Chapter 6
Acute Aortic Syndromes in the ER

Carlos Jerjes-Sánchez and Felipe Valdés

6.1 The Scope of the Problem

The high mortality rate and the associated morbidity of acute aortic syndromes (AAS) are directly related to diagnostic delay, since the onset of symptoms [1–4]. Acute aortic syndromes are rarely seen outside an ER scenario. Patients presenting with acute aortic dissections (AAD) tend to manifest life-threatening signs and symptoms, which need prompt diagnostic evaluation. Quick detection and expedite treatment are fundamental to reduce morbidity and mortality [5]. If left untreated, mortality can spike from 20% during the first 24 hours after symptom onset up to 62% by the 7th day [6].

Acute aortic syndromes are considered an infrequent spectrum of diseases, perhaps that is why medical personnel rarely consider them as a first-line diagnosis. AAS comprise a series of potentially fatal conditions which include aortic dissection, penetrating aortic ulcer (APU), and intramural aortic hematoma (AIMH). These AAS are clinically indistinguishable one from another, and their cardinal manifestation is acute onset of severe chest pain, probably because AAS share common pathophysiology which relates to the breakdown and tearing of the intima and media [1, 7].

6.2 Prevalence

The incidence of aortic syndromes is roughly around 2.5 to 4 cases per 100,000 patients per year, the vast majority comprised by AD [1, 2, 5, 7–9]. Of all AAS, AAD represent approximately 80% of all cases, followed by AIMH at 15% and APU with less than 5%. Present-day data derived from the International Registry of Acute Aortic Dissections shows that two-thirds of the affected patients are men, about two-thirds also suffer from high blood pressure, and AAD is more common in patients between their sixth and seventh decade of life. Traditionally if the age of presentation is below 40 years, the single most common predisposing condition is connective tissue diseases [1, 2, 7, 8].

C. Jerjes-Sánchez, *Cardiology in the ER*,
https://doi.org/10.1007/978-3-030-13679-6_6

Generally, the ascending aorta is affected in the majority of patients with an estimated incidence of 60–70% [1, 2, 6, 7]. Proper classification is key to determine treatment. Considering the high mortality of AAS, early treatment is one of the main determinants of survival, particularly for AAD, which if left untreated has an average mortality between 1 and 2% per hour during the first 24 hours upon symptom presentation. Survival declines with time since symptom onset and symptomatology progression can be considered a stronger prognostic factor, even more than the selected treatment approach [1, 9].

6.3 Patient-Related Risk Factors

Table 6.1 lists the most important risk factors for AAD patients presenting to the ER.

6.4 Types and Mechanisms of the Problem

According to the anatomical structures affected by the tear (layers of the aorta), acute aortic syndromes can be classified as seen in Table 6.2.

Table 6.1 Prevalent risk factors reported in patients with aortic dissections [1–3, 8]

Risk factor	Prevalence
Hypertension	65–77%
Age (beyond sixth decade of life)	32%
Genetic diseases (bicuspid aortic valve, Marfan syndrome, Ehlers-Danlos, autoimmune disease)	20%
High-speed deceleration (aortic trauma)	15–20%
Sex (male)	66%
Iatrogenic	0.06%
Vasculitis	NA
Other	NA

NA not available

Table 6.2 Classification and characteristics of the aortic syndromes [1, 2, 6]

Aortic dissection 60–70%	Intramural hematoma 5–30%	Penetrating ulcer 2–7%
The formation of true and false lumens	Hematoma formation in the media	Ulceration of atherosclerotic plaque
Intramural bleeding	No blood flow must supply the hematoma.	Penetrates internal lamina
Break in the medial layer	No flap and no intimal tear	Reaches medial layer
Inflammatory response and bleeding cause aortic dilation and eventually rupture	Rupture of the vasa vasorum	Risk of rupture or progression toward acute aortic dissection

6.5 Clinical Presentation

The most common clinical presentation (>75%) for both type A and B dissections is moderate to severe pain, usually abrupt in onset, starting at the chest or abdomen and described as tearing, stabbing, or penetrating. Sometimes the pain tends to radiate to the back and can migrate toward the lower abdomen and legs [1–3, 5, 7, 8].

Accompanying symptoms are very diverse and usually derive from ischemia to affected organs along the tear's path (Table 6.3) or hypotension and circulatory shock due to sudden and massive blood loss. When the tear starts at the aortic root, patients can exhibit aortic regurgitation and acutely decompensated heart failure [1, 2, 5, 7, 8].

6.6 Physical Examination

Usually patients tend to present with a hypertensive response. Nonetheless circulatory shock can be the initial presentation. Physicians should be aware of any neurological manifestation and perform a quick neurological checkup. Aortic murmurs can be present, especially in the setting of acute aortic regurgitation. Patients with aortic regurgitation can present wide pulse pressure variations along with classic aortic regurgitation signs. Others experience heart failure symptoms such as jugular reflux, lung rales, and edema. If the dissection flap excludes limb arteries along the way, pulse deficit can be noted on the affected extremity [1, 5, 7, 8].

Table 6.3 Signs and symptoms of ER patients with acute aortic syndromes

Signs/symptoms	Affected patients (%)
Chest pain	80–89%
Aortic regurgitation (acute)	40–75%
Hypertension (SBP >150 mmHg)	25–65%
Pain migration	15–50%
Back pain	40%
Abdominal Pain	24–41%
Neurological deficit or paraplegia	7–40%
Circulatory shock	5–33%
Pulse deficit	9–36%
Pericardial tamponade	<20%
Pleural effusion	15–20%
Renal failure	10–20%
Syncope	5–20%
Myocardial ischemia	10–<15%
Congestive heart failure	<10%
Tamponade	5%
Mesenteric ischemia	<5%

SBP systolic blood pressure

6.7 Classification

AAS is a spectrum of diseases closely related to pathological vessel walls. The vessel's walls are constantly withstanding variations in hemodynamic sheer stress which favor fluctuations along the disease's spectrum, meaning that no syndrome remains quiescent. AIMH and APU eventually may evolve to the most lethal of all AAS, the AAD.

AAD can also be classified based on different properties, such as its site of origin, extension, time from symptom onset, and etiology of the intimal tear; different schemes are recommended for these purposes [1–3, 9]. Table 6.4 and Fig. 6.1 show DeBakey and Stanford classifications.

Considering the time elapsed since symptom onset, the American Heart Association (AHA), the American College of Cardiology (ACC), and the European Society of Cardiology (ESC) classify AAD into three main categories, acute, subacute, and chronic dissections. Nonetheless, considering more recent data collected by the International Registry of Acute Aortic Dissections, a different and more contemporary chronological classification (Table 6.5) is proposed, adding a new time-based category, the hyper AAD. The global mortality is better matched according to this new arrangement, and it tends to increase substantially as time passes by. The patients whose symptoms have lasted for less than 24 hours and received proper and expeditious treatment exhibit the lowest mortality rates of all [1–3, 8].

