CASE REPORTS

COMPLETELY HEALED DISSECTION OF THE AORTA WITH DEATH FROM PHLEGMONOUS COLITIS

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Complete healing of a dissection of the aorta is very rare, only two reported instances having been found (Shennan, 1932; Case 41091, 1955). This contrasts with numerous reports of partial healing where a "double-barrelled" aorta has been formed, the dissection having established two connexions with the systemic circulation and become lined by endothelium. In the patient described below healing coincided with treatment by ganglion-blocking drugs, including pentolinium tartrate. Death 16 months later was partly due to the effects of these drugs and partly due to the precarious blood supply to the intestine.

Case Report

Clinical History. A man, aged 67, was admitted to hospital in October 1955 with severe localized epigastric pain which had lasted one day. His blood pressure was 220/120 mm. Hg and his heart was enlarged. The white cell count was 20,000 per c.mm. of which 87 per cent were polymorphonuclear neutrophils. There was transient proteinuria. The electrocardiogram revealed right bundle-branch block but there was no evidence of myocardial infarction. The Wasserman reaction and the Kahn precipitation test were negative. Initially requiring morphia, he was thereafter treated with a low salt diet and a series of hypotensive drugs. Control was finally obtained, after a second period in hospital, with reserpine 0·25 mg. three times a day and methylcholine hydrochloride 25 mg. and subcutaneous pentolinium tartrate 16 mg. twice a day.

He was admitted to hospital again in January 1957 because of lower abdominal pain and nausea for two days. He had been constipated for weeks and had not defaecated for five days. His abdomen was found to be tender and rigid and bowel sounds were diminished. The blood pressure was 210/100 mm. Hg. He was treated with morphia, intravenous fluid, gastric suction, and antibiotics, and approximately 300 ml. of hard faeces were removed manually from the rectum. He died nine hours after admission.

Autopsy. There was severe atheroma and calcification of the aorta: from the left subclavian artery to the celiac axis it was slightly dilated and thickened anteriorly (up to 0·6 cm.) by firm white and pale brown tissue which appeared to be outside the media. A maximum of 5 cm. out of a total aortic circumference of 6·5 cm. was involved. The posterior limits roughly coincided with the intercostal arteries which were so narrowed by atheroma that it was impossible to determine whether they were involved, but the major branches of the aorta were not. The abdominal aorta was dilated to form an anterior fusiform aneurysm (9 × 4 cm.) extending to the bifurcation. This contained old layered thrombus which obscured the origin of the inferior mesenteric artery. The superior mesenteric artery appeared to be occluded by atheroma and old thrombus.

Faeces distended the terminal 25 cm. of sigmoid colon and rectum to a maximum circumference of 17 cm. The bowel wall was thin and discoloured, with slight dulling of the serosa and there were several stercoral ulcers up to 3 cm. diameter. The peritoneal cavity contained 50 ml. of cloudy red-brown fluid. The cæcum and ascending and transverse colon were normal but the distal three-quarters of the small intestine were slightly reddened and dilated.

The heart was hypertrophied (465 g.) and dilated. The valves were normal. A raised plaque of fibrosis was present in the endocardium on the right side of the ventricular septum, immediately inferior to the anterior end of the septal leaf of the tricuspid valve. There was moderate patchy coronary atheroma.
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FIG. 1.—Transverse sections of the thoracic aorta. The elastic laminae of the media stain black. The layers are: 1, intima; 2, inner layer of media, containing pale-staining patches of muco-cystic degeneration in A; 3, organized dissection containing large thin-walled blood vessels; 4, outer layer of media; 5, adventitia. In A the outer layer of media is much thicker than in B. The great width of the scar in the media is illustrated in B. Both sections: Weigert's elastin and Curtis' Ponceau S; x 21.5.

**Histological Findings.** The thoracic aorta was thickened by a layer of dense collagenous tissue which was external to the greater part of the media but separated from the adventitia by a layer of media of varying width (Fig. 1). In some sections there were gaps in the outer layer of media where the collagenous layer merged with the adventitia, and this also was thickened. The layer of dense collagen contained a moderate number of thin-walled blood vessels and a small amount of brown pigment that contained iron. In some places there were fine fibres which stained with Weigert's method for elastin. There was much muco-cystic degeneration of the aorta and of elastic arteries. In some sections the media was very thin or absent beneath atheromatous plaques so that the latter were in direct contact with the collagenous layer.

