

controlled at 10.44mg/dl, and his renal function improved to a serum creatinine of 3.08mg/dl. Prior to discharge patient developed acute left knee pain, and was found to have an inflammatory arthritis, with urate crystals seen. The patient was diagnosed with an acute gout flare, which responded well to colchicine and was discharged. The patient eventually underwent parathyroidectomy, which showed a large 4cm left superior parathyroid as well as a large right superior parathyroid gland extending into the mediastinum. Pathology was consistent with parathyroid hyperplasia. After surgery, the patient developed hungry bone syndrome, with an admitting serum level calcium of 6.05mg/dl, serum magnesium of 2.00mg/dl, serum phosphorus of 2.8mg/dl, and serum potassium of 5.1mg/dl, with clinical features of tetany and weakness that resolved after two days with calcium and calcitriol administration. He again had an acute monoarticular arthritis prior to discharge that had both urate and calcium pyrophosphate crystals in the joint fluid and again responded well to colchicine and glucocorticoids. He was eventually discharged on vitamin D and calcium supplementation, with cinacalcet and colchicine.

This case illustrates the multiple clinical teaching points that exist in primary hyperparathyroidism, including both types of presentation and potential complications. It also expresses the need to be vigilant of some rarer clinical features, such as potential hyperparathyroid crisis and multiple enlarged parathyroid glands. Physicians should also be wary of both gout and CPPD, as well as complications that occur post parathyroidectomy such as hungry bone syndrome.

Reproductive Endocrinology

MALE REPRODUCTIVE CASE REPORTS

Megestrol Acetate for Bodybuilding Resulting in Abrupt Hypothalamic-Pituitary Dysfunction

Kimberly Kochersperger Lessard, DO, Jeffrey L. Miller, MD, Luis Arzeno, MD.

Thomas Jefferson University, Philadelphia, PA, USA.

SUN-031

Introduction: Megestrol acetate (MA) is a synthetic progestin often used for appetite stimulation and weight gain in patients with cachexia related to AIDS, cancer or terminal illness. MA is frequently implicated in development of both glucocorticoid excess and adrenal insufficiency (AI) in a somewhat unpredictable manner. Less commonly reported adverse effects of MA include dysfunction of the hypothalamic pituitary axis. Few cases in literature exist regarding the use of MA in young, healthy patients and the potential clinical severity of pituitary dysfunction in this population.

Case: A 24-year-old male was previously healthy but frustrated by an inability to gain muscle mass. Evaluation one-year prior was unrevealing and included a total testosterone of 1100ng/dL (264-916ng/dL). In the interim, he was prescribed MA 625/5mg for 2 months with rapid development of abdominal adiposity, depressed mood, low libido and erectile dysfunction. Repeat evaluation revealed a precipitous drop in total testosterone to 66 ng/dL at which point he self-discontinued MA. He then began to experience

palpitations, sweats, poor concentration, and undesirable weight loss along with worsening symptoms of sexual and erectile dysfunction.

Three months after MA discontinuation, he appeared well with physical examination and vital signs within normal limits. Surprisingly, repeat studies revealed total testosterone <3, free testosterone <0.2 (9.3 -26.5 pg/mL), LH 1.3 (1.7-8.6 mIU/mL), FSH 2.2 (1.5-12.3 mIU/mL), Prolactin 33.4 (4.0-15.2 ng/mL) as well as 8AM cortisol 2.2 ug/dL and ACTH 22.7 pg/mL. A basic metabolic panel, thyroid function studies and pituitary MRI were unremarkable. The patient was empirically placed on prednisone 5mg daily pending ACTH stimulation testing for HPA axis re-assessment. Topical testosterone replacement therapy was initiated due to intolerance of hypogonadal symptoms.

Discussion: MA, by virtue of affinity for glucocorticoid receptors, has the potential to cause hyperglycemia and Cushing's syndrome. Secondary AI often results from withdrawal of MA however central AI can also occur with active administration by unclear mechanisms. Hypogonadism and hyperprolactinemia are additional under-reported adverse effects of MA with symptoms often masked by AI or elements of chronic illness. In study of men age 60-85 year on MA, a mean percentage change of ACTH -89.5%, LH -49%, TSH -14.7% and Prolactin +150% were seen from baseline after 12 weeks of therapy as was decreased cortisol. Limited data exists in young patients such as ours given limited indications for the agent's use. Importantly, MA should be prescribed with understanding that weight gain is predominantly adipose rather than muscle mass and that its safety is limited by potential HP axis dysfunction, namely adrenal and gonadal deficiencies.

