

Thoracic Aortic Syndromes



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KEYWORDS

- Aortic dissection • Aortic intramural hemorrhage • Penetrating aortic ulcer

KEY POINTS

- Aortic dissection is an uncommon disease that often presents with varied and atypical findings suggestive of more frequently encountered conditions; therefore, it poses an exceptional diagnostic challenge to emergency providers.
- Mortality associated with aortic dissection is significant at presentation and advances with every hour the lesion is left untreated.
- Although almost all patients who have symptoms possibly caused by aortic dissection will not have aortic dissection, key features of the disease, including risk factors, pain characteristics, physical examination findings, and routine ancillary studies, allow clinicians to develop a rational approach to diagnostic testing.
- When the diagnosis is sufficiently likely to indicate definitive testing, computed tomography angiography is the advanced imaging test of choice in most centers, but transesophageal echocardiography and MRI may be appropriate alternatives in certain circumstances.
- Patients with diagnosed or strongly suspected aortic dissection require expeditious surgical evaluation, aggressive analgesia and anxiolysis, and treatment with rapid-acting, titratable agents to first lower heart rate, and then blood pressure, to specific targets.

INTRODUCTION

Aortic dissection (AD) is among the most immediately lethal diseases in medicine, with a mortality of 1% per hour,¹ and has effective temporizing medical therapies and a surgical cure. AD is, therefore, among the disorders of greatest interest to emergency physicians, yet is not diagnosed on its initial presentation in up to half of cases.²⁻⁴ In fact, 1 expert asserts that “difficulty in diagnosis, delayed diagnosis or failure to diagnose are so common as to approach the norm for this disease, even in the best hands...”⁵ This article explains why AD poses a diagnostic dilemma, proposes a strategy for its rational evaluation, and describes the principles of treatment.

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PATHOPHYSIOLOGY

AD occurs when the innermost layer of the aortic vessel wall is torn, creating a false lumen that transmits a longitudinal column of blood. It is sometimes referred to as a dissecting aortic aneurysm; however, this term is discouraged because it is both inaccurate and conflates AD with aortic aneurysm, a distinct clinical entity. AD is thought to result from the hydrostatic pressure accumulated as blood is pumped through the aorta, as well as movement of the aorta itself, with every cardiac cycle. Histologically, AD is associated with characteristic changes in the vessel wall known as medial degeneration. The former term, cystic medial necrosis, has fallen out of favor because the observed lesion demonstrates neither cysts nor necrosis.

Conditions that increase the pressure exerted by blood on the vessel wall predispose patients to AD. These include hypertension, pregnancy, stimulant use (eg, cocaine), weight-lifting, and pheochromocytoma. AD is more likely in conditions that weaken the vessel and accelerate medial degeneration, such as large-vessel vasculitides and congenital connective tissue disorders, including Marfan, Loeys-Dietz, Ehlers-Danlos, and Turner syndromes. Finally, AD may be caused by lesions of the aortic valve itself, such as bicuspid aortic valve, aortic valve instrumentation or aortic surgery, and syphilitic aortitis.

The Stanford classification designates type A dissections as lesions involving the ascending aorta, whereas type B dissections are confined to the descending aorta. Type A dissections are more common and much more dangerous, which drives differences in the therapeutic approach. Variants of AD include aortic intramural hemorrhage, which is a hematoma completely contained within the vessel wall, and penetrating aortic ulcer, which is a disruption in the vessel wall that usually leads not to dissection but to aneurysm. These lesions are both treated similarly to AD.

AD causes morbidity and mortality by several mechanisms. Type A dissections can progress proximally to cause pericardial effusion with tamponade, as well as acute aortic valve insufficiency. Both types of dissections can breach the outer adventitial layer of the vessel, leading to free rupture and exsanguination into the chest or abdomen. Most sequelae of AD, however, result from the false lumen extending across ostia of branch arteries, leading to acute ischemia of potentially any organ in the body.

CLINICAL FEATURES AND EPIDEMIOLOGY

AD is an uncommon disease, with prevalence estimates ranging from 3.5 to 6.0 per 100,000 patient-years in the general population.⁶ Untreated, AD carries a devastating mortality of 40% on presentation and an additional 1% rate of death per hour, to a 1-year mortality of 90%.¹ In a center where postmortem CT is routinely performed on patients with out-of-hospital cardiac arrest of uncertain cause, AD was determined to be the cause in 7% of cases.⁷

