

Computed Tomography Imaging Findings of Acute Aortic Pathologies

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Abstract

Acute aortic syndromes (AAS) encompass a spectrum of life-threatening conditions characterized by acute aortic pain. AAS include acute aortic dissection, intramural hematoma, penetrating atherosclerotic ulcer, and aneurysm rupture. The prognosis of AAS is clearly related to prompt diagnosis and appropriate management. The different types of AAS cannot be reliably differentiated solely based on clinical presentation since the clinical features are indistinguishable. Multidetector-row computed tomography (MDCT) with electrocardiographic gating (ECG-gated MDCT) has been used in the acute emergency setting as a powerful clinical tool, which enables rapid and specific diagnosis of aortic pathologies. ECG-gated MDCT significantly reduces motion artifact and avoids potential pitfalls in the diagnosis of AAS. The aim of this review is to evaluate the role of MDCT imaging in the assessment of AAS and to discuss the differentiation of this spectrum of aortic diseases with reference to the key imaging findings.

Categories: Cardiology, Emergency Medicine, Radiology

Keywords: acute aortic syndrome, aortic dissection, intramural hematoma, penetrating atherosclerotic ulcer, multidetector-row computed tomography

Introduction And Background

Acute aortic syndromes describe the acute presentation of patients with characteristic aortic pain caused by one of several life-threatening conditions of aortic diseases. AAS is an umbrella term that traditionally includes four aortic diseases: aortic dissection (AD), intramural hematoma (IMH), penetrating atherosclerotic ulcer (PAU), and rupture of an aortic aneurysm, often due to trauma [1-3]. The incidence of AAS is 2.6-3.5 cases/100,000/year; two-thirds are male, with an average age of 63 years [4]. Predisposing factors, such as hypertension, Marfan's syndrome, atherosclerosis, cardiac surgery, inflammatory changes, and thrombi, are important in the process of identifying patients at risk both in adult and juvenile populations [5-7]. The clinical presentation of AAS is a real-time emergency, often presenting with acute chest pain, varyingly described as severe, tearing, or migratory [2,8]. Correlation of the clinical history, cardiac enzymes, and electrocardiography (ECG) can help differentiate AAS from acute coronary syndrome, however, it is essential to remember that acute coronary syndrome may occur as a result of AAS [3,8].

Multidetector-row computed tomography (MDCT) with electrocardiographic gating (ECG-gated MDCT), trans-esophageal echocardiography (TEE), and magnetic resonance imaging (MRI) are valuable tests in the diagnosis of AAS. TEE and MRI could be considered complementary

Received 06/26/2019

Review began 06/28/2019

Review ended 07/22/2019

Published 08/30/2019

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How to cite this article

Duran E S, Ahmad F, Elshikh M, et al. (August 30, 2019) Computed Tomography Imaging Findings of Acute Aortic Pathologies. Cureus 11(8): e5534. DOI 10.7759/cureus.5534

modalities in defining particular diagnostic aspects, such as the functional evaluation of an aortic valve, heart function, floating thrombus, or in the case of a contraindication of iodinated contrast agent. The major advantage of echocardiography is the assessment of valve lesions, specifically aortic regurgitation in AAS [9]. The role of chest radiography for diagnosis or surveillance in this setting is very limited [10]. Conventional catheter angiography may be preferred as a means for treating complications of the disease. The main objectives of imaging are to confirm the diagnosis of an aortic wall lesion and to ascertain the site, extension, and complications of the disease. MDCT angiography is the modality of choice to evaluate patients with suspected AAS, as it has rapid acquisition and high diagnostic accuracy in detecting of the AAS [11-12].

In this review, our aim is to illustrate the MDCT findings of the most frequently encountered non-traumatic aortic emergencies. The imaging technique and typical imaging findings of the AAS will be discussed in this review.

Review

MDCT angiography imaging technique in acute aortic syndrome

The MDCT imaging protocol depends on the technical characteristics of tomography available such as the number of detectors (16, 64, 128, 256, 320), rotation tube speed, and table feeding. Typically, axial reconstruction thickness should be between 1 mm and 3 mm, using 16 x 1.25 mm on 16-row scanners, 64 x 0.5 mm on 64-row scanners and 128 x 0.6 mm on the new 128-row scanners. Sagittal, coronal, and multiplanar reconstructions (MPRs) should be generated on three-dimensional (3D) workstations [11-13]. The protocol should be optimized to reduce examination times, to improve spatial resolution, and to apply ideal total contrast material volume and exposure dose.

Non-contrast images provide important information concerning the presence of calcification and intramural hematoma, size of the aorta, general status of the lung parenchyma, mediastinum, and heart size, presence of pleural effusion, abdominal organs, bowel, intra, and retroperitoneal space, and fasciae fluid collections [14-15].

