During follow-ups, he had issues with compliance with the antithyroid therapy. However, the latest thyroid function test in February 2026 showed Free T4 of 20.5 pmol/L with suppressed TSH of <0.01 mIU/L. He remains clinically euthyroid throughout the follow-up.

CONCLUSION

This case highlights a rare but clinically relevant coexistence. Clinicians managing symptomatic CF patients should vigilantly screen for thyroid dysfunction to ensure early diagnosis and timely intervention. Early recognition and treatment may improve patient outcomes. Further research is needed to clarify the immunological link between CF and autoimmunity.

EP_A171

THYROID–LIVER INTERPLAY: EARLY RECOGNITION OF CARBIMAZOLE-INDUCED CHOLESTASIS AMID THYROTOXICOSIS

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INTRODUCTION

Carbimazole is a first-line therapy for thyrotoxicosis and is generally well tolerated. Drug-induced liver injury is rare (<1%) and typically presents as cholestatic hepatotoxicity, in contrast to propylthiouracil, which more commonly causes hepatocellular injury. Clinical presentation may mimic obstructive jaundice, and delayed recognition can lead to unnecessary investigations and interruption of definitive thyroid management.

CASE

A 70-year-old female with toxic multinodular goiter developed painless jaundice 4 weeks after starting carbimazole 20 mg daily for thyrotoxicosis precipitated by urinary tract infection. She had no prior liver disease or alcohol exposure. Examination revealed isolated icterus without features of chronic liver disease.

Initial thyroid function tests showed suppressed thyroid-stimulating hormone (<0.01 mIU/L) with markedly elevated free T4 (>100 pmol/L), improving after 4 weeks (free T4 29.1 pmol/L). She subsequently developed progressive jaundice without abdominal pain, fever, pruritus, or encephalopathy. Liver biochemistry demonstrated a cholestatic pattern (R factor 1.1) with conjugated hyperbilirubinemia (peak bilirubin 227 µmol/L), mild transaminitis, and elevated alkaline phosphatase.

Imaging, including hepatobiliary ultrasonography, contrast computed tomography, and endoscopic ultrasound, excluded biliary obstruction. Viral, autoimmune, and structural causes were negative. Carbimazole-induced cholestatic jaundice was diagnosed based on temporal association and exclusion of alternatives. Carbimazole was discontinued, ursodeoxycholic acid was initiated, and radioactive iodine therapy was performed, followed by gradual recovery.

CONCLUSION

Carbimazole-induced hepatotoxicity (0.1–0.2%) is likely idiosyncratic and not dose dependent. Differentiating drug-induced liver injury from thyrotoxicosis-related liver dysfunction is critical, as restoration of euthyroidism alone may normalize liver enzymes. Diagnosis relies on the exclusion of obstruction and recognition of drug chronology. Early drug withdrawal and multidisciplinary management are essential to prevent progression while ensuring timely definitive therapy.

EP_A172

THE EVOLVING BIOCHEMICAL PROFILE: FROM LOW FT4/LOW TSH TO ISOLATED HYPOTHYROXINEMIA IN PREGNANCY

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INTRODUCTION

Isolated hypothyroxinemia (IH) in pregnancy, characterized by low free thyroxine 4 (FT4) with normal thyroid-stimulating hormone (TSH), has a reported prevalence of 1.3–8%. We present a diagnostically challenging case that initially mimicked central hypothyroidism before evolving into classic IH.

CASE

A 37-year-old G4P3 female at 22 + 3 weeks gestation with β-thalassemia trait presented with palpitations and tachycardia (150–184 bpm). A bedside echocardiogram showed volume depletion. Her heart rate improved to 108–116 bpm following a 1.5 L normal saline bolus. Incidentally, thyroid function tests (TFTs) revealed both low FT4 6.04 pmol/L (7.86–14.41) and low TSH 0.009 mIU/L (0.38–5.33). Anti-thyroid peroxidase antibody was markedly elevated at 360 IU/mL (<35).

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.