Penetrating ulcer of the thoracic aorta: What is it? How do we recognize it? How do we manage it?

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Background: **Although classic type A and B aortic dissections have been well described, less is known about the natural history of penetrating atherosclerotic ulcers of the thoracic aorta. This study differentiates penetrating ulcer from aortic dissection, determines the clinical features and natural history of these ulcers, and establishes appropriate correlates regarding optimal treatment.**

Methods: **A retrospective review of patient records and imaging studies was conducted with 198 patients with initial diagnoses of aortic dissection (86 type A, 112 type B) at our institution from 1985 to 1997.**

Results: **Of the 198 patients, 15 (7.6%) were found to have a penetrating aortic ulcer on re-review of computed tomographic scans, magnetic resonance images, angiograms, echocardiograms, intraoperative findings, or pathology reports. Two ulcers (13.3%) were located in the ascending aorta; the other 13 (86.7%) were in the descending aorta. In comparison with those with type A or B aortic dissection, patients with penetrating ulcer were older (mean age 76.6 years, $p = 0.018$); had larger aortic diameters (mean diameter 6.5 cm); had ulcers primarily in the descending aorta (13 of 15 patients, 86.7%); and more often had ulcers associated with a prior diagnosed or managed AAA (6 of 15 patients, 40.0%; $p = 0.0001$). Risk for aortic rupture was higher among patients with penetrating ulcers (40.0%) than patients with type A (7.0%) or type B (3.6%) aortic dissection ($p = 0.0001$).**

Conclusions: **Accurate recognition and differentiation of penetrating ulcers from classic aortic dissection at initial presentation is critical for optimal treatment of these patients. For penetrating ulcer, the prognosis may be more serious than with classic type A or B aortic dissection. Surgical management is advocated for penetrating ulcers in the ascending aorta and for penetrating ulcers in the descending aorta that exhibit early clinical or radiologic signs of deterioration. (J Vasc Surg 1998;27:1006-16.)**

In 1934, Shennan[1] was the first to describe penetrating atheromatous ulcers of the thoracic aorta. These atheromatous plaques, characterized further by Stanson et al. in 1986,[2] ulcerate and disrupt the internal elastic lamina, burrowing deeply through the intima into the aortic media.[3] The plaque may precipitate localized intramedial dissection associated with a variable amount of hematoma within the aortic wall, may break through into the adventitia to form a pseudoaneurysm, or may rupture completely into the right or left hemithorax. The diagnosis is made at computed tomography (CT) with demonstration of a contrast material–filled outpouching in the aorta in the absence of a dissection flap or false lumen and often in the presence of extensive aortic calcification. The clinical presentation of penetrating aortic ulceration is similar to that of classic aortic dissection; however, penetrating ulceration represents a unique pathologic event that may have distinct prognostic and therapeutic implications.[4] Classic type A and B aortic dissections differ from penetrating ulcer. Dissection usually begins with an intimal tear at the points of greatest hydraulic stress—the

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Table I. Characteristics used to differentiate penetrating ulcer from aortic dissection

Characteristic	*Penetrating ulcer*	*Aortic dissection*
Typical patient	Elderly with hypertension	Often young with hypertension Occasionally with bicuspid aortic valve Marfan syndrome
Symptoms	Severe chest or midscapular pain	Severe chest or midscapular pain
Signs	Absent	Aortic insufficiency (type A) Pulse inequality Neurologic deficits Compromise of blood flow to visceral vessels
Diagnostic features	No intimal flap Intramural hematoma Localized ulceration penetrating internal elastic lamina	Intimal flap with contrast material filling false lumen
Extent of lesion	Focal	Usually extensive
Degree of atherosclerosis	Always severe	Variable (often minimal)

right lateral wall of the ascending aorta or the descending thoracic aorta close to the ligamentum arteriosum.[3] Imaging studies of classic type A or type B dissection show two or more channels separated by an intimal flap.

Because preliminary reports[2-4] indicate that penetrating ulcer is distinct from aortic dissection, we sought to isolate true cases and study the natural history of these atheromatous ulcers. Table I shows the characteristics used to differentiate penetrating ulcer from classic aortic dissection. Because penetrating ulcer is much less common than classic aortic dissection, it may not be recognized and therefore may be misclassified as aortic dissection on presentation.

To confirm the existence of penetrating ulcers, a retrospective review of the records of every patient admitted with a preliminary diagnosis of aortic dissection at Yale–New Haven Hospital from January 1985 through July 1997 was conducted. Of 198 patients classified as having dissection, 15 were found on re-review of CT scans, magnetic resonance (MR) images, angiograms, echocardiograms, intraoperative findings, or pathologic findings to have a penetrating ulcer. This analysis was aimed at defining the clinical features and natural history of penetrating atherosclerotic ulcers and determining correlates regarding optimal treatment.

METHODS

Patients were enrolled in the study after a computerized search of the records of all patients who underwent magnetic resonance imaging (MRI), CT, angiography, or echocardiography of the thoracic aorta at Yale–New Haven Hospital from January 1985 through July 1997. A search also was conducted to find patients who underwent operations on the aorta and patients with chronic dissection treated in the Center for Thoracic Aortic Disease at Yale University. Autopsy records were examined for all patients who died of aortic disease during this time period. Despite our attempt to isolate all patients with aortic disease, we recognized that it was possible that there were persons with asymptomatic disease who did not undergo imaging studies.

The database included 431 imaging studies (MRI, CT, angiography, and echocardiography) of the 198 patients originally classified as having aortic dissection (86 type A and 112 type B dissections). On re-review of the studies 15 of these 198 patients (7.6%, 9 men and 6 women) were found to have a penetrating aortic ulcer on the basis of inspection of CT scans, MR images, angiograms, echocardiograms, intraoperative findings, or pathology reports. The cases of these 15 patients were compared with those of 82 patients with type A dissection and 101 patients with type B dissection who were found not to have penetrating ulcers.

