Unusual cause of pyopericardium with tamponade

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ABSTRACT

Acute pyopericardium due to tuberculosis is uncommon even in high prevalence countries. We report an unusual case of acute pyopericardium with cardiac tamponade complicating tuberculosis pneumonia of lingula in an adult. Prompt catheter drainage of pyopericardium under echocardiographic guidance and six month course of anti-tuberculosis treatment resulted in complete recovery without any sequelae during the follow-up period of ten months.

Key words: Pneumonia, Pyopericardium, Tuberculosis, Tamponade

INTRODUCTION

Pyopericardium is an uncommon presentation of tuberculosis (TB) and reported in 6.98% cases of pyopericardium. Pyopericardium has been documented in less than 3% cases of large TB pericardial effusions even in high prevalence areas of TB and human immunodeficiency virus (HIV) infection. Pulmonary TB affects pericardium in 1%-2% cases and pericardial TB is responsible for 7% cases of cardiac tamponade. Pericardial TB is usually an insidious illness and may present as acute pericarditis, chronic pericardial effusion, cardiac tamponade or pericardial constriction but purulent pericarditis is rare.

We are not aware of any previous report from India of acute pyopericardium with tamponade as a complication of TB lingular pneumonia. We present an adult case of TB pneumonia with pyopericardium and cardiac tamponade.

CASE REPORT

A 28-year-old non-smoker male was admitted with acute onset of fever, cough and breathlessness of 7 days duration. Fever was high grade and intermittent with chills and rigors. Cough was productive of scanty sputum and associated with left sided pleuritic chest pain. Breathlessness was precipitated by minimal activity. Medical history was unremarkable. On physical examination, patient was sick looking and had bilateral pitting oedema. There was no digital clubbing or cyanosis. He had pulse rate of 128 beats/min, respirations 32 breaths/min, blood pressure 90/60 mmHg, body temperature 102.4°F and room air oxygen saturation 88%. Chest examination revealed features of consolidation over left mammary area. Arterial blood gases at room air were normal. Laboratory reports revealed total leucocyte count of 20,000/mm³ with a differential of 64% polymorphonuclear leucocytes and 29% lymphocytes, haemoglobin 12.1g/dL and erythrocyte sedimentation rate 32 mm at the end of the first hour. Serum biochemistry was normal. Sputum smears were negative for acid-fast bacilli (AFB) and bacteria. Postero-anterior (PA) and left lateral chest radiograph (Figure 1) showed cardiomegaly and lingular consolidation. HIV serology was negative. A 12-lead electrocardiogram showed global...
elevation of ST segment with low voltage complexes. Two dimensional transthoracic echocardiography (Figure 2) showed moderate pericardial effusion. Ultrasound examination of abdomen was normal. Poor general condition of the patient did not allow us to perform flexible fibreoptic bronchoscopy. Computed tomography (CT) of chest could not be done. A provisional diagnosis of acute bacterial pneumonia with pericardial effusion was made and patient was given supplemental oxygen, intravenous antibiotics (ceftriaxone and ciprofloxacin), intravenous corticosteroid (Hydrocortisone hemisuccinate) and other supportive treatment. Blood cultures were negative. *Klebsiella oxytoca* species was reported to have grown in the sputum specimen and antibiotics changed to piperacillin plus tazobactum and gentamycin as per sensitivity report. After two days of treatment, patient complained of worsening chest pain and breathlessness. Clinical examination revealed tachycardia, hypotension, increased jugular venous pressure and pulsus paradoxus indicating development of cardiac tamponade. Patient was shifted to intensive coronary care unit and pericardial drainage was done with placement of 6F pigtail catheter under echocardiographic guidance. Pericardial fluid showed polymorphonuclear leucocytosis and all reports were negative. Approximately 700 ml of thin purulent pericardial fluid drained and catheter was removed after 48 hours. Three days later, patient again developed cardiac tamponade due to re-accumulation of pericardial pus and this time, catheter drainage of pericardial cavity yielded about 550 mL of purulent fluid within 24 hours. Repeat smears and cultures of pericardial fluid were also negative. Mantoux test [1 tuberculin unit (TU)] was positive (12.15 mm). Sputum smears were again negative for acid fast bacilli. As there was no response to seven day course of broad range antibiotics and pericardial drainage, we started daily self-supervised anti-TB treatment with isoniazid, rifampicin, pyrazinamide and ethambutol. Patient improved clinically within seven days and pericardial drainage ceased. Chest radiograph (Figure 3) showed complete pericardial drainage and the pericardial drainage catheter was removed. Patient was discharged with daily anti-TB treatment. At review after four weeks of anti-TB treatment, chest radiograph (Figure 4) showed clearance of alveolar opacities and normal cardiac shadow. The sputum specimen grew *Mycobacterium tuberculosis* at the end of four weeks of inoculation in Lowenstein-Jensen’s (LJ) medium. After two months, pyrazinamide and ethambutol were stopped and anti-TB treatment was continued with isoniazid and rifampicin.
echocardiogram were normal. The patient completed six month course of anti-TB treatment regularly and remained well during the follow up period of ten months.

