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An Evidence-Based Approach To Acute Aortic Syndromes

Abstract

Aortic dissection, intramural hematoma, and penetrating atherosclerotic ulcer are parts of a spectrum of acute aortic syndromes that represent uncommon - but potentially deadly - diagnoses seen in the emergency department. The differential for acute aortic syndromes is large, as many conditions (including the much more common conditions of acute coronary syndromes and pulmonary embolism) present with many of the same chief complaints. This review looks at the features and classifications of acute aortic syndromes and presents evidence regarding the risk factors and chief complaints that can assist emergency clinicians in identifying the patients who require further investigation. Although no set of clinical factors has been shown to rule out aortic dissection, elements of a complete history and physical examination are critical in identifying patients who may be at risk for these diseases. In addition, the advantages and disadvantages of the various available advanced imaging strategies, the evidence regarding efficacy of laboratory testing (including D-dimer), as well as surgical and nonsurgical treatment options are reviewed.

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CME Objectives

After reading this article, participants should be able to:

1. Review the pathophysiology of aortic dissection, intramural hematoma, and penetrating atherosclerotic ulcer.
2. Identify patients who are high risk for an aortic dissection.
3. Choose appropriate diagnostic testing for patients suspected of an aortic dissection.
4. Manage patients with a diagnosis of an aortic dissection.

Prior to beginning this activity, see "Physician CME Information" on the back page.

Case Presentations

You are working an overnight shift when EMS arrives with a 55-year-old male with severe substernal chest pain and shortness of breath for the past 2 hours. The patient has a history of hypertension and type II diabetes. His blood pressure is 200/110 mm Hg, and his heart rate is 110 beats/min. A prehospital ECG was performed, which shows ST elevation in leads II and III as well as a VF consistent with an acute myocardial infarction, and you call a STEMI alert. The cardiologist calls back from your STEMI alert and states that she is coming in from home to see the patient. To save time, she wants to meet the patient directly in the heart catheterization lab on the second floor. After you get off the phone, you quickly look at the chest radiograph and notice that the mediastinum appears widened. You wonder if it's a good idea to start anticoagulation and send the patient to the catheterization lab right away…

The next week, you are working at a free-standing ED where the patients are checking in at record volume. You are getting pressure to see and discharge patients as fast as possible when you see a 21-year-old male presenting with chest pain radiating to his back, along with some shortness of breath. The patient reports no improvement in symptoms with over-the-counter analgesics. The patient plays on the local varsity basketball team. He has no known medical history, and his social history is negative for tobacco, alcohol, or illicit drugs. He appears slightly anxious and has a blood pressure of 155/90 mm Hg and a heart rate of 95 beats/min. He is tall and thin and has reproducible chest tenderness. Your CT scanner has unexpectedly gone down and is unavailable for the rest of the night. ECG shows a normal sinus rhythm without evidence of ischemia and a plain chest radiograph appears normal. As you start to watch your department getting backed up, the nurse states that he is concerned about this patient. You assess the patient as low risk for pulmonary embolism, so you decide to get a D-dimer, which comes back negative. You wonder if this patient has something more significant and what your diagnostic options are…

Introduction

Over 250 years ago, the first description of aortic dissection was made during an autopsy of King George II after he suddenly died "while straining on the toilet." Over 60 years later, the term "dissecting aneurysm" was coined by René Laennec, which brought both recognition and confusion to this disease, confusion that persists to the present day.[1] In 1955, Dr. Michael DeBakey and his team were the first to successfully repair a dissecting aortic aneurysm; ironically, many years later, Dr. DeBakey himself suffered an acute aortic dissection.[1]

While the term "dissecting aneurysm" is still used, aortic dissection and aortic aneurysm are 2 distinct disease processes. Acute aortic dissection is part of a spectrum often referred to as *acute aortic syndrome.* This encompasses not only aortic dissection but also its variants, including aortic intramural hematoma and penetrating atherosclerotic ulcer.[1] While aortic dissection usually presents with severe pain, its presentation can be more subtle and should be considered in anyone with chest pain and pain with either syncope or focal neurological deficits.

Aortic dissection is a potentially life-threatening diagnosis, and it requires a heightened suspicion. Unfortunately, the diagnosis of aortic dissection in the emergency department (ED) is missed 16% to 38% of the time.[2,3] Litigation surrounding missed aortic dissection has also become more common.[4] Morbidity and mortality of aortic dissection is high, and it requires aggressive management to prevent poor outcomes. Mortality for an untreated type A dissection is thought to be approximately 1% to 2% per hour in the first 48 hours, 50% by day 3, and 80% by 2 weeks.[1,5] Type B dissection has a mortality of approximately 10% at 30 days for lower-risk patients and up to 70% in high-risk groups.[1,6] However, recent advances in surgical repair have greatly improved outcomes in these patients. This issue of *Emergency Medicine Practice* will discuss nontraumatic aortic dissection and its anatomic variants, intramural hematoma and penetrating atherosclerotic ulcer. A review of the pathophysiology, risk factors, and appropriate imaging for aortic dissection will be discussed. Treatment options, both surgical and nonsurgical, will also be reviewed, including new noninvasive management techniques.

Critical Appraisal Of The Literature

A literature search of Ovid MEDLINE®, PubMed, and the Cochrane Database of Systemic Reviews was performed using the search term *aortic dissection*. The search was limited to the English language, humans, and adults, from 1993 through July 2013. Abstracts and articles were reviewed for applicability related to the acute management and diagnosis of aortic dissection. Policy statements from the American College of Cardiology Foundation/American Heart Association (ACCF/AHA) (including 8 other societies), the European Society of Cardiology, the Japanese Circulation Society, and the American College of Radiology (ACR) were also reviewed.[7-10] Currently, the American College of Emergency Physicians (ACEP) is developing a clinical policy for aortic dissection, but it was not available at the time of writing of this article.

Because of the low incidence of aortic dissection, the majority of literature on this topic comes from registry data such as the International Registry of Acute Aortic Dissection (IRAD). IRAD was established in 1996 and now consists of 30 large referral centers in 11 different countries, and it assesses clinical features, treatment, and outcomes for pa-

tients with an acute aortic dissection from this large retrospective database.[11] While the IRAD database is one of the most important sources of data on aortic dissection, it is important to understand the limitations of registry data, which include the lack of controls as well as the heterogeneity in data from protocol revisions over time. Other registry data also exist, such as the German Registry for Acute Aortic Dissection Type A (GERAADA), which consists of 50 cardiac centers and includes data for new treatment strategies.[12] Very few prospective studies exist in regard to acute aortic dissection, and these studies are generally observational in design. The remaining literature consists of case reports and series.

Epidemiology

The incidence of aortic dissection is 6000 to 10,000 cases per year in the United States, or approximately 4 to 30 cases per 1,000,000 person-years. This incidence can be compared to the much more common condition of acute myocardial infarction, which occurs at approximately 4400 cases per 1,000,000 person-years.[6,13,14] However, this incidence for aortic dissection comes from retrospective data and autopsy studies and likely underestimates its true incidence. Approximately 75% of aortic dissections occur in patients who are aged 40 to 70 years, with the majority occurring between the ages of 50 and 65 years.[14] Aortic dissection is reported to affect men 3 times more often than women, although women are more likely to present later and have worse outcomes.[15] Data from the IRAD database suggest that acute aortic dissections are more likely to occur in the morning (6:00 AM to noon) and in the wintertime, similar to other cardiovascular conditions. While some theories suggest that rhythmic variation in the sympathovagal balance and in the hemorheologic properties of circulating blood play a role, no definitive evidence exists.[16]

Pathophysiology And Etiology

Acute aortic syndrome is defined as 3 related conditions: (1) aortic dissection, (2) intramural hematoma, and (3) penetrating atherosclerotic ulcer. Their pathology, diagnosis, and treatment are similar.[7] Aortic dissection is defined as *acute* if it occurs within 2 weeks of the onset of symptoms, *subacute* if it occurs between 2 and 6 weeks, and *chronic* if it occurs more than 6 weeks from the onset of pain. (Some authors describe aortic dissections > 2 weeks as chronic).[7,14]

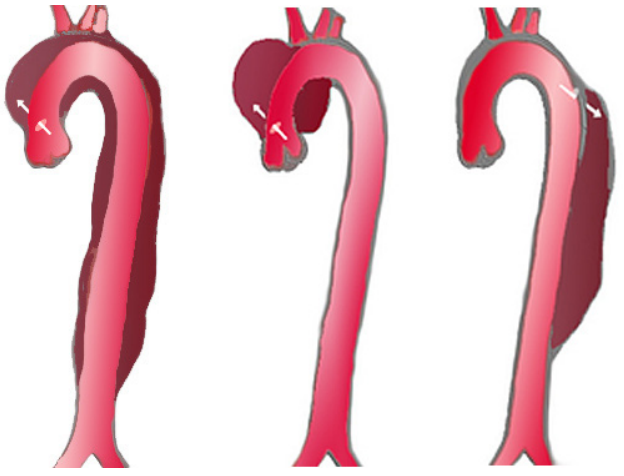
The classification of aortic dissection is based on pathological and anatomical features. In the pathologically based classification, there are 5 classes: [7,8]

- Class 1 – classic aortic dissection: separation of intima from media and/or adventitia with intimal flap
- Class 2 – intramural hematoma: hemorrhage within aortic wall without obvious intimal flap
- Class 3 – subtle-discrete dissection: localized intimal tear with no dissection flap or medial hematoma
- Class 4 – penetrating atherosclerotic ulcer: usually localized to descending aorta with significant atheroma; found usually in the adventitia with localized hematoma or saccular aneurysm. May convert to classic aortic dissection
- Class 5 – iatrogenic or traumatic dissection: following cardiac catheterization or cardiac surgery or decelerating chest trauma

There are 2 main anatomic classification systems for aortic dissections that are defined based on the involvement of the proximal aorta: (1) the DeBakey classification, and (2) the Stanford classification.[7,8] **(See Figure 1.)** In the DeBakey classification, there are 3 types. Type I originates in the ascending aorta and extends into the aortic arch and descending aorta. Type II is confined only to the ascending aorta. Type III originates in the descending thoracic aorta and is further subdivided into type IIIa, which is limited to the descending thoracic aorta, and type IIIb, which extends below the diaphragm. The proximal aorta is defined as the aorta proximal to the brachiocephalic artery; the descending aorta is defined as the aorta distal to the left subclavian artery.[7]

In the Stanford classification system, aortic dissection is defined according to whether the ascending aorta is involved or not. Stanford type A dissections involve the ascending aorta (similar to

Figure 1. Classification Systems For Aortic Dissections



DeBakey I	Debakey II	DeBakey III
Stanford A (proximal)		Stanford B (distal)

DeBakey type I and II). Stanford type B dissections involve the descending aorta (similar to DeBakey type III).[7] Approximately two-thirds of acute aortic dissections are Stanford type A and one-third are type B. Of the type B aortic dissections, approximately 1% are isolated to the abdomen only.[1,17] Stanford type B dissections are further classified as *complicated* or *uncomplicated*. Approximately 30% will be classified as complicated dissections, which refers to either radiographic evidence of thoracic aortic rupture (eg, blood outside the aortic wall); ischemia involving the viscera, kidneys, spinal cord, or lower extremities; persistent pain; or rapid expansion in the distal arch or proximal descending aorta to a total aortic diameter of > 4.5 cm.[1,18,19,20] These findings require immediate intervention due to the threat to life and limb.