Table 6.4 Classification of aortic dissections

DeBakey Origin of the tear	Svensson Etiology	Stanford Extension of the tear
Category 1 Dissection tear starting at the ascending aorta and disseminates to the descending aorta (aortic arch involvement is possible)	*Class 1* Dissection with true and false lumens (classical dissection)	*Type A* All dissections involving the ascending aorta
Category 2 Tear confined to ascending aorta	*Class 2* Intramural hematoma(or hemorrhage)	
Category 3 Tear starts at the descending aorta and propagates distally	*Class 3* Subtle dissection without hematoma	*Type B* All dissections that do not involve the ascending aorta (regardless of aortic arch involvement)
Category 3a Tear limits to the descending thoracic aorta	*Class 4* Penetrating atherosclerotic ulcer	
Category 3b Tear extends beyond the diaphragm	*Class 5* Iatrogenic or traumatic dissection	

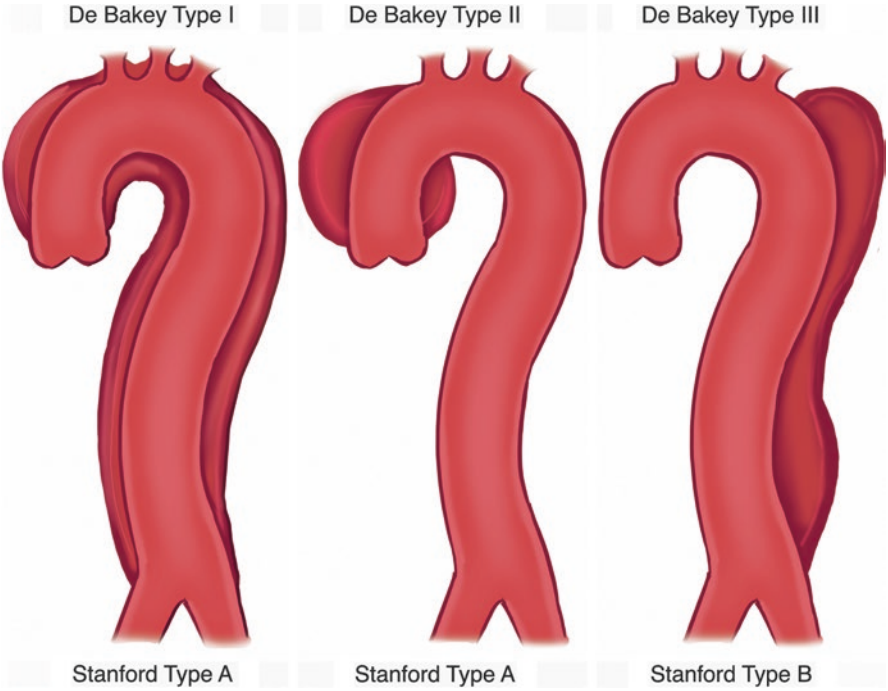

Fig. 6.1 Anatomical classification of aortic dissections. (Courtesy of Celina Ortiz)

Table 6.5 Aortic dissection chronological classification

	Hyperacute	Acute	Subacute	Chronic
AHA/ACC	NA	<2 weeks	2–6 weeks	>6 weeks
ESC	NA	<14 days	15–90 days	>90 days
IRAD[a]	<24 hours	2–7 days	8–30 days	>30 days

Classification is according to time since onset of symptoms
NA not applicable, *ESC* European Society of Cardiology, *AHA* American Heart Association, *ACC* American College of Cardiology, *IRAD* International Registry of Acute Aortic Dissections
[a]New proposed classification

6.8 Differential Diagnosis

The most important differential diagnostics in the ER setting in a patient presenting with sudden-onset chest pain and cardiovascular risk factors are acute coronary syndromes (ACS), pulmonary embolism, hypertensive emergencies, and acute heart failure. Other non-life-threatening conditions are esophageal diseases (spasm, reflux, achalasia) and pleural conditions (pleural effusion, pleuritis); finally we should consider musculoskeletal diseases that can mimic cardiopulmonary pathology.

6.9 Diagnostic Strategy

High-clinical suspicion is key. Patients with risk factors and characteristic symptomatology should undergo urgent contrast angiography CT scan and transthoracic echocardiography. Blood samples should be drawn searching for blood loss, hypoperfusion, and end-organ damage. D-dimer levels below the 500 ng/mL threshold make the diagnosis of aortic syndrome less probable; normal D-dimer does not exclude the possibility of AIMH. Biomarkers such as cardiac troponin I (cTi) and B-type natriuretic peptide (BNP) are of special importance while assessing myocardial stress [1, 5, 7, 8, 10]. High-clinical suspicion for AAD must be considered in all patients with sudden abdominal or chest pain, especially in high-risk populations with or without systemic hypoperfusion syndrome until proven otherwise (Fig. 6.2) [2, 7, 10, 11]. A fast diagnosis, risk stratification, and clinical decision making are mandatory to improve the outcome.

6.10 Multimodal Diagnostic Approach

6.10.1 Electrocardiogram

There is no specific electrocardiographic pattern for the diagnosis of an acute aortic syndrome. Usually nonspecific changes can be observed. Up to 40% of electrocardiograms (ECG) tend to be within normal limits [1, 12]. Around 35% of patients exhibit changes related to left ventricular (LV) hypertrophy, LV strain, or LV overload (Fig. 6.3). LV overload is a regular finding in patients presenting with aortic regurgitation. Less often, myocardial necrosis patterns, Q waves, and bundle branch blocks are seen in up to 5–10% [1, 6, 12]. ST and T wave changes were found in 34–47% of patients; most often ST changes were nonspecific. A smaller portion (around 15–20%) of these patients may exhibit ST depression and T wave inversion compatible with acute ischemia. ST elevation, an uncommon finding, was observed in about 5–8% of all ECGs [8, 9, 12]. This finding is of extreme importance because it should prompt the clinician to rule out acute myocardial infarction as a differential diagnosis and take into consideration that right coronary artery involvement is a possibility, especially in proximal type A dissections. If the coexistence of MI and ADD is confirmed, antiplatelet, anticoagulant, and thrombolytic agents must be avoided because they increase the risk of fatal bleeding boosting in-hospital mortality to numbers more than 70% [9].

6.10.2 Laboratory Tests

Lab testing is usually complimentary since no test offers robust evidence to diagnose an acute aortic syndrome properly. Its usefulness comes in the detection of alternate differential diagnosis. Excluding D-dimer, biomarker and blood analysis

