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Recognition and initial management of acute aortic dissection

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Abstract

Acute aortic dissection is a cardiovascular emergency that should be recognised on presentation in the Emergency Department (ED) because clinical outcome is time-dependent.

In suspected cases of acute aortic dissection, immediate imaging with chest computed tomography scan followed by transthoracic echocardiography (TTE) is essential to confirm diagnosis.

Immediate medical management is aimed at controlling the heart rate (60–80 beats/min), systolic blood pressure (100–120 mmHg) and pain. Patients with Type A acute aortic dissection should immediately be referred to the cardiothoracic surgeons for emergency aortic surgery while those with Type B acute aortic dissection should be referred to the vascular surgeons for surgical/endovascular interventions if indicated.

Key words: Aortic dissection; Type A dissection; Type B dissection; Malperfusion syndrome

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Introduction

Acute aortic dissection (AAD) is the most common life-threatening disorder affecting the aorta and results in a high rate of morbidity and mortality (Hagan et al, 2000). Hence, AAD should be promptly recognised and treated.

The mean age at presentation is 63 years (Hagan et al, 2000). It has a male predominance of 65% (Hagan et al, 2000). Females tend to present at an older age and have a worse outcome (Hagan et al, 2000; Coyle et al, 2014). They are also more likely to present in altered mental status (Nienaber et al, 2004). Acute aortic dissection may occur at a younger age in patients with connective tissue disorders like Marfan syndrome and Loeys-Dietz syndrome (Uehara et al, 2018). The incidence is also higher in patients with bicuspid aortic valve, and among cocaine users and weightlifters (Elsayed et al, 2017).

Acute aortic dissection is commonly classified into Stanford Type A and Type B dissection.

Although the most common presentation is sudden onset of severe chest pain, a high clinical index of suspicion is necessary as clinical presentation may be diverse (Hagan et al, 2000). Electrocardiogram (ECG)-gated chest computed tomography (CT) scan followed by echocardiography is required for definitive diagnosis. However, if ECG-gated CT aorta is negative for AAD, there is no need for further investigations. Transthoracic echocardiography is useful in assessing complications of confirmed AAD such as acute severe aortic regurgitation and/or cardiac tamponade.

Initial medical therapy which includes control of heart rate, blood pressure, and pain is necessary before prompt referral to a cardiac or vascular unit, depending on the type of AAD.

If untreated, mortality rate of Type A AAD is as high as 1% per hour (Braverman, 2010). The most common cause of death following Type A AAD is aortic rupture, cardiac tamponade and visceral ischaemia, whereas visceral ischaemia is most common cause of death in Type B AAD (Hagan et al, 2000).

Pathophysiology

Acute aortic dissection is defined as tear in the intima of the aorta causing propagation of blood into the media.

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The underlying factor is increased aortic wall stress augmented by medial layer degeneration. It follows the law of LaPlace, which states that wall stress is directly proportional to pressure and radius, and inversely proportional to thickness of the vessel wall (Patel and Arora, 2008).

The tear creates an intimal flap which separates the false lumen from the true lumen. Intimal tear is usually located in the proximal part of the ascending aorta in Type A AAD and immediately below the origin of the left subclavian artery in Type B AAD. The process of dissection propagates in an antegrade or retrograde fashion, or both. Propagation can lead to compression of the true lumen by the false lumen resulting in ischaemia (static obstruction) or pressurised false lumen causing the intimal flap to obstruct of the orifice of the vessel (dynamic obstruction). When it propagates in a retrograde fashion, it can cause prolapse of aortic valve commissure or distortion of aortic valve leaflet arrangement resulting in aortic regurgitation. It can also lead to coronary artery obstruction and pericardial effusion (Rylski et al, 2023). Antegrade propagation can cause narrowing of aortic side branch arteries resulting in cerebral, mesenteric, renal, and limb ischaemia or even paraplegia, depending on the vessel involved (Patel and Arora, 2008).

Classification of acute aortic dissection

This will depend on the following factors:

Chronicity (Lombardi et al, 2020).