The contrast agent administration protocol is based on the patient's weight and potential abnormalities in kidney function. After a variable time of 10 to 30 seconds after contrast media injection, intravascular contrast enhancement increases and lumen opacification appears linearly correlated with dilution effect; this is influenced by several parameters, such as contrast media concentration, flow rate and pressure of infusion, cardiac output, scan parameters, and the presence of saline flush following the injection of contrast media when double injectors are used. Bolus timing is crucial in MDCT angiography; therefore, especially in critical patients, the automatic detection of bolus such as bolus tracking may be utilized. The protocol of contrast agent delivery depends on the characteristics of computed tomography available. Generally, non-ionic iodinated contrast agent at a high concentration (≥ 350 mg/ml) at a dosage max of 0.1- 0.2 ml/kg body weight with moderate-high flow rate (4-4.5 ml/s) with a bolus (30-50 ml) saline solution at the same flow rate following contrast injection using a double pump injector, could be considered the simplest and most efficient protocol, especially in emergency cases. The contrast agent dosage should be calculated in relation to the scan duration in order to avoid the scan time to exceed the infusion time delivery [16-18].

ECG-gated MDCT of the thoracic aorta significantly reduces the motion artifacts when compared with non-ECG-gated studies [19-21]. ECG gating of the aorta and coronary arteries can be performed either prospectively or retrospectively. During prospective ECG gating, the image is obtained usually during late diastole. However, this method is particularly susceptible

to artifacts due to rapid changes in heart rate. The retrospective ECG-gating method reveals data continuously throughout the cardiac cycle. Images can also be viewed at any point along the R-R interval, thus allowing the selection of the phase with the least motion artifact for reconstruction [22]. However, the retrospective method causes higher radiation exposure than prospective ECG gating due to continuous versus intermittent X-ray exposure [23].

Aortic dissection

Aortic dissection is characterized by the separation of the aortic intima from the media, caused by the shearing forces of blood under high pressure, with variable longitudinal and circumferential extension, resulting in the formation of a double-channel aorta [24]. High blood pressure with the concomitant degenerative changes in the aortic media is the most common trigger for aortic dissection. Marfan syndrome, Turner syndrome, other connective tissue diseases, congenital aortic valvular defects, aortic coarctation, aortic aneurysm, aortitis, and pregnancy are among the most common causes of separation of the aortic intima and media. Since the right lateral wall of the ascending aorta and the proximal segment of the descending thoracic aorta have maximum hydraulic stress, intimal tears, leading to aortic dissections, often occur in these sites of the aorta [1,5,25].

The clinical presentation of aortic dissection can be very misleading and findings on physical examination may be non-specific. Patients may present with a classic history of acute-onset central chest pain that radiates to the back. Syncope can result from acute dissections and occurs in 9% of cases; syncope may be caused by hypotension secondary to cardiac tamponade, aortic rupture, cerebral vessel obstruction, or the activation of cerebral baroreceptors [26-27].

The intimal tear causes blood to enter the media from the vessel lumen. The blood-filled space within the medial layer creates a false lumen. This results in two lumina: a true and a false lumen with the false lumen having pressures greater than or equal to those in the true lumen [28]. Because of pressure differences, the false lumen may compress or obstruct the true lumen. Thus, the dissection can move in either an antegrade or a retrograde direction. The dissections may remain patent as a false lumen, thrombose, recommunicate with the true lumen through fenestrations, or rupture (Figure 1) into potential spaces such as the pericardial, pleural, or peritoneal cavities [29].

