parathyroidectomy, nephrolithiasis, and alcohol-induced chronic pancreatitis. Initial laboratory studies revealed elevated corrected calcium (11.5 mg/dL, n:7.6-10.6 mg/ dL) with a subsequent elevated parathyroid hormone level (105, n:14-72 pg/mL). CT scan of the abdomen and pelvis revealed fatty liver changes, nonobstructive nephrolithiasis and pancreatic calcifications. Due to his recurrent symptoms, gastroenterology was consulted for esophagogastroduodenoscopy that revealed erosive esophagitis with multiple duodenal ulcers. The patient was started on a high dose Proton Pump Inhibitor IV and treated supportively. His symptoms improved within a few days. Additional studies were obtained including a gastrin level and H. pylori antigen testing that required sending out to an outside laboratory for analysis. He was discharged with close outpatient follow up scheduled, but then was readmitted two days later with recurrent symptoms. The patient was again treated with supportive care, and was able to provide additional family history revealing parathyroid and ulcer disease in both his father and paternal grandmother, which triggered suspicion for multiple endocrine neoplasia type 1. His gastrin level returned, and was found to be elevated (489 pg/mL, n < 100) with a negative H. pylori antigen, supporting the diagnosis of MEN1. Further diagnostic testing was performed, revealing an elevated serum chromogranin A level (117 ng/mL, n < 15) and abnormalities on MEN1 genetic testing. An Octreotide scan was then performed, showing a single intense area of uptake near the caudate lobe of liver. At this time, he was transferred to a tertiary medical center for further evaluation and management.

Conclusion: This case illustrates the significance of suspicion for multiple endocrine neoplasia and the value of a complete history. Additionally, while a serum chromogranin A level can be nonspecific, it can be obtained in an outpatient setting to help differentiate neuroendocrine pathology from other etiologies. Though the symptoms of MEN1 patients can mimic many other disease processes, recognition of symptoms is critical for medical communities to provide swift treatment.

Adrenal

PROGRESS IN ADRENAL CORTEX AND MEDULLA RESEARCH

Identification of a Novel Stem/Progenitor Population of the Adrenal Medulla

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The adrenal glands regulate multiple physiological processes including the stress response, the immune system and metabolism. The adrenal is composed of an outer cortex that produces steroids, and an inner medulla that produces catecholamines. Tissue-specific stem/progenitor

populations have been identified in the adrenal cortex, while the presence of a functional stem/progenitor population in the adrenal medulla is unclear. The adrenal medulla derives from the neural crest and contains chromaffin cells, neurons and sustentacular (support) cells. Establishing cell hierarchy and elucidating mechanisms of regulation of the different cell types is important to understand normal homeostasis and disease pathogenesis, such as of pheochromocytomas. Using genetic approaches in mouse, we have established that a subpopulation of sustentacular cells express the stem/progenitor marker SOX2. Through genetic lineage-tracing using the Sox2-CreERT2 strain, we demonstrate that these are an expanding population, capable of giving rise to chromaffin cells and neurons throughout life, consistent with a stem/progenitor role in vivo. We further demonstrate the self-renewal and differentiation potential of SOX2+ cells through in vitro isolation and expansion. Through analysis of FFPE sections of human adrenals, we confirm the presence of SOX2+ cells in the normal adult organ, as well as in pheochromocytomas. Taken together, our data support the identification of a previously undescribed stem/progenitor cell in the mammalian adrenal medulla, and confirm its functional relevance.

Diabetes Mellitus and Glucose Metabolism

CLINICAL STUDIES IN OBESITY, DIABETES RISK, AND CARDIOVASCULAR OUTCOMES

Racial and Ethnic Differences in Metabolic Disease in Obese Adolescents with Polycystic Ovary Syndrome Stanley Andrisse, MBA, PhD¹, Yesenia Garcia Reyes, MS², Laura Pyle, PhD³, Kristen Nadeau, MD, MS⁴, Megan Moriarty Kelsey, MD⁵, Melanie Cree-Green, MD, PhD⁶.

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SAT-627

Background: Polycystic ovary syndrome (PCOS) affects up to 15% of women and is associated with a multitude of metabolic complications including insulin resistance, type 2 diabetes, cardiovascular disease, and hepatic steatosis. In the general population, metabolic disease rates vary by race and ethnicity. The interaction of race and ethnicity with PCOS-related metabolic disease in adolescent youth has not been extensively examined. Methods: Secondary analysis of data from overweight and obese (>90 BMI%ile) adolescent (12-21 years) female participants with PCOS enrolled across 4 protocols. Measurements included fasting hormone and metabolic measures, a 2-hour oral glucose tolerance test and MRI for hepatic fat. Groups were compared by ANOVA, with and without correction for BMI or chi-square tests for proportions. Results: Participants included 39 white (NHW 15.7±0.2 years; 97.7±0.2 BMI%ile), 50 Hispanic (15.2±0.3 years; 97.9±0.3 BMI%ile) and 12 black (NHB