Information on patients with aortic disease was maintained in a computerized database, and all patients' images studies were studied longitudinally. Demographic data, including risk factors and associated disease (hypertension, pulmonary disease, renal insufficiency, and cardiac disease) were classified and graded as mild, moderate, or severe according to the suggested standards for reports dealing with lower extremity ischemia as formulated by the Ad Hoc Committee on Reporting Standards of the Society for Vascular Surgery and International Society for Cardiovascular Surgery, North American Chapter.[5] The follow-up period for patients in our

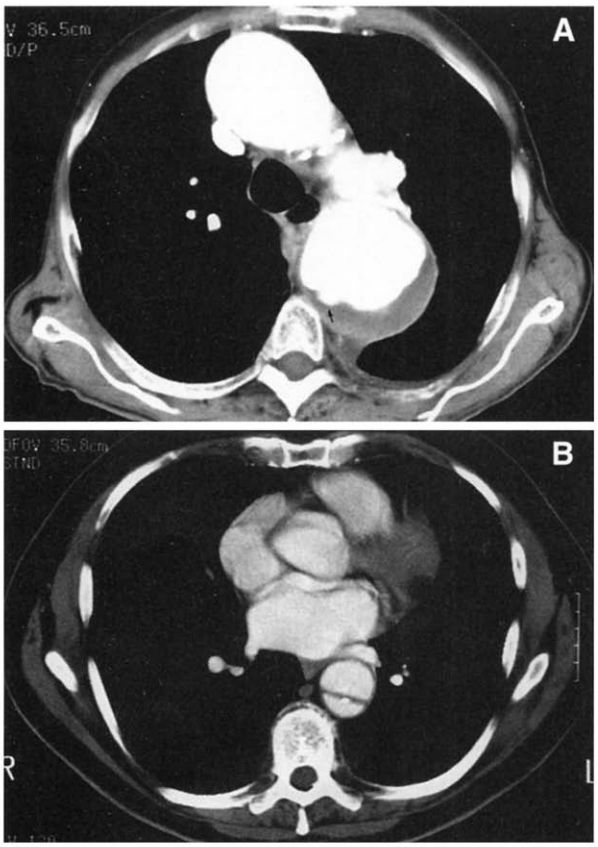

Fig. 1. A, Computed tomographic scan of patient 3 (Table II) at middle level of the descending thoracic aorta. Clearly demonstrated is a contrast medium–filled penetrating ulcer. No intimal flap is seen. **B,** Computed tomographic scan shows classic type B aortic dissection for comparison. Intimal flap and both true and false lumens are present.

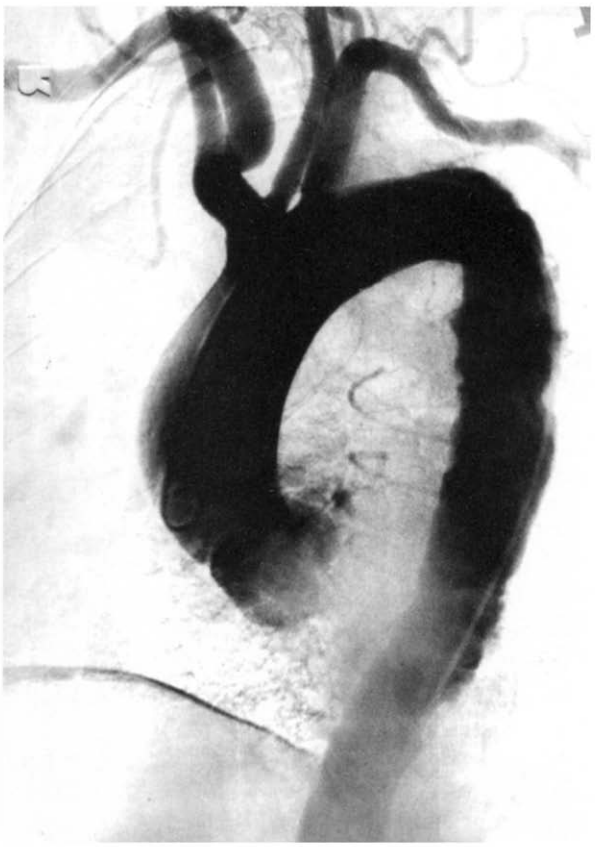

Fig. 2. Aortic angiogram confirms diffuse atherosclerotic disease in patient 3 (Table II). This patient had a penetrating ulcer confirmed by means of CT scan.

database ranged from 2 to 107 months, with a mean of 26 months.

A hospital chart review was conducted for all patients with type A dissection, type B dissection, or penetrating ulcer. The width of the penetrating ulcer, maximum aortic diameter at the ulcer level, and aortic wall thickness at the ulcer margin were measured. The diagnosis of penetrating ulcer was established by means of review of images with a radiologist specifically trained in vascular imaging. On CT scans, the diagnosis was established with demonstration of a contrast medium–filled outpouching in the aorta in the absence of a dissection flap or false lumen and often in the presence of extensive aortic calcification (Fig. 1*A*). On MR images, penetrating ulcers were diagnosed by means of visualization of a distinct ulcer crater in the absence of an intimal flap or false lumen. Transesophageal echocardiography (TEE) demonstrated a craterlike ulcer with jagged edges usually in the presence of extensive aortic atheroma and absence of an intimal flap or false lumen.

Angiography demonstrated a characteristic localized contrast medium–filled outpouching in the absence of a dissection flap or false lumen. Cobblestoning in the region of the ulcer was consistent with diffuse atherosclerosis (Fig. 2). Accompanying localized aortic dissection, intramural or surrounding hemorrhage, and pleural effusion often were found. Patients with classic type A or type B dissection (Fig. 1*B*), however, had two or more channels separated by an intimal flap on CT scans, MR images, aortograms, or echocardiograms.

