Aortic Emergencies

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KEYWORDS

- Aortic dissection • Abdominal aortic aneurysm
- Thoracic aortic aneurysm

Patients with aortic emergencies are some of the highest acuity patients that the Emergency Medicine (EM) physician encounters. These emergencies are divided into 2 primary groups: those related to aortic dissection and those related to an abdominal aortic aneurysm (AAA). Thoracic aortic aneurysms without dissection comprise a smaller subset of patients with aortic emergencies. Because there are varying presenting complaints, these diagnoses can be challenging to make, and a missed diagnosis often leads to significant morbidity and mortality. This article discusses the clinical presentations, available diagnostic tools, and treatment considerations of aortic dissection, AAA, and thoracic aortic aneurysm.

AORTIC DISSECTION
Causes and Risk Factors

Acute aortic dissection occurs when there is a tear in the aortic intima, resulting in separation between the aortic intima and the aortic media. Blood flows into this space, creating the false lumen. The initial tear may propagate proximally and/or distally and affect any arteries branching from the aorta, resulting in varied clinical presentations. Because of this, as well as the relative infrequency of the diagnosis, aortic dissection is a diagnosis that can be challenging for the emergency physician.

Several risk factors have been associated with aortic dissection.[1] These include:

- Hypertension
- Stimulant use
- Trauma
- Genetic conditions including Marfan syndrome, Ehlers-Danlos syndrome, bicuspid aortic valve
- Inflammatory vasculitides including Takayesu arteritis, giant cell arteritis, and Behçet arteritis

- Family history of aortic disease
- History of recent aortic manipulation
- History of known thoracic aortic aneurysm.

Clinical Presentation

The most common presenting symptoms of an aortic dissection are chest pain and/or back pain.[2] Chest pain that is sudden in onset, tearing in quality, or of severe intensity suggests an aortic dissection.[3] In addition to chest and/or back pain, hypertension is a common finding at the time of presentation and is seen in greater than two-thirds of patients.[2] Additional signs and symptoms that may be present include:

- Abdominal pain
- Migrating pain
- Pulse deficit
- Focal neurologic deficit
- Diastolic murmur (aortic insufficiency)
- Flank pain.

Although a pulse discrepancy between extremities is classically associated with aortic dissection, this sign has been found to have a sensitivity of only 31%. When it is present, though, it strongly suggests aortic dissection. Focal neurologic complaints also suggest dissection when present, but the lack of neurologic deficits does not help to exclude the diagnosis.[3]

Classification

Aortic dissections are classified by their anatomic location. Aortic dissections of the ascending aorta are twice as common as those involving the descending aorta. There are 2 different classification systems for aortic dissections (**Fig. 1**). One classification system is the Debakey system, which divides aortic dissections into 3 types based on the origin of the intimal tear:

- Type I: originates in the ascending aorta and involves both the ascending and the descending aorta
- Type II: originates in and involves only the ascending aorta
- Type III: originates in and involves only the descending aorta.

The Stanford system divides aortic dissections into 2 groups based on involvement of the ascending aorta, correlating with the likely treatment course:

- Type A (proximal): involves the ascending aorta with or without involvement of the descending aorta (usually surgical management)
- Type B (distal): involves only the descending aorta (usually medical management).

Diagnostic Modalities

Chest radiography

Chest radiography is easily obtained in the emergency department and is often one of the first tests available in the evaluation of a patient with aortic dissection. The presence of a widened mediastinum (>8 cm) is concerning for dissection (**Fig. 2**). Other findings include an abnormal aortic contour, the calcium sign (separation of calcific intima from outer aortic soft tissue), left pleural effusion, and depression of the left mainstem bronchus. Abnormalities on chest radiography are present in greater than 80% of patients with aortic dissection.[4]
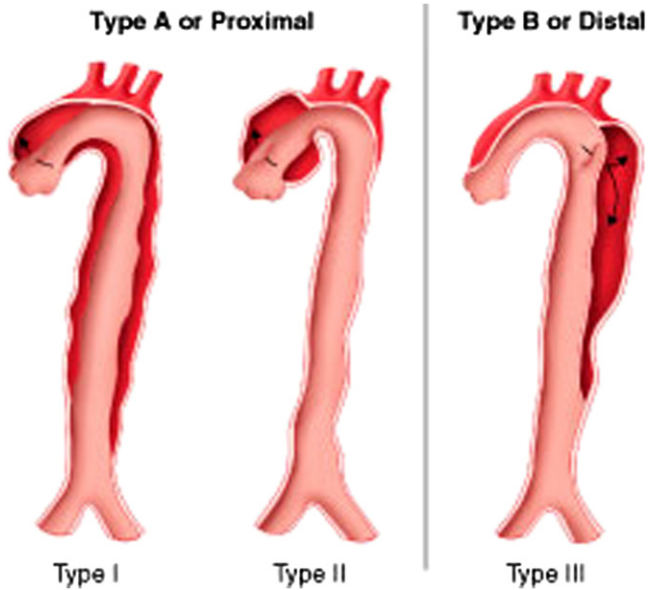
Type A or Proximal

