ml (4-15), normal ACTH 10 pg/ml, cortisol 13.4 mcg/dl, low total testosterone 48.2 ng/dl (193-836), normal free testosterone 7.92 ng/dl (4.85-19), normal TSH 1.55 mc unit/ml (0.27-4.2) and free T4 1.17 ng/dl (0.93-1.7). The patient was discharged home on 120+ units of total daily dose of insulin, after initial hospital admission.
He underwent trans sphenoidal resection of pituitary macro adenoma one month after his initial presentation. Surgical pathology confirmed growth hormone producing adenoma. He was successfully weaned off from insulin in one month following surgery.
Conclusion: DKA is an unusual initial presentation of growth hormone producing tumors. As more cases are being reported it is important to be vigilant to look for DKA presentation in these patients and adjust/wean patients insulin once the growth hormone producing tumor is treated either with surgery or medications.

Thyroid

THYROID DISORDERS CASE REPORTS II

Anticoagulation Conundrum: A Case Of Embolic Stroke Due To Thyrotoxic Atrial Fibrillation

Laishiya Munshi, DO[1], Yamuna Gorantla, MD[1], Susana Mascarell, MD[2].
[1]Rush University Medical Center, Chicago, IL, USA, [2]John H. Stroger Hospital of Cook County, Chicago, IL, USA.

SAT-468

Background: Thyrotoxicosis is not incorporated into any of the atrial fibrillation (AF) CVA risk stratification methods, including CHADS2VASc, as neither the American College of Cardiology/American Heart Association nor the American College of Chest Physicians view it as an independent risk factor of CVA. However, the literature has reported numerous cases of patients with thyrotoxic AF and low CHADS2VASc scores who developed CVA. It is unclear which patients with thyrotoxic AF would benefit from prophylactic anticoagulation (AC).
Clinical Case: A 50-year-old male without past medical history presented for palpitations. He was found to be in AF with rapid ventricular rate. Labs revealed thyrotoxicosis (TSH <0.010 [RR 0.34-5.6 μIU/mL], FT4 3.57 [RR 0.61-1.64 ng/dL], FT3 5.26 [RR 2.5-4.5 pg/mL], and TSI negative). He was started on methimazole but no AC, as his CHADS2VASc score was 0. A few hours after admission, he developed a right-sided facial droop and weakness & aphasia. CT head showed acute left MCA infarct. CTA head/neck revealed occlusive embolus in the distal left M1 segment. He underwent IR-guided embolectomy and received intra-arterial tPA. Echocardiogram did not show thrombi or atrial level shunt but showed right atrial dilation. After extensive work-up, etiology of CVA was determined most likely a cardioembolic source due to thyrotoxic AF. Patient was treated with PTU and steroids initially for two days due to recent contrast exposure (CTA and embolectomy) and poor neurologic status, in order to obtain the added benefit of decreased T4 to T3 peripheral conversion. Patient was started on AC two weeks after his CVA (given initial risk of hemorrhagic conversion due to large CVA burden), at which time his weakness and aphasia were slowly improving, but he remained with significant neurologic deficits.
Discussion: The current thought that thyrotoxicosis is not an independent risk factor of CVA in patients with AF has led to recommendations against prophylactic AC for this specific group. Although recent research has suggested that patients with thyrotoxic AF are at a lower risk of CVA than patients with non-thyrotoxic AF, there have still been many reported cases of CVA in patients who have thyrotoxic AF but no other risk factors for CVA. This discrepancy in association between thyrotoxic AF and CVA needs to be clarified.
Conclusion: This case of a middle-aged man with thyrotoxic AF who developed a debilitating CVA after being treated according to his CHADS2VASC score of 0 (he was not given prophylactic AC) mirrors multiple cases in the literature. It highlights the potential benefit of examining thyrotoxic patients with AF more closely in order to more effectively risk stratify them for CVA or further exploring the relationship between thyrotoxicosis and CVA. This may help to identify more patients who could benefit from AC and thus prevent devastating consequences of CVA.

