[1]St Vincents University Hospital, Dublin 4, Ireland, [2]St Vincent's University Hospital, Dublin, Ireland.

SAT-185

Introduction: Primary adrenal lymphoma (PAL) is a rare cause of adrenal enlargement with approximately 200 cases reported in the literature to date. It tends to affect elderly men and has a high incidence of bilateral involvement at diagnosis. We report the case of a 66 year old man, whose PAL manifested with symptomatic hypercalcaemia.

A 66 year old male, originally from the Philippines, was referred to the emergency department with nausea, vomiting, weight loss and right flank pain. His past medical history was significant for hypertension, gout and stage 3b chronic kidney disease. His medications were amlodipine, losartan and febuxostat. His family history was significant for hypertension. On examination he was hypertensive (blood pressure, 160/100 mmHg) and hyperpigmented. His laboratory investigations revealed; corrected calcium of 3.79 mmol/l, undetectable PTH, vitamin D 49 nmol/l. He was treated with intravenous (IV) 0.9% saline and IV zoledronic acid and his calcium levels improved.

To investigate causes of non-PTH mediated hypercalcaemia, computerised tomography of the thorax, abdomen and pelvis (CT TAP) as well as a positron emission tomography (PET) scan were performed. These demonstrated bilateral, large, metabolically active adrenal masses with no evidence of extra-adrenal disease. Differential diagnosis at this point included bilateral adrenal hyperplasia, metastases, lymphoma or adrenal TB. There were no radiological features of adrenocortical carcinoma (ACC) or phaeochromocytoma and subsequent biochemical investigations confirmed no evidence of cortisol, androgen or catecholamine excess. Adrenocorticotrophic hormone (ACTH) levels were elevated however, and a synacthen test revealed inadequate adrenal reserve (peak cortisol 214 nmol/l). The patient was commenced on maintenance steroids and with stress dose steroid cover, proceeded to adrenal biopsy. Histology confirmed diffuse large B cell non-Hodgkin's lymphoma. Haematology became involved in his care and he commenced polychemotherapy in the form of R-CHOP, 1 week post confirmation of the diagnosis. His treatment is ongoing and he has tolerated it well with minimal side effects, except a flare of gout.

Learning points: PAL should be considered in the differential diagnosis in patients with bilateral adrenal masses. Image guided adrenal biopsy is the gold standard for diagnosis, though caution must be exercised and an ACC or phaeochromocytoma should be excluded prior to biopsy. The prognosis of PAL is poor and therefore early diagnosis and prompt initiation of treatment are required to improve outcomes.

Thyroid

BENIGN THYROID DISEASE AND HEALTH DISPARITIES IN THYROID II

Risk Factors Associated with Thyroid Nodules in Type 2 Diabetes

Sonia Gisella Chia Gonzales, MD[1], victor noriega ruiz, MD[1], Miguel Eduardo Pinto, MD, MSc, FACE[2], eduardo pretell zárate, MD[1], Segundo Seclen Santisteban, MD[3].

[1]CAYETANO HEREDIA HOSPITAL, lima, Peru, [2]Cayetano Heredia Hospital, Endocrine Service, Lima, Peru, [3]Sociedad Peruana de Endocrinologia, Lima, Peru.

SUN-422

Objective: To determine risk factors associated with the presence of thyroid nodules in patients with type 2 diabetes

Material and methods: A case control prospective study, matched 1:1, according to age, sex and smoking habit was conducted. Patients with type 2 diabetes were recruited from the Endocrinology Service of Cayetano Heredia Hospital, Lima-Perú. Clinical evaluation, laboratory tests and thyroid ultrasound were performed to indentify patients with type 2 diabetes with and without thyroid nodules. The association was evaluated by calculating Odds ratios (OR) by conditional multivariate logistic regression techniques.

Results: 83 patients with type 2 diabetes and thyroid nodule and 83 patients with type 2 diabetes without thyroid nodule; were obtained. The risk factors associated with the presence of thyroid nodules in patients with type 2 diabetes were HbA1c (OR= 4.12, P= 0.002), BMI (OR= 1.13, P 0.030) and TSH (OR= 3.27, P= 0.0001), with cut-off points according to ROC curve of HbA1c ≥ 8%, TSH in the normal upper limit ≥ 2.3 μIU / mL and BMI ≥ 27 kg / m2. Patients with type 2 diabetes and thyroid nodule had higher values of blood glucose, thyroid volume, abdominal and neck circumference; greater frequency of acantosis nigricans and acrochordons compared to controls.

