Painless Dissecting Aneurysm of the Aorta

Report of a Case

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With the development of successful surgical treatment of dissecting aortic aneurysm, accurate diagnosis of this condition has become increasingly important. Pain in the torso, usually in the chest or back, is considered to be a classical feature of dissecting aneurysm of the aorta, but reliance on the presence of pain may result in unnecessary delay in diagnosis and treatment. Without treatment, approximately 25 per cent of patients survive the acute aortic dissection for more than 2 weeks, and approximately 10 per cent live for more than 6 months. Although dissecting aneurysm occurs most commonly in the sixth and seventh decades of life and is known to be associated with abnormal cardiovascular conditions, such as the Marfan syndrome, hypertension, and coarctation of the aorta, it may also occur in young adults with otherwise normal cardiovascular systems and in pregnant women.

The present report was prompted by experience with a patient who had a painless dissecting aneurysm of the aorta and who exhibited certain features that allowed the correct clinical diagnosis to be made in the absence of pain in the torso.

Report of a Case

A 53-year-old man first was seen at the Mayo Clinic on October 23, 1962, at which time he was complaining of dyspnea on exertion. Six weeks previously, he had suddenly become aware of rapid loss of sensation in his left leg, which became increasingly painful. He broke out into a profuse sweat and fainted for a few moments. His physician arrived within an hour and, noting absence of pulses in the left leg, sent the patient to the hospital where he underwent operation an hour later for acute occlusion of the left iliac artery.

Postoperatively, the patient had noted a return of function and sensation to his left leg and had been told that a clot had been removed from an artery above the groin. He was given anticoagulants. After the operation he also was told that he had a leaking heart valve and that this was probably due to rheumatic heart disease. Digitalis and an oral diuretic were prescribed. He was asymptomatic at the time of his dismissal from the hospital 3 weeks later.

Shortly thereafter, he had begun to experience progressive exertional dyspnea and, during the week prior to admission here, he had had several attacks of paroxysmal nocturnal dyspnea. He had not experienced any subsequent symptoms of peripheral vascular insufficiency. The patient denied a history of rheumatic fever and no heart murmur had been noted on several routine examinations prior to the present illness.

He was 64½ inches tall and weighed 158 pounds. He appeared chronically ill and was dyspneic at rest. Positive physical findings were limited to the cardiovascular system. The blood pressure was 145 mm. of mercury systolic and 65 diastolic in both arms. His pulses were collapsing in character and the rate was 124 per minute and regular. The left femoral, popliteal, and posterior tibial pulses were markedly diminished and the left dorsalis pedis pulse was barely perceptible. There was slight elevation pallor of the left foot and the venous filling time with dependency was 20 seconds.

The venous pressure was estimated to be approximately 14 cm. of water. Marked suprasternal and precordial pulsation was visible. There was a basal diastolic thrill, and the cardiac apical impulse was felt in the sixth left intercostal space in the anterior axillary line. A grade-IV (on the basis of I to VI) decrescendo aortic diastolic murmur, well transmitted to the apex, was also heard posteriorly just medial to the right scapula. A grade-II (on the basis of I to VI) aortic systolic murmur was also present. A separate systolic murmur could be followed from the epigastrium down to the left femoral artery and also into the right lower quadrant of the abdomen. Posteriorly, a loud systolic and a faint diastolic murmur could be heard, local-

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ized to the left of the lower lumbar region at the level of L2 to L5.

Routine studies of blood and urine, including serologic tests for syphilis, gave normal or negative results. Serum electrolytes, serum lipids, blood sugar, and blood urea also were normal. The electrocardiogram showed a sinus tachycardia and the pattern of left ventricular hypertrophy. Roentgenographic (fig. 1) and fluoroscopic examination of the thorax demonstrated cardiac enlargement with a prominent left ventricle and slight but definite dilation of the entire thoracic aorta. No valvular calcification was evident. A small amount of pleural fluid was present bilaterally.

A diagnosis of dissec ting aneurysm of the aorta with aortic valvular insufficiency and prior left iliac artery occlusion was made.

Shortly after his initial examination here, the patient was admitted to the hospital for control of acute left heart failure. Treatment consisted of bed rest, administration of a mercurial diuretic, and restriction of dietary salt. Treatment with digitalis was continued but, because of the diagnosis of dissecting aneurysm, the anticoagulant therapy was discontinued.

