CONCLUSION

This case highlights an atypical subacute presentation of hypothyroid myopathy following RAI, with evolving weakness and transient worsening after starting thyroid hormone therapy. Although other serious causes should be excluded, clinicians must maintain a high index of suspicion to avoid unnecessary investigations and ensure timely optimization of thyroid hormone therapy, as clinical improvement parallels biochemical recovery.

EP_A155

HASHIMOTO'S THYROIDITIS PRESENTING WITH RECURRENT MASSIVE PLEURAL EFFUSION SUGGESTIVE OF LYMPHOCYTIC INTERSTITIAL PNEUMONITIS

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INTRODUCTION

Hashimoto's thyroiditis is a common autoimmune thyroid disease; however, pulmonary involvement is a rare manifestation. Lymphocytic interstitial pneumonitis is commonly associated with autoimmune diseases, but its association with Hashimoto's thyroiditis remains poorly understood.

CASE

A 63-year-old male with prior diagnosis of Hashimoto's thyroiditis presented with recurrent massive pleural effusion and progressive dyspnea. He also experienced fatigue, cold intolerance, and constipation. Physical examination revealed facial puffiness, diffuse goiter, and dry skin. Thyroid function tests performed 6 months before admission confirmed overt hypothyroidism (thyroid-stimulating hormone [TSH] 80.30 µIU/mL, free thyroxine 4 [FT4] 0.46 ng/dL). One month prior to admission, laboratory findings showed subclinical hypothyroidism (TSH 9.09 µIU/mL, FT4 1.42 ng/dL). Despite levothyroxine treatment TSH levels remained elevated at 21.8 µIU/mL during admission. Anti-thyroid peroxidase antibodies were positive (13.95 IU/mL). Repeated thoracentesis revealed exudative pleural effusion with lymphocytic predominance and no evidence of malignancy in cytological examination. Infectious etiologies and malignancy were ruled out. Thyroid ultrasonography demonstrated diffuse hypoechoic enlargement of the thyroid gland. Chest radiography showed massive unilateral pleural effusion, while thoracic computed tomography revealed ground-glass opacities with a reticulogranular pattern and interlobular septal thickening, accompanied by multiple thin-walled cysts, highly suggestive of lymphocytic interstitial pneumonitis. Thyroid immunohistochemical analysis was performed (CD 20, CD 3, CD 10, CD 138, Ki 67, BCL 2, BCL 6, Thyroglobulin, MUM 1, Cyclin D1), consistent with lymphocytic thyroiditis. Significant clinical and laboratory improvement was achieved after combined treatment with levothyroxine and corticosteroid.

CONCLUSION

This case underscores the importance of considering autoimmune-related lymphocytic interstitial pneumonitis in patients with Hashimoto's thyroiditis who present with unexplained recurrent pleural effusion after exclusion of more common etiologies, such as infection and malignancy. Early recognition may lead to favorable improvement.

EP_A156

A RARE CASE OF COMPLETE HEART BLOCK SECONDARY TO NON-AUTOIMMUNE NON-FAMILIAL FORM OF THYROTOXICOSIS

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INTRODUCTION

Thyrotoxicosis typically manifests as a hypermetabolic state characterized by tachyarrhythmias, such as sinus tachycardia or atrial fibrillation. Bradyarrhythmia, specifically atrioventricular block (AVB), is a rare and atypical cardiac manifestation. While Graves' disease is the leading cause of hyperthyroidism, non-autoimmune etiologies must be considered when thyroid-stimulating antibodies are absent. The exact mechanism for AVB in thyrotoxicosis remains unclear but may involve myocardial inflammation of the conduction system or autonomic dysfunction.

