Acute dissection of the thoracic aorta

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Aortic dissection is an acute medical emergency with a high mortality. Crucial to improving survival is early recognition and appropriate treatment. This review describes the presenting clinical features and imaging techniques used in the diagnosis of aortic dissection and outlines the treatment modalities.

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Patients with aortic dissection usually present as acute medical emergencies. The diagnosis may not be obvious and a high index of clinical suspicion and early imaging is essential to confirm the diagnosis. Mortality is high and these patients should be managed in a tertiary cardiac centre with early involvement of cardiologists and cardiothoracic surgeons. Aortic dissection can be classified according to the pathology or anatomical features (Svensson et al, 1999).

PATHOLOGICAL CLASSIFICATION

Classical aortic dissection (class 1)

This is the most common type of aortic dissection (70% of all cases). It occurs as a result of a tear in the aortic wall with progressive separation of the intima from the underlying media and/or adventitia. In some cases, a secondary distal intimal disruption occurs resulting in the re-entry of blood from the false to the true lumen. The dissection often extends in a spiral manner along segments of the aorta, in an antegrade or retrograde direction to involve the aortic root, arch and side branches.

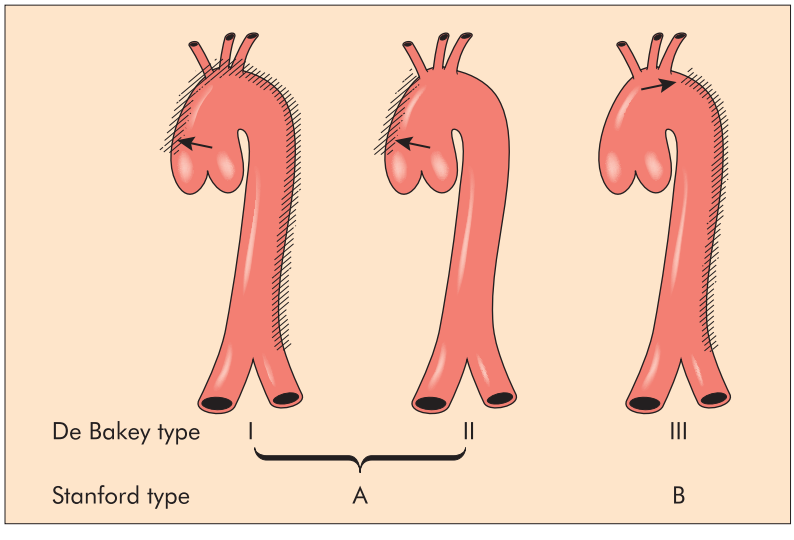

Figure 1. Classification of aortic dissection.

Intramural haematoma or haemorrhage (class 2)

Direct haemorrhage can occur into the aortic wall with no obvious intimal flap (10–30% of all cases). This is probably caused by rupture of the vasa vasorum. The haematoma can expand and perforate through the adventitia or intima and transform into a class 1 aortic dissection. The natural history and prognosis is similar to class 1 aortic dissection (Shimizu et al, 2000).

Subtle–discrete aortic dissection (class 3)

This rare form of aortic dissection (5%) occurs when an intimal tear remains localized with no dissection flap or haematoma, forming a discrete area of weakness in the aortic wall. Angiography shows a localized bulge in the aortic wall usually confirmed at surgery.

Aortic plaque rupture or ulceration (class 4)

Severe atheroma most commonly affects the descending thoracic or abdominal aorta. Atherosclerotic ulceration can progressively penetrate deep into the adventitia leading to weakness of the aortic wall, aneurysm formation or rupture.

Iatrogenic or traumatic dissection (class 5)

Iatrogenic dissections of the aorta (<5%) occur rarely following cardiac catheterization when the catheter tip causes a tear in the intima or following cardiac surgery where the aorta can be damaged by cross clamping. Deceleration chest trauma can also cause dissection of the ascending or upper descending thoracic aorta just below the ligamentum arteriosum.

