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Benign Tuberculous Constrictive Pericarditis: A Case of Complete Resolution with Empirical Antimicrobial Treatment

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Case Report

Abstract

Tuberculous (TB) pericarditis is a well-known manifestation of tuberculosis, particularly in endemic regions. The progression to constrictive pericarditis, while under anti-tuberculosis treatment, is reported to be as high as 30%. This report presents a case of a 56-year-old female patient who complained of cough, pleuritic chest pain, night sweats, and fever, followed by orthopnea, dyspnea, and peripheral edema. Transthoracic echocardiography revealed the early stages of constrictive pericarditis and a small pericardial effusion. Chest computed tomography (CT) showed a thickened pericardium, small pericardial and pleural effusions, and multiple mediastinal lymphadenopathies. Due to a high suspicion of tuberculous pericarditis, the patient was administered empirical anti-TB treatment. A follow-up after two months showed complete resolution of symptoms and echocardiographic findings. Empirical antimicrobial treatment in endemic areas is a well-established strategy for managing tuberculous infection and proved successful in this patient. The early presentation and the significant improvement in signs and symptoms following the medical anti-TB regimen, without the need for pericardiectomy, were unique aspects of this case.

Keywords: Constrictive Pericarditis, Tuberculosis, Empirical Antimicrobial Treatment

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Introduction

Tuberculous pericarditis is a significant complication of tuberculosis, often overlooked or diagnosed late [1]. Tuberculosis is the leading cause of pericarditis in Africa and other regions where TB remains a significant public health issue [2]. Tuberculous pericarditis is found in 1% of all autopsied cases of TB infection and in 1-2% of cases of pulmonary tuberculosis [3].

The general symptoms of tuberculous pericarditis, such as fever, weight loss, and night sweats, are nonspecific and typically precede cardiopulmonary complaints [1]. The nature of symptoms depends on the stage of infection, the extent of tuberculous disease outside the pericardium, and the severity and duration of pericardial involvement [4].

Establishing a diagnosis can be challenging and is often delayed or missed, resulting in late complications like constrictive pericarditis with increased mortality [1]. Progression to constrictive pericarditis, even with optimal anti-tuberculosis therapy (without concurrent corticosteroid treatment), is reported in up to 30% of cases [5].

This report describes a case of constrictive pericarditis due to tuberculosis infection, primarily presenting with longstanding fever and dyspnea.

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Case Presentation

A 56-year-old female, residing in a TB-endemic area in the southern part of Iran, presented to our clinic with complaints of dyspnea and a prolonged fever of unknown origin. Her medical history was unremarkable, except for hypothyroidism and hypertension, which had been treated with levothyroxine, amlodipine, and valsartan for over ten years. She reported experiencing cough, pleuritic chest pain, night sweats, and fever for six months. These symptoms were followed by orthopnea, dyspnea of New York Heart Association (NYHA) functional class III, and peripheral edema. Her medical records indicated that she had undergone pericardial fluid drainage for tamponade about six months ago, with the pericardial fluid being bloody and the cytology analysis for malignant cells returning negative, with no additional data. Subsequently, she was treated with aspirin and colchicine, but no significant clinical improvement was observed.

Upon presentation at this tertiary referral center, the patient was a healthy and alert middle-aged woman. She was febrile (oral temperature: 38.5°C) with stable blood pressure (BP: 140/90 mmHg), negative pulsus paradoxus, and a pulse rate of 100 beats per minute, without respiratory distress (respiratory rate: 20 per minute and hemoglobin O2 saturation-SPaO2- of 95% on room air). Physical examination revealed no peripheral lymphadenopathy, normal heart sounds without friction rub, and mildly elevated Jugular venous pressure. Bilateral basilar fine crackles were evident upon lung auscultation, with 2+ pitting peripheral edema observed during the examination of the lower extremities.

A twelve-lead surface electrocardiogram revealed sinus tachycardia with ST segment depression and inverted T waves on leads II, III, aVF, and V2-V6. Apart from mild normochromic normocytic anemia (Hemoglobin: 9.8 mg/dL), an elevated erythrocyte sedimentation rate, and C-reactive protein (ESR: 118 mm/hr and CRP 2+), other laboratory assessments, including serum electrolytes, renal and hepatic function tests, as well as immunologic parameters such as anti-nuclear antibody (ANA), anti-double-stranded DNA (Anti-dsDNA) antibody, Anti-cyclic citrullinated peptides (anti-CCP), anti-Ro, and Anti-La antibodies, were within the normal range. Serum antibodies for the human immunodeficiency virus (HIV test) and a nasopharyngeal swab for SARS-COV2 were reported to be negative.

