBESITY IN INDIAN ADOLESCENTS


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Obesity

Objective: To study association of body mass index (BMI), waist height ratio (WHtR) and country specific waist circumference (WC) charts with other anthropometric markers and biochemical markers of insulin resistance. Methods: 96 children in the 11-16 yr age group had multiple anthropometric markers like BMI, WC, WHtR, TSFT and fat percentage measured and were also evaluated biochemically for serum insulin, HOMA-IR, hs-CRP, fasting lipid profile and adiponectin levels, after a 10 hour overnight fast.

Case Presentation: BMI, increased WC(>75th centile for age) and higher WHtR (>0.5) showed significant associations with each other and with other anthropometric markers like fat percentage and TSFT; and the biochemical markers like serum fasting insulin, HOMA-IR, hs-CRP and triglyceride levels (p< 0.05). Surprisingly, adiponectin did not show a significant association with any of the biochemical or anthropometric markers.40 children out of the total 96 had a BMI< 85th centile, among whom, 8 children had an elevated WHtR (>0.5) and greater incidence of increased fat percentage (p< 0.05). 11 children in this group of 40 had greater WC (>75th centile for age) and had statistically increased fat percentage and hs-CRP levels (p< 0.05).

Discussion: Pediatric obesity is a stark reality in India today, but tools to quantify progression to Type 2 diabetes are still limited. Community based studies especially in resource limited settings need an easily measurable and reproducible anthropometric marker to quantify this risk, which also correlates well with biochemical markers of IR. In the present study, WHtR >0.5 showed good correlation with other anthropometric markers and also biochemical markers. It has an added advantage of a single value (>0.5) utilizable as a cutoff across all ages, which makes it useful in population studies. WC also showed similar associations and has also validated percentile charts obtained from urban schoolchildren, but needs the use of age based percentile charts, which limits utility in population studies. Among biochemical markers, HOMA-IR, fasting insulin and hs-CRP underlined their utility as markers of IR. Total adiponectin levels did not show significant associations, once again bringing into sharp focus the debate about high molecular weight vs total adiponectin as an IR marker.

Conclusion: Biochemical markers of IR and cardiovascular risk had significant association with anthropometric markers. WHtR and WC performed as well as BMI in assessing obesity & association with risk of type 2 diabetes and cardiovascular risk, and further, indicated future metabolic and cardiovascular risk among the children with normal BMI.

Abstract # 1333

TWO SIBLINGS WITH OBESITY

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Obesity

Objective: We present a case report of two young siblings with obesity and hyperphagia since 1 year of age, who were identified to have monogenic obesity on endocrine and genetic testing. Their parents were heterozygous for same mutation.

Case Presentation: A 6 year 9 month old boy was referred for evaluation of obesity and hyperphagia noticed since 8 months of age. History was not suggestive of any endocrine disorder. No history of consanguinity. Birth and development were normal. No genital ambiguity or micropenis were noticed at birth. His daily calorie intake is 2,500 Kcal. Was advised 1300 Kcal, but could not comply. On examination, height is 118 cm (50th centile) and weight is 60 kg (>97th centile). BMI is 28 kg/m2 (>95th centile). Both testes descended with volume of 2 ml. Penile length was 4.6 cm, which corresponds to -2 SD for age. Acanthosis nigricans is present and skin and hair colour are normal. His younger sister is 5 years 3 months old, with obesity and hyperphagia noticed since 1 year of age. Birth and development are normal. Dietary intake is 2,300 Kcal. On examination, height is 102cm...
(25th centile) and weight is 28 kg (>97th centile). BMI is 26.9 kg/m2 (>95th centile). Acanthosis nigricans is present. Thyroid function tests, serum cortisol and blood sugars were normal and triglycerides and total cholesterol were elevated in both siblings. Bone ages were age appropriate. Genetic analysis revealed a mutation in Mineralocorticoid Receptor 4 (MC4R) at locus L300P which is present in homozygous form in the two children and in heterozygous form in both their parents. Father was obese with a BMI of 31, while the mother had a BMI of 22. Parents were unrelated.

Discussion: MC4R has a central role in weight regulation. Mutations that lead to partial or complete dysfunction of MC4R lead to a clinical phenotype with lack of satiety, extreme continuous hyperphagia, a decline in energy utilization, and consequently, severe early-onset obesity. It is the commonest cause of monogenic obesity. Heterozygous mutations are common and homozygous mutations relatively rare. The prevalence of carriage of heterozygous MC4R mutations is estimated to be as high as 0.5–6% in obese individuals. While there is no treatment at present, future development of small molecule MC4R agonists holds hope for patients with this disorder.

Conclusion: Not all obesity in children is exogenous. Monogenic obesity should be considered, especially when children present with hyperphagia and morbid obesity early in life and kindred are affected.

Abstract # 1334

PREVALENCE OF METABOLIC SYNDROME AMONG MUSLIM CLERGY OF SRI LANKA

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Obesity

Objective: To study the prevalence metabolic syndrome among the Muslim clergy of Central Sri Lanka and compare it with the national data.

Methods: The study was conducted at Care Endocrine Research Centre, Akurana, Sri Lanka. A total of 130 clergy from Central Province of Sri Lanka participated in the study. All the participants were subjected to the procedures like anthropometric measurements, biochemical analysis for fasting blood glucose, fasting serum triglycerides, total cholesterol, high density lipoprotein, low density lipoprotein and very low density lipoprotein. All of their personal details and past medical history were obtained through interviewer administered questionnaire.

Results: Mean age of study sample was 41.99±13.8 years. Prevalence of metabolic syndrome was 29.2%. Subjects with metabolic syndrome (MS) and non metabolic syndrome (non MS) show a significant difference in the mean values of weight (79.1±8.4 vs 63.5±10.3), waist circumference (101.0±7.4 vs 85.0±10.3), BMI (28.8±2.7 vs 23.7±3.8), systolic (136.1 ±16.9 vs 124.4 ±19.9) and diastolic (84.8±12.2 vs 73.3±10.7) pressure, fasting blood glucose (126.5±37.1 vs 105.1 ±36.7), triglycerides (236.3±133.5 vs 170.4±82.8), HDL (45.2±8.6 vs 48.8 ±8.9) and VLDL (42.8±13.6 vs 33.0±14.1) cholesterol at 95% confidential level (P < 0.05). Mean fasting blood glucose of the sample was 111.3 ±38.0 mg/dl and prevalence of diabetes (169.24±39.83) and IFG (118.15±4.33) were 25.4% and 10.0 % respectively. According to 2003 WHO criteria, prevalence of hypertension was 23.8% while according to the current criteria its 28.5%. Moreover 48.5% of Muslim clergy were general population of Sri Lanka. Body weight misperception is common among Muslim clergy in Sri Lanka.6.9%, 26.9% and 13.8% were misperceived themselves as underweight, normal and overweight respectively. Only 1.5% have perceived themselves as obese, while 49.2% are actually obese.

Discussion: Our finding suggest that the Muslim Clergy of Central Sri Lanka, compared to general population of Sri Lanka are having higher prevalence of metabolic syndrome and other cardiovascular risk factors. They are more obese. This phenomena needs to be studied in clergy of other religions as well.

Conclusion: According to the survey of multiple risk factors, Muslim clergy of Central Province of Sri Lanka are having significantly higher prevalence of metabolic syndrome and diabetes, compared to the general population of Sri Lanka. Perception of obesity among the clergy was poor.
Abstract # 1335

MULTIFACTORIAL HYPERCALCEMIA

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Objective: To highlight the possibility of concomitant Parathyroid hormone (PTH) and non Parathyroid hormone induced hypercalcemia in an 82 year old male patient.

Case Presentation: An 82 year old male with diabetes and hypertension found to have corrected plasma calcium of 14.12 mg/dl with iPTH of 930 pg/ml (nl 24-85 pg/ml). Sestamibi scan was positive for a 5 cm left parathyroid adenoma and patient underwent left parathyroidectomy with intraoperative PTH decrease from 930 to 28.2 pg/ml. Pathology was benign. Post-op calcium levels remained in the upper range of normal and serum protein electrophoresis, sent preoperatively by the primary team, showed monoclonal IgG kappa spike in the alpha region. Patient was referred to Hematology and was diagnosed with monoclonal gammopathy of uncertain significance. During follow up, patient’s calcium remained normal but PTH levels increased again (87 – 96pg/ml range). 24 hr urine calcium was low (25 mg/24 hrs), with calcium/creatinine clearance ratio of 0.0019 on calcium and vitamin D supplements. His concomitant vitamin D 25-OH level was 32ng/ml and 1-25 hydroxyvitamin D 69 pg/ml. Patient’s sister is recently diagnosed with elevated PTH levels, undergoing work-up.

Discussion: The presentation of our patient had several atypical features. Patient had a very large adenoma with very elevated PTH levels. Postoperatively calcium level went down only to 9.8 mg/dl, when one would expect lower calcium levels, thus raising suspicion for additional causes of hypercalcemia. Patient was indeed subsequently diagnosed with MGUS. Several case reports, support that multiple myeloma may coexist with PHPT and in a prospective study of 101 patients with PHPT, monoclonal gammopathy was detected in 10% of the patients. This case underlines the importance of recognizing that PHPT might mask concomitant non PTH induced hypercalcemia. Post-op PTH levels were trending up, while patient remained normocalcemic. Differential diagnosis included coexistent Familial Hypercalcemic Hypercalciuria (FHH), recurrent hyperparathyroidism or rarely reported parathyromatosis. His 24 hr urine calcium was surprisingly low, and in a setting of normal vitamin D level and calcium supplementation, is very suggestive of FHH, which at this point seems to be the only explanation of his elevated PTH levels.

Conclusion: It is important to recognize that hypercalcemia can be multifactorial. Primary hyperparathyroidism not uncommonly coexists with monoclonal gammopathies. In our patient, an underlying FHH seems to be an underlying diagnosis to both, making this case a real eye opener to the challenges of diagnosis and management of multifactorial hypercalcemia.

Abstract # 1336

RELATIONSHIP OF CLINICAL RESPONSE & ADVERSE EVENTS TO DOSE OF MIFEPRISTONE (KORLYM®) IN THE SEISMIC STUDY: HIGHER DOSES ASSOCIATED WITH INITIAL CLINICAL RESPONSE. HIGHER DOSES NOT ASSOCIATED WITH INCREASED FREQUENCY OF SERIOUS ADVERSE EVENTS (SAES) OR 4 M

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Objective: In SEISMIC, a 24-week, multicenter open label study of mifepristone (MIFE) in Cushing’s syndrome (CS) patients (pts), investigators (PIs) were allowed to titrate beyond the starting dose (300mg/d). PIs could titrate up to 1200mg/d in 300mg increments at Day14, Wk6, & Wk10 at PIs’ discretion based on assessments of tolerability & clinical benefit. Recent prescription data indicates that many physicians are not titrating beyond 300mg/d, potentially limiting pts’ clinical response. This analysis assesses the relationships between MIFE dose & the safety & response profile from SEISMIC.

Methods: The frequency of SAEs & 4 most common AEs (headache, fatigue, nausea, & hypokalemia) were tabulated using descriptive statistics. The rate of SAEs were assessed by MIFE dose for all pts(N=50). The rate of the 4 most common AEs were compared between wk1-10 (pts were titrating) & after wk10 (pts on more stable doses) in study completers(N=34 completers).

Global clinical response (GCR), a key study parameter of efficacy, was assessed by an independent panel based on 8 clinical categories (glucose, BP, lipids, body composition/bone, clinical appearance, strength, psych/cognitive, & QOL). The dose of initial GCR response (n=40 GCR responders) were analyzed.

