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

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Shu May Chong and Lay Ang Lim
Hospital Seberang Jaya, Malaysia

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.

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

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Adilah Zulaikha Abd Latib,[1] Yik Zhi Kum,[1] Siti Nabilah Atiqah Othman,[1] Ooi Chuan Ng[2]
[1]*Department of Medicine, Hospital Sultan Abdul Aziz Shah, Universiti Putra Malaysia*
[2]*Faculty of Medicine and Health Sciences, University Putra Malaysia*

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.