cord palsy. Investigations revealed markedly elevated creatine kinase (7,950 U/L), aspartate aminotransferase (349 U/L), and alanine aminotransferase (179 U/L), with euthyroid biochemistry (thyroid-stimulating hormone 5.21 mIU/L, free thyroxine 4 16.6 pmol/L). Hypokalemia correction failed to improve symptoms, excluding periodic paralysis. ANA, C3, and C4 were normal. Myositis panel showed strong anti-Cytosolic 5′-nucleotidase 1A positivity with borderline anti-Ro-52. A diagnosis of IIM with bulbar involvement was made. She was treated with intravenous methylprednisolone and intravenous immunoglobulin, with clinical improvement.

### CONCLUSION

This case highlights the diagnostic challenge of muscle weakness in thyroid disease. While thyrotoxic myopathy is often presumed, markedly elevated creatinine kinase, rash, and bulbar involvement should prompt suspicion of IIM. Early immunosuppressive therapy is essential to achieve favorable outcomes.

## EP_A159

## GRAVES' DISEASE PRESENTING WITH PANCYTOPENIA: A RARE REVERSIBLE HEMATOLOGICAL ABNORMALITY IN THYROTOXICOSIS

**Wei Ton Wong[1], Che Azzah Hanim Che Yahya[1], Nurul Atikah Abdul Aziz[1]**
[1]*Internal Medicine Unit, Hospital Besut*

### INTRODUCTION

Graves' disease is associated with various hematological abnormalities, including anemia, leucopenia, and thrombocytopenia. However, pancytopenia involving all three cell lines is a rare and often under-recognized manifestation of thyrotoxicosis. We present a case of newly diagnosed Graves' disease complicated by pancytopenia, in which cell counts normalized rapidly following carbimazole initiation.

### CASE

A 51-year-old female with underlying type 2 diabetes mellitus, hypertension, and dyslipidemia presented with dysphagia for 3 months, significant weight loss (from 95 to 75 kg over 6 months), and a 1-week history of fever, palpitations, tremors, orthopnea, and bilateral lower limb swelling. On examination, she was febrile (38.2°C) with bibasal crepitations, pitting edema up to the mid-shins, bilateral hand tremors, and a multinodular neck mass moving with deglutition. Investigations confirmed thyrotoxicosis (thyroid-stimulating hormone [TSH] 0.01 mIU/L, free T4 130.1 pmol/L, T3 >30.8 pmol/L) with positive autoantibodies (thyroid receptor antibody 31.9 IU/L, anti-thyroid peroxidase 195 IU/mL). Full blood count showed pancytopenia: white cell count 2.73 × $10^9$/L, hemoglobin 10.3 g/dL, and platelets 108 × $10^9$/L. Peripheral blood film suggested normocytic normochromic anemia with leucopenia and thrombocytopenia. Chest radiography showed cardiomegaly with fluid overload, and echocardiography revealed an ejection fraction of 77%. She was treated for impending thyroid storm secondary to pneumonia with Lugol's iodine, hydrocortisone, propylthiouracil, and intravenous antibiotics. She was discharged on day 3 with a transition to carbimazole. At outpatient follow-up approximately 9 days later, thyroid function had improved significantly (free thyroxine 4 reduced from 130.1 to 29.34 pmol/L with suppressed TSH), and repeat full blood count demonstrated complete normalization of all three cell lines.

### CONCLUSION

This case illustrates that pancytopenia can be a direct consequence of severe thyrotoxicosis and may reverse completely with effective antithyroid therapy. The temporal relationship between biochemical improvement and hematological recovery supports a causal link. Clinicians should be aware of this rare association to avoid misdiagnosis and unnecessary invasive investigations.

## EP_A160

## A COSTLY ASSUMPTION: MISINTERPRETATION OF THYROID FUNCTION TESTS DELAYING GUILLAIN-BARRÉ SYNDROME DIAGNOSIS IN PREGNANCY

**Chen Huey Ng[1] and Wei Ton Wong[2]**
[1]*Hospital Sultan Ismail Petra*
[2]*Hospital Besut*

### INTRODUCTION

A common diagnostic error is the tendency to attribute new symptoms directly to the most obvious laboratory abnormality. In pregnancy, a suppressed thyroid-stimulating hormone (TSH) with elevated free T4 is frequently presumed to indicate primary hyperthyroidism, overlooking the possibility of benign gestational transient thyrotoxicosis (GTT). We report a case where this pattern led to the misattribution of acute flaccid paralysis to thyrotoxic periodic paralysis, critically delaying the diagnosis of Guillain-Barré syndrome (GBS).

