Aortic pathology: Aortic trauma, debris, dissection, and aneurysm

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The aorta is a conduit from the left ventricle that delivers pulsatile blood distally in either a compliant or stiffened vessel to organs and tissue beds. Only recently, since the advent of transesophageal echocardiographic imaging, did its presence and associated pathologies become more profound and more prominent for the intensivist. Angiography, the "gold standard" for diagnostic imaging, now seems to be in question since the advent of ultrasound (transesophageal echocardiography), improvements in magnetic resonance imaging, and particularly the advancement to 64-slice computed tomography. It is now a revelation of how revealing these newer imaging tools have expanded our knowledge potential of pathologies that involve the aorta. The latter three imaging modalities are continuing to improve, with established efficacy, particularly in the critically ill and injured patient. This article will enlighten the intensivist and others of their potential and contrast each imaging device in several prominent pathologies common to the critical care physician. The disadvantages of all will be brought forth. Evidence will be presented revealing the dynamic nature of imaging technologies that will continue to affect the outcome of our patients. The most common indications for interrogation of the aorta are in traumatic events in which there might be a catastrophic transection, intimal tear or flap, or subadventitial tear. The identification of hematomas (by these imaging devices) in the mediastinum might be associated with significant physical forces, and this article will show the relevance. The significance of atherosclerotic plaques, ulcers, and debris will also be debated. Finally, imaging of a patient with aortic dissection or aneurysm will be discussed, as its pathology and pathogenic process are well known, and the changing nature or paradigm shift in the imaging of this life-threatening disease will be addressed. (Crit Care Med 2007; 35[Suppl.]:S392–S400)

Key Words: aorta; pathology; transesophageal echocardiography; magnetic resonance imaging; computed tomography; critical care

Mechanical trauma is usually sudden and unexpected, and it can have devastating or subtle results that later may manifest into dire consequences. Appropriate and quick diagnosis may avoid a preventable mishap with significant potential of morbidity and even death. One such circumstance is in the rapid acceleration and deceleration involving motor vehicle accidents, motorcycle accidents, falls, industrial accidents, and domestic violence. In a motor vehicle accident, there are significant physical forces, resulting in compression and distortions, even to the extent of compression of chest wall contents, particularly the aorta against the spinal column.

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Traumatic aortic injury is one such disease that is life threatening, ranging from intimal tears, to mediastinal hematomas, to transection. The most common sites of injury are the aortic isthmus and the ascending aorta, just proximal to the origin of the brachiocephalic vessels (Table 1). Transection of the ascending aorta is relatively uncommon (10%). Cammack et al. (1) and others (2–4) showed that vertical forces of deceleration may lead to rupture of the ascending aorta and arch. The majority of the time (65% to 80%), the occupants with an aortic injury in motor vehicle accidents will succumb at the scene or at admission because of rapid hemorrhage. If the hemorrhage is contained in the chest or if there is the presence of cardiac tamponade (falls), initially, the patient may survive (5). Of the patients who arrive alive to the hospital, 33% will become hemodynamically compromised. The mortality in this latter subgroup (unstable) approaches 100%, whereas the remaining two thirds who are stable result in 25% mortality, even if handled carefully. Most of those deaths will be attributed to other associated injuries (3, 4, 6–8) (Table 2).

The mechanism of injury, ejection, unrestrained occupant, or death at the scene may trigger uncertainty. The astute physician should regard the presence of aortic injury early in the primary and secondary surveys. Chest radiography may or may not reveal a widened mediastinum. In addition, other radiographic signs may be present, such as rightward shift of the trachea, blurring of the aortic knob or descending thoracic aorta, opacification of the juncture between the aorta and pulmonary trunk, and collection of fluid in the left lower thorax. This latter sign may be associated with other injuries, such as bleeding from intercostals or pulmonary vessels (1, 5, 6, 9). These patients may seem stable or have other profound or grave injuries with hypotension (severe traumatic brain injury, crushed limb injuries, vertical shear of the pelvis, hemorrhage into any body cavity [liver laceration or splenic rupture], retroperitoneal bleeding, or any combination of the above), thus diverting attention from an existing aortic injury (5). If there is a positive Focused Assessment with Sonography for Trauma (FAST) examination or affirmative results from computed tomography, it is not unusual that the patient will undergo emergent laparotomy for intra-abdominal hemorrhage, not knowing of the potential of an aortic in-

Table 1. Aortic pathology: trauma

Injury location	%
Proximal descending	54–64
Aortic arch	10–14
Multiple sites	13–18
Distal aorta	12
Diaphragmatic	

Table 2. Aortic pathology: trauma

- Epidemiology
  - Falls, motor vehicle accidents, industrial accidents, blast injuries, domestic violence
  - Blunt and/or penetrating injuries
- Clinical presentation
  - Mortality at scene of 65% to 80%
  - At arrival, 33% become unstable
    - Mortality, unstable: 100%
    - Mortality, stable: 25%

Table 3. Aortic pathology: trauma

- Diagnostic imaging modalities
  - Chest radiography
  - Angiography
  - Computed tomography (CT)
    - Helical CT
    - Multidetector CT
    - 64-slice CT
  - Echocardiography
    - Transthoracic
    - Transesophageal
  - Magnetic resonance imaging

jury pattern. So, when time permits in the perioperative resuscitative portion, transesophageal echocardiography (TEE) should be considered. In addition, the clinician will also interrogate, in real time, ventricular function and volume, ventricular interactions, and other contiguous structures.

