ANEURYSM OF THE CORONARY ARTERIES

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Aneurysm of the coronary arteries is rare. Since the first case in 1812 up to the review by Scott in 1948, 47 cases of localized aneurysm have been reported. Two further cases are described below.

Case I

A retired butcher, aged 66, gave a history of increasing breathlessness for one year. He also had occasional sharp pain in the chest, not typically anginal.

On examination, he was cyanotic and orthopnoic and had consolidation of the upper lobe of the right lung, with bilateral basal crepitations. No clinical abnormality was noted in the heart, but an X-ray was reported as showing cardiac enlargement. Three days later his dyspnoea had increased and he complained of severe pain down both arms and across the chest. His blood pressure at this time was 100/65; he had

FIG. 1.—Case 1. X-ray showing the outline of the blood clot over the right atrium.

œdema over the sacrum and a blood count showed 13,700 white blood corpuscles with 79 per cent polymorphs. His W.R. and Kahn tests were negative. He was thought to have had a further coronary thrombosis with increasing heart failure, and he died the next day.

Post-mortem examination. The pericardium was lightly adherent over the whole heart. Over the

posterior surface of the right atrium there was a small blood clot showing early organization. The heart weighed 615 g. The muscle was pale and friable. There was a small thrombus attached to the commissure of the two posterior cusps of the aortic valve, apart from which the cusps were normal. Immediately above the aortic valve there were two small saccular aneurysms of the aorta, which bulged posteriorly and upwards. The larger was 3.7 cm. in diameter; the smaller was adjacent to the larger with a strip of atheromatous aorta between. The rest of the aorta showed only slight atheroma. The left coronary artery, the first part of the circumflex branch and of the descending interventricular branch were completely obliterated by calcifying atheroma. The right coronary artery was hypertrophied, and 0.6 cm. from its origin there was an aneurysm 1.9 cm. in diameter partly filled with old laminated clot. There were a few ounces of fluid in the right pleural cavity, œdema particularly of the right lung, and some recent adhesions over the right apex. The liver showed chronic passive venous congestion.

FIG. 2.—External view of the heart from Case 1 with diagram.

FIG. 3.—Internal view of the left ventricle and aorta from Case 1, showing the site of the aortic aneurysm.

Histology. The wall of the left ventricle showed hypertrophy of muscle fibres, and patchy diffuse fibrosis. There were also areas of more recent necrosis with polymorph reaction. The aneurysm of the ascending aorta appeared to originate in an area of necrosis of the media. The walls were formed by collagen from the adventitia, and were lined by clot and fibrin. In the aorta adjacent to the aneurysm there was extensive vascularization of the outer third of the elastica, and perivascular infiltration with lymphocytes. The wall of the coronary aneurysm consisted of collagen only, with perivascular round cell infiltration.

Case II

A doctor's widow, aged 82 years, felt ill at supper, following a car drive in the afternoon, and retired to bed. In the morning she said that she had a "bad night." During the day she collapsed and the doctor was called but on arrival found her dead.

Her son said that his mother had suffered from a weak heart for 50 years and that recently her heart attacks had been worse. She had moved several times and only one doctor who had attended her could be found. The only information he could give was that she had had a retinal hæmorrhage and that she complained of dyspnœa on exertion and occasional œdema of the ankles.

Post-mortem examination. No abnormality was found in the respiratory and alimentary tracts. The liver showed moderate fatty change. The spleen was normal. The kidneys showed a slight reduction in the cortex with occasional small scars and cyst formation, the small arteries were prominent and the capsule did not strip very readily, the surface left being finely granular. The picture was that of renal arteriosclerosis.

