Surgery of the thoracic aorta

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Abstract

Surgery for thoracic aortic pathology is challenging and is associated with significant early and late mortality and morbidity. In recent decades advances in diagnostic imaging, surgical techniques, myocardial and cerebral protection and graft design have significantly improved outcomes. Patient assessment is focused on timely diagnosis and intervention to prevent the catastrophic complications. The major challenge perioperatively is to minimize ischaemic injury to end organs, particularly the brain and spinal cord. Acute aortic dissection is the most common life-threatening thoracic aorta emergency and carries a high mortality rate with 40% of patients dying immediately. Early recognition and diagnosis of aortic dissection is therefore essential. Surgical intervention is required for ascending aortic dissection. Medical or endovascular management may be appropriate if the ascending aorta is not involved. Thoracic aortic aneurysms are usually asymptomatic and chronic in nature. Surgery is usually indicated for large or rapidly expanding aneurysms, or once a patient develops symptoms. Earlier surgery is advocated for patients with connective tissue disorders such as Marfan syndrome because of an increased risk of rupture. The advent of endovascular stent technology has opened the door for a minimally-invasive approach to the treatment of aortic pathology with encouraging early and medium-term outcomes reported.

Keywords Aneurysm; dissection; endovascular stent; thoracic aorta; trauma

Anatomy

The thoracic aorta may be subdivided into four distinct anatomical sections, each posing different challenges to the cardiac surgeon:

Aortic root: the aortic root forms a bridge between the left ventricle and the ascending aorta and includes the aortic valve annulus, aortic valve cusps and sinuses of Valsalva.

Ascending aorta: the tubular portion of ascending aorta, commencing from the sinotubular junction up to the origin of the brachiocephalic artery.

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Aortic arch: originates just proximal to the origin of the innominate artery and terminates just beyond the left subclavian artery.

Descending aorta: begins at the isthmus between the origin of the left subclavian artery and the ligamentum arteriosum and passes through the diaphragm into the abdomen.

Aortic dissection

Aortic dissection is defined as an intimal tear that allows blood to enter into the media resulting in disruptions in the layers of the aorta. Aortic dissection forms part of a triad of interrelated conditions known as acute aortic syndromes which also include intramural haematoma and penetrating aortic ulcer. An intramural haematoma is thought to arise from spontaneous rupture of vasa vasorum into the media, resulting in a concentric haematoma in the wall of the aorta. A penetrating aortic ulcer arises from an atheromatous plaque ulcerating and eroding the internal elastic lamina, exposing the media to pulsatile blood flow and haematoma formation. It can progress into a dissection or intramural haematoma.

The incidence of acute aortic dissection is estimated at 2–3 per 100,000 per year in the UK with 400–500 cases reported each year in the UK. Men are affected two to three times more frequently than women. Aortic dissection is a catastrophic condition if not recognized early and treated promptly with 30% of untreated patients dying within 24 hours and 50% by 48 hours, with an estimated 1% mortality per hour.

The dissection may be classified according to timing with acute dissection defined as occurring within first 2 weeks, subacute 2 weeks to 2 months, and chronic dissection presenting more than 2 months after initial event.

Risk factors for aortic dissection include hypertension, atherosclerosis, degenerative (cystic medial necrosis), bicuspid aortic valve, coarctation and connective tissue disorders (e.g. Marfan syndrome, Ehlers–Danlos syndrome, Loeys–Dietz syndrome) and pregnancy. Hypertension is present in over 75% of patients with aortic dissection. Dissection following trauma and iatrogenic dissection (e.g. during endovascular intervention) are less commonly seen.

Pathogenesis

A tear in the aortic intima leads to redirection of blood flow into the media creating an inner true lumen and an outer false lumen. This tear can be attributed to cystic medial necrosis, intramural haematoma or a penetrating ulcer. The cleavage plane between the two lumina is the dissection flap. The intimal tear usually occurs in the ascending aorta about 2–3 cm distal to the aortic valve and usually involves >50% of the aortic circumference. The dissection may extend distally (antegrade) for a variable distance and may compromise the perfusion of organs supplied by side branches of the aorta causing end-organ ischaemia (malperfusion syndrome). The false lumen may communicate distally with the true lumen through fenestrations. Retrograde extension, towards the coronary ostia and the aortic valve, may cause myocardial ischaemia and aortic regurgitation. Rupture of the false lumen into the pleura or pericardium is rapidly fatal.