Statistical methods were used to make comparisons between the three groups—penetrating ulcer, type A dissection, and type B dissection. Univariate analyses were performed with the SAS program, version 6.12, (SAS Institute, Cary, N.C.). Analysis of variance was performed to compare means between

the three groups. One-year survival estimates were calculated by means of life-table analysis (Kaplan-Meier) with the Lifereg procedure in the SAS program. The variables tested for survival differences included type A and B dissection versus penetrating ulcer. The log-rank test was used for examination of statistical significance ($\alpha = 0.05$).

RESULTS

Demographic considerations and clinical features. Table II summarizes demographic and clinical features of the 15 patients with penetrating ulcers of the thoracic aorta. The mean age was 76.6 years, which was significantly older than that of patients with type A (mean 54.7 years, $p = 0.018$) or type B dissection (mean 67.1 years, $p = 0.010$) (Table III). Penetrating ulcers occurred within a narrow age range (63 to 85 years) as opposed to type A (16 to 90 years) or type B dissection (18 to 89 years).

Anterior chest or midscapular back pain was the presenting symptom for 12 patients (80.0%). The other three patients (20.0%) had no symptoms (Table II). Accompanying medical problems were common among the 15 patients with penetrating ulcers, particularly hypertension (15 patients, 100.0%), chronic obstructive pulmonary disease (11 patients, 73.3%), cardiac disease (7 patients, 47.7%), diabetes mellitus (2 patients, 13.3%), and chronic renal insufficiency (4 patients, 26.7%).

The most common site of penetrating ulcer was the descending thoracic aorta, where 13 (86.7%) were found. In two of these patients, the ulcer caused localized intraaortic hemorrhage extending above the ligamentum arteriosum into the aortic arch. The other two ulcers (13.3%) were in the ascending aorta.

Table III displays the initial size of the aorta for patients with penetrating ulcers, type A dissection, and type B dissection. Although not statistically significant, the greatest aortic diameter among patients with penetrating ulcers tended to be larger on average than that among patients with type A or B dissection. Concomitant abdominal aortic aneurysms were found in 6 of 15 patients (40.0%) (Table III). This was significantly higher than the incidence of abdominal aortic aneurysm with type A dissection (8 of 86 patients, 9%) or type B dissection (35 of 112 patients, 31%) ($p = 0.0001$).

Four of the 15 patients with penetrating ulcer (26.7%) underwent aortic replacement (Table II). One of these patients was believed to have ruptured type A dissection (patient 13, Table II). This patient was an 84-year-old woman with a history of hypertension who sought treatment after 6 hours of anterior chest pain. An initial chest radiograph revealed a widened mediastinum. Subsequent TEE demonstrated 2+ aortic insufficiency, moderate pericardial effusion, aortic atheromatous plaques, and a circumferential intramural hematoma of the ascending aorta. The maximum transverse diameter of the aorta at this level of the ascending aorta was 6.8 cm. The patient was treated with sodium nitroprusside and a β-blocker and taken to the operating room for emergency resection and graft repair of the ruptured ascending aorta. During the operation extensive aortic calcification was found. The pathology report confirmed the presence of an intramural hematoma of the ascending aorta and a moderate-sized ulcer penetrating through the aortic media into the adventitia. The patient did well initially; however, progressive adult respiratory distress syndrome and cardiac failure developed 3 days postoperatively. The patient died on the sixth postoperative day.

Patient 8 (Table II) was a 72-year-old woman with left chest pain. A CT scan at admission revealed contained rupture of the descending aorta; the aorta measured 7 cm in transverse diameter at this level. The patient underwent emergency repair of the descending aorta. Extensive calcification of the aorta and a contained rupture were found during the operation. The patient's postoperative course was complicated by left hemiparesis. The pathology report confirmed the presence of large atheromatous plaques, a penetrating ulcer, and aortic rupture through the adventitia.

An 82-year-old woman (patient 12, Table II) with severe hypertension was admitted with significant midscapular back pain. Initial TEE revealed extensive calcification of the descending aorta and a penetrating ulcer, large intramural hematoma, and no intimal flap. The patient underwent successful operative repair.

Another patient was believed to have ruptured type B dissection (patient 14, Table II). This 75-year-old woman with a history of hypertension and chronic obstructive pulmonary disease had midscapular chest pain. An initial CT scan demonstrated left pleural effusion and an intramural aortic hematoma without an intimal flap that began just distal to the left subclavian artery. The patient underwent medical therapy with antihypertensive agents and afterload-reducing agents. On the sixth hospital day, a CT scan demonstrated a new right pleural effusion and an interval increase in the size of the left pleural effusion. Recurrent chest pain and hemodynamic instability developed despite medical management, and the patient was taken to the operating room. She was found to have a rupturing

Table II. Demographic and clinical characteristics of patients with penetrating ulcers of the thoracic aorta

Patient No.	*Age (yr)*	*Sex*	*Hypertension*	*Cardiac disease*	*Renal dysfunction*	*Pulmonary disease*
1	77	F	Moderate	None	None	Severe
2	79	M	Severe	Mild	Moderate	None
3	76	M	Mild	Mild	None	Moderate
4	85	M	Severe	None	None	Severe
5	63	M	Moderate	Severe	Mild	Moderate
6	82	M	Moderate	Moderate	None	Moderate
7	64	M	Severe	None	None	None
8	72	F	Moderate	None	None	Moderate
9	83	F	Severe	Moderate	None	Severe
10	77	F	Moderate	None	Moderate	Moderate
11	79	M	Severe	Severe	None	Moderate
12	82	F	Severe	None	Moderate	None
13	84	F	Moderate	None	None	None
14	75	F	Moderate	None	None	Severe
15	69	M	Moderate	None	None	Moderate

AAA, Abdominal aortic aneurysm; *Desc,* descending thoracic aorta; *Asc,* ascending aorta; +, study performed for either diagnosis or follow-up evaluation; –, study not performed; *Med,* medical management with antihypertensive drugs and afterload reduction; *Surg,* patient underwent an operation; *Discharged,* patient was treated successfully and released from the hospital; *Rupture/death,* patient had an acute rupture and died in the hospital; *Rupture/discharge,* patient had an acute rupture before operation, underwent resection of the penetrating ulcer, and was later released from the hospital.

