Special review

Diseases of the Aorta in the Critically Ill

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ABSTRACT

Objective: *To review diseases of the aorta that commonly require management in the critical care unit.*

Data sources: *Articles and published reviews on aortic dissection, traumatic aortic rupture and aortic aneurysm.*

Summary of review: *The aorta is the largest arterial vessel of the body and disorders that can lead to rupture (e.g. aortic dissection, traumatic aortic rupture and aortic aneurysm) are life threatening.*

Aortic dissections are usually classified for surgical purposes as those that involve the ascending aorta (i.e. type A dissections) which are usually managed surgically and all other dissections (i.e. type B dissections) which are usually managed non surgically. Recently, endoluminal aortic stents have been used to manage type B dissections. Traumatic aortic rupture usually follows an antero-posterior thoracic injury with 60% occurring just distal to the origin of the left subclavian artery and 25% at the ascending aorta. Treatment consists of open surgical repair ensuring that the aortic cross clamp times are less than 30 minutes to reduce the development of ischaemic cord lesions. Aortic aneurysm commonly occurs in the abdominal aorta and is usually surgically resected if > 5 cm in diameter. Post-operative care is commonly undertaken in the intensive care unit to monitor haemodynamic, respiratory and fluid and electrolyte status to reduce the incidence of renal and respiratory failure.

While angiography is often used to diagnose these disorders, transoesophageal echocardiography, helical computed tomography (CT) and magnetic resonance imaging are becoming more commonly used and in selected conditions are recommended as the investigations of choice.

Conclusions: *Aortic dissection, aortic aneurysm and aortic trauma may lead to aortic rupture with exsanguination and management commonly requires surgical intervention and postoperative care in a critical care unit.* (**Critical Care and Resuscitation 2000; 2: 117-124**)

Key Words: Aortic dissection, aortic aneurysm, traumatic aortic rupture

The aorta is the largest arterial vessel of the body and the conduit through which blood from the left ventricle is delivered to the systemic arterial bed. The elastic properties of the aorta provide a mechanical buffer to the circulation; by distending during systole and recoiling during diastole the left ventricular pulse pressure is reduced and a constant flow of blood is provided to the peripheral tissues.

In adults the diameter of the aorta is approximately 3 cm at its origin, 2.5 cm throughout the descending thoracic portion and approximately 2 cm throughout the abdominal portion. The normal maximum infrarenal aortic diameter is 2.5 cm in patients older than 50.[1] Diseases of the aorta that usually require management in the intensive care unit are aortic dissection, traumatic aortic rupture and aortic aneurysm.

Aortic dissection

Dissection of the aorta commonly starts as a tear in the intima which is perpendicular to the long axis of the thoracic aorta. This permits blood to enter the aortic wall causing an intramural haematoma that progresses within the outer third of the media proximally and distally from the site of the tear. The tear usually begins

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in one of two sites. The proximal portion of the ascending aorta (within a few centimetres of the aortic valve) is the commonest site (e.g. 60% cases). The next most common location is at the beginning of the descending aorta just distal to the origin of the left subclavian artery in the region of insertion of the ligamentum arteriosum (e.g. 30% cases).[2] Both sites are thought to bear the brunt of each systolic beat, accounting for the high incidence of an intimal tear at these two points in the aorta.[3] The arch vessels (i.e. innominate, left common carotid and left subclavian), right coronary, left intercostal, left renal and the left iliac arteries are often involved.[4] The ratio of males to females affected is 4:1.[5]

Causes

Intimal defects alone are not sufficient to explain the dissection and an abnormality of the media (e.g. cystic medial necrosis) is usually required. Atherosclerosis is thought to play a minimal role only.[3] Systemic hypertension is present in more than 70% of patients and is thought to provide the direct mechanical force needed to provoke the dissection. Both the mean arterial pressure and the rate of rise of the blood pressure are important in the pathogenesis of the disorder. Diseases commonly associated with aortic dissection are listed in table 1.[3,6]

