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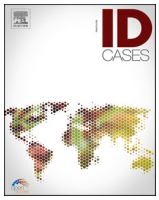

Case Report

Tuberculous pericarditis in a 71-year-old immunocompetent patient: Case report

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ABSTRACT

Tuberculosis is a transmissible disease caused by the bacterium *Mycobacterium tuberculosis*. It is the leading cause of mortality due to infectious diseases. Tuberculous pericarditis is one of the manifestations of extrapulmonary tuberculosis and represents the primary cause of pericardial effusion in developing countries. We present the case of a 71-year-old male patient with a history of 1 month of dyspnea, accompanied by paroxysmal nocturnal dyspnea and unintentional weight loss. An echocardiogram revealed the presence of severe pericardial effusion, for which pericardiocentesis was performed, and the pericardial fluid was analyzed using the Xpert MTB/RIF test, which confirmed the presence of *Mycobacterium tuberculosis* without resistance to Rifampicin. This is a case of Tuberculous Pericarditis as the cause of pericardial effusion, in which the etiological diagnosis was made possible through molecular biological analysis of the pericardial fluid. The importance of disseminating such cases lies in emphasizing that, even in the 21st century, in developing countries like Honduras, it is crucial not to dismiss tuberculosis infection, as it remains the leading cause of pericardial effusion in endemic regions.

Introduction

Tuberculosis is an infectious disease transmitted by the bacillus Mycobacterium tuberculosis [1,2]. It ranks second, after HIV, as the most prevalent infectious disease [3]. In 2022, it was estimated that 10.6 million people contracted tuberculosis, 87 % of whom were in low- and middle-income countries. Twenty percent of these cases correspond to the extrapulmonary form of the disease [1,2]. Tuberculous pericarditis occurs in 1 to 2 % of patients with pulmonary tuberculosis [4,5]. In developed countries, it accounts for less than 5 % of pericardial diseases, in contrast to what is reported in developing countries, where it represents 50 to 70 % of the causes of pericardial effusion [1,3,6].

Tuberculous pericarditis develops when Mycobacterium tuberculosis bacilli enter the pericardium through various routes. Since the lungs are the primary entry point for this microorganism, retrograde lymphatic dissemination through the mediastinal lymph nodes is typically the most common route. Hematogenous dissemination from a primary infection focus can also occur, and to a lesser extent, direct spread from adjacent infected structures such as the pleura, lungs, and spine may contribute [1,3,7–9]. Tuberculous pericarditis progresses through four pathological stages: 1) fibrinous exudation, 2) serosanguineous effusion, 3) absorption of the effusion, and 4) constrictive scarring. [1,3,5] These stages correspond to the various clinical manifestations observed, and thus, they may overlap and are not mutually exclusive [1,7].

Tuberculous pericardial effusion typically develops insidiously [8,10,11] and presents with nonspecific systemic symptoms such as fever, night sweats, fatigue, and weight loss, as well as dyspnea, chest pain, and cough [3,10,11]. Less commonly, it may present as acute pericarditis, characterized by severe, stabbing chest pain, pericardial friction rub, and generalized electrocardiographic abnormalities in the ST segment, T wave, and PR segment depression [1,3,11].

The diagnosis of tuberculous pericarditis can be made through the following methods: pericardial fluid analysis; in the early stages, the cytochemical analysis will predominantly show neutrophils, and in advanced stages, mononuclear cells will predominate [7,8]. Biomarkers such as adenosine deaminase (ADA), with a sensitivity of 88 % and specificity of 83 %, and interferon gamma (IFN-γ), with a sensitivity of 92 % and specificity of 100 % [1,3,12], can also be used. Bacteriological studies, such as pericardial fluid culture, remain the gold standard for laboratory diagnosis [1,3,7,8]. Nucleic acid amplification tests, such as Xpert MTB/RIF, which demonstrated a sensitivity of 63.8 % with high specificity (100 %) [3,7,8], are also valuable. Finally, histopathological

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Table 1
Laboratory test results.

Test	Result	Range
Hemoglobin	13.3 g/dl	12–16.5 g/dl
White Blood Cells	6230	5000–10,000
Platelets	210,000	130,000–400,000
Glucose	87 mg/dl	74–106 mg/dl
Creatinine	0.98 mg/dl	0.5–1.3 mg/dl
Sodium	138 mmol/L	136–145 mmol/l
Potassium	4.0 mmol/L	3.4–5.4 mmol/L
Aspartate Aminotransferase (AST)	21 U/L	15–37 U/L
Alanine Aminotransferase (ALT)	35 U/L	12–78 U/L
Lactate Dehydrogenase (LDH)	145 U/L	85–227 U/L
Creatine Phosphokinase (CPK)	73 U/L	39–308 U/L
Albumin	2.8 g/dl	3.4–5.0 g/dl
Procalcitonin	0.11 ng/mL	Menor 0.5 ng/mL
Antinuclear Antibody (ANA)	Negative	< 1:20
Complement C3	178 mg/dl	75–135 mg/dl
Complement C4	24.4 mg/dl	9–36 mg/dl
Total Prostate-Specific Antigen (PSA)	1.27 ng/mL	0–4.0 ng/mL
Thyroid Stimulating Hormone (TSH)	2.81 µU/mL	0.4–4.0 µU/mL
Chagas Serology	Non-reactive	
Human Immunodeficiency Virus (HIV) Serology	Negative	

