Mental status returned to baseline with improvement in memory function and in gait and balance. Creatinine kinase trended down to 688 U/L over the next 2 days. She did not exhibit any additional behavior of eating non-food objects during the hospital stay. Patient was diagnosed to have severe hypothyroidism presenting as pica along with hyponatremia and rhabdomyolysis. Patient was discharged on levothyroxine 75 mcg daily.
Clinical lessons/conclusions:
Hyponatremia, rhabdomyolysis, and psychosis are relatively uncommon but known reported complications of untreated primary hypothyroidism. Acute psychosis has been reported as a presenting feature of undiagnosed hypothyroidism, but hypothyroidism presenting as pica as seen in our patient is extremely rare. This case highlights the importance of screening for hypothyroidism in patients presenting with unexplained pica.

Thyroid

THYROID DISORDERS CASE REPORTS II

Thyrotoxicosis Presenting as Right Sided Heart Failure: Two Case Reports, Two Differing Outcomes Illustrating the Importance of Early Aggressive Treatment to Reverse Cardiac Dysfunction

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SAT-495

Background: Graves' thyrotoxicosis can lead to life-threatening right heart failure requiring urgent treatment. We present two such cases to illustrate management challenges and the importance of early definitive treatment. **Clinical cases:** The first case is a 41 year old female with Graves' hyperthyroidism and severe right heart failure. Cardiac ECHO showed LVEF 76% with severe biatrial enlargement, TV regurgitation, and an enlarged RV. The liver was enlarged, transaminases were high, bilirubin was 24 mg/dl (0-1.4), and clotting was prolonged. Due to hepatic dysfunction, methimazole was held. She was not a candidate for I-131 as she had received iodinated contrast. Treatment with dexamethasone and SSKI normalized thyroid function over four days. She was discharged but presented two months later with worsened hyperthyroidism and right heart failure. She was a poor surgical candidate and I-131 was administered as definitive therapy. She developed PEA arrest the following day without recovery leading to withdrawal of care and death eight days later.
The second case is also a 41 year old female with an 18 year history of Graves' disease, intermittently compliant with methimazole. She presented with hyperthyroidism and severe right heart failure. The liver was enlarged, transaminases were high, bilirubin was 5.1 mg/dl, and clotting was prolonged. Cardiac ECHO showed LVEF 66% with severe biatrial enlargement, TV and MV regurgitation, enlarged RV, and increased RA pressure. Due to hepatic dysfunction, methimazole was held. She was not a candidate for I-131 as she had received iodinated contrast. She was prepared for thyroidectomy with aggressive heart failure treatment, dexamethasone, SSKI, and cholestyramine. Thyroid function normalized over six days and she underwent total thyroidectomy eight days after admission without complications. Postoperative cardiac ECHO showed marked improvement of cardiac parameters. **Conclusion:** Right heart failure is an uncommon and often overlooked complication that can arise in poorly managed or treatment-resistant Graves' disease. It is life-threatening and requires aggressive normalization of thyroid function, treatment of heart failure, and timely definitive therapy with I-131 or thyroidectomy. Frequently complicated by liver abnormalities, the use of thionamides is questionable. Additionally, hospitalized patients have often been evaluated with iodinated contrast studies or treated with SSKI during the acute phase, which limit the use of I-131. Our cases show that dexamethasone, SSKI, and cholestyramine can rapidly normalize thyroid function. The first case ended in death, probably due to delay in definitive treatment, whereas the second patient had an early thyroidectomy with good outcome. We recommend aggressive treatment to normalize thyroid function and reverse cardiac dysfunction followed by early definitive therapy.

Pediatric Endocrinology

PEDIATRIC GROWTH AND ADRENAL DISORDERS

Unraveling the Connection Between Cortisol and Pediatric Idiopathic Intracranial Hypertension

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SAT-104

Background: Idiopathic intracranial hypertension (IIH) is a condition of elevated intracranial pressure without identifiable secondary causes. The childhood incidence is 0.7 per 100,000 and increases with age, obesity, and female gender. Few case reports in the literature, and our own experience, suggest there may be an association between IIH and adrenal insufficiency (AI) but the real extent is unknown. **Aim:** To describe the prevalence of AI in children presenting with IIH to a large pediatric referral center. **Methods:** Retrospective chart review identified all children who presented with IHH and had cortisol measured between January 2010 and September 2019. Based on morning, random or 1 mcg ACTH stimulated cortisol levels, adrenal functioning was classified as: (1) deficient (peak cortisol <16 μg/dl, AM cortisol <5 μg/dl), (2) at risk (peak cortisol 16 - 20 μg/dl, AM cortisol 5 - 13 μg/dl or random < 13 μg/dl), or (3) sufficient (peak cortisol >20 μg/dl, AM or random cortisol >13 μg/dl). Descriptive data present mean (+/- standard deviation), and chi-squared (χ^2) tests of differences are used to examine differences between the adrenal functioning groups. **Results:** Participants (N=64) were 40.6% male, of mixed ancestry (61% non-Hispanic white; 19% African-American, 16% Hispanic White and 5% Asian), with a mean age of 10.8 (4.8) years. Cortisol levels were obtained at an average of 0.6 (1.9) years after diagnosis of IIH; 23% and 52% of patients had insufficient or at risk cortisol levels. The majority of those in the insufficient (70%) or at risk (80%) groups were exposed to

