control. Given the underlying pathology, early definitive surgical management was planned with multidisciplinary input, and she subsequently underwent total abdominal hysterectomy with bilateral salpingo-oophorectomy successfully.

Hyperthyroidism in GTD is well described, but patients may remain clinically asymptomatic despite significant biochemical derangement, as seen in this case. Markedly elevated β-hCG can mimic primary thyroid disease and may lead to misinterpretation if the underlying cause is not recognized. While antithyroid drugs such as carbimazole are commonly initiated, they may have limited effect in this setting, as the hyperthyroidism is driven by hCG rather than intrinsic thyroid overactivity. Beta-blockers play an important role in controlling symptoms and reducing peripheral conversion of T4–T3. Early definitive treatment of the underlying trophoblastic disease remains the key to resolution.

CONCLUSION

Trophoblastic hyperthyroidism is a reversible condition secondary to the underlying disease process. Treatment of the trophoblastic tumor results in resolution of the thyrotoxic state. Early recognition and appropriate preoperative optimization are essential to ensure safe patient outcomes.

EP_A178

A DIAGNOSTIC MASQUERADE: RESISTANCE TO THYROID HORMONE MIMICKING TSH-SECRETING PITUITARY ADENOMA

Deviga Lachumanan[1]
[1]*Department of Medicine, Hospital Port Dickson*

INTRODUCTION

Discordant thyroid function tests (TFTs), characterized by elevated free thyroxine 4 (FT4) with non-suppressed thyroid-stimulating hormone (TSH), pose a significant diagnostic challenge. Differentiating between Resistance to Thyroid Hormone (RTH) and TSH-secreting pituitary adenoma (TSH-oma) is essential, as management strategies differ substantially.

CASE

A female with a history of hyperthyroidism diagnosed in 2011 was treated with antithyroid drugs for 1 year before defaulting on follow-up. She was later found to have discordant TFTs at a private centre, where a brain computed tomography scan was reportedly normal. She was referred to our centre for optimization of thyroid function prior to planned thyroidectomy for a solitary large right thyroid nodule measuring 4.2 × 2.8 × 4.7 cm. Fine-needle aspiration cytology demonstrated a benign follicular lesion (Bethesda II).

Despite restarting antithyroid medication, she remained clinically euthyroid with no overt thyrotoxic symptoms apart from intermittent palpitations without documented tachycardia. Antithyroid therapy was discontinued. Serial TFTs across multiple assay platforms consistently demonstrated elevated FT4 with inappropriately normal TSH levels. Thyroid autoantibodies, including TSH receptor and anti-thyroid peroxidase antibodies, were negative.

Pituitary magnetic resonance imaging showed no evidence of adenoma, and serum α-subunit level was normal (0.3 ng/mL), making TSH-oma unlikely. Dynamic testing was not performed as thyrotropin-releasing hormone stimulation was unavailable at our centre, while T3 suppression testing was deemed inappropriate due to symptomatic palpitations. Family screening was not possible as the patient was not in contact with her relatives. In the absence of pituitary pathology and given her largely euthyroid clinical status, RTH was considered the most likely diagnosis.

CONCLUSION

This case highlights the importance of considering RTH in patients with persistent discordant TFTs, particularly when clinical findings do not correlate with biochemical abnormalities. Early recognition and appropriate pituitary evaluation are essential to prevent misdiagnosis and avoid unnecessary antithyroid therapy or thyroidectomy.

EP_A179

DYNAMIC AUTOIMMUNE THYROIDITIS IN MYELODYSPLASTIC SYNDROME: FROM PAINLESS THYROTOXICOSIS TO OVERT HYPOTHYROIDISM

Dekritiana Dian Pratiwi[1] **and Hemi Sinorita**[1]
[1]*Department of Internal Medicine, Faculty of Medicine, Public Health and Nursing Gadjah Mada University/ Dr. Sardjito Hospital, Indonesia*

INTRODUCTION

Autoimmune thyroid disease can follow a triphasic course: thyrotoxicosis, a transient euthyroid period, and eventual hypothyroidism, and may coexist with myelodysplastic syndrome (MDS) in the context of immune dysregulation.

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

Suprhamanyam Evali[1], Siew Huang Lee[2], Karen Christelle[3], Anilah Abdul Rahim[4]
[1]*Endocrinology Unit, Hospital Putrajaya*
[2]*Pharmacy Department, Hospital Kuala Kangsar*
[3]*Family Medicine, Klinik Kesihatan Felda Bersia*
[4]*Endocrinology Department, Hospital Raja Permaisuri Bainun*

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

Fang Chan Lim[1]
[1]*Department of Internal Medicine, Hospital Tawau*

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.