

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**

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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**

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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