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EMERGENCY RADIOLOGY SPECIAL FEATURE: REVIEW ARTICLE

MDCT evaluation of acute aortic syndrome (AAS)

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ABSTRACT

Non-traumatic acute thoracic aortic syndromes (AAS) describe a spectrum of life-threatening aortic pathologies with significant implications on diagnosis, therapy and management. There is a common pathway for the various manifestations of AAS that eventually leads to a breakdown of the aortic intima and media. Improvements in biology and health policy and diffusion of technology into the community resulted in an associated decrease in mortality and morbidity related to aortic therapeutic interventions. Hybrid procedures, branched and fenestrated endografts, and percutaneous aortic valves have emerged as potent and viable alternatives to traditional surgeries. In this context, current state-of-the-art multidetector CT (MDCT) is actually the gold standard in the emergency setting because of its intrinsic diagnostic value. Management of acute aortic disease has changed with the increasing realization that endovascular therapies may offer distinct advantages in these situations. This article provides a summary of AAS, focusing especially on the MDCT technique, typical and atypical findings and common pitfalls of AAS, as well as recent concepts regarding the subtypes of AAS, consisting of aortic dissection, intramural haematoma, penetrating atherosclerotic ulcer and unstable aortic aneurysm or contained aortic rupture. MDCT findings will be related to pathophysiology, timing and management options to achieve a definite and timely diagnostic and therapeutic definition. In the present article, we review the aetiology, pathophysiology, clinical presentation, outcomes and therapeutic approaches to acute aortic syndromes.

INTRODUCTION

Acute aortic syndrome (AAS) is a medical emergency that requires an immediate and accurate diagnosis and treatment.¹ The term AAS includes all of the following: aortic dissection (AD), intramural haematoma (IMH), penetrating atherosclerotic ulcer (PAU) and large unstable aortic aneurysm.^{2–7} All these conditions are associated with a series of signs and symptoms. The most common symptom is chest pain. The grave concern regarding AAS is the possibility of rupture; diagnosis and treatment are vital for patient morbidity and mortality.⁶ Because the symptoms are vague, they can mimic other acute diseases processes. The incidence of AAS is estimated at 2–6/100,000/year⁻¹ with two-thirds of cases affecting males 63 years old and over.^{2–4} Multidetector CT (MDCT) is the modality of choice because it has a sensitivity of 100% and specificity of 98–99%.^{7–9} MDCT is also readily available in most emergency departments and can be performed urgently.

Aortic dissection

In 1760, Dr Frank Nicholls discovered on necropsy an AD in King George II, which resulted in his demise.¹⁰ In the majority

of patients (90%), an intimal disruption is present that results in tracking of blood in a dissection plane within the media.⁶

While the exact mechanism of AD is unclear, it is believed to be most commonly secondary degeneration of the medial layer of the aortic wall (cystic media necrosis), which results in the loss of normal aortic wall compliance and elasticity. It can be accelerated by other conditions such as hypertension and genetic mutations, such as Marfan syndrome, that predispose to premature degeneration of collagen and elastin within the aorta, but can also occur in a normal aorta. Histologically, AD is characterized by an entry intimal tear or primary intimal tear, allowing blood to penetrate and disrupt the aortic media. This subsequently forms a false lumen (FL) parallel to the original aortic true lumen (TL), with the FL causing pressure greater than or equal to the true lumen (Figure 1).^{1,11} The typical tear is transverse and does not involve the entire circumference of the aorta.¹² As the dissection flap is composed of intima and the inner two-thirds of media, intimomedial flap is a more appropriate term. The intima

Figure 1. An acute aortic syndrome drawing. (a) Aortic dissection (AD) is defined as disruption of the aortic intima and inner layer of the aortic media creating an entry intimal tear (black arrow) that enables blood to split the aortic media, resulting in a double-channel aorta. (b) An acute intramural haematoma results from haemorrhage into the arterial media (asterisk), which leads to weakening of the aortic wall. The distinguishing feature of this entity is the absence of the intimal disruption that characterizes classic AD. (c) Penetrating atherosclerotic ulcer is an ulcerating atherosclerotic lesion that penetrates the elastic lamina (star) and is often associated with haematoma formation within the media of the aortic wall. (d) According to Laplace's law, in unstable thoracic aortic aneurysm, wall tension (white arrows) is proportional to the mean vessel radius, and the maximum diameter of the aneurysmal sac and its rapid enlargement rate are the most significant predictive findings of impending rupture. FL, false lumen; TL, true lumen.

is displaced inward, along with any intimal calcifications.^{12,13} The intimomedial layer is cleaved both longitudinally and circumferentially for a variable distance. An entry tear can progress along the aortic lumen in either an antegrade or the less common retrograde direction. As the proportion of media involved in the intimomedial flap increases, the external wall of the FL becomes thinner. This increases the risk of aortic rupture.

Aortic Dissection risk factors, clinical presentation and classification

Risk factors

The most common comorbidity for media layer degeneration and AD is severe arterial hypertension. The constant exposure of the aorta to high blood pressure causes medial disruption and degeneration of the aortic wall. Multiple risk factors are associated with weakening of the aortic media or constant exposure to hydraulic pressure (Table 1).^{2,3,14,15}

Clinical presentation

The symptoms of AD are severe sudden onset of chest or back pain. It is described as a tearing or ripping pain of knife-like or sharp quality in older adults (*i.e.* sixth or seventh decade) with poorly controlled hypertension. There is a wide spectrum of pathological conditions associated with AD because of the variable extent of dissection along the blood vessel (Figure 2).^{6,11} The diagnosis of AD is challenging because there are no biomarkers; thus, imaging is necessary. In 2010, the American College of Cardiology/American Heart Association guidelines proposed

a priori a risk assessment tool based on three groups of information—predisposing conditions, pain features and clinical examination. The proposed scoring system graded these factors from 0 (none) to 3.⁶ The “DISSECT” mnemonic takes several clinical and imaging key factors into account; thus, it can aid in treatment planning (medical, endovascular or open surgical repair) (Table 2).^{16,17}

Classification

AD can be classified according to the extent of dissection (Stanford and DeBakey classifications), to the status of blood flow in the FL (communicating and non-communicating types) or to disease phase (acute, subacute and chronic phases). The preferred Stanford classification, proposed by Dailey et al,¹⁸ is based on the extent of intimomedial flap, rather than the location of the entry tear. The Stanford Type A dissection (more common in autopsy series) involves the ascending aorta, regardless of the site of origin and is a surgical emergency, whereas the Stanford Type B dissection (more common in radiological and surgical series) affects only the descending aorta and is typically treated with medical management.^{5,6,12} Accurate classification is important, as it drives decisions regarding surgical vs non-surgical management. The term “complicated” Type B AD means persistent or recurrent pain, uncontrolled hypertension despite full medication, early aortic expansion, organ malperfusion and signs of rupture (haemothorax, increasing periaortic and mediastinal haematoma) and is usually managed by stent-graft endovascular repair (TEVAR).^{19,20} Imaging and accurate reporting play an essential role in the determination of the therapeutic strategy (Table 3).

Table 1. Risk factors predisposing to acute aortic syndrome

Hypertension
Genetic disease
Marfan syndrome
Loeys–Dietz syndrome
Vascular Ehlers–Danlos syndrome (Type 4)
Turner syndrome
Coarctation of the aorta
Bicuspid aortic valve
Familial thoracic aortic aneurysm and dissection syndrome
Inflammatory vascular disease
Syphilis
Behcet's disease
Takayasu arteritis
Giant-cell arteritis
Aortic wall infection (bacteraemia or extension of adjacent infection)
Iatrogenic factors
Aortic catheterization
Cardiac valve or aortic surgery
Miscellaneous conditions
Crack cocaine or other drug abuse
Chronic corticosteroid or immunosuppressant therapy
Phaeochromocytoma
Polycystic kidney disease
Pregnancy
Traumatic injury
Atherosclerotic disease
Typically in penetrating aortic ulcer

Imaging of aortic dissection

Patients with acute chest pain will almost invariably undergo chest radiography (CXR). This is of minimal utility in AAS (sensitivity of 64% and specificity of 86% for overt AD),^{21,22} apart from its ability to establish or exclude alternate diagnoses. A CXR may show non-specific findings of widening of the aortic contour and mediastinal shadow enlargement; other features may include medially displaced intimal calcification, aortic kinking or opacification of the aortopulmonary window (Figure 3),^{21–23} Most importantly, the CXR cannot be relied upon to definitively exclude acute aortic disease. Up to 20% of patients with AD will have a normal or near normal CXR.²⁴

The diagnosis of AD by standard transthoracic echocardiography is based on detecting intimal flaps in the aorta. Transthoracic echocardiography is restricted in patients with abnormal chest wall configuration, narrow intercostal spaces and obesity, and these limitations are usually overcome by transoesophageal echocardiography (TOE). A major advantage of echocardiography is its

portability, which is useful in patients who are unstable. The disadvantages of TOE are operator dependency, limited acoustic window, innate blind spot (*i.e.* the distal ascending aorta and proximal aortic arch owing to air in the trachea) and inability to visualize the entire aorta.^{4–6}

A major advantage of MDCT over TOE is the ability to look beyond the aorta for alternative diagnoses such as pulmonary embolism.²⁵ Because of its long acquisition time and inability to monitor patients who are acutely unwell in the MRI suite, MRI is mainly used for the follow-up of chronic AD.^{24,26} Therefore, the choice of imaging modality for evaluating AD, considering the excellent accuracy of all modalities, should adapt to local expertise and be individualized according to the specific clinical situations.

