CORE COMPETENCY REVIEW

Acute Aortic Dissection

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Acute aortic dissection is an uncommon but lethal cause of acute chest, back, and abdominal pain. Establishing a timely diagnosis is paramount, as mortality from acute aortic dissection rises by the hour. Physical findings are protean and may include acute aortic valve insufficiency, peripheral pulse deficits, a variety of neurologic deficits, or end-organ ischemia. The keys to establishing a timely diagnosis are maintaining a high index of suspicion and quickly obtaining a diagnostic study. CT angiography, magnetic resonance imaging, transesophageal echocardiography, and, to a lesser extent, aortography are all highly accurate imaging modalities. The choice of study should be driven by the clinical stability of the patient, the information required and the resources available at presentation. Proximal dissections are surgical emergencies, but distal dissections are generally treated medically. Endovascular stents are gaining favor for use in the repair of both acute and chronic distal dissections. Long-term outcome data for endovascular stenting are still limited, and it remains unclear when stenting should be favored over surgery or medical therapy. ***Journal of Hospital Medicine* 2006;1: 94–105.** *© 2006 Society of Hospital Medicine.*

KEYWORDS: aortic diseases, chest pain, hypertensive emergency, shock, Marfan syndrome, adrenergic beta-antagonists, vascular surgery, thoracic surgery

Aortic dissection is an uncommon but highly lethal disease with an incidence of approximately 2,000 cases per year in the United States.[1] It is often mistaken for less serious pathology. In one series, aortic dissection was missed in 38% of patients at presentation, with 28% of patients first diagnosed at autopsy.[2] Early recognition and management are crucial. If untreated, the mortality rate for acute aortic dissection increases by approximately 1% per hour over the first 48 hours and may reach 70% at 1 week. As many as 90% of untreated patients who suffer aortic dissection die within 3 months of presentation.[3,4] Generally, cardiothoracic surgeons or cardiologists experienced with managing aortic dissection should direct patient evaluation and treatment. Hospitalists, however, are increasingly assuming responsibility for the initial triage and management of patients with acute chest pain syndromes and therefore must be able to rapidly identify aortic dissection, initiate supportive therapy, and refer patients to appropriate specialty care.

PATHOPHYSIOLOGY

Aortic dissection occurs when layers of the aortic wall separate because of infiltration of high-pressure arterial blood. The proximate causes are elevated shear stress across the aortic lumen in the setting of a concomitant defect in the aortic media. Shear stress is caused by the rapid increase in luminal pressure per unit of time (dP/dt) that results from cardiac systole. As the aorta

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traverses away from the heart, an increasing proportion of the kinetic energy of left ventricular systole is stored in the aortic wall as potential energy, which facilitates anterograde propagation of cardiac output during diastole. This conversion of kinetic to potential energy also attenuates shear stress. As the proximal aorta is subject to the steepest fluctuations in pressure, it is at the highest risk of dissection. Degeneration of the aortic media is part of the normal aging process but is accelerated in persons with a bicuspid aortic valve, Turner's syndrome, inflammatory arteritis, or inherited diseases of collagen formation.

Once the aortic intima is compromised, blood dissects longitudinally through the aortic media and propagates proximally or distally, creating a false lumen that may communicate with the true lumen of the aorta. Blood may flow through the true lumen, the false lumen, or both. Propagation of the dissection causes much of the morbidity associated with aortic dissection by disrupting blood flow across branch vessels or by directly compromising the pericardium or aortic valve. Over time, the dissection may traverse the entire aortic wall, causing aortic rupture and exsanguination.

CLASSIFICATION

Acute aortic dissection is classified as any aortic dissection diagnosed within 2 weeks of the onset of symptoms, which is the period of highest risk of mortality. Patients who survive more than 2 weeks without treatment are considered to have chronic dissection. Aortic dissections are further classified according to their anatomic location. The fundamental distinction is whether the dissection is proximal (involving the aortic root or ascending aorta) or distal (below the left subclavian artery). The Stanford and DeBakey classification systems are the classification systems most commonly used (Figure 1).

Some variants of aortic dissection are not described in either the Stanford or DeBakey systems. Aortic intramural hematomas (IMH) are caused by intramural hemorrhage of the vasa vasorum without an identifiable intimal tear.[5–7] Penetrating atherosclerotic ulcers (PAUs) are focal defects in the aortic wall with surrounding hematoma but no longitudinal dissection across tissue planes, typically resulting from advanced atherosclerotic disease.[8] The pathophysiologic distinctions between IMH, PAU, and classic aortic dissection remain somewhat controversial. Both IMH and PAU may progress to aortic aneurysm formation, frank dissection, or aortic rupture, suggesting that these entities represent a spectrum of diseases with broad overlap (Table 1).[9,10]

EPIDEMIOLOGY

Aortic dissection is a rare disease, with an estimated incidence of approximately 5-30 cases per 1 million people per year.[11–14] Fewer than 0.5% of patients presenting to an emergency department with chest or back pain suffer from aortic dissection.[15] Two thirds of patients are male, with an average age at presentation of approximately 65 years. A history of systemic hypertension, found in up to 72% of patients, is by far the most common risk factor.[2,14,16] Atherosclerosis, a history of prior cardiac surgery, and known aortic aneurysm are other major risk

