CASE REPORTS

DOUBLE-BARRELLED AORTA WITH BACTERIAL ENDARTERITIC VEGETATIONS

BY

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With the rising incidence of dissecting aneurysm of the last few decades (Maniglia and Gregory, 1952), valve-like arterial artefacts are likely to increase, and upon some tapping edge, sooner or later, bacterial vegetations might be expected. Such a case is here reported, apparently for the first time.

Case History

A male wood-machinist, aged 45, became febrile after a single dose of penicillin given at the end of a course of arsenical injections in a clinic for venereal disease, and was admitted to hospital in April, 1951. In 1945 he had begun to sleep upright in bed and thereafter attacks of breathlessness and tachycardia were increasingly frequent. He also felt that his memory was deteriorating. On examination, he was dyspnoeic with congestion of neck veins, pulse of 168 a minute and blood pressure of 160/115. Systolic and aortic diastolic murmurs were heard. Wassermann and Kahn reactions negative. Blood counts: hæmoglobin, 13.4 g. per 100 ml., white cells, 8400 per cu. mm. Blood culture sterile. The electrocardiogram showed paroxysmal tachycardia. During treatment the rhythm reverted through fibrillation to normal. At the same time a systolic thrill became palpable, from which it was concluded that aortic stenosis must be added to the original diagnosis of aortic regurgitation, and that the cause must be rheumatism not syphilis.

He was henceforward in and out of hospital at intervals of a few months until his death in July, 1953. Signs of an infective process first appeared on his fourth admission in September, 1952, when he was delirious and sweating, had a white cell count of 11,000 per cu. mm., and gave a growth of *Streptococcus viridans* in blood culture. On treatment, the patient became mentally normal and began putting on weight within a fortnight, but relapsed and was treated in another hospital where *Streptococcus viridans* was again isolated from the blood. Presence of an aortic aneurysm was noted in the chest film at this time. He was transferred to us again in March 1953. A large abscess of sterile pus in the right thigh had to be opened during the eleventh week, and in the seventeenth week the patient died suddenly.

Necropsy

The heart showed great left ventricular hypertrophy and weighed 666 grams, the left ventricle being 23 mm., the right 4 mm. thick. The aortic cusps were thicker than normal, though still fairly flexible, and slightly shortened by fusion of their ends, with a calcified nodule in one angle. A tear in the inner wall (Fig. 1), which was sharply defined but with the edge rounded off by healing, ran spirally upward round the ascending aorta from just above the right coronary orifice to a point 5 cm. distal to the commissure. Anteriorly the two step-like edges of the tear had retracted to leave a bare area 2 cm. wide floored by re-endothelialized outer coat, with which they had fused. Posteriorly and to the right the separated inner layer of intima-media (6 cm. from side to side) was extended across the centre of the aortic lumen, while deep to it a free blood channel, nearly equal in capacity to that of the aorta itself, had been formed. This channel was re-endothelialized, though the deep surface of the separated lamina bore fibrous tags, the largest of which (up to 8 mm. long $\times$ 1 mm. diam.) stretched across the angle of separation from the rest of the wall on the right side like a strut, resembling one of the chordæ. A tear in the inner wall of the aortic arch formed the distal end of the aneurysmal passage (Fig. 2), which was between 6 and 7 cm. long. This second orifice, by which the blood returned to the main aorta, was situated between the openings of innominate and left common carotid arteries, while, at its lower and slightly less advanced end, there was

a depressed area (1 cm. diameter) of retraction and re-endothelialization similar to the larger area described in the ascending aorta. The free edge (5 cm. long) was the site of smooth firm yellow nodular projections (streaked by a little red clot) with numerous small glistening translucent granules situated on the deep (outer) surface of the edge opposite the re-endothelialized media of the main wall. The spleen (335 g.) contained the scar of an old infarct (2.5 cm. diameter). A bullet-ended thrombus was loose in the right common iliac vein. Arachnodactyly was absent.

FIG. 1.—Ascending aorta, showing separated intimal-medial layer throwing its shadow on the channel outside it.

Microscopic sections showed only the changes attributable to trauma in the ascending aorta, while the distal flap of the aneurysm was seen to consist of dense hyaline tissue, in one place calcified, covered by endothelium to which small fibrin plugs were adherent in places. Beneath one of these latter a very few coccal forms were found. *Streptococcus viridans* was grown *post mortem* both from a vegetation and from the spleen.

Discussion

The reverse process to that described here, namely the formation of aortic dissecting aneurysm from bacterial endocarditis of the aortic valve ulcerating the commissure, has been described (Bartol *et al.*, 1943), and two traumatic aneurysms of the femoral artery, one gunshot (Lipton and Miller, 1944) and one stab wound (Heckler, 1952), have developed the vegetations of bacterial endarteritis. The term “double-barrelled aorta” occurs in the description (Levine *et al.*, 1951) of a dissecting aneurysm of some five months’ duration which split the ascending aorta of a thirty-eight-year-old man. The heart (740 grams) had a hypertrophied left ventricle, and a transverse tear 2 cm. above the aortic valve opened into a bulging aneurysmal sac traversed by strands of fibrous tissue and filled by blood clot.

FIG. 2.—Distal end of the aneurysmal channel shown by glass tube passing behind flap bearing vegetations of subacute bacterial endocarditis. (Cut for micro-section closed by stitches.) The dark colour of the upper part is due to adherent clot.

A case of double-barrelled aorta of fifteen years' duration in a young athlete with a grossly hypertrophied heart (860 grams), who died of cardiac failure at the age of 32, was recorded by Hall (1926). The aneurysmal tunnel, completely endothelialized, originated in an annular tear 1 cm. distal to the aortic valve and broke back into the main lumen by another annular tear at the site of a patent ductus arteriosus. The lesion occurred after running a mile race; and the evidence here, as in certain other cases where the ventricle was hypertrophied, might be taken to suggest that tears at the pericardial anchorage of the ascending aorta could be due to abnormally forceful ventricular movements, combined with an unluckily placed patch of atheroma, rather than to medial disease. Since histological degeneration is always found in the neighbourhood of aneurysmal dissections, as was pointed out (Erdheim, 1930) in the original description of *medio-necrosis aortæ idiopathica cystica*, it is not easy to separate effect from cause.

The present case seems almost a replica of Hall's, with the addition of bacterial vegetations on the distal end of the tunnel, and with the origin of the hypertrophied ventricle in a rheumatic incompetence instead of a congenital defect. In this case the force of the heart beats was one of the patient's complaints—a feature that might prove of some help in the discrimination of future cases. For instance, in a recent report of an ascending aortic dissection with the common termination of hæmopericardium (Bingle, 1957) the heart beats are mentioned as shaking the bed. The dissecting aneurysm seems likely to have occurred before our patient's first admission here (two years before his death) and might have been the cause of his admission to another hospital three years before that. The development of bacterial endarteritis can be reasonably assigned to the period between his third and fourth admissions, proving fatal therefore in about ten months.

Summary

A case is recorded in which a dissecting aneurysm of the ascending aorta ruptured back into the aortic arch, where the free edge of the tunnel became the site of subacute bacterial vegetations.

I should like to thank Dr. Ronald Hartley, under whose care this patient was admitted.

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