The heart was 20 cm. long, 12 cm. across at the auriculo-ventricular sulcus and 10 cm. thick. It weighed 460 g. The left atrium had projecting from it a globular mass 6×7×6 cm. over which the pericardium was attached by fibrous tags. This mass had a wall 2 mm. thick, which obviously consisted of layers, some patches being yellow. Inside was firm laminated clot, mainly yellow, but with occasional hæmorrhagic places. On section the myocardium of the left ventricle was 18 mm. thick while that of the right was 8 mm. thick. The wall of the left ventricle showed a number of small white infarcted areas. The tricuspid valve was 11.5 cm. in circumference and the pulmonary 7 cm., both appearing normal. The mitral valve (9 cm. in circumference) was thickened with shortened chordæ tendineæ and under its posterior cusp just inside the left ventricle was a calcified mass. The aortic valve was much thickened and was 7 cm. in circumference. Attached to the posterior and medial angle of the left atrium was a dark reddish-brown almond shaped ante-mortem clot, 3.5×1.5 cm. No other abnormality was found in the left atrium. The orifices of the coronary arteries were very large, being about 1 cm. in diameter on either side. Each had a patch of atheroma round the mouth. Both coronary arteries were much dilated and tortuous and the site of well developed

FIG. 4.—Anterior view of the heart from Case 2 showing the large coronary aneurysm.

atheroma. Their average circumference was about 2 cm. The left coronary took a normal course; so did the right until, at the point where the circumflex branch turned down, on the back of the left ventricle, just inferior and posterior to the aortic valve, it entered a small saccular atheromatous aneurysmal cavity about 2 cm. in diameter, containing no clot. From this sac sprang a completely calcified non-patent artery which penetrated the muscle of the left ventricle and traversing it, appeared just under the posterior cusp of the mitral valve as the calcified mass previously mentioned. From here it ran laterally and anteriorly curving round the ventricle under the posterior mitral cusp to end in the inferior-medial wall of the large aneurysm. No other vascular attachment of the large aneurysm could be found and it "shelled off" the left atrium with ease except where the calcified vessel entered.

Histologically the large aneurysm contained laminated clot. The wall showed marked atheromatous change, much fibrosis with cholesterol clefts, scanty frayed elastic tissue, patches of calcification and a few collections of small round cells. The vessel under the mitral valve was completely calcified and its previous microscopical structure as an artery could just be discerned.

The aorta showed little atheroma in the ascending part but there was severe atheroma in the arch and descending parts, patches of which were breaking down. The lower part of the descending aorta where it bifurcates was occupied by a large aneurysm $12 \times 8 \times 5$ cm containing a very firm yellow, hæmorrhagic clot. Near its upper pole the aneurysm had ruptured filling the retroperitoneal space with a vast quantity of blood and causing the woman's death.

DISCUSSION

Scott in 1948 collected 47 cases of coronary aneurysm of which 15 were congenital, 12 mycotic-embolic, 6 syphilitic, 6 arteriosclerotic, 4 of other types, and 4 unclassified. He considered that these aneurysms were localized or diffuse, and that all the diffuse aneurysms were congenital while the localized had various origins. He found that in cases of localized aneurysm the left coronary was more frequently involved. Men were more often affected than women, and there were no pathognomonic clinical findings. The condition was frequently associated with cardiac infarction.

In our first case, arteriosclerosis and syphilis were considered as possible ætiological factors, the negative W.R., the history, and the age of the patient favouring the former diagnosis and the appearance of the aortic aneurysm and some parts of the histological picture favouring the latter. A suggestion that the aortic aneurysm resulted from an avascular necrosis, due to occlusion of the left coronary artery, from branches of which the root of the aorta partly derives its blood supply, was not supported by much evidence. Radiographs of the coronary arteries injected with bismuth in gelatin showed the root of the aorta to be one of the areas where there was a particularly rich anastomosis between right and left coronary arteries (Shillingford, 1950).

In our second case the lack of clinical data made it difficult to prove the cause of the aneurysms but the appearances post mortem strongly supported an arteriosclerotic origin. This was favoured by the presence of coronary and myocardial degeneration and by severe arteriosclerotic changes in the aorta particularly the abdominal part terminating in its rupture. The case was unusual in several features; it occurred in a woman, the right coronary was affected and there was more than one aneurysm.

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