Table III. Comparison of penetrating ulcer with type A and B aortic dissection

Variable	*Penetrating ulcer*	*Type A dissection*	*Type B dissection*
No. of patients (sex)	15 (9M, 6F)	82 (58M, 24F)	101 (63M, 38F)
Age (yr)	76.6 ± 7.0 *†	54.7 ± 16.9†	67.1 ± 13.3*
Initial aortic diameter (cm)	6.45 ± 1.9	5.63 ± 1.5	5.4 ± 1.6
No. of patients with prior AAA (%)	6 (40)	8 (9.3)‡	35 (31.2)‡

Values are mean ± SD.
AAA, Abdominal aortic aneurysm.
*$p = 0.018$.
†$p = 0.01$.
‡$p = 0.0001$.

descending penetrating ulcer and underwent successful operative repair. Pathologic examination confirmed the presence of a penetrating atherosclerotic ulcer at the site of transmural rupture. The postoperative course was uneventful.

Three patients (20.0%) who were initially believed to have type B dissection died of acute aortic rupture while being treated medically with β-blockers and afterload reduction in the intensive care unit (patients 2, 3, and 4, Table II). For patient 2, the site of aortic rupture was visualized, and there was clear communication with the esophagus. The patient died of rupture of the atherosclerotic ulcer with exsanguination into the esophagus and left hemithorax. Patients 3 and 4 died of a perforated ulcer in the descending aorta. Autopsy demonstrated a mediastinal hematoma and hemothorax with a penetrating ulcer at the site of aortic rupture through the adventitia.

Eight patients (Table II) were treated medically and were pain free and in hemodynamically stable condition at discharge. These patients were undergoing follow-up serial imaging studies on an outpatient basis. Patient 6 (Table II) was found to have a penetrating ulcer in the ascending aorta but was treated medically because of advanced age and substantial comorbidity. The mean follow-up period for the patients who underwent medical therapy was 30.3 months (range 3 to 84 months). Only one patient (patient 15, Table II) had marked reduction of the intramural hematoma, which was evident on the first follow-up CT scan.

The incidence of acute rupture of penetrating ulcers was 40.0% (6 of 15 patients) as opposed to 7.3% (6 of 82 patients) for type A dissection and 4.0% (4 of 101 patients) for type B dissection ($p = 0.0001$).

Diagnostic studies. A number of different imaging modalities were used to establish the diagnosis and for follow-up examinations of patients

Presenting symptom	*AAA*	*CT*	*MRI*	*TEE*	*Angiography*	*Site*	*Management*	*Outcome*
Midscapular pain	Yes	+	−	+	−	Desc	Med	Discharged
Mild left chest and midscapular pain	Yes	+	+	+	−	Arch/desc	Med	Rupture/death
Midscapular pain	Yes	+	+	+	+	Desc	Med	Rupture/death
Left chest pain and hypotension	No	+	+	+	−	Desc	Med	Rupture/death
Left chest pain	Yes	+	+	−	+	Arch/desc	Med	Discharged
Asymptomatic	Yes	+	−	+	−	Asc	Med	Discharged
Midscapular pain	Yes	+	+	+	+	Desc	Med	Discharged
Left chest pain	No	−	+	−	−	Desc	Surg	Rupture/discharged
Asymptomatic	No	+	−	−	−	Desc	Med	Discharged
Asymptomatic	Yes	+	−	−	−	Desc	Med	Discharged
Left chest pain	No	+	−	+	−	Desc	Med	Discharged
Midscapular pain	No	−	+	+	−	Desc	Surg	Discharged
Anterior chest pain	No	+	−	+	−	Asc	Surg	Rupture/death
Midscapular pain	No	+	+	+	−	Desc	Surg	Rupture/discharged
Substernal pain	No	+	+	+	−	Desc	Med	Discharged

with penetrating aortic ulcers. More than one method was used for every patient (Table II). CT was the most commonly used imaging modality (13 patients, 86.7%). MRI was used in the diagnosis and follow-up evaluation of penetrating ulcers for 9 patients (60%). Fig. 3 is an MR image of a descending aortic pseudoaneurysm formed by an ulcer that bore through the media into the adventitial layer of the aortic wall. TEE was performed for 11 patients with penetrating ulcers (73%), and aortic angiography was used to examine 3 patients (20%).

Short-term survival. Fig. 4 and Table IV display the 1-year survival rates for the 15 patients with penetrating ulcers as opposed to patients with type A and type B dissection. Although not statistically significant, the 1-year survival rate was less for patients with penetrating ulcers (73.3%) than for patients with type A (90.2%) or B dissection (90.7%).

DISCUSSION

Severe atherosclerosis in the aorta commonly gives rise to distal embolic events and arterial flow obstruction. Although ulceration of an aortic atheroma occurs among patients with advanced atherosclerosis, it is usually asymptomatic, confined to the intimal layer, and not associated with intramural hematoma.[3] When an atherosclerotic plaque penetrates into the media, the media is exposed to pulsatile atrial flow, causing hemorrhage into the wall without an intimal flap. This leads to localized "dissection," and the intramural hematoma is prevented from extension by surrounding transmural inflammation and relative fusion of the layers of the aortic wall.[6] The adventitial layer is frequently dissected from the media in patients who have penetration through the media.[7] The adventitia also may rupture, and then only the surrounding mediastinal tissues contain the hematoma (Fig. 5). Patients with penetrating ulcers have a localized "dissection" limited by areas of severe calcification associated with usually severe atherosclerotic disease; this represents aortopathy different from that of classic aortic dissection. The site of entry into the "dissection" in patients with penetrating ulcers is the ulcer itself. Postmortem examination may show several atherosclerotic ulcers, often extending into the adventitia.[7]

The other cause of aortic intramural hematoma in the absence of an intimal flap includes rupture of the vasa vasorum in patients with cystic medial necrosis. This may occur spontaneously among patients with hypertension or after blunt chest trauma.[8] It has been speculated that aortic intramural hematoma may also originate from ulceration in an atherosclerotic aorta; thus the cause may be multifactorial.[9] These patients may have no evidence of an atheromatous ulcer on imaging studies, however, and an intimal tear is not visualized. Imaging studies may show an intramural hematoma, which is characteristically smooth in contour (Fig. 6).

