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Tuberculous pericarditis: a case report

Affan Denk[1], Mehmet Ali Kobat[2], Safak Ozer Balin[3], Sumeyye Selim Kara[1], Orhan Dogdu[2]

[1]Department of Infectious Diseases and Clinical Microbiology, Faculty of Medicine, Firat University, Elazig, Turkey;
[2]Department of Cardiology, Faculty of Medicine, Firat University, Elazig, Turkey;
[3]Department of Infectious Diseases and Clinical Microbiology, Elazig Training and Research Hospital, Elazig, Turkey

SUMMARY

Pericardial effusion is common disease and difficult to diagnose. Tuberculosis accounts for up to 4% of acute pericarditis and 7% of cardiac tamponade cases. Quick treatment can be lifesaving but requires accurate diagnosis. We report a case of a 65-year-old man who presented with a 3-week history of fever with chills, non-productive cough and dyspnea. The case was diagnosed by positivity of acid-fast staining, culture and polymerase chain reaction (PCR) of the aspirated pericardial fluid and treated promptly with antituberculosis drugs. The patient showed complete recovery.

Keywords: tuberculosis, pericarditis.

INTRODUCTION

Although there has been a significant decline in tuberculosis in wealthy industrialized countries over last years, Africa, Asia, and Latin America with 86% of the world's population, are home to 95% of all cases of active tuberculosis and 98% of nearly two million deaths resulting from this disease each year [1]. Tuberculous pericarditis, caused by *Mycobacterium tuberculosis* is found approximately in 1% of all autopsied cases of tuberculosis and in 1-2% instances of pulmonary tuberculosis [2]. Quick treatment of tuberculous pericarditis can be lifesaving. Effective treatment requires a rapid and accurate diagnosis for disease but it is frequently difficult [3].

We report a case of tuberculous pericarditis in a 65-year-old man that was diagnosed by the positivity of acid fast staining, culture and polymerase chain reaction (PCR) of the aspirated pericardial fluid, and promptly treated with antituberculosis drugs.

Corresponding author
Affan Denk
E-mail: affandenk23@yahoo.com

CASE REPORT

A 65-year-old man patient presented with a 3-week history of fever with chills, non-productive cough and dyspnea. There were no history of tuberculosis, alcohol and IV drug abuse, certain diseases such as diabetes, cancer, and HIV infection, immunosuppression, use of corticosteroids and occupational risk (for example, health-care worker) as well as no familiar risk factors for tuberculosis. On examination he was febrile with temperature of 38.8°C, tachycardia (heart rate of 112 beats/min), blood pressure of 120/80 mmHg, and respiratory rate of 23 breaths/min. Jugular venous pulse was raised. The heart sounds were muffled and associated with a pericardial rub on auscultation. He had a body weight of 62 kg.

Laboratory tests revealed haemoglobin of 12.4 g/dL, white blood cell count of 12000/mm^3 with polymorphs 60% and lymphocytes 37%, erythrocyte sedimentation rate of 38 mm/h and C-reactive protein level of 4 mg/L (normal <5 mg/L). He was seronegative for HIV. The hepatic function tests were within normal limits. Creatinine level was 1.3 mg/dL. Tuberculin skin test (TST) was positive at 17 mm aftre 24 hours. Electrocardiography (ECG) showed low voltage complex-

es with sinus tachycardia. Chest X-ray indicated left basal consolidation with effusion along with cardiomegaly. Ultrasound-guided thoracentesis was performed. Microbiological examination of thoracentesis fluid for acid fast staining, and PCR test for *M. tuberculosis* was found negative. Within the same day, pericardiocentesis was performed by transthoracic echocardiography. In a few days, 800 ml of straw-colored pericardial fluid was drained. On microbiological examination, PCR test for *M. tuberculosis* was found positive. The acid fast staining smears showed acid fast bacilli along with pus cells. Culture on Lowenstein Jensen (LJ) media showed rough and buff colonies suggestive of *M. tuberculosis* after four weeks of incubation and was confirmed by acid fast staining, and biochemical tests. The patient responded well to drainage of the pericardial fluid and dyspnea subsided. He was orally started on isoniazid (INH) 300 mg, rifampicin (RIF) 600 mg, ethambutol (EMB) 1500 mg, and pyrazinamide (PZA) 2000 mg daily. The fever subsided two weeks after this treatment. One month later, the patient was discharged from hospital. The patient was responding well to the treatment, with no recurrence of symptoms or any signs of deterioration when last followed up, six weeks after the start of therapy. The anti-tuberculosis 4-drug regimen was given for two months, afterwards 2-drug regimen (INH+RIF) was given for four months. The patient showed complete recovery.

DISCUSSION

Extrapulmonary tuberculosis occurs in 20% of patients with tuberculosis. Tuberculous pericarditis is shown in 1-8% of these patients. Tuberculous pericarditis is the most common cause of pericarditis in Africa and other countries where tuberculosis remains a large problem [4]. Tuberculosis accounts for up to 4% of acute pericarditis and 7% of cardiac tamponade. The mortality rate of tuberculosis still ranges from 14 to 40%. Tuberculous pericarditis is still a lethal condition. This effusion is mainly due to hypersensitivity to tubercular protein [5]. The route of spread of the organisms to pericardium is usually from mediastinal or hilar lymph nodes or from lungs or rarely as a part of miliary tuberculosis. Typically, the process begins as effusive constrictive pericarditis. In ultimate stages, acid fast bacilli are usually not detected but caseating granulomas involving the pericardium and epicardium may be present [6].

The clinical presentation of tuberculous pericarditis is variable and non-specific with symptoms including fever, night sweats, fatigue, and weight loss. Most common symptoms are cough, chest pain and dyspnea [7, 8]. In some cases, evidence of chronic cardiac compression mimicking heart failure can be present [6]. Cardiac tamponade may present as a complication of pericardial effusion. Sometimes vague symptoms make the condition difficult to recognize. Prompt treatment of tuberculous pericarditis may be lifesaving. Effective treatment requires a rapid and accurate diagnosis, which is often difficult [3]. Chest radiograph which shows an enlarged cardiac shadow in more than 90% cases, demonstrates features of active pulmonary disease in 30% cases [4-7]. The ECG is abnormal in virtually all cases of tuberculous pericarditis as observed in the present case [9].

In the present case, the diagnosis was confirmed by demonstration of acid fast bacilli in aspirated material and isolation on LJ medium. The variability in the detection of tubercle bacilli in a direct smear of pericardial fluid is well documented but the yield may range from 0 to 42% [10]. Thus, culture is said to be the gold standard. Rapid diagnosis by PCR was considered an important diagnostic tool, till a study by Cegielski et al. [11], demonstrated that the sensitivity of PCR of pericardial tissue is comparatively lower than culture. In this case, we have shown that tuberculosis PCR of pericardial fluid. Antibiotic therapy dramatically increases survival in tuberculous pericarditis. A regimen consisting of INH, RIF, EMB, and PZA for at least two months followed by INH and RIF (total six months of therapy) have been shown to be highly effective [3, 6].

In conclusion, tuberculous pericarditis is a rare manifestation of tuberculosis and is associated with considerable mortality and morbidity which decrease with effective early diagnosis and treatment. In patients admitted to hospital with long-lasting fever, cough, and shortness of breath, especially in endemic areas, tuberculous pericarditis should be kept in mind in the differential diagnosis.

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