ANATOMICAL CLASSIFICATION

Aortic dissections can be classified anatomically according to the De Bakey and Stanford classification systems (*Figure 1*). In the simpler Stanford classification, type A dissections

involve the ascending aorta while type B dissections involve the descending aorta only (Crawford et al, 1989). De Bakey further subdivided type A dissections into type I with involvement of ascending and descending thoracic aorta and type II with involvement of the ascending aorta only. Type III dissections are confined to the descending aorta, the same as Stanford type B (De Bakey et al, 1982). This anatomical classification system is important for management, as involvement of the ascending aorta (type A dissections) requires surgical repair while type B dissections can often be managed medically. Type A dissections are commoner than type B dissections (62% *vs* 38%) (Hagan et al, 2000). A dissection is said to be chronic when >14 days have elapsed since the acute event.

Predisposing factors

Aortic dissection affects men twice as commonly as women. The peak age is 50–55 years for type A and 60–70 years for type B dissections. The most common predisposing factors are systemic hypertension (78%) and cystic medial necrosis. Aortic dilation, aortic aneurysm, annuloaortic ectasia, hereditary connective tissue diseases (e.g. Marfan's syndrome or Ehlers–Danlos syndrome), chromosomal abnormalities (e.g. Turner's syndrome and Noonan syndrome), coarctation of the aorta and bicuspid aortic valve are recognized risk factors. Most of the above conditions increase the risk of aortic dissection by causing degeneration of the aortic media and reduced cohesiveness between the tissue layers in the aortic wall.

Hereditary conditions, such as Marfan's syndrome, which is associated with cystic medial necrosis, account for most cases of aortic dissection in patients <40 years of age. Vascular inflammation as a result of giant cell arteritis, Takayasu arteritis and Beçhet's disease can also predispose to aortic dissection. In young women, aortic dissections can occur during pregnancy, especially in the third trimester or during labour.

Prior cardiac surgery is an important risk factor for aortic dissection and was present in 18% of all cases recorded in the International Registry of Aortic Dissection (Hagan et al, 2000).

Clinical presentation

One fifth of patients with aortic dissection will die suddenly at home. Pain is the most common presenting symptom, and aortic dissection should be in the differential diagnosis of any patient presenting with sudden onset of severe chest, back or abdominal pain. The pain is often abrupt and maximal at the time of onset, and can be described as tearing, stabbing or sharp. Anterior chest pain is suggestive of type A dissections while interscapular or back pain characterizes type B dissections. Hypertension at initial presentation is more common in type B dissections.

Acute chest pain may be associated with syncope, haemodynamic collapse, stroke including hemiplegia, hemianaesthesia or spinal cord ischaemia and paraplegia. Any other branch of the aorta can be involved leading to ischaemia of the bowel, kidneys or legs. Involvement of the coronary arteries with myocardial ischaemia or infarction can also occur. Chest pain may be absent and up to 20% of patients may present with syncope (Slater and DeSanctis, 1976). Syncope on presentation is more common with type A dissections. If a second bout of acute pain occurs, this suggests extension of the dissection and is associated with a deteriorating clinical picture.

Differential diagnosis

Up to 30% of patients with aortic dissection are initially suspected of having other conditions such as acute coronary syndromes, aortic aneurysms, pulmonary embolus or aortic stenosis (Spittell et al, 1993). Aortic dissection needs to be considered in patients presenting with unexplained syncope, heart failure, stroke, myocardial infarction and acute ischaemia of extremities or viscera, even if chest pain is absent.

Physical examination

A pulse deficit with a weak or absent carotid, brachial or femoral pulses may be present in up to 20% of all cases occurring when branch vessels of the aorta are involved as a result of extension of the false lumen or compression by surrounding haematoma. Involvement of the left subclavian artery results in a lower blood pressure in the left compared to the right arm. An early diastolic murmur of aortic regurgitation is present in about half of the patients with type A aortic dissection, along with the physical signs of bounding pulse, wide pulse pressure and congestive cardiac failure. Rupture of the aorta into the pleural space may present with signs of a pleural effusion frequently on the left side. An exudative pleural effusion can also occur as an inflammatory reaction from the dissected aorta. The patient may be in shock as a result of cardiac tamponade or bleeding into the pleural cavity or mediastinum.

