

Hypothyroidism Induced Cardiac Tamponade in Intensive Care Unit—A Rare Presentation

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Abstract

Hypothyroidism is common disease with multi-system involvement. Pericardial tamponade secondary to hypothyroidism is extremely rare. In current

paper we describe a case of middle age Hispanic man presenting with pericardial tamponade due to severe hypothyroidism, and discuss the relevant literature.

Keywords: Hypothyroidism, pericardial effusion

Introduction

Hypothyroidism is a disease with multi-system involvement, which may present in various forms, one being unusual pericardial effusion [1,2]. Pericardial effusion secondary to hypothyroidism has been well-described [1]. Moderate to large pericardial effusion are rare and associated tamponade is extremely rare [3,4]. Most reports have emphasized the rarity of presentations involving cardiac tamponade, and few have discussed difficulties in early diagnosis [3,5]. Identification of cardiac tamponade in hypothyroidism is therefore, difficult and commonly mistaken for cardiac failure due to its symptoms of tachycardia, rise in venous pressure, lower limb edema, and increased cardiac silhouette on radiography. (Presentation of this rare condition includes; respiratory failure, muscular weakness and elevated creatine kinase (CK), makes it an unusual presentation, critical care physicians need to look for). Critical care physicians need to familiarize themselves with this presentation for early recognition of a pericardial effusion and its underlying cause are important for improving prognosis.

Case report

A 44 year old Hispanic male with morbid obesity weighing

400 lbs, and a history of hypothyroidism presented to the Emergency Department complaining of shortness of breath progressively worsening over one year duration. Patient on presentation to the Emergency Room was found to be hypoxic with PaO₂ of 42 torr and PCO₂ of 60 torr on Room air. Chest radiograph revealed an enlarged heart with increased vascular markings suggestive of congestive heart failure, but patient's B-Natriuretic Peptide was 52. He was found to be in moderate respiratory distress, and admitted to telemetry, where over the next few hours the patient's condition deteriorated. His thyroid stimulating hormone (TSH) level was 37.3 mIU/ml (normal range 0.27-4.2) despite supplementation of synthroid (thyroxine) of 100 micrograms daily. Patient's total creatine kinase level was 2090 U/L (normal 26-140), creatine kinase-MB (CK-MB) was 18.1 ng/L (normal 0.6-6.3), and troponin I was <0.04 (normal <0.04).

Patient was transferred to the Intensive Care Unit, where he was intubated, and placed on mechanical ventilation, his blood pressure was 80/60 mmHg, heart rate was 100 beats/minute with distant heart sound, and jugular venous distension was difficult to assess secondary to patient obesity and short neck. Lungs were clear to auscultation and abdomen examination was benign. Repeat chest radiograph revealed increase in size of cardiac silhouette (**Figure 1**). An emergent echocardiogram was performed, revealing massive pericardial effusion. A bedside pericardiocentesis was performed emergently draining 550 ml of straw color fluid, immediately improving his blood pressure. Protein content of pericardial fluid was high 7 gram/dl. Pulmonary artery catheter was placed for closer monitoring of hemodynamic status. Two days later, he started to show hemodynamic deterioration with equalization of right heart pressure, emergent echocardiogram was

