

Constrictive pericarditis due to *Mycobacterium tuberculosis*

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Abstract

Tuberculosis is an infectious disease caused by *Mycobacterium tuberculosis*, which is widely disseminated in developing countries. It can affect any organ or system, leading to high morbidity and mortality if untreated.

A 22-year-old incarcerated patient presented to the emergency room complaining of epigastric pain, moderate dyspnea on exertion which progressed rapidly to resting dyspnea, and ankle edema. A chest x-ray showed massive right pleural effusion, with an enlarged cardiac silhouette due to severe pericardial effusion, confirmed by echocardiography.

Pericardiocentesis revealed hemorrhagic fluid, with *Mycobacterium tuberculosis* documented through molecular tests and culture media. Despite starting antitubercular treatment and oral steroids, the patient developed constrictive pericarditis that required phrenic pericardiectomy. (*Acta Med Colomb* 2025; 50. DOI: <https://doi.org/10.36104/amc.2025.3756>).

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Introduction

Pericarditis is a rare complication of tuberculosis, occurring in 1-2% of cases. This infection accounts for less than 5% of pericarditis cases in low-prevalence countries. However, in areas with a high incidence, like Asia and Africa, it may account for over 60% of the causes of pericardial effusion in non-HIV positive patients, increasing to more than 90% with human immunodeficiency virus (HIV) coinfection. This condition's mortality increases with tuberculosis-HIV coinfection, ranging from 17-34%, respectively (1).

In Colombia, there was a 20.6% increase in the number of cases in 2022 compared to 2021. As far as the incidence rate, there were 31.69 cases per 100,000 inhabitants recorded in 2022, higher than the 28.24 cases per 100,000 inhabitants in 2019, and a 19.4% increase compared to the 2021 rate (26.53 cases per 100,000 inhabitants). The rate of extrapulmonary tuberculosis is 4.5 cases per 100,000 inhabitants, with tuberculous pericarditis being the least frequent (2).

Delayed diagnosis often leads to more late complications, like constrictive pericarditis and cardiac tamponade. This scenario is aggravated by the high microbacterial load, resulting in increased mortality (1). Pericardial infection occurs due to retrograde dissemination of the bacillus from adjacent lymph node structures or hematogenous spread, and progresses through four phases: first, a polymorphonuclear-rich fibrinous exudate; then a lymphocytic serosanguinous exudate; next, exudate resorption; and, finally, pericardial thickening due to fibrin, scarring and fibrosis (3, 4).

Case presentation

A 22-year-old incarcerated patient presented to the emergency room with epigastric pain, moderate dyspnea on exertion that progressed rapidly to dyspnea at rest, and ankle edema. On admission, a chest x-ray showed a massive right pleural effusion with enlargement of the cardiac silhouette. A cardiac point-of-care ultrasound (POCUS) showed severe pericardial effusion with internal septations and signs of imminent tamponade, requiring emergency pericardiocentesis. Approximately 500 ml of hemorrhagic fluid with low glucose and abundant lymphocytes were drained, characteristic of an exudate (Table 1).

The accompanying symptoms were notable for weight loss over the previous month and an occasional cough. Con-

Table 1. Pericardial fluid cytochemistry.

Pericardial fluid cytochemistry	Values	Reference range
Appearance	Hemorrhagic	Clear
Glucose	<1.11 mmol/L	2.2-4.4 mmol/L
Protein	5.6 gr/dL	1-3 g/dL
LDH	1,222	276-517 UI/L
Albumin	2.20 gr/dL	1.19-3.06 g/dL
LDH: Lactate dehydrogenase.		

sidering his history and the characteristics of the pericardial fluid, a real-time PCR was ordered using GeneXpert MTB/RIF Ultra, which showed *Mycobacterium tuberculosis*. Molecular PCR with GenoType MTBDR plus and the sensitivity test confirmed sensitivity to rifampin and isoniazid, and therefore antitubercular treatment was started with rifampin/isoniazid/pyrazinamide/ethambutol. Corticosteroids and colchicine were also started.

Despite medical treatment, the pericardial effusion recurred with echocardiographic signs of high filling pressure due to constriction. Right heart catheterization showed mild pulmonary hypertension, moderate to severely elevated end-diastolic pressures with pressure equalization between the ventricles (left ventricular pressure of 19 mmHg and right ventricular pressure of 18 mmHg), and slightly elevated pulmonary resistance; additional findings are shown in Figures 1-3. In light of these results, a phrenic pericardiectomy was performed, with satisfactory clinical improvement. The pericardial biopsy was consistent with necrotizing chronic granulomatous pericarditis.

Discussion

Cardiovascular impairment from *Mycobacterium tuberculosis* mainly affects the pericardium and, in exceptional cases, the myocardium or other heart structures. Tuberculosis-related pericarditis usually occurs in immunocompromised patients with HIV infection and, less frequently, in immunocompetent patients. In rare cases, with or without pericarditis, left ventricular systolic dysfunction may occur. Heart failure symptoms may be overlooked, as they overlap with the general symptoms of tuberculosis (5).