Table 1 Diseases associated with aortic dissection

Pregnancy
Marfan's syndrome
Coarctation of the aorta
Turner's syndrome
Bicuspid aortic valve, aortic stenosis
Aortic surgery
Cardiopulmonary bypass
Coronary artery vein graft
Aortic aneurysm repair
Aortography
Giant cell arteritis
Ehlers-Danlos syndrome
Polycystic renal disease

Clinical features

The history is important in diagnosing this disorder with one study finding that a diagnosis of an aortic dissection was made in > 90% of cases from the history alone.[7]

Symptoms include, an abrupt, sharp anterior chest pain (rather than the classic 'tearing' or 'ripping' pain) which occurs in more than 70% of patients with a proximal (type A) dissection, whereas in patients with distal (type B) dissection the pain is experienced more often in the back (it may present as interscapular pain only[8]) or abdomen.[9] The pain is maximum in intensity at its beginning (contrasting to the pain of myocardial infarction which is crescendo in nature) and often makes the patient writhe (which is uncommon with myocardial infarction pain, which is usually oppressive and makes the patient lie still).[2] It may also be migratory (in up to 16%) as the dissection progresses.[9] If the pain does not radiate to the back, the dissection is more likely to be limited to the ascending aorta.[8] Syncope occurs in up to 12%[9] and suggests that cardiac tamponade is likely.[8]

Signs include, diaphoresis, tachycardia, hypertension (which is commonly recorded when patients are first seen, even if they have poor peripheral perfusion) or hypotension (particularly if aortic regurgitation, tamponade or aortic rupture has occurred) and pulmonary oedema (indicating aortic regurgitation or dissection of a coronary artery with myocardial infarction).

Compression and occlusion of any branch of the aorta may also occur, with clinical features of obstructive vascular lesions presenting as a painful white leg (indicating that one of the iliac arteries may be involved), TIAs, an altered state of consciousness and hemiparesis or hemiplegia, (indicating involvement of the carotid arteries),[10] or paraparesis or paraplegia (indicating the involvement of spinal arteries).[10] In one study the incidence of absent femoral pulses and a pulse deficit (when the blood pressure was measured in both arms) was 20%, renal artery ischaemia with acute renal failure 15%, coronary artery occlusion 10%, carotid artery compression with cerebral ischaemia 5% and mesenteric artery ischaemia and spinal artery ischaemia 3%.[11] Compression of adjacent structures may also cause superior vena cava syndrome, Horner's syndrome or, rarely, vocal cord paralysis.

Investigations

While tests are characteristically used to diagnose the presence of an aortic dissection, some may be used to detect the involvement of the ascending aorta, extent of the dissection (i.e. sites of entry and re-entry), presence of thrombus in the false lumen, branch vessel involvement (e.g. coronary artery involvement and coronary artery disease), aortic insufficiency, and pericardial fluid (e.g. effusion or tamponade), to guide any planned surgical intervention. These include:

Chest X-ray: this may show a widening of the superior mediastinum (although in one series it was absent in 37.4% of cases[9]) and aorta (the descending aorta may appear wider than the ascending aorta) and left sided pleural effusion. In one series a normal chest X-ray was found in 12.4% of cases.[9]

ECG: this is characteristically normal, although it may show non specific T wave changes. ECG features

of myocardial ischaemia or infarction may also occur rarely with obstruction of a coronary ostium.

Plasma enzymes: lactic acid dehydrogenase isoenzyme 1 may be elevated (due to haemolysis), without elevation of creatine phosphokinase isoenzyme MB, or cardiac troponin T or I.

Haemoglobin: anaemia may occur, although it will usually take 2-4 days to develop.

Transoesophageal echocardiography: this is becoming the initial imaging modality of choice. It has a sensitivity of 99% and a specificity of 98% in diagnosing the presence of aortic dissection and is also of use in the assessment of left ventricular function, pericardial tamponade and aortic valve involvement.[12-15] However, intimal tears of the arch may be difficult to demonstrate and its accuracy is operator dependent.

