Diagnosis and management of aortic dissection

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Key points

Aortic dissection can be easily missed. A high index of suspicion is important in patients who have predisposing risk factors.

Classification is based on the location of dissection and its duration. Stanford type A dissections require surgery; type B dissections may be managed non-surgically under most conditions.

Multiple diagnostic imaging modalities can be used to complement each other depending upon the availability of facilities and patient condition.

Anaesthetic management of these patients is challenging because of significant haemodynamic instability.

Chances of survival are improved with prompt diagnosis, blood pressure and heart rate control, and early surgical repair if indicated.

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Aortic dissection is a rare but potentially fatal event resulting in separation of the layers of the tunica media by ingress of blood, producing a false lumen with variable proximal and distal extension. Ascending aortic dissection is the most common catastrophe of the aorta; it is 2–3 times more common than rupture of the abdominal aorta. Mortality of untreated acute dissection involving the ascending aorta is about 1–2% per hour during the first 48 h. The first documented famous case was King George II.

Classification

Several different classifications have been advocated to describe aortic dissection. The classification systems in common use are either based on the duration of onset of symptoms prior to presentation or the anatomy of the dissection.

Aortic dissection is acute if the diagnosis is made within 2 weeks following the initial onset of symptoms, and chronic if present for more than 2 weeks. Recently, the European Society of Cardiology Task Force on Aortic Dissection has come up with a more comprehensive etiological classification (Table 1) (Fig. 1). Advanced imaging technology has demonstrated that intramural haemorrhage, intramural haematoma, and aortic ulcers may be signs of evolving dissections or dissection subtypes. All these are grouped under acute aortic syndromes.

Classical anatomical aortic dissection is classified according to DeBakey or Stanford. The most commonly used is the Stanford classification, which is based on involvement of the ascending aorta.

Stanford classification

Type A involves the ascending aorta but may extend into the arch and descending aorta (DeBakey type I and II).

Type B involves the descending aorta only (DeBakey type III).

In Stanford type A, the ascending aorta is always involved. In Stanford type B, the dissection is distal to the origin of the left subclavian artery. The Stanford system also helps to delineate two distinct risk groups for management. Usually, type A dissections require surgery, while type B dissections are best managed conservatively with medical treatment under most conditions.

DeBakey classification

Type I involves ascending aorta, aortic arch, and descending aorta.

Type II is confined to ascending aorta only.

Type III is confined to descending aorta distal to the left subclavian artery only; IIIa extends up to diaphragm, IIIb extends beyond the diaphragm.

Pathophysiology

Aortic dissection is more common in males with a peak incidence at 50–70 yr of age. Aortic dissection can result either from a tear in the intima and propagation of blood into the media or from intramural haemorrhage and haematoma formation in the media followed by perforation of intima; the former is more common. The characteristic picture of aortic dissection is the presence of an intimal flap in the aorta. These are commonly preceded by medial wall degeneration or cystic medial necrosis. Blood may re-enter the true lumen at any point, thus making it a communicating dissection.

An intimal tear can occur in the regions of the aorta that are subjected to the greatest stress and pressure fluctuations. Because mechanical stress in the aortic wall is proportional to intramural pressure and vessel diameter, hypertension and aortic dilatation are known risk factors for dissections. Integral wall abnormalities such

Table 1 European Society of Cardiology Classification[4]

Class 1	Classical aortic dissection
Class 2	Intramural haematoma/haemorrhage
Class 3	Subtle-discrete aortic dissection
Class 4	Plaque rupture/ulceration
Class 5	Traumatic/iatrogenic aortic dissection

as Marfan's syndrome may also predispose to dissection. While no single disorder is responsible, several risk factors have been identified that can damage the aortic wall and lead to dissection (Table 2). Most aortic dissections occur with an initial transverse tear along the greater curvature of the aorta, usually within 10 cm of the aortic valve. The aortic root motion has a direct impact on the mechanical stresses acting on the aorta.[1] The next most common site is the descending thoracic aorta immediately distal to the origin of the left subclavian artery.

Clinical features

Clinically, aortic dissection presents as a two-step process. The first event is the interruption of the intima which is associated with severe pain and loss of pulse volume. The second event sets in when the pressure exceeds a critical limit and rupture occurs.