Extension of the dissection distally can lead to ischaemia of the legs, such as Leriche's syndrome, with obstruction of the iliac bifurcation and loss of pulses in both legs. Neurological deficits can occur in up to 40% of patients with type A dissec-

tions. Renal ischaemia causes haematuria, oliguria or anuria and mesenteric ischaemia is associated with abdominal pain, metabolic acidosis and elevated lactate dehydrogenase.

On rare occasions, symptoms such as vocal cord paralysis (resulting from compression of the left recurrent laryngeal nerve), superior vena cava compression (Spitzer et al, 1975), Horner's syndrome (resulting from compression of the superior cervical sympathetic ganglion) and haemoptysis or haematemesis (resulting from haemorrhage into the tracheobronchial tree or perforation into the oesophagus) may be encountered (Roth and Parekh, 1978).

INVESTIGATIONS

Electrocardiogram

An electrocardiogram (ECG) is important to distinguish ST elevation acute myocardial infarction from aortic dissection. Both conditions can coexist when the dissection flap extends into a coronary ostium causing acute myocardial ischaemia. In such cases thrombolytic therapy is detrimental. However, most patients with aortic dissection and coronary involvement have non-specific ST-T segment changes (Kamp et al, 1994). Changes reflecting left ventricular hypertrophy as a result of long-standing systemic arterial hypertension may be evident.

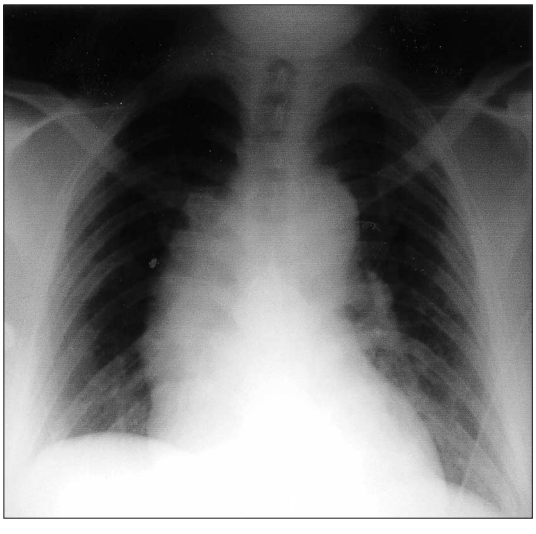

Figure 2. Chest X-ray showing widened mediastinum in patient with type A dissection.

TABLE 1.
Sensitivity and specificity of different imaging techniques used to diagnose aortic dissection

Imaging modality	Sensitivity	Specificity
Magnetic resonance imaging	98%	98%
Computed tomography	94%	87%
Transoesophageal echocardiography	98%	77%
Aortogram	87%	87%

Chest X-ray

The classic features of mediastinal widening may occur in both type A (*Figure 2*) and type B aortic dissections, as may a pleural effusion. A normal chest X-ray does not exclude aortic dissection.

Imaging

Computed tomography (CT), magnetic resonance imaging (MRI) and transoesophageal echocardiography (TOE) are useful non-invasive techniques for imaging all classes of aortic dissection, although aortography may be the best for class 3 dissections. The choice of technique will depend on availability and local expertise (*Table 1*). The key to radiological diagnosis of class 1 aortic dissection is the demonstration of an intimal flap separating a true and false lumen. The features of the dissection that need to be defined by imaging are:

- Type of dissection: A or B
- Extent of dissection
- Involvement of side branches
- Site of intimal tear and communication between the true and false lumens
- Involvement of aortic valve and extent of aortic regurgitation
- Signs of aortic perforation (blood in the pericardium, pleura or mediastinum).

Computed tomography

CT is the most widely used imaging modality in patients with suspected aortic dissection (Sommer et al, 1996) (*Figures 3a–d*). It requires the use of intravenous contrast and the extent of aortic dissection, including side branch involvement, can usually be clearly identified. Pleural and pericardial effusions are well seen. Its main disadvantage is the inability to diagnose aortic regurgitation and to localize entry tears precisely. Spiral (helical) CT is better than standard CT with images acquired rapidly in a single breath hold. This allows for better evaluation of the aorta as all images are obtained during optical contrast enhancement with less motion artefact.

