On examination, she appeared cachectic. She had a blood pressure of 137/72 mmHg and a heart rate of 120 bpm. Thyroid function tests showed severe hyperthyroidism with TSH <0.01 m IU/L and FT4 100 pmol/L. She had elevated TSH receptor antibodies of 32.7 IU/L. Her abdominal CT revealed a grossly distended stomach filled with oral contrast and significant narrowing at the D4 level of the duodenum. She was diagnosed with SMA syndrome secondary to Graves' disease. Hence, she was treated with nasogastric intubation for gastric decompression, total parenteral nutrition, antiemetic, PTU per rectal, Lugol's iodine and intravenous propranolol to control her thyrotoxicosis. Despite conservative treatment and normalisation of FT4 level, the patient had persistent symptoms hence she underwent exploratory laparotomy and duodenal kocherisation. Postoperatively, her symptoms improved. She was able to resume a normal diet and continued to gain weight appropriately.

CONCLUSION

This case highlights the importance of considering SMA syndrome in patients with Graves' disease presenting with gastrointestinal symptoms and rapid weight loss. Prompt treatment of thyrotoxicosis alongside nutritional optimization and duodenal obstruction relief by conservative or surgical management is equally crucial.

EP_A180

NAVIGATING THE CONUNDRUM: ACUTE LIVER FAILURE IN HYPERTHYROIDISM AND THE TREATMENT DILEMMA

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INTRODUCTION/BACKGROUND

Hyperthyroidism is a complex endocrine disorder associated with various systemic manifestations. Liver dysfunction in hyperthyroidism is a relatively rare but potentially serious complication. We present a case of a patient with hyperthyroidism who initially received inadequate treatment and subsequently developed acute liver failure. The causative role of hyperthyroidism itself versus antithyroid medication-induced liver injury remains elusive, posing a therapeutic challenge. A comprehensive review of the patient's medical records, laboratory findings, imaging studies, and clinical progress was undertaken. Additionally, relevant literature concerning liver dysfunction associated with hyperthyroidism and drug-induced liver injury was explored.

CASE

Two months after initiating carbimazole therapy, a 42-year-old male with a history of hyperthyroidism presented with jaundice. Subsequent liver function tests indicated significant conjugated hyperbilirubinemia, accompanied by abnormalities in prothrombin time, development of hepatorenal syndrome, and encephalopathy. Imaging studies detected no structural abnormalities. Despite thorough evaluation, the exact cause of his liver failure remained elusive, posing challenges in distinguishing between exacerbation of hyperthyroidism and carbimazole-induced hepatotoxicity. Close monitoring ensued, with consideration given to liver transplant if necessary. Discontinuation of carbimazole and initiation of Lugol's iodine and cholestyramine led to clinical improvement. Radioactive iodine therapy was planned as the definitive treatment.

CONCLUSION

While acute liver failure in Graves' disease is rare, its management poses significant hurdles. Despite cholestasis and liver dysfunction, meticulous methimazole administration can effectively control hyperthyroidism with careful monitoring. However, when the cause of liver injury remains elusive—whether from the disease itself or its treatment—crafting an appropriate management plan becomes particularly complex. A different treatment approach may be necessary to achieve euthyroid state, often necessitating definitive therapy in such cases.

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BATTLE OF AUTO-IMMUNITIES: GRAVES' DISEASE AND RHEUMATOID ARTHRITIS: A BIDIRECTIONAL CAUSAL EFFECT

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INTRODUCTION/BACKGROUND

Graves' disease is an autoimmune disorder characterized by hyperthyroidism secondary to circulating thyroid autoantibodies. Co-existence with other autoimmune diseases such as vitiligo, chronic autoimmune gastritis and rheumatoid arthritis (RA) have been reported. We report a patient who developed RA more than 10 years following her diagnosis of Graves' disease.