function and the patient was discharged home with plans for emergent thyroidectomy.This case demonstrates the importance of considering plasmapheresis as a life saving measure in the treatment algorithm of patients with extreme thyrotoxicosis and thyroid storm in which traditional therapies such as thionamides, steroids, and beta-blockers cannot be used or are ineffective. Reference:Ono, Y., Ono, S., Yasunaga, H., Matsui, H., Fushimi, K., & Tanaka, Y. (2016, February 18). Factors Associated With Mortality of Thyroid Storm: Analysis Using a National Inpatient Database in Japan. Retrieved April 6, 2019, from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4998648/

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS I

A Genetic Cause of Primary Hyperparathyroidism

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SAT-334

Background: Primary hyperparathyroidism is a relatively common endocrine condition, affecting up to 7 out of every 1000 adults. Median age of onset is during the sixth decade of life. When present in younger patients primary hyperparathyroidism may indicate an underlying genetic cause. Case: A 23-year-old woman is referred for evaluation of hypercalcemia that was found on labs drawn after surgery for bilateral ovarian cyst removal. At the time of presentation she felt well and was without complaint. Her medical history is otherwise unremarkable. Her family history includes multiple family members with nephrolithiasis. Physical exam revealed a well appearing Caucasian woman without a palpable neck mass. Laboratory results showed: serum calcium 11.7 mg/dL (ref 8.4–10.2), ionized calcium 1.44 mmol/L (ref 1.12–1.32) and serum PTH 192 pg/mL (ref 11–65). Technetium-99 sestamibi scan revealed a low attenuating mass with focal and persistent uptake just inferior to the left thyroid lobe. She subsequently underwent left inferior parathyroidectomy, which confirmed parathyroid adenoma, with resultant normalization of serum calcium and PTH. Due to her young age of diagnosis genetic testing was performed which revealed a mutation if CDC 73. Discussion: CDC 73 mutation is known cause of inheritable neoplasia's with a high prevalence of parathyroid dysfunction. Although penetrance and expression is variable, the mutation is associated primarily with Hyperparathyroidism Jaw Tumor Syndrome (HPT-JT), Familial Isolated Hyperparathyroidism and sporadic parathyroid carcinoma. Our patient had an allele mutation associated with HPT-JT. This syndrome classically presents with parathyroid adenoma, ossifying tumors of the mandible and renal or uterine neoplasms. The mutation is inherited in an autosomal dominant pattern and family history plays a key role in diagnosis of this rare condition. This patient had an early age of onset for hyperparathyroidism and a family history suggestive of an inherited calcium metabolism disorder. A single parathyroid adenoma is the most common presentation and surgical resection is often curative of hyperparathyroidism. In Hyperparathyroidism Jaw Tumor Syndrome patients classically develop ossifying tumors of the mandible and renal or uterine neoplasms in addition to parathyroid lesions. Rarely patients with CDC 73 mutation can develop parathyroid carcinoma. For this reason it is recommended that all first degree relatives undergo testing for CDC 73 mutation. This patient currently has no evidence of jaw, uterine or renal tumors on screening imaging. Ovarian tumors have been described separately to be associated with CDC 73 mutation, this patient may actually have an unrecognized phenotype of CDC 73 mutation. Given the potential impact of inheritable neoplasia, all young patients with unexplained hyperparathyroidism should be considered for genetic screening.

Diabetes Mellitus and Glucose Metabolism

DIABETES DIAGNOSIS, TREATMENT AND COMPLICATIONS

Prediction of Hypertension, Diabetes and Fractures in Eucortisolemic Women by Measuring Parameters of Cortisol Milieu

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SUN-614

Purpose. Cortisol secretion, peripheral activation and sensitivity seem to be associated with hypertension (HY), type-2 diabetes (T2D) and fragility fractures (FX) even in eucortisolemic subjects. The aim of the present study was to determine the cut-off(s) of the parameters of cortisol secretion and peripheral activation for predicting the presence of HY, T2D and FX (comorbidities). **Methods.** In 206 postmenopausal females (157 with ≥1 comorbidities and 49 without any), we assessed the ratio between 24-hour urinary free cortisol and cortisone (R-UFF/UFE, cortisol activation index), cortisol after 1mg-overnight-dexamethasone (F-1mgDST, cortisol secretion index), and the GC receptor N363S single-nucleotide polymorphism (*N363S*-SNP, cortisol sensitivity index). **Results**. The cut-offs for F-1mgDST and R-UFF/UFE set at 0.9 μg/dL (Area Under the Curve, AUC 0.634±0.43, p=0.005) and 0.17 (AUC 0.624±0.5, p=0.017) respectively, predicted the presence of ≥1 comorbidities. The presence of F-1mgDST >0.9 μg/dL *plus* R-UFF/UFE >0.17 showed 82.1% specificity for predicting the presence of ≥1 comorbidities, while the simultaneous presence of F-1mgDST ≤0.9 μg/dL and

R-UFF/UFE ≤0.17 showed 88% sensitivity for predicting the absence of comorbidities. The F-1mgDST >0.9 μg/dL or R-UFF/UFE >0.17 was associated with 2.8 and 2.1 fold increased risk of having ≥1 comorbidities, respectively. The F-1mgDST ≤0.9 μg/dL *plus* R-UFF/UFE ≤0.17 or F-1mgDST >0.9 μg/dL *plus* R-UFF/UFE >0.17 was associated with 2.8 fold reduced or 4.9 fold increased risk of having ≥1 comorbidities regardless of age, BMI and *N363S*-SNP. **Conclusions.** F-1mgDST >0.9 μg/dL and R-UFF/UFE >0.17 may be used for predicting the presence of ≥1 among HY, T2D and fragility FX.