Abrupt sharp high-intensity chest pain at the onset is the most specific characteristic of aortic dissection. It has been described as stabbing, tearing, or ripping in nature. Analysis of the International Registry of Acute Dissection (IRAD), noted that severe chest pain

is more common with type A dissections, whereas back pain and abdominal pain are more common in type B dissection. The pain may be migratory and follow the path of propagation of the dissection. The clinical manifestations are diverse and overlap.

Physical examination may reveal tachycardia, usually accompanied by hypertension in the setting of baseline primary hypertension and increased catecholamine levels from anxiety and pain. Tachycardia and hypotension result from aortic rupture, pericardial tamponade, acute aortic valve regurgitation, or even acute myocardial ischaemia with involvement of the coronary ostia. Differential or absent pulses in the extremities and a diastolic murmur of aortic regurgitation may also be present. Syncope, stroke, and other neurological manifestations secondary to malperfusion syndrome may develop. A complete neurological examination is essential and findings should be documented.

Diagnosis

Aortic dissection has a wide range of clinical presentations. A high index of suspicion is important in patients with predisposing risk factors, e.g. hypertension, aneurysmal disease of the aorta, or a familial connective tissue disorders. Typically the patient is a hypertensive male in his 60s, with a history of abrupt onset of chest pain.

In all patients, an immediate ECG must be done to exclude acute myocardial infarction for which the treatment is very different and may involve thrombolysis. About 20% of patients with type A dissection have ischaemic changes on ECG due to
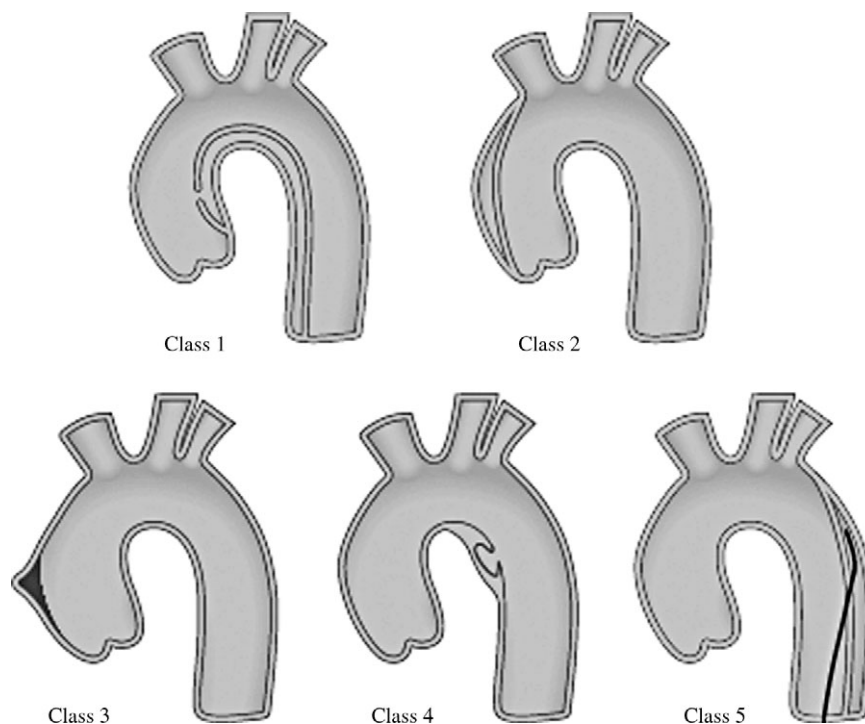

Class 1   Class 2

Class 3   Class 4   Class 5

Fig. 1 European Society of Cardiology Classification.[4]

Table 2 Risk factors for aortic dissection (Adapted from Erbel *et al.*[1])

Long standing arterial hypertension
    Advanced age
    Smoking
    Dyslipidaemia
    Cocaine/crack
Connective tissue disorders
    Hereditary fibrillinopathies
        Marfan's syndrome
        Ehlers-Danlos syndrome
        Turner's syndrome
    Hereditary vascular diseases
        Bicuspid aortic valve
        Coarctation
    Vascular inflammation
        Giant cell arteritis
        Takayasu arteritis
        Syphilis
Aortic aneurysm
Pregnancy
Deceleration trauma
    Accident
    Fall from height
Iatrogenic factors
    Catheter/Instrument intervention
    Aortic surgery
        Cross-clamp or side clamp
        Graft anastomosis
    Cannulation site

