TUBERCULAR PERICARDIAL EFFUSION COMPLICATED BY CARDIAC TAMPONADE: A RARE CASE REPORT

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ABSTRACT

Cardiac tamponade is a medical emergency which requires early diagnosis and intervention. Tubercular pericardial effusion is an uncommon manifestation of extra pulmonary TB with cardiac tamponade being even rarer. Here, we have a case of 78-year-male who presented with fever, chest pain and shortness of breath for 4 days. He was in respiratory distress, hypotensive along with raised JVP and muffled heart sounds. Echocardiography demonstrated cardiac tamponade for which immediate pericardiocentesis was performed. Pericardial fluid analysis was suggestive of tubercular origin for which ATT and steroids started.

KEYWORDS

Cardiac tamponade, Pericardiocentesis, Tuberculosis

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CASE REPORT

INTRODUCTION

Tubercular pericarditis is a rare form of extra pulmonary TB, accounting for only 1–2% of all TB infections.¹ Among those patients with TB pericarditis, 10% can develop cardiac tamponade which is a life-threatening emergency.² Cardiac tamponade is characterized by progressive intrapericardial accumulation of fluid leading to increased intrapericardial pressure and compression of the heart subsequently resulting in obstructive circulatory shock or even cardiac arrest.³

CASE REPORT

A 78-year-old non-smoker male with history of pulmonary tuberculosis 20 years back which was cured after full course of Anti-tubercular therapy, presented to our emergency department in February 2024 with fever for 4 days. Fever was associated with shortness of breath which progressed from NYHA grade I to IV in 4 days duration. He also complained of chest pain, pricking type exacerbated by leaning forward. At presentation, his GCS was 15/15. No signs of pallor, icterus, lymphadenopathy, cyanosis, clubbing or edema were observed. The patient was in respiratory distress with a respiratory rate of 28 breaths per minute, using accessory muscles of respiration and SPO₂ of 82% in room air. Jugular venous pressure was raised (10 cm). Blood pressure was only 80/60 mmHg and heart rate were 156 beats per minute (irregular, thready). On auscultation, there was minimal wheeze on bilateral chest. The first and second heart sounds were muffled with no added sounds, murmur or pericardial rub.

ECG demonstrated global ST segment elevation, electrical alternans and atrial fibrillation with fast ventricular rate (Figure 1). Chest x-ray showed right upper lobe fibrosis with cardiomegaly (Figure 2). Total leucocyte count was 14,000 (N27 L67 E01 M05 B0), hemoglobin 12.8 gm/dL and platelet count of 2,99,000 cells/cumm. HIV serology was non-reactive. Echocardiography revealed thickened pericardium with moderate pericardial effusion and echogenic strands inside pericardial sac (figure 3). Diastolic collapse of right ventricle and septal bouncing was also noted.

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Figure 1. ECG showing global ST segment elevation, electrical alternans and atrial fibrillation with fast ventricular rate



Figure 2. Showing chest x-ray with right upper lobe fibrosis with cardiomegaly

The patient was kept on ionotropic support and oxygen echocardiography guided supplementation. Urgent pericardiocentesis was done. Around 100 ml of thick purulent fluid drained and sent for laboratory evaluation (Figure 4). As the patient's ECG was still denoting atrial fibrillation with ventricular rate of 162 bpm, the patient was started on amiodarone infusion. The pericardial fluid report was suggestive of tuberculosis: Total cell count 21,000/cumm with 90% lymphocytes and 10% neutrophils. Glucose was low (53 mg/dL), protein was high (5.9 g/dL) and ADA value was 132.2 U/L. Likewise, gram stain and AFB stain of fluid were negative. Hence, patient started on anti-tubercular drugs along with steroids. After 6 hours, the patient's BP was maintained at 100/60 mmHg on dual inotropic support with noradrenaline and dobutamine, heart rate was 132 bpm. Antitubercular drugs along with supportive therapies were continued and planned for pericardial catheterization.



Figure 3. Echocardiography revealing thickened pericardium with moderate pericardial effusion and echogenic strands inside pericardial sac

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Figure 4. Pericardial fluid sample

DISCUSSION

This case demonstrates various clinical as well as ECG/ echocardiographic findings typical of cardiac tamponade which is quite rare in clinical practice. Patients with tuberculosis make up only 1-2 % of pericardial effusion. Other common causes include trauma, viral/bacterial infections. renal failure, autoimmune disease, hypothyroidism, ovarian hyperstimulation syndrome, and idiopathic.⁴ The occurrence of classic Beck's triad in cardiac tamponade is unusual. A study done on 150 patients by Stolz et al found that none of the patients with cardiac tamponade had all three findings of Beck's triad.⁵ Interestingly, our case had classical triad of tamponade which is uncommon. The classically taught ECG finding in cardiac tamponade is low voltage ECG with electrical alternans. Low voltage QRS is thought to exist due to the presence of fluid that occurs in between the myocardium and ECG lead. Electrical alternans is present due to the swaying heart phenomenon as it sways from side-to-side within the pericardial effusion. In a recent study by Chandra et al, these ECG findings were found to be neither sensitive nor specific. Low voltage QRS was found in 29% of patients while electrical alternans was found in 23%.⁶ Our case did not have low voltage ECG but electrical alternans was noticed. Additionally, diffuse ST elevation was present which is more specific for pericarditis. This implies that though ECG can be a good first line screening tool, the diagnostic accuracy is usually not so precise.

Besides history, examination and ECG, echocardiography remains the mainstay of tool for prompt diagnosis of pericardial effusion and tamponade. Echocardiography still remains the imaging modality of choice to assess the pericardium due to its ease of use, availability, cost-effectiveness and its comprehensive appraisal of the heart and its hemodynamics.⁷ The common signs on echocardiography are pericardial effusion, right atrial and right ventricular collapse during relaxation phase, diastolic ventricular size variability with respiration, IVC plethora (92% sensitivity), and septal bounce.⁷ Our patient had all of the above signs except IVC plethora which could not be demonstrated.

Pericardiocentesis is the most useful therapeutic procedure for the early management or diagnosis of large, symptomatic and cardiac tamponade.8 Jn pericardial effusion hemodynamically unstable patients, an emergent procedure is mandatory because only the removal of fluid allows a normal ventricular filling and restores an adequate cardiac output.9 As our patient was also hemodynamically unstable, we performed urgent echocardiography guided guided pericardiocentesis. Echocardiography pericardiocentesis is the best available therapy for initial management of cardiac tamponade. It is simple, safe, and effective for removing pericardial fluid and reversing hemodynamic instability, and the use of a pericardial catheter for extended drainage has been associated with significant reduction in recurrence of fluid accumulation.¹⁰ For the diagnosis of tubercular pericardial effusion, pericardial fluid ADA value ≥ 40 U/L has 87% sensitivity and 89% specificity.¹¹ Our case had pericardial fluid ADÅ value of 132.2 U/L suggestive of tuberculosis. Additionally, fluid had high protein (5.9 g/dL) and low sugar (53 mg/dL) which further suggest towards tuberculosis. As per the WHO guidelines, antitubercular drugs and steroid were instituted on him although the WHO recommendation for use of steroid in tuberculous pericarditis is conditional with very low certainty in evidence.

CONCLUSION

The early diagnosis of cardiac tamponade is crucial as the delay can be life threatening. Moreover, tuberculosis as a cause of pericardial effusion is a rare entity which requires a high index of suspicion. Though challenging, emergency pericardiocentesis can be a lifesaving procedure that every physician should be competent in.

CONFLICT OF INTEREST

None

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