CONCLUSION
Thyroid dermopathy can present in an atypical manner, hence, physicians should be aware of this. Treatment of hyperthyroidism may not have any significant effect on the cutaneous lesions. PM may occur even after successful control of the disease.

EP_A174

NEUROLOGIC MANIFESTATION AND PERICARDIAL EFFUSION UNVEILING AN AUTOIMMUNE HYPOTHYROIDISM

https://doi.org/10.15605/jafes.039.S1.185

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INTRODUCTION/BACKGROUND
Autoimmune hypothyroidism is an antibody-mediated chronic inflammatory process. Thyroid destruction may be intermittent. Given its chronic and progressive nature, the diagnosis is often challenging since the exhibited signs and symptoms are often subtle and non-specific. We report a middle-aged male with bilateral upper and lower limb weakness and pericardial effusion. Investigation led to a diagnosis of autoimmune hypothyroidism.

CASE
A 64-year-old male presented with a two-week history of lethargy, poor appetite, and lower limb swelling. Initially, he was treated for pneumonia and cardiac failure due to chest radiography showing obscured cardio-phrenic angle. Further history revealed he had inability to walk for one year due to bilateral lower limb weakness. He had proximal muscle weakness of all four limbs. Sensation and reflexes were preserved in the upper limbs but absent in the lower limbs. His nerve conduction study and electromyography revealed myopathic changes involving all four extremities with absent neurosensory responses in both lower extremities. His cranial CT scan showed bifronto-temporal subdural effusion while his echocardiography exhibited pericardial effusion with cardiomegaly. His thyroid function tests revealed profound hypothyroidism (TSH>100, fT4 <1). Together with the presence of markedly raised anti-TPO antibodies, he was diagnosed to have autoimmune primary hypothyroidism. He also had normocytic normochromic anaemia and hypercholesterolemia consistent with severe hypothyroidism. He was eventually started on oral L-thyroxine (1.6 mcg/kg/day).

CONCLUSION
This case report highlights the potential for severe neuromuscular and cardiovascular consequences due to untreated chronic autoimmune hypothyroidism. Thyroid dysfunction is a consideration in a patient with neurologic manifestation. Early diagnosis and prompt treatment of hypothyroidism can potentially avert long-term hypothyroid sequelae.

EP_A175

THYROID FUNCTION ABNORMALITIES: CONNECTING THE DOTS BETWEEN GENETICS AND CLINICAL PRESENTATION

https://doi.org/10.15605/jafes.039.S1.186

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INTRODUCTION/BACKGROUND
Subclinical hyperthyroidism presents with low or undetectable levels of thyroid stimulating hormone (TSH), alongside normal levels of free thyroid hormones (fT4 and fT3). While certain individuals may not show any symptoms, others may experience hyperthyroid symptoms like palpitations, weight loss, and heat intolerance. Early recognition and prompt appropriate management are crucial to prevent potential complications, including atrial fibrillation, osteoporosis, and progression to overt hyperthyroidism.

CASE
A 59-year-old female was referred to our endocrine clinic due to abnormal thyroid function tests (TFTs) revealing subclinical hyperthyroidism. She was asymptomatic.

Her thyroid function tests 5 years ago showed similar results, however, she had not received proper consultation or treatment during that time.

She has no significant medical history. She has been in menopause since 50 years old with regular menses before that. She had five pregnancies, four of which were preterm, with her eldest child having cerebral palsy and her fourth child deceased due to prematurity. Two of her children have hyperthyroidism. One son is on carbimazole while one daughter has subclinical hyperthyroidism.

A repeat thyroid function test still showed suppressed TSH level of 0.04 m IU/L and normal FT4 level of 13.2 pmol/L and free triiodothyronine (fT3) level of 4.3 pmol/L. Molecular studies showed polymorphism of exon 3 of the TSHR gene from the son.

CONCLUSION
In conclusion, this case report emphasized the need for thorough evaluation and appropriate management of abnormal thyroid function tests, particularly in the presence of familial clustering. Early recognition and treatment can prevent potential complications and improve patient outcomes. Additionally, the potential role of genetic factors, such as polymorphisms in exon 3 of the TSHR gene, should be considered in cases of familial clustering of thyroid disorders. Genetic testing and clinical correlation may be necessary for a comprehensive assessment and management of thyroid disorders associated with genetic polymorphisms.