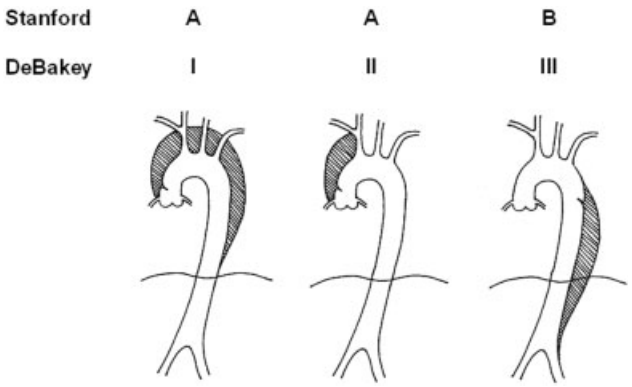

FIGURE 1. Types of dissection per Stanford and DeBakey.

TABLE 1
Composite Schema for Classification of Aortic Disssection, Incorporating Acuity, Anatomy, and Pathophysiology

Acuity	
• **Acute**	<2 weeks after onset
• **Chronic:**	>2 weeks after onset
Anatomic location:	
• **Ascending aorta:**	Stanford Type A, Debakey Type II
• **Ascending and descending aorta:**	Stanford Type A, Debakey Type I
• **Descending aorta:**	Stanford Type B, Debakey Type III
Pathophysiology:	
Class 1:	Classical aortic dissection with initimal flap between true and false lumen
Class 2:	Aortic intramural hematoma without identifiable intimal flap
Class 3:	Intimal tear without hematoma (limited dissection)
Class 4:	Atherosclerotic plaque rupture with aortic penetrating ulcer
Class 5:	Iatrogenic or traumatic aortic dissection (intra-aortic catherterization, high-speed deceleration injury, blunt chest trauma)

TABLE 2
Risk Factors for Aortic Dissection

Hypertension
Atherosclerotic disease
History of cardiac surgery
Aortic aneurysm
Collagen diseases (eg, Marfan syndrome and Ehlers-Danlos)
Bicuspid aortic valve (BAV)
Aortic coarctation
Turner syndrome
Strenuous exercise
Large vessel arteritis: giant cell, Takayasu's, syphilis
Cocaine and methamphetamine ingestion
Third-trimester pregnancy
Blunt chest trauma or high-speed deceleration injury
Iatrogenic injury, typically from intra-aortic catheterization

factors.[14] The epidemiology of aortic dissection is substantially different in young patients (<40 years of age). Hypertension and atherosclerosis become significantly less common, as other risk factors, such as Marfan syndrome, take precedence[17] (Table 2). Other risk factors for aortic dissection include:

- **Collagen diseases** (eg, Marfan syndrome and Ehlers-Danlos): In the International Registry of Acute Aortic Dissection (IRAD), the largest prospective analysis of aortic dissection to date, 50% of the young patients presenting with aortic dissection had Marfan syndrome.[17]
- **Bicuspid aortic valve (BAV):** Individuals with BAV are 5-18 times more likely to suffer aortic dissection than those with a trileaflet valve.[18,19] In one survey, 52% of asymptomatic young men with BAV were found to have aortic root dilatation, a frequent precursor of dissection.[20] Vascular tissue in individuals with BAV has been found to have increased levels of matrix metalloproteinases, which may degrade elastic matrix components and accelerate medial necrosis.[21]
- **Aortic coarctation:** Aortic coarctation is associated with upper extremity hypertension, BAV and aortic dilatation, all of which predispose to aortic dissection.
- **Turner syndrome:** Aortic root dilatation with or without dissection has been incidentally noted in 6%-9% of patients with Turner syndrome.[22,23]
- **Strenuous exercise:** Multiple case reports have associated aortic dissection with high-intensity weightlifting. Many affected individuals were subsequently found to have at least one other risk factor, including hypertension, anabolic steroid abuse, and cocaine abuse.[24–26]
- **Large vessel arteritis:** Large vessel arteritides, specifically giant cell arteritis, Takayasu's disease, and tertiary syphilis have long been associated with aortic dilatation and dissection.
- **Cocaine and methamphetamine ingestion:** Sympathomimetic drugs cause rapid increases in heart rate and blood pressure, markedly increasing aortic intraluminal shear stress. Furthermore, cocaine is thought to be directly toxic to vascular endothelium and may accelerate medial necrosis.[27–30]
- **Third trimester pregnancy,** especially in patients with diseases of collagen[31]; The significance of pregnancy has recently been called into question by data from the IRAD trial. Of 346 enrolled women with aortic dissection, only 2 were pregnant, suggesting that the previously held association of pregnancy with aortic dissection may be an artifact of selective reporting.[1]
- **Blunt chest trauma** or high-speed deceleration injury.
- **Iatrogenic injury,** typically from intra-aortic catheterization.

