CONCLUSION
Painful Hashimoto's thyroiditis should be considered in pregnant patients with known autoimmune thyroid disease presenting with acute thyroid pain despite normal thyroid function. Diagnosis requires integration of clinical, biochemical, and imaging findings to guide appropriate management.

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THE HIDDEN HAZARD AFTER TOTAL PARATHYROIDECTOMY: A CASE REPORT

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INTRODUCTION
Palpation thyroiditis is an underrecognized condition that occurs after parathyroidectomy. Symptoms range from mild to overt thyrotoxicosis that may result in life-threatening cardiac arrhythmias and acute coronary syndrome. Diagnosis is made by having suppressed thyroid-stimulating hormone (TSH) and elevated free thyroxine 4 (fT4) or free thyroxine 3 (fT3) and low radionuclide uptake on thyroid scan.

CASE
We report a case of a 72-year-old female with underlying end-stage renal disease on regular hemodialysis who underwent total parathyroidectomy for tertiary hyperparathyroidism. Her thyroid function test (TFT) preoperatively was normal, and cardiac assessment showed normal echocardiogram findings and sinus rhythm on electrocardiogram. Her operation was uncomplicated, involving neck exploration and all 4-gland removal. Postoperatively, she had new-onset atrial fibrillation (AF) with rapid ventricular response at day 5, requiring electrical and chemical cardioversion. She had recurrent tachyarrhythmias on day 6. Her repeated TFT showed suppressed TSH, 0.036 mIU/L (NR 0.38–5.33) and raised fT4, 35.4 pmol/L (NR 7.9–14.4), fT3–fT4 ratio 0.26 (<0.3), consistent with thyrotoxicosis. She was treated as thyroid storm (Burch-Wartofsky Point Scale 45), after ruling out other causes. She was started on glucocorticoids, propylthiouracil, and Lugol's iodine. Despite biochemical improvement, she continued to develop recurrent AF up till Day 12 post-op, requiring multimodal treatment, including beta-blocker and antiarrhythmic agents. She was later discharged well on low-dose carbimazole. Her ultrasound neck showed a normal thyroid gland, and her thyroid antibodies were negative.

CONCLUSION
A high level of suspicion is needed to detect thyroiditis post-parathyroidectomy due to its potential adverse impact on surgical outcomes. Treatment is mainly symptomatic, and the disease is self-limiting. Risk factors identified include secondary/tertiary hyperparathyroidism and bilateral neck exploration that involves manipulation of the thyroid gland.

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SUBACUTE HYPOTHYROID MYOPATHY AS AN ATYPICAL PRESENTATION FOLLOWING RADIOIODINE THERAPY

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INTRODUCTION
Hypothyroidism is the most common outcome following radioiodine (RAI) therapy for Graves' disease, affecting up to 80% of patients, usually within 6 months. While symptoms are often nonspecific, musculoskeletal complaints may be the predominant or sole manifestation. Hypothyroid myopathy occurs in 30–80% of patients, typically causing myalgias, cramps, fatigue, and slowly progressive, symmetric proximal weakness with delayed reflex relaxation. We report an atypical case with subacute and evolving weakness after levothyroxine initiation.

CASE
A 43-year-old female with Graves' disease underwent RAI therapy (25 mCi) and developed hypothyroidism 7 weeks later. She was started on levothyroxine 50 mcg daily. Two weeks into treatment, she presented with progressive proximal lower limb weakness (power 4/5), while distal strength and reflexes remained intact. Labs revealed elevated creatine kinase (259 U/L), hypokalemia (3.3 mmol/L), creatinine (57 µmol/L), and severe hypothyroidism (thyroid-stimulating hormone [TSH] 52.88 mIU/L, free thyroxine 4 [FT4] 7.79 pmol/L). Levothyroxine was increased to 100 mcg daily. Two weeks later, she developed proximal upper limb weakness (power 4/5), while lower limb strength had normalized. Nerve conduction studies and electromyography were unremarkable. Labs showed creatine kinase (244 U/L) and creatinine (58 µmol/L). As she remained hypothyroid (TSH 20.85 mIU/L, FT4 11.61 pmol/L), levothyroxine 100 mcg daily was continued. Her symptoms gradually improved alongside biochemical recovery (TSH 8.83 mIU/L, FT4 15.90 pmol/L) after 4 weeks, consistent with hypothyroid myopathy.

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.

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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