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CASE REPORT

Acute Aortic Dissection Following Cannulation of the Iliac Artery

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The aorta of a patient with aortic valve stenosis may be unduly thin, fragile, and liable to dissect irrespective of the presence or absence of atheroma. Indeed, Coleman (1955) stated that aortic dissection in young people was most commonly associated with coarctation or with bicuspid aortic valve. Petch (1952) reported fatal hæmopericardium from ruptured dissecting aneurysm in a man of 35 years with calcific aortic stenosis; a similar example in a man of 65 years was described by Heath, Edwards, and Smith (1958). Hudson (1965) also refers to the association between aortic valve stenosis and aortic dissection, sometimes with Marfan's disease also.

In the following fatal case this complication followed unsuccessful cannulation of the left external iliac preliminary to establishing cardiopulmonary bypass for surgical replacement of the diseased aortic valve.

Case Report

A man of 59 years with a history of increasing dyspnœa and angina pectoris for 3 years was admitted to hospital with a diagnosis of non-rheumatic calcific aortic valve stenosis. This was verified by the usual confirmatory investigations. The rhythm was normal and the blood pressure 130/80 mm. Hg.

Operation. In January 1965, Mr. Donald Ross undertook replacement of the aortic valve by a stored homograft using full-flow normothermic cardiopulmonary bypass.

Cannulation of the left external iliac artery was apparently successful, but on starting bypass the arterial line was found to be obstructed. The cannula was, therefore, withdrawn and inserted into the corresponding artery on the right side. The heart was electively fibrillated. When the ascending aorta was clamped, it was noted that its proximal portion did not decompress. Incision of the aortic wall, however, produced decompression, and this revealed that the aortic wall had dissected, almost to the origins of the coronary arteries. Further incision of the intima exposed a heavily-calcified valve, with only one recognizable commissure, quite unsuitable for reconstruction. The valve was excised and replaced by a stored homograft. The dissection space was then closed just proximal to the aortic clamp by a continuous whip-stitch inserted circumferentially through all coats. The heart defibrillated readily and subsequently took over the circulation without difficulty.

At 10 a.m. on the day after operation the diastolic blood pressure, which had been 70 mm. Hg, fell suddenly to zero. There was no change in systolic pressure. No diastolic murmur was noted. A satisfactory urinary output was maintained.

The extent of the dissection was then investigated—first by retrograde, and then by cine-aortography; in addition, the aorta was injected to show filling of both renal arteries from the true lumen.

Further surgery seemed unwarranted. On the third post-operative day, the patient suddenly collapsed with ventricular fibrillation which resisted all efforts at resuscitation.

Necropsy (Dr. Reginald E. B. Hudson).

Macroscopic Appearances (Fig. 1). The *heart*, together with the thoracic *aorta*, weighed 800 g., chiefly on account of hypertrophy of the left ventricle to a wall thickness of 2·1 cm. (B). The aortic homograft was firmly and correctly sutured into position below the coronary ostia, and the main coronary arteries were widely patent, with moderate atheroma only. The aorta was widely dissected from both its iliac branches to the occluding line of sutures just above the aortic valve; the dissection involved the renal arteries (which were both occluded by thrombus), and all three arch branches. The distal abdominal aortic intima showed severe atheroma, with calcification, ulceration, and thrombosis; both iliac arteries were also atheromatous (A).

The *lungs* were œdematous and congested and the pleural surfaces showed several dark nodules resembling small damsons. The *small intestine* carried a Meckel's

