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REVIEW ARTICLE

Cardiology

Diagnosing aortic dissection: A review of this elusive, lethal diagnosis

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Abstract

Aortic dissection (AD) remains a difficult diagnosis in the emergency setting. Despite its rare occurrence, it is a life-threatening pathology that, if missed, is typically fatal. Previous studies have documented minimal improvement in timely and accurate diagnoses despite the advancement of computed tomography. Previous literature has highlighted aortic dissections as a major cause of serious misdiagnosis-related harm. The aim of this article is to review the available literature on AD, discussing the diversity in presentations and the prevalence of historical and exam features to better aid in the diagnosis of AD. AD remains a difficult diagnosis, even with the widespread prevalence of computed tomography angiography usage. No single feature of the history or physical examination is enough to raise suspicion. The diagnosis should be strongly considered in any patient with chest pain that is severe and unexplained by other findings or testing. Those who do not present with acute pain are often complicated by neurologic deficits, hypotension, or syncope. These patients suffer from a change in mental status limiting their ability to participate in the history and physical examination and have a higher rate of complications and mortality. An educated understanding of the atypical presentations of aortic dissection helps the clinician to realistically rank it on the differential diagnosis, culminating in judicious use of definitive imaging.

KEYWORDS
aortic dissection, diagnostic errors, misdiagnosis, ultrasonography

1 | BACKGROUND

More than 30 years since the advent of computed tomography angiography (CTA), clinicians still face difficulty in accurately diagnosing aortic dissection.[1] In 2000, Mészáros and colleagues reported a series of 86 cases for whom only 13 (15%) were correctly suspected as having aortic dissection from the initial clinical presentation.[2] Untreated, it is a fatal condition, with a mortality rate of 40% on initial presentation, increasing by 1% each hour up to an annual mortality rate of 90%.[3]

One pathologic study queried cardiac specimens from autopsies with aortic dissection (AD) and compared two cohorts from 1956 to 1992 and 1993 to 2015. They found that 63% of AD cases were first identified during autopsy.[4] Both cohorts had no significant difference in miss-rates. Improved radiologic techniques and widespread availability of CTA have not enhanced diagnostic acumen of this condition. In December 2022, the Agency for Healthcare Research and Quality released a controversial report covering diagnostic errors in the emergency department. The report highlighted aortic dissections as a top-15 clinical condition associated with serious misdiagnosis-related harms.[5]

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The classically taught triad of chest pain that is sudden, tearing, and radiating to the back does occur, but distinctions must be made.[6]

1.1 | Objective

The aim of this article is to review the available literature on AD, discussing the diversity in presentations and the prevalence of historical and examination features to better aid in the diagnosis of AD.[7]

2 | METHODS

A literature review of PubMed databases was performed for articles up to December 31, 2022. Keywords used when searching included "aortic dissection" and "acute aortic syndrome." The authors included systematic reviews, clinical guidelines, as well as retrospective and prospective studies. Studies published outside the United States were included. The authors evaluated each study and decided which studies were appropriate to include based on their relevance to emergency medicine. A total of 56 studies were selected for inclusion.

3 | REVIEW

3.1 | Pathophysiology

The aorta, the largest artery in the body, supplies blood to every major organ system.[8] AD occurs when there is a tear in the intima, leading to the creation of a false lumen that expands into the media.[9] AD occurs in approximately 2.6–3.5 per 100,000 person-years.[2] Both the Stanford and DeBakey Classifications are used to categorize types of aortic dissection (Figure 1).[10,11] Of the two, the Stanford System is the most widely recognized classification and has the advantage of classifying dissections into ones which require immediate surgical intervention.[12] Type A dissections involve any portion of the ascending aorta, specifically proximal to the left subclavian artery.[10] Type B dissections involve the descending aorta, defined as distal to the left subclavian takeoff.[10] Ascending AD is nearly twice as common as descending.[11,13,14]