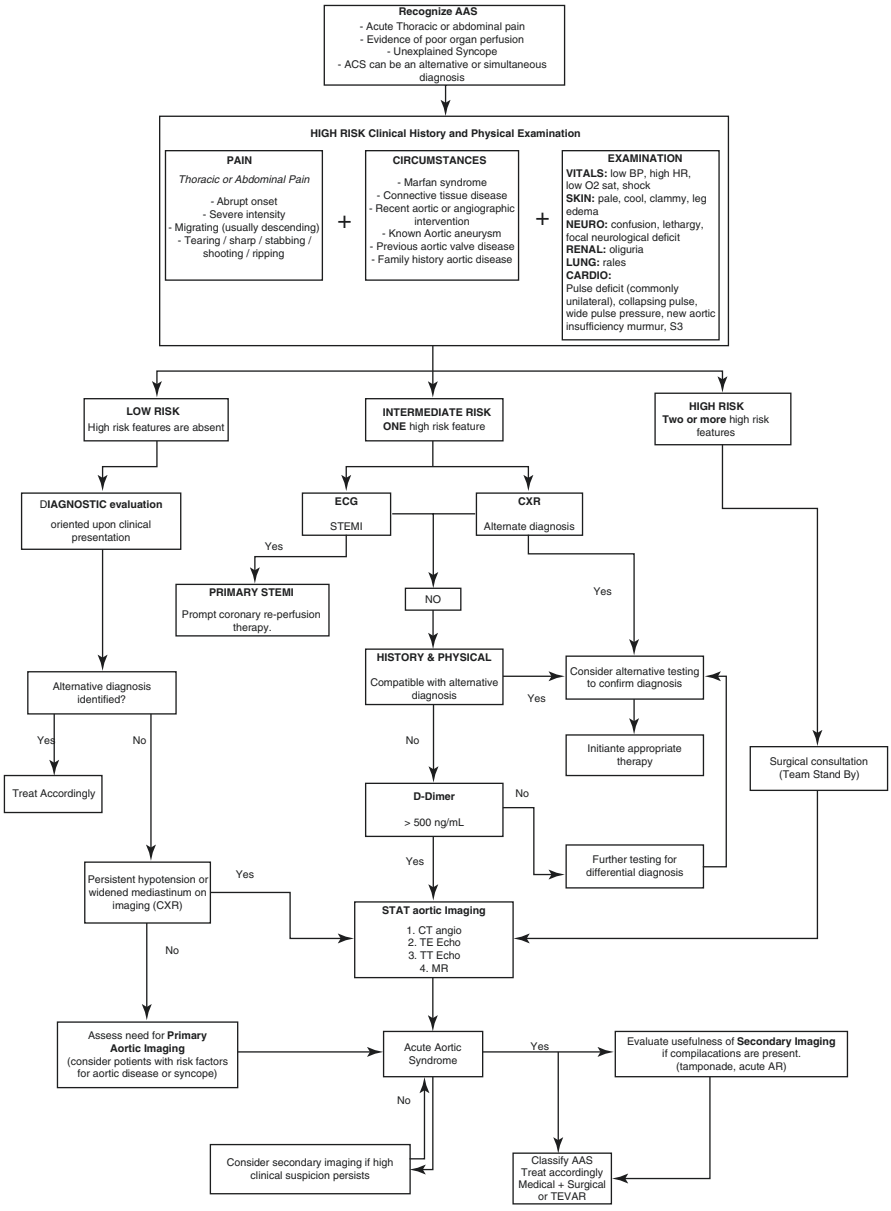

Fig. 6.2 Quick decision ER flowchart. STEMI, ST myocardial infarction. ECG electrocardiogram, CXR chest X-ray, TE echo transesophageal echocardiogram, TT echo transthoracic echocardiogram, MR magnetic resonance, AR aortic regurgitation

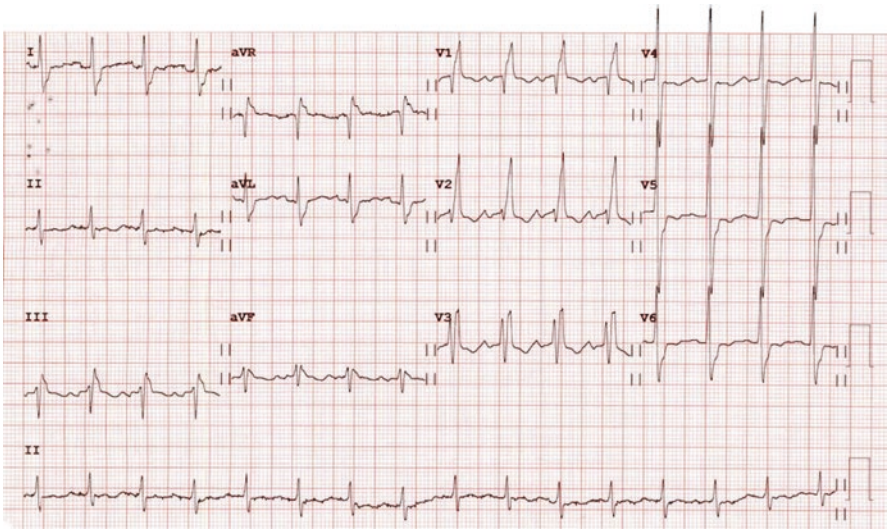

Fig. 6.3 ER ECG of a patient with aortic dissection and acute aortic regurgitation. The ECG shows sinus tachycardia, right bundle branch block and inferior subepicardial ischemia

lack sensitivity and specificity to accurately rule in or rule out AAS. BNP and cTi are adding information about myocardium response to an AAD. In the ER environment, lab results offer clues about blood loss, procoagulant activity, and end-organ damage.

6.10.3 Complete Blood Count

The complete blood count (CBC) is particularly useful for the assessment of dynamic changes in the total red blood cell and platelet count, even though the frequency in which changes are manifested in the CBC has not yet been defined. The detection of thrombocytopenia is an indirect indicator of bleeding which reflects platelet consumption and can be observed in the presence of ADD or large AIMH. Other indicators of hemorrhage are decreased hemoglobin, hematocrit, or a low red blood cell count.

6.10.4 Blood Chemistry

Several other biomarkers can be used as indicators of AAS severity beside CBC for blood loss. Low organ perfusion and ischemia can be inferred in the presence of high serum lactate, creatinine, and transaminase elevations [1].

6.10.4.1 C-Reactive Protein and Procalcitonin

C-reactive protein and white blood cell elevation suggest an inflammatory response. Even so, C-reactive protein and white blood cell tend to be elevated; both are not specific for AAS diagnosis. AAS differentials rarely involve infectious etiologies. Nonetheless, the measurement of procalcitonin and infection-related antibodies or biomarkers can assist clinicians in the differentiation of infection from inflammatory response.

6.10.4.2 Cardiac Troponin I or T

Traditionally myocardial infarction is a differential diagnosis of AAS, but according to the International Registry of Acute Aortic Dissections, up to 7% of acute AAD present with accompanying myocardial infarction. Troponin will always be useful in the context of acute chest pain and differential diagnostic considerations of ACS.

Physicians should invariably acknowledge troponin expression as myocardial ischemia consistent with an AC, particularly in the setting of acute chest pain and/or ST-T wave abnormalities presenting in a patient with cardiovascular risk factors. Current evidence suggests that cardiac troponin elevation can be present in patients with the AD and concomitant myocardial infarction or in patients with aortic regurgitation presenting with pressure/volume overload and hemodynamic stress.

In the ER, cardiac troponins rule out many non-cardiac causes of acute chest pain, but ultimately may not be such a powerful discriminator between AAD and myocardial infarction. High-clinical suspicion is key to quickly identify patients with synchronous AAD and differentiate them from MI. Bear in mind that this binomial condition is of high mortality. Several studies such as an ECG and coronary CT angiogram can aid in the diagnosis, without delaying proper treatment (which is often surgical for type A AAD and endovascular for type B AD). This spike in mortality is often iatrogenic and precipitated by inappropriate administration of anticoagulant and antiplatelet medications before the AAD is adverted. Standard cTi and high-sensitivity cTi expression (up to 25% of patients) have been related to higher in-hospital mortality and longer ICU and hospital stays, as well as elevated creatinine levels and the need for catecholamine infusion therapy [4]. High-sensitivity cTT (found in more than 60% of patients) have shown a similar correlation regarding mortality [13].

Recently, from 6455 consecutive patients with acute chest pain admitted to the ER, 15 (0.23%) of whom had AAD diagnosed and biomarker data collected. AAD was confirmed on transthoracic esophageal echocardiogram and computed tomography. Patients with abnormal cTnI concentrations had a higher rate of mortality. In univariate analysis, elevated.

cTnI was an independent predictor of in-hospital mortality (relative risk 27.46, 95% confidence interval 1.20–629.31). No relationship between mortality and D-dimer, BNP, or the DeBakey classifications was identified. These findings suggest that cTnI may be a promising tool for rapid risk stratification of patients with AAD [14].

6.10.4.3 D-Dimer

D-dimer (DD) elevation is often observed in patients with AAS. A cutoff level of 500 ng/mL has been suggested in several studies [1, 8]. DD is a test that is notably useful within the first 6 hours of symptom onset [6]. DD levels above the 500 ng/mL threshold are known to have a sensitivity ranging amid 96–98% with a low specificity just around 40–60% [5, 15–17]. It is important to take into consideration that a normal DD does not exclude the possibility of an AAS, particularly for those patients with whom there is high-clinical suspicion or for those presenting with AIMH; the displayed values for DD levels tend to be within normal ranges (below 500 ng/mL).