Multidetector CT technique in the assessment of aortic dissection

The advantage of MDCT is in the acquisition of isovolumetric three-dimensional information without loss of spatial resolution in a single breath-hold. The exact protocol for each patient will vary, based upon the vendor and patient body habitus. At our institution, the evaluation of an AD is performed with triphasic CT angiography (CTA), which consists of unenhanced, arterial and then venous phase injection.^{27,28} Pre-contrast low-dose CT scan is performed with thick collimation and its coverage is from the lung apex to the upper abdomen. Then, a bolus-tracked CT angiogram is performed from the lung apex to the groin using 60–120 ml of 370 mgI ml⁻¹ iodinated contrast material (CM) delivered at a rate between 3 and 6 ml s⁻¹, according to patient body weight, to achieve a target opacification of the aorta of 250 HU (Table 4). Delayed (venous) scans 1–2 min after injection are obtained selectively, to assess for late filling of an FL lumen and to clearly depict abdominal organ malperfusion in AD or contrast extravasation from aortic rupture. Contrast media are administered using automated pump injectors to facilitate multiphasic injections, with sequential administration of CM and normal saline, to enable homogeneous concentrated bolus of CM through the aorta.²⁹ The required amount of CM for CTA may be better predicted by weight-based calculations (average iodine concentration accounting for a flow rate of 1.0–1.6 g s⁻¹) than fixed-dose estimation.²⁸ Oral contrast is not necessary unless gas is identified within the endovascular or perivascular soft tissue, or if there is a suspected bronchial/oesophageal vascular fistula. The images, acquired from software-assisted centreline reconstructions, can be used to either generate reliable and reproducible measurements or carefully assess changes in the luminal diameter and contours.

Streak artefacts are generated by high-attenuation prior surgery material, high-contrast interfaces and cardiac motion. Several periaortic structures, such as origins of the aortic arch vessels, left brachiocephalic, superior intercostal and pulmonary veins, may be misinterpreted as double lumina or intimal flaps.^{29–32} To avoid diagnostic errors, a contrast-enhanced thoracic acquisition should be always obtained with electrocardiogram (ECG) gating to reduce motion artefacts, especially if there is concern of complications involving the aortic valve, aortic sinus, valve plane, aortic root or proximal ascending aorta (Figure 4).³³ ECG gating synchronizes the CT scan and the cardiac cycle.

Figure 2. Wide spectrum of pathological conditions related to aortic dissection and some reported frequencies in the Stanford classification (A = Stanford Type A; B = Stanford Type B). Depending on the length of the dissection flap, a wide spectrum of clinical symptoms may occur. Actually, lethal complications do not arise from the intimal tear itself, but rather from the subsequent course taken by the dissecting aorta, such as vascular compromise (dilatation, stenosis or occlusion) or aortic rupture. N/A, not assigned; SVC, superior vena cava.

A	B		A	B
Sudden death			Hoarseness, dysphagia	
< 10% Cerebral ischemia	<5%		Upper limb ischemia	
Mediastinal hematoma			Intrathoracic bleeding	
SVC syndrome			80% Severe chest pain	70%
Angina			40% Severe back pain	70%
10-15% Myocardial infarction	10%		Pulse deficit	
<20% Cardiac tamponade	N/A		Pressure difference between the two extremities	
40-75% Aortic valve regurgitation			Paraplegia	
Aorta aneurysm formation or rupture			Retroperitoneal hematoma	
Abdominal cavity bleeding			<20% Renal failure	
< 5% Intestinal tract ischemia			<10% Lower limb ischemia	<10%
Paralytic ileus			Disseminated intravascular coagulation syndrome	
Systemic inflammatory response syndrome				

A prospective gating is performed only during a desired phase of cardiac cycle, which is during left ventricle diastole, because the proximal aorta has less motion. In a retrospective scan, the CT scanner is on for the entire cardiac cycle, allowing increased scope for correction of artefacts from dysrhythmias or motion, but comes at the cost of increased radiation exposure.

Since retrospective ECG gating is associated with a significant increase in radiation dose, various dose reduction techniques may be used, such as prospective ECG triggering, ECG-based tube current modulation, automatic exposure control, lower peak kilovoltage and iterative reconstruction algorithms.³³ Various post-processing techniques such as multiplanar reformation, maximum intensity projection and volume rendering help facilitate understanding of

complex aortic pathology and to expedite communication with the surgeons and the attending physicians.

Typical and atypical MDCT findings of classic double-barrel AD

Although typical AAS cases demonstrate characteristic imaging features of each disease, imaging findings may also

Table 3. The multidetector CT radiology “perfect” report in aortic dissection (AD)

AD extent and involvement of aorta (<i>i.e.</i> , Stanford classification)
Site of entry tear (crucial for TEVAR procedure) and intimomedial flap course
Re-entry tear/s; differentiation between the TL and FL and their diameters at proximal/distal landing zones, at entry and at minimum; degree of compression of TL by FL; FL diameter as a predictor for aortic rupture; FL mural thrombus
Aortic valve and side branch (originating from TL/FL) involvement (coronary, carotid, subclavian, celiac, superior mesenteric, inferior mesenteric, renal and iliac artery, femoral and axillary arteries for cannulation for potential cardiopulmonary bypass)
Evidence of end-organ malperfusion (one-third mortality by organ failure)
Morphology and diameter of the aorta along with the patency, size and tortuosity of the iliac and femoral arteries (useful for TEVAR/EVAR planning)
The presence/absence of aortic rupture

TEVAR, endovascular repair; FL, false lumen; TEVAR; stent-graft endovascular repair; TL, true lumen.

Table 2. The DISSECT classification

Duration of disease (acute for symptoms <14 days; subacute = 14 days to 3 months; chronic for >3 months)
Intimal tear location (for example a Type A AD with an entrance tear in the descending aorta distal to the subclavian artery)
Size of the dissected aorta (in millimetres) and growth also in follow-up examinations
Segmental extent (<i>i.e.</i> thoracic only vs thoracoabdominal or abdominal aorta)
Clinical complications of the dissection (complicated vs uncomplicated AD)
Thrombus (thrombosis) within the aortic FL. Complete FL thrombosis is the key to long-term clinical success and long-term survival

AD, aortic dissection; FL, false lumen.