Conclusions: The risk factors associated with the presence of thyroid nodules in patients with type 2 diabetes were HbA1c, BMI and TSH, with cut-off points according to ROC curve of HbA1c ≥ 8%, TSH in the normal upper limit ≥ 2.3 μIU / mL and BMI ≥ 27 kg / m2.

Reproductive Endocrinology

TRANSGENDER MEDICINE AND RESEARCH

Cardiometabolic Effects of Cross-Sex Hormone Therapy in Transgender Patients

Lana Kosi Trebotic, MD[1], Carola Deischinger, MD[1], Anita Thomas, BSc[1], Ivica Just-Kukurova, PhD[2], Siegfried Trattnig, Prof, MD[2], Ulrike Kaufmann, MD[3], Alexandra Kautzky-Willer, Prof, MD[1].

[1]Medical University Vienna, Division of Endocrinology and Metabolism, Gender Medicine Unit, Vienna, Austria, [2]Medical University Vienna, Centre of Excellence, High-Field MR, Department of Radiodiagnostics, Vienna, Austria, [3]Medical University Vienna, Department of Obstetrics and Gynecology, Vienna, Austria.

SUN-048

Background: Sex hormones are believed to play an important role in development and progression of cardiovascular disease. However, the gender gap in onset and mortality is not yet completely understood. Transsexuals undergoing hormone therapy are a promising collective for analysing the effects of sex hormones on cardiometabolic disease.

Methods: Aim of this study is to identify gender specific cardiovascular changes attributed to high-dose cross-sex hormone therapy (HT) in male-to-female (MtF) and female to male (FtM) transgender patients by performing an oral glucose tolerance test (OGTT) and 3 Tesla magnet

resonance spectroscopy for hepatic (HCL) and myocardial (MYCL) lipid content analysis. The control group (CON) is conducted by age, sex and BMI matched healthy individuals. **Results:** Until now we included 26 MtF,14 FtM patients and 12 age and BMI matched healthy controls. The mean age was comparable in all 3 groups (MtF 30.12±2.31, FtM 29.72±1,91, CON 30,23±1.22 as well as BMI (22.59±3.81, 21.62±2.53, 21.33±1.20 kg/m2, p=ns, respectively). The mean hormone therapy duration was similar in both groups (MtF 4.58±1.20 vs FtM 2.35±0,95, p=0,29). HOMA Index did not significantly differ between the groups (MtF 1,78±0,92 vs FtM 1,96±1.22 vs CON 1,8±1.01, p=0,3 vs 0,4 vs 0,3 respectively). HCL was significantly higher in MtF than FtM (1,50±0,41 % vs 0.54±0,33 %, p=0,022, respectively). We also found a significant correlation between ejection fraction (EF) and Testosterone levels (Spearmans Rho 0,80, p=0.002). **Conclusio:** These preliminary data could indicate a positive effect of Testosterone therapy on heart function. Contradictory to current data we found a higher HCL in MtF than FtM suggesting a not so protective estrogen effect when looking at the liver. Long-term studies are warranted to assess whether cross-sex HT results in different outcomes regarding cardiovascular disease.

Diabetes Mellitus and Glucose Metabolism

CLINICAL AND TRANSLATIONAL STUDIES IN DIABETES

Course of Puberty and Growth Spurt in Boys with Type 1 Diabetes

Hana Shpitzer, MD[1], Liora Lazar, MD[2], Shlomit Shalitin, MD[1], Moshe Phillip, MD[3], Liat de Vries, MD[1].

[1]Schneiders' Children Medical Ctr in Israel, Herzeliya, Israel, [2]Schneider children's medical center of Israel, Petah Tikva, Israel, [3]Schneider Children's Med Ctr, Petah Tiqwa, Israel.

MON-659

Course of puberty and growth spurt in boys with type 1 diabetes

Background: Data on the course of puberty and pubertal growth in boys with Type 1 diabetes (T1D) are sparse.

