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Pericardial Effusion with Tamponade in Untreated Hypothyroidism

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Patient: Female, 44-year-old
Final Diagnosis: Hypothyroidism • pericardial effusion • tamponade
Symptoms: Dyspnea • fatigue • weight gain
Medication: —
Clinical Procedure: Pericardial drainage
Specialty: Cardiology • Endocrinology and Metabolic • General and Internal Medicine

Objective: Unusual clinical course

Background: Small pericardial effusions are common with chronic hypothyroidism, but large pericardial effusion with tamponade or pre-tamponade physiology is a rare complication of severe uncontrolled hypothyroidism. Presentation of pericardial effusion of any etiology can range from being asymptomatic to hemodynamic instability with cardiac tamponade, depending on the amount and speed of accumulation of pericardial fluid, but pericardial effusion associated with hypothyroidism are usually small. Protracted medication non-adherence was a key factor in our patient's presentation.

Case Report: We present a case of a woman in her 40s with a known history of autoimmune hypothyroidism with medication non-adherence for longer than 9 months who presented with fatigue, weight gain, limited physical activity, and exertional dyspnea with bilateral swelling of the upper and lower extremities. Examination revealed muffled heart sounds, positive JVD, and positive pulsus paradoxus. She had an elevated TSH, low free T4, and a high anti-thyroid peroxidase antibody level. Echocardiography revealed a large pericardial effusion with impending tamponade. Pericardiocentesis with pericardial drain was done and the patient's symptoms resolved quickly. The patient was restarted on a prior dose of levothyroxine 175 mcg. She had improved by the 3rd day of hospitalization; the pericardial drain was removed, and she was discharged with access to medication. Follow-up revealed complete resolution of her symptoms.

Conclusions: This case emphasizes the importance of recognition of hypothyroidism as the etiology of life-threatening large pericardial effusions, as it is treatable and recurrences are preventable. To prevent recurrence, it is important to achieve euthyroidism after treating an episode of pericardial effusion.

Keywords: Cardiac Tamponade • Hypothyroidism • Pericardial Effusion

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/938520

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Background

Small pericardial effusions are commonly associated with hypothyroidism. Some authors reported that over 80% of cases of long-standing or severe hypothyroidism are associated with pericardial effusion [1]. The presentation of pericardial effusion of any etiology can range from being asymptomatic to hemodynamic instability with cardiac tamponade, depending on the amount and speed of accumulation of pericardial fluid [2], but the amount of pericardial effusion associated with hypothyroidism is usually small [1]. It is very rare that a patient would present with a massive symptomatic pericardial effusion due to untreated hypothyroidism, and this usually happens in patients with severe disease or myxedema. Tamponade with massive pericardial effusion due to hypothyroidism is rarely reported in the literature [1]. We present a similar case of large pericardial effusion in a young female patient with untreated severe hypothyroidism.

Case Report

A woman in her 40s with a past medical history of autoimmune hypothyroidism, right-sided atrophic kidney, and tobacco abuse presented with a chief concern of worsening dyspnea with exertion, bilateral upper- and lower-extremity edema, and difficulty laying down flat since the last 2 weeks. She also noted recent puffiness of her eyelids and face. She also noted weight gain, fatigue, cold intolerance, and generalized weakness, which worsened with limited physical activity. She denied nighttime awakening with shortness of breath, chest pain, palpitations, lightheadedness, syncopal episodes, hair loss, or constipation. She was diagnosed with autoimmune hypothyroidism at 30 years of age and had been managed with 175 mcg of levothyroxine. She reported that she had been adherent to her thyroid medication until 9 months ago, when she was no longer able to pay for her health insurance and could not afford the medication. There was no history of use of amiodarone, lithium, or radiation to the neck. Her family history was significant for hypothyroidism on her maternal side in all the female relatives. She had a 15 pack-year smoking history, smoking half a pack a day at the time of admission. Her medical records confirmed positive anti-thyroid peroxidase antibodies and a prior thyroid ultrasound that showed an atrophic thyroid gland consistent with a diagnosis of Hashimoto's thyroiditis.