Computed tomography (CT) and Magnetic resonance (MR) imaging: while CT scanning with contrast has been used to diagnose aortic dissection and sensitivities of greater than 90% and specificities approaching 100% have been reported,[13] the site of entry may not be identified and it cannot identify the presence of aortic insufficiency or the involvement of aortic branch vessels.[15] MR imaging has a sensitivity and specificity of 98% in the diagnosis of aortic dissection and has been used as the investigation of choice in the haemodynamically stable patient.[16] Recent reports of helical CT have reported sensitivities and specificities of 100% in diagnosis of an acute aortic dissection and may now be the investigation of choice in both haemodynamically stable and unstable patients.[17] It also has the advantages of being able to identify atypical forms of aortic dissection (with atypical configurations of the intimal flap), intramural haematoma, penetrating atherosclerotic ulcer and ruptured type B dissection.[17] Helical CT has also been useful in follow-up assessment of early and late changes after surgery or medical treatment.

Aortography: this is usually performed if surgery is contemplated and is the definitive investigation to assess the site of entry tear, extent of the dissection, severity of aortic valve regurgitation and to clarify aortic branch involvement (e.g. coronary artery involvement).

Treatment

All patients with an aortic dissection should be monitored with direct arterial pressure measurements. The blood pressure should be controlled in the acute stage with beta-adrenergic receptor blockers (e.g. esmolol 500 μg/kg i.v. bolus over 1 minute followed by 25 - 200 μg/kg/min or metoprolol 5 - 15 mg i.v. over 5 - 15 minutes) and vasodilators (e.g. sodium nitroprusside 10 - 300 μg/min), rather than vasodilator agents alone, to reduce both the mean arterial pressure (MAP) and rate of rise of aortic pressure (to reduce shear stress). A pulse rate of approximately 60 beats per minute and a mean arterial blood pressure of 80 - 90 mmHg (100/70 - 110/80) with adequate renal function is optimal, although a reduction in the MAP by greater than 30%, may compromise renal, coronary and cerebral circulation and should be undertaken with care.

For surgical purposes, the dissection is often classified as either type A or type B. Type A includes all dissections involving the ascending aorta, regardless of site of entry (i.e. proximal dissections); all other dissections are classified as type B (i.e. distal dissections).[2] Type A dissections may be localised to the ascending aorta only (i.e. De Bakey II, 4% incidence) or extend to the descending aorta (i.e. De Bakey I, 64% incidence). Type B dissections usually begin beyond the origin of the subclavian artery and are confined to the descending aorta (i.e. De Bakey III, 30% incidence).[5,18] The types of aortic dissection are shown in figure 1.

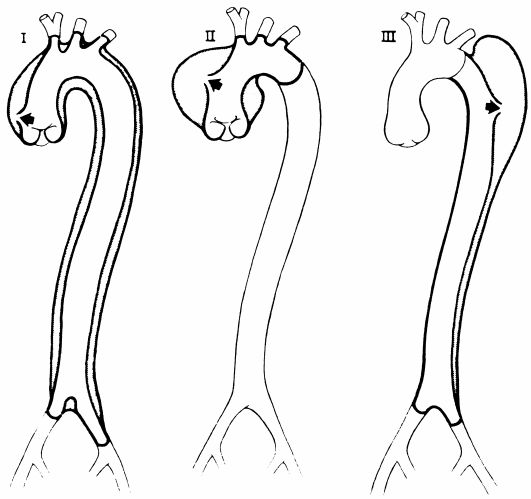

Figure 1. The three types of aortic dissection. Left: De Bakey Type I (Stanford Type A)-proximal tear, dissection involves the ascending and descending aorta. Centre: De Bakey Type II (Stanford Type A)-proximal tear, dissection confined to the ascending aorta. Right: De Bakey Type III (Stanford Type B)-tear beyond subclavian artery, dissection confined to the descending aorta. (Modified from Treasure T and Raphael MJ. Investigation of suspected dissection of the thoracic aorta. Lancet 1991;338:490-495.)

