Spontaneous Laceration of Ascending Aorta

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SUMMARY
Spontaneous laceration of the ascending aorta may result in (1) through-and-through laceration causing hemopericardium, (2) limited intramural dissection of blood (incomplete dissecting aneurysm), or (3) classical dissecting aneurysm. The latter is the most common manifestation. Background conditions include either extensive cystic medial necrosis (as may be seen in Marfan's syndrome) or hypertension. The latter condition is universally associated with cystic medial necrosis but classically the medial lesion is of minimal degree. Aortic stenosis may be an underlying cause of spontaneous laceration of the ascending aorta.

Additional Indexing Words:
Aortic stenosis  Cystic medial necrosis  Dissecting aneurysm of aorta  Aortic aneurysm  Hypertension  Hemopericardium

Laceration of the wall of the ascending aorta may occur in the absence of infection or trauma. Spontaneous laceration involves the intima and, from case to case, varying thicknesses of the related underlying media. The consequences of such a laceration are one of three as follows: (1) simultaneous through-and-through laceration of the media and overlying adventitia, (2) limited intramedial dissection of blood (so-called incomplete dissecting aneurysm), or (3) classical dissecting aneurysm in which blood passes from the lumen of the aorta through the laceration into a plane of cleavage within the media (fig. 1).

In order to determine the relative incidence of the aforementioned three possible consequences of spontaneous laceration of the ascending aorta and to relate the laceration to underlying causes, a review was made of 56 autopsied cases. Cases of so-called congenital aortic sinus aneurysm were not included, nor were cases with a traumatic back-
ground for laceration. With the exception of one case, no examples of inflammatory disease of the aorta were included. In the exceptional case, a chronic inflammatory process of unknown etiology was present in the aorta.

The ages of the 56 patients ranged from 13 to 84 years, half being between 50 and 70 years of age. Among the 54 patients in whom the sex was known, 11 were female and 43 were male.

Before presenting our observations, it seems appropriate to define the ascending aorta anatomically. The ascending aorta runs from the origin of the vessel to the level of origin of the innominate artery. Two subdivisions may be recognized, the sinus and tubular portions. The sinus portion runs from the origin of the aorta to the level of the upper edges of the aortic cusps. That part of the aorta which receives the aortic commissures is considered part of the sinus portion. Between the sinus portion and the origin of the innominate artery lies the tubular portion of the ascending aorta.

**Effects of Spontaneous Laceration**

In all of the specimens, laceration of the aortic media was associated with laceration of the overlying intima. In 52 cases, the tear was in the

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**Figure 2**

*Recent spontaneous laceration of the ascending aorta.*

(a.) Transverse laceration (between arrows) at junction of sinus and tubular portions. (b.) Transverse circumferential laceration (between arrows) of lowermost portion of tubular part. (c.) Vertical laceration (between arrows) in lower portion of tubular part.

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tubular portion, usually at the lower part, and in the remaining four cases it involved the upper part of the sinus portion of the ascending aorta. In 52 cases, including the four involving the sinus portion, the tear was perpendicular to the long axis of the ascending aorta (figs. 2a, b). In four specimens, each involving the tubular part, the tear was parallel to the long axis of the ascending aorta (fig. 2c).

In a few cases, the tear involved the entire circumference of the aorta (fig. 2b) but, in the majority, less than half of the circumference was involved. In those cases involving the tubular portion, the tear was located relatively close to the aortic valve, usually in the segment within 4 cm of the junction of the sinus and tubular portions.

In cases in which death occurred less than several days following the tear, the edges were distinct, resembling a laceration that might have been made with a sharp instrument. In such cases, there was relatively little retraction of the edges of the tear from each other. When death occurred weeks or longer after the tear, there was considerable retraction of the edges of the laceration. The space between the retracted edges was gray and rough, and the edges of the tear were adherent to this tissue. Characteristically, atheromas were uncommon in relation to the laceration.

**Through-and-Through Laceration**

While complete or incomplete dissecting aneurysm may eventuate in rupture of the outer layers of the aorta, there were eight specimens which exhibited simultaneous through-and-through laceration of the aorta, the external laceration appearing to have occurred as part of the fundamental feature of the primary laceration and without dissecting aneurysm being present (fig. 3). Hemopericardium and sudden death were characteristic consequences.