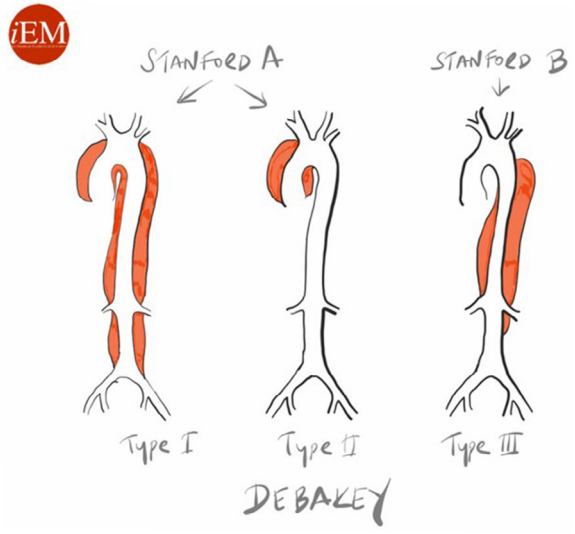

FIGURE 1 De Bakey and Stanford classification of aortic dissection. Illustrated by AA Cevik by using figure in Ref. 15.

Aortic rupture is the most common cause of death, but due to the visceral innervation of this large artery and depending upon exactly where the tear begins, a variety of pathologies with diverse presentations may result.[15] If the dissection moves proximally, cardiac complications including aortic regurgitation, tamponade, or myocardial infarction may occur.[16–18] If the tear moves into the cranial arteries, neurologic events occur ranging from minor to stroke-like symptoms.[14] Finally, a tear moving distally into the abdomen causes mesenteric, renal, or limb ischemia.[19]

3.2 | Risk factors

AD can occur at any age, with a male to female ratio of 2:1 and a mean age of 63.[20] The most common risk factors to consider are hypertension, Marfan's syndrome, and preexisting aortic aneurysm. History of hypertension is present in nearly 75% of patients, making it the most common comorbid condition.[13] There are multiple conditions that predispose younger patients to AD. A preexisting aneurysm is more common in those <40 years old (19% vs. 13% across all ages).[8] In those aged <40, about 50% of dissections are associated with Marfan's syndrome.[21]

Patients with a recent cardiac catheterization may present the bias of diagnostic momentum toward chest pain being from acute coronary syndrome. Indeed, cardiothoracic surgery and cardiac catheterization have been found to be risk factors, albeit uncommon.[22] One urban study found that 37% of AD are associated with cocaine or methamphetamines.[23]

Pregnancy alone is a fourfold risk.[24] Bicuspid aortic valves have been implicated in 9% of AD in those aged <40 years, and those with a bicuspid valve are eight times more likely to suffer from AD.[25]

3.3 | Clinical symptoms specific for AD versus nonspecific

While pain is observed in 90% of patients, this is not very specific.[7] There are certain descriptors of pain highly associated with AD. Sudden onset pain (+LR 2.6), tearing or ripping pain (+LR 10.8), and migratory pain (+LR 7.6) all have impressive likelihood ratios and should raise concern if mentioned.[26] Unfortunately, patients describe their pain as tearing or ripping only 30%–40% of the time, with the descriptor of sharp being more common. Migrating pain is only seen in 16% of cases.[3] Regardless, sudden onset of severe, acute pain that is maximal at onset and migrates should be very concerning for AD.[27] The fact that pain may decrease after initial onset is falsely reassuring, leading to delayed

Typical Presentation

Patient presents with acute onset of chest pain.

- Sudden onset of pain
- Pain described as sharp or tearing
- Pain radiates to back or abdomen
- Pain severe enough to require: frequent and/or larger doses of analgesics
- Patient has a personal or family history of Marfan's, Ehlos-Danlos, or Bicuspid aortic valve
- Patient endorses recent cocaine or amphetamine usage

Atypical Presentation

- Patient is hypotensive with complaints of chest, back, abdominal pain, and/or syncopal episode
- Patient has stroke-like symptoms
- Patient has altered mental status with an abnormal neurovascular exam concerning for pulse or neurologic deficits

FIGURE 2 Two key presentations of patients with suspected aortic dissection.

TABLE 1 Incidence of key symptoms in aortic dissection based on Stanford Classification type from the International Registry of Acute Aortic Dissection.

Symptom	Stanford A	Stanford B
Chest pain	79%	63%
Back pain	43%	64%
Abdominal pain	22%	43%
Elevated blood pressure	<35%	70%
Low blood pressure	25%	<5%
Syncope	19%	3%
Stroke-like symptoms	18%	4%

TABLE 2 Key objective findings that may be seen in aortic dissection.

