control. Given the underlying pathology, early definitive surgical management was planned with multidisciplinary input, and she subsequently underwent total abdominal hysterectomy with bilateral salpingo-oophorectomy successfully.

Hyperthyroidism in GTD is well described, but patients may remain clinically asymptomatic despite significant biochemical derangement, as seen in this case. Markedly elevated β-hCG can mimic primary thyroid disease and may lead to misinterpretation if the underlying cause is not recognized. While antithyroid drugs such as carbimazole are commonly initiated, they may have limited effect in this setting, as the hyperthyroidism is driven by hCG rather than intrinsic thyroid overactivity. Beta-blockers play an important role in controlling symptoms and reducing peripheral conversion of T4–T3. Early definitive treatment of the underlying trophoblastic disease remains the key to resolution.

CONCLUSION

Trophoblastic hyperthyroidism is a reversible condition secondary to the underlying disease process. Treatment of the trophoblastic tumor results in resolution of the thyrotoxic state. Early recognition and appropriate preoperative optimization are essential to ensure safe patient outcomes.

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A DIAGNOSTIC MASQUERADE: RESISTANCE TO THYROID HORMONE MIMICKING TSH-SECRETING PITUITARY ADENOMA

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INTRODUCTION

Discordant thyroid function tests (TFTs), characterized by elevated free thyroxine 4 (FT4) with non-suppressed thyroid-stimulating hormone (TSH), pose a significant diagnostic challenge. Differentiating between Resistance to Thyroid Hormone (RTH) and TSH-secreting pituitary adenoma (TSH-oma) is essential, as management strategies differ substantially.

CASE

A female with a history of hyperthyroidism diagnosed in 2011 was treated with antithyroid drugs for 1 year before defaulting on follow-up. She was later found to have discordant TFTs at a private centre, where a brain computed tomography scan was reportedly normal. She was referred to our centre for optimization of thyroid function prior to planned thyroidectomy for a solitary large right thyroid nodule measuring 4.2 × 2.8 × 4.7 cm. Fine-needle aspiration cytology demonstrated a benign follicular lesion (Bethesda II).

Despite restarting antithyroid medication, she remained clinically euthyroid with no overt thyrotoxic symptoms apart from intermittent palpitations without documented tachycardia. Antithyroid therapy was discontinued. Serial TFTs across multiple assay platforms consistently demonstrated elevated FT4 with inappropriately normal TSH levels. Thyroid autoantibodies, including TSH receptor and anti-thyroid peroxidase antibodies, were negative.

Pituitary magnetic resonance imaging showed no evidence of adenoma, and serum α-subunit level was normal (0.3 ng/mL), making TSH-oma unlikely. Dynamic testing was not performed as thyrotropin-releasing hormone stimulation was unavailable at our centre, while T3 suppression testing was deemed inappropriate due to symptomatic palpitations. Family screening was not possible as the patient was not in contact with her relatives. In the absence of pituitary pathology and given her largely euthyroid clinical status, RTH was considered the most likely diagnosis.

CONCLUSION

This case highlights the importance of considering RTH in patients with persistent discordant TFTs, particularly when clinical findings do not correlate with biochemical abnormalities. Early recognition and appropriate pituitary evaluation are essential to prevent misdiagnosis and avoid unnecessary antithyroid therapy or thyroidectomy.

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DYNAMIC AUTOIMMUNE THYROIDITIS IN MYELODYSPLASTIC SYNDROME: FROM PAINLESS THYROTOXICOSIS TO OVERT HYPOTHYROIDISM

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INTRODUCTION

Autoimmune thyroid disease can follow a triphasic course: thyrotoxicosis, a transient euthyroid period, and eventual hypothyroidism, and may coexist with myelodysplastic syndrome (MDS) in the context of immune dysregulation.