CONCLUSION

This case highlights a rare and potentially life-threatening association of thyroid storm and AMAN. Severe thyrotoxicosis can precipitate atypical autoimmune complications, underscoring the need for vigilance. Clinicians should consider neurological evaluation in patients with severe thyrotoxicosis presenting with new-onset motor weakness.

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SYNERGISTIC USE OF PLASMAPHERESIS AND LITHIUM IN REFRACTORY THYROID STORM

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INTRODUCTION

Thyroid storm is a life-threatening endocrine emergency with a mortality rate of 8–25% despite optimal therapy. Some patients exhibit a refractory phenotype characterized by rapid clinical deterioration and failure of conventional treatment, necessitating early escalation. Therapeutic plasmapheresis and lithium represent adjunctive therapies targeting different aspects of thyroid hormone physiology, yet their combined use remains underexplored.

CASE

A 55-year-old male with Graves' disease, non-adherent to treatment since 2020, presented with fever, palpitations, and dyspnea for 2 days. He recently started on carbimazole 30 mg daily and propranolol 1 week prior. On examination, blood pressure was 158/74 mmHg, heart rate 180 bpm, Glasgow Coma Scale 15/15 with bibasal crepitations. Electrocardiogram showed atrial fibrillation at 168 bpm. His Burch-Wartofsky score was 95, consistent with thyroid storm. Standard therapy with propylthiouracil 250 mg QID, Lugol's iodine, intravenous hydrocortisone 100 mg TDS, and carvedilol was commenced. However, after 3 days of treatment, he developed acute confusion and persistent fast atrial fibrillation requiring cardioversion. Liver function remained normal. Plasmapheresis was initiated on day 4 for six sessions. Propylthiouracil was switched to methimazole due to a declining white cell count from (5.5–3.2 $\times 10^9$/L). Lithium 300 mg BD was added on day 13 due to inadequate free thyroxine 4 (FT4) reduction. After 1 week of combined therapy, FT4 decreased from 70 to 35 pmol/L.

CONCLUSION

Early recognition of refractory disease and timely escalation are critical as refractory thyroid storm carries high mortality, especially with cardiovascular and neurological involvement. When conventional therapy fails, plasmapheresis facilitates rapid clearance of circulating thyroid hormones and inflammatory mediators, while lithium inhibits thyroid hormone release, providing an alternative mechanism when thionamides alone are insufficient. Their combined use offers a synergistic approach and targets both circulating and intrathyroidal hormone pools, suggesting that early dual-modality intervention is essential to overcome therapeutic resistance and improve overall outcomes in refractory disease.

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HYPERTHYROIDISM AND GESTATIONAL TROPHOBLASTIC DISEASE: A CASE REPORT

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INTRODUCTION

Gestational trophoblastic disease (GTD) is an uncommon but important cause of secondary hyperthyroidism, termed trophoblastic hyperthyroidism, resulting from the structural similarity between human chorionic gonadotropin (hCG) and thyroid-stimulating hormone (TSH). Excessively elevated hCG levels can stimulate the TSH receptor, leading to increased thyroid hormone production and clinically significant thyrotoxicosis. Early recognition is essential as uncontrolled hyperthyroidism may lead to serious perioperative complications.

CASE

We report a 50-year-old female who presented with persistent vaginal bleeding following a prior uterine evacuation. Clinical examination and ultrasonography revealed a uterine mass corresponding to approximately 14 weeks' gestation. Serum β-hCG was markedly elevated at >1,000,000 IU/L. Histopathological evaluation confirmed choriocarcinoma. Thyroid function tests demonstrated severe biochemical hyperthyroidism, with suppressed TSH and elevated free thyroxine levels. Notably, the patient did not exhibit classic symptoms or signs of hyperthyroidism such as palpitations, tremor, goiter, or thyroid eye signs. She was started on beta-blockers and carbimazole for initial

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.