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.

EP_A166

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.

EP_A167

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.

CASE

A 44-year-old male with chronic alcohol use presented with epistaxis. On examination, he was deeply jaundiced and had tremors on outstretched hands. Initial investigations showed severe hepatitis with AST 2029 U/L, ALT 698 U/L, total bilirubin 236 µmol/L, alkaline phosphatase 138 U/L, and thrombocytopenia (34 $\times 10^9$/L). Viral hepatitis HBV, HCV, HIV, and autoimmune hepatitis screen were negative. Dengue IgM was positive.

Thyroid function tests demonstrated overt thyrotoxicosis with free thyroxine 4 (FT4) 58.6 pmol/L and suppressed thyroid-stimulating hormone.

He was managed as severe thyrotoxicosis with impending thyroid crisis in view of systemic illness and multiorgan involvement. Due to significant hepatic dysfunction, he was treated with propranolol, dexamethasone, and lithium carbonate 300 mg BD. Over 5 days, there was marked clinical improvement with resolution of tachycardia and tremors. FT4 decreased to 44.8 pmol/L. Liver function tests improved significantly (AST 157 U/L, ALT 195 U/L, bilirubin 163 µmol/L), and thrombocytopenia resolved (platelets 323 $\times 10^9$/L). He remained hemodynamically stable with normal Glasgow Coma Scale throughout admission.

CONCLUSION

This case highlights severe thyrotoxicosis with acute hepatitis and dengue infection, where management was complicated by contraindication to conventional antithyroid therapy. Lithium and corticosteroids provided effective biochemical and clinical improvement. Early recognition and individualized therapy are crucial in complex multisystem thyrotoxic presentations.

EP_A168

PRESENCE OF MACRO-TSH: A RARE MIMICKER OF SUBCLINICAL HYPOTHYROIDISM

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INTRODUCTION

Macro-thyroid-stimulating hormone (macro-TSH) is a rare complex formed by monomeric TSH with anti-TSH autoantibodies. Macromolecules of TSH are not renally excreted due to its large size, leading to elevated TSH measurements without clinical consequences. This rare condition frequently mimics subclinical hypothyroidism, often leading to misdiagnosis and inappropriate levothyroxine therapy.

CASE

A 17-year-old female with major depressive disorder was biochemically diagnosed with subclinical hypothyroidism (TSH 39.06 mIU/L, free thyroxine 4 12.9 pmol/L, anti-thyroid peroxidase negative) and commenced on levothyroxine. Over 5 years, her TSH levels heavily fluctuated (0.48–82.59 mIU/L) and remained persistently elevated with high-normal free T4 levels despite treatment adherence. An endocrinology consult was obtained, and clinical evaluation revealed a clinically asymptomatic and euthyroid patient, with no family history of thyroid disease or supplement use, and there was no goiter. Assay interference was excluded by analyzing her thyroid function tests on a different platform, which yielded similar biochemical results. Subsequently, a polyethylene glycol (PEG) precipitation test was performed. Her pre-PEG TSH of 29.24 mIU/L decreased significantly to 2.78 mIU/L post-PEG. This yielded a remarkably low TSH recovery rate of 9.5%, strongly indicating the presence of macro-TSH. Levothyroxine was then stopped, and she remained clinically euthyroid.

CONCLUSION

While gel filtration chromatography remains the gold standard for diagnosing this condition, PEG precipitation is a more accessible, cost-effective, and reliable screening method in clinical practice. A TSH recovery rate below 20% is considered highly suggestive of macro-TSH. Clinicians