Table 3 Diagnostic goals (Adapted from Erbel *et al.*[4])

Confirm diagnosis
Classify the dissection/delineate the extent
Differentiate true and false lumens
Localize intimal tear; intimal flap, entry sites
Distinguish between communicating and non-communicating dissection
Assess side branch involvement (i.e. coronary, carotid, subclavian, ceoliac, and renal arteries)
Detect and grade aortic regurgitation
Detect extravasations (peri-aortic or mediastinal haematoma, pleural or pericardial effusion, tamponade)

extension of the dissection into a coronary ostium. In such patients, further imaging should be done before thrombolysis or revascularization procedures are attempted. Biochemical markers of myocardial damage may help in the diagnosis.[2] The most promising biochemical marker to date for diagnosing acute aortic dissection is an elevated circulating smooth muscle myosin heavy chain protein; this is released from damaged aortic medial smooth muscle.[3]

Imaging

Multiple modalities (CT, MRI scanning, and echocardiography) can be used to complement each other to facilitate diagnosis depending upon availability. The overall diagnostic accuracy of these different modalities is similar.[4] Table 3 outlines the diagnostic goals.

On chest X-ray (CXR), aortic knuckle changes may be observed, with intimal calcification separated more than 6 mm from the edge. A widened mediastinum, cardiomegaly (pericardial effusion), and loss of costo-phrenic angle secondary to the presence of a haemothorax may also be noted. Further management should not be delayed in an unstable patient.

Aortography is the historical 'gold standard' for diagnosis. This distinguishes the origin of branch arteries from true or false lumens. This is not appropriate in the unstable patient. With the availability of advanced non-invasive imaging techniques, aortography is nowadays rarely performed.

A CT scan is relatively rapid and non-invasive and with contrast image enhancement the extent of the dissection along with the true and false lumens can be identified. This technique is not appropriate if the patient is haemodynamically unstable. MRI gives high-resolution images without contrast dye, but can be time consuming. It is not advocated in haemodynamically unstable patients.

Transthoracic echocardiography (TTE) is easily available and the ascending aorta and aortic arch can be visualized well. In obese or chest trauma patients, image quality may be inadequate due to poor echo windows. Transoesophageal echocardiography (TOE) has become more popular as experience and availability increase. It is useful perioperatively in the haemodynamically unstable patient. TOE images the entire thoracic aorta except for the most distal ascending aorta and a part of the arch obscured by the trachea or right main bronchus. Echocardiography can be used with high accuracy for decision-making in acute dissection. Echo and acoustic artifacts can be misleading and should be differentiated from the intimal flap by examining the pathology in several image planes.[4] Intravascular ultrasound is a catheter-based imaging study which provides dynamic imaging of the aortic wall and intimal flap.

Initial management

Acute type A and complicated type B dissections should be managed surgically in a regional cardiothoracic centre. This often requires transfer from a peripheral hospital. Initial management depends upon clinical presentation (Table 4). The patient should be cared for in a critical care environment and early surgical involvement is essential. Depending on the urgency, coexisting medical conditions should be investigated and treated. Pain should be treated with adequate analgesics.

The primary goal is to reduce the force of left ventricular contraction without compromising perfusion, thus reducing shear forces and preventing further extension of the dissection or possible rupture. Beta-blockers (e.g. esmolol, metoprolol) and labetalol (beta- and alpha-blocker) can be used. If further reduction in BP is required, sodium nitroprusside, glyceryl trinitrate, or hydralazine are appropriate. Beta-blockers should be given first before vasodilators, as the reflex catecholamine release due to vasodilatation may increase left ventricular contractions. If the patient has a low

Table 4 Initial management of patients with suspected aortic dissection (Adapted from Erbel et al.[4])