TEE is one of the most powerful diagnostic tools immediately available in a portable fashion for the critical care physician in the assessment of the critically ill injured patient, and this tool can follow the patient to various settings. It can be readily available in any situation, as long as there are appropriately trained individuals with immediate access to the equipment and who are clinically inclined to these types of patients (7, 10, 11). There is still some controversy about the overall acceptance of TEE as a first-line diagnostic tool when an aortic injury may be under question, probably because of historical reasons (angiography as a "gold standard," lack of excellent randomized, prospective investigations, and bias) (12). Other imaging modalities are available, such as computed tomography (CT), angiography with or without contrast and with digital subtraction, and magnetic resonance imaging (MRI) (13) (Table 3). All of the imaging tools have their advantages and disadvantages. Timing is of the essence for appropriate care of these patients.

Table 4. Transesophageal echocardiography (TEE) and angiography (aortography or contrast-enhanced spiral computed tomography for traumatic aortic imaging [TAI])

	Sensitivity, %	Specificity, %	NPV, %	PPV, %
Minor TAI (n = 7)				
TEE (n = 208)	100	100	100	100
Angiography (n = 206)	84	100	97	100
Major TAI (n = 33)				
TEE (n = 208)	97	100	99	100
Angiography (n = 206)	97	100	99	100
All TAI (n = 41)				
TEE (n = 208)	98	100	99	100
Angiography (n = 206)	83	100	96	100

NPV, negative predictive value; PPV, positive predictive value.
Adapted from Goarin (10).

Table 5. Transesophageal echocardiography (TEE) and helical chest computed tomography (CT) for the identification of traumatic arterial injuries in severe blunt trauma

	Sensitivity (%)	Specificity (%)	NPV (%)	PPV (%)
Multiplane TEE (n = 106)	93 (68–100)	100 (96–100)	99 (94–100)	100 (77–100)
Helical CT (n = 99)	73 (45–92)	100 (96–100)	95 (89–99)	100 (71–100)

NPV, negative predictive value; PPV, positive predictive value. Reprinted with permission from Vignon et al (20).

The early survival of these patients depends on the initial resuscitation and timeliness of correct diagnostic modalities implemented. In the not-so-distant past, angiography was considered the gold standard for detecting an aortic rupture (2, 3, 7, 10, 11, 14, 15). Fortunately, other modalities came to bear, especially TEE multiplanar technology, with the packaging of pulsed-wave Doppler, continuous-wave Doppler, color flow, tissue Doppler imaging, harmonic imaging, and two-dimensional imaging changing to reconstruction imaging methods for three-dimensional input and eventual strain-rate development. Particularly in the hemodynamically unstable patient, TEE should be the first line of choice of diagnostic methods (7, 10, 11).

The difficulty with CT and MRI is transportation of the critically ill, especially in remote areas, and the time involved (60 to 70 mins). If time permits and the patient can withstand transportation, helical or multidetector CT is also a viable option, rendering similar sensitivities and specificities to that of aortography (13, 16–18) (Table 4). In an investigation by Dyer et al. (4) over a 5½-yr period, in which 1,561 patients were studied for the potential of traumatic aortic injury at two trauma centers, CT found only 30 aortic injuries. They conclude that liberal use of CT should be implemented in these situations. CT examination in this series reveals a 100% nonpredictive value (4, 19). In a smaller prospective series (n = 110), patients with a severity score ≥34 underwent TEE and CT. The results were that imaging modalities correctly identified subadventitial tear (confirmed at surgery) (20) (Table 5).

It is not unusual to use TEE first in the diagnostic schematic, complemented by reconstruction of CT images. MRI views the aorta with excellent quality but should be used only in a stable patient in the chronic phase of the aortic disease state. The entire thoracic aorta is easily visualized by imaging devices, including the use of transthoracic echocardiography (TTE) via the suprasternal notch for the ascending aorta and its arch. The FAST examination is limited and does not include the aorta. The interrogation of the thoracic aorta in the comprehensive TEE examination is crucial. The limitation of TEE is difficulty in interrogation of the distal thoracic aorta and arch vessels. Occasionally, reverberation artifacts become problematic. When this occurs, other modalities such as CT may complement the TEE images (if the patient has time to be transported to radiology). The

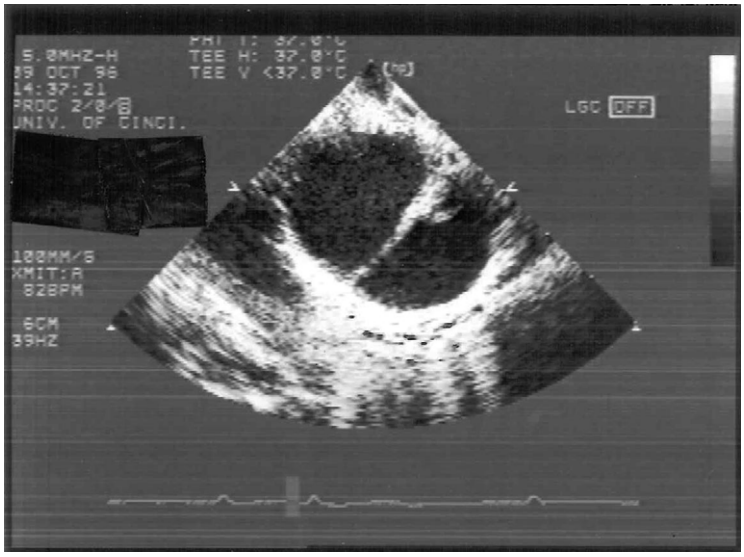

Figure 1. Transection of proximal distal thoracic aorta adjacent to the isthmus.

