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RESEARCH ARTICLE

UNCOMMON MANIFESTATION OF TUBERCULOSIS IN AN IMMUNOCOMPETENT HOST

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Abstract

Tuberculous myocarditis is a very unusual manifestation of tuberculosis.Our case serves to highlight a potentially life threating and rare presentation of tuberculosis.We discuss here the epidemiology,clinical presentation , the diagnostic approach and the management of the myocardial tuberculosis.

Introduction:-

Tuberculosis is a major public health problem, with an estimated 10 million cases of the disease worldwide in 2018[1].

Cardiac tuberculosis is a rare but well-recognised condition, it is estimated that 1-2% of all cases of tuberculosis have cardiac involvement [2]; it mostly affects the pericardium but myocardial involvement through known, is exceedingly rare and associated with a high mortality.

We are presenting unusual manifestation of tuberculosis in an immunocompetent host with disseminated tuberculosis revealing tuberculous myopericarditis.

Case report:

A 23-year-old from Equatorial Guinea currently resident in Morocco; never treated for tuberculosis, with no notion of contagious tuberculosis and no toxic habits. Hewas admitted to our department with a 2 months history of dry cough, unencrypted fever, night sweats, an important weight lost and alteration of general condition; he also reported a right pleuritic chest pain and the apparition of multiples masses in the low back over a period of 3 weeks.

Upon admission, the patient was afebrile with a pulse of 110 beats/min, blood pressure of 100/60 mmHg, respiratory rate at 22/min and normal oxygen saturation. Physical examination revealed a dullness to percussion and the absence of breathing sounds in the lower right lung fields, a multiple paraspinal collection; cardiovascular examination was unremarkable, with no sign of right heart failure, peripheral pulses were seen without added murmur.

Laboratory analysis revealed microcytic hypochromic anemia (haemoglobin 8,7g/dl), lymphopenia (500/mm3), hyponatremia (129 mmol/l), and elevated C-reactive protein levels at 183.82 mg/dl. His thyroid function, troponin levels and creatine phosphokinase were within normal; hepatitisserologiesand HIV serology were negative.

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His chest radiograph showed a right pleural effusion and a cardiomegaly, the electrocardiogram shows a sinus rhythm, constant PR, without cavitary hypertrophy or repolarization disorder(figure 1).

A chest CT scan revealed an encysted focal pleural fluid collection in right lung field(figure 2,3) and moderate pericardial effusion, large paravertebralabscessesextented to the spinal cord associated with multiple lytic lesions of thoracic vertebral bodies and posterior ribs arch. (figure 4, 5)