Objectives: To study the course of puberty, pubertal growth and final height in boys with T1D as well as possible factors affecting these.

Methods: In this retrospective longitudinal study, 68 boys diagnosed with T1D between 1996-2009 who were prepubertal at diagnosis and had completed puberty served as the cohort. Collected were data on anthropometric measurements, Tanner stage, and HbA1c levels from diagnosis to final height (F-Ht). F-Ht was compared to parental height and to the data of the national health survey

Results – In the study cohort final height-SDS was lower than that at diagnosis. It was similar to parental Ht-SDS as well as to that of the national health survey (p=0.126). F-Ht was inversely related to average HbA1c during puberty (R=-0.27, p=0.045). Boys who presented with diabetic ketoacidosis at diagnosis were shorter than those who did not throughout the entire follow-up. Age at onset of puberty was significantly related to the age of maternal menarche (R=0.44, p=0.01) and to HbA1c levels in the year preceding puberty onset (R=0.36, p=0.01). Total pubertal growth was inversely related to HbA1c levels in the year preceding onset of puberty (average R=-0.3, p=0.03)

Conclusions: Boys with T1D diagnosed before puberty achieve final height similar to that of their parents and that of the general population. Diabetic ketoacidosis at the diagnosis is associated with diminished F-Ht. Age of pubertal onset and F-Ht are affected by genetic factors as well as by glycemic control before and during puberty.

These results emphasize the importance of tight metabolic control in adolescents, to enable growth within the genetic target.

Pediatric Endocrinology

PEDIATRIC ENDOCRINE CASE REPORTS II

Ketotic Hypercalcemia; A Possible Side Effect of Managing Refractory Epilepsy with Ketogenic Diet

Bassem Dekelbab, MD[1], Yafa Davydova, MD[1], Michael A. Levine, MD[2].

[1]Beaumont Children's, Royal Oak, MI, USA, [2]Children's Hospital of Philadelphia, Philadelphia, PA, USA.

MON-074

Background: Patients with epilepsy have multiple risk factors for low bone mass, including immobility, reduced muscle mass, and use of multiple anticonvulsant medications. The ketogenic diet (KD) has been used as an effective treatment of refractory epilepsy for decades, with multiple reports in medical literature describing adverse effects on bone and mineral metabolism including gradual loss of bone mineral density. There has been only one report of hypercalcemia in three children on the KD for refractory seizures. We now describe another case, highlighting the importance of considering the KD as a cause of hypercalcemia.. **Case presentation:** A 14-year-old girl with Rett syndrome, epilepsy, global developmental delay and gastrostomy tube dependency presented to the emergency room with marked dehydration. She had been on the KD for several years due to refractory epilepsy. Her parents had recently noticed thick oral secretions. The only change in her management plan was the recent change of her formula from Ketocal 3:1 to Ketocal 2.5:1. Lab studies showed hypercalcemia 15 mg/dL (ref 9.2-10.7) with ionized calcium of 7.99 mg/dL (ref, 4.8-5.2). She had normal serum calcium levels on multiple previous occasions, including 10.2 mg/dL 4 months prior to presentation. Other studies included increased BUN 37 mg/dL (ref, 7-21) and Creatinine 2.31 mg/dL (ref, 0.53-0.8). She had a low normal PTH 14 pg/ml (ref, 8-72), PTH-related peptide 0.6 pmol/l (ref, < 4.2), and 25-hydroxyvitamin D 66 ng/ml (30-100). 1,25-dihydroxyvitamin D level was low at 12.5 pg/ml (ref, 19.9-79.3), with increased urine calcium/creatinine ratio 1.00 mg/mg creat (Ref, 0.02-0.26). Beta Hydroxybutyrate was 3.45 mmol/l (ref, 0.02 - 0.27), without major change over the last year. Renal US was normal. She received IV hydration with improvement in serum calcium, BUN, and creatinine and was discharged on increased intake of free water and adjustment of KD to maintain Beta Hydroxybutyrate around 2 mmol/l. Her serum calcium currently ranges 10.5- 12 mg/dl, and will soon begin therapy with subcutaneous calcitonin. **Conclusion:** The KD has adverse effects on bone and mineral metabolism, and can lead to severe