

normetanephrine 52,3 pg/mL (N<196). Her remaining laboratory values were within normal values including potassium and sodium values.

Due to the size, rate of growth and atypical features of the mass, right laparoscopic adrenalectomy was performed. The resected adrenal weighed 66.6g and within it there was an intraparenchymal nodule, measuring 7.5 x 3 x 3.5 cm, which on histologic examination proved to be an adrenal myelolipoma.

Conclusion: Adrenal myelolipomas are usually clinically silent. However, their incidental diagnosis should warrant careful diagnostic study. Although the pathogenesis of these tumors remains unclear, theories include retention of embryonic rests, adrenocortical metaplasia and extramedullary hematopoiesis. In this patient with sickle cell disease, bone marrow elements within the myelolipoma may have grown as a result of the persistent anemia. Particular to this case is the patient's young age, since most cases reported have occurred during the fourth-sixth decades of life. In addition, the association with sickle cell anemia has only been reported in a few cases.

Diabetes Mellitus and Glucose Metabolism

METABOLIC INTERACTIONS IN DIABETES

Elevated Osmolal Gap in Long-Term Complication of Type 2 Diabetes

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Osmolal gap (OG) is the difference between the measured osmolality and calculated osmolality estimated by using the mathematical equation. Elevated OG indicates the presence of osmotically active particles undetected in the plasma of healthy individuals. Elevated OG has been observed in multiorgan trauma as a useful prognostic factor of patient survival [1, 2]. We hypothesized that elevated OG may occur in patients with type 2 diabetes (T2D) and may become a useful indicator of unmeasured endotoxins. One of the major mechanisms of this phenomenon may be non-enzymatic glycation of proteins in hyperglycemia, in which the osmotic active carbonyl compounds such as glyoxal, methylglyoxal, and 3-deoxyglucosone, are formed. Testing was performed for participants with T2D, aged 18 to 85. The osmolality measurement was performed with venous blood using the osmometer. The concentration of individual osmols (sodium, potassium, glucose, urea) was determined and on this basis, osmolality was calculated using the Dorwart-Chalmers formula: $\text{osmolality (mOsm/kg H}_2\text{O)} = 1.86 \times [\text{Na}^+] + \text{glucose} + \text{urea} + 9$ (Dorwart, 1975). The clinical profile of patients was established based on history and physical examination (age, sex, age, duration of T2D, complications of T2D, HbA1c, LDL-cholesterol, triglyceride, BMI, coexisting diseases, medication). Data were analyzed using descriptive statistics. This study is currently ongoing, but preliminary data from the pilot study suggest an increased mean measured osmolality as well as elevated OG in patients with T2D compared to the reference values for healthy adults. These values differed

depending on the type of long-term complications and the duration of the disease. In the pilot study, the highest OG was reported in diabetic retinopathy. A follow-up study with a larger sample may have a better ability to detect the statistical significance of the association of OG and complications of T2D. The association of the osmolal gap and complication of type 2 diabetes is poorly understood, and further investigation is warranted.

References:

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Reproductive Endocrinology

BASIC MECHANISMS IN REPRODUCTION: FROM BEGINNING TO END

Maternal Adiponectin Prevents Against Metabolic Dysfunction in Prenatally Androgenized PCOS-Like Mice

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More than 10% of women worldwide are diagnosed with polycystic ovary syndrome (PCOS), causing reproductive and metabolic disease. Hyperandrogenism is the main characteristic and elevated levels of androgens during pregnancy affect placenta function and fetal programming, which leads to reproductive and metabolic dysfunction in the offspring. Adiponectin secreted from adipose tissue improves whole-body metabolism, but its role during pregnancy is under explored. Adiponectin affects placental nutrient transport during pregnancy allowing for speculation that adiponectin can exert endocrine effects on the developing fetus. This study aims to investigate if, in prenatally androgenized (PNA) mice, adiponectin can prevent metabolic and reproductive dysfunction in female offspring. Adiponectin transgenic (APNtg) and wildtype (wt) female mice were mated with wt males, and received dihydrotestosterone or vehicle injections between gestational days 16.5-18.5 to induce a PCOS-like phenotype. The anogenital distance, a marker of *in utero* androgen exposure, was measured at 22 days of age, estrus cyclicity was recorded at 6 weeks of age, and metabolic measures were performed at 4 months of age.

APNtg dams gave birth to significantly smaller offspring, independent of genotype, than wt dams. PNA increased f-insulin in all groups but insulin sensitivity was higher in wt mice from APNtg dams compared to wt mice from wt dams. Insulin resistance correlated with subcutaneous and visceral fat mass. PNA increased visceral fat % and adipocyte size in wt offspring from wt dams while wt and

APNtg offspring from APNtg dams were protected against this effect. Visceral adipose tissue gene expression was unaltered in PNA wt offspring, regardless of the dam's genotype, while APNtg offspring, regardless of PNA, had increased expression of adipogenic genes. Anogenital distance was increased in all PNA wt offspring independent of the dam's genotype. There was, however, no difference between APNtg-vehicle and APNtg-PNA mice, suggesting that adiponectin overexpression protects against this effect. PNA leads to disrupted estrous cycle and fewer ovulations, but this effect was less pronounced in PNA wt mice from APNtg dams. Our data suggests that elevated maternal adiponectin protects the offspring against PNA induced metabolic dysfunction, and to a lesser extent reproductive dysfunction.

