

Penetrating Atherosclerotic Ulcer of the Aorta: Imaging Features and Disease Concept¹

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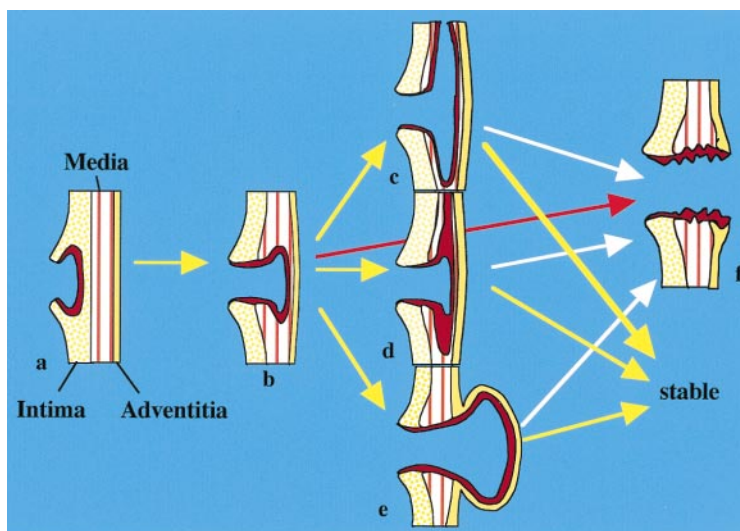
Penetrating atherosclerotic ulcer is an ulcerating atherosclerotic lesion that penetrates the elastic lamina and is associated with hematoma formation within the media of the aortic wall. This pathologic condition is distinct from classic aortic dissection and aortic rupture; however, care should be taken in making the diagnosis, particularly if the disease is discovered incidentally. At computed tomography (CT), penetrating atherosclerotic ulcer manifests as focal involvement with adjacent sub-intimal hematoma and is often associated with aortic wall thickening or enhancement. Magnetic resonance imaging is superior to conventional CT in differentiating acute intramural hematoma from atherosclerotic plaque and chronic intraluminal thrombus and allows unenhanced multiplanar imaging. Spiral CT involves shorter examination times and allows high-quality two- and three-dimensional image reconstruction. CT angiography can demonstrate complex spatial relationships, mural abnormalities, and extraluminal pathologic conditions. Transesophageal echocardiography has been reported to be highly sensitive and specific in the differentiation of aortic disease, and intravascular ultrasonography may also be useful in this setting. Although rupture or other life-threatening complications are rare, patients with penetrating atherosclerotic ulcer must be followed up, particularly during the 1st month after onset. Surgical treatment may become necessary in cases involving evidence of intramural hematoma expansion, signs of impending rupture, inability to control pain, or blood pressure changes.

Index terms: Aneurysm, aortic, 56.731 • Aorta, diseases, 56.731, 56.74, 56.75 • Aorta, dissection, 56.74 • Arteriosclerosis, 56.75

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Figure 1. Course of atheromatous ulcer. Although these ulcers occur in patients with advanced atherosclerosis, they are usually asymptomatic and confined to the intimal layer (a). Penetrating atherosclerotic ulcer is defined as a deep atheromatous ulcer that penetrates through the elastic lamina and into the media (b). Penetrating atherosclerotic ulcer can lead to aortic dissection (c, d), aortic aneurysm (e), or rupture (f). Yellow arrows indicate course of penetrating atherosclerotic ulcer that is stabilized with appropriate treatment, white arrows indicate course of rupture of aortic dissection and aortic aneurysm, and the red arrow indicates course of spontaneous aortic rupture.



Introduction

Penetrating atherosclerotic ulcer of the aorta was first described as a distinct clinical and pathologic entity by Stanson et al (1) in 1986. The condition is characterized by ulceration that penetrates through the elastic lamina and into the media and is associated with a variable amount of hematoma within the aortic wall (1,2). Since 1986, several articles have appeared describing the imaging appearances, prognosis, and management of this disease entity (1–9).

Penetrating atherosclerotic ulcer has been described as a process involving the thoracic aorta that is distinct from aneurysm and classic aortic dissection (1–5). However, distinguishing between these disease entities is sometimes difficult, and there seems to be some confusion regarding the concept of penetrating atherosclerotic ulcer.

In this article, we discuss and illustrate the concept of penetrating atherosclerotic ulcer of the aorta as well as its course, imaging appearances, prognosis, and management.

Disease Course

Penetrating atherosclerotic ulcer refers to an ulcerating atherosclerotic lesion that penetrates the elastic lamina and is associated with hematoma formation within the media of the aortic wall (1–3).

Initially, atheromatous ulcers develop in patients with advanced atherosclerosis. At this stage, the lesions are usually asymptomatic and confined to the intimal layer (3). In the next stage, the lesion progresses to a deep atheromatous ulcer that penetrates through the elastic lamina and into the media (ie, penetrating atherosclerotic ulcer) (Fig 1).