CASE

A 16-year-old male with no previous medical history presented with a sudden syncopal attack. Clinical evaluation revealed a complete heart block (CHB) in association with biochemical evidence of thyrotoxicosis, requiring a temporary transcutaneous pacemaker insertion. There were no features of Graves' disease, such as exophthalmos and thyroid acropachy, and further investigation showed a negative thyroid receptor antibody (TRAb) titer with no family history of thyroid disorders. Thyroid ultrasonography showed increased vascularity, while scintigraphy imaging showed diffuse, homogeneous, and increased uptake in both thyroid lobes. The combination of

negative serology and the absence of a family history, along with a hyperfunctional state on imaging, likely suggests a rare presentation of sporadic, non-autoimmune, non-familial form of thyrotoxicosis. Following the initiation of anti-thyroid therapy, the CHB completely resolved without the need for further cardiological intervention. He achieved a complete clinical remission after a few months and is being planned for radioactive iodine therapy.

CONCLUSION

Most of the thyrotoxicosis-associated CHB reported in the literature was due to Graves' disease or some forms of autoimmune thyrotoxicosis, with auto-antibodies and activated lymphocytes having a role in the pathogenesis of the CHB. CHB in association with a non-autoimmune, non-familial form of thyrotoxicosis is indeed rare, and as this case illustrates, it completely went into spontaneous sinus rhythm upon initiation of conventional anti-thyroid therapy. This rare presentation of CHB is believed to have a benign clinical course.

EP_A157

STEROID-RESPONSIVE ENCEPHALOPATHY ASSOCIATED WITH THYROIDITIS

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INTRODUCTION

Steroid-responsive encephalopathy associated with thyroiditis (STREAT) is a rare clinical entity. Clinical presentations of STREAT can range from a stroke-like presentation to psychiatric symptoms. STREAT is diagnosed based on four major criteria which include altered cognitive function, new or worsening psychiatric symptoms, elevated antithyroid antibodies, and exclusion of infectious, toxic, metabolic, or neoplastic causes. Corticosteroids are the cornerstone of treatment, with reported excellent response in neurocognitive symptoms resolution.

CASE

This was a case of a 48-year-old male with Graves's disease who underwent radioiodine therapy 4 months earlier and was started on levothyroxine 100 mcg 2 weeks prior to presentation. He presented with 4 days of behavioral symptoms, irritable mood with auditory and visual hallucinations. There was no history of fever, headache, limb weakness, or seizure. On general appearance, the patient was agitated, talking incoherently, and disorientated with normal vitals. There was no delayed relaxation of the reflex. The examination of the cranial nerve, motor, sensory, and cerebellar system was normal. A lumbar puncture showed normal opening pressure with a high protein CSF content of 1,596 mg/L. The thyroid-stimulating hormone (TSH) was elevated at 72.7 uIU/mL and T4 at 9.56 pmol/L. Serum anti-thyroid peroxidase was elevated at 877.42 IU/mL, and anti-TSH receptor antibody at 21.30 IU/L. CSF oligonal band, serum aquaporin-4, and viral screening were negative; non-contrasted computed tomography brain revealed normal findings. Based on the clinical history and examination, a diagnosis of STREAT was made at the emergency department. The patient was initiated on hydrocortisone 100 mg three times a day. Within 24–48 hours of steroid therapy, marked improvement and subsequent resolution of the mental status and behavioral symptoms were seen.

CONCLUSION

Hashimoto's thyroiditis can rarely lead to STREAT, a condition with diverse neurological manifestations, where early recognition and prompt corticosteroid therapy are essential for favorable outcomes.

EP_A158

MUSCLE WEAKNESS IN THYROID DISEASE: WHEN IT IS NOT THYROTOXIC MYOPATHY?

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INTRODUCTION

Muscle weakness in thyroid disease is commonly attributed to thyrotoxic myopathy or hypokalemic periodic paralysis. Nevertheless, autoimmune conditions such as idiopathic inflammatory myopathies (IIM) and myasthenia gravis (MG) should be considered, as they may coexist with Graves' disease.

CASE

A 54-year-old female with hypertension and Graves' disease, treated with carbimazole for 2 years, had her therapy discontinued after remission. She was restarted on low-dose carbimazole following symptom recurrence. Three weeks later, she developed progressive proximal weakness, dysphagia, hoarseness, anorexia, and weight loss. On examination, body mass index was 22 kg/m^2 with mild proptosis, symmetrical proximal weakness (MRC 4/5), and erythematous rashes on thighs and shins. Otorhinolaryngology evaluation confirmed bilateral vocal