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%; χ²=3.07, df=1, P=.001) or sufficient (81%; χ²=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 Pseudotumor Cerebri: Old, Still Not so Well Known Relationship

Madhuri Patil, MD, Valerie Butler, DO, Benjamin Cameron, DO, Nadia Jamil, MD, Tiffany Egbe, MD.

University of Texas Health Science Center at Tyler / CHRISTUS Good Shepherd Health System, Longview, TX, USA.

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 Trouseau’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-Dihydroxy Vitamin D 14.1 pg/mL, alkaline phosphatase 131 IU/L, 24-hour urinary calcium 48 mg/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

Viraj V. Desai, DO, MBA, Pratima V. Kumar, MD.

Dell Medical School at The University of Texas at Austin, Austin, TX, USA.

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°F 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 bruft. 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.
Her home doses of calcitriol and calcium were resumed. She was referred for total thyroidectomy as an outpatient once she became euthyroid. The confirmatory Western blot test for HIV was negative. It was determined that presence of thyroid stimulating immunoglobulin resulted in the false positive ELISA test.

Discussion: Hyperthyroidism with Graves disease seen in PHP has only very rarely been reported. (1) It has been postulated that abnormal electrolytes and elevated parathyroid hormone from PHP may lead to stimulation of the thyroid gland and perpetuate Graves disease symptoms. (2) Furthermore, the presentation of thyrotoxicosis despite TSH resistance in PHP indicates that there may be other mechanisms for TSH receptor antibodies to take effect in these patients which have not yet been determined. (3) Lastly, autoimmune diseases, including Graves disease, can cause a false-positive HIV ELISA as seen in our patient.

Conclusion: Although rare, thyrotoxicosis may present in patients with PHP1B. Additionally, it should be kept in mind that autoimmune diseases such as Graves disease can cause a false positive HIV ELISA, and follow-up Western blot testing should therefore be performed.

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Adipose Tissue, Appetite, and Obesity
RARE CAUSES AND CONDITIONS OF OBESITY: PRADER WILLI SYNDROME, LIPODYSTROPHY
U.S. Prevalence & Mortality of Prader-Willi Syndrome: A Population-Based Study of Medical Claims

SUN-604
Prader-Willi syndrome (PWS) is a complex developmental genetic disorder associated with hypotonia, poor feeding in neonates, onset of hyperphagia in early childhood, and shorter overall life expectancy. Prior epidemiology studies of PWS have examined smaller populations, with limited research in a US population. The aim of this study was to provide a contemporary estimate of PWS prevalence and annual all-cause mortality in the US using a large administrative medical claims dataset.

Methods: PWS patients were identified between 2012-2014 via the presence of ≥2 claims with a diagnosis code for