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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.