EP_A176

MASSIVE PERICARDIAL EFFUSION AS A PRIMARY MANIFESTATION OF HYPOTHYROIDISM

https://doi.org/10.15605/jafes.039.S1.187

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INTRODUCTION/BACKGROUND
Hypothyroidism is an endocrine disorder with multiorgan involvement and various complications. Mild pericardial effusion is a common cardiovascular complication but massive pericardial effusion with cardiac tamponade as initial presentation of hypothyroidism is rare.

CASE
We report a 70-year-old female with a history of hyperthyroidism who was treated with radioiodine ablation more than 20 years ago. She defaulted follow-up and hence was not on L-thyroxine. She presented with progressive exertional dyspnoea and hypothyroid symptoms (weight gain, fatigue, cold intolerance) for a month. On examination, she had coarse dry skin, periorbital oedema, and bradycardia. She was normotensive. Her heart sounds were not muffled. Biochemically she was in overt hypothyroidism, TSH 16.825 m IU/L (0.35-4.94), T4 <5.41 pmol/L (9.01-19.05). She also had hyponatremia with a sodium level of 118-125 mmol/L and hyperlipidaemia. She had cardiomegaly on a chest x-ray. Her electrocardiogram showed normal voltage complexes with no electrical alternans. Her echocardiography showed massive pericardial effusion (3.1 cm) with a collapsible right atrium. She had normal ventricular function. Pericardiocentesis was performed and 150 cc straw-coloured fluid was aspirated. The pericardial fluid was exudative. Cultures were negative for bacteria and acid-fast bacilli. There were no malignant cells. She was treated with L-thyroxine 75 mcg daily. TFTs repeated six weeks later were already normal with TSH of 2.521 m IU/L (0.35-4.94) and T4 of 12.76 pmol/L (9.01-19.05). Repeat echocardiography showed resolution of the pericardial effusion. Clinically, she remained asymptomatic.

CONCLUSION
Although massive pericardial effusion is an uncommon initial presentation of hypothyroidism, it can occur in long-standing untreated cases. Pericardial effusion can resolve with adequate thyroid hormone replacement therapy.

EP_A177

VANISHING THYROID NODULES: SUBACUTE THYROIDITIS MIMICKING SUSPICIOUS THYROID NODULES IN A PATIENT ON TYROSINE KINASE INHIBITOR

https://doi.org/10.15605/jafes.039.S1.188

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INTRODUCTION/BACKGROUND
Dasatinib is a tyrosine kinase inhibitor (TKI) used as a second-line treatment for chronic myeloid leukaemia. Thyroid dysfunction is rare with dasatinib. We report a patient with chronic myeloid leukaemia on Dasatinib who developed subacute thyroiditis mimicking a suspicious thyroid nodular disease.

CASE
A 57-year-old female was started on dasatinib in June 2021. She presented with a one-month history of fever, palpitations, heat intolerance, and neck swelling in April 2023. Her thyroid function tests (TFTs) showed elevated free-T4 30.9 pmol/ and suppressed thyroid stimulating hormone (TSH), <0.008 m IU/L, hence, carbimazole 20 mg daily was initiated. Thyroid ultrasound revealed hypoechoic solid nodules at both upper poles, measuring 1.7 x 2.1 x 4.7 cm and 1.7 x 2.0 x 3.4 cm, respectively. Both nodules had TIRADS scores of 5. Another hypoechoic solid nodule with a TIRADS score of 4 was also found at the right mid-pole. However, during the scheduled ultrasound-guided fine needle biopsy two months later, the repeat ultrasound no longer showed any thyroid nodule. TSH-receptor antibody was negative. Her thyroid function normalised and her carbimazole dose was tapered off after 2 months of treatment. Repeat neck ultrasound six months later demonstrated a normal thyroid gland. The subsequent serial TFTs remained normal. Dasatinib was continued throughout this period.

TKI-induced thyroid abnormality usually appears within the first 6 months but can still manifest after the first year of treatment. Ultrasound descriptions of subacute thyroiditis