Type B or Distal

Type I

Type II

Type III

Fig. 1. Classification of aortic dissection by the Stanford system (labels at top) and Debakey system (labels at bottom). (*From* Isselbacher EM. Diseases of the aorta. In: Braunwald E, Zipes DP, Libby P, et al, editors. Braunwald's heart disease: a textbook of cardiovascular medicine. 7th edition. Philadelphia: Saunders; 2004. p. 1416; with permission.)

Computed tomography imaging

Computed tomography (CT) scans are widely available, and CT with angiography (CTA) has become the imaging modality of choice for the evaluation of stable patients with acute aortic dissection. Advantages of CT scans include the ability to evaluate the location of the dissection flap and to aid in operative planning for ascending aortic dissections. **Fig. 3** illustrates a Stanford type A dissection. The disadvantage of the CTA is that it requires a contrast bolus, which is not ideal for patients with renal

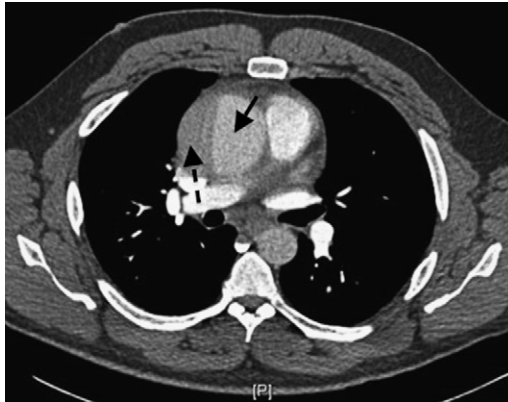**Fig. 2.** A widened mediastinum in a patient with aortic dissection.

Fig. 3. Axial image from a chest CT scan showing an aortic dissection involving the ascending aorta. Both the true lumen (*solid arrow*) and false lumen (*dashed arrow*) are visible.

insufficiency. In addition, unstable patients may not be able to leave the emergency department bay for the study and other modalities must be considered.

Transesophageal echocardiography
Transesophageal echocardiography (TEE) can be performed at the bedside, which makes it an ideal study for the evaluation of unstable patients. Although extremely sensitive and specific in experienced hands, TEE is operator dependent. It has the advantage compared with CT imaging of being able to evaluate the aortic root to assess for acute aortic insufficiency associated with the dissection.

Other imaging modalities
Magnetic resonance imaging (MRI) is sensitive for the identification of an aortic dissection but is less often used to make the diagnosis. Many patients with suspected aortic dissection are not stable enough for MRI, and MRI availability is variable. The advantages associated with MRI are the lack of radiation and the ability of MRI to detail the location and extension of the dissection.

Transthoracic echocardiography (TTE) may be able to visualize an aortic dissection, but TTE is not sensitive for this diagnosis and should not be used on its own to rule out the disease process. TTE is often used in combination with TEE for better visualization of the aortic arch (there is a blind spot in the distal portion of the aortic arch on TEE). **Fig. 4** depict an image obtained with TTE correlating with the CT image in **Fig. 3**.

Laboratory studies
There is no universally accepted biomarker or assay to diagnose or rule out aortic dissection. The D dimer assay has been suggested as an option to rule out low-risk patients for aortic dissection, much as it is used to rule out low-risk patients for pulmonary embolism. Multiple studies have shown increased D dimer levels in patients with aortic dissection.[5–8] Meta-analyses have shown high sensitivity of the D dimer in identifying patients with aortic dissection, ranging from 94% to 97%, with lower specificity.[9,10] A subset of patients with aortic dissection who have a thrombus in the false lumen and a short dissection length have been noted to have negative D dimers.[11] Although it is appealing to avoid the need for advanced imaging studies to exclude the
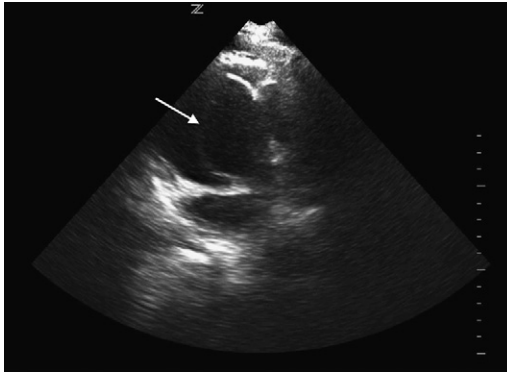
Fig. 4. Transthoracic ultrasound image of the same patient as in **Fig. 3**. The arrowhead points to the dissection flap.

diagnosis of aortic dissection, it is unclear at this time whether the D dimer can do this for the diagnosis of aortic dissection.

