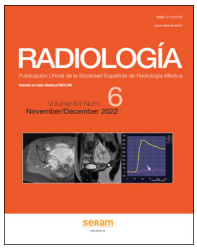
Radiología 67 (2025) 101613

RADIOLOGÍA

www.elsevier.es/rx



UPDATE IN RADIOLOGY

Acute aortic syndrome: What do you need to know?

S. Pereiro Pérez*, **S. Lojo-Lendoiro, M. Pérez Costas, A. Robles Gómez, S. García Benito, P. Rodríguez Fernández**

Servicio de Radiodiagnóstico, Hospital Álvaro Cunqueiro, Vigo, Pontevedra, Spain

Received 13 March 2024; accepted 24 May 2024
Available online 11 June 2025

KEYWORDS
Blood pool;
Aortic dissection;
Aortic intramural hematoma;
Aortic rupture;
Acute aortic syndrome;
Ulcer-like projection;
Penetrating aortic ulcer

Abstract Acute aortic syndrome is a potentially fatal clinical entity that encompasses different pathologies affecting the wall of the aorta: aortic dissection, intramural haematoma, penetrating aortic ulcer and other less common entities. All are characterised by the sudden onset of thoracic-abdominal pain and all share similar features, preventing differentiation. Computed tomography plays a crucial role in the diagnostic process, enabling a rapid and accurate assessment and contributing to early therapeutic management.

This article reviews the main entities that make up acute aortic syndrome, their aetiologies, pathophysiology and characteristic radiological findings. Imaging tests are mainly used to confirm the diagnosis and determine the location and extent as well as possible complications.

PALABRAS CLAVE
Acúmulo de contraste;
Disección aórtica;
Hematoma intramural aórtico;
Rotura aórtica;
Síndrome aórtico agudo;
Proyección simuladora de úlcera;
Úlcera aórtica penetrante

Síndrome aórtico agudo ¿qué necesitas saber?

Resumen El síndrome aórtico agudo es una entidad clínica potencialmente mortal que engloba diferentes patologías que afectan a la pared de la arteria aorta: la disección aórtica, el hematoma intramural, la úlcera aórtica penetrante y otras entidades menos habituales. Todas ellas se caracterizan por un cuadro de dolor tóraco-abdominal de instauración súbita con características similares, que no permite diferenciarlas. La tomografía computarizada, juega un papel crucial en el proceso diagnóstico, permitiendo una valoración rápida y precisa, contribuyendo a un precoz manejo terapéutico.

En el presente artículo se realiza una revisión de las principales entidades que componen el síndrome aórtico agudo, sus etiologías, fisiopatología y los hallazgos radiológicos característicos. El objetivo principal de las pruebas de imagen es confirmar el diagnóstico, determinando la localización, extensión y posibles complicaciones.

* Corresponding author.
E-mail address: samupereiro@gmail.com (S. Pereiro Pérez).

Introduction

Acute aortic syndrome (AAS) encompasses a group of potentially life-threatening conditions affecting the wall of the aorta. They are clinically indistinguishable and manifest with sudden chest/abdominal pain of varying intensity. This syndrome encompasses various conditions, including the most common condition, aortic dissection (AD), as well as intramural haematoma (IMH) and penetrating aortic ulcer (PAU). Finally, there is a catch-all category, which some authors include traumatic aortic pathology and rupture of aortic aneurysm in.[1–4]

The incidence is three to seven cases per 100,000 patient-years, although it is probably underdiagnosed as it is associated with cases of sudden death and pre-hospital mortality which go unnoticed if a directed autopsy is not performed.[5] The prevalence is higher in males (2:1) around the 6th-7th decades of life.[6–9] It appears to have a temporal pattern, with the prevalence of AD higher during the winter months and in the morning.[1,10]

Several risk factors common to all the conditions have been identified. The most common of these is hypertension (HT), but others include atherosclerosis, being male, smoking, having undergone previous aortic surgery, having an aneurysm and having a collagen disorder.[8]

The gold standard imaging technique is computed tomography (CT), which plays a crucial role in the management of AAS, as it not only provides a rapid diagnosis, but also provides the detailed anatomical characterisation necessary for therapeutic management.

Our main aim with this article was to provide a comprehensive review of the conditions that make up AAS, describing their epidemiology and pathophysiology and giving special emphasis to the characteristic radiological findings and technical aspects of the CT.

Technique

Currently, there is no standardised CT protocol, mainly because of the wide variability in CT equipment, contrast medium concentrations, administered doses and acquisition delay times.[11] The choice of protocol is made by each institution according to its preferences and available tools. At our centre we perform multiphase CT, which provides precise three-dimensional information and has a sensitivity and specificity close to 100%.[8,9,12–16] The study extends from the base of the neck to the height of both trochanters[1,17] and the iodinated contrast is preferably administered in the right arm to avoid flow artefacts in the brachiocephalic trunk, with an approximate volume of 70 ml and a concentration of 370 mg/ml at an approximate flow rate of 4–5 ml/s.

The study includes the following phases:

- A first phase without contrast, which is of special importance for the assessment of findings such as IMH, haemopericardium, haemomediastinum, haemothorax and displacement of intimal calcifications.[1,14,18,19]
- There are two arterial phases: an early phase and a late phase. The early phase is used to confirm dissection and study the dynamic behaviour of both vascular lumens and entry tears. The late phase is used to assess re-entry tears, which may be located in segments distal to the entry tear and cannot be adequately assessed in the early phase.
- Finally, we sometimes include a venous phase in cases of suspected visceral ischaemia, which provides us with information on possible signs of visceral hypoperfusion and the behaviour of the false lumen in later phases (assessment of false lumen thrombosis).[11]

The CT scan is performed using the *bolus tracking* technique,[20] which consists of placing a cursor (ROI) in the aorta. After contrast administration, multiple axial acquisitions are performed at that point until a pre-established Hounsfield unit threshold (150 HU in our centre) is reached at the ROI. Once reached, acquisitions are made at 8, 17 and 50 s respectively (early, late arterial and portal). An important technical aspect to consider in preparation is the placement of the ROI. There is a risk of this being placed in the false lumen and the aortic staining may be suboptimal for assessment (Fig. 1). Visualisation of the non-contrast phase prior to ROI localisation is particularly important, as identifying displaced intimal calcifications can guide us toward the existence of different lumens and so help us choose the most appropriate image from which to fire the helix. At our centre, to avoid motion artefacts, the ROI is located in the proximal descending thoracic aorta and once the true lumen enhancement begins, its correct placement within the aorta is verified. Otherwise, the acquisition is carried out manually.

Pitfalls

To make a correct diagnosis, CT must be assessed taking into account artefacts and potential errors of interpretation (*pitfalls*) caused by different factors (for example, anatomical, technical or physiological).

Heartbeat artefact

The movement derived from heart contraction, more accentuated in young patients with tachycardia, can produce multiple linear artefacts in the periaortic structures, especially in the ascending aorta and at the origin of the coronary arteries, which could be confused with true intimal *flaps*.[21,22] To avoid this, acquisition can be synchronised with an electrocardiogram (ECG), or using rapid acquisition equipment with *high pitch*, which improves the images obtained, especially where the disease is suspected to be affecting the aortic root or ascending aorta[4,9,14,23] (Fig. 1a,b). If not performing this type of acquisition, the different CT phases available will need to be evaluated; a heartbeat artefact will not be constant, while a true intimal tear will be visible in all phases.

Flow artefact

In older adult patients with low cardiac output, linear artefacts can be identified which can simulate *flaps*[21] and are the result of flow turbulence inside the vessel. They are most common in the lesser curvature of the aortic arch and in the

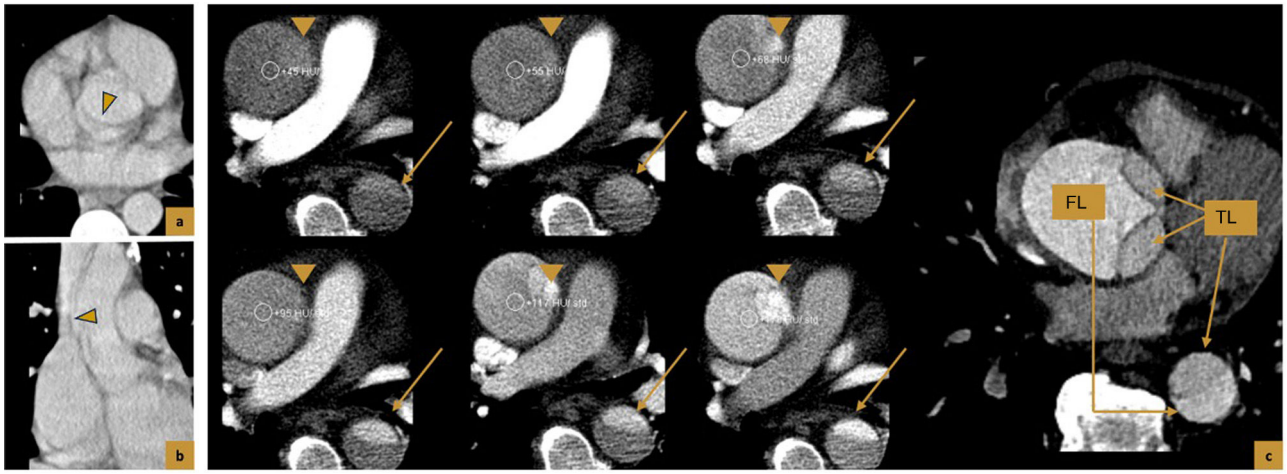


Figure 1 Common *pitfalls*. (a) and (b) Non-ECG-synchronised portal-phase contrast-enhanced CT, axial and coronal slices. Pronounced motion artefact in the aortic root simulating a dissecting *flap* (arrowheads) within the supravalvular aorta, a typical location of motion artefact. (c) Accidental placement of the ROI in the false lumen, axial slices from the scan sequence. Early staining of the true lumen is seen at the medial margin of the ascending aorta (arrowhead) and at the anterior margin of the descending aorta (arrows), with the ROI located in the false lumen. As a result, the study acquisition was performed late, once the threshold in the false lumen was reached, resulting in late suboptimal staining of the study. The image on the right corresponds to the helix of the study and shows a collapsed true lumen (TL) in the medial margin of the ascending aorta with a lower degree of attenuation than would be expected.