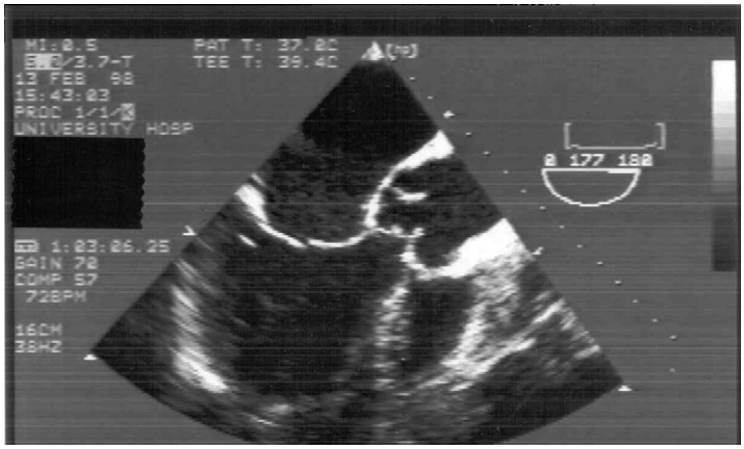

Figure 2. Intimal tear of proximal posterior aorta.

difficulty with CT is that it is not in real time and cannot assess left ventricular performance, valvular integrity, and cardiac hemodynamic factors. The benefit of angiography is the ability to see extravasations, defects of the aortic wall with the presence of aneurysmal dilation. However, it does not have the capability to interpret intraluminal defects. TEE easily identifies the intimal flap, tear, or thrombus. With color flow Doppler, integrity of the aorta and its contents can be assessed, showing flow patterns or turbulence (aliasing) as a sign of a potential injury (21–29).

Numerous studies have shown the extreme benefit of TEE and some of its limitations (2, 7, 11) (Figs. 1 and 2). One investigation overwhelmingly reveals the efficacy of TEE. It is quite evident that when there is a seasoned TEE operative with experience in trauma patients, the results are formidable (30). In this series, they immediately performed TEE and aortography in sequential fashion. The positive predictive value of TEE is 90.9%. A false-negative result did not undergo correction and it is believed that angiography in this case is also nonconclusive (31). Similarly, Vignon et al. (32) studied 32 consecutive patients who exhibited a widened mediastinum with a associated violent deceleration impact. The majority of injury pattern is subadventitial (n = 10) and intimal tears (n = 3). One medial tear was missed by TEE and confirmed at necropsy. The sensitivity and specificity of TEE for the diagnosis of subadventitial tear are 91% and 100%, respectively. A widened mediastinum on the chest radiograph (>8 mm) is a red flag, increasing the suspicion for an aortic injury. In an investigation by Vignon et al. (33), this marker was analyzed. They had two

Table 6. Aortic pathology: diagnostic imaging of trauma with computed tomography

Disadvantages
Time consuming
Transportation to remote area
Not real time
Intraluminal defects not observed
Small, multiple, irregular

groups; one with an enlarged mediastinum and one with a mediastinum of <8 mm. The total group consisted of 40 consecutive patients. They revealed that lack of a widened mediastinum does not obviate the need for a TEE examination. They believe that TEE is really the only first-line tool in these diagnostic dilemmas. One of the benefits of TEE is the availability of other diagnostic tools in real time. A prospective study by Catoire et al. (34) found how efficacious TEE is in these inherently complicated patients (n = 70). Within 48 hrs of admission, a comprehensive examination was completed. Myocardial contusion was identified in 25 patients, invalidating 18 suspected contusions and visualizing five positive cases that were unsuspected. Of importance, pericardial effusions were suspect in one case, and TEE identified 13 unsuspected cases. In 13 patients with a widened mediastinum, aortic injury was excluded in all of them. Hypovolemia was easily detected in seven of these patients in whom it otherwise might not have been appreciated. In one group, 70% of the patients had a new diagnosis confirmed only by TEE. Over an extended period or time, as follow-up (9 yrs), Goarin et al. (10) evaluated TEE in traumatic aortic injury in a substantial cohort (n = 209). Of patients identified with an aortic injury, angiography (aortography, contrast-enhanced CT, or both) was less accurate (sensitivity of 83%, specificity of 100%) than TEE (sensitivity of 98%, specificity of 100%). It became apparent that smaller lesions (intimal flaps) were easily detected by TEE. However, when there were larger lesions, the sensitivities and specificities became more equivalent (Tables 6 and 7).

Despite the apparent overwhelming data for TEE in the diagnosis of traumatic aortic injuries, controversy exists. Fabian et al. (3) and Cinnella et al. (12) do point out some differences. In the 50-center trial by Fabian et al. (3), only 274 blunt aortic injury cases were detected. The majority (81%) of these vascular injuries were attributed to motor vehicle acci-

Table 7. Aortic pathology: Computed tomography for trauma

Parameter	Hematoma Direct Signs	Periaortic Direct Signs	Direct Signs
Patients, n	1346	1346	1346
TN	671	1258	1299
FP	656	69	28
NPV, %	100	100	99.9
Sensitivity, %	100	100	95
Specificity, %	50	95	98
TAI	19	19	19
TP	19	19	18
PPV, %	3	22	39
FN	0	0	1

TN, true negative; FP, false positive; NPV, negative predictive value; TAI, traumatic aortic injury; TP, true positive; PPV, positive predictive value; FN, false negative.