Pulse deficit in >1 extremity
Neurologic deficit in >1 extremity
ST elevation myocardial infarction on ECG
Widened mediastinum on chest x-ray
Loss of the aortic knob on chest x-ray
Pericardial effusion on bedside ultrasound
Free fluid in the abdomen on bedside ultrasound

Abbreviation: ECG, electrocardiogram.

recognition.[28] Location of the pain varies with site of the initial tear and the progression of the dissection. The incidence of chest pain is 79% and 63% in Stanford type A and type B dissections, respectively[29] (see Table 1).

In one study of 141 cases of AD, patients presenting without chest pain were more likely to complain of back pain (44.3% vs. 27.5%) or abdominal pain (36.1% vs. 12.5%) as well as experience shock (21.3% vs. 7.5%).[30] Furthermore, the onset of the pain in those patients without chest pain was less likely to be abrupt (31.1% vs. 47.5%, see Figure 2).

Elevated blood pressure cannot be completely relied upon when patients present with AD. Only 49% will present with a systolic blood pressure > 150 mmHg (less than 35% of Type A; 70% of Type B).[6] In contrast, hypotension may be present in up to 25% of those with Type A AD and <5% in those with Type B.[13]

Rarely, patients may present with "painless dissections," where acute pain is not reported.[31] One retrospective review found "painless dissections" at a rate of <6%.[28] Another retrospective review from Japan stated that of 98 patients with AD, 17% had a "painless" presentation.[32] Those with a painless dissection are more likely to have a disturbed consciousness, either from syncope or neurologic deficits. These patients have worse outcomes and may have a delay in diagnosis.[33] Patients who are hypotensive (SBP < 80 mmHg) are more likely to have painless dissections. Hypotension on initial presentation to the ED has been found to be an independent correlate of in-hospital mortality.[34]

3.4 | Physical findings

In terms of the physical examination, the traditional teaching regarding inter-arm blood pressure differences may be true but is not sensitive or specific enough to help in diagnosis. One office study found that 20% of patients without AD had a blood pressure differential >20 mmHg between both upper extremities, and 50% had a difference >10 mmHg.[35] One retrospective analysis found that inter-arm blood pressure difference was not associated with Type B dissections.[36] Up to 40% of patients will have a new murmur, specifically a diastolic murmur that suggests aortic regurgitation, but this is difficult to auscultate in the ED setting.[6]

A meta-analysis on the exam features most predictive of AD included pulse and neurologic deficits.[3] Occurring in 19%–30% of AD cases, pulse deficits are the most common abnormal examination finding (+LR 5.7).[37] Their presence is concerning for increased in-patient complications and mortality.[38] Pulse deficits provide better diagnostic accuracy than inter-arm difference in blood pressure[39] (see Table 2).

Neurologic symptoms, specifically focal deficits, may disguise the underlying diagnosis of AD. Up to one-third of AD patients with neurologic symptoms do not complain of chest pain.[40] These patients are more likely to have a delay in diagnosis as well as higher in-patient mortality.[41]

3.5 | Ancillary testing

While the electrocardiogram (ECG) is normal in 30% of AD patients, 19% of patients can have various ST segment and T wave changes suggestive of ischemia. Myocardial infarction has been found in 7%.[6] The clinician should properly assess their patient and challenge themselves to think of alternative explanations when a patient does not fit the classic presentation of acute coronary syndrome with ECG changes.[16] Any ischemic changes of ECG have been found to be independently associated with increased in-hospital mortality.[42]

The classic findings on chest radiograph often are not there to raise suspicion.[43] Nearly 60% of patients will have a widened mediastinum.[5] Most radiologic guidelines agree that a mediastinum greater than 6–8 cm is considered wide, and the current chest radiograph should be compared to a prior one if possible.[44] Loss of the aortic knob (flattening of the aortic arch) and the "calcium sign" (separation of the internal calcification from the outer aortic knob by >0.5 cm) are both uncommon.[6]

