Adult E-Poster

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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.

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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.