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.

EP_A181

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.

CASE
A 37-year-old female with Graves' disease diagnosed at age 22 years presented with a 2 to 3-week history of multiple joint pains and morning stiffness involving both knees, ankles, elbows, and metacarpophalangeal joints (MCPJs). She did not have any preceding trauma, fever, genitourinary or gastrointestinal infections. There were no sicca symptoms, rashes, oral ulcers or constitutional symptoms. Examination revealed right knee and multiple MCPJ synovitis. She had a small diffuse goitre and no exophthalmos. Inflammatory markers were elevated with an ESR of 70 mm/H and CRP of 21.6 mg/L. She had mild hypochromic microcytic anaemia (Hb11.8 g/dL) and lymphopenia (1.3 103/uL). Rheumatoid factor and anti-Ro60 were both positive. Uric acid (277 umol/L) and FT4 (21.07 pmol/L) levels were normal. Knee joint aspirate culture and AFB were negative. Joint x-rays were unremarkable. Thyroid ultrasound showed enlarged thyroid lobes with coarse echotexture and increased vascularity. A rheumatology consult was obtained, and she was diagnosed with RA. She commenced on methotrexate and oral steroids which resulted in marked improvement in her joint condition. Her fT4 remained stable on low dose carbimazole.

CONCLUSION
Graves' disease is associated with an increased risk of RA and vice versa. Hence, it is important to screen patients with inflammatory joint pain for RA to ensure prompt management and prevent long-term joint damage and other complications.

EP_A182

A CASE OF MASSIVE PERICARDIAL EFFUSION IN SUBCLINICAL HYPOTHYROIDISM

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INTRODUCTION/BACKGROUND
Hypothyroidism is associated with multiorgan involvement and various complications. Pericardial effusion is a rare complication of hypothyroidism. However, if left untreated, it may progress to critical, life-threatening conditions such as cardiac tamponade and hemodynamic instability. Early identification of the diagnosis, with effective management of pericardial effusion in hypothyroidism, is essential.

CASE
A 67-year-old female with hypothyroidism since 2016 presented with worsening exertional dyspnoea, bilateral lower limb swelling, and fatigue. She had a background history of hypertension and bronchial asthma. She had elevated jugular venous pressure, but no muffled heart sounds. Her ECG showed small-voltage QRS complexes, and chest X-ray revealed cardiomegaly with pulmonary congestion. Her echocardiography showed a large pericardial effusion with a collapse of the right ventricle. An urgent pericardiocentesis was performed, and her symptoms improved after draining 500 cc of pericardial fluid. TFT showed elevated TSH (83.42 m IU/L) with normal free T4 (13.5 pmol/L). She had markedly elevated anti-thyroid peroxidase (>600 IU/mL) and anti-thyroglobulin (>4000 IU/mL). Her pericardial fluid investigations were unremarkable. The patient has been taking her levothyroxine inconsistently with her meals. Her levothyroxine dose was increased from 100 mcg to 150 mcg daily. She showed improvement by the third day of hospitalisation. She was discharged and advised to adhere to the levothyroxine. Her subsequent TFTs normalised with normal echocardiography during the follow-up visit.

Hypothyroidism causes protein-rich pericardial effusion due to increased membrane permeability, increased albumin distribution volume, and diminished lymphatic drainage, which happens gradually over time.

CONCLUSION
Pericardial effusion in hypothyroidism is an infrequent entity. It is more frequent in long-standing clinical hypothyroidism than subclinical hypothyroidism. An early cardiac assessment, adequate thyroid replacement therapy, and medication adherence can help mitigate the risk of pericardial effusion or cardiac tamponade.

EP_A183

SEVERE HYPOTHYROIDISM IN CHRONIC HEPATITIS C INFECTION: A QUANDARY OF AUTOIMMUNITY VERSUS ANTIVIRAL THERAPY

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INTRODUCTION/BACKGROUND
Chronic Hepatitis C virus infection (HCV) may have extrahepatic manifestations, mainly related to autoimmune and malignant disorders. Autoimmune thyroid disease (AITD) may present in up to 10% of cases as influenced by the presence of autoantibodies as well as direct virus invasion that promotes T-cell mediated cytotoxicity.