Case reports


Mycotic aneurysms of coronary arteries

B. R. M. Crook, E. B. Raftery, and S. Oram
From the Cardiac Department, King's College Hospital, London

A patient with a history of repeated episodes of subacute infective endocarditis associated with gross mitral incompetence, and electrocardiographic evidence of an inferior infarct, was found to have aneurysms of both right and left coronary arteries. Mycotic aneurysms of the coronary arteries are rare and have not previously been diagnosed during life.

Bacterial endocarditis may give rise to mycotic arterial aneurysms in any part of the body, but particularly in the thoracic aorta, intracranial vessels, superior mesenteric artery, and the large arteries to the extremities (Weintraub and Abrams, 1968). Aneurysms occurring in the coronary arteries are commonly arteriosclerotic or congenital and are very rarely mycotic in origin (Daoud et al., 1963). This report concerns a patient in whom the diagnosis of mycotic coronary arterial aneurysms was made in life by means of coronary arteriography.

Case report

A 56-year-old man was admitted to hospital in April 1969, with a history of loss of weight for three months, together with night sweats and progressive exertional dyspnoea. The presence of a heart murmur had been noted a year before his admission and he was known to have had scarlet fever at 4 years of age. Examination revealed finger clubbing, splenic enlargement, and the signs of mitral regurgitation with pulmonary congestion. A fever was recorded before treatment was started. His Hb was 11·8 g/100 ml and the erythrocyte sedimentation rate was 87 mm/1 hr (Westergren). A chest x-ray showed cardiac enlargement and pulmonary congestion. *Streptococcus faecalis* was consistently grown from blood cultures and a diagnosis of bacterial endocarditis was made. He was treated with ampicillin and streptomycin for 7 weeks, and also with digoxin and diuretics. He left hospital 2 months after admission, by which time it was evident that the bacterial endocarditis was no longer active though signs of severe mitral incompetence remained. In June 1969 he was readmitted to hospital with acute urinary retention. This resolved spontaneously but during this admission further blood cultures yielded *Staphylococcus albus* though there was no other evidence to suggest recurrence of bacterial endocarditis. After discharge he continued to attend outpatients and during the following weeks became more short of breath. He could manage to walk only 25 yards on the flat and also began to experience paroxysmal nocturnal dyspnoea. In August 1969 he was readmitted to hospital with congestive heart failure. Blood cultures failed to reveal any infection and the erythrocyte sedimentation rate was not raised. After resting in bed for one month, he returned home again, still on digoxin and diuretics.

In October 1969 he was admitted to hospital for consideration for mitral valve replacement. At that time his chest x-ray showed gross cardiac enlargement and an electrocardiogram showed changes compatible with ischaemia and a possible old inferior myocardial infarct. Left ventricular cineangiography showed gross mitral regurgitation with an immobile and calcified mitral valve. Both the left ventricle and atrium were dilated. Selective coronary arteriography was carried out by the Judkins technique (1968). There was no circulatory obstruction of the right or left coronary arteries but there was a loculated smooth-walled aneurysm on the main trunk of the right coronary artery and a small aneurysm on a single branch of the anterior descending branch of the left coronary artery (Fig. 1).

Mitrval valve replacement was carried out in November 1969. Both cusps of the mitral valve were perforated and there were many ruptured chordae tendineae. The cusps were excised and replaced with a Cutter valve. Severe hypotension followed operation and the chest was reopened on the following day, but no evidence of tamponade or excessive bleeding was found. The patient died two days after operation from hypotension and asystolic cardiac arrest.

At necropsy the heart was greatly enlarged with dila-

1 Present address: Northwick Park Hospital and Clinical Research Centre, Harrow, Middlesex.
FIG. 1 Left: left coronary artery, and right: right coronary artery, in the left anterior oblique position. Arrows indicate the position of saccular aneurysms on a branch of the anterior descending artery and on the right coronary artery. Note the absence of arterial wall irregularities, such as would be expected with atherosclerotic coronary arterial disease.

FIG. 2 Right coronary artery. (van Gieson, x 40.) There is aneurysmal dilatation which contains thrombus (see text).
tation of all its chambers. The aortic, pulmonary, and tricuspid valves were dilated but the cusps appeared normal. At 5 cm from the origin of the right coronary artery there was an aneurysm 1 cm in diameter. No gross abnormality of the left coronary circulation was seen on macroscopical examination and only mild atherosclerotic disease of both arteries was found. Histological examination of the myocardium showed considerable recent necrosis, especially of the papillary muscles and inner layer of the left ventricular myocardium, which was attributed to ischaemia at the time of operation. The right coronary artery was distorted by an aneurysm containing a thrombus. Histologically (Fig. 2) at the neck of the aneurysm there was abrupt cessation of medial muscle and elastic lamina. The wall of the aneurysm was composed of relatively acellular fibrous tissue. Scattered in the wall were focal areas of calcification. Chronic inflammatory aggregates were present, mainly in the adventitia and outer wall of the aneurysm. It was not possible to demonstrate organisms. There was pulmonary congestion and an enlarged liver showed nutmeg changes. An infarct was present in the left kidney and an old infarct in the enlarged spleen. No aneurysms of other arteries were found.

Discussion

The first major review of coronary arterial aneurysms was that of Packard and Wechsler (1929) who found 31 examples. Since then there have been further comprehensive reviews, and in 1963 Daoud and his co-workers were able to survey 89 cases diagnosed at necropsy, which included 10 of their own among the 66 men and 23 women in this series. The causation of the aneurysms, from the diagnosis offered by the original authors, was arteriosclerotic 52 per cent, congenital 17 per cent, mycotic or embolic 11 per cent, dissecting 11 per cent, and luetic 4 per cent. There was an almost equal distribution of the aneurysms in the left and right coronary arteries, and in 18 patients the aneurysms were multiple.

The diagnosis of coronary arterial aneurysms has only rarely been made in life. Munkner, Petersen, and Vesterdal (1958) reported a congenital coronary arterial aneurysm which was demonstrated by aortography in a child aged 6. Sherkat, Kavanagh-Gray, and Edworthy (1967) reported aneurysms of both coronary arteries in a 9-month-old infant, which were also demonstrated by thoracic aortography. In the absence of any other cause these aneurysms were presumed to be congenital. Sayegh, Adad, and McLeod (1968) described multiple fusiform aneurysms of the coronary arteries which were demonstrated by coronary arteriography in a 43-year-old man with severe atherosclerotic coronary artery disease. Konecke et al. (1971) described a traumatic aneurysm of the left coronary artery in a 17-year-old patient, also revealed by selective coronary arteriography. In our patient, aneurysms of the right and left coronary arteries were demonstrated in life by selective coronary arteriography, the presence of an aneurysm of the right coronary artery being subsequently confirmed at necropsy, though the small aneurysm of the left coronary artery was not commented on. The history of subacute bacterial endocarditis, the absence of any evidence of obstructive disease of the coronary arteries, and the appearance of the aneurysms arising as diverticula from the arteries, led us to believe that they were mycotic aneurysms. It is possible that the electrocardiographic evidence of inferior myocardial infarction was due to an infected embolus involving the right coronary artery during life.

Our thanks are due to Professor E. A. Wright who carried out the necropsy.

References


Requests for reprints to Dr. Samuel Oram, Cardiac Department, King's College Hospital, Denmark Hill, London S.E.5.
Mycotic aneurysms of coronary arteries.

B R Crook, E B Raftery and S Oram

Br Heart J 1973 35: 107-109
doi: 10.1136/hrt.35.1.107

Updated information and services can be found at:
http://heart.bmj.com/content/35/1/107.citation

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/