Approximately 1 in 10,000 emergency department (ED) patients will have AD, a number so small that emergency providers may only see several cases in their career. Only one-quarter of patients with AD present with a combination of classic features (pain of sudden onset or ripping or tearing quality, blood pressure differential, and widened mediastinum on chest xray [CXR]); 1 in 25 patients diagnosed with AD has none of the classic features.¹ Furthermore, AD can cause myriad symptoms localizing to any organ system or body part, and each of these symptoms can be explained by more common conditions, often by more common dangerous conditions that quite reasonably establish the focus of care but ultimately turn out to be distractors.⁸

Emergency clinicians are thus confronted with innumerable patients whose symptoms could be caused by AD but almost certainly are not. AD could, therefore, be said to represent not just a needle in a haystack but a needle in a haystack that is disguised as a blade of hay. Consequently, physicians evaluating patients whose symptoms may be caused by AD must understand the clinically relevant risk factors and clinical manifestations of this condition and develop a risk stratification strategy that identifies as many patients with the disease as possible without overusing advanced imaging studies that subject patients to important harms.

Despite the rarity of the disease, good data are available on clinical features of AD, owing to the International Registry of Acute Aortic Dissection (IRAD) and a variety of other longitudinal studies.⁹ Pain is more likely to be abrupt and most severe at onset than to be tearing or ripping. Pain location is a reflection of the site of the lesion and includes chest pain radiating to the neck, jaw, or classically, the back; thoracic or lumbar back pain; and abdominal pain. Although chest pain is the most common presenting symptom, 1 study found that of ED subjects diagnosed with AD, more than 40% did not have chest pain on presentation.¹⁰ Another study found 1 subject diagnosed with AD for every 980 subjects presenting with atraumatic chest pain.¹¹ Seventeen percent of subjects in 1 series had painless AD. These subjects presented predominantly with transient or persistent disturbance of consciousness or focal neurologic deficits.¹²

Constitutional symptoms are often marked and include nausea, diaphoresis, and (classically) extreme apprehension with a (justified) sense of impending doom. Patients with AD present with focal neurologic symptoms in 17% and syncope in 9% of cases. Though scenarios classically associated with AD, such as migratory pain, chest pain with neurologic deficits, and chest pain of sudden onset or with pulse deficit, occur in only a minority of cases, their presence strongly suggests the disease. It is commonly believed that patients with AD must be very ill or distressed with abnormal vitals, but ambulatory mode of arrival is an important risk factor for missing the disease at initial presentation.¹³

Many patients with AD will present with acute on chronic hypertension and AD is a cardinal hypertensive emergency. Hypotension is ominous in the setting of AD because it often indicates either proximal extension with cardiac tamponade or free or contained rupture. Pseudohypotension, peripheral hypotension with central normotension, may be caused by dissection across the subclavian arteries. Subclavian or iliac artery embarrassment may also lead to a pulse deficit or blood pressure differential across limbs; this classic finding is present in only 20% to 30% of cases; its absence should not be reassuring.¹⁴ The murmur of aortic regurgitation, or signs of cardiac tamponade, may be present. If the dissection involves the left or (more commonly) right coronary artery, acute myocardial infarction (AMI) and its attendant signs and symptoms can result. A variety of neurologic deficits, including weakness or even coma, may be caused by AD, depending on the cerebral or spinal branch arteries affected. Distal AD can cause ischemia to either kidney, lower extremity ischemia, mesenteric ischemia, and resulting abdominal pain, back pain, or diarrhea.

DIAGNOSIS

Routine laboratory testing is not helpful for ruling in or ruling out AD. Troponin positivity is much more likely to represent primary cardiac pathology condition but is common in AD.¹⁵ Therefore, when clinical features suggest dissection, a positive troponin should not dissuade the practitioner from ruling out the diagnosis of AD.

The use of serum quantitative D-dimer testing has been proposed as a strategy to rule out AD because blood in the false lumen activates the clotting cascade, generating fibrin degradation products detected by modern D-dimer assays with high sensitivity.^{16–20} Unfortunately, further work has demonstrated an unacceptably high false-negative rate, and the ACEP clinical policy recommends that clinicians “not rely on D-dimer alone to exclude the diagnosis of aortic dissection.”²¹ In 1 study, D-dimer was falsely negative in 9 of 113 confirmed AD cases,²² perhaps due to an AD variant in which the thrombosed lumen does not communicate with circulating blood, isolating the clot from detection by serum testing.²³ Furthermore, although a decision analysis of D-dimer and computed tomography (CT) angiography testing thresholds has been performed,²⁴ there is no evidence that D-dimer testing can be incorporated into a larger risk stratification strategy that would allow clinicians to sensitively exclude AD without greatly expanding the number of patients who receive advanced imaging studies. Given the experience with D-dimer testing to rule out pulmonary embolism, which has increased the number of advanced imaging studies ordered without increasing the number of pulmonary embolism diagnoses,²⁵ a comprehensive approach that accounts for false negatives and false positives should be validated before D-dimer testing is used routinely in the diagnosis of AD.