Hematoma formation may extend along the media, resulting in either “double-barreled” or “thrombosed” aortic dissection (Fig 1). Double-barreled aortic dissection demonstrates communication between the true and false lumina, whereas thrombosed aortic dissection shows no opacification of the false lumen. Thrombosed aortic dissection is probably more common than double-barreled aortic dissection because severe atherosclerosis is believed to prevent the extension of hematoma and the creation of reentry (10).

In some cases, a disease entity that has been identified as penetrating atherosclerotic ulcer would be more accurately described as aortic dissection, which is defined as the penetration of the circulating blood into the wall of a vessel for a varying distance (11).

In some cases, hematoma extension causes stretching of the weakened aortic wall, leading to the formation of a saccular aortic aneurysm. The aortic aneurysm and dissection may eventually rupture (Fig 1).

Spontaneous rupture of the thoracic descending aorta is a rare condition that occurs in the absence of a true aneurysm (12–15). Most cases involve predisposing conditions such as hypertension and atherosclerosis. The precise mechanism of spontaneous rupture is not well understood. However, Castleman and McNeely (12) have speculated that there may be pressure atrophy of the media due to overlying intimal atherosclerotic plaque with localized ballooning of the aortic wall prior to perforation. Most spontaneous aortic ruptures are believed to be associated with perforation through the atheromatous plaque (Fig 1) (12–15).

Imaging Appearances

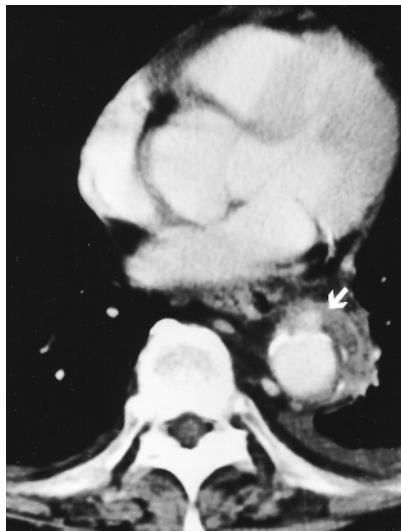
Because penetrating atherosclerotic ulcer, classic aortic dissection, and aortic rupture have similar clinical manifestations, they must be distinguished radiologically. Although angiography was previously considered the standard of reference for the diagnosis of many aortic diseases (16), it has largely been replaced by computed tomography (CT) and magnetic resonance (MR) imaging (4,5,17).

CT features of penetrating ulcers include focal involvement with adjacent subintimal hematoma located beneath the frequently calcified and inwardly displaced intima in the middle or distal third of the thoracic aorta. The ulcer is often as-

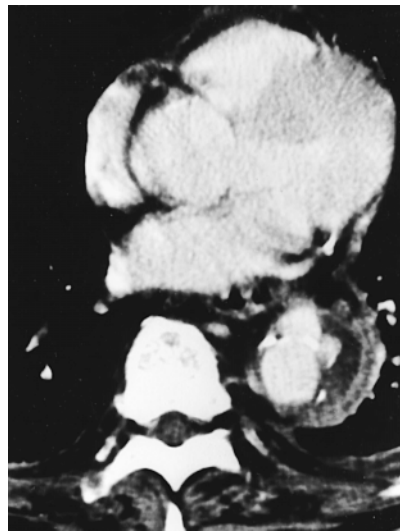
sociated with thickening or enhancement of the aortic wall (Figs 2–4) (4). Yucel et al (5) demonstrated that MR imaging is superior to CT in differentiating acute intramural hematoma from atherosclerotic plaque and chronic intraluminal thrombus. MR imaging has the additional advantage of allowing multiplanar imaging without use of contrast material (Fig 4). Transesophageal echocardiography has been used successfully and has been reported to be highly sensitive and specific in the differentiation of aortic disease (17). Vilacosta et al (9) described penetrating atherosclerotic ulcer as a craterlike or focal outpouching in the atherosclerotic wall that occurs in the middle to distal portion of the descending thoracic aorta.

We generally use spiral CT to evaluate aortic disease because of the shorter examination times. Moreover, high-quality two- and three-dimensional image reconstructions are possible with this modality. Spiral CT may not depict small, penetrating atherosclerotic ulcers because of its lower spatial resolution compared with conventional angiography. However, CT angiography can demonstrate complex spatial relationships, mural abnormalities, and extraluminal pathologic conditions, which may offset this weakness (18).

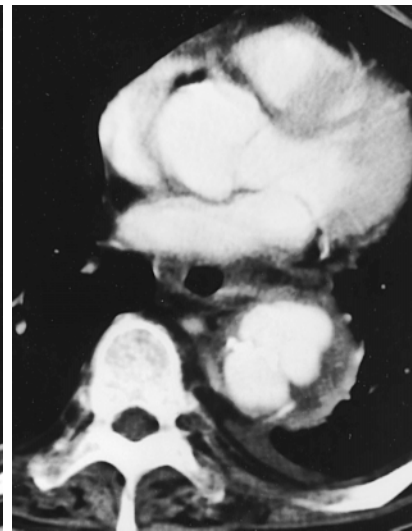
Atheromatous ulcers that are confined to the intimal layer sometimes appear radiologically similar to penetrating atherosclerotic ulcers. Therefore, care should be taken in making a diagnosis of penetrating atherosclerotic ulcer, particularly if the disease entity is discovered incidentally. Intravascular ultrasonography, which can help accurately assess the aortic layer, may be useful in the evaluation of penetration by atheromatous ulcers (19).



