

Aortic dissection

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Aortic dissection is caused by a tear in the aortic intima allowing haemorrhage into the media, separating the intima from the adventitia and creating a false lumen. Dissection usually proceeds distally but may extend proximally. Blood may re-enter the true lumen at any point along the dissection creating an exit flap as well as an entry flap. The dissection may compromise the origin of arterial branches arising from the aorta creating ischaemic complications or rupture of the weakened arterial wall. This is the most common cause of death in patients with dissection.

The majority of dissections originate either at one end of two points, the ascending aorta within a few centimetres of the aortic valve or in the descending aorta just distal to the origin of the left subclavian artery. Aortic dissection may occur in a non-aneurysmal aorta, however the degenerative changes which lead to aneurysm formation predispose to aortic dissection. These degenerative changes may be associated with Marfan syndrome, or other connective

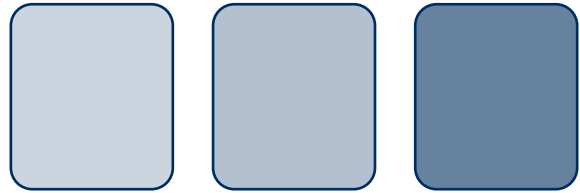
tissue disorders, e.g. Erlos-Danlos. Such disorders predispose particularly to proximal dissection.

Predisposing factors

Hypertension is the most important predisposing factor for aortic dissection. It co-exists in 70-90% of patients in most series, and accelerates the mild degree of medical degeneration which occurs with ageing. Other specific disorders predisposing to dissection include coarctation, bicuspid aortic valve, Turner syndrome, Noonan syndrome, giant cell aortitis, SLE and polychondritis. Although dissections predominate in males, with a male to female ratio of 3:1, there is an association with pregnancy – half the dissections in women under 40 years occur during pregnancy.

Trauma, particularly rapid deceleration injuries, may cause aortic dissection, as may cannulation or catheterisation of the aorta during diagnostic procedures or surgery. Dissections have been reported following prosthetic aortic valve repair or at the site of saphenous vein graft anastomosis after coronary artery bypass grafting. Penetrating atherosclerotic ulcers of intramural haematomas which are

Figure 1. DeBakey and Stanford classifications



distinct from dissection may also predispose to dissection and rupture.

Classification

The classification of aortic dissection has changed as management of the condition has evolved. In the 1950s, De Bakey devised a classification system involving nine different subgroups (or types) based on the extent of aortic involvement. He revised this in 1965 to a scheme which classified the dissection according to site of origin of the intimal tear (see figure 1).

Type I and II both originate in the ascending aorta. Type II is confined to the ascending aorta type but type I may extend to the arch and descending aorta. Type III dissections originate in the descending thoracic aorta, type III A extending as far as the diaphragm and type III B beyond. More recently, Stanford classification has been used. This divides dissections into type A, which involves the ascending aorta irrespective of the site of origin, and type B with other dissections.

Temporarily, dissections are divided into acute (defined as onset of symptoms less than two weeks before presentation) or chronic (presentation more than two weeks after onset of symptoms). This distinction is important as 65-75% of untreated patients with dissection are done within the first two weeks of onset. Among those who survive, the outer layer may expand to develop an aneurysm, which may ultimately rupture. Chronic dissecting aneurysms have a much higher rate of rupture than non-dissecting aneurysms.

Clinical findings

Dissection is usually accompanied by severe chest pain (90% of patients). Typically this pain is appropriately described as ripping or tearing and may radiate to the back. As the dissection progresses, the location of the pain may migrate accordingly.

Patients may present with neurological manifestations of the dissection, a CVA or paraparesis due to impairment of arterial supply to the brain or spinal cord. Rarely acute severe aortic regurgitation may precipitate a congestive cardiac failure presentation, although aortic insufficiency of some degree is found in approximately two-thirds of cases.