central aspect of the descending aorta, giving a *smoke-like* image. In case of doubt, a later CT acquisition can be considered, which will homogenise the contrast and confirm the absence of AD.[12]

Aortic dissection

AD is the most common condition coming under AAS. The incidence is estimated at 2.6 to 3.5 cases per 100,000 people per year.[5,24]

Pathophysiology

The pathophysiological mechanism is still not fully understood. It is believed that most are secondary to a degeneration of the middle layer of the aorta (cystic necrosis) which results in a loss of elasticity that reduces resistance to mechanical stress, perpetuating weakness and predisposing to dissection.[25] It originates from an intimal tear (entry point) (Fig. 2), which separates the layers of the wall, generating two vascular lumens and allowing the passage of blood from the true lumen to the false lumen, separated by an intimal flap (Fig. 2b–d). Hypertension and degenerative changes in the medial layer are the most important predisposing factors. Other conditions that have been linked include various connective tissue diseases such as Marfan syndrome, Ehlers-Danlos syndrome and Turner syndrome, and congenital defects of the aortic valve.[1]

Classification

AD classifications are based on the anatomical extension (Fig. 3a) of the intimal *flap* and the most widely used and well-known are those of DeBakey and Stanford[26] (Fig. 3b).

- Stanford Classification: preferred in the radiological setting for its simplicity and its therapeutic implications. It categorises both AD and IMH into two groups: those that involve the ascending aorta regardless of the location of the entry tear (type A) and therefore require surgical management[27]; and those that do not affect the ascending aorta (type B) and whose urgent management is initially conservative. Some authors propose using the term n̈on-A non-Bd̈issection to refer to those dissections with the entry point in the descending aorta and retrograde progression of the *flap* to the aortic arch and those that have the entry point between the brachiocephalic trunk and the subclavian artery and do not affect the ascending aorta. It is important to be aware of n̈on-A non-Bd̈issections because they are associated with higher complication rates and worse prognoses.[28,29]
- DeBakey Classification[30]: used exclusively for AD and classifies it as type I if it involves the ascending aorta, aortic arch and descending aorta, type II if it is limited to the ascending aorta, and type III if it affects the aorta distal to the exit of the subclavian, sub-classified as IIIa if it exclusively affects the descending thoracic aorta and IIIb if it extends to the abdominal aorta.

Imaging findings

CT enables us to assess the entry tear, the identification of both lumens, their extension and possible complications.[1,13] It is important to perform a systematic assessment which provides an accurate and detailed diagnosis, facilitating proper patient management. For this to be done, the following findings will have to be assessed to enable the preparation of a brief, structured and concise radiological

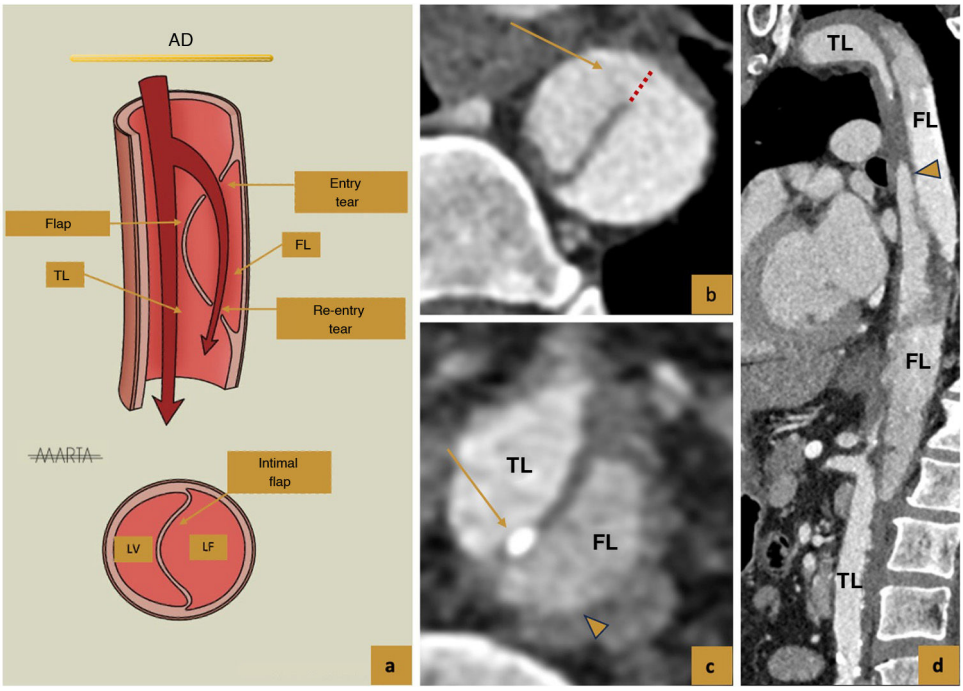


Figure 2 Aortic dissection. (a) Classic illustration of aortic dissection. An entry tear is identified that allows blood to pass through the medial layer, creating two vascular lumens, one true (TL) and one false (FL), separated by an intimal *flap*. (b) Contrast-enhanced CT scan in the arterial phase, axial slice. Entrance tear, identifying the defect (dashed line) of the intimal *flap* that allows communication between the two lumens. (c) Contrast-enhanced CT in arterial phase, axial slice, intimal *flap* with intimal calcification (arrow) in the external wall of the left ventricle. The calcification is located in the intima, which has displaced from the external wall of the aorta (arrowhead) medially into the lumen. In addition, less contrast opacification characteristic of false lumen can be seen. (d) Late arterial phase contrast-enhanced CT, sagittal slice. Type B dissection. An intimal *flap* (arrowhead) with spiral progression (not shown) is identified separating the two vascular lumens, a true one (TL) with homogeneous contrast opacification and a false one (FL) with a larger lumen and less contrast enhancement and more heterogeneous, reflecting turbulent flow.

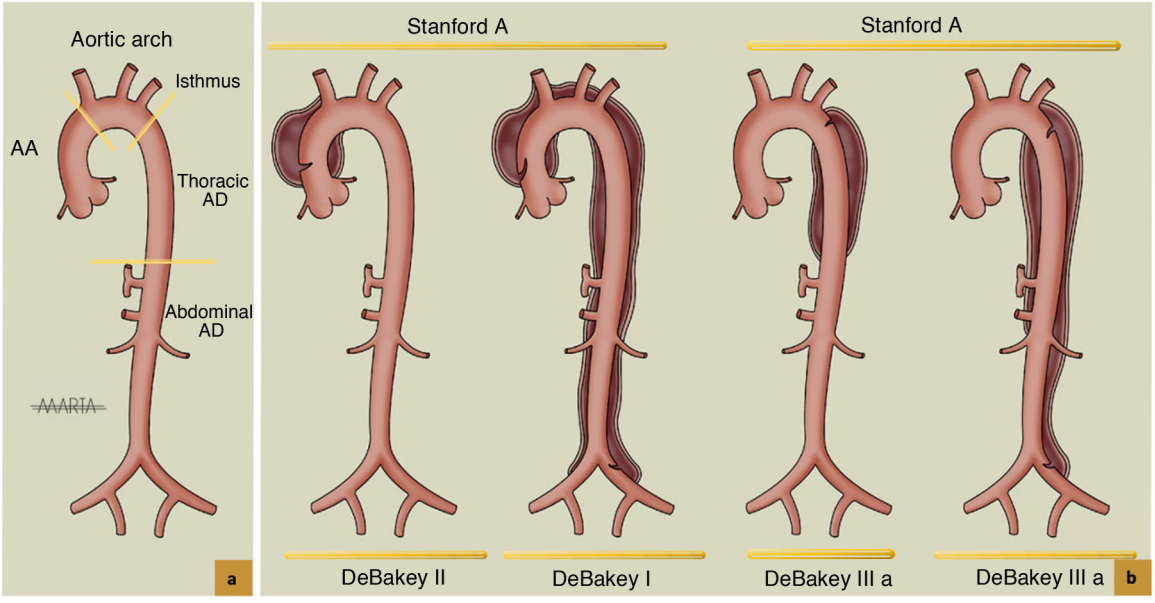


Figure 3 Classic classification of AD. (a) Illustration of the aorta and its anatomical divisions. (b) Stanford and DeBakey classification. The Stanford classification classifies as A if it involves the ascending aorta, otherwise it is classified as B. The DeBakey classification classifies as type I, II or III depending on whether both the ascending and descending aorta are affected, only the ascending or only the descending respectively. DeBakey III is sub-classified as IIIa or IIIb depending on whether only the descending thoracic aorta is involved or the abdominal aorta is affected respectively.

Table 1 Proposal for structured report.

- Stanford Classification (A/B).
- Location of entry and re-entry tear(s) and approximate sizes.
- Morphology and proximal and distal extension of the intimal *flap*.
- Description of TL and FL: morphologies, maximum diameters, presence of false lumen thrombosis.
- Assessment of the origin of the main visceral branches (type of obstruction, signs of hypoperfusion/infarcts).
- Assessment of complications (haemopericardium/haemomediastinum/haemothorax, rupture of the pulmonary artery sheath, rupture of the false lumen)
- Other findings (anatomical variants, incidental findings).

TL: true lumen; FL: false lumen.

report which includes the assessment of the most relevant aspects (Table 1).