examination is more specific than sensitive, diagnosing only 10–64 % of cases [3,5,7,8].

Tuberculosis is the leading cause of death from infectious diseases worldwide [1,2,13,14], representing a serious public health problem in developing countries. The mortality rate of tuberculous pericarditis in HIV-negative patients ranges from 8 % to 17 %, and it can rise to as high as 85 % if early and appropriate treatment is not provided, due to the development of cardiac tamponade [3,15]. For this reason, we present the case of a male patient, HIV-negative, with pericardial effusion secondary to tuberculous pericarditis.

Case report

We present the case of a 71-year-old male patient from Comayagua, a construction worker, with a history of hospitalization for COVID-19 pneumonia 6 months ago and benign prostatic hyperplasia. He presented with a 1-month history of dyspnea on exertion, which progressed to rest dyspnea over the last 3 days, accompanied by paroxysmal nocturnal dyspnea and unintentional weight loss over the past month. As a result, he consulted a cardiologist in Comayagua, who performed an echocardiogram and detected severe pericardial effusion, prompting a referral to the emergency department of the Escuela Hospital.

Upon arrival, he was normotensive with a blood pressure of 120/70 mmHg, tachycardic with a heart rate of 107, and without tachypnea (respiratory rate: 18). He had jugular venous distension, but no Kussmaul sign. Cardiac auscultation revealed normal heart sounds with a regular rhythm, no murmurs, and a slight decrease in vesicular breath sounds at the lung bases, without adventitious sounds. He was subsequently presented to the Cardiovascular Surgery service for pericardiocentesis.

Laboratory tests revealed the following results: complete blood count with no alterations in the three cell lines, hypoalbuminemia (2.8 g/dL), normal thyroid function tests, and normal results for antinuclear antibodies and anti-DNA antibodies. Chagas disease and HIV serology were negative (Table 1). Chest X-ray (PA view) showed cardiomegaly with an inverted cup sign (Fig. 1). The electrocardiogram showed signs of low voltage in the limb leads (Fig. 2). Echocardiography revealed normal cardiac chambers with normal global and segmental left ventricular mobility, ejection fraction > 60 %, and pericardial effusion both anterior and posterior, with a fluid collection of 21 mm but no signs of cardiac tamponade.

A subxiphoid pericardial window was performed, draining 1000 mL of yellowish fluid with pericardial thickening. Additionally, a pericardial drainage system was placed and maintained for 3 days. When the daily fluid output decreased to less than 10 mL, the drainage system was removed without complications.

Regarding the pericardial fluid studies, cytochemistry showed a cell count of 648 cells/µL, predominantly neutrophils, with a protein level of 67.36 g/dL. The Xpert MTB/RIF test reported the presence of Mycobacterium tuberculosis without rifampicin resistance, and the histopathological examination of the pericardium revealed fibroadipose tissue partially lined by mesothelium, with both acute and chronic inflammation (Table 2). The patient was then started on a quadruple drug regimen: Isoniazid 300 mg, Rifampicin 600 mg, Pyrazinamide 1.6 g, and Ethambutol 1.1 g, along with Prednisone 50 mg/day. On the third day of therapy, the patient showed minimal pleural fluid production, allowing for the removal of the drainage system. By the fifth day, the patient reported no sensation of dyspnea and tolerated the supine position. This was considered satisfactory progress, allowing for the continuation of strictly supervised outpatient antitubercular treatment, along with Prednisone 50 mg/day for 1 month, followed by 30 mg/day for 1 month, 15 mg/day for 2 weeks, and finally 5 mg/day for 1 week, before discontinuing the corticosteroid.

