Unexpected outcome (positive or negative) including adverse drug reactions

Reversible severe biventricular dysfunction postpericardiocentesis for tuberculous pericardial tamponade

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Summary

Transient severe biventricular systolic dysfunction is a rare phenomenon postpericardiocentesis. Here, the authors report the clinical scenario of a young male who presented with pericardial tamponade that was tuberculous in origin. Soon postpericardiocentesis, he had biventricular dysfunction that recovered spontaneously. The authors discuss the clinical presentation, investigations and pathogenesis of this complication.

BACKGROUND

This case represents a phenomenon of a rare complication that can develop postpericardiocentesis. The marked deterioration of both ventricles postprocedure, and subsequent rapid recovery with inotropic support, led us to investigate such behaviour in the literature and report this particular case as one the few available for consideration.

CASE PRESENTATION

The patient was a 17-year-old Indian male student, with no significant medical history. He had spent his summer vacation in India, 2 months prior to being admitted to the medical ward with epigastric pain, vomiting and diarrhea. Although he had developed a fever a few days earlier, he denied chest pain, shortness of breath or palpitations. Upon admission, he was mildly pyrexemic with a temperature of 37.8°C, heart rate of 114 beats/min and blood pressure of 99/63 mm Hg. Cardiovascular examination was unremarkable, apart from tachycardia attributed initially to his mild fever. Abdominal examination revealed tender hepatomegaly 3–5 cm below the costal margins, with no splenomegaly.

INVESTIGATIONS

His initial laboratory results were as follows:

- ▶ White blood cell count: $11.1 \times 10^9/l$
- ▶ Haemoglobin: 11.8 mg/dl with normocytic and hypochromic indices
- ▶ Platelets: $261 \times 10^9/l$
- ▶ Serum amylase: 49 U/l

Routine and microscopic urinalysis was normal. Liver function tests revealed a low albumin level of 29 g/l with a mild elevation of alanine transaminase level at 62 U/l. Urea and electrolyte levels were normal. Septic investigation was performed, and empirical antibiotics were started. HIV and hepatitis screenings were negative. Abdominal ultrasonography showed moderate hepatomegaly with mild ascites. Plain chest x-ray showed evidence of bilateral pleural effusion.

Engorgement of the neck veins was noticed on the second day of admission.

ECG changes showed a sinus rhythm of 97 beats/min with electrical alternans noticed in the precordial leads (figure 1).

Bedside echocardiography showed massive pericardial effusion consistent with pericardial tamponade. The right ventricle and right atrium were collapsed, and the septum was noted to have paradoxical movements. The estimated ejection fraction was 62%. The hepatic veins were non-collapsing.

TREATMENT

The patient was transferred to the cardiac unit for pericardiocentesis, which was performed under aseptic conditions and local anaesthesia with echocardiographic guidance through the left fourth intercostal space. Seventy millilitres of fluid was aspirated. A clamped pigtail catheter was left in the pericardial space and was intermittently released to gradually drain the remaining fluid. The plan was to drain 100 to 150 ml gradually over 6 h. The procedure was uneventful, but 4 h later, after draining 150 ml of pericardial fluid, he developed hypotension with blood pressure of 88/45 mm Hg. Therefore, fluid therapy and inotropic support (dopamine and dobutamine) were administered.

The pigtail drain was kept in place until the next day, and a total of 450 ml was drained. Repeated echocardiography showed normal left ventricle dimensions; however, severe biventricular systolic dysfunction was noted with an ejection fraction of 10–15%. The ejection fraction was calculated using the four- and two-chamber Simpson's method.

Furthermore, mild pericardial effusion with moderate mitral and tricuspid regurgitation was also noted. Serial ECGs showed no evidence of ischaemia to support traumatic myocardial or coronary injury. Cardiac marker levels (creatinine kinase/troponin I) were normal pre and postpericardiocentesis.

CT of the chest and abdomen showed bilateral moderate to large pleural effusion with compressive atelectasis; a lymph node measuring 1–2 cm was also noted in the pretracheal region. Multiple necrotic lymph nodes were noticed

Figure 1 ECG showing electrical alternans.

in the mesentery in the lower abdomen with thickened lower bowel loops. Infectious and Rheumatology teams were consulted, and tuberculous aetiology was considered as the primary differential diagnosis; however, collagenosis was also considered since it shares similar clinical manifestations with the former.

The results of pericardial aspirate examination were as follows:

- ▶ Albumin: 20 mg/l
- ▶ Glucose: 5.8 mmol/l
- ▶ Lactate dehydrogenase enzyme level: 685 U/l
- ▶ Protein: 36 mg/l
- ▶ Red blood cells: 28 000/mm²
- ▶ White cell count: 1730/mm² (lymphocytes: 92%; polymorphonuclear leucocyte: 2%; macrophages: 6%)

Gram staining showed intense lymphocytosis reaction, but the initial cultures were sterile and the acid fast bacilli (AFB) smear was negative.