Adapted from Dyer et al (4).

Table 8. Aortic pathology: transesophageal echocardiography for diagnostic imaging of trauma

Disadvantages
Reverberation artifacts
Difficulty in visualizing distal ascending aorta
Difficulty in interrogation arch vessels
Operator dependent
Experience and training
Availability

dents. Of note, only 88 and 30 patients underwent a CT and TEE, respectively. The diagnostic potential was described as 75% and 80%, respectively. In this author's opinion, this is an inherent bias because lack of the number of patients with a comprehensive TEE examination. Also, it seems that operators of TEE, and their experience and expertise, were not controlled and were quite varied (3) (Table 8). However, in the review by Cinnella et al. (12), the controversy is more established, as numerous investigations are critically evaluated. They found 17 relevant TEE studies out of 758 during a time period from 1994 to 2002. They developed several quality scores that met certain criteria. They believe that TEE is a highly useful diagnostic tool for detecting traumatic aortic injury but noticeably equivalent to angiography. However, they did state that smaller lesions and multiple lesions were easily detected with certainty by the TEE method (12). In this author's opinion, I believe this critical appraisal confirms the benefit of TEE. It reaffirms that experience is vital, as is timing of the study, and that TEE complements other diagnostic modalities and should be used in a critical fashion. TEE should still be the first line of defense in the detection of these critical injuries. An echocardiographic team with extensive training (the team does not have to be just cardiologists) and with expertise in TEE should be immediately available. For practical purposes, this could only be possible in level I trauma centers. As imaging tools improve in quality (64-slice CT), the diagnostic scheme may alter, but if timing is of the essence, particularly in critically ill multiple trauma patients who may be unstable, it behooves the clinician to bypass TEE.

Aortic Atheroma

Echocardiography is a valuable tool in the evaluation of aortic atheroma. Interestingly, most of the TEE/TTE studies are performed on patients who already have had an event (stroke, traumatic aortic injury, or systemic emboli) necessitating evaluation. Occasionally, aortic atheroma is detected incidentally on TEE performed to evaluate other pathologic conditions.

However, this entity is largely underdiagnosed, as shown by the Stroke Prevention: Assessment of Risk in a Community (SPARC) study (35). This investigation enrolled 588 randomly sampled subjects (average age of 66.9 yrs) from 1993 to 2000 who underwent medical record review, home medical interview, TEE, carotid ultrasound, and repeated blood pressure measurement. The incidence of detecting a simple aortic plaque (<4 mm) in the ascending arch and descending aorta was 43.7%. Of those, 29.9% were located in the ascending aorta and the aortic arch. The presence of complex plaque (mobile or ≥4 mm) was 7.6% in the ascending arch and descending aorta, with 2.4% located in the ascending aorta and aortic arch (35).

Classification of Grades of Atheroma. The importance of detecting aortic atheroma is based on its association with other significant diseases that might be encountered in the intensive care unit (Fig. 3).

Stroke is one of the leading causes of morbidity and mortality in the United States. Many studies examined the association between aortic atheroma and stroke (36–38). One of these studies found that atheroma in the ascending aorta and aortic arch was a significant and independent risk factor for cerebral ischemia. The odds ratio for a cerebrovascular event in the presence of simple atheroma was 2.3, and for complex atheroma (≥5 mm, mobile or ulcerated surface), it was 7.1 (36).

A French study (38) of aortic plaques showed that, in the stroke group, aortic atheroma of ≥4 mm was an independent risk factor for recurrent brain infarction after adjusting for other factors. These patients had a significantly higher incidence of recurrent brain infarction of 11.9 per 100 person-years in patients with aortic atheroma of ≥4 mm in the ascending aorta and proximal arch, as compared with 3.5 per 100 person-years in patients with aortic atheroma of 1–3.9 mm and 2.8 per 100 person-years in patients with aortic atheroma of <1 mm ($p < .001$).

In the same study (38), the presence of aortic atheroma of ≥4 mm was associated with all vascular events (stroke, myocardial infarction, peripheral embolism, and death from vascular causes) at an incidence of 26 per 100 person-years.

Furthermore, the presence of aortic atheromatous plaque on TEE has been correlated with a higher prevalence of coronary artery disease (39), and the lack of aortic plaque with TEE has been shown to predict the absence of coronary artery disease (40).

Patients with significant carotid stenosis also have a higher prevalence of aortic atheroma (41). The incidence of abdominal aneurysm is ten times higher in patients with severe thoracic aorta atherosclerosis (41).

However, the proper treatment for this condition is unknown so far. It was found in a large retrospective, nonrandomized analysis that only statins showed a significant reduction in recurrent strokes and embolic events. Interestingly, the use of warfarin or antiplatelet agents was not beneficial in this study (42).

