Aortic Infarction Following Dissecting Aortic Aneurysm

SANFORD H. BARSKY, M.D. AND SEYMOUR ROSEN, M.D.

SUMMARY Aortic infarction was observed in 21 of 34 cases of dissecting aortic aneurysm. This lesion occurred as a central zone of necrosis with preserved elastic laminae, sparing media adjacent to the true and false lumens. In cases where the false lumen was occluded, the central infarction extended to this lumen. The infarction followed rather than preceded dissection, took approximately 48 hours to develop, and did not organize with time. The lesion occurred exclusively in the thoracic aorta, and bore no relationship to medial cystic necrosis. Present surgical therapy does not extirpate these areas, and the implication of these lesions in terms of management remains to be determined.

DISSECTING ANEURYSM, the most common life-threatening disease involving the aorta, has been the subject of extensive investigative work. Its relation to hypertension and underlying diseases of the aorta such as medial cystic necrosis (MCN) has been noted, although the role of these entities in the pathogenesis of aortic dissection is controversial. Similarly, the nature of dissection, its location, and consequent organ compromise have been analyzed in detail. In contrast, the effects of dissection on the aorta per se have largely been ignored. This report describes a lesion of aortic infarction, distinct from medial cystic necrosis, that occurs following dissecting aortic aneurysm. It is confined to the aortic segment involved in the dissection and is exclusively present in the thoracic aorta.

Materials and Methods

We reviewed all the cases of dissecting aortic aneurysm seen at the Beth Israel Hospital, Boston over a 25-year period (1953-1978) from which either autopsy or surgical material had been obtained. Thirty-four cases were found and included in this study. Formalin-fixed, paraffin-embedded sections of the aorta were stained with hematoxylin and eosin, elastic Van Gieson, Gomori trichrome, periodic acid Schiff (PAS) and Alcian blue stains. Thirty-four age-matched controls were obtained from patients who died of other causes; sections from these aortas were processed in the identical manner.

Results

Aortic infarction was present in 21 of the 34 dissection cases, but absent in all 34 controls. This lesion is readily identified by routine hematoxylin and eosin staining, but is highlighted by Gomori trichrome stains (fig. 1). Histologically, it is defined by a sharply delimited, centrally located zone of necrosis, characterized by loss of smooth muscle nuclei and cell dropout, and occupying one-fourth to one-half of the media that lies between the true and false lumens. This band of necrosis begins either at the point of dissection or a few millimeters distal to it and is not present in the aorta proximal to dissection. The intima and adjacent media bordering the true lumen are preserved, as is the media adjacent to the false lumen. Alcian blue stains reveal no increase in mucopolysaccharides, and elastic stains reveal preserved elastic laminae with no evidence of disruption. Inflammatory cells are consistently absent.

The classic lesion of medial cystic necrosis was present in 60% of cases, ranging from mild to severe involvement. This lesion is characterized by an increase in the amount of mucopolysaccharides in the aorta demonstrated with Alcian blue stains, and by a disruption of the elastic laminae. In contrast to aortic infarction, trichrome staining did not define MCN. The 21 cases of aortic infarction following dissection showed no increase in the amount of MCN compared with the 13 cases of dissecting aneurysm lacking aortic infarction. When MCN occurred with aortic infarction, the degree of MCN was always the same in both infarcted and uninfarcted segments of the aorta. When aortic infarction and MCN occurred together, the histology reflected the properties of both lesions superimposed on one another.

In the nine cases of aortic dissection in which the dissection began in the thoracic aorta and extended to the carotids, abdominal aorta, or iliacs, infarction was present exclusively in the thoracic aorta.

The presence or absence of this lesion was correlated with the clinical duration of the dissection (table 1). In the majority of cases, the diagnosis of dissection was based on aortography or chest x-ray, while in a minority of cases, its occurrence was inferred by a suggestive clinical history and physical findings such as severe chest pain radiating to the back and absent femoral pulses.