Physical findings

Hypertension occurs in up to two-thirds of cases. More specifically, a pressure difference may exist between arms or between upper and lower extremities depending on the site dissection. Absent pulses are an important finding as up to half of patients with proximal dissection have loss of one or more pulse.

New aortic regurgitation must be carefully examined

for, as the diastolic murmur may not be immediately apparent. Aortic valve incompetence may be due to displacement of the valve leaflets by the pressure of the false lumen, in which case resuspension of the valve with decompression of the false lumen may correct the incompetence. It may also be due to disruption of the annulus in which case aortic valve replacement is necessary.

Signs of rupture into the pericardium, a pericardial rub or Beck's triad associated with pericardial tamponade are all associated with a poor outcome. Although severe chest pain with normal ECG and cardiac enzymes is the typical presentation of dissection, the ECG may be abnormal and cardiac enzymes may be elevated in the 1-2% of cases who have myocardial infarction due to compromise of one of the coronary ostia.

Mesenteric, renal or peripheral ischaemia may occur with compromise of the relevant branch of the dissecting aorta. Other far less common findings include acute aortic stenosis, Horner syndrome, vocal cord paralysis, right atrial obstruction, superior vena cava syndrome, haemoptysis, haematemesis, rupture into the right atrium or ventricle, heart block and unexplained fever.

Diagnosis

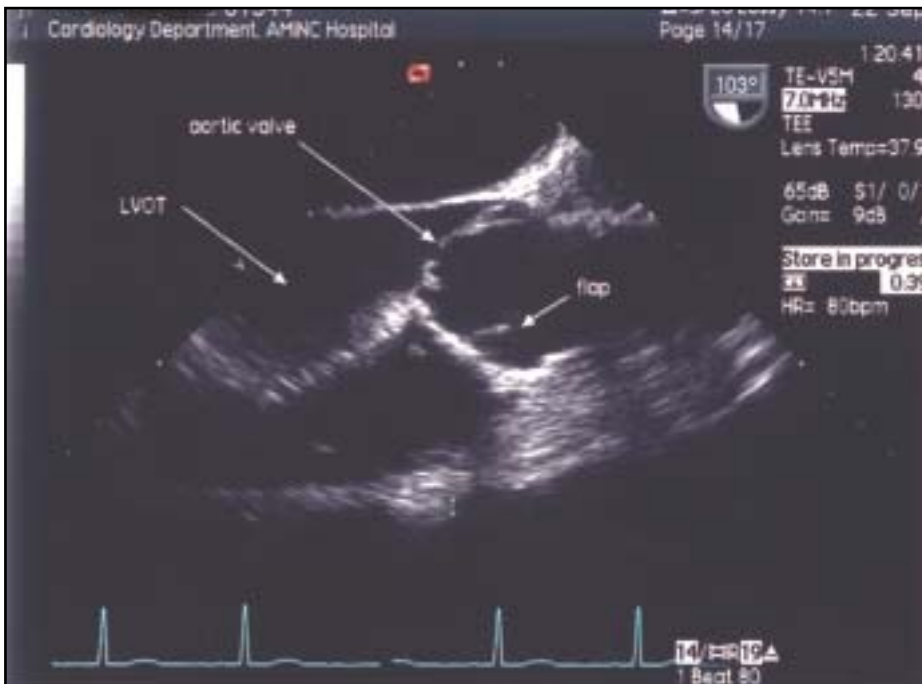
A high index of suspicion is of paramount importance in establishing the correct diagnosis in aortic dissection. The humble chest x-ray may provide valuable information, indeed 90% of patients with dissection will have an abnormal aortic silhouette on x-ray, usually moderate widening of the aorta in the mediastinum.



Figure 2. TOE showing dissection extending from aortic root



Figure 3. TOE showing false lumen with thrombus in descending thoracic aorta



Although aortic angiography was for many years the diagnostic procedure of choice in the evaluation of dissection, the rapid pace of development in non-invasive technology has yielded highly sensitive and specific tests which do not have the attendant risks involved in aortography, e.g. contrast CT, MRI and transoesophageal echo.

