Myocardial tuberculoma with rupture and pseudoaneurysm formation—successful surgical treatment

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SUMMARY A case of left ventricular pseudoaneurysm due to rupture of a myocardial tuberculoma is presented. The diagnosis of pseudoaneurysm was initially suggested by echocardiography and was confirmed by angiocardiography. The aetiology was suggested at operation and confirmed by histological examination. This is a very rare condition which is usually diagnosed only at necropsy.

Case report

A 38 year old African hospital employee had a severe, central, crushing chest pain which lasted for four hours. For the next five days he had intermittent pleuropericardial type pain in the left side of the chest. He then sought medical advice. He gave no history of chest pain before this. Five years ago he had had fever associated with patchy bronchopneumonic infiltration of the right lung base; sputum analysis and culture were negative for pathogens. Two months after that he had transient atrial fibrillation which spontaneously reverted to sinus rhythm. A tuberculin skin test had been positive in 1976. The patient had never had treatment for tuberculosis.

Physical examination at the time of admission showed a healthy looking adult in no acute distress. Pulse rate was 80 beats per minute and regular, temperature was 37°C, and blood pressure was 140/100 mm Hg. Jugular venous pressure was not raised; there was no cardiac enlargement; first and second heart sounds were normal. There was no pericardial rub. The chest was clear; the liver and spleen were not palpable and there was no oedema in the legs.

The electrocardiogram showed changes of an inferior myocardial infarction. An electrocardiogram recorded three years earlier had been normal. The chest x ray film showed a heart of normal size with clear lung fields. Serum concentrations of cardiac enzymes were normal and the erythrocyte sedimentation rate was 35 mm/hour (normal 0–5). The cross sectional echocardiogram in the long axis parasternal view showed a pouch posterior to the left ventricle communicating with the left ventricular cavity through a narrow necked channel (Fig. 1). M mode echocardiography showed a large echo free space posterior to the left ventricular epicardium. These findings suggested a pseudoaneurysm in the left ventricle. Coronary angiography showed normal left and right coronary systems; left ventricular angiography demonstrated a multi-loculated pseudoaneurysm (Fig. 2).

Four weeks later the patient underwent surgery. The posterior pericardium was found to be thickened and there was a haemorrhagic pericardial effusion. A hole, 1 cm in diameter, was found in the lower left ventricular wall. The hole was patched and the pericardium and pseudoaneurysm were resected and sent for histological examination. The
early and late postoperative periods were without complications.

Histopathological examination showed that the pseudaneurysm lining was a mixture of myocardium and pericardium. There was extensive fibrosis and a large number of granulomas with areas of central caseation surrounded by epithelioid cells and multinucleated giant cells. Although no acid fast bacilli were seen and culture remained sterile, the presumptive diagnosis was tuberculoma of the myocardium with rupture and pseudaneurysm formation. The patient was treated with isoniazid, rifampicin, and ethambutol. In the next two months his weight increased by 8 kg and he remains symptom free 10 months after operation.

Discussion

Tuberculosis of the myocardium was rare even in the early 1900s and it is becoming more rare with improved medical treatment. Custer and Charr reviewed more than 14,000 deaths due to tuberculosis and found only 64 cases (0·44%) of tuberculosis of the heart. Three types of myocardial involvement are described: localised mass (tuberculoma), diffuse myocarditis, and miliary lesions. The mechanisms of myocardial involvement are direct retrograde spread from mediastinal lymph nodes, extension from a pericardial focus, and blood dissemination. There are few reported cases of myocardial tuberculoma, and all were diagnosed at necropsy. Reported presentations include complete heart block, right ventricular outflow obstruction, aortic regurgitation, superior vena cava obstruction, or left ventricular aneurysm. Our patient appears to be the first case of myocardial tuberculoma with rupture and pseudaneurysm formation which has been treated successfully.

The diagnosis of pseudaneurysm was suspected from the echocardiogram which had features similar to those described by Katz et al.,13 Roelandt et al.,14 and Sears et al.,15 except that the pseudaneurysm wall was more thickened in our patient, probably due to the inflammatory aetiology. The diagnosis was confirmed by angiography; the aetiology was demonstrated histologically. Although no acid fast bacilli were seen or grown, tuberculosis is the most likely diagnosis. Sarcoidosis can produce a similar picture with localised aneurysm16 but this entity is very rare in Saudi Arabia whereas tuberculosis is common. A tuberculous aetiology is further supported by the histological demonstration of central caseation, and the patient's clinical improvement after surgery and antituberculous therapy.

References

5 Claiborne TS. Caseating granulomas of the heart. Am J Cardiol 1974; 33: 920–3.

Fig. 2 Left ventricular angiogram showing contrast material filling a pseudaneurysm on the inferior surface of the left ventricle (right anterior oblique projection). LV, left ventricle; PA, pseudaneurysm.