- Hyperacute: Less than 24 hours.
- Acute: 1–14 days.
- Subacute: 15–90 days.
- Chronic: More than 90 days.

Anatomic: Based on the entry tear or longitudinal extent of AAD.

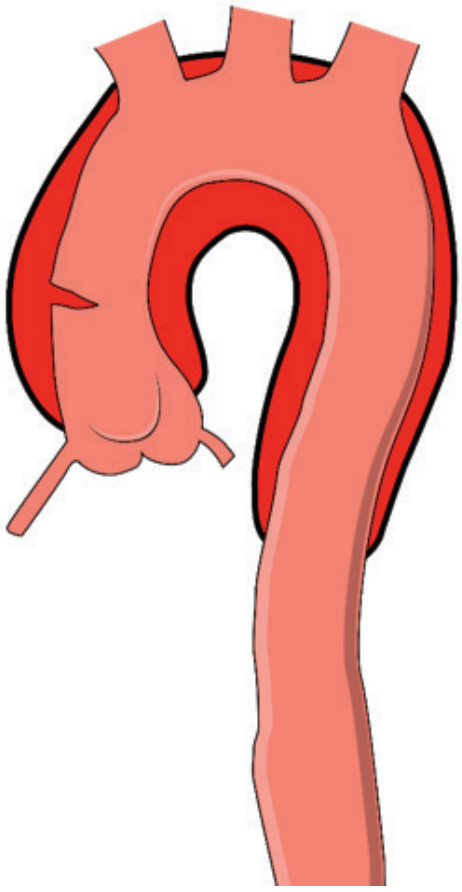

Figure 1. Stanford Type A acute aortic dissection (AAD).

Stanford classification is most commonly used clinically. AAD is classified into Type A and Type B AAD (Elsayed et al, 2017).

Type A: Involves the ascending aorta but may extend into the arch and descending aorta, regardless of the site of origin (Figures 1,2).

Type B: Not involving the ascending aorta. It involves the descending aorta distal to the left subclavian artery (Figures 3,4,5).

Non-A Non-B AAD: Dissection confined to the aortic arch only or a retrograde dissection arising from the descending aorta that extends into the arch and stops before the ascending aorta (Urbanski and Wagner, 2016).

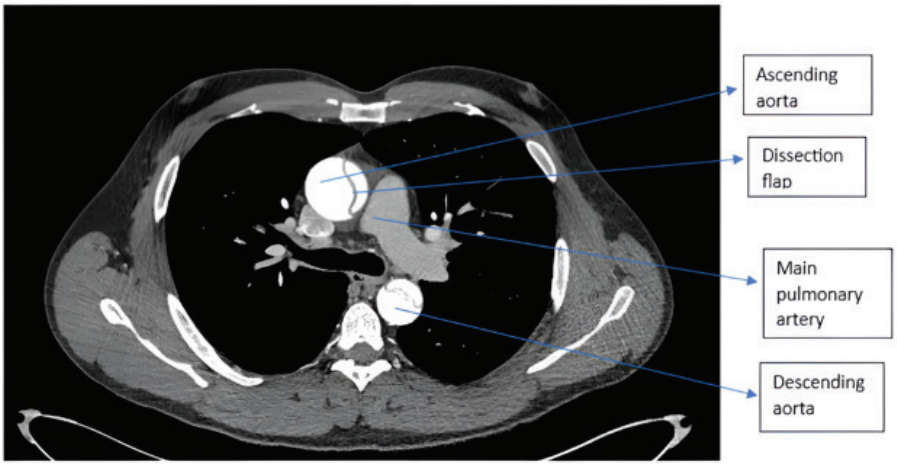

Figure 2. Chest computed tomography scan showing Type A acute aortic dissection.