Identification of the intimal flap and location of entry tear(s)

Identification of the intimal *flap* confirms the dissection. It is important to look for the entry and re-entry tear(s), as they will be one of the therapeutic targets. The entry tear is typically located in areas of greatest hydraulic stress such as the right lateral wall of the ascending aorta or the segment proximal to the ligamentum arteriosum in the descending aorta.[1,9] Entry tears longer than 10 mm are associated with a worse prognosis, as are those located in the internal curvature of the aorta, as they have a tendency to retrograde progression towards the ascending aorta[31] Re-entry tears are usually located in the abdominal aorta and are frequently not identified due to their small size.

Differentiating between the two lumens

Differentiating between the two lumens is essential for therapeutic management and is not always an easy task. The false lumen will generally be larger, more heterogeneous due to the presence of non-physiological turbulent flow, and will show less contrast opacification in the early arterial phase. The true lumen will be smaller and have greater contrast enhancement in the early phase.

There are many signs which can help with correct differentiation[1,3]:

- Peak sign: an acute angle is visualised in the false lumen between the intimal *flap* and the external wall (Fig. 4.1).
- Intimal calcifications: always located in the intima, they move into the vascular lumen and are located on the external wall of the true lumen (Fig. 4.2).
- Cobweb sign: residual remains of the intimal tear, which show up as multiple small linear filling defects inside the false lumen (Fig. 4.2).

The presence of thrombi in the false lumen is associated with a rapid increase in the size of the vascular lumen due to greater pressurisation, with a worse prognosis in terms of survival.[1] To detect this, it is useful to examine the false lumen in the different CT acquisitions in order to differentiate thrombi from possible flow artefacts (Fig. 4.3).

Segments involved and proximal and distal extension

AD can involve any segment of the aorta. Stanford type A accounts for 60–70% of dissections and requires urgent surgical intervention, with mortality rates of up to 50% in the first 48 h without treatment.[32] It is important to assess and

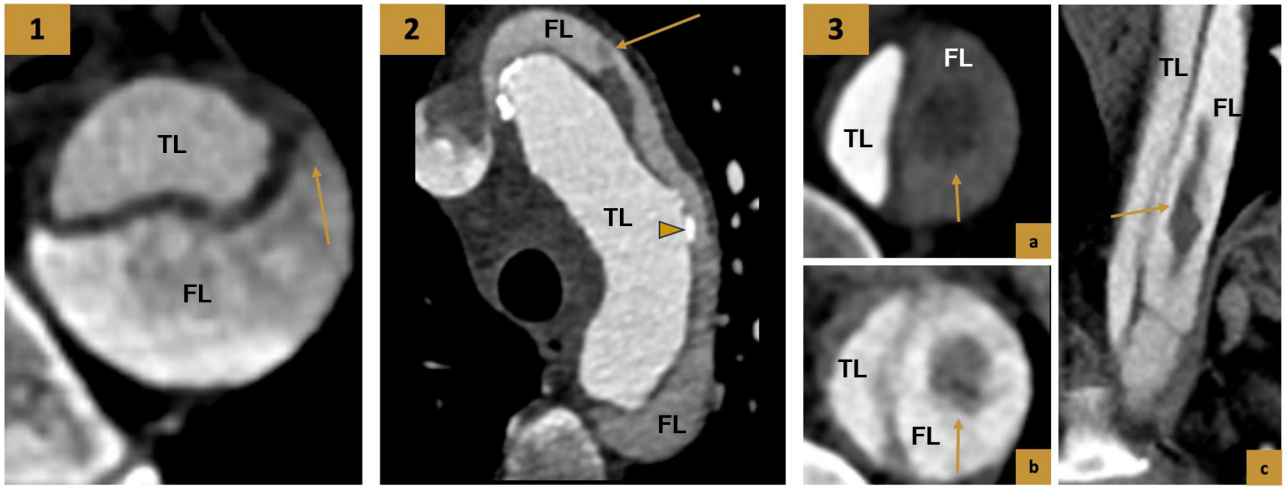


Figure 4 Characteristics to differentiate the two lumens. (1) Peak sign. Contrast-enhanced CT in arterial phase, axial slice. The *intimal flap* forms an acute angle with the external wall of the false lumen (arrow). (2) Contrast-enhanced CT in arterial phase, axial slice. Cobweb sign and intimal calcifications. Linear low attenuation band (arrow) inside false lumen representing torn medial layer fragments. Intimal calcifications displaced into the lumen and on the external wall of the true lumen (arrowhead). (3) Thrombus formation in the false lumen. (a and b) CT in early and late arterial phases, axial slices. Filling defect within the false lumen that persists into the late arterial phase and confirms the presence of thrombus. (c) Contrast-enhanced CT in the late arterial phase, coronal slice. The elongated linear filling defect is identified in association with partial thrombosis of the false lumen.

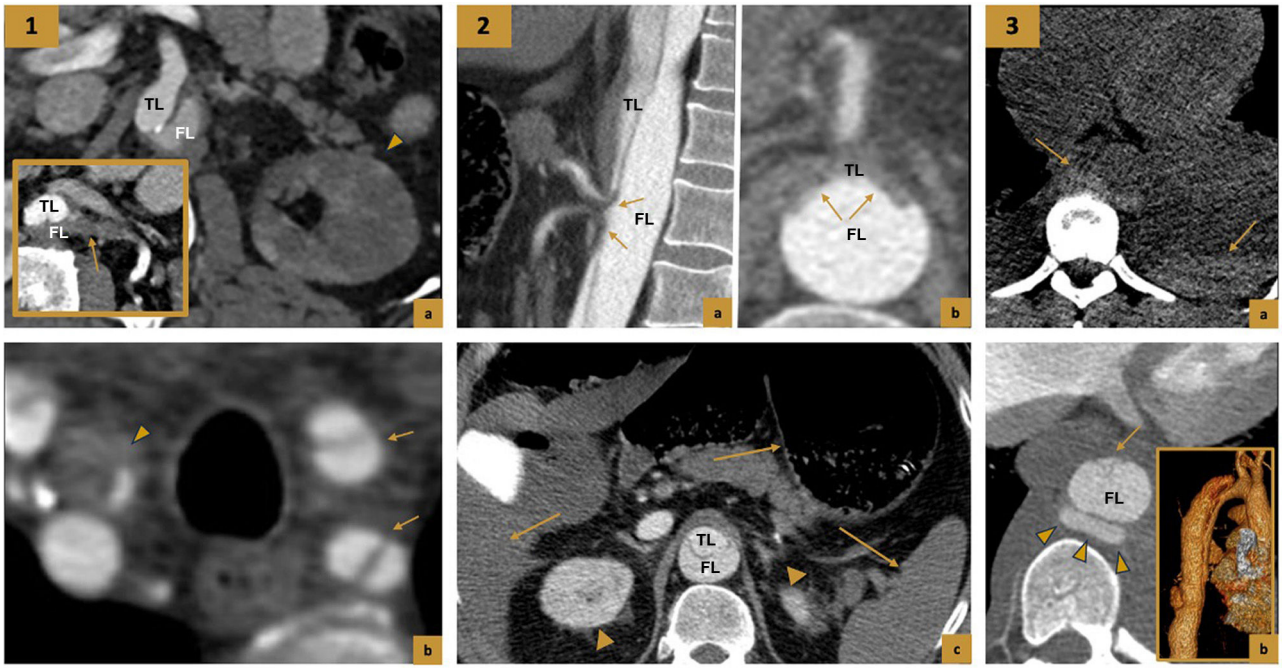


Figure 5 Complications of aortic dissection. 5.1 Renal hypoperfusion and right-sided stroke. (a) Contrast-enhanced CT scan in early arterial (inset) and portal phases, axial slices. Dissection *flap* extending into the left renal artery, with partial thrombosis of the lumen and severe hypoperfusion of the left kidney (arrowhead). (b) CT with contrast, axial slice. Extension of the dissection *flap* to the left carotid and subclavian arteries (arrows) and the right carotid artery, revealing a severely collapsed true lumen and thrombosis of the false lumen, which caused a right-sided stroke (not shown). 5.2 Contrast-enhanced CT scan in late arterial phases, sagittal and axial slices. Mesenteric ischaemia secondary to type B dissection due to severe pressurisation of the false lumen (a and b) which displaces the intimal *flap* anteriorly, occluding the origin of the coeliac trunk and superior mesenteric artery (arrows). (c) Contrast-enhanced CT in the portal phase, axial slice. Severe generalised hypoperfusion of the liver, spleen, and small intestine and colon loops (arrows) with respect to both kidneys (arrowheads), which were spared, consistent with massive mesenteric ischaemia, which was later confirmed in the operating theatre. 5.3 Rupture of the false lumen. Non-contrast CT, axial slice (a) Periaortic haematoma in the posterior region and moderate left haemothorax (arrows). (b). Late arterial phase contrast-enhanced CT, axial slice showing a severely collapsed true lumen in the anterior region with significant distension of the false lumen and a contrast-enhanced image (volumetric reconstruction) at its posterior margin (arrowheads) consistent with rupture of the false lumen.

reflect the distal extension in the report, as involvement of the iliac arteries may lead to changes when planning an endovascular therapeutic approach.

In addition to assessing the extent of the dissection, all the aortic visceral branches need to be evaluated, establishing their origin in the true or false lumen.

Assessment of complications

Obstruction of visceral branches. Two types of lumen obstruction have been described, one static and the other dynamic.[33] Static obstruction is produced by the entry of the *flap* into the ostium of the arterial branch (Fig. 5.1) generating a displacement of the true lumen and a stenosis that will cause hypoperfusion and infarction of the organ if sustained over time.[31,32,34] The treatment for static obstructions is the placement of a *stent* in the true lumen to allow re-expansion and restoration of vascular flow.

