CLINICAL IMAGES

Intramyocardial tuberculosis – a rare underdiagnosed entity

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In contrast to tuberculous pericarditis, the myocardium is resistant to inflammation but can rarely present with tuberculomas. Ziehl-Neelsen staining often fails to demonstrate acid-fast bacilli, and diagnosis rests on the detection of typical histological changes.

Case 1

A 38-year-old man, a fit kick-boxer, known anabolic steroid user and marijuana smoker, collapsed with shortness of breath in the casualty department and died. On autopsy, he was found to be muscular and heavily built, and had cardiomegaly, enlarged paratracheal lymph nodes and pulmonary oedema. His heart had a large fibrous nodular mass in the mitral valve cusp; there were numerous small, infiltrative, white, cheese-like lesions proximal to the aortic valve cusp and extending to the myocardium, and extensive fibrous thickening of the tricuspid valve cusp (Figs 1 and 2).

Histological analysis revealed necrotising caseating granulomatous inflammation, and the Ziehl-Neelsen stain was positive for acid-fast bacilli. The subject's HIV status was not ascertained.

Case 2

A 21-year-old man had been nauseous and vomiting for one day, and had bought Gaviscon; he collapsed and died while vomiting into a toilet. He was thin but not emaciated. At autopsy, cardiomegaly was evident, with a firm, hollow, yellowish mass with a thrombus within the cavity, just below the aortic outflow tract in the left ventricle. The aortic valve cusps had been replaced by the mass. The lungs were normal and the lymph nodes enlarged. The subject's HIV status was not ascertained. No acid-fast bacilli were noted on Ziehl-Neelsen staining.

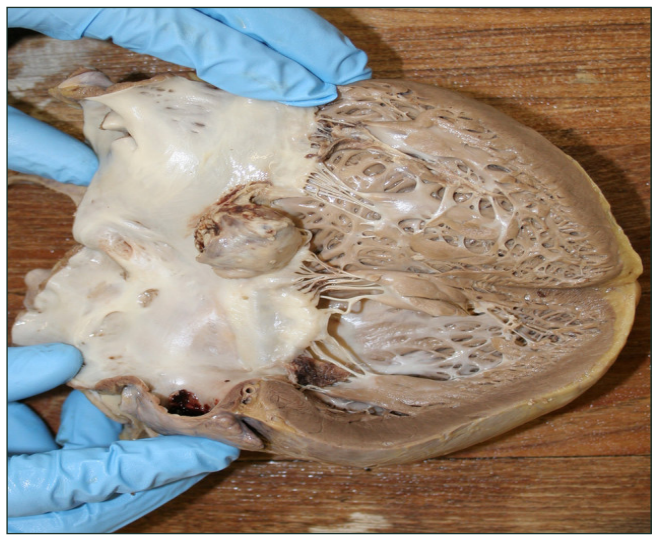

Fig. 1. Large fibrous nodular mass in the mitral valve cusp.

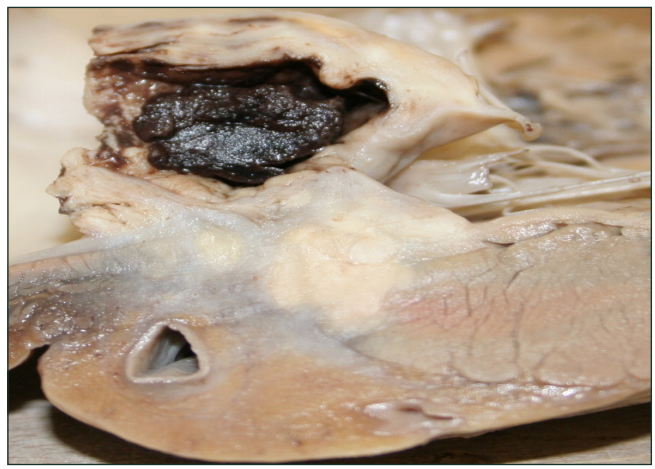

Fig. 2. Numerous small, infiltrative, white, cheese-like lesions proximal to the aortic valve cusp and extending to the myocardium.

Discussion

Tuberculosis is rampant in South Africa and, with the exacerbating effects of AIDS, the incidence is likely to rise. Tuberculosis is generally thought to spare the heart, thyroid, pancreas and skeletal muscle; cardiovascular manifestations are largely restricted to chronic pericardial inflammation.

Cardiac tuberculoma was first reported by Maurocordat in 1664 and Morgagni in 1761; Virchow denied the existence of myocardial tuberculosis in 1865 but, in 1902, 72 cases were

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reported.[1] In a series of 19 patients at Groote Schuur Hospital over a 27-year period,[2] 1 patient was diagnosed *ante mortem*, 11 were found on autopsy to have nodular lesions, and 7 had miliary lesions; acid-fast bacilli were demonstrated in only 1!

Myocardial tuberculosis spreads from mediastinal lymph nodes, tuberculous pericarditis or retrograde lymphatic and haematogenous spread. The favoured sites are the right atrium and left ventricle. Pathological varieties are nodular/miliary tubercles or diffuse infiltrative tuberculosis of the myocardium.

Ante mortem diagnosis is rare; patients may present with arrhythmias, superior vena cava obstruction, right ventricle outflow tract obstruction or left ventricular obstruction. The mechanisms of death include arrhythmias, impaired myocardial contractility, cardiac rupture, coronary occlusion, and obstruction to pulmonary blood flow leading to fatal haemorrhage.

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2. Rose AG. Cardiac tuberculosis: a study of 19 patients. *Arch Pathol Lab Med* 1987; 111: 422-426.