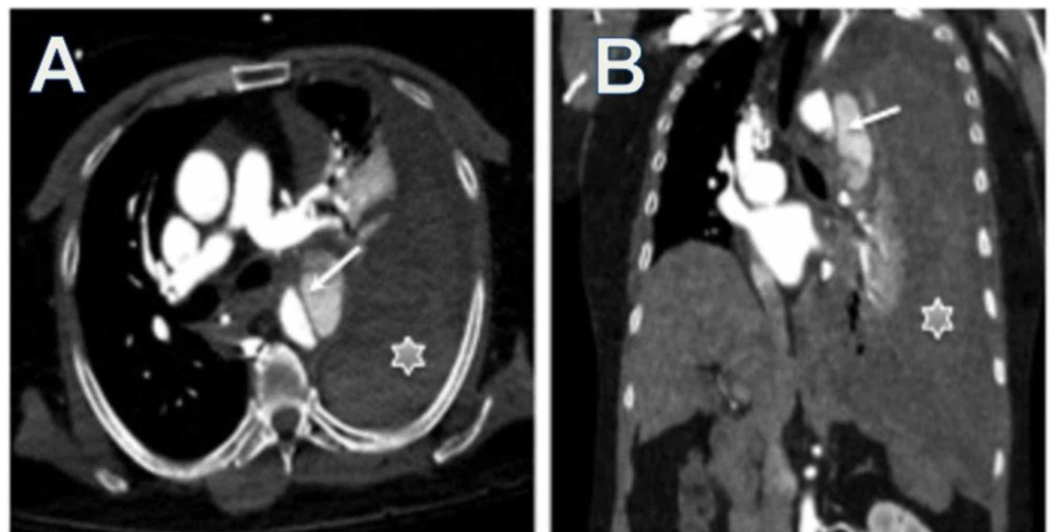


FIGURE 1: Type B Aortic Dissection

Axial oblique (A) and coronal oblique (B) reformats and 3D volume rendering image of thoracic CT angiography images demonstrate Stanford type B thoracic aortic dissection (arrow) and rupture,

with left hemothorax, pleural effusion, and near complete atelectasis of the left lung (star).

The classification of aortic dissection is based on the location and extension of the dissection, and the DeBakey and Stanford classification systems are most commonly utilized [30-31]. In general, the Stanford classification is preferred due to its capacity to propose immediate clinical management: surgical (type A) versus medical (type B) [1,5]. Stanford type A dissection includes the ascending aorta and can extend into the descending aorta (Figure 2). Stanford type B dissection includes the descending aorta beyond the origin of the left subclavian artery [30].

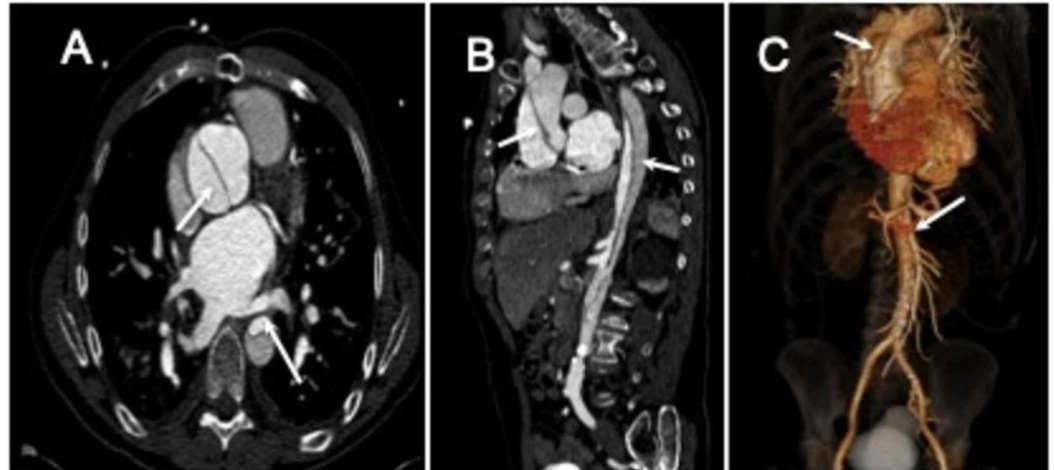


FIGURE 2: Type A Aortic Dissection

A. Axial contrast-enhanced CT image demonstrating an intimal flap (arrows) consistent with a Stanford Type A dissection with extension into the distal thoracic aorta.

B. 3D volume rendering image of the thoracic aorta visualization of the type A dissection (arrows).

C. Sagittal reformat image shows the extension of the intimal flap into the abdominal aorta (arrows). The false lumen hypoattenuating as compared to the true lumen.

A widened mediastinum is the most common imaging finding on the radiography. In a study published in JAMA, a widened mediastinum was noted in 61.1% of aortic dissection cases, displacement of calcification of the aorta was reported in 14.1% of cases, with an abnormal cardiac contour being noted in 25.8% [32]. TEE has a reported sensitivity of 59%-83% and a specificity of 63%-93% for the diagnosis of aortic dissection. The sensitivity of transthoracic echocardiography is between 78% and 100% for the diagnosis of a type A dissection, but it is only 31%-55% for dissections involving the descending aorta [30,33].

In practice, ECG-gated MDCT should be preferred since it allows more precise delineation of the proximal extent of the intimal flap in relation to the aortic valve and coronary arteries and, more importantly, helps avoid the overdiagnosis of aortic dissections caused by the misinterpretation of a motion artifact as an intimal flap. Non-contrast MDCT images help obtain the extent of inward displacement of intimal calcification. The two most useful indicators of the false lumen are the beak sign and the cobweb sign [1,5,27]. The differences between true and false lumen MDCT imaging findings are summarized in Table 1.