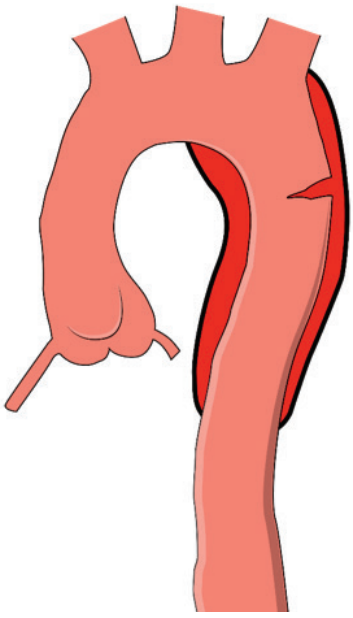

Figure 3. Stanford Type B acute aortic dissection.

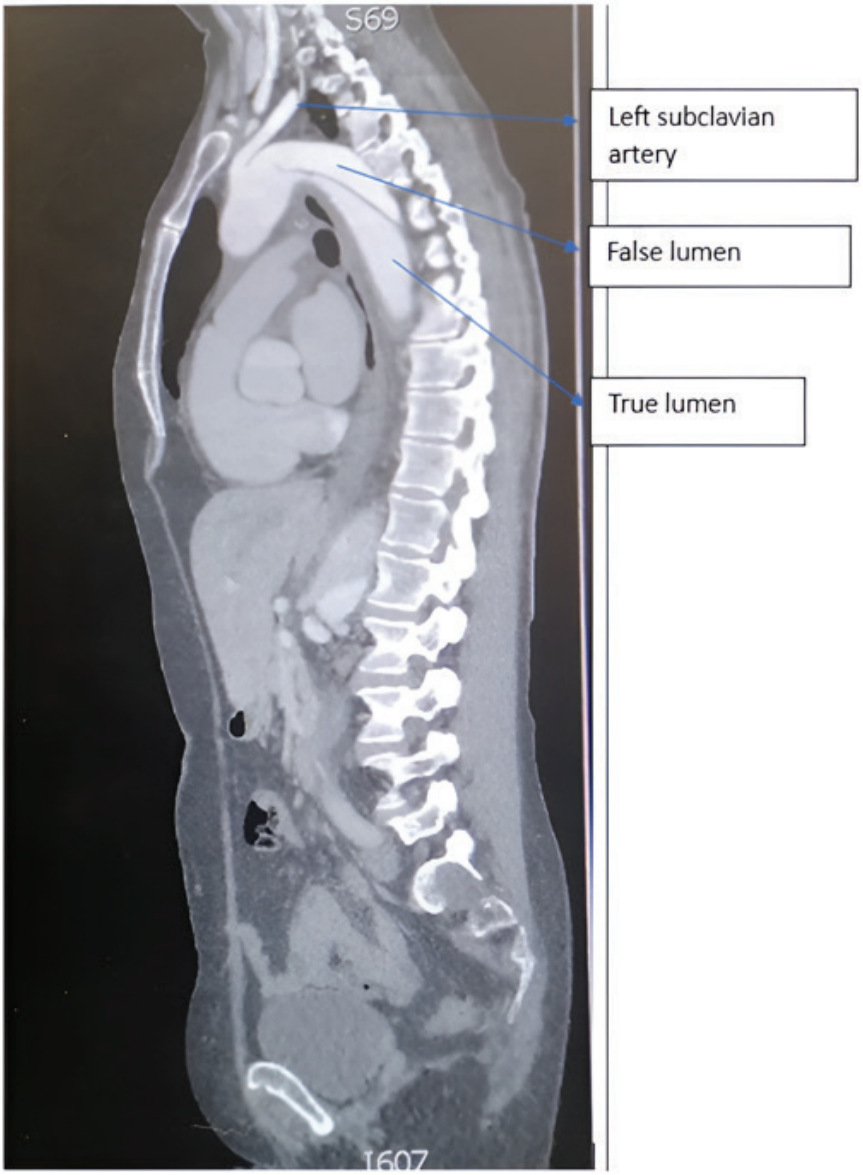

Figure 4. Computed tomography scan showing Type B acute aortic dissection (saggital view).