Recently, we observed concentrations above the upper limit of the assay in all patients with AAD except one, who had localized intramural hematoma. As previously reported, a trend toward higher DD levels in patients who died was detected; however, no relationship with mortality was observed. The results confirm previous observations and suggest that testing for DD should become part of the initial screening of patients with the suspicion of AAD. A negative test result makes the presence of the disease unlikely [14].

6.10.4.4 B-Type Natriuretic Peptide

Frequently, an acute heart failure syndrome is a clinical complication of patients with proximal AAD; BNP could offer a secondary phenomenon biomarker to understand this complication better. Recent evidence linking BNP and AAD showed high plasma concentrations in patients with AAD (667 ± 703 pg/mL) or a chronic aneurysm (593 ± 964 pg/mL) compared with a control group [14].

Recently, BNP concentrations identified a subgroup with acute heart failure syndrome and preserved ejection fraction. BNP concentrations established severe ventricular dysfunction and adrenergic, renin-angiotensin-aldosterone systems and vasopressin activation in some patients, but not in others. Although abnormal BNP concentrations could be attributed to heart failure secondary to aortic valve disruption and/or acute ischemia, long-standing hypertension and subclinical heart failure has to be considered in some cases. Diagnosis of heart failure with preserved ejection fraction could be established through normal ejection fraction and elevated plasma BNP concentrations. Although BPN determination does not replace echocardiography in the diagnostic approach to AAD, adding a rapid plasma BNP assay could be useful for detecting early ventricular dysfunction stages when an echocardiogram is not available or the ejection fraction is normal. Although BNP values had no relationship with in-hospital mortality, these findings could be the first clinical evidence linking BNP and ejection fraction in AAD patients [14].

6.11 Imaging Modalities

Aortic imaging is the most reliable approach to the diagnosis of aortic pathology. Imaging techniques are not only used for the detection of aortic pathology but rather can be used for monitoring purposes as well. Imaging can diagnose disease in at-risk patients, or patients with known stable aortic diseases (like aneurysms) can benefit from repeated imaging and anatomical landmark measurement and comparison [3].

6.11.1 Chest X-Ray

The chest X-ray (CXR) is a broadly available imaging study. Its chief advantages are its speed and its cheap cost; nonetheless, it has a low diagnostic performance in the detection of aortic dissections. CXR's poor sensibility around 64% and specificity of 86% and high observer variability represent a diagnostic challenge while searching for aortic pathology [18]. A normal CXR may be observed up to 20–50% of patients, while a widened mediastinum is the most common finding in around 50–60% of patients [1, 6]. Other frequent findings in CXR of patients presenting with AAD are changes in the aortic contour, an enlarged aortic knob (double density), aortic silhouette dilation (due to hematoma, aneurysm or edema), and mass effects that usually displace contralaterally structures such as the trachea and esophagus [18] (Fig. 6.4).

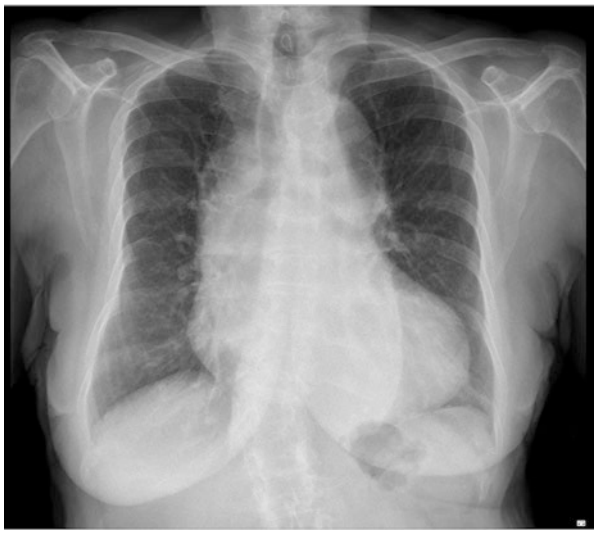

Fig. 6.4 Chest X-ray of a patient presenting with a dissected thoracic aneurysm. A widened mediastinum is observed. A prominent aorta and cardiomegaly are also distinctive features

6.11.2 Echocardiography

Ultrasonographic imaging comes in handy in the approach of a hemodynamically unstable patient inside the emergency room because it is quick, cheap, and mobile. These studies are safe, lack the added risk of radiation exposure, and can be performed bedside [1, 3]. A minor inconvenience is that both echo modalities are operator-dependent [9].

Transthoracic echocardiography (TTE) has a sensitivity which ranges around 77–100% for AAS type A AAD with a specificity of 93–96% and sensitivity around 31–55% for AAS type B AAD. Its usefulness comes to play as a quick screening tool for the detection of proximal (or type A) AAD [3, 7, 9]. TTE is particularly advantageous in the ER because it offers a fast approach to the patients' anatomy [1, 7]. TTE helps clinicians rule out multiple complications associated to AAD, principally the ones related to type A AAD, such as aortic root involvement, aortic regurgitation, pericardial effusion, pericardial tamponade, and abnormalities in wall motion [1, 2, 7].

Transesophageal echocardiography (TEE) has a higher sensitivity (88–99%) and specificity (80–100%) for unmasking aortic pathology, except the proximal aortic arch [1, 3, 9]. Even IMHs can be detected through the identification of wall thickening while in the absence of a false lumen and an intimal flap [3, 7]. The drawbacks of this imaging study are related to the need for proper sedation and that it cannot be carried out as fast as TTE (Figs. 6.5 and 6.6).

6.11.3 Computed Tomography

Emergency CT imaging usually is the first-line imaging option. Multidetector helical CT angiogram offers high sensitivity and specificity for the detection of AAD and AIMH, both nearing 100% [2, 3, 9]. It has the capability to rule out other

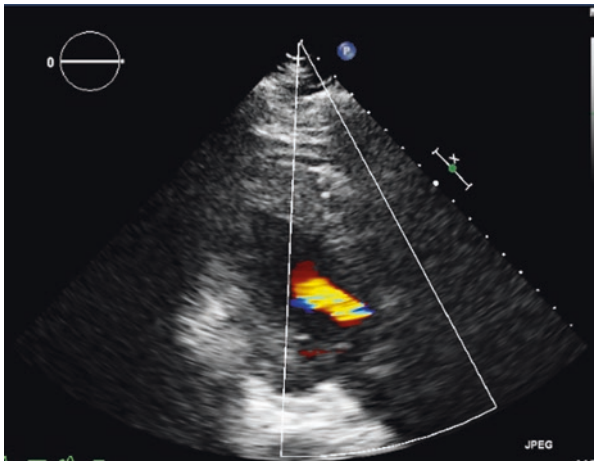

Fig. 6.5 Transthoracic echocardiogram is featuring aortic regurgitation in a patient with an acute aortic dissection

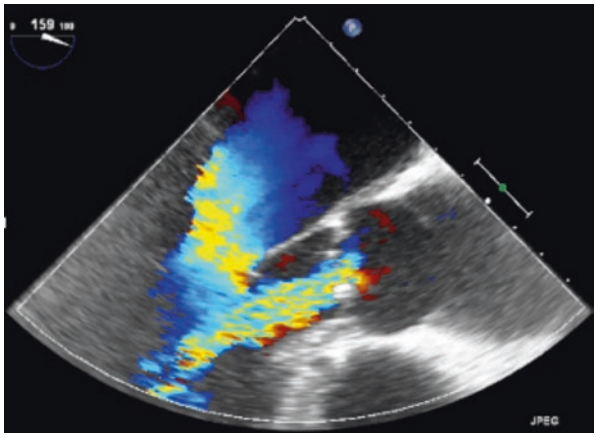

Fig. 6.6 Transesophageal echocardiogram is depicting aortic regurgitation in the same patient with an acute aortic dissection