True Lumen	False Lumen
Smaller than a false lumen	Larger than a true lumen
Directly communicates with the aorta	Not connected to the unaffected aorta
Intima displaced inwards	Beak sign: Acute angle in the corner of the false lumen with true lumen
Calcification along the intimal flap	Cobwebs sign: Band of connective tissue crossing the false lumen
Calcification along the intimal flap	The surface of the intimal flap is convex
More enhanced than false lumen during the peak of aortic enhancement	Hypodense compared to the true lumen during the peak of aortic enhancement due to the presence of slow flow
Wrapped around the false lumen	Wrapped around the true lumen

TABLE 1: MDCT findings to differentiate true and false lumen

MDCT: multidetector-row computed tomography

Contrast-enhanced magnetic resonance angiography is more available for the investigation of aortic dissection in medically stable patients or those with chronic dissections. It has several advantages over MDCT angiography, including a lack of non-ionizing radiation, multiplanar evaluation, and greater vessel coverage at high resolution. Three-dimensional magnetic resonance angiography can reveal a complete and dynamic display of aortic dissection and display the true and false lumina [34-35].

Recently, triple rule-out MDCT protocol is used to assess the aorta, coronary arteries, and pulmonary arteries during a single scan with the use of several optimally timed boluses of contrast material and ECG gating in patients who are at low risk for an acute coronary syndrome. The main goal is to minimize the contrast material dose and radiation exposure while achieving optimal image quality, providing coronary artery image quality equivalent to that of dedicated coronary MDCT angiography, pulmonary artery image quality equivalent to that of dedicated pulmonary MDCT arteriography, and high-quality images of the thoracic aorta without pulsation artifact. In addition, the presence of acute coronary syndrome and aortic dissection can be evaluated via a triple rule-out technique [34]. This technique can evaluate and rule out pulmonary emboli, aortic coronary syndrome, and AAS all through the same imaging study.

Intramural hematoma

Intramural hematoma (IMH) is a variant of dissection and is characterized by the presence of hemorrhage into the aortic media from the vasa vasorum. Tears seen in classic aortic dissection are absent. IMHs are thought to comprise 10%-30% of all AAS [36-37]. IMH may originate spontaneously as a consequence of a penetrating ulcer or after thoracic trauma. It may be a precursor of aortic dissection, and many investigators have suggested that IMH is synonymous with a thrombosed type or noncommunicating aortic dissection [9]. Between the 50% and 85% of IMH occur in the descending aorta and are typically associated with hypertension. IMH is the cause of 5%-20% of acute aortic dissections. The clinical findings of IMH are similar to those of other acute aortic syndromes and patients predominantly present with acute chest pain [5,28].

The hyperdense crescent-shaped or ring-like thickening of the aortic wall is often detected on the non-contrast MDCT images, and precontrast imaging is essential in this protocol (Figure 3). The absence of an obvious communication between the true and false lumen explains the absence of flow and the lack of enhancement with contrast administration on MDCT or MRI [1,11]. On contrast-enhanced MDCT series, IMH may be easily confused with atherosclerotic thrombus, as the mildly increased attenuation of IMH compared to thrombus may be overlooked by the window level settings used to look at contrast-enhanced images. On the contrary to aortic dissection, the configuration of the IMH usually does not spiral around the aortic lumen (Figure 4). Additionally, IMH cannot be easily differentiated on the contrast-enhanced images. However, the thrombosed false lumen in classic aortic dissection has a pattern that spirals longitudinally around the aorta while an IMH pattern generally maintains a circumferential and eccentric relationship with the aortic wall. The advanced spatial resolution of MDCT allows us to visualize these features and differentiate the two pathologies [38-39].

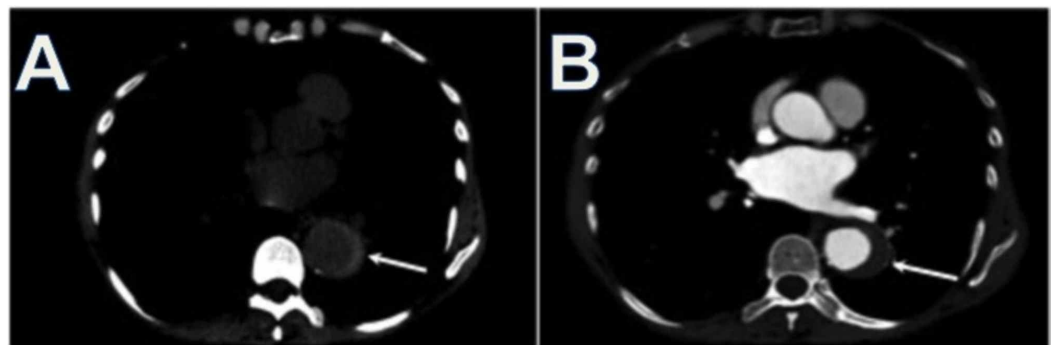


FIGURE 3: Intramural Hematoma

A. Intramural hematomas is visualized as a crescent-shaped or ring like hyperdensity on non-contrast enhanced CT imaging.
B. Contrast-enhanced CT demonstrates crescent-shaped hypoattenuation, not to be confused with atherosclerotic thrombus.



