CT Features of Acute Aortic Pathology

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Learning Objectives

To illustrate the features and pitfalls of acute aortic pathology, including both traumatic and non-traumatic, with the aim to improve the ability and confidence of the radiology trainee in interpreting such findings.

Background

Acute aortic syndromes describe pathologies that share clinical and prognostic characteristics which include aortic dissection, intramural haematoma and penetrating atherosclerotic ulcer. As the clinical presentation of these pathologies may be similar, imaging plays an important role in differentiation. The underlying process is disruption of the layers of the aortic wall. The most common entity, aortic dissection, is characterised by laceration of the aortic intima, allowing blood to enter the aortic media forming a double channel with a flap separating the true and false lumina. Isolated intramural haematoma is thought to begin with rupture of the vasa vasorum that supply the aortic media, leading to intramedial haematoma whilst the intimal layer remains intact. Rarely, intramural haematoma may be post-traumatic. In penetrating atherosclerotic ulcers, atheromatous plaques erode the internal elastic lamina, burrowing through the intima into the media and may propagate transmurally to involve the adventitia. The media is exposed to pulsatile arterial flow, causing limited haemorrhage into the wall and the formation of an associated intramural haematoma [4,29]. Both intramural haematoma and penetrating atherosclerotic ulcers may serve to precede propagation to typical dissection.

Published data on incidence of acute aortic syndromes is sparse and targeted typically to aortic dissections, ranging from 2.9-3.5 per 100,000 person-years based on American and Western European populations [1,2]. It is relatively rare compared to other conditions with similar presenting complaints, with an estimation of 3 aortic dissections per 1000 patients presenting to the emergency department with chest and/or back pain [3].

Patients with type A dissections and intramural haematoma typically present with sudden onset sharp anterior chest pain rather than 'tearing' or 'ripping' pain. Type B dissections are associated with back and abdominal pain. Abruptness of onset is the most sensitive characteristic of the pain, being present in 90% of patients [4,5]. Differential pulses and a diastolic murmur of aortic regurgitation are significant examination findings at time of presentation, and have prognostic implications for poorer outcomes such as neurological deficits, coma and shock [6].
The mortality of acute aortic syndromes varies with type and location and can be as high as 91% by one week if left untreated [7]. Aortic pathologies involving the ascending aorta are surgical emergencies due to proximal extension, valvular dysfunction, tamponade, arch side-branch occlusion or aortic rupture [8]. Type A dissections have 1-2% mortality per hour during the first 48h [8,11]. Type B dissections can be managed medically with critical care support unless there are complicating features, such as side-branch occlusion, end-organ ischaemia or rupture [8].

Due to the high mortality and potential for life saving surgical intervention, prompt diagnosis is imperative. Acute aortic syndromes are recognised by the RANZCR as a 'key condition' [9].

**Imaging Findings OR Procedure Details**

**Typical Dissection**

The most common pattern of non-traumatic aortic injury is classical dissection, with an intimal flap reported in 70%-90% of cases [10,15]. The underlying pathogenesis in the majority of cases is mechanical forces, pressure and shear stress at fixed sites associated with hypertension and, ultimately, leading to a tear in the intima [29]. Less common entities that may cause dissection include connective tissue disorders, vascular inflammation, pregnancy, drug use, particularly cocaine, and iatrogenic factors, such as following intervention [11].

The Stanford Classification is the commonly used standard of classification for dissection. The proximal extent of the dissection has important implications on management. Stanford Type A dissections (Fig. 1 on page 10) are the most common variety, beginning with the intimal tear occurring proximal to the left subclavian artery and require surgical repair in all cases [12,13,15]. The intimal tear classically occurs at the lateral wall of the ascending aorta [29]. Stanford Type B dissections (Fig. 2 on page 11) begin with an intimal tear distal to the left subclavian artery and may be managed medically via aggressive blood pressure control, although individual management will vary based on associated complications [13]. The distal extent of the dissection may involve any part of the aorta and often extends into side and terminal branches.