Pediatric Endocrinology

SEXUAL AND GENDER DEVELOPMENT IN THE PEDIATRIC POPULATION

High Prevalence of Gene Variants in Boys of Prepubertal Age with Clinical Suspicion of Central Hypogonadism and Low AMH

Franco Brunello, MSc¹, E. Gabriela Sansó, PhD¹, Paula Scaglia, MSc¹, María Esnaola Azcoiti, MSc¹, Ariel Berenstein, PhD², Agustín Izquierdo, MSc¹, Patricia Bedecarrás, MSc¹, Marcelo Martí, PhD³, M. Gabriela Ropelato, PhD¹, Rodolfo Alberto Rey, MD, PhD¹, Romina Grinson, MD, PhD¹, Analía Freire, MD, PhD¹.

¹Centro de Investigaciones Endocrinológicas "Dr. César Bergadá" (CEDIE), CONICET-FEI-División de Endocrinología, Hospital de Niños Ricardo Gutiérrez, Buenos Aires, Argentina, ²Instituto Multidisciplinario de Investigaciones en Patologías Pediátricas (IMIPP), CONICET-GCBA, Hospital de Niños Ricardo Gutiérrez, Buenos Aires, Argentina, ³Instituto de Química Biológica de la Facultad de Ciencias Exactas y Naturales (IQUIBICEN), CONICET-UBA, Buenos Aires, Argentina.

OR15-03

Introduction: In boys of prepubertal age, the diagnosis of central hypogonadism may be difficult to ascertain since gonadotropins and testosterone are normally low. Sertoli cell markers, like AMH and inhibin B, may be useful. In recent years, with the development of next generation sequencing (NGS) technology, the number of

genes associated with central hypogonadism has had an exponential increase. However, even with these advanced techniques, the gene variants with potential pathogenicity can be found at present in only 30-50% of the patients. **Hypothesis of the study:** Low serum AMH is an appropriate screening biomarker to select patients for NGS, in order to make a genetic diagnosis in boys of prepubertal age with suspected central hypogonadism. **Patients and methods:** All patients aged 1-10 yr referred between 2001 and 2018 with clinical suspicion of central hypogonadism (micropenis and cryptorchidism and/or microorchidism), with low serum AMH (<10th centile) were included. Serum AMH was determined by ELISA (Beckman-Coulter), and LH, FSH and testosterone (T) by ECLIA (Roche). NGS was performed with the TruSight™ One Sequencing Panel in a NextSeq® 500 sequencer (Illumina). Results are expressed as medians (range). **Results:** 13 patients were included. Age at first visit was 4.4 (0.1-9.2) yr. Cryptorchidism was present in all of them, micropenis in 10 and microorchidism in 11. Orchiopexy was required in 11 boys and the other 2 responded to hCG treatment. 4 patients had olfactory disturbances, 1 had sensory deafness and 1 had piebaldism. 2 patients had a family history of olfactory disturbances and/or central hypogonadism. 7 patients could be followed up to pubertal age, and the diagnosis of central hypogonadism was clinically confirmed. At age 6.1 yr (1.2-10), AMH was 159 pmol/L (65-363), LH was <0.1 IU/L in all, FSH was 0.61 IU/L (<0.1-1.9). 17 variants in 9 genes associated with central hypogonadism were found in 10 of 13 patients. 5 boys had 1 gene variant, while 4 had 2 gene variants and 1 had 3 gene variants indicating probable oligogenicity, in the following genes: *FGFR1* (n:4), *CHD7* (n:3), *PROKR2* (n:2), *SOX10* (n:2), *AXL* (n:2), *HS6ST1* (n:1), *AMHR2* (n:1), *NSMF* (n:1), *DCC* (n:1). **Conclusion:** A high prevalence of gene variants was found in boys of prepubertal age with a suspicion of central hypogonadism based on micropenis and cryptorchidism and/or microorchidism with low serum AMH.