FIGURE 4: Aortic Dissection Spiraling

Spiral course of the dissection flap of the aorta is seen in the sagittal reformat image.

Progression to aortic dissection occurs in 28%-47% of patients with IMH [40]. Similar to Stanford's classification in the aortic dissection, surgery is suggested in patients with type A IMH and initial medical therapy is suggested in patients with type B IMH [41].

Penetrating atherosclerotic ulcer

An atherosclerotic plaque erodes the internal elastic lamina into the media of the aortic wall in patients with a penetrating atherosclerotic ulcer (PAU). These ulcers may be complicated by true aneurysm formation, erosion through the media to form a pseudoaneurysm or dissection [1,42]. PAU generally occurs in an elderly individual with multiple risk factors for atherosclerosis and the associated comorbidities of atherosclerotic disease such as coronary artery disease and peripheral arterial disease. The clinical findings of PAU may usually be the same as those of aortic dissection. In the absence of atherosclerosis, it can also occur in young

patients with connective tissue disorder or after the rupture of a mycotic plaque [40,43]. Since the atheromatous plaque can rupture and precipitate intramural hemorrhage, early diagnosis of the PAU is essential. Most cases with PAU (approximately >90%) occur in the aortic arch or descending aorta; atherosclerotic plaques are rarely located in the ascending aorta [44].

In extensive atherosclerosis, high-density hematoma surrounding the ulceration and variable size IMHs can be detected in non-contrast enhanced images. In contrast-enhanced images, ulceration of the atherosclerotic plaque can be seen and would show protrusion beyond the intimal level into the medial layer of the aortic wall, together with a focal outpouching of the outer aortic contour. The protrusion and focal contour change may differentiate the PAU from the common atheromatous ulcer (Figure 5) [36,44].

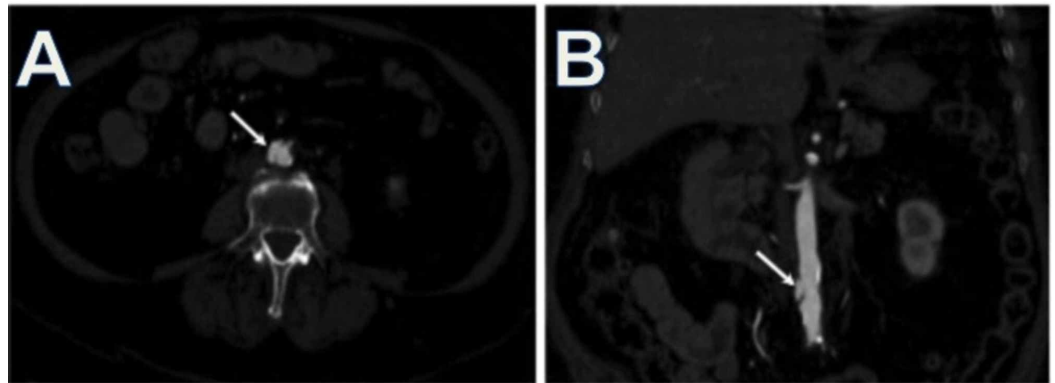


FIGURE 5: Penetrating Atherosclerotic Ulcer

Axial image (A) and coronal reformat (B) of the contrast-enhanced CT demonstrate an atheromatous plaque in the abdominal aorta extending beyond the intima into the aortic media. PAU will increase the patient's risk of intramural hemorrhage, pseudoaneurysm, or dissection formation.

Invasive treatments such as surgery and stent-grafting are required in acute and symptomatic cases, but course observation, including periodical evaluation using imaging techniques is recommended in asymptomatic or chronic cases [41,45].

Aortic aneurysm and rupture

Aortic aneurysmal enlargement is defined as a permanent dilatation to at least 150% of the normal size. According to their contents, aneurysms are divided into two: true and false. “True” aneurysms include all the layers of the aortic wall whereas “false” aneurysms are contained ruptures and usually comprise just the adventitia, surrounded by fibrosis and hematoma. Localized aneurysms are usually divided into two: “saccular” and “fusiform,” with fusiform defined by more diffuse dilatation [2,5,46]. Most of the aneurysms involve the aortic isthmus; aneurysm rupture occurs when the mechanical stress on the wall exceeds the strength of the wall tissue. The main events of the rupture of the aneurysm include the formation of an intramural hematoma and hemorrhagic leak into the mediastinum through the aortic leak, with the progressive invasion of the pleural cavity, and pericardium. Sometimes, the hematoma may separate the parietal pleura from the endothoracic fascia, leading to an extrapleural hematoma [47-49].

