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

https://doi.org/10.15605/jafes.039.S1.193

Sarojini Devi, Yit Hern Chen, Siow Ping Lee
Department of Internal Medicine, Hospital Melaka, Jalan Mufti Haji Khalil, Melaka, Malaysia

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

https://doi.org/10.15605/jafes.039.S1.194

Mas Suria,[1] Abdullah Shamshir AM,[2] Md Syazwan MA,[1] Goh KG[1]
[1]Medical Department, Hospital Tengku Ampuan Afzan, Kuantan, Malaysia
[2]Endocrinology Unit, University Malaya Medical Centre, Kuala Lumpur, Malaysia

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.

Besides host factor, interferon previously used as a standard treatment in HCV, is known to cause thyroid dysfunction either by direct inhibitory effect on the thyroid gland or immune activation particularly in those with genetic predisposition to autoimmune disease

Direct-acting Antivirals (DAA) targeting specific non-structural proteins of the virus, hinders viral replication. Since it was introduced, there are few studies demonstrating the effect of DAA on thyroid dysfunction.

We report a case of new-onset hypothyroidism in a patient with HCV soon after commencement of DAA.

CASE

A 53-year-old male, former intravenous drug user with Chronic Hepatitis C Child Pugh A, was treated with DAAs (sofosbuvir and daclatasvir) and ribavirin based on viral load and genotyping. He denied preceding hypothyroid symptoms or family history of thyroid disorder. Two months into treatment, he complained of facial puffiness, weight gain and was eventually admitted for heart failure. Biochemical investigations revealed overt hypothyroidism with FT4 <3.2 pmol/L (7.86 - 14.41) and TSH >300 uIU/mL (0.38 - 5.33) with positive thyroid peroxidase antibody. Levothyroxine was started subsequently, however due to poor compliance, he showed poor clinical and biochemical response in HCV viral suppression and thyroid disorder.

CONCLUSION

Hypothyroidism related to HCV infection is a relatively uncommon association but an important one to diagnose, nonetheless. The condition can be part of the extrahepatic viral manifestation or may be treatment related. Supported by previous study that demonstrated similar effect of DAA on the thyroid gland, further larger RCTs are needed to substantiate this association.

EP_A184

CHALLENGES IN THE DIAGNOSIS AND DIFFERENTIATION OF THYROID HORMONE RESISTANCE FROM TSHOMA

https://doi.org/10.15605/jafes.039.S1.195

Shu Xian Lai and Eunice Yi Chwen Lau
Endocrine Unit, Department of Medicine, Sibu Hospital, Sarawak, Malasyia

INTRODUCTION

An elevated fT4 with non-suppressed TSH levels may present as a diagnostic challenge resulting in inappropriate treatment. Although rare, resistance to thyroid hormone (RTH) and TSHomas can present in this manner and diagnosis is important to guide management.

CASE

A 43-year-old male was referred from a district hospital for evaluation of atypical chest pain. He was noted to have an elevated fT4 with normal TSH for the past four years and had received carbimazole previously. He reported symptoms of hyperthyroidism including intermittent palpitations, tremor and anxiety. No goitre was noted clinically. Both his mother and maternal aunt had undergone thyroid surgery. After stopping treatment, he had elevated fT4 (28.31 and 19.39 pmol/L) and normal TSH (1.55 and 1.619 m IU/L) performed on two different platforms. Sex hormone binding globulin (22.6 nmol/L), alpha-subunit (0.22 IU/L) and neck ultrasound were normal. Pituitary MRI showed an ill-defined hypo-enhancing nodule measuring 2.0 mm x 2.2 mm x 1.9 mm. Other pituitary hormones were unremarkable. He went on to have a thyrotropin stimulation test which showed an exaggerated TSH response with an 11-fold increase at 20 minutes, supporting the diagnosis of RTH. Genetic testing was not performed due to resource limitations. Subsequently, he was managed symptomatically with beta blockers.

CONCLUSION

When managing discordant thyroid function tests, a high index of suspicion and proper clinical assessment, including laboratory and imaging studies, are needed to ensure precise diagnosis and avoid potentially harmful or unnecessary treatment such as radioactive iodine, anti-thyroid medication or pituitary surgery. Small non-functioning pituitary adenomas are not uncommon. Abnormal imaging needs to be correlated carefully.

EP_A185

UNVEILING PRETIBIAL MYXEDEMA: A CASE REPORT OF GRAVES' DISEASE

https://doi.org/10.15605/jafes.039.S1.196

Uswaton Hasanah Hashim, Jen Hoong Oon, Noor Lita Adam
Department of Medicine, Hospital Tuanku Ja'afar, Seremban, Negeri Sembilan, Malaysia

INTRODUCTION

Pretibial myxedema (PTM), a rare manifestation of Graves' disease, holds importance as it constitutes a component of the classical triad associated with the condition. Historically observed in up to 5% of Graves' disease patients, the incidence of pretibial myxedema has notably decreased, likely attributed to advancements in early diagnosis and prompt initiation of antithyroid therapy.