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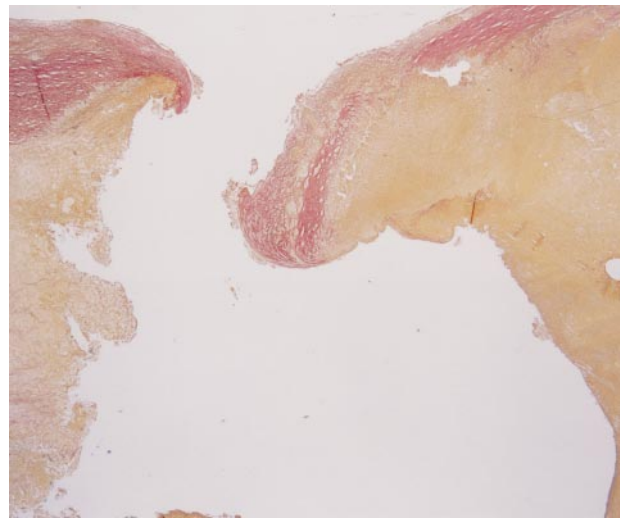
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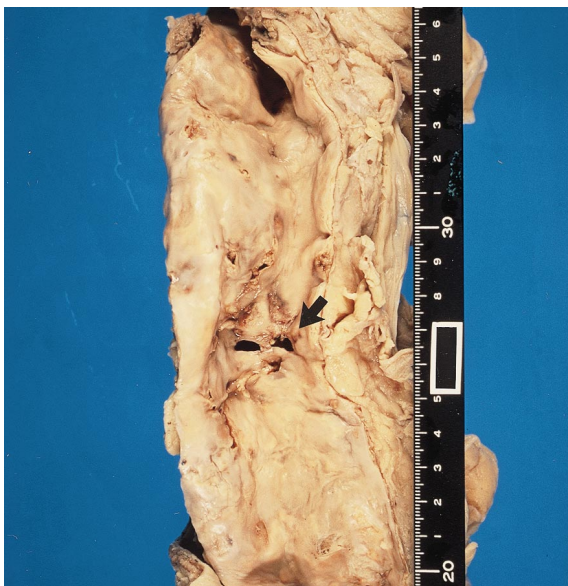
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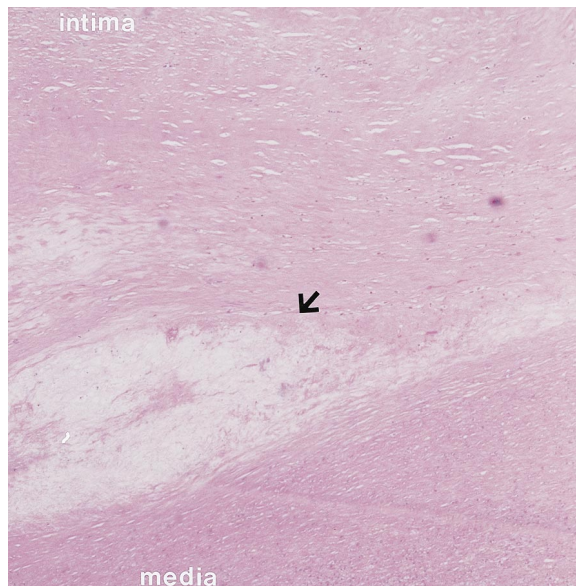
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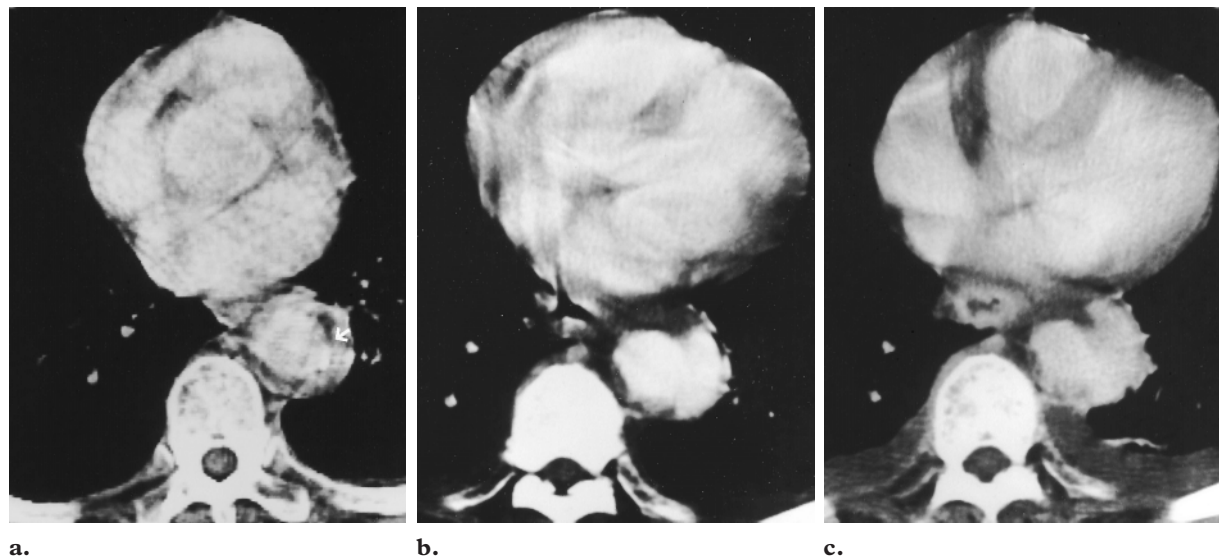
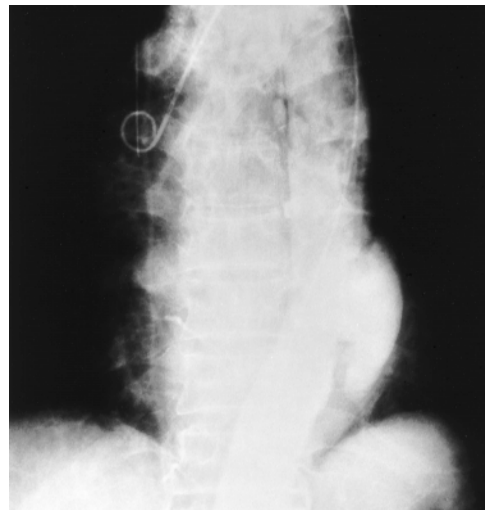