Hyperdense thickening of the aortic wall that represents blood collection between partially disrupted wall layers and mediastinal hematoma is usually detected on MDCT angiography images. This mediastinal hematoma may extend from the site of the aortic lesion into the

periaortic mediastinal fat. Pleural and rarely pericardial effusion can also be detected on MDCT images. MDCT angiography also may be helpful to depict signs of impending hypovolemic shock via presenting reduction in the caliber of central vessels and excessive contrast enhancement of the aorta relative to the injection parameters [9,11,47-48]

Conclusions

Acute aortic syndromes are important emergency conditions of the aorta and can often lead to the patient's death. Early diagnosis and treatment are essential for improving prognosis. MDCT allows the imaging of the entire aorta with rapid acquisition and data reconstruction to provide a prompt and accurate diagnosis, including all of the AAS types and helps identify relevant complications that may have an impact on surgical planning or management.

Additional Information

Disclosures

Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Maddu KK, Shuaib W, Telleria J, Johnson JO, Khosa F: Nontraumatic acute aortic emergencies: part 1, acute aortic syndrome. *AJR Am J Roentgenol.* 2014, 202:656-665. [10.2214/AJR.13.11437](https://doi.org/10.2214/AJR.13.11437)
2. Vilacosta I, San Roman JA: Acute aortic syndrome. *Heart.* 2001, 85:365-368. [10.1136/heart.85.4.365](https://doi.org/10.1136/heart.85.4.365)
3. Vilacosta I, Aragoncillo P, Canadas V, San Román JA, Ferreirós J, Rodríguez E: Acute aortic syndrome: a new look at an old conundrum. *Heart.* 2009, 95:1130-1139. [10.1136/hrt.2008.153650](https://doi.org/10.1136/hrt.2008.153650)
4. Corvera JS: Acute aortic syndrome. *Ann Cardiothorac Surg.* 2016, 5:188-193. [10.21037/acs.2016.04.05](https://doi.org/10.21037/acs.2016.04.05)
5. Clouse WD, Hallett JW Jr., Schaff HV, Spittell PC, Rowland CM, Ilstrup DM, Melton LJ III: Acute aortic dissection: population-based incidence compared with degenerative aortic aneurysm rupture. *Mayo Clin Proc.* 2004, 79:176-180. [10.4065/79.2.176](https://doi.org/10.4065/79.2.176)
6. Meszaros I, Morocz J, Szlavi J, Schmidt J, Tornóci L, Nagy L, Szép L: Epidemiology and clinicopathology of aortic dissection. *Chest.* 2000, 117:1271-1278. [10.1378/chest.117.5.1271](https://doi.org/10.1378/chest.117.5.1271)
7. Nienaber CA, Fattori R, Mehta RH, et al.: Gender-related differences in acute aortic dissection. *Circulation.* 2004, 109:3014-3021. [10.1161/01.CIR.0000130644.78677.2C](https://doi.org/10.1161/01.CIR.0000130644.78677.2C)
8. Wooley CF, Sparks EH, Boudoulas H: Aortic pain. *Prog Cardiovasc Dis.* 1998, 40:563-589.
9. Ueda T, Chin A, Petrovitch I, Fleischmann D: A pictorial review of acute aortic syndrome: discriminating and overlapping features as revealed by ECG-gated multidetector-row CT angiography. *Insights Imaging.* 2012, 3:561-571. [10.1007/s13244-012-0195-7](https://doi.org/10.1007/s13244-012-0195-7)
10. von Kodolitsch Y, Nienaber CA, Dieckmann C, et al.: Chest radiography for the diagnosis of acute aortic syndrome. *Am J Med.* 2004, 116:73-77.
11. Salvolini L, Renda P, Fiore D, Scaglione M, Piccolid GP, Giovagnonia A: Acute aortic syndromes: role of multi-detector row CT. *Eur J Radiol.* 2008, 65:350-358. [10.1016/j.ejrad.2007.09.020](https://doi.org/10.1016/j.ejrad.2007.09.020)
12. Berger FH, van Lienden KP, Smithuis R, Nicolaou S, van Delden OM: Acute aortic syndrome and blunt traumatic aortic injury: pictorial review of MDCT imaging. *Eur J Radiol.* 2010, 74:24-39. [10.1016/j.ejrad.2009.06.023](https://doi.org/10.1016/j.ejrad.2009.06.023)
13. Brink M, de Lange F, Oostveen LJ, et al.: Arm raising at exposure-controlled multidetector trauma CT of thoracoabdominal region: higher image quality, lower radiation dose. *Radiology.*

