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 lifethreatening 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 Alfonso Hoyos-Martinez, MD¹, Vincent Horne, MD¹,

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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