Decision rules

One of the challenges in evaluating patients for aortic dissection is that there have been no well-established decision rules to help categorize patients as low risk for aortic the way the Wells criteria do for the evaluation of patients with suspected pulmonary embolism. In 2010, a guideline was published for the evaluation and treatment of patients with thoracic aortic disease.[1] Within this guideline, the investigators present a risk-assessment tool to identify patients as low, moderate, or high risk for aortic dissection based on the presence of high-risk conditions, high-risk pain features, and high-risk examination features. Within the pathway, patients classified as moderate or high risk have aortic imaging.

Recently, this risk-assessment tool was applied to the International Registry of Acute Aortic Dissection and was found to have a sensitivity greater than 95%.[12] However, 4.3% of patients in this registry with known aortic dissection would have been classified as low risk and potentially would have been missed. It is also unclear how the tool will perform in an undifferentiated patient population with suspected aortic dissection. Prospective investigation may shed further light on the general applicability of this tool.

Treatment

Medical

All patients with aortic dissections require aggressive blood pressure and heart rate control to limit shear force on the aorta, which can lead to propagation of the dissection. The target systolic blood pressure is 100 to 120 mm Hg and goal heart rate is 60 beats per minute.[13] There are several classes of medications that are used to reach these targets.

β-Blockers β-Blockers are first-line therapy for aortic dissection because of their combined effects in lowering both blood pressure and heart rate. Esmolol is a good choice given that it is a short-acting agent and can be titrated to effect (starting dose 500 μg/kg bolus, followed by infusion at 50 μg/kg/min; rebolus and increase drip rate by 50 μg/kg/min every 4 minutes until target vital signs have been reached). If esmolol is not available, labetolol is an alternative choice.

Nitroprusside Once the heart rate has been well controlled, vasodilators such as nitroprusside can be added if additional blood pressure reduction is needed. Nitroprusside acts by reducing both preload and afterload. It is important that nitroprusside is only added after β-blockers because it can cause reflex tachycardia when used independently, thereby increasing aortic stress and potentially resulting in a worsening dissection. Nitroprusside is given as a continuous infusion at a rate of 0.5 to 3 μg/kg/min. Higher doses should be avoided because of the risk of cyanide toxicity.

Calcium channel blockers Although calcium channel blockers such as verapamil or diltiazem are not commonly used in the medical management of aortic dissection, they can be substituted if the patient has a contraindication to β-blocker administration.

Surgical

Most patients with an aortic dissection involving the ascending aorta require surgical intervention. If cardiac surgery is not available at the diagnostic center, patients require emergent transfer to a tertiary care hospital. While awaiting surgical intervention or transfer, it is crucial to continue aggressive medical management. Some patients with a descending aortic dissection are also considered for surgical intervention, including those with aortic rupture or evidence of visceral or limb ischemia. Older patients (>70 years) and those with preoperative shock have been shown to have higher surgical mortality.[14]

Other interventions

Endovascular stenting is a newer intervention used in the treatment of aortic dissection, particularly as an alternative to surgery for patients with Stanford type B aortic dissections with evidence of limb or visceral ischemia. Its feasibility was first studied in the 1990s.[15,16] Refractory or recurrent pain and/or refractory hypertension are considered poor prognostic indicators for survival in patients with descending aortic dissections. Investigators of the International Registry of Acute Aortic Dissection (IRAD) studied this population and showed that these patients have lower mortalities with endovascular intervention compared with medical management alone.[17] For patients with ruptured descending thoracic aortic aneurysms, a recent meta-analysis showed lower 30-day mortality for patients treated with an endovascular approach versus open surgery (19% vs 33%).[18]

 An alternative to endovascular stenting, aortic fenestration may help to restore perfusion to patients with evidence of end-organ injury. With this procedure, communication is established between the true and false lumens of the aorta to allow blood flow to arteries originating from the false lumen. Surgical aortic fenestration has been suggested as an alternative option to aortic replacement as well as in the case of contraindicated or failed endovascular stenting.[16,19,20]

AAA

Causes and Risk Factors

A ruptured AAA is a catastrophic cardiovascular condition with high morbidity and mortality.