Pathophysiology

The aortic wall consists of 3 layers: the intima, media, and adventitia. The intima is the innermost layer and contains fairly loose connective tissue, allowing for motion relative to the cardiac cycle. The media is the middle layer and contains multiple layers of elastin, collagen, and smooth muscle that give the aorta its properties for both distensibility and integrity. Lastly, the adventitia is the outer layer that consists primarily of collagen and connective tissue that also contribute to the integrity of the aorta. The vasa vasorum lies within the adventitia and provides nutritional support for the aorta.[1,14]

Constant exposure to high pulsatile pressure and shear stress can lead to weakening of the aortic wall and eventual injury to the aorta, which can lead to the disruption of the aortic media. When an intimal flap or tear is created, the aortic media is connected to the aortic lumen, causing an aortic dissection. A distal tear may occur back through the intima, but it can also be through the adventitia.[7] Distal tears are usually in the distal thoracic aorta, but they can be in the abdominal aorta or iliac arteries. The dissection creates a new channel called the *false lumen*, which separates the media from the adventitia and can extend either anterograde or retrograde involving the aortic root, arch, or any of the main aortic branches, giving the appearance of a "double-barreled" aorta. Due to increased pressures within the false lumen, it can expand often 50% or more of the aortic circumference and compress the true lumen, thus compromising blood flow to distal arteries and causing ischemic complications such as renal failure, stroke, spinal infarction, limb ischemia, and myocardial events.[1,14,21] The dissection flap can also prolapse across a vessel, obstructing blood flow (dynamic obstruction), or it can extend directly into the vessel (static obstruction).[7]

Etiology

Risk factors for nontraumatic aortic dissection include poorly controlled hypertension, connective tissue disorders (eg, Marfan syndrome, Ehlers-Danlos syndrome), congenital valvular disorders (eg, bicuspid aortic valve), aortic coarctation, metabolic disorders (eg, homocystinuria), previous cardiac surgery, familial aortic dissections, aortic aneurysms, pheochromocytoma, inflammatory vasculitis, weight lifting, and sympathomimetics (eg, cocaine).[6,7,14] Fewer than 10% of patients with aortic dissection are aged < 40 years, and they are more likely to have connective tissue disorder (eg, Marfan syndrome), bicuspid aortic valve, or prior aortic surgery.[1,22] Degenerative changes such as medial degeneration (formerly known as cystic medial necrosis) can occur with aging, leading to the breakdown of collagen, elastin, and smooth muscle, with an increase in basophilic ground substance in the aorta. This causes stiffness and increases the susceptibility of the aorta to shear forces.[7,14] Conflicting evidence exists as to whether pregnancy is a risk factor. Pregnant patients, especially with connective tissue disorders such as Marfan syndrome, are thought to be at an increased risk,[5,14,23] though at least 1 study disputes this.[13] Iatrogenic causes of aortic dissection can occur and are usually associated with cardiac surgery (eg, aortic valve replacement, coronary artery bypass grafting) or percutaneous catheter placement (eg, cardiac catheterization), and they account for approximately 4% of all aortic dissections.[6,14]

Connective Tissue Disorders

Marfan syndrome occurs in approximately 1 in 3000 to 5000 patients and results from the mutation of the fibrillin-1 (FBN1) gene. Fibrillin is the major component of fibrils, which serve as the primary substrate for elastin, and abnormality in this leads to weakening of the aorta.[24] Marfan syndrome is inherited through an autosomal dominant pattern, but 25% of cases occur without a family history of Marfan syndrome. The most common aortic pathology in patients with Marfan syndrome is dilatation of the proximal aorta, which increases the risk of a type A dissection.[23] Patients should be suspected of having Marfan syndrome if they have certain physical characteristics such as being tall and thin with unusually long arms, legs, or fingers or if they have pectus excavatum.[24] Mortality has been high in patients with Marfan syndrome related to aortic rupture, but recent advances in screening and surgical management for aortic disease has improved life expectancy from 45 years to about 70 years.[23]

Ehlers-Danlos syndrome is another connective tissue disorder that has a heterogeneous presentation. Ehlers-Danlos syndrome type IV is a rare autosomal dominant disorder resulting from mutations in the gene COL3A1, which is responsible

for encoding type III procollagen synthesis. The mutation causes vascular tissue fragility and has been associated with aortic dissections and dissecting aneurysms.[25] Patients may present with joint hypermobility, increased skin elasticity, and tissue fragility. While 80% of patients with Ehlers-Danlos syndrome type IV will have experienced a complication by the age of 40, most will be unaware of their condition until they have a vascular complication.[25]

Intramural Hematoma

Intramural hematoma is thought to be a variant of an aortic dissection and occurs in approximately 10% to 20% of acute aortic syndromes.[7,26] Patients tend to be older and are more frequently from Asian countries, compared to Western countries.[27] Intramural hematoma is distinct from an aortic dissection because of its lack of blood flow in the false lumen or its lack of an intimal flap or entry point.[7,26] While the pathophysiology for intramural hematomas is still unclear, some believe that it originates from a rupture of the vasa vasorum within the medial layer; others suggest that it results from microscopic tears in the intima.[7] Regardless of the origin, the approach to an intramural hematoma is similar to a classic aortic dissection.

Intramural hematoma has a similar clinical presentation compared to classic aortic dissection, and it is classified using the Stanford system. Type A intramural hematomas (compared to classic aortic dissection) are less likely to have a pulse deficit (15% vs 31%, $P = .012$) or aortic regurgitation (35% vs 56%, $P = .003$), but they are more likely to have pericardial effusion (67% vs 43%, $P < .001$) and periaortic hematomas (46% vs 21%, $P < .001$). However, in type B intramural hematomas, only a pulse deficit was less likely, compared with classic aortic dissection (7.6% vs 19%, $P = .013$).[26] The natural history of intramural hematomas is variable, with approximately 10% reabsorbing without intervention. Others will expand until rupture. Approximately 3% to 14% of type B intramural hematomas and 11% to 88% of type A intramural hematomas will convert to a classic aortic dissection.[7]

Penetrating Atherosclerotic Ulcer

Penetrating atherosclerotic ulcer is defined as ulceration from a focal atherosclerotic lesion. While the exact pathophysiology is unknown, penetrating atherosclerotic ulcer may progress to intramural hematoma via erosion of the vasa vasorum creating a hematoma within the medial wall of the aorta.[7,28] Intramural hematoma has also been seen to form a penetrating atherosclerotic ulcer with either pseudoaneurysm formation or conversion into a classic aortic dissection.[28] In 90% of cases, penetrating atherosclerotic ulcer is found in the descending aorta (where atherosclerotic lesions of the aorta are found), and it is usually diagnosed by computed tomography (CT).[7] The incidence is thought to be around 2% to 11% of all acute aortic syndromes, and up to 60% will have concurrent abdominal aortic aneurysms.[28,29] Patients with penetrating atherosclerotic ulcer are older (typically aged > 70 years), present with chest or back pain, and have less aortic regurgitation and malperfusion compared to classic aortic dissection. They are often found incidentally in patients without symptoms.[7,28] Older age and urgent presentation are associated with worse outcomes.[30] Because of the potentially devastating progression of penetrating atherosclerotic ulcer, aggressive management is usually warranted in these patients.

Differential Diagnosis

Aortic dissection has many presentations, depending on which organ system is involved; thus, it is a diagnostic consideration in a large number of patients presenting to the ED with a variety of chief complaints. **(See Table 1.)** Classically, aortic dissection will present with abrupt chest or back pain, but approximately 5% of patients with an acute aortic dissection will have no pain.[6] A 2011 retrospective study showed that the diagnosis of aortic dissection was more likely to be missed if the patient entered the ED as a walk-in than by emergency medical services (EMS), likely due to the patient presenting with milder symptoms.[2] Although aortic dissection is rare, it should always be considered in the differential diagnosis of patients with suspected acute coronary syndromes or pulmonary embolism. Atypical presentations can also occur. Syncope has been reported in up to 17% of patients, and acute neurological deficits or coma occur in 30% of patients presenting with an acute type A dissection.[31] Aortic dissection should be considered when other diagnoses such as pericarditis, cholecystitis, gastritis, syncope, or cerebrovascular accident are contemplated.

Prehospital Care

Limited data exist on prehospital cardiac arrests from aortic dissections. In a retrospective study of 1990 patients who were diagnosed with cardiac arrest in the ED over a 11.5-year period, 46 patients were ultimately diagnosed with an aortic dissection.[32] Of these patients, 26 of 46 (57%) had suffered

Table 1. Chief Complaints That Include Aortic Dissection In The Differential Diagnosis

- Chest pain
- Back pain
- Abdominal pain
- Syncope
- Acute neurological deficit (especially with chest or back pain)

cardiac arrest in the prehospital setting. Historical clues (such as previous aortic pathology) may alert the prehospital provider to a possible acute aortic dissection. However, since aortic dissection cannot be diagnosed by history and physical examination alone, the focus should be on stabilizing patients and rapidly transporting them to the appropriate facility. While there are no studies evaluating diversion of patients to a facility with cardiovascular surgery capability, if there is a high clinical suspicion (eg, history of aortic dissection presenting with similar symptoms), it would not be unreasonable to discuss direct transfer from the field to a facility with expertise in treating aortic dissections with local medical control physicians.

All patients with a suspected or confirmed diagnosis of aortic dissection should be transported via advanced life support. Intravenous access should be obtained and the patient should be placed on a cardiac monitor. Hypoxia should be treated with supplemental oxygen, and intravenous fluids should be given if the patient is hypotensive, with close monitoring of vital signs. Standing protocols should be implemented based on the patient's presenting complaint. Prehospital providers should be in contact with local medical control physicians for additional orders.