Magnetic resonance imaging

MRI is the most accurate method for diagnosing aortic dissection (*Figure 4*). No contrast material is required, with excellent visualization of the intimal flap, extent of dissection, tear localization, aortic regurgitation, side branch involvement and complications. However, the technique is often not available on an emergency basis and examination of haemodynamically unstable patients is difficult. The examination times are

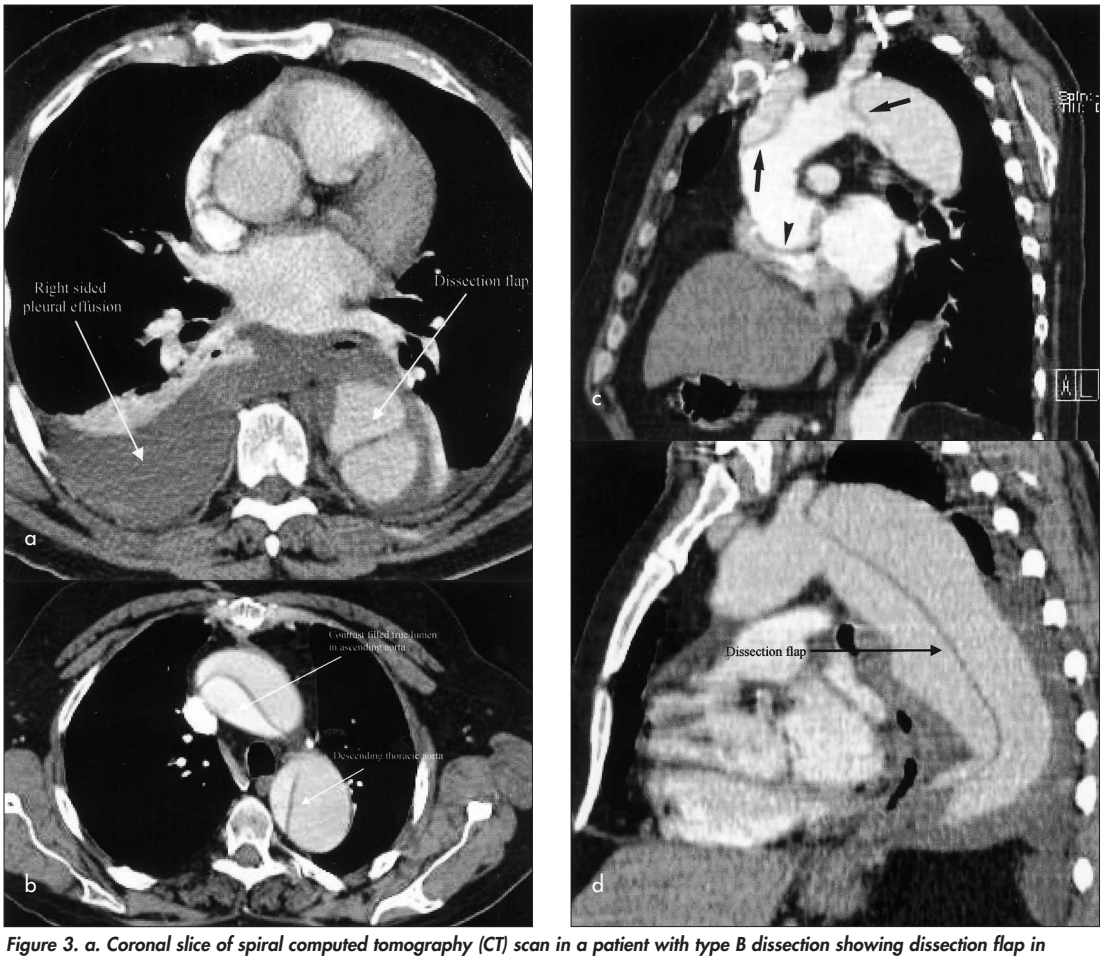

Figure 3. a. Coronal slice of spiral computed tomography (CT) scan in a patient with type B dissection showing dissection flap in descending thoracic aorta and right-sided pleural effusion. b. Coronal slice of spiral CT scan following contrast showing dissection flap in both ascending and descending thoracic aorta (type A dissection). Contrast is seen in the true lumen and not in the false lumen. c. Sagittal slice of spiral CT scan following contrast showing type A dissection (arrows) starting from aortic root and extending along the arch in a spiral fashion. d. Sagittal slice from spiral CT scan showing dissection flap in the descending thoracic aorta.