INITIAL EVALUATION

The differential diagnosis for acute aortic dissection includes acute coronary syndrome, pulmonary embolus, pneumothorax, pneumonia, musculoskeletal pain, acute cholecystitis, esophageal spasm or rupture, acute pancreatitis, and acute pericarditis. Acute aortic dissections are rarely asymptomatic; in fact, the absence of sudden-onset chest pain decreases the likelihood of dissection (negative LR 0.3).[32] In the IRAD trial, approximately 95% of patients with aortic dissection complained of pain in the chest, back, or abdomen, with 90% characterizing their pain as either "severe" or "the worst ever" and 64% describing it as "sharp."[14] Although the presence of "tearing" or "ripping" chest or back pain suggests aortic dissection (positive LR 1.2-10.8), its absence does not reliably exclude this diagnosis.[32] The wide variability in the presentation of aortic dissection increases the challenge of establishing a diagnosis. Clinical findings depend largely on the anatomical location of the dissection and may include pulse deficits, neurologic deficits, hypotension, hypertension, and end-organ ischemia. Women who develop aortic dissection are generally older and present later than men. Their symptoms are less typical and are likely to be confounded by altered mental status.[1] A diagnosis of

TABLE 3
Clinical Findings in Acute Aortic Dissection

Hypotension or shock due to:
a. Hemopericardium and pericardial tamponade
b. Acute aortic insufficiency due to dilatation of the aortic annulus
c. Aortic rupture
d. Lactic acidosis
e. Spinal shock
Acute myocardial ischemia/infarction due to coronary ostial occlusion
Pericardial friction rub due to hemopericardium
Syncope
Pleural effusion or frank hemothorax
Acute renal failure due to dissection across renal arteries
Mesenteric ischemia due to dissection across intra-abdominal arteries
Neurologic deficits:
a. Stroke due to occlusion of arch vessels
b. Limb weakness
c. Spinal cord deficits due to cord ischemia
d. Horner syndrome due to compression of superior sympathetic ganglion.
e. Hoarseness due to compression of left recurrent laryngeal nerve

aortic dissection should be strongly considered for patients presenting with acute chest or back pain and otherwise unexplained aortic insufficiency, focal neurologic deficits, pulse deficits, or end-organ injury (Table 3).

Electrocardiogram: Electrocardiographic abnormalities are commonly seen in aortic dissection and may include ST-segment or T-wave abnormalities or left ventricular hypertrophy.[14] Proximal aortic dissections may compromise coronary artery perfusion, generating electrocardiogram (ECG) findings compatible with acute myocardial infarction, which may lead the clinician to diagnose and treat myocardial infarction while missing the underlying diagnosis.[33] In a recent survey, 9 of 44 patients (21%) presenting with acute aortic dissection were initially diagnosed with acute coronary syndrome and anticoagulated, with 2 deaths.[34] ECGs must therefore be interpreted with extreme caution in aortic dissection.

Chest x-ray: In the emergency department, chest radiography is a mainstay of the evaluation of acute chest pain. Unfortunately, plain-film radiography has limited utility for diagnosing aortic dissection.[35] In the IRAD trial, mediastinal widening (>8 cm) and abnormal aortic contour, the classic radiographic findings in aortic dissection, were present in only 50%-60% of cases. Twelve percent of patients had a completely normal chest x-ray.[14] A pooled analysis of previous studies demonstrated that the sensitivity of widened mediastinum and abnormal aortic contour was 65% and 71%, respectively.[32] Nonspecific radiographic findings, most notably pleural effusion, were common.[36] Thus, if the index of suspicion for aortic dissection is elevated, a confirmatory study must be obtained (Figure 2).

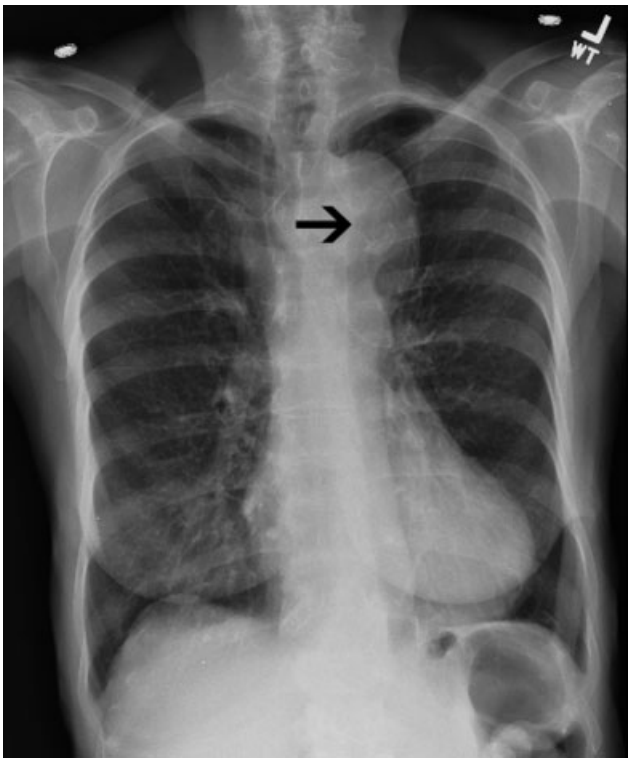

FIGURE 2. Chest radiograph showing classic findings of a proximal aortic dissection. The aortic knob (arrow) has been displaced superiorly and laterally because of mass effect from the false lumen. (courtesy of Dr K. Ed Adib, UW Health and Meriter Hospital, Madison, Wisc).

