CASE REPORT

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Traumatic False Aneurysm of Descending Aorta with Aortoesophageal Fistula

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ABSTRACT: We report a case of traumatically induced false aneurysm with secondary bacterial aortitis of the descending aorta and fistula formation between the aorta and esophagus. This lesion was diagnosed at autopsy and caused death in a 68-year-old female 19 years after an automobile accident. Aortoesophageal fistula (AEF) formation as a result of any cause is rare. To our knowledge, this is the second reported case of traumatic aortic aneurysm as a result of an automobile accident with subsequent AEF formation.

KEYWORDS: pathology and biology, cardiovascular system, aortoesophageal fistula, motor vehicle accidents, aneurysms

Aortoesophageal fistula (AEF) formation is an uncommon event; only 109 cases have been reported in the English literature since 1916 [*1-38*]. In the majority of cases, fistula formation is secondary to a thoracic aneurysm, usually syphilitic or atherosclerotic in origin. Rarer causes of AEF include esophageal carcinoma [*5,7,14,28,32,37,38*], tuberculosis [*8,17,24*], and swallowed foreign bodies [*3,4*]. Only 3 cases of AEF secondary to a traumatic aortic aneurysm have been reported: 1 from shell fragments [*23*], another from gunshot injury [*36*], and a third from a motor vehicle accident (MVA) with chest injury [*12*]. We report the fourth fatal case of AEF as a result of traumatic aortic aneurysm and the second as a result of an MVA.

Report of Case

Clinical

A 68-year-old white female with a normal habitus was reported to be well until three months before death when she visited her physician complaining of chest pain and heartburn. Physical examination, electrocardiogram, and chest roentgenograms were normal. The pain was diagnosed as musculoskeletal, and the patient was prescribed nonsteroidal

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anti-inflammatory agents. The medicine was without effect and was soon discontinued. Her chest pain continued, and minutes before death she became acutely agitated, vomited large quantities of fresh blood, and died as a result of exsanguination.

After an autopsy was performed, further information revealed that 19 years prior to death she was involved in a "head-on" MVA with an impact speed of greater than 60 mph (96 km/h). Her injuries were not well documented, but she was known to have fractured her right radius.

Autopsy Findings

Approximately 8 cm distal to the ligamentum arteriosum the anterior, medial portion of the aorta had a 3- by 2-cm rectangular perforation with smooth, rolled edges (Fig. 1*a*). This opened into an aneurysmal sac (Fig. 2), which in turn communicated with the esophagus. The esophageal perforation was a 2- by 1-cm oval filled with thrombus (Fig. 1*b*). The esophagus and aorta were closely applied (2 cm at the closest point) and tightly adhesed together in the area surrounding the fistula. The fistula had approximate greatest dimensions of 4 by 3 by 2 cm. The degree of atherosclerosis present at this level of the aorta was minimal (Fig. 1*a*). The aorta and esophagus showed no evidence of other traumatic injury, syphilis, carcinoma, or foreign body by gross examination or specimen radiography. The stomach, duodenum, and jejunum were filled with fresh blood. Microscopically, the fistulous tract (aneurysmal sac wall) was lined by sequential layers of recent thrombus, acute inflammatory

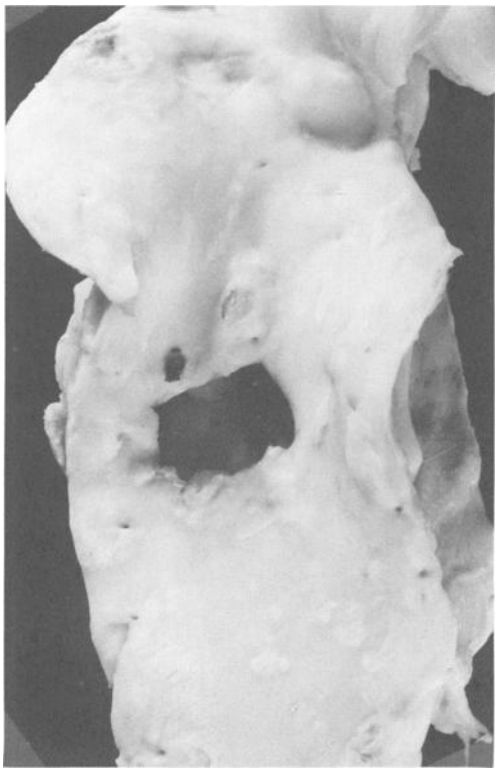

FIG. 1*a*—*Intimal surface of thoracic aorta demonstrating rectangular perforation and few atherosclerotic streaks.*

FIG. 1*b*—*Mucosal surface of esophagus demonstrating oval perforation in esophagus plugged with thrombus.*

