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Tuberculous Pericarditis, Two Case Report

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ABSTRACT

Background: *Mycobacterium tuberculosis* is among the leading causes of death, usually in underdeveloped countries, being the state of Baja California with most cases and deaths in Mexico. This infection can affect pericardium, although infrequent and it represents 1-4% of the cases of tuberculosis. The relevance of this presentation is the difficulty of diagnosis and high mortality.

Objective: Demonstrate the importance of keeping the high suspicion that this type of tuberculosis in endemic entities because of high mortality and the existence of treatment.

Description Of The Cases: 39 year old male who practiced sex with men and had multiple comorbidities. Referred to the emergency department to start antituberculous treatment. During his stay he presented deterioration of health. Pericardiocentesis was performed with results suggestive of *Mycobacterium tuberculosis*. He subsequently presents cardio respiratory arrest without spontaneous return of the circulation. The second case it's about a 69 year old male who came into the emergency room with lower respiratory tract infection symptoms with two months of evolution and signs of cardiac insufficiency. An echocardiogram was performed meeting pericardial effusion which confirms the etiology of tuberculosis and started an antituberculous treatment with good evolution until discharge.

Conclusion: Currently, in endemic zones of *Mycobacterium tuberculosis* is important to suspect this agent in view of typical presentation of infection and atypical presentation of frequent pathologies.

KEYWORDS: tuberculous, tuberculous pericarditis, tamponade cardiac

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BACKGROUND:

Internationally, *Mycobacterium tuberculosis* is among the top 10 causes of death, especially in low- and middle-income countries (1). In Mexico, it is considered an endemic health problem and its incidence has increased (2), with the states with the highest number of cases and deaths being: Baja California, Veracruz, Guerrero; Mexicali appearing as one of the cities with the highest concentration of affected people (3). In Mexico during 2023 at the time of epidemiological week 9, a total of 628 reported cases of non-respiratory or non-meningeal tuberculosis have accumulated, of which 71 cases are from Baja California (4).

Tuberculous pericarditis represents 1-4% of all forms of tuberculosis and 1-2% of extrapulmonary tuberculosis (5), and has a prevalence of 1-2% in endemic areas (6) (7). On the other hand, it represents 10% of all cases of pericarditis, reaching a prevalence of up to 50-70% of constrictive pericarditis in countries with a high incidence (5).

Immunosuppression is a crucial risk factor for the acquisition of tuberculosis (1), showing frequent comorbidity between tuberculosis and HIV. Thus, it is expected that in developing countries where there is a high prevalence of tuberculosis, pericardial tuberculosis represents more than 90% of HIV-positive patients (5). Due to the above, *Mycobacterium tuberculosis* etiology should always be suspected as the cause of constrictive pericarditis in patients who live in endemic regions, receive immunosuppressive treatment or biological therapies, who have coinfection with HIV, diagnosed with chronic kidney disease and especially if they receive dialysis, have diabetes mellitus, or are alcohol or drug users (5). In addition to this, mortality in untreated patients with HIV is three times higher than in patients without it (7).

By itself, pericardial tuberculosis has a mortality of 17 to 40%, but if it does not receive adequate treatment, up to 50% of patients can develop cardiac tamponade and their mortality increases to 90% at 6 months (1, 6).

Mycobacterium tuberculosis can enter the pericardium by retrograde lymphatic dissemination from the mediastinal lymph nodes, the peritracheal, and peribronchial area; by hematogenous route in primary tuberculosis or, infrequently, by direct transmission from an adjacent infected structure such as the lungs, pleura and spine (8, 9).

Two mechanisms can carry on clinic presentation of tuberculous pericarditis: fluid buildup in the pericardium space which compresses the heart chambers during the cardiac cycle, without being able to reach filling and cardiac contraction (8). On the other hand, it could be caused by thickening of the pericardium or absence of effusion resulting in diastolic impairment (8). Being the majority of the manifestations due to the loss of pericardial compliance (10). Part of the problem with this condition is that the classic symptoms of acute pericarditis such as sharp stabbing pain and pericardial friction rub are not usually common in tuberculous pericarditis, which is a more insidious and systemic presentation (1). The most common symptoms and signs in this presentation are usually increased cardiothoracic index on chest x- ray, increased central venous pressure, fever, muffled heart sounds, sinus tachycardia, palpitations, cough, dyspnea, Kussmaul's sign (which consists of an inspiratory increase in systemic venous pressure that is reflected as increased jugular venous distension), hepatomegaly, angina pectoris, pleural effusion (1, 6) and the most predominant clinical manifestation being similar to heart failure (8).