Clinical Prediction Tool

Three clinical features were demonstrated to be effective in identifying aortic dissection in patients presenting with acute chest or back pain: immediate onset of tearing or ripping chest pain, mediastinal widening or aortic enlargement/displacement observed on chest x-ray, and arm pulse or blood pressure differential exceeding 20 mm Hg. When all 3 findings were absent, dissection was unlikely (7% probability, negative LR 0.07 [CI 0.03-0.17]). If either chest pain or radiographic findings were present, the likelihood was intermediate (31%-39% probability). With any other combination of findings, dissection was likely (83-100% probability).

This prediction tool effectively identified 96% of all patients who presented to an emergency department with acute aortic dissection.[15] However, 4% of patients categorized as low risk were ultimately diagnosed with aortic dissection. Given the exceptionally high mortality resulting from a missed diagnosis, a 4% false-negative rate is unacceptably high. Thus, the absence of any of the aforementioned findings should not dissuade the clinician from obtaining a confirmatory imaging study if the pretest probability for acute aortic dissection is elevated.

CONFIRMATORY IMAGING STUDIES

The ideal confirmatory imaging modality should identify aortic dissection with high sensitivity and specificity. It should also identify the entry and exit points of the dissection and provide information about the extent of compromise of the aortic valve, pericardium, and great vessels. Four imaging modalities sufficiently meet these criteria in order to be considered diagnostically useful.

Aortography: Previously the "gold standard" for diagnosing aortic dissection, aortography is no longer a first-line imaging modality. The sensitivity and specificity of aortography are at best equivalent and probably inferior to less invasive imaging modalities.[37,38] False negatives may occur if both the true and false lumens opacify equally with contrast, or if the false lumen is sufficiently thrombosed to preclude any instillation of contrast. Aortography cannot identify aortic intramural hematomas, is invasive and highly operator dependent, requires nephrotoxic contrast, and generally takes longer to obtain than other modalities.[39]

Aortography uniquely offers excellent visualization of the coronary arteries and great vessels and is preferred when such information is necessary. Percutaneous aortic endovascular stent grafting has been recently employed to repair distal aortic dissections.[40–43] As a result, aortography is gaining new life as a therapeutic modality.

CT angiography: Spiral CT angiography (CTA) is the most commonly used modality for diagnosing aortic dissection.[44] It is emergently available at most hospitals, and images can be obtained in minutes. Sensitivity and specificity may approach 100%, and CTA may be more sensitive than MRA or TEE in evaluating arch vessel involvement.[45–47] Like conventional angiography, CTA requires administration of nephrotoxic contrast. It frequently cannot visualize the entry and exit sites (intimal flaps) of a dissection and provides limited information about the coronary arteries and no information about the competency of the aortic valve.[48,49] Thus, if aortic dissection is identified by CTA, a second study may be needed to provide further diagnostic information and to guide surgical intervention (Figures 3 and 4).

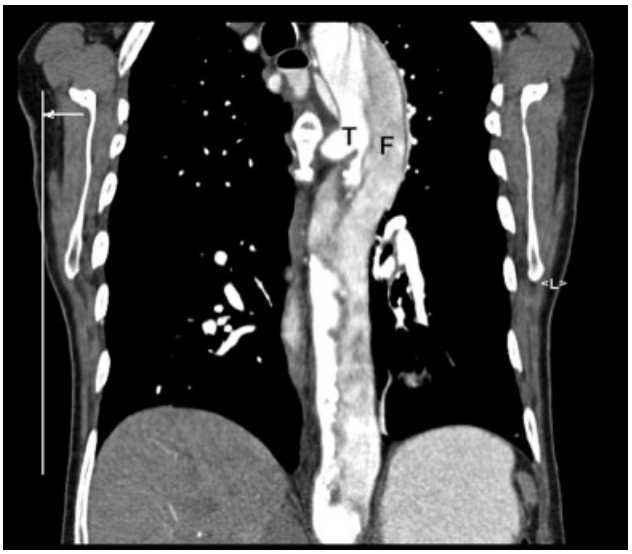

FIGURE 3. Coronal CT angiogram of extensive Stanford type A aortic dissection. The dissection extends the entire length of the thoracic aorta. (T: true lumen, F: false lumen). (courtesy of Dr K. Ed Adib, UW Health and Meriter Hospital, Madison, Wisc).

