



## A case of primary hypothyroidism initially presenting with massive pericardial effusion

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### ABSTRACT

While mild pericardial effusion generally accompanies overt hypothyroidism, massive pericardial effusion or pericardial tamponade is rare and more frequently occurs in patients with severe hypothyroidism. Similarly, massive pericardial effusion as the initial presenting feature of newly diagnosed hypothyroidism is also quite uncommon. A 64 year old diabetic (DM) male patient presented to our clinic with a recent worsening of shortness of breath. Transthoracic echocardiography demonstrated the presence of massive pericardial effusion and a pericardiocentesis was performed. Laboratory examination showed elevated thyroid stimulating hormone (TSH), and decreased free thyroxine (fT4) and free tri-iodothyronine (fT3) levels. A diagnosis of primary hypothyroidism was made. A diagnosis of hypothyroidism should be considered in the differential diagnosis of patients presenting with unexplained pericardial effusion, even in the absence of accompanying signs and symptoms of hypothyroidism.

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### Introduction

Although pericardial effusion is a common finding in patients with primary hypothyroidism, massive pericardial effusion or pericardial tamponade is rare. Pericardial effusion secondary to hypothyroidism may present diagnostic challenges due to the general discordance between the volume of effusion and clinical symptoms [1]. Herein, we describe a newly diagnosed case of primary hypothyroidism initially presenting with massive pericardial effusion.

### Case history

A 64 year old male patient presented to the emergency unit with worsening dyspnea over the past 1 month. He denied chest pain, fever, chills, diaphoresis or palpitations. His history was unremarkable except for type 2 diabetes. Blood pressure was 110/70 mmHg, respiratory rate was 20/min, pulse was 60 bpm, and the body temperature was 37.1 °C. The patient didn't have any symptoms of overt hypothyroidism including weight gain, fatigue, cold intolerance,

constipation, dry skin, edema, and muscular weakness. On physical examination assessments, there was no gallop or heart murmur, jugular vein engorgement, or reduced deep tendon reflexes.

ECG showed a low voltage QRS wave. Cardiac troponin I and CKMB levels were normal 0.009 ng/ml (0–0.06) and 12.66 U/l (0–25) respectively. Chest X-ray showed increased cardio-thoracic index in favor of the cardiac silhouette. No infiltration could be observed in the lung parenchyma. Echocardiographically ejection fraction was 60%. Due to the detection of pericardial fluid levels of 7 cm, 2 cm, 6 cm, and 2.6 cm at the adjacency of the posterior wall, right ventricle, lateral margin, and right atrium, respectively, a diagnosis of massive pericardial effusion was made. A pericardiocentesis was performed and a total of 2800 cc of fluid was evacuated. Complete blood count, electrolytes, renal and hepatic function tests, and erythrocyte sedimentation rate were normal. No bacterial growth was observed in pericardial fluid samples and cytological examination revealed no findings suggesting malignancy. Also, adenosine deaminase level was normal and no growth occurred in Lowenstein-Jensen medium. Rheumatoid factor, anti-nuclear antibody profile, and viral markers (HIV and HBsAg) were negative. Thyroid function tests were as follows: TSH > 150 mIU/l (N: 0.5–4.7), fT4 0.2 ng/dl (N: 0.8–1.7), fT3 1.2 pg/ml (N: 1.8–4.6 pg/ml), antiTPO negative and anti TG negative. A 3 × 3 × 3 mm cystic nodule was found in the R lobe on US examination. Consequently, a diagnosis of primary hypothyroidism was made and replacement treatment was initiated

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with 50 mcg/day of levothyroxine with gradual dose titration. fT4 level was checked again and detected as 0.7 ng/dl. Echocardiogram showed that the pericardial effusion had decreased considerably in size. The patient's condition improved and then he was discharged. Follow-up examination after 6 months showed normal TSH, fT4, and creatine kinase levels. EKG was normal, with no pericardial effusion detected by echocardiography.

## Discussion

Any defect in the hypothalamic–pituitary–thyroid axis may result in the development of hypothyroidism. Low serum free T4 accompanied by elevated TSH suggests a diagnosis of hypothyroidism and points out to the pathological conditions involving the thyroid gland. Overt hypothyroidism causes a number of symptoms including weight gain, fatigue, cold intolerance, constipation, dry skin, edema, muscular weakness, and reduced deep tendon reflexes.

Overt hypothyroidism is associated with some cardiovascular findings; for instance, increased systemic vascular resistance, decreased cardiac contractility, decreased cardiac output, atherosclerosis, coronary artery disease, bradycardia and conduction abnormalities. Another cardiac finding is pericardial effusion [2,3].

Pericardial effusion can be detected in approximately 25% of the hypothyroid patients [4]. Hypothyroidism leads to a decreased synthesis of albumin. Increased permeability of the capillaries results in the loss of albumin in the intravascular compartment through increased transcapillary escape rate of albumin, eventually leading to reduced plasma volume. Thus, the consequent increase in the concentration of the albumin in extravascular compartment causes increased interstitial fluid volume and impaired lymphatic drainage [5,6]. In a study involving rats with hypothyroidism, absence of thyroid hormones was associated with alterations in the structure of the interstitium, reduced interstitial compliance, and disturbance in transcapillary fluid balance [7].

The pericardium is composed of two layers, i.e. visceral and parietal, and normally contains approximately 10 to 50 ml of fluid, which is produced by the pericardium through ultrafiltration of plasma. The abovementioned pathological mechanisms cause an increase in the volume of pericardial fluid. Significant increase in pericardial fluid volume without obvious signs and symptoms has been reported to occur in patients with hypothyroidism [6]. Similarly, our patient presented with massive pericardial effusion without the common symptoms and signs of hypothyroidism. Pericardial tamponade is uncommon in these patients due to the elasticity of the pericardium and low accumulation rate of the pericardial fluid, obviating the need for pericardiocentesis in the majority of the patients. Also, pericardial effusion generally disappears within months of starting L-thyroxine. Cardiac tamponade is an indication for emergency pericardiocentesis [1,8,9].

Clinical triad of cardiac tamponade (Beck's triad) includes hypotension, distant heart sounds, and jugular vein engorgement. Our

patient lacked clinical signs of cardiac tamponade but moderate respiratory stress and worsening dyspnea might be present early in the tamponade physiology.

Because of the insidious and non-specific characteristics of the signs and symptoms of hypothyroidism along with rare occurrence of massive pericardial effusion in some patients, the possibility of hypothyroidism may be overlooked in the differential diagnosis of massive pericardial effusions. Since hypothyroidism represents a treatable cause of massive pericardial effusion, the diagnosis must be established and treatment be started promptly [10]. In patients with unexplained pericardial effusion, thyroid function tests and echocardiography should always be performed. After obtaining an euthyroid status with several months of treatment, pericardial effusion slowly disappears, preventing unnecessary pericardiocentesis in these patients [9].

Massive pericardial effusion is a rare complication of hypothyroidism and patients frequently exhibit other signs and symptoms of hypothyroidism before the development of pericardial effusion. However, in the patient described herein, initial presentation involved massive pericardial effusion associated with dyspnea. In patients with pericardial effusion even if no overt symptoms of hypothyroidism appear, differential diagnosis should include hypothyroidism. Also, it should be borne in mind that the presence of diabetes may further complicate the diagnosis due to neuropathy.

## Conflict of interest

The authors declare they have no conflicts of interest.

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