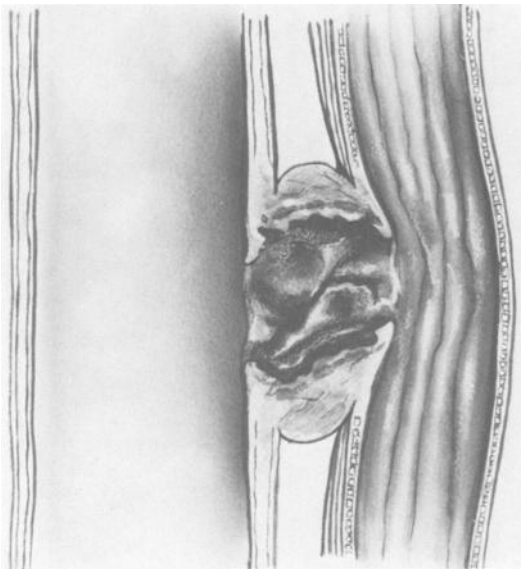

FIG. 2—*Idealized drawing of longitudinal section through esophagus* (right), *aorta* (left), *and false aortic aneurysm* (middle) *with esophageal perforation comprising the aortoesophageal fistula.*

cells, reactive fibroblasts, organizing thrombus, and fibrosis (Fig. 3). The latter adhesed the aorta and esophagus together to a focal area of the left pleura. Components of normal aortic wall were not present in the fistula tract. Microabscesses were noted in and near the aneurysmal sac and the aortic wall (Figs. 3 and 4). The acute inflammation extended from the false aneurysm wall to dissect the media of the aorta at the edge of its perforation (Fig. 4). The esophagus was histologically normal (Fig. 5) in areas not affected by the perforation or the surrounding fibrosis.

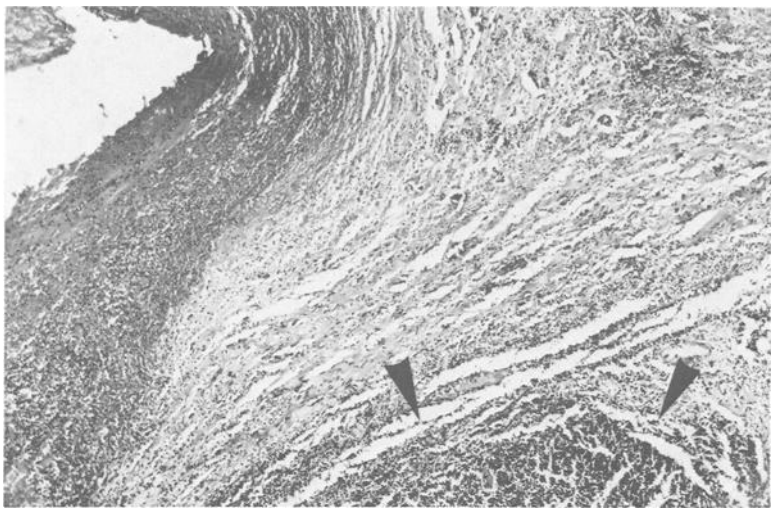

FIG. 3—*Wall of aneurysm* (lumen to the left at top) *illustrating thrombus, acute inflammation with focal abscess formation* (arrowheads) *and fibrosis. (Hematoxylin and eosin, ×50).*

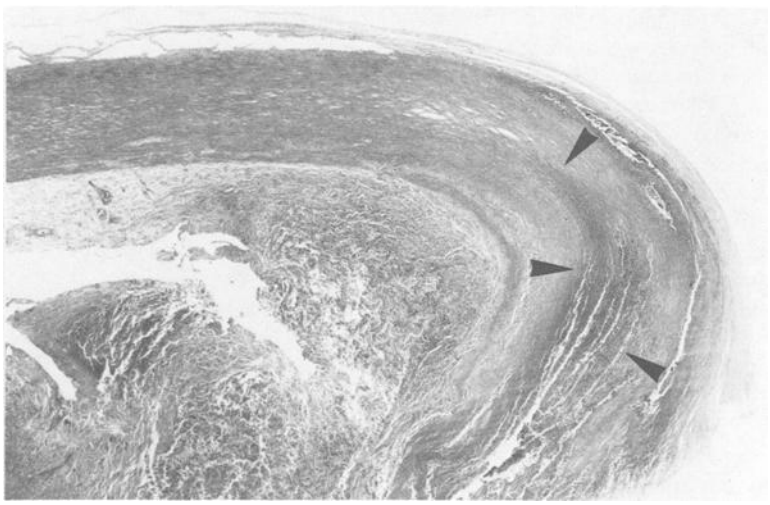

FIG. 4—*Essentially normal aortic wall in continuity with perforation and entrance to aneurysm. Arrowheads demonstrate dissection of aortic media by acute inflammation extending from aneurysm wall. (Hematoxylin and eosin, ×12.5).*

