CASE REPORT

Tuberculous myopericarditis: a rare presentation in an immunocompetent host

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SUMMARY

Tuberculosis is a common cause of pericardial disease in India. Myocardial involvement, although well described in the literature, is a rare manifestation of tuberculosis. We report a patient with disseminated tuberculosis and myopericarditis manifesting as cardiogenic shock. The patient gradually improved on antituberculosis drug therapy, steroids and an evidence-based guideline driven therapy for heart failure. Follow-up imaging showed calcification of the pericardium and improvement of his left ventricular systolic function.

BACKGROUND

Myocarditis secondary to *Mycobacterium tuberculosis* is an uncommon manifestation of a very common and prevalent infection. We present this case as few causes of myocarditis are reversible; tuberculosis being one of the exceptions. Hence, it is important to consider this diagnosis in patients presenting with a cardiogenic shock from myocarditis.

CASE PRESENTATION

A 28-year-old Indian man presented with 20 days history of low-grade fever, abdominal discomfort, oliguria and facial and pedal oedema. He reported exertional dyspnoea with orthopnoea, which worsened to class IV over a period of 3 days. There was no history of cough or haemoptysis. There was no known history of tuberculosis, rheumatic or ischemic heart disease or any other medical illness. Physical examination revealed a tachycardic (126/min regular), a hypotensive (60 mm Hg) man with elevated jugulovenous pressure of 14 cm of water, S3 gallop, bibasilar crackles and pedal edema. A pericardial friction rub was present.

Peripheral extremities were cold and clammy. No lymphadenopathy, clubbing or central cyanosis was appreciated.

INVESTIGATIONS

Intensive supportive care for his cardiogenic shock was begun, while the diagnostic work-up to determine the aetiology was started. Basic work-up begun with chest radiography that showed cardiomegaly and an ECG that revealed incomplete right bundle-branch block with diffuse ST-segment elevations (figure 1). Cardiac troponins were positive. For the diagnosis of myocarditis he underwent a 2-Dimensional (2D) echocardiogram which revealed severe global hypokinesia and left ventricular systolic dysfunction (ejection fraction: 35%). Cardiac MRI was performed to rule out any infiltrative cardiomyopathy, which showed an evidence of non-infiltrative cardiomyopathy as there was late gadolinium enhancement. After haemodynamic stabilisation, there was a high clinical suspicion of tuberculosis based on the presentation, hence we followed this with a CT scan of the chest and the abdomen which revealed enlarged mediastinal, mesenteric and ileocaecal lymph nodes suggestive of disseminated tuberculosis. Fine needle aspiration (FNA) of an enlarged lymph node revealed caseating granulomas with the identification of acid-fast bacilli positive consistent with *M tuberculosis*. HIV serology was negative.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis of myocarditis

Figure 1 ECG on presentation.

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Figure 2 Pericardial calcification.

1. Infections: viral, bacterial, protozoal, helminthic, fungal, rickettsial, spirochetal and mycobacterial;
2. Autoimmune disease: giant cell, hypereosinophilic, sarcoidosis and collagen vascular disease;
3. Hypersensitivity reaction to drugs;
4. Toxins.

TREATMENT

The patient was initially started on inotropes for a cardiogenic shock and intravenous steroids were initiated. On the basis of the CT scan, the cardiac MRI and FNA of the lymph node, a diagnosis of disseminated tuberculosis involving the myocardium manifesting as a cardiogenic shock, was made. Four-drug ATT was initiated with adjunctive glucocorticoids.

OUTCOME AND FOLLOW-UP

Follow-up at 2 months showed complete resolution of the ST changes, with T-wave inversions and normal left ventricular systolic function on 2D echocardiogram. Repeat CT of the chest revealed pericardial calcifications with regression in the size of lymph nodes (figure 2). At the end of the second month of treatment he complained of severe left hypochondriac pain without any associated nausea or vomiting. CT scan of the abdomen revealed a cold abscess with destruction of the ninth rib (figure 3). Ultrasound-guided aspiration of the cold abscess was performed and tissue was sent for culture and histopathology. The results of which were consistent with *M tuberculosis*.

Figure 3 A cold ninth rib abscess on the left side with abdominal lymphadenopathy.

He completed a total treatment course of 9 months without any further complications or toxicities from ATT. He continues to follow-up biannually for the evaluation of any signs of constrictive pericarditis.

DISCUSSION

Cardiac tuberculosis is a rare but well-recognised condition that was first reported several decades ago.¹ It is estimated that 1% of all the cases of tuberculosis have cardiac involvement, predominantly affecting the pericardium.² Endocardial, myocardial, valvular or coronary arterial involvement are rare. An anatomical predilection for the right-sided mediastinal lymph nodes has been described in this condition, making the right side of the heart the most vulnerable area of the myocardium owing to the potential for contiguous spread.³

Horn and Saphir¹ have described three histological types of myocardial tuberculosis: (1) nodular tubercles (tuberculomas) of the myocardium, varying from pea to egg size, with central caseation, (2) miliary tubercles of the myocardium complicating generalised miliary disease and (3) the uncommon diffuse infiltrative type usually associated with tuberculous pericarditis in which the myocardium is diffusely infiltrated by the granulation tissue containing the giant cells, endothelial cells and the lymphocytes. Tubercular myocarditis can remain clinically asymptomatic (with a diagnosis being made at autopsy) or can present with sudden cardiac death,^{4–6} intractable ventricular arrhythmias, long QT syndrome,⁷ high-grade heart-block⁸ or congestive heart failure.⁹ Sudden cardiac death related to tuberculosis is rare and is usually attributable to bronchopneumonia and complications of pulmonary cavitation leading to massive haemoptysis from rupture of Rasmussen's aneurysm.¹⁰

Increasing recognition of the entity and the use of endomyocardial biopsy may help in detecting more cases of such a curable form of cardiomyopathy. The case presented in this report is a singular example of myopericardial involvement in tuberculosis.

Learning points

- Recognise the presentation of myocarditis.
- Identify tuberculous myopericarditis as a reversible cause of myocarditis.
- Know the aetiology of myocarditis.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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