**CASE**

A 21-year-old female Malay primigravida at 19 weeks and 5 days presented with a 2-week history of progressive, descending bilateral lower limb weakness culminating in paralysis, associated with vomiting and 5 kg weight loss. On admission, she was febrile (38.0°C) and tachycardic (150 bpm). Neurological examination revealed proximal-predominant flaccid paralysis and hyporeflexia with intact sensation, without thyroid eye signs or goiter. Thyroid function tests showed profound thyrotoxicosis (TSH <0.005 mIU/L, free thyroxine 4 20.16 pmol/L) with hypokalemia (2.9 mmol/L). A neck ultrasound was normal. A provisional diagnosis of thyrotoxic periodic paralysis with impending storm was made, leading to treatment with potassium replacement, propylthiouracil, and hydrocortisone. Despite biochemical improvement, her paralysis persisted. On day 5, she developed acute bulbar palsy and respiratory failure requiring intubation. A subsequent comprehensive workup for infectious, autoimmune (including thyroid antibodies), and nutritional causes was unremarkable. Nerve conduction studies confirmed the acute motor axonal neuropathy (AMAN) variant of GBS. Treatment with a 5-day course of intravenous immunoglobulin resulted in neurological improvement and successful extubation.

**CONCLUSION**

This case highlights the critical pitfall of prematurely attributing acute neurological deficits to abnormal thyroid function tests in pregnancy. Biochemical thyrotoxicosis, including GTT, should not preclude urgent evaluation for life-threatening neurological conditions such as GBS, particularly when weakness is progressive or refractory to metabolic correction.

## EP_A161

## BETA BLOCKER-INDUCED RAYNAUD PHENOMENON IN A CASE OF GRAVES' DISEASE

**Lui Tjun Yew[1] and Gerard Jason Mathews[1]**
*[1]Hospital Canselor Tuanku Muhriz UKM*

**INTRODUCTION**

Beta-blockers are frequently used as adjunctive therapy in hyperthyroidism to manage adrenergic symptoms, yet their potential to precipitate secondary Raynaud's phenomenon remains under-recognized. This adverse effect is attributed to β2-adrenoceptor blockade, which impairs peripheral vasodilation and promotes reflex vasoconstriction. Non-selective agents such as propranolol carry this particular risk.

**CASE**

A 22-year-old female presented with a 6-month history of palpitations, 11 kg weight loss, and fine tremors. There was no personal or family history of autoimmune or connective tissue disease. She denied any malar rash, joint swelling, alopecia, myopathy, oral ulcers, or uveitis. She also denied using any supplements or any herbal or traditional remedies. Examination revealed tachycardia, fine tremors, and Graves' ophthalmopathy, but no acropachy or organomegaly. Investigations confirmed Graves' disease: Free T4 of 73 pmol/L, thyroid-stimulating hormone <0.05 mIU/L, with elevated anti-thyroid peroxidase and anti-thyroglobulin antibodies. Full blood count, renal, and liver function were unremarkable.

She was commenced on carbimazole 20 mg daily and propranolol 40 mg three times daily. After 8 months of treatment, she developed cold-induced digital color changes and skin tightening consistently, suggestive of Raynaud's phenomenon. Dose reduction of propranolol to 20 mg BD only yielded partial improvement. Propranolol was subsequently substituted with verapamil, a non-dihydropyridine calcium channel blocker, resulting in significant resolution of Raynaud's phenomenon.

**CONCLUSION**

Propranolol-induced Raynaud's phenomenon is an under-recognized complication in the management of Graves' disease, and this case illustrates that it may develop insidiously after months of therapy and can persist despite dose reduction. Substitution with verapamil achieves rate control without β2-adrenergic antagonism. Clinicians should maintain a low threshold for recognizing this adverse effect and consider early substitution with verapamil in affected patients. While verapamil does not confer the same adrenergic blockade as propranolol, its vasodilatory properties make it a rational and effective alternative in this context.