Results: 90% of pts received a daily dose of ≥600mg (N=45/50), 68% ≥900mg (N=34/50), 44% 1200mg (N=22/50). 85% of GCR responders (N=34/40 responders)
had their initial clinical response at a dose $\geq 600\text{mg}$, & 35% at $\geq 900\text{mg}$ (N=14/40). The total number of SAEs was 26; 10 occurring at 300mg, 8 at 600mg, 3 at 900mg, 3 at 1200mg, & 2 while off drug. When accounting for the number of pts that ever reached a given dose, the pts’ observed SAE frequency was similar across doses (10/16/15/14 % of pts at 300mg/600mg/900mg/1200mg, respectively). The 4 most common AEs decreased after wk10 compared to wk1-10 (study completers, N=34); % of pts with headache, fatigue, nausea and hypokalemia in wk1-10 was 44, 41, 41, and 24, respectively, and 24, 24, 21, and 18 after wk10, respectively. The mean dose was 588mg/d during wk1-10 & 895mg/d after wk10. 16 pts withdrew from the study, including 7 due to AEs. The mean dose of pts who did not complete the study was 551mg/d compared to 773mg/d in N=34 completers.

Discussion: Higher doses of MIFE were not associated with increases in SAEs or the 4 most common AEs. The 4 most common AEs decreased in frequency after wk10. Higher incidence of adverse events was not observed at higher doses. Initial clinical response was more prevalent in pts on $\geq 600\text{mg/d}$.

Conclusion: A large majority of pts from the SEISMIC trial titrated above the starting dose of 300mg/d MIFE. A higher incidence of adverse events was not observed at higher doses. Initial clinical response was more prevalent in pts on $\geq 600\text{mg/d}$.

Abstract # 1337

RARE CASE OF KLINEFELTERS SYNDROME WITH 48XXYY

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Other

Objective: Describe Klinefelters Syndrome with 48XXYY

Case Presentation: A 28 year old male with klinefelter syndrome(KF) diagnosed during learning disability evaluation at age of 13 presented for follow up. He was on testosterone supplementation and his examination revealed tall stature, atrophic testis, gynecomastia. Intriguingly his medical history for his young age was very complicated. He had diagnosis of childhood asthma that was poorly controlled until the initiation of immunoglobulin therapy for common variable immunodeficiency. He had cerebrovascular accident at age 14, atrial septal defect repaired at age of 20, pulmonary embolism managed with IVC filter and anticoagulation. Imaging of his cervical spine showed loss of cervical lordosis. His diabetes was uncontrolled due to his non compliance and learning difficulties to understand diabetes and its management. Since his records of KF diagnosis was unavailable and he had multiple complex medical problems lab testing to confirm diagnosis karyotyping was done. Karyotyping revealed 48,XXYY that explained many of his diverse medical problems which are typical of Klinefelters 48, XXYY syndrome. Despite his multiple medical problems and learning difficulties he works as high school basketball manager.

Discussion: KF is the most common congenital cause of primary hypogonadism, occurring in 1 : 1000 males. While KF results from extra X chromosome in 80% of cases, in the remaining cases this results from numeric sex chromosome abnormalities (48,XXXY, 48,XXYY,49,XXXXY) or 46,XY/47,XXY mosaicism, or structurally abnormal sex chromosomes greater and lesser numbers of X chromosomes. The physical features of KF XXYY are similar to KF 47,XXY but with more pronounced phenotypic abnormalities including mild craniofacial dysmorphism, skeletal anomalies such as radioulnar synostosis and clinodactyly, lower cognitive function resulting in developmental delays, congenital cardiac anomalies and asthma manifest at younger age. Adulthood is characterized by hypogonadism, deep vein thrombosis, intention tremor, and type II diabetes. Their behavior abnormalities include hyperactivity, attention problems, impulsivity, aggression, mood instability, ‘autistic-like’ behaviors, and poor social function. These behavioural issues make management of multiple medical problems such as diabetes and anticoagulation very difficult.

Conclusion: KF 48, XXYY is a severe form of KF with additional clinical, neurodevelopmental & behavioural manifestation that make the management of KF challenging. Care of these individuals requires a multidiciplinary approach.

Abstract # 1338

NON-SURGICAL TREATMENT FOR PARATHYROID ADENOMA

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Other

Objective: Currently endocrinologists can accurately localize the parathyroid adenoma with ultrasound guided biopsy of possible parathyroid adenoma and measure PTH level on the aspirate. Percutaneous ethanol injection (PEI) is safely used to treat local recurrence of papillary thyroid carcinoma with no complications. In addition, there is a publication from the Mayo Clinic (AJR:191, Dec 2008) of 41 parathyroid hyperplasia in 22 patients treated with PEI and treatment was proven safe and effective. From reviewing
all publications about non-surgical parathyroid adenoma treatment, there were two published abstracts from Europe (Eur Radiol1998;8(9):1565-9) (Clin Endo (Oxf). 2008 Oct;69(4):542-8). The response rate was around 60%. No publication from the United States was found.

**Methods:** Patients with parathyroid adenoma localized using the ultrasound and confirmed with biopsy that showed elevated PTH level on the aspirate were offered PEI if they refused surgery. Topical analgesic cream was used to numb the injection site. Using 10cc syringe with 25 gauge needle we injected between 0.5 to 3 ml of dehydrated ethanol inside the parathyroid adenoma in multiple areas. The procedure preparation and actual technique is the same as diagnostic FNA except for injecting alcohol instead of tissue aspiration. Calcium and PTH level were measured at 1 and 4 weeks after the PEI.

**Results:** 9 patients with parathyroid adenomas were treated with PEI. The calcium level normalized after 1 week in 8 patients. At 4 weeks, 6 of 9 patients (67%) had normal calcium. 8 months of data is available on the first patient. The original size of parathyroid adenoma was 23mm, calcium 11.6 and PTH 108 pre-ablation. The ultrasound was repeated 8 months after treatment and the adenoma was not visible, calcium 9.5, and PTH 65. Patients 1 and 2 had pain that started the night of procedure and continued for 3 days. The pain was described as a tender lump in the injection site.

**Discussion:** More patients need to be treated to have a better understanding of PEI safety profile and efficacy. PEI is a simple, local treatment that should be evaluated to replace surgery as first line. If the first treatment fails to normalize calcium it could be repeated or go for surgery. Another advantage of PEI (if it is simple, low cost, and low risk) is that we can treat any patient where the parathyroid adenoma can be localized and decrease the need for years of follow up and monitoring and the constant discussion about every symptom patient has if it is related to the hyperparathyroidism or not.

**Conclusion:** Since localization of a parathyroid adenoma is possible and accurate, PEI is another treatment option that is a simple, low cost, low risk outpatient procedure.

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**Abstract # 1339**

**1,25 OH VITAMIN D MEDIATED HYPERCALCEMIA IN A PATIENT WITH MALIGNANCY WITHOUT LYMPHOMA**

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**Other**

**Objective:** Describe an unusual case of calcitriol mediated hypercalcemia in a patient with colonic adenocarcinoma and mesothelioma.

**Case Presentation:** A 62-year-old male presented with constipation and left abdominal pain of 1-month duration. Abdominal CT revealed rectal thickening concerning for cancer with metastatic lymphadenopathy, metastatic peritoneal lesion and a 20cm liver mass. Biopsy of sigmoid colon showed invasive moderately differentiated adenocarcinoma. Four months prior, his labs were calcium 9.8mg/dL, albumin 4.6gm/dL, eGFR 96 with normal BUN and creatinine. At presentation his labs were Ca 10.5, alb 4.2, BUN 29, creatinine 1.59 and eGFR 46. During subsequent hospitalization for right leg cellulitis, he had Ca 11.9 and ionized calcium 6.45 with low albumin 3.4. He developed sepsis and due to concern for liver abscess the liver mass was biopsied. Pathology showed high grade malignant neoplasm consistent with epithelioid mesothelioma. Septicemia and renal failure requiring dialysis complicated his course. Hypercalcemia workup showed no evidence of osteolytic lesions on imaging, normal 25 OH vitamin D 46, low intact PTH 12, normal PTHrP at 1.1 pmol/L and absent monoclonal proteins on serum and urine protein electrophoresis. Calcitriol was high on two samples at 95 and 77pg/mL. The patient’s calcium gradually improved with administration of pamidronate to 7.3 with an albumin of 1.9 and a corrected calcium 8.9. Despite being on 3 vasopressors, antibiotics and continuous renal replacement therapy, his condition deteriorated and patient died on comfort measures.

**Discussion:** Hypercalcemia resulting from calcium homeostasis imbalance is seen in one third of malignancies and is a poor prognostic factor. Solid malignancies cause hypercalcemia from high PTHrP and less commonly from osteolytic cytokines. Excessive calcitriol seen mostly in lymphoma causes hypercalcemia by 1-hydroxylation of 25 OH vitamin D. This mechanism has also been reported in dysgerminoma, myofibroblastic tumour and gastrointestinal stromal tumours. This patient had hypercalcemia from high calcitriol with mesothelioma and colon cancer. Ex vivo upregulation of 1-hydroxylase in colon cancer has been described but there are no colon
cancer case reports of calcitriol mediated hypercalcemia. Hypercalcemia in mesothelium from high calcitriol with calcium normalization following resection has been reported. We could not establish which malignancy caused elevation of calcitriol in this patient. 

**Conclusion:** Elevated calcitriol could be a hypercalcemia etiology in non lymphoma malignancies and needs to be considered when work up for elevated PTHrP and osteolytic hypercalcemia is negative.

**Abstract # 1340**

**HYPERPARATHYROIDISM SECONDARY TO PARYTHYROID CARCINOMA: A RARE CASE PRESENTATION**

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**Other**

**Case Presentation:** We present a 66-year-old female who presented to the hospital after falling and sustaining a spiral fracture of the left humeral shaft. Her past medical history was significant for breast cancer status-post lumpectomy and radiation therapy 7 years prior, hyperparathyroidism, severe chronic obstructive pulmonary disease, and morbid obesity with BMI of 56. Physical exam on admission demonstrated morbid obesity and mild confusion. Laboratory evaluation pertinently showed total serum calcium 14.3 mg/dL, phosphorus 2.1mg/dL, and intact PTH (iPTH) of 627.8pg/mL. Diagnosis of primary hyperparathyroidism was made. Neck ultrasound showed a >4cm hypoechoic lesion posterior to the right thyroid lobe with no vascularity, consistent with a parathyroid adenoma. She was initially taken for surgical repair of her humeral fracture and later for parathyroidectomy. Intact PTH was 705.6pg/mL pre-operatively and decreased to 54.8pg/mL post-operatively. Final pathology showed atypical features including dense fibrous bands throughout the neoplasm with focal areas suggestive of capsular invasion and a focal area of vascular invasion. Final diagnosis of parathyroid carcinoma was made based on the clinical presentation with very high serum calcium and iPTH levels, and atypical pathologic features.

**Discussion:** Parathyroid carcinoma is a rare malignancy with incidence of 0.005% of all cancers. The first case was described by Fritz De Quervain in 1904. However, since then, diagnosis and treatment of parathyroid carcinoma is still not well defined. Not only is its diagnosis difficult to make preoperatively, the pathologic diagnosis is challenging as well. Due to its rarity, difficulty in diagnosis, and limited cases described in literature, the knowledge about treatment and postoperative course is limited. 

**Conclusion:** Our goal is to increase physician awareness about this rare presentation so as to bring more cases to light. Though the best chance for cure exists with complete resection of the tumor with margins free of malignancy, recurrence rates remain high. Not much is known about long term follow up and treatment and this may be due to the paucity of reported cases. By increasing awareness of this malignancy, we hope to increase the understanding of its management.

**Abstract # 1341**

**24-HOUR URINE CALCIUM IN 5600 PATIENTS WITH PRIMARY HYPERPARATHYROIDISM SHOWS LIMITED USEFULNESS AND LACK OF PREDICTIVE VALUE**

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**Other**

**Objective:** 24-hour urine calcium analysis is thought to help in the diagnosis of primary hyperparathyroidism (pHPT), predicting stone formation and distinguishing pHPT from familial hypocalciuric hypercalcemia (FHH), yet the test is obtained in less than half of cases and the utility is unclear.

**Methods:** A single-center review of patients with surgically proven pHPT over a 10-year period analyzed 24-hour urine calcium levels and characterized correlations with age, gender, preoperative serum calcium, PTH, and presence of nephrolithiasis.