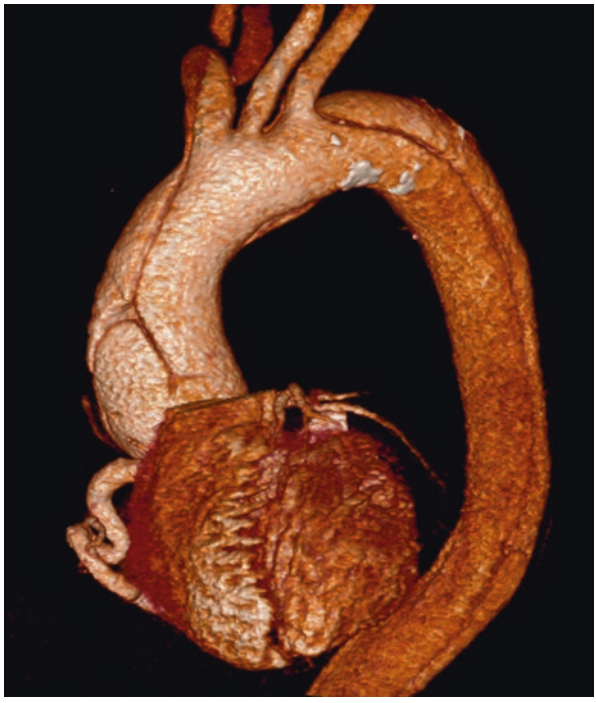

Fig. 6.7 CT angiography 3D reconstruction of a DeBakey type I dissection which starts at the aortic root and extends all the way through the ascending aorta, supra-aortic arch and supra-aortic vessels

differential diagnoses (such as lung or coronary pathologies) [1, 5, 7]. Images are acquired in a speedy fashion, which is ideal inside the ER environment or while evaluating unstable patients.

CT imaging offers high spatial resolution and isn't limited to a few windows nor by the patient's anatomical variants. Using this imaging study, CT technicians can display a 3D reconstruction that allows clinicians to better understand the patient's anatomical variants and disease burden (Figs. 6.7, 6.8, 6.9, 6.10, 6.11, and 6.12).

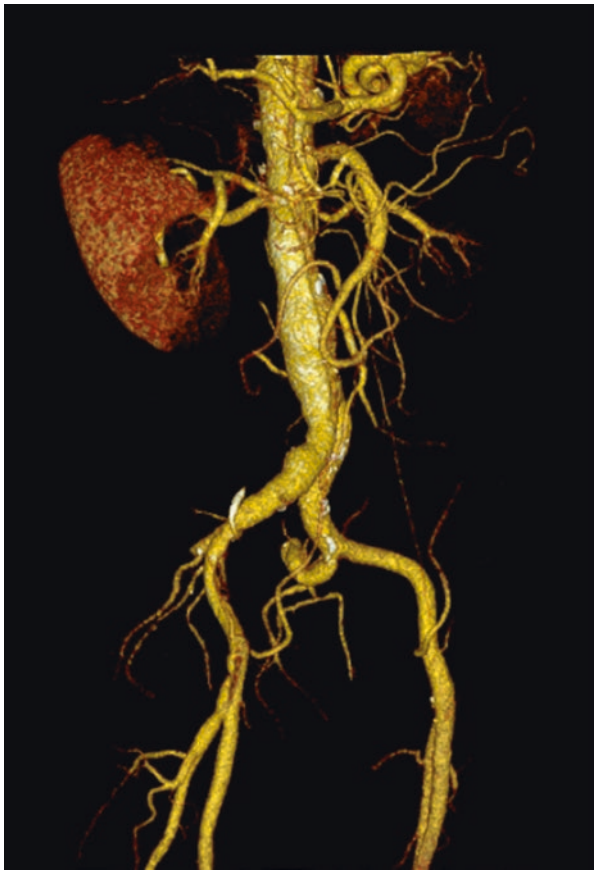

Fig. 6.8 CT angiography 3D reconstruction of a DeBakey type I dissection extending from the ascending aorta to both iliac arteries. During its trajectory along the abdominal aorta, the dissection tear excluded the left renal artery

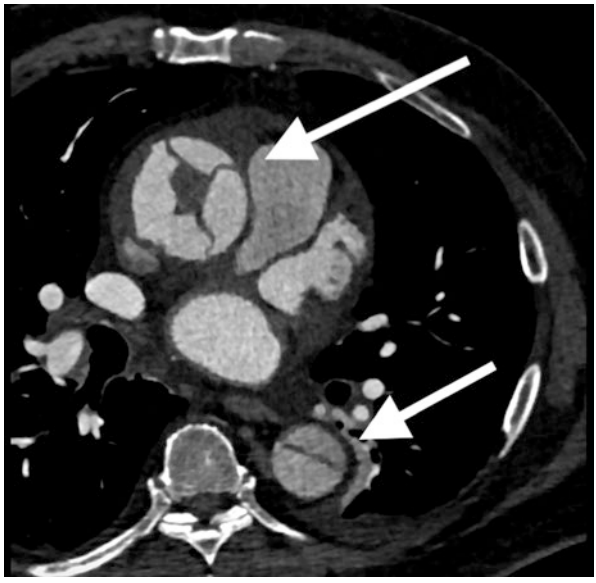

Fig. 6.9 CT angiography transverse projection of a patient with an acute aortic dissection. The tear can be seen in the thoracic ascending and descending aorta (arrows) along with its true and false lumens

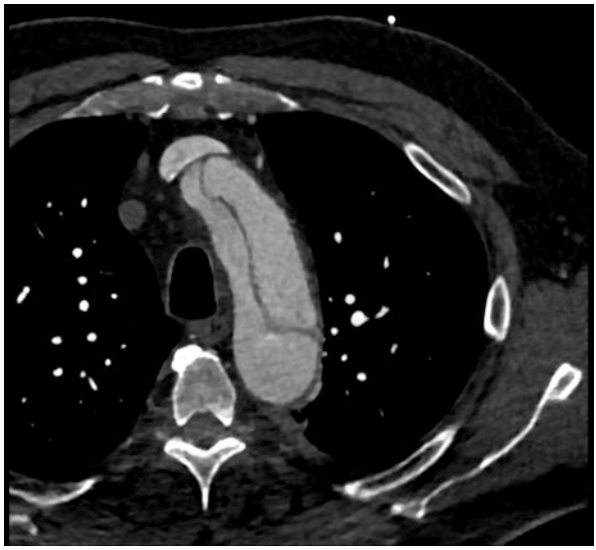

Fig. 6.10 CT angiography transverse projection of a patient with an acute aortic dissection. The arrow shows the tear extending along the aortic arch