MRI and transoesophageal echo have emerged as the most sensitive and specific non-invasive/semi-invasive investigative procedures. However CT has the advantage of being more readily available in most centres. MRI yields excellent definition of the dissection including the site of

the initial flap surrounding structures and presence of thrombus. Transoesophageal echo has comparable sensitivity, although it is less specific and has the advantages of speed, ability to be performed at the bedside (particularly important in a critical patient) and prompt identification of aortic regurgitation.

Management

The modern surgical treatment of aortic dissection was pioneered, as that of thoracic aneurysm repair in the 1950s and has advanced with improvements in cardiopulmonary bypass and hypothermic circulatory arrest.

The objective of surgical repair is to excise the intimal tear where possible, obliterate the false channel proximally and distally, and reconstitute the aorta, usually with a synthetic graft. Aortic valve replacement or repair may also be necessary.

Medical treatment of dissection was introduced in the 1960s, the primary focus of which is to reduce arterial blood pressure, and the velocity of ventricular contraction to reduce shear stress on the aortic wall.

Once the diagnosis of aorta dissection is considered, medical treatment must be instituted urgently even before definitive diagnostic procedures have been performed. Initial treatment is with intravenous betablockade and, if necessary, the addition of sodium nitroprusside or other antihypertensive agents. Vasodilating agents which produce a hyperdynamic circulation

should be avoided. There is general agreement that acute proximal dissections should be treated surgically, with the exception of cases where there is extensive irreversible neurological damage. The operative mortality is now in the range of 15-20%, although rates as low as 7% have been reported.

However, the treatment of acute distal dissection is more controversial. Excellent results have been obtained with medical treatment of stable acute distal dissections, comparable or even superior to results obtained with surgery. Survival probabilities at 1, 5 and 10 years of 0.94,

0.87, 0.30 (medical) and 0.90, 0.80 and 0.50 (surgical) have been reported.

Patients with distal dissection tend to have generalised atherosclerosis, are more frequently elderly, hypertensive have chronic pulmonary disease and pose a formidable operative risk. In addition, the devastating complication of paraplegia/paraparesis is feared. Thus surgery in cases of distal dissection is reserved for complicated dissections or in patients with Marfan syndrome.

Complicated dissections

Leaking or rupture of aorta
Arterial compromise, jeopardising a limb or organ
Continued or recurrent pain
Uncontrollable hypertension

Betablockade is the mainstay of medical treatment of chronic dissections, with the addition of other antihypertensive agents as necessary. Survivors of surgery should also receive long-term medical treatment, particularly patients with Marfan syndrome.

The long-term survival of patients with treated aortic dissection has improved from 5-10% one year survival in the pre-treatment area to 40% actuarial survival at 10 years (60% for those who leave hospital). Re-operation may be necessary for late complications including aortic regurgitation, localised aneurysm formation and redissection. Up to one-third of all late deaths are due to rupture of the aorta, so close postoperative surveillance is advised. However, a large proportion of patients will succumb to

co-morbid conditions, e.g. ischaemic heart disease.

Recently endovascular procedures to treat type B aortic dissection have been reported with very promising preliminary results. However, numbers to date have been relatively small and consensus as to the role of endovascular reconstruction has not yet been reached. Other interesting topics of research include the genetics of non-syndrome-associated familial aortic dissection and risk of dissection associated with specific mutations in Marfan syndrome.

The diagnosis and treatment of aortic dissection and thoracic aneurysm have advanced considerably in the latter half of this century. Combined medical and surgical input is necessary to obtain optimal outcomes. It is hoped that further advances in diagnostic and preventative measures over the next half a century will match improvements in surgical techniques in the last to limit the potentially devastating consequences of these conditions.

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