In another study, the detection of aortic thrombi by TEE changed the secondary prevention to oral anticoagulation for ≥4–6 wks in patients with acute brain ischemia; later in the course of the dis-

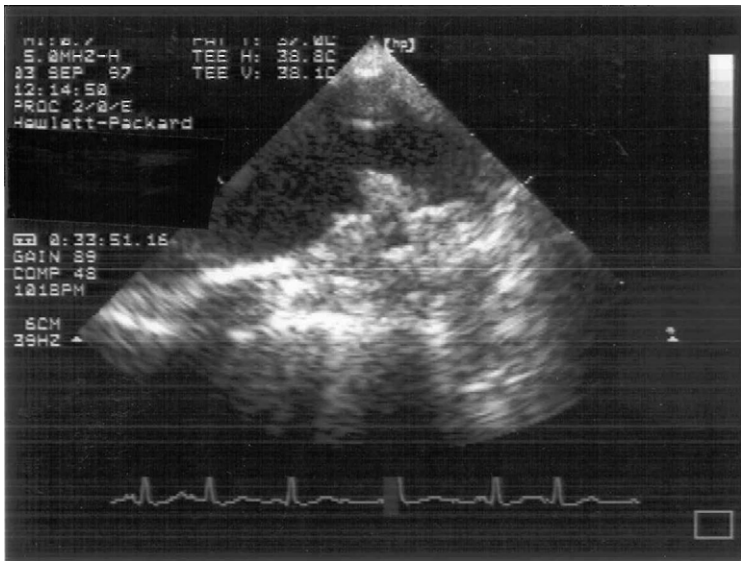

Figure 3. Large free-flowing thrombus with plaques on the anterior arch.

ease, platelet inhibitors and statins were used for plaque stabilization (43). The ongoing Aortic arch Related Cerebral Hazard (ARCH) trial, comparing clopidogrel plus aspirin vs. warfarin, will contribute substantial information for the optimal treatment (43).

Thoracic Aortic Dissection

Aortic dissection can be associated with a high mortality rate, especially if not diagnosed and treated rapidly. In the following section, the etiology, classification, and diagnosis of aortic dissection will be discussed.

All of the causes that lead to aortic aneurysms, as discussed before, can lead to weakening of the aortic wall, with resultant dissection or rupture. Other causes include trauma and iatrogenic dissection, such as after cardiac catheterization or aortic cannulation and manipulation during heart surgery.

There are two widely known classifications, Stanford and De Bakey. Stanford type A includes dissection that involves the ascending thoracic aorta, whereas type B dissection does not involve the ascending thoracic aorta. De Bakey type I dissection involves the whole aorta, type II dissection involves the ascending aorta, and type III is a dissection of the descending aorta. Thus, Stanford type A dissection includes De Bakey types I and II, and Stanford type B equals De Bakey type III. Because intramural hematoma, intramural hemorrhage, and aortic ulcer may be a sign of evolving dissection, a new differentiation has been proposed (44) (Fig. 3).

Class 1: Classic Aortic Dissection. Aortic dissection is characterized by the presence of an intimal flap that separates between the true and false lumen. This can further divide into communicating and noncommunicating dissection. Communicating dissection has an intimal tear with unidirectional or multidirectional flow between the true and false lumen. On the other hand, noncommunicating dissection, no flow, and no intimal tear can be detected. That dissection can spread either in an antegrade fashion, with involvement of the distal part of the aorta, or extend to different branches, like the carotid, subclavian, and renal. It can also spread in a retrograde fashion to involve the coronaries (44).

Class 2: Intramural Hematoma/Hemorrhage. Intramural hematoma/hemorrhage may be the result of rupture vasa vasorum and may be the initial lesion in cases of cystic medial degeneration. It often coexists with or progresses to class 1 dissection. It is present in 10% to 15% of patients with suspected aortic dissection (44, 45) (Fig. 1).

Class 3: Subtle-Discrete Aortic Dissection. This form of dissection is characterized by a stellate or linear intimal tear with the exposure of the underlying media and adventitia but without progression to separation of medial layers. Svensson et al. (46) described the occurrence of this rare type of dissection in nine patients with suspected aortic dissection, but diagnostic tests failed to diagnose it, including TEE, CT, and MRI. They concluded that aortography should be done if dissection is still a highly likely diagnosis, but other noninvasive tests were negative. In aortography, the dissection may look like an eccentric bulge (Fig. 2).

Class 4: Plaque Rupture and Ulceration. Ulceration of the aortic plaques can lead to aortic dissection or aortic rupture. The natural history and progression of aortic ulcer and the treatment is of controversial issue. In a symptomatic patient, it might be similar in presentation to other causes of acute chest pain, or it might be asymptomatic and diagnosed accidentally (47) (Fig. 3).

Class 5: Traumatic and Iatrogenic Aortic Dissection. Blunt trauma may cause dissection at the level of aortic isthmus. Iatrogenic dissection may be seen after aortic angioplasty for aortic coarctation or after cross-clamp of the aorta during heart surgery (44).

Clinical Presentation and Diagnosis. Acute sudden onset of severe pain is the typical manifestation of aortic dissection, but a wide variety of symptoms can be present. The patient may have symptoms suggestive of congestive heart failure, stroke, shock, or loss of distal pulse (Table 9).

Other features might include diastolic murmur of aortic regurgitation and neurologic deficits. If the dissection causes bleeding into the pericardium, distant heart sounds secondary to pericardial effusion may be noted, and symptoms and signs of tamponade may be seen in extreme cases.

Early diagnosis is critical because early intervention can decrease the mortality rate, which is estimated to be 1–2% per hour in the first 48 hrs of ascending aortic dissection (44).

