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

Endah Pratamaningtias[1] and Nyoman Satvika Dharma Yudha[1]

[1]Division of Endocrinology, Metabolism and Diabetes, Department of Internal Medicine, Saiful Anwar Hospital-Faculty of Medicine Brawijaya University, Indonesia

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

Ket Meng Chin[1], Katherine Khor[2], Nor Azmi Kamaruddin[3]

[1]Department of Internal Medicine, Hospital Tuanku Fauziah
[2]Department of Endocrinology, Hospital Tuanku Fauziah
[3]Department of Cardiology, Institut Jantung Negara

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