Electrical alternans in a cardiac tamponade – the first sign of tuberculosis

Alternanța electrică din tamponada cardiacă – prim semn al tuberculozei

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Abstract

Pericarditis is a rare manifestation of tuberculous disease, occurring in approximately 1 to 2 percent of patients with extrapulmonary tuberculosis. Large pericardial effusions are uncommon, even fewer patients developing tamponade as the initial manifestation. Tuberculous pericardial effusion usually develops insidiously, presenting with nonspecific systemic symptoms, and is associated with immunosuppression, as in HIV infection, but extremely rare it can be the first manifestation of tuberculosis, as in the case of our patient.

Keywords: pericarditis, tuberculosis, cardiac tamponade

Rezumat

Pericardita este o manifestare rară a bolii tuberculoase, apărând la aproximativ 1-2% dintre pacienții cu tuberculoză extrapulmonară. Pericardita cu lichid în cantitate mare este rară, cu atât mai rară fiind tamponada cardiacă drept manifestare inițială. De obicei, pericardita tuberculoasă se dezvoltă insidios, cu simptome nespecifice, fiind asociată cu o scădere severă a imunității, ca în sindromul imunodeficienței umane dobândite, dar extrem de rar poate fi și prima manifestare a tuberculozei, așa cum este cazul pacientului pe care îl prezentăm.

Cuvinte-cheie: pericardită, tuberculoză, tamponadă cardiacă

Introduction

Tuberculosis (TB) can involve any organ, lungs being the most common. Extrapulmonary tuberculosis (EPTB) is the term used to describe the occurrence of TB at body sites other than the lungs. It commonly involves lymph nodes, pleura, gastrointestinal tract, bone, central nervous system, or genitourinary system. TB-related pericardial disease is uncommon (<1% of EPTB), and the cases complicated by life-threatening cardiac tamponade are extremely rare(1).

The total of TB cases in industrialized countries is constantly declining, pericardial tuberculosis being particularly related to immunosuppression due to Human Immunodeficiency Virus (HIV)(2,3). We report a case of cardiac tamponade as first sign of tuberculosis (no TB history) in a mid-age patient without immunosuppression factors.

Case report

A 43-year-old man without medical history was admitted for increased fatigue, fever and dry cough. As clinical features, the patient presented acute dyspnea with tachypnea, tachycardia, hypotension with reduced pulse pressure, jugular turgescence, cardiac impulse difficult to palpate and muffled heart sounds. Laboratory studies showed currently inflammatory syndrome and mild iron deficiency anemia. Sinus tachycardia and QRS electrical alternans 2:1 were present on the electrocardiography (Figure 1). The chest radiograph revealed rounded appearance of the antero-posterior silhouette of the heart, mild bilateral pleural effusion, calcified lymph nodes and pleural fluid within horizontal fissure (Figure 2a, Figure 2b).

Emergency transthoracic echocardiography showed a large circumferential pericardial effusion, with early diastolic collapse of the right ventricle (Figure 3a); late diastolic collapse of the right atrium (Figure 3b) and “swinging heart” (Figure 3b, Figure 3c); distention of the inferior vena cava that didn’t diminish with inspiration (Figure 3d).

Emergency pericardiocentesis was needed, with drainage of 2.000 ml of hemorrhagic exudate, with intense monocytesis, adenosine deaminase (ADA) = 90 U/L, glucose 55 mg/dl, lactate dehydrogenase (LDH) = 120 mg/dl. The patient was redirected to the department of pneumology, where diagnostic thoracentesis evacuated 20 ml of haemoragic pleural effusion, exudate, ADA=16 U/L, glucose 60 mg/dl, LDH 138 mg/dl, pH, lactate- and amylase-levels were in normal range in both pleural and pericardial liquid. The cytological exam for neoplasia was negative. Pleural biopsy was performed and the histopathological examination was highly suggestive for tuberculosis: pleural epithelioid cell granuloma and Langhans giant cell (Figure 4).

It is worth mentioning that HIV test was negative and the patient remembers no TB contact in the last months.