long, with patients having to remain motionless in a confined space with limited access. MRI is the method of choice for monitoring chronic aortic dissections but is not suitable for patients with metallic implants.

Transthoracic echocardiography

Transthoracic echocardiography (TTE) is unable to adequately visualize the distal ascending, transverse and descending thoracic aorta. Its sensitivity and specificity for diagnosing aortic dissection is much less than that of CT, MRI or TOE. Dissection flaps in the proximal ascending aorta can be visualized, and cardiac complications such as pericardial effusion, aortic regurgitation and regional left ventricular systolic function are readily identified.

Transoesophageal echocardiography

TOE allows for detailed examination of the ascending and descending thoracic aorta (*Figures 5a* and *b*) because of the close proximity of the aorta and oesophagus. TOE is the preferred imaging technique for acutely unstable patients and may be done in theatre immediately before operation. The distal ascending aorta and anterior aortic arch are not well seen with TOE. Only experienced operators should interpret TOE studies as false positive results occur from reverberation echos, calcified atheromatous

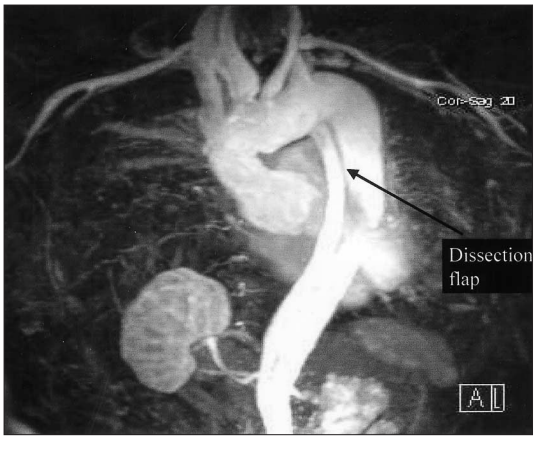

Figure 4. Magnetic resonance imaging scan showing dissection flap in the descending thoracic aorta.

plaque and motion artefacts (Erbel et al, 1989). When more spatial resolution is necessary, CT or MRI is used in addition.

Aortography

Aortography (*Figure 6*) was the first accurate diagnostic test for assessing patients with aortic dissection. However, the sensitivity is lower than other techniques as completely thrombosed false lumens or intramural haematomas may be missed (Chirillo et al, 1994). It is not readily available and is being replaced by other non-invasive tests. It is useful for imaging the aorta when performing stent graft implantation for type B aortic dissections. Coronary angiography is usually not required before surgery for acute aortic dissection, unless features suggest a high likelihood of significant coronary artery disease.

MEDICAL MANAGEMENT

Haemodynamic stabilization of the patient with adequate analgesia is the main aim of initial management. Patients should be in either a coronary or intensive care unit with blood pressure monitoring via an arterial line. Hypotension is suggestive of aortic perforation or cardiac tamponade, and these patients require fluid replacement and surgery. Percutaneous pericardiocentesis can lead to accelerated bleeding and is of no benefit (Isselbacher et al, 1994).

In most other patients, systolic blood pressure should be reduced to between 100 and 110 mmHg. Intravenous beta-blockers are the drugs of choice as they decrease shear stress on the aorta. Propanolol, labetolol or esmolol can be used. Labetolol blocks both alpha- and beta-receptors while esmolol has a short half-life and may be used in patients with potential intolerance to beta-blockers such as asthmatic individuals. If additional therapy is required to control blood pressure, vasodilators such as sodium nitroprusside or glycerol trinitrate may be added. Sodium nitroprusside should not be used without beta-blockade as it causes a reflex increase in sympathetic output and increases myocardial force of contraction. If no emergency surgery is planned then oral beta-blockers and other antihypertensive agents such as angiotensin-converting enzyme (ACE) inhibitors or calcium channel blockers can be started while intravenous therapy is weaned off.