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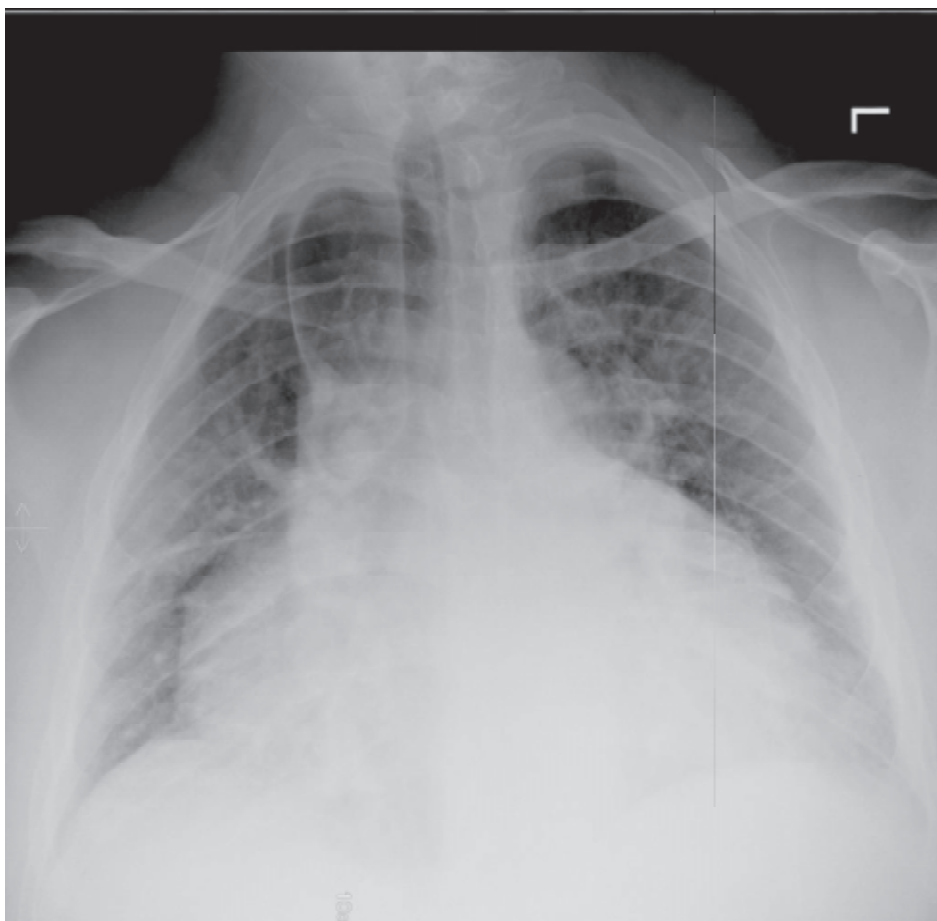


FIGURE 1. CHEST RADIOGRAPH OF PATIENT DISPLAYING ENLARGED CARDIAC SILHOUETTE.

obtained revealing significant pericardial effusion requiring repeat pericardiocentesis draining an additional 500 ml of fluid. Patient then underwent pericardial window with improvement in his hemodynamic and respiratory status, blood pressure 120/78 mmHg and heart rate decreased to 60 beats/minute. Patient was then treated with intravenous thyroxine resulting in improvement of creatine kinase, which normalized over the next three days, along with improvement in respiratory status. Culture of pericardial fluid was negative for viral or bacterial infection. Collagen vascular profile was normal.

Discussion

The systemic hypometabolism that is associated with hypothyroidism results in a decrease in cardiac output that is mediated by reductions in heart rate and contractility [6,7]. Thyroid hormone regulation of gene coding for specific myocardial enzymes involved in myocardial contractility and relaxation is responsible for the decrease in contractil-

ity [8]. The occurrence of a pericardial effusion in hypothyroidism seems to be related to the severity and duration of the disease. The incidence is reported to be as 3% in early mild stage to 80% when myxedema is present [1, 9]. In contrast to the frequent occurrence of small pericardial effusion in patients with hypothyroidism, moderate to large pericardial effusions are rare and associated tamponade is extremely rare. Cardiac tamponade in patients diagnosed with hypothyroidism is probably as rare as it is due to pericardial distensability and the slow accumulation of fluid, allowing significant fluid accumulation without hemodynamic compromise [3,10,11].

Many hypothyroid patients have high serum CK concentrations. The isoenzyme distribution is almost completely MM, with less than 4 percent constituting MB, indicating skeletal muscle, not myocardial, origin [12]. Approximately 14 percent of patients with hypothyroidism have a raised serum concentration of CK-MB that can be confusing in the evaluation of chest pain. This problem is obviated by measurement of serum troponin I, which is normal in hypothyroidism [13].

In conclusion, pericardial effusion with tamponade should be suspected in patients with high TSH, low blood pressure, relative bradycardia, and elevated CK level. Use

of ultrasound by Emergency Room physician and critical care specialist as a part of their armamentarium is highly recommended.

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