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.

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

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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

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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).