In the eight patients who died within 12 hours of dissection, there was no evidence of infarction. The lesion was present in two of six patients who died or were operated on 12-48 hours after dissection. In these later cases, aortic infarction was focal and involved more limited numbers of lamellae (fig. 2). The lesion was present in all but one patient dying or operated upon 48 hours to three years after dissection. The infarction did not organize with time but remained as a mummified band of dead, smooth mus-
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Figure 1. A) The area of aortic infarction is seen as a centrally located zone of necrosis with preservation of media adjacent to the true (upper) and false lumen. The dissection was 72 hours old; ×40 Gomori trichrome. B) Elastic stains reveal preserved laminae, making it difficult to detect the presence of this lesion; ×40 elastic Van Gieson.

Table 1. Clinical Duration of Dissection versus Occurrence of Aortic Infarction

<table>
<thead>
<tr>
<th>Group</th>
<th>Cases</th>
<th>Lesion present</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients dying suddenly or within 12 hours after dissection.</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Patients dying or operated on 12-48 hours after dissection.</td>
<td>6</td>
<td>2*</td>
</tr>
<tr>
<td>Patients dying or operated on 48 hours to three years after dissection.</td>
<td>20</td>
<td>19</td>
</tr>
<tr>
<td>Patients, age-matched, dying from other causes.</td>
<td>34</td>
<td>0</td>
</tr>
</tbody>
</table>

*The lesion is more focal in these cases (see text).
Discussion

Despite extensive literature concerning dissecting aneurysms, little has been published about aortic infarction as described in this paper. Braunstein, however, mentioned this lesion and recognized its ischemic nature. Acellular medial defects, more focal and much smaller than the lesion in this report, have also been described and have been related to hypertension and aging. Faria observed medial necrosis after shock similar to our findings in H/E preparations, but in contrast to the present observations, he noted concomitant degeneration of elastica, and regarded the lesions as an early phase of medial cystic necrosis. We have observed a necrotic lesion with maintained elastic laminae following prolonged shock in two cases of gram negative sepsis (authors' unpublished data).

Aortic infarction associated with dissecting aorta often occurs as an early phase of dissecting aneurysm. The false lumen is usually obliterated by thrombus, and the area of necrosis extends to the false lumen. The false lumen was obliterated by thrombus; × 40 hematoxylin and eosin. B) The elastic fibers are intact and well maintained. No progression to medial cystic necrosis has occurred; × 40 elastic Van Gieson.
aneurysm is characterized by midzonal medial necrosis which preserves both inner and outer medial zones. The pattern of necrosis noted here can be explained by recognizing that the inner zone is maintained by diffusion of nutrients from the true lumen, whereas the tissue bordering the dissection tract is maintained by blood flow through the dissection channel. The central zone is no longer supplied by the disrupted vasa vasorum and is thus infarcted. In our cases in which the false lumen is occluded with atheromata or thrombus, diffusion can not supply the media adjoining the dissection tract, and the zone of necrosis extends to the false lumen. In studying the aortas of various species, it has been found that by diffusion alone, nutrients can penetrate to a thickness of 29 lamellae, and that vasa vasorum are present only if the number of lamellar units exceeds 29.7 In the human abdominal aorta, there are only 28 lamellar units; the smaller arteries such as the carotids and iliacs have even fewer. However, the thoracic aorta consists of 58 lamellar units.8 Thus, the dependence of the thoracic aortic media on the vasa vasorum provides the explanation for the exclusive occurrence of aortic infarction in this segment.

The presence of this lesion provides a means of dating the age of a dissection. In other tissues, infarction is not recognizable by light microscopy in patients dying within 12 hours of acute aortic dissection. The earliest lesion occurs 12–48 hours after dissection and appears as focal areas of midzonal necrosis that progress to one large longitudinal central band of infarction, a finding observed in nearly all cases 48 hours or longer after dissection.

No inflammatory reaction or organization is seen in conjunction with these lesions. Cases with a history of a one-year-old dissection are similar histologically to those which are a few days old; the only difference we observed was flattening of the elastic laminae in the older lesions. Our oldest documented dissecting aneurysm, which lasted three years, showed an unorganized zone of necrosis extending to the false lumen, which was occluded by thrombus (fig. 3).