In a study in Africa in patients carrying the human immunodeficiency virus coinfected with *Mycobacterium tuberculosis*, it was found that 15-20% of the etiology of pericardial diseases is not diagnosed due to the lack of precision of diagnostic tests.(1).

The definitive diagnosis requires the isolation of bacillus in the pericardial fluid or histopathological tissue(1), pericardiocentesis is recommended in cases of diagnostic doubt and in effusions larger than 1 cm (12). The pericardial fluid has a very low concentration of mycobacteria and the diagnosis with acid-fast bacillus (AFB) can be difficult (12), so the levels of adenosine deaminase (ADA), gamma interferon and polymerase chain reaction for *M. tuberculosis* should be analyzed. In addition, here are also established diagnostic criteria (Table 1).

Table 1: Diagnostic criteria for tuberculous pericarditis in endemic settings (12).

Diagnostic criteria for tuberculous pericarditis in endemic settings	
Definitive	**The bacilli are found in stained smears or culture of pericardial fluid; and/or In the histopathological study of the pericardium, bacilli or caseating granulomas are found.**
Probable	**Evidence of pericarditis in a patient with tuberculosis demonstrated elsewhere in the body; and/or Lymphocytic pericardial exudate with elevated ADA activity; and/or Good response to antituberculosis chemotherapy.**

The treatment of this pathology consists of the use of the four classic drugs against tuberculosis (rifampicin, pyrazinamide, isoniazid and ethambutol) together with corticosteroids (1, 13).

Table 2: Diagnostic tests in pericardial fluid for tuberculosis

Diagnostic test	**Sensitivity**	**Specificity**
ADA	**90%**	**86%(14)**
Culture	**53-75%(8)**	**100%(15)**
Biopsy	**10-64%(12)**	**100% (5)**
Xpert MTB/RIF	**Líquid 63% (2 hrs) Biopsy 78%**	**≈100%(16)**
Gamma-interferon (IFN-γ)	**97%**	**100%(12)**

CASE REPORT 1

A 39 years old male, who practices sex with men. As pathological history he has type 2 diabetes mellitus, of two years of evolution treated with metformin 850 mg every 24 hours, with poor control, systemic arterial hypertension treated with losartan 50 mg every 24 hours, long term hypothyroidism treated with levothyroxine at a dose of 100 mcg per day and anxiety disorder, treated with sertraline at a dose of 50 mg every 24 hours.

The current condition begins 3 months prior to admission with a wet cough with greenish sputum and unquantified fever, nocturnal diaphoresis and weight loss of 10 kg. Days prior to his admission, he presented progressive dyspnea that led him to seek medical attention at a health center, underwent an AFB test on suspicion of *Mycobacterium tuberculosis*, which was reported positive and was referred to the secondary care center.

He is admitted with the following vital signs: heart rate of 106 beats per minute, respiratory rate of 23 breaths per minute, blood pressure of 140/85 mmHg, temperature of 37°C, room air saturation of 82%. On physical examination, the presence of thick bilateral rattling respiratory sounds, use of accessory muscles for breathing, heart sounds decreased in intensity, without the presence of heart murmurs. During hospital stay, intensive treatment for pulmonary tuberculosis plus intravenous antibiotic therapy with ceftriaxone 1 gram every 12 hours and clarithromycin 500 milligrams every 12 hours was started. Control laboratories are taken which are shown in the following tables (Tables 3, 4, 5). The chest x-ray and the electrocardiogram taken on admission are shown in Figure 1 and Figure 2 . During the evolution he developed deterioration of alertness, hypotension, jugular vein distention. Echocardiogram-guided pericardiocentesis was performed, from which 20 ml of pericardial fluid were extracted with the characteristics shown in Table 5. Hours later, ventricular tachycardia developed, which led to advanced cardiopulmonary resuscitation maneuvers without being able to obtain return of spontaneous circulation and death was declared.