- 2008, 249:661-670. [10.1148/radiol.2492080169](https://doi.org/10.1148/radiol.2492080169)
14. Lawler LP, Fishman EK: Multidetector row computed tomography of the aorta and peripheral arteries. *Cardiol Clin*. 2003, 21:607-629.
 15. Wintersperger BJ, Nikolaou K, Becker CR: Multidetector-row CT angiography of the aorta and visceral arteries. *Semin Ultrasound CT MR*. 2004, 25:25-40.
 16. Bae KT, Tran HQ, Heiken JP: Multiphasic injection method for uniform prolonged vascular enhancement at CT angiography: pharmacokinetic analysis and experimental porcine model. *Radiology*. 2000, 216:872-880. [10.1148/radiology.216.3.r00au43872](https://doi.org/10.1148/radiology.216.3.r00au43872)
 17. Fleischmann D: Use of high-concentration contrast media in multiple-detector-row CT: principles and rationale. *Eur Radiol*. 2003, 13:14-20.
 18. Fleischmann D, Rubin GD, Bankier AA, Hittmair K: Improved uniformity of aortic enhancement with customized contrast medium injection protocols at CT angiography. *Radiology*. 2000, 214:363-371. [10.1148/radiology.214.2.r00fe18363](https://doi.org/10.1148/radiology.214.2.r00fe18363)
 19. Cademartiri F, Runza G, Marano R, Luccichenti G, Midiri M: Thoracic cardiovascular imaging with 16-row multislice computed tomography and retrospective ECG-gating. technical note. *Radiol Med*. 2004, 108:487-493.
 20. Flohr T, Prokop M, Becker C, et al.: A retrospectively ECG-gated multislice spiral CT scan and reconstruction technique with suppression of heart pulsation artifacts for cardio-thoracic imaging with extended volume coverage. *Eur Radiol*. 2002, 12:1497-1503. [10.1007/s00330-002-1388-0](https://doi.org/10.1007/s00330-002-1388-0)
 21. Roos JE, Willmann JK, Weishaupt D, Lachat M, Marincek B, Hilfiker PR: Thoracic aorta: motion artifact reduction with retrospective and prospective electrocardiography-assisted multi-detector row CT. *Radiology*. 2002, 222:271-277. [10.1148/radiol.2221010481](https://doi.org/10.1148/radiol.2221010481)
 22. Ohnesorge B, Flohr T, Becker C, et al.: Cardiac imaging by means of electrocardiographically gated multisection spiral CT: initial experience. *Radiology*. 2000, 217:564-571. [10.1148/radiology.217.2.r00nv30564](https://doi.org/10.1148/radiology.217.2.r00nv30564)
 23. Morgan-Hughes GJ, Owens PE, Marshall AJ, Roobottom CA: Thoracic aorta at multi-detector row CT: motion artifact with various reconstruction windows. *Radiology*. 2003, 228:583-588. [10.1148/radiol.2282020873](https://doi.org/10.1148/radiol.2282020873)
 24. Nienaber CA, von Kodolitsch Y, Nicolas V, et al.: The diagnosis of thoracic aortic dissection by noninvasive imaging procedures. *N Engl J Med*. 1993, 328:1-9. [10.1056/NEJM199301073280101](https://doi.org/10.1056/NEJM199301073280101)
 25. Wheat MW Jr: Acute dissection of the aorta. *Cardiovasc Clin*. 1987, 17:241-262.
 26. Braverman AC: Acute aortic dissection: clinician update. *Circulation*. 2010, 122:184-188. [10.1161/CIRCULATIONAHA.110.958975](https://doi.org/10.1161/CIRCULATIONAHA.110.958975)
 27. McMahon MA, Squirrell CA: Multidetector CT of aortic dissection: a pictorial review. *Radiographics*. 2010, 30:445-460. [10.1148/rg.302095104](https://doi.org/10.1148/rg.302095104)
 28. Williams DM, Lee DY, Hamilton BH, et al.: The dissected aorta: part III. Anatomy and radiologic diagnosis of branch-vessel compromise. *Radiology*. 1997, 203:37-44. [10.1148/radiology.203.1.9122414](https://doi.org/10.1148/radiology.203.1.9122414)
 29. Patel PD, Arora RR: Pathophysiology, diagnosis, and management of aortic dissection. *Ther Adv Cardiovasc Dis*. 2008, 2:439-468. [10.1177/1753944708090830](https://doi.org/10.1177/1753944708090830)
 30. Daily PO, Trueblood HW, Stinson EB, et al.: Management of acute aortic dissections. *Ann Thorac Surg*. 1970, 10:237-247.
 31. DeBakey ME, Henly WS, Cooley DA, Morris GC Jr, Crawford ES, Beall AC Jr: Surgical management of dissecting aneurysms of the aorta. *J Thorac Cardiovasc Surg*. 1965, 49:130-149.
 32. Hagan PG, Nienaber CA, Isselbacher EM, et al.: The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. *JAMA*. 2000, 283:897-903.
 33. Meredith EL, Masani ND: Echocardiography in the emergency assessment of acute aortic syndromes. *Eur J Echocardiogr*. 2009, 10:31-39. [10.1093/ejechocard/jen251](https://doi.org/10.1093/ejechocard/jen251)
 34. Halpern EJ: Triple-rule-out CT angiography for evaluation of acute chest pain and possible acute coronary syndrome. *Radiology*. 2009, 252:332-345. [10.1148/radiol.2522082335](https://doi.org/10.1148/radiol.2522082335)
 35. Krinsky GA, Rofsky NM, DeCorato DR, et al.: Thoracic aorta: comparison of gadolinium-enhanced three-dimensional MR angiography with conventional MR imaging. *Radiology*. 1997, 202:183-193. [10.1148/radiology.202.1.8988210](https://doi.org/10.1148/radiology.202.1.8988210)
 36. Birchard KR: Acute aortic syndrome and acute traumatic aortic injury. *Semin Roentgenol*. 2009, 44:16-28. [10.1053/j.ro.2008.10.002](https://doi.org/10.1053/j.ro.2008.10.002)