Diabetes Mellitus and Glucose Metabolism

CLINICAL AND TRANSLATIONAL GLUCOSE METABOLISM AND DIABETES

Degree of Diabetes Control Determines the Admission Severity of Diabetic Ketoacidosis

Harshwant Grover, MD[1], Puneet Dhillon, MD[1], Neethu Gopisetti, MD[1], Daniel Evan Rosenberg, MD[2], Rajeshkumar Patel, MD[1].
[1]Abington Jefferson Health, Willow Grove, PA, USA, [2]Endocrine Specialists, Abington Jefferson Hospital, Horsham, PA, USA.

MON-614

Objectives: To determine whether the degree of diabetes control correlates with the admission severity of diabetic ketoacidosis (DKA).
Methods: A Retrospective chart review was performed for patients admitted with DKA to the medical ICU at Abington Memorial Hospital between January 1, 2017 and January 1, 2018. Laboratory Data required to determine an acute physiology and chronic health evaluation (APACHE) score, hemoglobin A1C, length of hospital stay was recorded. The APACHE score was used to determine the severity of disease at admission. Patients were divided into two groups: low severity (APACHE <15) and high severity (APACHE >15).
Results: A total of 50 patients were included in the analysis. The mean age of the patients was 47 yrs (range 17-85 yrs). 52%(n=26) of the population were males. The overall mean APACHE II at admission was 15 (range 3-28). The low severity group (APACHE <=15) and high severity group (APACHE >15) were equally matched at 25 patients each. The mean APACHE scores were 9.9 and 18.7 for the low and high severity groups respectively. The mean hemoglobin A1C values for the low and high severity groups were 10.5 and 15 respectively. The average length of ICU/hospital stay was 1.6/3.65 and 1.54/3.61 days for the low and high severity groups respectively.
Conclusions: According to our study, a higher severity of DKA (higher APACHE) was associated with a higher

hemoglobin A1C level. However, the study did not find any difference in the average length of ICU/hospital stay between the two groups.

Thyroid

THYROID DISORDERS CASE REPORTS II

Simultaneous Hashimoto/Graves Disease or Prolonged Hashitoxicosis? A Diagnostic Challenge with Therapeutic Implications

Carlos A. Penaherrera, MD, Valentina Rodriguez, MD.
Division of Endocrinology, Diabetes & Metabolism, University of Miami Miller School of Medicine, Miami, FL, USA.

SAT-482

Background: Some individuals with Hashimoto's thyroiditis (HT), characterized by anti-TPO antibodies (Abs), can also have positive TSI Abs in up to 20% of cases, without necessarily having Graves disease (GD). Patients with signs of both hyper-and-hypothyroidism with positivity to these two Abs can pose a diagnostic and therapeutic dilemma, as their course is often unpredictable. **Clinical Case:** A 49-year-old woman was diagnosed with hypothyroidism and took levothyroxine (LT4) for about 1 year, after which she developed symptoms of hyperthyroidism and was switched to methimazole (MMI), which she only took for 1 year. At her initial visit at our clinic she had been off of MMI for 12 months and she was biochemically hyperthyroid (TSH of 0.01 mcIU/ml, f-T4 of 2.26 ng/dl). TSI Abs were positive at 461, but she also tested positive for anti-TPO at 673. Thyroid receptor Abs (TRAb) were also elevated at 54.8. Her vital signs were stable, but she had marked proptosis and complained of eye dryness, so MMI was restarted. A RAIU scan could not be obtained, but a thyroid US showed a heterogeneous and hypervascular gland. On MMI, her thyroid function tests normalized, and her eye disease vastly improved over 2 years. Her MMI dose was progressively decreased until it was stopped completely. On re-evaluation a few months later, she had newly elevated TSH of 8.7 mcIU/ml and low f-T4 of 0.87 ng/dl, with no symptoms of hypothyroidism, so we opted for management with active surveillance instead of starting her on LT4. Her TSI level improved to 240, but remains elevated. **Discussion:** It is unclear if our patient has a mixed condition with features of both GD and HT, or if she has HT with a very prolonged hyperthyroid phase (hashitoxicosis). Extended periods of hashitoxicosis have been described, the longest reported lasted for 2 years[1]. Simultaneous presentation of GD and HT is very rare, with only a few cases described in the literature. RAIU scan is often diagnostic, showing increased uptake as seen in GD, but patchy areas of decreased uptake can also be seen. In our case it is likely that HT and GD were coexisting, with GD masking the hypothyroidism, until the former remitted with MMI, and her HT took over. Though no RAIU scan was available, the TSI positivity, clinical response of her hyperthyroidism to MMI and the presence of orbitopathy rule in favor of co-existing GD. Decision to treat with LT4 should be weighed against the risk of causing recurrence of hyperthyroidism. Special considerations should be taken in women of childbearing age due to the difficult management that overlapping hyper/hypothyroidism would entail during pregnancy. **References:**
1. Shahbaz, A et al. Prolonged Duration of Hashitoxicosis in a Patient with Hashimoto's Thyroiditis: A Case Report and Review of Literature. Cureus. 2018; 10(6):e2804

