to persons with colon and gynecologic cancers, but worse than breast cancer.[1] The SF-36 QOL questionnaire has shown to be a valid tool for assessing QOL in thyroid cancer subjects.[2] We evaluated QOL in both thyroid nodule and thyroid cancer patients in an institutional cancer registry.
METHODS: The Short Form-36 data version 2 (SF-36) QOL data was obtained from single institution prospective bioinformatics thyroid cancer and benign nodule registry. Physical and mental health scores from the SF-36 questionnaire were obtained from both thyroid cancer and nodule patients. Physical and mental QOL responses were scored on a scale from 20-80 and categorized as either "the same or better than the average population,"; "below average,"; or "well below average." A two sample Wilcoxon rank sum test and a chi-squared test were used to compare QOL between thyroid cancer and nodule subjects, using QOL as a continuous or categorical variable. Univariate descriptive statistics and bivariate analyses were performed using a Wilcoxon Rank Sum and Chi-squared test for categorical QOL data and Kruskal-Wallis for continuous QOL data.
RESULTS: We analyzed 321 thyroid cancer and 32 nodule subjects who completed the SF-36 at a single point in time after diagnosis. There was no difference between the groups with regard to sex, age or QOL scores overall. Average age was 43 and 48 years in cancer and nodule groups respectively. Average Physical QOL score was 50.8 (standard deviation SD + 8.8) and 29.6 (SD+ 12.1), respectively (p=0.42). Average Mental QOL score was 48.9 (SD + 9.9) and 48.3 (SD + 8.1) respectively, p=0.16. Physical QOL score was significantly decreased as cancer stage increased when evaluating results as a continuous variable: Stage 1: 51.8 + 8.7; Stage 2: 50.6 +9.5; Stage 3: 48.7 + 7.2; Stage 4: 47.4 + 9.2. Results for mental QOL as a continuous variable by cancer stage showed improvement in reported QOL with increasing cancer stage: Stage 1: 48 (SD+ 9.9); Stage 2: 50.6 (SD + 11.1); Stage 3: 51.1 (SD + 9.13); Stage 4: 53 (SD+ 6.4), p=0.03.
CONCLUSIONS: We found no overall difference in physical nor mental QOL between patients with thyroid cancer or benign thyroid nodules. Overall, QOL was in the "same or better than average" for all respondents, but low cancer stage was associated with higher physical and lower mental QOL scores.
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SAT-470
Hyperthyroidism is associated with multiple cardiac pathologies including dilated cardiomyopathy, isolated right ventricular heart failure, and atrial fibrillation (AF). Long standing untreated hyperthyroidism in conjunction with AF can cause severe dilated cardiomyopathy with reduced ejection fraction that is completely reversible with treatment. We present the case of a previously healthy male who presented with florid congestive heart failure (CHF) as an initial presentation for hyperthyroidism. A 37-year-old male presented to the emergency department with progressively worsening dyspnea on exertion and lower extremity edema for one month. His heart rate was noted to be 172 bpm and an EKG was done that showed AF. He was clinically noted to be in heart failure and was admitted for further management. He was started on metoprolol with good heart rate control and was started on furosemide for diuresis. A transthoracic echocardiogram was done and showed severe global hypokinesis with left ventricular ejection fraction reduced to 20% along with bi-atrial enlargement and dilated left ventricular cavity. Ischemic cardiomyopathy was ruled out with left heart catheterization. A TSH level was checked as a part of workup for non-ischemic cardiomyopathy and atrial fibrillation and was markedly reduced to <0.01mIU/L with free T4 of 1.49ng/dL and free T3 of 6.7ng/dL. A diagnosis of hyperthyroid cardiomyopathy with concomitant tachycardia induced cardiomyopathy was made. Autoimmune workup was negative for anti-thyroid-peroxidase and anti-thyroid-stimulating antibodies. Ultrasound of his thyroid gland revealed multiple thyroid nodules concerning for toxic multinodular goiter. He was started on methimazole and discharged after volume optimization with diuresis to closely follow up with endocrinology and cardiology for further management. CHF can be the primary presentation in about 6% of patients with hyperthyroidism. T3 is the main thyroid hormone that binds to cardiomyocytes. It increases the expression of beta-adrenergic receptors on cardiomyocytes and subsequently increases heart rate and contractility. T3 can also cause atrial arrhythmias such as AF by decreasing the parasympathetic tone. Concomitant AF and hyperthyroidism can cause reduced ejection fraction due to tachycardia induced cardiomyopathy and dilated cardiomyopathy. Treatment mainly is with beta-blockers that slow down the heart as well decrease serum T3 levels by blocking 5-monodeiodinase which converts T4 to T3. Our patient was started on beta-blocker and methimazole with good reduction in heart rate and improvement of symptoms. Recovery of cardiac function will be assessed with longitudinal follow up. As hyperthyroidism is one of the few causes of CHF that is completely reversible, clinicians must maintain low degree of suspicion in patients with new onset heart failure especially when associated with AF.

Thyroid

THYROID DISORDERS CASE REPORTS II

When The Heart Can't Clap To The Thyroid's Beat

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Tumor Biology

ENDOCRINE NEOPLASIA CASE REPORTS II

Carcinoid Causing Catastrophic Calcemia

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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

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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

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