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Case Report

A case of hypertensive emergency, primary hypothyroidism and large pericardial effusion with early tamponade

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ABSTRACT

Thyroid hormones and the cardiovascular system are strongly intertwined with known risk of coronary disease, atrial fibrillation, and cardiomyopathy. Pericardial effusions are commonly seen in cases of severe hypothyroidism, however large to massive pericardial effusions with cardiac tamponade are exceptionally rare. We report a case of a patient presenting with hypertensive emergency and a concomitant diagnosis of primary hypothyroidism with a large pericardial effusion and early echocardiographic features of tamponade. Following pericardiocentesis, hypertension management, and thyroid replacement therapy the patient's symptoms improved with no recurrence of pericardial effusion.

<Learning objective: Hypothyroidism is a common medical comorbidity with many clinical manifestations and cardiovascular effects including hypertension. Pericardial effusion is a known complication of hypothyroidism, however cases of massive effusion and tamponade are rare. Management of large effusions is unclear, with some patients treated with thyroid supplement and others requiring pericardiocentesis.>

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Introduction

Thyroid hormones are involved with both development and function of the cardiovascular system. Major effects of thyroid hormones are mediated by triiodothyronine (T3) that increases the force of systolic contraction and diastolic relaxation as well as decreases vascular resistance. Primary hypothyroidism is characterized by decreased levels of T4 and T3 with compensatory high levels of thyroid stimulating hormone (TSH). Most common cardiovascular manifestations include diastolic hypertension and sinus bradycardia. Other manifestations include pericarditis, dyslipidemia, myxomatous valvular changes, and pericardial effusion [1]. Hypothyroidism and pericardial effusion has an incidence of 3–6% in mild cases of hypothyroidism and up to 80% in severe hypothyroidism such as myxedema coma [2]. Large to massive pericardial effusion is infrequent with rare cases of tamponade. This is the first case report to our knowledge of a hypertensive emergency and large pericardial effusion presenting as primary hypothyroidism.

Case report

A male in his late 20s presented to emergency with a one month history of worsening intermittent blurred vision and was found to have a blood pressure of 224/140 mmHg with a temperature of 36.5 °C, heart rate of 62 bpm with no evidence of cardiac or respiratory distress. There was no jugular venous distension, pulsus paradoxus, or cardiac murmurs auscultated. Neurological examination was remarkable for bitemporal hemianopsia with fundoscopic examination revealing significant papilledema and hypertensive retinopathy. Clinical history did not have any concerning symptoms of overt hypothyroidism and he underwent emergent neuroimaging that showed no intra-cranial pathology. He was admitted to the internal medicine service and treated as a hypertensive emergency with an IV labetalol infusion with work-up for secondary causes of hypertension.

Electrocardiogram showed normal sinus rhythm with left ventricular hypertrophy (LVH) with repolarization changes (lateral T wave inversion leads I, aVL, V5, and V6) and QTc prolongation of 495 ms. There was no QRS alternans or low voltage (Fig. 1A). Echocardiography revealed a large circumferential pericardial effusion (36 mm) with diastolic compression of the right atrium and no right ventricle diastolic compression. There was blunted respiratory variation of the mitral valve and tricuspid valve inflow