Table 3. Sexually transmitted diseases (case report 1)

03/15/2023	Result
Syphilis	Reactive 3.6
HIV	Reactive 832.63
Hepatitis B surface antigen (HBsAg)	Non reactive 0.18
AC against Hepatitis (HCV)	Non reactive 0.07

Table 4. Laboratory result (case report 1)

Laboratories	Result 03/10/2023	Result 03/15/2023	Normal ranges
Hemoglobin	11.3	12.5	12.5 g/dL
Platelets	186	148	150-450 x10^9
Leukocytes	17.2	7.6	5-10 x10^9
Neutrophils	97.88	93.28	40-75%
Lymphocytes	0.86	3.5	20-55%
Glucose	144	84	60-100mg/dL
Creatinine	1.6	0.46	0.72-1.25 mg/dL
Urea	40	70.6	19-42.8mg/dL
BUN	15	33	5.1-16.8 mg/dL
Uric acid	4.4	8.2	3.5-7.2 mg/dL
Albumin	2.3	1.6	3.5-5.2 g/dL
Direct bilirubin	4.4	3.5	0-0.5 mg/dl
Total bilirubin	5.5	4.9	0.2-1.2mg/dl
Alkaline phosphatase	590	514	40-150 U/L
Lactate dehydrogenase	439	346	125-220 U/L
AST	138	115	5-34U/L

ALT	60	60	0-55 U/L
GGT	286	335	12-64 U/L
Chlorine	87	101	98-107mmol/L
Sodium	125	133	137-145 mmol/L
Potassium	4.5	4.3	3.6-5 mmol/L

Table 5. Pericardial fluid analysis (case report 1)

	Result	**Normal ranges**
Volume	20 ml	15-50 ml (24)
Aspect	Cloudy	Hemorrhagic(25)
Color	Red	Light - yellow (25)
pH	8.5	7.57 +-0.11 (26)
Leukocytes	Plenty	0
Mononuclear cells	10	0
Polymorphonuclear	90	0
Glucose	47	40-80 mg/dl (27)
Albumin	1.1	
Proteins	2.7	3.1 +- 0.6 g/dL (26)
Gram stain	gram + Cocci, gram + bacilli	Negative
AFB	Positive, moderate	Negative