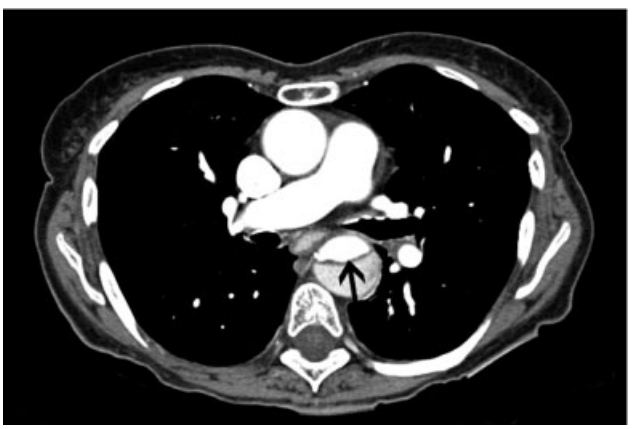

FIGURE 4. Axial CT angiogram of aortic dissection The arrow points to the intimal flap separating the true lumen from the false lumen. (courtesy of Dr Ed Adib, UW Health and Meriter Hospital, Madison, Wisc).

Magnetic Resonance Angiography: Magnetic resonance angiography (MRA) offers excellent noninvasive evaluation of the thoracic aorta. Sensitivity

and specificity are probably superior to spiral CTA, and MRA generally identifies the location of the intimal tear and provides some functional information about the aortic valve.[44,50,51] MRA is not emergently available at many hospitals. Scanning is time intensive, requiring the patient to remain motionless and relatively inaccessible for up to an hour. Furthermore, patient claustrophobia and the presence of implanted devices such as pacemakers or ferromagnetic foreign bodies may preclude MRA.

Transesophageal echocardiography: The sensitivity and specificity of transesophageal echocardiography (TEE) are also excellent—on a par with CTA and MRA. In addition to providing excellent visualization of the thoracic aorta, TEE provides superb images of the pericardium and detailed assessment of aortic valve function.[52] It also is extremely effective at visualizing the aortic intimal flap.[44,49,53] A significant advantage of TEE is its portability, allowing rapid diagnosis at the bedside. For this reason, it is particularly useful for evaluation of patients who are hemodynamically unstable and are suspected to have an aortic dissection. Because of the anatomic relationship of the aorta with the esophagus and the trachea, TEE more effectively identifies proximal than distal dissections.[43] TEE is also somewhat invasive, usually requires patient sedation, and is highly operator dependent, requiring the availability of an experienced and technically skilled operator (Figure 5).

Transthoracic echocardiography: Although it is an excellent tool for the evaluation of many aspects of cardiac anatomy and function, surface echocardiography can reliably visualize only limited portions of the ascending and descending aorta.[54,55] As a consequence, it is neither sensitive nor specific enough to diagnose aortic dissection. Transthoracic echocardiography (TTE) does, however, play a role in rapidly assessing patients at the bedside for aortic valve or pericardial compromise when these complications are suspected.

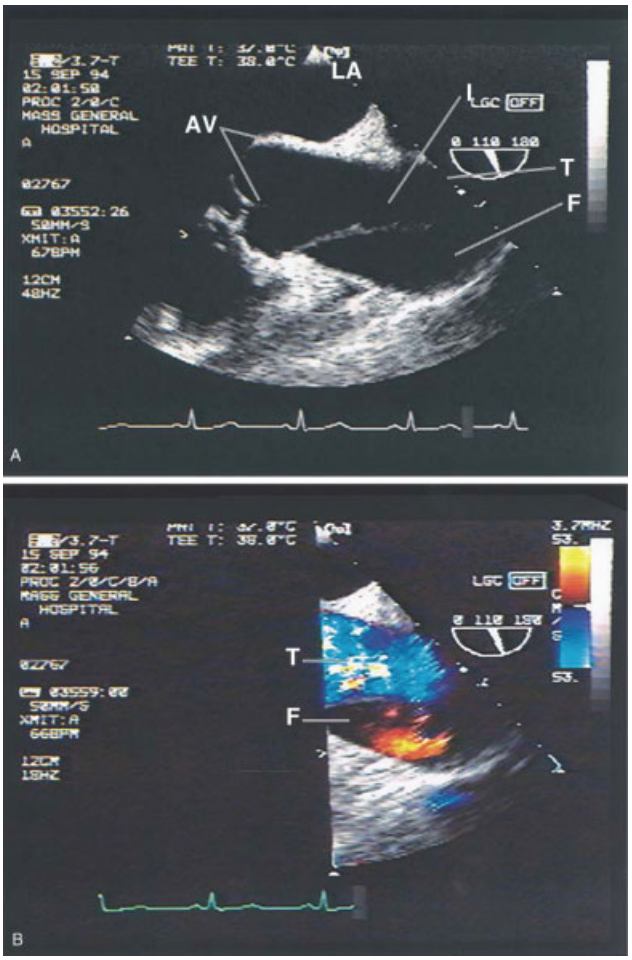

FIGURE 5. Transesophageal echocardiogram of aortic dissection AV. The top image demonstrates the aortic root and aortic valve. The bottom image demonstrates the proximal aorta just distal to the aortic valve (A: aortic valve, T: true lumen, F: false lumen, I: intimal flap). Reprinted with permission from Zipes, D. *Braunwald's Heart Disease.* 7th ed. New York, NY: Elsevier/Saunders; 2005.