Emergency Department Evaluation

History

The presentation of an acute aortic dissection is widely variable. Key historical features should be obtained, including time of onset of symptoms, location of pain (especially in the chest, back, or abdomen), character of pain (eg, ripping or tearing), radiation of pain, alleviating or aggravating factors, and other associated symptoms (eg, neurological, vascular, or cardiopulmonary). Obtaining a past medical history for aortic dissection risk factors is essential, including a history of long-standing hypertension, previous cardiac surgery (especially aortic valve replacement), previous aortic pathology, known connective tissue disorders, and vasculitis. **(See Table 2.)** Medications, especially anticoagulants, are important to inquire about. Providers should be especially be aware of the patient's history with novel anticoagulants such as apixaban (Eliquis®), rivaroxaban (Xarelto®), and dabigatran (Pradaxa®). Information about allergies to intravenous iodinated contrast will be important in order to help determine the appropriate imaging modalities. Other information, such as family history (especially connective tissue disorders and aortic pathologies) and social history (especially cocaine and amphetamine use) should also be obtained. Unfortunately, no set of clinical factors has been shown to rule out an aortic dissection.[33]

Data from the IRAD database have shown that 85% of patients with type A dissections will have sudden onset of pain, with 79% describing it as being in their chest.[6] Pain is described as "severe" or "worst ever" in 90% of patients. Pain may radiate to the extremities, the back, or the abdomen. If the aortic dissection extends into the spinal or the carotid arteries, the patient may present with focal neurological findings. Type B dissections can have similar presentations to type A dissections, with 84% of patients with type B dissections also having sudden onset of pain and 63% describing it as being in their chest. However, type B dissections are more likely to produce back and abdominal pain compared to type A dissections.[6] Isolated abdominal aortic dissections are more likely to produce abdominal pain, limb ischemia, or hypotension compared to classic type B dissections, which involve both thoracic and abdominal aorta.[17]

Historical predictors have been shown to affect the probability of an aortic dissection. In a 2002 review of 21 studies, the absence of sudden pain decreased the likelihood of an aortic dissection (likelihood ratio [LR], 0.3; 95% confidence interval [CI], 0.2-0.5). "Tearing" or "ripping" pain and pain that migrates may be useful signs, but more studies are needed to assess the significance.[33]

Physical Examination

Patients may present as hypertensive (49%), normotensive (35%), hypotensive (8%), or in shock (8%).[6] The presence of hypotension or shock is an ominous finding.[14,34] Blood pressure differential (pseudohypotension) can occur when a dissection extends into a branch of the aorta occluding the subclavian artery. A difference of 20 mm Hg between arms is considered positive and can be suggestive of an aortic dissection; however, 20% of the population will have a blood pressure differential without an aortic dissection.[14] Pulse deficit has also been described in aortic dissec-

Table 2. High-Risk Features For Aortic Dissection

Conditions
• Known or suspected connective tissue disorder (eg, Marfan syndrome)
• Family history of aortic pathology
• Known aortic pathology
• Previous cardiac surgery or recent catheterization
• Aortic valve pathology (eg, bicuspid)
• Cocaine or amphetamine use
• Vasculitis
Pain and Examination Features
• Sudden onset/severe pain **AND**
◦ Ripping/tearing pain
◦ Pulse deficit
◦ Blood pressure differential
◦ New aortic insufficiency
◦ Hypotension/shock

tion and should be looked for in the carotid, brachial, or femoral pulse. A pulse deficit is defined as having weak or no pulse on the affected side.[35] However, data from the IRAD database of 2538 patients found a pulse deficit or blood pressure differential in only 20% of patients.[36] Absence of a pulse deficit cannot be relied upon to rule out an aortic dissection; however, the presence of a pulse deficit does increase the likelihood of an aortic dissection (LR, 5.7; 95% CI, 1.4-23).[33] Moreover, when a pulse deficit is present in the setting of an acute type A dissection, patients have a 2 to 3 times greater risk of death.[33,37,38] The presence of shock/hypotension was also associated with a 3 to 7 times increased risk of death.[31,37,38]

The pulmonary examination focuses on evaluating for retractions, tachypnea, rales, and rhonchi. These findings may suggest acute congestive heart failure due to acute aortic insufficiency, acute myocardial infarction, or pericardial tamponade complicating the aortic dissection. Focal lung findings may suggest a large pleural effusion from an aortic rupture.[7]

The cardiac examination includes examining for new murmurs, distant heart sounds, jugular venous distension, and tachycardia. An aortic dissection may extend retrograde and involve the aortic valve or coronary arteries. A diastolic murmur can be heard in about 31% of patients with an aortic dissection and may represent severe aortic regurgitation;[6] however, the presence of a diastolic murmur is neither sensitive nor specific for aortic dissection.[33] The presence of jugular venous distension, distant heart sounds, and tachycardia may suggest pericardial tamponade, which is associated with worse outcomes.[37,39]

Neurological findings can be associated with aortic dissections secondary to the extension of the dissection into the aortic branches. These branches can be occluded by either expansion of the false lumen occluding the true lumen or by emboli from an expanding thrombus. Deficits may occur from insults to the brain or spinal cord. Coma can also occur from shock or hypotension. Neurological symptoms can be found in 17% to 40% of aortic dissection cases. Of these, cerebrovascular accidents account for 5% to 14% of cases.[6,40,41] In a retrospective study of 102 patients with aortic dissection, 94% of the patients without neurological symptoms experienced pain. Of the 30 patients who did report neurological symptoms, only 66% reported chest pain.[40] The IRAD database also found altered mental status to be present with aortic dissections, with coma being present in approximately 3% of patients with aortic dissection.[41] Altered mental status was also more common in the presence of cardiac tamponade in type A dissection patients (31.2% with cardiac tamponade vs 10.6% without).[39]

Mesenteric ischemia is the most common gastrointestinal complication from an abdominal aortic dissection, resulting from the dissection involving the mesenteric arteries. Although it is classically described as pain out of proportion to the physical examination, it can be nonspecific. Biomarkers for mesenteric ischemia (such as serum lactate) may not elevate until late in the presentation.[7] Gastrointestinal bleeding is a rare complication of aortic dissection and may represent mesenteric infarction, aortoesophageal fistula, or rupture into the small bowel.[7]

Syncope occurs in approximately 9% to 13% of patients with an acute aortic dissection.[6,7] Many etiologies exist as a cause of syncope, including cardiac tamponade, severe aortic regurgitation, decreased cerebral blood flow, vasovagal response secondary to pain, and aortic rupture.[7] Patients with aortic dissection presenting with syncope are more likely to die (34% vs 24%) and to have associated pericardial tamponade, stroke, decreased consciousness, and spinal cord ischemia.[7,39]

Risk Stratification

Several studies have attempted to risk stratify patients with potential aortic dissection. A 2000 study by von Kodolitsch et al prospectively evaluated 26 potential predictors in 256 patients presenting to the ED with a clinical suspicion of aortic dissection. They found 3 independent clinical predictors: (1) immediate onset of aortic pain (ie, chest or back pain) or pain described as tearing or ripping; (2) mediastinal widening or aortic widening on chest radiograph; and (3) pulse differential or blood pressure differential. Absence of all 3 variables conferred a probability of 7% for aortic dissection, while the presence of all 3 variables had a probability of 100% for aortic dissection.[42] However, the application of this study is limited by its small enrollment of 256 patients over an 8-year period, with a 49% positive rate for an aortic dissection. This study population with a very high rate of aortic dissection will likely not be applicable to other EDs.

The 2010 ACCF/AHA thoracic aortic disease guidelines created an aortic dissection detection score for risk stratification.[7] This score, which ranges from 0 to 3, assigns 1 point for each of 3 categories: (1) high-risk conditions (Marfan syndrome, family history of aortic disease, known aortic valve disease, recent aortic manipulation, and known thoracic aortic aneurysm); (2) high-risk pain features (chest, back, or abdominal pain described as abrupt, severe, tearing, or ripping); and (3) high-risk examination features (pulse deficit; blood pressure differential; focal neurological deficit; or new murmur associated with aortic regurgitation, hypotension, or shock state). This risk assessment score for aortic dissection was evaluated utilizing the IRAD database of 2538 patients. A score of 0 was considered low-risk and was found in 4.3% of patients with aortic dissection. If a chest radiograph was performed that did not show evidence

of widened mediastinum, then the probability went down to 3%.[36] While these tools seem promising, they have not been externally validated. Use of the aortic dissection detection score may potentially lead to overtesting, as many patients may fall into the intermediate-risk or high-risk categories.

Diagnostic Studies

The diagnosis of aortic dissection is primarily made by advanced imaging such as ultrasound, CT, or magnetic resonance imaging (MRI). However, other testing may be beneficial in the evaluation of patients with potential aortic dissection. Most recently, various biomarkers have been evaluated for use in evaluating aortic dissection.

Laboratory Testing

Laboratory testing should be focused on the differential from the history and physical examination. If suspicion of aortic dissection is high, testing should include a basic metabolic panel, complete blood count, and coagulation studies. Blood type and screen may be helpful in preparation for blood products, including reversal of anticoagulation.[7-9] Additional laboratory testing, including cardiac enzymes, liver function tests, pancreatic enzymes, and urinalysis, may be appropriate. Point-of-care (POC) testing may be helpful, including the evaluation of renal function, as some institutions have a cutoff for creatinine clearance for the use of intravenous contrast. Renal failure has been shown to be a marker for increased mortality in patients with aortic dissectons.[31,37,43]

Biomarkers

Various biomarkers have been tested in the evaluation of suspected aortic dissection, including D-dimer, highly sensitive C-reactive protein (CRP), and calponin.

Currently, D-dimer is the most studied biomarker in the evaluation of aortic dissection. D-dimer is a breakdown product of cross-linked fibrin by the endogenous fibrinolytic system. It is a test that has been validated in the evaluation of pulmonary embolism and is readily available in most EDs.[44] Multiple small retrospective and prospective observational studies have looked at the utility of D-dimer in aortic dissection, finding a sensitivity ranging from 91% to 100%.[45-54] Elevations of > 5000 ng/mL were more likely to distinguish aortic dissection and pulmonary embolism from acute myocardial infarction.[55] However, in a prospective study of 80 patients, 18% of patients with an aortic dissection had a D-dimer < 400 ng/mL, giving it a sensitivity of 82%.[56] It is important to note that many of these studies used varying cutoffs for a "negative" D-dimer, ranging from 400 ng/mL to 900 ng/mL. A recent meta-analysis evaluating 7 studies showed a sensitivity of 97% and a negative predictive value of 96% using a cutoff of < 500 ng/mL.[57] Patients with intramural hematoma have been shown to have lower D-dimer levels than patients with aortic dissections.[58] D-dimer may be "negative" in patients with an intramural hematoma, short dissections, or a thrombosed false lumen without ulcer-like projections.[52,58,59] While more and more evidence is accumulating in support of the use of D-dimer as a screening tool for aortic dissection similar to pulmonary embolism, exercise caution in using this method until larger prospective studies become available to validate its use.[60] Currently, the 2010 ACCF/AHA guidelines do not recommend using D-dimer for the evaluation of possible aortic dissection.[7]

Fibrin and fibrinogen degradation products (FDP) have also been studied to aid in the diagnosis of aortic dissection. Similar to D-dimer, FDP is expected to rise in the setting of an aortic dissection. Two studies have shown promise in the use of this biomarker, but more studies are needed.[54,61]

CRP is a marker of inflammation, and it has been studied in the evaluation of aortic dissection. Evidence suggests its value is in predicting the need for urgent surgical treatment and worsening outcomes;[50,62] however, it is neither sensitive nor specific in the diagnosis of aortic dissection.[47,53]

Novel biomarkers such as calponin and matrix metalloproteinases (MMPs) have been evaluated in the diagnosis of aortic dissection. In a prospective study of 207 patients, calponin was found to be elevated in aortic dissection, so it may become useful in the future.[63] MMP is another biomarker that has been shown to be elevated in acute aortic dissection.[64,65] However, a recent study showed relatively poor sensitivity for MMP by itself. MMP in combination with D-dimer may be more sensitive and specific than either biomarker alone.[59]

Electrocardiogram

An electrocardiogram (ECG) is usually obtained on patients who complain of chest pain, shortness of breath, syncope, stroke, or altered mental status. While ECG can be a valuable tool in the evaluation of cardiac lesions, it is neither sensitive nor specific for aortic dissection. ECG can show chronic changes such as Q waves and nonspecific ST-T wave changes, and these findings were noted in 49% of all patients in the IRAD database.[6] In a retrospective study of 159 patients, acute ECG changes were found in 50% of type A aortic dissections, with ST elevation in 8%.[66] In this same study, ST depression and T-wave inversions were also noted to be associated with an increased incidence of shock and cardiac tamponade.[66] While ischemic changes on ECG may suggest a cardiac etiology, aortic dissection should also be considered, especially if the patient presents with high-risk features for aortic dissection.