**Results:** Of 15,541 patients referred for surgery for pHPT, only 38% underwent preoperative 24-hour urine calcium testing. Average urine calcium concentration was 275 ± 132 mg/24hr. Just 19% had 24-hour levels over 400 mg/24hr. Ten percent had urine calcium <100 mg/24hr. Male gender and younger age were both associated with higher average urine calcium (p<0.001). Urine calcium was not correlated with preoperative serum calcium or PTH level (Pearson’s r=0.1 for both, p<0.001). Nephrolithiasis occurred at all urine calcium levels. Patients with urine calcium >400 mg/24hr were more likely to have nephrolithiasis than those with lower urine calcium (30% vs. 21%, p<0.001), though 70% of those with high urine calcium did not have stones, and even patients with urine calcium under 100 mg/24hr had stones in 15% of cases.

**Discussion:** 24-hour urine calcium levels are obtained in just over one-third of patients undergoing endocrinology
workup for pHPT. In virtually every case, the test did not assist in the diagnosis of pHPT, nor was it predictive of who would or would not develop nephrolithiasis. Ten percent of patients with surgically proven pHPT had urine calcium levels under 100 mg/24hr, the accepted cutoff for FHH. Given the rarity of FHH, most hypercalcemic patients with low urine calcium will have pHPT.

**Conclusion:** Overall, there seems to be very little value of routine 24-hour urine calcium assessment in the workup of pHPT.

**Abstract # 1342**

**DIABETES INSIPIDUS IN PATIENT WITH BODYDYSMORPHIC AND EATING DISORDER**

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**Pituitary Disorders/Neuroendocrinology**

**Objective:** Importance of diet and lifestyle assessment in diabetes insipidus.

**Case Presentation:** A 26 year old female had 6 months of polyuria and polydipsia. She had positive water deprivation test results for central diabetes insipidus during which she had 4lbs weight loss, serum sodium increased above 145meq, undetectable ADH and serum osmolality >300. DDAVP administration improved urine osm from 190 to 563 and serum sodium improved from 148 to 145. Since the urine osmolality increased by 100% she was diagnosed with central diabetes insipidus. She had a normal pituitary MRI without any abnormal enhancement, pituitary stalk displacement and sellar or para sellar lesion. Her menstrual cycles were normal and her serum pregnancy test was negative ruling out diabetes insipidus in pregnancy. Her BMI was 22.5 but complained of weight gain from lack of physical activity. Desmopressin 0.05mcg oral bid resolved her polyuria and polydipsia. Subsequently she acknowledged diagnosis of body dysmorphic disorder and reported a daily dietary intake of 1200 kcal and running atleast 3 miles a day. She had poor body image perception, along with diet pattern of binge eating and not eating few days. Clinically she did not have deficiency of pituitary hormones given her normal menstrual cycles. Also it was less likely that she had either adrenal insufficiency or hypothyroidism since these conditions could impair renal free water clearance masking diabetes insipidus. However lab results were normal with Total T4 7.2, T3uptake 29% and FTI 2.1(1.2-4.9), LH 11.2, FSH 16.6, random Cortisol 10.9, Estradiol 43.1 and Prolactin 10.7. The patient continues to seek psychiatric counselling for her eating disorders and is being treated with desmopressin for the management of diabetes insipidus.

**Discussion:** Hypothalamus is essential for integration and coordination through neuronal and hormonal pathways needed for homeostatic regulation and autonomic function. Like other hormones such as oxytocin that influences maternal behavior and corticotrop-releasing hormone that modulates stress-behavior, antidiuretic hormone influences behaviour through its vasopressin receptor. Eating disorders like anorexia nervosa are associated with multiple endocrine changes. Anorexia nervosa patient have lower ADH levels and suboptimal response of kidney to exogenous ADH. Latent DI could exist even before development of anorexia nervosa.

**Conclusion:** During evaluation of patients with eating disorder the focus is mostly on hypothalamic and pituitary dysfunctions attributable to hypothalamic–anterior pituitary axis. This case underscores that derangement of hypothalamic–posterior pituitary axis can be a manifestation of eating disorders.

**Abstract # 1343**

**YOUNG FEMALE WITH ACROMEGALOID FEATURES, PITUITARY MICROADENOMA AND AN UNCOMPLICATED PREGNANCY**

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**Pituitary Disorders/Neuroendocrinology**

**Case Presentation:** Thirty-two years old female G2P1A0 seen at our endocrinology clinic nine years ago complaining of persistent menstrual irregularities associated to elevated prolactin values. MRI done revealed a pituitary microadenoma. However, she refused treatment for microprolactinoma. Four years afterwards, patient was lost to follow up and on 2012 she visited our clinic at 24th weeks of gestation with history of progressive hands and feet enlargement. On physical examination marked prognathism, hands and feet enlargement, acanthosis nigricans and hirsutism were found, so acromegaly was suspected. Elevations of prolactin and IGF-1 levels were found during whole pregnancy. After six months postpartum, laboratories were repeated and basal and 2hr insulin showed marked elevation, increased HOMA index, and normal IGF-1 and GH values. Thus, the diagnosis of
Insulin Mediated Pseudoacromegaly was confirmed. Discussion: We present a 32 y/o female G2P1A0 on her 24th week gestation with clinical characteristics and computerized FIDA results compatible with acromegaloid features. Of interest was the elevated IGF-1 level during pregnancy most likely due to placental production, not pituitary in origin as confirmed post partum when IGF-1 levels normalized. Repeated laboratories six months post partum showed basal and 2 hours Insulin elevation, increased HOMA index, but suppressed GH and IGF-1 levels. Thus, this patient presents an unusual combination of pseudoacromegaly in the presence of insulin resistance, a macroadenoma and an uncomplicated pregnancy.

Conclusion: Pseudoacromegaly is a rare syndrome in which the biochemical hallmarks of Acromegaly as in our patient are lacking. Several mechanisms has been reported the last one being proposed by Karim Dib et al. suggesting the presence of a selective post receptor defect in IP-3 Kinase defect that results in impairment of normal function of the metabolic pathway but preservation and adequate function of the mitogenic signaling which is involved in cell growth stimulated by insulin. This leads to diversion towards the mitogenic pathway, potentiating the effects of insulin and other growth factors contributing to acromegaloid features seen in these patients. Although more research is needed in order to better understand pseudoacromegaly this review may help the clinician to better direct the evaluation of patients presenting to the clinic with characteristics similar to the ones seen in our patient.

Abstract # 1344

LANDERHANS CELL HISTIOCYTOSIS-ASSOCIATED CENTRAL DIABETES INSIPIDUS IN AN ADULT PATIENT

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Pituitary Disorders/Neuroendocrinology

Objective: Langerhans cell histiocytosis (LCH) is a rare disease affecting 1-2 adults per one million in the U.S. population. Central Diabetes Insipidus (CDI) occurs in 5% to 50% of patients with LCH. The correlation between the two results from disruption of ADH secretion due to histiocyte infiltration of the hypothalamus. This report describes a case of an elderly patient diagnosed with LCH who subsequently developed CDI.

Case Presentation: A 74 year old Caucasian female presented with tender erythematous plaques at the inframammary, truncal and inguinal regions. The lesions were refractory to conservative medical treatment and on biopsy revealed LCH of the skin. Additionally, the patient complained of unexplained polydipsia, polyuria and weight loss. MRI of the brain with contrast revealed an irregular enhancing mass measuring 1cmx9mmx9mm. The mass involved the hypothalamus, pituitary stalk, tuber cinereum and suprasellar cistern. Work-up revealed normal TSH, free T3, morning cortisol, ACTH, estradiol and IGF-1. Prolactin was elevated and free thyroxine, LH and FSH were decreased. Urine osmolality and urine sodium were low, while serum osmolality was elevated. Following cladribine infusions, the patient was started on radiation therapy with 14 Gray to the hypothalamus and pituitary gland. Desmopressin improved urine osmolality, urine sodium and serum osmolality. A chest CT scan revealed several nodules in both lungs. No focal bone lesions were identified on complete bone survey studies. Mammography found no significant evidence of malignancy. Follow-up MRI performed six months post-treatment showed a significant decrease in degree of enhancement of the hypothalamic mass and a reduction in size to 6mmx6mmx4mm. The patient was also taking methotrexate. Two years after radiation therapy, no significant residual enhancement in the hypothalamus was evident on brain MRI, and the pituitary appeared normal. A repeat skin biopsy was also normal.

Discussion: This patient presented with posterior pituitary insufficiency assessed as CDI from metastatic LCH. CDI was well-controlled with desmopressin. Once diagnosed, CDI is rapidly irreversible in most patients. Although the natural history, treatment and prognosis are not clearly established for LCH, radiation has been the standard of treatment for hypothalamic-pituitary lesions,with serial monitoring of hypothalamic-pituitary hormones post-radiation to evaluate for panhypopituitarism.

Conclusion: LCH is a multisystem disease that may affect hypothalamic-pituitary function, causing CDI. A high index of suspicion for CDI must be maintained when monitoring patients diagnosed with LCH in order to avoid the need for lifelong treatment with desmopressin.
Abstract # 1345
SELLAR GERMINOMA IN A MALE OF 26 YEARS OLD WITH SEVERE CHIASMATIC SYNDROME AND PANHYPOPITUITARISM

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Pituitary Disorders/Neuroendocrinology

Objective: To describe a case of sellar germinoma with high mitotic index.

Methods: We describe the clinical features of a young adult with sellar germinoma.

Results/Case Presentation: Male, 26 yo, had pleural TB incompletely treated by drug intolerance. His cousin had medulloblastoma. He had a 3-year disease evolution characterized by polyuria, polydipsia and hyporexia; erectile dysfunction is added later, was diagnosed with pituitary microadenoma MRI in Feb2011. Subsequently by persistent headache and decreased vision was evaluated by finding hypopituitarism and was prescribed LT4 100μg/d, and prednisone 15mg/d; and new MRI study he underwent transsphenoidal surgery for presence of sellar and suprasellar expansion process (Oct2012) whose pathological diagnosis ‘chromophobe adenoma.’ Then by disorder sensory and hyperpyrexia was transferred to another institution with hydrocephalus and intracranial hypertension, subsequently undergoing transcranial resection (Nov2012); because ventilatory and infectious postoperative complications is transferred to the ICU of our hospital. Pathologic review concluded germ cell tumor. He was admitted to our department in Jan2013 with persistence and intensification of polyuria, polydipsia, headache and fever, somnolence, bradilalia and decreased visual fields. Analysis: β-hCG:0.12, AFP:0.2, TSH:0.029, LH:0.11, FSH:0.31; Cortisol: <1.00, GH:0.179, IGF-1<25; PRL:9.64; Free Testo:2.34. After hemodynamic and metabolic compensation he underwent subtotal resection of suprasellar tumor transcranial; finding extensive tumor infiltrating across the opto-chiasmatic structure with severe lesion of the chiasm and both optic nerves; tumor dimensions 4.5x4.0x3.5cm. Histopathology:Germinoma,PLAP(+),CD117(+),Ki-67:50%, EMA(-),GFAP(-). After overcoming multiple Intercurrences the patient underwent 25 sessions of ERT with linear accelerator. His MRI showed a significant reduction of tumor volume. The patient improved clinically but persists with bilateral amaurosis.

Discussion: CNS germinomas correspond to 0.4-3.4% of primary intracranial tumors. It mainly affects prepubertal and usually located in the pineal gland or suprasellar region. They may present with diabetes insipidus, hypopituitarism and visual disturbances. CNS germinomas are β-hCG and AFP negative in 85-90% of cases. They are radiosensitive and potentially curable.

Conclusion: Thickening of the pituitary stalk on MRI suggest inflammatory, granulomatous and neoplastic lesions. Although in most cases with pituitary intrasellar masses and thickening, clinical and para- clinical may guide the diagnosis, a biopsy should be performed to allow an accurate diagnosis and appropriate treatment.

Abstract # 1346
LYMPHOCYTIC HYPOPHYSITIS WITH COMMITMENT SIXTH CRANIAL NERVE IN A MALE 35 YEARS

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Pituitary Disorders/Neuroendocrinology

Objective: Describe the case of a patient with pan-hypopituitarism secondary to pan-lymphocytic hypophysitis.

