Acute aortic syndrome: surgical, endovascular or medical treatment

Acute aortic syndrome encompasses classic aortic dissection, intramural haematoma, penetrating aortic ulcer, and iatrogenic or traumatic transection of the aorta. These acute aortic pathologies present with characteristic 'aortic pain', and are managed by several operative and endovascular techniques.

The term acute aortic syndrome embraces a heterogeneous group of aortic conditions where patients present acutely with characteristic chest or thoracic pain (ripping or tearing 'aortic pain') caused by one of several acute thoracic aortic pathologies. These include classic aortic dissection (with true and false lumens), intramural haematoma of the aortic wall, subtle dissection (typically found intraoperatively in Marfan patients), laceration of the aortic wall without dissection of the aortic media (incomplete dissection), penetrating aortic ulcer, and iatrogenic or traumatic transection of the aorta. In some patients, acute aortic syndrome may also be caused by symptomatic degenerative aortic aneurysm (Svensson et al, 1999, 2008). Any of these acute aortic syndrome pathologies may progress to frank aortic rupture, may precede each other or may simply coexist.

Presentation and epidemiology of acute aortic syndrome

Acute aortic syndrome is the most frequently fatal condition in patients with chest pain. Patients with acute aortic syndrome are characterized by aortic pain and a long-lasting history of severe hypertension. A severely intense, acute, tearing or ripping, pulsating and migratory chest pain suggests that the patient may have an acute aortic syndrome. In contrast with the more gradual increasing intensity of pain caused by coronary syndromes, pain from acute aortic syndrome has a sudden onset with maximal intensity often at the time of onset. Chest pain radiating to the neck, throat or jaw indicates that the aortic segment involved is the ascending aorta, whereas pain located in the back or the abdomen suggests that the diseased segment is most probably the descending aorta. The existence of a murmur of aortic regurgitation and pulse differentials may be used as additional diagnostic clues of acute aortic syndrome (Vilacosta et al, 2009).

Since aortic pain may be confused with that of acute coronary syndromes, laboratory tests, electrocardiographic abnormalities and chest X-ray examination may be helpful in differentiating those (Ohlmann et al, 2006). The acute rise in plasma concentrations of D-dimer and the absence of electrocardiographic changes favour the presence of acute aortic syndrome whereas a rise in myocardial enzymes with electrocardiographic changes are indicative of an acute coronary syndrome. D-dimer levels correlate significantly with acute aortic syndrome and are higher in patients with classic dissection than in those with intramural haematoma (Ohlmann et al, 2006). Aortic-specific clinical examination (Ashrafian, 2006), systemic vascular review (Ashrafian, 2007) and chest radiographs are usually performed in patients with acute chest pain.

It is important to remember that a normal chest radiograph does not exclude the presence of an acute aortic syndrome (von Kodolitsch et al, 2004). Several imaging modalities have been used in the assessment of patients with acute aortic syndrome, such as computed tomography, magnetic resonance and transoesophageal echocardiography. The combination of morphological and metabolic information provided by positron emission tomography and computed tomography imaging seems to be helpful in identifying patients with acute aortic syndrome with an increased risk for disease progression (Kuehl et al, 2008) (*Figure 1*).

From a surgical and prognostic standpoint, patients with raised aortic diameter can be categorized into predictive risk groups based on aortic size index (Ashrafian, 2008), while those presenting with acute aortic syndrome may be classified into two groups depending on the aortic segment involved: proximal acute aortic syndrome if the affected segment is the ascending aorta and/or the aortic arch, or distal acute aortic syndrome if the involved segment is the descending aorta (Vilacosta and Roman, 2001).

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Intramural haematoma

Intramural haematoma is commonly defined as bleeding into the outer layers of the aortic media, presumably caused by rupture of aortic vasa vasorum, which are prone to rupture as a result of alterations from chronic arterial hypertension (Erbel et al, 2001). By definition, intramural haematoma lacks a detectable intimal tear or disruption, and therefore has no communication with the aortic lumen, but is confined within the aortic wall.