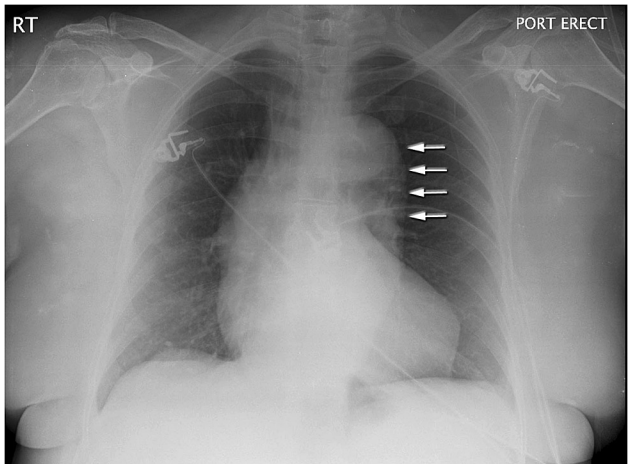
Imaging

The advancement of current noninvasive technology has given the emergency clinician several strategies for diagnosis of aortic dissection, including echocardiography, CT, and MRI. The choice of advanced imaging for the diagnosis of aortic dissection will depend on local expertise and availability; however, all 3 options can be used as a first-line test.[7,10,67]

Chest Radiograph

Chest radiography can suggest an aortic dissection. **(See Figure 2.)** Abnormalities are seen in approximately 88% of patients.[6] **(See Table 3.)** A widened mediastinum is commonly associated with aortic dissection and is seen in approximately 60% of patients.[6] A meta-analysis looking at 1337 radiographs of patients with an aortic dissection reported a sensitivity of 90% for no radiographic findings on chest x-ray. In particular, absence of a widened mediastinum and abnormal aortic contour had a negative LR of 0.3 (95% CI, 0.2-0.4).[33] However, interobserver and intraobserver agreement in evaluating chest x-ray was poor (kappa = 0.25 for interobserver agreement for suspicion for aortic dissection; kappa = 0.23-0.33 for interobserver agreement for widened mediastinum, irregularities of the aortic contour, and pleural effusion).[33] Patients with a missed diagnosis of aortic dissection were more likely to have a normal mediastinum.[2] While the chest x-ray in combination with other signs and symptoms can suggest an aortic dissection, it cannot be relied upon, by itself, to rule out an aortic dissection.[7,8]

Figure 2. Chest Radiograph In An Aortic Dissection



Note the widened mediastinum (arrows).

Table 3. Chest Radiograph Findings In Aortic Dissection

- Widened aortic knob or mediastinum
- Displaced intimal calcification
- Pleural effusion (left > right)
- Left apical pleural cap
- Indistinct or irregular aortic contour
- Tracheal or nasogastric tube displacement

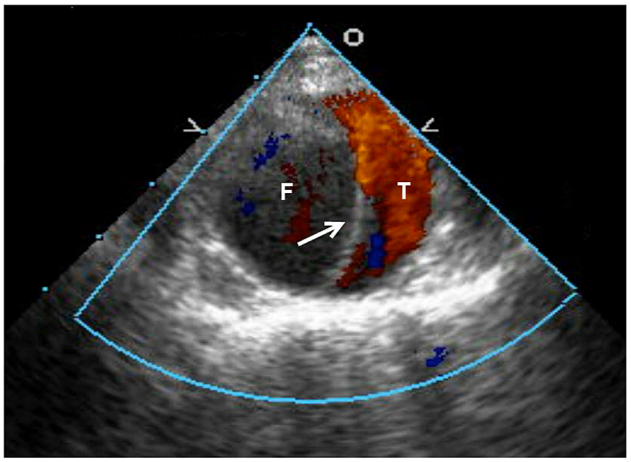
Transthoracic Echocardiogram

Transthoracic echocardiogram (TTE) can be used as a screening tool for the evaluation of possible aortic dissection, especially in the unstable patient. However, 2 retrospective studies and 1 prospective study have reported the sensitivity to be 59% to 88% in the hands of experienced operators.[68-70] Using older ultrasound technology, specificity for type A dissection ranged from 87% to 96%, and specificity for type B dissection ranged from 60% to 83%. Newer technology (such as harmonic imaging and contrast use) has improved the specificity for type A dissection to 97% and type B to 94%.[71] Technical challenges based on a patient's anatomy and difficulty in visualizing the whole thoracic aorta (especially the descending thoracic aorta) limit the usefulness of TTE; however, TTE can also reliably obtain information such as pericardial effusion/tamponade and left ventricular function, which can be helpful in evaluating critical patients.[71] While it may be used as a screening examination, it cannot be relied upon to rule out aortic dissection.

Transesophageal Echocardiogram

Because of its proximity to the aorta, transesophageal echocardiogram (TEE) is a much more sensitive test than TTE. **(See Figure 3.)** Several prospective studies show the sensitivity of TEE to be 86% to 100% and the specificity to be 68% to 100%.[70,72,73] Advantages of using TEE are its accuracy, lack of

Figure 3. Transesophageal Echocardiogram With Doppler Of The Ascending Aorta



Transesophageal echocardiogram showing an aortic dissection. Doppler flow is seen in the true lumen. T: true lumen; F: false lumen; Arrow: aortic dissection flap.

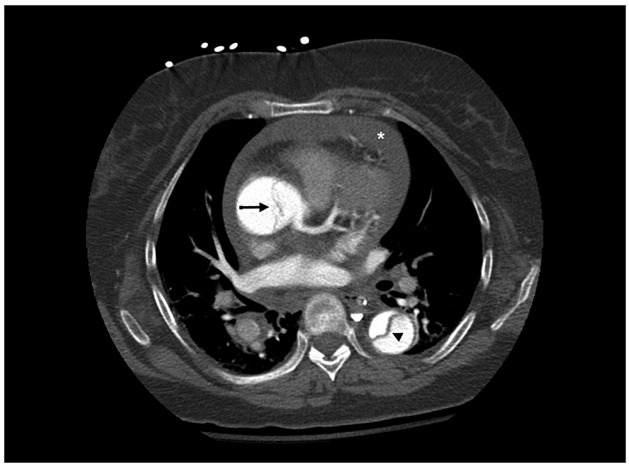
ionizing radiation and contrast exposure, ability to diagnose concurrent aortic valve pathology, evaluation of left ventricular dysfunction, evaluation of pericardial effusion and tamponade, and ability to be used at the bedside (especially in hemodynamically compromised patients). Disadvantages of TEE include the need for sedation or intubation, inadequate views of the aortic arch and its vessels due to interference of the trachea, the risk of esophageal injury (though rare), and operator-dependent quality.[7,71] The lack of availability of TEE at many facilities (including expertise in evaluating acute aortic syndromes, especially after hours) may preclude its use. Because of TEE's limitation in evaluating the descending abdominal aorta, patients with aortic dissections undergoing endovascular repair may require additional advanced imaging.

TEE can also diagnose intramural hematoma and penetrating atherosclerotic ulcer more reliably compared to TTE.[71] TEE may also be used intraoperatively to help with operative management, particularly with aortic valve pathology.[7] When available (especially in hemodynamically compromised patients) TEE may be considered a first-line diagnostic test.[7,10]

Computed Tomography

CT has become the mainstay in diagnosing acute aortic dissection, and it is usually the first-line test in the ED unless there is a contraindication.[10] Multidetector CT allows for rapid anatomical mapping of the entire aorta and branch vessels.[74] **(See Figure 4.)** A meta-analysis evaluating multidetector CT for aortic dissection found the sensitivity to be 100% (95% CI) and the specificity to be 98% (95% CI).[73]

Figure 4. Contrast-Enhanced Computed Tomography With Type A Aortic Dissection

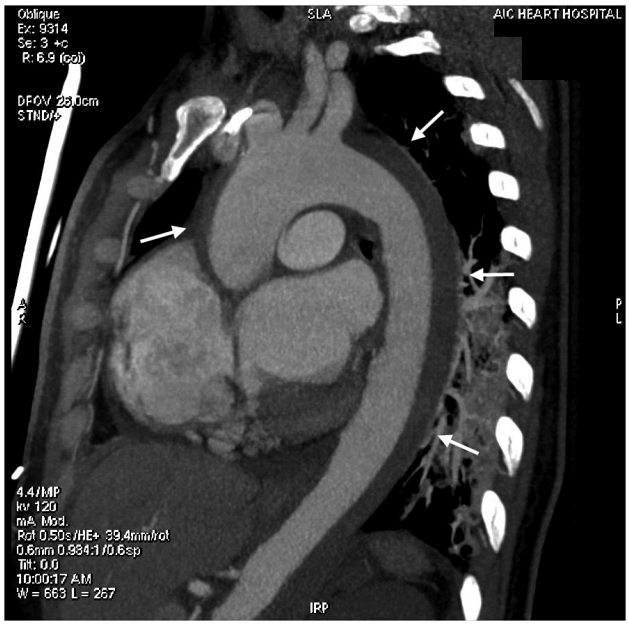


Computed tomography of the chest showing a dissection flap in both the ascending aorta (arrow) and descending thoracic aorta (arrowhead). Note also the pericardial effusion (asterisk) at the top right.