Oxygen (ABC as indicated)
Detailed medical history and complete physical examination (whenever possible)
HR, BP, and Sp_{O_2} monitoring
i.v. line, bloods (Cross match, CK, Troponin, FBC, U & Es, Myoglobin, D-dimer, LDH)
12-lead ECG: documentation of ischaemia
Pain relief (morphine sulphate)
Careful i.v. fluid infusion
BP titration to about 110–120 mm Hg systolic with i.v. esmolol, metoprolol, or labetalol first.
Sodium nitroprusside for further control of blood pressure (calcium channel blockers if beta-blockers are contraindicated)
Imaging studies at the earliest opportunity
Transfer to theatre/regional cardiothoracic centre/intensive care unit as appropriate

Glasgow coma scale (GCS <8) or profound haemodynamic instability, intubation and ventilation are indicated.

Surgical management

Acute type A aortic dissections are operated upon without delay, as rupture can be imminent. Possible contraindications include paraplegia and severe incurable comorbidities. Neurological involvement, metabolic acidosis, and acute renal impairment are associated with a poor prognosis.

Several surgical approaches are described. The goals of surgical therapy are to prevent extension, excise the intimal tear, and replace the segment of aorta susceptible to rupture with an interposition synthetic graft (elephant trunk technique). Combined aortic valve and ascending aorta replacement with re-implantation of coronary arteries using a composite graft is performed if the aortic valve is not salvageable.

In acute type B aortic dissections, surgical intervention is only indicated if there is persistent or recurrent intractable pain, aneurysm expansion, peripheral ischaemic complications, and rupture. This is because surgical repair has no proven superiority over nonsurgical treatment in stable type B dissection patients.

Anaesthetic considerations

Anaesthetists are involved in resuscitation and stabilization, pain relief, sedation for TOE, transfer, anaesthesia, and perioperative care of aortic dissection patients. The anaesthetist's role may also include diagnostic perioperative TOE to aid surgical decision making.

Type A dissection

Monitoring and lines

Standard anaesthetic principles for cardiac surgery are followed. Adequate peripheral venous access should be established. Titration of fluid infusions is paramount as over zealous fluid administration may lead to progression of dissection and rupture. As described earlier, antihypertensive therapy with beta-blockers should be continued during induction and maintenance of anaesthesia.

Left radial arterial pressure monitoring is preferred as the innominate artery may be involved in the dissection and therefore affect right radial artery pressures. Anatomy should be verified prior to insertion of the arterial line. A central line (CVC) is secured before or after induction of anaesthesia. Meticulous monitoring and treatment of haemodynamic instability, acidosis, coagulopathy, and low urinary output is of the essence. Continuous TOE monitoring is helpful to guide surgical decision making.

Induction and maintenance

Surgery of the ascending aorta is usually performed via a median sternotomy, while any procedure to the descending aorta requires a left thoracotomy carrying its own implications. A volatile anaesthetic agent like isoflurane or sevoflurane with its potential advantages to the myocardium, and or propofol is used for maintenance of anaesthesia. Further titration of opioid analgesia (fentanyl/alfentanil/morphine) and muscle relaxing agent will help to provide optimal surgical conditions and patient care. Prolonged muscle relaxation should be avoided to facilitate early postoperative extubation and neurological evaluation when appropriate.

Cannulation options and cardiopulmonary bypass

Cardiopulmonary bypass (CPB) is established using various cannulation sites depending on the anatomy and urgency. Arterial cannulation for antegrade perfusion is accomplished either via the distal aortic arch if not involved, right subclavian artery, innominate artery, or true lumen of the dissected ascending aorta. An alternative cannulation site for antegrade perfusion is through the left ventricular apex and aortic valve. Cannulation of either femoral artery will provide retrograde aortic perfusion with potential extension of the dissection area. Venous cannulation is most often through the right atrium using a two-stage venous cannula. Femoral or bicaval venous cannulation are other options. If the aortic valve is incompetent, a left-ventricular vent is necessary to prevent left-ventricular distension and subsequent subendocardial ischaemia. TOE is very useful in guiding various cannulation manoeuvres.

The procedure may require partial CPB or deep hypothermic circulatory arrest (18–20°C) while performing the distal aortic anastomosis. CPB time and aortic cross-clamp times may be prolonged because of the complexity of surgery. Circulation is re-established through the true lumen after surgical repair and then the patient is weaned off CPB appropriately.

