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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#### **SAT-245**

**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 cvsts 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 PRKAR1A which encodes the regulatory subunit type  $1\alpha$  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 ±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 ±0.9 cm. Average creatinine at diagnosis was  $0.8 \pm 0.2$  mg/dl. Of the patients with renal cysts, 22 were