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MON-246

Background Identifying the etiology of hormonal insufficiency is important for correct diagnosis and for appropriate hormone supplementation. Usually multiple hormone deficiencies are driven by pituitary pathology, but here we present a case that poses a unique diagnostic and therapeutic dilemma. **Clinical Case** A 55 y/o lady with HTN and recently diagnosed metastatic Renal Cell Carcinoma (RCC) presented with weakness, dizziness, altered mental status, polydipsia and polyuria. Her metastases included the bilateral adrenal glands, thyroid, brain, and she had a sellar lesion displacing the pituitary as well as infundibular thickening. She had been treated with dexamethasone due to vasogenic edema, whole brain radiation, and was going to start immunotherapy (Ipilumimab&Nivolumimab). On exam she had relative hypotension and tachycardia (BP 115/58, HR 108). She was diagnosed with non-PTH mediated hypercalcemia (Ca 13.9/15.3 corrected for albumin 2.3, PTH 6.5, PTH-RP 69, Vitamin D-25 19.4). Her calcium normalized after fluid resuscitation and bisphosphonate treatment, but weakness and hypotension persisted. We tested thyroid and adrenal function given the location of her lesions, recent whole brain radiation, and recent steroid use. Her TSH was 0.017 (0.270 - 4.200 μIU/mL), fT4 0.84 (0.80 - 1.50 ng/dL), T3 0.59 (72.0 - 153.0 ng/dL), ACTH was 1.7 (7.2-63.3 pg/ml), cortisol 1.3 (6.0 - 18.4 ug/dL), aldosterone 3 (< or = 28 ng/dL), renin 7.87 (0.25 - 5.82 ng/mL/h), renin/aldosterone 0.4 (0.9 - 28.9). At 30 minutes after cosyntropin administration cortisol was 7.7 ug/dL, aldosterone 26 ng/dL, and at 60 minutes cortisol was 10.3 ug/dL. Hydrocortisone was initiated while tapering off dexamethasone, and levothyroxine was offered but declined. She opted to postpone further workup. **Conclusion** This patient presents a dilemma in identifying the primary causes of hormonal abnormalities given potential pituitary and primary thyroid and adrenal disease. The thyroid abnormalities could represent sick euthyroid physiology with lower T4 to T3 conversion or the effects of steroids which can also suppress TSH vs. hypothalamic or pituitary disease. Primary hypothyroidism from gland destruction was unlikely. The low ACTH and cortisol levels were expected since she was on dexamethasone, but low aldosterone with raised renin activity was concerning for primary adrenal insufficiency. However, after cosyntropin the suboptimal cortisol yet preserved aldosterone response supported secondary or tertiary adrenal insufficiency, and only cortisol was supplemented. Understanding her endocrine disease had implications for longer term management given that replacement may only need to be temporary and adrenal recovery might be possible. Further, the use of a 30 minute aldosterone level can be helpful in cases where multiple factors and discrepant laboratory findings may exist.

Steroid Hormones and Receptors

STEROID AND NUCLEAR RECEPTORS

Functional Characterization of Estrogen-Regulated LncRNA16 in ER+ Breast Cancer

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SUN-748

Long noncoding RNAs (lncRNAs) have been demonstrated to be involved in diverse cellular processes as important regulators, such as in cancer. However, their roles in breast cancer biology are greatly unknown so far. In our study, integrated analysis of subcellular fractionation RNA-seq with gene expression profile from human reproductive tissues yielded a comprehensive catalog of estrogen-regulated reproductive tissue-specific lncRNAs. We selected long intergenic noncoding RNA 16 (*LINC16*) for further study as it was the top upregulated lncRNA by estrogen and associates with clinical outcome. Analysis of RNA-seq data from different human tissues, we found that *LINC16* is highly expressed in testis and followed by other reproductive organs, cervix and uterus. Interestingly, interrogation of expression data from human cancer tissues showed *LINC16* is highly expressed in breast cancer compared to other cancers. We have determined the 5' and 3' ends of *LINC16* and its exon/intron structure, and cloned *LINC16* to study its function in molecular and cell-based assays. Our preliminary results suggest that LINC16 plays a critical role in ERα-dependent pathways.

Adrenal

ADRENAL CASE REPORTS II

A Case of Deoxycorticosterone-Producing Malignant Adrenocortical Tumor

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SUN-180

Background: Hypermineralocorticism (hypertension, hypokalemia, and low plasma renin activity) due to deoxycorticosterone (DOC) excess associated with adrenocortical carcinoma is extremely rare. DOC-producing tumors cause primary aldosteronism-like symptoms presenting low plasma aldosterone with very high DOC levels, and due to weak hormonal DOC activity, its diagnostic is done lately. Generally, malignant cases are progressive with a dismal prognosis. **Clinical case:** A 61-year-old woman was admitted to our hospital presenting lumbar pain and weight loss of 8 kg, in 2018. Previously, arterial hypertension was diagnosed in 2015, showing a satisfactory control with two classes of antihypertensive drugs. **Physical exam:** The patient presented no features of Cushing syndrome, but a palpable abdominal mass was noticed in the right flank. Blood pressure was 160x100 mmHg, with sustained high levels, despite regular treatment. **Laboratory**