On arterial-phase, contrast-enhanced CT a typical dissection will demonstrate division of the aortic lumen into two compartments, separated by a hypo-attenuating intimal flap.
• The cross-sectional area of the true lumen is less than that of the false lumen in a majority of cases (85%), which has been attributed to contraction of the elastic lamina [14].
• The "beak sign" (Fig. 3 on page 12 and Fig. 5 on page 14) is present in dissections and indicates the false lumen [14].
• The disruption of the aortic media may leave residual strands of incompletely separated media (Fig. 6 on page 15), termed "cobwebs," which are also highly specific to the false lumen, but relatively insensitive, present only in a minority (9%) of cases [14].
• Intimal calcification internal displacement indicates dissection (Fig. 7 on page 16).
• Circumferential dissection gives a classical "windsock" appearance, demonstrating the true lumen centrally which is enveloped by the false lumen (Fig. 8 on page 17).

Other features are less beneficial in differentiating the true and false lumen [14].

• The attenuation of contrast within each lumen depends on contrast flow dynamics and scan technique (Fig. 9 on page 18).
• The convexity of the intimal flap is non-specific, as it varies between convex and concave during the cardiac cycle secondary to complex flow dynamics between the two lumina (Fig. 10 on page 19). Scan time and technical factors, such as cardiac gating, have a direct effect on this phenomenon. The convexity may vary throughout the CT scan volume.

Complications relate to both vascular compromise and structural instability.

Dissection will commonly propagate into the branches of the aorta. Supply to end-organs may arise from the false lumen or true lumen, either of which may contribute to tissue perfusion. Side-branch occlusion may be described as static or dynamic.

• Static occlusion describes a direct extension of the dissection into side-branches. Disruption to flow may be due to pressure causing expansion of the false lumen or thrombosis of the false lumen, either of which may lead to compression of the true lumen.
• Dynamic occlusion may be more subtle with transient obstruction of side-branches during systole when pressures in the false lumen rise above those within the true lumen (Fig. 10 on page 19). This results in collapse of the dissected intima into the ostium, thus preventing systolic perfusion. Dynamic occlusion may not always be evident on CT, but may be implied by the configuration of the dissection and can be confirmed with dynamic MR imaging.

The ultimate outcome to tissues lies on a spectrum between fully viable to ischaemia and necrosis. These complications can be lethal in Type A dissections due to potential
involvement of the coronary arteries, leading to myocardial infarction, and arch side-branches supplying the brain.

The most significant structural complication is that of rupture (Fig. 11 on page 20) and has been described as the cause of fatality in up to a third of cases [15,16]. This is a devastating outcome with a high associated mortality and morbidity. Rupture may be into the mediastinum, pleural space, retroperitoneum or pericardium (Fig. 12 on page 21). Retrograde pericardial haemorrhage and aortic valvular dysfunction is unique to Type A dissection with a significant risk of cardiac insufficiency and tamponade [17].

**Intramural Haematoma**

Intramural haematoma (IMH) presents and is managed similar to typical dissection, but is radiologically distinct [20]. The features of intramural haematoma are subtler than dissection and can missed without a high index of suspicion.

The underlying pathogenesis of spontaneous intramural haematoma begins with rupture of the vasa vasorum feeding the aortic media leading to intramural haemorrhage. Compared to typical dissection there is no direct communication with the aortic lumen. Propagation occurs relative to the site of initial haemorrhage and may be retrograde, antegrade or both. The configuration is again described as per the Stanford classification depending on the section of involved aorta. While classical teaching suggests equivalent treatment to typical dissection based on this classification, there is debate as to the extent that some Type A IMH may be individualised for more conservative medical management [18,21,24,28]. Type B IMH has a more favourable long-term prognosis than Type A IMH [19]. The natural history and development of complications are variable. IMH may regress, stabilise or completely resolve. Alternatively, the disease may progress, either rapidly or slowly, even when the intramural haematoma itself regresses [20,23].

