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LYMPHOCYTIC TURNED LYMPHOMATOUS: A CASE OF HASHIMOTO'S THYROIDITIS

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INTRODUCTION

Primary thyroid lymphoma (PTL) is a rare malignancy representing 1–5% of all thyroid cancers, most frequently arising on a background of chronic autoimmune thyroiditis.

CASE

We report a 73-year-old Malay male who initially presented with large goiter, symptomatic bradycardia, and subsequently developed severe primary hypothyroidism (thyroid-stimulating hormone 231 mIU/L, anti-thyroid peroxidase 643 IU/mL), consistent with Hashimoto's thyroiditis. During his clinical course, he also sustained a non-ST elevation Myocardial Infarction which was attributed to demand-supply mismatch of hypothyroidism. Computed tomography scan of the neck revealed a large multinodular goiter with retrosternal extension, tracheal compression, and extensive nodal lymphadenopathy. Initial Fine needle biopsy was non-diagnostic, but a repeat biopsy was performed due to rapidly enlarging goiter within weeks with suspicious nodules seen on bedside thyroid ultrasound. Ultrasound-guided core biopsy confirmed Diffuse Large B-cell Lymphoma (DLBCL), non-germinal centre B-cell (non-GCB) subtype. Despite initiation of levothyroxine and corticosteroid therapy, the patient deteriorated with remarkable rapidity over the ensuing weeks, culminating in a 15 × 10 cm compressive neck mass, Type 1 respiratory failure, hypercalcemia, complete dysphagia, and concurrent pneumonia. He was then transferred to hematology tertiary centre for commencement of his chemotherapy.

CONCLUSION

This case of PTL presenting in the background of Hashimoto's thyroiditis emphasizes the role of early tissue diagnosis in patients with rapidly enlarging goiter. Identification of PTL is critical because management shifts from surgical intervention to systemic chemotherapy.

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DISCORDANT TFT AFTER TOTAL THYROIDECTOMY: CHALLENGES OF DIAGNOSING RESISTANCE TO THYROID HORMONE

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INTRODUCTION

Thyroid hormone resistance syndrome (THR) is a disorder characterized by reduced responsiveness of target tissues to thyroid hormones with non-suppressed thyroid-stimulating hormone (TSH) despite elevated free thyroxine 4 (FT4). Diagnosing and managing THR in athyreotic patients, however, is challenging; indeed, TSH levels post thyroidectomy for Differentiated Thyroid Carcinoma in patients with THR have been reported to reach levels as high as 112.59 mIU/L despite high-dose thyroxine-suppressive therapy. This case highlights the complexities in diagnosing Resistance to Thyroid Hormone (RTH) post-thyroidectomy.

CASE

A 63-year-old female with end-stage renal failure (ESRF) on hemodialysis and prior parathyroidectomy for tertiary hyperparathyroidism underwent total thyroidectomy in 1997 for presumed benign goiter. Following surgery, she demonstrated persistently elevated TSH, ranging from 93.2 to 670.4 mIU/L (0.55–4.78 mIU/L), with normal to mildly elevated FT4 levels, ranging from 17 to 35 pmol/L (11.5–22.7 pmol/L), while on thyroxine replacement doses as low as 0.86 ug/kg. Discordant thyroid function tests (TFTs) were consistent across different assay platforms. Polyethylene glycol precipitation excluded macro-TSH interference.

Intermittent levothyroxine increments suppressed TSH but led to thyrotoxic symptoms, including weight loss, heat intolerance, insomnia, and fragility fracture. Uncontrasted pituitary magnetic resonance imaging (risk of nephrogenic systemic fibrosis with gadolinium in ESRF) showed a small pituitary gland without adenoma, excluding TSH-oma. T3 suppression test was relatively contraindicated due to her advanced age and co-morbidities. There was no family history of thyroid disorder; the patient's only child had a normal TFT, and her parents/siblings could not be tested. A working diagnosis of RTHβ was made. She declined genetic testing for RTH. She is currently on levothyroxine 50 mcg OD (1.07 ug/kg) with FT4 16.1 pmol/L and TSH 562 mIU/L with no symptoms/signs of hypo or hyperthyroidism.