data: a hypokalemia (K 2.4 mEq/L, nr 3.5 -5.0 mEq/L) and hypernatremia (Na 146 mEq/L, nr 135 to 145 mEq/L), with metabolic alkalosis (venous pH 7.46 and serum bicarbonate 32 mmol/L, nr 23-27 mmol/L) was confirmed. Hormonal tests excluded hypercortisolism and pheocromocytoma. Serum aldosterone and renin were suppressed. Mineralocorticoid precursors dosage was extremely high, DOC (654 ng/dL, nr < 25 ng/dL) and progesterone (5.0 ng/mL, nr <0.89 ng/mL), as well 11-deoxycortisol (7.2 ng/mL, nr <0.5 ng/mL). **Radiological imaging:** abdominal CT showed a heterogeneous hypervascular adrenal mass (13.0x13.0x21.0 cm) exhibiting central necrosis, suggesting malignancy. FDG-PET/CT scan showed a hypermetabolic adrenal mass (SUVmax=13.8). Also, two metabolically active pulmonary nodules (SUVmax=3.7) measuring 0.7 and 0.4 cm were detected. The patient underwent right adrenalectomy, and the tumor was removed (24x13x13 cm). According to Weiss criteria (8/9) and modified Weiss criteria (5/7), the tumor was considered an adrenocortical carcinoma. Immunohistochemistry revealed a low Ki-67 index (10%). After the surgical procedure, all adrenal steroid levels normalized, and mitotane was prescribed as adjuvant therapy. Although the pulmonary nodules were stable at the four-month follow-up, the abdominal CT-scan revealed a heterogeneous nodule (3.7cm) in the left adrenal gland, which was suspicious of metastasis. **Conclusion:** DOC-producing adrenocortical tumors are heterogeneous regarding tumor size, clinical behavior, hormonal and metabolites secretion, and disease-free and overall survival; however, it is common hypokalemia, hypertension, and other symptoms as abdominal pain, due to tumor growth, and weight loss. The association of arterial hypertension with hypokalemia and elevated 11-deoxycortisol, with normal aldosterone and renin, lead to the need for mineralocorticoid precursors evaluation in patients with adrenocortical tumor.

Thyroid

THYROID DISORDERS CASE REPORTS III

Elevated Thyroglobulin Level in Benign Thyroid Nodule

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MON-458

Background: Elevated thyroglobulin levels are most commonly associated with differentiated thyroid carcinoma and these levels are often used to monitor for disease recurrence. Thyroglobulin antibody levels can be elevated in both Hashimoto's Thyroiditis and Graves' Disease and these antibodies can interfere with thyroglobulin levels as detailed by Spencer, Wang (1). With benign thyroid nodules, there is limited research regarding thyroglobulin levels as studied in Chinnappa, et al (2).

Clinical Case: A 31-year woman presented with a palpable thyroid nodule and dysphagia. Her primary care physician ordered thyroid labs including normal TSH (1.3 mIU/L, n0.4-4 mIU/L), normal thyroid peroxidase antibody (56 units, n<250units), normal thyroglobulin antibody level (<1 IU/mL, n<1 IU/mL), and elevated thyroglobulin level (469.1 ng/mL, n2.8-40.9 ng/mL). Her thyroglobulin levels remained elevated on repeat testing (224.4 ng/mL, n2.8-40.9) one month later. In addition, her thyroglobulin lab studies were repeated with HAMA treatment and remained elevated (277.7 ng/mL, n2.8-40.9). Office ultrasound showed longest dimension of nodule to be 5 cm and patient received FNA biopsy. Biopsy results were reported as a benign nodule and it was recommended to follow-up in 12 months. Six months later the patient reported having increasing dysphagia. She underwent Barium swallow which showed no abnormalities. She had a growth increase of 35% on repeat imaging along with increasing neck pressure and discomfort and was referred to an ENT for surgery. Final pathology after left thyroid and isthmus thyroidectomy was reported as "Multinodular hyperplasia with background thyroid parenchyma histologically unremarkable. Negative for malignancy." Thyroglobulin levels subsequently returned to within the normal range and the patient's dysphagia resolved.

Conclusion: Thyroglobulin levels can be markedly elevated with benign thyroid nodules, which can mislead physicians and increase concern for thyroid cancer.

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Pediatric Endocrinology

SEXUAL AND GENDER DEVELOPMENT IN THE PEDIATRIC POPULATION

Mental Health Implications and Determinants of When Youth Present to a Gender Clinic

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OR15-02

Background/Aims: Gender incongruent (GI) youth experience high rates of mental health comorbidities. While gender-affirming medical care (GAMC) provides psychological benefit, GI youth often present to care at older ages. The goals of this study were to 1) assess the relationship between age at presentation to GAMC and rates of mental health comorbidities, 2) identify factors influencing when youth present to GAMC, and 3) determine whether older presenting youth face more barriers to care.