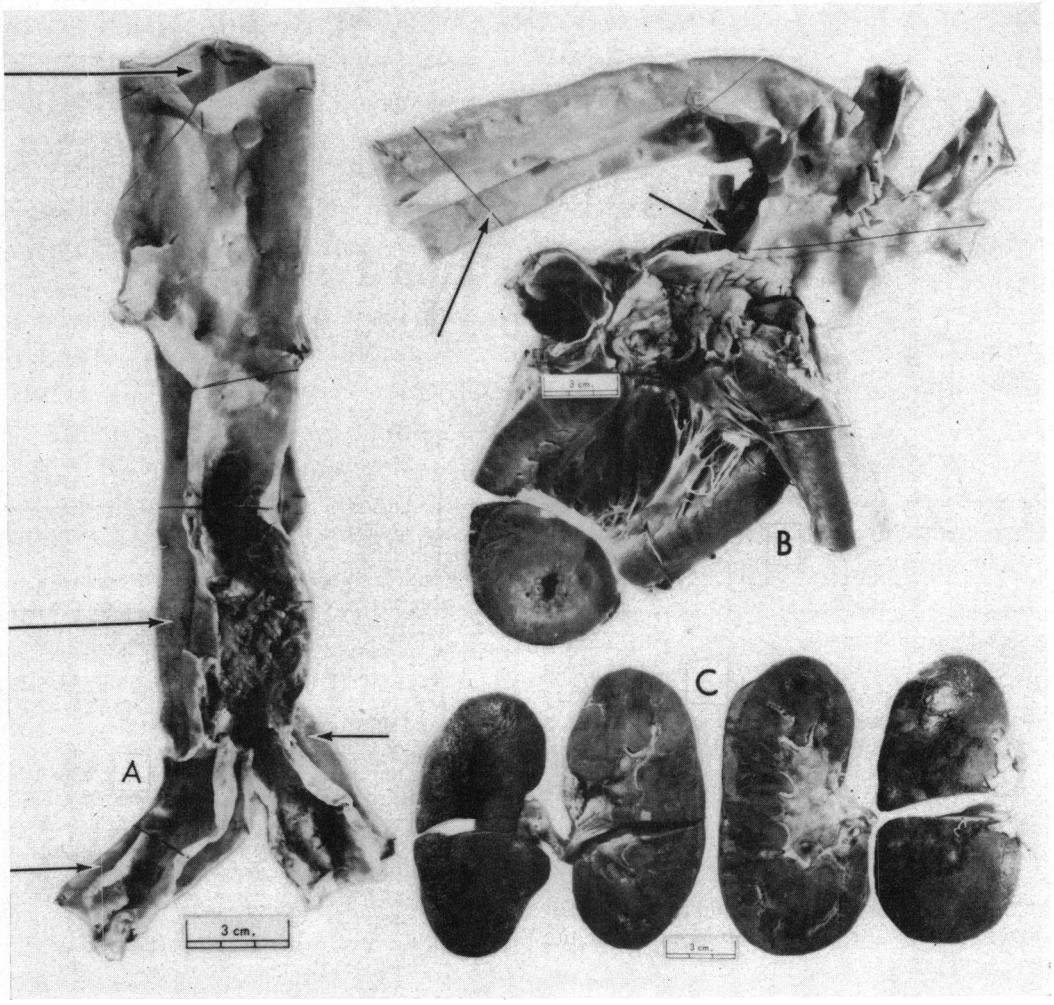

FIG. 1.—(A) Abdominal aorta showing dissection (arrows) which apparently originated in the left external iliac artery. There is severe calcific atheroma with mural thrombosis of the aorta above the bifurcation, and severe atheroma of both iliac branches, which are also dissected. (B) Heart with thoracic aorta. The dissection (arrows) extends to the row of sutures just above the homograft aortic valve; it also extends into the 3 arch branches. (C) Kidneys showing extensive recent infarction. (Photographs by courtesy of Dr. Reginald E. B. Hudson.)

diverticulum, 4 cm. long. The *liver* weighed 1700 g. Both *kidneys* were extensively and recently infarcted (C). The *brain* was normal and the *spleen* was enlarged to 360 g.

Microscopic Appearances (Fig. 2). Histology of the *aorta* showed that dissection had extended into the wall below the upper limit of the valve homograft. The dissection space was between the outer third and inner two-thirds of the media—the usual site. The wall just below the bifurcation showed severe atheroma with accompanying adventitial lymphocytic aggregations; in addition the media was thinned and infiltrated with some cells, histiocytes, lymphocytes, a few neutrophils, and an occasional eosinophil; there were no giant cells (B). Similar lesions were present in the dissected left iliac artery, the medial elastic being completely destroyed in places (C). All sections of the abdominal aorta showed excessive polychromasia to toluidine blue, and empty clefts between the elastic lamellæ were conspicuous in places; though these clefts are known to be a post-mortem artefact, they were more numerous than normally seen. The cell reaction in the media of the distal aorta and iliac arteries was probably due to the severe atheroma rather than to a primary mesarteritis, because no such lesions were found more proximally, where there was little abnormality apart from the dis-

