pup and maternal loss. We then elected to develop a milder undernutrition model, which may relate more closely to society’s nutritional challenges with the objective of determining if the timing of the leptin surge had shifted. This maternal undernutrition study consisted of dams fed ad libitum (fed) and pair fed dams receiving 20% reduced caloric intake (undernourished). Undernutrition started at E15 and ended with sacrifice at various times during the leptin surge. While nursing, the undernourished dams did not lose weight, but their weight gain was reduced to 45% of that of fed dams. We have collected data from 177 neonatal pups and 19 fed or undernourished dams. At PND5 and PND10, pups from undernourished moms weighed significantly less (16.3% and 21.8%) than pups from fed dams. Additionally, weanlings (PND 21) from underfed dams exhibited a 28.04% reduction in weight and an 8.43% reduction in nose to anus length (p = 0.0005) compared to pups from control fed dams. The timing of the leptin surge in pups from fed dams was normal in female pups. However, pups from mildly undernourished dams had “premature” leptin surges that peaked 2 days earlier than normal. Ongoing studies are testing metabolic function in these mice, as adults, to determine their sensitivity to a 45% high fat diet and the impact on somatotrope functions. This model demonstrates that even a 20% reduction in nutrition will negatively impact offspring and shift the timing of the leptin surge.

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Adrenal

ADRENA L CASE REPORTS II

Oncocytic Adrenocortical Carcinoma: A Case Report

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SUN-179

Adrenocortical carcinoma (ACC) is a rare tumor with a poor prognosis. Oncocytic variant of adrenocortical carcinoma is considered to be an even more rare malignancy with four reported in the literature in 2002 and thirty-six documented in 2015. This case report illustrates the case of oncocytic ACC.

A 40-year-old woman post-hysterectomy from cervical cancer presented to the ER with severe left flank and back pain for two weeks. There was no weight loss or early satiety. CT scan revealed a large left adrenal mass measuring 12×9×13 cm, displacing the left kidney inferiorly with no metastasis to nearby structures. We ruled out functional adrenal mass through a 24-hour urine collection and blood work, then performed open left adrenalectomy. Grossly, it was a large, tan-colored mass, weighing approximately 2,500 grams. On biopsy, we incidentally found the mass to be oncocytic adrenocortical carcinoma. Using the Lin-Weiss-Bisceglia criteria, it met two major and one minor criteria, when only one major criterion is required to indicate malignancy. Immunohistochemical stains showed that the tumor cells were positive for Mart-1, inhibin, and synaptophysin. Quantitative Ki-67 showed a proliferation index of 10% in average (>5% is considered malignant). Even though adrenocortical carcinoma, specifically the oncocytic variant of ACC, is a very rare occurrence, it should be on the differential diagnosis for a chief complaint of unilateral flank pain with a large adrenal mass on CT due to its poor prognosis.

Abstract keywords: Oncocytic adrenocortical carcinoma, ACC, oncocytic variant, adrenocortical carcinoma, adrenocortical carcinoma

Endnotes

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Adrenal

ADRENA L CASE REPORTS I

Adrenocortical Carcinoma in Untreated Congenital Adrenal Hyperplasia

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SAT-207

Background: Congenital adrenal hyperplasia (CAH) is a group of rare inherited autosomal recessive disorders characterized by a deficiency of various enzymes participating in steroid hormone synthesis. It occurs in 1 in 5000 to 1 in 15000 births. The most common (90-95%) cause of CAH is the absence of the enzyme 21-hydroxylase. We are presenting a case of a 34 year old male with untreated congenital adrenal hyperplasia due 21-hydroxylase deficiency and metastatic adrenocortical carcinoma.

Case: A 34 year old male with a history of classic congenital adrenal hyperplasia (CAH) untreated since childhood presented with symptoms of right abdominal pain, anorexia and weight loss. His family history was significant for one brother diagnosed with CAH, father with liver and pancreatic cancer and another brother with colon cancer. After 2 weeks of treatment with NSAIDs for the pain, his abdominal pain worsened and patient also reported dyspepsia with minimal exertion and dry cough. The patient was admitted
A 58-year-old post-menopausal white female presented to UPMC MERCY, PITTSBURGH, PA, USA. She presented with deepening of voice, male pattern hair loss, increased blood pressure, abdominal girth & skin smoothing. Her surgical pathology was diagnostic of adrenal cortical carcinoma.