Dynamic obstruction affects the branches that originate in the true lumen and is produced by the increase in pressure in the false lumen causing a displacement of the intimal *flap* towards the true lumen (Fig. 5.2). This causes difficulty in the passage of arterial flow to the adjacent visceral branches and consequent ischaemia. Treatment will be aimed at reducing pressure in the false lumen (fenestrations or *stents*).[31,32,34]

Coronary arteries may become dissected or occluded, with the right coronary artery generally being the most affected, causing myocardial ischaemia and infarction[33] (Fig. 6).

Rupture of the false lumen. False lumen distension secondary to weakness of its outer wall (it has no elastic layer) often contributes to dilation of the aorta and formation of pseudoaneurysms. In severe cases where a large amount of blood enters through the intimal defect, sudden intraluminal pressurisation may occur in the false lumen, causing rapid distal progression of the *flap* and possibly complicating the process with a rupture of the aortic wall with extravasation beyond the adventitia[31] (Fig. 5.3).

Aortic valve dysfunction

In type A dissections involving the aortic root, it will be essential to perform an emergency transthoracic echocardiogram,[5] to provide immediate and important information on the functioning of the aortic valve and dilation of the ascending aorta.[13]

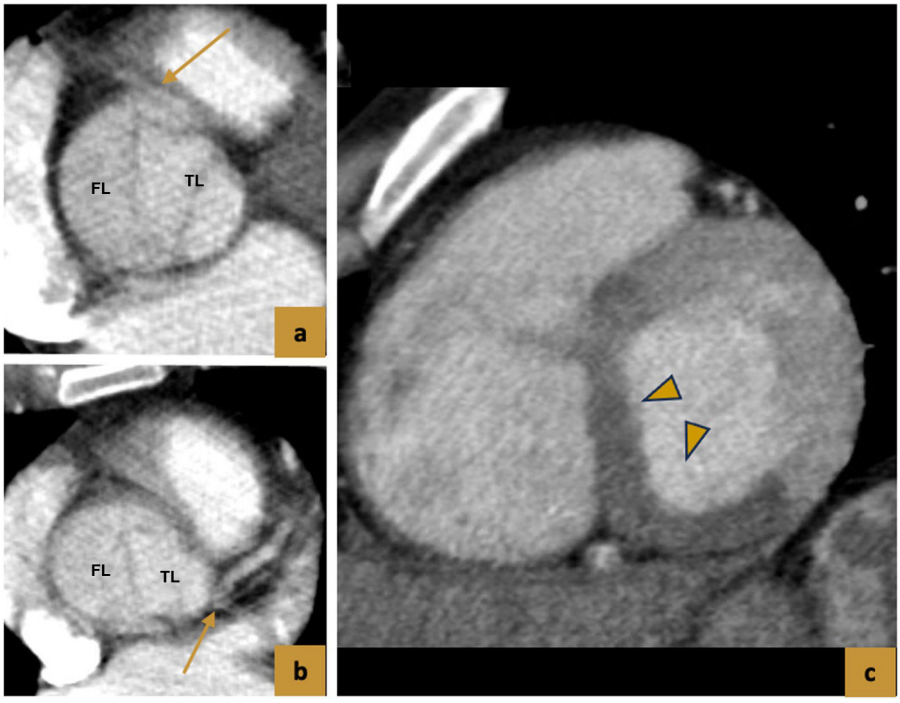


Figure 6 Type A dissection complicated by acute myocardial infarction. (a and b) Late arterial phase contrast-enhanced CT, axial slices showing the origin of the right coronary artery in the false lumen and the origin of the left coronary artery in the true lumen (arrows). (c) Portal phase contrast-enhanced CT, oblique slice. Transmural infarction in the inferior-septal and inferior basal segments (arrowheads) in relation to acute infarction in the right coronary artery territory.

Rupture to the pericardium and mediastinum

One of the possible complications of dissections affecting the ascending aorta is rupture and opening into the pericardium, identifying hyperdense contents in the pericardial sac. These findings are most easily visualised in the non-contrast phase (Fig. 7). In severe cases with massive haemopericardium, distension of the pericardium can lead to cardiac tamponade with collapse of heart chambers, especially the right ones, hypotension and cardiovascular shock.[5,6,32,33]

Haemorrhagic infiltration of the common aortopulmonary adventitia

The real prevalence of this complication is unknown, but it is estimated to be underdiagnosed as it can easily go unnoticed.[35] The ascending aorta and the pulmonary trunk share an embryological origin and, more importantly, a common adventitia. Dissections of the ascending aorta can open through this adventitia, extending the blood towards the pulmonary arteries, and can reach the bronchial tree and the pulmonary parenchyma, even becoming complicated with alveolar haemorrhage, which tends to show up with a peribronchovascular ground-glass pattern.[1,35,36]

The findings in the non-contrast phase are virtually pathognomonic. Hyperdense circumferential areas are seen surrounding the wall of the pulmonary arteries (Fig. 7), which adopt a peripheral halo morphology in the later ones. In severe cases, the haematoma can exert a mass effect on the vascular lumen, reducing the lumen of the pulmonary arteries. This complication is usually accompanied by haemopericardium and haemothorax.

Intramural haematoma

IMH is a potentially life-threatening condition characterised by the presence of an accumulation of blood in the thickness of the aortic wall. Like AD, it presents as sudden, severe chest pain. The incidence varies in the literature and it is estimated to account for 5–25% of aortic syndromes.[14,37–39]

Pathophysiology

Historically, the theory has been that the haematoma is produced by a spontaneous rupture of the *vasa vasorum* within the medial layer in the absence of an intimal defect (Fig. 8). However, a large number of studies have described the presence of small intimal defects found during surgery in cases of IMH.[39–41] The pathophysiology of the conditions that make up AAS is not fully understood and is widely discussed in the literature; the boundary between the three (AD, IMH and PAU) is not clearly defined, and they may be inter-related or even coexist; for example, an IMH could originate from a PAU or a small AD, and the IMH could itself lead to a true dissection.[39]

Classification

The Stanford classification is used (Fig. 3). Stanford A IMH has higher morbidity and mortality rates than type B and therapeutic management follows the dissection scheme, with type A being preferably surgical, although there is a certain amount of debate on the subject.[37,38]

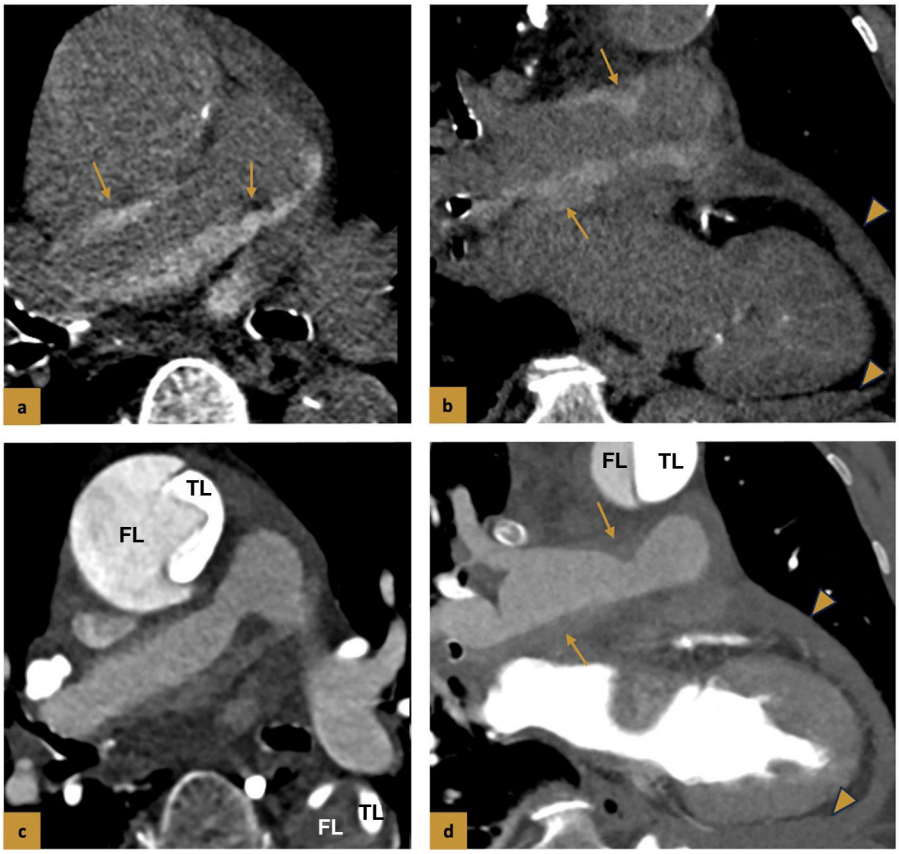


Figure 7 Type A aortic dissection with haematoma extending to the pulmonary artery adventitia. (a) and (b) Axial and coronal CT without contrast. Spontaneously hyperdense circumferential thickening surrounding the pulmonary artery trunk and extending to the right pulmonary artery (arrows). It is accompanied by a moderate haemopericardium (arrowheads). (c and d) Contrast-enhanced CT in the early arterial phase, axial and coronal slices, shows a dissecting flap separating two lumens, a very collapsed true lumen on the left lateral margin of the ascending aorta and anteriorly located in the descending aorta, and a highly pressurised false lumen. The peripheral thickening of the right pulmonary artery is more difficult to identify; the only notable feature is a slight mass effect on the lumen in the proximal segment secondary to the haematoma, which could go unnoticed if the non-contrast phase were unavailable.

Imaging findings

In the phase without intravenous contrast (IVC), a concentric thickening of the aortic wall can be seen with a crescent-like morphology, hyperdense (39–72 HU), which displaces the intimal calcifications in a curvilinear manner into the lumen (Fig. 8). Assessment using a narrow window is recommended (W: 200 HU; L: 40 HU) to facilitate identification.[39] In the phases with IVC it is iso/hypodense, sometimes going unnoticed due to the high attenuation of the aortic lumen.[38,39,42]

Intimal calcifications can help in the differential diagnosis with intraluminal thrombus[25]; while in IMH the intimal calcifications move into the aortic lumen, in intraluminal thrombus the calcifications remain peripheral.