Intramural haematoma accounts for approximately 6–10% of all acute aortic syndromes according to a European cohort (Evangelista et al, 2005) but can account for 30–40% of acute aortic syndrome patients in some Asian populations (Song et al, 2001). Symptoms of intramural haematoma may be very similar to those of classic dissection, and patients cannot be reliably distinguished by clinical presentation alone (Erbel et al, 2001). However, the risk of malperfusion is lower with intramural haematoma and thus symptoms of organ ischaemia may be absent in these patients. The natural history of intramural haematoma is not fully understood, but some patients undergo spontaneous reabsorption of intramural haematoma under medical treatment, although regression is less common.

Imaging

It is thought that intramural haematoma has probably been underdiagnosed in the past, because it is difficult to diagnose with angiography alone. The use of non-contrast computed tomography images is paramount in detection of an intramural haematoma: discrimination of an intramural haematoma from a dissection on a contrast-enhanced computed tomography alone is more difficult because the attenuation of acute haemorrhage in the wall of the aorta is higher than that of intraluminal blood. Magnetic resonance imaging is also useful for detecting intramural haematoma, as the signal characteristics of haemoglobin breakdown products help distinguish between acute and chronic blood (Birchard, 2009). In addition, transoesophageal echocardiography provides important information for diagnosing intramural haematoma.

Treatment

The management of intramural haematoma is not well established although it is similar to that for aortic dissection. Involvement of the ascending aorta (type A intramural haematoma) demands urgent surgical repair because of the risk of rupture or progression to frank dissection in other parts of the thoracic aorta (Eggebrecht et al, 2009). Groups from Japan and Korea have documented a benign course of type A intramural haematoma resulting in non-operative management (blood pressure control and 4-week bed rest) with imaging surveillance (Song et al, 2001; Evangelista et al, 2005). Intramural haematoma confined to the aortic arch or the descending thoracic aorta (type B intramural haematoma) may primarily be safely treated non-surgically with close imaging follow up (Eggebrecht et al, 2009) (*Figure 2*).

Penetrating aortic ulcer

Penetrating aortic ulcer has only recently been acknowledged as a distinct pathological variant of classic false-

Figure 1. Diagnostic algorithm of patients with acute aortic syndrome.