Figure 3. Chest radiography (CXR) in acute aortic syndrome (AAS). (a) Anteroposterior view and (b) close-up view of displaced calcification >1cm on the aortic knob (black arrows). (c) Lateral view of the porcelain aorta. (d) Lateral view of the proximal descending aortic aneurysm. (e) Posteroanterior and (f) lateral views show an aortic isthmus calcified pseudoaneurysm [white arrow in (e) and black arrow in (f)]. CXR is, however, only of limited value for diagnosing an AAS, particularly as a normal aortic silhouette is not sufficient to rule out the presence of an aneurysm or dissection of the ascending aorta.

overlap between different entities, especially when the process is dynamic and evolving; these transitional and overlapping features, both clinical and pertaining to imaging findings,

are sometimes definable as atypical and have led to misconceptions and controversies concerning the disease concept of AAS.^{34–37}

Table 4. Multidetector contrast-enhanced (CE) CT acquisition parameters (64-section multidetector CT)

Parameters	CE spiral CT	CE ECG-gated CT
Section thickness (mm)	0.5–0.625	0.5–0.625
Increment (mm)	0.4	0.4
Tube potential (kV)	100–120	100–120
Collimation (detectors × mm)	64 × 0.625	64 × 0.625
Pitch	About 1	BPM dependent
Rotation time (s)	0.5	Minimum
Field of view (mm)	210–260	210–260
Matrix (pixels)	512 × 512	512 × 512
Non-ionic CM ^a	50–120 ml (plus saline chaser)	50–120 ml (plus saline chaser)
Biphasic injection protocol	Yes	Yes
Injection rate (ml s ⁻¹)	3–6 (18–20 G preferably in the right arm)	3–6 (18–20 preferably in the right arm)
ROI	Distal AA/arch (bolus triggering)	Distal AA/arch (bolus triggering)
Study plan	Diaphragm to the groin	Lung apices to the diaphragm

AA, aortic aneurysm; BPM, beats per minute; CM, contrast material; ECG, electrocardiogram; G, gauge; ROI, region of interest.

^aHigh concentration (≥ 350 mg l ml⁻¹) CM.

Figure 4. The advantages of electrocardiogram (ECG)-gating CT imaging as a powerful tool to differentiate true intimomedial flaps from aortic pulsation artefacts. (a, b) Non-gated multidetector CT (MDCT) and (c, d) prospectively ECG-gated MDCT in a patient transferred to our cardiothoracic centre with suspected aortic dissection on the initial study (white arrows). The suspected dissection flap was confirmed to be cardiac motion artefact by its absence on the subsequently performed prospective ECG-gated study.

Classic multidetector CT findings of aortic dissection. The pre-contrast phase is needed to evaluate for displaced intimal calcifications which are suggestive of AAS, IMH and high-density blood in the pericardium, pleural space or mediastinum, indicating aortic rupture (Figure 5).

Typical MDCT findings of AD are direct visualization of media-intima entrance primary tear from TL to FL, as a distinct intimomedial flap defect. The TL is typically smaller and more intensely opacified than the FL in the early angiographic phase owing to higher pressure and faster mixing with blood. The FL is crescent shaped, with acute angles (beak sign = an acute angle between the dissection flap and the outer wall of the FL; the space formed by the acute angle could be filled with a high-attenuation material, contrast-enhanced blood, or a low-attenuation material in chronic dissections, haematoma) between the detached intima and the aortic wall (Figure 6).^{1,23,25,34-37}

Contrast enhancement between the arterial and venous phase is required to differentiate between the TL and partially thrombosed FL. In some cases, media-intima separation is not complete, and cobwebs or tendrils of the media layer (cobweb sign) persist between the intima and media, generally over short segments of the dissection. The TL is identified by tracing back or forth from an uninvolved portion of the aorta; this is not easy, if the aortic root is involved proximally or the dissection extends into the iliac vessels.³⁸

The intimomedial rupture sign is also helpful to distinguish the TL from FL. This sign refers to the discontinued ends of the

intimomedial flap at the site of the entry tear that point towards FL (Figure 7).³⁹ It indicates the direction of blood flow through entry tear from TL to FL. However, the direction of blood flow through the entry tear is bidirectional or reversed, depending on the cardiac phase. Intraluminal thrombus is more frequently encountered in the FL (46%) rather than TL (6%) because of a slow flow in the acute setting.^{36,38}

The most common locations of entry tear and maximum hydraulic stress are:

- the right lateral wall of the ascending aorta; the FL extends distally, in a spiral fashion, along the left posterolateral wall of the descending aorta
- the descending aorta just distal to the left subclavian artery, where the shearing stress against the aortic wall generated by hypertensive blood flow is maximal.⁶ The FL, adventitially bound, represents the blood-filled space between the dissected layers of the aortic wall. Fenestrations within the intimal flap downstream, typically occurring where branch ostia are cleaved off by the dissecting process, lead to sites of re-entry for flow into the TL, thus maintaining FL patency.

A re-entry tear can occur within the descending thoracic aorta, abdominal aorta or iliac arteries. It is not frequently identified because it typically consists of a minute defect.^{34,38,40}

Curved multiplanar reformatted images show entry and re-entry tears more intuitively (Figure 8). The differentiation between TL and FL is difficult, particularly in cases with involvement of the

Figure 5. The importance of multidetector CT unenhanced phase in acute aortic syndrome. (a) Displaced intimal calcification (arrow) along the true luminal aspect of the flap in a patient with Stanford Type B dissection; (b) spontaneous high-attenuation intimomedial flap (white arrows) in a right-side aortic arch; (c) diffuse circumferential calcifications of the ascending aorta (porcelain aorta) unable to cross the clamp or cannulate; (d) calcified intimomedial flap (arrows) in a left-side aortic arch extending into an “arteria lusoria”, and the accepted name of the aberrant retroesophageal right subclavian artery is not originating from the brachiocephalic trunk but directly from the aortic arch; (e) crescent of high attenuation (white arrows) within the wall of the aorta by acute false lumen thrombosis; (f) high-density intramural haematoma (black arrow); this is difficult to appreciate and easily overlooked on the arterial phase imaging; (g) high-density blood (spot) in the left pleural space and in the right mediastinum in aortic rupture. The different locations of intrathoracic haematoma may suggest the site of rupture.

aortic root and especially in those with circumferential dissection involving the root.³⁹ The celiac, superior mesenteric and right renal arteries typically emanate from the TL, and the left renal artery arises from the FL, but variations can occur.²⁵ Depending on the circumference and pressures involved, dilation of the false channel may diminish the TL diameter (compared with that of the FL) until TL thrombosis and collapse; this can cause malperfusion of visceral or peripheral arteries (Figure 9). According to Laplace's law, a large FL is more likely to be associated with aortic rupture than a small one.³⁷

Atypical findings

(a) The flap can have various configurations (Figure 10).^{7–43} A marked curvature of a mobile dissection flap is characteristic of acute dissections, whereas a rather flat and fixed appearance of an immobile and thickened flap (fibrosis leading to reduced flap mobility) is characteristic of a chronic dissection.³⁷ A circumferential intimomedial flap is caused by the complete dissection of the intima; the TL takes on a cylindrical or filiform shape, and this results in an intimointimal intussusception, producing a “windsock” appearance.^{44,45} In patients with a 360° intimal tear in the ascending aorta, the forward

aortic blood flow may push the curled-up intima against the great vessel origins in the aortic arch, compromising cerebrovascular blood flow. On the rare occasion, massive aortic valve regurgitation can occur owing to a circumferential intimal disruption and prolapse of the cylinder-shaped intimal flap into the left ventricle.⁴⁶ Rarely, flap continuity is not clearly visible on axial scans. In such cases, volume rendering reconstructions may be of value, showing greater sensitivity (Figure 11).⁴⁷

- (b) Rarely, a three-channel AD or an aorta with several false channels can be seen, if a secondary dissection occurs within one or more of the channels, with a resulting intimal flap giving rise to the Mercedes-Benz sign or other complex and bizarre morphologies. Pain recurrence in patients with AD should suggest the possibility of a three-channel dissection (Figure 12).^{25,35–37} This is more common in Marfan syndrome.
- (c) Limited intimal tear (Class 3), also known as “incomplete tear” or “subtle/discrete dissection”, is a rare variant of AD qualified as a subtle, discrete dissection, in which the limited stellate or linear intimal tear is associated with exposure of the aortic media or adventitial layer and focal eccentric bulge

Figure 6. Diagram and multidetector CT (MDCT) axial image of Stanford Type A aortic dissection (AD) typical findings. Axial MDCT scan shows the course of the intimomedial flap in ascending and descending aorta and differentiation between true lumen (TL) and false lumen in a 68-year-old male who presented with acute aortic pain. Note that TL is usually smaller and more intensely and early opacified because of the higher velocity of blood, surrounded by calcifications (if present).

at the tear site.^{5,48} It has no extensive progression, significant separation of medial layers and an intimomedial flap and does not result in a second flow channel, as seen in classic AD or an evident IMH. Although, the incidence of limited intimal tear is not known and is underestimated because of general unfamiliarity with this dissection variant, and imaging techniques such as CT, MRI or TOE may fail to detect this type of dissection.

