

Short reports

Sudden death due to myocardial tuberculosis

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Tuberculous myocarditis is rare and usually complicates typical tuberculous disease elsewhere. Although tuberculosis is usually associated with an insidious onset, symptoms being present for several weeks or even months before medical help is sought, in its miliary form it may cause prostration and sudden death, as this report illustrates.

Case report

A 31 year old Asian man resident in the United Kingdom for 12 years collapsed without warning at work. On arrival in hospital he was dead. Full resuscitative measures were unsuccessful. His body appeared well nourished and there were no signs of trauma or drug abuse. Fundoscopy showed a choroidal tubercle. Subsequent inquiry revealed no previous symptoms or ill health before his death. His sister, however, had recently had pulmonary tuberculosis.

Postmortem examination showed miliary tubercles in the liver, kidneys, and lungs with contraction and fibrosis at the apex of the right lung. Acid fast bacilli were identified in the caseating material of one enlarged mediastinal lymph node.

In addition, there was a severe tuberculous myocarditis. The heart was enlarged (weight 333 g) with patches of white discolouration and infiltration beneath the epicardium affecting both ventricles and the interventricular septum. The cardiac chambers, valves, coronary arteries, and pericardium were normal. Microscopic examination revealed a severe giant cell myocarditis with multiple granulomas throughout the myocardium. Acid fast bacilli and caseation were absent. Serial sections through the interventricular septum revealed an extensive granulomatous infiltration surrounding but not destroying the conducting tissue (fig).

Discussion

Tuberculous myocarditis is very rare and was at one time found most frequently in children and young adults, invariably coexisting with tuberculous disease elsewhere.¹ The myocardium is affected usually by direct extension, or less often by retrograde lymphatic drainage, from tuberculous mediastinal nodes. Infection via the haematogenous route

may develop in miliary disease and direct spread from tuberculous pericarditis can also occur.

Horn and Saphir¹ described three histological types of myocardial tuberculosis: (1) nodular tubercles (tuberculomas) of the myocardium, varying "from pea to egg size" with central caseation usually affecting the right side of the heart, particularly the right atrium; (2) miliary tubercles of the myocardium complicating generalised miliary disease; and (3) the uncommon diffuse infiltrative type, usually associated with tuberculous pericarditis, in which the myocardium is diffusely infiltrated by granulation tissue containing giant cells, endothelial cells, and lymphocytes.

In this case the diagnosis of tuberculosis was not immediately apparent at necropsy. The appearance of the heart was identical to that illustrated by Payling Wright² and suggested an apparently isolated giant cell myocarditis, a condition of unknown aetiology. Furthermore, the histological picture was also similar to that seen in sarcoid heart disease. Lesions of the heart and its conducting tissue caused by either condition may, of course, result in a variety of cardiac arrhythmias and, in particular, in sudden death.³ The man's origin and the presence of a choroidal tubercle, however, suggested a tuberculous myocarditis and this was confirmed by the discovery of acid fast bacilli in a tuberculous mediastinal node and of miliary tubercles elsewhere. Our case thus emphasises the importance of a careful search for acid fast bacilli and for similar lesions in other organs if granulomatous lesions are found in the heart.

Myocardial tuberculosis is rarely diagnosed during life. Nevertheless, it should be suspected in a patient with tuberculosis if a cardiac arrhythmia such as atrial fibrillation, paroxysmal ventricular tachycardia, ventricular fibrillation,⁴ or atrioventricular block⁵ occurs; if congestive heart failure supervenes⁶; or if valve dysfunction⁷ or obstruction of the superior vena cava,⁸ right ventricular outflow tract,⁹ or pulmonary veins¹⁰ develops. Such obstructive lesions may be due to large nodular tubercles of the right atrium, right ventricle, and left atrium respectively.

The striking feature about this case is the contrast between the apparent lack of symptoms before the young man's death and the extent and severity of the tuberculous myocarditis found at necropsy. The immediate cause of his sudden death was probably a fatal ventricular arrhythmia or conduction defect due to the extensive disease surrounding the conducting tissue of the heart.

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Section from interventricular septum showing the tuberculous infiltration (right bundle branch top left). (Haematoxylin and eosin, $\times 18$.)

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