Case report

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A case of tuberculosis pericarditis with an interesting echocardiographic image

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Abstract

Tuberculosis-associated pericardial disorders are an excessively rare manifestation of extrapulmonary tuberculosis. The patients may present with constrictive pericarditis or pericardial fluid accumulation leading to cardiac tamponade. This paper reports a case of tuberculosis-associated pericardial effusion with dense fibrinous material not causing tamponade in a foreigner presenting with nonspecific symptoms. It also provides a discussion about the diagnostic and therapeutic methods as well as interesting echocardiographic images of the patient.

Keywords: tuberculosis; pericarditis; effusion; echocardiography; fibrin

Introduction

Tuberculous pericarditis (TP) is a form of extrapulmonary tuberculosis that usually develops as a result of direct extension of primary pulmonary tuberculosis from trachea, peribronchial and mediastinal scrofula, or of hematogenous spread of pleural tuberculosis [1]. TP manifests itself with 3 main clinical forms, namely pericardial effusion, constrictive pericarditis, and a combination of effusion and constriction [2]. Rapid treatment of TP is life-saving, with an effective management primarily requiring rapid and accurate diagnosis, which is sometimes considerably difficult [3]. Herein we report a patient of TP with an interesting visual material, who presented with massive pericardial and pleural effusion that did not lead to tamponade. We also discussed diagnostic and therapeutic methods used for the condition.

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Case report

A 46-year-old woman of Middle Asian origin presented to emergency department with cough, dyspnea, and leg swelling that had become progressively worse for the last 2 weeks. She had no history of any disease or medication use. On admission, she had dyspnea and used her accessory respiratory muscles. Her blood pressure was 100/60 mmHg, pulse rate 108/min, body temperature 37°C, and oxygen saturation 97%. On physical examination her heart sounds were rhythmic but muffled, and there was an audible pericardial friction rub. Her respiratory sounds were reduced at the bases of her lungs. The liver was palpable 1 cm below the right costal margin. Her electrocardiogram was unrevealing except for sinus tachycardia. A posteroanterior chest X-Ray showed the obliteration of both pleural sinuses, with right side being affected more severely (fig 1a). A computerized tomography with contrast enhancement taken in emergency department showed pleural and pericardial effusion but no pulmonary parenchymal mass or filling defects in pulmonary artery branches (fig 1b). A transthoracic echocardiography demonstrated marked pericardial effusion around the heart. The effusion contained dense fibrinous material and surrounded the heart,

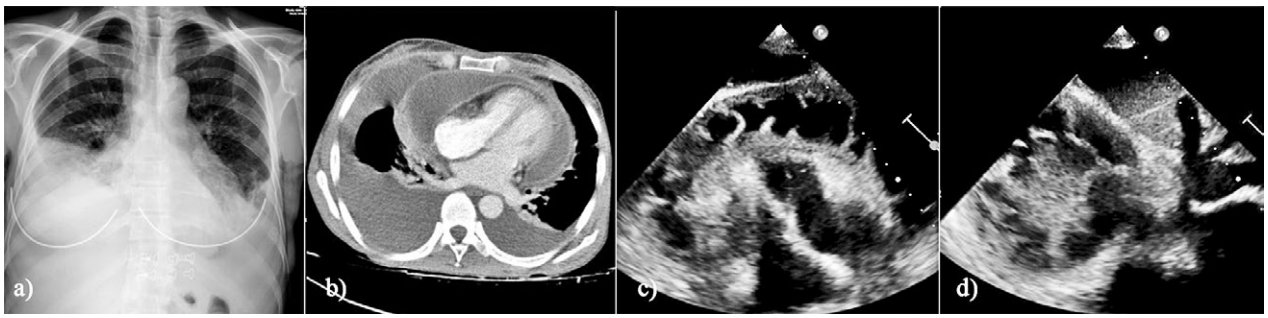

Fig 1. a) Chest X-Ray showing both sinuses obliterated by pleural fluid; b) Computer tomography of the chest showing both sinuses obliterated with pleural fluid and pericardial cavity filled with dense fibrinous material; c) and d) Pericardial effusion with dense fibrin materials around the heart.

