CASE REPORTS

CALCIFIED CARDIAC ANEURYSM

BY

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An account of calcified cardiac aneurysms and a summary of previously reported cases has been given by Epstein (1953), who found only 37 instances of myocardial calcification reported during the present century, and in 26 of these the calcification occurred in a ventricular aneurysm. In 15 of the 26 cases a history of coronary thrombosis was obtained, the survival time after the acute attack ranging from five to eighteen years, while in the case presented by Epstein the patient was still living twenty years after the acute episode. It was also pointed out that the presence of calcification in cardiac aneurysms and infarcts was associated with relative longevity. Two further examples have been reported by Broustet *et al.* (1954) and by Aravanis and Luisada (1955).

The case now presented is remarkable in that the patient survived for 28 years after the original attack of cardiac infarction, and the degree of calcification found in the aneurysm and in the coronary arteries was very advanced. In view of this exceptionally long history, the apparent rarity of the condition, and the clearly defined pathological features, it was thought desirable to put on record a brief account of this patient.

Case History

The patient, a man, aged 80, and a member of the medical profession, had suffered from an attack of coronary thrombosis in 1927 at the age of 52 and had been treated by prolonged rest in bed. He had continued subsequently to have moderate breathlessness and angina of effort, and there was an exacerbation of these symptoms in 1940 when he was aged 65 but clinical examination at that time did not suggest a further infarct, while the electrocardiogram showed evidence of only an old defect. The angina of effort persisted, but no other serious episode occurred until 1955 when he had severe dyspnoea and blood-stained sputum together with acute retention of urine. He was admitted to a local hospital where, he was found to be cyanosed and gasping for breath, with a pulse rate of 120 a minute, a blood pressure of 115/60 mm. Hg, and a temperature of 99° F. There were clinical signs of cardiac enlargement with an apical systolic murmur, and coarse râles were heard all over the chest. The urinary retention, which was due to an enlarged prostate, was relieved by an in-dwelling catheter.

The patient was transferred to this hospital and was still cyanosed and breathless. Clinical examination revealed no enlargement of the heart, but there was a systolic murmur at the apex conducted to the axilla; there was pre-systolic gallop rhythm, the pulse was regular with a rate of 100 a minute, and the blood pressure was 130/90 mm. Hg. Crepitations and coarse râles were present all over the lungs, and slight oedema of the legs was noted. The blood urea was 76 mg. per 100 ml., the hæmoglobin 78 per cent (Haldane), and the white blood count 18,000 per cu. mm. with 94 per cent neutrophil polymorphs. Radiological examination of the chest was carried out in the ward, owing to the patient's poor condition, but was unsatisfactory as much of the heart shadow was obscured by the diaphragm, and only in retrospect was the outline of the cardiac aneurysm distinguished. The cardiogram showed sinus tachycardia, and an old apical myocardial infarct was indicated by loss of R in V5 with T inversion in V7. More recent posterior infarction was also indicated by pathological QS in III and Q in VF with S-T elevation.

The clinical diagnosis was congestive heart failure due to myocardial infarction. Rapid deterioration followed and death occurred ten days later.

Pathological Findings. The pericardium was normal except for firm fibrous adhesions over the cardiac apex. The heart weighed 520 g., and all the cavities were somewhat dilated and contained dark red

post-mortem blood clot. The valves and orifices were normal except for slight atheromatous thickening of the aortic valve. At the apex of the left ventricle the myocardium was reduced in thickness to 0.2 cm. over an area 4.5 cm. in diameter, with the formation of a slight, aneurysmal bulge. The wall of the aneurysm was hard and rigid due to extensive calcification (Fig. 1) and the sac was completely filled by adherent thrombus. There was also evidence of a more recent infarction of the left ventricle in a high posterior position, the myocardium being softened and discoloured over an ill-defined zone of approximately 2.5 cm. in diameter and covered internally by mural thrombus. The coronary arteries showed very advanced atheroma with extensive calcification (Fig. 1) and narrowing of their lumina, and the right coronary was occluded 10 cm. from its origin by a small red thrombus.

Histological examination shows the wall of the aneurysm (Fig. 2) to be composed of dense hyaline fibrous tissue, with areas of calcification and very few remaining muscle fibres. The adherent thrombus shows hyaline change in its deeper layers. The posterior left ventricular wall shows evidence of both old and recent infarction, with areas of fibrosis as well as hæmorrhagic zones where there are necrotic muscle fibres and infiltrations of inflammatory cells.

Apart from the heart, other changes found post mortem included œdema of the lower limbs, bilateral pulmonary congestion, œdema, and organizing pneumonia; a mild grade of chronic venous congestion of the liver; fairly severe generalized atherosclerosis; and senile enlargement of the prostate.

FIG. 1.—X-ray photograph of the heart at necropsy, showing calcification of the aneurysm and of the coronary arteries.

FIG. 2.—Photomicrograph of the wall of the aneurysm, showing fibrous tissue with calcium deposits and remnants of muscle fibres. The mural thrombus (stained darkly, above) and the pericardium with a coronary artery are also shown. Hæmatoxylin and eosin. $\times 15$. The section was not decalcified.

Summary

A brief presentation has been made of a case of calcified left ventricular aneurysm in a man, aged 80, with an exceptionally long history dating from cardiac infarction occurring 28 years previously. A search for published records of similar cases showed that myocardial calcification, with or without aneurysm formation, is uncommon.

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