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MON-683

Objectives: To determine the frequency of diabetic foot risk of ulceration and associated factors in patients with type 2 diabetes mellitus.

Methods and materials: We used a cross-sectional descriptive design. Data collection was performed in the foot at risk office at Maria Auxiliadora hospital over a period of 3 years, dating from the october 2016 to september 2019.

Foot risk assessment was based on the International Working Group of Diabetic Foot (IWGDF) system, which evaluates peripheral neuropathy (with monofilament or tuning fork), biomechanical deformity, peripheral arterial disease (altered pulse or ankle branchial index) or a history of foot ulcer[1,2]. Foot risk frequency was found according to epidemiological characteristics, and the prevalence ratios with 95% confidence interval were calculated to association analysis, we perform bivariate and multivariate analysis using a generalized linear model. This study has the approval of the María Auxiliadora Hospital´s Ethics Committee.

Results: a total of 402 subjects were included in this study, 63.3% were women, average age 62 yo and diabetes duration <10 years in 56%. 76.6% presented risk to develop ulcer, of which 54.7% presented biomechanical deformation, 37.3% Peripheral Neuropathy (NP), 35.4% Peripheral Arterial Disease (PAD), and 12.7% history of foot ulcer. Patients with foot at risk were associated with older age (RP 1,006, 95% CI 1,001-1.01), and with a score> 5 with respect to <5 in the Total Symptom Score (RP 1.26, 95% CI 1.05-1.51).

Conclusions: 3 of 4 patients have a foot at risk of ulceration, predominantly due to biomechanical deformation and peripheral neuropathy, and this risk was associated with older age and greater pain symptomatology.

References

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Adrenal

ADRENAL CASE REPORTS II

Metastatic Spindle Cell Sarcoma Unmasked by Bilateral Adrenal Hemorrhage Resulting in Adrenal Insufficiency

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SUN-163

Introduction:

Adrenal hemorrhage (AH) is rare and can be life-threatening when bilateral AH causes adrenal insufficiency (AI). Risk factors include trauma, stress, sepsis, anticoagulant and antiplatelet use, hematologic disorders, and underlying adrenal tumors. We describe a patient whose bilateral AH led to a diagnosis of an underlying malignancy and caused AI.

Clinical case:

A 71-year-old man with well-controlled HIV presented with fatigue, weight loss, and acute lower abdominal pain.

Four months prior to presentation, he underwent hip arthroplasty. His post-operative course was complicated by multiple pulmonary emboli and a new left 11.6 x 7.3 x 8.9 cm cystic retroperitoneal lesion with a density of 29 Hounsfield units on CT, thought to be a pancreatic pseudocyst or adrenal or retroperitoneal hemorrhage. Since the size remained stable on repeat CT three days later, he was discharged on rivaroxaban. On the day of presentation, he acutely developed severe abdominal and back pain. CT scan revealed a new 8.0 x 7.8 x 7.8 cm right adrenal collection and increased size of the prior left adrenal lesion to 13.1 x 10.6 x 13.0 cm. MRI confirmed bilateral adrenal masses with intralesional AH, as well as numerous peritoneal and retroperitoneal implants not noted on prior imaging.

He remained stable and was managed non-operatively. Sodium (Na) and potassium (K) ranged 134-138 mmol/L (135-145) and 3.7-4.3 mmol/L (3.5-5.1), respectively. On presentation, morning cortisol and ACTH were 11 ug/dL and 27 ng/L (6-50), respectively, with an undetectable aldosterone and PRA 0.65 ng/mL/hr (0.25-5.8). Subsequent ACTH levels were 71 and 102 ng/L, and cortisol levels were 12 and 14 ug/dL. ACTH-stimulated cortisol was 15 ug/dL and free cortisol was 0.88 ug/dL. Plasma metanephrines were normal. Hydrocortisone was started and anticoagulation was held indefinitely. Biopsy of a retroperitoneal implant revealed metastatic spindle cell sarcoma.

Three weeks later, given a persistently low Na of 134 mmol/L and increased K of 4.7 mmol/L, although blood pressure and heart rate were normal, he was empirically started on fludrocortisone. He followed up with oncology and was started on palliative chemotherapy.

Clinical lessons:

AH should prompt evaluation for an underlying etiology. In our patient, we suspect he already had a unilateral adrenal metastasis causing the initial unilateral AH, as he had no other risk factor. Four months later, the subsequent bilateral AH was likely caused by further metastatic spread and exacerbated by anticoagulation therapy.

This case also suggests that AH may preferentially affect the zona fasciculata of the adrenal cortex and cause glucocorticoid deficiency, a phenomenon which has been noted on prior case reports. Our patient only needed hydrocortisone replacement initially, followed by fludrocortisone replacement three weeks later.

Pediatric Endocrinology

PEDIATRIC ENDOCRINE CASE REPORTS II

Precocious Puberty and Hypothyroidism in a Pediatric Case

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MON-058

Background: Hypothyroidism with secondary sellar/suprasellar mass is rarely associated with precocious puberty. Here we describe a rare case of pediatric hyperprolactinemia and precocious puberty secondary to hypothyroidism, marked TSH elevation, and pituitary hyperplasia.