Discussion

The clinical manifestations presented by the patient included dyspnea, paroxysmal nocturnal dyspnea, and unintentional weight loss, as well as signs of heart failure such as jugular venous distention, pleural

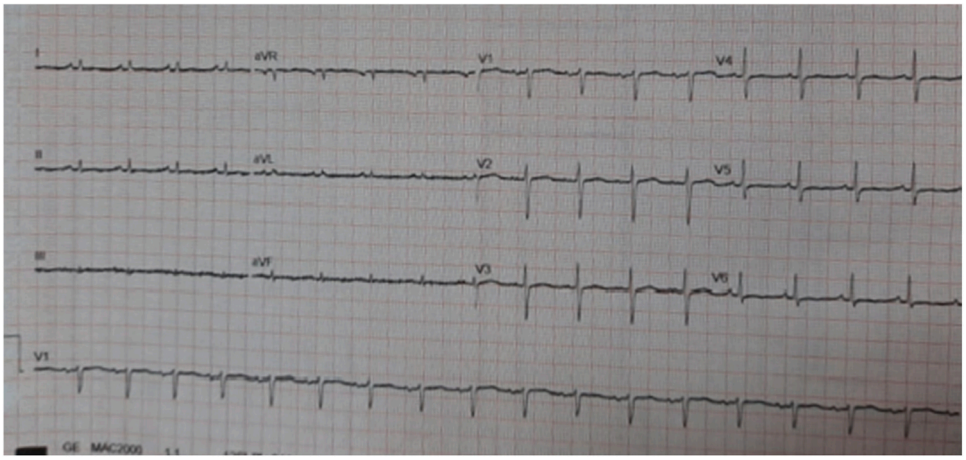

Fig. 1. Electrocardiogram: Poor R-wave progression in the limb leads and T-wave flattening in the limb leads and from V4 to V6.

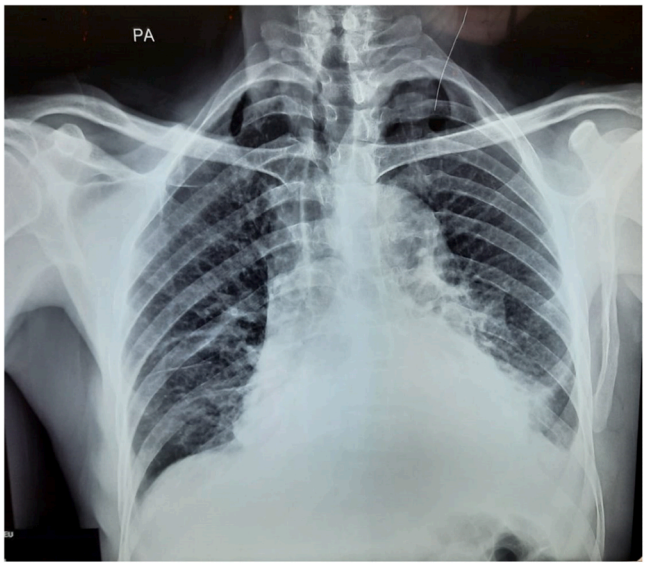

Fig. 2. Admission Chest X-ray, showing grade 2 cardiomegaly and the "inverted cup" sign.

Table 2
Pericardial fluid studies.

Test	Result
Cytochemistry	
Appearance	Slightly bloody
Cells	648 cel/μL
Neutrophils	95 %
Lymphocytes	5 %
Glucose	0 mg/dl
Proteins	67.36 g/dl
Xpert MTB-RIF	Positive for Mycobacterium tuberculosis with no rifampin resistance
Pericardium Biopsy	Fibroadipose tissue partially covered by mesothelium with acute and chronic inflammation

effusion, and cardiomegaly. The electrocardiogram revealed poor R-wave progression in the limb leads and T-wave flattening, while the echocardiogram showed pericardial effusion, consistent with some of the clinical characteristics of tuberculous pericarditis, such as the insidious onset of systemic symptoms [1,2,8,10,11] and manifestations of heart failure [7,11]. Furthermore, considering that in developing countries, 50–70 % of cases of pericardial effusion are due to tuberculosis in HIV-negative patients, this proportion rises to 87 % in HIV-positive patients [1,3,6,13], it is essential to consider tuberculosis as the underlying cause of pericardial effusion in countries like Honduras, which is endemic for tuberculosis.

The symptoms of tuberculous pericarditis vary depending on the characteristics of the pericardial fluid, such as the rate of fluid accumulation, hemodynamic effects, and the degree of inflammation[3]. This leads to the development of pericardial effusion, and whenever it exceeds 1 cm, pericardiocentesis is necessary to determine the cause of the effusion, provide therapeutic benefit, and reduce the risk of fluid reaccumulation, which may require repeated pericardiocentesis [1,3,7]. In this case, to address the cause of the pericardial effusion, a subxiphoid pericardiocentesis was performed, allowing for the collection of pericardial fluid samples for analysis. Moreover, this procedure improved the patient's symptoms and prevented the development of cardiac tamponade.