The chest X-ray did not reveal any specific pathology. Transthoracic echocardiography (TTE) showed a thickened pericardium with a small circumferential pericardial effusion, moderate left ventricular (LV) systolic dysfunction with an LV ejection fraction (LVEF) of 37%, abnormal septal motion typical for septal bounce and septal shudder, global hypokinesia, annulus reversus and paradox, significant respiratory variation in mitral valve (MV) and tricuspid valve (TV) inflows (50% and 100% respectively), and a top normal inferior vena cava (IVC) size (Figure 1, complementary video 1). Chest computed tomography (CT) revealed a thickened pericardium (7 mm), a small pericardial effusion, multiple small perihilar and mediastinal lymphadenopathies, and a left-sided pleural effusion, as well as some segmental collapses in the left lung field.

Considering the patient's history, residence in a TB-endemic area, and findings from the TTE and chest CT, a purified protein derivative (PPD) skin test and interferon gamma release assay (IGRA) test were performed, with the latter yielding a positive result. The sputum smear and culture were negative for Mycobacterium tuberculosis. Due to the small size of the pericardial and pleural effusions, pericardiocentesis and thoracentesis could not be performed. The patient had undergone PE drainage without biopsy six months prior to presenting at our center. Upon examination of the patient, given the typical history, CT and echo findings, and mild to moderate PE without signs of tamponade necessitating an open procedure, the most probable diagnosis was TB. The pathology findings also require an expert team for diagnosis.

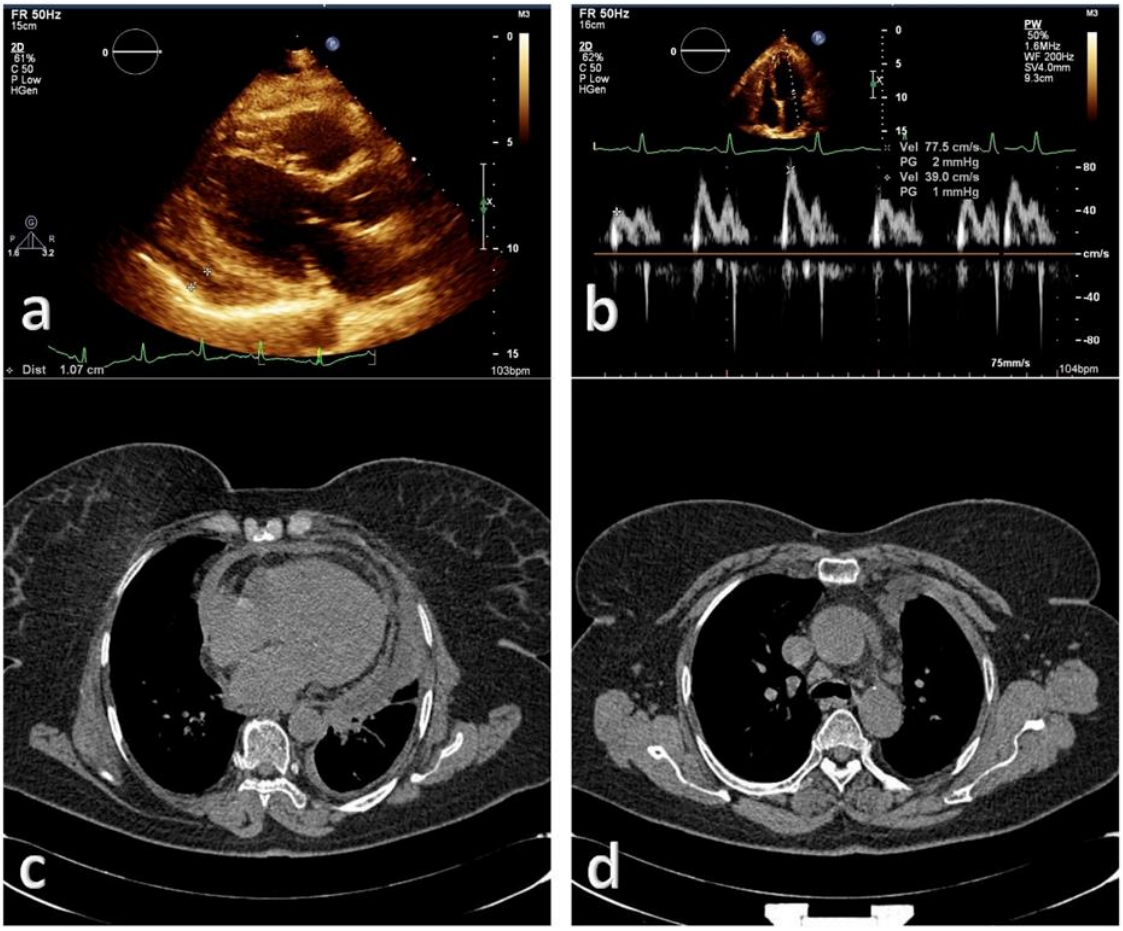

Figure 1. 1a. Parasternal long axis view reveals small circumferential pericardial effusion and septal shudder.
1b. Pulse wave Doppler of mitral inflow shows significant respiratory variation.
1c. Mid thoracic slice of chest CT shows thick pericardium, small pericardial and pleural effusions.
1d. Upper thoracic chest CT shows multiple small perihilar and mediastinal lymphadenopathies.
complementary video 1: Apical 4 chamber view shows LV systolic dysfunction and septal bounce.