Methods: We report the clinical features and analysis of a patient with autoimmune pan-hypophysitis.

Results/Case Presentation: Male, 35yo; measles, chickenpox and mumps in childhood. Suffering 1 year 3 episodes presenting headache in left frontal region, intensity 8/10 irradiated to orbital area associated to horizontal diplopy. Initially diagnosed with herpetic encephalitis without changes in EEG or MRI, He received corticosteroids and acyclovir IV with total remission. 05 months after new episode receiving corticosteroid PO 2 weeks. A month later, reappears headache and diplopy of the same features, adding fatigue, polydipsia, polyuria, decreased libido, changes in mood. On examination: paresis of the left eye lateral rectus, no ptosis and isochoric pupils photoreactive. New MRI showed marked thickening of the pituitary stalk with mass effect on the optic chiasm being transferred to Endocrinology. Analysis: Hb:12g%, hypopituitarism, Na:163mEq/L; K:3.68mEq/L, Uosm: 87mOsm/kg. No evidence of active autoimmune or infectious disease, RCP(-); ESR:22mm/h; CRF:colorless, transparent, protein:3.7g, glucose:59mg; BK(-), ADA(-) Flow cytometry: Lymphocytes NK 92%, Lymphocyte T:86% CD4:CD8 63.5% :18%. Biopsy: Infundibulum thickened and deformed by solid gray tissue that protrudes from the inside, right lateralized injury in addition to the presence of grayish tissue throughout the basal meninges covering sellar diaphragm based optical channel and right anterior tuber area Sellar; histopathological study concluded: fibrous tissue with marked infiltrate lymphoplasmacytic compatible with lymphocytic hypophysitis. Methylprednisolone 500 mg/d for 3 days IV was prescribed.
Discussion: Lymphocytic hypophysitis is rare, first described in young women during pregnancy or postpartum, may be associated with periods of remission with other exacerbation. The histopathology is characterized by the presence of inflammatory infiltrates rich in plasma cells and T lymphocytes to destroy the glandular tissue. There also fibroblast proliferation with collagen deposition. The treatment is controversial and in some cases reverse the hormone deficiency.

Conclusion: The pan-hypophysitis affects both lobes of the pituitary gland, at any age and in both sexes. The most common form of clinical presentation is that of a pituitary dysfunction ranging from panhypopituitarism and an isolated and in some cases can have a pseudotumor behavior with neighboring structures commitment hormone deficiency.

Abstract # 1347

DO WE NEED HORMONAL SCREENING IN PATIENTS WITH SUBCENTIMETER PITUITARY MICROADENOMAS?

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Pituitary Disorders/Neuroendocrinology

Case Presentation: A 54-year-old post-menopausal woman was seen at our endocrinology clinics. She presented with upper and lower extremity paresthesia, salt cravings, episodes of hypotension and a long term history of depression. Vital signs: BP: 90/65 HR 65 RR: 18 T: 37.5 C BMI: 26. Physical exam within normal limits. Cervical and brain MRI ordered by her neurologist three years ago revealed sella and pituitary normal in size, stable very small 3 mm pituitary incidentaloma and mild disc bulging at C4-5 and C6-7. Due to persistence of symptoms repeated pituitary MRI with IV contrast showed no significant changes from previous study done three years ago. Basal hormonal evaluation was compatible with a low cortisol: 1.16µg/dL and ACTH: < 5 pg/ml, elevated LH and FSH and low estradiol. The rest of the anterior pituitary hormones including IGF-1 and GH were normal. However, an Insulin Tolence Test was performed, a subnormal response in ACTH: < 5 pg/dl and cortisol: 10.3µg/dL was observed after patient achieved a good hypoglycemic level. Glucagon Stimulation Test revealed growth hormone deficiency. Past medical history is negative for autoimmune diseases, surgeries, radiotherapy, head trauma or use of any medications. Due to our findings in dynamic testing, ACTH and GH deficiency was established. Hydrocortisone 5 mg at AM and 2.5 mg at PM was started.

Discussion: ACTH deficiency and the rarity of its occurrence explain the uncertainties in its epidemiology and etiology. In adults, an autoimmune etiology is most often hypothesized as an alternative to traumatic cases. (1) Prevalence of reduced hormonal secretion in subjects with microadenomas is most frequent for somatotrophs, followed by thyrotrophs, gonadotrophs and rarely of corticotrophs.(2) GH and ACTH deficiency are underestimated disorders frequently misdiagnosed and can be a challenging task for physicians due to nonspecific symptoms. Routine hormonal screening may not be necessary in microincidentalomas because the rate of hypopituitarism is very low.(3)

Conclusion: Even though recent guidelines strongly favor hormonal screening for microincidentalomas of 6 mm to 9 mm, and not necessarily in smaller microincidentalomas, our case shows that hormonal deficiencies may occur in small tumors less than 6 mm.

Abstract # 1348

DIABETES INSIPIDUS UNMASKED BY GLUCOCORTICOID REPLACEMENT IN THE SETTING OF PITUITARY APOPLEXY

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Pituitary Disorders/Neuroendocrinology

Objective: Pituitary apoplexy is potentially life threatening if not diagnosed and treated accordingly. There are many documented cases of pituitary apoplexy, but there are few documented cases of pituitary apoplexy with diabetes insipidus (DI). The objective of this case presentation is to demonstrate a patient with apoplexy into a pituitary mass in whom diabetes insipidus was unmasked by glucocorticoid replacement.

Case Presentation: A fifty year old male with no known history of pituitary disease presented with four days of right sided vision loss and lateral deviation of the right eye, as well as nausea and vomiting. He also noted having polyuria, polydipsia, nocturia, and headaches in the preceding 3 months. He was hyponatremic with sodium of 130mmol/L and creatinine of 3.30 mg/dL. MRI without
contrast showed a 2.2x1.7x1.5cm enhancing sellar mass with increased signal on T1 images, compression of the optic chiasm and mass effect on the right cavernous sinus, most likely representing pituitary macroadenoma with hemorrhage. The neurology service began the patient on IV decadron for his visual loss. On hospital day 2, he was seen by endocrinology with urine output of 200ml/hr on 125ml/hr of normal saline. He craved ice water and complained of inability to quench his thirst though able to drink freely. Labs included normal prolactin and thyroid function testing, 8am cortisol 2.6 ug/dL, urine osmolality 329 mosm/kg, and plasma osmolality 305 mosm/kg, plasma sodium 135mmol/L, urine sodium 82 meq/L. Over the next 3 days on decadron 4mg IV q6h, he developed urine outputs of more than 7 liters/day as well as increased polydipsia, and repeat labs osmolality 295 mosm/kg, urine osmolality of 265 mosm/kg, plasma sodium 138 mmol/L, and urine sodium 38 meq/L. The patient’s urine output and symptoms improved on desmopressin 0.5mcg SQ daily. He underwent transphenoidal surgery. The final pathology showed fragments of necrotic tissue with acute inflammation and hemorrhage. His vision improved postoperatively, and he remained on desmopressin for control of DI.

**Discussion:** Our patient presented with polyuria, polydipsia and mild DI and evidence of secondary adrenal insufficiency. His secondary adrenal insufficiency was due to pituitary apoplexy of a pituitary macroadenoma. Treatment of his secondary adrenal insufficiency with glucocorticoids unmasked overt DI which improved with desmopressin.

**Conclusion:** Pituitary apoplexy is an uncommon complication of pituitary adenomas and rarely presents with the symptoms of DI. Postoperatively, DI can occur due to surgery or severing of the pituitary stalk. This case demonstrates that glucocorticoids may unmask DI more commonly in pituitary apoplexy than previously reported.

**Abstract # 1349**

**HYPERPROLACTINEMIA AND GROWTH HORMONE DEFICIENCY ASSOCIATED WITH MORNING GLORY SYNDROME**

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**Pituitary Disorders/Neuroendocrinology**

**Objective:** Morning Glory Syndrome (MGS) is a congenital anomaly of the optic disk. It can occasionally be associated with midline cranial defects, including hypopituitarism. We describe a case of MGS associated with Hyperprolactinemia, and Growth Hormone (GH) deficiency.

**Case Presentation:** A 32 year old white female who was diagnosed with MGS at age 3, and had no and family history of similar or related issues was seen by her physician at age 17 for primary amenorrhea, short stature (her height was 4ft 1 inch). She was diagnosed with MGS, left eye blindness hyperprolactinemia and GH deficiency. She was treated with GH replacement, birth control pill till age 28, and then birth control was discontinued. Due to hyperprolactinemia and unovulatory cycles she was treated with Dostinex. At age 31, underwent in vitro fertilization with successful delivery on her recent physical exam revealed high 5 feet, blood pressure 110/70 mmHg, pulse rate 72 per minute, weight 126 lbs. Visual acuity to finger counting 20/20 right eye / 20/200 left eye, hypertelorism and galactorrhea. Fasting Glucose: 87 mg/dl (65-99); Prolactin: 62 mg/l (2-14); GH: 0.2 ng/ml (0.0-10.0); GH: post arginine stimulation test <2ng/ml; IGF1: 22ng/ml (71-241); LH: 1.3 mIU/ml (0.0-4.0); FSH: 5.0 mIU/ml (0.0-5.0); Estradiol: 8.6 pg/ml (12.5-166.0); DHEA: 323 ng/dl (31-701); ACTH: 33.1pg/ml (7.2-63.3); TSH: 1.48 iu/ml (0.45-4.5); Free T4: 1.2ng/dL (0.58-1.6). MRI revealed congenital abnormality involving the optic chiasm left optic nerve. Mild compression of the pituitary gland by a basal encephalocele, with normal size pituitary gland. Genetic studies were positive for mutation in PAX 6 gene.

**Discussion:** MGS occur more commonly in females and occur rarely in African-Americans. It can be associated with congenital cataracts. Visual acuity typically is 20/200 to finger counting, although all levels of vision have been reported. Acquired visual loss associated with the morning glory disc anomaly can occur. Morning glory disc anomaly may be associated with midline cranial defects, including transphenoidal encephalocele, causing to have wide heads, flat noses, cleft lip and/or palate, hypertelorism, agenesis of the corpus callosum, hypopituitarism, and absence of the optic chiasm. The diagnosed of transphenoidal encephalocele is made with MRI. Interestingly, this patient had hypertelorism and loss of vision of the left eye. Her endocrine abnormalities were GH deficiency and hyperprolactinemia, possible due to compression of the pituitary gland from the basal encephalocele.

**Conclusion:** In young female with been evaluated primary amenorrhea and hyperprolactinemia, and MGS should be included in the differential diagnosis.
Abstract # 1350

EARLY TEMOZOLAMIDE TREATMENT OF ATYPICAL REFRACTORY PROLACTINOMA RESULTING IN REMISSION

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Objective: The management of refractory, invasive pituitary prolactinomas is a significant clinical challenge. Multimodal management strategies are typically employed, though there can be limitations, including dopamine agonist intolerance, radiation necrosis and surgical complications. Temozolamide has been used for intracranial malignancies including pituitary carcinoma and refractory prolactinomas with reasonable success. It has been advocated to consider temozolamide therapy early in the course of this disease.

Case Presentation: A 48 year old man was diagnosed with a prolactin secreting adenoma in 2003 (initial prolactin level >3000 ng/ml) with vision loss refractory to bromocriptine. He underwent transsphenoidal resection followed by stereotactic radiosurgery for the residual tumor. He had persistently elevated prolactin requiring high doses of bromocriptine and required repeat surgery. He ultimately developed Addisonian crisis, failed cabergoline therapy and presented to our institution with an increased prolactin of 2932ng/ml, optic chiasm compression and a progressively invasive atypical adenoma (Ki-67 ~ 10% with increased p53). He underwent a third endonasal endoscopic debulking operation and his prolactin level decreased from 2932 to 338ng/ml. Over the next year, he was maintained on increasing doses of cabergoline but his prolactin level continued to rise to 533ng/ml with associated tumor growth seen on MRI. Consequently, he was started on adjuvant temozolamide therapy one year after surgery. After starting temozolamide, the patient’s serum prolactin levels decreased from 696ng/ml to 8ng/ml after ten cycles of temozolamide over an eight month period. There was also a marked decrease in tumor size of over 90% on serial MRI scans. The patient remains in remission (prolactin level 5 ng/ml) over an eight month period. There were no significant complications of temozolamide therapy.

