Different Histopathological Appearances of Cardiac Tuberculosis: Presentation of a Rare Case with Review of Literature

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Abstract

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Tuberculosis is caused by a mycobacterium and is a multipotential, omnipresent infectious disease. Generally, two major categories, pulmonary and extrapulmonary exist. Whereas pericardial involvement is relatively common, myocardial tuberculosis has been reported in not more than 0.3% of all tuberculosis patients post-mortem.
A 21-year-old woman sought medical attention due to progressive dyspnea upon exertion, weight loss, and general weakness. She underwent a heart operation and a well-circumscribed, solid, and creamy egg-shaped mass with a lobulated surface was seen in the right atrium. The histopathological study showed multiple well-formed granulomas with multinucleated giant cells and extensive caseous necrosis. These were compatible with a diagnosis of tuberculoma; however, the Ziehl-Neelsen stain failed to reveal the acid-fast bacilli. Her postoperative course was uneventful and soon she was discharged with advice to continue her medical therapy***(Iranian Heart Journal 2011; 12 (2)55-58).***

Keywords: Mycobacterium tuberculosis■ Tuberculoma■ Tuberculous pericarditis

Tuberculosis is an infectious disease with the potential to involve almost any organ in the body. The pericardium is the usual site for cardiac tuberculous infection. The result is a granulomatous reaction that can be hemorrhagic. Involvement of the myocardium mainly stems from direct pericardial extension. The range of symptoms varies from heart failure and complete heart block to sudden death. Definitive diagnosis is established by isolating the organism, but adenosine deaminase activity and polymerase chain reaction also aid in the diagnosis. In the absence of these techniques, sarcoidosis is a close differential diagnosis and needs exclusion.

Caused by a mycobacterium, tuberculosis is an infectious disease with the potential to involve almost any organ in the body.

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symptoms are seen due to the type of involvement, which may vary from one layer to another. Here, we discuss not only the different layers of the heart that may be involved but also the varied histopathological pictures which are produced by this disease.

First of all, tuberculous pericarditis is dealt with, where the membrane that surrounds the heart is affected, causing the space between the pericardium and the heart to be filled with fluid. Restriction of the heart occurs when little blood fills its chambers, and inefficient beating is naturally the leading consequence.[1] The pericardium is the usual site for cardiac tuberculous infection.

The result is a granulomatous reaction that can be hemorrhagic. If tuberculous pericarditis is extensive and chronic, there can be massive fibrosis with calcification and thickening of the pericardium, leading to constrictive pericarditis.[2] Lymphatic, hematogenous, or contiguous dissemination from lungs or spine give rise to pericarditis.[3] Microscopically, this inflammation is granulomatous, with epithelioid macrophages and Langhans giant cells as well as lymphocytes, plasma cells, maybe a few PMN's, fibroblasts with collagen, and characteristic caseous necrosis in the center of the lesion. An acid fast stain (Ziehl-Neelsen) will show the organisms as slender red rods. Although the most common specimen screened is sputum, histologic staining can also be done on tissues or other body fluids.[2]

Three morphologic stages are noted here: acute, subacute, and chronic. In the acute stage, the effusive picture dominates, which is later superseded by granuloma formation, and finally constrictive pericarditis and calcification settle.[3] Involvement of the myocardium mainly results from direct pericardial extension. Localized necrotizing granulomas, also

complete heart block to sudden death. Definitive diagnosis is established by isolating the organism; however, adenosine deaminase activity and polymerase chain reaction also aid in the diagnosis.[3] In the absence of these techniques, sarcoidosis is a close differential diagnosis and needs exclusion.[4]

We now take into consideration the observation of a 21-year-old woman who was referred to our center with a diagnosis of an intracardiac mass. Progressive dyspnea upon exertion, some infrequent palpitations, a weight loss of about 7 kg, and general weakness over the past two months constituted her clinical findings and complaints. Her medical history was soon found to be positive for pulmonary tuberculosis.