Current multidetector CT protocols utilize both unenhanced CT as well as CT angiography with intravenous contrast. The noncontrast CT helps with the diagnosis of intramural hematoma, evaluation of possible hemorrhage, and identification of calcification within the aorta.[67,74] **(See Figure 5.)** CT angiography requires an intravenous line for contrast that can withstand pressures of up to 5 mL/sec and involves imaging starting from the thoracic inlet down to the femoral arteries with a slice thickness of 0.625 mm.[7,74] Advantages to CT scanning are that it is fast, is readily available in most EDs, can diagnose alternate pathology, is noninvasive, can detect pericardial effusion, and is the least operator-dependent modality compared to MRI and echocardiography.[7,67,73] CT can also render 3-dimensional mapping to help with preplanning for an endovascular procedure. The disadvantages of CT are the ionizing radiation and exposure to intravenous contrast, especially for patients with renal insufficiency.[67,73] For evaluating aortic dissection, the ACR gives CT angiography the highest rating of all advanced imaging techniques and recommends it as a first-line test.[10]

A recent retrospective study of 2868 patients showed that unenhanced CT may have utility in diagnosing aortic dissection, with a sensitivity of 94.4% for type A dissections.[75] Though unenhanced CT may be a reasonable first step in patients who cannot receive intravenous contrast, it should not be

Figure 5. Contrast-Enhanced Computed Tomography With Type A Intramural Hematoma



Computed tomography of the chest showing intramural hematoma from the ascending to the descending thoracic aorta (arrows). Note that no dissection flap is seen along the aorta.

used by itself to rule out aortic dissection.

Newer scanners (≥ 64-slice) can perform ECG-gated CT, which allows for scanning to be synchronized with cardiac contraction. This can minimize motion artifacts (especially in the aortic root) that can cause the appearance of an aortic dissection on CT.[76] However, for routine aortic dissection evaluation, the additional value of ECG-gated CT is unclear. Because of limited availability as well as increased radiation exposure for retrospective ECG-gated CT (but not prospective ECG-gated CT), it should be considered primarily for patients with indeterminate nongated ECG CT or known complex vascular anatomy.[67,74] In patients for whom there is concern for acute coronary syndromes or pulmonary embolism, evaluation by "triple rule-out" can also be performed by ECG-gated CT, but this exposes the patient to higher radiation and contrast volumes and can miss intramural hematoma and abdominal dissections, as most protocols do not perform a noncontrast CT and limit imaging to the thoracic aorta.[4,10] With the advent of dual-source, dual-energy multidetector CT, less intravenous contrast is needed and noncontrast images can be performed virtually, delivering less radiation to the patient.[10]

Magnetic Resonance Imaging

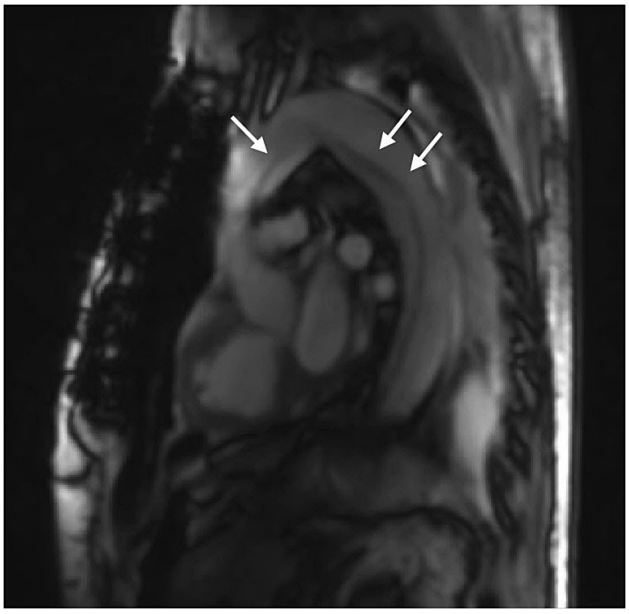
MRI and magnetic resonance angiography (MRA) have a role in certain cases where aortic dissection is suspected. A meta-analysis evaluating 7 studies for MRA showed a high sensitivity (98%; 95% CI, 95%-99%) and specificity (98%; 95% CI, 95%-100%) for aortic dissection, with some studies showing 100% sensitivity and specificity.[73] Earlier studies evaluating the use of MRI/MRA in the acute setting showed feasibility without complications.[70,72] Cine-MRA can also be performed to evaluate aortic valve and left ventricular function. Advantages of MRI include lack of ionizing radiation, lack of iodinated contrast, superb anatomic detail, the ability to detect aortic regurgitation and left ventricular dysfunction, and diagnosis of alternative extra-aortic pathology. Disadvantages include lack of availability and operator expertise, length of examination, incompatibility with implanted metal devices, need for sedation, and monitoring difficulties during examination (especially with hemodynamically unstable patients).[7]

MRA requires the use of gadolinium-based agents. Ideally, these studies are also ECG-gated to prevent motion artifact at the aortic root and ascending aorta.[7] However, patients with contraindications to gadolinium may undergo noncontrast MRA utilizing steady-state free precession protocols, though this is inferior to MRA with contrast.[10,67] **(See Figure 6.)** Due to limited availability and operator expertise, MRA is usually used as a secondary imaging study to confirm aortic dissection or as a follow-up study for chronic aortic dissections in order to reduce radiation exposure.[10,67] However, MRA may be considered a first-line study if there are contraindications to obtaining CT.[10]

Conventional Aortography/Angiography

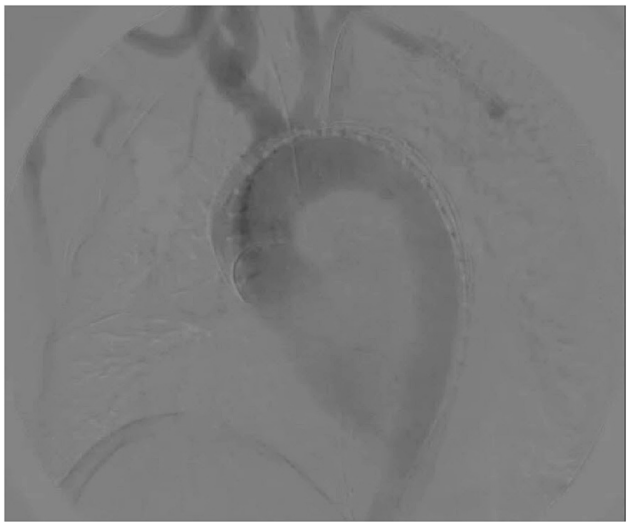
Historically, catheter-based angiography was considered the gold standard for the diagnosis of aortic dissection. Its use has fallen out of favor due to its lower sensitivity and specificity compared to noninvasive imaging such as TEE, CT, and MRI.[7,9,67] Specificity of aortography is approximately 95%, with a sensitivity of 77% to 88%; it tends to miss intramural hematoma and dissections with a throm-

Figure 6. Noncontrast Magnetic Resonance Angiogram Of Type A Aortic Dissection



Magnetic resonance angiogram of the chest showing a dissection flap (arrows) along the ascending and descending aorta.

Figure 7. Aortogram Of Aortic Dissection



bosed false lumen.[8,10] Aortography can localize the dissection flap, evaluate branch involvement, and evaluate aortic regurgitation. **(See Figure 7, page 11.)** Limited availability and risk of complications with this invasive technique limit its usage for the diagnosis of aortic dissection, though it may be used for operative planning (especially in the evaluation of coronary artery involvement). Generally, formal angiography should not be used as a first-line test.[10]

Which Diagnostic Modality To Use?

TEE, CT, and MRI all have excellent sensitivities and specificities; each imaging technique has its advantages and disadvantages. **(See Table 4.)** The decision on which modality to use as a first-line screen should be based on institutional availability and expertise. For most EDs, CT angiography will likely be the first-line test, due to its widespread availability.[10] However, in unstable patients (especially if there is a contraindication to CT), TEE may be the more appropriate first-line diagnostic test.[7,10] In 2002, results from the IRAD database showed that the initial modality choices for diagnosing aortic dissection were: CT (61%), TTE/TEE (33%), angiography (4%), and MRI (2%). More than two-thirds of patients received 2 or more diagnostic tests.[77] If clinical suspicion remains high and the initial diagnostic test does not show an aortic dissection, a second diagnostic test should be performed.[7]

Treatment

Aortic wall stress is directly affected by the velocity of ventricular contraction over time (dP/dt). Initiation of treatment to decrease these shear forces should occur as soon as the diagnosis is made or suspected. A target heart rate of < 60 beats/min and a systolic blood pressure between 100 and 120 mm Hg are recommended to prevent progression of dissection.[7,8] **(See Table 5.)** Intravenous narcotics should also be given and titrated to pain control.[7] These recommendations are from guidelines that are based on consensus statements made from animal and observational studies and not by randomized controlled trials.[7]

Intravenous beta blockers should be administered first, in order to control both heart rate and blood pressure. Options include propranolol, metoprolol, labetalol, or esmolol. Esmolol has the advantage of a very short half-life, while labetalol is an alpha- and beta-receptor antagonist and may be more effective in controlling both heart rate and

Table 4. Imaging Modalities For Acute Aortic Syndromes

Modality	Advantages	Disadvantages
TTE	• Can be done at bedside • Can diagnose pericardial tamponade	• Poor sensitivity and specificity
TEE	• High sensitivity and specificity • Can be done in unstable patients (at bedside) • No radiation or contrast • Can diagnose aortic valve function, LV function, and pericardial tamponade	• Requires an experienced operator • Limited availability • Requires sedation • Risk of esophageal injury/perforation • Limited visualization of arch and coronary arteries
CTA (first-line)	• High sensitivity and specificity • Fast and noninvasive • Widely available • Evaluates vascular anatomy, including aortic branches • Evaluates pericardial effusion • Can give alternative diagnosis	• Radiation and IV iodinated contrast • Unable to evaluate aortic pathology and LV dysfunction (note: cardiac CTA can evaluate LV function) • Cannot reliably diagnose pericardial tamponade
MRI/MRA	• High sensitivity and specificity • Noninvasive • No radiation or iodinated contrast • Can identify aortic pathology and LV dysfunction • Can give alternative diagnosis	• Prolonged duration of study • Transport from ED • Contraindications with implantable device • Requires radiology expertise for interpretation • May require sedation
Catheter angiography	• Assists in surgical planning • Localizes intimal tear	• Requires availability of interventionalist • Radiation and IV iodinated contrast • Transport from ED • May miss intramural hematoma • Invasive procedure with potential complications • May not give alternate diagnosis for symptoms

Abbreviations: CT, computed tomography; CTA, computed tomographic angiography; ED, emergency department; IV, intravenous; LV, left ventricular; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; TEE, transesophageal echocardiography; TTE, transthoracic echocardiography.