Discussion: This case demonstrates that temozolamide can be efficacious for the treatment of refractory, invasive atypical prolactinomas. The literature supports temozolamide therapy for these tumors, though early administration after surgical failure has not been reported. As soon as this patient’s refractory condition was recognized at our institution (within a year of evaluation, surgery and treatment), he was started on temozolamide therapy. As these patients may require numerous procedures and high doses of dopamine agonist therapy, it is pertinent to consider early temozolamide treatment after surgical and radiosurgical failure.

Conclusion: Early adjuvant temozolamide therapy should be administered for the management of refractory atypical prolactinomas.

Abstract # 1351

IMPACT OF SELECTIVE PITUITARY GLAND RESECTION OR INCISION ON HORMONAL FUNCTION IN ENDONASAL TUMOR OR CYST REMOVAL

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Objective: With the resection of pituitary adenomas or Rathke’s cleft cysts (RCC), the anterior pituitary gland often partially or completely obstructs transsphenoidal access to the lesion. In such cases, a gland incision and/or partial gland resection may be required to obtain adequate tumor/cyst exposure. We investigated the frequency with which this technique was performed in our practice and determined the associated risk of post-operative hypopituitarism.

Methods: All patients who underwent endoscopic-assisted or fully endoscopic removal of a pituitary adenoma or RCC between July 2007 and January 2013 (minimum 3 month follow-up) and had a gland incision or resection performed were identified. Each patient’s routine pre- and post-operative hormonal testing (minimum 3 month follow up) was then retrospectively evaluated in order to determine the overall impact on pituitary gland function. Total hypophysectomy patients were excluded.

Results: Of 372 total operations over this period, an anterior pituitary gland incision or partial gland resection was performed in 93 cases (25%). In 64 operations, a vertical or horizontal gland incision was made only while the remaining 29 cases involved some degree of gland resection including 17 partial hemi-hypophysectomies and 12 resections of thinned/attenuated anterior gland draped over a large macroadenoma. Diagnoses included 72...
pituitary adenomas (18 endocrine-inactive, 32 Cushing’s, 14 prolactinomas, 8 acromegaly) and 21 RCCs. Of 63 patients with complete endocrine follow-up data, new permanent hypopituitarism occurred in 4 patients (6.5%): two with macroadenomas (3.2cm and 3.5cm) and two with RCCs (2.7cm and 1.2cm). Of these 4 patients, one macroadenoma patient had apoplexy and one RCC patient had pre-operative hypopituitarism. All 4 developed permanent DI and one macroadenoma patient developed growth hormone deficiency. Four additional patients had transient post-operative hyponatremia. Overall gland function improvement occurred in 13/22 patients (59%) with pre-operative hypopituitarism, including 9 with resolution of 1 axis or of stalk-effect hyperprolactinemia and 4 with resolution of 2 or more axes.

**Discussion:** Selective gland incisions and gland resections which were performed in 25% of our cases, appear to minimize traction on compressed normal pituitary gland during removal of large tumors or cysts and facilitates better visualization and removal of both microadenomas and macroadenomas.

**Conclusion:** Judicious use of pituitary gland incisions and partial gland resections are generally well-tolerated with a relatively low risk of causing new hypopituitarism and can be utilized when necessary to gain surgical access to pituitary adenomas and RCCs.

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**Abstract # 1352**

**“I’M NOT THIRSTY!” CENTRAL DIABETES INSIPIDUS MASQUERADING AS BENIGN PROSTATIC HYPERPLASIA IN A NATIVE HAWAIIAN**

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Pituitary Disorders/Neuroendocrinology

**Objective:** Central diabetes insipidus develops when production or release of antidiuretic hormone is disrupted. A large number of cases are idiopathic with the vast majority of all remaining adult cases a result of primary or secondary hypothalamic tumor, infiltrative disease, neurosurgery or trauma. Pituitary adenoma is not typically a cause of central diabetes insipidus. We present a case of pituitary macroadenoma resulting in central diabetes insipidus that presented critically after months of polyuria and nocturia that had previously been attributed to benign prostatic hyperplasia.

**Case Presentation:** A 67 year old male with history of hypertension and on no medications presented with headache, nausea, nocturia and polyuria. He had ongoing symptoms for a year and presented to the ED after acute worsening. He was hypotensive and tachycardic. He was admitted to the ICU briefly requiring vasopressor support for blood pressure support. He was noted to have large volume output of dilute urine and elevated serum sodium of 148. An MRI demonstrated a concentric sellar mass with lymphocytic hypophysitis as a resultant working diagnosis. He was started on corticosteroids but after no improvement a repeat MRI showed a 2 x 1.3 x 2.5 cm lobulated, heterogeneously enhancing mass seen centered within the sella with T2 hyperintensity concerning for necrosis. He underwent a transsphenoidal excision of the mass with pathology consistent with nonsecreting pituitary adenoma. He responded appropriately to treatment with PO and nasal desmopressin.

**Discussion:** Central diabetes insipidus is frequently seen with tumors that involve the hypothalamus. It is also frequently seen in metastatic spread of lung or breast cancer to the posterior pituitary or hypothalamus. However, tumors originating from the anterior pituitary very rarely cause DI. This led to a working diagnosis of lymphocytic hypophysitis in this patient but a lack of response to therapy with corticosteroids led to excision and final diagnosis of adenoma. There is also an interesting cultural twist to this case. The patient was working as a construction worker on the Big Island of Hawaii. Several coworkers had similar symptoms of frequent urination and nocturia and had been diagnosed with BPH. They managed their symptoms with decreased PO water intake at work and the patient felt pressured to do the same. As a result he developed a significant ability to ignore his thirst response and this contributed to his not seeking medical care before becoming very ill. The patient required coaching prior to discharge in order to recognize his thirst response and achieve stable serum sodium levels.

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**Abstract # 1353**

**TESTOSTERONE THERAPY IS NOT ASSOCIATED WITH HIGHER RISK OF MYOCARDIAL INFARCTION OR STROKE: THE LOW T EXPERIENCE**

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Reproductive Endocrinology

**Objective:** Following recent studies on concerns for safety with testosterone therapy, our goal was to assess the association between testosterone therapy and MI, or stroke
among our Low T Center patients. These community based specialized centers have strict protocols requiring regular 2 week monitoring in the office for efficacy and safety.

Methods: Following IRB application and GCP training, we conducted a retrospective analysis of patients that had MI and strokes pre and post testosterone therapy. Data was extracted from our electronic health record (Advance MD) of the multi site Low T Centers across the United States. Altogether 40 Centers were examined. Prior to extraction of data; we held 3 nationwide conference calls with providers to ensure that all ICD-9 were updated, with particular attention to MI and strokes. We also interviewed families with patients that had fatal MI.

Results: 39,937 patients were seen between years 2009-2014 and approximately 50% met criteria for treatment. Of the treated patients, there were 4 non-fatal MI and 2 probable fatal MI; and rate of new MI was 30 per 100,000. There were 46 patients with pre therapy MI of which none had adverse outcomes after testosterone. Of the treated patients, there were 2 cases of stroke; and rate of new stroke was 10 per 100,000. There were 12 patients with pre therapy stroke and none had adverse outcomes after testosterone. The risks for new MI and stroke were compared to the Kaiser Permanente and Northern Manhattan Registry which was 208 per 100,000 and 93 per 100,000 respectively. Rate ratio (RR) for MI in testosterone treated patients is 0.14 (C.I. 0.098 to 0.211, p<0.0001) whereas strokes is 0.107 (C.I. 0.06 to 0.21, p<0.0001).

Discussion: There have been recent studies associating testosterone therapy with myocardial infarctions (MI). One study found 1223 hypogonadal veterans undergoing coronary angiography and who had used testosterone to have increased risk of adverse outcomes. The other study of 55,593 patients in an insurance database found that the risk of heart attacks was increased in older men and younger men with pre-existing heart disease. Our study showed the opposite in that carefully monitored testosterone treated patients has 7 and 9 times lower risk of developing MI and strokes respectively as compared to similar community data sets.

Conclusion: This study contradicts the 2 other studies, instead suggesting a protective effect of testosterone against MI and strokes. Further, there was no evidence of worsening of pre-existing MI or strokes in patients treated with testosterone.

Abstract # 1354

INCIDENCE OF LOW TESTOSTERONE IS 40% IN TESTED MEN > 40 YEARS; HIGHEST PREVALENCE FOUND IN MEN LIVING IN SOUTHERN STATES: RESULTS FROM A NATIONWIDE DATABASE

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Reproductive Endocrinology

Objective: To use a national lab test results database (Medivo Lab Value Exchange (LVX™), Medivo Inc., New York, NY) to review population-level data for the US low testosterone patient population using demographic, geographic, comorbidity, and physician specialty criteria.

Methods: We analyzed the results of all men >40 years of age in the LVX who were tested for testosterone levels between Jan 1, 2012 - Dec 31, 2013. We also used an algorithm based on testosterone test panels and ICD-9 codes to find all men of all ages in the LVX with low testosterone. Included in the analyses were the practice types where testing took place, the practice zip codes, and the rates of reported ICD-9 codes for 3 selected comorbidities known to be associated with low testosterone: diabetes, obesity and cardiovascular disease.

Results: The nationwide LVX included 100,263 men over age 40 tested for testosterone between Jan 1, 2012 - Dec 31, 2013. Of this group, 40,373 (40%) had low testosterone levels (< 300 ng/dL); 18,447 (18.3%) had levels ≤ 200 ng/dL; 10,550 (10.5%) had levels between 201 - 250 ng/dL; and 11,375 (11.3%) had levels between 251 - 299 ng/dL. Among a total of over 69,000 men with low testosterone in the LVX, the highest rates of men with low testosterone were found in the South (56%), followed by the West (26%), the East (16%) and the Midwest (10%). The highest rates of testosterone tests were ordered by primary care physicians (9%), followed by endocrinologists (7%) and urologists (7%). The most commonly reported comorbidity among men with low testosterone was diabetes (11.93%), followed by obesity (0.66%) and ischemic heart disease (0.06%). Looking at men with low testosterone and all 3 comorbidities, the highest rates were found in the South (6.6%), followed by the West (2.6%), the East (2.0%) and the Midwest (1.3%).

Discussion: We found that the prevalence of low testosterone among men >40 years of age tested for testosterone was high (40%). Most testing for testosterone levels is conducted by primary care physicians. The highest
rates of men with low testosterone occur in the South, as do the highest rates of men with low testosterone and comorbidities associated with low testosterone (diabetes, obesity and heart disease). Due to the availability of ICD-9 code data for only about 13% of men tested for low testosterone, we suspect that comorbidity rates may be higher in this population.

**Conclusion:** Research based on a nationwide laboratory results database can provide valuable insights on testosterone test patterns, including patient information such as demographics and geographic regions, and also testing rates by physician specialty.

Abstract # 1355

**PREVALENCE OF SHORT STATURE IN JUVENILE HYPOTHYROIDISM AND THE IMPACT OF TREATMENT ON GROWTH VELOCITY IN A TERTIARY CARE CENTER, WESTERN U.P, INDIA**

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**Thyroid Disease**

**Objective:** To study the prevalence of short stature in juvenile hypothyroidism, to study the various radiological manifestations of juvenile hypothyroidism and to study the impact of treatment on growth velocity and on various skeletal manifestations.

**Methods:** Out of total nine hundred hypothyroid patients, eighty seven patients found to be of juvenile hypothyroidism were enrolled in the study that were 6-18 years of age with newly diagnosed or on follow in the endocrine clinic over a period of 1 ½ years were evaluated clinically and by laboratory tests. Serial assays of TSH, T4, T3 and skeletal X rays and anthropometry were done at regular interval and clinical and radiological outcome of patients were analyzed.