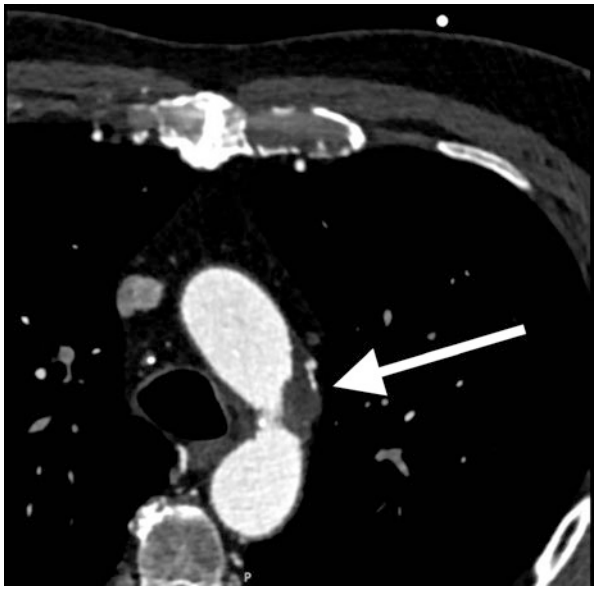

Fig. 6.11 CT angiography transverse cut in which an intramural hematoma is identified. A large hematoma (arrow) is seen at the aortic walls, along with some atherosclerotic plaque

CT scanning and image post-processing can also ascertain accurate measurements between anatomical landmarks. Computed tomography could characterize the aneurysmal aortic disease, the atherosclerotic burden, ulceration, intravascular thrombosis, the presence of flaps, dissection, calcification, or rupture. Anatomic areas of abnormal perfusion can be observed through irregular contrast distribution [2, 3] (Figs. 6.13 and 6.14).

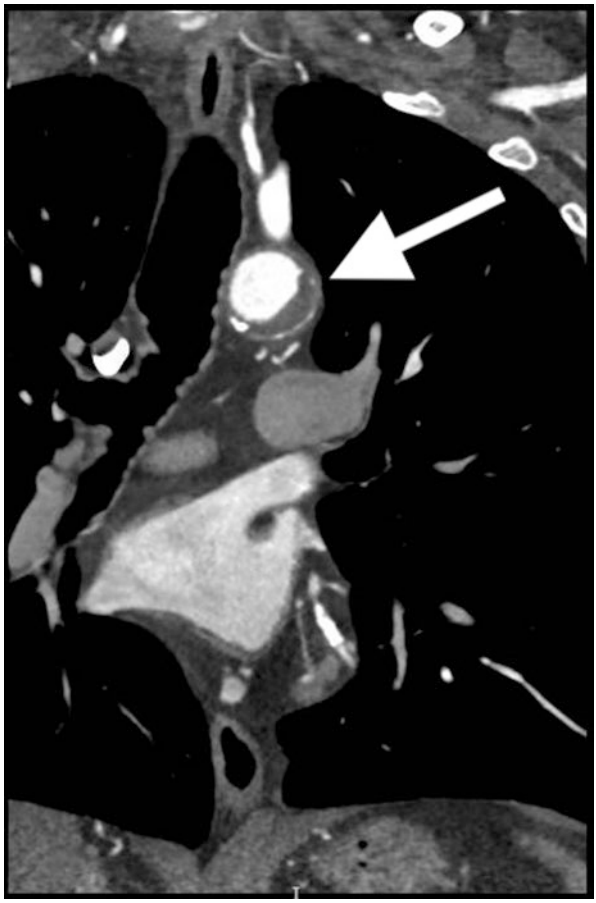

Fig. 6.12 CT angiography coronal projection of the same intramural hematoma

The downsides are that CTs are not readily available in many hospitals, they lack mobile capacities, patients are exposed to ionizing radiation (usually low doses), and to gain better imaging resolution, CT studies need IV contrast [1, 5, 7]. Iodinated contrast agents carry inherent risks such as allergic reaction and contrast nephropathy [1] (Figs. 6.15 and 6.16).

6.11.4 Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is another highly sensitive and specific imaging procedure, both ranging between 98 and 100% [3, 7]. It shares similar advantages with CT angiography, like the ability to deliver unlimited windows, and it possesses a high spatial resolution. MRI is notably helpful in patients presenting with aortic regurgitation because it can accurately calculate regurgitating volumes,

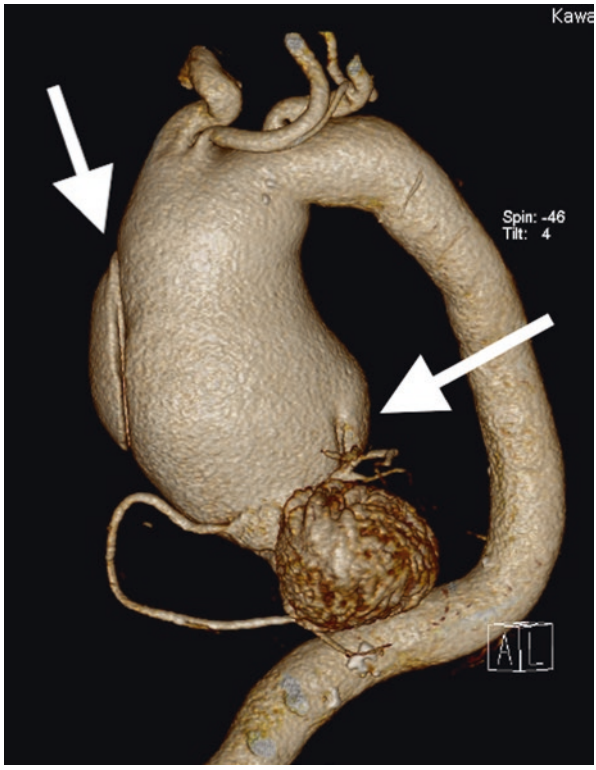

Fig. 6.13 CT angiography 3D reconstruction of an aortic aneurysm affecting the ascending aorta. An aneurysm presents a type I DeBakey Stanford A dissection with two tears (arrow)

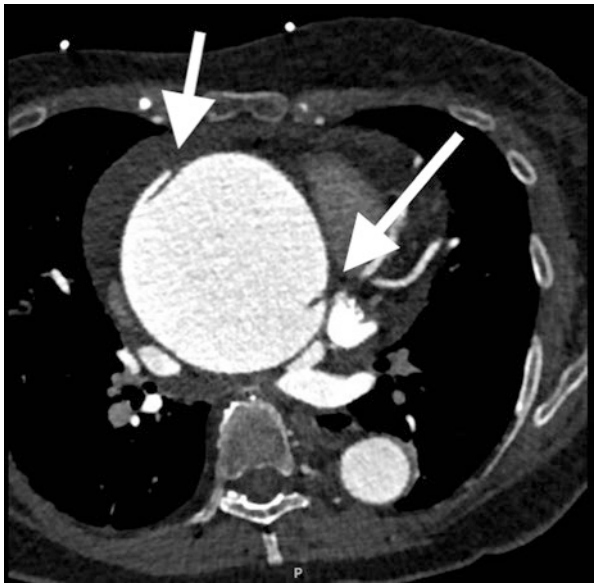

Fig. 6.14 CT angiography of the same patient presenting an aortic aneurysm with a type I DeBakey Stanford A dissection with two tears (arrows)

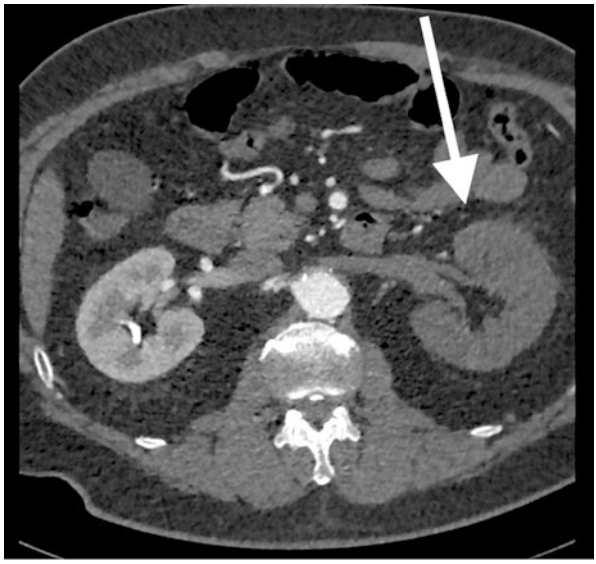

Fig. 6.15 CT angiography images showing two different reconstructions of a patient with an abdominal dissection tear. The left renal artery was adjacent to the false lumen. The lack of contrast in the left kidney is compatible with hypoperfusion

