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.

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

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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.