CASE REPORT

TUBERCULOMA OF THE INTERVENTRICULAR SEPTUM

BY

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Tuberculous involvement of the myocardium is unusual even in miliary tuberculosis and is almost unknown in the absence of active tuberculosis elsewhere in the body. Three forms are generally recognized. Of these, the nodular is the most striking and has been most frequently reported. In a few instances the nodules have been directly responsible for death. In the first two recorded cases (Townsend, 1832; Maurocordatus, 1664) myocardial tubercles obstructed the pulmonary veins, causing death from pulmonary hæmorrhage; ventricular rupture through a tuberculoma was described by Oudendal (1923) and Korybut-Daszkiewicz (1922); coronary artery occlusion by Campos and Maranzana (1950); and impaired atrial contraction by Adamson (1920). However, in most reports, myocardial nodules have merely formed part of a fatal tuberculous process, and there is only one instance in which no other focus of active tuberculosis was present (Barnes, Barnes, and Wilson, 1956).

Case Report

A man of 87 was admitted with one week's urinary retention. He had had winter bronchitis for the past seven years, had first become dyspnoëic on exertion about four years previously, and been dyspnoëic even at rest for the past six months. He gave a history of rheumatic fever fifty years ago, and no other previous illnesses.

Examination showed an extremely obese, emphysematous, elderly man with acute bronchitis, a distended bladder, and a moderately enlarged prostate. His blood pressure was 130/80 mm. Hg, and his heart was fibrillating at approximately 100 a minute. A chest radiograph showed bilateral basal congestion and left-sided cardiac enlargement. The cardiogram showed atrial fibrillation and possible left ventricular hypertrophy only.

Since operation was contraindicated, the urinary retention was treated conservatively, and the acute exacerbation of his chronic bronchitis was treated with tetracycline and aminophylline; but his condition deteriorated rapidly and he died four days after admission. The necropsy showed acute and chronic bronchitis and emphysema, with much generalized congestion of both lungs, moderately thickened bronchi, and œdematous and intensely congested bronchial mucosæ with large amounts of muco-pus. Bilateral chronic pleural adhesions were present but a careful examination failed to demonstrate any calcified or caseous foci or other evidence of pulmonary tuberculosis. There was a moderate degree of benign prostatic hyperplasia, and the kidneys showed early ascending pyelonephritis microscopically. Several 0.1–1.0 cm. irregular white subcapsular nodules were present in the liver. There was no significant abnormality in any other organ except the heart.

The heart weighed 460 g. There was a chronic adherent pericarditis and a moderate degree of left ventricular hypertrophy. An area of endocardium, high on the postero-superior aspect of the interventricular septum, appeared opaque, white, and thickened; and bulged slightly into the cavity of the left ventricle. Underlying this area was a thick-walled, irregularly calcified cavity, 2 cm. in diameter, containing caseous material and two irregular "calculi" (Fig. 1). This was occupying the area immediately below the membranous part of the interventricular septum, in which the left branch of the atrio-ventricular bundle is normally found. Both atria, the right ventricle, the coronary arteries, and the heart valves were normal.

Microscopy confirmed the tuberculous nature of the septal lesion, showing a fibrous and calcifying wall

FIG. 1.—Opened left ventricle showing the bisected tuberculoma in the interventricular septum and two calculi removed from the caseous contents.

surrounding caseous material. Active tuberculous changes were also present with foci of epithelioid cells, Langhans type giant cells, lymphocytic infiltration, and fibroblastic proliferation (Fig. 2). A few acid alcohol fast bacilli were seen in the Ziehl Neelsen stained sections. The hepatic nodules were all hæmangiomas at various stages of sclerosis, and there was no evidence of tuberculosis. Histological examination of the other organs showed nothing significant.

Discussion

Solitary tuberculomas are generally believed to be the result of retrograde spread from mediastinal lymph nodes to the cardiac lymphatic plexuses draining into them. Macroscopic involvement of the pulmonary hilar lymph nodes by tuberculosis has almost invariably been present in the cases previously described: Anders (1902), reviewing 72 cases of myocardial tuberculosis, found only 2 in which there was no local lymph node involvement. In the present case there was no local lymph node involvement, but clear evidence of old pericarditis in the form of numerous chronic pericardial adhesions. Since direct extension from the pericardium is anatomically improbable in this case, infection must have occurred either by retrograde spread from pericardial to endocardial lymphatic plexuses, or have arisen, together with the pericarditis, by hæmatogenous dissemination or lymphatic spread from pulmonary lymph nodes.

The absence of lymph node involvement is not surprising in this case, since the cardiac infection probably occurred fifty years before death, and the changes after several years of chronic and recurrent acute bronchitis would be sufficient to obliterate any small non-calcified lesions. The heavy

FIG. 2.—Photomicrograph showing Langhan's giant cell, epithelioid cells, lymphocytes, and caseous material at the periphery of the tuberculoma. (H. and E. $\times 240$.)

calcification and calculus formation indicate that the tuberculoma was of many years' standing, and the presence of tubercle bacilli is not inconsistent with a lesion of this duration. In retrospect, it appears likely that the previous diagnosis of rheumatic fever was actually based on tuberculous pericarditis and myocarditis. There was no macroscopic or microscopic evidence of rheumatic carditis at necropsy. From the relation of the tuberculoma to the conduction system, signs of myocarditis would be expected, particularly in the early stages of its development and, in view of its final size, it is perhaps rather surprising that there was no evidence of bundle-branch block in the cardiogram: however, this is in accordance with the concept that only a few intact fibres may be sufficient to maintain normal conduction (Lumb and Shacklett, 1960).

Summary

A calcifying myocardial tuberculoma, containing calculi, was an incidental autopsy finding in a man aged 87, whose history suggested that the infection had occurred 50 years previously. Although the lesion occupied the sub-membranous area of the interventricular septum, no bundle-branch block had been present in life.

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