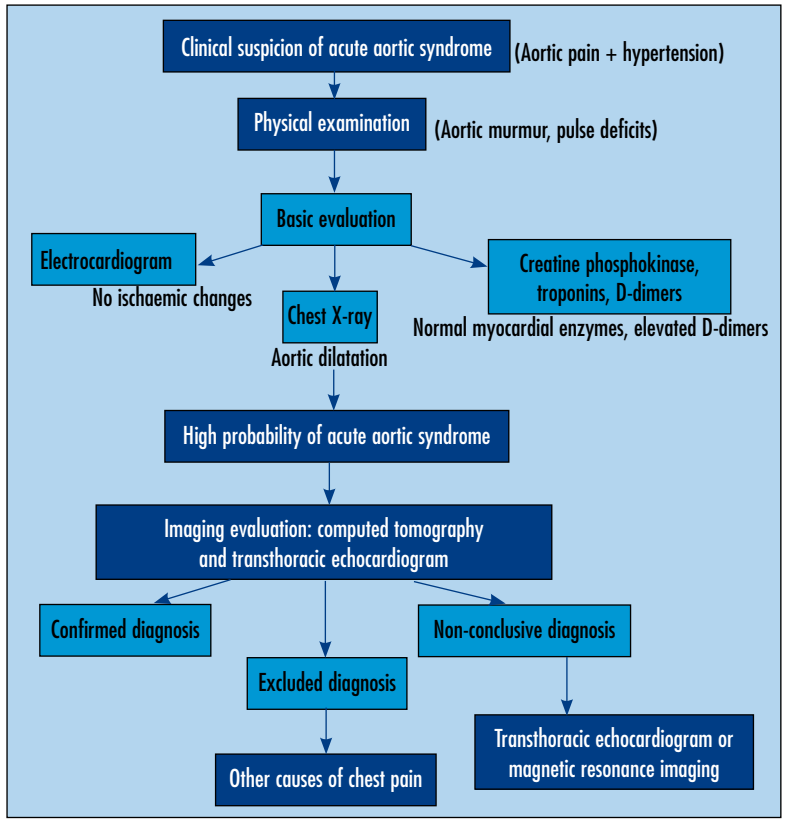
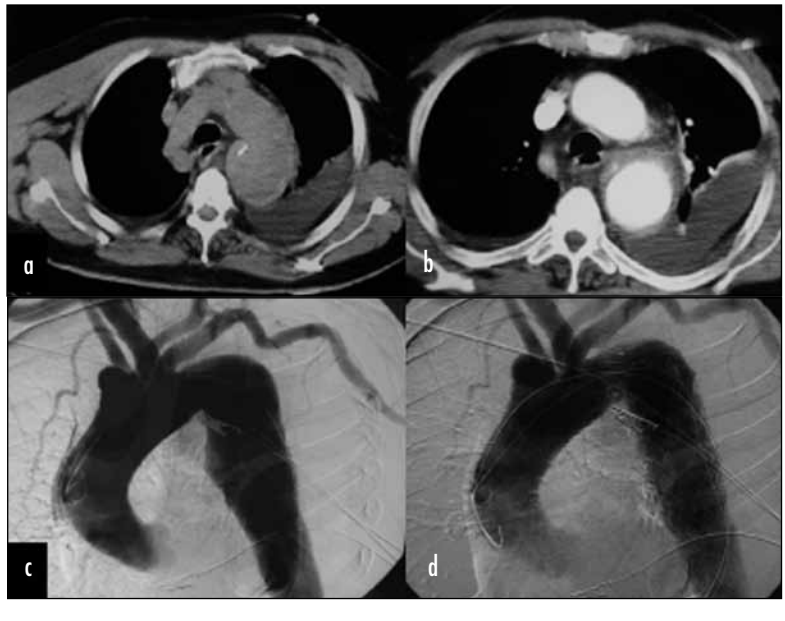


Figure 2. Intramural haematoma. a. Computed tomography scan plain image and (b) with contrast enhancement showing an intramural haematoma of the descending thoracic aorta. c. Conventional angiography images (c) before and (d) after stent graft deployment.