**DISCUSSION**

Pyopericardium (purulent pericarditis) occurs usually in severe bacterial infections; pneumonia being the commonest predisposing factor followed by sepsis. Pyopericardium is commonly associated with empyema. Involvement of pericardium usually occurs through retrograde lymphatic spread of TB from peritracheal, peribronchial and mediastinal lymph nodes. Contiguous spread from lung TB or by haematogenous spread from a distant primary TB infection is uncommon. In our case, direct spread from contiguous lingular tuberculous pneumonia is the most likely mechanism of development of pyopericardium. Echocardiography is the technique of choice for diagnosis of pericardial effusion and cardiac tamponade. Pericardial drainage under echocardiographic guidance through pigtail catheter is safe and effective. CT or magnetic resonance are better than echocardiography during evaluation of pericardial disease as they can simultaneously show anatomical abnormalities of lung and mediastinal structures with better soft tissue contrast and larger field of vision than echocardiography. Initially, we did not consider TB as aetiology as the illness resembled acute bacterial pneumonia with pyopericardium. As the patient did not show significant clinical improvement with antibiotic therapy and pericardial drainage, the only positive sputum bacterial culture report was considered to be probably due to contamination. In case of pericardial effusion, TB as the aetiological cause can be established through demonstration of AFB in smear or culture of pericardial fluid or pericardial biopsy specimen and histologic examination of pericardial biopsy specimen revealing caseating granulomatous inflammation. The diagnostic yield of AFB from smear and culture of pericardial fluid is variable and was 53% with conventional culture method using LJ medium and increased up to 75% with prompt bedside culture in double strength liquid medium of Kirchner. In our case, all reports of pericardial
not done. The initial negative sputum smear for acid fast bacilli could be due to patient’s inability to submit proper sputum sample due to chest pain. Molecular diagnostic techniques like polymerase chain reaction (PCR) assay of pericardial fluid is useful particularly in developed countries. In high prevalent countries of TB, PCR technique is less sensitive than culture as false positive results may occur due to contamination. In our case, atypical presentation of TB and inability to do early bronchoscopy or PCR led to delay in diagnosis. In our case, isolation of tubercle bacilli from sputum culture and excellent response to anti-TB treatment fulfilled Cherian’s diagnostic criteria of pericardial TB. Management of pyopericardium requires prompt pericardial drainage followed by treatment of underlying cause. But if the pericardial pus is thick, surgical procedures like pericardiectomy in addition to anti-TB treatment is required to manage tuberculous pyopericardium. In countries where TB is highly endemic like India, there should be a high index of suspicion to consider TB in the differential diagnosis of all respiratory infections and carry out appropriate work up for ruling out the same. In view of high prevalence of TB in our country, there is need to avoid use of fluoroquinolones if possible and carefully using fluoroquinolones as initial first line treatment of respiratory infections. In fact, our Revised National Tuberculosis Control Programme (RNTCP) advises that use of fluoroquinolones be restricted to bacterial respiratory tract infections. Since clinical presentation was like acute bacterial pneumonia, ciprofloxacin was added initially as an inexpensive empiric antibiotic to cover possible pseudomonal infection. We used corticosteroids for two weeks during hospital stay and achieved excellent therapeutic response with prompt pericardial drainage and anti-TB therapy alone. Our case highlights the importance of early empiric anti-TB treatment with pericardial drainage for acute pyopericardium in an hyperendemic area of TB like India if clinical response to broad range antibiotics and prompt pericardial drainage is poor.

REFERENCES