Clinical Case
A 63 year old male with past medical history significant for uncontrolled type 2 diabetes (10 year duration, HgA1c=11.2%, on insulins detemir and aspart, metformin, and empagliflozin), coronary artery disease, and treatment refractory antibody-negative polymyositis (baseline CPK levels ~1000-2000, on a burst of prednisone for flare) presented with fever (101.2F), fatigue, myalgias, and nausea with poor oral intake and insulin cessation after recent IV zoledronic acid infusion for prevention of steroid-induced osteoporosis. He was found to be acidemic with bicarbonate=16, AG=18, Cr=1.6 (baseline 1.1), lactic acid=2.9, glucose=245, glucosuria/ketonuria, serum osmolality=295, and CPK=3613. No infectious etiology was found. Differential diagnosis of precipitating factors of DKA includes: steroid-induced hyperglycemia with lipolysis and insulin resistance; starvation ketosis from poor oral intake due to bisphosphonate-induced flu-like illness; metformin-associated lactic acidosis in setting of acute kidney injury; ketone production secondary to insulin cessation in setting of febrile illness; and SGLT2-inhibitor use with dehydration secondary to decompensated hyperglycemia. He was treated for DKA with insulin and volume resuscitation. He was discharged with discontinuation of empagliflozin.
Conclusion
In people with type 2 diabetes and multiple medical problems, a collusion of clinical factors leading to acidemia can occur simultaneously and lead to a drastically increased risk of DKA, especially in the setting of SGLT2-inhibitor use. Clinicians should have heightened awareness of minor predisposing factors that in combination can increase risk of DKA in a patient with type 2 diabetes.

Thyroid

THYROID DISORDERS CASE REPORTS I

Graves'Disease and Autoimmune Hepatitis: Management Challenges.

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SUN-502
Graves' disease (GD) is the most common etiology of hyperthyroidism and may be associated with other autoimmune disorders. Case report: A.J.M.N., 27 years old, previously healthy, presented with abdominal discomfort, nausea and headache. She used paracetamol 750mg t.i.d for seven days. After that, she noticed jaundice and sought medical care. On admission, patient was icteric, oriented, afebrile, without signs of heart failure or alterations in the intestinal habit. Admission laboratory tests: AST 1287 U/L (RR<46), ALT 1090 U/L (RR < 50), total bilirubin (TB) 45.66mg/dL (RR<1.3), direct bilirubin 42.22mg/dL (RR<0.8), TSH 0.04 mcUI/ml, FT4 > 6.99 ng/dL (RR< 2.19). Serology for infectious diseases (A, B and C viral hepatitis; cytomegalovirus; Epstein-Bar Virus, syphilis; Dengue virus) were negative. Available antibodies for autoimmune hepatitis (anti-LKM1, anti-mitochondria, anti-smooth muscle, anti-SSB, anti-SSA, anti-Rnp / Sm, anti-DNA) were non-reactive. Ceruloplasmin and serum copper were normal. TRAB 3 IU/L (RR<1.75 IU /L); Thyroid scintigraphy showed homogeneous distribution of parenchymal contrast and regular contours of the gland; 15-minute uptake was 9.19% (RR: 1%-6%). Propranolol (40mg q.i.d) was prescribed. Burch and Wartofsky score was 30 (possible previous infection episode as precipitation factor = 10 points and unexplained jaundice = 20 points). Since the patient did not have diagnostic criteria for thyroid storm and since liver function was greatly altered, we opted to treat the thyroid disease with 12mCi of radioiodine, instead of antithyroid drugs (ATD). Differential diagnosis of the liver disease, whether due to autoimmunity or due to hyperthyroidism itself or both, were considered. Corticosteroid therapy (prednisone 40mg) was added due to the possibility of the coexistence of GD and autoimmune hepatitis previously reported as been 1.8% of the autoimmune hepatitis cases. Liver biopsy was performed 4 days later, and the findings were compatible with this condition. Ten days after prednisone and 20 days after radioiodine, we noticed a drop in TB (45 to 20mg/dL) and liver enzymes (AST= 69 and ALT 106) and she was discharged with normal FT4. Autoimmune hepatitis and GD presents a management challenge because sometimes it is not possible to confirm the etiology before treatment. The abnormalities could have been due to hyperthyroidism itself, since all autoantibodies to autoimmune hepatitis have been ruled out, but liver biopsy was very suggestive of the autoimmune cause. Initiating ATD for rapid improvement of hyperthyroidism could represent a risk due to hepatotoxicity of these drugs. On the other hand, withholding the treatment in cases of hepatic insufficiency due to hyperthyroidism, can have disastrous consequences. The option with beta-blocker, radioiodine and corticosteroid was successful and might be considered in similar cases.