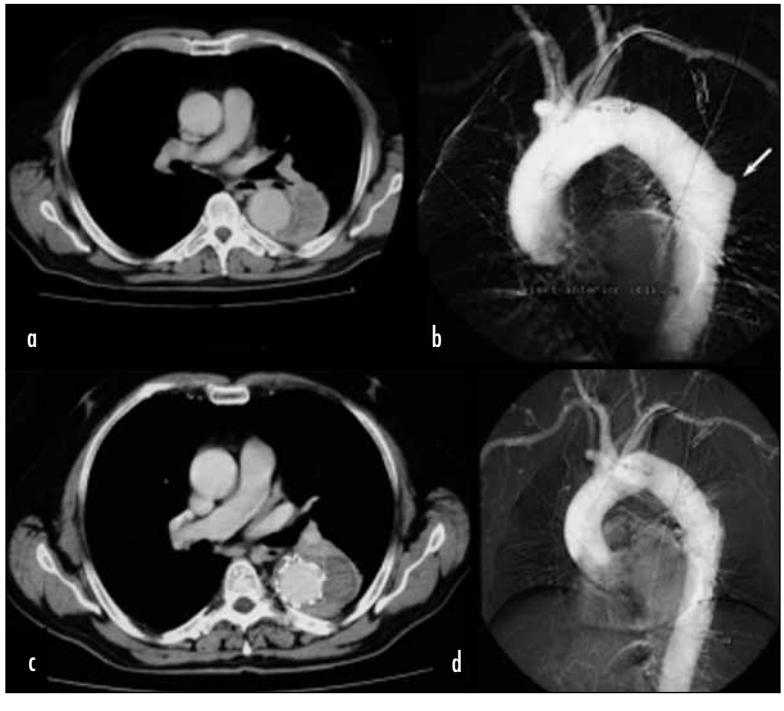


lumen aortic dissection. It is defined as an ulceration of an aortic atherosclerotic plaque penetrating through the internal elastic lamina into the aortic media. Penetrating aortic ulcer occurs most often in patients with extensive atherosclerotic disease. As a result, most patients with penetrating aortic ulcer present in their 7th decade. Previous investigators have estimated that 2.3–7.6% of acute aortic syndromes are caused by penetrating aortic ulcer. The presentation of penetrating aortic ulcer may not be distinguished from classic dissection and there is a strong association of penetrating aortic ulcer with concomitant abdominal aortic aneurysms (Eggebrecht et al, 2009). Although the natural history of penetrating aortic ulcer is not fully understood, patients presenting with acute aortic syndrome caused by penetrating aortic ulcer have a worse prognosis than those who are asymptomatic and in whom penetrating aortic ulcer is found incidentally on axial imaging (Eggebrecht et al, 2009).

Imaging

Penetrating aortic ulcer has a characteristic appearance on angiography, reminiscent of duodenal ulcer (although this is now rarely performed for penetrating aortic ulcer), whereas non-invasive imaging modalities (contrast or non-contrast computed tomography) are used more frequently and provide important information on periaortic tissues. Contrast-enhanced magnetic resonance is useful for confirming enhancement of the ulcerated tissue and adjacent aortic wall (Birchard, 2009).

Figure 3. Penetrating aortic ulcer. a. Computed tomography scan and (b) conventional angiography showing a penetrating aortic ulcer of the middle segment of the descending thoracic aorta (arrow). c. Computed tomography scan and (d) conventional angiography images after stent graft deployment and exclusion of the lesion.



Treatment

Penetrating ulcers of the ascending aorta (type A penetrating aortic ulcer) are rare, but require urgent surgical repair in most patients (Erbel et al, 2001; Eggebrecht et al, 2003). Endovascular stent placement is an accepted form of treatment, although determining which patients to treat and when to treat them remains controversial. Patients with symptomatic penetrating aortic ulcers of the descending aorta who are poor surgical candidates (typically in their 7th decade in the majority of cases with a life expectancy of less than 10 years) are good candidates for urgent stent graft placement (Svensson et al, 2008) (*Figure 3*).

Acute aortic dissection

Acute aortic dissection is the most common cause of acute aortic syndrome, and the most common predisposing factor for aortic dissection is systemic hypertension. Connective tissue disorders such as Marfan syndrome, presence of a bicuspid aortic valve, and aortic coarctation are also associated with increased incidence of aortic dissection. Atherosclerotic disease, however, is not directly related to development of aortic dissection, so its absence should not preclude a diagnosis of dissection. Overall, men are affected three times as often as women (Birchard, 2009). Patients with aortic dissection often present with back or chest pain. The pain may radiate posteriorly between the scapulae or may radiate anteriorly, depending on which portion of the aorta is affected.

Classic aortic dissection exhibits an intimomedial flap and an entrance tear, which typically occurs at sites of highest intraluminal pressure and wall tension: the right lateral wall of the ascending aorta or in the proximal segment of the descending thoracic aorta (Macura et al, 2003). Aortic dissection is characterized by a separation of the aortic media of variable longitudinal and circumferential extension. Blood under pressure dissects the media longitudinally and, as a consequence, a double-channel aorta is formed. The outer part of the aortic media together with the adventitia will form the false channel outside wall, whereas the rest of the aortic media together with the intimal layer will form the intimomedial flap. The proportion of media that remains at the external wall of the false channel differs from patient to patient and is a critical factor in subsequent patient outcome and management (Vilacosta et al, 2009).