Reproductive Endocrinology

MALE REPRODUCTIVE CASE REPORTS

Diabetes Mellitus Associated with Klinefelter Syndrome

Carolina Sarria, MD, William Iapa Yauri, MD.

HOSPITAL ARZOBISPO LOAYZA, Lima, Peru.

SUN-029

Background: It has not been clarified why diabetes mellitus develops in patients with Klinefelter's syndrome. However the association between both diseases is frequent. **Clinical Case:** A 31-years-old man with type 2 diabetes diagnosed 2 months ago who received metformin for treatment. He presented to the hospital with dysuria, polyuria, polydipsia, weight loss, hyporexia, vomiting and drowsiness. On examination: BP: 110/60 mmHg, HR: 108 lp, FR: 24 rp, T °: 37 ° C, BMI: 31.4 kg / m², oral mucosa dry, bilateral gynecomastia, subcutaneous cellular tissue of gynecoid distribution. His body hair was thin. His penis was small and both testicles were prepubertal. Laboratory: Glucose: 410 mg / dl; HbA1c 15.2%; creatinine: 1.01 mg / dl; arterial gases: pH 7.14 pCO₂: 20, HCO₃: 6.6, AG: 29.

Normal electrolytes. Ketonuria: 3+. We concluded: diabetic ketoacidosis. In addition, FSH 61.18 μU / ml (RR: 1.5-12.5); LH: 28.47 μU / ml (RR: 0.7-8.6); Total testosterone: 0.41 ng / ml (RR: 2.8-8), compatible with hypergonadotropic hypogonadism. Therefore a karyotype is requested in peripheral blood, resulting in 47, XXY in 20 metaphases analyzed. Klinefelter's syndrome was diagnosed from his physical characteristics, hormonal findings and his chromosomal aberration. He received testosterone undecanoate every 4 weeks, NPH insulin 12 IU / 8UI plus metformin 850 mg every 12 hours. Three months later: baseline glucose 89 mg / dl and HbA1C of 9.5%. **Conclusion:** We present the case of a young male with diabetic ketoacidosis and hypogonadism, secondary to Klinefelter syndrome. Klinefelter syndrome is associated with Diabetes mellitus with a RR that varies from 1.64 to 7.06 according to current literature. In addition, we highlight the importance of the medical history and physical examination for an adequate diagnosis of rare conditions such as Klinefelter Syndrome. **Reference:** 1. Kanakis GA, Nieschlag E. Klinefelter syndrome: more than hypogonadism. *Metabolism*. September 2018;86:135-44.

Thyroid

THYROID NEOPLASIA AND CANCER

A Descriptive Study of Clinical and Surgical Characteristics of Patients with Thyroid Cancer: A 10-Year Retrospective Study from UAE

Abdulla Mohamed Alnuaimi, MD¹, Majid Alameri, MD¹, Huda Mustafa, MD¹, Aly Bernard Khalil, MD².

¹Sheikh Khalifa Medical City, Abu Dhabi, United Arab Emirates,

²ICLDC, Abu Dhabi, United Arab Emirates.

MON-507

A descriptive study of clinical and surgical characteristics of patients with thyroid cancer: a 10-year retrospective study from UAE

Introduction: Thyroid cancer is the sixth most common type of cancer in the UAE. It has been observed that the incidence of thyroid cancer has been steadily increasing worldwide. However, limited studies about thyroid cancer has been reported from the Arab gulf region.

Objective: The objective of this study was to describe the clinical and surgical characteristics of patients with thyroid cancer in the UAE population.

Methods: Retrospective analysis was performed on all adult patients attending thyroid cancer clinic at Sheikh Khalifa Medical City (SKMC) in Abu Dhabi, UAE over ten years from 2008 to 2018. All patients with a confirmed histological diagnosis of thyroid cancer who had surgical intervention with long-term follow up data on cancer outcome have been included. Categorical variable analysis and descriptive analysis were used to identify factors associated with increased risk of developing thyroid cancer.

Results: Total number of 203 patients with confirmed diagnosis of thyroid cancer were included. Most of the patients were female (72.9 %, n=148). Mean age at the time of diagnosis was 40±13 years. Papillary thyroid carcinoma was the most common thyroid cancer observed (95.6%, n=194) followed by follicular thyroid carcinoma (2%), medullary thyroid carcinoma (1.5%) and mixed medullary-papillary