Risk factors

Hypertension: Hypertension is a very important risk factor for AAD because it is associated with aortic wall stress. This includes both elevated systolic and diastolic pressures (Elsayed et al, 2017; Hibino et al, 2022).

Connective tissue disorders: This causes inherent weakening of aortic wall. They include Marfan syndrome (mutation in Fibrillin-1 (*FBN1*) gene that encodes fibrillin), Loeys-Dietz syndrome (mutation in transforming growth factor beta receptor genes 1 and 2), Type IV Ehlers-Danlos syndrome (defect in the Collagen Type III Alpha 1 Chain (*COL3A1*) gene which encodes Type III collagen) and Turner's syndrome (absent or incomplete X-chromosome) (Elsayed et al, 2017; Thunström et al, 2019; Sayed et al, 2021).

Congenital abnormalities: The presence of a bicuspid aortic valve and aortic coarctation has been found to be associated with an increased risk of aortic dissection in Turner's syndrome (Elsayed et al, 2017; Thunström et al, 2019).

Familial aneurysmal disease: These include non-syndromic familial thoracic aortic aneurysm and familial aortic dissection (Sayed et al, 2021).

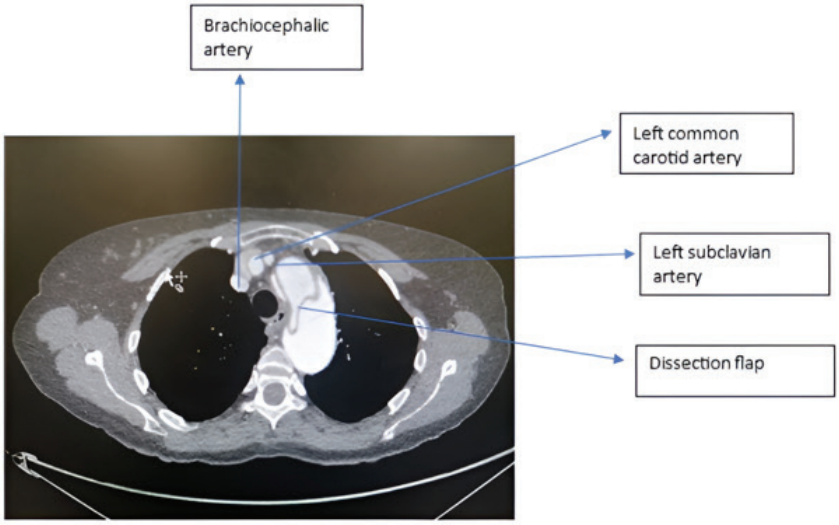

Figure 5. Computed tomography scan showing Type B acute aortic dissection (axial view).

Inflammatory vasculitis: These include giant cell arteritis, Takayasu arteritis, and rheumatoid arthritis (Elsayed et al, 2017).

Others: Old age, atherosclerosis, cocaine/amphetamine use, heavy lifting, surgical/catheter manipulation, and pregnancy (Elsayed et al, 2017).

Clinical features

This depends on the extent of dissection, malperfusion and haemodynamic stability (Table 1) (Hagan et al, 2000; Patel and Arora, 2008; Elsayed et al, 2017; Sayed et al, 2021; BMJ Best Practice, 2022; Acharya and Mariscalco, 2023).

Clinical signs

On physical examination, a weak or absent contralateral radial artery, brachial artery or femoral artery pulsations may be elicited in AAD (pulse deficit). Bradycardia may occur secondary to complete heart block due to AAD dissecting into the right coronary artery.

A difference in the blood pressure of more than 20 mmHg between both left and right arms may be a pointer to AAD involving the subclavian artery.

Hypertension may be due to preexisting hypertensive disease or secondary to increased sympathetic drive. It is more common in patients presenting with Type B dissection. In AAD patients presenting with hypotension, it is important to suspect life-threatening complications like severe aortic regurgitation, significant pericardial effusion/cardiac tamponade or even rupture. A combination of hypotension, pulsus paradoxus and diminished or distant heart sound suggests pericardial effusion/cardiac tamponade.