Classification

Aortic dissections may be classified according to aetiology (connective tissue disease, hypertension and atherosclerosis, trauma), acuity (acute, subacute, chronic), anatomical and prognostic features, with the later having the greatest clinical utility. The two most common classification systems with regard to location within the thoracic aorta are the Stanford and DeBakey classification systems. The Stanford system is more widely applied, listing type B dissections as involving the

descending thoracic aorta distal to the left subclavian artery while type A dissections (*Figure 4*) involve the ascending aorta. As intramural haematoma and penetrating aortic ulcer are now considered as variants of classic dissection, the European Working Group introduced a new classification system:

- Class 1: classic aortic dissection
- Class 2: formation of intramural haematoma
- Class 3: subtle dissection
- Class 4: plaque rupture with penetrating aortic ulcer
- Class 5: traumatic dissection (Erbel et al, 2001).

Finally, the IRAD classification combines anatomical and prognostic information, based on location of the most proximal entry site. Type B dissection is prognostically the same regardless of the entry site (B1–B3, with B1 closer to the left subclavian artery), while type A dissections show worse prognosis the closer the entry site is located to the aortic valve (A1–A3, with A1 closer to the aortic valve) (Suzuki et al, 2003) (*Table 1*).

Imaging

Chest radiography is performed routinely in patients with suspected aortic dissection. It has a sensitivity of 67% and a specificity of 70%. The chest radiograph may show an enlarged aortic contour or a widened mediastinum (Hagan et al, 2000), whereas a specific but less often encountered finding is inward displacement of aortic intimal calcifications toward the centre of the aortic lumen. Computed tomography, magnetic resonance and transoesophageal echocardiography have a similar diagnostic accuracy in classic aortic dissection.

Figure 4. Acute type A dissection.

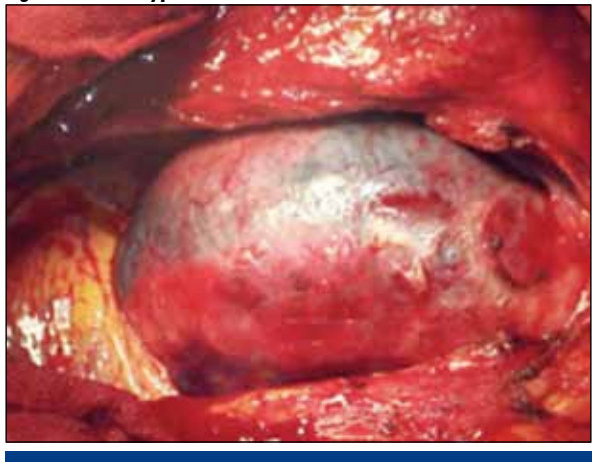


Table 1. Aortic dissection classification systems

Stanford classification	Type A: ascending aorta + arch + descending aorta
	Type B: descending aorta
DeBakey classification	Type I: ascending and descending aorta
	Type II: ascending aorta only
	Type III: descending aorta

Computed tomography angiography with intravenous contrast has a reported sensitivity of 83–100%, a specificity of 87–100% for aortic dissection, and is the imaging study used most often to evaluate for dissection in the emergency setting. On computed tomography angiography, the key finding in aortic dissection is identification of the intimal flap, appearing as a thin 'line' of soft tissue within the lumen of the aorta which effectively creates two lumens. The intimal flap may take a simple course within the aorta, or may have a complex spiral configuration (Birchard, 2009), where the true lumen usually contains 'brighter' contrast material than does the false lumen (as the false lumen fills more slowly). The development of computed tomography angiography with retrospective electrocardiographic gating provides additional benefits by eliminating cardiac pulsation artefacts so that it can now be used to image the aortic root (Fleischmann et al, 2008).

Management

Type A dissections require immediate surgery because of the potential for fatal complications. Rupture of a type A dissection into the pericardium may occur, causing haemopericardium, cardiac tamponade and death. A type A dissection may also propagate in a retrograde direction and dissect into the coronary arteries, resulting in ischaemia or infarct. Dissection into the right main coronary is more common than the left, typically because ascending aortic dissections tend to occur on the right lateral side of the ascending aorta. Surgical treatment has been guided by established principles, which include:

1. Resection and replacement of the aortic tear site, usually the ascending aorta or arch
2. Repair or replacement of the aortic sinus segments with false lumen obliteration to treat potential coronary malperfusion and late aortic root abnormalities
3. Resuspension or replacement of the aortic valve
4. Obliteration of the false lumen at the distal anastomosis and reestablishment of primary flow into the true lumen.