topical, nasal or inhaled corticosteroids, but not systemic. Only 60% and 12 % of those with IIH and insufficient or at risk cortisol testing, respectively, underwent definitive testing with a stimulation test. Adrenal function did not differ by age, race/ethnicity, zBMI, nor prolong exposure to steroids (> 2 weeks), time between IIH diagnosis and cortisol testing (all P>.05). Those in the deficient group were less likely to be female (33%) than those in the at risk (61%; χ^2=3.07, df=1, P=.001) or sufficient (81%; χ^2=7.30, df=1, P<.001) groups. Those with AI were more likely to have history of asthma (53%; vs: 18% at risk and 12% normal; both P>.05) **Conclusions:** Steroid use and AI are common in IIH and need consideration as a cause of IIH development. Appropriate diagnosis and treatment of AI in children who present with IIH may lead to its resolution, significantly impacting clinical outcomes of these children. In our cohort, the majority had AI or at risk cortisol levels, and many did not undergo further testing. All young children who present with IIH should be evaluated for steroid exposure, including non-systemic steroids, and undergo evaluation for AI. Caution should be utilized in pediatric providers prescribing these medications. More prospective studies are required to evaluate the effects of steroid use in relation to IIH development.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS II

Severe Hypocalcemia and Psuedotumor Cerebri: Old, Still Not so Well Known Relationship

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MON-342

Introduction:
Severe headaches with nonspecific characteristics raise concern for idiopathic intracranial hypertension (IIH) in a young adult female patient. Clinical features of severe headache, vision loss, and papilledema; normal neuroimaging and elevated opening pressure on lumbar puncture are diagnostic of IIH or pseudotumor cerebri. Etiology is often attributed to obesity in young female patients. We present a rare case of Pseudotumor cerebri in a non-obese female patient with severe hypocalcemia.
Case:
A 26 year old Hispanic female patient with past medical history of hypocalcemia, but off her medications, presented to ER for worsening headache for over a month. Without any specific triggers, she began having headaches in her bitemporal region. The pain was continuous, pressure-type, and 9/10 intensity at its worst. It was associated with nausea and blurring of vision and was refractory to ibuprofen. Patient endorsed severe muscle cramps on review of systems. Physical examination demonstrated mild distress due to the headache, positive Trousseau's sign, and bilateral papilledema. Laboratory studies revealed serum total calcium level of 4.8 mg/dL, albumin 3.5 g/dL, ionized calcium 0.71 mmol/L, serum phosphate 5.3 mg/dL, intact PTH 300.1 pg/mL, 25-OH Vitamin D 14.3 ng/mL and 1, 25-Dihydoxy Vitamin D 14.1 pg/mL, alkaline phosphatase 131 IU/L, 24-hour urinary calcium 48mg/d and 24 hour urinary calcium/creatinine ratio 50 mg/g. Her MRI brain and MR venogram were unremarkable. Lumbar puncture had elevated opening pressure of 46 cm of H2O. The patient was started on Acetazolamide, oral calcium, and calcitriol without resolution of headaches and hypocalcemia. Acetazolamide was discontinued when she developed severe hypokalemia. With diagnosis of pseudohypoparathyroidism, the dose of elemental calcium was doubled and calcitriol dose increased to 1 mcg BID with improvement of serum calcium, symptoms, and papilledema.
Discussion:
While IIH could be associated with certain medications and systemic conditions, obesity or recent weight gain are the most commonly cited causes of IIH. Severe hypocalcemia is a rare and less known etiology of IIH. Cases of adult patients are reported with a variety of etiologies of severe hypocalcemia. Severe hypocalcemia leading to hypersecretion of cerebrospinal fluid is postulated as a possible mechanism but evidence is lacking in literature. Chronic, rather than acute, severe hypocalcemia is likely to precipitate IIH. Visual symptoms, headache and papilledema are difficult to resolve with conventional medical therapy without correction of underlying severe hypocalcemia. We conclude that before considering surgical interventions for cases which are refractory to medical therapy, rare underlying conditions like severe hypocalcemia must be investigated and treated optimally.

Thyroid

THYROID DISORDERS CASE REPORTS I

Patient with Pseudohypoparathyroidism Type 1B, Graves Disease, and False Positive HIV Screen- a Rare Presentation

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SUN-524

Background: Pseudohypoparathyroidism 1B (PHP1B) is a disorder that can lead to thyroid stimulating hormone (TSH) resistance and hypothyroidism, although it is rarely associated with thyrotoxicosis.
Clinical Case: A 25-year-old female with a history of PHP1B, seizures due to hypocalcemia, and family history of PHP1B in her three sisters and brother presented to our emergency room with a fever of 103^0F and generalized malaise. Two months prior, she was seen at an outside hospital with palpitations and bulging of the left eye. There, she was diagnosed with hyperthyroidism, started on methimazole, and asked to continue levetiracetam and calcitriol upon discharge.
On our exam, she had tachycardia of 120 beats per minute, left eye proptosis, positive Chvostek sign, and a large goiter with bruit. Reflexes were 3+.
Laboratory evaluation revealed corrected serum calcium of 6.1 (8.5-10.5 mg/dL), TSH < 0.01 (0.34-5.60 mU/L), free T4 2.81 (0.60-1.60 ng/dL), free T3 13.0 (2.4-4.2 ng/dL), and iPTH 131 (12-88 pg/mL). ELISA testing for screening of HIV was positive. She was treated with IV calcium gluconate, methimazole, propranolol, and hydrocortisone.