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Case Report
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Pericardial Decompression Syndrome in a Case of Rheumatic Heart Disease

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Abstract

Pericardial decompression syndrome is a life-threatening condition. Pericardial effusion in the setting of rheumatic heart disease is rarely associated with cardiac tamponade. We hereby present a case of rheumatic heart disease with severe mitral stenosis and severe aortic regurgitation who had cardiac tamponade and required emergency pericardiocentesis. But thereafter, he developed acute pulmonary oedema and refractory hypotension due to pericardial decompression. Pericardial decompression can be prevented by the removal of fluid in aliquots.

Abbreviations

ADHF: Acute Decompensation of Heart Failure; AR: Aortic Regurgitation; LV: Left Ventricle; MS: Mitral Stenosis; NYHA: New York Heart Association Functional Class; PDS: Pericardial Decompression Syndrome; RA: Right Atrium; RV: Right Ventricle

Case Presentation

Introduction: Rheumatic heart disease is still prevalent in developing countries [1]. Pericardial effusion and cardiac tamponade are rare in rheumatic heart disease and infective endocarditis [2-4]. Pericardiocentesis is required in cardiac tamponade and pericardial decompression syndrome is one of its complications. Pericardiocentesis in presence of pulmonary artery hypertension and valvular heart diseases needs to be done with utmost care and hemodynamic monitoring. Sudden withdrawal of a large amount of pericardial fluid can result in hemodynamic instability.

History of present illness: A 20-year-old workman initially presented to a peripheral health center with a fever for the last 3 weeks, worsening dyspnea (NYHA Class I→ III), fatigue and retrosternal chest heaviness. He had a poor response to initial medical care received at the peripheral center and was referred to a higher center for further management. But he had progressive symptoms and presented to us in poor general condition with NYHA Class IV dyspnea with orthopnea. On admission, his pulse rate was 130/min, low volume, regular, blood pressure was 90/40 mmHg and respiratory rate of 38/min. His O_2 saturation was 99% on room air. His JVP was distended up to the angle of the jaw while sitting, with prominent × descent. The heart sounds were muffled with a gallop rhythm.

Past medical history: At around 9 to 10 years, he had migratory arthritis involving the right knee, left knee and right elbow. He was diagnosed with rheumatic fever and was advised of treatment and secondary rheumatic prophylaxis. Medical advice was not followed because of illiteracy and affordability. The patient remained in good functional capacity till the present episode of illness.

Investigations: Recent chest X-ray revealed cardiomegaly with straightening of the left heart border with wide carina and double atrial shadow suggestive of LA enlargement with prominent vascular markings in the upper lobes of both lungs (Figure 1a). His WBC count was 30.8 × 10^9/L with neutrophilia. ECG revealed sinus tachycardia with low voltage complexes.

Echocardiography: RHD was evident by the presence of thickened AML and restricted movement of PML with severe mitral stenosis (mitral valve area- 0.7 cm^2, mean gradients of 18 mmHg @ heart rate of 130 per minute. His aortic valve was thickened and there was vegetation on the non-coronary cusp leading to severe aortic regurgitation. There was moderate pulmonary hypertension with an estimated RV systolic pressure of 50 mmHg. Large circumferential pericardial effusion with maximum dimensions measuring 38 mm anterior to RV in RV diastole was noted with intra-pericardial strands. There was diastolic inversion of the right atrium. IVC was distended with <50% respiratory variability (Figure 2).

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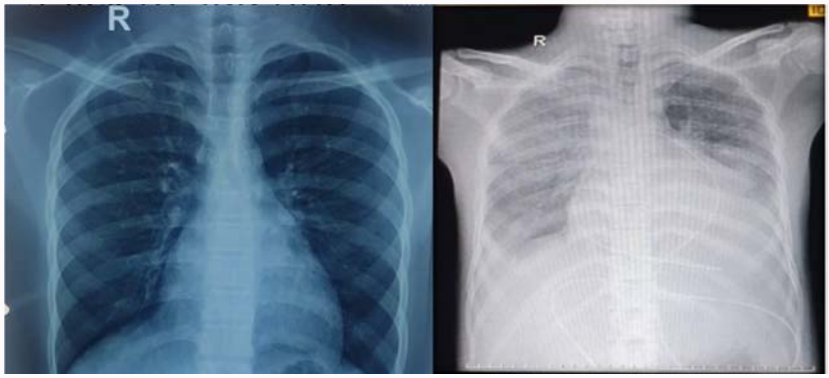

Figure 1: a) Chest X-ray (of the previous admission) showing straightening of the left heart border; b) Chest X-ray shows a pigtail catheter in the pericardial cavity and there is evidence of pulmonary oedema.