Plain CXR is indicated in patients with chest pain of uncertain cause and whenever AD is considered. The CXR is most useful when it provides an alternative explanation for the patient’s symptoms. A variety of CXR abnormalities are associated with AD (**Box 1**), the most important being mediastinal widening, which is present in more than half of cases but is nonspecific.²⁶ The absence of suggestive CXR findings makes AD less likely. However, 10% to 20% of patients with AD have a normal CXR; therefore, a negative study cannot exclude the disease and should not play a decisive role in the decision to pursue advanced imaging.²⁷

The electrocardiogram in AD is used to evaluate the differential diagnosis and usually demonstrates nonspecific findings but may also indicate acute myocardial ischemia.²⁸ This presents a clinical challenge because not only do the symptoms of AD overlap with the symptoms of myocardial ischemia, AD can *cause* myocardial ischemia when the dissection flap involves the ostium of the left or right coronary artery. When myocardial infarction complicates AD, treatment is directed at AD. Furthermore, usual therapies for AMI may directly worsen outcomes of patients with AD. The clinician caring for a patient whose symptoms may be caused by either diagnosis must, therefore, have a strategy for managing their possible convergence (see later discussion).

Transthoracic echocardiography (TTE) poorly visualizes much of the aorta and is limited by patient, operator, and machine characteristics. AD cannot be excluded

Box 1

Signs of aortic dissection on chest radiography

Mediastinal widening

Disruption of normally distinct contour of aortic knob

Calcium sign: separation of intimal calcification from the vessel wall greater than 5 mm

Double-density appearance within aorta

Tracheal deviation to the right

Deviation of nasogastric tube to the right

by TTE.²¹ However, point-of-care ultrasound by emergency physicians is recommended for all patients with suspected AD and should be considered for all patients with chest or abdominal pain of uncertain cause. In addition to evaluating alternative diagnoses, an intimal flap at either the aortic root or descending aorta may be distinguished by TTE, especially when augmented by suprasternal notch views, and is diagnostic.²⁹ TTE also reliably identifies complications of AD such as pericardial effusion and aortic regurgitation.^{30–32}

Transesophageal echocardiography (TEE) is very accurate in both ruling in and ruling out AD and can be performed in a critical care area as resuscitative efforts are ongoing, which is a distinct advantage compared with CT and MRI, the 2 other definitive imaging modalities. However, TEE is uncommonly performed by emergency clinicians and not widely available on a consultative basis in many EDs. Invasive echocardiography may play a more prominent role in the emergency evaluation of AD as the technique sees broader application by emergency providers.³³

Intravenous (IV) contrast-enhanced CT reliably confirms and excludes AD and may elucidate alternative diagnoses, including pulmonary embolism and obstructive coronary artery disease. Contemporary CT scanning is rapid and widely available. Therefore, CT is the most common definitive imaging study used in patients with suspected AD. Beyond the concerns raised by moving a potentially critically ill patient to the radiology suite, drawbacks of CT include the risks of IV contrast and ionizing radiation, as well as several diagnostic pitfalls (**Box 2**). Though CT angiography is an excellent test for AD, these harms are underappreciated by both physicians and patients.^{34,35}

MRI also accurately rules in and rules out AD,³⁶ and is free of contrast and radiation risk. However, limited availability and relatively long image acquisition times relegate MRI to a secondary imaging modality in most scenarios. MRI has a role in managing stable patients with an equivocal CT or TEE, or patients with known severe IV contrast allergy.

THE DECISION TO IMAGE

For emergency clinicians and other providers who manage patients with undifferentiated symptoms, the central challenge in the evaluation of AD is determining which patients require advanced imaging. Many reviews recommend that AD be considered in any patient presenting with chest pain, abdominal pain, back pain, or malperfusion of any organ, but this encompasses a set of patients so large as to be almost meaningless.

In 2010, the American College of Cardiology (ACC) Foundation and American Heart Association (AHA) published a multidisciplinary guideline on the diagnosis and management of patients with thoracic aortic disease, including AD.³⁷ This document

Box 2

Harms of computed tomography

Radiation or oncogenesis

Contrast harms (kidney injury, allergy, extravasation, volume overload)

False negatives, false positives

Incidental findings (may lead to further testing and therapies)

Overdiagnosis (true positives that would not have harmed patient)

Resource utilization (time, monetary cost, opportunity cost)

presents an evaluation pathway that guides clinicians in deciding which patients require advanced imaging to exclude the disease. The pathway hinges on AD risk markers from past medical history, history of present illness, and physical examination (**Box 3**). Patients with risk markers from more than 1 risk category are classified as high risk and assumed to have AD until proven otherwise. Patients with risk markers from 1 category are intermediate risk and should receive expedited aortic imaging. Finally, patients with no risk markers only require aortic imaging if no alternative diagnosis is identified and the patient has unexplained hypotension or widened mediastinum on CXR.