Despite surgical advances, many large series reveal a 25% perioperative mortality rate (Hagan et al, 2000; Bavaria et al, 2002). Nevertheless, improved patient outcomes may be achieved by enhanced surgical techniques and use of newer procedures such as the 'frozen elephant trunk' operation which offers a synergy between traditional aortic surgery and modern endovascular therapy to allow the management of complex and extended aortic disease (Vecht et al, 2010), whether located at the arch, ascending or descending aorta. In high risk cases hybrid approaches can be used including extra-anatomical bypass of head and neck vessels and consequently endoluminal stenting of the aortic arch or by using branched endografts. It is important to note that the durability of these options is currently unknown.

In uncomplicated type B dissection (*Figure 5*) surgery does not improve survival, so these patients are mainly

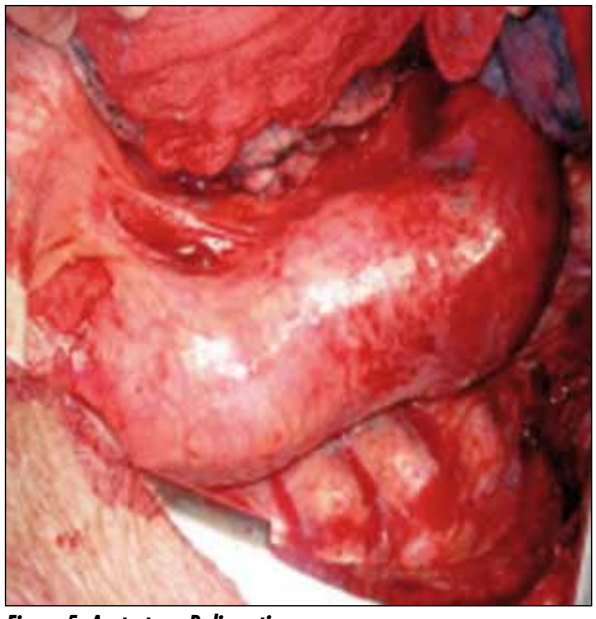


Figure 5. Acute type B dissection.

managed medically. In-hospital survival with medical treatment alone approaches 90% (Szeto et al, 2008). Surgery or percutaneous intervention are usually reserved for cases with ischaemic complications, impending rupture, severe aortic dilatation or intractable pain, but it may be considered in good surgical candidates or following poor response to drug therapy (Fuster and Andrews, 1999). Emergency stent grafting for acute complicated aortic dissection should not be considered as a 'curative' intervention but it will save lives if applied judiciously and quickly to salvage critically ill patients (*Figure 6*).

Whereas the decision to proceed with intervention or surgery is individualized, all patients should be managed medically with anti-impulse therapy. Although blood pressure control is fundamental, drugs with arterial vasodilatory properties may paradoxically increase the risk of wall rupture, as a result of reflex sympathetic responses leading to increased cardiac chronotropy and inotropy (Fuster and Andrews, 1999). Best results are reached with aggressive reduction in both blood pressure and maximum rate of change in left ventricular pressure constituting the basis of medical therapy of acute aortic syndromes. The initial agent of choice in most cases should be a beta-blocker, and then calcium-channel blockers if these are contraindicated. When blood pressure cannot be lowered to the desired target a second agent with vasodilatory effects may be considered such as sodium nitroprusside, enalapril or the newer fenoldopam (Sheinbaum et al, 2003) (*Figure 7*).

Acute traumatic aortic injury

Acute traumatic aortic injuries are most often the result of high-speed motor vehicle collisions. Acute traumatic aortic injury from blunt trauma is a substantial cause of morbidity and mortality, occurring in approximately 0.5–2% of all non-lethal motor vehicle collisions and 10–20% of all high speed deceleration fatalities (Steenburg et al, 2008). Nearly 80% of the victims die at the accident scene as a result of complete aortic transection (Neschis et al, 2008). Approximately 30% of surviving patients who are admitted to a hospital will die after 24 hours if they remain untreated. In surgically untreated survivors the natural course of aortic rupture is false aneurysm formation with secondary rupture after months or years (Buz et al, 2008). Computed tomography is the imaging modality of choice for acute traumatic aortic injuries and helical computed tomography is estimated to have a sensitivity of 100%, compared with 92% for angiography (Neschis et al, 2008).

