DISSECTING ANEURYSM OF THE AORTA ASSOCIATED WITH GOLDBLATT KIDNEY

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Cases of dissecting aneurysm have been reported in which the dissection sheared off the origins of the renal arteries and brought about death from anuria. In the case to be described, the dissection involved the left renal artery only and the obstruction to the circulation was incomplete. The result seems to have been the development of hypertension, through a mechanism analogous to the classical Goldblatt experiments.

Case Report

The patient, a single women aged 54 years, was first admitted to hospital in December 1950 because she had suddenly lost consciousness for a short period the day before, and this had been followed by weakness in the left leg. On admission her blood pressure was found to be 210/100 but settled to 160/80 within a few days. She made good progress, but ten days after admission experienced a sudden attack of severe pain behind the sternum. The pain was described as cutting, and lasted for four hours. Four days later there were signs of a pleural effusion and 150 ml of fluid was aspirated from the left chest. There were no further attacks of pain, and on discharge from hospital, one month later, X-ray films of the chest showed no abnormality. The blood pressure was 160/80 and there was a little residual weakness of the left leg.

The patient remained fairly well for a few months, but her blood pressure then began to rise and she experienced headaches and palpitation. In September 1952 her blood pressure was 260/160 and she was treated with hexamethonium bromide. Early in 1954 she was admitted for a review of her hypertension, and a routine X-ray of her chest revealed an aneurysm of the ascending aorta. Wassermann and Kahn tests were negative. The patient improved with rest and was discharged after one month.

In May 1954 she experienced her second attack of pain in the chest. She had no more pain in the chest that year, but in December she had severe and prolonged abdominal pain. Examined at her home, she was found to have an enlarged and very tender abdominal aorta.

During the next two and a half years she had few attacks of pain and was fairly comfortable, leading a restricted life. A further chest X-ray in April 1957, showed that the thoracic aneurysm was now very large. In May there was another attack of severe chest pain and further attacks of pain occurred during the remaining months of her life. In March 1958, signs of cardiac and respiratory embarrassment appeared and she died in hospital at the end of April.

Necropsy. The pleural cavities were occupied by large aneurysms, so that the right lung was completely, and the left lung almost completely, collapsed. The heart and the aorta, with the commencement of the main branches, were removed in one block (Fig. 1). There was no disease of the heart. A very large aneurysm of the ascending aorta and the first part of the arch was separated by an isthmus from a large aneurysm of the descending thoracic aorta. The aorta narrowed to pass through the diaphragm, to expand again into an aneurysm of the upper two thirds of the abdominal aorta. There were also aneurysmal dilatations of the innominate artery continuing into the right common carotid artery, and also of the left common iliac artery. All these aneurysms formed the parts of a system of intercommunicating dissections in the wall of the aorta, and an aorta of normal calibre traversed the centre of this system. The only communication between the interior of the aneurysmal system and the lumen of the aorta was a longitudinally running slit, 2·5 cm. in length, situated 3 cm. above the aortic valves. The intima of the aorta showed a moderate degree of atheroma in the thoracic part and considerable atheroma in the abdominal part.

The right kidney appeared to be normal, but the left kidney was only half the size of the right and the capsule was adherent and the surface somewhat granular (Fig. 2). The origins of the main branches of the upper part of the abdominal aorta were then examined. The aneurysm was situated posteriorly; the celiac axis, superior mesenteric, and right renal arteries originated from the aorta, but the origin of the left renal
artery was included in the dissection. The left renal artery thus communicated with the interior of the aneurysms, but there was no communication at all with the lumen of the aorta. The blood flow reaching the left kidney via the aneurysmal system must have been a very poor one.

Sections from various parts of the wall of the aorta showed that there was an extensive degeneration of the elastic fibres of the media which were to a large extent replaced by collagen. Sections from the left kidney (Fig. 3) showed many sclerosed and hyalinized glomeruli with similar changes in many of the cortical arterioles. Only occasional sclerosed glomeruli were seen in the right kidney.

Discussion

Dissecting aneurysm is usually an event of short duration, ending in death from external rupture. In the present case, symptoms that suggested episodes of dissection occurred over a period of seven years, and external rupture never occurred. It has commonly been thought that dissection occurs as a result of a sudden breach of the intima, in an aorta weakened by previous degeneration of the media. There was medial degeneration in the present case, but the history and the small size of the communication between the aorta and the aneurysms did not suggest an origin in this way. However, Schnitker and Bayer (1944) consider that dissecting aneurysm usually begins within the media, as a result of rupture of one, or several, of the vasa vaurorum; and a series of episodes of this type, some more extensive than others would explain both the history and the necropsy findings.

Hypertension is a frequent forerunner of dissecting aneurysm and is sometimes one of the factors determining its onset. Here probably, the hypertension was an effect, rather than a cause, and was the result of
interference by the aneurysm with the circulation in the left renal artery. The origin of this vessel was included in the aneurysm, and had been for a long time. An attack of pain, indicating an episode of dissection in the thoracic aorta, occurred before the patient had any serious hypertension, and the history suggests that silent dissection could have started in the abdominal aorta then or even earlier. Certainly extension of the dissections sometimes occurred silently in this patient, because early in 1951 X-rays of the chest showed no abnormality, whereas three years later they showed a thoracic aneurysm, without the patient having had any further chest pain.

Obstruction to the circulation in the main renal artery is becoming recognized as a possible cause of hypertension (Lancet, 1958; and Poutasses and Dustan, 1957). Eastcott and Sutton (1958) mention this as a possible cause of hypertension, in a case of chronic dissecting aneurysm diagnosed by aortography. There is further support for this view in the present case, by the finding at necropsy of a contracted kidney with sclerosed glomeruli on the left side, and an almost normal kidney on the right. Once established, hypertension would be an important factor in bringing about further dissections, and removal of the left kidney at an early stage might have arrested the growth of the aneurysms.

Summary

A dissecting aneurysm of the thoracic and abdominal parts of the aorta increased in size over a period of seven years. There was a series of attacks of pain occurring at intervals, each attack suggesting a fresh episode of dissection. The thoracic aneurysm became very large and caused death from cardiac and respiratory embarrassment. Hypertension was present, and is thought to have been caused by interference with the circulation in the left renal artery due to inclusion of its origin in the dissection of the abdominal aorta.

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References