Figure 3. Case 12. Penetrating atherosclerotic ulcer in a 74-year-old man with sudden onset of chest pain. **(a)** Contrast-enhanced CT scan shows a small penetrating ulcer in the descending thoracic aorta (arrow) with intramural hematoma. The patient was treated conservatively. **(b, c)** Follow-up CT scans obtained 7 **(b)** and 21 **(c)** days after onset show gradual enlargement of the penetrating atherosclerotic ulcer and gradual reduction of the hematoma. **(d)** Thoracic aortogram clearly delineates the extent of the ulcer. The patient underwent surgery, the results of which confirmed penetrating atherosclerotic ulcer.



d.

Figure 2. Case 7. Ruptured penetrating atherosclerotic ulcer in a 73-year-old man with terminal laryngeal cancer and sudden onset of chest pain. **(a)** Contrast material-enhanced CT scan shows a penetrating ulcer in the descending thoracic aorta with surrounding intramural hematoma (arrow). The patient was treated conservatively. **(b, c)** Follow-up CT scans obtained 14 **(b)** and 35 **(c)** days after onset show gradual enlargement of the penetrating atherosclerotic ulcer and hematoma. **(d)** Multiplanar reconstructed image clearly demonstrates the extent of the penetrating atherosclerotic ulcer and intramural hematoma. Surgical treatment was not attempted, and the patient died 36 days after onset with severe hematemesis due to an aorto-esophageal fistula. **(e)** Photograph of the autopsy specimen clearly depicts ulceration (arrow) and intramural hematoma extending to the esophageal wall. Scale is in centimeters. **(f)** Low-power photomicrograph (original magnification, $\times 35$; elastica van Gieson stain) demonstrates atheromatous ulcer penetrating the media and intramural hematoma extending under the thickened intima. **(g)** High-power photomicrograph of the unruptured portion of the ulcer (original magnification, $\times 175$; hematoxylin-eosin stain) demonstrates an intracellular lipid in the thickened intima. Extracellular lipid deposition is seen in the underlying intima (arrow).