The superior mesenteric artery contained a canalized organized thrombus. The sigmoid colon was congested and infiltrated by large numbers of neutrophil polymorphonuclear leucocytes. Serial sections showed that the right branch of the bundle of His was enclosed by the plaque of subendocardial fibrosis. There were conspicuous changes of benign hypertension in the kidney.

**Comment**

The aorta described by Shennan (1932) contained an 8 weeks old organized dissection and two subsequent separate dissections, one of which had ruptured into the pericardium. Shennan was able to demonstrate that the organized dissection had communicated with the lumen of the aorta and that this also had healed.

The case from the Massachusetts General Hospital in 1955 was a patient who had been treated with hypotensive drugs and lived 18 months after the dissection. Death was caused by a pontine haemorrhage. In this aorta also intimal tears were demonstrated.
In the present case it is reasonable to suppose that the aortic dissection was the cause of the pain that brought the patient to hospital 15 months before death. Although a considerable proportion of the aorta was involved, the situation of the dissection away from branches probably explains the relative paucity of symptoms. It is of interest that at no time was there any history of abdominal symptoms which can be related to the thrombus in the superior mesenteric artery. The gaps in the inner layer of media did not look like healed tears, but were all covered by thick atheromatous plaques and may have been a consequence of atheroma. However rupture may have taken place through, or a healed tear may have been covered by, an atheromatous plaque. On the other hand, the gaps in the outer layer of media indicate that the hæmorrhage extended into the adventitia. If there had also been a connexion with the lumen it is surprising that a severe or fatal hæmorrhage did not occur.

The illustrations in the two previous reports show that the dissection took place in the outer part of the media and this was largely true of the present instance. This may explain the complete organization of these dissections as this part of the media has the best blood supply. In a recently examined dissection of the aorta that ruptured into the thorax after 12 days, the media had split at varying depths but organization of the hæmatoma had begun only where there were few elastic lamæ separating it from the adventitia.

The fluctuating blood pressure that occurs in patients treated with ganglion-blocking drugs may be a factor in precipitating dissection of the aorta (Perry and Schroeder, 1954; Barnett, 1956; and Heaven and Murphy, 1956). Such drugs have also been advocated in the treatment of this condition (Pyke, 1956) and it is likely that, once dissection has begun, hypotensive drugs might limit its extension. This could have happened in two of these patients with completely healed dissection.

Fibrosis around the right branch of the bundle of His without any other demonstrable cardiac lesion is of considerable interest in a patient with right bundle-branch block. There was no indication of the origin of this lesion.

The severe constipation was responsible for the stercoral ulceration and phlegmonous colitis. Although ganglion-blocking drugs undoubtedly contributed to this constipation, the meagre blood supply to the intestine must have been an important factor. [The inferior mesenteric artery was obscured by thrombus and the superior mesenteric artery was almost obliterated.] Hypotensive therapy would further diminish the blood flow through the narrowed vessels. Thus the fate of this patient emphasizes the danger of constipation in treatment with ganglion-blocking drugs, specially if there is severe arteriosclerosis.

The dilatation of the distal three-quarters of the small intestine did not appear to be a direct result of mechanical obstruction of the large intestine as the bowel between the sigmoid colon and ileocecal valve was normal. The ileus may have been a result of peritonitis, but the limited distribution is similar to that seen radiographically or at operation in patients treated with ganglion-blocking drugs (Bourne and Horsford, 1951; Mackey and Shaw, 1951; Goldstone, 1952; McCalla, Creech, and Ford, 1953; Grant and Boyd, 1957; and Furste, Phelps, and Taylor, 1958). However it is likely that the early peritonitis was a factor in the development of this localized ileus. Barnett (1956) suggests that a similar combination of ganglion-blocking drugs and abdominal adhesions precipitated intestinal obstruction in three of his patients.

**Summary**

A large completely healed dissection of the aorta of 15 months’ duration was found at autopsy in a patient with benign hypertension treated with pentolinium tartrate.

Death was due to acute peritonitis and phlegmonous colitis caused by constipation. The intestinal blood supply was very poor. Ileus of the small intestine may have been precipitated by ganglion-blocking drugs.

Right bundle-branch block was associated with fibrosis around the bundle of His.

I am grateful to the late Dr. A. M. Barrett for his advice and criticism, to Dr. G. A. Gresham who examined the heart, and to Dr. A. P. Dick for permission to publish this case.
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References


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*Br Heart J* 1963 25: 532-535
doi: 10.1136/hrt.25.4.532

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