 Risk factors for AAA include

- Age greater than 60 years
- Male sex
- Tobacco use
- Family history of AAA
- History of heart disease or peripheral vascular disease
- Hypertension.

Clinical Presentation

Unruptured aneurysms are often asymptomatic, resulting in a diagnosis that is challenging to make. Multiple studies have evaluated the mortality benefit and cost-effectiveness of routine screening for AAA, with conflicting results.[21–28] The US Preventive Services Task Force recommends screening men aged 65 to 75 years with a history of smoking. The task force recommends against screening men without a smoking history or women because of the low incidence of AAA in these groups.[29]

Patients with a ruptured AAA usually present with severe abdominal pain. Other symptoms that may also be present include back or flank pain, hypotension, and syncope. The triad of syncope, abdominal pain, and hypotension is highly suggestive of a vascular catastrophe. A patient with a history of a prior AAA presenting with a catastrophic gastrointestinal bleed suggests the development of an aortoenteric fistula.

Physical examination can be limited for the diagnosis of an unruptured AAA. A pulsatile abdominal mass may be palpable. This finding increases in sensitivity with enlarging aneurysm size as well as with smaller abdominal girth.[30,31] One study has noted that the sensitivity of abdominal palpation increases from 29% for AAAs between 3.0 and 3.9 cm, to 50% for AAAs between 4.0 and 4.9 cm, to 76% for AAAs 5.0 cm or greater.[30] Once the AAA ruptures, abdominal tenderness is common. However, in the absence of hemodynamic instability, the diagnosis can remain challenging, which was shown in a review of a group of patients with ruptured AAA presenting to internists. In 61% of cases, the diagnosis was initially missed and only identified once there was hemodynamic compromise.[32]

Diagnostic Modalities

Ultrasonography is one of the most readily available modalities for the evaluation of patients with suspected AAA. It has a high sensitivity and specificity, making it an ideal test for both diagnosing and following an AAA. A normal-diameter aorta is defined as being smaller than 3.0 cm. When evaluating the aorta with ultrasound, it is important to obtain measurements at multiple levels and to include both axial and longitudinal views.

Although traditionally performed by ultrasound technicians and interpreted by radiologists, there have been multiple studies evaluating the use of bedside ultrasound by the emergency physician. Results suggest that, with minimal training, emergency physicians can identify AAAs with high sensitivity and specificity.[33,34] Advantages of performing ultrasound by emergency physicians for the diagnosis of AAA include the ability to make a rapid diagnosis at the patient's bedside as well as the ability to make the diagnosis in settings with limited ultrasound technician and radiology support. **Fig. 5** illustrates an AAA identified with bedside ultrasound in the emergency department.

CT scan imaging is an alternative to ultrasonography for the diagnosis of AAA. CT scans are limited in that they can only be performed in stable patients, but there are advantages to this modality. CT scans can characterize and confirm the extent of the lesion, which is helpful for operative planning. In addition, unlike ultrasound, CT scans are less operator dependent. MRI is not commonly used in the initial work-up and diagnosis of AAA, but an AAA may be seen on an abdominal MRI obtained for other reasons.

Treatment

Treatment of an AAA depends on its size as well as whether or not it is ruptured. Unruptured AAAs identified in asymptomatic patients may be monitored for
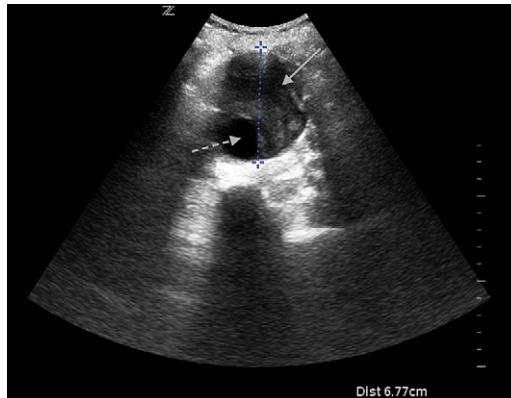
Fig. 5. Transverse view of the abdominal aorta obtained with bedside ultrasound. Antero-posterior measurement of the aorta shows a 6.77-cm aneurysm. Both the intramural thrombus (*solid arrow*) and the aorta lumen (*dashed arrow*) are depicted.

progression. Symptomatic and ruptured aneurysms require surgical intervention. Once an asymptomatic aneurysm is greater than 5 cm, operative repair is generally recommended.[35] Based on the law of Laplace (tension is proportional to pressure and radius), the rate of expansion increases as the lumen size increases. Beyond 5 cm, the risk of rupture generally exceeds that of operative risk.