Recommendations

CTA, MRA, and TEE are all highly sensitive and specific modalities for diagnosing aortic dissection. Therefore, the condition of the patient, the information needed, and the resources and expertise immediately available should drive the choice of study. MRA is considered the gold standard diagnostic study and is the preferred modality for hemodynamically stable patients with suspected aortic dissection. Because of slow data acquisition and the inaccessibility of patients in the scanner, it is generally unsuited for unstable patients, including those with ongoing pain. Bedside TEE is an excellent choice for patients who are too unstable for MRA but is less effective at visualizing distal dissections. Arch aortography is generally reserved for the confirmation of questionable diagnoses or to image specific branch arteries (Tables 4 and 5).

Most trials comparing CTA, MRA, and TEE were performed in the early 1990s. Computed tomography has evolved significantly over the intervening decade, and some of the diagnostic limitations previously ascribed to CTA, such as the inability to generate 3-D reconstructed images, no longer exist.

TABLE 4
Comparative Sensitivities of Imaging Modalities in Aortic Dissection

	Overall	Proximal	Distal
TEE	88%	90%	80%
CTA	93%	93%	93%
MRA	100%	100%	100%
Aortogram	87%	87%	87%

Adapted from Moore et al.[43]

TABLE 5
Comparative Diagnostic Utility of Imaging Techniques in Aortic Dissection

	TEE	CTA	MRA	Aortography
Sensitivity	++	++	+++	++
Specificity	+++	++	+++	++
Classification	+++	++	++	+
Intimal flap	+++	-	++	+
Aortic regurgitation	+++	–	++	++
Pericardial effusion	+++	++	++	–
Branch vessel involvement	+	++	++	+++
Coronary artery involvement	++	+	+	+++

Adapted from recommendations of the task force on aortic dissection, European Society of Cardiology.[61]

Furthermore, CT angiography is widely available and is gaining increasing acceptance as a first-line imaging modality for patients with noncardiac chest pain.[48] Medical centers that maintain round-the-clock CT capability may have limited or delayed access to TEE, MRA, or aortography. Given the potential for rapid and dramatic patient deterioration, it is imperative that a diagnosis be established quickly when aortic dissection is suspected. Thus, when the choice is obtaining an immediate CTA or a delayed TEE or MRA, CTA is generally the better choice (Figure 6).

MANAGEMENT

Acute Management:

Approximately half of all patients who present with acute aortic dissection are acutely hypertensive.[14] Hypertensive aortic dissection is a hypertensive emergency that mandates immediate decrease in blood pressure to the lowest level that maintains organ perfusion. As a rule, short-acting, parenteral, titratable antihypertensive agents should be used (Table 6). Intravenous beta-adrenergic blockers are the mainstay of acute and chronic therapy. Their negative inotropic and chronotropic effects decrease shear stress across the aortic lumen and decrease the likelihood of dissection propagation and aortic dilatation.[56,57] Parenteral vasodilators (eg, nitroprusside and nitroglycerin) should be initiated if beta-blockers prove insufficient for lowering blood pressure. They should never be used alone, as they may cause reflex tachycardia and consequently may increase intraluminal shear stress. The use of opiates for analgesia and benzodiazepines for anxiolysis further decreases blood pressure by controlling the severe pain and anxiety often associated with acute dissection.

Hypotension or shock, which develop in 15%-30% of patients with acute aortic dissection, are ominous findings that frequently portends impending hemodynamic collapse.[14,58] Patients who develop hypotension are at a fivefold increased risk of death (55.0% vs. 10.3%) and are at markedly increased risk of developing neurologic deficits, as well as myocardial, mesenteric, and limb ischemia. Hypotension may result from pump failure (due to acute aortic insufficiency, pericardial tamponade, or myocardial ischemia), aortic rupture, systemic lactic acidosis, or spinal shock. Bedside transthoracic echocardiography may be particularly useful for the evaluation of hypotensive patients, as it can be used to quickly and noninvasively determine the integrity of the aortic valve and pericardium. Although hypotension may transiently respond to volume resuscitation, all hypotensive patients with aortic dissection, regardless of type, should be immediately referred for emergent surgical evaluation. Pericardiocentesis in the setting of pericardial tamponade remains controversial; a small study suggested that decompression of the pericardial sac may hasten hemodynamic collapse by accelerating blood loss.[59]

Facilities that do not maintain urgent cardiopulmonary bypass capability should emergently transport patients with aortic dissection to a facility that provides a higher level of care. Transfer should not be delayed to confirm a questionable diagnosis. Proximal aortic dissection frequently compromises the pericardium, aortic valve, and arch vessels, and therefore emergent surgical repair is indicated. When treated medically, proximal dissection carries a dismal 60% in-hospital mortality rate.[14,60] In contrast, distal aortic dissection is generally treated medically, with surgical intervention generally reserved for patients with an expanding aortic aneurysm, elevated risk of aortic rupture, refractory hypertension, intractable pain, visceral hypoperfu-