Figure 7. Algorithm of management of acute aortic dissection.

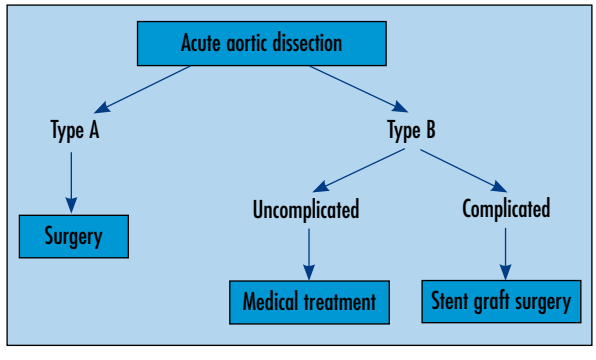
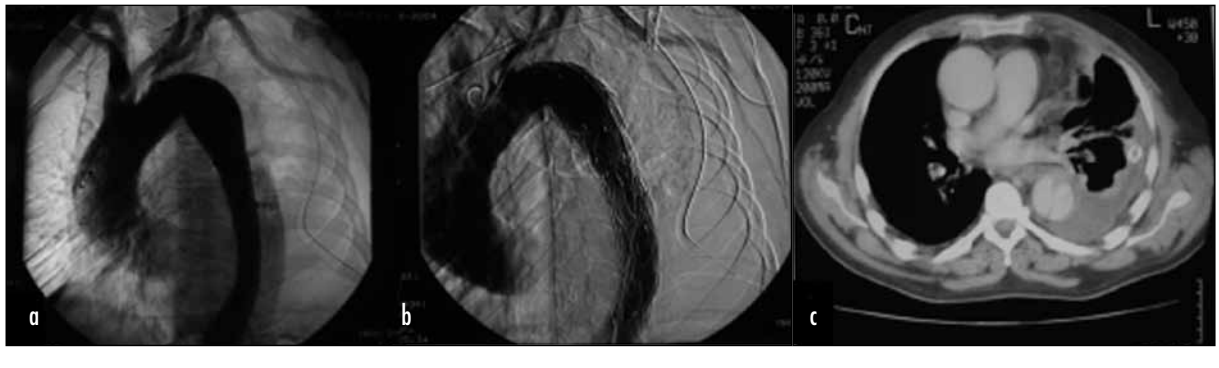


Figure 6. Acute type B dissection. a. Conventional angiography of a type B dissection showing a narrow true lumen. b. Stent graft deployment with covering of the proximal intimal tear, expansion of true lumen and flow restoration. c. Axial computed tomography scan image of a type B dissection showing an intimal flap in the descending thoracic aorta separating the true and false lumens.



The term 'minimal aortic injury' is often used to describe an aortic lesion associated with blunt injury that carries a relatively low risk of rupture. It can be defined as an intimal flap of less than 1 cm with no or minimal periaortic haematoma or pseudoaneurysm (Malhotra et al, 2001) which can be safely monitored by serial helical computed tomography. If, however, there is significant thrombus, periaortic haematoma, lumen encroachment or pseudoaneurysm it would be better to proceed with endograft coverage (Neschis et al, 2008). Several also demonstrate the relative safety of a delayed approach, particularly if there are substantial co-injuries, where beta-blockers and antihypertensive agents can decrease the shear force on the aortic wall (Neschis et al, 2008).

