CASE
We describe a 35-year-old male with MDS (multilineage dysplasia) receiving cyclosporine who initially presented with painless thyrotoxicosis, with suppressed thyroid-stimulating hormone (TSH) and elevated free thyroxine (FT4). During this hyperthyroid phase, thyrotropin receptor antibody (TRAb) was positive. Over time, his thyroid status fluctuated, progressing to overt hypothyroidism, and he had poor adherence to thyroid medications. On admission, he had prominent hypothyroid features (fatigue, cold intolerance, slowed movement, periorbital puffiness) with severe biochemical hypothyroidism (TSH 49.5 μIU/mL; FT4 0.419 ng/dL). Anti-thyroid peroxidase (anti-TPO) antibodies 12.62 IU/mL (negative <5.61 IU/mL; positive ≥5.61 IU/mL) showed an autoimmune response to the thyroid. Thyroid ultrasound showed heterogeneous, hyperechoic with some hypoechoic parts, and no significant increase in thyroid vascularity was observed, supporting autoimmune thyroiditis representation. His hematologic profile remained consistent with MDS, with chronic macrocytic anemia and thrombocytopenia.

CONCLUSION
This case highlights a dynamic autoimmune thyroiditis phenotype progressing from a hyperthyroid phase with TRAb positivity to higher anti-TPO overt hypothyroidism in a patient with MDS. In individuals with MDS and changing thyroid function tests, clinicians should keep autoimmune thyroiditis variants in mind and use thyroid antibodies together with ultrasound to secure the diagnosis when the clinical pattern is typical.

EP_A180

A THORN IN THE TREATMENT OF GRAVES' DISEASE: THE HIDDEN ALLERGEN

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INTRODUCTION
Graves' disease is typically managed with antithyroid drugs (ATDs) and beta-blockers like propranolol. While allergic reactions to ATDs are common, beta-blockers are rarely identified as allergens.

CASE
A 42-year-old female with Graves' disease was started on carbimazole 5 mg daily and propranolol 40 mg daily. She developed mild itchiness, which was tolerable. One month later, liver enzyme derangement led to the discontinuation of carbimazole. Propylthiouracil (PTU) 300 mg daily was initiated while continuing propranolol, but caused generalized urticaria, necessitating its cessation. Prednisolone was started, but her thyroid function worsened. Alternative therapies were proposed but declined by the patient. Upon resolution of urticaria, PTU was reintroduced at 50 mg daily without adverse effects, and propranolol was discontinued. Her cutaneous symptoms did not recur, implicating propranolol as the allergen.

CONCLUSION
In hyperthyroidism, increased hepatic clearance reduces plasma propranolol levels, minimizing the risk of adverse effects. However, as thyroid function normalizes, propranolol clearance slows, leading to drug accumulation and increased susceptibility to side effects. Clinicians should consider all medications as potential allergens and understand how thyroid states affect drug metabolism to optimize treatment.

EP_A181

TITRATING THE MIND: REFRACTORY SCHIZOPHRENIFORM PSYCHOSIS AS AN ISOLATED "CEREBRAL STORM" IN GRAVES' DISEASE

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INTRODUCTION
Neuropsychiatric manifestations of thyrotoxicosis range from mild anxiety to severe psychosis. While the Burch-Wartofsky Point Scale (BWPS) reliably identifies systemic thyroid storms, an isolated "cerebral storm"—where profound thyrotoxic encephalopathy presents without peripheral autonomic signs—remains a diagnostic challenge. This case highlights the neuromodulatory pathogenesis of thyrotoxic psychosis and the critical role of biochemical control for psychiatric resolution.

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.

EP_A182

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.