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Early diagnosis in an unusual presentation of tubercular pericarditis—A case report

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PEER REVIEW

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Comments

This is a good study and nicely presented. In which the authors evaluated the usefulness of early diagnosis of tubercular pericarditis. The results are interesting and highlight the importance of good clinical practices in the resource poor settings.

(Details on Page 163)

ABSTRACT

Patients with tuberculosis sometimes may present with atypical, unusual features. The reported case is of 57 years male admitted with chief complaint of fever and breathlessness. Spiral CT chest revealed significant pericardial effusion all around heart, bilateral pleural effusion and multiple ill defined alveolar nodules scattered in both the lungs, more so in upper lobes. No significant mediastinal lymphadenopathy was seen. Pericardial fluid on Ziehl–Neelsen staining revealed acid fast bacilli. Subsequently sputum examination also showed acid fast bacilli. Hence pericardial fluid examination is helpful in the diagnosis of tubercular pericarditis. Tuberculosis can have protean manifestations as in this case with bilateral pleural effusion, pericardial effusion and mottling of lungs. Therefore clinician needs to keep complex presentation of tuberculosis in mind to manage the case at its earlier stage to avoid residual complications.

KEYWORDS

Tuberculosis, Pericarditis

1. Introduction

Despite the global prevalence of tubercular pericarditis, it remains a diagnostic and therapeutic challenge for the clinical cardiologists specifically in developing countries. Being a disease of protean manifestation, it poses a diagnostic dilemma to medical practitioners even in endemic areas where clinical suspicion is high^[1–4]. In many patients, an exclusive workup fails to identify the etiological agent. Definitive diagnosis of tuberculosis requires demonstration of acid fast bacilli in the pericardial fluid or histopathological examination of pericardial biopsy specimen. However, yield of these tests has been very poor^[4]. If left untreated ultimately constrictive pericarditis develops in all the patients^[5,6]. Here we report a case of tubercular pericarditis in a 57 years old male, diagnosed by

demonstration of acid fast organisms in the pericardial fluid collected by pericardiocentesis. The patient was promptly treated with antitubercular drugs without any residual complications.

2. Case report

A 57 years old male, from low socioeconomic strata (farmer) with two months history of low grade fever, breathlessness and significant weight loss was admitted in the cardiology unit of Dayanand Medical College & Hospital, Ludhiana, Punjab. There was no history of cough, palpitation or syncope. There was no past history of diabetes, heart disease or tuberculosis.

On examination patient was conscious and oriented. There

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was no peripheral edema, cyanosis, pallor or jaundice. His Juglar venous pressure (JVP) was normal, pulse rate 110/min, respiratory rate was 18/min, bilateral vesicular breathing, temperature 99 °F, blood pressure 140/90 mm Hg and the heart sounds were muffled.

On investigations Hemoglobin was 11.4 g/dL; RBC count 4.72 million/mm³; WBC count 6.85×10⁹/L with polymorphs 81%, lymphocytes 15%, eosinophils 2% and monocytes 4%. Erythrocyte sedimentation rate was raised (85 mm/h). Hepatic and renal function tests were within normal limits. The patient was seronegative for HIV 1 & 2 antibodies, HBsAg and HCV antibodies.

Electrocardiograph showed sinus tachycardia with low voltage complexes in frontal leads. Spiral computerized tomography chest revealed significant pericardial effusion all around heart, bilateral pleural effusion and multiple ill defined alveolar nodules scattered in both the lungs more so in upper lobes. No significant mediastinal lymphadenopathy was seen and pericardium was normal in thickness. An ultrasound guided pigtail catheter was inserted and over the next few days approximately 700 mL of straw coloured pericardial fluid was drained. The drained pericardial fluid was sent for microbiological examination of *i.e.* Gram's stain, culture and antibiotic sensitivity for pyogenic organisms, Ziehl–Neelsen stain and culture for mycobacterium. The Gram's stain showed no microorganisms, culture for pyogenic organism showed no growth of pyogenic organisms, Ziehl–Neelsen stained smears showed acid fast bacilli (AFB) along with pus cells. Culture on Lowenstein Jensen media showed rough and buff colonies suggestive of *Mycobacterium tuberculosis* (*M. tuberculosis*) after 4 weeks of incubation and was confirmed by acid fast staining and biochemicals.

The pericardial fluid was also sent for biochemical investigations which showed adenosine–deaminase (ADA) level of 11.4 IU/L (within normal limits; cut off 30 IU/L), glucose 3 mg/dL; total protein 5.1 mg/dL; amylase 19 IU/L; and significantly raised lactate dehydrogenase (LDH) *i.e.* 2472 IU/L. cytological examination showed predominance of polymorphic–nuclear leukocytes (PMNLs) with lots of degenerated cells. There were no malignant cells seen.

The patient was put on antitubercular treatment (ATT) (four drug regimen) *i.e.* isoniazid 300 mg, rifampicin 450 mg, pyrazinimide 1250 mg and ethambutol 750 mg. Liver function Tests were found to be deranged following institution of ATT. Isoniazid, rifampicin and pyrazinamide were with–held. Flouroquinolones along with aminoglycosides were added. Gradually liver function tests normalized, isoniazid and rifampicin were reintroduced at low doses and revised to normal dose. Patient's condition improved over a period of 25 d from the date of admission and was discharged in a stable condition. The patient responded well to the treatment and follow up examination after eight weeks of initiation of therapy showed improvement.

