negative serology and the absence of a family history, along with a hyperfunctional state on imaging, likely suggests a rare presentation of sporadic, non-autoimmune, non-familial form of thyrotoxicosis. Following the initiation of anti-thyroid therapy, the CHB completely resolved without the need for further cardiological intervention. He achieved a complete clinical remission after a few months and is being planned for radioactive iodine therapy.

CONCLUSION

Most of the thyrotoxicosis-associated CHB reported in the literature was due to Graves' disease or some forms of autoimmune thyrotoxicosis, with auto-antibodies and activated lymphocytes having a role in the pathogenesis of the CHB. CHB in association with a non-autoimmune, non-familial form of thyrotoxicosis is indeed rare, and as this case illustrates, it completely went into spontaneous sinus rhythm upon initiation of conventional anti-thyroid therapy. This rare presentation of CHB is believed to have a benign clinical course.

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STEROID-RESPONSIVE ENCEPHALOPATHY ASSOCIATED WITH THYROIDITIS

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INTRODUCTION

Steroid-responsive encephalopathy associated with thyroiditis (STREAT) is a rare clinical entity. Clinical presentations of STREAT can range from a stroke-like presentation to psychiatric symptoms. STREAT is diagnosed based on four major criteria which include altered cognitive function, new or worsening psychiatric symptoms, elevated antithyroid antibodies, and exclusion of infectious, toxic, metabolic, or neoplastic causes. Corticosteroids are the cornerstone of treatment, with reported excellent response in neurocognitive symptoms resolution.

CASE

This was a case of a 48-year-old male with Graves's disease who underwent radioiodine therapy 4 months earlier and was started on levothyroxine 100 mcg 2 weeks prior to presentation. He presented with 4 days of behavioral symptoms, irritable mood with auditory and visual hallucinations. There was no history of fever, headache, limb weakness, or seizure. On general appearance, the patient was agitated, talking incoherently, and disorientated with normal vitals. There was no delayed relaxation of the reflex. The examination of the cranial nerve, motor, sensory, and cerebellar system was normal. A lumbar puncture showed normal opening pressure with a high protein CSF content of 1,596 mg/L. The thyroid-stimulating hormone (TSH) was elevated at 72.7 uIU/mL and T4 at 9.56 pmol/L. Serum anti-thyroid peroxidase was elevated at 877.42 IU/mL, and anti-TSH receptor antibody at 21.30 IU/L. CSF oligonal band, serum aquaporin-4, and viral screening were negative; non-contrasted computed tomography brain revealed normal findings. Based on the clinical history and examination, a diagnosis of STREAT was made at the emergency department. The patient was initiated on hydrocortisone 100 mg three times a day. Within 24–48 hours of steroid therapy, marked improvement and subsequent resolution of the mental status and behavioral symptoms were seen.

CONCLUSION

Hashimoto's thyroiditis can rarely lead to STREAT, a condition with diverse neurological manifestations, where early recognition and prompt corticosteroid therapy are essential for favorable outcomes.

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MUSCLE WEAKNESS IN THYROID DISEASE: WHEN IT IS NOT THYROTOXIC MYOPATHY?

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INTRODUCTION

Muscle weakness in thyroid disease is commonly attributed to thyrotoxic myopathy or hypokalemic periodic paralysis. Nevertheless, autoimmune conditions such as idiopathic inflammatory myopathies (IIM) and myasthenia gravis (MG) should be considered, as they may coexist with Graves' disease.

CASE

A 54-year-old female with hypertension and Graves' disease, treated with carbimazole for 2 years, had her therapy discontinued after remission. She was restarted on low-dose carbimazole following symptom recurrence. Three weeks later, she developed progressive proximal weakness, dysphagia, hoarseness, anorexia, and weight loss. On examination, body mass index was 22 kg/m^2 with mild proptosis, symmetrical proximal weakness (MRC 4/5), and erythematous rashes on thighs and shins. Otorhinolaryngology evaluation confirmed bilateral vocal

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.

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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).