The ACC/AHA evaluation pathway was found by its authors to have a sensitivity of 95.7%: 1 out of 23 patients diagnosed with AD had zero risk markers.² An external validation confirmed that the evaluation pathway accurately risk stratified patients' likelihood of having AD (more risk markers are associated with higher risk). However, in this cohort, 1 out of 11 subjects with AD had zero risk markers, and subjects with 5 risk markers were still as likely to not have AD as to have the disease.³⁸ The ACC/AHA pathway can, therefore, be used to inform the decision to image, but the absence of risk markers does not exclude the disease and the presence of risk markers does not mandate aortic imaging. Patient-specific factors must ultimately guide management. These factors include the results of ancillary testing and likelihood of an alternative diagnosis, how likely that patient is to be harmed by advanced imaging (eg, radiation harms are inversely proportional to age), candidacy for surgery if AD is diagnosed, and the patient's preferences.

Though many patients with AD do not have classic symptoms or signs, given the low prevalence of the disease, routinely excluding AD with CT angiography in

Box 3

American College of Cardiology and American Heart Association aortic dissection high-risk markers

High-risk conditions

- Marfan syndrome
- Family history aortic disease
- Known aortic valve disease
- Recent aortic manipulation
- Known thoracic aortic aneurysm

High-risk pain features

Chest, back, or abdominal pain, described as any of the following:

- Abrupt onset
- Severe intensity
- Ripping or tearing

High-risk examination features

- Evidence of perfusion deficit
 - Pulse deficit
 - Systolic blood pressure differential
 - Focal neurologic deficit (in conjunction with pain)
- Murmur of aortic insufficiency (new and with pain)
- Hypotension or shock state

patients without typical features would cause more harm than good. Efforts are underway to develop an accurate diagnostic test with a favorable benefit-to-harm profile that may allow practitioners to safely exclude AD in patients who present with the broad range of symptoms that could be due to dissection but almost always are not.³⁹ Until these efforts are successful, a sound approach to AD uses a knowledge of the risk factors for and clinical findings suggestive of the disease to indicate advanced imaging, understanding that with rational application of current diagnostic paradigms and technology, missing the diagnosis on initial presentation will remain the standard of care.

AORTIC DISSECTION AND ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

For patients who present with symptoms consistent with both AD and myocardial ischemia, if the electrocardiogram demonstrates evidence of ST-segment elevation myocardial infarction (STEMI), management should be directed at AMI unless other evidence of AD is present. This recommendation is based on estimates of primary AMI occurring more than 1000 times more frequently than AMI resulting from AD,⁴⁰ and the benefit of timely reperfusion in STEMI. When STEMI is present on electrocardiogram and clinical features raise concern for AD, patients who can receive immediate on-site percutaneous intervention (PCI) may be transferred to cardiac catheterization with the plan to perform aortic angiography before coronary artery angiography.^{41,42} If PCI is not immediately available, CT aortic angiography may be performed to exclude dissection as preparations are made for usual STEMI reperfusion care (eg, thrombolysis or transfer) (Fig. 1). Given the benefit-harm profile associated with usual antiplatelet or anticoagulant agents, in STEMI patients being evaluated for AD, it is reasonable to administer aspirin and hold other treatments until the diagnosis is clarified.