The biopsy procedure was refused by the surgeon, and an infectious disease specialist recommended empirical treatment (as noted in the consultation paper, the patient was definitively diagnosed with TB). The decision to select the preferred treatment strategy was made by a team comprising an infectious diseases specialist, internist, pulmonologist, and cardiologist. Ultimately, empirical anti-TB treatment was chosen, considering the early stages of TB effusive constrictive pericarditis as the most probable diagnosis. This decision was based on a high clinical suspicion for TB and the results of paraclinical assessments. The patient was administered a four-drug regimen consisting of isoniazid, rifampin, pyrazinamide, and ethambutol. At a two-month follow-up, she reported complete resolution of symptoms. ESR and CRP measures were within normal ranges, and a TTE examination showed dramatic improvement, including an improvement in LV function (LVEF: 45%), resolution of pericardial effusion, annulus reversus, and septal bounce. The patient continued anti-TB treatment for the next four months with isoniazid and rifampin.

Discussion

In developed countries, Tuberculous pericarditis is a very rare cause of acute pericarditis, and 30–60% of patients with TB

pericarditis may develop constrictive disease [6]. In sub-Saharan Africa and parts of Asia, TB is a common cause of pericardial effusion, cardiac tamponade, and constrictive pericarditis [7]. The mechanism of pericardial involvement may be due to retrograde lymphatic spread of Mycobacterium tuberculosis from peritracheal, peribronchial, or mediastinal lymph nodes, or hematogenous spread from primary tuberculous infection[8]. A definite diagnosis of TB constrictive pericarditis is based on the presence of acid-fast bacilli (tubercle bacilli) in the pericardial fluid or on a histological section of the pericardium, by culture or PCR testing; with the sensitivity of pericardial biopsy in a wide range of 10 to 64%. On the other hand, in endemic areas, a probable diagnosis can be made when there is an appropriate response to anti-tuberculosis chemotherapy [9].

In regions endemic for TB, a pericardial effusion, in the absence of other clear alternative diagnoses, is often attributed to tuberculosis, and anti-tuberculosis treatment often needs to be initiated before a definitive bacteriological diagnosis is made. In some patients, an adequate response to anti-tuberculosis therapy supports the diagnosis, even without a diagnosis based on bacteriology, histology, or pericardial fluid analysis [10].

In this case, due to the small amounts of pericardial and pleural fluids, pericardiocentesis and thoracentesis were not possible, leading to the decision to proceed with empirical therapy for TB constrictive pericarditis. Sameer Kushwaha et al. reported complete resolution of symptoms and increased LVEF after anti-TB treatment in a case of TB constrictive pericarditis. However, this response was observed after pericardiectomy, and they also used adjunctive prednisolone [11].

Another case presentation reported symptom improvement in a patient with TB constrictive pericarditis after six months of anti-TB treatment. However, this patient had also undergone pericardiectomy before the anti-TB treatment [12].

Pericardiectomy is indicated when anti-TB therapy and adjuvant steroids (when indicated) fail to prevent or reverse pericardial constriction after 4-8 weeks of treatment. However, this procedure is expensive, high risk, and poorly accessible in many developing countries [13,14].

The use of corticosteroids in patients with tuberculous pericarditis to attenuate the inflammatory response may improve outcomes and decrease mortality, but the clinical effectiveness of this treatment is unclear [7]. According to the European Society of Cardiology (ESC) guidelines, adjunctive corticosteroid therapy may be reasonable in HIV-negative patients with TB pericarditis (class IIb) [9].

In a case of TB constrictive pericarditis, clinical improvement and complete resolution of pericardial effusion and thickening, along with normalization of the myocardial longitudinal deformation, have been reported following standard anti-TB treatment with adjuvant corticosteroid therapy, and there was no need for pericardiectomy [13].

Also, Catez et al. reported a successful response to a standard 4-drug anti-TB treatment in a case of TB constrictive pericarditis without corticosteroid therapy and the need for pericardiectomy [14].

As stated above, the patient in this case exhibited a dramatic response to a four-drug regimen of anti-TB therapy without the need for corticosteroid therapy. This response strongly suggests that TB constrictive pericarditis was the most probable diagnosis for this case. Early diagnosis at the initial stages of constrictive pericarditis led to clinical and echocardiographic improvement of CP physiology, eliminating the need for pericardiectomy. This case underscores the importance of early diagnosis and treatment in managing TB constrictive pericarditis effectively.

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