In contrast to the detection of dissection, intramural haematoma is best demonstrated on non-enhanced CT as the presence of contrast within the aorta can obscure the features of IMH.

**Features on non-enhanced CT:**

- Un-enhanced blood is normally of similar attenuation to the wall and often indistinct. In the setting of intramural haematoma, the coagulated blood within the wall will be notably higher attenuation that that of flowing blood. On CT this classically manifests as a crescentic area of high attenuation eccentrically located along the aorta (Fig. 13 on page 21
The crescentic appearance is not essential and may occasionally be circumferential.

- Wall thickening with centrally displaced intimal calcification should be evident despite the presence of contrast material. Although this finding is also seen in dissection, the aortic media should not show contrast opacification nor will an intimal flap or tear be evident (Fig. 14 on page 22).
- Intramural haematoma causes aortic wall thickening, which is usually smooth. Atherosclerotic plaque and mural thrombus also cause wall thickening. Atherosclerotic plaque can be distinguished by irregular or nodular thickening (Fig. 14 on page 22). Mural thrombus typically occurs in a dilated vessel and is usually hypo-dense on a non-contrast scan.

Contrast enhanced CT should always be performed when aortic syndromes are suspected. Although the diagnosis is not made on enhanced CT, the following should be confirmed:

- Absence of an intimal tear or direct communication with the aortic lumen is the defining feature of this pathology.
- No contrast should be present within the aortic media to indicate dissection.
- Penetrating atherosclerotic ulcer (discussed below) should be differentiated. Although there is considerable overlap, important distinctions exist which impact on prognosis and management [27,28]

Various complications are well established despite considerable debate on clinical course and variability [20-24,29]:

- Progressive weakening of the intima may lead to intimal tear and subsequent conversion to typical dissection with associated potential complications. The risk of conversion to dissection and rupture is higher in Type A IMH, while Type B may be treated more conservatively.
- Outward rupture through the adventitia may occur with resultant extravasation or pseudoaneurysm formation.
- Cardiac tamponade may occur in IMH involving the ascending aorta.
- Development of aortic aneurysm which may be saccular or fusiform. This may occur even following resolution of the IMH.

**Penetrating Atherosclerotic Ulcer**

Penetrating atherosclerotic ulcer (PAU) occurs in the setting of established atherosclerotic disease and, therefore, predominantly occurs in elderly patients [29,30]. The offending lesion begins as an intimal plaque which progressively erodes through the
intimal layer into the media. Haemorrhage occurs into the media at the site of penetration but is initially limited by pre-existing fibrosis caused by atheromatous changes [25]. On imaging, the intramural haematoma accompanying a PAU is limited to the region of the ulcer. Features important for the diagnosis of PAU are best demonstrated with multi-phase CT including non-enhanced and arterial phase of intravenous contrast.

The natural history of penetrating atherosclerotic ulcer is less well established than dissection and intramural haematoma. Similar to IMH there may be progression to fusiform or saccular aneurysm with potential for complication by true dissection or rupture. Rupture is a life-threatening complication which is thought to be more prevalent in penetrating ulcers than in typical dissection [26]. Most studies suggest a poorer prognosis from penetrating ulcers than other aortic syndromes [27,30], while a few suggest slow progression and low prevalence of severe complications [30]. One study reports rupture rates as high as 40%, compared to Type A dissection (7.0%) and Type B dissection (3.6%) [26]. Due to the increased risk of life-threatening complications and poor prognosis, penetrating ulcers are typically treated invasively, such as with endovascular stenting. As IMH has a more favourable prognosis and may be treated medically, particularly when involving the descending thoracic aorta, the distinction between primary IMH and penetrating atherosclerotic ulcer is essential to offering the optimal management [27,28].

