After surgery, he received flare-dosing PVO at 20 mg/day for 4 weeks, then 10 mg/day for 8 weeks. Post-surgical imaging 12 weeks after the surgery showed new bridging HO at the site of intramedullary rod insertion and around the distal screw.

Nine months after the fracture the patient had a second fall resulting in a right intertrochanteric fracture. He underwent intramedullary nailing of the right hip, in a modified procedure which did not require distal screw placement. PVO was increased similarly to the above flare protocol, but, at the time of fracture occurrence rather than post-surgery. He had no skin or healing complications with either treatment regimen.

After each fracture the patient had prolonged recurrent flare-ups at the injury sites, significantly increasing his number of flare-ups per year. After the fractures there was new Brooker class D HO at the left hip, originating at the insertion of the intramedullary rod, and new class B HO at the right greater trochanter, again near the insertion site of the intramedullary rod compared to his pre-surgery baseline. In contrast, there was no new HO at the right distal intramedullary rod whereas HO occurred around the screw placement site at the left distal rod.

Conclusion: This case suggests that PVO in the dosing regimen received by this patient can be tolerated in an individual with FOP following major surgery. HO still occurred in this patient, particularly along the rod insertion track, suggesting that the PVO regimen may need to be optimized for surgical cases or that poly-trauma events may not be adequately blocked by the dosing regimen received by this patient. However, PVO did not negatively impact fracture healing or osteointegration, and no major skin healing effects were identified. Further investigation is needed to assess whether PVO can lead to a dose-dependent reduction in HO in the setting of trauma and surgery.

Thyroid

THYROID DISORDERS CASE REPORTS I

Dissociation of Clinical Course of Coexisting Autoimmune Hepatitis and Graves Disease

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SUN-508

Title: Dissociation of Clinical Course of Coexisting Autoimmune Hepatitis and Graves Disease

Introduction: The association between Graves’ disease and coexisting autoimmune hepatitis is well known. Treatment of autoimmune hepatitis with glucocorticoids can concurrently lower thyroid hormone levels. Additionally, recurrence of hyperthyroidism has been shown to be associated with recurrence of autoimmune hepatitis. We present a case of a patient with autoimmune hepatitis and Graves’ thyrotoxicosis, which initially improved with prednisone therapy, but thyrotoxicosis recurred during the prednisone taper while hepatitis stayed in remission.

Clinical Case: A 47-year old female initially presented with fatigue, nausea, and jaundice. Liver enzymes showed elevated AST (1634 U/L) and ALT (1956 U/L) with total bilirubin 15.1 mg/dL. Liver biopsy was consistent with autoimmune hepatitis. Treatment was initiated with ursodiol 300 mg TID and liver enzymes improved to ALT 579 IU/L and AST 544 IU/L with total bilirubin 1.9 mg/dL. Nine months later, she presented with worsening upper and lower extremity weakness, slurred speech, abdominal pain, and nausea and vomiting. AST and ALT were again elevated to >1,000 U/L. TFTs were checked due to symptoms of palpitations and heat intolerance. TSH was 0.024 uIU/mL with elevated free T4 of 3.74 ng/dL and TSI of 435%. She was treated with prednisone, cholestyramine, and propranolol and discharged with a one-month prednisone taper starting at 60 mg daily. LFTs and TFTs normalized after one month. Cholestyramine was discontinued and prednisone was tapered to 5 mg daily. Seven months later, she had symptoms of palpitations and heat intolerance. TFTs were consistent with hyperthyroidism (TSH 0.049 uIU/mL, free T4 3.96 ng/dL). LFTs remained in normal range. Since thionamides have relative contraindications in patients with liver disease, prednisone was increased from 5 mg to 20 mg daily and cholestyramine was resumed to treat hyperthyroidism. After five months, TSH remained suppressed to 0.019 uIU/mL however free T4 improved to 1.74 ng/dL. The patient was referred for total thyroidectomy.