Type A dissections, after medical management has stabilised the blood pressure, have a better prognosis if treated surgically (mortality is reduced from 58% to 26%).[2,5] Surgery is also indicated if haemo-pericardium, tamponade, acute aortic regurgitation or coronary artery occlusion are present.[19]

Type B dissections have a better prognosis if treated medically, compared with surgical treatment. However, favourable results have been achieved using endovascular stent-grafts to seal the entry tear and promote thrombosis of the false lumen in patients in

whom the dissection originates in the descending aorta.[20,21]

Irrespective of whether medical or surgical treatment is given, there is usually a 30-50 % mortality in the first 2 days. The 10-year survival of patients who leave hospital is approximately 60% and is related to the success of surgical treatment of type A dissections, effective medical control of hypertension and close follow-up with early operative intervention for late complications.[2]

Traumatic aortic rupture

The aorta is the commonest major vessel to be injured following blunt chest trauma. Approximately 60% rupture just distal to the origin of the left subclavian artery at the ligamentum arteriosum (i.e. at a junction between the relatively fixed and mobile portions of the aorta, making it prone to the shearing stress caused by acceleration and deceleration forces of trauma. Figure 2).

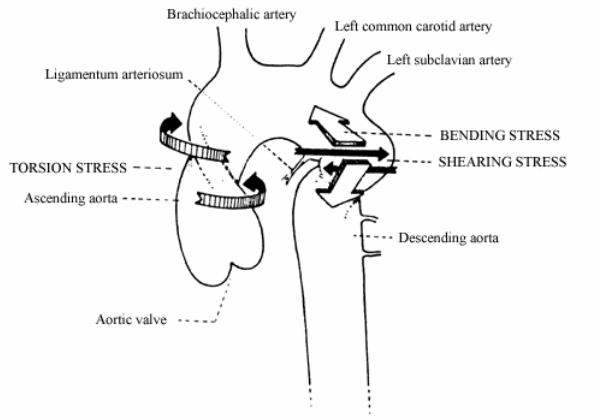

Figure 2. Diagrammatic representation of forces acting upon the thoracic aorta from blunt antero-posterior chest trauma (Modified from Symbas PN. Traumatic injuries of the heart and great vessels. Charles C Thomas, Springfield Ill. 1971 p 153).

Rupture of the ascending aorta accounts for 25% of cases and usually occurs just proximal to the origin of the brachiocephalic artery (i.e at a relatively mobile portion of the vessel making it a focal point of torsion injury. Figure 2). Survival without surgery may present as a false aneurysm as a delayed complication.

Investigations

These include,

Chest X-ray: this may show a widening of the superior mediastinum, blurring of the left margin of the aortic knuckle, tracheal shift and depression of the left main-stem bronchus (> 40 degrees below the horizontal), nasogastric tube deviation to the right of the T4 spinous process (due to deviation of the oesophagus), right paratracheal widening (i.e. enlargement of the paratracheal stripe) and a left pleural effusion.[22] However, a mediastinal haematoma may produce a similar chest X-ray picture and a ruptured aorta may even present with a normal chest X-ray.[23]

Aortic angiography: as a traumatic rupture of the aorta can only be definitively diagnosed by aortic angiography, a high index of suspicion in the presence of a rapid deceleration injury and the acceptance of a high percentage of negative aortographic investigations (e.g. 50% or greater) is often the only way to obtain a high diagnostic yield for this injury.