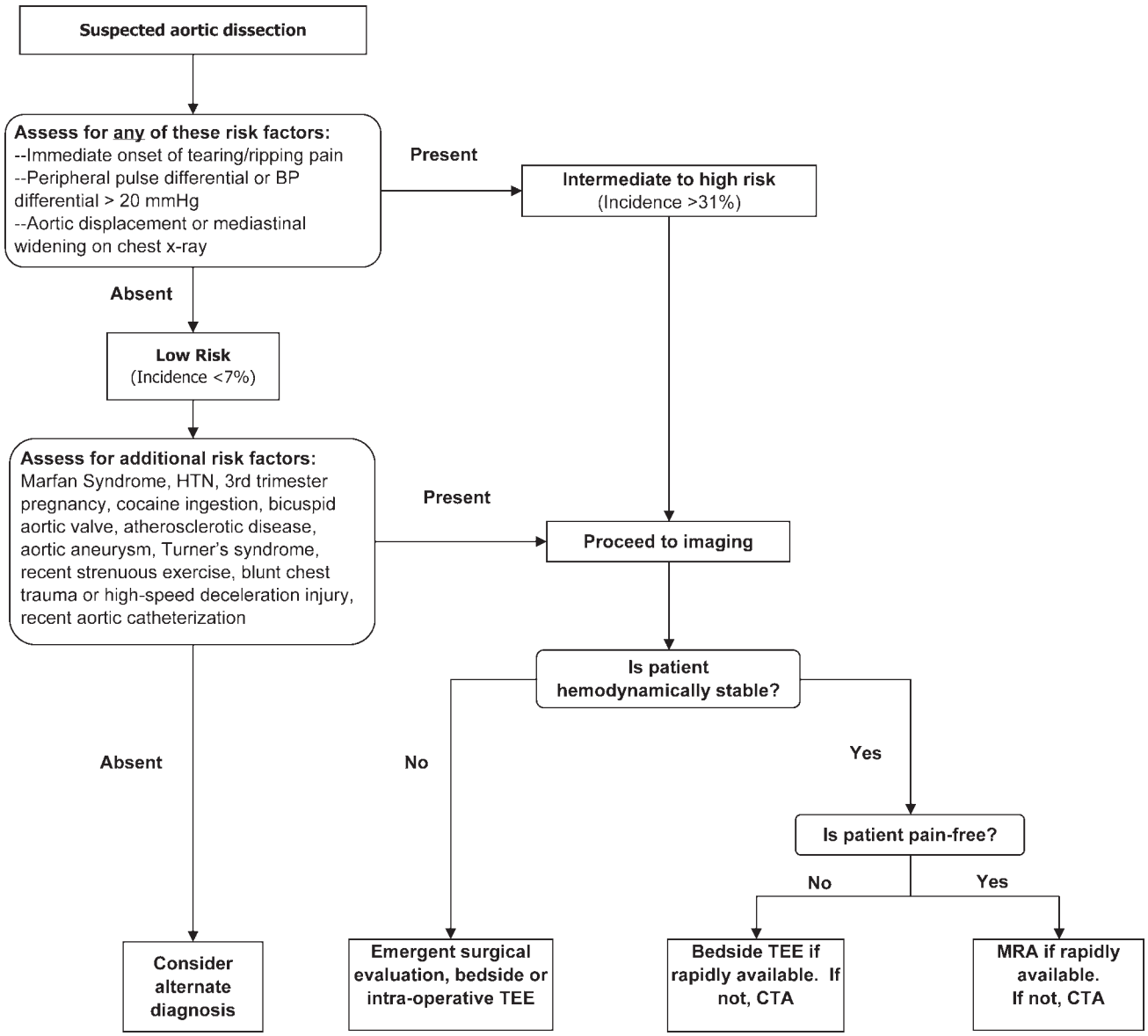

FIGURE 6. Decision tree for evaluating suspected aortic dissection. The flowchart incorporates the clinical decision tool per von Kodolitsch as well as known risk factors for aortic dissection.

sion, and limb ischemia or paresis.[11,61,62] Individual branch vessel occlusion may be effectively ameliorated with conventional arterial stenting or balloon fenestration.

Endovascular stent grafting has been used successfully in lieu of surgery for patients with acute or chronic distal (type B) aortic dissections.[39,40–42,63] The stent graft is deployed across the proximal intimal tear, obliterating the false lumen and facilitating aortic healing. Early studies suggested that endovascular stent grafting may be safer and more efficacious than conventional surgical repair of distal dissection.[41] A recent meta-analysis of published trials of endovascular aortic stenting found procedural success rates exceeding 95% and a major complication rate of 11%. Thirty-day mortality was approximately 5%, with 6-, 12- and 24-month mortality rates plateauing at 10%. Centers with high patient volume had fewer complications and much lower acute mortality rates.[14,64] These medium-term outcomes compare favorably with conventional therapy. Endovascular stenting has not been