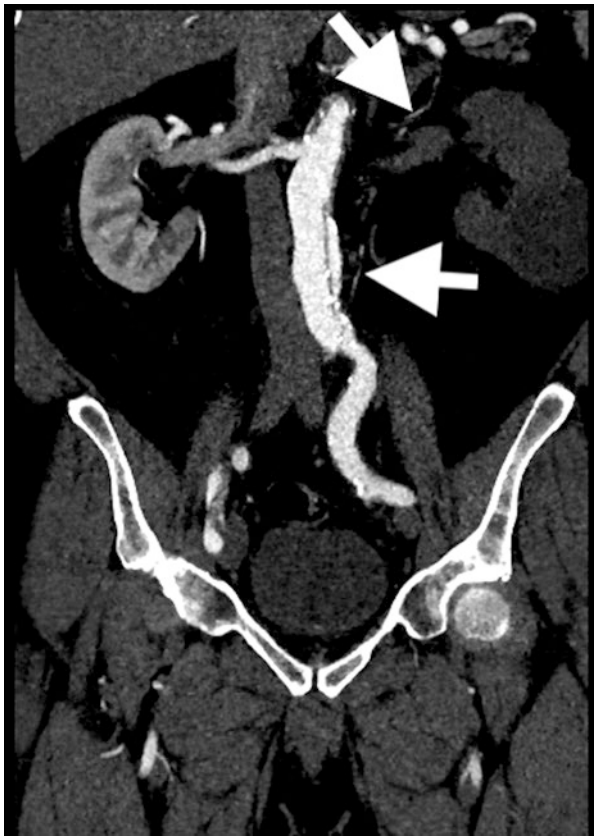

Fig. 6.16 CT angiography images showing two different reconstructions of a patient with an abdominal dissection tear. The left renal artery was adjacent to the false lumen. The lack of contrast in the left kidney is compatible with hypoperfusion

and normally no IV contrast is needed during the blood vessels evaluation. The reconstruction of 3D angiograms is also feasible using this imaging modality. An added benefit is that this imaging modality lacks the characteristic risk of radiation exposure [2].

The inconveniences it presents are its higher cost and poor availability; many hospitals are not equipped with MRI. Unstable patients are not the best candidates because the image-acquisition process tends to be time-consuming [1, 5, 7]. The prolonged duration of this process can be an issue with claustrophobic patients. Some other common contraindications are the presence of ferromagnetic implants, prosthesis, and pacemakers [3]. MRI falls short during the discrimination process of the coronary arteries and the aortic root; for these reasons it is the least used imaging study during emergencies. Information retrieved from the International Registry of Aortic Dissection reports a 1–5% usage in the ER setting, and normally patients who underwent MR imaging had a delayed diagnosis of AAD, fact that is related to increased mortality [3, 7]. Figures 6.17 and 6.18 show the MRI angiography of a patient with an acute dissection tear extending from the ascending to the descending aorta along with the aortic arch.

6.12 Treatment and Management

Aortic syndromes prompt strict blood pressure management. Normal blood pressure (BP) decreases aortic wall stress and lowers the possibility of wall rupture. Ideally, BP levels should be set below 110 mmHg systolic and 60 mmHg diastolic. The drugs of choice are intravenous beta-blockers (also helpful in patients with aortic regurgitation), and accepted choices are intravenous nitrates/vasodilators and oral calcium channel blockers. Intravenous beta-blockers (BB) not only help

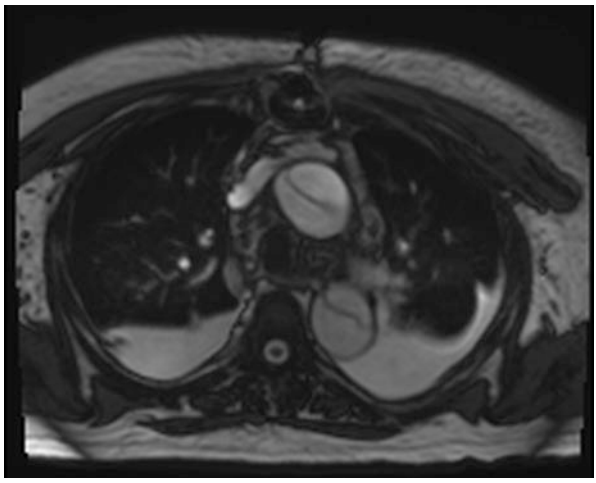

Fig. 6.17 MRI angiography of a patient with an acute dissection tear extending from the ascending to the descending aorta along with the aortic arch (arrow). Bilateral pleural effusions are observed. No contrast was needed for this MRI

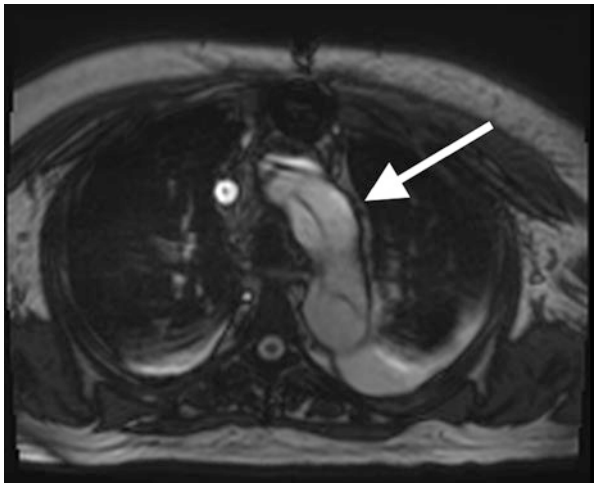

Fig. 6.18 MRI angiography of a patient with an acute dissection tear extending from the ascending to the descending aorta along with the aortic arch (arrow). Bilateral pleural effusions are observed. No contrast was needed for this MRI

lower BP, but they also offer heart rate control, which is also beneficial in lowering vascular wall stress. Usually, beta-blocking therapy is titrated to an HR goal of 60 bpm or less. ACE inhibitors or ARBs might be added to medical treatment if BBs have been initiated and systolic BP remains above 120 mmHg. Vasodilating agents should not be initiated as a first-line agent to avoid reflex tachycardia [3]. Systolic BP should be maintained between 100 and 120 mmHg, so careful monitoring is key to success [1].

Pain control is very important; strong analgesics, usually opioids, are needed. Avoid nonsteroidal anti-inflammatory drugs due to their intrinsic antiplatelet activity. Unstable patients should be stabilized and treated in an as-needed basis, inotropic support, central IV lines, and blood transfusions. Management of AAS is often surgical. All type A dissections should undergo surgical correction. Surgical treatment has shown to reduce 1-month and long-term mortality in patients with type A AD. Several factors, such as low coronary perfusion, pericardial tamponade, and stroke, have a negative impact on mortality. Regarding aortic regurgitation, a surgical team must evaluate the feasibility for the repair or replacement of the aortic valve. Grafting can be needed if the dissection extends into the supra-aortic branches [1–3].