Transoesophageal echocardiography: if this is performed and interpreted by an experienced operator, transoesophageal echocardiograph can be a very useful investigation. One study reported a 100% sensitivity and 98% specificity (i.e. one false positive test) in detecting injury to the thoracic aorta.[24] However, as transoesophageal echocardiography does not permit complete visualisation of the ascending aorta or the arch, can be interfered with by mediastinal emphysema, and is operator dependent, aortic angiography still remains the investigation of choice.[25]

Computed tomography (CT) and Magnetic resonance (MR) imaging: Spiral volumetric CT and MR angiography are techniques that have also been used to detect traumatic rupture of the aorta with a reported high degree of accuracy,[26] although sensitivities and specificities of these investigations have not yet been determined. MR imaging may also be particularly useful in confirming or excluding diaphragmatic injury, if spiral CT scan with reformations are equivocal, to help assess the aorta and mediastinum in cases of potential injury that are not clarified by CT scan or thoracic angiography and to help identify the true origin of trauma-related masses.[27]

Thoracoscopy: while thoracoscopy has been used in the management of a patient with chest trauma it is largely beneficial in evaluating a haemothorax or the integrity of the left hemidiaphragm, rather than aortic rupture.[28]

Treatment

Treatment of a ruptured aorta consists of an open surgical repair (although repair using an endovascular aortic stent has also been reported[29]), paying close attention to the spinal cord arterial supply during the procedure, as up to 5% of cases develop ischaemic cord lesions and paraplegia in the perioperative period,[30] increasing up to 10% if the aortic cross clamp times exceed 30 minutes.[31] Atrio-femoral bypass,[32] hypothermia (either total body or selective epidural spinal cooling), cerebrospinal fluid drainage and intravenous magnesium (acting as an N-methyl-D-aspartate antagonist[33]), have all been reported to offer some

protection to the spinal cord during the repair of the aorta.[31] However, while two studies concluded that partial bypass (e.g. atrio-femoral bypass) reduced the incidence of paraplegia in thoracoabdominal aortic aneurysm surgery,[32,34] one prospective randomised trial found that cerebrospinal fluid drainage did not.[35]

Aortic aneurysm

An aneurysm is an abnormal dilatation of a segment of a blood vessel. A true aneurysm is one that involves all three vessel wall layers (i.e. intima, media, adventitia) and a pseudoaneurysm is one that due to a breach in the intima and media involves only the outer adventitia (occasionally in association with a perivascular clot).

Causes

Atherosclerosis is the commonest pathological condition associated with an aortic aneurysm, although it may also be caused by cystic degeneration of the collagen and elastic fibres of the aortic media (i.e. cystic medial necrosis which may be associated with Marfan's syndrome, Ehlers Danlos syndrome or pregnancy), infections (e.g. syphilis, tuberculosis), arteritides (e.g. Takayasu arteritis, giant cell arteritis), arthritides (e.g. seronegative spondyloarthropathies, rheumatoid arthritis), and trauma. Atherosclerosis is often associated with abdominal or descending thoracic aortic aneurysms, whereas cystic medial necrosis is usually associated with ascending thoracic aortic aneurysms.

Approximately 5% of abdominal aortic aneurysms are inflammatory which have a higher incidence of renal failure, retroperitoneal fibrosis and ureteric strictures.[36-38]

Clinical features

An abdominal aneurysm is often detected as an incidental finding. For example, as a pulsating epigastric or central abdominal mass during a routine physical examination or on abdominal X-ray or ultrasound performed for another reason. It can also present with abdominal pain and tenderness (particularly when there is an aneurysmal leak with perivascular extravasation of blood), back pain, peripheral embolisation (e.g. ischaemic leg) or in the case of an acute rupture a pulseless (and pale) cardiac arrest.

Diagnosis

An abdominal aortic aneurysm is present when the infrarenal aortic diameter exceeds 3.0 cm.[39] While aortic angiography is often used as the investigation of choice, as it allows the extent (e.g. upper and lower borders) of the aneurysm to be delineated, abdominal ultrasound is also useful in diagnosing and following the rate of enlargement of an abdominal aneurysm. However, currently a spiral CT with contrast or MR imaging are often used to diagnose this disorder, as an angiogram may be associated with complications and may also underestimate the size in the presence of a mural thrombus.[40] A spiral CT to detail the intramural and extramural haematoma, may also be useful in a haemodynamically stable emergency.[41]

Treatment

Surgical repair is indicated for all abdominal aneurysms that are symptomatic or expanding rapidly. Acute abdominal pain with hypotension (i.e. rupture) is an emergency and requires immediate surgical repair. Intravenous fluid therapy is usually administered carefully or delayed (similar to penetrating torso injuries[42]) until surgical access has allowed bleeding to be controlled.