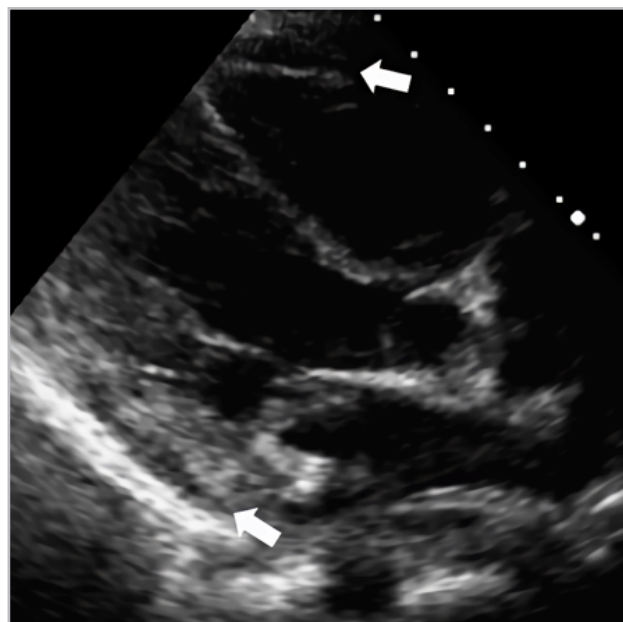


Figure 1. Lumpy pericardial effusion. Recurrence of pericardial effusion (White arrows).

The most common complication of pericardial tuberculosis is pericardial effusion, which occurs in up to 79.5% of cases. The second most common manifestation is constrictive pericarditis, with an incidence that reduces from 30-60% to 5-25% after starting antitubercular treatment (6).

Constrictive pericarditis is a consequence of the local inflammatory response to mycobacterial antigens, leading to a deterioration in cardiac function, with tamponade and chronic

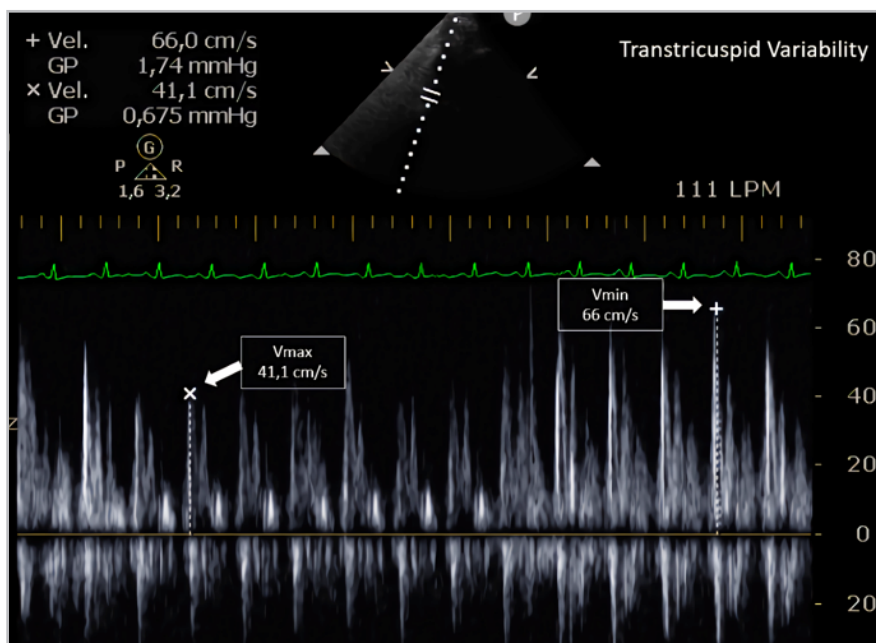


Figure 2. Transtricuspid variability ($V_{max} (66 \text{ cm/s}) - V_{min} (41.1 \text{ cm/s}) / V_{max} (66 \text{ cm/s}) = 37\%$). Greater than 35% is considered significant.