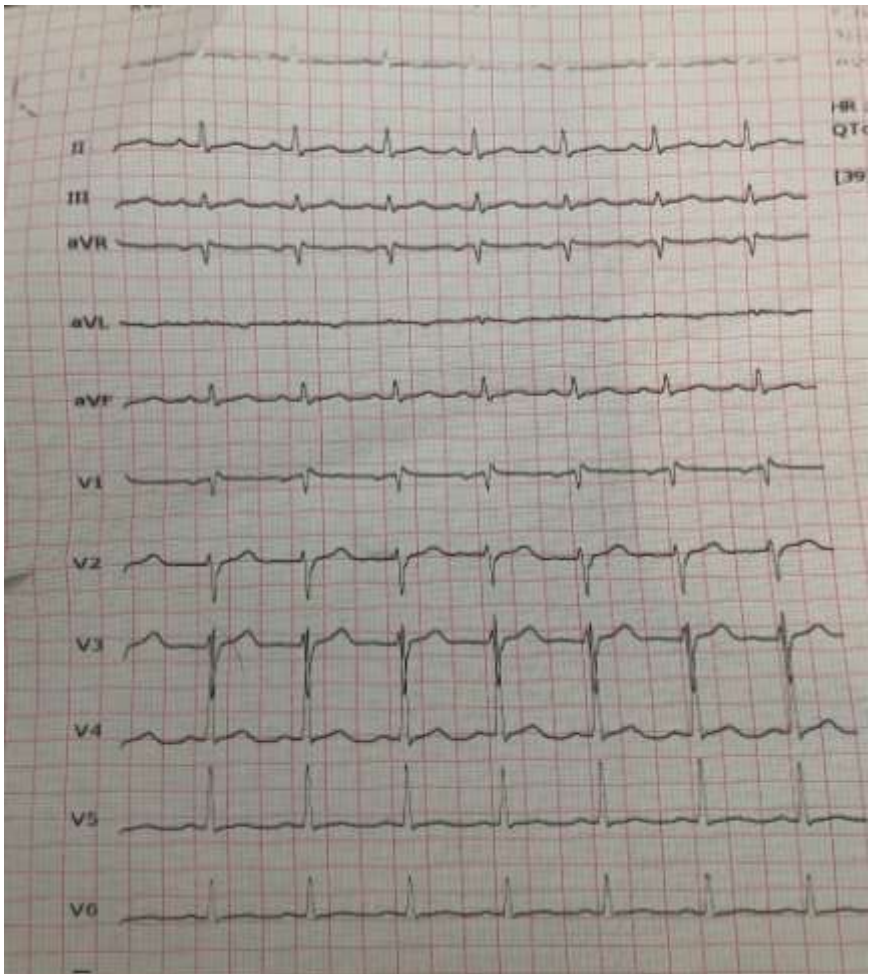


Figure 1:- Electrocardiogram without significant abnormalities.

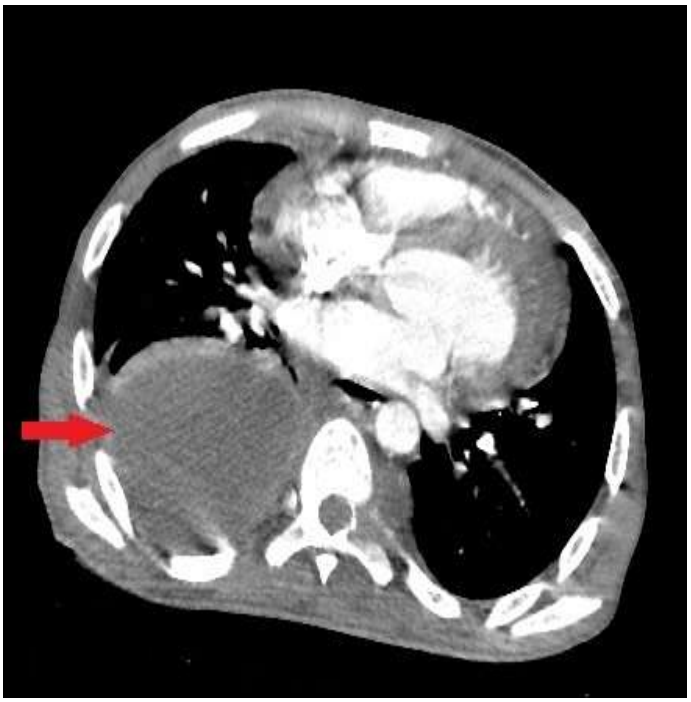


Figure 2:

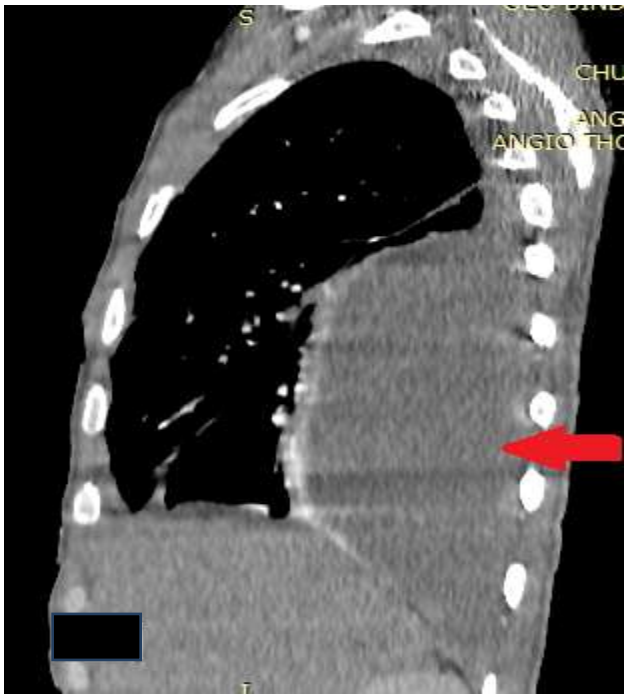
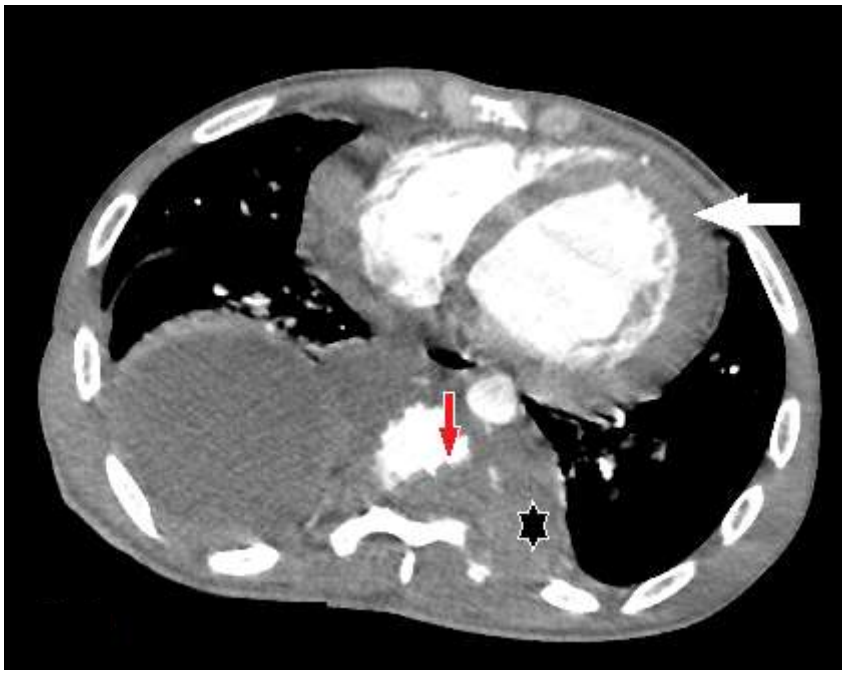


Figure 3:

Figures2,3:- Axial and sagittal contrast enhanced CT of chest showing an encysted focal pleural fluid collection in right lung field (red arrow) with no underlying parenchymal pathology



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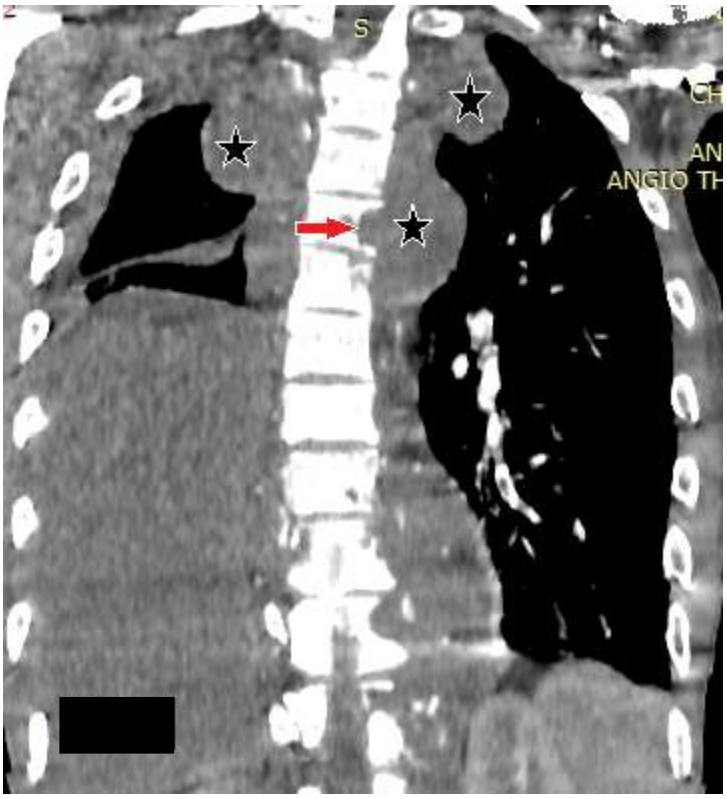

Figure 5:

Figures 4,5:- Axial and coronal view of a contrast enhanced Computed tomography (CT) of chest revealing : moderate pericardial effusion (white arrow), large paravertebral abscesses (black stars) extented to the spinal cord associated with multiple lytic lesions (red arrow) of thoracic vertebral bodies and posterior ribs arch.