On MDCT, limited intimal tear is described either as an eccentric one-sided bulge or as a minor contour abnormality of the aortic wall that may be the only imaging finding of this lesion. Asymmetrical bulges are sometimes accompanied by haemorrhagic content within the aortic wall on unenhanced MDCT imaging and linear filling defects from subtle undermined edges on MDCT angiography (Figure 13).⁴⁷

Multidetector CT findings of end-organ malperfusion syndromes

On cross-sectional imaging, impaired perfusion of end organs can be due to four mechanisms (Figure 14):

- (1) static = continuing dissection in the feeding artery may induce ostial or proximal stenosis (usually treated by stenting); the flap might be perpendicular or parallel to the trajectory of the side branch artery without or with compression of the TL by the FL
- (2) dynamic = dissection flap hanging in front of the ostium like a curtain (usually treated with fenestration)
- (3) a mixed type, corresponding to the compression of the TL caused by FL overpressure associated with extension of the flap into the visceral artery
- (4) ostial disconnection, corresponding to ostial avulsion of the visceral artery origin by dissection with a clean tear, persistent

cobwebs or complete avulsion with the residual intimal flap projecting in the branch, which may cause a stenosis.^{49–52}

Depending on the size of the entry tear in the artery, ischaemic organ changes may occur. If the entry tear is small, ischaemia of

Figure 7. The intimomedial rupture sign refers to the discontinued ends of the intimomedial flap at the site of the entry tear that point towards false lumen (arrow). However, the direction of blood flow through the entry tear can be bidirectional or reversed depending on the cardiac phase.

Figure 8. Multidetector CT axial and oblique sagittal and coronal multiplanar reformation images of entry and re-entry tears. (a) An entry tear (arrow) in the aortic root; (b) an entry tear distal to the left subclavian artery, recognized as a pinhole (arrow); (c) a large entry tear (arrow) in the aortic arch. (d) A re-entry tear (arrow) at the mediiodistal descending thoracic aorta; (e) a re-entry tear (white arrow) at the right common iliac artery; (f) a re-entry tear (arrow) at the medioabdominal aorta. Note that the jet phenomenon is due to the high velocity of the blood.

the organ will be due to hypoperfusion by either the TL or FL, inducing compression of the TL by pressure into the FL. Conversely, a large entry tear in the flap will not lead to vascular compression. Furthermore, the perfusion disturbance can be intermittent if caused by a dissection flap prolapse, or persistent in cases of obliteration of the organ arterial supply by FL expansion.^{49,50}

Prognostic factors and follow-up multidetector CT
A large entry tear (>10 mm) is a strong predictor of poor mortality and surgical or TEVAR intervention.⁵³ Patients with clear overall TL compression have a higher risk for rapid FL enlargement and further aortic complications. Major predictors of complications after acute phase during follow-up

Figure 9. Diagram and multidetector CT axial images of morphological features of (a, b) pressure competition between lumens until true lumen (c) collapse. f, false lumen; t, true lumen.

Figure 10. The multiple configurations of the flap. (a) Chronic Type A aortic dissection; a flat and fixed appearance of an immobile and thickened flap (fibrosis leading to reduced flap mobility) is more characteristic of a chronic dissection. (b) An aortic arch circumferential flap with oval morphology ("windsock" sign); (c) an abdominal aorta small circumferential flap; (d) unenhanced and (e, f) enhanced multidetector CT axial scans showing a flap with a very atypical shape.

are secondary dilatation of the aorta (a descending aorta diameter >45 mm or annual growth >5 mm) and a persistently patent FL.

Intramural haematoma

Aortic IMH is defined as a haematoma within the media of the aortic wall due to a spontaneous rupture of the vasa vasorum. In the absence

Figure 11. A 67-year-old male presenting to the emergency department with intense upper back pain. Multidetector CT (MDCT) axial images showing an (a) intimal flap (arrow) at the level of the aortic root; (b) absence of the flap at the level of the distal ascending aorta and (c) partial dissection flap (arrow) at the middle aortic arch associated with a small haemopericardium. (d) Volume-rendered MDCT reconstruction clearly demonstrating a flap (arrow) propagating from the aortic root to the mid-aortic arch. At surgery, an intimointimal aortic intussusception and complete flap tear was seen (Type A aortic dissection), and graft replacement of the ascending aorta and concomitant proximal hemiarch was performed.

Figure 12. A 52-year-old male complaining of acute chest pain for 2 days. (a, b) Axial and (c) sagittal maximum intensity projection (MIP) multidetector CT scans showing a three-channel dissection in the aneurysmal proximal descending aorta with an intimomedial tear (arrows) entering the false lumen from the true lumen, clearly demonstrated in the sagittal MIP reconstruction. (d) A volume-rendered image giving a suggestive and comprehensive view of the three-channel dissection.

of an FL, an intimal tear can develop. This concept is outdated, and modern imaging technology can often detect small communications between the aortic lumen and the haemorrhage within the wall.⁵⁴

Current opinion about IMH is that it can be a variant or a precursor of AD with a small intimal defect and thrombosed FL without re-entry tear.¹¹ This type was also defined as

Figure 13. Acute aortic syndrome due to a limited intimal tear (Svensson class 3 variant of aortic dissection) with intramural haemorrhagic content in a 58-year-old male with sudden onset of chest and back pain. (a) Contrast-enhanced multidetector CT (MDCT) axial image demonstrating a linear filling defect with subtle undermined edges (arrow) and eccentric medial one-sided bulge of the ascending aorta without a clear intimomedial flap or false lumen; this was initially diagnosed as a focal sinus projection. (b) Slab MIP coronal MDCT reconstruction image demonstrating the “eccentric one-sided bulge” (arrow) of the aortic left wall along the ascending aorta. (c) Three-dimensional volume-rendered oblique coronal reconstruction confirms the aortic bulging.

Figure 14. Multidetector CT of end-organ ischaemia. (a) A 66-year-old male with Type B aortic dissection (AD) and mesenteric malperfusion syndrome [right colon ischaemia (arrow)] treated with balloon fenestration. (b, c) A 58-year-old male with Type B AD and upper abdominal pain. Axial images show that (arrow in b) the dissection extended to the celiac trunk and hepatic artery within the porta hepatis with false lumen thrombosis and ischaemic hypodensity of II and III hepatic segments (arrows in c). In mesenteric ischaemia, clinical manifestation is frequently insidious; abdominal pain is often non-specific, and patients may be painless in 40% of cases; consequently, the diagnosis is often frequently too late to save the bowel and the patient.

“thrombosed-type acute AD”, where the separation of the aortic wall layers is filled with thrombus rather than free-flowing blood in what would otherwise be the FL of a classic dissection.

A circular or crescent-shaped thickening of >5 mm of the aortic wall in the absence of detectable blood flow suggests an IMH. Owing to the similarities with AD, IMHs are also classified

Figure 15. (a, b) Diagram and axial unenhanced and (c, d) diagram and enhanced multidetector CT (MDCT) scans of intramural haematoma (IMH). The distinguishing feature of this entity is the absence of the intimal disruption that characterizes classic aortic dissection. (b) Unenhanced MDCT axial image showing a crescentic hyperattenuating thickening of the aortic wall (circle). (d) Enhanced MDCT axial image depicting a smooth, non-enhancing, crescentic region of aortic wall thickening (circle) without a spiralling intimal flap.

Figure 16. Emergent pre-surgical multidetector CT (MDCT) in a rapidly progressive chronic aortic dissecting aneurysmal disease in a young child (<1 year of age) with Loeys-Dietz syndrome and transthoracic echocardiography diagnosis of ascending aorta aneurysm. (a) Axial unenhanced MDCT image, (b, c) axial early and delayed enhanced MDCT images, (d) maximum intensity projection sagittal MDCT reconstruction image and (e) volume-rendered MDCT post-processing image showing chronic dissecting aneurysmal dilatation of the root and ascending aorta. Hypodense halo at unenhanced MDCT refers to a chronic mural haematoma and excludes an acute aortic dissection. Note the associated pectus deformity (excavatum). Successful valve-sparing aortic root and ascending aorta replacement was performed. (f) Photograph of the resected specimen showing chronic dissection.

according to the Stanford classification.⁵⁵ IMH consists of 5–15% of AAS.