Mortality from surgery remains high at 12–26% as a result of associated injuries (Cook et al, 2006); endovascular and open surgery have been compared retrospectively to show a reduced morbidity and mortality and no cases of paraplegia in the endograft groups (Amabile et al, 2004; Rousseau et al, 2005). Endovascular repair offers several obvious advantages such as increased efficiency, improved outcomes with an overall early mortality lower by 3–6%, lower risk of paraplegia and minimized delay. Pending further evidence, this may be the preferred treatment of this disorder (Buz et al, 2008) (*Figure 8*).

Conclusions

Acute aortic syndrome encompasses a variety of challenging, acute aortic pathologies presenting with a similar clinical profile. The use of newer non-invasive imaging modalities has made the accurate detection of even subtle forms of these related pathologies. However, successful diagnosis of these entities equally depends on an exquisite, conscientious and profound clinical assessment. Future technologies include branched endografts, and application of novel pre- and post-treatment monitoring devices such as microelectromechanical systems used to continually measure aortic pressure using wireless technology (Chow et al, 2010). In the current era, use of endovascular techniques seem to offer significant survival benefits over open surgery. As device technology improves and clinical experience expands, stent-graft deployment for acute aortic pathologies will soon become the therapy of choice for a broad spectrum of acute aortic syndrome, even though a deeper knowledge of the natural history and recognition of important prognostic factors of these diseases is still needed. The value of these approaches to acute aortic syndrome could ultimately reduce the significant morbidity and mortality associated with this condition. **BJHM**

Conflict of interest: none.

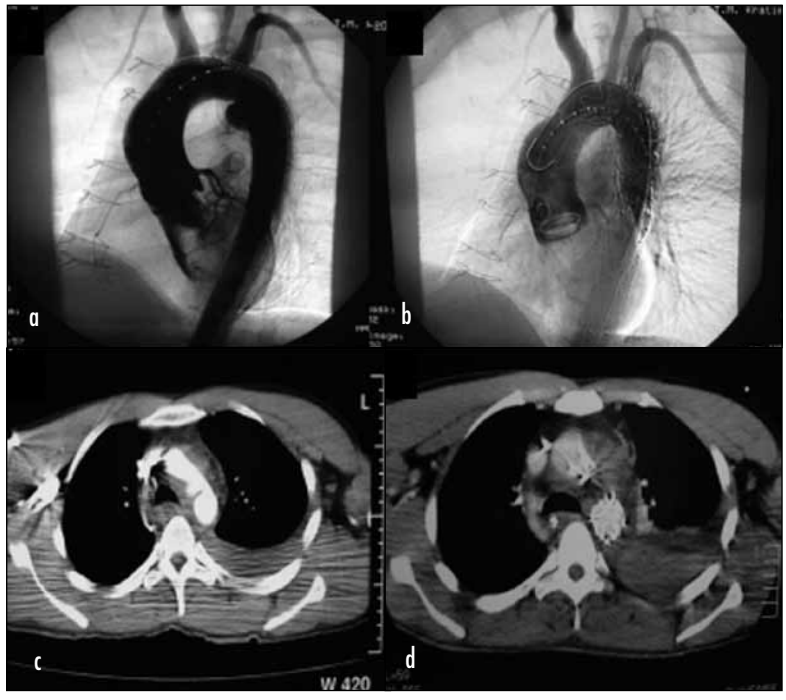


Figure 8. Acute traumatic aortic injury. a. Conventional angiography in left anterior oblique projection reveals an aortic pseudoaneurysm at the aortic isthmus. b. Stent graft deployment with successful exclusion of the pseudoaneurysm. c. Contrast-enhanced computed tomography scan shows aortic transsection with contained pseudoaneurysm. d. Contrast-enhanced computed tomography scan after stent graft deployment.

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KEY POINTS

- Acute aortic syndrome encompasses classic aortic dissection, intramural haematoma, penetrating aortic ulcer, and iatrogenic or traumatic transection of the aorta.
- It presents with characteristic chest or thoracic pain (also known as 'aortic pain').
- Imaging includes computed tomography, magnetic resonance imaging and both transthoracic and transoesophageal echocardiography.
- Treatment consists of medical management, surgical repair, endovascular stenting or a synergy between these therapies such as the 'frozen elephant trunk' operation.

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