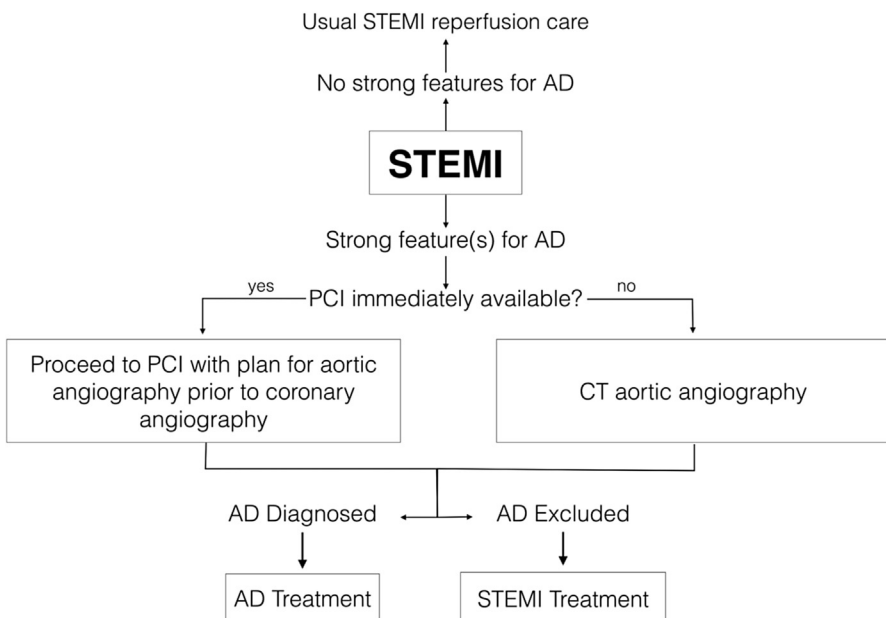


Fig. 1. Relationship between AD and STEMI.

TREATMENT

The primary concern for emergency physicians managing confirmed or highly suspected AD is to arrange for immediate surgical consultation. Though Stanford B dissections may ultimately be managed nonsurgically, all patients with AD should receive prompt surgical evaluation regardless of anatomic location³⁷ (Fig. 2).

The goal of medical therapy in the normotensive or hypertensive patient with AD is to reduce the frequency and magnitude of force bloodflow exerts on the aortic wall. Symptom control is the first priority and is easily overlooked. Patients with AD may have severe pain and anxiety, both of which merit attending to in their own right, but also produce a catecholamine response that directly undermines treatment objectives. Fortunately, unlike the underlying lesion, pain and anxiety are easily managed, and IV opioids (or similarly effective agents but not ketamine, which is catecholaminergic) should be immediately and aggressively titrated to relief of pain as soon as dissection is diagnosed or strongly suspected.

The cornerstone of medical management is beta blockade, titrated to a heart rate of 60 beats per minute. Widely available agents well suited to this purpose include metoprolol and esmolol, with esmolol offering the benefit of minute-to-minute titration. This is particularly advantageous in AD patients who may experience dramatic swings in blood pressure as the lesion evolves and, for example, causes pericardial tamponade or acute aortic insufficiency. Labetalol is widely recommended and is an acceptable alternative; however, labetalol tends to lower blood pressure more reliably than heart

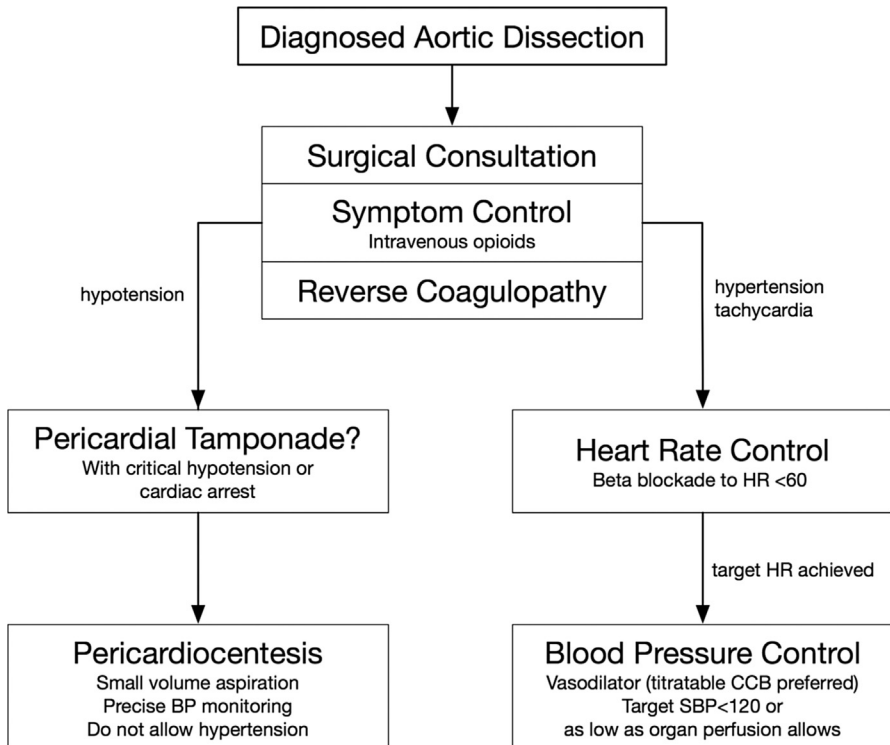


Fig. 2. Management of AD. BP, blood pressure; CCB, calcium channel blocker; HR, heart rate; SBP, systolic blood pressure.

rate.⁴³ Patients with a strong contraindication to beta blockade should receive IV diltiazem or verapamil for rate control.