INTERVENTIONAL TREATMENT

Open surgical repair

Type A aortic dissections affecting the ascending aorta should all be treated surgically if the patient is fit enough as there is a high risk of life-threatening complications such as aortic rupture, aortic regurgitation, cardiac tamponade or myocardial infarction. In untreated patients with type A dissection, 20% die within 24 hours, 30% by 48 hours, 40% by day 7 and 50% by 1 month.

Surgical intervention aims to excise the intimal tear and to obliterate the false lumen by replacing the ascending aorta with a synthetic vascular graft (*Figures 7a* and *b*). If the aortic valve is not suitable for repair than a composite graft (prosthetic aortic valve plus ascending aortic tube graft) is used with reimplantation of the coronary arteries into the graft. The distal connection of the tube graft is usually carried to the junction of the ascending aorta with the arch. Surgical risk is increased by the presence of extensive intimal tears that extend into the aortic arch. This requires more extensive surgery with arch replacement and reconnection of the supraaortic vessels.

Uncomplicated type B aortic dissections confined to the descending thoracic aorta are best treated medically. Healing is characterized by disappearance of the false lumen as a result of thrombosis. Surgical intervention is restricted to patients with evidence of aortic rupture, rapidly expanding aortic diameter, persistent pain or peripheral ischaemic complications. The aim of surgery is to replace the affected aorta with a tubular graft of appropriate length. Surgery is associated with a high incidence (up to 18%) of paraplegia as a result of spinal cord ischaemia and surgical mortality exceeds 80% in patients with end organ ischaemia. The most common causes of death are aortic rupture and visceral ischaemia. *Table 2* shows the inhospital mortality of patients with type A and type B aortic dissection treated surgically and medically.

FENESTRATION OF INTIMAL FLAP FOR PERIPHERAL ISCHAEMIC COMPLICATIONS

Peripheral ischaemic complications associated with aortic dissections are associated with a poor prognosis, with mesenteric and renal ischaemia having mortality rates of up to 87% and 70% respectively (Cambria et al, 1988). Ischaemia occurs when the false lumen compresses the true lumen preventing blood flow down the main aortic branches. The aim of percutaneous balloon fenestration is to create an artificial tear in the intimal flap to allow communication between the true and false lumen and an improvement in blood flow to ischaemic organs (Williams et al, 1990). The intimal flap is usually crossed from the true lumen to the false lumen using a Brockenborough needle. The needle is switched for a stiff wire and a tear is produced across the intimal flap using 10–14 mm diameter angioplasty balloons.

ENDOVASCULAR AORTIC STENTING

Stent grafts were first successfully used to treat aneurysms of the thoracic and abdominal aorta. They have been used as a less invasive alternative to surgery for the treatment of type B aortic dissections. The aim is to cover the entry tear and to induce thrombosis of the false lumen in order to stimulate the healing process. The first non-randomized study to compare stent grafting with surgery in type B aortic dissection included a total of 24 patients (Nienaber et al, 1999). There was no morbidity (paraplegia, stoke, embolization, branch occlusion or infection) or mortality in the 12 patients treated with stent grafts over a follow up of 1 year. In contrast, surgery was associated with a mortality of 33% and a 42% incidence of major adverse events. In a second series of 19 patients with more complex disease (14 with aortic branch involvement, four with retrograde dissections involving the ascending aorta) the 30-day mortality was 16% (Dake et al, 1999). Causes of death were rupture of the false lumen and ischaemia of the bowel or limbs. Among survivors, long-term results were excellent with no instances of aneurysm formation or aortic rupture during a mean follow-up period of 13 months. The stents are between 25 and 35 mm in diameter and up to 20 cm long (*Figure 8*) and are introduced via a 22–27 French sheath that often requires a surgical cut-down onto the femoral artery. Although a promising technique

