Imaging thoracic emergencies

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Introduction
Imaging, mainly using CT, plays a pivotal role in the assessment of patients presenting with severe pain arising from non-traumatic and traumatic disorders of the thoracic aorta. This article illustrates the different types of thoracic aortic pathology encountered in clinical practice.

Acute aortic syndrome
Acute aortic syndrome (AAS) is a term used to describe three closely related entities affecting the thoracic aorta. These are classic aortic dissection (CAD), intramural haematoma (IMH) and penetrating atherosclerotic ulcer (PAU). Vilaiesta and Roman first used this definition in an editorial in Heart in 2001,1 and later included incomplete dissection. Other authors have also incorporated ruptured aortic aneurysm and aortic transection, but in this article we will limit our discussion to the original definition.

The original three entities usually exist separately but may also coexist. In some patients, IMH may evolve into aortic dissection and PAU may act as the entrance tear for an aortic dissection. They are clinically indistinguishable as typically they present as interscapular chest pain in chronically severe hypertensive patients. As a cause of chest pain, AAS represents the spectrum of conditions most likely to result in death due to aortic rupture.2 These syndromes are non-trauma-related.

The main aim of radiological interpretation is not only to detect which entity but also to differentiate between their classification, Stanford type A or B, as this has an impact on how the patient is managed.

i) Classic aortic dissection
CAD is a life threatening condition requiring immediate diagnosis and treatment otherwise death results. It is the most common entity causing AAS and has an incidence of three to four per 100,000 persons/year.3 Pathologically, there is an intimal tear at the proximal end also known as the entrance tear. This propagates distally in a longitudinal fashion forming the double layer identified by the intimal flap. Distally, the false lumen re-enters the true lumen through another intimal or re-entrance tear, usually at the level of the abdominal aorta or iliac vessels. The re-entrance tear is difficult to visualise using any imaging technique.2

Aortic dissection may be described as acute, if symptoms last less than two weeks, or chronic if lasting longer. Several classification systems have been used to describe CAD. DeBakey, who was a cardiac surgeon and himself a sufferer of aortic dissection, devised the original classification. The Stanford classification later superseded this. In Stanford type A, the dissection involves the ascending aorta and aortic arch. It may or may not involve the descending aorta and accounts for 75% of CAD. This should be treated surgically or by endovascular repair to protect vital organs, namely the brain and heart, from retrograde dissection. Stanford type B is a dissection commencing distal to the left subclavian artery. This type of dissection can be treated medically with antihypertensive medication in the absence of rupture or ischaemic complications.4

Acute type A aortic dissection has a reported mortality rate of approximately 1% per hour if untreated, with 50% mortality by day three, and almost 80% by two weeks. Death rates are lower in acute type B aortic dissection with 10% mortality at 30 days but 70% or more if in the highest risk group.

The chest radiograph has a sensitivity and specificity of 64% and 86% respectively5 and hence cannot rule out dissection. One would expect to see widening of the mediastinum and if intimal calcification is present, it will be displaced by more than 5mm from the aortic border. This is also known as the ring sign and is considered to be a specific radiographic sign. CT, however, has a sensitivity and specificity of nearly 100% and can depict an intimal flap in 70% of cases. It is important to differentiate the true from false lumen for surgical planning. Features that can be useful in identifying the true lumen are a smaller cross-sectional area (due to the difference in pressures) and intimal calcification that is located adjacent to the flow channel. The false lumen can be seen to wrap around the true lumen in a spiral fashion, which is quite important to differentiate from mural thrombus, which maintains a congruous relationship with the aortic wall giving a crescent shape. The false lumen also demonstrates a smooth internal border in contrast to thrombus, which has an irregular border. Other features include the ‘beak sign’ where the intimal flap forms an acute angle with the outer wall of the dissection flap, and ‘aortic cobwebs’ which are strands of media that are incompletely sheared during the propagation of a dissection flap6 (figure 1). Beware of artefacts mimicking a dissection flap caused by streak artefacts from contrast in the superior vena cava, pulsation artefacts particularly if the scan is non-cardiac gated, improper timing of contrast and peri-aortic structures.7