**Results:** The mean age of diagnosis of juvenile hypothyroidism was 11.2 years, and the females had twice the incidence than that of males, the mean TSH value were 118±24.3 µIU/ml. Prevalence of short stature was found to be 45% while delayed bone age was found to be 72% in juvenile hypothyroid populations. Height SDS increased from –2.9±0.9 at the start of thyroidine therapy to –1.8±0.8 after 12 months later (p <0.001). Bone age SDS increased from 8.9±2.5 at the start of thyroidine therapy to 10.8±2.7 after 12 months later. Height velocity increased from 4.9±0.8 cm/year in the year before treatment to 8.7±1.3 during treatment (p <0.001). Serum TSH decreased and serum T4 levels increased after treatment (p < 0.002). The most resistant resistance to treatment was found to be on skull (enlarged sella, and wormian bones) and thickened band at metaphyseal ends.

**Discussion:** The importance of recognising the various skeletal manifestations and the prevalence of short stature in juvenile hypothyroidism and impact of treatment outcome in this condition can profoundly improve the growth velocity.

**Conclusion:** The presentations of juvenile hypothyroidism may be varied including short stature, spondylolisthesis, delayed bone age and irregular ossification of the epiphyses. Prompt recognition of the findings can lead to early and effective treatment, and improving the skeletal defects.

Abstract # 1356

**RAPID HIGH-DOSE LEVOTHYROXINE ABSORPTION TESTING FOR PSEUDOMALABSORPTION**

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**Thyroid Disease**

**Objective:** To describe a case of pseudomalabsorption, i.e., non-adherence to levothyroxine (LT4), and a successful rapid high-dose LT4 absorption test completed in 2 hours.

**Case Presentation:** A 41-year-old, 70 kg female with a history of primary hypothyroidism due to hashimoto’s thyroiditis presented with severe hypothyroidism despite being prescribed 200 mcg of LT4 daily. She reported taking the LT4 regularly in the fasting state without other medications. She underwent Roux-en-Y gastric bypass surgery 2 years prior to presentation and has a history of irritable bowel syndrome. TSH was 54.98 µIU/ml (0.5 - 4.70) and total thyroxine (T4) was 1.8 µg/dL (4.5 - 12.5). LT4 was increased to 300 mcg daily and a subsequent TSH was 69.59 µIU/L. After a further LT4 increase to 400 mcg daily the TSH was 90.21 µIU/L. We performed a rapid version of the LT4 absorption test over 2 hours. The patient was instructed to skip her usual dose of LT4 the morning of the test. At baseline, 1000 mcg of LT4 was administered orally to the patient in the fasting state. At 0, 1 and 2 hours, TSH was 0.09, 0.10 and 0.09 mIU/L, FT4 was 3.1, 5.4 and 6.1 ng/dL (0.8 - 1.8), and free triiodothyronine (FT3) was 12.3, 11, 10.8 pg/mL (2.3 - 4.2). The diagnosis of pseudomalabsorption was made. We decreased the LT4 dose to 300 mcg daily and re-counseled the patient on compliance. Subsequently the TSH was 0.01 and FT4 was 3.2 ng/dL. LT4 was then reduced to 150 mcg daily and follow-up testing is pending.
Discussion: Despite the low TSH and high FT4 at baseline, which indicate the patient had started taking her medication, there was a robust FT4 and FT3 response. This suggests normal LT4 absorption. Testing for LT4 absorption, usually performed over 6 hours or several days, can differentiate pseudomalabsorption from true malabsorption. Other studies have shown that peak serum levels of LT4 are achieved within 2 to 4 hours after ingestion. High-dose levothyroxine absorption testing over 2 hours offers a more rapid alternative to 6-hour or multi-day testing to evaluate pseudomalabsorption. Such testing may be contraindicated in patients with cardiac conditions.

Conclusion: High-dose levothyroxine absorption testing over 2 hours offers a more rapid alternative to 6-hour or multi-day testing to evaluate pseudomalabsorption.

Abstract # 1358

PERCUTANEOUS ETHANOL INJECTION (PEI) OF COMPLEX THYROID CYSTS: OUTCOME OF THREE CASES

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Thyroid Disease

Objective: Presented are three cases of complex cystic thyroid nodules which were treated with percutaneous ethanol injection.

Methods: Three complex cystic thyroid nodules were identified by thyroid ultrasound and were proven to be benign on fine needle aspiration biopsies. The volumes of these nodules were measured prior to percutaneous ethanol injections. Local anesthesia was used for the procedures using 5 cc of 2% Lidocaine. 18-22 gauge needles were used for cysts aspiration. The volumes of the drained fluid were used to calculate the amount of 200-proof Ethanol injected. Informed consents were obtained from the patients, explaining in details the potential complications and required precautions.

Case Presentation: 75, 71, and 31 year-old patients, a male and two females, included in the study with left sided complex solid cystic thyroid nodules. Pre-PEI volumes were 15.3, 3.6, and 35 cc, respectively. Post-PEI volumes were 4, 0.8, and 23 cc, respectively in a follow up ultrasound performed a few weeks following the procedure. Estimated amounts of fluid removed were 7, 3.5, and 15 cc; and estimated amounts of 200-proof Ethanol injected were 5 cc, 3.5, and 10 cc, respectively. No post-procedure complications such as tingling, voice change, dysphonia, bleeding, pressure, significant pain, or bruises reported. The patients reported immediate relief of the pressure symptoms in their necks and noted cosmetic improvements.

Discussion: PEI is a therapeutic procedure which involves thyroiditis or any other risk factor for malignancy. Since Amiodarone is a thyrotoxic drug and has been associated with thyroid neoplasm it is possible that the thyroid lymphoma in this patient was a result of long term Amiodarone use. Considering the fact that our patient had hypothyroidism prior to being put on Amiodarone, it is possible that he may be having an underlying undiagnosed Chronic Autoimmune Thyroiditis.

Conclusion: We believe it is important to document this association and Amiodarone should be kept in the differential diagnoses of risk factors associated with thyroid lymphoma.
an injection of ethanol subcutaneously into a variety of lesions to induce sclerosing properties and achieve measurable outcomes, such as reduction in the sizes of the lesions and relieve of pressure symptoms. It has been used for many years as part of management of hepatocellular tumors, renal cysts, among many other lesions. It has been also used in the management of cystic thyroid nodules in addition to other neck lesions.

**Conclusion:** PEI is a relatively safe outpatient procedure that could certainly minimize the need of primary surgeries or repeat surgical interventions. It is anticipated to contribute to the reduction of the costs to the health system and is particularly useful in selected patients who have contraindications for surgeries or radioactive iodine therapy.

**Abstract # 1359**

**DEVELOPMENT OF GRAVES’ DISEASE AFTER THYROID SURGERY**

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**Thyroid Disease**

**Objective:** To present a case of a patient developing Graves’ Disease after hemithyroidectomy.

**Case Presentation:** A 32 yo Hispanic female was referred to our clinic for a thyroid nodule. Thyroid ultrasound showed a mixed cystic/solid lesion in the right thyroid lobe measuring 5.2x2.4x3.4 cm. Fine needle aspiration showed a rare cluster of benign follicular epithelial cells and scanty watery colloid suggestive of a benign colloid nodule. Right thyroidectomy was recommended due to size. Two months later patient had a right thyroid lobectomy and isthmectomy. Histological examination revealed adenomatoid nodule with Hurthle cell change with background thyroid of Hashimoto’s thyroiditis. There was no evidence of malignancy. Postoperatively patient reported weight gain, but was not hypothyroid based on laboratory testing. Six months later patient returned because of troublesome anxiety, increasing weight gain, hair loss, constipation and dizziness. Thyroid testing revealed TSH 0.01 (reference range 0.4-4.2), Free T4 1.3 (reference range 0.8-1.8) and a thyroid stimulating immunoglobulin level of 160 (reference range <140). β-hCG testing was negative. Patient was diagnosed with subclinical hyperthyroidism. Thyroid testing revealed TSH 0.01 (reference range 0.4-4.2), Free T4 1.3 (reference range 0.8-1.8) and a thyroid stimulating immunoglobulin level of 160 (reference range <140). β-hCG testing was negative. Patient was referred for I-131 treatment.

**Discussion:** In our literature search there have been eight published case reports of Graves’ disease occurring after thyroid surgery. The previous cases reported patients with papillary thyroid cancer developing Graves’ disease after partial thyroidectomy. Unlike the previous case reports our patient was diagnosed with a benign thyroid nodule prior to partial thyroidectomy. Currently the mechanism of developing Graves’ disease after surgery is unknown. One hypothesis is that thyroid epithelial cell destruction during surgery releases TSH receptors. The released TSH receptors may prompt antibody production from the T-cells. An alternative theory suggests that stress from the anesthesia and surgery creates an immunological imbalance.

**Conclusion:** Developing Graves’ disease after thyroid surgery is an exceedingly rare complication. This case suggests that thyroid surgery could be a potential cause of Graves’ disease. Further research is required to identify the exact mechanism of this phenomenon.

**Abstract # 1360**

**THE PREVALENCE OF THYROID CANCER IN HYPERTHYROIDISM, STUDY FROM A TERTIARY ENDOCRINE CENTER IN OMAN**

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**Thyroid Disease**

**Objective:** The presence of thyroid cancer in patients with hyperthyroidism is well described. To date no report is available about the co- existence of hyperthyroidism and thyroid cancer from Arab countries. The purpose of this study was to analyze the clinical relevance of association between thyroid cancer and hyperthyroidism in patients who were treated and followed up in the National diabetes and endocrine center (NDEC) in Muscat, Oman after they underwent total thyroidectomy in the Royal hospital.

**Methods:** The data of 71 Omani patients underwent thyroidectomy in the Royal hospital between the years 2007 to 2013, for hyperthyroidism due to grave’s disease, toxic multi nodular goiter and solitary toxic adenoma were collected from the records of both the hospital and the NDEC. Patients who underwent thyroidectomy for other reasons like non toxic goiter or hypothyroidism with cancer were excluded from the study.

**Results:** Out of the 71 patients, thyroid cancer was identified in 32.8 % (n=23) patients with hyperthyroidism. Papillary thyroid cancer was the main histological type...
detected. Half of these patients 52.1% (n=12) patients had papillary micro-cancer (intra-thyroidal) and three patients with grave’s disease (13%) had lymph nodes metastasis (loco-regional infiltration). Thyroid cancer was detected more (60.8%) in patients with grave’s disease. The cancer preponderance was high in young (82.6%) and female patients (73.9%). The majority of patients with thyroid cancer had abnormal ultrasound neck findings (US) and thyroid scintigraphy (99mTc uptake).

Discussion: Hyperthyroidism does not protect patients from developing thyroid cancer as it was believed earlier TSH has a central role in the thyroid growth and functioning and appears to play same role in the development of thyroid cancer. The true incidence of thyroid cancer in patients with hyperthyroidism may be underestimated especially when treated with long term medical or radioactive iodine therapy. In this study we present the analysis of our experience of thyroid cancer in patients with hyperthyroidism at the National diabetes and Endocrine center .It showed around one third of the patients were affected by thyroid cancer. The cancer was detected mainly in patients with grave’s disease.

Conclusion: A substantial number of patients with hyperthyroidism have thyroid cancer. Clinicians managing patients with hyperthyroidism need to be aware of the possible increased risk of thyroid cancer in hyperthyroid patients , and a proper initial evaluation of these patients is required before taking decision about long term therapy.

Abstract # 1361

EFFECT OF PROPHYLACTIC CENTRAL LYMPH NODE DISSECTION AND RADIOACTIVE IODINE THERAPY ON THYROGLOBULIN LEVELS AND RECURRENCE IN PATIENTS WITH PAPILLARY THYROID CANCER

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Thyroid Disease

Objective: Prophylactic central lymph node dissection (CLND) in addition to total thyroidectomy (TTX) is controversial for patients with papillary thyroid cancer (PTC) when disease is clinically and radiologically limited to the thyroid. With radioactive iodine therapy (RAI) treatment post-operatively, the impact of CLND on thyroglobulin levels and recurrence remains unclear. This study examines the correlation between thyroglobulin levels and recurrence of disease in patients with PTC who underwent TTX with and without CLND, and RAI post-operatively.