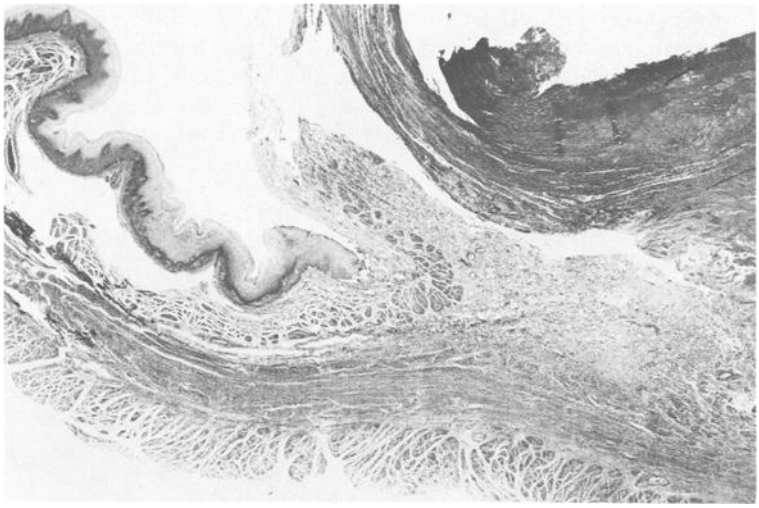

FIG. 5—*Normal esophagus in continuity with esophageal perforation filled by thrombus. (Hematoxylin and eosin, ×12.5).*

Tissue gram stain showed multiple clumps of bacterial organisms in the acutely inflamed areas, most prominently at the advancing inflammatory border of the contiguous aortic wall. The organisms were oval to round gram positive cocci in pairs and chains compatible with streptococci. A source of the bacteria was not identified at autopsy: endocarditis was notably absent. Similarly, there was no microscopic evidence of syphilis or cystic medial necrosis.

Additional findings included rare hepatic microabscesses, most likely a result of embolic spread from the infectious process in the aorta.

Discussion

Although gross and histologic examination of a longstanding false aortic aneurysm does not permit one to assign absolutely previous trauma as the etiology, we believe that the history of a previous automobile accident and the lack of evidence to support another etiology makes trauma the most likely cause for this aneurysm. We are unable to support a diagnosis of primary mycotic aneurysm with aortitis since there is no preexisting disease of the aorta nor is there any infection surrounding the aorta [*25*]. Secondary abscess formation in association with AEF has been reported elsewhere [*5,12,16,23,25,33,35*], however. Bacteria in the acutely inflamed areas of these lesions were not uniformly present. The bacterial seeding of the previously traumatically damaged aorta in the present case is most likely due to a transient bacteremia of unknown cause.

This case presents an aggregation of unusual findings. Whereas the majority of traumatic aneurysms occur near the isthmus [*39*], the area of maximal shear during deceleration injury, the present case, like two reported by Bennett and Cherry [*39*], occurred in the descending thoracic aorta. Another notable component is the exceedingly long interval between injury and clinical presentation. Our patient died 19 years after initial injury, similar to the case of Fleming et al. [*40*] which presented 21 years later. Also, virtually all reported aneurysms of traumatic origin have been radiographically evident before death. In only one other reported case of chronic traumatic thoracic aneurysm has normal chest roentgenographic findings been reported at the time of diagnosis [*1*].

Of the 109 cases of AEF reported in the English literature since 1916, only 1 of these is

similar to ours. In 1962, Garamella and colleagues [*12*] reported the only other case of chronic thoracic traumatic aneurysm caused by an MVA with eventual AEF formation. As with the current case, the aneurysm became clinically evident many (8) years following a car accident. It also was grossly adherent to surrounding tissues and had associated abscess formation and mild concomitant aortic atherosclerosis.

In summary, we illustrate a traumatic pseudoaneurysm of the descending thoracic aorta which became infected 19 years after its formation with resultant AEF formation. As is often the case in AEF, death soon followed as a result of exsanguination with the diagnosis established by autopsy. This case suggests that AEF caused by traumatic pseudoaneurysm may be underreported, since the pathologic findings are not specific and the interval between trauma and formation of the lethal fistula may be exceedingly long. Therefore, as presented here, a history of trauma should be sought in unexplained AEF in an attempt to establish the etiology of the aneurysmal component. If trauma is determined to be the cause of the aneurysm, then death as a result of AEF is traumatic even if delayed. In turn, the manner of death is not natural, but by definition either accidental, suicidal, or homicidal.

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