Adrenal

ADRENAL CASE REPORTS II

Giant Adrenal Myelolipoma Associated with Prolonged ACTH Elevation in a Patient with Congenital Adrenal Hyperplasia (CAH)

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SUN-197

Background: Myelolipomas are rare benign tumours which consist of adipose tissue and mature hematopoietic tissue. The incidence ranges from 0.08 to 0.4% and they constitute 15% of adrenal incidentalomas. We report the case of a massive adrenal myelolipoma in a patient with CAH. **Clinical Case:** A 51year old man with salt losing CAH due to 21 hydroxylase deficiency presented to the emergency department with abdominal pain. CT scan of the abdomen revealed a 15 x 16 x 19cm mass in the left adrenal gland. There were concerns about malignancy, however expert radiology review diagnosed a massive adrenal myelolipoma. The patient was diagnosed to have salt losing CAH during infancy and managed with cortisone acetate and fludrocortisone during childhood, then dexamethasone 0.5mg nocte, 0,25mg mane as an adult. At 27 years he was reviewed and found to be Cushingoid with abdominal striae, obesity and hypertension. A CT scan of the abdomen done at that time showed a normal left adrenal gland with nodular right adrenal gland. While he was on dexamethasone, his 17-OHP was 14 nmol/l (NR: 0–6.0 nmol/l). His dexamethasone was reduced, then he was transferred on to prednisolone and then to hydrocortisone. 17-OHP level and ACTH levels have risen with each change in medication. At the time of referral with abdominal pain, he was on hydrocortisone 10 mg mane, 10 mg midday and 5mg at 1700h and his biochemistry showed: ACTH 486ng/l (NR 7.2–63.3), 17-OHP 101.1nmol/l (0–6.0nmol/l), renin 5.5 nmol/l/hr (NR 0.2–3.3) and androstenedione 26.6 nmol/l (NR 0–7.8). He underwent laparoscopic hand-assisted left adrenalectomy with the removal of a 2030 gram adrenal myelolipoma. He made an uneventful recovery with remarkable clinical improvement in general wellbeing. Histology confirmed adrenal myelolipoma. **Conclusion:** Chronic exposure to high ACTH levels causes metaplasia of adrenocortical cells, but a direct causal relationship for myelolipomas is not established. The role of ACTH in development of myelolipomas is supported by the increased incidence in patients with Addison's disease, Nelson's syndrome and ACTH dependent Cushing Disease. Our case supports the role of ACTH as the myelipoma developed after the reduction of patient's glucocorticoid treatment and ACTH levels were high for years. Myelolipomas are usually asymptomatic but can present with abdominal pain, spontaneous rupture, and spontaneous retroperitoneal haemorrhage. There are no set guidelines but for large and rapidly growing tumours, surgery may be considered..

Pediatric Endocrinology

PEDIATRIC GROWTH AND ADRENAL DISORDERS

The Natural History of Pituitary Cysts in Patients with Growth Hormone Deficiency and Idiopathic Short Stature

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SAT-105

Background: The sequential follow-up of simple fluid-filled pituitary cysts (PC) has not been fully elucidated. In this study, we further report our follow up of PCs in a cohort of pediatric patients (PTs).

Objective: To further analyze the sequential cyst volume (CV) change in short children.

Patients and Methods: A pediatric endocrinology and neuroradiology center was queried for the presence of PCs. PTs who underwent multiple high resolution post-contrast MRIs (1mm slices) were subjects of this study. PTs with additional MRI abnormalities were excluded. Pituitary volumes (PV) and CVs were measured using the ellipsoid formula (LxWxH/2). The percentage of the gland occupied by the cyst (POGO) was measured and calculated. A cyst with a POGO ≤15% was defined as a small pituitary cyst (SPC), and a POGO >15% was defined as a large pituitary cyst (LPC). 34 PTs met inclusion criteria, all of whom were diagnosed with short stature (23 growth hormone deficient (GHD) PTs and 11 idiopathic short stature (ISS) PTs). All PTs were receiving GH during data collection.

Results: The mean (MN) and median (MD) ages for these subjects were 10.7 yrs ±3.5 and 11.1 yrs, respectively (RSP). Of the 34 PTs, 24 PTs' (71%) initial MRI demonstrated a SPC and 10 PTs' (29%) initial MRI demonstrated a LPC.

The MN and MD times between first and second MRIs were 1.23 yrs and 0.83 yrs RSP, with a range (RG) of 0.14 to 4.08 yrs.

The MN and MD ΔCV for all PTs was 23.33% ±179.17% and -25.94% RSP, with a RG of -100.00% to 763.94%.

The MN and MD ΔPOGO by the cyst for all PTs was 48.59% ±313.26% and -36.84% RSP, with a RG of -100.00% to 1734.79%.