

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.