When beta blockade has achieved its goal heart rate, blood pressure is the next therapeutic target. If, once heart rate is at or below target, systolic blood pressure is greater than 120 mm Hg, an additional agent should be added to lower blood pressure with a goal of less than 120 mm Hg, ideally titrated to as low a blood pressure as end organs (eg, mentation, skin perfusion) allow. Blood pressure should be measured in both arms and treatments directed at the highest reading. Nicardipine (or its more titratable cousin clevidipine), a parenteral dihydropyridine calcium channel blocker, has emerged as the first-line vasodilator infusion in many centers and is recommended in this context. Nitroprusside is effective and classically used for AD but is more difficult to manage and is associated with adverse effects such as cerebral blood vessel vasodilation⁴⁴ and cyanide or thiocyanate toxicity.⁴⁵ Fenoldopam, a peripheral dopamine agonist, and enalaprilat, an IV angiotensin-converting enzyme inhibitor, are variously recommended as vasodilator therapies and are both acceptable choices but less easily titratable than the alternatives.⁴⁴ Phentolamine, hydralazine, and nifedipine should be avoided in AD if possible. Vasodilator agents should not be administered before control of the heart rate is established with beta receptor or calcium channel blockade because this may result in reflex tachycardia and an increase in aortic wall stress (**Box 4**).

Medical therapies for patients with AD who are hypotensive are of minimal utility and limited to IV crystalloid and vasopressor support, pending surgical management.

Box 4

Medical therapies in aortic dissection (analgesia, heart rate control, blood pressure control)

Preferred therapies

Fentanyl^a: 1 mcg/kg bolus, then 1 mcg/kg/h

Esmolol^b: 500 mcg/kg bolus, then 50 mcg/kg/min (repeat bolus if titrating up infusion)

Nicardipine^c: 5 mg/h, titrated every 5 to 10 minutes, maximum 15 mg/h

Clevidipine^c: 2 mg/h, titrated every 1 to 2 minutes

Alternative therapies

Morphine^a: 0.1 mg/kg bolus, then 0.1 mg/kg/h

Labetolol^b: 0.4 mg/kg bolus, then 1 mg/min

Metoprolol^b: 0.05 mg/kg every 5 to 10 minutes, maximum 15 mg

Propranolol^b: 1 mg IV every 5 to 10 minutes, maximum 0.15 mg/kg

Diltiazem^b: 0.2 mg/kg bolus, then 0.2 mg/kg/h

Verapamil^b: 0.075 mg/kg bolus, then 0.075 mg/kg/h

Nitroprusside^c: 0.3 mcg/kg/min, maximum 2 mcg/kg/min

Fenoldopam^c: 0.1 mcg/kg/min, titrated every 10 to 15 minutes, max 1.6 mcg/kg/min

Enalaprilat^c: 1.25 mg over 5 minutes every 4 to 6 hours, maximum 5 mg every 6 hours

Nitroglycerine^c: 20 mcg/min, titrated every 3 to 5 minutes

Doses indicated are starting doses, titrate to effect

^a Analgesia

^b Heart rate control

^c Blood pressure control

Pericardial tamponade is a common cause of hypotension in these cases and small-volume pericardiocentesis, with careful attention not to precipitate hypertension, is an appropriate ED therapy in the arrested or critically hypotensive dissection patient with tamponade.^{46,47} Otherwise, all efforts should be focused on expeditious transfer of the patient to the operating theater.

SUMMARY

AD is an uncommon disease that often presents with varied and atypical findings suggestive of more frequently encountered conditions. Therefore, it poses an exceptional diagnostic challenge to emergency providers. Mortality associated with AD is significant at presentation and advances with every hour the lesion is left untreated. Although almost all patients who have symptoms possibly caused by AD will not have AD, key features of the disease, including risk factors, pain characteristics, physical examination findings, and routine ancillary studies, allow clinicians to develop a rational approach to diagnostic testing. When the diagnosis is sufficiently likely to indicate definitive testing, CT angiography is the advanced imaging test of choice in most centers, but TEE and MRI may be appropriate alternatives in certain circumstances. Patients with diagnosed or strongly suspected AD require expeditious surgical evaluation, aggressive analgesia and anxiolysis, and treatment with rapid-acting, titratable agents to first lower heart rate, and then blood pressure, to specific targets.