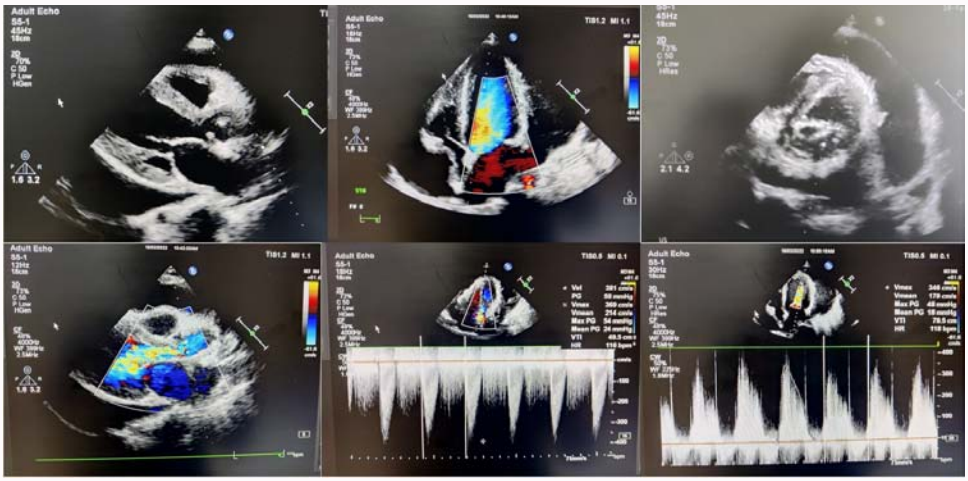

Figure 2: Echocardiographic findings: a) Large pericardial effusion, thickening of both mitral valve leaflets and aortic valve in PLAX view (Left upper); b) A4C view showing large pericardial effusion and inversion of RA (mid-upper); c) The PSAX view showing the fish mouth-like opening of the mitral valve with commissural fusion and valve area by planimetry was 0.7 cm^2 (right upper); d) Severe aortic regurgitation evident on PLAX view (left lower); e) The mean pressure gradient across the aortic valve is 24 mmHg (mid-lower); f) The mean pressure gradient across the mitral valve is 18 mmHg (right lower).

Management: The patient was admitted to the cardiac critical care unit. Emergency pericardiocentesis was planned for diagnostic as well as therapeutic intent. About 1200 ml of serosanguinous pericardial fluid was drained slowly under hemodynamic monitoring. There was a dramatic initial improvement in his symptoms. But the condition of the patient deteriorated suddenly after the procedure as soon as he was shifted back to CCU. He suddenly developed respiratory distress. An immediate echocardiographic evaluation didn't reveal re-accumulation of pericardial effusion. The patient was intubated and placed on mechanical ventilation after acute refractory respiratory distress. His chest X-ray was suggestive of pulmonary oedema (Figure 1b) and there was no residual pericardial fluid on repeat echocardiographic assessments. He had a rapid downhill course and also developed refractory hypotension; he collapsed despite intensive resuscitative measures.

Discussion

Rheumatic heart disease is endemic in India, with the highest estimated number of cases and deaths [1].

Rheumatic heart disease remains a leading cause of infective endocarditis in the adult population. Pericarditis is seen in ~10% of acute rheumatic carditis but cardiac tamponade is very rare. There are only isolated case reports of cardiac tamponade in acute rheumatic carditis [2,3]. Pericardial effusion in infective endocarditis is known to occur and cardiac tamponade is very rare in such settings and also results in poor prognosis [4,5].

The definite cause of pericardial effusion could not be established in this case and the possibilities include acute rheumatic pan carditis, tuberculosis, and bacterial infective endocarditis with rupture of aortic root leading to acute severe aortic regurgitation.

A possible explanation for acute hemodynamic collapse, after pericardiocentesis, is existing severe mitral stenosis and severe acute aortic regurgitation may have led to acute pulmonary oedema following an increase in RV preload after pericardiocentesis.

Pericardial decompression syndrome was considered as the patient had hypotension and pulmonary edema after pericardiocentesis.

The hemodynamic features of such a combination- severe MS with severe AR with cardiac tamponade have not been described. Our patient also had pulmonary hypertension and the safety of pericardiocentesis in presence of PAH is not established [6,7].

Pericardiocentesis is a lifesaving procedure in cardiac tamponade. Complications are known to occur. Pericardial Decompression

Syndrome (PDS) is rare and has poor outcomes.

The exact mechanism of PDS is still not clear. Various possible mechanisms have been proposed for PDS, such as preload-afterload mismatch, sudden sympathetic withdrawal following pericardiocentesis and cardiac tamponade related myocardial stunning.

In this case, sympathetic overdrive resulted in relatively high systemic vascular resistance. Removal of pericardial fluid resulted in a sudden increase in venous return. The presence of severe mitral stenosis and severe aortic regurgitation and increased SVR resulted in preload-afterload mismatch and thus worsened heart failure.

Removal of pericardial fluid under invasive monitoring with the right heart and pericardial pressure monitoring could have added to the management of our patient. Removal of pericardial fluid in aliquots has also been proposed to prevent PDS [8,9].

Conclusion

Pericardial decompression syndrome is a rare complication of pericardiocentesis in cases of cardiac tamponade. Pericardiocentesis should be done with intensive hemodynamic monitoring, especially in coexisting valvular heart disease.

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