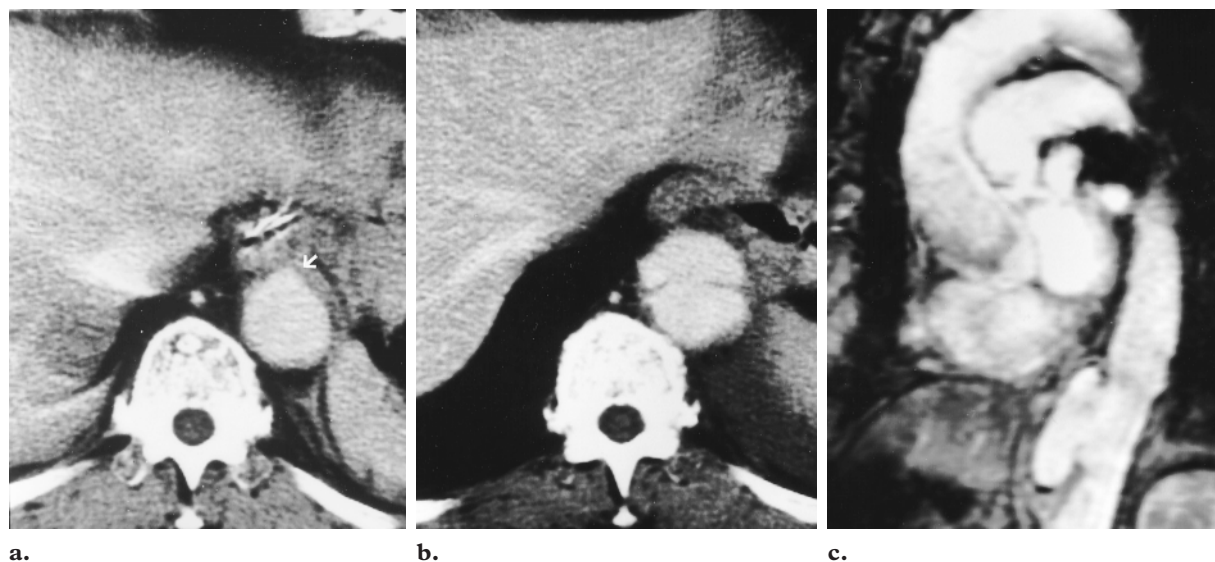


Figure 4. Case 5. Penetrating atherosclerotic ulcer in a 73-year-old man with back pain. **(a)** Contrast-enhanced CT scan shows a penetrating ulcer in the descending thoracic aorta (arrow) with intramural hematoma. The patient was treated conservatively. **(b)** Follow-up CT scan obtained 48 days after onset shows enlargement of the penetrating atherosclerotic ulcer and reduction of the intramural hematoma. **(c)** Oblique sagittal cine MR image obtained 3 months after onset clearly demonstrates the extent of the penetrating atherosclerotic ulcer. The ulcer has a smooth edge and has not changed in size (cf **b**). Over 5 years later, the patient remains asymptomatic and the penetrating atherosclerotic ulcer remains unchanged in size.

Although penetrating atherosclerotic ulcers typically involve the descending thoracic aorta, they can also involve the abdominal aorta, which is sometimes more severely atherosclerotic (Fig 5).

Multiple penetrating atherosclerotic ulcers may also be seen. Some of these ulcers may develop after the extension of hematoma, presumably secondary to weakening of the intimal layer (Fig 6).

Prognosis

Little is known about the natural history of penetrating atherosclerotic ulcer. This disease entity has been considered by most authors to have a

poorer prognosis than classic aortic dissection (1–3,6,7). However, Harris et al (8) reported that disease progression is slow, with a low prevalence of acute rupture or other life-threatening complications. We believe that this discrepancy can be explained by differences in patient selection. For example, in the study by Stanson et al (1), 13 of 16 patients presented with symptoms of back or chest pain, whereas in the study by Harris et al (8), only four of 18 patients presented with these symptoms. Furthermore, if many incidental cases of penetrating atherosclerotic ulcer are included, the survival rate for affected patients may increase. Pain is believed to be one of the most important variables in determining the appropriateness of surgical intervention (1).

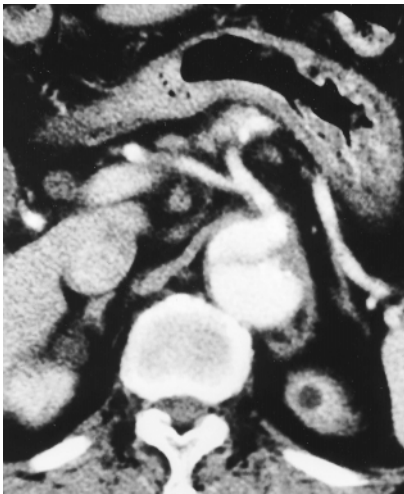
Figures 5, 6. **(5) Case 1.** Penetrating atherosclerotic ulcer in the abdominal aorta in a 73-year-old man with back pain. **(a)** Contrast-enhanced CT scan shows a penetrating ulcer in the abdominal aorta (arrow). The patient was treated conservatively. **(b, c)** On follow-up contrast-enhanced CT scans obtained 2 weeks **(b)** and 2 years **(c)** after onset, the size of the penetrating atherosclerotic ulcer is virtually unchanged (cf **a**). The celiac trunk is seen originating from the false channel. **(d)** Maximum-intensity-projection image obtained 2 months after onset clearly demonstrates the penetrating atherosclerotic ulcer and severe calcification of the aorta. **(6)** Multiple penetrating atherosclerotic ulcers in a 73-year-old man with back pain. **(a)** Unenhanced CT scan shows high-attenuation intramural hematoma in the descending aorta. **(b)** Follow-up contrast-enhanced CT scan shows a penetrating aortic ulcer with intramural hematoma. **(c)** Follow-up contrast-enhanced CT scan obtained 2 weeks after onset demonstrates multiple penetrating atherosclerotic ulcers in the descending thoracic aorta (arrows). **(d)** Maximum-intensity-projection image clearly demonstrates multiple ulcers (arrows). (This case was not included in the Table because the follow-up period was not long enough to allow prediction of the prognosis.)



