Section: Emergency Medicine

Case Report

CARDIAC TAMPONADE AS THE INITIAL PRESENTATION OF SEVERE PRIMARY AUTOIMMUNE HYPOTHYROIDISM: A RARE CASE REPORT

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ABSTRACT

Background: Pericardial effusion is a well-recognized cardiovascular manifestation of hypothyroidism; however, progression to cardiac tamponade is exceptionally uncommon because the fluid accumulates slowly, allowing gradual adaptation of the pericardium. Consequently, patients frequently lack the classical hemodynamic manifestations of tamponade, making diagnosis challenging. We report a rare case of severe primary autoimmune hypothyroidism presenting with massive pericardial effusion complicated by cardiac tamponade that was successfully managed with urgent pericardiocentesis and thyroid hormone replacement.

Case Presentation: A 45-year-old woman presented with progressively worsening dyspnea, generalized body swelling, fatigue, low-grade fever, and dull aching chest pain. She also gave history of constipation, dry skin and menstrual irregularities. Clinical examination revealed non-pitting pedal edema, elevated jugular venous pressure and muffled heart sounds. Chest radiography demonstrated marked cardiomegaly with a characteristic "water-bottle" cardiac silhouette. Electrocardiography was done which showed low-voltage QRS complexes. Transthoracic echocardiography confirmed a large circumferential pericardial effusion and features consistent with cardiac tamponade. Laboratory evaluation revealed profound primary hypothyroidism and strongly positive anti-thyroid peroxidase antibodies. Emergency pericardiocentesis was done and thyroid replacement therapy was started. Serial echocardiography demonstrated complete resolution of the effusion and the patient was eventually discharged in stable condition.

Conclusion: Cardiac tamponade should be considered in patients with unexplained large pericardial effusions, even in the absence of hypotension or tachycardia. Severe hypothyroidism remains an uncommon but reversible cause of tamponade. Early recognition through careful clinical examination, electrocardiography, and echocardiography, followed by prompt pericardiocentesis and thyroid hormone replacement, can be lifesaving and result in excellent clinical outcomes.

Keywords: Cardiac tamponade; Hypothyroidism; Pericardial effusion; Autoimmune thyroiditis; Pericardiocentesis; Echocardiography.

INTRODUCTION

Hypothyroidism is one of the most common endocrine disorders worldwide and affects multiple organ systems through reduced metabolic activity and impaired tissue function. Cardiovascular manifestations are particularly important because thyroid hormones exert significant effects on myocardial contractility, heart rate, vascular resistance, and intravascular volume regulation. Patients with untreated hypothyroidism frequently develop bradycardia, reduced cardiac output,

diastolic hypertension, dyslipidemia, and, less commonly, pericardial effusion. Although pericardial effusion is a recognized complication of severe hypothyroidism, progression to cardiac tamponade is exceedingly rare because the pericardial fluid accumulates slowly, permitting gradual stretching of the pericardial sac without immediate hemodynamic compromise.[1]

The reported prevalence of pericardial effusion among patients with hypothyroidism varies widely, ranging from approximately 3% in mild disease to as high as 80% in severe, longstanding hypothyroidism. The pathogenesis involves increased capillary permeability, accumulation of protein-rich mucopolysaccharides within the interstitial tissues, impaired lymphatic drainage, and reduced plasma protein clearance from the pericardial cavity. These mechanisms promote gradual accumulation of protein-rich fluid within the pericardial sac. Since fluid develops slowly, patients often remain asymptomatic until the effusion becomes massive, delaying diagnosis and treatment.[2]

Cardiac tamponade represents a medical emergency characterized by impaired ventricular filling due to elevated intrapericardial pressure. Classically patients of cardiac tamponade present with Beck's triad of hypotension that includes elevated jugular venous pressure, muffled heart sounds, often accompanied by tachycardia and pulsus paradoxus. However, tamponade secondary to hypothyroidism may differ from tamponade caused by other usual conditions. Because metabolic demands are reduced and sympathetic activation is blunted, affected patients may remain normotensive and relatively bradycardic despite large pericardial effusions. Consequently, diagnosis may be overlooked if clinicians rely solely on the classical clinical picture.[3]

The diagnosis of hypothyroid-associated cardiac tamponade requires a high index of suspicion and integration of clinical findings with laboratory and imaging investigations. Electrocardiography commonly demonstrates low-voltage QRS complexes and, occasionally, electrical alternans in patients with massive effusions. Chest radiography may reveal globular enlargement of the cardiac silhouette with the characteristic "water-bottle" appearance. Nevertheless, transthoracic echocardiography remains the diagnostic modality of choice because it accurately identifies the volume of pericardial fluid and demonstrates echocardiographic signs of tamponade physiology, including right atrial or right ventricular chamber collapse, respiratory variation in transvalvular flow velocities, and a swinging motion of the heart within the pericardial sac.[4]

Management depends on the patient's hemodynamic status. Small or asymptomatic hypothyroid-related pericardial effusions generally resolve gradually following initiation of levothyroxine replacement alone. Conversely, patients with evidence of cardiac tamponade require urgent pericardiocentesis to relieve cardiac compression, followed by definitive correction of the underlying thyroid dysfunction. Early intervention is associated with rapid symptomatic improvement and excellent long-term outcomes, whereas delayed recognition may result in circulatory collapse and death.