Typical MDCT findings of intramural haematoma

A subintimal hyperdense (60 ± 15 HU) crescent is the most common and important finding in pre-contrast images.⁷ The aortic lumen is patent: no intimal flap or aortic wall enhancement can be seen. IMHs have a smooth lumen–wall interface, with visualization of the subintimal semi-circular or curvilinear calcifications (Figure 15). The absence of obvious communication between the TL and FL explains the absence of flow on colour Doppler flow and the lack of enhancement on CT or MRI.

In acute IMH, imaging should always include a thorough attempt to localize a primary (micro) entry tear, which is very often present and directs the course of treatment, especially when considering TEVAR.⁵⁶ One way to differentiate IMH from a thrombosed FL of AD is that IMH maintains a constant circumferential relationship with the aortic wall, whereas an AD tends to spiral longitudinally (Figure 16).

Details required from imaging are localization and extent of aortic wall thickening, coexistence of atheromatous disease

(calcium shift) and the presence of small intimal tears.⁵ Patients should be closely followed up in the first 30 days.

If luminal dilatation, penetrating ulcer, enlargement of the IMH or dissection occur, surgical or endovascular treatment should be considered.^{55,56}

Atypical MDCT findings of intramural haematoma

The findings of a dissection-variant IMH are as follows:

- A small intimal disruption defined as “primary intimal tear” or “ulcer-like projection/lesion” is a localized blood-filled pouch protruding from the TL into the thrombosed FL of the aorta (Figure 17). This finding is an indicator of the formation of a flow channel between the two lumens, which can become an AD or can heal and resolve. An ECG-gated CTA improves the sensitivity for detecting small perforations between the TL and FL in IMH.⁵⁷
- Intramural blood pools or aortic branch artery pseudoaneurysms (BAP) are localized CM-filled pools inside the IMH on post-contrast MDCT images secondary to the damage caused by IMH propagation across the origin of the aortic branch (bronchial, intercostal, intercostobronchial, pericardial, lumbar) artery that is partially or completely torn (Figure 18). The communication between the contrast

Figure 17. Ulcer-like projections/primary intimal tear in the follow-up multidetector CT (MDCT) in a 74-year-old male 3 weeks after acute onset of Type B intramural haematoma and chest/back pain. No evidence of extraluminal contrast material was seen at early enhanced MDCT, and the haematoma was treated with conservative management. (a) Axial, (b) coronal and (c) sagittal MDCT reconstruction images showing an enhancing ulcer-like projection (arrows), a finding suggestive of a new intimal tear in the distal arch/proximal descending aorta. This patient was finally treated by surgical therapy.

medium-filled pool and the TL is absent or is a tiny orifice <2 mm in diameter. A possible distal connection with the aortic branch is often noted.^{58,59} Intramural blood pools/BAPs may propagate along the aortic circumference and in a craniocaudal direction, from a few millimetres up to several centimetres, with a possible confluence of BAP arising from aortic branch arteries adjacent to each other, giving a “Chinese ring-sword” sign.⁶⁰ The clinical course of BAP is usually benign and self-limited and not associated with a poor outcome; the majority of BAPs spontaneously regress in size over time and/or completely disappear on follow-up MDCT.^{60–62}

Multidetector CT findings of penetrating atherosclerotic ulcer

In PAU (estimated incidence between 2.3% and 7.6%), an atherosclerotic plaque ulcerates and disrupts the internal elastic lamina. This suggests a diseased intima and occurs more commonly in the descending thoracic aorta ($>90\%$) of elderly individuals with multiple risk factors for atherosclerosis and associated comorbidities of atherosclerotic disease.^{5,6,11,63} There is currently no clear cut-off for PAU diameter (depth) or neck diameter that warrants treatment; in one publication, a depth of >20 mm or a neck >10 mm was associated with higher complication rates.^{64,65}

On MDCT, localized ulceration penetrating through the aortic intima is the characteristic finding. There is also an outpouching of the outer aortic contour, which differentiates uncomplicated

atheromatous ulcer from PAU. A PAU is diagnosed by demonstrating a focal contrast-filled outpouching of the aortic wall with jagged edges usually in the presence of extensive aortic atheroma; unenhanced MDCT frequently (about 80%) shows high-density haematoma surrounding the ulceration (Figure 19).^{7,25} Invasive treatments such as surgery and stent grafting are indicated in acute unstable or symptomatic cases (rapid expansion of the aortic diameter, persistent or recurrent pain, development of pseudoaneurysm, pericardial effusion, bloody pleural effusion or distal embolization), but because they occur mainly in patients with severe comorbidities, course observation including periodical evaluation using imaging techniques is recommended in asymptomatic or chronic cases.^{66,67}

Multidetector CT findings of unstable aortic aneurysm

A true aortic aneurysm (AA) is defined as a dilatation of the aorta that contains all layers of the aortic wall and usually involves the entire circumference.^{68,69}

Unstable thoracic AA is a part of AAS, and if it is symptomatic, it is clinically indistinguishable from AD, IMH or PAU.^{9,34} A thoracic AA is unstable, if it shows rapid enlargement and/or signs of impending rupture:

- size increases especially with a rapid enlargement rate (>10 mm year⁻¹)
- focal discontinuity of the intimal wall calcification (missing calcium sign), especially if the patient lumen tapers towards the focal discontinuity (“tangential calcium sign”)

Figure 18. Intramural blood pools/branch artery pseudoaneurysms (BAP) in intramural haematoma (IMH) in a 61-year-old patient with acute chest pain who is hypertensive. (a) Maximum intensity projection (MIP) axial and (b) MIP coronal multidetector CT (MDCT) reconstruction images showing pseudoaneurysm (arrow) of the left intercostal artery at T9 level in the context of the thoracoabdominal IMH (arrow). (c) Enhanced MDCT axial image obtained 2 months later showing a partial BAP regression. (d-f) Enhanced axial MDCT images showing BAP as localized island-like contrast material-filled pools or collections (arrow), isodense with the aortic lumen and typically located along the non-pleural circumference of the aorta at the origin of the aortic branch arteries, inside the IMH.

- eccentric shape of the aortic lumen
- hyperattenuating crescent rim sign or “crescent sign”, seen at unenhanced MDCT, as a localized usually curvilinear zone

with higher attenuation in the thrombus, caused by fresh blood that first insinuates itself into the mural thrombus and later penetrates the aortic wall

Figure 19. Multidetector CT (MDCT) of penetrating aortic ulcer (PAU) in a 72-year-old male with sudden onset of chest pain; (a) enhanced MDCT axial scan showing a small penetrating ulcer in the proximal descending thoracic aorta (arrow). (b) Enhanced MDCT axial scan showing another flat penetrating ulcer (arrow) with associated intramural haematoma (IMH) (white circle). (c) Enhanced MDCT axial scan showing a small penetrating ulcer in the descending thoracic aorta with a focal IMH. (d) MDCT volume-rendered reconstruction showing a PAU of the isthmus lesser curvature (arrow).

Figure 20. Unstable thoracic aorta aneurysm. (a) Unenhanced and (b) enhanced multidetector CT (MDCT) axial images showing focal discontinuity of the intimal calcifications (arrows) or “missing calcium sign”; this finding should also be reported, as it is a sign of impending rupture, especially if the intimal calcification points away from the aneurysm (“tangential calcium sign”). Enhanced MDCT axial images demonstrating a (c) focal saccular, (d) triangular and (e) serpiginous active extravasation (arrow) of the contrast material into the thrombosed portion of thoracic aortic aneurysm (thrombus fissuration).

- thrombus fissuration, *i.e.* penetration of CM (bleeding) through longitudinal, transverse or oblique fissures, which directly communicates with the lumen and represents fissures or dissections between layers of the intraluminal low-attenuating thrombus with increasing wall tension (Figure 20)
- draped aorta sign (unidentifiable posterior aortic wall and the posterior aorta conforming to the contours of the neighbouring vertebral body)
- periaortic stranding.^{68,70–73}

The detection of these findings advocates urgent treatment (early surgery or TEVAR). Patients with contained rupture should be managed with permissive hypotension to prevent a free rupture and keep the patients stable until treatment.⁶⁹

Future directions

Advances in MDCT hardware (wide detector panel, gantry rotation speed reduction and new detector composition and structure) and software post-processing evolution (automatic CAD of vessel stenosis, three-dimensional modelling, calcification subtraction and enhancing) are opening a new era, which will improve patient safety (reduction of dose and CM), reduce cost and improve patient care in this group of patients.^{74–81} With better understanding of predisposition and genetic risk, acute AD might soon become predictable and, to some degree, preventable by new biomarkers. The description of any given dissection will

be individualized by addressing specific features rather than using simple classification systems of the past. The ongoing advancement of endovascular techniques (fenestrated and branched stent grafts) to the most challenging segment of the aorta (ascending and aortic arch) will revolutionize the treatment of aortic disease.