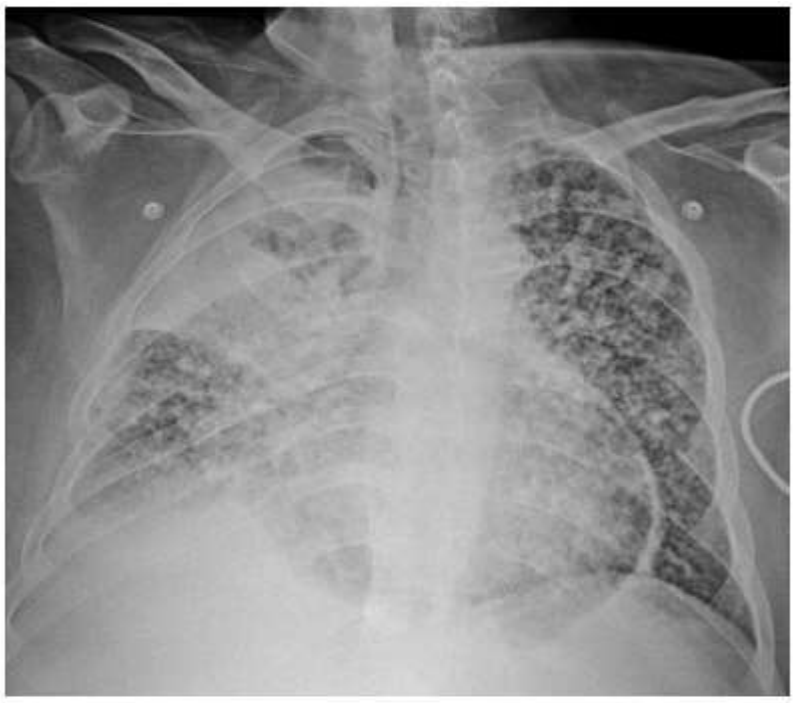
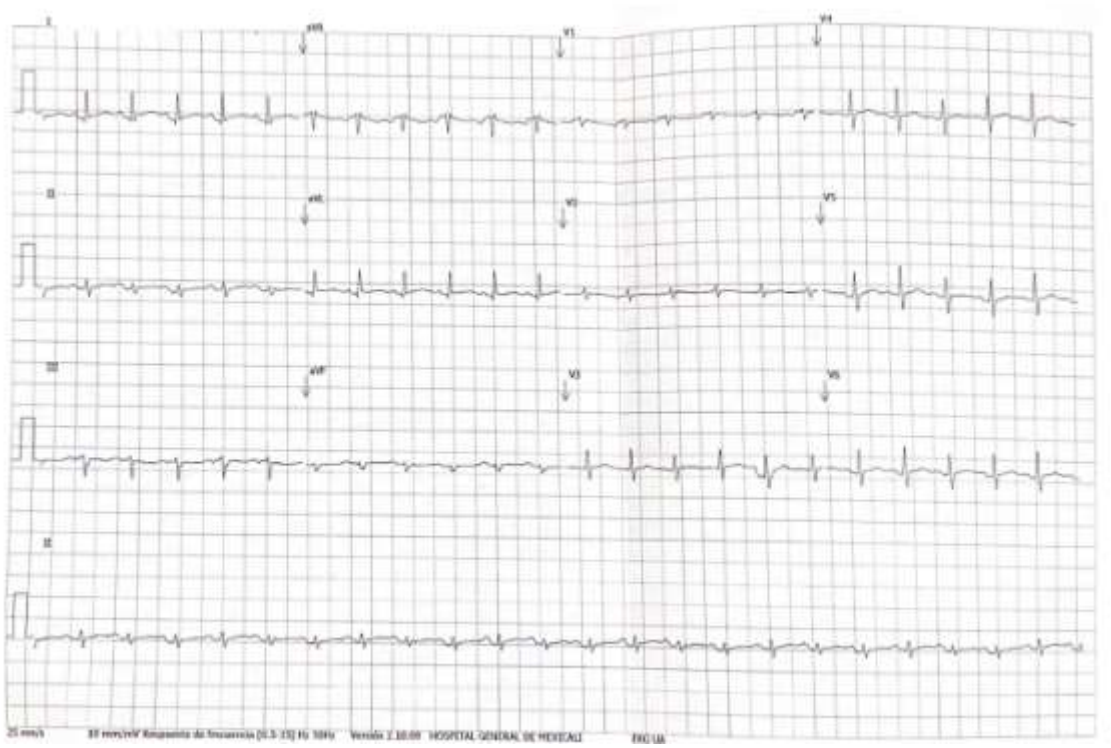


Figure 1. Anteroposterior chest X-ray showing “water bottle” sign, homogeneous radiopaque image in the right upper lobe and image suggestive of ipsilateral apical cavitation, with diffuse nodular infiltrate, and effacement of right costophrenic and cardiophrenic angles.

Figure 2.



Electrocardiogram: Electrocardiogram with sinus tachycardia, with normal axis, with electrical alternation phenomenon and microvoltage, without other alterations of waves and segments.

CASE REPORT 2

A 69-year-old male with history of a tonsillectomy and blood transfusion 40 years ago, alcoholism at the rate of 1 liter of fermented beverages daily, smoking, alkaloids and cannabis drug usage, native and resident of Mexicali, denied vaccination in recent years.

He began his current condition 2 months prior to his admission, presenting asthenia, adynamia, and unintentional weight loss of approximately 10 kg. One month prior to his admission, mMRC 2 dyspnea began, which decreased with rest in the semi-fowler position, however it was progressive dyspnea. Subsequently, back pain and a dry cough that became productive with whitish sputum a day prior to his arrival are added. Dyspnea worsens for which he is taken to the emergency unit by ambulance.

On arrival, he had heart rate 127 beats per minute, respiratory rate 33 breaths per minute, blood pressure 119/75 mmhg, temperature 36.7°C, with telangiectasias on the anterior side of the thorax, generalized coarse crackling rales predominantly parahilar, bilateral basal hypoventilation, hepatojugular reflux, lower limb edema +/+++, otherwise normal. Due to a history of drug and alcohol abuse and because he met the Framingham criteria with a classification of severity NYHA III, heart failure was suspected. A bedside transthoracic echocardiogram was performed, which revealed a global pericardial effusion causing a 27 mm separation of the pericardial sheets, with no signs of hemodynamic compromise nor cavity collapse. After this, the diagnosis of pericardial effusion is established, ceftriaxone is started, and the surgery department is consulted to perform pericardiocentesis.