Preexisting dilatation of the aorta was present in six of the specimens, and in the other two the ascending aorta was normal in appearance. Each of the six patients with preexisting dilatation had exhibited chronic aortic insufficiency and left ventricular hypertrophy. The aorta and aortic valve were normal in the other two specimens, and there was no clinical or necropsy evidence which suggested hypertension.

**Incomplete Dissecting Aneurysm**

Laceration of the intima and subjacent media without through-and-through laceration may be followed by varying degrees of intramedial dissection of blood. In some cases, classic dissecting aneurysms result. In others, there is no significant extravasation of blood into the media. If, following the underlying laceration no significant intramural dissection occurs, the process may be termed "incomplete dissecting aneurysm." Following the laceration, the edges retract and the "bare area" is filled in with a neointima composed primarily of fibrous tissue which becomes covered by endothelium (fig. 4).

The base of the bare area, which usually contains some medial tissue, may show localized aneurysmal dilatation. Incomplete dissecting aneurysm of the ascending aorta by itself is a relatively benign condition but it may be associated with aortic insufficiency. In our series of 56 cases involving laceration of the ascending aorta there were 12 examples of incomplete dissecting aneurysm of which four were incidental findings pathologically. In the remaining eight cases, however, aortic insufficiency was a dominant clinical manifestation.
Aortic insufficiency associated with incomplete dissecting aneurysm may either precede the aortic laceration (on the basis of preexisting aortic dilatation) or it may result from the laceration. The laceration may cause loss of commissural support (fig. 5) and/or be associated with laceration or distortion of one or more aortic cusps, as previously described.1

**Classic (Complete) Dissecting Aneurysm**

In contrast to the laceration followed by a limited degree of intramedial dissection of blood characteristic of incomplete dissecting aneurysm is classic or "complete" dissecting aneurysm. Classic dissecting aneurysm is characterized by extensive intramedial dissection of blood. The dissection may involve the entire length of the aorta and may also extend into aortic branches causing obstruction of those so involved (fig. 6a). Among the other consequences is loss of commissural support of the aortic valve resulting in aortic insufficiency (fig. 6b), as may occur in the incomplete type. The most common complication of those dissecting aneurysms that begin in the ascending aorta is secondary rupture of the intima and superficial part of the media has ruptured (arrows) leaving exposed a bare area which lies upon the remaining media (M). A neointima has formed to line the bare area. (b.) Left ventricle and ascending aorta showing an old incomplete dissecting aneurysm. The edges of the laceration (arrows) have retracted exposing a bare area (B.A.) which is covered by neointima.

![Image 5](http://circ.ahajournals.org/)

**Figure 5**

Incomplete dissecting aneurysm of ascending aorta. The edges (arrows) of the laceration have retracted somewhat. The lower edge of the laceration involves a commissural area so that the related cusps of the aortic valve (A.V.) have prolapsed resulting in aortic insufficiency.
the outer wall of the false passage leading to hemopericardium (fig. 6c). In contrast, when the primary laceration is more distant, as in the upper descending aorta, external rupture is less common. With time, such cases, in addition to showing a “double-barreled aorta,” may exhibit a saccular aneurysm corresponding with the position of the primary laceration.

If external rupture does not occur and the patient survives, a saccular aneurysm may develop in the thin outer wall of the aorta opposite the internal tear \(^2\) (fig. 6d). The false channel within the media may acquire a neointima and may continue as a functional channel along with the natural aorta. In some cases, the false channel may be the wider of the two in the so-called “double-barreled aorta.” The lining of the false passage may develop atherosclerosis and the latter lesion may calcify.

Among the 56 cases studied with a spontaneous laceration of the ascending aorta there were 36 cases (64%) of classic or complete dissecting aneurysm. Thirty-two of the patients died within hours or days after the onset, while in four cases the dissecting aneurysm was healed. In each of the latter, chronic aortic valvular insufficiency was a complication.

External rupture of the aorta was seen only among those patients dying shortly after the onset, this lethal complication being seen in 19 cases. In 17, the rupture occurred at the same level as the internal tear leading to hemopericardium while, in two, hemothorax resulted. In one of the latter, the rupture was into the left pleural cavity and, in one, into the right hemothorax. The latter location for hemorrhage is highly uncommon in dissecting aneurysm.

Signs of aortic insufficiency had been exhibited clinically by 12 of the patients with complete dissecting aneurysm, in four examples of healed dissecting aneurysm (fig. 7), and in eight patients who died during the acute phase.