Summary

AAS comprises interrelated emergent aortic conditions with a similar and overlapping clinical presentation. Recent advances in imaging and therapeutic techniques have further emphasized the importance of early diagnosis of AAS, which continues to be crucial to survival. Immediate diagnostic imaging (TOE and MDCT) plays a pivotal role in management in the emergency setting. To minimize diagnostic error, the radiologist should be familiar with the spectrum of clinical presentations for AAS, the selection and optimization of imaging techniques, as well as with the key concepts behind the common and uncommon imaging features encountered. Technological, biological and therapeutic advancements have already led to an important new clinical paradigm, a multidisciplinary team is necessary to provide optimal outcomes for these patients.

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REFERENCES

1. Vilacosta I, Román JAS. Acute aortic syndrome. *Heart* 2001; **85**: 365–8. doi: <http://dx.doi.org/10.1136/heart.85.4.365>
2. Howard DP, Banerjee A, Fairhead JF, Perkins J, Silver LE, Rothwell PM; Oxford Vascular Study. Population-based study of incidence and outcome of acute aortic dissection and premorbid risk factor control: 10-year results from the Oxford Vascular Study. *Circulation* 2013; **127**: 2031–7. doi: <http://dx.doi.org/10.1161/CIRCULATIONAHA.112.000483>
3. Olsson C, Thelin S, Ståhle E, Ekblom A, Granath F. Thoracic aortic aneurysm and dissection: increasing prevalence and improved outcomes reported in a nationwide population-based study of more than 14,000 cases from 1987 to 2002. *Circulation* 2006; **114**: 2611–8. doi: <http://dx.doi.org/10.1161/CIRCULATIONAHA.106.630400>
4. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. *JAMA* 2000; **283**: 897–903. doi: <http://dx.doi.org/10.1001/jama.283.7.897>
5. Erbel R, Aboyans V, Boileau C, Bossone E, Bartolomeo RD, Eggebrecht H, et al. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC). *Eur Heart J* 2014; **35**: 2873–926. doi: <http://dx.doi.org/10.1093/eurheartj/ehu281>
6. Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VE, Casey DE Jr, 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: executive summary. *J Am Coll Cardiol* 2010; **55**: 1509–44. doi: <http://dx.doi.org/10.1016/j.jacc.2010.02.010>
7. Chiu KW, Lakshminarayan R, Ettles DE. Acute aortic syndrome: CT findings. *Clin Radiol* 2013; **68**: 741–8. doi: <http://dx.doi.org/10.1016/j.crad.2013.03.001>
8. Yoo SM, Lee HY, White CS. MDCT evaluation of acute aortic syndrome. *Radiol Clin North Am* 2010; **48**: 67–83. doi: <http://dx.doi.org/10.1016/j.rcl.2009.09.006>
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**: 561–71. doi: <http://dx.doi.org/10.1007/s13244-012-0195-7>
10. Nicholls F. Observations concerning the body of His Late Majesty. *Philos Trans Lond* 1761; **52**: 265–75. doi: <http://dx.doi.org/10.1098/rstl.1761.0052>
11. JCS Joint Working Group. Guidelines for diagnosis and treatment of aortic aneurysm and aortic dissection (JCS 2011): digest version. *Circ J* 2013; **77**: 709–828.
12. Nienaber CA, Powell JT. Management of acute aortic syndromes. *Eur Heart J* 2012; **33**: 26–35b. doi: <http://dx.doi.org/10.1093/eurheartj/ehs186>
13. Braverman AC, Thompson RW, Sanchez LA. Diseases of the aorta. In: Bonow RO, Mann DL, Zipes DP, Libby P, eds. *Braunwald's Heart disease*. 9th edn. Philadelphia, PA: Elsevier Saunders; 2012. pp. 1309–1337.
14. Westerland O, Frigiola A, Robert L, Shaw A, Blakeway L, Katsanos K, et al. Vascular manifestations of syndromic aortopathies: role of current and emerging imaging techniques. *Clin Radiol* 2015; **70**: 1344–54. doi: <http://dx.doi.org/10.1016/j.crad.2015.08.008>
15. MacCarrick G, Black JH III, Bowdin S, El-Hamamsy I, Frischmeyer-Guerrero PA, Guerrero AL, et al. Loeys–Dietz syndrome: a primer for diagnosis and management. *Genet Med* 2014; **16**: 576–87. doi: <http://dx.doi.org/10.1038/gim.2014.11>
16. Rogers AM, Hermann LM, Booher AM, Nienaber CA, Williams DM, Kazerooni EA, et al; IRAD Investigators. Sensitivity of the aortic dissection detection risk score, a novel guideline-based tool for identification of acute aortic dissection at initial presentation: results from the international registry of acute aortic dissection. *Circulation* 2011; **123**: 2213–18. doi: <http://dx.doi.org/10.1161/CIRCULATIONAHA.110.988568>
17. Dake MD, Thompson M, van Sambeek M, Vermassen F, Morales JP; DEFINE Investigators. DISSECT: a new mnemonic-based approach to the categorization of aortic dissection. *Eur J Vasc Endovasc Surg* 2013; **46**: 175–90. doi: <http://dx.doi.org/10.1016/j.ejvs.2013.04.029>
18. Daily PO, Trueblood HW, Stinson EB, Wuerflein RD, Shumway NE. Management of acute aortic dissections. *Ann Thorac Surg* 1970; **10**: 237–47. doi: [http://dx.doi.org/10.1016/S0003-4975\(10\)65594-4](http://dx.doi.org/10.1016/S0003-4975(10)65594-4)
19. Grabenwöger M, Alfonso F, Bachet J, Bonser R, Czerny M, Eggebrecht H, et al. Thoracic Endovascular Aortic Repair (TEVAR) for the treatment of aortic diseases: a position statement from the European Association for Cardio-Thoracic Surgery (EACTS) and the European Society of Cardiology (ESC), in collaboration with the European Association of Percutaneous Cardiovascular Interventions (EAPCI). *Eur Heart J* 2012; **33**: 1558–63. doi: <http://dx.doi.org/10.1093/eurheartj/ehs074>
20. Fattori R, Cao P, De Rango P, Czerny M, Evangelista A, Nienaber C, et al. Interdisciplinary expert consensus document on management of type B aortic dissection. *J Am Coll Cardiol* 2013; **61**: 1661–78. doi: <http://dx.doi.org/10.1016/j.jacc.2012.11.072>
21. Eyster WR, Clark MD. Dissecting aneurysms of the aorta: roentgen manifestations including a comparison with other types of aneurysms. *Radiology* 1965; **85**: 1047–57. doi: <http://dx.doi.org/10.1148/85.6.1047>
22. von Kodolitsch Y, Nienaber CA, Dieckmann C, Schwartz AG, Hofmann T, Brekenfeld C, et al. Chest radiography for the diagnosis of acute aortic syndrome. *Am J Med* 2004; **116**: 73–7. doi: <http://dx.doi.org/10.1016/j.amjmed.2003.08.030>
23. Hayter RG, Rhea JT, Small A, Tafazoli FS, Novelline RA. Suspected aortic dissection and other aortic disorders: multi-detector row CT in 373 cases in the emergency setting. *Radiology* 2006; **238**: 841–52. doi: <http://dx.doi.org/10.1148/radiol.2383041528>
24. Souza D, Ledbetter S. Diagnostic errors in the evaluation of nontraumatic aortic emergencies. *Semin Ultrasound CT MR* 2012; **33**: 318–36. doi: <http://dx.doi.org/10.1053/j.sult.2012.02.001>
25. 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**: 656–65. doi: <http://dx.doi.org/10.2214/AJR.13.11437>
26. Holloway BJ, Rosewarne D, Jones RG. Imaging of thoracic aortic disease. *Br J Radiol* 2011; **84**: S338–54. doi: <http://dx.doi.org/10.1259/bjr/30655825>
27. Chung JH, Ghoshhajra BB, Rojas CA, Dave BR, Abbata S. CT angiography of the thoracic aorta. *Radiol Clin North Am* 2010; **48**: 249–64. doi: <http://dx.doi.org/10.1016/j.rcl.2010.02.001>
28. Fleischmann D, Chin AS, Molvin L, Wang J, Hallett R. Computed tomography angiography: a review and technical update. *Radiol*

