should maintain a high index of suspicion for macro-TSH in asymptomatic patients presenting with isolated TSH elevations that do not respond to thyroxine therapy. Prompt recognition prevents misdiagnosis and avoids the potential risks of unnecessary thyroid hormone replacement.

EP_A169

OVERT HYPOTHYROIDISM PRESENTING WITH ISOLATED LOWER MOTOR NEURON FACIAL NERVE PALSY

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INTRODUCTION
Cranial nerve palsies have been reported in patients with uncontrolled hypothyroidism. We describe a case of overt hypothyroidism due to treatment disruption following radioiodine therapy, presented with a unilateral lower motor neuron seventh cranial nerve palsy.

CASE
A 32-year-old female presented with hypertension, dyslipidemia, class III obesity, and Graves' disease post radioiodine therapy. She had stable thyroid function test with levothyroxine replacement post radioiodine. She presented with a 3-day history of right-sided facial droop associated with lethargy, weight gain, and constipation. She reported inadequate levothyroxine supply due to 2-month lapses in follow-up.

On examination, there was a right-sided lower motor neuron 7th cranial nerve palsy with delayed ankle reflexes. No additional neurological deficits, goiter, cutaneous rashes, or vesicular lesions were noted. There was no preceding viral illness or otologic symptoms.

Thyroid function tests showed overt hypothyroidism with thyroid-stimulating hormone 38 mIU/L, free thyroxine <5.4 pmol/L with a deranged lipid profile (total cholesterol: 8.8 mmol/L, low-density lipoprotein: 5.72 mmol/L, triglyceride: 1.77 mmol/L). Renal and liver function tests were within normal limits, with no leukocytosis. Neuroimaging of the brain was unremarkable.

She was commenced on a short course of high-dose prednisolone, with no improvement in symptoms. Levothyroxine replacement therapy initiated led to progressive restoration of thyroid function, accompanied by symptom recovery.

CONCLUSION
Isolated lower motor 7th nerve palsy has been reported as a manifestation of hypothyroidism. This case underscores the importance of early recognition to enable timely and appropriate management, as patients with hypothyroidism-induced isolated seventh nerve palsy demonstrate marked recovery with adequate levothyroxine replacement.

EP_A170

FIRE IN THE GLAND: A RARE CASE OF GRAVES' DISEASE IN CYSTIC FIBROSIS

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INTRODUCTION
Cystic fibrosis (CF) is an autosomal recessive disorder caused by mutations in the CFTR gene. Complications such as cystic fibrosis–related diabetes (CFRD) are well recognized. The association between CF and autoimmune thyroid disease, however, is rare and poorly understood. We report a case of CFRD complicated by Graves' disease.

CASE
A 22-year-old male was diagnosed with CF at age 5, confirmed by a positive sweat chloride test. Following the diagnosis, lifelong pancreatic enzyme replacement therapy (Creon) was initiated to treat exocrine pancreatic insufficiency. In 2021, he developed type 3c diabetes, attributed to endocrine pancreatic insufficiency, and required regular basal insulin therapy.

In early 2024, he developed hypokalemic periodic paralysis with proximal myopathy, despite potassium correction, and was admitted to the hospital. On admission, examination revealed a fine tremor and diffuse bilateral neck swelling. Biochemical evaluation showed thyrotoxicosis with Free T4 of 37 pmol/L and thyroid-stimulating hormone (TSH) <0.01 mIU/L. He started a tapering dose of carbimazole and propranolol. An urgent neck ultrasound showed a heterogeneous thyroid parenchyma with increased vascularity and no nodules. Elevated anti-thyroid peroxidase (anti-thyroid peroxidase, >600 IU/mL) and TSH receptor antibodies (thyrotropin receptor antibody, 2.57 IU/L) confirmed a diagnosis of Graves' disease.

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).