Aortic dissection, on the other hand, begins with an intimal tear, and blood courses rapidly along the outer third of the media. At one time it was thought

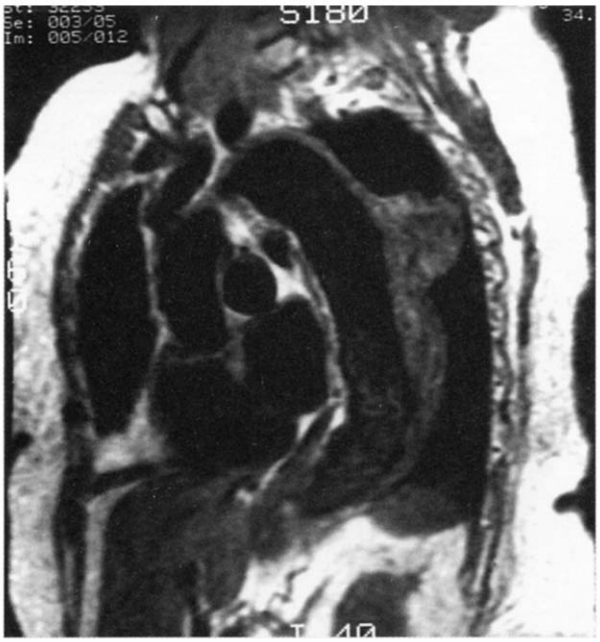

Fig. 3. Magnetic resonance image of the thorax demonstrates a pseudoaneurysm caused by a penetrating ulcer in the descending aorta.

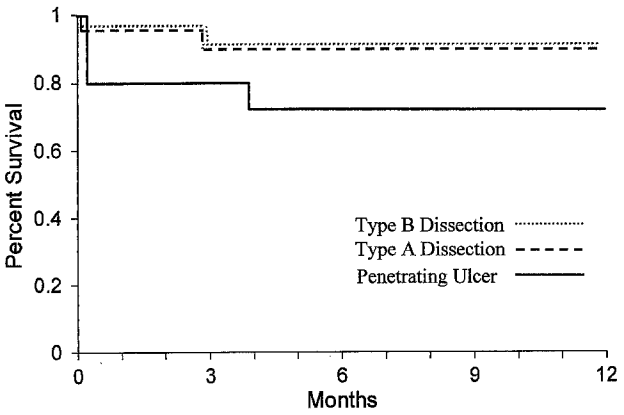

Fig. 4. Kaplan-Meier 1-year survival rates for patients with penetrating ulcer and type A and type B aortic dissection.

that atherosclerosis was the cause of acute aortic dissection. Among the earliest believers in this theory were Virchow (1851), Girode (1887), Ewald (1890), Rolleston (1893), and von Möller (1906).[1] In a classic paper titled "Dissecting Aneurysms," Shennan in 1934 began to recognize that although nodular atheromas were present in a large number of aortic dissections and ruptures, only a few displayed a relation of the atheroma to the exact location of primary rupture or dissection.[1] Only 6 of 218 dissections in Shennan's study demonstrated evidence of dissection at the base of an atheromatous ulcer.

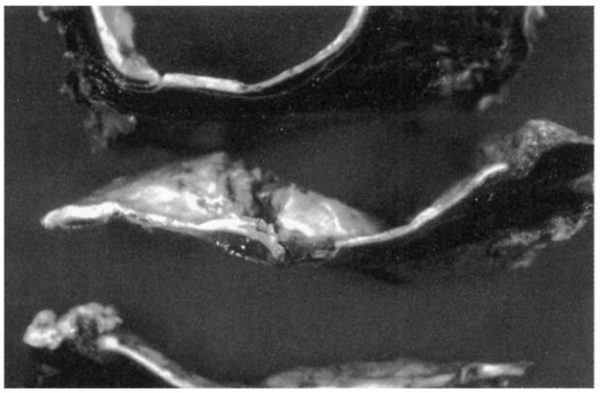

Fig. 5. Gross specimen. Serial sections through the aorta reveal a transmural penetrating ulcer in a patient who died of aortic rupture (patient 4, Table II).

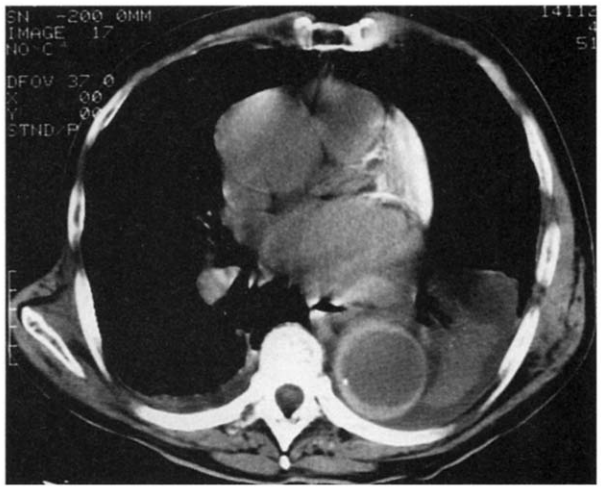

Fig. 6. Computed tomographic scan of a patient with an intramural hematoma.