Clinical course

Although IMH appears to have a better prognosis than dissection, there is a non-negligible risk of complications, with an estimated 28–56% of IMH progressing.[38,39] In the natural evolution there are three possible scenarios: in approximately 1/3 of patients complete resorption will eventually take place; 1/3 remain stable; and the remaining 1/3 progress and become more complicated. In this last scenario, the IMH can evolve into AD, aneurysm or even rupture of the arterial wall.[4]

Special mention should be made of two conditions which develop in the follow-up of patients with IMH, *ulcer-like projections* and focal contrast accumulations or *blood pools*. Both consist of small accumulations of contrast in the thickness of the wall and have different pathophysiology and prognostic implications (Fig. 9).

Focal accumulation of contrast in the thickness of the haematoma/*blood pool*

These are small accumulations of contrast appearing in the thickness of the IMH and showing communication with the aortic lumen which are imperceptible or less than 2 mm in size, unlike the projections mimicking an ulcer (Fig. 9a).[39] They are more common in the descending aorta and the theory is that they are related to pseudoaneurysms of the intercostal, bronchial or lumbar aortic branches. The natural evolution is towards resolution and they have prognostic

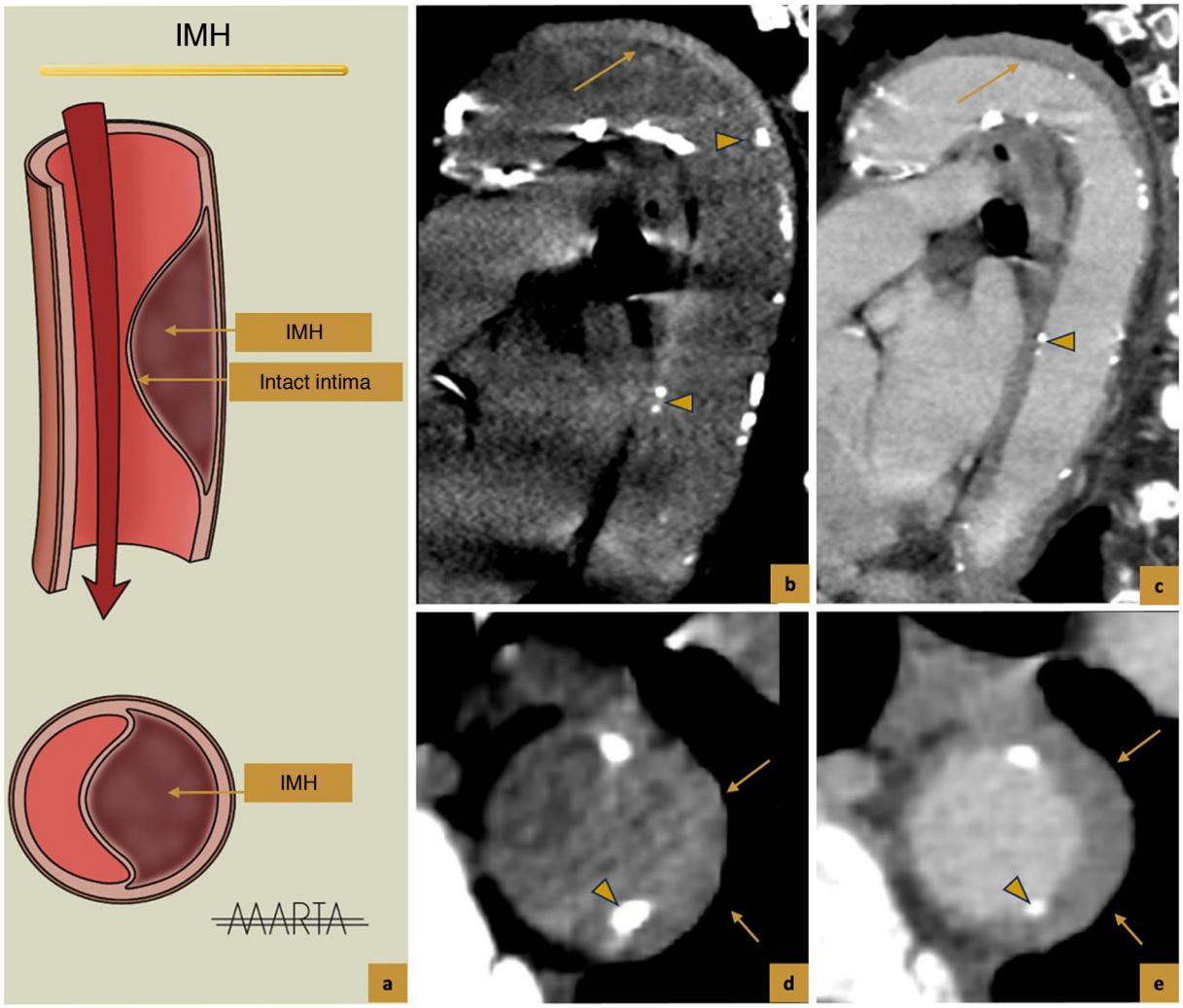


Figure 8 Aortic intramural haematoma. (a). Illustration of an intramural haematoma, showing semilunar thickening of the aortic wall secondary to blood accumulation within the wall, with no entry tears or *flaps*identified. (b, c, d, e) Sagittal CT scans with and without IVC, and axial CT scans with and without IVC respectively. Spontaneously hyperdense aortic semilunar parietal thickening (68 HU) (arrows). Displacement of calcified atheromatous plaques into the lumen (arrowheads) due to the position of the haematoma between the adventitia and the medial layer.

implications.[38,43] Multiple and contiguous blood pools in the descending aorta in relation to the intercostal arteries are uncommon and have been described in the literature as the *Chinese ring-sword sign*[44,45] (Fig. 10).

Ulcer-like projections

These are small extravasations of contrast located in the thickness of the IMH which show communication with the aortic lumen and similar enhancement in the phases with contrast, that is, we have a small focal intimal defect (>3 mm) which communicates with the extravasated contrast[39,43] (Fig. 9b).

The imaging findings are similar to those of penetrating ulcers, but it should be noted that these are two phenomena with distinct pathophysiology. While ulcer-mimicking projections appear in the natural course of an IMH, penetrating ulcers always form from the ulceration of an atheromatous plaque, which eventually comes into contact with the media.[38]

Their natural course is usually progression; they have been associated with a higher risk of complications such as aneurysm formation (most frequent sequela), dissection, rupture and even higher mortality rates.[38]

With regard to the utility of radiology in follow-up, different image predictors have been established which, together with other clinical and analytical data, help make an appropriate selection of patients in whom an active therapeutic approach would be indicated, thus preventing the development of complications[15,37,39]:

- IMH and ascending aortic diameter >4.8 cm or descending aortic diameter >4.1 cm.
- IMH > 11 mm thick or rapid progression in early checks.
- *Ulcer-like projection* > 20 mm in diameter and > 10 mm deep, located in the ascending aorta and aortic arch.

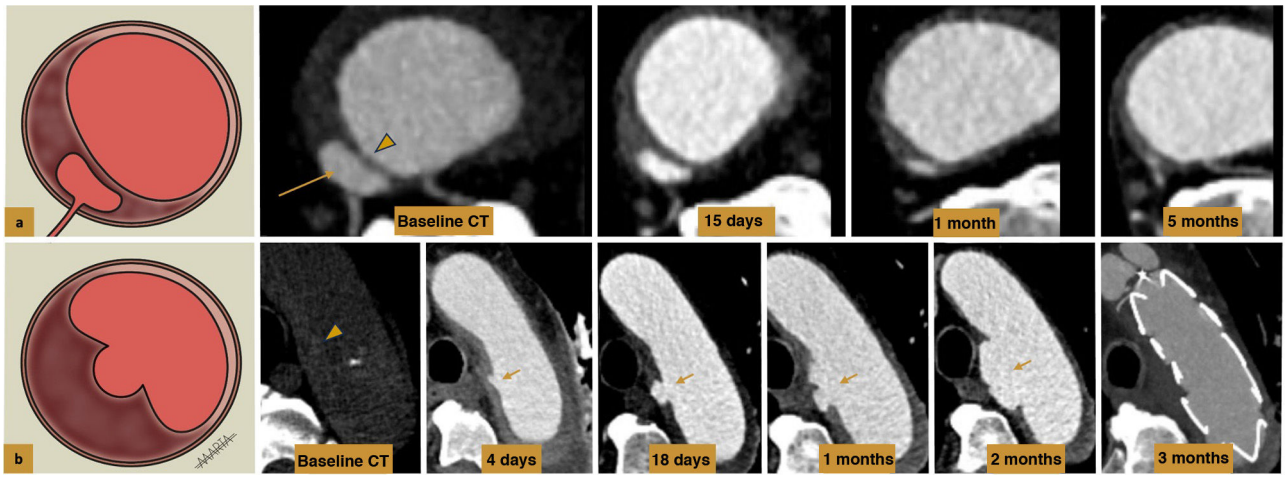


Figure 9 Focal accumulation of contrast vs ulcer-mimicking projection. (a). Focal accumulation of contrast (blood pool). Schematic representation. Accumulation of contrast in the thickness of the haematoma (arrow) with no communication with the arterial lumen and in contact with a small-lumen artery on its right lateral margin. Late arterial phase contrast-enhanced CT, axial slices, focal accumulation of contrast in the thickness of an IMH in the descending aorta without communication with the aortic lumen (arrowhead) and in relation to an intercostal artery at the right lateral margin (arrows). Subsequent follow-up examinations show a tendency toward regression, consistent with the natural evolution of the lesions. (b) Illustration of a projection mimicking an ulcer with contrast accumulation in the thickness of the haematoma communicating with the arterial lumen. Late arterial phase contrast-enhanced CT, axial slices. A spontaneously hyperdense IMH is identified at the inner margin of the aortic arch (arrowhead). In the successive scans, an image of contrast addition is seen in the thickness of the haematoma which communicates with the aortic lumen through an intimal defect (arrows) and progressively increases in thickness. Given the progression and associated risk factors, endovascular treatment with a stent was decided on, excluding the focal defect.

