Saccular aneurysms of left ventricle

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In this report two patients with most unusual aneurysms of the left ventricle are described, and the subject of rare ventricular aneurysms (including both true aneurysm and pseudo-aneurysm formation) is briefly discussed. In both patients the diagnosis of left ventricular aneurysm was unsuspected on clinical or on radiological grounds until the lesion was shown by selective left ventricular angiography.

The majority of left ventricular aneurysms develop in middle age or in the elderly as a sequel to coronary arterial disease, and the well-known clinical, radiological, and electrocardiographic features of these post-myocardial infarction aneurysms are characteristic. The diagnosis is usually first established when a localized bulge in the cardiac silhouette is seen on the routine chest x-ray, and on screening paradoxical pulsation is evident in this localized area. The configuration of this type of aneurysm is fairly stereotyped in that the mouth of the aneurysm is at least as large as the greatest diameter, and the greatest diameter is rarely more than 6 cm. (Dubnow, Burchell, and Titus, 1965).

A few instances of left ventricular aneurysm not caused by coronary artery disease have been described, and the aetiology of these has included congenital defects of the myocardium (Lovitt and Lutz, 1954), trauma (Hall, 1903), and rheumatic myocardial necrosis (Burn, Hollander, and Crawford, 1943; Parkinson, Bedford, and Thomson, 1938). Aneurysm of the left ventricle in youth is rare. In 102 cases of cardiac aneurysm reported by Schlichter, Hellerstein, and Katz (1954), the youngest patient was 36 years old, and Parkinson's youngest patient was aged 30. In this communication, 2 examples of most unusual aneurysms of the left ventricle are described.

Case Reports

Case 1 A 21-year-old woman was admitted to another hospital at the age of 14 with episodes of palpitations and congestive cardiac failure. Rheumatic fever was suspected as the erythrocyte sedimentation rate was high, the heart was enlarged, and there was peripheral oedema. The joints were

not affected and there were no other manifestations of rheumatic fever. During treatment in hospital the patient developed atrial fibrillation and this has persisted since that time, but with bed-rest, digitalis, and diuretics she made a good recovery from heart failure and led a normal life from the age of 15 up to her present admission. She was admitted to the Cardiac Unit at the age of 21 for further studies.

On examination the patient was a normally developed woman with a normal venous pressure and normal arterial pulses. The rhythm was atrial fibrillation and on palpation of the praecordium a diffuse heave, synchronous with the heart beat, was felt over the lower sternal region extending outwards to the left of the sternum. The impulse

FIG. 1 Phonocardiogram (Case 1) shows a systolic murmur at the cardiac apex, loudest in early and mid-systole, a normally moving second sound, and an early diastolic click, 80 milliseconds after A₂.

Received 2 June 1969.

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FIG. 2 *Electrocardiogram (Case 1) showing widespread Q waves.*

was not expansile in nature. On auscultation at the cardiac apex there was a soft systolic murmur occupying early and mid systole at the cardiac apex. The second heart sound split normally during inspiration and there was in addition a click in early diastole, shown on the phonocardiogram to occur 80 milliseconds after aortic valve closure (Fig. 1).

FIG. 3 *Case 1. Considerable enlargement of the cardiac outline.*

The electrocardiogram (Fig. 2) shows Q waves in aV1, V4, V5, and V6, and the chest x-ray (Fig. 3) shows gross enlargement of the cardiac silhouette with a normal vascular pedicle and normal lung fields. At cardiac catheterization the pressures in the right ventricle and pulmonary artery were normal, the pressures in the left ventricle and aorta were 100/12 mm. Hg and 100/60 mm. Hg, respectively, and the cardiac output 4 l./min.

A selective right ventricular angiocardiogram showed that the right ventricular outflow tract was displaced towards the mid line (Fig. 4), and a further injection of contrast into the left ventricle revealed a very large thin-walled sac in communication with the left ventricle (Fig. 5). The sac occupies the entire lateral area of the cardiac outline in the antero-posterior view and is seen to extend posteriorly to the cardiac silhouette in the lateral view. On aortography normal right and left coronary arteries were shown arising in the usual position from the aorta. The patient remains well and no operation has yet been performed.

Case 2 A 44-year-old housewife was admitted with a history of increasing shortness of breath on exertion over a period of 2 years and frequent attacks of palpitations over the same period. On examination she was found to have the signs of severe aortic valve stenosis. The arterial pulses were small, anacrotic, and the rhythm was sinus with frequent ventricular extrasystoles. The venous pressure was normal, there was a forceful left ventricular apex beat in the 5th left space in the mid-clavicular line, and no other praecordial impulses. On auscultation a harsh aortic ejection murmur radiating into the neck, preceded by a loud aortic ejection click, was heard. The chest x-ray (Fig. 6) showed slight cardiac enlargement with normal lung fields and no calcium was visible in the aortic valve. The electrocardiogram confirmed severe left ventricular hypertrophy and there were frequent ventricular extrasystoles.

Right heart and transseptal left heart cardiac catheterization showed a pulmonary artery pressure of 30/10 mm. Hg, a left atrial pressure mean of 6 mm. Hg, left ventricle 215/14 mm. Hg, aorta 100/65 mm. Hg, and a cardiac output of 4.8 l./min. Selective left ventricular angiography showed aortic stenosis due to thickened fused aortic valve cusps and also a smooth-walled aneurysm, with a narrow neck arising from the cavity of the left ventricle near to the anterior inferior margin (Fig. 7). The neck was seen to contract down during ventricular systole.