37. Evangelista A, Dominguez R, Sebastia C, et al.: Long-term follow-up of aortic intramural hematoma: predictors of outcome. *Circulation*. 2003, 108:583-589. [10.1161/01.CIR.0000081776.49923.5A](https://doi.org/10.1161/01.CIR.0000081776.49923.5A)
38. Chao CP, Walker TG, Kalva SP: Natural history and CT appearances of aortic intramural hematoma. *Radiographics*. 2009, 29:791-804. [10.1148/rg.293085122](https://doi.org/10.1148/rg.293085122)
39. Gutschow SE, Walker CM, Martinez-Jimenez S, Rosado-de-Christenson ML, Stowell J, Kunin JR: Emerging concepts in intramural hematoma imaging. *Radiographics*. 2016, 36:660-674. [10.1148/rg.2016150094](https://doi.org/10.1148/rg.2016150094)
40. Patel PJ, Grande W, Hieb RA: Endovascular management of acute aortic syndromes. *Semin Intervent Radiol*. 2011, 28:10-23. [10.1055/s-0031-1273936](https://doi.org/10.1055/s-0031-1273936)
41. Akin I, Kische S, Ince H, Nienaber C: Penetrating aortic ulcer, intramural hematoma, acute aortic syndrome: when to do what. *J Cardiovasc Surg (Torino)*. 2012, 53:83-90.
42. Hayashi H, Matsuoka Y, Sakamoto I, Sueyoshi E, Okimoto T, Hayashi K, Matsunaga N: Penetrating atherosclerotic ulcer of the aorta: imaging features and disease concept. *Radiographics*. 2000, 20:995-1005. [10.1148/radiographics.20.4.g00j101995](https://doi.org/10.1148/radiographics.20.4.g00j101995)
43. Coady MA, Rizzo JA, Hammond GL, et al.: Penetrating ulcer of the thoracic aorta: what is it? How do we recognize it? How do we manage it?. *J Vasc Surg*. 1998, 27:1006-1015.
44. Cho KR, Stanson AW, Potter DD, et al.: Penetrating atherosclerotic ulcer of the descending thoracic aorta and arch. *J Thorac Cardiovasc Surg*. 2004, 127:1393-1399. [10.1016/j.jtcvs.2003.11.050](https://doi.org/10.1016/j.jtcvs.2003.11.050)
45. Mozes G, Gloviczki P, Park WM, Schultz HL, Andrews JC: Spontaneous dissection of the infrarenal abdominal aorta. *Semin Vasc Surg*. 2002, 15:128-136.
46. Manghat NE, Morgan-Hughes GJ, Marshall AJ, Roobottom CA: Multidetector row computed tomography: imaging congenital coronary artery anomalies in adults. *Heart*. 2005, 91:1515-1522. [10.1136/hrt.2005.065979](https://doi.org/10.1136/hrt.2005.065979)
47. Downing SW, Sperling JS, Mirvis SE, Cardarelli MG, Gilbert TB, Scalea TM, McLaughlin JS: Experience with spiral computed tomography as the sole diagnostic method for traumatic aortic rupture. *Ann Thorac Surg*. 2001, 72:495-501.
48. O'Connor CE: Diagnosing traumatic rupture of the thoracic aorta in the emergency department. *Emerg Med J*. 2004, 21:414-419.
49. Richens D, Kotidis K, Neale M, Oakley C, Fails A: Rupture of the aorta following road traffic accidents in the United Kingdom 1992-1999. The results of the co-operative crash injury study. *Eur J Cardiothorac Surg*. 2003, 23:143-148. [10.1016/s1010-7940\(02\)00720-0](https://doi.org/10.1016/s1010-7940(02)00720-0)