Features on non-enhanced CT include [29,30]

- Established atherosclerotic disease as evidenced by the presence of aortic intimal calcification, which is usually marked.
- In contrast to dissection and IMH, penetrating ulcers are more common in the descending thoracic aorta, but may occur at any site where atherosclerotic plaques exist [31].
- Aortic mural thickening (>7mm) associated with a focal area of hyper-attenuation representing limited intramural haematoma is seen in a majority of cases (Fig. 15 on page 22) [31].
- Centrally displaced intimal calcification should be present but does not allow differentiation between dissection, IMH and PAU.

Additional information provided by enhanced (arterial phase) CT include

- Saccular or crater-like contrast extending into a thickened aortic wall extrinsic to the aortic lumen (Fig. 15 on page 22).
- Contrast should extend deep to the level of intimal calcification. Care should be taken to ascertain the presence of this features as ulceration confined to the intimal layer does not convey the same prognostic and management implications.
• Absence of intra-medial contrast should be confirmed to differentiate from dissection.
• Pseudo-aneurysm or frank extravasation with high attenuation contrast extrinsic to the aortic lumen may be present in the setting of acute rupture.

**Acute Traumatic Aortic Injury**

The pathogenesis of acute traumatic aortic injury (ATAI) is distinct from the non-traumatic syndromes described above. However, there is some overlap with pathological outcome and, therefore, with the imaging features. Acute aortic injury occurs in high-velocity blunt trauma resulting in violent deceleration with a majority of cases occurring in motor vehicle collisions above 50km/h [32,33]. The remainder are largely represented by falls from considerable height and crush injuries [37].

The mechanism of ATAI is incompletely understood, although various complex mechanisms have been suggested. The mechanism is likely a combination of hydraulic and mechanical forces. Mechanical stress at sites of relative immobility of the aorta occur at the ligamentum arteriosum, aortic root and diaphragm. When injury occurs it usually manifests as transverse tears of the aorta at these particularly vulnerable locations. Tears can involve all or part of the circumference. The majority of ATAI that reach the hospital are partial tears involving only the inner two layers of the aortic wall. Conversely, transmural tears, or complete transection, involve the adventitia and are rapidly fatal due to massive haemorrhage with death usually occurring at the scene [37].

Contrast enhanced, multi-detector row CT performed in the arterial phase is regarded as the investigation of choice for the diagnosis and evaluation of ATAI with direct features of aortic injury approaching 100% specificity when present [34-36]. These include:

• Presence of an intimal flap. Despite an intimal flap frequently being evident, a true "double-barrel" dissection is uncommon. The most common site is the aortic isthmus (Fig. 16 on page 22), while injuries to the aortic root are likely under-represented due to high incidence of death at the scene [37].
• A pseudo-aneurysm or contained rupture (Fig. 16 on page 22). Contrast will be seen extending beyond the margins of the aortic lumen. Active extravasation may be seen, but is rare, often indicating impending exanguination.
• Unexplained contour abnormality of the aorta, best seen on multiplanar reformats.

Due to the mechanism, traumatic aortic injury rarely occurs in isolation. Indirect signs should serve to raise the index of suspicion for ATAI, but also increase false positive rates.
when direct signs are absent. Indirect findings largely refer to mediastinal haematoma. Peri-aortic haematoma should be evident with particular care to assess for loss of normal intervening fat planes between the haematoma and aortic wall. Sites of review should include the aortic root and pericardium, aortic isthmus and retrocrural space. Preserved fat planes may indicate that the haemorrhage does not originate from the aorta and should prompt careful review of other thoracic vessels. Co-existing thoracic and abdominopelvic injuries are independently predictive of ATAI, but lack specificity [38].

While the majority of those that receive definitive endovascular or open surgical management are likely to survive [39-41], the ongoing imaging of ATAI is generally that of the post-surgical aorta.