The murmur of aortic regurgitation, typically a diastolic decrescendo murmur may be heard in the 3rd intercostal space. This is likely to be accompanied by widened pulse pressure, hypotension and heart failure. Presence of continuous murmur may indicate rupture of dissection into the atria or left ventricle (Elsayed et al, 2017).

Decreased breath sound may be due to rupture of AAD into the pleural space (acute aortic rupture or leaking dissection) or sympathetic left pleural effusion (Acharya and Mariscalco, 2023). Rhonchi may result from compression of tracheobronchial tree from periaortic haematoma.

Malperfusion syndrome may manifest on physical examination as neurologic dysfunction. This may present in the form of syncope, altered mental status, stroke, and paraplegia. Other neurological signs that can occur rarely in AAD include Horner syndrome (due to compression of the ascending sympathetic supply within the carotid sheath in AAD involving

Table 1. Table showing symptoms of acute AAD and the possible explanations for these symptoms

Symptoms	Explanation
A. Location of pain: This may be of help in localising the part of the aorta involved in the dissection. Pain of Type A dissection is described as an abrupt onset of intense chest pain, often described as ripping or tearing in nature and located retrosternally or substernally and propagates as the dissection evolves. Pain of Type B dissection is experienced in the intrascapular region or the back. In patients with aortic aneurysm, AAD may not be associated with pain	A. Pain is due to the dissection stretching the adventitial nerve fibres of the aorta
B. Symptoms due to extension of dissection to the aortic branch arteriesa a. Syncope b. Stroke c. Abdominal pain d. Paraplegia e. Low urine output or anuria	B. a. AAD complicated by carotid dissection, cardiac tamponade, and aortic rupture b. AAD involving the brachiocephalic artery or extending into the carotid artery c. AAD involving the mesenteric artery d. AAD involving the intercostal and lumbar arteries e. AAD involving the renal arteries
C. Symptoms due to rupture or compression secondary from haematoma a. Haemoptysis b. Hoarseness c. Dysphagia d. Haematemesis e. Pulsating neck mass	C. a. Rupture into the tracheobronchial tree b. Compression of the recurrent laryngeal nerve c. Compression of the oesophagus d. Rupture into oesophagus

the carotid artery), and hoarseness (due to compression of the left recurrent laryngeal nerve) (Khan et al, 1999; Kasravi et al, 2010). The degree of preoperative neurological involvement must be adequately documented.

Abdominal tenderness is suggestive of mesenteric ischaemia and limb ischaemia is due to involvement of artery of the lower limb in AAD.

Some phenotypic appearances may suggest the underlying risk factor for AAD. Marfanoid characteristics of a tall, thin individual with arm span to height ratio > 1.05, pectus deformities and scoliosis suggests Marfan syndrome. Phenotypic features of Ehlers-Danlos syndrome include a typical facial appearance (prominent ears, hollow cheek), thin translucent skin, and excessive bruising while hypertelorism, bifid uvulae or cleft palate is typically seen in patients with Loeys-Dietz syndrome.

Investigations

Electrocardiogram (ECG)

ECG findings in AAD may range from normal to acute ischaemic changes due to myocardial ischaemia complicating AAD. Acute ischaemic ECG changes in patients with low risk of AAD suggest acute coronary syndrome. However, in those with risk factors for AAD, additional investigative modalities should be undertaken to rule out AAD before an anticoagulant or a thrombolytic therapy is given to the patient as this will potentially worsen the prognosis in AAD (Coyle et al, 2014).

Radiologic investigations

Chest radiograph

A normal chest x-ray is found in 15% of patients with AAD, hence it has limited benefits in AAD and should be omitted in suspected AAD patients who are clinically unstable. The chest x-ray is only relevant in patients where the diagnosis is unclear, however some findings may be a pointer to AAD. These findings may include widened mediastinum in 60–70% of cases, double aortic shadow or irregular aortic contour, widened aortic knob or displacement of aortic knob calcification and calcium sign which indicate the separation of the intimal calcification from the outer aortic soft tissue border > 5 mm. Others include deviation of trachea, left main bronchus, oesophagus and nasogastric tube to the right, left pleural effusion and apical cap (Callaway et al, 2021; Acharya and Mariscalco, 2023).

