MON-908

Intro: Carcinoid tumors are rare, slow growing, indolent neuroendocrine tumors typically originating from enterochromaffin in the gastrointestinal tract and bronchopulmonary tree[1]. While often found to be secreting serotonin, many different secretory products have been described[2]. We present the case of a patient with refractory hypercalcemia due to a carcinoid tumor producing parathyroid hormone related peptide (PTHrP). Case: A 65-year-old male was found to have hypercalcemia of 14.7 mg/dL after presenting for nausea and vomiting. He was treated with Zolendronic acid and intravenous (IV) fluids as initial work-up revealed an appropriately suppressed parathyroid hormone level, no monoclonal spike, and a PTHrP that was dramatically elevated. He refused further work-up initially but was admitted two months later for persistent severe hypercalcemia. Computed tomography imaging showed innumerable liver lesions. Histologic analysis of the largest liver lesion was consistent with carcinoid tumor. For the next two years, he was managed outpatient with Pamidronate, Denosumab, and Sandostatin, along with two liver embolizations. Control of serum calcium levels became more difficult and he had multiple hospitalizations for symptomatic hypercalcemia until chemotherapy, Sunitinib, was initiated. Calcium levels normalized for one year after starting Sunitinib prior to onset of suspected medication-induced pancreatitis. He was switched to Everolimus but did not respond to that and was readmitted mere weeks later for symptomatic hypercalcemia and a combination of Folinic acid, Fluorouracil, and Oxaliplatin (Folfox) was started. He continued to get frequent bisphosphonates and IV fluids along with Folfox but several months later he stopped responding to all medical options. His calcium level climbed to 19.9mg/dL and he underwent a technically complicated surgical procedure in which significant tumor burden was removed from his liver. Since surgery, the patient has remained normocalcemic without additional medical therapy. Discussion: Carcinoid tumors are uncommon with reported incidence of 40 per one million people[2]. PTHrP is most commonly produced by squamous cell lung cancer, renal cell cancer, gynecologic cancers, and lymphoma[3]. Carcinoid tumors producing PTHrP with resultant hypercalcemia is rare with a few cases reported in literature[4]. Our patient had a complex treatment course including IV fluids, anti-resorptive agents, somatostatin analogs, liver embolization, chemotherapeutic agents, and eventual surgical debulking. Surgical intervention is not commonly required for carcinoid tumors[5]. This patient had a rare tumor, producing an uncommon hormone, and required extensive treatment. This case shows the importance of a multidisciplinary approach in patients with hypercalcemia secondary to carcinoid tumors but refractory to traditional therapy.

Pediatric Endocrinology

PEDIATRIC ENDOCRINE CASE REPORTS II

Persistent Progressive Clitoromegaly Is Not Always Hormonal: When One Disease Fits All

Meenal Gupta, MD[1], Vincent Horne, MD[1], Abhishek Seth, MD[1], Duong Tu, MD[1], Yemi Adeyemi-Fowode, MD[1], Lefkothea P. Karaviti, PHD,MD[2].

[1]Texas Children's Hospital, Houston, TX, USA, [2]Baylor College of Medicine, Houston, TX, USA.

MON-064

Introduction: Clitoromegaly presenting in childhood can be congenital or acquired. The most common cause is exposure to excess androgens in fetal or neonatal life. However, non-hormonal causes like neurofibromatosis type 1 (NF-1), epidermoid cysts, tumor syndromes have been reported. An asymmetric or irregular appearing clitoris is usually caused by a non-hormonal process.

Clinical Case: A 6-year-old female with NF-1 and right-sided hemihypertrophy was referred to endocrinology due to progressive clitoromegaly since birth. NF-1 features included café-au-lait spots, bilateral optic nerve gliomas, plexiform neurofibroma, Lisch nodule, first degree relatives with NF-1 (sister and mother). At age 1.5, a hormonal work up was negative for hyperandrogenism. At age 2, patient was seen by genetics, and by urology for removal of a bladder neurofibroma, but did not return to these specialties for follow up. Lumbar spine MRI, obtained for back pain, revealed a large sciatic plexiform neurofibroma. She followed with oncology for cancer surveillance and due to parental concern for progressive clitoromegaly was referred to endocrinology at age 6. At the endocrinology visit, parents denied breast development, vaginal discharge or bleeding, axillary or pubic hair, body odor or acne. Her genital exam revealed a clitoris 3 x 1.5 cm in size, Tanner 1 pubic hair, no palpable gonads, no labial fusion but asymmetric labial sizes (right>left). A hormonal workup was normal including 41 ng/dL 17-hydroxyprogesterone (n ≤137 ng/dL), 20 ng/dL androstenedione (n ≤ 45 ng/dl), 42 ng/dL unconjugated DHEA (n ≤ 487 ng/dL), 11 mcg/dL DHEA Sulfate (n ≤ 34 mcg/dL), 3 ng/dL total testosterone (n ≤ 21 ng/dL) and pre-pubertal LH, FSH and estradiol levels. Patient was referred to a multi-disciplinary DSD (Disorders of Sexual Differentiation) clinic for further evaluation and potential surgical options. A pelvic ultrasound and subsequent pelvic MRI revealed that the large sciatic plexiform neurofibroma, detected on the prior MRI, had now extended into the clitoris and right labia. Uterus and ovaries were pre-pubertal in size. Surgical options were discussed in a multi-disciplinary approach. Since clitoral enlargement was contiguous with posterior bladder mass and vital organ functions were not affected, resection was not recommended. Clitoral reduction for cosmetic reasons had a potential risk of recurrence. Since benefits did not outweigh the risks, family chose to not pursue any surgical intervention.

Conclusions: NF-1 is a rare but potential non-hormonal cause of clitoromegaly. In the absence of clinical evidence of hyperandrogenism, clitoromegaly in a patient with NF-1 does not warrant an extensive hormonal work up. Pelvic imaging should be pursued first, to search for local neurofibromas. Decision for surgical interventions requires a multi-disciplinary approach with detailed discussion of benefits vs. risks.

Diabetes Mellitus and Glucose Metabolism

DIABETES COMPLICATIONS II

Assessment of Features Associated with Diabetic Foot Risk in General Hospital in Lima-Peru

Marlon Augusto Yovera-Aldana, MD, MSc, Sonia Helen Perez-Cavero, MD, Candy Ivoone Sipiran, MD, Haydee Barrios, MD, Eduardo Callacna, MD, Delia Cruz-Estacio, MD, Diana Consuelo Flores, MD, Lorena Roncal, MD, Julio Huayta, MD,