were acromegaly types 2 and 3 (OR=2.7; IC95% 1.9-6.7, p=0.005), and radiotherapy (OR=0.36, 0.18-0.70, p=0.003). **Conclusions:** Type 3 acromegaly patients showed higher frequency of comorbidities, disease activity, and risk of mortality, adjusted for treatment, than type 2 and type 1 patients.

References

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Tumor Biology

ENDOCRINE NEOPLASIA CASE REPORTS II

Testosterone Secreting Ovarian Tumor: A Rare Cause of Erythrocytosis and Pulmonary Embolus

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Background: Ovarian testosterone producing tumors are an uncommon cause of hirsutism, pulmonary embolus and polycythemia.

Clinical Case: A 67-year-old Caucasian postmenopausal woman was referred for Endocrine evaluation of a one-year history of hirsutism, weight gain and elevated total testosterone level of 672 ng/dL (n <75 ng/dL). She reported increased hair growth on her chin for the past year. She denied any male pattern hair loss or any increased hair growth on her legs or chest. She also reported unintentional weight gain of thirteen pounds and low energy levels. Past medical history is remarkable for gastroesophageal reflux disease. Her last menstrual period was at age forty and periods occurred at regular intervals without heavy bleeding. She never became pregnant by choice. At this time physical exam was notable for shaved terminal hair on her chin, vellus hair on abdomen, there was no hirsutism or voice change noted. There were no abdominal striae, moon facies, buffalo hump.

Labs from the initial visit included a total testosterone of 672 ng/dL (n<75 ng/dL), 2pm cortisol 5.6 ug/dL (2.7-10.5 ug/dL in PM), TSH 2.28 uiU/mL (0.27 – 4.20), Androstenedione 133 ng/dL (n<10-93 ng/dL), 11 deoxycortisol 29.3ng/dL (n <32), ACTH 12 pg/mL (n<46), DHEAS 87 ug/dL (13-130 ug/dL) free testosterone index 24.2 (n<2.1), 17-hydroxyprogesterone 207ng/dL (n<272), FSH 26.1 miU/mL (25.8-134.8), LH 17.9 IU/L (7.7-58.5), Hemoglobin 18. g/dL (11.7-15g/dL), hematocrit 50.4% (35-45%), HbA1c 5.4%.

Four months after this initial presentation, the patient developed acute onset of chest pain and shortness of breath and was diagnosed with a right pulmonary embolus on CT Chest Angiogram. Evaluation with imaging for an ovarian mass revealed a negative workup. CT Abdomen Pelvis did not reveal any adrenal masses. Transvaginal Ultrasound did not demonstrate any ovarian masses. MRI of pelvis did not show any adnexal masses.

Despite the negative imaging, because of the markedly elevated testosterone levels, this presentation was thought to correspond to a testosterone secreting ovarian tumor. Furthermore, the normal adrenal hormone levels and significantly elevated testosterone level is consistent with neoplasm range. High levels of testosterone are known to cause a hypercoagulable state leading to a PE. Furthermore, testosterone causes an increase in RBC mass leading to marked elevations in hemoglobin. The patient was referred for bilateral oophorectomy. Pathology of the right ovary revealed a 2 cm steroid cell tumor, not otherwise specified.

Conclusion: This is the first known case of a steroid cell ovarian tumor, not otherwise specified, that secretes testosterone leading to pulmonary embolism and erythrocytosis. Furthermore, testosterone secreting tumors should be considered in the diagnosis of PE as a rare cause of hypercoagulable state.

Reproductive Endocrinology

HYPERANDROGENISM

Developmental Programming: Depot-Specific Inflammatory State and Distribution of Thermogenic Adipocytes Programmed by Gestational Testosterone Excess

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Prenatal testosterone excess induces insulin resistance, dyslipidemia and cardiovascular defects in ewes. Considering adipose depots influence systemic metabolic status, disruptions in visceral (VAT), subcutaneous (SAT), pericardiac (PCAT) and perirenal (PRAT) adipose depots may contribute to these metabolic defects. While the changes in PCAT and PRAT in the prenatal T-treated sheep are unknown, we found an increase in the oxidative stress and inflammatory state in the VAT but not SAT, while insulin sensitivity is maintained in both depots (Biol Reprod 2016;94:113). Although all four depots are rich in white adipocytes that specialize in lipid storage, it is not clear what underlies the depot-specific differences. A potential contributor may relate to depot-specific differences in presence of brown/beige adipocytes. Brown/beige unlike white adipocytes are thermogenically active due to their expression of uncoupled protein 1 (UCP1), which uncouples mitochondrial oxidative phosphorylation to release stored energy as heat. Because they favor lipid utilization rather than storage they promote a metabolically healthy phenotype. We hypothesized that the relative distribution of brown/beige adipocytes may contribute to the depot-specific changes in inflammatory state. To test this, adipose depots from control (n=6) and prenatal T- (100mg T propionate twice a week from days 30-90 of gestation)-treated (n=5) female sheep were studied at 21 months of age. The changes in expression of inflammatory and thermogenic adipocyte markers were assessed by real time RT-PCR and data analyzed by Student's t-test and Cohen's effect size analysis. Prenatal T-treatment induced 1) significant increase in inflammatory cytokine interleukin 6 in VAT and PRAT while decreasing it in PCAT 2) significant increase in tumor