Several authors have emphasized the rarity of cardiac tamponade as the initial manifestation of severe hypothyroidism. Hypothyroid cardiac tamponade has been described as an uncommon clinical entity in which patients frequently lack the classical hemodynamic findings of tamponade because of slow fluid accumulation. Massive pericardial effusions may remain clinically silent until advanced stages, highlighting the importance of thyroid function testing in unexplained pericardial effusions. Prompt recognition and combined management with pericardiocentesis and thyroid hormone replacement have been shown to result in favorable clinical outcomes.[5]

We report a rare case of severe primary autoimmune hypothyroidism presenting with massive pericardial effusion complicated by cardiac tamponade. This case is important because it highlights the diagnostic challenges posed by the absence of classical tamponade features. It also underscores the importance of early echocardiographic evaluation and timely management of cases with cardiac tamponade in preventing potentially fatal complications.

CASE REPORT

A 45-year-old woman presented to the Emergency Department with progressively worsening exertional dyspnea of 15 days' duration, which had advanced from New York Heart Association (NYHA) functional class II to class III over the preceding three months. She also complained of generalized body swelling, easy fatigability, low-grade intermittent fever, and dull aching retrosternal chest pain. On further inquiry, she reported constipation, dry skin, slowing of speech, and menstrual irregularities over several months, suggestive of underlying hypothyroidism. There was no history of hypertension, diabetes mellitus, ischemic heart disease, chronic kidney disease, tuberculosis, connective tissue disorders, malignancy, previous thyroid disease, or prior cardiac intervention. There was no history of trauma or anticoagulant use.

On physical examination the patient appeared lethargic. She was conscious, oriented and afebrile at the time of presentation. Blood pressure was within the normal range while peripheral pulses were regular. General examination revealed pallor and bilateral non-pitting pedal edema. Jugular venous pressure was elevated [Figure 1] and a positive Kussmaul's sign was observed.

Figure 1: Clinical photograph showing prominent distension of the external jugular veins in the neck, consistent with elevated jugular venous pressure (JVP).

On Cardiovascular examination heart sounds were found to be muffled and there were no audible murmurs on auscultation. Respiratory examination revealed reduced air entry over the left infra-axillary region. Abdominal examination showed presence of shifting dullness suggesting presence of ascites. The absence of hypotension and tachycardia despite a large pericardial effusion made the diagnosis of cardiac tamponade clinically difficult and challenging.

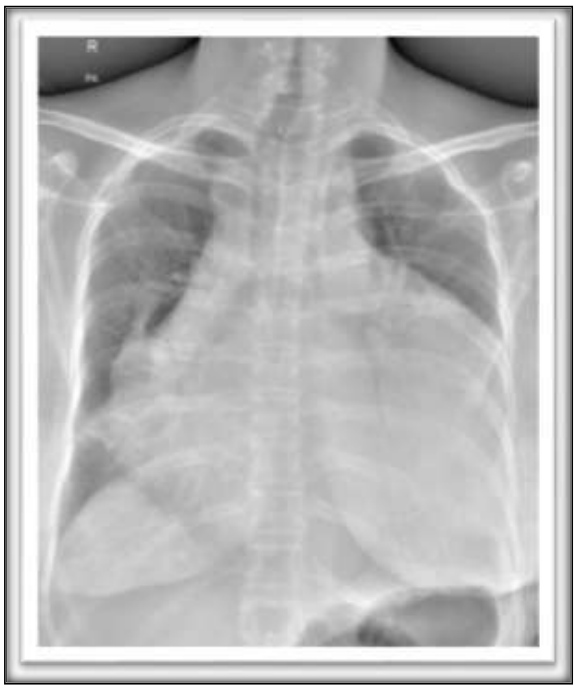

Figure 2: Posteroanterior chest radiograph demonstrating marked globular cardiomegaly ("water-bottle" cardiac silhouette), characteristic of a massive pericardial effusion.