There remains some controversy about whether atherosclerosis causes aortic dissection.[10,11] Most authorities believe it does not. There are those who believe it does not reason that aortic dissection is more frequently found in the ascending aorta, where atherosclerosis is less common. In addition, dissection in the area of gross atherosclerosis is usually limited because of neighboring fibrosis and calcification. Variables identified as being intimately associated with classic aortic dissection, include medial degenerative disease and hypertension.[12,13] Congenital aortic stenosis, bicuspid aortic valve, and coarctation of the aorta also are associated with a high incidence of aortic dissection, particularly

Table IV. Life-table analysis of cumulative survival rates for penetrating ulcer, type A dissection, and type B dissection

Interval (mo)	*No. at risk at start of interval*	*No. with failed treatment*	*No. withdrawn*	*Survival rate*	*Survival standard error*
Penetrating ulcer					
0-1	15.0	3	0	1.000	0.000
1-3	12.0	0	0	0.800	0.103
3-6	12.0	1	0	0.800	0.103
6-9	10.5	0	1	0.733	0.114
9-12	9.5	0	1	0.733	0.114
Type A dissection					
0-1	82.0	5	0	1.000	0.000
1-3	76.5	1	1	0.939	0.026
3-6	75.0	2	0	0.927	0.029
6-9	71.5	0	3	0.902	0.033
9-12	69.0	0	2	0.902	0.033
Type B dissection					
0-1	101.0	2	0	1.000	0.000
1-3	95.5	5	7	0.980	0.014
3-6	87.0	1	0	0.928	0.026
6-9	85.0	1	2	0.918	0.027
9-12	81.5	0	3	0.907	0.029

among young patients.[14] Medial degenerative disease is more frequently observed among patients with Marfan syndrome and is present in two thirds of all patients who need surgical treatment of aortic dissection.[15]

Penetrating ulcers, similar to aortic dissection, occur among persons with hypertension but affect an older age population (Tables I through III). This observation is consistent with those reported in other studies.[2] Penetrating ulcers are associated with a larger aortic size at the time of initial diagnosis, and many patients have a concomitant abdominal aortic aneurysm (Table III).

Although aortography once was the standard for the diagnosis of many aortic diseases, it has largely been replaced by CT (Fig. 1) and MRI. Yucel et al.[16] reported that MRI helped identify a discrete ulcer in 6 of 7 patients with angiographically proved ulceration. TEE has been used with success and has been reported to be highly sensitive and specific in the differential diagnosis of aortic disease.[17] Although all images in our study (CT scans, MR images, and echocardiograms) were reviewed by a radiologist, there remains a possibility that a tear may have been missed. Most of the 15 patients with penetrating ulcer, however, underwent multiple imaging studies without demonstration of an intimal flap, and all 15 patients had an ulcer visualized (Table II).

The condition of patients with penetrating aortic ulcers is stabilized with antihypertensive drugs and afterload-reducing agents in the intensive care unit until pain relief is achieved and hemodynamic stability is preserved. Persistent pain, recurrent pain, hemodynamic instability, and rapidly expanding aortic diameters have been indications for surgical treatment.[2] Emergency treatment also has been advocated for patients whose condition is hemodynamically unstable or who have pseudoaneurysm formation caused by a penetrating ulcer.[18] Several reports have shown that penetrating aortic ulcers in the ascending aorta or arch are prone to transmural rupture and therefore should be managed surgically.[11,19,20]

Kazerooni et al.[11] described eight patients with aortic ulceration complicated by intramural hematoma whose conditions were hemodynamically stable. These patients were treated conservatively with resolution of pain. Four of the eight patients underwent follow-up CT at a mean of 18.2 weeks. Intramural hematoma had resolved completely in three patients and diminished in size in one patient. Yucel et al.[16] reported similar results. Stanson et al.,[2] however, reported a high incidence of recurrent pain (7 of 16 patients) and contained rupture (7 of 16 patients). Cooke et al.[3] therefore recommended expeditious surgical treatment once the diagnosis has been confirmed for a patient with symptoms.

Two patterns of analysis in our series demonstrated the potentially dangerous nature of penetrating ulcers. Observational data demonstrated that the ulcers of 40% of patients (6 of 15 patients) ruptured during observation in the intensive care unit. Most of these patients were being treated for type B dissection (Table II). In addition, survival analysis suggested a trend toward a lower short-term survival rate

among patients with penetrating ulcers as opposed to type A or B dissection with the drop in survival manifested during the early acute phase. Perhaps the most likely reason for the poorer prognosis is that more layers of the aorta are penetrated by an ulcer in contradistinction to classic dissection, which occurs in the middle to outer third of the media.

Regardless of the location of a penetrating ulcer, patients should undergo immediate medical treatment to control pain and prevent rupture. In 1934, Shennan[1] found that 40% of patients with proximal dissection died immediately, 70% within 24 hours, 94% within 1 week, and 100% within 5 weeks. Because rupture and cardiac tamponade may occur at any time, patients with ascending penetrating ulcers should be treated with an immediate operation to prevent these complications.

Penetrating ulcers in the descending aorta should be managed initially with aggressive medical management and a high index of suspicion for rupture. Operative treatment becomes necessary in the case of clinical or radiologic deterioration, which includes evidence of expansion of intramural hematoma, signs of impending rupture (pseudoaneurysm formation and pericardial or bloody pleural effusion), or inability to control pain. Because penetrating ulcers primarily affect older persons, one must consider the patient's age, overall physical condition, and anticipated life expectancy in the decision to use early operative treatment.

Urgent operations for penetrating ulcers can be technically difficult because of heparinization and tissue friability. We do not routinely administer heparin during these repairs and have not found any thrombosis or thromboembolism with use of a Bio-Medicus left heart centrifugal pump in the absence of heparin. We do, however, instill 50 ml of heparinized saline solution into the left femoral artery before clamping. We have found treatment of the ascending aorta with 25% glutaraldehyde for several minutes extremely helpful in toughening the tissues in penetrating ulcers and type A dissection.[21,22] Although we have not used this technique on the descending thoracic aorta, Chen et al.[23] had success with glutaraldehyde, particularly in the presence of ruptured or impending rupture of the descending aorta. We also routinely used Hemashield grafts, secure friable suture lines with polytetrafluoroethylene (Teflon) felt, and biologic thrombin glue.