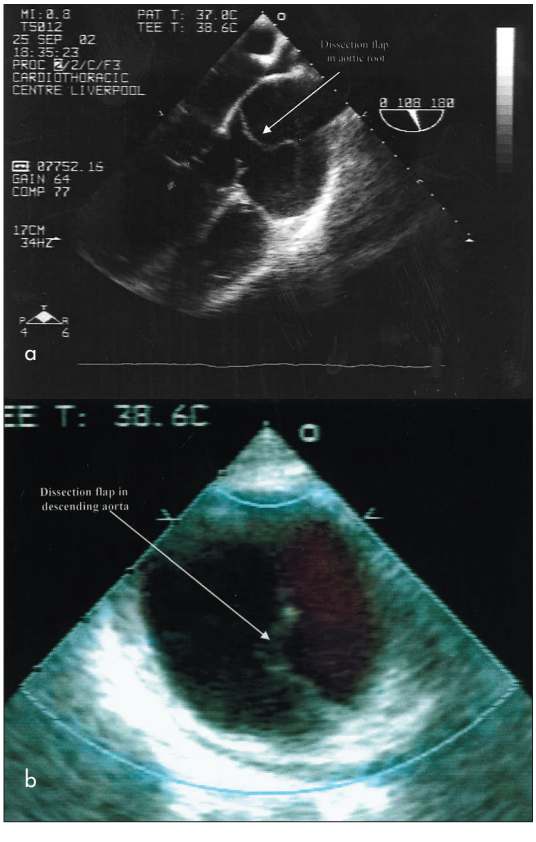

Figure 5. a. Transoesophageal echocardiograph (TOE) long axis image showing undulating dissection flap in the aortic root in a patient with Marfan's syndrome and type A dissection. b. TOE short axis view showing dissection flap in the descending thoracic aorta with colour flow Doppler (in red) demonstrating flow in the true lumen.

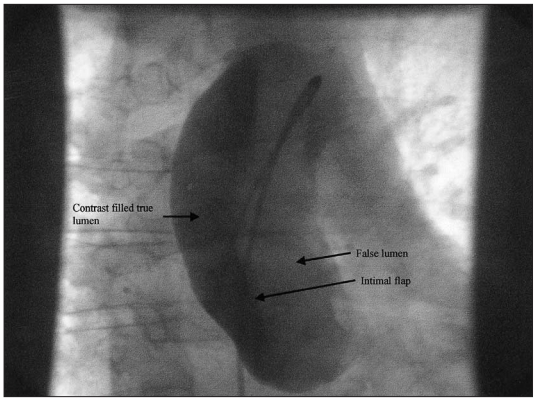

Figure 6. Aortogram showing contrast opacification of the true lumen separated by an intimal flap from the false lumen.

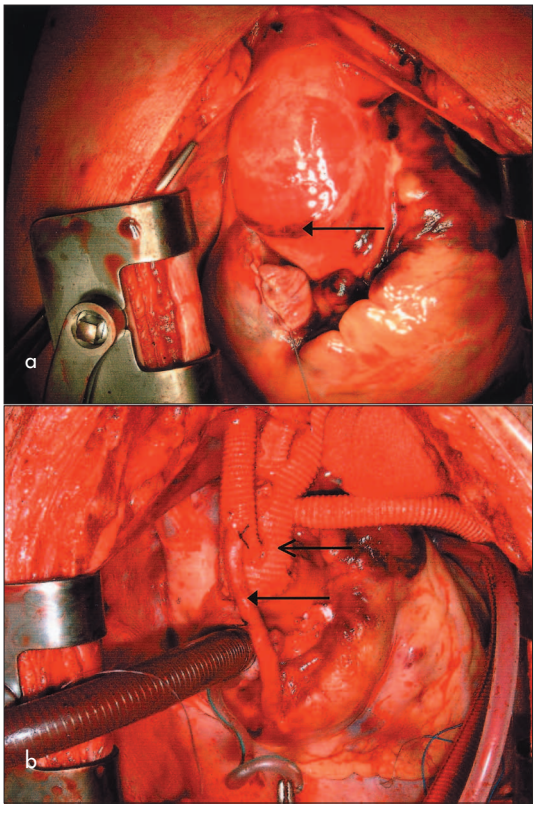

Figure 7. a. Dilated swollen ascending aorta with adventitial haematoma (arrow) seen at the time of surgery in a patient with type A dissection. b. Surgical repair of aortic dissection showing replacement of the aortic arch with a dacron graft (open arrow head), connected to prosthetic aortic valve (not seen) and root replacement. A saphenous vein graft (closed arrow head) is seen connected from the dacron graft to the right coronary artery which was involved in the dissection.