Our patient had a favorable clinical outcome, without relapse under antituberculous therapy: the six-month course of therapy was completed uneventfully, with no evidence of the reconstitution of the inflammatory syndrome. Corticotherapy was unnecessary. Ultrasound at the end of the 6 month-treatment demonstrated no residual pericardial or pleural effusion.
**Figure 1.** Sinus tachycardia, QRS electrical alternans 2:1

**Figure 2.** Chest XRay; (a) postero-anterior projection and (b) lateral projection: rounded appearance of the antero-posterior silhouette of the heart, mild bilateral pleural effusion, calcified left hilar lymph nodes, pleural fluid within horizontal fissure of the right pulmonary lobe

**Figure 3.** Transthoracic echocardiography; (a) large circumferential pericardial effusion from about 55 mm, with early diastolic collapse of the right ventricle; (b) late diastolic collapse of the right atrium; (b) and (c) “swinging heart”; (d) distention of the inferior vena cava
Discussion

Tuberculosis presents with symptoms that vary with the anatomical site involved. The clinical manifestations of tuberculous pericarditis are various – chest pain, cough, dyspnea, fever, night sweats and fatigue may commonly arise. Patients may present subacutely with the development of constrictive pericarditis or, as commonly arise. Patients may present subacutely with the development of constrictive pericarditis or, as described in our case, acutely, with signs of heart failure due to pericardial fluid accumulation leading to cardiac tamponade(4).

Tuberculosis can be a cause of acute pericarditis, but usually has more chronic symptoms. It can be accompanied by signs and symptoms of acute pericardial inflammation, but these patients are generally critically ill and other components of their illness typically dominate. However, in a tuberculous pericarditis, emergency management and subsequent therapeutic interventions, mandatory in our case, are unusual(5).

Regarding the diagnosis of pericardial tuberculosis, the yield of Mycobacterium tuberculosis isolation from pericardial fluid is low. The probability of making a diagnosis is increased if both fluid and biopsy specimens are examined early in the effusive stage. Thus, there is a definite role for biopsy, the pericardial or pleural tissue revealing either granulomas or organisms in 80% to 90% of cases. Measurement of ADA, an enzyme produced by white blood cells, improves markedly the accuracy and speed of diagnosis, so that ADA higher than 40 units/liter in the fluid has a sensitivity of approximately 88% and a specificity of approximately 83%(6). Increased interferon-gamma in pericardial fluid is an additional marker that, when combined with ADA, provides an even greater accuracy. Thus, measurement of ADA and interferon-gamma should be routine whenever tuberculous pericarditis is suspected or when there is no clue for diagnosis. PCR to detect Mycobacterium tuberculosis can be performed, but its usefulness is disappointing, with a sensitivity lower than 30%(7).

The short-term goal of therapy is to treat the symptoms and tamponade, and the long-term goal is to prevent progression to constriction. Multidrug antituberculous therapy is mandatory: a 6-month therapy is the standard recommendation and has proved to greatly decreased mortality(8). However, effusions are likely to reaccumulate before the benefits of antimicrobial therapy are obtained that is why the role of corticosteroids is still debated(9). In our case the cortico therapy was unnecessary, considering the rapid response to the antituberculous therapy, with no reoccurrence of the pericardial fluid. Pericardiocentesis relieves tamponade, but open drainage and the establishment of a window along with pericardial biopsy are recommended, especially when needed for diagnosis.

In our patient, due to the nonspecific nature of the presenting symptoms and physical findings, the initial high values of ADA in pericardial fluid and then the paradoxically low values in pleural fluid, the final diagnosis was histological. However, the delays involved in making a diagnosis by culture were avoided and antituberculous drugs were early initiated. As a result, the patient had no recurrent pleural or pericardial effusion. Also, as a singularity of our patient, we would like to note the lack of the immunodeficiency status (tuberculous disease being often a finding in HIV-infected individuals) and, of course, cardiac tamponade as being the clue that lead to the discovery of a TB disease.

References