CT chest which revealed a 2cm lung nodule. She had video assisted thoracoscopic (VATS) left lower lobe wedge resection with completion left lower lobectomy for a 3 cm lung adenocarcinoma with negative margins and 33 negative lymph nodes. She was subsequently treated with RAI after recovery from VATS procedure. Pretreatment thyroglobulin was 0.8 ng/ml with negative thyroglobulin antibodies. One month after her RAI treatment, ultrasound of the neck revealed suspicious bilateral level IV lymph nodes which increased in size during short term follow up. Serum thyroglobulin was 0.3ng/ml with negative antibodies and TSH 0.29 mIU/L. Biopsy of right level IV lymph node was positive for PTC with thyroglobulin washout >5000 while left level IV lymph node was negative for PTC and Tg washout was 0.1. She subsequently underwent right-sided modified radical neck dissection, with lymph nodes revealing PTC also involved by small lymphocytic lymphoma. She had repeat RAI ablation for thyroid cancer and is being actively monitored for her small lymphocytic lymphoma and lung adenocarcinoma.
Conclusion: We present a patient with no known history of malignancy who presented with 3 de novo primary malignancies. This case may demonstrate an increased risk of malignancy in patients with thyroid cancer not necessarily related to radioactive iodine treatment.

Tumor Biology

ENDOCRINE NEOPLASIA CASE REPORTS II

Diabetic Ketoacidosis Following Treatment of Endogenous Hyperinsulinism

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MON-909

Background: There has been only one case of Diabetic Ketoacidosis (DKA) reported following treatment of endogenous hyperinsulinism in a 16 month old.[1] This has not yet been described in adults.
Clinical Case: An 85 Y/O M with a PMH of metastatic gastric adenocarcinoma complicated by gastric outlet obstruction requiring TPN was admitted for symptomatic hypoglycemia. On the day of admission, his wife noted that he appeared confused and checked his capillary blood glucose, which was 35, prompting her to call EMS who gave him IV dextrose 50% (D50).
In the ED, he was placed on continuous dextrose 10% (D10) due to persistent hypoglycemia. To investigate the cause of hypoglycemia, D10 was stopped and a fast test was initiated. The patient developed symptomatic hypoglycemia 9 hours after stopping the D10. Laboratory results showed: plasma glucose 43 mg/dL, c-peptide 3.1 nmol/L, pro-insulin 18.7 pmol/L, insulin 10.7 uU/mL, beta-hydroxybutyrate (BHOB) 0.08 mmol/L. Insulin antibody and screen for oral hypoglycemic drugs were negative. Glucagon administration raised his blood glucose from 43 mg/dL to 50 mg/dL, 84 mg/dL, and 106 mg/dL after 10, 20, and 30 minutes, respectively. A diagnosis of endogeneous hyperinsulinism was made and the patient was started on Diazoxide 50 mg TID which was increased to 150 mg TID 2 days later. On day 3, Prednisone 20 mg daily was started due to inability to come off the D10 drip completely. On day 4 he was taken off D10. Due to plasma glucose >150 mg/dL, prednisone dose was reduced to 10 mg and then 5 mg on day 5 and 6, respectively. On day 8, he was found to be in DKA with a plasma glucose of 250 mg/dL, metabolic acidosis with an anion gap of 20, HCO3 of 15 mg/dL, undetectable insulin levels and BHOB of 5.49 mmol/L. Prednisone and Diazoxide were discontinued and he was started on an intravenous insulin infusion. Within 24 hours he became persistently hypoglycemic requiring D50 and prednisone was restarted. DKA developed once again and the patient was subsequently made comfort measures only. Further investigation of his endogenous hyperinsulinism was not pursued. The patient was transferred to inpatient hospice, where he passed away several days later.
Conclusion: This is the first reported case of an adult patient with documented endogenous hyperinsulinism developing DKA following treatment with diazoxide and prednisone. **Reference:** (1) Mangla et al. J Ped End Met. 2018; 31(8): 943-945.

Cardiovascular Endocrinology

ENDOCRINE HYPERTENSION AND ALDOSTERONE EXCESS

Sparing Confirmatory Tests in Primary Aldosteronism

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SAT-551

Context: The current clinical guidelines suggest that confirmatory tests for primary aldosteronism (PA) may be excluded in some of patients who have elevated plasma aldosterone concentration (PAC) under plasma renin suppression. However, this has low priority evidence and is under debate in use of serum potassium. **Objective**: This study aimed to investigate an appropriate setting for sparing confirmatory tests in PA. **Design and Setting**: A retrospective cross-sectional study in a single referral center. **Participants**: This study included 327 patients who had hypertension under plasma renin suppression and underwent captopril challenge test (CCT) between January 2007 and April 2019. CCT results were used to diagnose PA. **Main outcome measure**: Diagnostic value of PAC and serum potassium in confirmation of PA. **Results**: Of the studied patients, 252 of 327 (77%) were diagnosed with PA. All 61 patients with PAC >30 ng/dl were diagnosed with PA. In patients with PAC between 20 and 30 ng/dl, 44 of 55 (80%) were diagnosed with PA, while all 26 with PAC between 20 to 30 ng/dl who had spontaneous hypokalemia were diagnosed with PA. Areceiver operator curve analysis showed that the sensitivity of diagnosis of PA is 100% in our patients, when PAC set at > 28.8 ng/dl and showed that the sensitivity of diagnosis of PA is 100% in our patients with spontaneous hypokalemia, who had PAC < 30 ng/dl, when PAC was set at > 19.2 ng/dl. While, the prevalence of PA was higher in patients with hypokalemia, who had PAC between 10 and 20 ng/dl than in those with PAC < 10 ng/dl. Collectively, 100 out of 102 (98%) with hypokalemia, who

had PAC > 10 ng/dl were diagnosed as PA. The proportion of unilateral PA determined by adrenal vein sampling (AVS) was higher in patients who had PAC >30 ng/dl or those with spontaneous hypokalemia who had PAC between 20 and 30 ng/dl than those who did not meet the criteria (76% vs. 17%, P<0.001). **Conclusion**: Confirmatory tests in PA could be spared in patients who have typical features of PA and these patients had a high probability of unilateral PA on AVS.