REFERENCES

1. Klompas M. Does this patient have an acute thoracic aortic dissection? *JAMA* 2002;287(17):2262–72.
2. Rogers AM, Hermann LK, Booher AM, et al, IRAD Investigators. Sensitivity of the aortic dissection detection risk score, a novel guideline-based tool for identification of acute aortic dissection at initial presentation: results from the international registry of acute aortic dissection. *Circulation* 2011;123(20):2213–8.
3. Hansen MS, Nogareda GJ, Hutchison SJ. Frequency of and inappropriate treatment of misdiagnosis of acute aortic dissection. *Am J Cardiol* 2007;99(6):852–6.
4. Sullivan PR, Wolfson AB, Leckey RD, et al. Diagnosis of acute thoracic aortic dissection in the emergency department. *Am J Emerg Med* 2000;18(1):46–50.
5. Elefteriades JA, Barrett PW, Kopf GS. Litigation in nontraumatic aortic diseases—a tempest in the malpractice maelstrom. *Cardiology* 2008;109(4):263–72.
6. Mussa FF, Horton JD, Moridzadeh R, et al. Acute aortic dissection and intramural hematoma: a systematic review. *JAMA* 2016;316(7):754–63.
7. Tanaka Y, Sakata K, Sakurai Y, et al. Prevalence of type A acute aortic dissection in patients with out-of-hospital cardiopulmonary arrest. *Am J Cardiol* 2016;117(11):1826–30.
8. Grock A, Weinstock MB, Jhun P, et al. Aortic dissection! Or is it? Sigh. *Ann Emerg Med* 2016;68(5):640–2.
9. Hagan PG, Nienaber CA, Isselbacher EM, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. *JAMA* 2000;283(7):897–903.
10. Fan KL, Leung LP. Clinical profile of patients of acute aortic dissection presenting to an emergency department without chest pain. *Am J Emerg Med* 2017;35(4):599–601.

11. Alter SM, Eskin B, Allegra JR. Diagnosis of aortic dissection in emergency department patients is rare. *West J Emerg Med* 2015;16(5):629–31.
12. Imamura H, Sekiguchi Y, Iwashita T, et al. Painless acute aortic dissection. - Diagnostic, prognostic and clinical implications. *Circ J* 2011;75(1):59–66.
13. Kurabayashi M, Miwa N, Ueshima D, et al. Factors leading to failure to diagnose acute aortic dissection in the emergency room. *J Cardiol* 2011;58(3):287–93.
14. Pape LA, Awais M, Woznicki EM, et al. Presentation, diagnosis, and outcomes of acute aortic dissection: 17-year trends from the international registry of acute aortic dissection. *J Am Coll Cardiol* 2015;66(4):350–8.
15. Vrsalovic M. Acute aortic dissection associated troponin leak. *Am J Emerg Med* 2017;35(4):655–6.
16. Akutsu K, Sato N, Yamamoto T, et al. A rapid bedside D-dimer assay (cardiac D-dimer) for screening of clinically suspected acute aortic dissection. *Circ J* 2005;69(4):397–403.
17. Eggebrecht H, Naber CK, Bruch C, et al. Value of plasma fibrin D-dimers for detection of acute aortic dissection. *J Am Coll Cardiol* 2004;44(4):804–9.
18. Sodeck G, Domanovits H, Schillinger M, et al. D-Dimer in ruling out acute aortic dissection: a systematic review and prospective cohort study. *Eur Heart J* 2007;28(24):3067–75.
19. Suzuki T, Distanto A, Zizza A, et al. Diagnosis of acute aortic dissection by D-dimer: the international registry of acute aortic dissection substudy on biomarkers (irad-bio) experience. *Circulation* 2009;119(20):2702–7.
20. Weber T, Högler S, Auer J, et al. D-Dimer in acute aortic dissection. *Chest* 2003;123(5):1375–8.
21. Diercks DB, Promes SB, Schuur JD, et al. Clinical policy: critical issues in the evaluation and management of adult patients with suspected acute nontraumatic thoracic aortic dissection. *Ann Emerg Med* 2015;65(1):32–42.e12.
22. Hazui H, Nishimoto M, Hoshiga M, et al. Young adult patients with short dissection length and thrombosed false lumen without ulcer-like projections are liable to have false-negative results of D-dimer testing for acute aortic dissection based on a study of 113 cases. *Circ J* 2006;70(12):1598–601.
23. Sutherland A, Escano J, Coon TP. D-Dimer as the sole screening test for acute aortic dissection: a review of the literature. *Ann Emerg Med* 2008;52(4):339–43.
24. Taylor RA, Iyer NS. A decision analysis to determine a testing threshold for computed tomographic angiography and D-dimer in the evaluation of aortic dissection. *Am J Emerg Med* 2013;31(7):1047–55.
25. Kabrhel C, Matts C, McNamara M, et al. A highly sensitive ELISA D-dimer increases testing but not diagnosis of pulmonary embolism. *Acad Emerg Med* 2006;13(5):519–24.
26. Zhan S, Hong S, Shan-Shan L, et al. Misdiagnosis of aortic dissection: experience of 361 patients. *J Clin Hypertens (Greenwich)* 2012;14(4):256–60.
27. Harris KM, Strauss CE, Eagle KA, et al, International Registry of Acute Aortic Dissection (IRAD) Investigators. Correlates of delayed recognition and treatment of acute type A aortic dissection: the International Registry of Acute Aortic Dissection (IRAD). *Circulation* 2011;124(18):1911–8.
28. Pourafkari L, Tajlil A, Ghaffari S, et al. Electrocardiography changes in acute aortic dissection-association with troponin leak, coronary anatomy, and prognosis. *Am J Emerg Med* 2016;34(8):1431–6.