Thyroid

BENIGN THYROID DISEASE AND HEALTH DISPARITIES IN THYROID I

Insulin Resistance, Lipid Profile and High-Sensitivity C-Reactive Protein in Patients with Autoimmune Thyroiditis

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SAT-431
Introduction: Thyroid function and autoimmunity has been associated with cardiovascular events in patients with

autoimmune thyroiditis. **Objectives**: To evaluate the association between thyroid function, antithyroid antibodies levels, insulin resistance and markers of cardiovascular risk in patients with autoimmune thyroiditis. **Methods**: We evaluated 228 patients with autoimmune thyroiditis, 93.9 % female, with a mean age of 47.06 ± 15.35 years. We analyzed thyroid function, anti-thyroglobulin antibodies (anti-Tg), anti-thyroid peroxidase antibodies (anti-TPO), HOMA-IR, HOMA-B, QUICKI, HISI (Hepatic Insulin Sensitivity Index), WBISI (Whole-Body Insulin Sensitivity Index), the levels of lipid profile, high-sensitivity C-reactive protein (hs-CRP), homocysteine, folic acid, and vitamin B12. We defined 3 groups based on TSH levels: TSH between 0.35-2.49 μUI/ml, (n = 166), TSH between 2.50-4.94 μUI/ml, (n = 43) and TSH over 4.95 μUI/ml, (n = 19), and normal levels of free T4 and free T3. A 75-g OGTT was performed in the morning and blood samples were obtained every 30 min for 120 min for measurements of plasma glucose, insulin, and C-peptide. For the statistical analysis we used the Mann-Whitney test and Spearman correlations. Results are expressed as means ± SD or percentages. A two-tailed $p<0.05$ was considered statistically significant. **Results**: There were no significant differences regarding median age or median BMI between groups. We did not find any significant differences comparing group with TSH 0.35-2.49 and group with TSH 2.50-4.94, in all parameters evaluated. Group with TSH 2.50-4.94 had higher indexes of QUICKI (0.69 ± 0.39 vs 0.48 ± 0.13; $p = 0.02$) and HISI (79.83 ± 63.72 vs 41.73 ± 29.02; $p = 0.01$) than group with TSH over 4.95. The group with TSH over 4.95 demonstrated a higher index of HOMA-IR than group with TSH 2.50-4.94 (3.77 ± 2.93 vs 1.95 ± 1.24; $p = 0.01$). In the TSH 0.35-2.49 group we found significant correlations between TSH and HOMA-IR ($r= 0.18$; $p = 0.01$), total cholesterol and anti-TPO ($r =0.23$; $p = 0.002$), anti-Tg and HDL-cholesterol ($r= -0.17$; $p=0.002$), anti-Tg and triglycerides ($r=0.34$; $p < 0.001$), and anti-Tg and LDL-cholesterol ($r=0.16$; $p=0.03$). In the TSH 2.50-4.94 group we observed positive correlation between Apo A1 and HOMA-B ($r=0.58$; $p<0.001$), HOMA-IR and LDL-cholesterol ($r=0.34$; $p=0.02$) and WBISI and HDL-cholesterol ($r=0.34$; $p=0.02$). In the TSH over 4.95 group we observed a correlation between TSH and triglycerides ($r=0.70$; $p<0.001$) and between anti-Tg and hs-CRP ($r=0.64$; $p=0.004$). **Conclusions**: The association among TSH, lipid profile, insulin resistance, hs-CRP and antithyroid antibodies in patients with autoimmune thyroiditis may contribute to an increased cardiovascular risk, not only in patients with subclinical hypothyroidism but also in those classified as euthyroid.

Pediatric Endocrinology

PEDIATRIC ENDOCRINE CASE REPORTS II

Unusual Association Between Congenital Hyperinsulinism and Neurofibromatosis Type 1

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MON-080

Background: Neurofibromatosis type 1 (NF1) is an autosomal dominant multisystemic disorder characterized by an increased risk of benign and malignant tumor formation affecting skin, bone and nervous system. In children with NF1, endocrine manifestations include central precocious puberty, growth hormone deficiency and growth hormone hypersecretion, resulting from complications of optic pathway gliomas involving the hypothalamic and sellar region. A few reports of adults with NF1 have been described to have hypoglycemia due to insulinoma. However, hypoglycemia due to hyperinsulinism has not been described in children with NF1. We present a case of NF1 diagnosed during neonatal period associated with congenital hyperinsulinism.

Case: Patient was delivered at 36 weeks by C- section with birthweight of 2780 grams which was appropriate for her gestational age. There was no maternal history of diabetes. Pertinent exam findings included microcephaly and multiple café-au-lait spots. She developed hypoglycemia at DOL1 with blood glucose of 26 mg/dl which normalized with IV dextrose at a glucose infusion rate of 6 mg/kg/min. At DOL8, an attempt to wean IV dextrose failed and she developed hypoglycemia with blood glucose of 47 mg/dl. Critical sample showed insulin level 3.3 uU/ml, betahydroxybutyrate 0.08 mg/dl (0.2-2.8), cortisol 18.1 mcg/dL and GH 13.2 ng/mL. A glucagon stimulation test showed an increase in glucose of 30 mg/dl. She was diagnosed with hyperinsulinism and started on Diazoxide (8 mg/kg/day) with improvement of blood glucose with prefeed glucose of > 70 mg/dl. She had normal 8- hour fasting tolerance with all BG > 70 while on Diazoxide. Genetic test for known mutations causing hyperinsulinism was negative. Microarray confirmed a 1.42Mb interstitial deletion at chromosome 17q11.2 which encompasses NF1 gene confirming the diagnosis of NF1. Additionally, she has an Xp22.33 duplication of uncertain clinical significance.

Conclusion: Our patient presented with an unusual association between congenital hyperinsulinism and NF1. Further testing needs to be performed to determine whether this association is coincidental or whether congenital hyperinsulinism is a rare manifestation of NF1.

Thyroid

THYROID NEOPLASIA AND CANCER

Cell Adhesion Molecules mRNA Expression in Thyroid Tumors

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