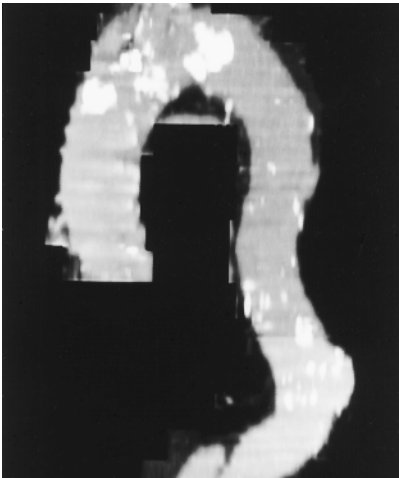
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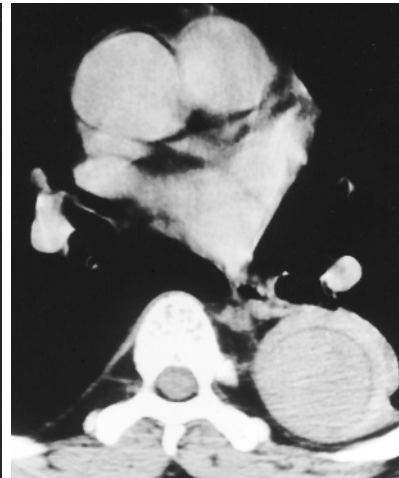
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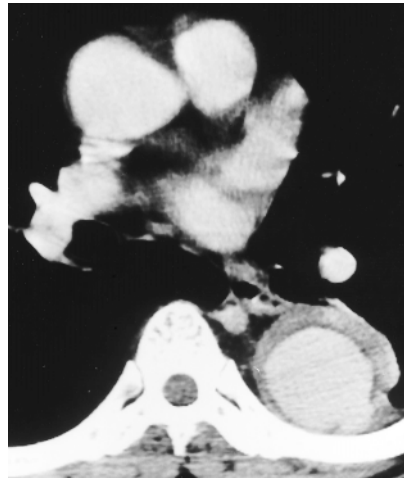
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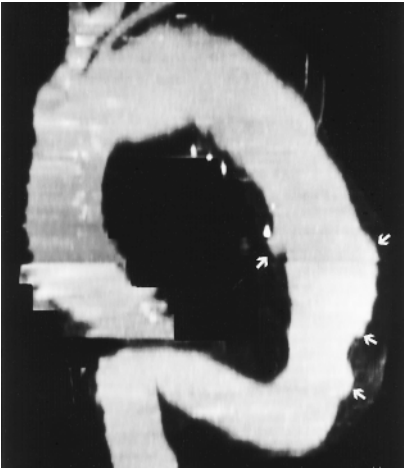
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Lesion Characteristics and Patient Outcome in 12 Cases of Penetrating Atherosclerotic Ulcer

Patient/ Age(y)/Sex	Follow-up Period	Lesion Location	Lesion Depth (mm)			Lesion Contour	Compli- cating Aneurysm	Patient Outcome
			At Onset	1 m after Onset	Final [‡]			
1/73/M	2 y	Abdominal aorta	6	6	6	Smooth	Yes*	Living
2/67/M	1 y 9 m	Proximal descending thoracic aorta	5	12	12	Smooth	No	Living
3/65/M	4 y	Proximal descending thoracic aorta	9	15	21	Smooth	No	Died of unrelated disease
4/73/M	3 y	Proximal descending thoracic aorta	3	15	15	Irregular	Yes [†]	Living
5/73/M	5 y 5 m	Distal descending thoracic aorta	7	21	21	Smooth	No	Living
6/71/M	2 y 2 m	Proximal descending thoracic aorta	11	16	0	Irregular	Yes [†]	Living
7/73/M	1 m 1 w	Distal descending thoracic aorta	8	22	NA	Irregular	No	Died of rupture
8/75/M	1 m	Distal descending thoracic aorta	5	16	NA	Irregular	Yes [†]	Died of rupture
9/69/M	2 w	Proximal descending thoracic aorta	6	8	NA	Smooth	No	Underwent surgery
10/73/M	2 w	Distal descending thoracic aorta	5	5	NA	Smooth	Yes* [†]	Underwent surgery
11/77/F	1 m 1 w	Proximal descending thoracic aorta	7	12	NA	Irregular	No	Underwent surgery
12/74/M	1 m	Distal descending thoracic aorta	4	22	NA	Irregular	Yes [†]	Underwent surgery

*Thoracic abdominal aneurysm.
[†]Abdominal aortic aneurysm.
[‡]NA = not applicable.

The Table shows ulcer location, depth, and contour as well as patient outcome in 12 patients with penetrating atherosclerotic ulcer who were followed up from onset for varying periods of time. All six patients with poor prognoses (cases 7–12) either died ($n = 2$) or underwent surgical resection for recurrent pain or enlargement of the ulcer ($n = 4$) within 1 month of onset. We believe the hematoma had not yet organized at this point. In the remaining six patients (cases 1–6), the size of the ulcer stayed the same or increased very slowly, although one of these patients died from unrelated disease. Although cases of penetrating atherosclerotic ulcer typically have many features in common (eg, elderly patients with evidence of advanced atherosclerosis and hypertension, involvement of the descending thoracic aorta or ab-

dominal aorta), the prognosis was different from case to case. In case 7, hematoma in the media enlarged rapidly and resulted in rupture (Fig 2). The patient in case 12 underwent surgical resection because of ongoing chest pain (Fig 3), and the patient in case 5 has been treated conservatively for more than 5 years (Fig 4). The radiologic size and shape of acute-stage penetrating atherosclerotic ulcers was the same whether the prognosis was good or bad.

