

CONCLUSION

This case highlights the diagnostic challenges in managing possible RTH β post-total thyroidectomy. Treatment should be guided by clinical status and target of mid-to-normal FT4 rather than TSH to avoid iatrogenic thyrotoxicosis.

EP_A164

FROM STABILITY TO STORM: THYROID STORM AFTER A DECADE OF ANTITHYROID DRUG

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INTRODUCTION

Long-term antithyroid drug (LT-ATD) has emerged as a feasible treatment strategy for patients who decline radioactive iodine (RAI) or thyroidectomy for relapsed or refractory Graves' disease (GD). Benefits include faster achievement of euthyroidism, lower risk of hypothyroidism, a more favorable cardiovascular profile, and avoidance of surgical risks. Although fluctuations in thyroid status may occur despite good compliance, thyroid storm is exceedingly rare in patients on LT-ATD. While there is no specific data on the incidence of thyroid storm in this cohort, surveys suggest an overall low incidence (0.2–0.76 cases per 100,000 annually). We report a patient with stable GD who developed a thyroid storm despite more than 10 years of LT-ATD.

CASE

A 40-year-old female was diagnosed with GD 11 years earlier during pregnancy. Treatment was stopped at 25 weeks' gestation, but she relapsed at 7 months postpartum and was started on carbimazole. She declined RAI or surgery following a relapse and remained on carbimazole 5–10 mg daily, with good compliance. She presented with a 1-day history of fever, cough, rhinorrhea, diarrhea, and palpitations. She was compliant with carbimazole 5 mg daily. On presentation, BP was 132/70 mmHg, HR 140 bpm, temperature 38.5°C, and SpO₂ 98% on air. She was alert without agitation, had a diffuse goiter, mild proptosis, and conjunctival injection, with otherwise normal findings. Electrocardiogram showed sinus tachycardia. Laboratory investigations demonstrated mild transaminitis, leukocytosis, markedly elevated free T₄ (>154 pmol/L), suppressed thyroid-stimulating hormone (<0.008 mIU/L), and elevated thyroid-stimulating immunoglobulin (2.25

IU/L, reference <0.55). Thyroid function tests 1 month ago was normal. Her Burch-Wartofsky score was 60, consistent with thyroid storm, likely precipitated by upper respiratory tract infection. She improved with treatment and was discharged with carbimazole 30 mg daily with planned tapering, subsequently agreeing to RAI as definitive treatment.

CONCLUSION

Infection may trigger thyroid storm despite good compliance with LT-ATD. Patients should be counselled regarding this risk and advised to seek early medical attention if thyrotoxic symptoms recur.

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UNEXPECTED CARDIOVASCULAR COLLAPSE AFTER RADIOIODINE THERAPY IN A PATIENT WITH SEVERE GRAVES' CARDIOMYOPATHY

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INTRODUCTION

Graves' disease may lead to thyrotoxic cardiomyopathy, a potentially reversible condition following restoration of euthyroidism. Radioiodine (RAI) therapy is commonly used as definitive treatment for patients with long-standing Graves' disease or poor compliance with antithyroid medications. However, individuals with severe underlying cardiomyopathy may remain vulnerable to cardiovascular instability during the peri-treatment period.

CASE

We report a 39-year-old male with a 10-year history of Graves' disease who had previously defaulted on follow-up and was admitted with thyroid storm. Echocardiography during that admission demonstrated severe dilated cardiomyopathy with global hypokinesia and an ejection fraction (EF) of 26%. Following treatment with carbimazole and propranolol, he achieved biochemical euthyroidism and remained clinically stable (NYHA class I). In view of his long-standing disease and prior thyroid storm, he underwent RAI therapy as definitive therapy as per the guidelines.

One week following RAI, he developed a persistent cough and progressive dyspnea. Six weeks later, he presented to the emergency department with hypotension (BP 60/31 mmHg) and new-onset atrial fibrillation (heart rate 100 bpm), requiring intubation and inotropic support for cardiogenic shock. Laboratory evaluation showed low free

T4 (5.31 pmol/L) with normal thyroid-stimulating hormone (2.1 mIU/L). Repeat echocardiography demonstrated persistent severe left ventricular dysfunction (EF ~25%) without new structural abnormalities. The patient improved rapidly with supportive management and was successfully extubated after stabilization.

Although RAI is considered a safe and effective definitive therapy, transient thyroid hormone fluctuations and inflammatory thyroid destruction may occur following treatment. In patients with severe pre-existing cardiomyopathy, these physiological changes may precipitate arrhythmias or acute cardiac decompensation. In this case, new-onset atrial fibrillation in the setting of markedly reduced myocardial reserve likely triggered cardiogenic shock despite biochemical euthyroidism.

CONCLUSION

This case highlights that patients with advanced Graves' cardiomyopathy may remain at risk of acute cardiovascular deterioration following RAI therapy. Careful cardiovascular risk assessment, optimization of heart failure therapy, and close monitoring after definitive treatment should be considered in this high-risk population.

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ACUTE ISCHEMIC STROKE MASKING UNDERLYING HYPERTHYROIDISM: A DIAGNOSTIC PITFALL OF NON-THYROIDAL ILLNESS SYNDROME

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INTRODUCTION

Non-thyroidal illness syndrome commonly occurs during acute systemic illnesses and is characterized by suppressed or inappropriately normal thyroid-stimulating hormone with low or low-normal thyroid hormone levels. This biochemical pattern may obscure or delay the diagnosis of underlying hyperthyroidism, especially in mild or borderline cases. Acute stroke is a recognized trigger of non-thyroidal illness syndrome, yet its masking effect on hyperthyroidism is under-recognized in clinical practice.

CASE

A 58-year-old Malay male with cardiovascular risk factors presented with an acute ischemic stroke involving the left occipital lobe and right internal capsule. He developed new-onset atrial fibrillation (CHA₂DS₂-VASc 3) and mild left ventricular systolic dysfunction. Initial thyroid function tests during the acute stroke phase showed mildly suppressed thyroid-stimulating hormone (TSH) (0.30 mIU/L) with high-normal free T4 (21.2 pmol/L), interpreted in the context of acute illness. The patient was clinically stable without overt thyrotoxic features.

One month post-stroke, repeat testing revealed further TSH suppression (0.09 mIU/L) and rising free T4 (27.6 pmol/L). Detailed history uncovered prior Graves' disease in 2019 with treatment default. Examination revealed a small diffuse goiter without ophthalmopathy. The biochemical evolution following recovery from acute illness confirmed recurrent hyperthyroidism previously masked by non-thyroidal illness syndrome.

CONCLUSION

Acute stroke can induce cytokine-mediated suppression of the hypothalamic-pituitary-thyroid axis and altered peripheral deiodination, leading to misleading thyroid function tests. In this case, non-thyroidal illness syndrome blunted the biochemical severity of hyperthyroidism, delaying recognition despite high-risk features such as atrial fibrillation and prior Graves' disease. Reliance on a single thyroid function test during acute illness may therefore result in underdiagnosis.

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WHEN THYROID MEETS DENGUE AND HEPATITIS: A CASE OF SEVERE THYROTOXICOSIS WITH MULTIORGAN DYSFUNCTION

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INTRODUCTION

Severe thyrotoxicosis is an endocrine emergency that may present with multiorgan dysfunction, particularly when precipitated by systemic infection. Management becomes challenging when hepatic injury limits the use of standard antithyroid therapy. We report a case of severe thyrotoxicosis with acute hepatitis in the setting of dengue IgM positivity.