**Pitfalls**

Pitfalls in imaging may be encountered during evaluation of the acute aorta which may either simulate or mask disease. While some of these may be obvious, there exists the potential for some to pose a diagnostic dilemma to the radiology trainee. These pitfalls may be attributed to a variety of causes including technical factors, artefacts, normal peri-aortic structures and anatomical variants [42]

- Improper timing of contrast material - Appropriate timing and high flow rate of the contrast bolus is essential to diagnosis of dissection. Insufficient enhancement may produce a false-negative diagnosis if there is inadequate contrast resolution to differentiate the presence of an intimal flap (Fig. 18 on page 24).
- Flow related artefact - Related to timing of contrast bolus and complex flow dynamics in the false lumen. Relative to the site of intimal tear, the flow of blood and contrast may be antegrade, retrograde or both and may be further complicated by potential for to-and-fro motion in the false lumen throughout the cardiac cycle. Insufficient time for adequate opacification may lead to misinterpretation as thrombosis (Fig. 19 on page 26 and Fig. 20 on page 27). Similar phenomena occurs in large pseudo-aneurysms, the size of which may be grossly underestimated. Such artefacts may be reduced or avoided with utilisation of delayed-phase scans or a pre-bolus of contrast.
- Streak artefact - Produced as a largely unavoidable consequence of the back-projection technique in image reconstruction, these appear as linear streaks directed from high attenuation or high-contrast interfaces (Fig. 21 on page 24). This includes metal (eg. surgical staples) and undiluted contrast. Undiluted contrast within the left brachiocephalic vein or within a central venous catheter in the SVC may produce streak artefact over the ascending aorta, simulating Type A dissection. A true intimal flap should be smooth and sharply defined with a clear interface with, and not extending beyond, the aortic wall.
• Motion artefact - Respiratory motion and, in particular, cardiac motion will produce artefact due to overlap of structures occupying the same space at different times over the duration of the rotation, which are then averaged into a single slice. The aortic root is particularly predisposed to cardiac motion artefact and, therefore, may be misinterpreted as Type A dissection (Fig. 22 on page 25). An example includes the aortic annulus or valve leaflets appearing in the region of the proximal ascending aorta (Fig. 23 on page 29). Cardiac gating will help to reduce the appearance of these artefacts by only including images from a particular phase of the cardiac cycle. However, this has the draw-back of increasing radiation dose. Further, not all phases of the cardiac cycle are free of artefact and additional phases may need to be reconstructed to optimise visualisation (Fig. 24 on page 26). Appearances are similar to streak artefact and can be differentiated from true pathology on the same basis noted above.

• Normal peri-aortic structures - Numerous normal peri-aortic structures have been described a potential mimics for pathology. The origin of the arch vessels, left brachiocephalic, superior intercostal, bronchial and pulmonary veins may be misinterpreted to represent a false lumen or intimal flap (Fig. 25 on page 28). The superior pericardial recess, atelectasis or consolidated lung, residual thymus, pleural effusions or thickening could also be thought to represent dissection, haematoma or extravasation (Fig. 19 on page 26). Many of these are unlikely to represent a diagnostic problem with modern availability of MDCT, thin section images and multiplanar reformats.

• Atherosclerotic plaque and mural thrombus - The presence of atherosclerotic wall thickening or mural thrombus can mimick dissection. IMH and dissection should demonstrate smooth margin and curvature, which can often be contrasted with an irregular or lobulated contour of mural thrombus (Fig. 14 on page 22). Presence of centrally displaced calcification is a reliable indicator of dissection or IMH in the acute setting.