blood pressure as a single agent.[7] Patients with contraindications to beta blockers (eg, severe asthma, chronic obstructive pulmonary disease, acute congestive heart failure, or cocaine toxicity) should be given intravenous calcium-channel blockers such as verapamil or diltiazem. In the setting of severe aortic regurgitation with the use of intravenous beta blockers or calcium-channel blockers, caution is advised. In the case of cocaine toxicity, intravenous benzodiazepines should be given to decrease the sympathetic drive.[7,8]

To further reduce blood pressure, intravenous vasodilators or angiotensin-converting enzyme (ACE) inhibitors should be administered. Traditionally, nitroprusside has been described, but other agents (such as nicardipine, nitroglycerin, or clevidipine) may be used.[7,78,79] Beta-blocker therapy should be given prior to initiating intravenous vasodilators to prevent reflex tachycardia. Aggressive pain control can augment the effects of heart rate and blood pressure control.[7]

Shock

The literature provides little guidance regarding the management of hypotension and shock in aortic dissection. Hypotension and shock have been associated with worse outcomes and may be caused by aortic rupture, pericardial tamponade, or severe aortic regurgitation. Shock is an indication for immediate repair.[7,31,37,38] Pseudohypotension should be excluded prior to aggressive resuscitation. Intravenous fluids may be used initially, with the addition of vasopressors, to carefully restore perfusion. However, inotropes should be used with caution, as they may increase shear forces, potentially worsening the dissection.[7]

Pericardial tamponade is a common complication of type A aortic dissection and intramural hematoma.[7,39] One case series of 10 patients showed an increase in recurrent pericardial bleeding and increased mortality following emergent pericardiocentesis.[80] However, another study showed no increase in mortality with pericardiocentesis in patients with type A intramural hematoma.[81] Current guidelines suggest that pericardiocentesis should only be performed if the patient is unlikely to survive to surgery.[7]

Complications

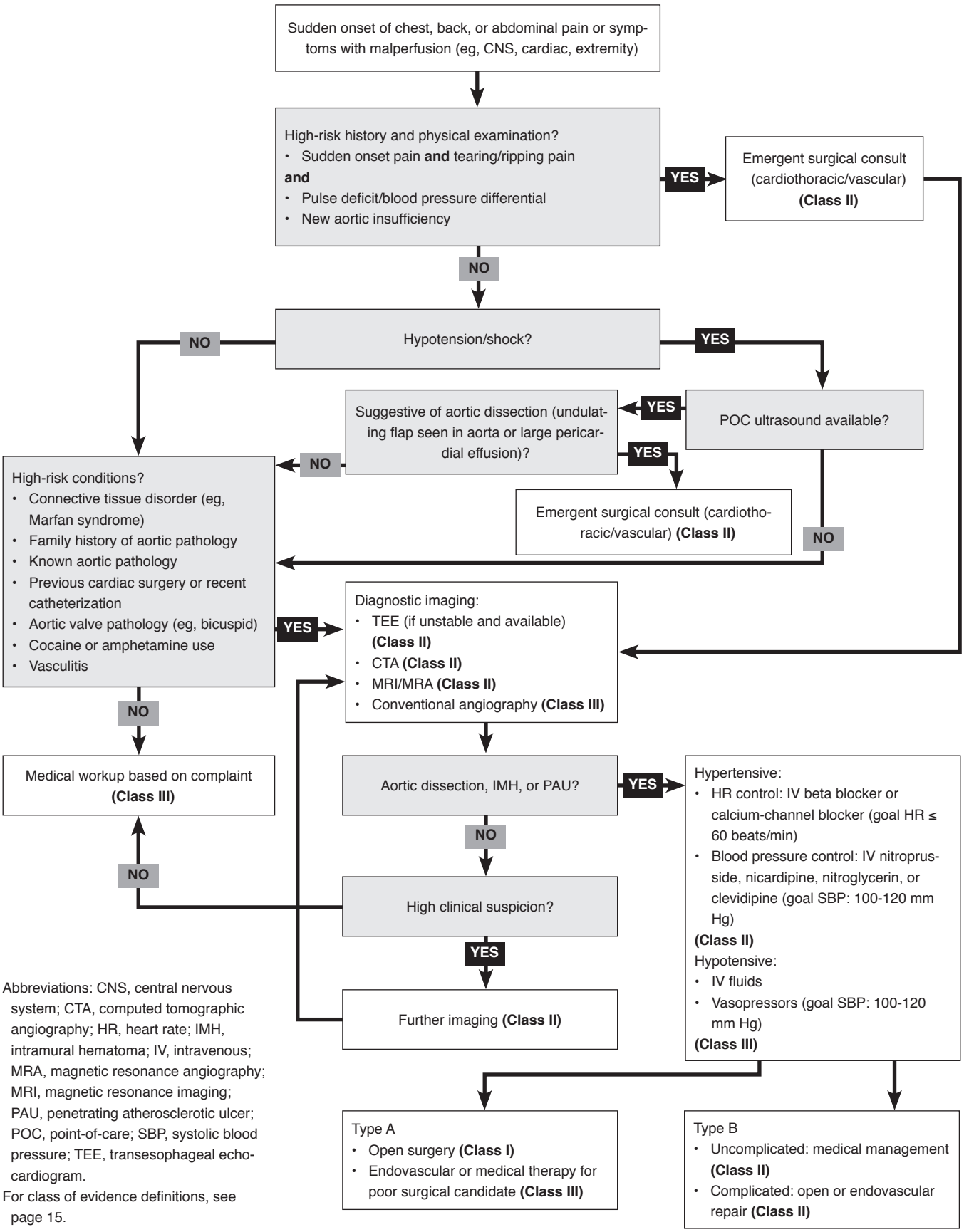
For any patient in whom aortic dissection may be in the differential, careful evaluation should be performed prior to initiating anticoagulation treatment. In a retrospective study of 44 patients with aortic

Table 5. Medication Therapies For Acute Aortic Syndromes[7,78,79]

Medication	Dosage	Comments
Beta blockers (recommended as first-line treatment; target heart rate < 60 beats/min		
Esmolol Beta 1-receptor blocker	Bolus 500 mcg/kg IV, then infusion at 50-200 mcg/kg/min	Preferable due to short half-life and easy titration; may be preferred in asthma/COPD
Labetalol Alpha 1-, beta 1-, and beta 2-receptor blocker	10-20 mg IV push q10min up to 300 mg maximum; infusion 0.5-2.0 mg/min	May be used as a single agent
Metoprolol Beta 1-receptor blocker	5 mg IV q5min up to 15 mg maximum	No IV infusion available
Propranolol Beta 1-, beta 2-receptor blocker	1 mg IV q5 min up to 0.15 mg/kg maximum	No IV infusion available
Calcium-channel blockers (target heart rate < 60 beats/min)		
Diltiazem	Bolus 0.2-0.25 mg/kg IV, then infusion 5-15 mg/hr	Second-line for heart rate control when beta blockers are contraindicated (eg, cocaine toxicity, COPD, or asthma exacerbation)
Verapamil	5-10 mg IV	NA
Vasodilators (give beta blocker first to prevent reflex tachycardia; target SBP 100-120 mm Hg)		
Clevidipine	Start 1-2 mg/h infusion. Titrate every 90 sec to a maximum of 32 mg/h	May be used as a first-line vasodilator
Nicardipine	Start 2.5-5 mg/h infusion. Titrate 2.5 mg/h q5min to a maximum of 15 mg/h	May be used as a first-line vasodilator
Nitroprusside	Start 0.3-0.5 mcg/kg/min infusion. Titrate to a maximum of 2 mcg/kg/min	May be used as a first-line vasodilator; protect from light; increased risk of cyanide toxicity when used in patients with renal or hepatic impairment
Nitroglycerin	Start 10-20 mcg/min infusion. Titrate 5-10 mcg/min q10min to a maximum of 100 mcg/min	Not a first-line vasodilator

Abbreviations: COPD, chronic obstructive pulmonary disease; IV, intravenous; NA, not applicable; q, every; SBP, systolic blood pressure.

Clinical Pathway For Management Of Acute Aortic Syndromes In The Emergency Department



Abbreviations: CNS, central nervous system; CTA, computed tomographic angiography; HR, heart rate; IMH, intramural hematoma; IV, intravenous; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; PAU, penetrating atherosclerotic ulcer; POC, point-of-care; SBP, systolic blood pressure; TEE, transesophageal echocardiogram.

For class of evidence definitions, see page 15.

dissection, 9 patients were anticoagulated due to suspicion for acute coronary syndromes. Of these, 4 patients suffered complications, including 1 bleeding complication after surgery, 1 stroke during reversal of anticoagulation, and 2 deaths from bleeding complications. In the patients who were anticoagulated, ECG changes (either ST depression or ST elevation) were more common (89% vs 6%), and there was a normal mediastinum on chest radiograph (0% vs 67%).[82] However, unless there are high-risk features for dissection, treatment of acute myocardial infarction should not be delayed to rule out dissection.

Aortic dissection can also present as an acute stroke. While the majority of these patients will have some pain, only two-thirds will have chest pain.[40] Poor outcomes have been described in giving thrombolytics for acute stroke in the setting of an acute aortic dissection, though 1 case report described a favorable outcome after intravenous tissue plasminogen activator (tPA).[83,84] Ultrasound of the carotids can be performed to evaluate for possible carotid dissection in the setting of an acute stroke, which may further suggest an aortic dissection. Isolated carotid dissection in acute stroke does not increase the risk of bleeding, and it is not a contraindication for thrombolytics.[85]

Type A Dissections

Because of the high mortality rate of type A dissections, surgical therapy is generally recommended.[7-9] In the IRAD database, patients undergoing surgery had a survival rate of 96.1% and 90.5% at 1 and 3 years, respectively, versus 88.6% and 68.7% in patients without surgery.[86] Inhospital mortality was higher in unstable patients compared to stable patients (25% vs 11%).[87] If extension of the dissection involves the aortic valve or coronary arteries, aortic valve replacement and/or coronary artery bypass grafting may be indicated.[7]

In patients presenting with coma (Glasgow Coma Scale score < 11) and type A dissection, immediate surgical repair is associated with improved outcomes. In a retrospective study of 27 patients presenting with an acute aortic dissection and coma, hospital mortality was 14% in the immediate surgical group compared to 67% in the delayed group. Full recovery of consciousness occurred in 86% of patients in the immediate surgical group compared to only 17% in the delayed group.[88] In the IRAD database, 5-year survival for patients with type A dissection presenting to stroke or coma was 23.8% and 0%, respectively, after medical management compared with 67.1% and 57.1%, respectively, after surgery.[41] Type A dissections with associated coma or cerebrovascular accident should be surgically managed.