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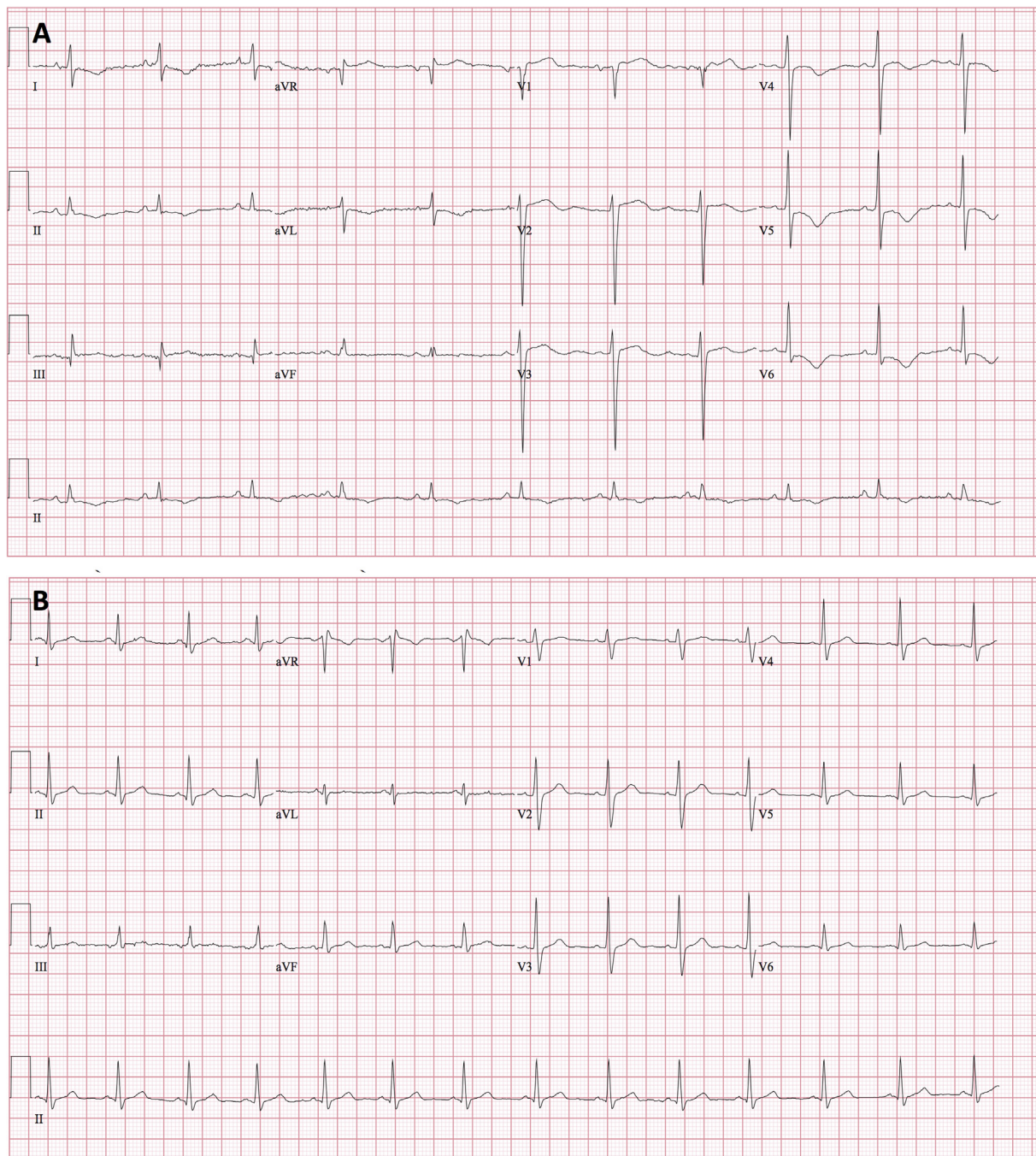


Fig. 1. (A) Electrocardiogram of patient upon presentation [heart rate (HR) 65 bpm, QTc of 495 ms]. (B) Electrocardiogram 10 months post pericardiocentesis and thyroid replacement (HR 81 bpm, QTc of 439 ms).

suggestive of increased pericardial pressure. Left ventricular systolic function was preserved with mild concentric LVH (Fig. 2). There was no pericardial pathology identified either on trans-thoracic echocardiography or computed tomography imaging (Figs. 2 and 3).

Laboratory work was remarkable for a TSH of 503.50 mIU/L (0.20–4.00 mIU/L) with a free T4 and T3 of 0.7 (10.0–25.0 pmol/L) and 1.0 pmol/L (3.5–6.5 pmol/L), respectively. Complete blood count, serum electrolytes, renal function, and liver chemistry were all within the normal limits. Ultrasound imaging of the thyroid and secondary causes of hypertension were unremarkable.

The patient was diagnosed with primary hypothyroidism and started on high-dose thyroid replacement. His blood pressure slowly improved with oral anti-hypertensive agents, however he began to develop worsening shortness of breath and repeat echocardiogram revealed early findings of cardiac tamponade (Fig. 2F–H). This prompted an echocardiography-guided pericardiocentesis with 750 cc of transudative straw-colored fluid with negative cytology and infectious cultures. He improved post pericardiocentesis and was discharged in stable condition on oral thyroid replacement therapy and anti-hypertensives.

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