

CASE

A 31-year-old male with Graves' disease and poor medication adherence presented with a 2-week history of aggressive behavior and auditory hallucinations. Initial investigations revealed overt thyrotoxicosis (thyroid-stimulating hormone <0.01 mIU/L; free thyroxine 4 (FT4) 59.53 pmol/L) and an unremarkable computed tomography brain. Despite his severe neuropsychiatric decompensation, a BWPS of 20 precluded a clinical diagnosis of systemic thyroid storm.

His symptoms remained refractory to aggressive psychotropic polypharmacy, including haloperidol, olanzapine, lithium, and diazepam. Management was therefore focused on the underlying thyrotoxicosis with an optimized antithyroid regimen of carbimazole and propranolol. Clinical resolution of psychotic symptoms was achieved only after FT4 declined significantly to 20.46 pmol/L, allowing the successful tapering of all psychiatric medications. He was discharged in a stable, clinically euthyroid state with integrated medical and psychiatric follow-up.

CONCLUSION

The failure of standard antipsychotics suggests that thyrotoxic psychosis is driven by distinct neuromodulatory mechanisms rather than primary dopaminergic dysfunction. The chronic thyrotoxic state secondary to prolonged treatment non-adherence likely upregulates and sensitizes dopamine receptors while simultaneously suppressing inhibitory GABAergic pathways. This neurochemical imbalance lowers the neurological threshold, precipitating an isolated cerebral storm. This case illustrates the limitations of the BWPS in purely neuropsychiatric presentations and reinforces that restoring euthyroidism is the definitive treatment for thyrotoxic psychosis.

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SUBACUTE THYROIDITIS VS SUPPURATIVE THYROIDITIS: A DIAGNOSTIC CHALLENGE

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INTRODUCTION

Subacute thyroiditis (SAT) and acute suppurative thyroiditis (AST) are two distinct inflammatory conditions but share many overlapping clinical and biochemical features, which may pose a diagnostic challenge. We report a case of SAT mimicking AST.

CASE

A 39-year-old female presented with a 3-week history of painful neck swelling and symptoms of thyrotoxicosis, including palpitations, heat intolerance, and diaphoresis. She had no family history of thyroid disease. On examination, she was febrile and had a smooth and tender goiter. Her blood tests showed leukocytosis with elevated erythrocyte sedimentation rate and C-reactive protein. Her thyroid function test (TFT) showed thyroid-stimulating hormone of 0.023 mIU/L and free thyroxine 4 of 32.15 pmol/L.

An urgent ultrasound revealed a large, ill-defined heterogeneously hypoechoic lesion predominantly at the left lobe of the thyroid gland with mild intralesional vascularity. These were reported as features of thyroiditis with infective or early suppurative changes. Fine needle aspiration (FNA) of the lesion was attempted by the radiologist but was unsuccessful.

She was treated with intravenous antibiotics and non-steroidal anti-inflammatory drugs (NSAIDs), but symptoms persisted. Repeated ultrasound and computed tomography scans about 1 week later confirmed an enlarged thyroid gland with no obvious lesion or collection within the gland. The diagnosis was revised to SAT, and prednisolone 15 mg OD was started. This resulted in rapid improvement in her symptoms and inflammatory markers.

Four weeks later, she remained asymptomatic, and prednisolone was gradually tapered off. Serial TFT monitoring showed a classic triphasic pattern with an initial hyperthyroid phase, followed by hypothyroidism before returning to euthyroidism.

CONCLUSION

SAT can present with a focal lesion on the ultrasound during the early stage of the disease, making it difficult to distinguish from a thyroid abscess due to overlapping features. Good clinical judgement guided by FNA and appropriate follow-up imaging may be helpful in differentiating between the two conditions.

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RESCUE PLASMA EXCHANGE IN THYROID STORM: SUCCESSFUL BRIDGING TO THIONAMIDE THERAPY

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INTRODUCTION

Therapeutic plasma exchange (TPE) has emerged as a rescue therapy in thyroid storm. Multiple cycles are often required to achieve adequate reduction in circulating thyroid hormone levels. We report a case of thyroid storm with multiorgan failure successfully managed with only two cycles of TPE, followed by carbimazole.

CASE

A 43-year-old female with no significant past medical history presented with a 5-month history of altered bowel habits. She has a small goiter, which was not investigated before. She underwent elective esophagogastroduodenoscopy and colonoscopy at a private centre, both of which were unremarkable. Post-procedure, she developed unstable tachyarrhythmia and cardiac arrest. Return of spontaneous circulation was achieved after three cycles of cardiopulmonary resuscitation, and she was transferred to the intensive care unit. Biochemical evaluation revealed severe thyrotoxicosis (thyroid-stimulating hormone [TSH] <0.008 mIU/L, free thyroxine 4 [FT4] >100 pmol/L). Burch-Wartofsky Point Scale was 90.

Her clinical course was complicated by multiorgan failure, including ischemic hepatitis with coagulopathy and oliguric acute kidney injury requiring continuous veno-venous hemodialysis. Lugol's iodine and corticosteroids were initiated as thionamides were contraindicated due to severe hepatic dysfunction. She developed refractory tachyarrhythmia despite electrical cardioversion. Following a multidisciplinary discussion, TPE was initiated due to the limited choice of antithyroid medication.

After two TPE sessions, thyroid function improved markedly (TSH 0.01 mIU/L, FT4 26.2 pmol/L), accompanied by improvement in hepatic and renal function, allowing safe commencement of carbimazole. Thyroid function remained stable with FT4 of 19.3 pmol/L 1 week post TPE. Echocardiography demonstrated mildly reduced left ventricular ejection fraction (45–50%) with bi-atrial dilatation.

CONCLUSION

This case highlights the role of limited-cycle TPE as an effective rescue therapy and bridging strategy to definitive treatment in severe thyroid storm with multiorgan failure. Timely TPE initiation may improve biochemical control and clinical outcomes in critically ill patients.