A diagnostic thoracentesis showed biological characteristic of neutrophilic exudate with pleural fluid genexpert was positive.

In view of the presence of cardiomegaly, we had done a transthoracic echocardiography revealing dilated cardiomyopathy with severe left ventricular dysfunction (LVEF 20%), undilated right ventricle with borderline systolic function, no significant valvulopathy. The pericardium is thickened with minimal detachment without any consequences. On cardiac magnetic resonance imaging (MRI), we find dilated biventricular cardiomyopathy in severe left ventricular dysfunction with appearance of myopericarditis explained by inflammatory pericarditis with signs of adhesion and myocarditis in the lateral and inferior wall area(Figures 6,7). MRI also shows a costochondral bone lesions related to tuberculous spondylodiscitis, given the context (Figure 8)

The diagnosisretained: Dilated cardiomyopathy as a result of tuberculous myopericarditis in the context of disseminated tuberculosis.

A standard antituberculosis regimen (Isoniazid, Rifampicin , Ethambutol, Pyrazinamide) was started and a chest tube was inserted to allow drainage of the pleural effusion out of the chest.

Corticosteroids (1mg/kg/day), therapy of heart failure (Beta-blockers, diuretic and Angiotensin-converting-enzyme inhibitors) was added.

The evolution was favourable within 6 weeksmarked by clinical improvement weight gain (14kgs),decrease in the size of paraspinal collections and radiological improvement diminution of his pleural effusion on the chest radiograph .

Myocardial biopsy was not performed taking into account the risks of this invasive examination, the lack of clinical severity criteria requiring a diagnosis of certainties and disseminated tuberculosis impairment making myocardial impairment possible.

The favourable clinical evolution under antibacillary treatment, corticosteroids and therapy of heart failure is a strong retrospective argument to assert the tubercular origin of the heart involvement.

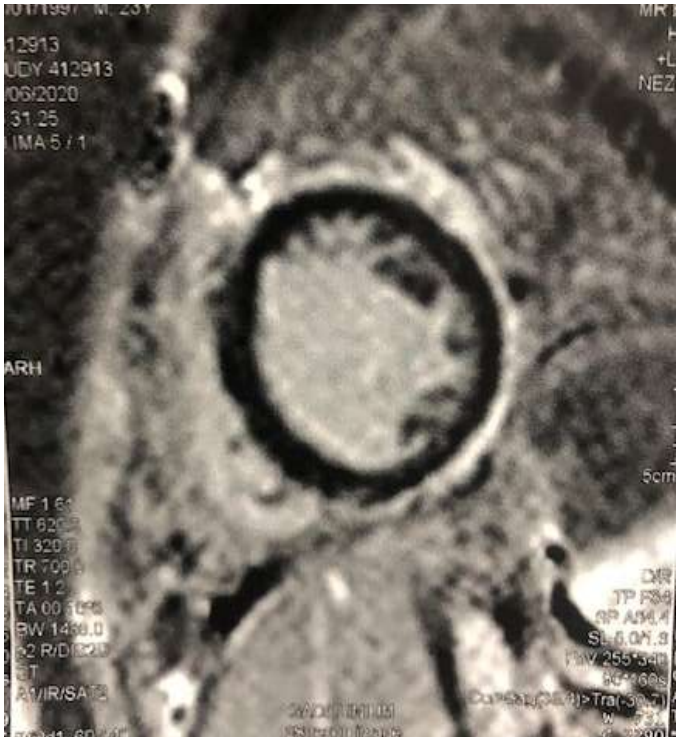

Figure 6:

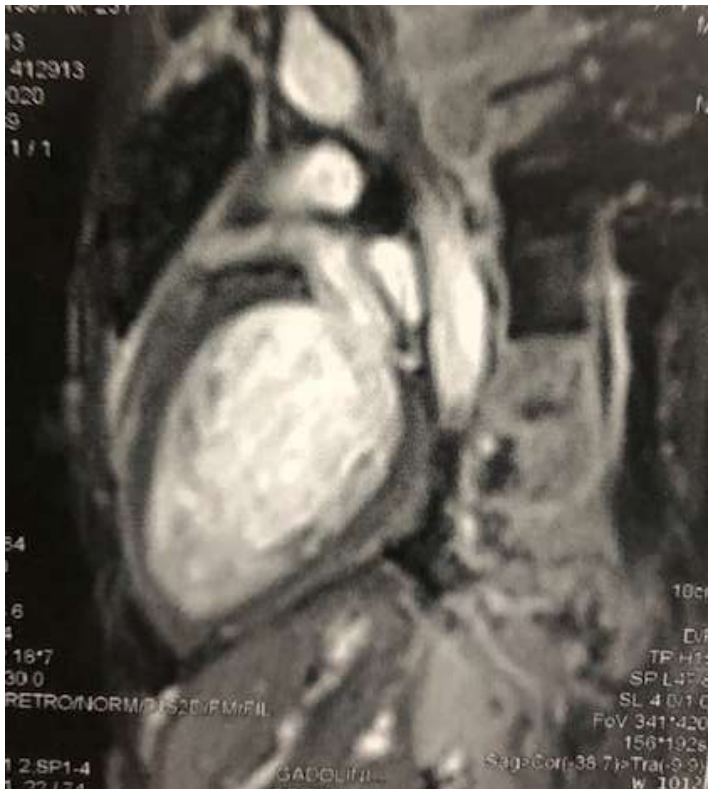

Figure 7:-

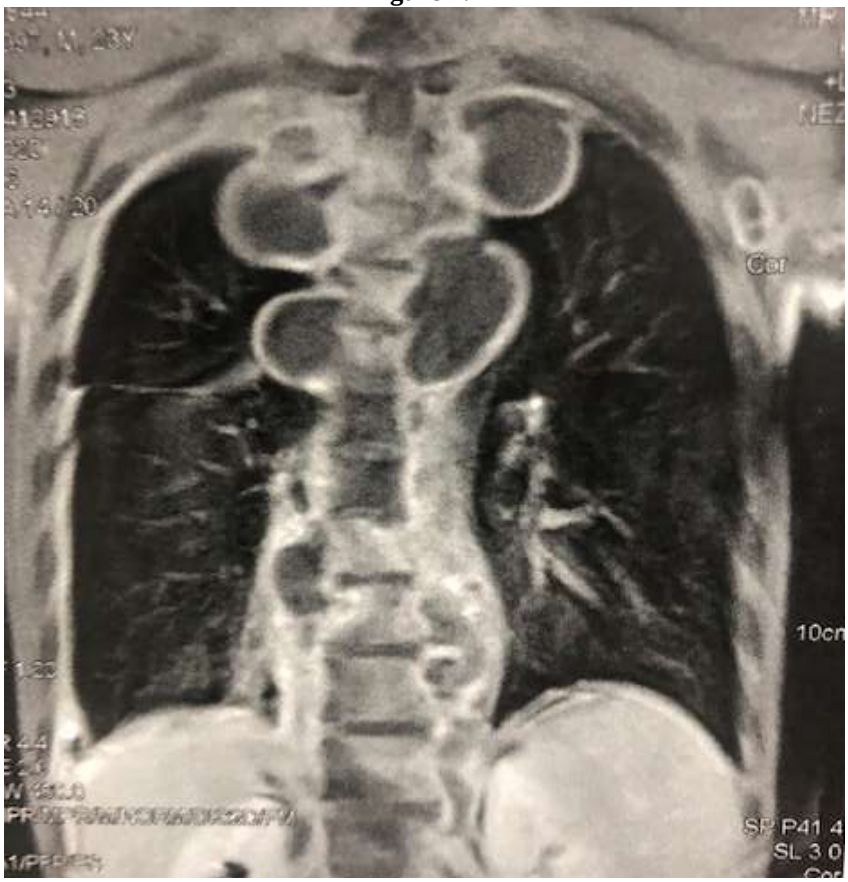

Figure 8:-

Discussion:-

Cardiac tuberculosis is a rare manifestation and it is becoming even less frequent because of better control of tuberculosis in the developed countries during the last couple of decades.

Tuberculosis may affect the heart in several ways. It often complicate typical tuberculous disease of other organ in the body (specially the lung), however it may be seen primarily in the heart.

It mostly affects the pericardium; involvement of myocardium is very rare.

Myocardial tuberculosis is rarely diagnosed during life and most of the literature is based on autopsy reports, Maurocordat initially reported it in 1664 then Morgagni in 1761 after a gap of 97 years [3].