In summary, we confirmed the existence of penetrating atheromatous ulcer as distinct from aortic dissection. Penetrating ulcer was found to be a life-threatening condition that may not be detected when a patient seeks treatment unless the clinician is aware of its distinguishing radiologic features. Characteristic features of penetrating ulcer include midscapular pain affecting an elderly patient with hypertension. The disease process usually affects the descending aorta. A concomitant abdominal aortic aneurysm often is present. The diagnostic studies most commonly used include CT, MRI, and TEE. In the case of penetrating ulcer, these imaging modalities demonstrate an ulcer, intramural hematoma, and absence of a flap.

Although our sample size was small, penetrating ulcers appeared to have a greater propensity to rupture in the acute setting during conservative treatment. Thus aggressive management is recommended for penetrating ulcers, and a high index of suspicion must be maintained for rupture. Like classic type A dissection, ascending penetrating ulcers should be managed with emergency operative repair because of malignant behavior. Penetrating ulcers in the descending aorta also should be considered strongly for early surgical intervention. A high index of suspicion for rupture must be maintained. Because penetrating ulcers primarily affect older persons, one must consider the patient's age, overall physical condition, and anticipated life expectancy in the decision to use early operative treatment. If patients tolerate early medical management without clinical deterioration, our experience suggests that they may continue conservative follow-up care with reasonable safety. These are provisional recommendations regarding the treatment of penetrating ulcer, the behavior of which is not fully understood. As we continue to expand our database, we hope to formulate a more accurate definition of the natural history of penetrating ulcer and isolate specific risk factors for early morality among these patients.

REFERENCES

1. Shennan T. Dissecting aneurysms. Medical Research Council, Special Report Series, No. 193. 1934.
2. Stanson AW, Kazmier FJ, Hollier LH, Edwards WD, Pairolero PC, Sheedy PF, Joyce JW, et al. Penetrating atherosclerotic ulcers of the thoracic aorta: natural history and clinicopathological correlations. Ann Vasc Surg 1986;1:15-23.
3. Cooke JP, Kazmier FJ, Orszulak TA. The penetrating aortic ulcer: pathologic manifestations, diagnosis, and management. Mayo Clin Proc 1988;63:718-25.
4. Mozsowitz HD, Lampert C, Jacobs LE, Kotler MN. Penetrating atherosclerotic aortic ulcers. Am Heart J 1994;128:1210-7.
5. Rutherford RB, Flanigan DP, Gupta SK, Johnston KW, Karmody A, Whittemore AD, et al. Suggested standards for reports dealing with lower extremity ischemia. J Vasc Surg 1986;4:80-94.

6. Roberts WC. Aortic dissection: anatomy, consequences, and causes. Am Heart J 1981;101:195-214.
7. Svensson LG, Crawford ES. Occlusive disease of the aorta. In: Cardiovascular and vascular disease of the aorta. Philadelphia: WB Saunders Company; 1997.
8. Alfonsa F, Goicolea J, Aragoncillo P, Hernandez R, Macaya C. Diagnosis of aortic intramural hematoma by intravascular ultrasound imaging. Am J Cardiol 1995;76:735-8.
9. Muluk SC, Kaufman JA, Torchiana DF, Gertler JP, Cambria RP. Diagnosis and treatment of thoracic aortic intramural hematoma. J Vasc Surg 1996;24:1022-9.
10. Hirst AE Jr, Johns VJ Jr, Klime SW Sr. Dissecting aneurysm of the aorta: a review of 505 cases. Medicine (Baltimore) 1958;37:217-79.
11. Nakashima Y, Kurozumi T, Sueishi K, Tanaka K. Dissecting aneurysm: a clinicopathologic and histopathologic study of 111 autopsied cases. Hum Pathol 1990;21:291-6.
12. Svensson LG, Crawford ES. Aortic dissection and aortic aneurysm surgery: clinical observations, experimental investigations, and statistical analyses; II. Curr Probl Surg 1992;29:915-1007.
13. DeBakey ME, Henly WS, Cooley DA, Morris GC, Crawford ES, Beall AC Jr. Surgical management of dissecting aneurysms of the aorta. J Thorac Cardiovasc Surg 1965; 49:130-49.
14. Gore I, Seiwert VJ. Dissecting aneurysm of the aorta: pathological aspects—analysis of 85 fatal cases. Arch Pathol 1952; 53:1022.
15. Svensson LG, Crawford ES. Aortic dissection. In: Cardiovascular and vascular disease of the aorta. Philadelphia: WB Saunders; 1997.
16. Yucel EK, Steinberg FL, Egglin TK, Geller SC, Waltman AC, Athanasoulis CA. Penetrating aortic ulcers: diagnosis with MR imaging. Radiology 1990;177:779-81.
17. Sommer T, Fehske W, Holzknecht N, Smekal AV, Keller E, Lutterbey G, et al. Aortic dissection: a comparative study of diagnosis with spiral CT, multiplanar transesophageal echocardiography, and MR imaging. Radiology 1996; 199:347-52.
18. Kazerooni EL, Bree RL, Williams DM. Penetrating atherosclerotic ulcers of the descending thoracic aorta evaluation with CT and distinction from aortic dissection. Radiology 1992;183:759-65.
19. Patterson DL, Brennan S, Cartwright T, Jolly W, Adlam JH, Waller BF. Traumatic rupture of an aortic ulcerative atherosclerotic plaque producing aortic dissection: a complication of interscapular back blows used to dislodge objects from the esophagus. Clin Cardiol 1993;16;741-4.
20. Calick A, Vance ZB, Berger SA. Spontaneous rupture through atheromatous plaque. NY State Med J 1973; 73:2068-70.
21. Vasseur B, Hammond GL. New technique for repair of ascending thoracic aortic dissections. Ann Thorac Surg 1989; 47:318-9.
22. Hammond GL. 1989: new technique for repair of ascending aortic dissections. Updated in 1997. Ann Thorac Surg 1997;63:287.
23. Chen YF, Chou SH, Chiu CC, Lin YT, Wang HJ. Use of glutaraldehyde solution in the treatment of acute aortic dissections. Ann Thorac Surg 1994;58:833-5.

