At 24 + 4 weeks, TFTs showed persistently low FT4 (5.84 pmol/L) and TSH (0.022 mIU/L). Differential diagnoses included central hypothyroidism or assay interference. Tests were repeated using different platforms. Both the Beckman system (FT4: 5.73 pmol/L, TSH: 0.086 mIU/L) and Roche system (FT4: 8.83 pmol/L [12.0–22.0], TSH: 0.11 mIU/L [0.27–4.20]) confirmed low values, ruling out assay interference. Following endocrinology consultation and given her asymptomatic status, IH was considered most likely, and a plan for close monitoring was initiated.

Serial follow-up demonstrated biochemical evolution. By 31 + 6 weeks, TFTs showed a low TSH (0.753 mIU/L) with an FT4 level (5.86 pmol/L) within the normal reference range, a pattern typical of IH in pregnancy. Repeat TFTs 6 weeks postpartum showed both TSH and FT4 within normal range.

CONCLUSION

This case illustrates a rare and unexpected biochemical progression of IH in pregnancy, initially presenting with a pattern indistinguishable from central hypothyroidism. It underscores that in an asymptomatic, fertile patient, IH can manifest with an atypical pattern early in gestation. The findings suggest that vigilant serial monitoring, rather than immediate extensive pituitary workup, may be a prudent initial approach in such scenarios, as the biochemical profile can normalize toward the classic IH pattern as pregnancy advances.

EP_A173

WHEN TSH SUPPRESSION BECOMES HARMFUL: THYROXINE OVER-REPLACEMENT DRIVING CARDIOVASCULAR DECOMPENSATION IN ADVANCED HEART FAILURE

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INTRODUCTION

Thyroid stimulating hormone (TSH) suppression following differentiated thyroid carcinoma is widely recommended to reduce recurrence risk. However, this strategy assumes cardiovascular tolerance to supraphysiologic thyroid hormone exposure. In patients with advanced structural heart disease, this assumption may fail, exposing a critical limitation of guideline-directed TSH suppression.

CASE

We report a 71-year-old male with end-stage renal failure on hemodialysis and severe ischemic cardiomyopathy (ejection fraction 23%) who presented with acute decompensation characterized by dyspnea, rapid atrial fibrillation, and non–ST-elevation myocardial infarction. He had a history of papillary thyroid carcinoma treated with total thyroidectomy and radioactive iodine over 20 years prior and was maintained on levothyroxine 200 mcg daily for TSH suppression. Despite biochemically euthyroid indices (TSH 1.8 mIU/L, free thyroxine 4 17 pmol/L), he developed recurrent arrhythmia with heart failure decompensation.

This case highlights a dissociation between biochemical euthyroidism and tissue-level thyrotoxicity in a structurally compromised myocardium. Papillary thyroid carcinoma after definitive therapy typically follows an indolent course with low short-term mortality. In contrast, in severe left ventricular dysfunction, excess thyroid hormone increases adrenergic sensitivity and myocardial oxygen demand, precipitating arrhythmia and ischemia. This risk is amplified in end-stage renal disease, where altered hormone handling renders biochemical indices less reliable.

CONCLUSION

Biochemical euthyroidism does not equate to physiological safety. In patients with advanced cardiovascular disease, thyroid hormone therapy should be titrated to cardiovascular tolerance rather than oncologic targets alone, and routine TSH suppression may be inappropriate.

EP_A174

SEVERE BIOCHEMICAL THYROTOXICOSIS WITHOUT CLINICAL HYPERTHYROIDISM IN ESRF FOLLOWING PARATHYROIDECTOMY: A DIAGNOSTIC AND THERAPEUTIC PITFALL

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INTRODUCTION

Thyrotoxicosis following neck surgery is typically attributed to transient destructive thyroiditis from follicular disruption. In end-stage renal failure (ESRF), however, altered thyroid hormone kinetics, including reduced protein binding, impaired peripheral metabolism, and decreased clearance, can distort biochemical interpretation.

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

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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.