In our experience, the prevalence of critical cases of symptomatic penetrating atherosclerotic ulcer is higher than that of classic aortic dissection. Furthermore, Coady et al (7) reported that the risk of aortic rupture was considerably higher among patients with penetrating atherosclerotic ulcer (40% of cases) than among patients with type A or type B aortic dissection (7.0% and 3.6%, respectively).

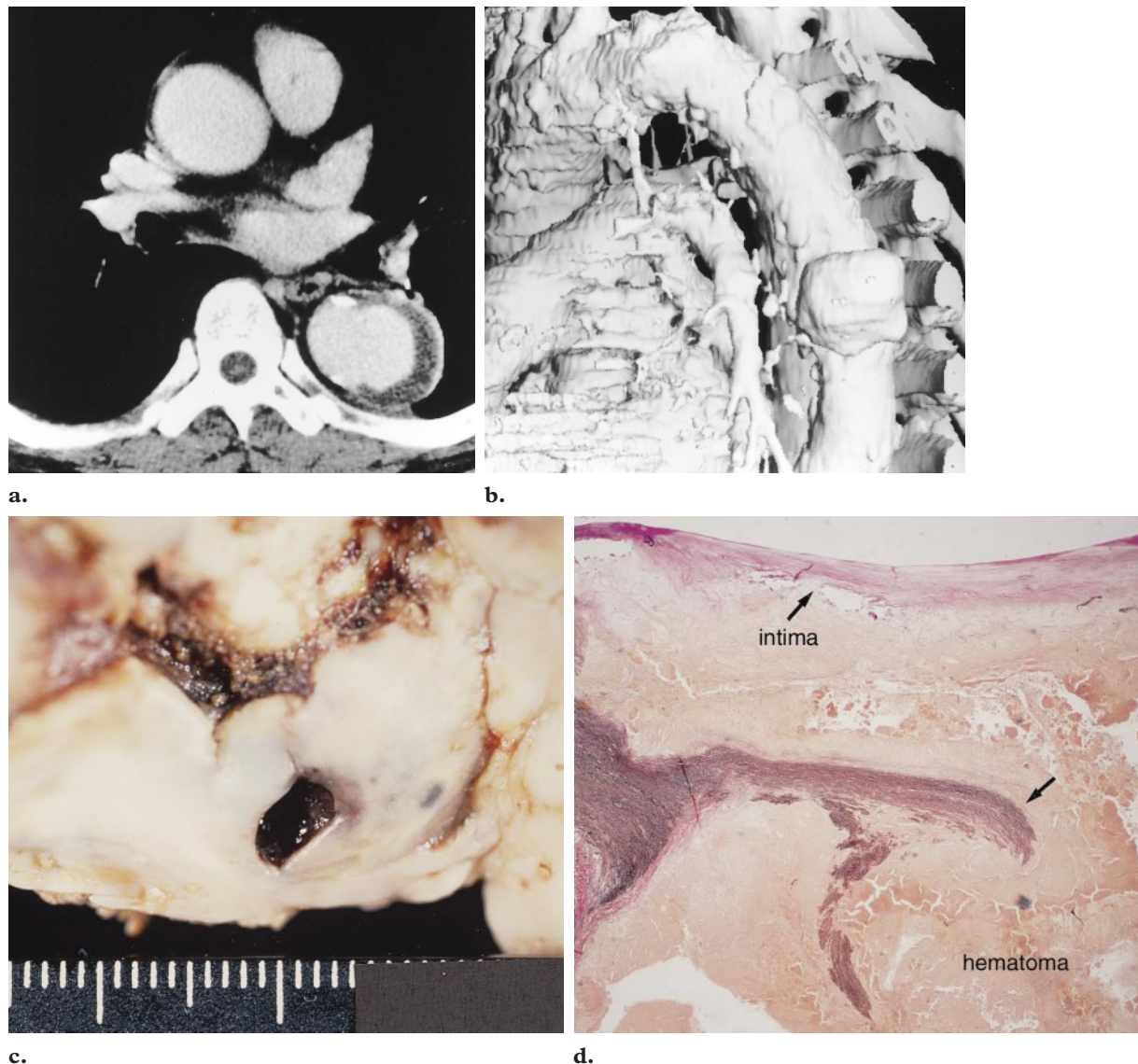


Figure 7. Saccular aneurysm caused by penetrating atherosclerotic ulcer in an asymptomatic 65-year-old man. **(a)** CT scan shows a contrast material–filled ulcer and mural thrombus. **(b)** Shaded-surface-display image clearly demonstrates the aneurysm. **(c)** Photograph of the surgical specimen shows ulceration penetrating the intima and forming a saccular aneurysm. Scale is in millimeters. **(d)** Low-power photomicrograph (original magnification, $\times 35$; elastica van Gieson stain) demonstrates the false aneurysm with complete disruption of the media (arrow). Hematoma can be seen under the atherosclerotic intima and the media. (This case was not included in the Table because the lesion was discovered incidentally, making follow-up from onset impossible.)