On arrival at the emergency room, her vital signs were stable. She was noted to have a BMI of 37.46 kg/m^2. Physical examination showed generalized, 2+ non-pitting edema of woody consistency in the bilateral upper and lower extremities, with facial puffiness, most pronounced around the eyelids, with a dull facial expression. She looked older than her stated age and had rough, dry skin. A cardiovascular exam revealed muffled heart sounds, no pericardial rub, positive JVD, and positive pulsus paradoxus with a drop in systolic blood pressure by 24 mmHg, but she remained hemodynamically stable with BP 120-140/70-80 mmHg and heart rate 70 s to 80 s. Her lungs were clear to auscultation bilaterally. She had delayed relaxation phase of deep-tendon reflexes, especially prominent at the patellar and ankle reflexes bilaterally.

Lab investigations revealed unremarkable CBC, troponins, BNP, ESR, CRP, lipid panel, ANA screen, troponin, and BNP. CMP showed an acute kidney injury with creatinine of 1.56 mg/dl (baseline creatinine 0.9 mg/dl) and GFR of 36. She had an elevated TSH >500, low free T4 of 0.4, and a high positive anti-thyroid peroxidase antibody of 122 (**Table 1**). EKG showed low-voltage QRS in all leads with electrical alternans, QTc 456 (**Figure 1**). A chest X-ray showed cardiomegaly (**Figure 2**). Trans-thoracic echocardiography (TTE) showed a large pericardial effusion measuring 2.3 cm in the posterior region and 1.6 cm in the anterior region, with significant respiratory variation in mitral inflow plus a diastolic collapse of the RV (**Figures 3-5**). Overall findings were suggestive of impending cardiac tamponade.

With no fever, upper-respiratory infection symptoms, or chest pain, an infectious etiology of pericardial effusion was unlikely. With no weight loss or any B symptoms, malignancy was low on the differential as well. Severe hypothyroidism can cause pericardial effusion but is a rare cause of massive pericardial effusion causing tamponade physiology, which became the most probable etiology in this patient given her severely elevated TSH levels.

Pericardiocentesis with a pericardial drain placement was performed. A total of 240 mL of serosanguinous pericardial fluid was drained followed by brisk improvement of clinical and

Table 1. Thyroids laboratory test results.

	On admission	At 3-month follow-up	Normal values
TSH	>500	0.401	0.300-5.000 mIU/L
Free T4	0.4	Not done	0.7-1.9 ng/dL
Anti-thyroperoxidase antibody	122	Not done	<0.4 IU/mL