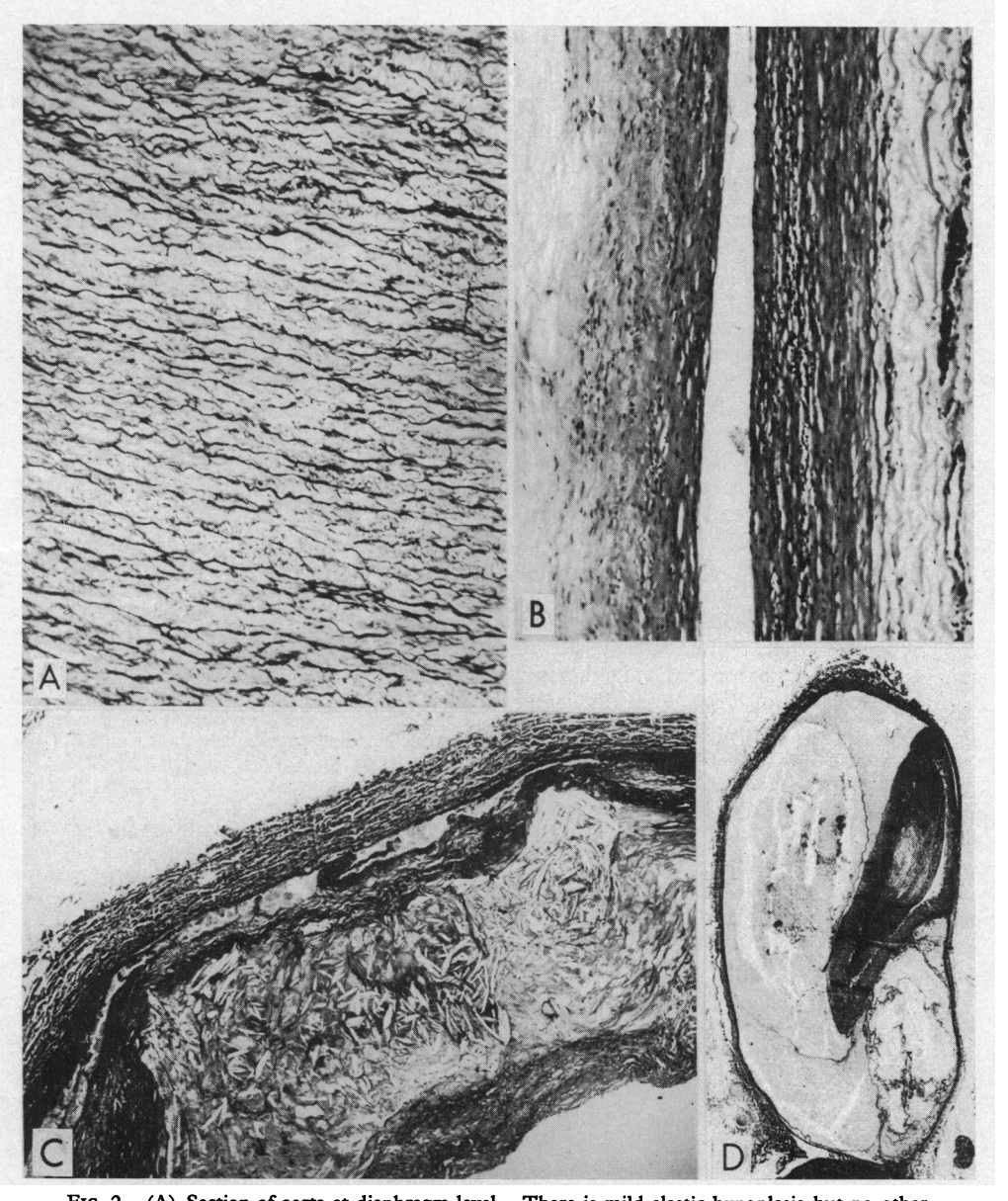

FIG. 2.—(A) Section of aorta at diaphragm level. There is mild elastic hypoplasia but no other abnormality. (Verhoeff's elastic—van Gieson. × 80.) (B) Aortic dissection at bifurcation to show the cell infiltration of the media bordering both sides of the dissection space running vertically down the picture. (H. and E. × 80.) (C) Left common iliac artery showing an area of almost complete destruction of the dissected medial coat, deep to a thick intimal plaque of atheroma containing numerous cholesterol clefts (below). The adventitia (top) is intact, and the narrow dissection space, in the outer-most media, is just below it. (Verhoeff's elastic—van Gieson. × 80.) (D) Renal artery dissection. The outermost rim of media has become dissected away by a hæmatoma which is compressing the original lumen to a narrow cleft inside the remainder of the vessel (top right) which also shows intimal atheroma. (Verhoeff's elastic—van Gieson. × 8.) (Photographs by courtesy of Dr. Reginald E. B. Hudson.)

section and possibly some elastic hypoplasia (A) and increase of polychromasia (a variable feature even in the normal aorta).

The occlusion of the *renal arteries* was found to be due to compression of the lumen by a dissecting hæmatoma in the outermost rim of the media (D). The pleural nodules proved to be hæmatomata, possibly traumatic.

Discussion

In the present case, when the dissecting aneurysm was first observed at operation, obliteration of the false lumen by approximation of the inner and outer layers of the dissection by continuous sutures, both proximally and distally, was thought to be the proper course in view of the serious nature of the operation to be performed, i.e. replacement of the aortic valve by a homograft. However, DeBakey *et al.* (1962, 1965) in their reviews of the surgical treatment of dissecting aneurysms suggested that obliteration of the false lumen by the above procedure should be followed by end-to-end anastomosis of the transected aorta.