Methods: A retrospective review of prospectively collected data from 485 patients with PTC who underwent TTX with or without CLND, and RAI, at a single institution from 2004-2012 was performed. Patients were stratified into two groups: Group 1 patients underwent TTX only; and Group 2 patients underwent TTX and CLND; all underwent RAI post-operatively. Thyroglobulin levels were compared between groups in the immediate post-operative period and post-RAI treatment. Patients were followed for a minimum of two years, and PTC recurrence was compared between the two groups.

Results: A total of 187 patients met inclusion criteria. 93 patients (49%) in group 1 and 94 patients (51%) in group 2. There was a significant difference in the thyroglobulin levels pre-RAI compared to post-RAI treatment in each group (group 1, p=0.0051; group 2, p=0.002). But post-operative thyroglobulin levels were not significantly higher for group 1 (13.04 ng/mL) compared to group 2 (7.87 ng/ml) (p=0.2125). In addition, there were no significant differences in mean thyroglobulin levels between group 1 (1.91 ng/ml) and group 2(1.89 ng/mL) (p=0.988) post-RAI treatment. Finally, there was no significant difference seen in PTC recurrence between group 1 (2.7%) and group 2 (4.6%) (p=0.46).

Discussion: Differences in thyroglobulin levels were not appreciated in PTC patients who underwent TTX alone compared to patients who underwent TTX with CLND in both the immediate post-operative period and post-RAI treatment. RAI treatment significantly lowered thyroglobulin levels, independent of CLND. Recurrence was also not significantly different between the two groups.

Conclusion: RAI treatment can negate prophylactic CLND. However, with the current trends of using less RAI to treat PTC, a CLND may be necessary for disease control and recurrence prevention.
Abstract # 1362

THE PREVALENCE OF COEXISTENCE THYROID CARCINOMA AND THYROID NODULES IN HYPERTHYROID PATIENTS OF SAN JUAN CITY HOSPITAL

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Thyroid Disease

Objective: Hyperfunctional nodules of the thyroid are rarely associated with thyroid cancer, for these reason are rarely biopsied. Although multiple theories have being proposed, the relationship is still uncertain. After performing a MEDLINE literature search, we found in multiple retrospectives analysis, that patients with hyperthyroidism with hyperfunctional nodules have an estimated of 0.3% to 16.3% prevalence of malignancy.

Results: In our study forty-eight hyperthyroid cases were prospectively investigated to provide information about the association between hyperthyroidism and thyroid cancer. Historical, biochemical and radiological characteristics of the case subjects and their nodules were also analyzed. All nodules greater than 1cm in diameter, nodules 5-10 mm size diameter if they had calcification were fine-needle biopsied (FNAB) under ultrasound guidance. The biopsy samples were cytologically asses (by the BETHESDA classification) and we found 77% of benign nodules, 2% of nodules presented with atypia of undetermined significance or follicular lesion of undetermined significance, 8.3% were malignant nodules, and 10.4% were nondiagnostic or unsatifactory. All patients with a biopsy diagnosis of malignant underwent surgery. Thyroid malignancy (micro- or macrocarcinoma) diagnosed pre-operatively in all 4 cases by US-guided FNAB was confirmed by the pathology obtained after the surgery. Papillary thyroid carcinoma was identified in 2 patients (4.17%), and Follicular thyroid carcinoma was found in 2 patients (4.17%). These data demonstrate a higher than expected incidental cancer rate in hyperthyroid patients compared with euthyroid subjects with nodular goiter.

Conclusion: Our purpose is to stress the point that, although hyperfunctioning thyroid nodules are rarely described as malignant in the literature, FNAB should not be restricted to cold nodules, in view of our data and others published reports.

Abstract # 1363

TALL CELL VARIANT OF PAPILLARY THYROID CANCER TREATED WITH MULTIPLE ROUNDS OF RADIOIODINE THERAPY

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Thyroid Disease

Case Presentation: 45 year old Hispanic man with no past medical or family history and no risk factors, presented with dysphagia and hoarse voice for 2 years. Exam was unremarkable except for an enlarged, hard and asymmetric thyroid with decreased vocal cord motility on laryngoscopy. Ultrasonography revealed an enlarged, heterogeneous gland with microcalcifications and bilateral, hypervascular lymphadenopathy. CT showed an enlarged, heterogeneous thyroid with coarse calcifications and bilateral level I-VI lymph nodes (LN) with increased enhancement and low attenuation. Ultrasound guided fine needle aspiration (FNA) of the thyroid revealed clusters of atypical follicular cells, favoring follicular neoplasm. FNA of LNs showed clusters of thyroid follicular cells suggestive of nodal metastasis. Total thyroidectomy with neck dissection and debulking of involved paratracheal soft tissues and neck musculature was performed without complication. Pathology revealed diffuse, infiltrating papillary thyroid carcinoma (PTC), tall cell variant, involving entire thyroid, extending to bilateral paratracheal soft tissue and striated muscle, with 19/21 LNs positive for tumor (T4a, N1b, Mx). RAI Whole Body Scan (WBS) showed foci of increased uptake in thyroid bed, neck and diffuse uptake throughout both lungs, consistent with Stage IVC disease. There was no evidence of neoplastic range, hypermetabolic activity on FDG PET scan. Following thyroxine withdrawal, he was treated with 209mCi of I131, with post ablation WBS showing widespread pulmonary and mediastinal metastases. During thyroxine suppression therapy there was no sonographic evidence of residual thyroid tissue or suspicious lymph node recurrence. Thyrogen stimulated thyroglobulin was elevated to 45ng/mL with negative antibodies, and withdrawal WBS showed thoracic metastases in both lung fields and mediastinum. He was treated again with 256mCi of I131, for a total dose of 465mCi. Post ablation WBS again showed pulmonary metastases. Thyroxine suppression was continued and 6 months post RAI, Thyrogen stimulated thyroglobulin.
declined to 13ng/mL and stimulated WBS showed no radiotracer uptake. Over the next 2 years of thyroxine therapy, stimulated thyroglobulin declined to 3ng/mL with no evidence of recurrence on imaging.

**Discussion:** Tall cell, is a rare and aggressive variant of PTC that is usually metastatic at diagnosis and can be non-RAI avid. It is, however, a form of differentiated thyroid carcinoma (DTC) and when initial approach follows accepted guidelines desired results can be achieved.

**Conclusion:** Despite aggressive variants, distant metastasis, and lack of initial response to RAI, DTCs can be responsive to multiple rounds of RAI therapy.

**Abstract # 1364**

**“HASHIMOTO’S ENCEPHALOPATHY”: SYNDROME OF CONFUSION TO PATIENTS AND DIAGNOSIS OF EXCLUSION FOR PHYSICIANS**

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**Thyroid Disease**

**Objective:** Hashimoto’s encephalopathy is a rare syndrome that is not easily identified due to its wide-ranging presentation along with absence of specific and sensitive diagnostic studies.

**Case Presentation:** A 29-year old male presented with acute onset of seizures along with loss of consciousness for the first time. While in the hospital he had witnessed seizures and described as generalized tonic clonic. No previous head trauma, psychiatric illness, or drug abuse. He was alert but disoriented. Neurological exam was within normal. Routine workup was within normal. Urine drug screen was negative. MRI of head was normal and lumbar puncture showed elevated CSF protein with no evidence of infection. HSV and West Nile were negative. Initially, he was treated with anti-seizure medications but showed no improvement. He continued to have seizures, spells of agitation and odd behaviors including undressing himself. Bedside EEG showed no epileptiform activity; however, 24 hour EEG monitoring revealed generalized slowing. Anti-seizure medications were discontinued. He was started on a trial of steroids but remained confused. Syphilis IgG, HIV, hepatitis panel, lead, arsenic, and mercury were negative along with antibodies against AMPA, GABA-B and NMDA. While the patient had an elevated TSH, the low T3 and elevated antithyroglobulin level raised concerns for Hashimoto’s encephalopathy. He was started on levothyroxine, liothyronine, and high dose steroids. Patient showed progressive improvement back to his cognitive baseline and continued to improve. He was seen two months later with complete resolution of his symptoms.

**Discussion:** Hashimoto’s encephalopathy is a rare syndrome of encephalopathy with elevated antithyroid antibodies that responds well to steroid. It has variety of presentation ranging from seizures, to stroke-like symptoms, behavioral and cognitive changes. Furthermore, sterile CSF with an elevated protein, generalized slowing on EEG, normal imaging and elevated antithyroid antibodies, are indicative of the diagnosis. Management is dependent upon early suspicion, testing for antithyroid antibodies and high dose steroids. Studies have shown that early diagnosis along with aggressive steroid treatment has better outcomes and complete remission was achieved in 80% of cases.

**Conclusion:** Hashimoto’s encephalopathy is a challenging diagnosis and needs a high level of suspicion. Nevertheless, debate still persist due its broad clinical spectrum, nonspecific laboratory findings, along with lack of definite pathology. Further studies into the underlying pathophysiology will be helpful in guiding diagnosis and management.

**Abstract # 1365**

**MEDULLARY THYROID CARCINOMA PRODUCER MELANIN**

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**Thyroid Disease**

**Objective:** We present a case of Medullary Thyroid Carcinoma (MTC) producing melanin, coexisting with papillary thyroid carcinoma (PTC).

**Methods:** We report the clinical features and analysis of a patient with coexistence of PTC and CMT.

**Case Presentation:** Female 64 yo, with a condition 3 months, noticed tumor in right anterior cervical region; denies dysphagia , dyspnea, hoarseness , sore throat and diarrhea. Take 75ug of LT4. Analysis:TSH:0.055uU/mL; Thyroid Ultrasound:RTL 38x10x14mm, with homogeneous parenchyma and focal hypoechoic lesion of 4mm without vascular representation, LTL occupied by nodular lesion measuring 41x41x43mm spherical heterogeneous diffuse vascularity and sharp edges, no cervical lymphadenopathy. FNA:cystic tumor with atypia. She underwent total thyroidectomy with resection of nodal center compartment. Pathology review: LTL: CMT producing melanin (6x4.5x4cm), with extension to thyroid capsule with lymphatic tumor emboli, no venous vascular
emboli or perineural infiltration, IHC:Calcitonin(+), thyroglobulin(-), TTF-1(-), CK high molecular weight(-), Congo Red(+), focal. In RTL papillary microcarcinoma. Classic variety of 0.3cm diameter capsule was found partially infiltrates. Thyroid remnant: chronic thyroiditis, 6 nodes without metastases. Prior to radioiodine: TSH:>75μU/mL, Tg < 0.20, TgAb/AbTPO(-), Calcitonin 33.3pg/ml. CEA:4.5

Discussion: The MTC corresponds to 0.3% of nodular goiter and 3% of the thyroid malignancies diagnosed. In most cases, the preoperative diagnosis is safe, based on FNA (sensitivity: 30-50%), calcitonin measurement and study of the RET (not available for our environment). In 10-15% are diagnosed in the pathology of total thyroidectomy. The concurrence of MTC and PTC is rare, have been reported in the literature 20 cases. The CMT have a confirmed ability multidirectional differentiation, tumor cells can secrete their hormonal and nonhormonal substances, products including mucin, peptides and amines. The prognosis for this variant is not known exactly, due to shortage of presentation of the reported cases, not yet I infer a behavior, not being able to determine whether aggression was mitotic activity of CMT per se or the presence this contributes to melanin.

Conclusion: MTC producing melanin is extremely rare, since 1982 to 2008 have been reported in the literature 10 cases. The respective be tracked in order to contribute to the knowledge of the behavior of this variant of CMT.

Abstract # 1366

A CASE OF MYXEDEMA COMA COMPLICATED BY SEVERE CARDIOMYOPATHY

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Thyroid Disease

Objective: To discuss the presentation, pathogenesis and management of cardiomyopathy in Myxedema Coma.

Methods: We review a rare case of Myxedema Coma with cardiomyopathy and discuss the relevant literature regarding pathogenesis, treatment and outcomes.