Elderly patients, especially those aged > 70 years, are thought to be at a higher risk of death.[31,37,89] However, data from the GERAADA database have shown acceptable survival rates in the elderly, with a mortality of 11% to 14% for patients 20 to 40 years of age and peaking at 25% for octogenarians, with no increased risk of stroke or length of stay.[90]

Patients with type A dissection (especially those with chronic dissection, intramural hematoma, or penetrating atherosclerotic ulcer without involvement of the aortic valve or coronary arteries) who are unfit for open surgical repair may undergo endovascular repair. Limited data exist for this therapy, and it should be considered only in high-risk patients who are not suitable for open surgical repair.[91,92]

Type B Dissections

Patients with uncomplicated type B dissections are managed medically, usually in the intensive care unit, with strict blood pressure and heart rate control.[7-9,93,94] Shock/hypotension, the absence of chest or back pain, and branch vessel involvement are often referred to as the "deadly triad," and this portends a worse outcome.[95] Previous outcomes data have shown increased survival with medical therapy compared to surgical therapy.[7] However, in patients with complicated type B dissection, surgical repair is indicated; if untreated, it is associated with a mortality of 50% to 85%.[93] "Complicated" dissection is defined as end-organ ischemia, leaking or rupture; aorta dilation; or continued pain.[18-20]

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

In complicated type B dissections, open surgical repair has traditionally been done; however, the use of thoracic endovascular aortic repair (TEVAR) has recently become an attractive option, as it is less invasive and has a shorter recovery time (especially in patients who are poor surgical candidates).[7,18] TEVAR has been shown to improve perfusion to end organs by increasing true lumen size and decreasing false lumen and aortic size.[18,19] Complications of TEVAR include neurologic deficits, retrograde progression to type A dissection, renal failure, and endoleaks requiring revision or open repair.[7,19,96] As a result, TEVAR is usually performed only in centers with significant expertise.[7] There are no randomized trials comparing TEVAR with open repair or medical therapy for complicated type B dissections, and recommendations are based on observational cohort studies, often in comparison with historical controls.[7] Data from the IRAD database showed an in-hospital mortality of 11% in the endovascular group compared to 34% in the open repair group.[20] Other trials have shown favorable survival rates with TEVAR (90% at 1 year and 87% at 5 years), with a favorable aortic remodeling of 30% at 1 year.[97, 98] Tight, long-term heart rate management is recommended in all patients with type B dissections.[93] One retrospective study of 171 patients found that tight heart rate control of < 60 beats/min decreased long-term aortic events compared to heart rates > 60 beats/min (odds ratio [OR], 0.2; CI, 0.08-0.77).[94]

Special Circumstances

Intramural Hematoma

The presentation, evaluation, and treatment of intramural hematomas are similar to classic aortic dissection.[7] Diagnostic imaging such as CT, MRI, or

Risk Management Pitfalls For Acute Aortic Syndromes (Continued on page 17)

1. **"The D-dimer was negative, so I assumed the patient did not have an aortic dissection and sent him home."**
   While D-dimer has shown promise in the evaluation of possible acute aortic dissection, there have been no large prospective studies to validate this strategy. Unfortunately, no biomarkers currently have been validated to rule out an aortic dissection. Intramural hematoma or aortic dissections with a thrombosed false lumen can have a false negative D-dimer. If there is sufficient clinical suspicion for an acute aortic dissection, advanced imaging is indicated.

2. **"The patient didn't have tearing chest pain, a pulse or blood pressure differential, or a widened mediastinum on chest x-ray; therefore, he couldn't have an aortic dissection."**
   While studies have shown a decreased probability for an aortic dissection when none of these features are present, this strategy has not been validated to safely rule out an aortic dissection. Approximately 5% of acute aortic dissections will not have any associated pain, and 38% will not have a widened mediastinum on chest x-ray.

3. **"The patient was only 28 years of age and had reproducible chest pain. I thought the patient had costochondritis, so I sent him home with NSAIDs. I didn't know he could have had an aortic dissection!"**
   While acute aortic dissections usually occur in those who are older, they can occur at any age. Patients with a history of (or suspected) Marfan syndrome, another connective tissue disorder, or a history of a bicuspid aortic valve should always have aortic dissection in the differential. A thorough history and physical examination should be performed to evaluate for all possible risk factors.

4. **"She was 56 years of age, with chest pain and a slightly elevated troponin. I thought it was acute coronary syndromes, and I anticoagulated her while waiting for the cardiologist to see her."**
   Although acute coronary syndromes are more common than acute aortic dissection, aortic dissection should always be considered in anyone who presents with chest pain, even with elevations in cardiac biomarkers. History of connective tissue disorder, bicuspid valve, or illicit drug use (such as cocaine) should increase suspicion. Studies have shown missed aortic dissections to be more likely diagnosed as acute coronary syndromes. Evaluation of the chest x-ray and a good history and physical examination can help risk stratify patients for possible aortic dissection.

5. **"She was 65 years old and presented with syncope without chest pain or shortness of breath. I thought it might have been an arrhythmia, so I just admitted her to a telemetry bed."**
   Approximately 12% of patients with aortic dissection will have syncope. Elderly patients may not have classic symptoms associated with aortic dissection.

TEE may be used. Approximately 16% of intramural hematomas will progress to a classic aortic dissection on serial imaging.[99] Because of the high mortality associated with type A intramural hematomas, immediate surgery is often recommended for these patients, especially if it is complicated by pericardial effusion or rupture.[26,100] Mortality is similar for both type A intramural hematoma compared to type A classic aortic dissection and for type B intramural hematoma compared to type B classic aortic dissection.[26,101] Since studies have shown a higher mortality rate in patients with type A intramural hematoma who are managed medically compared to those managed surgically, early surgery is recommended for type A intramural hematoma. One study showed a mortality rate of 40% for medical management versus 24% for surgical management,[26] and another showed a mortality rate of 55% with medical management versus 8% with surgery.[102] However, a prospective observational study in Korea did show a comparable outcome for medical therapy compared to surgical therapy in type A dissection.[103] In select stable type A intramural hematoma cases, medical management may be reasonable and should be decided by the surgical consultant. Increased age, no beta-blocker therapy, and initial aorta diameter and hematoma thickness may predict worsening outcomes in type A intramural hematoma.[102,103] For type B intramural hematoma or intramural hematoma confined to the aortic arch, medical management is recommended unless complications arise.[26,104] In all cases, strict blood pressure and heart rate control is recommended.[7]

Penetrating Atherosclerotic Ulcer

Due to a lack of well-controlled trials, controversies exist regarding the appropriate management for

Risk Management Pitfalls For Acute Aortic Syndromes (Continued from page 16)

6. **"The patient had an inferior wall STEMI on ECG and the cardiologist was unavailable to take the patient emergently to the heart catheterization lab. I gave thrombolytics because that is what I was told to do."**
   Two percent to 5% of patients with aortic dissections will have concurrent myocardial ischemia. Proximal aortic dissections can dissect into the right coronary artery, causing occlusion, and can present with a STEMI. If clinical suspicion for aortic dissection is present, other diagnostic modalities should be used to evaluate for proximal aortic dissection prior to anticoagulation or thrombolytics.

7. **"The patient was 28 years old and had Marfan syndrome. I was concerned about an aortic dissection, but he couldn't get a CT scan due to a contrast allergy. I got a TEE and it didn't show an aortic dissection, so I sent him home and told him to follow up with his primary care provider."**
   Advanced imaging such as TEE, CT, and MRI are all very sensitive and specific; however, no modality is 100%. If there is a high clinical suspicion for an aortic dissection, a second modality should be obtained to rule out aortic dissection.

8. **"The patient presented within an hour, had right hemiparesis, and was unable to speak. He was having a stroke and was within the window for thrombolytics."**
   Acute neurological deficits can be found in up to 30% of acute type A aortic dissections, as the dissection extends into the internal carotids or to the spinal arteries. Thrombolytics in the setting of an acute aortic dissection can be fatal. Consider aortic dissection in patients who present with stroke symptoms, especially when patients have concurrent chest or back pain.

9. **"The patient had a type B aortic dissection. These patients are only treated medically, so I didn't consult surgery."**
   While uncomplicated type B aortic dissections are usually managed medically, those with a potential for rupture or organ dysfunction as a result of the aortic dissection will be considered for intervention, either with TEVAR or open surgical repair.

10. **"I ordered a pulmonary embolism study for the patient, and it didn't show a pulmonary embolism or aortic dissection. I sent the patient home and told him to follow up with his primary care provider."**
    CT angiography for pulmonary embolism is a different protocol than for an aortic dissection. While aortic dissections can be seen in a CT for pulmonary embolism, it might miss small dissections, as CT protocol for dissection uses smaller cuts. If clinical suspicion remains high for an aortic dissection, consider ordering a second imaging study.

penetrating atherosclerotic ulcer. Consensus statements have been made with regard to initial treatment, similar to classic aortic dissection (ie, heart rate and blood pressure control).[7,29] Some authors suggest aggressive treatment for patients with penetrating atherosclerotic ulcer who are symptomatic. In a retrospective study of 37 patients, urgent or emergent need for intervention was found in 40% of patients presenting with penetrating atherosclerotic ulcer, and it was associated with an increase in mortality (OR, 14.7).[105] Complications from penetrating atherosclerotic ulcer (including rupture) occur in approximately 40% of patients, with an inhospital mortality rate of 15%.[28] Indications for immediate intervention include: (1) an increase in the diameter of the aorta or the pain from the aorta or penetrating atherosclerotic ulcer, (2) rupture of a penetrating atherosclerotic ulcer, and (3) a type A penetrating atherosclerotic ulcer.[28,29]

For patients with asymptomatic penetrating atherosclerotic ulcer, medical management has been advocated. While open repair has been the standard treatment, TEVAR has been advocated as a treatment option for symptomatic penetrating atherosclerotic ulcer.[28] One study found a higher short-term mortality rate with open repair than with TEVAR but no difference in long-term mortality, with an overall survival rate of 48%.[30] Another small retrospective study of 37 patients using TEVAR for type B penetrating atherosclerotic ulcer found an overall freedom from treatment failure (as defined by aortic reintervention, rupture, or aortic-related death) of 82% at 5 years.[105] Isolated abdominal penetrating atherosclerotic ulcer is rare and is thought to have an overall worse outcome compared to thoracic penetrating atherosclerotic ulcer and these lesions require aggressive treatment.[106]

Chronic Dissections

Chronic type B dissections have a 5-year mortality of approximately 50% and an expansion rate of 0.10 to 0.74 cm/year depending on initial aortic size and blood pressure control.[93] Patients are usually followed serially (through outpatient imaging), typically at 1 year, then 2 to 3 years for interval change, due to the risk of a secondary acute aortic dissection developing. Imaging can be done with CT, MRI, or TEE;[7] ideally, similar modalities are used in follow-up to keep consistent measurements. If complications occur from the dissection, immediate repair may be needed. For patients with a chronic descending aortic dissection (especially those with connective tissue disorder) but without major comorbidities or chronic ascending aortic dissection, expansion of > 5.5 cm is an indication for open repair.[7]

Chronic dissections can be challenging in terms of repair, as the clot in the false lumen matures, making endovascular stenting more difficult. Strict blood pressure and heart rate control are needed to prevent progression of disease. Expansion of a chronic dissection involving the arch usually requires replacement of the aortic arch.[7] In some countries, the use of TEVAR has become increasingly common in uncomplicated type B dissections due to difficulties in surveillance of certain populations. A nonrandomized prospective study of 303 patients in China evaluated TEVAR against optimal medical therapy in chronic uncomplicated type B dissections. At 2- and 4-year followup, TEVAR was found to improve aorta-related mortality but not all-cause mortality, when compared to medical therapy.[19] Similarly, a prospective randomized controlled study found no statistical difference in all-cause mortality at 2 years between medical therapy and TEVAR.[107]

Controversies And Cutting Edge

Noninvasive Surgical Management

Traditionally, open surgical repair has been the mainstay for the treatment of aortic dissection. However, the use of TEVAR for type B dissection has become more and more common. Experienced centers are using this as first-line therapy in complicated type B dissections as well as in patients with select type A dissections who are not candidates for open surgical repair.[7] Some cases utilize a hybrid approach, with partial repair using open surgical techniques and partial repair with stent placement.[93] However, as newer techniques evolve and long-term data become more robust, TEVAR will likely become the standard of care over open surgical techniques in the majority of patients.