Treatment of patients with unruptured AAAs smaller than 5 cm centers around risk factor modification and monitoring for expansion of the aneurysm. Smoking cessation should be encouraged. Initiation of β-blocker therapy is also recommended in patients with AAAs. Although studies are limited, β-blockers have been shown to reduce the rate of aneurysm expansion, and the American College of Cardiology and American Heart Association recommend initiation of β-blockers in patients with AAAs.[36]

The initial treatment of a patient with a ruptured AAA is focused on hemodynamic stabilization. Multiple large-bore intravenous lines should be placed, and infusion of crystalloid should begin immediately in hypotensive patients. Uncrossmatched packed red blood cells can be initiated and then switched to crossmatched packed red blood cells when available. Platelets and fresh frozen plasma will likely be needed, given the large-volume transfusions that occur with these patients. It is important to know the capabilities of the blood bank in the practice setting. If there are limited blood products available, rapid patient transfer becomes more critical.

There is no consensus on specific vital sign goals of resuscitation in hypotensive patients with ruptured AAAs. Instead, the focus is to resuscitate to vital organ function. Follow mental status and urine output to assess brain and kidney function. Serial electrocardiograms assessing for evidence of cardiac ischemia may be useful. Over-resuscitation may lead to possible clot disruption. There is also a concern for dilution of clotting factors if resuscitation is limited to crystalloid and packed red blood cells.

When the diagnosis of a ruptured AAA is made, a vascular surgeon should be consulted immediately. Depending on operative capabilities of the treating institution, patients with ruptured AAAs may require emergent transfer to tertiary care centers if they can be stabilized for transport. There are 2 different options for the repair of a ruptured AAA: open versus endovascular repair. Multiple studies have compared the mortalities of the 2 options. The endovascular approach has been shown to have a lower short-term mortality, but this advantage is lost over time because of the incidence of graft failure.[37–40]

THORACIC AORTIC ANEURYSM
Causes and Risk Factors

Thoracic aortic aneurysms (TAAs) are less common than AAAs. They are described based on their location within the thoracic aorta: the aortic root, the ascending thoracic aorta, the aortic arch, or the descending thoracic aorta.[41] Aneurysms may also extend into the abdominal aorta and are then called thoracoabdominal aneurysms. Risk factors for TAAs are similar to those for AAAs.

Clinical Presentation

As with AAAs, TAAs are often asymptomatic until they rupture. The most common presenting symptom of rupture is pain in the chest or back. Additional symptoms are determined by the location and size of the aneurysm. Aortic root and ascending TAAs can cause aortic insufficiency, resulting in heart failure. They can also obstruct the superior vena cava if they are large, resulting in distended neck veins on examination. TAAs can cause hoarseness from stretching of the recurrent laryngeal nerves, and descending TAAs can compress the trachea, resulting in respiratory symptoms such as wheezing, cough, or dyspnea. Compression of the esophagus causes dysphagia.[41]

Diagnostic Modalities

TAAs are often first suspected after chest radiography is obtained for another indication. Findings include a widened mediastinum, tracheal deviation, and enlargement of the aortic knob. However, normal chest radiography does not rule out a TAA. Similarly to the work-up for aortic dissections, CT scan imaging is the most common modality used. Echocardiography is a good alternative if a CT scan cannot be obtained, and has the functional advantage of being able to assess the aortic valve function. Although MRI provides detailed characterization of TAAs, it is less commonly used in the work-up of a TAA.

Treatment

Patients with ruptured TAAs require resuscitation in the same manner as those with ruptured AAAs, as well as emergent cardiothoracic surgical consultation. Asymptomatic patients with TAAs may be monitored with serial imaging for aneurysm growth. β-Blockers are recommended in an attempt to slow aneurysm growth. The decision for operative intervention in asymptomatic patients with TAA is determined by the aneurysm size and rate of expansion, similarly to the approach of AAAs. The annual risk of TAA rupture has been shown to increase from 2% for aneurysms less than 5 cm, to 3% for those 5 cm to 5.9 cm, to 7% for those greater than 6 cm.[42] Studies evaluating the risk of rupture in untreated TAAs have led to a recommendation that ascending TAAs greater than 5.5 cm and descending TAAs greater than 6.5 cm should receive intervention.[43,44] Patients with Marfan syndrome are considered for earlier intervention because of the higher risk of rupture or dissection.

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