Case Presentation: A 49 year old female without significant medical history was admitted for 3 weeks of worsening dyspnea, lethargy, hypothermia, bradycardia, hypoxemic and hypercarbic respiratory failure, and hypotension. Physical examination revealed disorientation, dry skin, alopecia, peri-orbital and peri-oral edema and pre-tibial myxedema. Labs were remarkable for hyponatremia, TSH 52, Free T4 0.18, and T3 0.00. She was intubated and started on intravenous Dopamine and Dobutamine drips for hemodynamic stability. Levothyroxine and Lithotrybine were administered intravenously. Bedside echocardiogram revealed diffuse hypokinesis with a 20% ejection fraction, so digoxin was initiated. Empiric hydrocortisone was briefly administered for supportive measure. Symptoms and vitals improved, so vasopressors were tapered off and our patient was successfully extubated. Ejection fraction improved to 25% and she was discharged with outpatient follow-up.

Discussion: Myxedema Coma is a rare medical emergency with a 25-60% mortality rate. It is a severely decompensated hypothyroid state manifesting with multi-organ suppression. Thyroid hormone replacement, supportive care and hemodynamic support are the primary treatments. Cardiomyopathy is a rare feature, as in our case. In PTU-induced hypothyroid rats, Tang et al observed an increased left ventricular systolic diameter, reduced ejection fraction by up to 36% and a reduction in wall thickness and myocardial blood flow. Ladenson et al. reported a decrease in cardiac α-myosin heavy chain mRNA levels secondary to thyroid hormone deficiency, and a reversal of cardiomyopathy within 9 months after a euthyroid state was achieved.

Conclusion: Myxedema Coma is a state of severe thyroid decompensation, which rarely presents with reversible cardiomyopathy, as in our case. Studies suggest that thyroid hormones play a role in gene expression of cardiac α-myosin heavy chain, and appropriate thyroid hormone replacement in myxedema may reverse the cardiomyopathy. It is therefore important to consider severe hypothyroidism in patients with unexplained heart failure, as this may be reversible.

Abstract # 1367

REFRACTORY HYPOTHYROIDISM FROM PSEUDOMALABSORPTION- DIFFERENTIATING FACT FROM FACTITIOUS

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Thyroid Disease

Case Presentation: A 41 year old female with hypothyroidism presented with generalized muscle cramping, constipation and hair loss. Patient was on 350 micrograms of Levothyroxine per day. Her labs suggested bio-chemical hypothyroidism. History was
negative for lactose intolerance, prior gastro-intestinal surgery, malabsorption symptoms, over the counter medications use. Physical exam was normal. Human anti-mouse antibody test (HAMA) to detect any antibodies which could possibly interfere with the thyroid function tests (TFT) was negative. Subsequently she underwent levothyroxine absorption test which showed –Thyroid stimulating hormone (TSH) - 40.9 uU/mL (normal 0.5-5 uU/mL), Free thyroxine (FT4), at 0 hours – 1.1 ng/dL (normal - 0.8-2.8 ng/dL), FT4 (1hour) – 2 ng/dL, FT4 (2hours) -3.1 ng/dL, FT4 (4hours) – 3.3 ng/dL and anti microsomal antibody was 13.2 IU/mL (normal - 0-35 IU/mL) suggesting psuedomalabsorption. On further questioning the patient admitted taking her thyroid medications along with food to avoid stomach upset. She underwent counseling and had improved TFT’s and symptoms after resuming her usual dose of oral thyroxine.

Discussion: Work up of persistent clinical and biochemical hypothyroidism despite high doses of thyroid supplementation/replacement should entail meticulous history taking, checking for malabsorption symptoms and ruling out any possible drug interactions. Pseudomalabsorption which is a factitious disorder very common yet not identified timely thereby delaying the right management. The test is based on the bioavailability of oral levothyroxine which is about 64% in non-fasting and 79-81% in fasting state and the t max(time from administration to reach maximum plasma concentration) of 2–4 hrs. Test is started after loading 1000 micrograms of oral levothyroxine under supervision with close vitals monitoring. Baseline TSH and FT4 with subsequent hourly FT4 for next 4 hours is measured. Rise in the FT4 towards end of test helps rule out true malabsorption with good certainty. Testing with lower doses of lower doses of 500 mcg or 750 mcg should be considered in elderly and those with pre-existing cardiac conditions. The test is not standardized and influenced by conditions like mucosal edema from congestive heart failure, liver cirrhosis, nephrotic syndrome and overt hypothyroidism etc. We however emphasize that if used in the right context as discussed above, it can serve as a very useful tool in identifying pseudomalabsorption and saving time and cost by avoiding further unnecessary testing or initiating parenteral levothyroxine.

Conclusion: We conclude that our patient had refractory hypothyroidism secondary to pseudomalabsorption.

Abstract # 1368

HYPOTHYROIDISM WITH ANTIBODY INTERFERENCE IN THYROID ASSAY: A CASE REPORT

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Thyroid Disease

Objective: To report a case of an assay interference in free T4 (FT4) levels complicating the course of management.

Case Presentation: A 46 YO female presented to the endocrine clinic with an abnormal thyroid panel. Her most recent labs showed TSH of 15.56 mIU/L (0.34-5.60) and FT4 of 2.52 ng/dL (0.58-1.64). She has been taking Levothyroxine for many years and had been taking 100 mcg daily for last 2 years. She denied heart palpitations, sweating, anxiety, heat intolerance or chest pain. She also reported having a goiter since age 11 and abnormal lab findings for many years. A significantly elevated TSH and FT4 were measured on multiple tests performed at Diagnostic Lab and Levothyroxine was initially discontinued. Repeat thyroid panel after discontinuing Levothyroxine for 4 weeks showed elevation in TSH to 26.71 mIU/L (0.34-5.60) and FT4 of 1.97 ng/dL (0.8-1.8). Levels of SHBG and an alpha subunit were measured to evaluate for TSH secreting pituitary adenoma and were found to be negative. Heterophile mono screen measuring antibody level was also negative. Interference with immunoassay was suspected at the Diagnostic Lab. Unfortunately, the patient was lost to follow up for 6 months and was taking Levothyroxine 137 mcg/day on her return visit. To evaluate for interference, samples collected on the same day were sent to two different labs. Her thyroid panel at Diagnostic Lab showed TSH of 12.99 mIU/L (0.34-5.60) and FT4 of 2.61 ng/dL (0.58-1.64) while Quest Diagnostics showed TSH of 9.91 mIU/L (0.40-4.50) and FT4 of 1.1 ng/dL (0.8-1.8). Results from Quest Diagnostics confirmed our hypothesis of an assay interference. Both labs were using chemiluminescence immunoassay technique but different automated system platform. A diagnosis of hypothyroidism was confirmed and her repeat thyroid function tests were within the normal reference range after adjusting levothyroxine dose accordingly.

Discussion: Interference from serum antibodies with immunoassay has been reported in the past but not many cases have reported interference in free T4 measurements while using similar immunoassay techniques on different automated systems. Prevalence of
immunoassay interference is variable and it depends on the specific analyte and assay techniques. Ismail A et al (2002) reported a prospective study of 5310 cases with an antibody interference in 0.53% of cases and results found to be significant enough to potentially affect cost and clinical care. This case illustrates the difficulties and limitations of management when faced with immunoassay interference.

**Conclusion:** Interference of immunoassays should be considered when evaluating patient with abnormal lab findings without clinical symptoms to avoid unnecessary investigation.

**Abstract # 1369**

A CASE OF RADIATION-INDUCED THYROIDITIS AND WARTHIN-LIKE PAPILLARY ADENOCARCINOMA IN A HYPERTHYROID ADOLESCENT AFTER RADIOACTIVE IODINE THERAPY

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**Thyroid Disease**

**Objective:** We present an unusual case of an adolescent young woman who underwent radioactive iodine ablation for hyperthyroidism, who subsequently developed thyroiditis and Warthin-like papillary adenocarcinoma of the thyroid.

**Case Presentation:** A 15-year-old young Caucasian woman presented with occasional difficulty swallowing and was found to have goiter and hyperthyroidism. An initial radioactive iodine scan showed 40.8% uptake, consistent with hyperthyroidism; however, her TSH was not suppressed. Medical therapy made little clinical difference. About six months later, her thyroid was 1.5 to 2 times normal size, but she had no clinical signs of hyperthyroidism aside from a mild palmar tremor. Both thyroid peroxidase and thyroglobulin antibodies were very significantly elevated. Another six months later, the patient noted increased neck swelling but denied any other symptoms. A subsequent radioactive iodine scan showed 69% uptake. She was then treated with I-131 therapy. As expected, her thyroid stopped functioning, but her thyroid peroxidase and thyroglobulin antibodies remained very elevated two months later, consistent with thyroiditis. After her thyroid initially decreased in size following the ablation, it enlarged to about 4-5 times normal size by clinical exam, two months post-procedure. Thyroid ultrasound showed some hypervascularity but no discrete mass. She was placed on high-dose thyroid replacement therapy. As the patient continued to complain of neck swelling, she had an MRI done, which showed a heterogeneous thyroid gland containing multiple small nodules and a 1.5-cm nodule involving the thyroid isthmus. At age 17 she underwent a total thyroidectomy, which revealed papillary adenocarcinoma, Warthin-like variant, in the left lobe and isthmus. Margins were negative, lymph nodes were negative (6/6), and there were no distant metastases. In addition, advanced lymphocytic thyroiditis was observed.

**Discussion:** Papillary thyroid adenocarcinoma is a common malignancy occurring in patients with a history of radiation exposure. This has been best characterized in individuals, especially young children, exposed to chronic environmental radiation, such as from nuclear power plant fallout. The risk of thyroid carcinoma from I-131 therapy has been shown to be quite small, especially because the high dose of radiation minimizes the persistence of residual thyroid tissue. The Warthin-like variant is characterized by extensive lymphocytic infiltration, which in this patient’s case likely correlates with her radiation-induced thyroiditis.

**Conclusion:** Though unusual, there remains a small probability of thyroid malignancy developing after radioactive iodine therapy, especially in younger patients.

**Abstract # 1370**

ISOLATED METASTASIS OF RENAL CELL CARCINOMA TO THYROID GLAND TWENTY YEARS FOLLOWING NEPHRECTOMY

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**Thyroid Disease**

**Objective:** Presented here is an interesting rare case of metastatic renal cell carcinoma to the thyroid gland.

**Case Presentation:** The case presented here is about a 68 year-old lady with a history of renal cell carcinoma for which she underwent left nephrectomy in 1994, and that according to the patient, she has been in remission ever since, and did not require additional therapy. The patient presented over the last 2 to 3 years with a progressively enlarging left sided thyroid mass, measuring over 5 cm in maximum diameter. This mass was biopsied twice: in 2011 and in 2013, and both were suggestive of a benign thyroid nodule and no malignancy was reported. However, due to compressive neck symptoms and the size of her mass, thyroidectomy was recommended. Total thyroidectomy performed in the earlier part of 2014, and
the pathology confirmed the presence of metastatic renal clear cell carcinoma. Interestingly, post operatively, PET CT and MRI brain performed, and no metastatic disease was evident elsewhere in her body.

**Discussion:** The thyroid gland is one of the most vascular organs in the body and therefore, it would be expected to be the site of where several primaries could be seeding to. Breast cancer and melanoma were reported among the primary neoplasms that would metastasize to the thyroid gland. Renal cell carcinoma has been reported too, and it could happen during the same time of the primary lesion, or several years later. However, isolated metastasis of renal cell carcinoma to the thyroid gland is exceedingly rare.

**Conclusion:** Metastasis of renal cell carcinoma to the thyroid gland is very rare; however, it would need to be strongly considered in the proper clinical setting. Our case was interesting in a sense that metastasis of the renal cell carcinoma in this patient appeared almost 20 years following her nephrectomy, and that enlights how indolent could be renal cell carcinoma. Furthermore, there was no evidence of metastatic disease elsewhere in her body. Another interesting fact is, although the mass was biopsied twice and no malignancy was evident, a strong clinical suspicion should be considered even with utilization of very accurate diagnostic modality such as FNA, especially in progressively growing lesions and the presence of compressive neck symptoms. Lastly, one would need to interpret biopsy result of large thyroid lesions with caution, as the accuracy would depend on the place in the lesion where sampling was obtained.
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