She had formerly received medications over a period of 6 months. Upon examination, her vital signs were stable, the lung fields were clear, and the heart sounds were normal. No murmurs or pericardial friction rub could be heard.

Hepatomegaly without ascites or peripheral edema featured the other findings. The laboratory tests results were indicative of a hypochromic microcytic anemia (Hb=9.3 g/dl).

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The erythrocyte sedimentation rate was moderately increased, and the c-reactive protein level had also risen. The routine blood culture results were, as was expected, negative.

The patient underwent a heart operation, which revealed pericardial thickening with signs of dense pericardial adhesion. A well-defined, solid, and creamy ovoid mass with a lobulated surface was seen in the right atrium, measuring 4×3.5×2 cm.

Because complete excision was impossible, the mass was to be debulked as much as possible to preserve the tricuspid valve leaflets.

The histopathological slides of the endocardial mass divulged multiple well-formed granulomas with multinucleated giant cells and extensive caseous necrosis.

These were compatible with a diagnosis of tuberculoma; nevertheless, the Ziehl-Neelsen stain failed to reveal the acid-fast bacilli.

The patient had an uneventful course and was discharged with advice for a referral to a TB specialist.

Later, the transthoracic echocardiographic examination showed tricuspid valve leaflet thickening and moderate regurgitation but no distinct intracavitary mass or residual atrial septal defect.

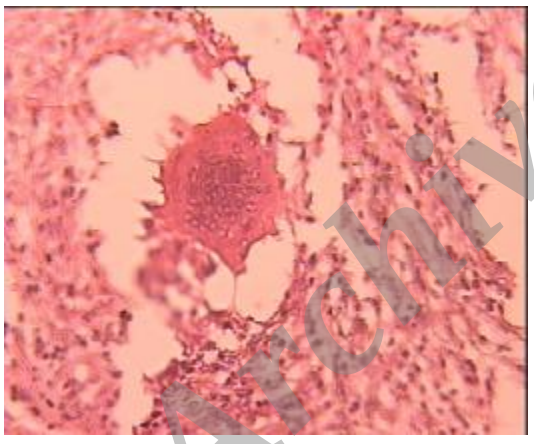

Fig. 1. Microscopically, there are scattered Langhans giant cells, lymphocytes, plasma cells, and the characteristic caseous necrosis (H&E, X 400).

Discussion

Tuberculomas among various pictures of tuberculosis in the heart are very rare. A thorough literature review shows that these have been reported in not more than 0.3% of all tuberculous patients post-mortem.[5-7] Aside from the pericardium, it is extremely uncommon to involve the heart. The first report came by Morgagni in 1761.[5] As was stated before, the myocardium becomes involved as a consequence of the direct extension from the pericardium or due to spread from the mediastinal lymph nodes or possibly secondary to hematogenous dissemination.[8] Three distinct morphologies depict the myocardial involvement: 1) diffuse infiltration, the most common form, characterized microscopically by giant cells and lymphocytes; 2) the miliary variant, resulting from hematogenous spread; and 3) nodular type, showing central caseation necrosis.[9-10] The Ziehl-Neelsen staining of specimens often fails to reveal the acid-fast bacilli, and the definitive diagnosis obviously relies on the distinction of the above typical histological changes.[11] No specific features distinguish tuberculoma on echocardiography or CT-scan from other cardiac neoplastic or non-neoplastic masses.

We emphasize again that isolated cardiac tuberculomas are exceedingly rare.[12] Cardiac tuberculomas, either single or multiple in number, most often are observed in the right-heart chambers, particularly in the right atrial wall.[5-6] These mass lesions, are usually well-circumscribed and are liable to erode the underlying myocardium, thus creating lesions that in turn can lead to thrombus formation and subsequent embolism. This phenomenon may help hematogenous seeding and dissemination.[13]

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