A 61-year-old woman

Steroid Hormones and Receptors

Steroid and Nuclear Receptors

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

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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 seler 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-PITH 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), T4 0.84 (0.80 - 1.50 ng/dL), T3 0.59 (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.

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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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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