- Clin North Am* 2016; **54**: 1–12. doi: <http://dx.doi.org/10.1016/j.rcl.2015.09.002>
29. Bae KT. Intravenous contrast medium administration and scan timing at CT: considerations and approaches. *Radiology* 2010; **256**: 32–61. doi: <http://dx.doi.org/10.1148/radiol.10090908>
 30. Baliga RR, Nienaber CA, Bossone E, Oh JK, Isselbacher EM, Sechtem U, et al. The role of imaging in aortic dissection and related syndromes. *JACC Cardiovasc Imaging* 2014; **7**: 406–24. doi: <http://dx.doi.org/10.1016/j.jcmg.2013.10.015>
 31. Meier C, Lichtenberg M, Lebiecz P, Breuckmann F. Pitfalls in suspected acute aortic syndrome: impact of appropriate and if required repeated imaging. *Case Rep Emerg Med* 2015; **2015**: 573256. doi: <http://dx.doi.org/10.1155/2015/573256>
 32. Harris KM, Strauss CE, Eagle KA, Hirsch AT, Isselbacher EM, Tsai TT, et al; International Registry of Acute Aortic Dissection (IRAD) Investigators. Correlates of delayed recognition and treatment of acute Type A aortic dissection: the International Registry of Acute Aortic Dissection (IRAD). *Circulation*. 2011; **124**: 1911–8. doi: <http://dx.doi.org/10.1161/CIRCULATIONAHA.110.006320>
 33. Bolen MA, Popovic ZB, Tandon N, Flamm SD, Schoenhagen P, Halliburton SS. Image quality, contrast enhancement, and radiation dose of ECG-triggered high-pitch CT versus non-ECG-triggered standard-pitch CT of the thoracoabdominal aorta. *AJR Am J Roentgenol* 2012; **198**: 931–8. doi: <http://dx.doi.org/10.2214/AJR.11.6921>
 34. Agarwal PP, Chughtai A, Matzinger FR, Kazerooni EA. Multidetector CT of thoracic aortic aneurysms. *Radiographics* 2009; **29**: 537–52. doi: <http://dx.doi.org/10.1148/rg.292075080>
 35. Valente T, Rossi G, Lassandro F, Marino M, Tortora G, Muto R, et al. MDCT in diagnosing acute aortic syndromes: reviewing common and less common CT findings. *Radiol Med* 2012; **117**: 393–409. doi: <http://dx.doi.org/10.1007/s11547-011-0747-9>
 36. Castañer E, Andreu M, Gallardo X, Mata JM, Cabezu MA, Pallardó Y. CT in non-traumatic acute thoracic aortic disease: typical and atypical features and complications. *Radiographics* 2003; **23**: S93–110.
 37. McMahon MA, Squirrell CA. Multidetector CT of aortic dissection: a pictorial review. *Radiographics* 2010; **30**: 445–60. doi: <http://dx.doi.org/10.1148/rg.302095104>
 38. LePage MA, Quint LE, Sonnad SS, Deeb GM, Williams DM. Aortic dissection: CT features that distinguish true lumen from false lumen. *AJR Am J Roentgenol* 2001; **177**: 207–11. doi: <http://dx.doi.org/10.2214/ajr.177.1.1770207>
 39. Kapoor V, Ferris JV, Fuhrman CR. Intimo-medial rupture: a new CT finding to distinguish true from false lumen in aortic dissection. *AJR Am J Roentgenol* 2004; **183**: 109–12. doi: <http://dx.doi.org/10.2214/ajr.183.1.1830109>
 40. Maldjian PD, Partyka L. Intimal tears in thoracic aortic dissection: appearance on MDCT With virtual angiography. *AJR Am J Roentgenol* 2012; **198**: 955–61. doi: <http://dx.doi.org/10.2214/AJR.11.7327>
 41. Khan IA, Nair CK. Clinical, diagnostic, and management perspectives of aortic dissection. *Chest* 2002; **122**: 311–28. doi: <http://dx.doi.org/10.1378/chest.122.1.311>
 42. Nienaber CA. The role of imaging in acute aortic syndromes. *Eur Heart J Cardiovasc Imaging* 2013; **14**: 15–23. doi: <http://dx.doi.org/10.1093/ehjci/jes215>
 43. Chin AS, Fleischmann D. State-of-the-art computed tomography angiography of acute aortic syndrome. *Semin Ultrasound CT MR* 2012; **33**: 222–34. doi: <http://dx.doi.org/10.1053/j.sult.2012.01.003>
 44. Lovy AJ, Rosenblum JK, Levsky JM, Godelman A, Zalta B, Jain VR, et al. Acute aortic syndromes: a second look at dual-phase CT. *AJR Am J Roentgenol* 2013; **200**: 805–11. doi: <http://dx.doi.org/10.2214/AJR.12.8797>
 45. Jánosi RA, Erbel R, Konorza TF. Recent advances in the diagnosis of acute aortic syndromes. *Expert Opin Med Diagn* 2012; **6**: 529–40. doi: <http://dx.doi.org/10.1517/17530059.2012.704362>
 46. Ko SM. MDCT findings of acute aortic dissection with diastolic prolapse of the intimal flap into the left ventricle. *Br J Radiol* 2009; **82**: e95–7. doi: <http://dx.doi.org/10.1259/bjr/28896686>
 47. Valente T, Rossi G, Lassandro F, Rea G, Marino M, Urciuolo S, et al. MDCT distinguishing features of focal aortic projections (FAP) in acute clinical settings. *Radiol Med* 2015; **120**: 50–72. doi: <http://dx.doi.org/10.1007/s11547-014-0459-z>
 48. Svensson LG, Labib SB, Eisenhauer AC, Butterly JR. Intimal tear without hematoma: an important variant of aortic dissection that can elude current imaging techniques. *Circulation* 1999; **99**: 1331–6. doi: <http://dx.doi.org/10.1161/01.CIR.99.10.1331>
 49. Gaxotte V, Cochetoux B, Haulon S, Vincentelli A, Lions C, Koussa M, et al. Relationship of intimal flap position to endovascular treatment of malperfusion syndromes in aortic dissection. *J Endovasc Ther* 2003; **10**: 719–27. doi: [http://dx.doi.org/10.1583/1545-1550\(2003\)010<0719:ROIFPT>2.0.CO;2](http://dx.doi.org/10.1583/1545-1550(2003)010<0719:ROIFPT>2.0.CO;2)
 50. Willoteaux S, Lions C, Gaxotte V, Negaiwi Z, Beregi JP. Imaging of aortic dissection by helical computed tomography (CT). *Eur Radiol* 2004; **14**: 1999–2008. doi: <http://dx.doi.org/10.1007/s00330-004-2441-y>
 51. Eggebrecht H, Kahlert P, Kaiser GM, McDougall I, Sarraf-Cave Z, Herrmann RA, et al. Technical development and initial animal experience with a novel, uncovered, self-expanding, highly flexible aortic stent with improved side branch access. *J Endovasc Ther* 2009; **16**: 539–45. doi: <http://dx.doi.org/10.1583/09-2776.1>
 52. Midulla M, Renaud A, Martinelli T, Koussa M, Mounier-Vehier C, Prat A, et al. Endovascular fenestration in aortic dissection with acute malperfusion syndrome: immediate and late follow-up. *J Thorac Cardiovasc Surg* 2011; **142**: 66–72. doi: <http://dx.doi.org/10.1016/j.jtcvs.2010.07.081>
 53. Evangelista A, Salas A, Ribera A, Ferreira-González I, Cuellar H, Pineda V, et al. Long-term outcome of aortic dissection with patent false lumen: predictive role of entry tear size and location. *Circulation* 2012; **125**: 3133–41. doi: <http://dx.doi.org/10.1161/CIRCULATIONAHA.111.090266>
 54. Harris KM, Braverman AC, Eagle KA, Woznicki EM, Pyeritz RE, Myrmel T, et al. Acute aortic intramural hematoma: an analysis from the international registry of acute aortic dissection. *Circulation* 2012; **126**(11 Suppl. 1): S91–6. doi: <http://dx.doi.org/10.1161/CIRCULATIONAHA.111.084541>
 55. Schlatter T, Auriol J, Marcheix B, Lebbadi M, Marachet MA, Dang-Tran KD, et al. Type B intramural hematoma of the aorta: evolution and prognostic value of intimal erosion. *J Vasc Interv Radiol* 2011; **22**: 533–41. doi: <http://dx.doi.org/10.1016/j.jvir.2010.10.028>
 56. von Kodolitsch Y, Csösz SK, Koschik DH, Schalwat I, Loose R, Karck M, et al. Intramural hematoma of the aorta: predictors of progression to dissection and rupture. *Circulation* 2003; **107**: 1158–63. doi: <http://dx.doi.org/10.1161/01.CIR.0000052628.77047.EA>
 57. Kitai T, Kaji S, Yamamuro A, Tani T, Kinoshita M, Ehara N, et al. Detection of intimal defect by 64-row multidetector computed tomography in patients with acute aortic intramural hematoma. *Circulation* 2011; **124**(Suppl. 11): S174–8. doi: <http://dx.doi.org/10.1161/CIRCULATIONAHA.111.037416>
 58. Bosma MS, Quint LE, Williams DM, Patel HJ, Jiang Q, Myles JD. Ulcerlike projections developing in noncommunicating aortic dissections: CT findings and natural history. *AJR Am J Roentgenol* 2009; **193**: 895–905. doi: <http://dx.doi.org/10.2214/AJR.08.2073>
 59. Wu MT, Wang YC, Huang YL, Chang RS, Li SC, Yang P, et al. Intramural blood pools accompanying aortic intramural hematoma: CT appearance and natural course. *Radiology*

