Tuberculous pericarditis in an older adult: a case report

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ABSTRACT
We report a case of tuberculous pericarditis in a 65-year-old man. Tuberculous pericarditis is an extra-pulmonary tuberculous in endemic areas. Its complications include constrictive pericarditis and cardiac tamponade. Computed tomography and cardiovascular magnetic resonance imaging are important tools to detect pericardial disease. Measurement of adenosine deaminase level in the pericardial fluid is a valuable diagnostic test, with good specificity and sensitivity. Timely administration of anti-tuberculous and steroid therapy reduces the risk of constrictive pericarditis.

Key words: Adenosine deaminase; Pericarditis, tuberculous; Pleural effusion; Steroids

CASE PRESENTATION
In May 2019, a 65-year-old man was admitted to the North Lantau Hospital with a 2-month history of coughs with sputum, weight loss of 2 kg, low-grade fever, and increasing exertional dyspnoea. The patient was a chronic smoker, with chronic obstructive pulmonary disease.

On examination, the patient had high fever, with blood pressure of 100/60 mmHg, pulse rate of 110 per min, and oxygen saturation of 98%. He had no pulsus paradoxus. Chest examination showed reduced air entry with stony dullness on percussion over the left lower zone. The heart sound was normal with no murmur.

Chest radiography showed cardiomegaly and left pleural effusion (FIGURE). Electrocardiography showed sinus tachycardia with reduced voltage, whereas echocardiography revealed a large pericardial effusion but no definite cardiac tamponade. The left ventricular systolic function was normal. Blood tests showed normochromic normocytic anaemia, with haemoglobin of 10.1 g/dL and white cell count 6 ×10⁹/L. There was mild hyponatraemia (sodium level, 128 mmol/L) but otherwise normal renal function and liver transaminases levels. The serum albumin level was 18 g/L and the serum globulin level was 44 g/L. The inflammatory markers were raised, with C-reactive protein of 60 mg/L and erythrocyte sedimentation rate of 58 mm/hr.

Thoracentesis and pericardiocentesis were performed for relief of dyspnoea. Both pleural and pericardial fluids were exudative in nature, and cytology showed no malignant cell. Results of Gram-staining for micro-organism, acid fast bacilli smear, and bacterial culture were all negative. The adenosine deaminase level in the pericardial fluid was 49 U/L (≥40 U/L is suggestive of tuberculous pericarditis with 88% sensitivity and 83% specificity), whereas the level in pleural fluid was 6.9 U/L. The pleural biopsy showed chronic inflammation but no granuloma or malignancy.

The patient was treated with intravenous ceftriaxone. However, he showed no improvement, with progressive shortness of breath requiring supplemental oxygen at 2 L/minute. Serial chest radiography showed further increase in bilateral pleural effusion. The functional status was greatly impaired. Contrast computed tomography of the thorax showed bilateral pleural effusions and mild to moderate pericardial effusion with thickened pericardium. Multiple enlarged bilateral paratracheal
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and hilar lymph nodes were noted but no other lung or mediastinal mass lesion.

In view of the raised pericardial fluid adenosine deaminase level (49 U/L) and the computed tomography findings suggestive of tuberculous pericardial disease with signs of constrictive pericarditis, empirical anti-tuberculous treatment with isoniazid, rifampicin, pyrazinamide, and levofloxacin was started. Ethambutol was not considered because of the patient's poor vision. Oral prednisolone was given in view of features of constrictive pericarditis. After 2 weeks of antituberculous therapy, the patient's condition gradually improved and was discharged.

Serial chest radiography and computed tomography of the thorax showed resolution of pericardial effusion, pleural effusion, and pericardial thickening. Echocardiography revealed only small amount of residual pericardial effusion. After 6 weeks of incubation, the pericardial fluid for acid fast bacilli culture grew Mycobacterium tuberculosis. This confirmed the diagnosis of tuberculous pericarditis. After 6 months of anti-tuberculous therapy plus the initial 6 weeks of oral prednisolone, chest radiography showed complete resolution of bilateral pleural effusion with normal cardiothoracic ratio (Figure).

DISCUSSION

Tuberculous pericarditis is an uncommon manifestation of extra-pulmonary tuberculosis, which can be fatal if treatment is delayed. It is usually due to disease dissemination from the lung, spine or mediastinal lymph node, as well as part of miliary tuberculous infection.1 The clinical presentation is often non-specific, with fever, weight loss, and fatigue. The most common long-term complication of pericardial tuberculosis is constrictive pericarditis, which are reported in 30% to 60% of patients.2 Occasionally, it could present as chronic heart failure or acute cardiac tamponade.

Pathologically, the diseased pericardium will progress from the initial exudative stage to the late adsorptive and constrictive stage, in which pericardial thickening, fibrosis and calcification lead to the clinical syndrome of constrictive pericarditis.3

Figure. Chest radiographs showing (a) cardiomegaly with left pleural effusion on admission and (b) resolution of pericardial and pleural effusion after completion of anti-tuberculous therapy.
Chest radiography may reveal cardiomegaly, and electrocardiography may show non-specific ST segment and T-wave changes. Echocardiography can confirm pericardial effusion and guide subsequent pericardiocentesis. Computed tomography may show features of pericarditis and coexisting pulmonary tuberculosis. Cardiovascular magnetic resonance imaging may delineate the extent of pericardial fluid and confirm any pericardial thickening.

Pericardiocentesis is recommended in all patients with suspected tuberculous pericarditis, and absolutely indicated in the presence of cardiac tamponade. Tuberculous pericardial effusions are typically exudative in nature, with high protein content and lymphocytic-monocytic leukocyte predominance.

The detection rate of tubercle bacilli by direct smear examination of pericardial fluid ranges from 0 to 42%. Positive culture yield of tubercle bacilli from pericardial fluid with conventional method is around 50%. Pericardial biopsy with direct histological identification of tubercle bacilli may be used but is seldom performed, because of the associated operative risk.

Adenosine deaminase is an enzyme whose activity correlates closely with the activation of T-lymphocytes involved in the immune responses to tuberculosis. Measurement of pericardial fluid adenosine deaminase level at a cut-off range of 30 to 60 IU/L gives a diagnostic yield with 90% sensitivity and 70% specificity.

The anti-tuberculous treatment for pericardial tuberculosis is the same for pulmonary tuberculosis, with the standard 6-month regimen. As constrictive pericarditis is a major sequela of pericardial tuberculosis, adjunctive corticosteroid therapy should be used to decrease the pericardial inflammation, which may lead to fibrosis and constriction. However, the American Thoracic Society does not recommend the routine use of steroids in the treatment of tuberculous pericarditis. Steroids might be appropriate in patients at higher risk of inflammatory complications, including those with large pericardial effusions, with high levels of inflammatory cells in pericardial fluid, or with early signs of pericardial constriction.

CONTRIBUTORS
All authors designed the study, acquired the data, analysed the data, drafted the manuscript, and critically revised the manuscript for important intellectual content. All authors had full access to the data, contributed to the study, approved the final version for publication, and take responsibility for its accuracy and integrity.

CONFLICTS OF INTEREST
All authors have disclosed no conflicts of interest.

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DATA AVAILABILITY
All data generated or analysed during the present study are available from the corresponding author on reasonable request.

ETHICS APPROVAL
The patients were treated in accordance with the tenets of the Declaration of Helsinki. The patients provided written informed consent for all treatments and procedures and for publication.

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