ii) Intramural haematoma
IMH was first described by Krukenberg in 1920 and follows an atherosclerotic plaque, eroding the inner layer of the aortic wall reaching the media where it produces a haematoma. It occurs in patients with severe
atherosclerosis. It is located mainly in the aortic arch and descending thoracic aorta. Atheroma is less common in the ascending thoracic aorta as it is protected by the high velocity blood flow. PAU is often multiple, therefore surgical treatment is difficult and complex, and associated with higher morbidity hence most are treated medically. Extensive atheroma is seen on the unenhanced CT associated with high density IMH reflecting its aggressive nature. When contrast is given, it is seen pooling in the ulcer crater deep into the media. PAU leading to rupture through the adventitia is rare but may result in pseudoaneurysm formation or dissection which tends to be more localised than CAD, as propagation is limited by the atheromatous plaques. PAU is thought to represent a prelude of most aortic saccular aneurysms and the need for surveillance is recommended.

**Thoracic aortic aneurysm**

A true aneurysm is defined as permanent abnormal focal dilatation of the thoracic aorta, which contains all three anatomical layers of the vessel wall and is most commonly due to atherosclerosis. It is known that the aortic diameter increases with age, nevertheless the normal diameter of the mid ascending aorta should always be less than 4cm, and the descending aorta should be no more than 3cm. Often, it is detected as an incidental finding on a chest radiograph.

A false aneurysm has fewer than three of the anatomical layers involved. It is typically saccular with a narrow neck and is most commonly due to trauma but may also be secondary to penetrating atherosclerotic ulcers or infection, otherwise known as mycotic aneurysms. Post-traumatic pseudoaneurysm should not be confused with a ductus diverticulum, a normal variant. Both are located at the isthmus but the latter is seen as a focal, smooth bulge along the antero-inferior surface of the aortic arch and forms an obtuse angle with the aortic wall.

The risk of rupture of a thoracic aneurysm increases with size, therefore surveillance of aneurysms is advocated. Rupture is best assessed with CT and is identified by high density IMH reflecting its aggressive nature.

**Tramautic aortic injury**

It is estimated that 75-80% of thoracic aortic injuries are as a result of high-speed road traffic collisions with most occurring after rapid deceleration above 50km/h. It is a fatal condition as 85-90% of patients die before reaching hospital and approximately 50% of those who survive and reach hospital die within one week if not treated appropriately. Endovascular stenting is now the treatment of choice.

Typically, the sites of thoracic aortic injury occur at aortic attachments located most commonly at the proximal descending aorta or isthmus in more than 90% of cases, followed by the aortic arch, aortic root and distal descending aorta at the diaphragmatic hiatus. The mechanism is thought to be multifactorial – osseous pinch of sternum with thoracic spine, torsion and hydrostatic forces acting on the aortic isthmus which is the weak point.

Signs of suspected thoracic aortic rupture on chest radiography include mediastinal widening (>6cm), irregular aortic margin, right deviation of the trachea, depression of the left main bronchus and a left apical pleural cap. The left apical cap is the result of blood migrating from the mediastinum into the extrapleural space (figure 4).

Partial transection seen as traumatic pseudoaneurysm, intimal flap, abnormal aortic contour and sudden change in calibre are direct signs of traumatic aortic injury, usually accompanied by mediastinal haematoma (figure 5).

**Summary**

Imaging is vital in the assessment of disorders of the thoracic aorta. In non-traumatic cases, an unenhanced CT scan of the thorax is recommended to detect intramural haematoma. The Stanford classification is used to determine therapy – medical or surgical. In the traumatic setting, CT is highly sensitive and specific in determining the presence of aortic injury and as a planning study prior to stent placement.

**References**

Figure 2
Unenhanced axial sequences (A) showing crescent shaped IMH (arrow) and haemopericardium (*). This is much harder to appreciate on the post contrast sequences (B) at the same level hence the importance of performing an initial unenhanced scan. There is also an associated acute dissection in the descending thoracic aorta. Courtesy of Dr Dan Barnes, Glenfield Hospital, Leicester.

Figure 3
Leaking thoracic aortic aneurysm diagnosed by the presence of mediastinal haematoma (*) and a left haemothorax with layering of haemorrhage posteriorly (white arrow).

Figure 4
Chest radiograph as seen in traumatic aortic rupture demonstrating mediastinal widening, left lung contusions and a left apical pleural cap.
Figure 5
Axial (A) and coronal (B) CT of partial aortic transection at the isthmus (black arrow). There is active contrast extravasation (white arrow) resulting in mediastinal haematoma (*) and left pleural effusion.