Cerebral protection

Repair of dissection involving the arch requires disruption of blood flow to the brain. Cerebral protection during this period can be achieved through either deep hypothermia with cessation of electrical activity or continued cerebral perfusion by retrograde or antegrade fashion. The patient's head should be packed in ice during total circulatory arrest. A short duration of aortic cross-clamp and deep hypothermic cardiac arrest is essential to limit cerebral and cardiac damage. Methylprednisolone, thiopental, and lidocaine administration during cooling are adjunctive measures thought by

some to decrease cerebral metabolic requirements and limit neurological damage. There are potential protective properties of halogenated anaesthetics such as isoflurane and sevoflurane since the concept of protective ischaemic preconditioning by these compounds is well established in other organs, particularly in the heart.

Furosemide and mannitol may be administered to initiate diuresis and promote free radical scavenging following circulatory arrest. New EEG processing monitors such as the Bispectral Index has the ability to give dynamic information about the state of the brain during various stages of anaesthesia and surgery.

Temperature regulation

A fluid warmer, warming blanket, and warming water mattress are helpful to rewarm and prevent hypothermia after weaning from CPB.

Coagulation

Disseminated intravascular coagulopathy can occur because of continuing major haemorrhage, prolonged CPB or circulatory arrest time, hypothermia, massive blood transfusion, or drugs. Prophylactic use of aprotinin (infusions before, during and after CPB) and tranexamic acid have been advocated. A cell saver should be used if available. Thromboelastography can give useful information to guide coagulation therapy. Liaison with a haematologist regarding blood product replacement is helpful as these patients often have complex transfusion requirements.

Pain relief

Perioperative pain relief is usually obtained through medium to high doses of opioids. The role of epidural analgesia is controversial because of its potential to cause or mask spinal damage.

Type B dissection

Surgery involving the descending or thoracoabdominal aorta requires a left lateral thoracotomy. One-lung ventilation with a double-lumen endotracheal tube is standard procedure in such cases. A right radial arterial pressure line is essential as the left subclavian artery may be clamped. Femoral arterial pressure is also monitored to ensure adequate perfusion of the lower body.

Postoperative management

The patient should be closely monitored in a critical care unit after surgery. Mechanical ventilation is continued until the patient is warm and haemodynamically stable with minimal surgical bleeding and satisfactory gas exchange. Blood pressure should be well controlled with adequate analgesia and antihypertensive agents to prevent further complications. A full physical examination including complete peripheral vascular exam is performed on admission to critical care unit. Gross neurological examination should be carried out at the earliest possible time. ECG and CXR are performed to reveal any abnormalities and serve as a baseline for postoperative progress.

At this time, patients may require continuous correction of coagulopathy together with renal replacement therapy. Visceral ischaemia, metabolic acidosis, infection, and respiratory complications are potential problems.

Interventional management

Endovascular interventions are gaining popularity in type B aortic dissections, especially in patients at high risk for thoracotomy because of severe coexisting cardiopulmonary abnormalities or other medical problems. The advent of percutaneous stenting and/or fenestration technology provides an alternative to open surgery for selected patients. Aims of the treatment include reconstruction of the thoracic aortic segment containing the entry tear, induction of thrombosis of the false lumen, and re-establishment of the true lumen and side branch flow.[3]

Medical management

Medical management is preferred for uncomplicated descending aortic dissections. Adequate pain relief is provided as required. The primary focus of medical management is to reduce blood pressure and hence prevent extension of the dissection.

Prognosis and follow-up

Aortic rupture, cardiac tamponade, circulatory failure, stroke, or visceral ischaemia are the most common causes of death. According to the IRAD,[5] current overall in-hospital mortality rates for type A dissections was 26% for surgically managed and 58% for medically managed patients (advanced age, comorbidities). Mortality rates for type B dissection were 31.4% and 10.7%, respectively. Once the patient is discharged, medical follow-up with a focus on tight blood pressure control should be performed regularly. Serial aortic imaging surveillance is recommended. The overall 10 yr mortality is about 55% in treated patients. In-hospital mortality remains high, despite recent advances. Survival rates are improved by prevention, prompt diagnosis, and timely management.

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