The most common modalities for diagnosing aortic dissection include TEE/TTE, helical CT, and MRI (Table 10). Aortography is another method for making the diagnosis, but it is invasive and requires the use of contrast. The utility is also limited in critically ill and unstable patients. Nevertheless, it remains the method of choice in diagnosing class 3 dissection, as previously mentioned. Each test has its advantages and limitations and must be evaluated carefully in the context of the patient's medical condition, need to transfer outside the intensive care unit, feasibility, and cost-effectiveness.

Table 9. Demographics and history of patients (n = 464) with acute aortic dissection

Variable	n (%)	Type A n (%) (n = 289)	Type B n (%) (n = 175)	*p* Value Type A vs. B
Patient history				
Hypertension	326/452 (72.1)	194 (69.3)	132 (76.7)	.08
Atherosclerosis	140/452 (31.0)	69 (24.4)	71 (42)	<.001
Previous cardiac surgery	83 (17.9)	46 (15.9)	37 (21.1)	.16
Previous aortic aneurysm	73/453 (16.1)	35 (12.4)	4 (2.3)	.006
Previous aortic dissection	29/453 (6.4)	11 (3.9)	18 (10.6)	.005
Diabetes	23/451 (5.1)	12 (4.3)	11 (6.6)	.29
Marfan syndrome	22/449 (4.9)	19 (6.7)	3 (1.8)	.02

Adapted from Ince H, Nienaber CA: Diagnosis and main management of patients with aortic dissection. *Heart* 2007; 93:266–270.

Table 10. Aortic pathology: dissection and aneurysm

Variable	Aortography	CT	MRI	TEE
Sensitivity	**	**	***	***
Specificity	***	***	***	**/***
Site of intimal tear	**	*	***	*
Thrombus	***	**	***	*
Aortic insufficiency	***	—	*	***
Pericardial effusion	**	***	***	***
Branch vessel involvement	***	*	**	*
Coronary artery involvement	**	—	—	**

CT, computed tomography; MRI, magnetic resonance imaging; TEE, transesophageal echocardiography.

Asterisks, gradation of importance, sensitivity, or detection.

Table 11. Aortic pathology: dissection and aneurysm

Echocardiographic characteristics
Small true lumen
Larger false lumen
Intimal flap
Entry site, single vs. multiple
Presence of thrombus in false lumen
Intramural hematoma, rupture of vasa vasorum within aortic media

In the Task Force on Aortic Dissection study by the European Society of Cardiology and others (44), they found that most centers use an average of 1.8 methods to diagnose aortic dissection, revealing the uncertainty of this potentially life-threatening disease.

A recent meta-analysis by Shiga et al. (48) reviewing published studies of the diagnosis of aortic dissection by TEE, helical CT, and MRI showed that tests yield clinically equally reliable diagnostic values. TEE had a 99% sensitivity and 95% specificity, helical CT had a 100% sensitivity and 98% specificity, and MRI had a 98% sensitivity and 98% specificity.

Echocardiography TTE and TEE. Echocardiography is a bedside test that is simple to perform, with proven value and accuracy. It might be the most appropriate test for a patient who is hemodynamically unstable. It also provides important information regarding the function of the heart, presence of pericardial effusion, involvement of the aortic valve, and aortic regurgitation.

Other complications of aortic dissection, such as mediastinal hematoma, might show as echo-free space between the esophagus and left atrium during TEE exam. Pleural effusion may also develop.

The TTE can only evaluate the ascending aorta and arch, but it might be difficult to visualize in mechanically ventilated patient. On the other hand, TEE will provide better images to the ascending, arch and descending aorta but will not visualize the distal ascending aorta because of the presence of the left main bronchus in between (Table 11). It is important to differentiate between different classes of aortic dissection because treatment and prognosis vary accordingly. For example, classic type A dissection needs rapid surgical intervention, whereas classic type B will need medical management. It is important to localize the tear, if possible, because the main goal of intervention is to occlude the entry point. By two-dimensional echocardiography, the intimal flap, the point of entry, and true and false lumens can be easily seen. The true lumen is usually smaller, expands during systole, and by the use of color flow, it has faster flow compared with the false lumen, which might be larger in size, become compressed during systole, and may have slow or no flow, as evident by presence of smoke or thrombus (Fig. 4). Another limitation of echocardiography is the inability to evaluate extension to the abdominal aorta or other branches.

Helical CT. Helical CT is the most widely used method for diagnosis, with very high sensitivity and specificity. In a meta-analysis, helical CT was found to have the highest negative predictive value, making it the best test to rule out aortic dissection (48).

MRI. MRI has the highest accuracy and sensitivity for detection of all types of dissection, with the exception of class 3, which is only diagnosed with aortography (44, 46, 48).

Problems with MRI include patient hemodynamic instability, length of time needed to do the study, many incompatibility problems, and contraindications like pacemakers. It is also expensive and not readily available at all times and in all centers.

Aortic Aneurysms

Aortic aneurysmal disease is a condition that carries significant risk of morbidity and mortality. Aortic aneurysms are considered to be one of the major causes of mortality in the western countries (ranked 13th). The incidence of aortic aneurysms is estimated to be 4.5 per 1,000 (49).