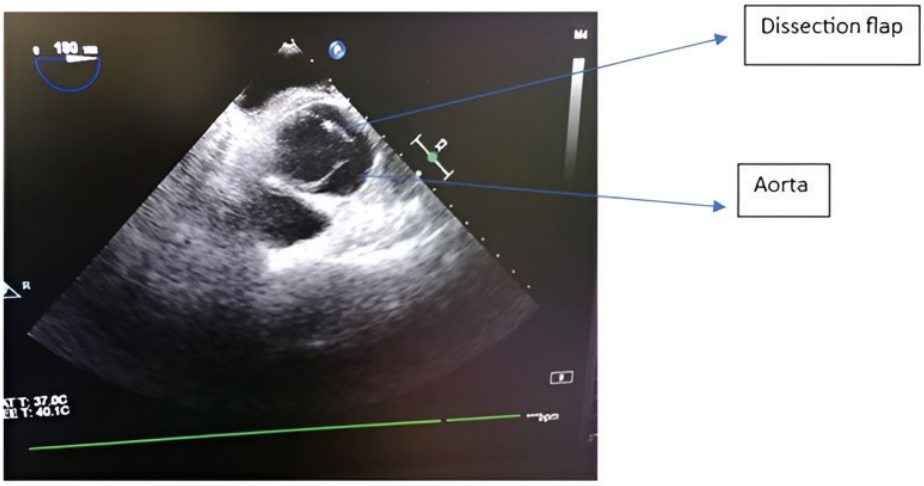

Figure 6. Transoesophageal echocardiography showing dissection flap.

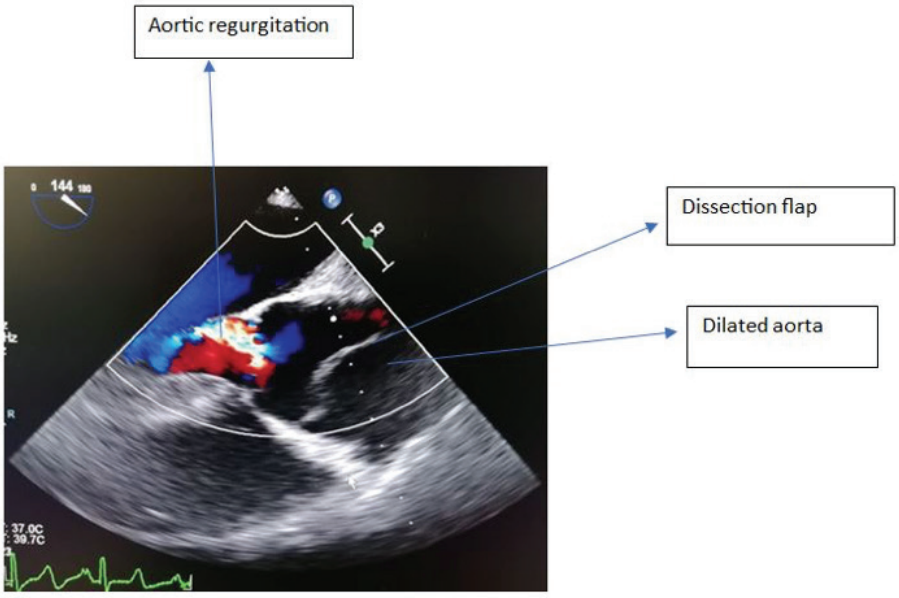

Figure 7. Transoesophageal echocardiography showing aortic regurgitation.

Echocardiography

Transthoracic echocardiography (TTE) can be performed in the ED and in patients suspected to have AAD, contrast administration is recommended when images are not definitive (Evangelista et al, 2019). Transoesophageal echocardiography (TOE) produces better images but require sedation and is often performed in the intensive care unit (ICU) or theatre (**Figures 6,7**).