Vasculitis screening was also negative.

The patient continued to be pyrexial despite antibiotic therapy. In view of the patient's ethnic group, recent travel to India, lymphocytosis in pericardial fluid and the presence of necrotic lymph nodes in the abdomen, along with the clinical presentation, antituberculosis (anti-TB) therapy in the form of isoniazid, rifampicin, pyrazinamide, ethambutol and vitamin B6 was instituted.

Upon the commencement of anti-TB therapy, the patient began to show dramatic improvement in his appetite and was afebrile within the first 24 h. Left ventricular systolic dysfunction began to recover by the third day postpericardiocentesis, and by the fifth day, it had almost normalised (figure 2). The blood pressure started to normalise; subsequently, inotropic drug support was tapered off and discontinued. Prednisolone therapy was started on the fourth day of commencing anti-TB therapy with a dose of 30 mg/day. The steroid therapy was delayed in order to avoid masking

other aetiologies like systemic lupus erythematosus (SLE) that can respond to high doses of steroids.

OUTCOME AND FOLLOW-UP

The rest of the patient's clinical course was uneventful, and he was discharged on the ninth day after commencing anti-TB therapy. Upon serial follow-up in the cardiology outpatient department, he was noted to be asymptomatic and maintained good health. Subsequent AFB cultures at 4 weeks were noted to be positive.

The patient was discharged from the cardiology clinic at the ninth month after commencing anti-TB chemotherapy, with no recurrence of pericardial or pleural effusions. He maintained good health and appetite and remained asymptomatic in the follow-up period.

DISCUSSION

While this rare phenomenon of transient systolic dysfunction postpericardiocentesis has been recognised in recent literature, unique to this reported scenario is the presentation of florid biventricular deterioration of cardiac function (few hours postprocedure) and its rapid restoration (fifth postprocedure day) (figure 1).

This phenomenon has mostly been reported to develop soon after relieving malignant pericardial effusion.¹⁻⁴ Over the past 3 years, however, the tuberculous aetiology is being recognised and noted as a cause.^{1 5} While this is the third reported case of severe systolic dysfunction post-tuberculous pericardial drainage, it is the first in regards to a patient of such an age and gender^{1 8} (table 1).

The pathogenesis remains obscure despite the diversity of the available speculations. One of the anticipated mechanisms is coexistent left ventricular dysfunction at the time of the tamponade that is unmasked after relieving the pericardial effusion.^{1 4 6} However, the rapid recovery of the contractile reserve with simple supportive therapy where

Figure 2 Ejection fraction over time.

Table 1 Summary of reported cases of tuberculous pericardial tamponade and systolic dysfunction postpericardiocentesis

Reference No	Age	Gender	Complications postpericardiocentesis
1	25	Male	Cardiogenic shock
8	42	Female	Left ventricular apical akinesia with thrombus formation and pulmonary oedema
Current case	17	Male	Cardiogenic shock

no heart failure medications are instituted (β -blockers or ACE inhibitors) argued against this possibility. Further supporting evidence against the theory of coexistent left ventricular dysfunction is the prolonged recovery time needed for myocardial dysfunction in patients who present with both pericardial effusion and left ventricular dysfunction, as in SLE where the recovery can take weeks despite active antitardiac failure and immunosuppressive therapy.⁷

The next theory is referred to as the *haemodynamic hypothesis*, which is related to the rate and volume of the pericardial fluid initially drained. The pulmonary oedema in this scenario is precipitated by the mismatch between the preload and after-load volumes.^{2 8 9} Such a complication was well-anticipated: a vigilant draining procedure allowed for an initial 70 ml of pericardial fluid to be accurately removed. The patient, however, went into severe biventricular failure. This also contradicts the haemodynamic theory.

The report by Skolidis *et al* that describes coronary circulation during pericardial tamponade warrants special attention. They demonstrated the gradual decline in coronary perfusion during the tamponade despite a normal response to adenosine. In other words, the decline in the coronary perfusion is mainly related to hyperstimulated catecholaminic response rather than to the elevated pericardial pressure.¹⁰

The physiology of the human coronary circulation during tamponade supports the role of the over-activated

sympathetic nervous system. The compromised coronary circulation may lead to myocardial stunning and hibernation precipitated by the sudden withdrawal of the overactive catecholaminic drive after the pericardium is relieved.^{1 2 4 6} As the bulk of the patients who have exhibited this complication are known to harbour neoplasms or tuberculosis as the underlying disease – both being leading causes of constriction – tuberculosis may contribute to this complication.^{1 8}

Learning points

- The main mechanism explaining the transient systolic dysfunction postpericardiocentesis is the coronary hypoperfusion related to augmented coronary vascular resistance.
- The role of pericardial constriction needs to be further elucidated.

Competing interests None.

Patient consent Obtained.

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