3.6 | Decision rules

D-Dimer testing has received more attention as a possible screening test for AD. The ADvISED trial in 2017 examined the use of the Aortic Dissection Detection Risk Score (ADD-RS) paired with D-dimer testing. The authors found when the D-dimer was <500 ng/mL within 24 h of symptom onset, it had a negative LR of 0.07 for aortic AD.[45] A negative D-dimer test paired with an ADD-RS score of 0 had a sensitivity of 99.6% for AD, while an ADD-RS of 1 with a negative D-dimer test had a sensitivity of 98.8% for AD.[46] Despite its promise, this was an observational study with no external validation. It contained many confounding variables, including lack of physician blinding, no patient follow up after 14 days, and no established, acceptable miss rate of AD in the medical community.[41,47] Another scoring system, the aorta simplified (AORTAs) score, has six criteria that may be scored by the clinician. If a score ≥2 points is present, there is a high probability of AD. Like the ADD-RS, the AORTAs score has not been externally validated and was only evaluated in a prospective derivation cohort.[48] Consequently, risk scoring systems with biomarkers may be useful to rule out AD, but further validation is required before widely accepted implementation can begin.[49] In a clinical policy statement from the American College of Emergency Physicians, D-dimer testing and risk scoring systems received a Level C recommendation, and they specifically advised clinicians to not rely on D-dimer testing as a means to exclude AD.[47]

3.7 | Bedside imaging

Bedside transthoracic echocardiography has a role in assessing for AD. Bedside ultrasound is heavily operator dependent and requires practice.[50] Bedside ultrasound views can visualize dissection flaps within the proximal 8 mm of the ascending aorta, as well as the descending aorta.[51] The presence of a hyperechoic dissection flap is highly specific for AD.[52] Bedside ultrasound is useful to identify complications associated with AD, including aortic regurgitation, aortic valve dilation, regional wall motion abnormalities, and pericardial effusion, with the last being the easiest to identify. Each of these findings are concerning for AD, but their absence does not rule out the diagnosis.[43] In the undifferentiated hypotensive patient, bedside ultrasound has proven utility.[53] The ability to rapidly evaluate for causes of shock can improve timely diagnosis and change in management. A FAST examination may be utilized to identify free fluid in the abdomen, which would suggest aortic rupture.[54] Pericardial effusions have been found in 33% of all AD patients, while tamponade has been found to complicate 10% of all cases in one study.[17] Most recently, one observational retrospective study found that a bedside ultrasound protocol dedicated to evaluation of AD had an overall sensitivity of 93.2%.[55] Forty-one of the 44 cases had at least one of the following three sonographic signs that were specifically evaluated: pericardial effusion, intimal flap, or aortic outflow track diameter more than 35 mm.

3.8 | Definitive diagnosis

CTA of the aorta is gold standard for diagnosis. One systematic review of 82 studies involving 57,311 patients found CTA to have a sensitivity of 100% when performed with the correct contrast timing. Standard contrast-enhanced CT chest imaging highlights lung and mediastinal pathology at the expense of vascular structures and should be avoided, as it drastically reduces specificity, identifying the intimal flap in <75% of cases.[56] The key finding is the presence of an intimal flap in the aorta.[15]

3.9 | Overview of management

Once the diagnosis is confirmed, immediate cardiac surgical consultation is needed. If these services are not available, transfer to the nearest facility for definitive management is critical.[49] As cardiac surgical consultation is initiated, management priorities include pain control and rapid lowering of blood pressure if the patient is hypertensive. Both therapies are considered "anti-impulse" and reduce the rate of false lumen expansion. Preferred agents include fentanyl for pain control, along with esmolol or labetalol[49] titrated to pulse rate goal of ~60 beats/min and systolic blood pressure goal of <120 mmHg.

3.10 | Future research

Despite its rare occurrence, AD is a life-threatening pathology that, if missed, is fatal.[3] Despite the proliferation of CT availability in the acute care setting, there is still opportunity to reduce the burden of morbidity and mortality from AD. Future research should be directed at risk stratification, with development and multicenter validation of clinical decision rules to aid in the diagnosis of AD. D-Dimer testing shows promise as a potential sensitive assay, combined with the use of risk scoring.

4 | CONCLUSION

AD remains a difficult diagnosis, even with the widespread prevalence of CT usage.[4] No single feature of the history or physical exam is enough to raise suspicion.[6] The diagnosis should be strongly considered in any patient with chest pain that is severe and unexplained by other findings or testing. An educated understanding of the atypical presentations of aortic dissection helps the clinician to realistically rank it on the differential diagnosis, culminating in judicious use of definitive imaging.

AUTHOR CONTRIBUTIONS

Blake Briggs wrote manuscript. David Cline developed figures and tables, reviewed manuscript, and provided reference organization.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

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