Initial laboratory investigations demonstrated presence of anemia (Hb=8.4 g/dL) and a total leukocyte count of 4,000/mm³. Liver and renal function tests were normal except for mildly elevated serum transaminases. Thyroid function test revealed presence of severe primary hypothyroidism with a markedly elevated thyroid-stimulating hormone (TSH) level (350.7 mIU/mL), significantly reduced free thyroxine (FT4) (0.454 ng/dL), and free triiodothyronine (FT3) of 0.199 pg/mL. Anti-thyroid peroxidase antibody levels were markedly elevated (700 IU/mL). All these findings supported the diagnosis of autoimmune thyroiditis as the underlying etiology.

Chest radiography showed marked cardiomegaly with a characteristic globular enlargement of the cardiac silhouette. This classic "water-bottle" appearance was suggestive of massive pericardial effusion.

Electrocardiography demonstrated presence of diffuse low-voltage QRS complexes without any evidence of acute ischemic changes. Bedside transthoracic echocardiography confirmed presence of a large circumferential pericardial effusion and swinging motion of the heart within the pericardial sac was clearly visible. Echocardiographic evidence of right atrial systolic collapse was also present, although right ventricular collapse was absent. This was consistent with evolving cardiac tamponade physiology.

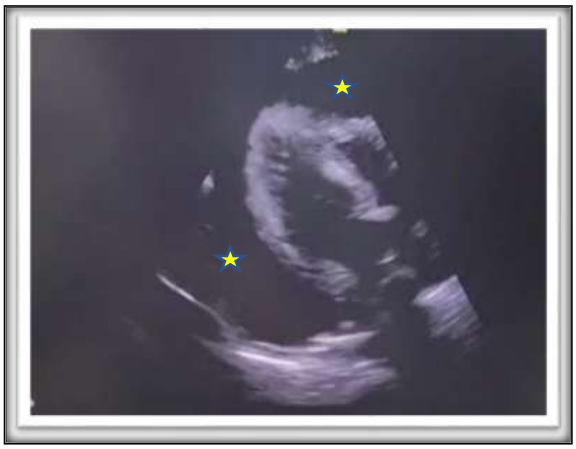

Figure 3: Transthoracic echocardiography (Subcostal view) demonstrating a large circumferential pericardial effusion (star marks) surrounding the heart. Echocardiographic findings were consistent with evolving cardiac tamponade physiology.

In conjunction with the clinical findings of elevated jugular venous pressure and muffled heart sounds the diagnosis of cardiac tamponade secondary to severe primary hypothyroidism was established.

The patient was immediately initiated on oral levothyroxine at a dose of 100 µg daily together with supportive medical therapy, including diuretics and prednisolone 30 mg daily. Considering the echocardiographic evidence of tamponade and the risk of further hemodynamic deterioration, emergency ultrasound-guided pericardiocentesis was performed through the subcostal approach under continuous cardiac monitoring. Approximately 1,000

mL of clear pericardial fluid was drained during the procedure and a pigtail catheter was left in situ for the purpose of continued drainage.

Pericardial fluid analysis revealed a clear, pale-yellow fluid with a low total leukocyte count. There was no evidence of bacterial or tuberculous infection. Acid-fast bacilli staining and cartridge-based nucleic acid amplification testing (CBNAAT) for Mycobacterium tuberculosis were found to be negative. Cytological examination did not show presence of any malignant cells. These findings excluded infectious as well as malignant causes of the pericardial effusion thereby supporting hypothyroidism as the primary etiology for cardiac tamponade.

Over the following six days an additional 730 mL volume of pericardial fluid was drained through the indwelling pigtail catheter. Serial transthoracic echocardiography demonstrated progressive reduction of the pericardial effusion with disappearance of tamponade physiology. The patient's dyspnea, peripheral edema and constitutional symptoms improved significantly during hospitalization. She remained hemodynamically stable throughout the recovery period and was discharged after ten days in good clinical condition. At the time of discharge, she was counselled to continue lifelong levothyroxine replacement therapy and to attend regular outpatient follow-up.

DISCUSSION

Pericardial effusion is a well-recognized cardiovascular manifestation of hypothyroidism, whereas progression to cardiac tamponade remains exceptionally uncommon. The rarity of tamponade is attributed to the slow accumulation of pericardial fluid, which allows progressive stretching of the pericardium and accommodation of large fluid volumes before significant impairment of cardiac filling occurs. Consequently, many patients remain asymptomatic until the effusion becomes massive, and the classical hemodynamic features of cardiac tamponade may be absent. This characteristic presentation was evident in our patient, who had a large pericardial effusion with echocardiographic evidence of tamponade physiology despite preserved blood pressure and the absence of significant tachycardia. Such atypical presentations emphasize the importance of maintaining a high index of suspicion in patients with severe hypothyroidism presenting with unexplained cardiomegaly or dyspnea.[6]

The pathophysiology of hypothyroid-associated pericardial effusion involves increased systemic capillary permeability, reduced lymphatic drainage and accumulation of hydrophilic glycosaminoglycans within interstitial tissues resulting in leakage of protein-rich fluid into the pericardial cavity.[7] Reduced metabolic activity as well as diminished sympathetic stimulation further contribute to the absence of compensatory tachycardia. All these pathophysiological features cause masking of the severity of cardiac compression. The marked elevation of thyroid-stimulating hormone, and strongly positive anti-thyroid peroxidase antibody levels and absence of any alternative explanation of pericardial effusion in the present case confirmed severe autoimmune primary hypothyroidism as the underlying cause of the massive pericardial effusion.