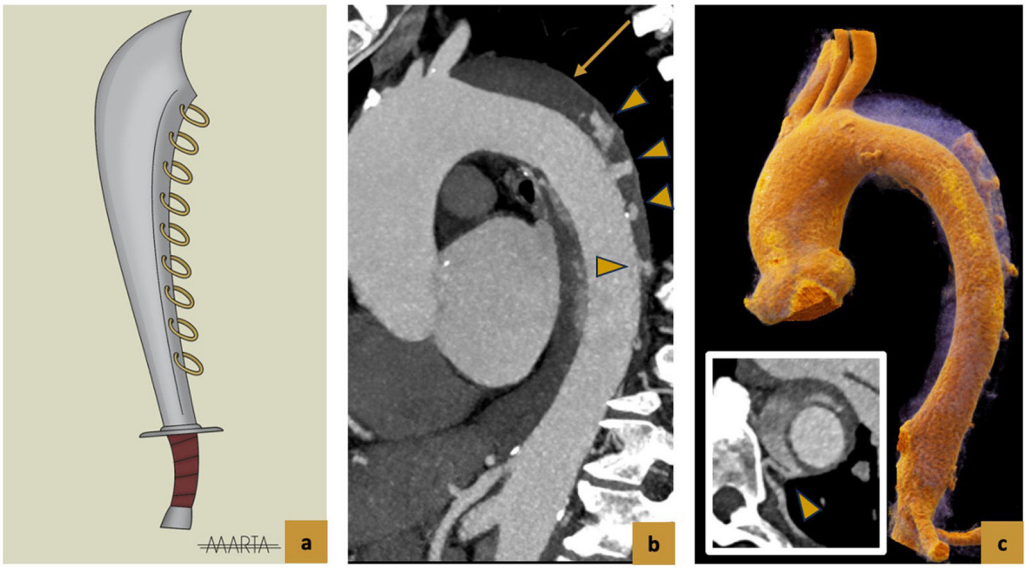


Figure 10 Chinese 9-ring-sword sign. (a). Schematic representation of the sword. (b). Contrast-enhanced CT in early arterial phase, oblique sagittal slice. Type B intramural haematoma (arrow) extending from the left subclavian artery to the iliac arteries (not shown). Small focal accumulations of contrast (arrowheads) with a serpenginous morphology resembling rings are identified in the thickness of the haematoma. (c). Contrast-enhanced CT in the early arterial phase, oblique axial reconstruction showing the connection of these contrast accumulations with an intercostal artery (arrowhead), without communication with the aortic lumen, and 3D volumetric reconstruction in which these accumulations are evident in the thickness of the haematoma, giving the aorta the morphological appearance reminiscent of this characteristic sword.

Table 2 Proposal for structured report of the IMH.

- Stanford Classification (A/B).
- Maximum aortic diameters.
- Maximum thickness of the haematoma and location.
- Focal accumulations of contrast, if any, number and location.
- Projections simulating ulcers, if any, number, location, and largest diameters and thickness.
- Pleural effusion/haemothorax/periaortic haematoma.

IMH: intramural haematoma.

- Stanford type A IMH.
- Pleural or pericardial effusion.
- Persistent or poorly controlled chest pain.

Table 2 shows a proposed structured report which includes the most important elements when assessing an IMH.

Penetrating atherosclerotic ulcer

PAU accounts for 2.3–7.6% of all aortic syndromes and is more common in older, male patients who have multiple comorbidities and cardiovascular risk factors which contribute to the development of atheromatous plaques (for example, HT, hyperlipidaemia, smoking).[3,9,31,46,47]

Pathophysiology

The PAU originates from an atheromatous plaque which erodes the intima until it reaches the internal layer of the media[31,48,49] (Fig. 11a). Progression of the ulcer reaches the adventitial layer of the aorta, weakening the wall and promoting the formation of pseudoaneurysms. The transmission of high intraluminal arterial pressures to the medial layer can lead to the development of IMH, aneurysms, AD or aortic rupture.

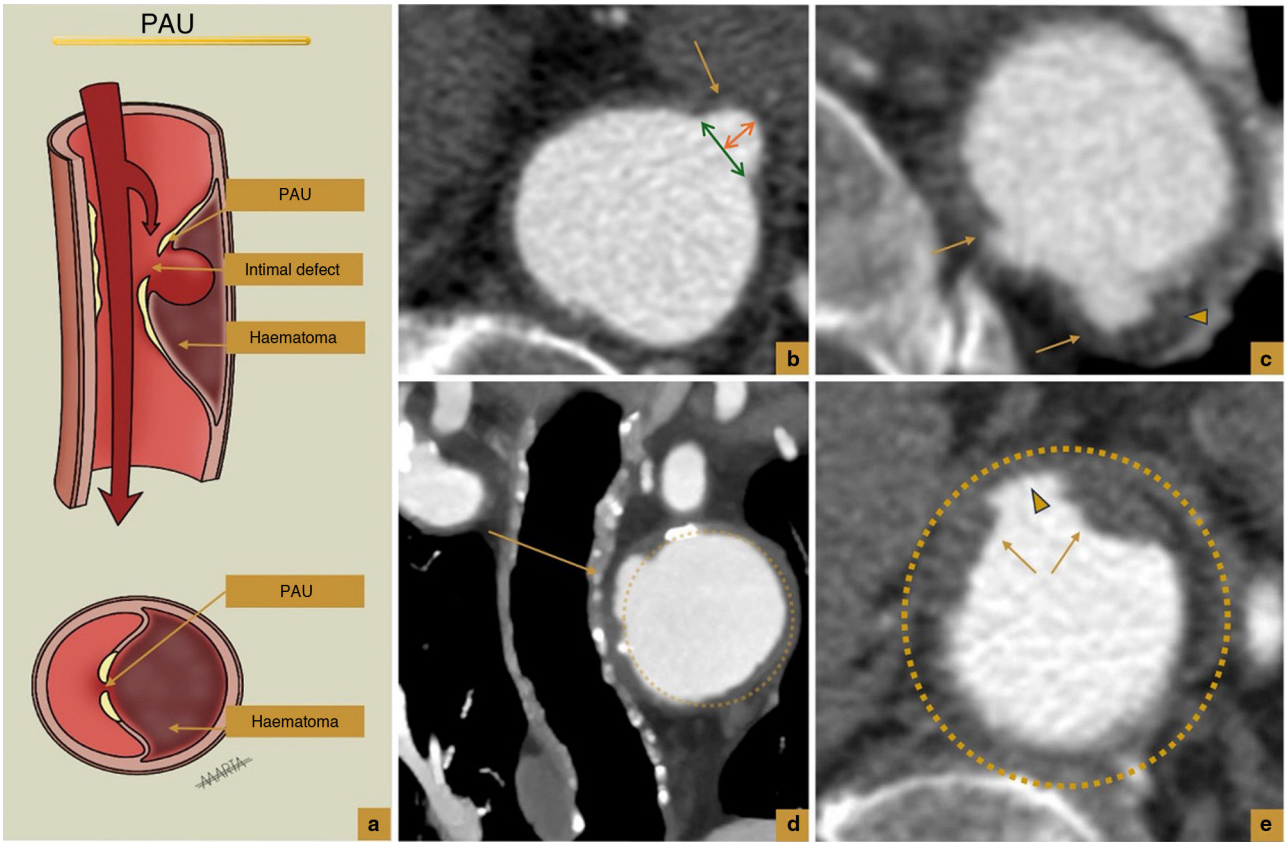


Figure 11 (a) Schematic representation of a penetrating aortic ulcer, which is the result of an ulcerated plaque which crosses the intima and reaches the elastic layer of the media, and may be accompanied by an IMH. (b and c) Contrast-enhanced CT, axial slices. Image of contrast addition which extends beyond the aortic wall and deforms its contour. This disruption in the intima facilitates the association with small haematomas in the wall (arrowhead). (b) Example of neck measurement (double green arrow) and thickness (double orange arrow) of a PAU (d) CT with contrast, coronal slice. Atheromatous plaque on the inner margin of the aortic arch with loss of aortic sphericity and contour alteration (arrow) consistent with penetrating ulcer. (e) Contrast-enhanced CT, axial slice. Ulcerated atheromatous plaque. Soft atheromatous plaque with irregular contours and ulceration up to the intima (arrowhead). Unlike penetrating ulcers, it does not alter the aortic contour.