CONCLUSION

This case highlights the diagnostic challenges in managing possible RTHβ post-total thyroidectomy. Treatment should be guided by clinical status and target of mid-to-normal FT4 rather than TSH to avoid iatrogenic thyrotoxicosis.

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FROM STABILITY TO STORM: THYROID STORM AFTER A DECADE OF ANTITHYROID DRUG

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INTRODUCTION

Long-term antithyroid drug (LT-ATD) has emerged as a feasible treatment strategy for patients who decline radioactive iodine (RAI) or thyroidectomy for relapsed or refractory Graves' disease (GD). Benefits include faster achievement of euthyroidism, lower risk of hypothyroidism, a more favorable cardiovascular profile, and avoidance of surgical risks. Although fluctuations in thyroid status may occur despite good compliance, thyroid storm is exceedingly rare in patients on LT-ATD. While there is no specific data on the incidence of thyroid storm in this cohort, surveys suggest an overall low incidence (0.2–0.76 cases per 100,000 annually). We report a patient with stable GD who developed a thyroid storm despite more than 10 years of LT-ATD.

CASE

A 40-year-old female was diagnosed with GD 11 years earlier during pregnancy. Treatment was stopped at 25 weeks' gestation, but she relapsed at 7 months postpartum and was started on carbimazole. She declined RAI or surgery following a relapse and remained on carbimazole 5–10 mg daily, with good compliance. She presented with a 1-day history of fever, cough, rhinorrhea, diarrhea, and palpitations. She was compliant with carbimazole 5 mg daily. On presentation, BP was 132/70 mmHg, HR 140 bpm, temperature 38.5°C, and SpO2 98% on air. She was alert without agitation, had a diffuse goiter, mild proptosis, and conjunctival injection, with otherwise normal findings. Electrocardiogram showed sinus tachycardia. Laboratory investigations demonstrated mild transaminitis, leukocytosis, markedly elevated free T4 (>154 pmol/L), suppressed thyroid-stimulating hormone (<0.008 mIU/L), and elevated thyroid-stimulating immunoglobulin (2.25 IU/L, reference <0.55). Thyroid function tests 1 month ago was normal. Her Burch-Wartofsky score was 60, consistent with thyroid storm, likely precipitated by upper respiratory tract infection. She improved with treatment and was discharged with carbimazole 30 mg daily with planned tapering, subsequently agreeing to RAI as definitive treatment.

CONCLUSION

Infection may trigger thyroid storm despite good compliance with LT-ATD. Patients should be counselled regarding this risk and advised to seek early medical attention if thyrotoxic symptoms recur.

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UNEXPECTED CARDIOVASCULAR COLLAPSE AFTER RADIOIODINE THERAPY IN A PATIENT WITH SEVERE GRAVES' CARDIOMYOPATHY

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INTRODUCTION

Graves' disease may lead to thyrotoxic cardiomyopathy, a potentially reversible condition following restoration of euthyroidism. Radioiodine (RAI) therapy is commonly used as definitive treatment for patients with long-standing Graves' disease or poor compliance with antithyroid medications. However, individuals with severe underlying cardiomyopathy may remain vulnerable to cardiovascular instability during the peri-treatment period.

CASE

We report a 39-year-old male with a 10-year history of Graves' disease who had previously defaulted on follow-up and was admitted with thyroid storm. Echocardiography during that admission demonstrated severe dilated cardiomyopathy with global hypokinesia and an ejection fraction (EF) of 26%. Following treatment with carbimazole and propranolol, he achieved biochemical euthyroidism and remained clinically stable (NYHA class I). In view of his long-standing disease and prior thyroid storm, he underwent RAI therapy as definitive therapy as per the guidelines.

One week following RAI, he developed a persistent cough and progressive dyspnea. Six weeks later, he presented to the emergency department with hypotension (BP 60/31 mmHg) and new-onset atrial fibrillation (heart rate 100 bpm), requiring intubation and inotropic support for cardiogenic shock. Laboratory evaluation showed low free