This creates a high-risk scenario where laboratory values overestimate tissue thyrotoxicity, predisposing to inappropriate antithyroid therapy. We present a case demonstrating marked clinical–biochemical dissociation, reframing postoperative thyrotoxicosis in ESRF as a disorder of hormone handling rather than hormone overproduction.

CASE

A 45-year-old female with ESRF on maintenance hemodialysis and tertiary hyperparathyroidism underwent total parathyroidectomy. Preoperative thyroid function was consistently euthyroid. Within 48 hours postoperatively, she developed severe biochemical thyrotoxicosis (thyroid-stimulating hormone 0.28 mIU/L, free thyroxine 4 [FT4] 68 pmol/L). Despite this, she remained clinically euthyroid, with stable hemodynamics, absence of adrenergic or neuropsychiatric features, and no evidence of thyroid eye disease.

The temporal relationship strongly suggested destructive thyroiditis secondary to surgical manipulation, with passive release of preformed thyroid hormone. In the context of ESRF, impaired hormone clearance and altered binding likely amplified circulating free hormone levels without proportional end-organ effect, resulting in striking clinical–biochemical dissociation.

A conservative strategy was adopted. Antithyroid drugs were withheld, given the non-synthetic mechanism of hormone excess, and the patient was managed with close monitoring and symptom-guided beta-blockade. Serial thyroid function demonstrated spontaneous improvement without complications.

CONCLUSION

Post-parathyroidectomy thyrotoxicosis in ESRF represents exaggerated biochemical derangement without true tissue toxicity. Management must prioritize physiology over laboratory values, as misclassification risks iatrogenic harm. This case demonstrates that in ESRF, elevated FT4 may not reflect true tissue thyrotoxicity, and reliance on biochemical severity alone can lead to inappropriate antithyroid therapy and iatrogenic harm.

EP_A175

THYROTOXICOSIS ASSOCIATED WITH GUILLAIN–BARRÉ SYNDROME: A RARE AUTOIMMUNE OVERLAP

Nur Asmak Abdullah[1], Rabeah Md Zuki[2], Mohamed Azlam Micdhadhu[3]

[1]*Internal Medicine Unit, Department of Medicine, Hospital Sultan Abdul Halim (HSAH)*

[2]*Endocrinology Unit, Department of Medicine, Hospital Sultan Abdul Halim (HSAH)*

[3]*Neurology Unit, Hospital Seberang Jaya*

INTRODUCTION

The coexistence of thyroid storm and Guillain–Barré syndrome (GBS) is rare, with few reported cases. A shared autoimmune mechanism has been suggested, although the exact pathophysiology remains unclear. In severe thyrotoxicosis, new neurological symptoms may be overlooked or attributed to metabolic causes, delaying diagnosis and posing a diagnostic and therapeutic challenge. We report a case of a 43-year-old female with Graves' disease complicated by thyroid storm and Acute Motor Axonal Neuropathy (AMAN), a variant of GBS, highlighting the importance of early recognition and multidisciplinary management.

CASE

A 43-year-old female with no prior medical illness presented with fever, generalized weakness, fine tremors, and 10 kg weight loss over 5 months. On arrival, she was lethargic, febrile (40.2°C), and tachycardic (148 bpm), consistent with thyroid storm by Burch–Wartofsky criteria. She denied preceding diarrheal illness or upper respiratory tract symptoms. The thyroid function test showed markedly elevated free thyroxine 4 (>64.35 pmol/L) and suppressed thyroid-stimulating hormone (TSH) (<0.008 mIU/L). Subsequent testing revealed elevated TSH receptor antibodies (>40 IU/L), confirming Graves' disease.

Her course was complicated by anaphylactic shock with transient cardiac arrest, followed by acute kidney injury and respiratory failure requiring intensive care unit admission and mechanical ventilation. Following extubation, symmetrical limb weakness with generalized areflexia and bilateral foot drop was observed. Nerve conduction studies demonstrated a symmetrical axonal motor-predominant polyneuropathy consistent with AMAN.

She received five sessions of plasma exchange and showed marked neurological improvement, while thyroid and renal function normalized at discharge.

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.

EP_A176

SYNERGISTIC USE OF PLASMAPHERESIS AND LITHIUM IN REFRACTORY THYROID STORM

Humaira Nuraqilah Mohd Yusof[1], Nur Aini Eddy Warman[1], Nur Haziqah Baharum[1], Aimi Fadilah Mohamad[1], Mohd Hazriq Awang[1], Fatimah Zaherah Mohamed Shah[1], Rohana Abdul Ghani[1]

[1]Department of Internal Medicine, Faculty of Medicine, Universiti Teknologi MARA

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.

EP_A177

HYPERTHYROIDISM AND GESTATIONAL TROPHOBLASTIC DISEASE: A CASE REPORT

K.J. Lingeswary[1], Jean Mun Cheah[1], Fei Bing Yong[1], Jen Hoong Oon[1], Aniqah Shamimi[1], Sharifah Noor Adrilla binti Long Mohd Noor Affendi[1], Gayathri Devi A/P Krishnan[1], Shazatul Reza Binti Mohd Redzuan[1], Subashini Rajoo[1]

[1]Endocrine Unit, Hospital Kuala Lumpur

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