• Ductus bump or non-patent ductus arteriosus remnant - A developmental normal variant occurring at the aortic isthmus which can simulate traumatic transection and can pose particular difficulty when appearing in the context of trauma. This variant is seen at the same location as the most common site for transection to occur. This appears as a out-pouching at the anteromedial aspect of the aorta at the site of developmental insertion of the ductus arteriosus. In contrast to transection, a ductus bump should have smooth, rounded shoulders at the interface with the aorta (Fig. 17 on page 23). Conversely, acute margins, contour irregularity and presence of an intimal flap are direct features of transection. An indirect sign of peri-aortic haematoma should be considered highly suspicious. [43,44]
Fig. 1: Stanford Type A dissection. Curvilinear low attenuation intimal flap is seen within ascending (black arrowhead) and descending aorta (white arrow). The ascending aortic intimal flap has a tear.
Fig. 2: Stanford Type B dissection with rupture and active contrast extravasation in sagital-oblique section. The dissection flap commences at the left subclavian artery. The flow in the false lumen is slower than in the true lumen. CT scan has been acquired in phase on contrast where the false lumen is of higher attenuation than the true lumen. Black arrow-head indicates site of extravasation and adjacent low density indicates mediastinal haematoma.
**Fig. 3:** Section of descending thoracic aorta in Type B dissection demonstrates intimal flap. The false lumen on the left and is evident due to the "beak sign" (arrowhead).
Fig. 4: Patient with Type A dissection treated with ascending aorta interposition graft. There is a residual dissection flap in the arch and descending aorta. The interposition graft feeds both the true and false lumina. White arrow indicates residual dissection flap adjacent to the upper anastomosis.
**Fig. 5:** Cross-sectional CT showing dissection within the abdominal aorta. The false lumen is on the left with acute angles at the margins (arrowheads). This is the "beak sign." In this case, the true lumen is smaller, which is the more common scenario, but is not always the case.
Fig. 6: Stanford Type B dissection demonstrates incomplete separation of a strand of media within the false lumen (white arrowhead). This is the "cobweb sign," which indicates the false lumen. In this case, the true lumen is larger than the false lumen, but this is a dynamic situation and may change with the cardiac cycle.
Fig. 7: Selected image from ruptured Stanford Type B dissection demonstrates centrally displaced intimal calcification in the descending thoracic aorta (white arrow). The false lumen on the left is crescent-shaped. There is differential contrast enhancement of the two lumina. There is mediastinal haematoma (white arrowheads).
Fig. 8: "Windsock" appearance of circumferential Type A dissection on cross-sectional CT at level of aortic arch. Notice equivalent attenuation within true lumen (black arrowheads) and false lumen (white arrow).
Fig. 9: Sagital section of contrast enhanced CT shows a Type B aortic dissection. True lumen enhancement occurs earlier than false lumen enhancement. There is differential contrast enhancement between the two lumina. In the upper descending thoracic aorta the false lumen has relatively low attenuation while in the lower descending aorta it is of higher attenuation than the true lumen. This scan was performed on a slow 16-slice CT from cranial to caudal, accounting for longer acquisition time and differential enhancement.
**Fig. 10:** Cine MR imaging of Type B aortic dissection. Images (a) and (b) are at the level of the upper thoracic aorta. Images (c) and (d) are at the level of the upper abdominal aorta. Images (a) and (c) show the relatively larger size of the true lumen in systole compared to (b) and (d) where there is collapse of the true lumen in diastole.

**Fig. 11:** Contrast enhanced CT scan with patient with aortic dissection shown at the level of the upper ascending/descending aorta and at the level of the main pulmonary artery.
This shows aortic rupture from the lateral wall of the descending aorta (arrowhead), active contrast extravasation and mediastinal haematoma (arrows).

**Fig. 12:** Non-enhanced CT at the level of main pulmonary artery shows relatively high attenuation mediastinal haematoma compared to blood (arrowhead).
**Fig. 13:** Non-enhanced (left) shows relatively high attenuation "crescent sign" of intramural haematoma within the wall of the descending thoracic aorta (arrowhead). Contrast in the aorta on arterial phase contrast enhanced CT (right) masks the appearance of the haematoma. Wall thickening and intimal calcification internal displacement remains evident despite contrast.