Point-Of-Care Ultrasound

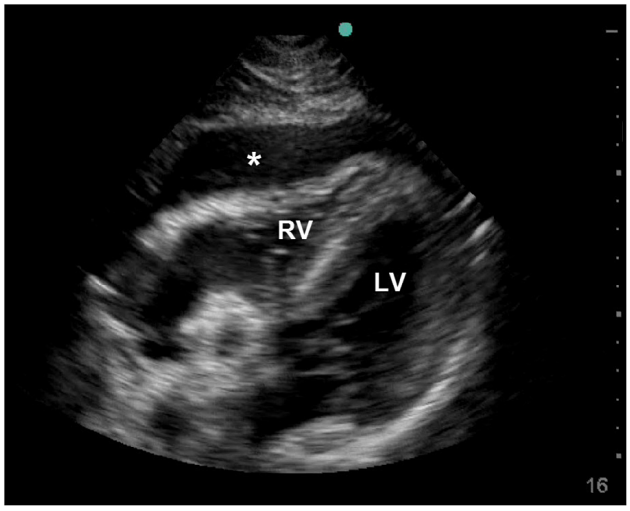
Many emergency clinicians have access to POC ultrasound. This can give the emergency clinician rapid information to help make the diagnosis, especially in critically ill patients. ACEP has produced comprehensive clinical policies regarding the use of ultrasound in emergency medicine. ACEP's clinical policy was first approved in 2001 and then updated in 2008.[108] One of the core applications is the focused cardiac ultrasound, which has been endorsed by the American Society of Echocardiography and ACEP.[109] Studies have shown that emergency physicians are capable of diagnosing pericardial effusions, with 1 study showing a sensitivity and specificity of 96% and 98%, respectively.[110] **(See Figure 8.)** This can alert the emergency clinician to a life-threatening condition (such as an aortic dissection). However, there are only a few case series and case reports published in regard to emergency physicians' ability to diagnose aortic dissection by TTE.[111,112] An undulating flap within the abdominal aorta or proximal aorta can be highly suggestive of an aortic

dissection. **(See Figure 9.)** This can be observed via transabdominal view of the aorta in either the long or short axis or transthoracic view by parasternal long axis or suprasternal notch view. The "Mercedes-Benz" sign (which has a resemblance to the Mercedes-Benz symbol within the aortic valve in the subxiphoid view) may suggest a proximal aortic dissection.[112] However, studies have shown a relatively low sensitivity for TTE.[71] While POC ultrasound can suggest possible aortic dissection, it should not be used to rule out aortic dissection.

Use Of D-dimer

Currently, only a few small studies have looked at the sensitivity of D-dimer in ruling out aortic dissection.[57] These studies were limited, as they were based on different D-dimer test characteristics and thresholds as well as spectrum bias in patients enrolled. While the use of D-dimer in low- to moderate-risk patients has been validated in patients suspected of having pulmonary embolism, no such studies exist for aortic dissection.[44] One issue is that there are no validated clinical decision rules to determine pretest probability for aortic dissection.[60] A recent paper determined thresholds for appropriate pretest probability using D-dimer testing in ruling out aortic dissection. Because of the high mortality associated with aortic dissection, they found D-dimer testing to be appropriate, with a pretest probability of 0.01% to 0.6%.[113] While a "negative" D-dimer may lower the probability of an aortic dissection, at this time, D-dimer should not be routinely used to rule out aortic dissection.[7,60]

Figure 8. Subxiphoid View Of Point-Of-Care Ultrasound With Pericardial Effusion



Point-of-care ultrasound in the subxiphoid view showing pericardial effusion (asterisk). RV = right ventricle, LV = left ventricle.

Disposition

All patients with aortic dissection should be admitted to the intensive care unit for close monitoring as well as strict blood pressure and heart rate control. Type A aortic dissections will usually require transfer to a center with cardiac surgery capabilities. Type B aortic dissections may be cared for by either a cardiothoracic surgeon or a vascular surgeon, depending on the institution. Coordination of care with the treating surgeon will determine whether or not surgical repair is indicated.

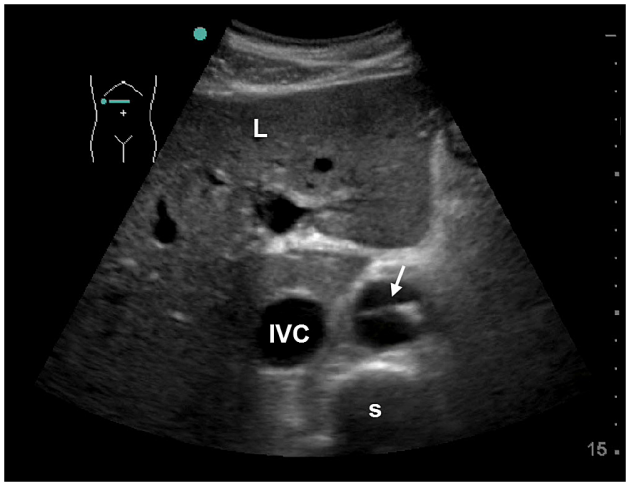
Summary

Aortic dissection is an uncommon but deadly disease. Patients with high-risk features should have an evaluation for possible aortic dissection. Current biomarkers, especially D-dimer, have shown promise in the evaluation of aortic dissection, but they should not be used alone to rule out aortic dissection. While advanced imaging options have been shown to have excellent sensitivity and specificity, if clinical suspicion for aortic dissection is high, a negative first study should be followed with another diagnostic modality. Further studies are needed to provide better algorithmic pathways and treatment strategies in the evaluation of aortic dissection.

Case Conclusions

Prior to anticoagulating the 55-year-old patient with a STEMI, you considered whether the patient might have an aortic dissection, based on his chest radiograph. On

Figure 9. Transabdominal Point-Of-Care Ultrasound Showing An Aortic Dissection



Point-of-care ultrasound showing a dissection flap (arrow) in the descending aorta. IVC = inferior vena cava; S = spine; L = liver.

further examination, the patient had decreased pulses and blood pressure between the right and left arms. You held off on anticoagulation and obtained a CT angiogram, where a type A aortic dissection was found. You called the cardiologist back to update her on the patient and contacted the cardiothoracic surgeon for evaluation. The patient was sent emergently to the operating room where he underwent operative repair for his aortic dissection and coronary artery bypass grafting. The patient was admitted to the ICU in critical but stable condition.

Although you were tempted to discharge the 21-year-old patient with reproducible chest pain with costochondritis, you noticed that he appeared to have marfanoid features. Since your CT scanner was down, you decided to do a bedside ultrasound, which showed a large pericardial effusion with early signs of tamponade and an undulating flap within the descending abdominal aorta. You immediately started an esmolol and nicardipine drip and transferred the patient to the local tertiary care center where he was ultimately diagnosed with a type A dissection and underwent immediate repair. You reminded yourself to thank the nurse the next time you see him for not letting you dismiss the patient so quickly.

Time- And Cost-Effective Strategies

1. **Use your gestalt.** Currently, no validated clinical decision rule exists to rule out an acute aortic dissection. Historical and physical findings can be used to increase or decrease probability, but atypical presentations are not uncommon. Although rare, aortic dissection should always be in the differential for patients with chest pain, back pain, or abdominal pain.
2. **Be aggressive in the evaluation and management of these patients.** Mortality rates are up to 1% to 2% per hour for an acute aortic dissection. Initial stabilization and surgical consultation are urgent once a diagnosis is strongly suspected or made.
3. **Choose the best diagnostic tool.** CT will often be the first-line tool in diagnosing an aortic dissection in the ED. The use of POC ultrasound can be helpful in unstable patients in the initial evaluation, but it should not be relied upon to rule out an aortic dissection. TEE, when available, should also be considered in unstable patients. MRI may also be used in stable patients and has the advantage of not needing contrast. Remember, no test is 100% sensitive. If a high suspicion exists for aortic dissection and the initial TEE, CT, or MRI is negative, additional studies may be needed. D-dimer alone should not be used to "rule out" aortic dissection.

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Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study will be included in bold type following the reference, where available.

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1. Acute aortic syndrome encompasses all of the following EXCEPT:
   a. Aortic aneurysm
   b. Aortic dissection
   c. Intramural hematoma
   d. Penetrating atherosclerotic ulcer

2. A Stanford type B dissection refers to:
   a. DeBakey type I and II dissection
   b. Dissection of the ascending aorta only
   c. Dissection of both the ascending and descending aorta
   d. Dissection of the descending aorta only

3. **Compared to a type A intramural hematoma, a classic type A aortic dissection is more likely to:**
   a. Be associated with a penetrating atherosclerotic ulcer
   b. Cause the patient to complain of chest pain
   c. Cause aortic regurgitation
   d. Cause pericardial tamponade

4. **Which of the following laboratory tests has been validated in ruling out aortic dissection?**
   a. Calponin
   b. D-dimer
   c. Matrix metalloproteinases
   d. None of the above

5. **Which of the following is true in regard to diagnostic imaging for aortic dissection?**
   a. CT is not as sensitive as TEE
   b. When compared to TEE, TTE has equal specificity but lower sensitivity
   c. MRI always requires intravenous contrast
   d. TEE should be considered in unstable patients

6. **Which radiographic modality has the lowest sensitivity and specificity in the diagnosis aortic dissection?**
   a. CT b. MRI
   c. TEE d. TTE

7. **To decrease shear force, which of the following should be the initial medical therapy for aortic dissection?**
   a. Esmolol
   b. Clevidipine
   c. Nicardipine
   d. Nitroprusside

8. **Initial management of intramural hematoma and penetrating atherosclerotic ulcer is the same as for a classic aortic dissection.**
   a. True
   b. False

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