The patient subsequently underwent surgical exploration and repair on October 29, 1962. Exposure was through a primary median sternotomy (fig. 2). The left ventricle and ascending aorta were noted to be enlarged. There was an obvious diastolic thrill over the left ventricle and a less intense systolic thrill over the proximal ascending aorta. The aorta appeared tense and edematous with a slightly dusky hue. Extracorporeal circulation was instituted with use of the right atrium for venous drainage and the right common femoral artery for hypothermic perfusion, and the patient was cooled to an esophageal temperature of 30 C. The ascending aorta was cross clamped proximal to the right innominate artery and the aorta was incised transversely at a point 3 cm. above the sinuses of Valsalva. Coronary perfusion was instituted and a left ventricular vent was inserted.

It was apparent, on cutting through the thin adventitious outer layer of the aorta, that the intima had been completely stripped from the ascending aorta and that this dissection had originated from a T-shaped laceration in the intima, which was almost circumferential (fig. 2a). The vertical limb of the T extended down to the com-

Figure 1

Thoracic roentgenogram of patient.

Circulation, Volume XXIX, May 1964

Figure 2

Appearance of the ascending thoracic aorta at surgical exploration. a. T-shaped laceration in the intima is shown with the vertical limb of the tear extending down to the commissure between right and noncoronary aortic cusps. The horizontal tear was not completely circumferential and there was no separation of intima from the outer layers of the aorta in the area above the commissure between the right and left aortic cusps. b. Nature of the proximal dissection of the aneurysm about the base of the heart and aortic valves. Note the placement of the anchoring stitch at one of the commissures. Although not shown, the placement of these stitches at each of the affected commissures effected a return of competence to the otherwise normal aortic valve. b’. Mechanism of aortic insufficiency, showing the drooping aortic cusp failing to make contact with its opposite member. c and d. The technic employed in repair (see text).
misuse between the right and noncoronary cusps of the aortic valve. The proximal dissection between the intima and adventitia had extended into the sinuses of Valsalva and had detached all but the commissure between the left and right coronary cusps, resulting in insufficiency of the otherwise normal aortic valve (fig. 2b and b').

By reattaching the involved commissures to the outer wall of the aorta with interrupted sutures, the free margin of each cusp was elevated and competency was restored (fig. 2b). The vertical tear in the intima of the proximal part of the aorta was repaired and anchored to the adventitia with running silk sutures, after excising a V-shaped wedge from an attenuated bulge of adventitia (fig. 2c) to permit reattachment of intima to adventitia in this area. The circumferential tear in the intima was parallel to and superimposed on the transverse aortotomy incision. Prior to closure of this incision, the intima was reattached to adventitia, thus completely closing the origin of the dissecting hematoma and obliterating the false channel at that point (fig. 2d).

On closure of the aorta and resumption of cardiac function, it was apparent that the aortic valve was competent. The patient was rewarmed before the extracorporeal circulation was discontinued.

The postoperative hospital course was uneventful. The patient was dismissed from the hospital on the sixteenth postoperative day. At the time of dismissal there were no detectable cardiac murmurs, but systolic bruits over the left femoral artery and abdominal aorta persisted.

At re-examinations, in May 1963 and March 1964, the patient reported that he had been free from symptoms in the interval since the operation. Blood pressure measured 140 systolic and 85 diastolic. There were no cardiac murmurs and the previously noted back and abdominal bruits were unchanged. The peripheral pulses were normal in character but remained diminished in amplitude in the left leg.

Discussion

This case illustrates a number of features worthy of emphasis. Certain symptoms and signs were present that allowed the diagnosis of dissecting aneurysm of the aorta to be made clinically in the absence of the often emphasized pain in the chest, back, or abdomen. The clinical features of note included sudden onset of symptoms, syncope, acute iliac artery occlusion, appearance of aortic insufficiency, and bruits over the back and abdomen.

Certain conditions predispose a patient to the development of aortic dissection. The commonest associated condition by far is hypertension; aortic dissecting aneurysm characteristically occurs in hypertensive middle-aged men.3 Approximately 60 per cent of patients with dissecting aneurysm have hypertension,5,6 and it has been stated that,7 in the absence of predisposing conditions such as the Marfan syndrome, the disease is nearly always associated with hypertension. It is of interest, therefore, that there was no history of hypertension in our patient and that his blood pressure remained at normal levels during and after his illness.

The clinical manifestations of dissecting aneurysm may be produced directly by the dissection itself or indirectly as a consequence of occlusion of the arteries arising from the aorta. Occlusive manifestations may be complete or may be transient and partially reversible due to cessation of spread of the dissecting hematoma or to the re-entry of the dissecting hematoma into the lumen of an artery or the aorta.

One of the truly constant clinical features of dissecting aneurysm is the abrupt onset of symptoms.8,9 Although the symptoms may vary widely, depending on the location of the dissection and the aortic branches involved, the abruptness of onset warrants emphasis as a feature of note in this disease.