- 2011; **258**: 705–13. doi: <http://dx.doi.org/10.1148/radiol.10101270>
60. Wu MT, Wu TH, Lee D. Images in cardiovascular medicine. Multislice computed tomography of aortic intramural hematoma with progressive intercostal artery tears: the chinese ring-sword sign. *Circulation* 2005; **111**: e92–3. doi: <http://dx.doi.org/10.1161/01.CIR.0000154547.65893.45>
61. Ferro C, Rossi UG, Seitun S, Scarano F, Passerone G, Williams DM. Aortic branch artery pseudoaneurysms associated with intramural hematoma: when and how to do endovascular embolization. *Cardiovasc Intervent Radiol* 2013; **36**: 422–32. doi: <http://dx.doi.org/10.1007/s00270-012-0512-z>
62. Seitun S, Rossi UG, Cademartiri F, Maffei E, Cronin P, Ferro C, et al. MDCT findings of aortic branch artery pseudoaneurysms associated with type B intramural haematoma. *Radiol Med* 2012; **117**: 789–803. doi: <http://dx.doi.org/10.1007/s11547-011-0779-1>
63. Nathan DP, Boonn W, Lai E, Wang GJ, Desai N, Woo EY, et al. Presentation, complications, and natural history of penetrating atherosclerotic ulcer disease. *J Vasc Surg* 2012; **55**: 10–5. doi: <http://dx.doi.org/10.1016/j.jvs.2011.08.005>
64. Evangelista A, Carro A, Moral S, Teixido-Tura G, Rodríguez-Palomares JF, Cuéllar H, et al. Imaging modalities for the early diagnosis of acute aortic syndrome. *Nat Rev Cardiol* 2013; **10**: 477–86. doi: <http://dx.doi.org/10.1038/nrcardio.2013.92>
65. Patatas K, Shrivastava V, Ettles DF. Penetrating atherosclerotic ulcer of the aorta: a continuing debate. *Clin Radiol* 2013; **68**: 753–9. doi: <http://dx.doi.org/10.1016/j.crad.2012.11.014>
66. Botta L, Buttazzi K, Russo V, Parlapiano M, Gostoli V, Di Bartolomeo R, et al. Endovascular repair for penetrating atherosclerotic ulcers of the descending thoracic aorta: early and mid-term results. *Ann Thorac Surg* 2008; **85**: 987–92. doi: <http://dx.doi.org/10.1016/j.athoracsur.2007.10.079>
67. 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**(1 Suppl. 1): 83–90.
68. Booher AM, Eagle KA. Diagnosis and management issues in thoracic aortic aneurysm. *Am Heart J* 2011; **162**: 38–46. doi: <http://dx.doi.org/10.1016/j.ahj.2011.04.010>
69. Guo DC, Papke CL, He R, Milewicz DM. Pathogenesis of thoracic and abdominal aortic aneurysms. *Ann N Y Acad Sci* 2006; **1085**: 339–52. doi: <http://dx.doi.org/10.1196/annals.1383.013>
70. Bhalla S, West OC. CT of nontraumatic thoracic aortic emergencies. *Semin Ultrasound CT MR* 2005; **26**: 281–304. doi: <http://dx.doi.org/10.1053/j.sult.2005.07.002>
71. Schwartz SA, Taljanovic MS, Smyth S, O'Brien MJ, Rogers LF. CT findings of rupture, impending rupture, and contained rupture of abdominal aortic aneurysms. *AJR Am J Roentgenol* 2007; **188**: W57–62. doi: <http://dx.doi.org/10.2214/AJR.05.1554>
72. Rakita D, Newatia A, Hines JJ, Siegel DN, Friedman B. Spectrum of CT findings in rupture and impending rupture of abdominal aortic aneurysms. *Radiographics* 2007; **27**: 497–507. doi: <http://dx.doi.org/10.1148/rg.272065026>
73. Ahmed MZ, Ling L, Ettles DF. Common and uncommon CT findings in rupture and impending rupture of abdominal aortic aneurysms. *Clin Radiol* 2013; **68**: 962–71. doi: <http://dx.doi.org/10.1016/j.crad.2013.04.007>
74. Chaikriangkrai K, Choi SY, Nabi F, Chang SM. Important advances in technology and unique applications to cardiovascular computed tomography. *Methodist Debaque Cardiovasc J* 2014; **10**: 152–8. doi: <http://dx.doi.org/10.14797/mdcj-10-3-152>
75. Johnson TR. Dual-energy CT: general principles. *AJR Am J Roentgenol* 2012; **199**(Suppl. 5): S3–8. doi: <http://dx.doi.org/10.2214/AJR.12.9116>
76. Vliegenthart R, Pelgrim GJ, Ebersberger U, Rowe GW, Oudkerk M, Schoepf UJ. Dual-Energy CT of the Heart. *AJR Am J Roentgenol* 2012; **199**(Suppl. 5): S54–63. doi: <http://dx.doi.org/10.2214/AJR.12.9208>
77. Henzler T, Fink C, Schoenberg SO, Schoepf UJ. Dual-Energy CT: radiation dose aspects. *AJR Am J Roentgenol* 2012; **199**(Suppl. 5): S16–25. doi: <http://dx.doi.org/10.2214/AJR.12.9210>
78. Yanagawa M, Gyobu T, Leung AN, Kawai M, Kawata Y, Sumikawa H, et al. Ultra-low-dose CT of the lung: effect of iterative reconstruction techniques on image quality. *Acad Radiol* 2014; **21**: 695–703. doi: <http://dx.doi.org/10.1016/j.acra.2014.01.023>
79. Nitta N, Ikeda M, Sonoda A, Nagatani Y, Ohta S, Takahashi M, et al. Images acquired using 320-MDCT with adaptive iterative dose reduction with wide-volume acquisition: visual evaluation of image quality by 10 radiologists using an abdominal phantom. *AJR Am J Roentgenol* 2014; **202**: 2–12. doi: <http://dx.doi.org/10.2214/AJR.12.10364>
80. Yamada Y, Jinzaki M, Hosokawa T, Tanami Y, Sugiura H, Abe T, et al. Dose reduction in chest CT: comparison of the adaptive iterative dose reduction 3D, adaptive iterative dose reduction, and filtered back projection reconstruction techniques. *Eur J Radiol* 2012; **81**: 4185–95. doi: <http://dx.doi.org/10.1016/j.ejrad.2012.07.013>
81. Pasricha SS, Nandurkar D, Seneviratne SK, Cameron JD, Crossett M, Schneider-Kolsky ME, et al. Image quality of coronary 320-MDCT in patients with atrial fibrillation: initial experience. *AJR Am J Roentgenol* 2009; **193**: 1514–21. doi: <http://dx.doi.org/10.2214/AJR.09.2319>