Clinical Case: A 9-year-old female, with onset of thelarche and menses occurring at age 7 and 8 respectively, presented with primary hypothyroidism (Free T4 <0.11; n=4.9-11.4mcg/mL), elevated TSH (1620.0mU/mL; n=0.3-4.7mU/mL), hyperprolactinemia (108.6ng/mL; n=3.0-23.1ng/mL), and elevated serum estradiol (37.6pg/mL; n=10pg/mL). The patient had coarse scaly skin, diminished energy, and poor growth lasting 1 year. There were no associated gastrointestinal issues, temperature intolerance, nor visual impairments noted during this time.

Magnetic resonance imaging revealed a large mass (1.48cm) with suprasellar extension and a mass effect on the optic chiasm. The patient was then started on Levothyroxine and Cabergoline, to reduce serum prolactin levels. However, upon follow-up two months later, the patient had hypoprolactinemia (2.0ng/mL; n=3.0-23.1ng/mL). The patient was referred to neurosurgery for resection of the sellar mass. Endocrinology was also consulted, at which point Cabergoline was discontinued and Levothyroxine was gradually increased.

Follow up 4 months later showed prolactin levels had normalized to 11.4ng/mL (3.0-23.1ng/mL). Serum LH and FSH were within normal ranges (1.2mIU/mL and 4.2mIU/mL, respectively). TSH, though still elevated (47.35mU/mL; n=0.3-4.7mU/mL), was significantly reduced compared to the prior measurement (1620mU/mL). Serum levels of Free T4 increased to 1.06mcg/mL (n=4.9-11.4mcg/mL). Levothyroxine was titrated up and a repeated pituitary MRI demonstrated a significant decrease in the size of the mass with resolution of the suprasellar extension and mass effect on the optic chiasm. Further, the patient's menses ceased and thelarche resolved upon correction of T4 and regression of the pituitary mass.

Conclusions: While rare, primary hypothyroidism and TSH-driven pituitary hyperplasia can result in a large mass effect with suprasellar extension, causing secondary hyperprolactinemia by a mass effect and central precocious puberty. This case highlights the benefits for evaluating underlying hypothyroidism as a cause for hyperprolactinemia and sellar/suprasellar mass.

Reproductive Endocrinology

TRANSGENDER MEDICINE AND RESEARCH

Hormone Replacement Therapy in Transgender Unmasks Testicular Tumor

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SUN-040

Introduction In transwomen, hormonal replacement therapy typically usually consists of anti-androgen and/or estrogens. Per Endocrine Society 2017 guidelines, patients should be evaluated every 3 months in the first year for appropriate signs of feminization and adverse reactions. This includes measuring serum testosterone every 3 months initially and the testosterone level should be < 50 ng/dL. We describe a case of a transwoman patient diagnosed with testicular seminoma during surveillance of hormonal replacement therapy (HRT). **Case presentation** A 31-year-old male-to-female transgender presents to the endocrine clinic for HRT. She previously had seen another provider for HRT and was started on estradiol valerate 5 mg weekly and leuprolide 3.75 mg every month. After 5 months of therapy, she reported that her testosterone level remained elevated, so spironolactone 100 mg BID was subsequently added. Despite adherence, she was dismayed at how little physical changes she achieved after 1.5 years of HRT. She denies taking exogenous substances or OTC mediation containing androgens. Her examination reveals a male habitus and musculature with male voice, male diamond pubic hair pattern, adult penis size and scrotum measuring 20 cc's bilaterally. Initial labs revealed total testosterone 131 ng/dl, free testosterone 30.8 ng/dl, bioavailable testosterone 64.6 ng/dl, SHBG 12 nmol/L, LH < 0.07 IU/L, FSH < 0.1 IU/L, and estradiol 146 pg/ml. Due to non-suppressed testosterone level despite undetectable gonadotropins, adrenal androgen labs were obtained which was normal. However, her HCG-beta tumor marker was elevated, 12 IU/L (0-3). This prompted a scrotal ultrasound which revealed 3.2 cm right testis mass. Follow-up PET/CT revealed increased activity localized to the right testis without findings of metastasis. Patient underwent right orchiectomy with pathology revealing seminoma stage 1A pT1bMx. At 2 months postop, her total testosterone is now 8 ng/dl, and she reports that her breast tissue has increased and skin softened. **Conclusion** Hyperandrogenism can be easily diagnosed in females given more obvious clinical features; however, except for precocious puberty, there are typically no obvious features of exogenous testosterone production in males. Thus, typically no workup is undertaken in males to look for underlying cause, including testicular cancer. While presence of scrotal mass is the most common initial presentation, patients can be asymptomatic until tumor burden is high and there is metastasis. Transwomen should be monitored by obtaining estradiol and testosterone levels following the Endocrine Society guidelines. If patient is on GnRH agonist, her testosterone level should be almost completely suppressed (T <50). A testosterone level > 50 ng/dL while on GnRH therapy should warrant workup for exogenous sources, including testicular cancer and adrenal abnormalities.

Tumor Biology

TUMOR BIOLOGY: DIAGNOSTICS, THERAPIES, ENDOCRINE NEOPLASIAS, AND HORMONE DEPENDENT TUMORS

Luminescence-Based Drug Screen for Novel Androgen Receptor Antagonists for Prostate Cancer Therapy

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