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DISCUSSION

Dr. Magruder C. Donaldson (Boston, Mass.). Dr. Coady and his colleagues have done a great service by discussing a distinctly threatened subset of patients with conditions that masquerade as aortic dissections. Although their conditions are similar in clinical presentation from the standpoint of symptomatology, patients with penetrating thoracic aortic ulcer were older, were more commonly afflicted with associated chronic aortic atherosclerosis and abdominal aneurysms, and had harbored aortas generally of larger diameter than those with true dissection. Radiologic and pathologic findings revealed a focal process of contained intramural disruption rather than a diffuse axial pattern of medial dissection. Most importantly, this process was associated with rupture in 40% of cases as compared with 5.5% overall among patients with aortic dissection and 4% among patients with type B descending aortic dissections. Although the study design does not elucidate precise natural history, it is clear that penetrating aortic ulcers are "bad actors" worthy of our full attention.

To those of us familiar with mural disruption of the abdominal aorta, penetration and rupture of the thoracic aorta is not a surprising event. I wonder if Dr. Coady would care to elaborate on his thoughts on why some old atherosclerotic aortas penetrate and rupture and others prefer to expend their explosive energy by merely dissecting?

Of 15 penetrating ulcers, 13 were found hiding among the generally older patients with type B dissections involving the descending aorta. Because therapy is preferentially medical for type B dissections, patients with misdiagnosed penetrating ulcers of the descending aorta are in real danger. Given the importance of rapid and precise differential diagnosis in the heat of battle, can you elaborate on the currently best algorithm for this critical diagnosis?

Rupture had either occurred on presentation or followed soon thereafter only among the 12 patients with symptoms. Were any other clinical findings—such as pleural effusion, aortic diameter, response of pain to antihypertensives, or radiologic characteristics (ie, the relative depth and penetration)—at all helpful in predicting early rupture and thus selecting patients for urgent surgery?

Finally, eight of your patients did reasonably well when treated medically as presumptive dissections. In retrospect, can you clarify circumstances under which you would knowingly choose observational medical management? What impact has the increasing experience with endovascular graft exclusion had on your philosophy of management?

Dr. Richard P. Cambria (Boston, Mass.). I certainly agree that there is considerable confusion in disease of the thoracic aorta with the terms dissection, penetrating ulcer,

thoracic aortic intramural hematoma, and degenerative aneurysm, but I am unsure that the way you gathered your patients in this study design helps sort out the issue. On the basis of the data and the computed abdominal tomography scans you showed, I think that the high rupture rate in the patients with "penetrating ulcer" represented rupture of destabilizing thoracic aortic aneurysms. The mean aortic size of your patients with penetrating ulcers was 6.5 cm. I believe that because the study was designed in terms of retrieval, you missed patients with penetrating aortic ulcer in small-sized or modest-sized aortas. I would like to add to your management guidelines that the size of the thoracic aorta at the time the patient is first seen should be an important component in deciding between medical and surgical therapy.

We presented 15 cases of thoracic aortic intramural hematoma last year before the Eastern Vascular Society, and we agree with your recommendations that hematoma ought to be treated surgically when the process occurs in the ascending aorta. When the hematoma occurs in the descending aorta, we believe the presence or absence of concomitant aneurysm should be the guiding principle in deciding between medical and surgical treatment.

Dr. Michael A. Coady. Penetrating ulcers of the thoracic aorta occur in elderly hypertensive patients. It is unclear why some individuals rupture and others do not. The difference may be related to the depth of ulcer penetration into the aortic wall and the degree of surrounding localized fibrosis. Other possibilities may relate directly to LaPlace's law, reflecting the effects of hypertension, aortic diameter, and wall thickness. Our experience with 15 patients has been limited, and this is an attempt to disentangle this entity from classic aortic dissection. In reality, a broad spectrum of dissecting aortic pathologies may exist, with penetrating ulcers representing a single variant.

As far as distinguishing penetrating ulcers from aortic dissection in the acute setting, a thorough review of imaging studies with a radiologist may aid in differentiating these ulcers from classic dissections. This is especially worthwhile if a flap is not visualized easily on initial imaging studies.

For patients with penetrating ulcer, poor pain control while receiving maximal medical treatment does correlate with early rupture. However, because the majority of our patients were first seen acutely without prior imaging studies, we cannot directly correlate a rate of change in aortic diameter with a risk of impending rupture. The relationship of an increasing aortic diameter with risk of rupture makes intuitive sense, however, and has been an important consideration for early resection in patients with intramural hematoma and thoracic aortic aneurysms. The presence of a pleural effusion in our experience has not been particularly helpful in predicting rupture. A pleural effusion may occur with frank rupture or may be present acutely in absence of rupture, representing an inflammatory pleural reaction—we have seen both.

The majority of our patients who did well when treated medically were those who were first seen with penetrating ulcers located in the descending aorta. We advocate medical management of penetrating ulcers in the descending aorta in patients who are well controlled on "antiimpulse" therapy and who demonstrate no evidence of continued pain, aortic expansion on serial imaging studies, or pseudoaneurysm formation.

There have been several early studies on the treatment of aortic dissection with minimally invasive procedures employing endovascular stents. We have no experience with these stents in the thoracic aorta at our institution.

Finally, I agree with Dr. Cambria's opinion that size of the thoracic aorta should be an important additional guideline in the management of these lesions. We recently published a paper outlining the management of thoracic aortic aneurysms (J Thorac Cardiovasc Surg 1997;113:476-91). That paper shows the importance of increasing aortic size as a risk factor for acute dissection or rupture.