Pediatric Endocrinology

PEDIATRIC ENDOCRINE CASE REPORTS I

Hidden in Plain Sight: Rethinking Our Approach to Allan-Herndon-Dudley Syndrome

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SAT-081

Background: Allan-Herndon-Dudley (AHD) is a rare X-linked disorder with neurological manifestations secondary to a mutation in monocarboxylate transporter 8, a protein that transports T3 into nerve cells in the brain. AHD is characterized by increased serum free T3, decreased serum free T4 and normal serum TSH levels as well as the severe neurological manifestations including global developmental delay, hypotonia, and joint contractures (1). A phase 2 trial using triodyothyroacetic acid has shown promise in treating this disorder (2). We report on three children who were diagnosed by whole exome sequencing after presenting with neurological manifestations.

Clinical Cases: Patient 1 presented at 4 months to the neurology clinic for seizures. He had a normal newborn screen. Worsening developmental delays and central hypotonia prompted a brain MRI that revealed delayed myelination for age. At 6 months a chromosomal microarray and metabolic work-up were performed and were nondiagnostic. Whole exome sequencing was obtained at the age of 4.5 years revealing a mutation in the SLC16A2 gene (p.Ser210Tyr). Thyroid studies were consistent with the diagnosis.

Patient 2 presented to neurology at 9 months for developmental delay. A brain MRI was obtained which was within normal limits. At 14 months an acylcarnitine profile was obtained which indicated a possible CPT1 deficiency, which did not fit his clinical picture. Chromosomal microarray as well as work-up for inborn errors of metabolism were performed and were nondiagnostic. Thyroid studies were obtained which showed low free T4 with normal TSH. Whole exome sequencing was obtained at the age of 2.5 years, which revealed a mutation in SLC16A2 (p.R371C).

Patient 3 presented as sibling of patient 2 with known AHD syndrome. Testing for SLC16A2 was performed at the age of 5 months and returned positive for same mutation as sibling (p.R371C).

Conclusion: Allan-Herndon-Dudley syndrome is a rare neurological disease secondary to a mutation in the T3 transporter protein to nervous tissue. A high index of suspicion as well as thyroid studies should be obtained in patients presenting with central hypotonia and global developmental delay with normal newborn screens, particularly in states that use TSH as a screening test. This is especially important as treatments are becoming available that may help prevent neurological devastation seen in these patients.

References:
1. Dumitrescu AM, Fu J, Dempsey MA, Refetoff S. MCT8-Specific Thyroid Hormone Cell-Membrane Transporter Deficiency. In: Adam MP, Ardinger HH, Pagon RA, et al., eds. *GeneReviews®*. Seattle (WA): University of Washington, Seattle; 1993
2. Groeneweg S, Peeters RP, Moran C, et al. Effectiveness and safety of the tri-iodothyronine analogue Triac in children and adults with MCT8 deficiency: an international, single-arm, open-label, phase 2 trial. *Lancet Diabetes Endocrinol*. 2019;7(9):695-706.

Thyroid

HPT-AXIS AND THYROID HORMONE ACTION

Influence of Smoking on Thyroid Function in Japanese Subjects: Longitudinal Study for One Year of On-Off Smoking

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SAT-437

Recent studies showed that various factors, including age, gender, race, iodine intake, obesity, the thyroid peroxidase antibody (TPO-Ab), and/or smoking, influence the thyroid status. In the present study, we analyzed and investigated the effects of these factors, particularly smoking and the thyroid peroxidase antibody (TPO-Ab) in Japanese euthyroxinemia individuals with serum free T4 level within normal range. A total of 12,289 subjects who underwent health check-ups were analyzed in a cross-sectional and longitudinal study. The mean age of subjects was 50 ± 10 years (age range: 21–88 years). Serum TSH levels and the prevalence of positivity for TPO-Ab increased with age in Japanese euthyroxinemia subjects. Mean and median serum TSH levels increased with age in smokers and non-smokers, but were significantly lower in smokers than in non-smokers among men and women in most age groups; the median 97.5th percentiles of TSH levels were 1.2 mU/liter and 2.9 mU/liter in smokers, and 1.4 mU/liter and 3.9 mU/liter in non-smokers in 31- to 40-year-old men, p<0.01, and 1.4mU/liter and 4.3 mU/liter, and 1.8mU/liter and 6.2 mU/liter in 61- to 70-year-old men, p<0.01. However, smoking had a negligible effect on serum TSH levels in women older than 50 years; 1.3 mU/liter in smokers and 1.6 mU/liter in non-smokers in 31- to 40-year-old women, p<0.01, and 1.5 mU/liter and 1.8 mU/liter in 51- to 60-year-old women, p=0.3. Furthermore, the present study confirmed that serum free T4 levels in men progressively decreased with age, whereas no significant change was observed in women. Smoking did not affect the relationship