Histologically, the lesions of medial cystic necrosis and aortic infarction are distinctly different. With MCN, there is an increase in mucopolysaccharides and a disruption of the elastica, determined by Alcian blue and elastic stains.9 In aortic infarction, there is no increase in mucopolysaccharides and no disruption of the elastica. Aortic infarction is highlighted by the Gomori trichrome stain because the smooth muscle cells die, leaving intact the less oxygen-dependent fibrous and elastic fibers, both of which stain intensely with trichrome. The equal occurrence of MCN with and without aortic infarction also shows that the two lesions are independent of each other. Furthermore, the three cases in our series in which the lesion of aortic infarction was present years after a documented dissection provide evidence that this lesion does not evolve into MCN.

Our findings correlate well with dog studies in which ligation of intercostal arteries was carried out, thereby occluding vasa vasorum and inducing a midzonal medial necrosis indistinguishable from the lesion of aortic infarction described in this report.9 The lesion in dogs took approximately 48 hours to develop, was characterized by an absence of inflammation with preservation of elastic fibers, and showed no evidence of organization. At two weeks, some PAS staining material was noted at the inner margin of the midzonal necrosis, and it was concluded that this experimentally induced lesion might be related to medial cystic necrosis, in spite of their finding that the elastic laminae were preserved. In our cases of aortic dissection, which range up to three years in length, there was no increase in the amounts of PAS or Alcian blue material in or adjacent to the infarcted areas.

Current surgical therapy for aortic dissection is based on removing the site of intimal tear, obliterating
the false lumen, and inserting a synthetic tubular graft.\textsuperscript{10, 11} With blood no longer entering the false lumen, the dissection tract is decompressed and obliterated. The aortic infarction persists, however, and may even widen following this procedure, since after treatment no diffusion occurs from the false luminal side. The patient is left with a surgically repaired dissection, but with a thoracic aorta that has one-fourth to one-half or even more of its wall infarcted. One might suppose that the retained infarcted media would constitute a nidus for continuing or for future dissections, but our data provide no evidence for this. Consequently, the implication of this lesion in management remains to be determined.

Acknowledgments

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Platelet Aggregation in Aortic and Coronary Venous Blood in Patients With and Without Coronary Disease

3. Role of Tachycardia Stress and Propranolol

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with the technical assistance of Carol Burger, B.A.

SUMMARY We studied 16 patients with coronary artery disease (CAD) to evaluate platelet aggregation in blood samples withdrawn simultaneously from the aorta and coronary sinus. At rest, mean platelet aggregation in coronary venous blood was significantly lower than that in aortic blood. Platelet counts in coronary venous blood were also lower than in the aortic blood in each of the six CAD patients in whom counts were done. Platelet aggregation was lower in seven patients who were taking propranolol than in the remaining nine who were not taking propranolol. During tachycardia stress, platelet aggregation increased in all patients, but the magnitude of increase was greater in patients not taking propranolol. In four other patients without CAD, platelet aggregation and counts were also studied in the same fashion and were similar in both the aortic and coronary venous blood. These data suggest that in certain CAD patients, platelet consumption or destruction within atherosclerotic vasculature may occur. Propranolol may reduce platelet aggregation at rest and modify excessive aggregation during tachycardia stress in certain CAD patients.

ABNORMAL PLATELET FUNCTION has been reported in several ischemic disease states, such as coronary heart disease and cerebrovascular disease. Abnormal platelet function has also been found in association with hyperlipidemia, diabetes mellitus and hypertension, conditions that are “risk factors” in the development of ischemic syndromes. Animal studies suggest that extension of myocardial infarction may relate to excessive platelet aggregation and that induction of platelet aggregation may result in focal myocardial necrosis. Recently, increased formation of platelet aggregates in the coronary circulation of dogs with narrowed coronary arteries has been suggested as additional evidence implicating platelets in myocardial ischemia and necrosis.

The purpose of this study was to evaluate possible changes occurring in platelet aggregation as platelets traverse the coronary circulation in patients with coronary artery disease. Platelet aggregation studies were performed in aortic and coronary sinus blood samples from subjects with and without coronary disease. The influence of tachycardia stress on platelet aggregation was also assessed.

Materials and Methods

Patient Selection

Twenty patients, ages 46–64 years, were included in this study. Sixteen patients had angiographically documented coronary artery disease (CAD) defined as > 50% diameter narrowing. All of these CAD patients had typical stable angina pectoris ranging from one month to five years. Each had evidence for transient ischemia (electrocardiographic ST changes) on exercise by generally accepted criteria. These patients...
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