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.

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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.

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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).