They are usually located in the aortic segments where the deposit of atheromatous plaques is common, such as the descending thoracic aorta, the abdominal aorta and the aortic arch (in descending order of frequency). High flow in the ascending aorta plays a protective role for atheroma deposition.[2]

Imaging findings

It manifests on CT as a broad-based contrast enhanced image which exceeds the intimal calcifications and alters the aortic contour with pseudoaneurysm morphology[50] (Fig. 11). Unlike penetrating ulcers, chronic atheromatous plaques and ulcerated plaques (Fig. 11e) do not produce alteration of the wall contour, this finding being key for the differential diagnosis.[25]

Clinical course

It is important to highlight that the vast majority of PAU are not symptomatic and nor do they evolve into AAS.[5,12] The prognosis will depend on its size, presence of associated symptoms and whether there is an associated haematoma in the wall. There is currently no established cut-off point that determines the need for treatment, but it has been shown that PAU with a thickness greater than 20 mm or a neck greater than 10 mm are associated with higher rupture rates and a worse prognosis.[46,51] Therapeutic management depends on the symptoms, the location of the aortic segment involved, the associated imaging predictors (for example, size and signs of imminent rupture), and the experience of each centre. In small asymptomatic PAU, treatment is medical (aimed at reducing mechanical stress on the aortic wall) with serial imaging checks. Given the risk of aortic complications, it is recommended to perform repeat CT scans and clinical checks before the patient is discharged and at one, three, six and 12 months, and then annual follow-up in symptomatic cases and every six months in asymptomatic cases for the first three years, with subsequent annual follow-up.[5,46,52] In cases of larger or symptomatic PAU, a surgical approach will be necessary, with endovascular management being preferable to open surgery as it is associated with lower complication rates.[3,46,48,51]

Ruptured aneurysm and aortic trauma

Ruptured aortic aneurysms are a potentially life-threatening condition which often present with haemodynamic instability. One of the risk factors for rupture is the size of the aneurysmal sac, with an annual risk of rupture of 1–11% in aneurysms of 50–59 mm, 10–22% in those of 60–69 mm and 30–33% in those larger than 70 mm^3.

In suspected ruptured aneurysm, CT is the technique of choice and characteristic findings suggestive of risk of imminent rupture include irregularity of the aortic wall, wall disruption, periaortic haematomas and trabeculation of periaortic fat (Fig. 11.1). In cases of ruptured aortic

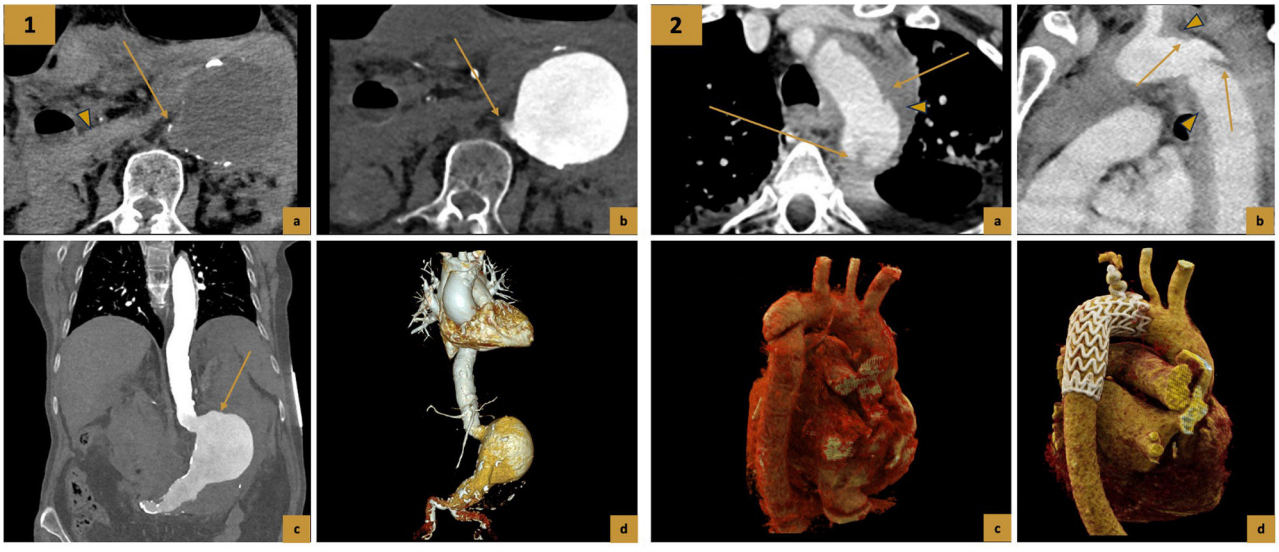


Figure 12 **1.** Ruptured abdominal aortic aneurysm. (a, b, c, and d) Non-contrast CT, axial slice, contrast-enhanced CT in the arterial phase, axial and sagittal slices, and 3D volumetric reconstruction respectively. Large aneurysmal sac with peripheral intimal calcifications, extensive haemoretroperitoneum (arrowhead), and alteration of the contour of the aneurysmal sac wall at its right lateral margin (arrows). **2.** Aortic pseudoaneurysm in the isthmus resulting from high-impact chest trauma in a road traffic accident. (a and b) Contrast-enhanced CT scan in the portal phase, axial and oblique sagittal slices. *Flap* (arrows) at the isthmus with pseudoaneurysm formation, periaortic haematoma (arrowheads), and associated haemothorax. (c) 3D reconstruction, posterior view, pseudoaneurysm in the isthmus, immediately distal to the subclavian artery exit. (d) 3D reconstruction, posterior view, result after aortic endograft implantation, excluding the chimney aneurysm in the left subclavian artery due to the short distance from the neck when fixing the prosthesis to its proximal segment.

aneurysm, treatment is always surgical, and an open or endovascular approach may be used depending on the morphological characteristics and location of the aneurysm, the age and comorbidities of the patient, and the experience of the working team.[53,54]

Traumatic injury of the aorta is a condition with a high burden of morbidity and mortality in terms of survival, and an imminent risk of death of around 80–90% of cases.[55] The most common causal mechanism is high-impact chest trauma, mostly vehicle-related,[55] which causes sudden chest deceleration, most frequently injuring the transition zone of the mobile aortic segment (ascending aorta and aortic arch) with the fixed portion (descending aorta), known as the aortic isthmus, immediately distal to the exit of the left subclavian artery.[1,3,13,56] CT is the test of choice to look for signs such as intimal flaps, aortic contour irregularities, periaortic haematoma, haemothorax/haemopericardium and formation of aneurysms (Fig. 12.2).

Conclusions

AAS is a relatively rare emergency in the Accident and Emergency department. It covers a set of inter-related conditions with a high burden of morbidity and mortality. All of them present with a similar and non-specific clinical picture of sudden onset chest/abdominal pain. CT plays a fundamental role in distinguishing between the conditions, providing a rapid diagnosis which does not delay therapeutic management, and has an immediate and emerging impact on the definitive treatment of these patients. Radiologists play a central role in the management of AAS. It is therefore important that we understand the basic concepts to enable us to collaborate with the other specialist areas, in order to achieve optimal multidisciplinary management in benefit of the patient.

CRediT authorship contribution statement

Conception and design of the study, data collection and the analysis and interpretation of the data: not applicable.

Drafting of the article and critical review of the intellectual content: SPP, SLL, MPC, ARG, SGB and PRF.

Final approval of the submitted manuscript: SPP, SLL, MPC, ARG, SGB and PRF.

Funding

This study received no specific grants from public agencies, the commercial sector or non-profit organisations.

Declaration of competing interest

The authors declare that they have no conflicts of interest.

Acknowledgements

We would like to thank the staff of the chest radiology department at Hospital Álvaro Cunqueiro, especially Dr Moure, for her dedication and commitment to teaching.

References

1. Murillo H, Molvin L, Chin AS, Fleischmann D. Aortic dissection and other acute aortic syndromes: diagnostic imaging findings from acute to chronic longitudinal progression. Radiographics. 2021;41:425–46.
2. Modares M, Hanneman K, Ouzounian M, Chung J, Nguyen ET. Computed tomography angiography assessment of acute aortic syndromes: classification, differentiating imaging features, and imaging interpretation pitfalls. Can Assoc Radiol J. 2022;73:228–39.
3. Voitle E, Hofmann W, Cejna M. Aortic emergencies—diagnosis and treatment: a pictorial review. Insights Imaging. 2015;6:17–32.
4. Wu MY, Bang TJ, Restauri N, Chawla A, Khawaja RDA, Vargas D. Imaging acute aortic syndromes. Semin Roentgenol. 2022;57:335–44.
5. Bossone E, Labounty TM, Eagle KA. Acute aortic syndromes: diagnosis and management, an update. Eur Heart J. 2018;39:739–49.
6. Vilacosta I, San Román JA, di Bartolomeo R, Eagle K, Estrera AL, Ferrera C, et al. Acute aortic syndrome revisited: JACC state-of-the-art review. J Am Coll Cardiol. 2021;78:2106–25.
7. DeMartino RR, Sen I, Huang Y, Bower TC, Oderich GS, Pochettino A, et al. Population-based assessment of the incidence of aortic dissection, intramural hematoma, and penetrating ulcer, and its associated mortality from 1995 to 2015. Circ Cardiovasc Qual Outcomes. 2018;11:e004689.
8. Mussa FF, Horton JD, Moridzadeh R, Nicholson J, Trimarchi S, Eagle KA. Acute aortic dissection and intramural hematoma a systematic review. JAMA. 2016;316:754–63.
9. Valente T, Rossi G, Lassandro F, Rea G, Marino M, Muto M, et al. MDCT evaluation of acute aortic syndrome (AAS). Br J Radiol. 2016;89:1–19.
10. Vitale J, Manfredini R, Gallerani M, Mumoli N, Eagle KA, Ageno W, et al. Chronobiology of acute aortic rupture or dissection: a systematic review and a meta-analysis of the literature. Chronobiol Int. 2015;32:385–94.
11. Reginelli A, Capasso R, Ciccone V, Croce MR, Di Grezia G, Carbone M, et al. Usefulness of triphasic CT aortic angiography in acute and surveillance: our experience in the assessment of acute aortic dissection and endoleak. Int J Surg [Internet]. 2016;33:S76–84.
12. Steinbrecher KL, Marquis KM, Bhalla S, Mellnick VM, Ohman JW, Raptis CA. CT of the difficult acute aortic syndrome. Radiographics. 2022;42:69–86.
13. 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.
14. Evangelista A, Sitges M, Jondeau G, Nijveldt R, Pepi M, Cuellar H, et al. Multimodality imaging in thoracic aortic diseases: a clinical consensus statement from the European Association of Cardiovascular Imaging and the European Society of Cardiology working group on aorta and peripheral vascular diseases. Eur Heart J Cardiovasc Imaging. 2023;24:E65–85.
15. Abbas A, Brown IW, Peebles CR, Harden SP, Shambrook JS. The role of multidetector-row CT in the diagnosis, classification and management of acute aortic syndrome. Br J Radiol. 2014;87.
16. Guideline BP [Accessed Feb 2024]. Available from: The Royal College of Emergency Medicine The Royal College of Radiologists Best Practice Guideline Diagnosis of Thoracic Aortic Dissection in the Emergency Department; 2024. p. 1–12 https://www.rcr.ac.uk/our-services/all-our-publications/clinical-radiologypublications/diagnosis-of-thoracic-aortic-dissection-in-the-emergencydepartment/