Adipose Tissue, Appetite, and Obesity

RARE CAUSES AND CONDITIONS OF OBESITY: PRADER WILLI SYNDROME, LIPODYSTROPHY

Pubertal Timing and Hormonal Correlates in Male Obesity.

Jean De Schepper, PhD[1], Evy Berlanger, Student[1], Monique Van Helvoirt, MD[2], Ann De Guchtenaere, PhD[2], Eddy Basslé, MT[2], Ellen Anckaert, PhD[1], Jesse Vanbesien, MD[1], Inge Gies, PhD[1].
[1]UZ Brussel, Brussel, Belgium, [2]Zeepreventorium De Haan, De Haan, Belgium.

SUN-594

An early, normal or delayed pubertal onset have been described in overweight/ obese males(1). A greater prepubertal adiposity has been associated with a greater risk for delayed puberty in males, but an underlying mechanism was not explored(2). We investigated whether an increased testosterone aromatization or an higher degree of low-grade inflammation might be more prevalent in obese males with a delay in genital development. Pubertal status assessment by Tanner staging and measurement of morning serum testosterone, estradiol, leptin, and hSCRP by standard laboratory methods were performed in 191 male adolescents, aged between 10 and 18.6 yr (median 12.8 yr) with overweight (BMI z-score > 1.3), starting an ambulatory (n = 138) or a residential weight loss program (n = 55). Their median (range) BMI z-score was 2.32 (1.34 – 3.38). Delayed / slow and early / rapid genital development was defined by a Tanner genital stage respectively above the 90th or below 10th percentile age distribution (national Flemish standards of 2004). In 3 males pubertal development was advanced, while in 34 it was delayed. In the remaining 154 adolescents genital stage was normally timed. Males with a delayed timing or progression of genital development were older (median(range) age:14.8 (11.6-18.6) yr vs 12.3 (10-18.6) yr; p< 0.005) and shorter (height sds: -0.55 (-1.90- 1.48) vs 0.49 (-3 – 3.19); p < 0.005), and had a higher birthweight (birthweight z-score: 0.15(-3.51-2.75) vs -0.34(-4.7-3.30); p = 0.058), but a similar BMI and waist z-score in comparison with males with a normally timed puberty. Median serum estradiol, leptin, and hSCRP concentrations did not differ significantly between those with a normal or a delayed pubertal onset or progression. In conclusion, pubertal delay is more frequently observed than early puberty in males referred to obesity clinics. Neither low grade inflammation nor increased estradiol production appear to be associated with a later onset of slower progression of genital development in male obesity. References (1) Li W et al. Int J Environ Res Public Health. 2017 Oct 24;14(10) (2) Lee JM et al. Arch Pediatr Adolesc Med. 2010 Feb;164(2):139-44.

Adrenal

ADRENAL CASE REPORTS II

A Rare Case of Untreated Congenital Adrenal Hyperplasia Leading to Gender Dysphoria and a Female to Male Transgender

Dana Awad, MBBS[1], Htet Htet Win, MBBS[2], Mohsen Zena, MD[3].