To determine the cause of pericardial effusion, it is necessary to address differential diagnoses, starting with the analysis of pericardial fluid, as was done in our patient. The cytochemical examination showed an increase in cellularity due to neutrophils, accompanied by elevated protein levels, suggesting an acute infectious cause, likely bacterial, similar to the findings observed in the first pathological stage of tuberculous pericarditis [3,5,8]. It is important to note that there are other infectious causes of pericardial effusion; the main ones are outlined in Table 3.

Table 3
The characteristics of the main causes of infectious pericarditis.

Characteristics	Tuberculous Pericarditis	Staphylococcal Pericarditis	Influenza-Induced Pericarditis
Epidemiology	In developing countries, more than 50–70 % of cases of pericarditis [1,3,6]	In the antibiotic era, it causes 22 % of purulent pericarditis cases 18	Cardiac involvement by influenza is variable, ranging from 0–10 % [19]
Clinical Manifestations	Nonspecific systemic symptoms, fever, night sweats, weight loss, dyspnea, intense and sharp chest pain, pericardial friction rub [1,3,10,11]	Chest pain, tachycardia, dyspnea, fever, hypotension, paradoxical pulse, muffled heart sounds, and pericardial friction rub [20]	Progression of dyspnea, chest pain, and hemodynamic instability [21]
Evolution	Insidious onset [8,10,11]. Occasionally acute presentation that progresses to cardiac tamponade [8]	Acute clinical presentation, symptom duration 1–18 days [20,22]	Cardiac symptoms begin rapidly, 4 to 9 days after the onset of influenza symptoms [21,23] Typically self-limiting [24]
Pathogenesis	-Lymphatic dissemination -Hematogenous dissemination -Direct dissemination [1,3,7–9]	-Invasion from contiguous foci (pneumonia) -Traumatic implantation (cardiothoracic surgery) -Hematogenous spread [18,22]	-Direct viral invasion and increased expression of TNF-α and its myocardial receptors [24]
Treatment	Antituberculous therapy: -Rifampicin -Isoniazid -Pyrazinamide -Ethambutol, minimum duration of 6 months [1,7,8,16]	Penicillins or cephalosporins are the drugs of choice; in cases of methicillin-resistant Staphylococcus, use vancomycin for 3 to 4 weeks 18	-Neuraminidase inhibitors, NSAIDs combined with colchicine [21,23]-In fulminant myopericarditis, immunomodulatory therapy with intravenous human immunoglobulin is studied [21]
Use of Corticosteroids	Indicated especially in individuals without HIV and in people with HIV who are not on antiretrovirals [17]	In some cases, it may be beneficial [25]	Corticosteroids are not indicated as they can reactivate viral infections and cause persistent inflammation [21,24]
Mortality	8–17 % mortality, and 85 % without treatment [1,3,6,26]	Without treatment, mortality is 100 %; with appropriate management, it can decrease to 40 % [18,22]	24 % to 35 % [21]

To identify the causative agent, the Mycobacterium tuberculosis bacillus, various diagnostic methods are available. The gold standard remains the culture [1,3,7,8], but its long waiting time for results and the need to begin anti-tuberculosis treatment as soon as possible complicate its practical utility. Therefore, other diagnostic methods such

as molecular tests can be used; the Xpert MTB/RIF test, which has high specificity and also indicates rifampicin resistance. In our patient's case, the Xpert MTB/RIF result was obtained in 4 h, revealing Mycobacterium tuberculosis without rifampicin resistance, which allowed for the early initiation of treatment.

The treatment of tuberculous pericarditis is based on anti-tuberculosis chemotherapy combined with four drugs: Rifampicin, Isoniazid, Pyrazinamide, and Ethambutol, with a minimum duration of 6 months [1,7,8,16]. The concomitant use of corticosteroids has been controversial, but it is currently recommended, especially in cases involving HIV-negative patients and large volumes of pericardial effusion [1,7,8,15], as corticosteroids help prevent the development of constrictive pericarditis, reduce mortality from pericarditis, and decrease the need for repeated pericardiocentesis [17]. In the case of our patient, the early initiation of anti-tuberculosis therapy along with the use of prednisone contributed to the patient's good progress, as it helped prevent further accumulation of pericardial effusion, facilitating the early removal of the pericardial drainage system. As a result, an improvement in symptoms was observed, which allowed for discharge and continued outpatient management through adherence to the anti-tuberculosis and corticosteroid treatment regimen with a tapering schedule.

Author statement

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We also warrant that the article is not being considered for publication elsewhere.

Publication of the article is approved by all authors and tacitly or explicitly by the responsible authorities where the work was performed.

If accepted, the article will not be published elsewhere in the same form, in English or any other language, including in electronic format, without the written consent of the copyright holder.

Consent

Written informed consent is obtained from the patient. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Ethical approval

The study is a case report, only information from the patient's file was used, no type of intervention was performed with the patient, so it does not have approval from the ethics committee.

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Conflict of interest

No author has any conflict of interest.

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