The predictors of rupture of an aortic abdominal aneurysm are the initial size (e.g. > 4 cm in diameter[40]) and the presence of diastolic hypertension.[43] Graft replacement in patients without rupture is usually associated with an operative mortality of 2% or less and survival of 63% at 5 years and 18% at 15 years[44] (or a 9% mortality each year following the operation[45]). The early mortality rate (i.e. during the first 30 days) after an operation in patients with an acute ruptured aneurysm ranges from 25 to 50% and depends on the haemodynamic abnormality at the time of the operation.[44]

Most favour observation of asymptomatic aortic aneurysms up to 4 cm in diameter (as the risk of rupture in an aneurysm < 4.0 cm is small). In centers with an operative mortality of 2% or less, most favour an operation if the aneurysm is greater than 5 cm in diameter,[45] as the risk of rupture for a 5 - 6 cm abdominal aneurysm is 5% each year, increasing exponentially for larger aneurysms.[39] While patients who have an abdominal aneurysm between 4 - 5 cm in diameter (or twice the normal diameter of the infrarenal aorta) and who are in good health, are often advised to have a prophylactic graft replacement,[40,45] some recommend serial noninvasive follow-up as an alternative, particularly in centers with an operative mortality rate of greater than 2%.[46] In centers with elective mortality rates of greater than 10% for abdominal aortic aneurysm repair, the benefit to the patient of any surgical intervention for an asymptomatic abdominal aortic aneurysm of less than 6.0 cm in diameter is questionable.[47]

If the patient is in poor health, then the operation may be deferred until hypertension is controlled (which may cause a reduction in size of small aneurysms[48]), and progressive growth of the aneurysm or symptoms of

abdominal or back pain have been confirmed within the 3-monthly check-up period.[40] Endovascular repair of the aortic aneurysm (using a percutaneously inserted aortic stent) may also be considered in this group of patients[49,50] and even in selected cases of ruptured aortic aneurysms.[51,52]

The indications for surgical management of a thoracic aortic aneurysm are different compared with abdominal aortic aneurysms and depend on whether it is an ascending or descending thoracic aortic aneurysm. For example, 5.5 cm is an acceptable size for elective resection of an ascending aortic aneurysm, as it can be performed with relatively low mortality. For aneurysms of the descending aorta, in which perioperative complications are usually greater compared with surgical repair of the ascending aortic aneurysm, surgical intervention is usually only recommended for aneurysms of 6.5 cm or greater.[53] Endovascular repair of a thoracic aneurysm (particularly descending thoracic aorta) has also been used to reduce perioperative mortality.[49,50]

A first generation cephalosporin (1 gm cephalexin), 1 hr before the incision and thereafter 6-hourly for the first 24 hr post-operatively, will reduce the incidence of wound and graft infection.[54] Postoperative care is usually in an intensive care for the first 24 -48 hr, to monitor haemodynamic and respiratory status. This may also require a right heart catheter, and mechanical ventilation.

Postoperative complications include renal failure, cholesterol embolism of lower limbs ('trash foot'),[55] acute respiratory distress syndrome, disseminated intra-vascular coagulation, myocardial infarction, jaundice, ischaemic or infarcted bowel[56] and paraplegia (even with an infrarenal abdominal aortic aneurysm repair[57]). Hyperkalaemia may also occur in the immediate postoperative period if the patient has been transfused with scavenged blood. Late complications include graft infection, aorto-caval (and other arterio-venous) fistula and aorto-enteric fistula. Long-term beta-blocker therapy should be administered to all patients following surgical repair.

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