Definition and Classification. An aortic aneurysm is a localized dilation of the aorta. There are two types of aneurysms: a true aneurysm, in which the dilated segment involves all three layers of the vessel, and a false or pseudoaneurysm, which is a contained hematoma outlined by adventitia or surrounding tissue. Aortic aneurysms can be further classified according to their morphology into fusiform or saccular.

Aneurysms can affect different locations of the aorta: the aortic root, ascending aorta, aortic arch, or the descending aorta. Sixty percent of thoracic aneurysms involve the aortic root or the ascending aorta, 40% involve the descending aorta, 10% involve the arch, and 10%

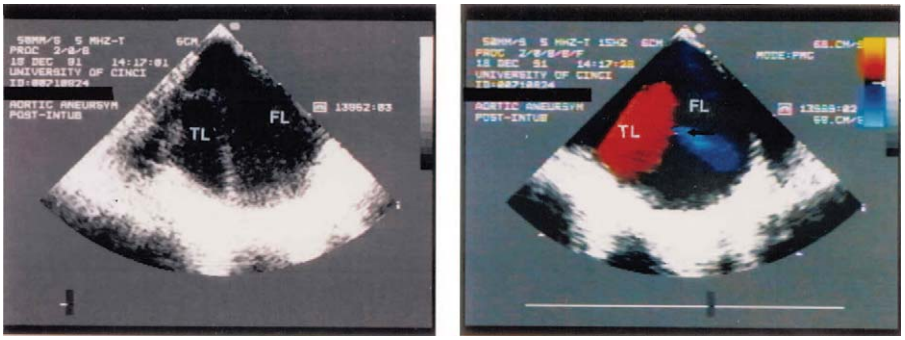

Figure 4. *Left*, aortic dissection with intimal flap and entry site of distal thoracic aorta; *Right*, aortic dissection renal flow via color flow Doppler through intimal tear. *TL*, true lumen; *FL*, false lumen.

are thoracoabdominal aorta, with some involving more than one segment (50).

Etiology. There are a variety of conditions that lead to weakening of the aortic wall (most importantly the media), causing aneurysmal formation. Interestingly, the etiology may differ depending on the location of the aneurysm. The most common cause of the descending aortic aneurysm is atherosclerosis, whereas the etiology for aortic root aneurysm could be associated with connective tissue disorders like Marfan syndrome, Ehlers-Danlos syndrome, and bicuspid aortic valve disease. Other etiologies include infectious, inflammatory, trauma, dissection, and idiopathic (49).

Marfan syndrome is an autosomal dominant connective tissue disorder with a mutation in one of the genes for fibrillin-1, which is the major component of elastin. Ehlers-Danlos syndrome is another connective tissue disorder and is associated with aortic aneurysm, articular hypermobility, skin hyperextensibility, and tissue fragility (44, 50). Familial thoracic aortic aneurysm syndrome is associated with cystic medial degeneration of the aorta but no other connective tissue abnormalities. It is believed to be an autosomal dominant disorder, with marked variability in expression and penetration (44, 50). Bicuspid aortic valve is also associated with aortic aneurysmal formation. Not only is it related to poststenotic dilation, but interestingly, it was found that up to 52% of patients with a normally functioning bicuspid aortic valve have aortic dilation (51). Inadequate production of fibrillin-1 is thought to be the underlying pathology (50).

Inflammation could also lead to aneurysm formation. Syphilitic aortitis is somewhat rare now, but this used to be a common cause of aortic root and ascending aortic aneurysms. Other inflammatory disorders include Takayasu arteritis, which occurs more in women with a mean age of 29 yrs. It mainly causes obstructive lesions, but aortic dilation is present in 15% of cases. Giant-cell arteritis is another rare disease, which commonly affects the temporal artery but also may affect the ascending aorta.

Trauma may lead to dissection or transection, with subsequent formation of pseudoaneurysm, which commonly affects the descending aorta distal to the origin of the left subclavian artery. Chronic dissection can dilate with time, causing aneurysmal formation (44, 49, 50).

Natural History of Aortic Aneurysms. Aortic aneurysm is usually a progressive disease that needs to be monitored closely or treated by intervention. As aneurysms grow in size, there is an increased incidence of rupture, dissection, and death. Ascending aortic aneurysms will grow an average of 1–4 mm every year, but in patients with bicuspid aortic valve and Marfan syndrome, the rate of growth is more rapid (49). After aneurysmal size exceeds 6 cm, the risk for rupture and dissection is $\geq$6.9% per year and risk of death is 11.8% per year (52).

Clinical Manifestations. Most patients are asymptomatic, and the aneurysm is discovered incidentally by chest radiography, echocardiography, or CT. Aortic aneurysms may manifest with symptoms late in the course of the disease.

Progressive dilation of the ascending aneurysm may cause dilation of the aortic annulus, with resultant aortic regurgitation. This represents a significant volume overload on the left ventricle, resulting in progressive left ventricular dilation and failure. Compression of the adjacent structure may lead to chronic chest pain, but when a patient experiences a new onset of severe chest pain, this may be an early indication for dissection.

Role of Echocardiography in the Evaluation of Aortic Aneurysms. Transthoracic two-dimensional echocardiography is very effective in evaluating the aortic root, but the mid and distal ascending aorta, aortic arch, and the descending aorta are not seen. Thus, it is a valuable and useful noninvasive test to evaluate and follow up on patients with an aortic root aneurysm (e.g., Marfan syndrome). It also provides important information on aortic valvular regurgitation and on the function and dimensions of the left ventricle.