Transthoracic echocardiography can visualise an intimal flap in the proximal aorta and its sensitivity has improved to 75–85% with current echo-technology and contrast enhancement (Cecconi et al, 2012; Evangelista et al, 2023; Muratori et al, 2023). TTE can detect complications of AAD, including aortic regurgitation, presence of pericardial effusion, and regional wall motion abnormalities. TTE can also assess right ventricular size and function, pulmonary artery pressure and left ventricular function (Evangelista et al, 2023; Muratori et al, 2023). A negative TTE does not rule out AAD and it has a low sensitivity for Type B dissection (Evangelista et al, 2023; Muratori et al, 2023).

Transoesophageal echocardiography has a higher sensitivity than TTE and it is a very useful tool in evaluating the primary entry tear (presence, site, and size), presence of secondary communications, thrombus in the false lumen, and in defining the mechanism of aortic regurgitation (Evangelista et al, 2023; Muratori et al, 2023). The true lumen is often smaller than the large lumen, exhibit systolic expansion on pulsation and has a systolic antegrade flow direction (Evangelista et al, 2023).

Computed tomography (CT) scan

Computed tomography scan of the chest, abdomen, and pelvis is the diagnostic modality of choice and must be performed without delay in suspected AAD. ECG synchronisation (ECG-gated CT scan) is preferred as it removes motion artefacts and produce motion free images in most cases (Callaway et al, 2021).

Computed tomography scan will clearly reveal the presence of an intimal flap which separates the true lumen from the false lumen. The true lumen is smaller than the false lumen, has calcifications along its outer wall, directly communicates with aorta and shows more enhancement unlike the false lumen. False lumen is also recognised by beak sign (acute angle formed between the wall of false lumen and intimal flap) and cobweb sign (fine strands of dissected tissue of the media crossing the false lumen) (Hallinan and Anil, 2014).

It can also show the segment of the aorta involved in AAD, the proximal and distal extent of AAD, and the origin of visceral arteries from the true or the false lumen.

Other findings may include the presence of thrombus in false lumen, the diameter of involved aorta (underlying pathology), identification of contrast leak and haemopericardium (Pepper, 2016; Singh et al, 2018; Acharya and Mariscalco, 2023).

Magnetic resonance angiography (MRA)

Magnetic resonance angiography does not require contrast and hence suitable for patients who are allergic to contrast or have poor renal function (Sayed et al, 2021). It possesses a high degree of precision in identifying the site of tear, presence of thrombus, pericardial effusion and has the capacity for assessing functional cardiac information which include the left ventricular function and presence and degree of aortic regurgitation (Coyle et al, 2014).

Magnetic resonance angiography is not as available as a CT scan. It is also not ideal for critically ill patients as time is consumed completing the investigation and adequate monitoring may be inadequate in the process.

Blood investigations

D-dimers: In the first 24 hours of AAD, D-dimer assay is elevated in AAD. Within this time, values < 500 ng/mL are unlikely to be due to AAD. It is not a specific marker for AAD, and it is also elevated in pulmonary embolism and myocardial infarction which are important differential diagnosis of acute chest pain (Acharya and Mariscalco, 2023). Hence, D-dimer assay could assist the emergency physician in stratifying patients presenting with chest pain within 24 hours of onset of pain. In patients at low risk of AAD, a negative D-dimer assay may be useful in ruling out AAD while those with a higher assay > 500 ng/mL warrant further investigation with diagnostic imaging (Asha and Miers, 2015).

Cardiac troponin: Elevation of cardiac troponin in AAD suggests myocardial ischaemia complicating AAD (Coyle et al, 2014; Pepper, 2016). In Type A AAD, coronary malperfusion may be caused by compression, coronary dissection or coronary disruption and may be associated with mortality up to 33.3% (Kawahito et al, 2003).

Blood gases: This may reveal severe acidosis and elevated lactate which may be pointers to mesenteric ischaemia complicating AAD (Pepper, 2016). This is associated with an almost 5-fold increased risk of perioperative mortality (Tsagakis et al, 2013).