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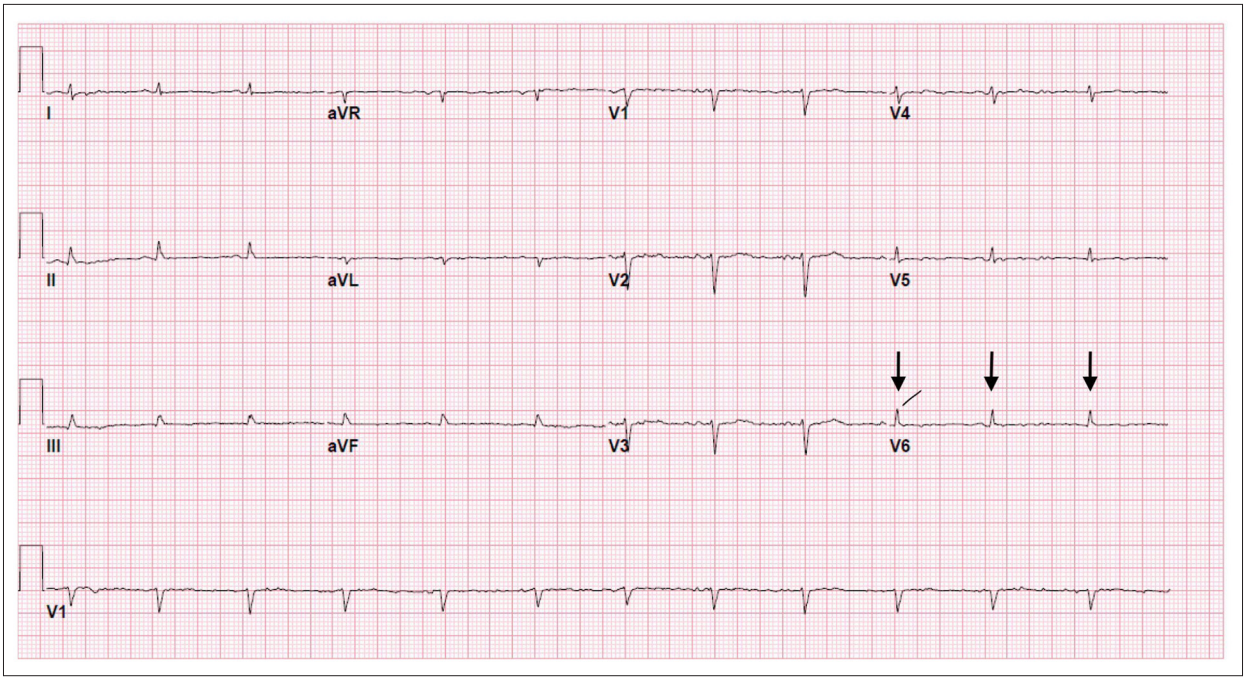

Figure 1. EKG of patient at admission showing low-voltage QRS and electrical alternans as shown by the solid black arrows.

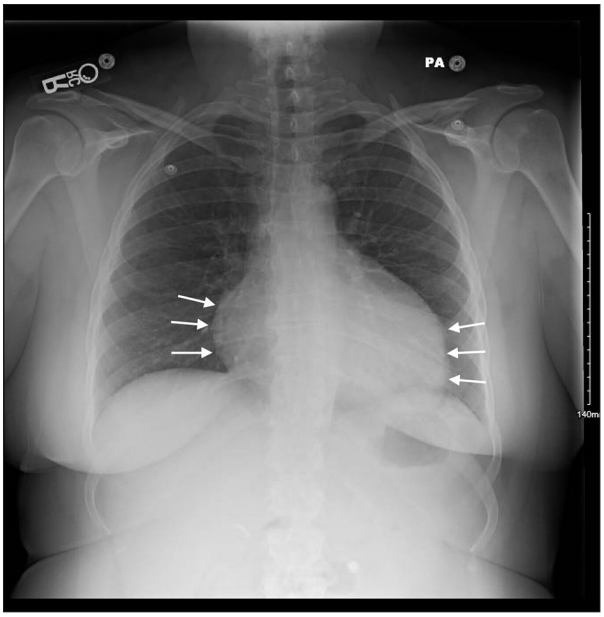

Figure 2. Chest X-ray of patient at admission showing cardiomegaly with boot-shaped appearance as demarcated by the solid white arrows.

echocardiographic parameters, with a resolution of tamponade physiology. Post-procedure TTE showed a minimal remnant pericardial effusion. Over the next 3 hospital days, another 350 ml of fluid drained from the pericardial drain until the drainage ceased, after which the drain was removed. The patient was also started back on daily intravenous 175 mcg of levothyroxine, which was switched to oral after 3 days.

She showed significant improvement during her hospital stay, with complete resolution of her dyspnea on exertion. Pericardial fluid analysis with gram stain, culture, LDH, protein, cell differential, and cytology were all unremarkable, and the acute kidney injury had resolved. The patient was counseled on adherence to her thyroid medication and provided resources to obtain medications. At 2-week and 3-month follow-up appointments with her primary care physician, the patient continued to feel well, with marked improvement and complete resolution of her symptoms and normalization of her thyroid labs.