Conclusion: This case illustrates an example of the rarely reported occurrence of thyrotoxicosis recrudescence despite initial improvement with treatment of underlying autoimmune hepatitis. Recognition of recurrence of hyperthyroidism independent of recurrence of autoimmune hepatitis indicates the need for early definitive therapy for hyperthyroidism.
study. Chronic heart failure and chronic renal failure were determined according to reported diagnosis and drug treatment. Chronic renal failure patients were included if the glomerular filtration rate (GFR) in the first hospitalization was below 30 ml/min/1.72 square meter. Results: Overall, 90,199 TSH tests were sent from the non-surgical wards, most of them as part of the admissions profile. Of these, 2,116 hospitalizations met the inclusion criteria of the first hospitalization. In the final analysis, 126 inpatients with at least one re-hospitalization were included, of whom 43 (34.1%) had chronic heart failure and 22 (17.5%) had chronic renal failure. According to the most recent re-hospitalization, thyroid function was worse in 11 (8.7%), 4 (9.3%) and 2 (9.1%) patients of the total, heart failure and renal failure groups respectively. The TSH level was found to be normal in re-hospitalization in 81.4% of those with heart failure and 86.4% of those with renal-failure. No association between heart failure or renal-failure and thyroid function worsening was found (p = 1.00 for both). Of 34 patients with chronic heart failure re-hospitalized after 1/2-1 year, in 29 (85.3%) the repeated TSH was normal, in 3 (8.8%) it was unchanged and in 2 (5.9%) it was worse. In most re-hospitalization the worsening was due to initiation of Levothyroxin treatment and because of the retrospective nature of the study we cannot be sure whether the initiation was justified; therefore, it is likely that the worsening percentage is even lower. Conclusions: An isolated TSH elevation in hospitalized patients with past medical history of chronic heart-failure or chronic renal failure does not indicate thyroid disease, in most cases.

Adrenal
ADRENAL - CORTISOL EXCESS AND DEFICIENCIES
Adrenal Androgen Control and Steroidal Side Effects in Adolescents and Adults with Congenital Adrenal Hypoplasia Treated with Glucocorticoids
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MON-183
Introduction: Classic congenital adrenal hyperplasia (CAH) due to 21-hydroxylase deficiency is a rare autosomal recessive disorder that results in little or no cortisol biosynthesis, increased production of precursor steroids, and excess production of adrenal androgens. Glucocorticoid (GC) treatment, the current standard of care for CAH, is used to correct cortisol deficiency and reduce excessive androgens. Elevated 17-hydroxyprogesterone (17OHP) is used for diagnosis and management. GC titration to achieve 17OHP <1000 ng/dL may be targeted for adrenal androgen control; however, patients with 17OHP <1000 ng/dL might be at risk for complications of long-term GC excess. This real-world study evaluated adrenal androgen levels and potential GC complications in adolescents and adults with CAH.

Methods: TriNetX, a research network that includes electronic medical records from >37 million U.S. patients, was searched on 30/Aug2019 for patients who met the following criteria: diagnosis code of E25.0 (ICD-10) or 255.2 (ICD-9); history of GC use; available 17OHP laboratory result; and ≥15 years of age (“grown”) at the most recent 17OHP assessment. Patients were categorized as “adequately controlled” (17OHP <1000 ng/dL) or “poorly controlled” (17OHP ≥1000 ng/dL). Assessments included: demographics; laboratory results for 17OHP, adrenocorticotropic hormone (ACTH), and androstenedione (A4); and low-density lipoprotein (LDL). Adequately vs. poorly controlled groups were compared using Chi-square tests and t-tests.

Results: Of 511 grown CAH patients, 352 were adequately controlled and 159 were poorly controlled. Mean concentrations for 17OHP were 244 and 5393 ng/dL in the adequately and poorly controlled cohorts, respectively (p<0.01). Adequately controlled patients also had lower ACTH and A4 than poorly controlled patients: ACTH (72 vs 389 pg/mL, p<0.01); A4 (82 vs 256 ng/dL, p<0.01). Compared to poorly controlled patients, adequately controlled patients were more likely to be female (81% vs 57%, p<0.01) and older (mean birth year: 1981 vs 1986, p<0.01). Adequately controlled patients also had evidence of more metabolic and infection complications, including higher mean LDL (105 vs 94.3 mg/dL, p=0.02), more type 2 diabetes mellitus (9% vs 4%, p=0.08), and more respiratory tract infections (21% vs 11%, p=0.01).

Conclusions: In this retrospective analysis, patients with adequately controlled CAH (17OHP <1000 ng/dL) had better adrenal androgen control (lower A4) but also higher rates of complications potentially related to excessive GC exposure. These findings highlight the current challenges of managing CAH with GC regimens alone.

Neuroendocrinology and Pituitary
NEUROENDOCRINE & PITUITARY PATHOLOGIES
Acromegaly Significantly Impacts Employees' Health Benefit Costs and Increases Work Absenteeism
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SUN-296
Background: Acromegaly (ACRO) is a rare, chronic disorder of growth hormone hypersecretion associated with increased morbidity that can affect work productivity. Data on ACRO employees’ health costs and work absenteeism are limited.

Aims: To assess the impact of ACRO on employees’ health benefit costs and absenteeism.

Methods: A US employee database of prescription (Rx) drug, medical claims, and absenteeism (payment and time) from Jan 2010 to Apr 2019 was analyzed. Employees with the diagnosis (Dx) of ACRO were identified based on claims with ICD-9/-10 codes 253.0x/E22.0. A 12 month study period followed each employee’s first ACRO Dx in the database.