Classically, the intramural hematoma of the aorta may extend into the walls of various branches and yield particular clinical manifestations.\(^3,4\) In branches, the lumen is narrowed by the intramural hematoma (figs. 8-11). Rupture of the hematoma may occur into the arterial lumen beyond the obstruction to reconstitute flow of blood to the involved organ, a process which has been termed “rerupture” (fig. 11b). The rerupture may be circumferential. In the latter situation, if the
involved vessel is large, the circumferential rerupture may be associated with prolapse of the tissues so as to obstruct the lumen (fig. 11c) or the distal segment may be fed through the false passage (fig. 11d).

Any of the branches of the aorta may be involved by the dissecting hematoma but there is a particular tendency which may be related to the curves of the aorta. The ascending aorta, the arch, and the descending aorta form an arc with a wider outer curve and a more narrow inner curve. As blood is ejected into the media through the aortic laceration, it tends to proceed along the wider and outer curve of the aorta so that branches dominantly involved arise from the outer curve (fig. 12). Thus, in the ascending aorta, the right coronary artery tends to be involved more commonly than the left (figs. 13a, b). The branches of the arch arise from the outer aortic curve and are susceptible to involvement (fig. 13c). In the descending aorta, the left intercostals (fig. 13d) are more commonly affected than the right intercostals. Sparing of the intercostal arteries on one side (usually the right) serves to provide the spinal cord with adequate nourishment. Involvement of both sets of intercostal arteries underlies the uncommon complication of dissecting aneurysm in the form of infarction of the spinal cord.  

In the abdominal aorta, the left renal artery is commonly involved, as is the left common iliac artery. While the foregoing indicates a distinct tendency, variations occur (fig. 14).
Intramural hemorrhages in dissecting aneurysm: internal carotid artery. The hematoma causes some distortion of the lumen (L.).

**Background Conditions**

Spontaneous laceration of the ascending aorta may be considered an expression of weakness of the aorta relative to the intraarterial pressure. Therefore, laceration may be a manifestation either of primary weakness of the aorta or of excessive pressure. While the two conditions may be associated, usually one is clearly dominant. With the exception of one of our 56 cases of laceration of the ascending aorta, cystic medial necrosis of some degree was present in each. In the exceptional case, an inflammatory condition of unknown etiology was present.

Cystic medial necrosis was graded histologically into four grades of severity in conformity with the classification of Carlson and associates. In this,
grade I represents just barely visible change while in grade IV there is major interruption of elastic elements of the media. The grades of cystic medial necrosis were correlated with age, sex, and blood pressure. There was no particular pattern characteristic of either sex.

In general among patients with spontaneous laceration of the ascending aorta, there was an inverse relationship between age and the grade of cystic medial necrosis.

 Thirty-two patients manifested grade I or II cystic medial necrosis and, with the exception of three subjects, each was over 40 years of age. Grade III cystic medial necrosis was observed in 13 subjects, eight being under 40 years of age. Of the eight subjects showing grade IV change, five were less than 40 years old.

 Five of the patients among the 56 studied manifested features of Marfan's syndrome (four male: one female). Each was in the group with either grade III or IV cystic medial necrosis.

Hypertension was identified in 22 of the 55 cases with cystic medial necrosis of some degree. It was associated with grade I and grade II lesions in 18 instances and with grade III in only four instances. In no instance did grade IV cystic medial necrosis and hypertension coexist. With the exception of a single case, each of the hypertensive patients was over 40 years of age.

Specimens from patients with and without hypertension were compared according to the severity of cystic medial necrosis. Grade I and II lesions were present in approximately equal numbers in the hypertensive and normotensive groups. Grade III and grade IV lesions were much more frequent in normotensive than hypertensive patients (fig. 15).

Manley compared the histologic appearance of the ascending aorta in 27 cases of dissecting

**Figure 11**
Classic dissecting aneurysm. Variations in extension of the intramural hematoma into an arterial branch. (a.) The hematoma has caused major narrowing of the lumen. (b.) The hematoma has ruptured into the lumen so that beyond the obstruction flow is reconstituted. (c. and d.) Circumferential laceration. In c, the distal edges of the circumferential laceration in the branch have prolapsed in such a way as to obstruct flow into the vessel beyond. In d, the edges of the laceration have retracted toward the wall of the vessel so that the false passage feeds the vessel.

