

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