By the 3rd day of hospitalization, he is hemodynamically unstable, with tachycardia, hypotension, and tachypnea, unable to tolerate the supine position, for which norepinephrine 8 mg/24 h is started in an infusion pump to maintain MAT of 70 mmHg. After this, he is maintained hemodynamically stable with amines and the following day pericardiocentesis is performed obtaining 315 ml of bloodish fluid, which is sent for cytological, cytochemical, AFB, Gram stain, papanicolaou test (PAP smear) and ADA study. On the 5th day of hospitalization, a contrast-enhanced chest tomography was performed to address the etiological diagnosis of neoplasia against pulmonary tuberculosis against viral pericarditis. In this study, the pericardial effusion was confirmed, and we observed its intrapericardial catheter for drainage as well as left basal atelectasis and air bronchogram area, suggestive of an added pneumonic process, as well as fibrosis in this same lobe and minimal laminar thickening of the pleura adjacent to these areas. One week after admission, 80 mL of pericardial fluid is drained and negative serial AFB results are obtained.

On day 9 of the hospital stay, he presented clinical improvement, decreased intensity of dyspnea, and 100 cc of pericardial fluid was drained, with a total of 665 ml drained up to that day. The next day the PAP smear result arrives, reporting 99% lymphocytes, 500 per field, and 1% macrophages. On his 16th day, the result of the ADA of pericardial fluid arrived, which was positive 105. Subsequently, treatment for tuberculosis was started, the

catheter was removed and after 24 hours of observation he was discharged from the hospital.

He is scheduled for control and follow-up, however contact is lost in post-discharge follow-up.

Table 6. Serial smears

BAAR	Result
02/08/2020	Negative
02/09/2020	Negative
02/10/2020	Negative

Table 7. Sexually transmitted diseases (case report 2)

06/02/2020	Result
HIV	Non reactive
Hepatitis B surface antigen	Non reactive
AC against Hepatitis (HCV)	Gray zone

Table 8. Laboratory result (case report 2)

Laboratories	02/05/2020	02/09/2020	Normal ranges
Hemoglobin	8.5	10	12.5 g/dL
Platelets	239.000	233.000	150-450 x10^9
Leukocytes	6.58	3.32	5-10 x10^9
Neutrophils	92.8	54.2	40-75%
Lymphocytes	2.0	18.6	20-55%
Glucose	130	84	60-100mg/dL
Creatinine	0.63	0.70	0.72-1.25 mg/dL
BUN	8.0	17.8	5.1-16.8 mg/dL
Uric acid	3.2	5.3	3.5-7.2 mg/dL
Albumin	2.6	-	3.5-5.2 g/dL
Direct bilirubin	1.13	-	0-0.5 mg/dl
Total bilirubin	2.0	-	0.2-1.2mg/dl
Alkaline phosphatase	86	-	40-150 U/L
Lactate dehydrogenase	488	-	125-220 U/L
AST	63	-	5-34U/L
ALT	24	-	0-55 U/L
GGT	59	-	12-64 U/L
Chlorine	96	98.2	98-107mmol/L
Potassium	3.66	3.7	137-145 mmol/L
Sodium	129.7	138.9	3.6-5 mmol/L

CONCLUSIONS

In endemic areas of *Mycobacterium tuberculosis*, it is important to suspect this agent in typical presentations of tuberculosis infection and in atypical presentations of frequent pathologies. In our town, due to the high prevalence of this microorganism, this microorganism is often thought of early, which is why the infectious disease department grants treatment early. However, as we were able to observe with the first case, which coincides with international survival statistics, antituberculous treatment may not be enough to improve the prognosis of a tuberculous pericardial effusion, since these patients usually have comorbidities that worsen the outcome.

It is important not to forget that *Mycobacterium tuberculosis* is a "mimic" bacterium, and we must keep in mind the strong relationship that HIV has with this disease, likewise the presentation of pericardial effusion by *M. tuberculosis* is usually silent, so in our country a high diagnostic suspicion in a patient with hemodynamic deterioration is essential for an adequate approach and management.

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