17. Bastarrika G, Schoepf UJ. El radiólogo de urgencias ante el dolor torácico agudo: ¿cómo y para qué debo utilizar los equipos TC multicorte? Radiologia. 2011;53 Suppl. 1:30–42.
18. Ko JP, Goldstein JM, Latson LA, Azour L, Gozansky EK, Moore W, et al. Chest ct angiography for acute aortic pathologic conditions: pearls and pitfalls. Radiographics. 2021;41:399–424.
19. Varela C, De Luccas V, Gutiérrez JM, Mantelli E, Ríos P, Zúñiga JM, et al. Estudio imagenológico del síndrome aórtico agudo. Rev Med Chil. 2019;147:1579–93.
20. Hinzpeter R, Eberhard M, Gutjahr R, Reeve K, Pfammatter T, Lachat M, et al. CT angiography of the aorta: contrast timing by using a fixed versus a patient-specific trigger delay. Radiology. 2019;291:531–8.
21. Husainy MA, Sayyed F, Puppala S. Acute aortic syndrome—pitfalls on gated and non-gated CT scan. Emerg Radiol [Internet]. 2016;23:397–403.
22. Yoo SM, Lee HY, White CS. MDCT evaluation of acute aortic syndrome. Radiol Clin North Am [Internet]. 2010;48:67–83.
23. Wielandner A, Beitzke D, Schernthaner R, Wolf F, Langenberger C, Stadler A, et al. Is ECG triggering for motion artefact reduction in dualsource CT angiography of the ascending aorta still required with high-pitch scanning? The role of ECG-gating in highpitch dual-source CT of the ascending aorta. Br J Radiol. 2016;89:7–9.
24. Gouveia e Melo R, Mourão M, Caldeira D, Alves M, Lopes A, Duarte A, et al. A systematic review and meta-analysis of the incidence of acute aortic dissections in population-based studies. J Vasc Surg [Internet]. 2022;75:709–20.
25. Macura KJ, Corl FM, Fishman EK, Bluemke DA. Pathogenesis in acute aortic syndromes. Am J Roentgenol. 2003;181:309–16.
26. Daily PO, Trueblood HW, Stinson EB, Wuerflein RD, Shumway NE. Management of acute aortic dissections. Ann Thorac Surg [Internet]. 1970;10:237–47.
27. Pape LA, Awais M, Woznicki EM, Suzuki T, Trimarchi S, Evangelista A, et al. Presentation, diagnosis, and outcomes of acute aortic dissection: 17-year trends from the international registry of acute aortic dissection. J Am Coll Cardiol. 2015;66:350–8.
28. Howard C, Ponnapalli A, Shaikh S, Idhrees M, Bashir M. Non-A non-B aortic dissection: a literature review. J Card Surg. 2021;36:1806–13.
29. Czerny M, Schmidli J, Adler S, van den Berg JC, Bertoglio L, Carrel T, et al. Editor's choice – current options and recommendations for the treatment of thoracic aortic pathologies involving the aortic arch: an expert consensus document of the European Association for Cardio-Thoracic Surgery (EACTS) & the European Society for Vascu. Eur J Vasc Endovasc Surg [Internet]. 2019;57:165–98.
30. DeBakey ME, Beall AC, Cooley DA, Crawford ES, Morris GC, Garrett HE, et al. Dissecting aneurysms of the aorta. Surg Clin North Am [Internet]. 1966;46:1045–55.
31. Lombardi JV, Hughes GC, Appoo JJ, Bavaria JE, Beck AW, Cambria RP, et al. Society for Vascular Surgery (SVS) and Society of Thoracic Surgeons (STS) reporting standards for type B aortic dissections. J Vasc Surg [Internet]. 2020;71:723–47.
32. McMahon MA, Squirrell CA. Multidetector ct of aortic dissection: a pictorial review. Radiographics. 2010;30:445–60.
33. Carrel T, Sundt TM, von Kodolitsch Y, Czerny M. Acute aortic dissection. Lancet [Internet]. 2023;401:773–88.
34. Jonker FHW, Patel HJ, Upchurch GR, Williams DM, Montgomery DG, Gleason TG, et al. Acute type B aortic dissection complicated by visceral ischemia. J Thorac Cardiovasc Surg [Internet]. 2015;149:1081–6.e1.
35. Sueyoshi E, Matsuoka Y, Sakamoto I, Uetani M. CT and clinical features of hemorrhage extending along the pulmonary artery due to ruptured aortic dissection. Eur Radiol. 2009;19:1166–74.
36. Guilmette J, Semionov A, Dennie C, Gahide G, Pressacco J, Fraser R, et al. Hemorrhagic infiltration of the aortopulmonary adventitia: a complication of acute aortic dissection. Eur J Radiol. 2016;85:239–47.
37. Spanos K, Kölbel T, Giannoukas AD. Current trends in aortic intramural hematoma management—a shift from conservative to a more aggressive treatment. Ann Cardiothorac Surg. 2019;8:497–9.
38. Kruse MJ, Johnson PT, Fishman EK, Zimmerman SL. Aortic intramural hematoma: review of high-risk imaging features. J Cardiovasc Comput Tomogr [Internet]. 2013;7:267–72.
39. Gutschow SE, Walker CM, Martínez-Jiménez S, Rosado-de-Christenson ML, Stowell J, Kunin JR. Emerging concepts in intramural hematoma imaging. Radiographics. 2016;36:660–74.
40. Estrera AL, Sandhu HK, Leake SS, Charlton-Ouw KM, Afifi RO, Miller CC, et al. Early and late outcomes of acute type A aortic dissection with intramural hematoma. J Thorac Cardiovasc Surg [Internet]. 2015;149:137–42.
41. Sawaki S, Hirate Y, Ashida S, Takanohashi A, Yagami K, Usui M. Clinical outcomes of medical treatment of acute type a intramural hematoma. Asian Cardiovasc Thorac Ann. 2010;18:354–9.
42. Ahn JM, Kim H, Kwon O, Om SY, Heo R, Lee S, et al. Differential clinical features and long-term prognosis of acute aortic syndrome according to disease entity. Eur Heart J. 2019;40:2727–36.
43. 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.
44. Revels JW, Akaike G, Kicska G, Cham MD. Chinese Ring-Sword sign in intramural hematoma: feature of intramural blood pool. Abdom Radiol [Internet]. 2020;45:234–6.
45. Wu M-T, Wu T-H, Lee D. Multislice computed tomography of aortic intramural hematoma with progressive intercostal artery tears. Circulation. 2005;111:92–3.
46. Dev R, Gitanjali K, Anshuman D. Demystifying penetrating atherosclerotic ulcer of aorta: unrealised tyrant of senile aortic changes. J Cardiovasc Thorac Res [Internet]. 2021;13:1–14.
47. Flohr TR, Hagspiel KD, Jain A, Tracci MC, Kern JA, Kron IL, et al. The natural history of penetrating ulcers of the iliac arteries. J Vasc Surg [Internet]. 2016;63:399–406.
48. Yugueros X, Mestres G, García-Madrid C, Riambau V. Actualización sobre las características pronósticas y el manejo terapéutico de la úlcera aórtica penetrante. Angiologia [Internet]. 2016;68:304–10.
49. Stanson AW, Kazmier FJ, Hollier LH, Edwards WD, Pairolero PC, Sheedy PF, et al. Penetrating atherosclerotic ulcers of the thoracic aorta: natural history and clinicopathologic correlations. Ann Vasc Surg. 1986;1:15–23.
50. Evangelista A, Maldonado G, Moral S, Teixido-Tura G, Lopez A, Cuellar H, et al. Intramural hematoma and penetrating ulcer in the descending aorta: differences and similarities. Ann Cardiothorac Surg. 2019;8:456–70.
51. Salim S, Locci R, Martin G, Gibbs R, Jenkins M, Hamady M, et al. Short- and long-term outcomes in isolated penetrating aortic ulcer disease. J Vasc Surg. 2020;72:84–91.
52. Kyaw H, Sadiq S, Chowdhury A, Gholamrezaee R, Yoe L. An uncommon cause of chest pain - penetrating atherosclerotic aortic ulcer. J community Hosp Intern Med Perspect [Internet]. 2016;6:31506.
53. Lojo-Lendoiro S, Calvín Álvarez P, Sobral Viñas P. Aneurisma de aorta abdominal roto, ¿qué necesitan el radiólogo intervencionista y el cirujano vascular de nuestro informe? Radiologia. 2023;65:S53–62.

54. Wanhainen A, Van Herzeele I, Bastos Goncalves F, Bellmunt Montoya S, Berard X, Boyle JR, et al. Editor's Choice – European Society for Vascular Surgery (ESVS) 2024 clinical practice guidelines on the management of abdominal aorto-iliac artery aneurysms. Eur J Vasc Endovasc Surg. 2024;67:192–331.
55. Steenburg SD, Ravenel JG, Ikonomidis JS, Schönholz C, Reeves S. Acute traumatic aortic injury: imaging evaluation and management. Radiology. 2008;248:748–62.
56. 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 [Internet]. 2010;74:24–39.