29. Sparks SE, Kurz M, Franzen D. Early identification of an atypical case of type A dissection by transthoracic echocardiography by the emergency physician. *Am J Emerg Med* 2015;33(7):985.e1-3.
30. Evangelista A, Flachskampf FA, Erbel R, et al. Echocardiography in aortic diseases: EAE recommendations for clinical practice. *Eur J Echocardiogr* 2010; 11(8):645–58.
31. Fojtik JP, Costantino TG, Dean AJ. The diagnosis of aortic dissection by emergency medicine ultrasound. *J Emerg Med* 2007;32(2):191–6.
32. Perkins AM, Liteplo A, Noble VE. Ultrasound diagnosis of type A aortic dissection. *J Emerg Med* 2010;38(4):490–3.
33. Blaivas M. Transesophageal echocardiography during cardiopulmonary arrest in the emergency department. *Resuscitation* 2008;78(2):135–40.
34. Lumbreras B, Vilar J, González-Álvarez I, et al. Evaluation of clinicians' knowledge and practices regarding medical radiological exposure: findings from a mixed-methods investigation (survey and qualitative study). *BMJ Open* 2016; 6(10):e012361.
35. Lee CI, Haims AH, Monico EP, et al. Diagnostic CT scans: assessment of patient, physician, and radiologist awareness of radiation dose and possible risks. *Radiology* 2004;231(2):393–8.
36. Laissy JP, Blanc F, Soyer P, et al. Thoracic aortic dissection: diagnosis with transesophageal echocardiography versus MR imaging. *Radiology* 1995;194(2): 331–6.
37. Hiratzka LF, Bakris GL, Beckman JA, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with thoracic aortic disease: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. *Circulation* 2010;121(13):e266–369.
38. Nazerian P, Giachino F, Vanni S, et al. Diagnostic performance of the aortic dissection detection risk score in patients with suspected acute aortic dissection. *Eur Heart J Acute Cardiovasc Care* 2014;3(4):373–81.
39. Ranasinghe AM, Bonser RS. Biomarkers in acute aortic dissection and other aortic syndromes. *J Am Coll Cardiol* 2010;56(19):1535–41.
40. Hermann L. Current guidelines for diagnosis and management of thoracic aortic disease in the emergency department. *EM Pract Guidel Update* 2010; 2(6):1–12.
41. Lentini S, Perrotta S. Aortic dissection with concomitant acute myocardial infarction: from diagnosis to management. *J Emerg Trauma Shock* 2011;4(2):273–8.
42. Ramanath VS, Eagle KA, Nienaber CA, et al. The role of preoperative coronary angiography in the setting of type A acute aortic dissection: insights from the International Registry of Acute Aortic Dissection. *Am Heart J* 2011;161(4):790–6.e1.
43. Silke B, Nelson GI, Ahuja RC, et al. Comparative haemodynamic dose response effects of propranolol and labetalol in coronary heart disease. *Br Heart J* 1982; 48(4):364–71.
44. Haas AR, Marik PE. Current diagnosis and management of hypertensive emergency. *Semin Dial* 2006;19(6):502–12.
45. Nelson N. Nitroprusside toxicity. *Emerg Med* 2000;32(10):71–5.

46. Cruz I, Stuart B, Caldeira D, et al. Controlled pericardiocentesis in patients with cardiac tamponade complicating aortic dissection: experience of a centre without cardiothoracic surgery. *Eur Heart J Acute Cardiovasc Care* 2015;4(2): 124–8.
47. Adler Y, Charron P, Imazio M, et al, European Society of Cardiology (ESC). 2015 ESC Guidelines for the diagnosis and management of pericardial diseases: the Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC) Endorsed by: the European Association for Cardio-Thoracic Surgery (EACTS). *Eur Heart J* 2015;36(42):2921–64.