Clinical diagnosis of hypothyroid-related cardiac tamponade may be particularly challenging because the classic manifestations of Beck's triad are frequently incomplete. Although our patient demonstrated elevated jugular venous pressure and muffled heart sounds, blood pressure remained within the normal range throughout presentation. Similar atypical clinical findings have been described by Chahine et al who reported that patients with hypothyroid cardiac tamponade often lack hypotension because the slow accumulation of pericardial fluid permits gradual physiological adaptation of the cardiovascular system.[8] Their review emphasized that delayed recognition remains common unless thyroid dysfunction is actively considered in the differential diagnosis of unexplained pericardial effusion.

Electrocardiography and chest radiography provide valuable initial diagnostic clues but lack specificity. Our patient demonstrated diffuse low-voltage QRS complexes together with a characteristic globular "water-bottle" cardiac silhouette on chest radiography, findings commonly associated with massive pericardial effusion. Definitive diagnosis was established by transthoracic echocardiography, which demonstrated a large circumferential pericardial effusion with a swinging heart and right atrial systolic collapse, confirming tamponade physiology. Echocardiography remains the gold standard for evaluating both the severity of pericardial effusion and its hemodynamic consequences, enabling prompt therapeutic intervention before circulatory deterioration occurs.

Several published reports have described similar clinical presentations. Kabadi UM et al reported patients with severe primary hypothyroidism who developed massive pericardial effusions without overt hemodynamic instability. All these cases underscore importance of thyroid function testing in patients presenting with otherwise unexplained large pericardial effusions.[9] Similarly Wang et al. documented cardiac tamponade as the initial manifestation of severe hypothyroidism. The authors further demonstrated complete clinical recovery following urgent pericardiocentesis combined and initiation of prompt thyroid hormone replacement.[10] These observations are quite similar to the clinical course observed in this patient. As seen in this case as well as earlier similar cases early diagnosis and definitive treatment result in excellent outcomes

despite the potentially life-threatening nature of the condition.

Management of hypothyroid-associated pericardial effusion depends primarily on the presence or absence of tamponade physiology. Small or asymptomatic effusions generally resolve gradually following levothyroxine replacement alone as normal thyroid function is restored.[11] However, when cardiac tamponade develops, immediate pericardial drainage becomes mandatory to relieve intrapericardial pressure and restore normal ventricular filling. Current European Society of Cardiology guidelines recommend urgent pericardiocentesis for all patients with clinically significant tamponade irrespective of the underlying etiology. In the present case, emergency pericardiocentesis resulted in drainage of approximately 1,730 mL of pericardial fluid over six days, followed by complete echocardiographic resolution after initiation of levothyroxine therapy, consistent with current recommendations.

The present case highlights several important clinical lessons. First, severe hypothyroidism must be considered in the differential diagnosis of unexplained large pericardial effusions. This is more so if pericardial effusion is accompanied by constitutional symptoms of thyroid dysfunction. Second, the absence of hypotension or marked tachycardia should not automatically exclude the possibility of cardiac tamponade in chronic hypothyroidism cases because gradual fluid accumulation often masks classical hemodynamic findings in these cases. Finally, early echocardiographic assessment, prompt pericardial decompression when indicated and prompt thyroid replacement therapy remain the cornerstones of successful management. High index of suspicion for this rare but reversible complication may facilitate earlier diagnosis and substantially improve patient outcomes.[12]

CONCLUSION

Cardiac tamponade is an uncommon but potentially life-threatening complication of severe primary hypothyroidism. Pericardial tamponade in these cases may occur despite the absence of classical hemodynamic features such as hypotension and marked tachycardia. This case highlights the importance of considering thyroid dysfunction in the differential diagnosis of unexplained massive pericardial effusion particularly in cases who display subtle clinical features of hypothyroidism. Early diagnosis through careful clinical assessment, electrocardiography and prompt echocardiographic evaluation is essential for timely diagnosis. In patients with tamponade physiology interventions such as urgent pericardiocentesis combined with prompt thyroid hormone replacement therapy is known to result in rapid clinical improvement and favorable outcomes. A high index of suspicion for this rare presentation among clinicians may facilitate earlier diagnosis and reduce morbidity associated with delayed treatment.

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