Neuroendocrinology and Pituitary CASE REPORTS IN CLASSICAL AND UNUSUAL CAUSES OF HYPOPITUITARISM

A Rare Case of Clival Primary Diffuse Large B-Cell Lymphoma Presenting with Panhypopituitarism and Unmasked Diabetes Insipidus

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Background: Central adrenal insufficiency (AI) is known to conceal diabetes insipidus (DI) by impairing free water clearance from the renal system and inhibiting polyuria, which is later unmasked by the initiation of glucocorticoids. The instances of masked DI in the literature are confined to case reports - the actual incidence is unclear. There have been no documented cases of masked DI secondary to a clival tumor, which remains a rare cause of hypopituitarism. Here we discuss a case of primary diffuse large B-cell lymphoma (DLBCL) of the clivus presenting with panhypopituitarism and the development of DI upon glucocorticoid initiation.

Clinical Case: A 60-year-old man with DLBCL initially presented to an outside institution with headache, diplopia, and right eye ptosis. Brain MRI showed enhancement of the clivus without obvious pituitary gland involvement. He underwent biopsy with partial resection of the tumor and pathology confirmed DLBCL. His course was complicated by persistent fevers. Extensive work-up was unrevealing and he was transferred to our institution for further management. On admission, infectious work-up was positive for C. Difficile. He later developed septic shock requiring vasopressors and broad-spectrum antibiotics. Despite prolonged antibiotics, he remained hypotensive. Due to proximity of the clivus and sella, there was concern for pituitary involvement. A random cortisol was 9.1 [3.7-19.4 ug/dL], subsequent 250ug cosyntropin stimulation test yielded 13.1 and 14.9 at 30- and 60-minutes, respectively. Given inadequate stimulation and persistent hypotension in this critically ill patient, he was then started on stress dose hydrocortisone (HC) with prompt resolution of hypotension. Further work-up of the pituitary axes was consistent with anterior hypopituitarism: ACTH 5 [6-50 pg/mL], TSH 0.336 [0.350-4.940 uIU/mL], free T4 0.5 [0.7-1.5 ng/dL], FSH 0.3/

LH 0.1 [1-10 mIU/mL], IGF-1 <16 [41-279 ng/mL], prolactin <1 [3-16 ng/mL]. On day two of HC stress dosing, he developed hypernatremia and polyuria. Urine studies were consistent with DI and desmopressin was started with subsequent improvement.

Conclusion: This is the first documented case of DLBCL of the clivus leading to panhypopituitarism. For tumors in close proximity to the pituitary, there should be a low threshold for pituitary axes evaluation. In addition, masked DI from central AI remains rare and requires close attention by the Endocrinologist following initiation of glucocorticoids.

Genetics and Development (including Gene Regulation)

GENETICS AND DEVELOPMENT AND NON-STEROID HORMONE SIGNALING I

Prevalence of Renal Cysts in Patients with Carney Complex

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In the general population renal cysts appear most commonly in patients >50 y and in men. Among published studies, the prevalence of renal cysts detected by MRI was 27%, detected by CT was 20-41%, and detected by US was 4-17% (Mensel, et al., 2018; Choi, et al., 2016). In these studies, the male to female ratio in patients with renal cysts ranged from 1.4:1 to 2.93:1. Carney complex (CNC) is an autosomal dominant multiple endocrine neoplasia and lentiginosis syndrome predominantly caused by aberrant cAMP-protein kinase A (PKA) signaling mostly (but not always) due to germline inactivating defects in *PRKARIA* which encodes the regulatory subunit type 1 α of PKA. In a small retrospective study, 5 of 9 subjects with CNC had renal cysts on MRI or CT (Ye, et al., 2017). This same study evaluated the development of renal cysts in kidney-specific *Prkar1a* knockout mice, where all mice developed a renal cystic phenotype. To determine the prevalence of renal cysts, we performed a retrospective cohort study of patients with CNC evaluated at our institution between 1984 and 2019 who underwent renal imaging with MRI, CT, and/or US. We hypothesized that CNC leads to renal formation of cysts in humans, with increased number of renal cysts and earlier age at detection. 117 patients with CNC (69 female [59%], 48 male [41%]) were evaluated with renal imaging (56% MRI, 41% CT, 3% US). Of these, 39 (33%) patients had renal cysts that were first detected on imaging between the ages of 13 and 58 y (mean age at diagnosis 37.1 \pm 12.7 y; 5 [13%] 12-19 y, 5 [13%] 20-29 y, 10 [26%] 30-39 y, 11 [28%] 40-49 y, and 8 [21%] 50-59 y). The mean number of cysts was 1.3 \pm 0.7, and mean dominant cyst size was 1.2 \pm 0.9 cm. Average creatinine at diagnosis was 0.8 \pm 0.2 mg/dl. Of the patients with renal cysts, 22 were