necrosis factor in the VAT and a trend (evident by effect size analysis only) for an increase in SAT and PCAT and 3) significant increase in PRAT and a trend toward increase in the macrophage marker CD68 expression in VAT. Among the thermogenic gene markers, the expression of UCP1 was significantly increased in VAT and PCAT with a trend for an increase in PRAT. The expression of UCP2 and PPAR gamma co-activator 1 beta (PPARGC1B) were also significantly elevated in VAT with a trend for an increase in SAT. These findings are indicative of depot-specific differences in prenatal T-induced inflammatory status with effects being pronounced in VAT compared to other depots. The increases in thermogenic markers in adipose depots do not support our hypothesis but rather are reflective of a compensatory response to promote adipose depot insulin sensitivity and may have a bearing on function of organs in the proximity of respective depots. These findings are likely of translational significance in metabolic disorders associated with hyperandrogenic state.

Tumor Biology

ENDOCRINE NEOPLASIA CASE REPORTS II

Primary Neuroendocrine Tumor of the Central Nervous Sistem, a Case Report and Literature Review

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Introduction

Neuroendocrine Neoplasms are rare, with an incidence of 5 to 100,000 inhabitants, constituting 1% of all malignancies, presenting high survival rates in general, even in metastatic diseases. However, in those poorly differentiated, as in the following case, survival is around 4% in 5 years. We will describe a case of primary neuroendocrine tumor in the brain, of which is uncommon in the literature.Clinical case

A 26 years women was referred to the ER of Santa Casa de São Paulo, in January 2019, to be evaluated by neurosurgery, due to progressive left hemiparesis and headache for 3 months, which got worse in 4 days. On CT scan, there was a 6 x 6 cm solid-cystic, expansive, lesion in the right frontal lobe, with perilesional edema and contralateral midline 1.3cm deviation and subfalcine herniation.

Thus, the tumor was resected soon, with anatomopathological analysis showing poorly differentiated tumor of cells with scarce cytoplasm, hyperchromatic nuclei and high mitotic activity.

Immunohistochemical analysis finds 50% Ki67, with focal p53, TTF1, CD99, CD 56 and synaptophysin positivity. The main hypotheses, then, consisted of Neuroendocrine Carcinoma.

Four months after surgery, the patient reported worsening deficit, headache, pain, weight loss, being referred to the Emergency Room, once more. In RM an expansive lesion was found 6.6 x 4.4 cm, in the right

frontoparietal surgical cavity, edema, compression and 0,4 cm midline deviation.

The patient was once again submitted to emergency neurosurgery, with microsurgical resection. The pathology was identical to the previous one.

We proceed with hormonal evaluation, regarding to Medular Thyroid carcinoma, Gastrinoma, Insulinoma, Pheochromocytoma, Carcinoid tumor and others.

Imaging exams were also performed to investigate other primary sites: no changes in CT scan of the chest and abdomen and PET CT FDG. However, this one showed recurrence of the intracranial lesion, with three sites of involvement, all hypermetabolic: one of 4.1 x 2.9 cm (SUV 4.9) and another of 3.9 x 3.3 cm (SUV 8, 4) in the right frontoparietal region and medial nodule to the right thalamus of 1.2 cm (SUV 6.1).

Patient currently maintain left hemiparesis, frequent pain, taking carbamazepine due to epileptic seizures, and considerable weight loss. She has an important limitation of daily activities and basic self-care, with 50% Karnofsky scale. Due to relapse, palliative radiotherapy was initiated in the region of the tumors.

Conclusion

The patient had a poor outcome in relation to cancer, with little possibility of treatment due to poor tumor differentiation and poor performance status.

Adipose Tissue, Appetite, and Obesity

RARE CAUSES AND CONDITIONS OF OBESITY: PRADER WILLI SYNDROME, LIPODYSTROPHY

Identification of NASH Using Data from NHANES III

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Nonalcoholic steatohepatitis (NASH) is a serious liver condition marked by hepatic steatosis (HS), cell damage and inflammation. Patients with NASH are at risk for developing cirrhosis and hepatic cancer. Currently, the definitive method of diagnosing NASH is by liver biopsy. To avoid the costs and risks associated with biopsy procedures, there has been considerable effort to develop a non-invasive method of identifying patients with NASH. However, none of these methods has become accepted as a "gold standard." Our objective was to compare three non-invasive methods of identifying NASH by using data from NHANES III (1988-1994) to determine variables associated with published formulas to identify NASH. We used ultrasound data to identify subjects with moderate - severe HS. Among those with HS, we identified the NASH population using either the HAIR score, the NASH liver fat score, or the Gholam score. The HAIR score was developed in a sample of obese patients, is based on hypertension, insulin resistance and alanine transaminase (ALT) levels, and had an AUROC of 0.9, a sensitivity of 0.8, and a specificity of 0.89. The NASH liver fat score was developed in a Finnish population undergoing gastric bypass, and validated in an Italian population of liver biopsy patients. This score incorporates metabolic syndrome, type 2 diabetes, serum insulin, AST, and ALT. In the Finnish and Italian populations, respectively,