**Figure 12**
Classic distribution of intramural hematoma in dissecting aneurysm of the aorta. The hematoma tends to occupy the outer and larger curve of the aorta so that the branches which are particularly susceptible to involvement include the right coronary artery, the branches of the arch, the left intercostals, the left renal artery, and the left common iliac artery. The variations from this classic distribution, however, occur.
(a. and b.) Limited dissecting aneurysm of ascending aorta showing occlusion of the left main coronary artery. (a.) The edges of the laceration are identified by the arrows. (b.) The aorta below the laceration has been retracted to expose the false passage which has extended into the wall of the left coronary artery (L.C.). (c.) The aortic arch. Within the wall of each branch is a hematoma (H.) causing various degrees of compression of the lumen. I.A. = innominate artery; L.C. = left coronary artery; L.S. = left subclavian artery. (d.) Interior of false passage within descending aorta. Arrows indicate sites of acutely left intercostal arteries.

aneurysm with 27 age- and sex-matched normotensive controls and 27 age- and sex-matched hypertensive controls. Elastic fragmentation was seen to be of similar degree in each. There was a tendency for subjects with dissecting aneurysm to show more metachromasia of the ground substance than was apparent in the two control groups.

Our observations support the concept that spontaneous laceration of the ascending aorta results primarily either from the weakness of
extensive cystic medial necrosis or from excessive arterial pressures. While cystic medial necrosis was seen in all hypertensive patients, the degree of such involvement was usually minor. Only in exceptional cases were both hypertension and significant degrees of cystic medial necrosis present. Atherosclerosis did not appear to be a significant factor in the cause of laceration. Forty-one specimens manifested atherosclerosis of the ascending aorta of varying degrees of severity. In 21 specimens, atherosclerosis was associated with cystic medial necrosis without hypertension. Nineteen patients with hypertension and cystic medial necrosis showed atherosclerosis of the ascending aorta, and the single specimen with aortitis also exhibited atherosclerosis. In three instances, laceration appeared to be initiated in relation to atheromatous foci. In these cases, normal as well as atheromatous intima was involved in the site of laceration.

Coarctation of the aorta is commonly recognized as being potentially complicated by laceration of the aorta and dissecting aneurysm. Less commonly identified as a background for laceration of the ascending aorta is aortic stenosis. Each of these conditions was represented among our cases.

Of the 56 examples of spontaneous laceration of the ascending aorta, aortic stenosis was present in five, in one of which coarctation had also been present and repaired. Coarctation of the aorta was a condition in three patients, one of which was the aforementioned one in which aortic stenosis was also present. In the other two, no aortic stenosis was present.

In each of the patients with coarctation (age ranges 11–29 years), the coarctation had been

Figure 14
Abdominal aorta viewed from front. The intramural hematoma (F.P.) is partially shown. There has been extension into both the left (L.I.) and right (R.I.) common iliac arteries of the hematoma, but more pronouncedly into the right. Each renal artery (R.R. and L.R.) is involved and is shown in greater detail in b. (b.) The hematoma (F.P.) within the media of the aorta has extended into each renal artery. In the right artery (R.R.), the hematoma has narrowed the lumen, while in the left renal artery there has been circumferential “rerupture.”

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resected within 2 years of the time of laceration of the aorta. In each, the postoperative clinical picture was that of successful repair of the coarctation.

Among the five subjects with aortic stenosis, complete dissecting aneurysm resulted from the laceration in three (one of the cases being the one with coexistent aortic stenosis and coarctation), incomplete dissecting aneurysm with saccular aneurysm in one, and through-and-through rupture of the aorta with hemopericardium in the fifth.

Of the two patients with coarctation but without aortic stenosis, one showed through-and-through laceration of the aortic wall resulting in hemopericardium, and the other, complete dissecting aneurysm and complicating hemopericardium. The four patients with isolated aortic stenosis ranged in age from 42 to 78 years, while the three patients with coarctation of the aorta ranged in age from 11 to 29 years.

Among the four patients with isolated aortic stenosis, the stenosis resulted from calcification of a congenital bicuspid aortic valve in two (fig. 16), while in the other two the stenosis was fibrous, resulting from rheumatic endocarditis. In the patient with coarctation and aortic stenosis, the latter was of the congenital unicommissural type.

References

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