3. Discussion

Extra–pulmonary tuberculosis occurs in 20% of patients with tuberculosis[7,8], which rises to over 50% in people with HIV[9].

Tubercular pericarditis is seen in 1%–8% of these patients. The route of spread to pericardium is usually from mediastinal or hilar nodes or from lung and rarely as part of miliary tuberculosis. Tubercular pericarditis can present with recurrent pericardial effusion without any history or symptoms of tuberculosis. It is the most common pericardial disease in Sub–Saharan African and other developing countries[10]. TB accounts for <5% cases of pericardial disease in developed world, yet is the cause of 50%–70% of cases in developing world. Being a disease of protean manifestation, it poses a diagnostic challenge to the clinician even in endemic area where clinical suspicion is very high[11–13]. Rapid diagnosis and treatment are crucial in reducing the mortality, morbidity and residual complications of tubercular pericarditis[14].

Tubercular pericarditis may present as purulent pericardial effusion with predominance of PMNLs and relatively abundant mycobacteria; sero sanguinous effusion with predominantly lymphocytic exudates; absorption of effusion with pericardial thickening and ultimately fibrosis and constrictive pericarditis[15]. The pericardial effusion is mainly due to hypersensitivity to tubercular protein[16]. Tubercular pericarditis has a variable clinical presentation and should be considered in the evaluation of all cases of pericarditis[17].

Tubercular pericardial effusion usually develops insidiously, presenting with symptoms such as fever, night sweats, fatigue, weight loss, chest pain, cough and breathlessness are common, though severe acute pericardial pain is usually absent. Advent of echocardiography has made it possible to have an accurate, noninvasive method for diagnosing the presence of pericardial effusion, but this cannot determine the pathogenesis of effusion[4]. The ECG is abnormal in virtually all cases of tubercular pericardial effusion including the present case. Imaging by CT scan or MRI can also be used to diagnose the pericardial effusion as in the present case also, but is seldom available in rural areas in developing countries.

In the present case pericardial effusion was typically exudative and was characterized by high protein content and increased leukocytes with a predominance of PMNLs and increased LDH levels. An exudate is defined as one or more of the following: fluid protein/serum protein ratio>0.5; Fluid LDH/Serum LDH ratio>0.6 and/or fluid LDH levels>200 IU/L[18]. These criteria can be extrapolated to pericardial fluid as well and are the most reliable tools for identifying pericardial exudates[16,19].

The tubercular pathogenesis of pericarditis must be established as far as possible by demonstrating acid fast bacilli in the sputum, lymph nodes and pericardial fluid[20–22]. The variability in the detection of tubercle bacilli in the direct smear examination of pericardial fluid is well documented; the yield ranges from 0–42%[17,23]. Definite tubercular pericarditis can be diagnosed by one or more of the following criteria: (a) Isolation of *M. tuberculosis* from pericardial fluid or pericardial biopsy; (b) Demonstration of granulomatous inflammation on histological examination of pericardial biopsy; (c) Isolation of *M. tuberculosis* from sputum or non pericardial effusion exudates in the presence of clinical and/or radiological evidence of tuberculosis, associated with positive response to ATT and in the absence of any other obvious cause of pericarditis[24]. In the present case, the diagnosis was confirmed by demonstration of

AFB in pericardial fluid and isolation of *Mycobacterium* from the same on Lowenstein Jensen medium. Subsequently sputum smear was also found to be positive for AFB.

Antibiotic chemotherapy increases survival dramatically in tubercular pericarditis. A regimen consisting of isoniazid, rifampicin, pyrazinamide and ethambutol for at least two months followed by INH and rifampicin (total six months of therapy) have been shown to be highly effective.

On conclusion, it appears that this patient had pulmonary tuberculosis resulting in bilateral pleural effusion and pericardial effusion. The patient was hospitalized in the early effusive phase of tubercular pericarditis as indicated by the demonstration of AFB in the fluid with increase in leucocytes and predominance of PMNLs. It has been documented that probability of obtaining definitive bacteriological diagnosis is greatest when pericardial fluid is examined in the early effusive phase of the disease^[17]. The patient was successfully treated with ATT without any residual complications due to correct diagnosis made at an early stage of disease.

Conflict of interest statement

We declare that we have no conflict of interest.

Comments

Background

Tuberculous pericarditis is a major cause of constrictive pericarditis requiring pericardiectomy. Rapid diagnosis and treatment are crucial in reducing the mortality, morbidity and residual complications of tubercular pericarditis.

Related reports

In some cases, evidence of chronic cardiac compression mimicking heart failure is common presentation. Cardiac tamponade may present as a complication of pericardial effusion. In the present case, any such complications were averted due to the early diagnosis and prompt treatment. Prompt treatment of tuberculous pericarditis may be life saving (Reuter H, et al. 2006). Effective treatment requires a rapid and accurate diagnosis, which is often difficult (Mayosi BM, et al. 2005).

Peer review

This is a good study and nicely presented. In which the authors evaluated the usefulness of early diagnosis of tubercular pericarditis. The results are interesting and highlights the importance of good clinical practices in the resource poor settings.

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