We occasionally encountered a saccular aneurysm, which seemed to be caused by a penetrating atherosclerotic ulcer (Fig 7). Although rupture or other life-threatening complications are rare, affected patients must be followed up because such complications can progress slowly (11).

Management

Most authors suggest that surgical intervention with grafting of the affected area is the treatment of choice because of possible malignant involvement (1–3,6,7). In particular, persistent or recurrent pain, hemodynamic instability, and a rapidly

expanding aortic diameter have been considered indications for surgical treatment (1).

On the other hand, some authors believe that immediate surgical treatment is not always required because the disease may have a benign course (4,8). In the study by Harris et al (8), few patients developed aortic dissection or aortic rupture during follow-up. These authors emphasized that most patients with penetrating atherosclerotic ulcers are at high risk for surgical intervention because of their advanced age and poor general health.

Because we could not predict rupture from the early imaging findings, we concluded that penetrating ulcers of the descending thoracic aorta should initially be treated aggressively and that pertinent clinical and radiologic data should be monitored carefully during the 1st month after onset. Surgical treatment may become necessary in cases involving evidence of intramural hematoma expansion, signs of impending rupture, inability to control pain, or blood pressure changes.

Murgo et al (20) emphasized that surgical repair of the descending thoracic aorta is frequently complicated by respiratory disease, renal insufficiency, or spinal ischemia and recommended the transluminal placement of endovascular stent-grafts for penetrating atherosclerotic ulcers.

Relationship between Penetrating Atherosclerotic Ulcer and Aortic Dissection

We believe that penetrating atherosclerotic ulcer penetrates through the elastic lamina and into the media and can cause aortic dissection, aortic aneurysm, or spontaneous rupture (Fig 1). Segmental dissection of the aortic wall at the site of atherosclerotic plaque has also been described. In his classic 1933 monograph on dissection, Shennan (21) showed that some of the wall hematoma had begun in an atheromatous ulcer. However, it remains controversial whether atherosclerosis causes aortic dissection.

Some investigators do not consider atherosclerosis a risk factor for aortic dissection, and Roberts (10) suggested that medial atrophy and fibrosis secondary to atherosclerosis may actually limit rather than promote intramural cleavage. Moreover, classic aortic dissection usually begins in the proximal aorta, whereas atherosclerosis is rarely seen in this location (11). In contrast, several reports have described aortic dissection originating at the base of an atherosclerotic ulceration (22,23). Larson and Edwards (23) reported that rupture of ulcerocalcific aortic atheromas may have initiated the intimal tear in some type III dissections.

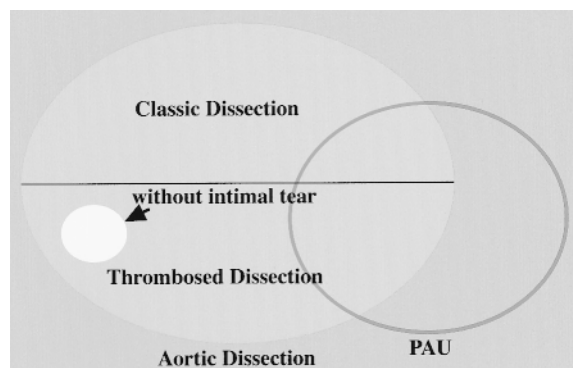


Figure 8. Diagram illustrates the relationship between penetrating atherosclerotic ulcer and aortic dissection. Considerable overlap is seen.

Penetrating atherosclerotic ulcer is typically seen in elderly individuals with hypertension and atherosclerosis and usually involves the descending thoracic aorta (1–3). Stanford B dissection also occurs frequently in elderly individuals with an atherosclerotic aortic wall. Moreover, atherosclerosis prevents extension of hematoma along the aortic wall and the creation of reentry. Therefore, we speculate that substantial numbers of aortic dissections are caused by penetrating atherosclerotic ulcer, particularly in elderly patients with Stanford B and thrombosed type dissections.

Figure 8 illustrates our concept of the relationship between aortic dissection and penetrating atherosclerotic ulcer. A subgroup of thrombosed aortic dissections is believed to have no intimal tear. Penetrating atherosclerotic ulcer cannot be definitely distinguished from aortic dissection (particularly thrombosed dissection) because of overlapping characteristics.

Conclusions

Differentiation of penetrating atherosclerotic ulcer from other causes of aortic disease such as aortic dissection, aortic aneurysm, and spontaneous aortic rupture is difficult or impossible in some cases. Because critical cases of penetrating atherosclerotic ulcer cannot be identified on the basis of initial imaging findings, careful follow-up is needed in affected patients, particularly during the 1st month after onset.

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