At operation using cardiopulmonary bypass, aortic valve stenosis was found to be due to a hypoplastic commissure between the two coronary cusps and to partial fusion of the anterior and posterior commissure of this essentially bicuspid aortic valve. At the apex of the heart there was an aneurysm of the left ventricle with an outside diameter of 4 cm. and a narrow neck of 1 cm. leading into the left ventricular cavity.

The aortic valve stenosis was relieved by dividing the areas of commissural fusion, leaving the

FIG. 4 Displacement of right ventricular outflow tract shown on right ventricular angiography.

hypoplastic commissure intact; the ventricular aneurysm was excised, and the neck closed with interrupted black silk stitches. Further examination of the aneurysm confirmed the dimensions to be 4.0×2.0 cm. with an average wall thickness of 0.5 cm. The wall of the aneurysm was formed by myocardium which on histological examination showed areas of extensive replacement by fibrous tissue. The remaining muscle fibres in the wall were conspicuously hypertrophied.

The patient remains well three years after operation.

Discussion

Two main types of ventricular aneurysm may be distinguished, the true and the false, according to the composition of the wall (Roberts and Morrow, 1967). The wall of the true left ventricular aneurysm, though thinned and replaced by fibrous tissue, contains remnants of left ventricular muscle and the aneurysm was originally formed by a localized dilatation of the left ventricular wall.

By contrast, the wall of the false ventricular aneurysm is composed of dense fibrous tissue which may contain calcium deposits, but it is

FIG. 5 Selective left ventricular angiography. (A) Antero-posterior projection: huge thin-walled sac outlined. (B) Lateral: the huge sac partly overlies and extends posteriorly to the left ventricle.

FIG. 6 Case 2. Chest x-ray.

devoid of myocardial fibres and of coronary arteries (Hurst, Fine, and Keyes, 1963). A false aneurysm occurs as a sequel to rupture of infarcted left ventricular wall when the extravasated blood is confined by an adherent pericardium. The wall of the aneurysm becomes thickened, presumably as a response to the intraventricular pressure, and the sac progressively enlarges. The false, or pseudo-aneurysm, of the left ventricle is exceedingly rare and, according to Roberts and Morrow (1967), only about 5 patients have been described.

Myocardial fibres were detected in the wall of the aneurysm in Case 2, providing conclusive proof that this is a true aneurysm of the left ventricle. The small mouth and narrow neck of this aneurysm give it an unusual elongated shape. This, then, is a saccular aneurysm, an exceedingly rare type of left ventricular aneurysm, of which practically all instances described have been encountered in young subjects (Higginson and Keeley, 1951; Roberts and Morrow, 1967).

The aetiology of this small saccular aneurysm has not been determined with any degree of certainty. No evidence of coronary arterial disease was noted during cardiac operation, and in any event the shape of this aneurysm is quite unlike that of the usual post-myocardial infarction dilatation. One possi-

bility is that a small area of congenitally defective myocardium may have bulged out as a consequence of the abnormally high left ventricular systolic pressure, and one similar example of a congenital diverticulum extending from the apex of the ventricle was described by Skapinker (1951).

The very large saccular aneurysm in Case 1 has the angiographic features of a false aneurysm. It is excessively large, arises from the posterior or postero-lateral surface of the left ventricle from a mouth much smaller than the largest internal diameter of the aneurysm, and shares many of the appearances of the pseudo-aneurysm described by Roberts and Morrow (1967). The cause of pseudo-aneurysm is almost always coronary arterial disease followed by myocardial infarction and rupture into an adherent pericardium (Hunter and Benson, 1933; Hurst *et al.*, 1963; Roberts and Morrow, 1957), but there was no evidence of coronary arterial disease in the patient described here. The larger coronary vessels were shown to be normal in size and position on aortography, and this patient is in a young age-group for coronary atheroma. False ventricular aneurysm formation after both accidental and surgical trauma has been described (Bailey *et al.*, 1958), but there has been no suggestion of any severe chest wall trauma at any time in this patient.

The occurrence of a cardiac arrhythmia was a prominent feature in both of these patients and might have presented a clue to the diagnosis (Zeeman *et al.*, 1962). There are numerous reports of ventricular arrhythmias, particularly ventricular tachycardia, in the presence of ventricular aneurysm (Parkinson *et al.*, 1938; Wasserman and Yules, 1953; Couch, 1959). Ventricular aneurysmectomy for relief of life-threatening arrhythmias has been effective in at least 2 patients (Couch, 1959; Hunt, Sloman, and Westlake, 1969), and since the relief of the aortic stenosis and excision of the aneurysm there have been no further arrhythmias in the patient reported here. The prognosis for false aneurysm of the left ventricle has been uniformly bad, and most of the reported cases died of rupture of the aneurysm (Hunter and Benson, 1933; Hurst *et al.*, 1963). Operative removal of the pseudo-aneurysm appears to offer the best hope but this has not so far been attempted in the patient described here.

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FIG. 7 Selective left ventricular angiogram. (A) Antero-posterior projection: aneurysm with narrow neck arising from region of cardiac apex; aortic valve cusps thickened. (B) Lateral: diverticulum-like aneurysm extends anteriorly and downwards from the left ventricle.

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