Adult E-Poster

EP_A101

THYROID-ASSOCIATED ORBITOPATHY IN HASHIMOTO'S THYROIDITIS: A RARE AUTOIMMUNE OVERLAP

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INTRODUCTION/BACKGROUND

Thyroid-Associated Orbitopathy (TAO), or Graves' Orbitopathy (GO), is an immune-mediated inflammatory disorder of the orbit most commonly associated with hyperthyroidism in Graves' disease. It is primarily driven by TSH receptor antibodies (TRAb), which stimulate orbital fibroblasts and induce tissue remodelling. In contrast, Hashimoto's thyroiditis is characterized by gland-destructive autoimmunity, with elevated anti-thyroid peroxidase (TPO) antibodies and progressive hypothyroidism. The occurrence of GO in patients with overt hypothyroidism due to Hashimoto's thyroiditis is rare and represents a unique overlap of autoimmune thyroid diseases.

CASE

We present the case of a 56-year-old male with no prior history of thyroid disease who presented with progressive, bilateral eye discomfort, photophobia, eyelid swelling, and intermittent diplopia over the preceding eight months. He also reported experiencing fatigue, cold intolerance, dry skin, myalgia, weight gain, and constipation. The patient's medical history included hypertension, and he was an active smoker; both are known risk factors for orbitopathy.

Physical examination revealed eyelid lag, dry skin, and bilateral exophthalmos. His Clinical Activity Score (CAS) indicated active Graves' orbitopathy (GO). No goitre or tremor was noted. Thyroid function tests confirmed overt hypothyroidism (TSH 19.515 μIU/mL; FT4 0.59 ng/dL), with significantly elevated anti-thyroid peroxidase (anti-TPO) antibodies (9,307.99 IU/mL) and borderline-positive TSH receptor antibodies (TRAb) (1.85 IU/L). Thyroid ultrasound demonstrated reduced thyroid volume and heterogeneous echotexture, consistent with Hashimoto's thyroiditis. Orbital computed tomography (CT) showed bilateral rectus muscle thickening, further supporting the diagnosis of Graves' orbitopathy. Clinical improvement was observed following treatment with levothyroxine and selenium.

CONCLUSION

This case underscores the concept of autoimmune thyroid disease existing on a spectrum, wherein features of both Hashimoto's thyroiditis and Graves' disease can coexist. Early recognition of this overlap is crucial for accurate diagnosis, appropriate treatment guidance, and prevention of long-term ocular complications.

EP_A102

CLOTS AND CRACKS: OSTEOPOROSIS AS A CONSEQUENCE OF PROTEIN C DEFICIENCY AND WARFARIN USE

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INTRODUCTION/BACKGROUND

Activated protein C is essential in anticoagulation. Protein C deficiency results in inappropriate blood clot formation due to dysregulated coagulation. We report a case of osteoporosis secondary to protein C deficiency and warfarin use.

CASE

A 30-year-old male initially presented with a superior sagittal sinus thrombosis, complicated with a left frontal lobe venous infarct at the age of 21. He reported a family history significant for venous thromboembolism. A thrombophilia screen done during presentation revealed a moderately low protein C activity at 44.6% (reference interval 70 – 140) with normal protein S levels. Autoimmune workup including anticardiolipin, and lupus anticoagulants were negative. Long term warfarin was initiated for the treatment of the cerebral venous thrombosis (CVT).

Three years after CVT and warfarin use, old compression fractures involving the T5 and T7 vertebrae were found on routine X-rays done during an admission for rhabdomyolysis. A bone mineral densitometry (BMD) showed a Z score of -2.8 at the femoral neck, and -1.5 at the L1-L4 vertebrae. Screening for causes of secondary osteoporosis, specifically hyperparathyroidism, hyperthyroidism, hypogonadism, acromegaly, chronic kidney disease, and Cushing syndrome were negative. The patient's inability to attain peak bone mass would likely be due to severe illness (CVT) suffered at a young age.

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Upon diagnosis of osteoporosis, warfarin was replaced with rivaroxaban for anticoagulation and vitamin D replacement and calcium supplements were started, while no anti-osteoporosis medications were initiated. Annual BMD was done, and the latest imaging showed an improvement of 2.4% in the femoral neck compared to the previous year. Apart from the previously noted vertebral compression fractures, no new fractures were appreciated during follow-up. BMD monitoring will continue every 2 years.

CONCLUSION

Osteoporosis in the young should be thoroughly investigated and managing the underlying condition is key to proper treatment.

EP_A103

LIPOPROTEIN X-MEDIATED PSEUDOHYPONATREMIA IN A PATIENT WITH TYPE 2 DIABETES

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INTRODUCTION/BACKGROUND

Pseudohyponatremia is a lab abnormality commonly caused by hypertriglyceridemia, hyperglycemia or hypergammaglobulinemia. Lipoprotein X (LpX) is an abnormal lipoprotein that most commonly appears in the plasma of patients with cholestasis. LpX mediated pseudohyponatremia is rare but has been described in the literature. We report a patient with type 2 diabetes mellitus (T2DM) and LpX-mediated pseudohyponatremia due to severe cholestatic hepatitis.

CASE

A 31-year-old female was admitted with newly diagnosed T2DM and severe DKA secondary to bilateral calf abscesses. She was treated with insulin and intravenous cefazolin as intraoperative tissue culture grew MSSA. Three days after starting cefazolin she developed progressively worsening severe cholestasis [peak total bilirubin (TB) 245 umol/L(reference interval (RI) <17), conjugated bilirubin 175 umol/L (RI <6), peak ALP 1027 U/L (RI 45-129), with normal to marginally elevated transaminases] with negative viral and autoimmune serologies including AMA. Malignancy, biliary stones, and extra-hepatic cholestasis were excluded by imaging including CECT liver. Liver biopsy showed non-caseating granulomatous hepatitis, consistent with drug-induced liver injury secondary to cefazolin.

Concurrently, she developed hyponatremia despite adequate glycemic control on insulin therapy, that was established to be secondary to severe hypercholesterolemia [nadir serum sodium (sNa) 125 mmol/L (RI 136-145), serum osmolality 308 mmol/kg (RI 275-295), total cholesterol (TC) 30.6 mmol/L (RI <5.2), triglyceride 5.3 mmol/L]. Serum protein electrophoresis showed a supernumerary peak between albumin and alpha-1 region, suggestive of the presence of LpX. Cefazolin was discontinued and she was given a course of ursodeoxycholic acid (UDCA) for three months. Subsequently, TB and ALP dramatically improved, TC gradually declined and serum sodium became normal. During her most recent follow-up, her liver panel and serum sodium remained normal. TC, triglyceride, and LDL, while markedly improved, remained slightly elevated, compatible with her diagnosis of metabolic syndrome.

CONCLUSION

Recognition of the relationship of cholestasis, elevated LpX and pseudohyponatremia is important to avoid mismanagement of hyponatremia. Electrophresis confirms the diagnosis of LpX and diagnosed patients should subsequently be monitored for hyperviscosity secondary to hypercholesterolemia.

EP_A104

A CASE OF LATE-ONSET HYPOPARATHYROIDISM FOLLOWING RECURRENT ANTERIOR NECK SURGERY RESULTING IN RHABDOMYOLYSIS

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INTRODUCTION/BACKGROUND

Hypoparathyroidism is a known complication of anterior neck surgery, with 1.5% becoming permanent. Delayed-onset hypoparathyroidism can manifest years postoperatively due to progressive scar tissue formation. It is often overlooked, causing complications. We present such a patient complicated by rhabdomyolysis and renal failure.