TABLE 2.
Inhospital mortality of patients with type A and type B aortic dissection treated surgically and medically

Diagnosis	In-hospital mortality
Type A dissection treated medically	50%
Type B dissection treated surgically	24%
Type A dissection treated surgically	20%
Type B dissection treated medically	10%

in development, experience is limited to a small number of patients and longer follow up is required. The procedure requires a multidisciplinary team consisting of cardiologist, interventional radiologist and vascular surgeon.

LONG-TERM PROGNOSIS AND FOLLOW UP

Long-term prognosis for patients who survive the acute episode is good with 5- and 10-year survival rates of 68% and 52% respectively for type A dissection treated surgically, and for medically-treated type B dissections of 80% and 40% respectively. The best prognosis is for non-communicating retrograde type B dissections treated medically (Erbel at al, 2001).

Risk factors for dissection are systemic factors that will affect the entire length of the aorta. It is estimated that one third of all patients who survive will experience dissection extension, aortic perforation or require surgery for aortic aneurysm formation within 5 years of initial presentation.

The single most important factor for long-term follow up is excellent blood pressure control with a target of <135/80 mmHg. The majority of late deaths are caused by aortic rupture and patients need regular MRI scans to look for signs of aortic expansion, aneurysm formation and leaks at anastomoses or stent sites. A specialist aortic aneurysm surgical team should ideally follow all patients. HM

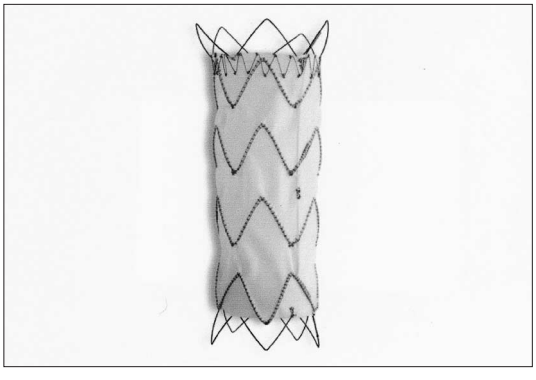

Figure 8. A Talent (Medtronic, Minneapolis, Minnesota, USA) covered stent used for endovascular repair of type B aortic dissection.

KEY POINTS

- A high index of clinical suspicion and early imaging are essential to diagnose aortic dissection.
- Acute severe chest pain with additional features of syncope, haemodynamic collapse or stroke is the most common presentation, although atypical cases are common.
- Class 1 dissections are the commonest and can be classified anatomically according to Stanford and De Bakey systems.
- Computed tomography scanning is the most common diagnostic imaging technique used although transoesophageal echocardiography and magnetic resonance imaging are equally effective and aortography may still be useful.
- Early liaison with cardiologists and/or cardiothoracic surgeons at a cardiac surgical centre is essential to optimize treatment.
- Blood pressure should be lowered to between 100 and 110 mmHg systolic with intravenous beta-blockers unless hypotensive.
- Surgical repair of the aorta is the treatment of choice for all type A dissections and for type B dissections where there is evidence of aortic rupture, branch vessel occlusion, persistent severe pain and aortic expansion.
- New techniques such as fenestration for branch vessel occlusion and endovascular stents are being developed as alternatives to surgery for type B dissections and initial results are very promising.
- Long-term follow up with a target blood pressure <135/80 mmHg and serial imaging to look for aortic aneurysm formation, anastomotic leaks or dissection extension is essential for all patients who survive the initial presentation.

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