Type A AIMH and APAUs when complicated (pericardial effusion, aortic hematomas, rapidly growing lesions) are subject to urgent medical care [2].

Most uncomplicated type B dissections, intramural hematomas, and penetrating ulcers can be subject to endovascular aortic repair. Uncomplicated AAS are those in which circulatory shock and low perfusion are absent. Thoracic endovascular aortic repair has shown lower rates of aortic remodeling and lower mortality compared to patients receiving only medical therapy [7]. This device directs blood flow through the true lumen improving distal perfusion and promoting the thrombosis of the false lumen [2, 3].

Table 6.6 summarizes key clinical aspects for the evaluation and treatment of acute aortic syndromes.

Table 6.6 Quick evaluation guide

Describe chest pain	Intense, sharp or stabbing Sudden-onset Radiation to back or abdomen
Risk factors	Smoker, history of high blood pressure Abnormal phenotype (Marfan) Recent aortic valve or aortic manipulation
Physical examination	Wide pulse pressure (>60 mmHg) Abnormal pulse (collapsing) Absent pulse (unilateral) Aortic regurgitation murmur (new/not known)
Diagnostic testing	Chest X-Ray (wide mediastinum, calcified aortic knob) Electrocardiogram (ST-elevation myocardial infarction, Left ventricular hypertrophy/overload) D-dimer (>500 ng/mL) Troponin (1/4 patients are Tn positive)
CT angiogram	Quick and with high sensibility and especificity for acute aortic syndrome Establishes the classification and extension Helps evaluate the alternate diagnosis 1. Coronary artery disease 2. Lung/pleural pathology Affected adjacent organs
Treatment	Pain management Blood pressure and heart rate control
TTE	Consider if patient's vitals are unstable

TTE transthoracic echocardiogram

6.13 Additional Clinical Practice Takeaways

- Be highly suspicious of patients presenting to the ER with acute, stabbing, and severe thoracic pain.
- Particularly young males with an abnormal phenotype or older ones with long-standing high blood pressure who present with pain and difficult control high blood pressure in the ER setting.
- Coronary risk factors may be a common denominator, and usually ACS are ruled out, but still, severe pain persists.
- Due to its high diagnostic yield and fast imaging processing, consider expediting aortic imaging, especially if patients present with hemodynamical deterioration.
- AAS, including aortic dissection, penetrating aortic ulcer, and aortic intramural hematoma, are clinically indistinguishable one from another, and their cardinal manifestation is acute onset of severe chest pain.
- Specific groups of patients tend to be at high risk for aortic pathology (Marfan syndrome, smokers, hypertension, high-speed trauma, or deceleration).

- D-dimer levels below the 500 ng/mL threshold make the diagnosis of aortic syndrome less probable; D-dimer levels below the 500 ng/mL threshold make the diagnosis of aortic syndrome less probable.
- Normal D-dimer does not exclude the possibility of AIMH biomarkers such as cardiac troponin I.
- Biomarkers such as cardiac troponin I and B-type natriuretic peptide are of special importance while assessing myocardial stress.
- Transthoracic echocardiography has a sensitivity which ranges around 77–100% for AAS type A AAD with a specificity of 93–96% and sensitivity around 31–55% for AAS.
- Transesophageal echocardiography (TEE) has a higher sensitivity (88–99%) and specificity (80–100%) for unmasking aortic pathology, except for the proximal aortic arch.
- Multidetector helical CT angiogram offers high sensitivity and specificity for the detection of AAD and AIMH, both nearing 100%.

Acknowledgments Images are courtesy of Erasmo de la Peña-Almaguer, Head of Cardiovascular Imagen at Instituto de Cardiologia y Medicina Vascular, Tec Salud, Hospital Zambrano Hellion, Escuela de Medicina y Ciencias de la Salud, Tecnologico de Monterrey.

References

1. Bossone E, LaBounty TM, Eagle KA. Acute aortic syndromes: diagnosis and management, an update. Eur Heart J. 2018;39:739–749d.
2. Erbel R, Aboyans V, Boileu C, Bossone E. 2014 ESC guidelines on the diagnosis and treatment of aortic diseases. Eur Heart J. 2014;35:2873–926.
3. Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey DE, 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. Circulation. 2010;121:e266–369.
4. Bonnefoy E, Godon P, Kikorian G, Chabaud S, Touboul P. Significance of serum troponin I elevation in patients with acute aortic dissection of the ascending aorta. Acta Cardiol. 2005;60:165–70.
5. Bustamante-Munguira J, Juez M. Síndrome aórtico agudo. Cir Cardiovasc. 2016;23:38–44.
6. Evangelista A. Avances en el síndrome aórtico agudo. Rev Esp Cardiol. 2007;60:428–39.
7. Wells CM, Subramaniam K. Acute aortic syndrome. In: Subramaniam K, Park KW, Subramaniam B, editors. Anesthesia and perioperative care for aortic surgery. New York: Springer; 2011. p. 17–36. https://doi.org/10.1007/978-0-387-85922-4_2. Accessed 5 Sept 2018.
8. Evangelista A, Maldonado G, Gruosso D, Teixido G, Rodríguez-Palomares J, Eagle K. Insights from the international registry of acute aortic dissection. Glob Cardiol Sci Pract. 2016;8:1–14.
9. Coyle S, Moriarty T, Melody L, Ryan D. Diagnostic testing in acute aortic dissection. Curr Emerg Hosp Med Rep. 2014;2:97–103.
10. Rogers AM, Hermann LK, Booher AM, Nienaber CA, Williams DM, Kazerooni EA, et al. 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;123:2213–8.

11. Strayer RJ, Shearer PL, Hermann LK. Screening, Evaluation, and early management of acute aortic dissection in the ED. Curr Cardiol Rev. 2012;8:152–7.
12. Hirata K, Wake M, Kyushima M, Takahashi T, Nakazato J, Mototake H, et al. Electrocardiographic changes in patients with type A acute aortic dissection. J Cardiol. 2010;56:147–53.
13. Li G, Wu X-W, Lu W-H, Cheng J, Wu X-Y, Ai R, et al. High-sensitivity cardiac troponin T: a biomarker for the early risk stratification of type-A acute aortic dissection? Arch Cardiovasc Dis. 2016;109:163–70.
14. Jerjes-Sanchez C, Garcia N, Diaz de Leon-Gonzalez E, Garcia-Sosa A, Sanchez-Ramirez CJ. Significance of biomarker panel including cardiac troponin I, D- dimer, and B-type natriuretic peptide in acute aortic dissection. J Cardiol Ther. 2013;1:58–63.
15. Nazerian P, Mueller C, Soeiro A de M, Leidel BA, Salvadeo SAT, Giachino F, et al. Diagnostic accuracy of the aortic dissection detection risk score plus D-dimer for acute aortic syndromes: the ADvISED Prospective Multicenter Study. Circulation. 2018;137:250–8.
16. Bonaca MP, O'Gara PT. Diagnosis and management of acute aortic syndromes: dissection, intramural hematoma, and penetrating aortic ulcer. Curr Cardiol Rep. 2014;16:1–13.
17. Asha SE, Miers JW. A systematic review and meta-analysis of D-dimer as a rule-out test for suspected acute aortic dissection. Ann Emerg Med. 2015;66:368–78.
18. Damberg A, Ziganshin BA, Elefteriades JA. Chest X-ray in aortic disease. In: New approaches to aortic diseases from valve to abdominal bifurcation. Elsevier; 2018. p. 129–31. https://linkinghub.elsevier.com/retrieve/pii/B9780128099797000122. Accessed 5 Sept 2018.