Primary Hypothyroidism Presenting as Cardiac Tamponade

Prasan K Panda, Shridhar Pattar, Budha O Singh, Taranjeet Cheema

Abstract

Hypothyroidism is commonly associated with pericardial effusion, but it can be rarely complicated by cardiac tamponade. We report a case series of two patients who presented with shortness of breath and distension of the abdomen, progressing to generalized edema. Each of them was found to have cardiac tamponade at presentation and eventually diagnosed with hypothyroidism. They were managed by urgent pericardiocentesis followed by intermittent drainage of the collected pericardial effusion along with thyroxine replacement to which they responded. The presence of cardiac tamponade with bradycardia should raise a suspicion of a hypothyroid etiology. Early diagnosis and treatment of hypothyroidism are essential to prevent such complications.

Keywords: Hypothyroidism, Pericardial effusion, Pericardiocentesis, Thyroxine replacement.

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Sir,

A 21-year-old woman, daily laborer, without any comorbidities, presented with generalized body swelling for the last 3 months that started from the ankle and gradually progressed toward the abdomen and face. She also had a dry cough and an undue intolerance to cold along with chills suspected to be fever that was, however, undocumented. She had shortness of breath for 15 days, progressing from class 1 to 4 NYHA and was associated with orthopnea. There was no history of chest pain/palpitations/decreased urine output.

On presentation, she appeared pale and had a normal sensorium with low BP of 84/50 mm Hg, PR 64 bpm, RR 26/minute, and raised JVP which was more at the time of inspiration. She had cold, clammy coarse skin and had a hoarse voice. There was bilateral pitting pedal edema and tensed abdomen with shifting dullness. Heart sounds were muffled on auscultation.

Necessary investigations at the point of care were performed (Table 1). Chest X-ray and ECG showed massive cardiomegaly and low voltage pattern, respectively. Point-of-care ultrasound revealed bilateral pleural effusion along with a massive pericardial effusion with right ventricle (RV) diastolic collapse. Ascites was also confirmed. Ultrasonography of the thyroid gland showed normal size and vascularity of both the thyroid lobes and isthmus. There was a slightly heterogeneous echotexture with ill-defined fibrotic strands.

Urgent pericardiocentesis was done by inserting a pigtail catheter via subxiphoid approach, and around 1100 mL of pericardial fluid was removed in a single setting. The pericardial fluid was clear and straw yellow in appearance and investigations sent ruled out any infective pathology including tuberculosis. The remaining accumulated fluid was drained gradually over a period of ten days through the pigtail catheter.

On confirmation of primary hypothyroidism, she was treated with levothyroxine 200 µg stat followed by 100 µg once daily (OD). Over the course of 14 days of her stay in the hospital, her general condition improved. Serial echocardiographic monitoring was done, and the pigtail was finally removed after confirming that there was no residual effusion or reaccumulation. The patient was discharged in a hemodynamically stable condition. She was advised to continue thyroxine supplementation and follow up regularly.

Although there are a variety of causes for pericardial effusion, with few leading to cardiac tamponade, a temporal association with severe hypothyroidism was demonstrated in the above case. Clinical presentation, echocardiographic findings, and a raised JVP during inspiration (Kussmaul sign) were suggestive of a tamponade physiology. This evidence is supported by the gradual resolution of the pathology and clinical improvement with supplementation of thyroxine. The diagnosis of hypothyroidism as the cause of cardiac tamponade is at times difficult because of its slow onset, nonspecific signs and symptoms.1 These confounding presentations were observed in the above case. Also, the absence of sinus tachycardia in patients diagnosed with cardiac tamponade as in the above cases should arise a strong suspicion of hypothyroidism.2

Clinical features of cardiac tamponade depend upon how rapidly the fluid accumulates in the pericardial space.3 Based on this, cardiac tamponade can be clinically classified as acute, subacute, regional, or low-pressure tamponade. The symptoms are usually of a sudden onset cardiovascular collapse and may be associated with chest pain, tachypnea, and dyspnea. Dyspnea is frequently acute, which, however, was gradual in onset and progressive in our patients. Patients with chronic fluid accumulation remain asymptomatic in the initial phase. Still, when the pressure rises above a threshold, they develop dyspnea, fatigue, chest discomfort, and peripheral edema, due to decreased cardiac output.4 Rarely they can present with left lung collapse or even cough syncope.5,6

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Hypothyroid patients with pericardial effusion respond well to thyroid hormone replacement. But those with severe hemodynamic compromise, i.e. cardiac tamponade need emergency pericardiocentesis or surgical drainage. The present case was managed by pericardiocentesis followed by thyroxine replacement and her condition improved significantly.

In summary, hypothyroidism can present in varied ways at any age; hence, the clinician needs a high degree of suspicion to diagnose the condition and order a thyroid function test. The presence of cardiac tamponade with bradycardia should raise a high degree of suspicion of a hypothyroid etiology.

**Contributors**

SP, TC, and BS contributed to the data collection and data analysis and were involved in manuscript writing. PKP gave the concept, interpreted analysis, critically reviewed the draft, and approved it for publication along with all authors.

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Table 1: Basic and advanced investigation performed in the case

| Investigations                       | Reference range (adults) | Findings |
|--------------------------------------|--------------------------|----------|
| Hemoglobin (gm/dL)                   | 13.5–17.5                | 9.5      |
| White cell count (per μL)            | 4,500–11,000             | 6,500    |
| Differential count (per μL)          |                          |          |
| Neutrophils                          | 1,800–7,700              | 4,550    |
| Lymphocytes                          | 1,000–4,800              | 1,625    |
| Monocytes                            | 200–1,200                | 175      |
| Eosinophils                          | 0–900                    | 390      |
| Basophils                            | 0–300                    | 380      |
| Platelets (per μL)                   | 150,000–400,000          | 178,000  |
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**Pericardial Fluid Analysis**

- **Appearance**
- **Color**
- **Total leukocyte counts**
- **Differential leukocyte counts**
- **Sugar (mg/dL)**
- **Protein (mg/dL)**
- **Microscopy**
- **CB-NAAT**
- **Acid-fast bacilli**
- **Culture**

**Microscopic Examination**

- **Findings**
  - Clear
  - Straw yellow
  - 50 cells/cumm
  - Mononuclear/lymphocyte: 70/30
  - 72
  - 2.9
  - Apoptotic cells and a few reactive mesothelial cells. No malignant cells seen
  - Mycobacterium tuberculosis not detected
  - Not seen
  - Sterile

**2D ECHO**

- **Findings**
  - Massive pericardial effusion with RA, RV collapse

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