Red cell count: Low haemoglobin suggests blood loss or active bleeding (Pepper, 2016; BMJ Best Practice, 2022).

Urea and Creatinine: High blood levels of urea and creatinine may suggest renal malperfusion (Pepper, 2016; BMJ Best Practice, 2022).

Liver function test (LFT): Elevated blood levels of aspartate transaminase and alanine aminotransferase may suggest hepatic malperfusion (Acharya and Mariscalco, 2023).

Management

Admit the patient where he/she can be adequately monitored, e.g., intensive care unit.

Ensure adequate oxygenation and ventilation. AAD patients with profound hemodynamic instability may require intubation and mechanical ventilation.

Insert two large bore cannulas for resuscitation. Intravenous resuscitation may involve the use of normal saline or balanced crystalloids. In hypotensive patients, blood transfusion may be required if haemoglobin is low.

Insert arterial line and monitor vital signs (blood pressure, ECG) and urinary output.

Monitor continuous mental and neurologic state.

Group and save ± crossmatch blood for surgical or endovascular intervention.

Blood pressure management: The objective is to maintain target blood pressure, heart rate control and to reduce the rate of change in pressure over time (dP/dt) which is associated with propagation of intimal tear (Patel and Arora, 2008). Intravenous beta blocker can be initiated to reduce shear stress on the aortic wall, reduce contractility and prevent rupture of the diseased aortic wall in AAD not complicated by aortic regurgitation. Target systolic blood pressure should just be adequate to maintain organ perfusion, between 100–120 mmHg and pulse rate of 60–80 beats/min (Acharya and Mariscalco, 2023). The most commonly used beta blockers include (Bristol Bath Weston Vascular Network, 2022):

- Labetalol at a dose of 0.25 mg/kg bolus over 2 min, followed by 2 mg/min infusion for 10 min and increase infusion up to 5 mg/min.
- Esmolol at a dose 250–500 µg/kg bolus over 1–3 min, followed by 50 µg/kg/min infusion for 4 min and increase infusion up to 200 µg/kg/min.

In the presence of contraindications or intolerance to beta blockers, non-dihydropyridine calcium channel blockers like verapamil and diltiazem are used. It is not advisable to use vasodilators alone as they can cause compensatory tachycardia and increase stress in the aortic wall (Patel and Arora, 2008).

Pain control: Pain can lead to increase in wall stress of aorta by increasing blood pressure and heart rate, so pain control is vital. Opioids are useful in reducing sympathetic tone. Morphine sulphate is given at a dose of 2–5 mg intravenously every 5–30 min (Bristol Bath Weston Vascular Network, 2022).

Emergency referral to the regional cardiac surgery unit should be made in case of Stanford Type A AAD and the vascular surgery unit for Type B AAD.

Conclusion

Acute aortic dissection can be rapidly fatal, hence prompt recognition and institution of appropriate line of management remain vital. Immediate surgical repair is indicated for Type A AAD. Uncomplicated Type B dissection is treated medically while the complicated cases will require open surgical or endovascular intervention.

Key points

- AAD is a life-threatening emergency and mortality is time-dependent.
- A high index of suspicion is needed. ECG-gated CT scan ± Echocardiography is required to establish a diagnosis.
- Appropriate medical management must be instituted and prompt transfer to cardiothoracic or vascular service depending on the type of AAD.

Curriculum checklist

This article relates to the necessary requirement for the specialist training in general internal medicine, and is linked with the following capabilities in practice:

- Managing an acute unselected take.
- Managing an acute specialty-related take.
- Delivering effective resuscitation and managing the acutely deteriorating patient.

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Availability of data and materials

All the data of this study are included in this article.

Author contributions

EBK, COI and DLN designed the research study. EBK, COI and DLN analyzed the data. EBK drafted the manuscript. All authors contributed to the important editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics approval and consent to participate

Not applicable.

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Conflict of interest

The authors declare no conflict of interest.

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