![Fig. 13](image13.png)

**Fig. 14:** Selected axial images from arterial phase contrast enhanced CT shows atherosclerotic plaque (left), intramural haematoma (middle) and dissection (right). The atherosclerotic plaque is irregular with peripheral calcification. Intramural haematoma is smooth with centrally displaced calcification. The dissection shows differential enhancement of the true and false lumina. There is evidence of rupture and mediastinal haematoma in the dissected image.

![Fig. 14](image14.png)

**Fig. 15:** Non-enhanced axial CT (left) at the level of the aortic root shows limited intramural haematoma (arrowhead) and centrally displaced intimal calcification. Arterial phase contrast enhanced CT (right) at the same level shows crater-like area of contrast extending deep to the level of intimal calcification. This represents a penetrating atherosclerotic ulcer.

![Fig. 15](image15.png)
Fig. 16: Selected images from a contrast enhanced CT performed in the setting of a high speed motor vehicle accident. Axial and sagital slices shown are targeted to the region of the aortic isthmus. There is a traumatic isthmus pseudo-aneurysm and mediastinal haematoma. Arrows highlight the intimal flap.
**Fig. 17:** Sagital slices from arterial phase, contrast-enhanced CT highlighting the distal aortic arch and descending thoracic aorta. There is a prominent ductus bump (arrows). There is no acute aortic pathology. This was reported as a normal finding and was unchanged on subsequent imaging.

**Fig. 18:** Axial CT slices at the level of the main pulmonary trunk obtained during contrast bolus tracking. The image on the right shows differential attenuation between the true and false lumina and the intimal flap. The image on the left shows isodense contrast making differentiation of the intimal flap difficult.
Fig. 21: Axial contrast enhanced CT at the level of the aortic root. Contrast has been injected through a peripherally inserted central catheter (white arrow). There is streak artefact simulating an intimal flap in the ascending aorta (black arrowhead). Cardiac motion causes double-wall appearance of the left ascending aortic wall (above black arrowhead).
**Fig. 22:** Contrast enhanced axial CT at the level of the aortic root in the same patient. Non-gated CT (left) shows double aortic wall appearance in the ascending aorta (arrowhead) due to cardiac motion. This artefact is not present with ECG-gated CT (right).

**Fig. 24:** Axial slices from CT angiogram at the aortic root performed with retrospective ECG-gating. All images are reconstructed at the same level. The 70%, 75% and 80% phase reconstructions show aortic root motion artefact (arrowhead). Artefact is eliminated in the 40% early diastolic phase (top right).
**Fig. 19**: Series of axial images from contrast enhanced CT in a post-surgical patient with an ascending aortic interposition graft. There is a dissection flap in the descending aorta with faint enhancement within the false lumen posteriorly (arrowheads). There is no contrast in the superior false lumen, which may suggest slow flow or thrombosis. This can be avoided by giving a pre-bolus or imaging with a longer delay. Left lower lobe collapse may mimic contrast extravasation (white arrow). Pleural effusions and mediastinal haematoma is also present.
Fig. 20: Coronal and sagital slices from arterial phase, contrast-enhanced CT from the same study shown in the previous image. This further illustrates the differential contrast gradient within the false lumen. The superior portion does not contain contrast. This may represent thrombosis or slow flow.
**Fig. 25:** Sagital slice from CT angiogram profiling the aortic arch. A bronchial artery simulates contrast within the aortic wall (arrowhead). There is no acute aortic pathology.
**Fig. 23:** Selected axial slices from contrast enhanced CT at the level of the aortic root. Three artefacts are shown. The aortic cusp margins simulate a dissection flap (arrowhead). There is also cardiac motion artefact at the aortic root and streak artefact from undiluted contrast arriving from the SVC.
Conclusion

Acute aortic syndromes include traumatic and non-traumatic pathologies. CT scanning is the mainstay of diagnosis at most institutions. Familiarity with the CT signs is important for the radiology trainee to alleviate uncertainty and for correct diagnosis.

Personal Information

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References


