

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 μ IU/mL; FT4 0.419 ng/dL). Anti-thyroid peroxidase (anti-TPO) antibodies 12.62 IU/mL (negative <5.61 IU/mL; positive $\geq$ 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.

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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.

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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.