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Recommended Citation
Chun Kit Wong, Yeuk Chi Choi, Tuberculous pericarditis Journal of the Hong Kong College of Cardiology
2022;29(2):95-99 https://doi.org/10.55503/2790-6744.1203

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CASE REPORT

Tuberculous Pericarditis

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Abstract

Tuberculous (TB) pericarditis, a severe extrapulmonary manifestation of Mycobacterium tuberculosis, is notorious for its challenging presentation. We report a case of effusive-constrictive pericarditis due to tuberculosis infection.

Keywords: Tuberculosis, Mycobacterium tuberculosis, Pericarditis, Constrictive pericarditis, Effusive-constrictive pericarditis, Pericardial effusion

Introduction

Mycobacterium tuberculosis (MTB) is notorious for its extrapulmonary involvement. Cardiac involvement, in the form of effusive-constrictive pericarditis, has been scarcely described in the literature. We report a case of tuberculous pericarditis, successfully managed after timely echocardiographic, microbiological, and endoscopic workup.

Case report

A 63-year-old man was admitted to the intensive care unit for high fever and shortness of breath. He reported increasing exertional dyspnea and lower limb swelling for one week. On admission, the white cell count was normal (8.2 × 10^9/L); there was mild anemia (hemoglobin 12.1 g/dL), with a high erythrocyte sedimentation rate (103 mm/hr). His renal function was normal (creatinine 61 μmol/L), and there were elevated liver enzymes (alkaline phosphatase 142 U/L, alanine aminotransferase 89 U/L). Electrocardiogram demonstrated low voltage over limb leads, and the chest X-ray showed gross cardiomegaly. Bedside focused echocardiogram to evaluate cardiomegaly found massive circumferential pericardial effusion, more than 4 cm measured from the apical window, which quickly led to a pericardiocentesis draining around 2 L of blood-stained pericardial fluid.

However, the patient's clinical condition continued to deteriorate despite drainage of pericardial effusion, with worsening respiratory distress and multiorgan dysfunction. Urgent tracing of the biochemistry of pericardial fluid revealed an exudative nature, with high protein (55 g/L) and lactate dehydrogenase level (521 U/L); however, bacterial culture, acid-fast bacilli smear, MTB PCR of the pericardial fluid were negative. The only hint of MTB as a unifying diagnosis over other differentials e.g. rheumatological disease or neoplastic processes was an elevated pericardial adenosine deaminase (ADA) level up to 45 U/L, with a cut off quoted to be 40 U/L.

Urgent computed tomography of the thorax with contrast was ordered, which revealed apart from nonspecific consolidative changes of lung parenchyma, multiple prominent paraaortic, paratracheal and hilar lymph nodes, with the largest one measuring 2.3 cm in the right paratracheal region. Thickened pericardium with contrast enhancement was suggestive of active pericarditis. Repeat trans-thoracic echocardiogram showed interval reduction of pericardial effusion (Figure 1), but overt constrictive features: septal bounce, dilation of both...
atria with congested inferior vena cava, exaggerated respiro-phasic changes of mitral, tricuspid, and pulmonary vein diastolic inflow (Figure 2), expiratory diastolic flow reversal in hepatic vein (Figure 3), annulus reversus, and pulsus paradoxus, consistent of an effusive-constrictive physiology, and thus explaining the clinical deterioration with pulmonary congestion and hepatic and renal derangement.

Bronchoscopy was scheduled urgently as microbiological proof is the key. No endobronchial lesions were noted upon examination of the airway; bronchoalveolar lavage was performed to the right upper lobe; and lastly endobronchial ultrasound was able to guide needle biopsy of the right paratracheal lymph node transbronchially. It was the lymph node that finally gave microbiological proof of MTB, with MTB PCR positive in the cell block and histology showing granuloma. Antimicrobials were streamlined to amikacin, ethambutol, and levofloxacin; and corticosteroids, initially with intravenous hydrocortisone.
100 mg every eight hours, was started for effusive-constrictive pericarditis.

The patient improved rapidly after commencement of anti-tuberculous medications and corticosteroids, weaning off noninvasive ventilation support, and evaded the need of acute dialysis. His organ parameters normalized, and standard anti-tuberculous therapy was introduced carefully, using isoniazid, rifampicin, ethambutol and levofloxacin. 1 week after the introduction of rifampicin, a strong enzyme inducer, moderate doses of hydrocortisone were switched to oral prednisolone at 0.5 mg/kg/day, tapering by 10 mg/day each week over 4 weeks, while carefully monitoring for any rebound inflammation. Progress echocardiogram showed resolution of all constrictive features; right heart catheterization was deemed not necessary due to dramatic clinical improvement. The patient was discharged at week 3 of hospitalization. On clinical follow up subsequently, *Mycobacterium tuberculosis* was grown from pericardial fluid and paratracheal lymph node.

**Discussion**

This case illustrates the difficulty of diagnosing extrapulmonary *Mycobacterium tuberculosis* infection, and the race with time in attaining microbiological proof of tuberculosis before correct treatment can be initiated. The laboratory use of adenosine deaminase level in pericardial fluid aids to reduce diagnostic uncertainties, by providing clear cutoff with acceptable sensitivity (88%) and specificity (83%) [1]. PCR tests of MTB DNA have further reduced the time in diagnosis.

The diagnosis of constrictive pericarditis has gradually moved from gold-standard invasive right heart catheterization to multiple specific echocardiographic parameters. The Mayo Clinic Criteria [2] selected five principal echocardiographic variables, all independently associated with surgically proven constrictive pericarditis. Apart from the well-recognized respiration-related ventricular septal shifts and preserved or increased medial mitral annular e'...
velocity, hepatic vein expiratory diastolic flow reversal has gained its place as one of the good differentiators with other cardiomyopathies e.g., restrictive cardiomyopathy. In our case, the transient nature of inflamed pericardium and the expectation of rapid response to corticosteroids led to the cautious omission of right heart catheterization, which may prove difficult to perform in acutely ill patients.

The addition of corticosteroids in tuberculous pericarditis with effusion received a Class IIb (Level C) evidence in the 2015 European Society of Cardiology Guideline [3]; while the 2016 American Thoracic Society Clinical Practice Guideline advised against routine corticosteroid therapy for tuberculous pericarditis [4], citing low quality evidence of benefit [5,6]. However, we elected to start corticosteroids in this case due to biochemical and imaging evidence of severe inflammation, and tapered off rapidly once clinical improvement was seen, to avoid the side effects of prolonged steroid therapy [7].

This case report has illustrated timely diagnosis of Mycobacterium tuberculosis infection causing effusive-constrictive pericarditis, with multiorgan dysfunction.
Ethics information
Not applicable.

Acknowledgement and funding
None.

Conflict of interest
None declared.

References