In two autopsy series of patients who died of pulmonary tuberculosis, the frequency of myocardial involvement is estimated between 0.14% and 0.3%, it is higher in cases of disseminated tuberculosis reaching 7.8% [3].

In Morocco one case of myocardial tuberculomawas described in 2016 [4]

The reason for this lower affinity of mycobacterium tuberculosis to the myocardium compared with the pericardium is not clear. Raviart proposed that lactic acid produced by muscular activity protected cardiac muscle against the tubercle bacilli [5].

Three possible routes of spread have been suggested: haematogenous, lymphatic via infiltrated mediastinal nodes and directly from the adjacent pericardium or a pulmonary cavity lesion. The right heart, particularly the right atrium are reportedly more vulnerable, probably because of the frequent involvement of the right mediastinal lymph nodes, with consequent involvement of the myocardium [6]. Although right and left ventricle have been found to be involved in different series.

Horn and Saphir have described three histological types of myocardial tuberculosis [7]: - Nodular tubercles of the myocardium (Tuberculomas).

1. Miliary tubercles of the myocardium complicating generalised miliary disease.
2. Diffuse infiltrative form (uncommon) associated tuberculous pericarditis.

Tuberculous myocarditis can remain clinically asymptomatic and be diagnosed at autopsy or present with : ventricular arrhythmias, long QT syndrome, heart block , valve dysfunction, obstruction of the superior veina cava, right ventricular outflow tract or pulmonary vein, congestive heart failure and even sudden cardiac death.

The imaging modalities have already become the primary tool for non-invasive confirmation of myocardial inflammation, and they are indicated in all patients with symptoms of myocardial involvement.

Echocardiography can be useful in assessing systolic and diastolic function or the heart chamber sizes in cases of cardiomyopathies or visualizing the presence of cardiac masses. In our case the transthoracic echocardiography was a major aid in the diagnosis orientation.

Cardiac MRI is an interesting technique to identify non-invasively and specify the extension of myocardial involvement. It provides an accurate assessment of myocardial lesions, both for initial diagnosis and for progression after treatment. It can reveal lesions of late enhancement in the myocardial wall as evidence of focal inflammation and fibrosis.

Diagnostic criteria for myocardial inflammation, according to consensus-based recommendations, are at least two of the following:

1. Regional or global elevation of myocardial T2 signal intensity;
2. Increased global myocardial T1 enhancement ratio to skeletal muscle after early administration of gadolinium;
3. Presence of at least one focal lesion exhibiting delayed T1 gadolinium enhancement.

Endomyocardial biopsy offers tissue examination, however it is an invasive technique with low diagnostic yield (sensitivity <35%) but it remains indicated in case of unexplained cardiomyopathy and when etiologic confirmation may affect treatment or prognosis.

Most authors argue, that in the context of clinical or radiologic suspicion of tuberculosis and the presence of evocative image at the cardiac MRI the diagnosis can be safely made by sampling or culturing more accessible involved sites (Lung, pleura and lymph nodes…)[2]

Antituberculous drugs are the cornerstone of therapy, they should be initiated once the diagnosis is confirmed; although the ideal duration of therapy is currently unclear, most investigators have applied extended courses of 9 to 12 month. The place of corticosteroids is not clearly defined given the small number of reported cases; however, the experience acquired with cardiac sarcoidosis could be an argument in favour of corticosteroids [8].

For our patient we indicated a durationof 9 months therapy, corticosteroids were also added: 1mg/kg/day during 4 weeks then we progressively decreased.

Antiarrhythmic drugs and diuretic are used depending on the presenting clinical signs. In cases of ventricular arrhythmias, the insertion of an implantable cardioverter-defibrillatoris indicated. In cases of large tuberculomas, surgical therapy can be considered, the presence of several loss of systolic ventricular function can lead to an indication of heart transplantation.

Conclusion:-

Tuberculous myocarditis usually remain undiagnosed in antemortemcases, as most of the patients are asymptomatic. It should be searched in case of clinical or paraclinical signs in favour of myocardial involvement or in case of dissimilated tuberculosis; the combination of echocardiography, cardiac MRI, and culture or histology from appropriates sites affirm the diagnosis.

The prognosis is severe and the antituberculous drugs and corticosteroids are the cornerstone of therapy.

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