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 (CHA_2DS_2-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.