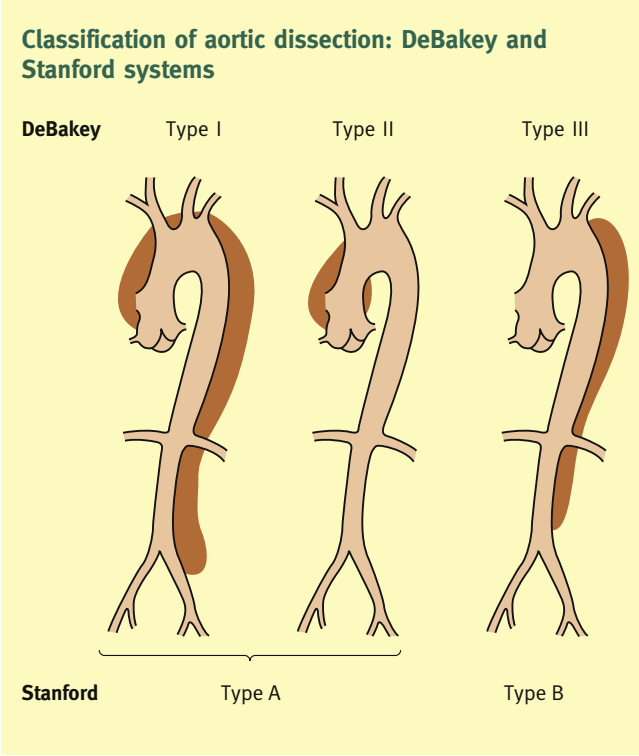

Figure 1

Classification

Figure 1 shows the two commonly used classification systems — the DeBakey and Stanford. In Stanford type A, the dissection involves the ascending aorta, whether or not it also involves the arch and the descending aorta, and regardless of the site of origin (Figure 2). In Stanford type B the dissection does not involve the ascending aorta. DeBakey type I involves the ascending, arch and descending aorta, Type II involves only the ascending aorta while type III is dissection restricted to the descending aorta. The Stanford classification is simpler and thus is more widely used. The Stanford classification also has implications in deciding how the dissection is to be managed. Stanford Type A dissections are managed surgically whereas type B dissections are primarily managed medically unless an indication for surgery is present.

Clinical presentation and diagnosis

The diagnosis of acute aortic dissection requires a high index of suspicion from the outset as up to 30% of patients eventually diagnosed with aortic dissection are thought to have other conditions such as acute coronary syndrome or pulmonary embolism.

The typical presentation for AAD is sudden onset severe, unrelenting, 'tearing', 'ripping', retrosternal or interscapular chest pain. The pain is often migratory, indicating extension of the dissection. The associated clinical picture depends on the extent of the dissection and the involvement of the aortic side branches. Patients may have signs or symptoms related to malperfusion of the brain (e.g. stroke), limbs (pulse deficit and limb ischaemia), or visceral organs. Involvement of the mesenteric and renal arteries may, for example, manifest as abdominal pain and oliguria. Syncope is an ominous feature often heralding cardiac tamponade or obstruction of cerebral vessels. A diastolic murmur of aortic regurgitation may be present due to prolapse of unsupported right or non-coronary valve cusps. A complete neurological examination is essential to document any baseline neurological deficit.

Diagnosis

An ECG should be performed in all patients and may demonstrate ischaemic changes. Chest X-ray is abnormal in 60—90% of patients with AAD, with findings such as widened mediastinum, irregular aortic contour, opacification of aortopulmonary window, loss of aortic knob and left pleural effusion.

Diagnostic imaging studies should confirm the diagnosis, localize the tear, define the extent of dissection and classify the type of aortic dissection.

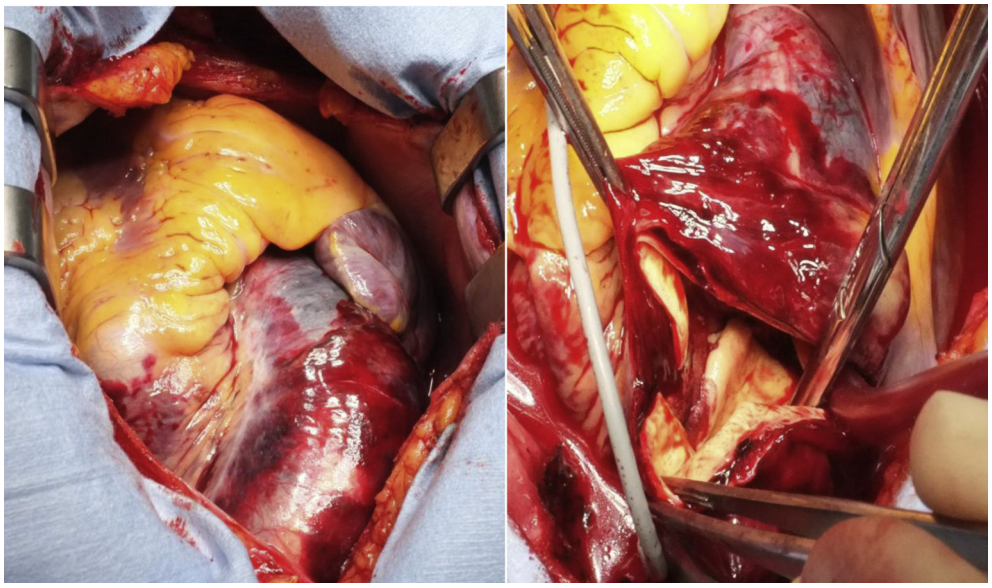

Figure 2 Acute type A aortic dissection.