His baseline morning cortisol level was 22.45 mcg/dL, ACTH 13.2 (n=7.2-63.3), androstenedione 291 ng/dL (n=27-152), 17-hydroxy progesterone 10,850 ng/dL (n=27-199), testosterone 140 mg/dL, renin 1.295 ng/ml/h (n=0.167-5.38), aldosterone unable to assay due to interference and LDH 2011 U/L (n=87-241). He failed an overnight 1mg dexamethasone suppression test with cortisol of 20.17 in the morning. During hospitalization, his clinical condition gradually deteriorated with hypotension, altered mental status, acute respiratory failure and acute liver failure with an AST 2787 units/L (n <39), ALT 399 (n=30-65) and ALP 1013 units/L (n=40-120). Oncology decided that the patient was a poor candidate for antineoplastic treatment therefore he was offered hospice care and eventually expired.

Discussion:
There have been reports of benign and malignant adrenal tumors in patients with CAH. It has been surmised that ACTH is the driver of adrenal tumor transformation in these patients. Our patient with adrenocortical carcinoma presented at a late stage with widespread metastases resulting in death. His elevated cortisol level occurred in association with low normal ACTH and the failed 1mg overnight dexamethasone suppression test are consistent with tumor production of cortisone. Considering that he had untreated CAH since childhood, we assume elevated ACTH levels were present until tumor transformation occurred.

Tumor Biology

TUMOR BIOLOGY: GENERAL, TUMORIGENESIS, PROGRESSION, AND METASTASIS

Postmenopausal Virilization: Rare Case of an Ovarian Tumor Not Easily Identified on Imaging
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SAT-134

Postmenopausal virilization: rare case of an ovarian tumor not easily identified on imaging

Introduction:
Ovarian sex cord-stromal tumors are a rare type of ovarian tumor which can be benign or malignant. Steroid cell tumors are a subtype of these tumors, representing <0.1% of all ovarian neoplasms (1). Here, we present the case of a 58 year old post-menopausal female who presented with virilization in the setting of bilateral adrenal adenomas & pelvic ultrasound without a definitive mass.

Case Report:
A 58 year old post-menopausal white female presented with deepening of voice, male pattern hair loss, increased muscle mass, weight gain, citoromegaly, acne, increased axillary & facial hair growth. This had occurred over a period of 1.5 years. Initial investigatory labs revealed markedly elevated testosterone level of 630 ng/dL (n 2-45ng/dL). Normal FSH/LH, morning ACTH and cortisol, DHEAS, 17 OHP, androstenedione, prolactin and IGF1. Exogenous intake of testosterone was excluded. A transvaginal ultrasound showed thickened and cystic endometrial lining but no cysts or masses in the ovaries. An endometrial biopsy was normal. In the absence of a definitive source of elevated androgens, CT abdomen pelvis was done & showed remarkable for bilateral adrenal adenomas. Serum metanephrines were normal. An overnight dexamethasone suppression test was abnormal; morning cortisol level 3.1 (n 4.0-22.0). With a normal DHEAS these adenomas were considered to be the less likely etiology of her virilization. Given concern for an ovarian malignancy, a hysterectomy was recommended.

Her total testosterone level right before surgery was 954 ng/dL. Post-operatively, not only did the total testosterone levels drastically fall to 18 ng/dL merely on POD 4, but the patient reported new scalp hair growth, decreased abdominal girth & skin smoothing. Her surgical pathology was with that of a steroid tumor of the ovary.

Conclusion
Steroid cell tumors of the ovary can be benign or malignant. A prompt diagnosis is critical. In the presence of elevated testosterone levels or virilizing symptoms, an ovarian etiology must be suspected. Given the malignant potential of these tumors, if there is clinical suspicion, an expedited total hysterectomy and bilateral salpingo-oophorectomy is recommended. The objective of surgical treatment is to relieve symptoms and for staging in the case of malignancy.

Timely management of the tumor can reduce metastasis and significantly improve quality of life, as seen in our case.

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