Syncope is probably the commonest neurologic symptom associated with dissecting aneurysm.8, 10 This fact may help in differentiating this condition from myocardial infarction in which syncope is rare7 and pain is also common. Neurologic symptoms, including syncope, are of importance in the clinical diagnosis of dissecting aneurysm because the correct diagnosis can be made much more readily when they are present than when they are absent.11

Amer and his colleagues12 recently have called attention to dissecting aneurysms presenting as iliac artery occlusions. It is of interest that only one of the four patients in their report experienced chest pain with the dissection; the others had no pain in the torso. Of the 53 cases in which pulses were recorded in both lower limbs in the series of dissecting aneurysms reported by Hirst and co-workers,3

Circulation, Volume XXIX, May 1964
in 37 (70 per cent) there was an inequality of pulses between legs and in 13 (25 per cent) no pulses were felt. Inequality of pulses in the neck and in the arms may also be an important sign in dissecting aneurysm, as may inequality of the blood pressure.

Aortic diastolic murmurs are common in patients with aortic dissection: the reported incidence ranges from 20 per cent to 56 per cent.14 The presence of an aortic diastolic murmur greatly facilitates the correct diagnosis of dissecting aneurysm with involvement of the ascending aorta.9 In our patient, it was observed at operation that the dissection extended into the sinuses of Valsalva and had detached all but the commissure between the left and right coronary cusps, resulting in insufficiency of an otherwise normal aortic valve (fig. 2b').

A bruit over the back or abdomen may be an important sign in the diagnosis of dissecting aneurysm of the aorta, as indeed it was in our patient. Bruits have been recorded over the thoracic and lumbar parts of the spinal column and over various parts of the abdomen. A good correlation between the site of an intimal tear and the point of maximal intensity of the bruit was noted in some cases, although in others this correlation did not exist. The lumbar bruit in our patient persisted after surgical correction of the intimal tear in the ascending aorta, allowing speculation that another intimal (re-entry) tear may have been present in the abdominal aorta.

Although the incidence of painless dissection of the aorta is difficult to estimate, Hirst and colleagues, 3 in a review of 505 cases, noted that pain was insignificant or absent in 15 per cent of the cases in which symptoms were recorded. The highest incidence of painless dissecting aneurysm was reported by Baer and Goldburgh: 24 (55 per cent) of their 44 patients were stated to have had no pain. It should be noted that, in some patients with dissecting aneurysms, there are circumstances that preclude a record of pain, namely, sudden death, loss of consciousness, confusion, and dysarthria.

The fact that aortic dissection may or may not be associated with pain raises the question of the origin of the pain when present, and of its significance. The cause of the pain is unknown, although several theories have been proposed and were briefly outlined by Kirkpatrick.18 Pressure of the dilated aorta on neighboring structures and ischemia of tissues whose arterial supply has been compromised, while undoubtedly causes of pain, are hardly sufficient to account for the sudden severe tearing pain so characteristic in many cases of acute aortic dissection.

That distention of the aortic adventitia causes the pain is probably the oldest theory. However, it does not account for the absence of pain in certain cases. Wood and co-workers advanced two possible explanations for this: firstly, that the dissecting hematoma might bulge the intima inward and re-enter the true aortic lumen early, without displacing the adventitia outward; and secondly, that the dissection might occur very slowly without sudden violent distention of the adventitia and might thus be painless.

Pain is absent twice as commonly in chronic as in acute dissecting aneurysm.9 It would thus appear that the presence or absence of pain has prognostic import in dissecting aneurysm of the aorta.

Summary

A case is reported of a man who had an acute painless dissecting hematoma of the aorta, which was diagnosed clinically in the absence of hypertension or known precipitating cause. The successful surgical treatment is described and illustrated.

The following diagnostic features were stressed in the absence of pain: sudden onset of symptoms, syncope, acute peripheral arterial occlusion, appearance of aortic insufficiency, and finding of bruits over the course of the aorta. The significance of the symptom of pain in the torso and the possible modes of its production in this condition are considered, and it is suggested that pain in dissecting aneurysm may imply a poor prognosis.

References

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Scientific Reasoning

Above all, it is essentially requisite that the physician should learn the art of reasoning; or that facility of distinguishing or rightly classing ideas, which must necessarily flow from the habitual application of the mental faculties to various branches of science, and which he, who has been merely occupied with what is called the practice of the profession, can rarely hope to possess.—Preface. Collections from the Unpublished Medical Writings of the Late Caleb Hillier Parry, M.D.F.R.S. Vol. I. London, Underwoods, Fleet-Street, 1825, p. 4.
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