causing minimal compression of the base of the right atrium at end-diastole. Fibrinous material conglomerated at the right ventricular free wall. Fibrinous material moved synchronously with each heart beat and appeared like "cheering fans" (fig 1c,d Video 1 - movement of fibrin materials synchronous with each heartbeat - on the journal site). Laboratory tests were in normal range. The patient had a stable hemodynamics but dyspnea, and the latter was attributed to pleural effusion which was therefore drained. One day later, a total of 450 cc pericardial effusion was drained for diagnostic and therapeutic purposes. Both fluids were of exudative properties. The cell count showed lymphocyte predominance with a number of 40/mm^3. Simultaneous pleural and pericardial adenosine deaminase levels were 18.30 IU/L and 28.5 IU/mL, respectively. No cell could be identified in gram staining. Acid-fast bacteria were not detected at first. Pericardial biopsy could not be obtained due to scarce resources of our hospital. Considering her anamnesis and simultaneous pleural and pericardial effusion, the patient was diagnosed with TP and quadruple therapy (isoniazid 3x100 mg, rifampicin 2x300 mg, ethambutol 3x500 mg, streptomycin 2x500 mg) was commenced. Although AARB was not proliferated in the cultures, polymerase chain reaction was positive. The patient's dyspnea was relieved on the 10th day of therapy. Effusion markedly regressed at follow-up. Fibrin materials were still in place despite being markedly reduced. The patient was then lost to follow-up because she returned to her home country.

Discussions

Extrapulmonary tuberculosis affects 20% of patients with tuberculosis. TP develops in 1% to 8% of these cases. TP is still one of the major etiologies of pericarditis in underdeveloped countries where tuberculosis remains a significant public health problem [4]. Tuberculosis is responsible for up to 4% of acute pericarditis cases and still has a high mortality (14-40%) [5]. Hypersensitivity reaction against the tuberculin protein is considered the main cause of pericardial effusion [5]. The difficulty of making the diagnosis of TP stems from the diversity and vagueness of symptoms. Affected patients usually present with fever, weight loss, cough, night sweating but also with acute cardiac tamponade, albeit rare [6]. Four phases of tbc pericarditis have been described: 1) the dry phase when early immune response takes place and fibrin material's exudation develops; 2) the effusive phase when a serosanguinous fluid is formed; 3) the absorption phase when pericardial thickening and granulomatous caseation organize; and 4) the constrictive phase [7]. The diagnosis of the condition requires pericardial fluid or tissue analysis. Adenosine deaminase and interferon gamma activity may also be beneficial in addition to PCR test, histopathological examination, and cultures. However, culture is the gold standard for a definitive diagnosis [3]. The diagnosis of the condition is based on 4 criteria, which include the isolation of M. Tuberculosis from pericardial tissue or fluid cultures, demonstration of the bacilli or granuloma in pericardial tissue on histopathological examination, showing pericardial granuloma formation in pericardial tissue in the presence of an extracardiac tuberculosis focus, and response to specific treatment. Although it is quite difficult to isolate bacteria from pericardial fluid samples, with cultures frequently returning non-diagnostic, a proliferation can be observed in a third of cases [8-9].

The treatment of tbc pericarditis resembles that of pulmonary tuberculosis, which involves a quadruple therapy consisting of rifampicin, isoniazid, ethambutol, and pyrazinamide for 2 months, followed by rifampicin and isoniazid for 4 months [10].

Our patient had recently come to our country from Middle Asia. According to her anamnesis, she resided in a place with poor hygienic conditions. Presence of pleu-

ral and pericardial fluid at admission, particularly dense fibrinous material adhering to each other and pericardial membranes, made us think TP as a provisional diagnosis. The synchronous movement of dense fibrinous material inside pericardial fluid with heart beats was quite interesting. M. Tuberculosis did not proliferate from the samples obtained from both pericardium and pleura. The patient was administered anti-tuberculosis treatment consisting of four drugs. Steroids may also have been commenced on the basis of treatment response and echocardiographic signs. Nevertheless, as the patient returned to her country, her treatment was left half finished. Our patient's anamnesis and symptoms, dense fibrinous materials seen in echocardiography, and elevated adenosine deaminase level all made us start anti tuberculosis treatment as rapidly as possible, without waiting for culture proliferation. PCR test becoming positive at her follow-up cleared our doubts about our diagnosis. Obtaining a pericardial biopsy and observing granulomas in pathology examination would have confirmed the diagnosis but we could not perform it due to our hospital's scarce resources.

In conclusion TP could be a fatal disease requiring urgent diagnosis and treatment. Medical treatment should be commenced as rapidly as possible, and effusion should be drained whenever needed. Patient anamnesis, symptoms, and echocardiographic findings as well as culture proliferation should be taken into account for diagnosis. Particularly, if there are fibrin materials within effusion, TP should be primarily considered.

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