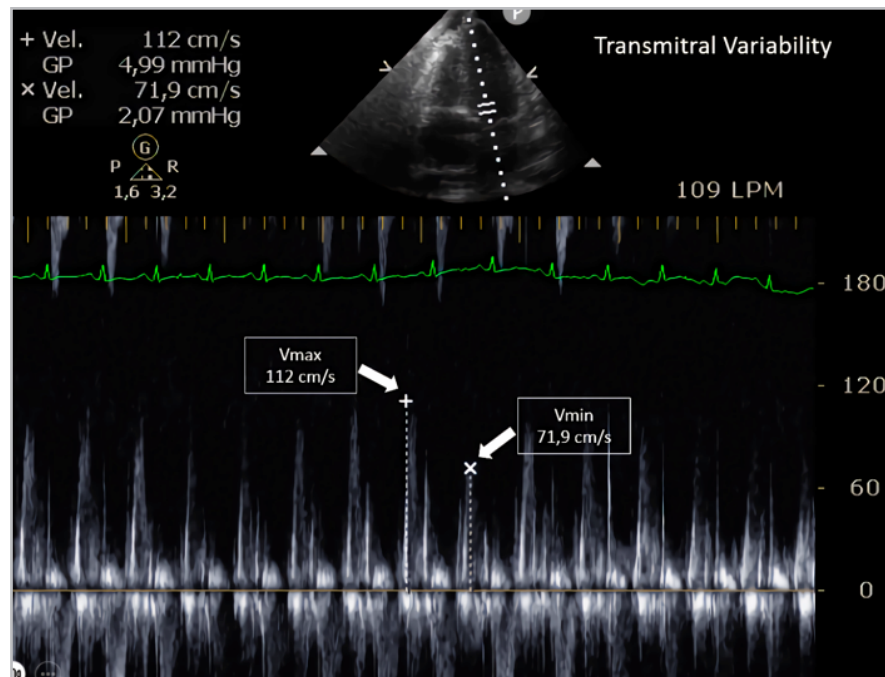


Figure 3. Transmitral variability. ($V_{\max}$ (112 cm/s) - $V_{\min}$ (71.9 cm/s) / $V_{\max}$ (112 cm/s) = 35%). Greater than 25% is considered significant.

pericardial damage (7). However, not all patients develop this complication after acute phase resolution. A retrospective study in Korea analyzed the medical records of 50 patients with tuberculous pericarditis who underwent echocardiography one, three and six months after beginning antitubercular treatment with or without steroids. Min Sun Kim et al. found that the initial echocardiographic characteristics seemed to predict the risk of developing constrictive pericarditis. Patients with pericardial effusion and no constrictive physiology had a much lower risk (3%) when they received antitubercular treatment and steroids. Patients who initially had pericardial effusion and constrictive physiology improved with medical treatment, as the constrictive pericarditis was reversible with proper treatment in 80% of cases (8).

Due to its immune-mediated nature, corticosteroids have been proposed as an adjuvant to antitubercular treatment for more than 40 years, to reduce the rate of constrictive pericarditis and mortality. In one of the first studies on this subject, published in *Lancet* in 1987, the use of corticosteroids in addition to antitubercular treatment for 11 weeks showed more rapid clinical improvement with lower rates of pericardiectomy, but a non-significant reduction in mortality (9). Although these results were refuted years later with shorter prednisolone courses that achieved significant mortality reductions, this study only included 58 HIV-positive patients (10).

The IMPI study provided a more robust analysis, with 1,400 patients, in which BM Mayosi et al. found that a six-week course of prednisolone did not achieve a significant effect on the primary composite outcome of death, cardiac tamponade requiring pericardiocentesis or constrictive pericarditis (23.8 vs. 24.5% HR 0.95; 95% CI, 0.77-1.18; $P =$

0.66). However, corticosteroids were associated with significant reductions in the incidence of constrictive pericarditis (4.4 vs. 7.8% HR 0.56; 95% CI, 0.36-0.87; $P = 0.009$) and hospitalizations (20.7 vs. 25.2% HR 0.79; 95% CI, 0.63-0.99; $P = 0.04$), despite a concerning increase in some types of HIV-related malignancies (11).

In light of the current evidence, the Infectious Diseases Society of America (IDSA) does not recommend routine use of steroids, except in selected cases like those with massive effusions, high levels of inflammatory cells in the pericardial fluid or early signs of constrictive physiology (12).

Inflammation and pericardial fibrosis lead to diastolic heart failure, for which pericardiectomy continues to be the treatment of choice. In a meta-analysis that included 12 articles and 859 patients with tuberculous constrictive pericarditis, total pericardiectomy was the most frequently used treatment. A significant reduction in all-cause mortality was found in patients with tuberculous pericarditis, compared to other etiologies (combined RR 0.34, CI [0.12, 1.01], $I^2 = 61\%$), with significant improvement in these patients' New York Heart Association (NYHA) functional class one year after pericardiectomy (RR 8.04, CI [5.20; 12.45], $I^2 = 0\%$) (13). However, this procedure is not challenge-free, as some centers have recorded perioperative mortality rates of up to 12%. This has encouraged exploration of alternatives like intrapericardial fibrinolysis, currently being studied in the IMPI-2 trial (14).

Conclusion

Tuberculosis is a widely distributed infection around the world, which can affect any organ in the body. Although

pericardial involvement is relatively rare, lack of timely detection leads to a high risk of morbidity and mortality, and therefore a high index of suspicion is required. The antitubercular treatment is the same as that used for pulmonary tuberculosis, and although the use of corticosteroids has not proven to reduce mortality, it does impact the progression to constrictive pericarditis. When the latter occurs, the prognosis worsens and pericardiectomy is justified as a salvage measure to improve cardiac filling pressures and hemodynamics in general.

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