Massive pericardial effusion in primary hypothyroidism

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Summary

A 44 year old lady with primary hypothyroidism presented with massive pericardial effusion without cardiac temponade. Pericardial tap was done twice and the effusion resolved as the hypothyroid state improved. She remained hypertensive despite the euthyroid state. She was discharged well with L-thyroxine and anti-hypertensive therapy.

Key words: Hypothryoid, pericardial effusion, hypertension, hyperprolactinaemia.

Introduction

The clinical manifestations of hypothyroidism are highly variable, depending on its cause, duration and severity. Cardiovascular dysfunction includes bradycardia, poor peripheral circulation, fatigability and decreased exercise tolerance. Occasionally, hypothyroid cardiomyopathy or pericardial effucison may be seen. Pericardial effusion has been reported to occur in 30% of patients with hypothyroidism.[1] Hypertension reversible by thyroxine may also be found.[2] The association of primary hypothyroidism with hyperprolactinemia has been well established with some patients presenting with galactorrhoea.[3]

The patient presented with several interesting but rare features of primary hypothyroidism. These include massive pericardial effusion, hypertension and galactorrhoea.

Case Report

A 44 years old lady with no previous history of thyroid disorder was admitted with symptoms of hypothyroidism. She had lethargy, constipation, cold intolerance and poor effort tolerance for the past 10 years. Bilateral expressible milky galactorrhea was noted 2 months prior to admission, associated with 3 years of secondary amenorrhoea. She also had anginal pain on exertion, but no symptoms of heart failure.

Physical examination revealed a lethargic lady with coarse voice and not in respiratory distress. There were no macroglossia, goitre or surgical scar. Her blood pressure on admission was 150/1000 mmHg and pulse rate 100/min, without pulsus paradoxus. Her lung was clear. There was no hepatosplenomegaly. She had marked prolongation of relaxation phase of ankle jerks and bilateral expressible galactorrhoea. There were no features of autoimmune or pituitary disease.

Biochemically she had primary hypothyroidism i.e. serum thyroxine (T4) < 39 nmol/L and thyrotrophin (TSH) 129.5 uiu/ml. Serum prolactin was 1954 mu/l (normal: 117–468 mu/L). Anti-thyroid antibodies were negative. The chest Xray showed massive globular cardiomegaly suggestive of massive pericardial effusion. The electrocardiogram (ECG) showed diffuse small voltage but no electrical alternans. There was no evidence of acute myocardial infarction. The 2 dimensional (2D) echocardiogram confirmed a large circumferential pericardial effusion, anteriorly and posteriorly with diastolic collapse of right ventrical suggesting pretemponade. CT Scan of the chest showed the thyroid gland in the neck with no retrosternal extension. The superior vena cava and inominate veins were grossly distended. Although her skull Xray showed enlarged pituitary fossa with double flooring, the CT Scan did not show evidence of pituitary tumour.

Pericardial tap was done and showed a thick "jelly-like" haemorrhagic effusion. The pericardial fluid was also cultured for acid-fast bacilli which was negative. She was treated with a low dose of L-thyroxine 0.05 mg daily which was gradually increased up to 0.2 mg daily. Four weeks later, she improved with serum T4 74 nmol/L and TSH 21.3 uiu/ml. Subsequent ECG, and chest Xray and 2D-echocardiogram showed marked improvement. The hypertension was well controlled with Verapramil 80 mg tds, and the serum prolactin was 638 mu/L. On discharge, she remained euthyroid on L-thyroxine 0.2 mg daily.

Discussion

This lady had typical clinical features of primary hypothyroidism of a long duration. The high TSH was suppresible with an elevation of the T4 level, indicating an intact negative feedback mechanism and that a pituitary feedback tumour had not developed. The TSH value would remain high if the pituitary secretion was autonomous as in adenoma. The CT Scan did not show any pituitary tumour.

Hypothyroidism with pericardial effusion has been well described. The study done by Hardisty et al showed that 30% of untreated hypothyroidism will develop pericardial effusion.[4] The basic pathology is accumulation of glycosaminoglycans – mostly hyaluronic acid, due to a decreased destruction rather than excessive synthesis.[6] Although hypothyroidism can cause massive pericardial effusion, it is rarely complicated with cardiac temponade.[5] This is due to slow formation of the pericardial effusion and the ability of the pericardium to distend. The effusion will resolve once the hypothyroid state improves with thyroxine therapy. [5,7,8] This lady was relatively comfortable at rest despite the massive pericardial effusion and features of pretemponade on the 2D-echocardiogram. It has been suggested that hypothyroidism should be considered in investigating idiopathic pericardial effusion and secondary temponade especially in elderly.[9]

Galactorrhoea is a rare feature of primary hypothyroidism. Her prolactin level was high (1954 uiu/ml). The hyperprolactinaemia could be due to stimulation of the lactotroph by the high thyrotrophin releasing hormone (TRH), reduced hypothalamic dopamine concentration and decreased prolactin clearance. The galactorrhea and hyperprolactinaemia usually resolved with thyroxine therapy.[3] In this lady, on discharge she still had minimal galactorrhea which could be due to her being subthyroid biochemically (Serum T4 74 nmol/L TSH 21.3 uiu/mL) even though she was clinically euthyroid.

Hypothyroidism is often listed as an endocrine cause of diastolic hypertension as compared to systolic hypertension of hyperthyroidism. The incidence is variously estimated as 50% to

75%.[10,11] The hypertension is secondary to sodium retention and reversible with thyroxine therapy. This lady has persistent hypertension despite euthyroid state. This could be due to concomittent essential hypertension.

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