TABLE 6
Recommended Drugs for Treating Hypertensive Acute Aortic Dissection

Name	Mechanism	Dose	Cautions/contraindications
Esmolol	Cardioselective beta-1 blocker	Load: 500 μg/kg IV Drip: 50 $\mu g\ kg^{-1}\ min^{-1}$ IV. Increase by increments of 50 μg/min	• Asthma or bronchospasm • Bradycardia • 2nd- or 3rd-degree AV block • Cocaine or methamphetamine abuse
Labetalol	Nonselective beta 1,2 blocker Selective alpha-1 blocker	Load: 20 mg IV Drip: 2 mg/min IV	• Asthma or bronchospasm • Bradycardia • 2nd or 3rd degree AV block • Cocaine or methamphetamine abuse
Enalaprilat	ACE inhibitor	0.625-1.25 mg IV q 6 hours. Max dose: 5 mg q 6 hours.	• Angioedema • Pregnancy • Renal artery stenosis • Severe renal insufficiency
Nitroprusside	Direct arterial vasodilator	Begin at 0.3 $\mu g\ kg^{-1}\ min^{-1}$ IV. Max dose 10 $\mu g\ kg^{-1}\ min^{-1}$	• May cause reflex tachycardia • Cyanide/thiocyanate toxicity—especially in renal or hepatic insufficiency
Nitroglycerin	Vascular smooth muscle relaxation	5-200 μg/min IV	• Decreases preload—contraindicated in tamponade or other preload-dependent states • Concomitant use of sildenafil or similar agents

prospectively compared against conventional therapy in randomized trials, and it therefore remains unclear who should be referred for endovascular stenting instead of conventional therapy.

Long-term Management

Survivors of aortic dissection, especially those with diseases of collagen, have a systemic disease that predisposes them to further aortic and great vessel events. Almost one third of survivors of acute aortic dissection will develop dissection propagation or aortic rupture or will require aortic surgery within 5 years of presentation.[41,60] Young patients who present for aortic dissection should be screened for Marfan syndrome according to the Gent nosology.[65] To reduce shear stress to the aortic lumen, all patients should be treated with beta-blockers for life, with blood pressure targeted to be below 135/80.[60,66] Patients who do not tolerate beta blockade may benefit from treatment with diltiazem or verapamil. Progression to aortic aneurysm is common, and patients should undergo serial imaging of the aorta at 1, 3, 6, and 12 months after discharge and annually thereafter. Dilatation of the proximal aorta to $>$5.0 cm and of the distal aorta to $>$6.0 cm should prompt referral for surgical or possibly endovascular repair.[41,67] Although supporting data are limited, it is generally accepted that patients should moderate their physical activity to avoid extremes of tachycardia and blood pressure elevation. Sports that involve high speed or sudden deceleration, such as ice hockey, downhill skiing, and football, should be strictly avoided. Patients should be warned to seek immediate medical attention if they develop recurrent chest or back pain or focal neurologic deficits.

PROGNOSIS

Despite significant medical and surgical advances, aortic dissection remains exceptionally lethal. Patients with proximal dissections are more likely to die than those with distal dissections. Using data from the IRAD trial, Mehta et al determined that age $\geq$ 70 years (OR, 1.70), abrupt onset of chest pain (OR 2.60), hypotension/shock/tamponade (OR, 2.97), renal failure (OR, 4.77), pulse deficit (OR, 2.03), and abnormal ECG (OR, 1.77) were independent determinants of death.[59] Medical treatment of proximal dissection is generally reserved for patients too ill, unstable, or frail to undergo surgery. In contrast, most patients with distal dissection are managed medically, with surgery generally reserved for those with acute complications. Hence, patients with proximal dissections who are managed medi-

TABLE 7
Mortality in Acute Aortic Dissection[14]

	Proximal (DeBakey I, II; Stanford A)		Distal (DeBakey III; Stanford B)	
	Surgical	Medical	Surgical	Medical
In-hospital mortality	26%	58%	31%	11%
Average	35%		15%	

cally and those with distal dissections who are managed surgically have the worst outcomes. Outcomes for women are worse than those for men, which is probably attributable to several factors. Women dissect at an older age, present later after the onset of symptoms, and are more likely to have confounding symptoms that may delay timely diagnosis[1] (Table 7).

CONCLUSION

Aortic dissection is a rare and acutely life-threatening cause of acute chest and back pain. Delays in diagnosis and misdiagnoses are common, frequently with catastrophic consequences. The key to diagnosis is maintaining a high index of suspicion for dissection, especially in patients who present with acute severe chest, back, or abdominal pain in the setting of unexplained acute pulse deficits, neurologic deficits, or acute end-organ injury. Three clinical findings have been shown to be diagnostically useful: immediate onset of tearing or ripping chest or back pain, mediastinal widening or abnormal aortic contour on chest radiograph, and peripheral pulse deficits or variable pulse pressure (>20 mm Hg). If all 3 findings are absent, acute aortic dissection is unlikely. The presence of any of these findings should prompt further workup. A normal chest radiograph does *not* rule out aortic dissection. Only TEE, CT, and MR angiography are sufficiently specific to rule out dissection. Aortography is rarely used as a first-line diagnostic tool but may be useful as a confirmatory test or to provide additional anatomic information. Patients who present with proximal aortic dissection or with any aortic dissection with concomitant hypotension are at exceptionally high risk of death and should be immediately referred for surgical evaluation. Beta-blockers are the mainstay of acute and chronic therapy of aortic dissection. Survivors of aortic dissection are at a markedly elevated risk for further aortic events and should be followed vigilantly posthospitalization.

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