Angiography is the most reliable technique available in the diagnosis of dissecting aneurysm. Hirst, Johns, and Kime (1958) in an extensive review recommended that it be carried out in departments equipped for such specialized radiology.

The commonest causes of death from dissecting aneurysm, after cardiac failure and myocardial infarction, are progression of the dissection and renal failure. In this case the aneurysm had dissected to all the major branches of the aorta as far as the femoral arteries, and the idea of any further operation had, therefore, to be abandoned. Moreover, necropsy showed that both kidneys were extensively infarcted.

Summary

A case is reported of dissection of the whole aorta, originating in traumatic cannulation of the left iliac artery, in a man of 59 years, preparatory to establishing bypass for homograft replacement of a calcified aortic valve.

The accident demonstrates the danger of dissection when perfusing the atheromatous aorta of a patient with aortic valve stenosis.

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References

Coleman, P. N. (1955). A case of dissecting aneurysm in a child. *J. clin. Path.*, **8**, 313.

DeBakey, M. E., Henly, W. S., Cooley, D. A., Crawford, E. S., Morris, G. C., and Beall, A. C., Jr. (1962). Aneurysms of the aortic arch: factors influencing operative risk. *Surg. Clin. N. Amer.*, **42**, 1543.

——, ——, ——, Morris, G. C., Jr., Crawford, E. S., and Beall, A. C., Jr. (1965). Surgical management of dissecting aneurysms of the aorta. *J. thorac. cardiovasc. Surg.*, **49**, 130.

Heath, D., Edwards, J. E., and Smith, L. A. (1958). The rheologic significance of medial necrosis and dissecting aneurysm of the ascending aorta in association with calcific aortic stenosis. *Proc. Mayo Clin.*, **33**, 228.

Hirst, A. E., Jr., Johns, V. J., Jr., and Kime, S. W., Jr. (1958). Dissecting aneurysm of the aorta: a review of 505 cases. *Medicine (Baltimore)*, **37**, 217.

Hudson, R. E. B. (1965). *Cardiovascular Pathology*, Vol. 1, p. 512. Edward Arnold, London.

Petch, C. P. (1952). Congenitial aortic aneurysm with valvular stenosis and dissecting aneurysm. *Brit. Heart J.*, **14**, 147.