Discussion

Hypothyroidism is ubiquitous, affecting anywhere from 4% to 10% of the general population. Low thyroid hormone can affect nearly all organs in the body, including the heart. Physical examination and history findings with hypothyroidism can include dyspnea, fatigue, decreased exercise tolerance, constipation, dry skin, poor memory, and non-pitting edema. Cardiovascular manifestations of low T3 include diastolic hypertension, sinus bradycardia, pericarditis, dyslipidemia, and pericardial effusion. Hypothyroidism is the cause of 3% to 37% of pericardial effusions, and the vast majority of these are small [3,4]. Small pericardial effusion in chronic hypothyroidism is common, but development of cardiac tamponade or pre-tamponade secondary to hypothyroidism is rare [5].

Pericardial effusion in hypothyroidism is a chronic process, developing over time, allowing for the pericardium to stretch and

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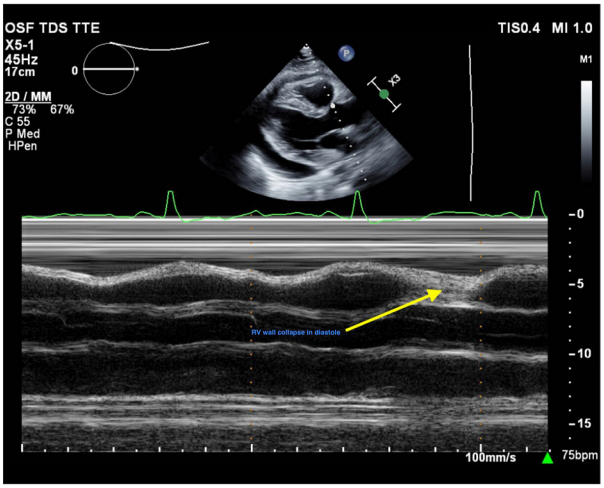

Figure 3. Parasternal long axis M-mode showing collapse of the free right ventricular wall in diastole as portrayed by the solid yellow arrow.

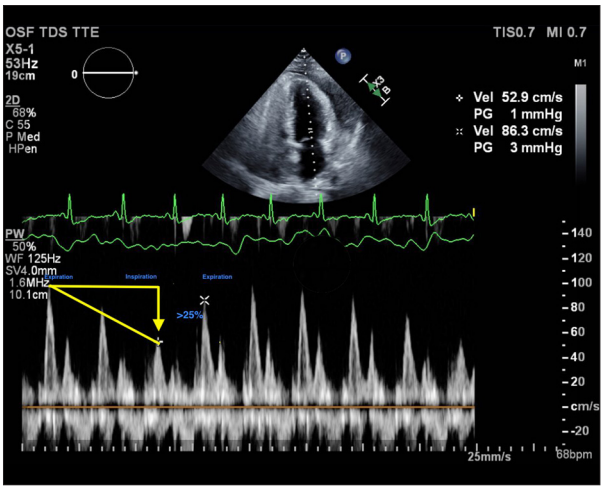

Figure 4. Apical 4-chamber view pulsed wave Doppler interrogation of the mitral valve showing significant respiratory variation (>25%) of the mitral inflow velocities as shown by the solid yellow lines and arrow.

adapt to accommodate the large fluid volume without causing cardiac compromise. Hypothyroidism is thought to cause effusions due to increased pericardial permeability and decreased lymphatic drainage of albumin, leading to leakage of protein into the pericardial space. This increase of albumin in the extravascular space leads to decreased reabsorption of fluid at the venous end of capillaries and a chronic accumulation of fluid in the pericardial space [4]. The compliant pericardium keeps expanding to compensate for the increasing volume of pericardial fluid [6], but at a certain volume, the pericardial space can no longer accommodate the fluid, leading to tamponade physiology. The increased pericardial pressure causes chamber collapse because during diastole the heart cannot expand into the pericardial space due to the fluid [3,4]. This manifests as