Newer technology might improve the visualization of the thoracic aorta, as detailed in case report of visualization of a mycotic descending aneurysm using three-dimensional transthoracic echocardiography (53). TEE allows better visualization of the aortic root, ascending aorta, aortic arch, and descending aorta. Images are usually accurate, with the exception of part of the aortic arch and the distal part of the ascending aorta due to the position of the left bronchus between the probe and aorta. TEE allows near-complete evaluation of the aorta due to the proximity of the high-frequency probe to the aorta. Images are far superior to TTE, especially in intubated patients or patients with severe pulmonary disease and poor echocardiographic windows. TEE also provides significant data on cardiac structural abnormalities, valvular disorders, and cardiac function. It is a semi-invasive test that can be performed at the bedside in the critical care unit or in the operating room, without having to transfer unstable patients, unlike other imaging modalities. Disadvantages of TEE include being invasive, need for an experienced operator, and the inability to perform the test in patients with significant esophageal pathology.

There are other imaging modalities for aortic aneurysmal disease. Helical CT and CT angiography allow for the full evaluation of the entire aorta and the extension of the aneurysm. They are also superior in evaluating the aortic branches. Three-dimensional reconstruction allows optimal measurement of the aneurysm size, especially when the aorta is tortuous. Another important tool that CT can provide is the visualization and localization of the artery of Adamkiewicz, which supplies the anterior spinal artery. Most causes of paraplegia after repair of thoracoabdominal aneurysm or dissection are related to interrupted blood supply to the anterior spinal artery. Detection of the artery of Adamkiewicz can help in the surgical decision and may

prevent postoperative paraplegia. In one study, Takase et al. (54) was able to detect it in 63 of 70 patients (90%). The main disadvantage of CT is the need for contrast administration and radiation exposure. It is not the optimal test for critically ill patients who are not stable for transfer to the radiology suite.

MRI is another modality for evaluating aortic aneurysms. It is very sensitive and requires no radiation exposure, but it is expensive and time consuming and might not be suitable in critically ill patients. It is also possible to detect the artery of Adamkiewicz with MRI angiography with great sensitivity. In a study by Hyodoh et al. (55) they were able to detect the artery of Adamkiewicz in 42 of 50 patients (84%), which led to a change in the surgical approach to preserve the artery. In the 42 patients, there was no postoperative paraplegia. Of the other eight patients in whom the artery of Adamkiewicz was not visualized, two had postoperative paraplegia.

Finally, chest radiography may show enlarged cardiac silhouette, aortic knob, and tracheal deviation by the large aneurysm, but smaller aneurysms will not be shown on radiography.

Sinus of Valsalva Aneurysm

Sinus of Valsalva aneurysm (SVA) is a rare cardiac anomaly, which may be either congenital or acquired. Congenital SVAs may be attributed to lack of continuity between aortic media and annulus fibrosis (56). Acquired aneurysms may result from trauma (57), infection, endocarditis (58, 59), syphilis, Marfan syndrome, and senile dilation. A supracristal ventriculoseptal defect may frequently coexist with the right SVA (60). Dilation of all three sinuses of Valsalva may be seen with aging, Marfan syndrome, syphilis, and other connective tissue diseases.

Aneurysm of one or two of the sinuses is unusual and may be related to congenital or, more commonly, endocarditis. The right coronary sinus is involved most often, followed by the noncoronary sinus, and least frequently is the left coronary sinus (60).

The natural history of the SVA is hard to study due to the rarity of the disease, but it can either present with or without rupture (60, 61). A nonruptured SVA may remain asymptomatic but might be complicated with right ventricular outflow obstruction, infective endocarditis, arrhythmias and myocardial infarction, and ischemia due to distortion of the coronary Ostia or compression of the coronary trunk. Progressive dilation may lead to aortic regurgitation. There may be associated symptoms of dyspnea, chest pain, syncope, or congestive heart failure.

An intracardiac rupture of the aneurysm into the right atrium, right ventricle, or both right atrium and ventricle is possible, leading to shunting with progressive congestive heart failure; also, the aneurysm might dissect into the interventricular septum (62), which may result in complete heart blockage, requiring a permanent pacemaker. An extracardiac rupture of the aneurysm into the pericardium might be fatal.

An SVA (ruptured or nonruptured) can be seen with TTE (transthoracic echocardiography) parasternal short-axis view or TEE aortic valve short-axis view. A nonruptured SVA will be seen as a thinner wall and is larger than other sinuses. If ruptured, color Doppler will show a continuous turbulent jet within the aneurysm into the receiving chamber. If the aneurysm ruptured into the right atrium, the flow will be continuous during both systole and diastole. If it ruptured into the left ventricle, the flow will occur only during diastole. Other features might include volume overload in the right atrium and ventricle in the case of right sinus of Valsalva to right atrium shunt (the most common). In cases of left SVA to left ventricle shunt, the volume overload will be seen in the left ventricle. An associated ventriculoseptal defect may also be seen with the SVA.

The treatment options are beyond the scope of this article, but the main strategies for treatment include:

Observe (noncomplicated asymptomatic cases) for this case, follow-up with echocardiography to evaluate the size of the aneurysm is needed.

Catheter closure, which is done with the help of fluoroscopy and TEE (63–66).

Surgical treatment, for which echocardiography is needed to look for the associated lesion before surgery and to make sure the aneurysm is adequately treated and no further shunt exists after bypass.

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