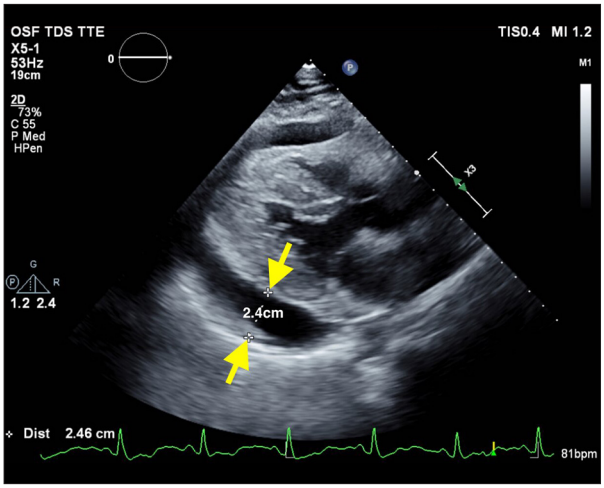

Figure 5. Parasternal long axis view showing large effusion with 2.4 cm posteriorly between the 2 solid yellow arrows.

RV collapse, such as seen in our patient. Another unique aspect of hypothyroidism associated with cardiac tamponade is that patients may not have tachycardia, which was the case with our patient [7]. The prolonged course of severe, untreated hypothyroidism, along with lack of cardiovascular system capacity led to the development of massive pericardial effusion in our patient. T3 increases myocardial adrenergic sensitivity and has positive ionotropic and chronotropic effects [8]; thus, in severe hypothyroidism due to lack of this chronotropic effect, tachycardia is not seen, even in cardiac tamponade.

The management of pericardial effusions is with thyroid replacement with or without an intervention to drain the fluid. Several studies have shown successful management of hypothyroid-induced pericardial effusion with echocardiographic tamponade physiology, but not clinical cardiac tamponade, to be treated effectively with levothyroxine alone [9]. However, most patients with hypothyroid-induced pericardial effusion with clinical cardiac tamponade (eg, pulsus paradoxus), such as in the patient we present, require medication as well as pericardial drainage through pericardiocentesis or pericardial window to stabilize the patient. Chahine et al presented a scoring system developed by the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases to determine how urgently pericardiocentesis should be performed in cardiac tamponade [4]. It is important to achieve euthyroidism in the weeks ensuing pericardiocentesis as there is a significant risk of recurrence [7].

We opted to treat the medication non-compliance-associated severe hypothyroidism prior to investigating other differentials of pericardial effusion, kidney injury, and hypothyroidism such as amyloidosis and IGG4 disease to prevent unnecessary testing. The acute kidney injury improved greatly with pericardiocentesis and diuresis, confirming the kidney injury was most likely cardio-renal in nature. Her edema in the extremities also improved

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significantly. In the following outpatient clinic visits, she reported medication compliance and non-recurrence of symptoms.

The treatment of pericardial effusion remains the same, but identifying the underlying cause of hypothyroidism can prevent further recurrences. Thus, given the ubiquity of hypothyroidism, it is important for clinicians to be aware of it being a possible cause of large, life-threatening pericardial effusions.

Conclusions

Severe hypothyroidism can cause pericardial effusion but is a rare cause of massive pericardial effusion causing tamponade or pre-tamponade. Pericardial effusion due to hypothyroidism presenting with tamponade physiology may need emergent pericardiocentesis along with thyroid replacement therapy. To prevent recurrence, it is important to achieve euthyroidism after treating the episode of pericardial effusion. This case report emphasizes the importance of recognition of hypothyroidism as the etiology of life-threatening large pericardial effusions as it is treatable and recurrences are preventable.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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