Seminar

Aortic dissection

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Since Morgagni's first comprehensive description in 1761, aortic dissection has challenged us as to find an appropriate management. Because uncertainties still surround this potentially lethal disease and because its management is shared by many specialties, no fewer than 200 contributions on the subject are published yearly in the medical literature.

Epidemiology

The incidence of aortic dissection is unknown. Published figures may not be representative of the population because as many as one-third of aortic dissections are never diagnosed.[1] Necropsy series from industrialised countries have reported incidences between 5 and 10 per million people per year,[1,2] and a population-based study of 45 000 white people living in Minnesota, USA, identified 37 new cases of aortic dissection over a 30-year period, giving a calculated incidence of 27 cases per million habitants per year.[3] The incidence of aortic dissection is related to the prevalence of risk factors, the most important being untreated hypertension, advanced age, and disease of the aortic wall.[2]

Pathogenesis and predisposing factors

Two factors—an initiating event and a structural weakness of the arterial wall—contribute to dissection of the aorta (table 1). Dilatation of the aorta or high blood pressures tears the intima;[4] the tear of the intimal allows a surge of blood into the aortic wall. The blood, driven by the pulsatile pressure of the circulation, separates the layers of the arterial wall. Local factors determine the site of dissection. The convexity of the ascending aorta (1 or 2 cm above the aortic sinuses) is involved in 60% of cases, the aortic arch in 10%, and the origin of the descending aorta (just below the left subclavian artery) in 30%.[1,5] The proximal ascending aorta is vulnerable to dissection because, during systole, it expands the most and its convexity is repeatedly exposed to maximum arterial pressure. Expansions of the aorta concentrate mechanical stress on points of relative fixation, such as the origin of the descending aorta, where the relatively mobile aortic arch is anchored to the thoracic wall.

That only a minority of patients with dilatation of the aorta or high blood pressure get a dissection suggests that a structural weakness of the arterial wall is also necessary. Various diseases with abnormal connective tissue are associated with high rates of aortic dissection (table 1), among them Marfan's syndrome which is linked to a chromosomic mutation altering synthesis of a fibrillin polypeptide chain.[6] Many genotypes and phenotypes of the disease are being identified and might include a spectrum of disorders ranging from neonatal full-blown Marfan's syndrome to possible "formes frustres", like the marphanoid phenotype or annuloaortic ectasia.[7]

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Most common factors increasing stress on the arterial wall
Arterial hypertension
Aortic dilatation and wall thinning
Unicommissural or bicuspid aortic valve
Aortic coarctation
Aortic arch hypoplasia
Iatrogenic (cardiac surgery and percutaneous catheters)
Most common factors reducing resistance of the arterial wall
Aging
Marfan syndrome
Pregnancy

Table 1: **Most common factors predisposing to aortic dissection**

In a minority of patients (probably less than 10%), dissection of the aorta is initiated by spontaneous rupture of the vasa vasorum. The haematoma spreads within the medial layer of the aorta and disrupts the intima. This pathogenic mechanism, which has long been suspected by pathologists,[8] has been shown to happen by serial echocardiography in the initial stages of the process.[9]

Classification

Various classifications have been proposed based on anatomical characteristics (extension of dissection, site of intimal rupture, fate of the false lumen) and on the duration of dissection at the time of first presentation. DeBakey's classification recognises a Type I and II which originate in the ascending aorta (in Type I, the dissection extends distally to the aortic arch; in Type II, it is confined to the ascending aorta). And a type III which originates in the descending aorta.[10] The widely used Standford classification (employed in this review) designates as Type A all dissections involving the ascending aorta (regardless of the entry site location), and type B those distal to the left subclavian artery (sparing the ascending aorta) (figure 1).[11] A few cases, however, do not fit in this simplified classification and have been called nonA/nonB dissections by some authors (figure 2).[12] "Acute" applies when the diagnosis is made within 14 days after the first symptoms, and "chronic" when made later.

Clinical presentation and diagnosis

Abrupt and severe retrosternal or interscapsular chest pain, sometimes felt as a tear, with subsequent migration down the back occurs in 90% of patients and is due to the dissecting process itself. Complications of the dissection produce additional signs and symptoms. Acute aortic insufficiency and tense pericardial effusion (from transudation of blood through the thin wall of the false lumen) are common when the ascending aorta is involved and produce various degree of cardiac failure. Secondary rupture of the adventitia is usually rapidly fatal from acute cardiac tamponade, or massive bleeding into the mediastinum of left pleural cavity.[5,13,14] Dissection may compress or occlude a branch of the aorta and produce acute ischemia. Ischemia occurs in arms or legs in 20% of patients, in the kidney in 15%, myocardium in 10%, brain in 5%, and mesentery or spinal cord in 3%.[15,16]

Echocardiography plays a central role in the diagnosis of patients with a suspected aortic dissection (table 2). Transthoracic echocardiography is diagnostic in 75% of

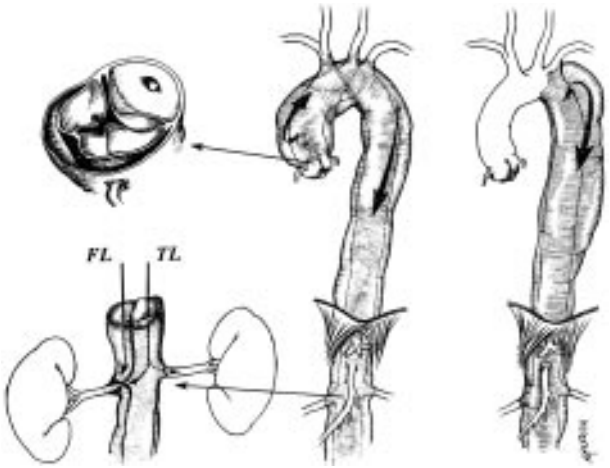

Figure 1: **Standford classification of aortic dissection**
Typical type A (centre) and type B (right) dissection. Complications of the dissected aorta (left) may include aortic insufficiency from prolapsed commissure, (top) or organ ischaemia from aortic branch compression (bottom). FL=false lumen, TL=true lumen.

Type A and 40% of Type B dissection, and shows aortic regurgitation, pericardial effusion, and areas of cardiac dyskinesia. This rapid evaluation can usually be followed by a single additional diagnostic step before treatment is started. Unstable patients are best evaluated by transoesophageal echocardiography in the emergency room or in the operating theatre. Stable patients can be evaluated by computed tomography or magnetic resonance imaging. Angiography is recommended for stable patients in whom diminished perfusion of an aortic branch is suspected or when other diagnostic methods are not available.

High-resolution imaging techniques now allow precise delineation of intraparietal lesions in the aorta like penetrating ulcers or intramural haematoma. Intramural haematoma may be detected within a few hours after onset of pain in as many as 12% of patients with an acute aortic syndrome and is probably a consequence of rupture of vasa vasorum. Left untreated, it progresses to overt dissection or adventitial rupture.[9] Haematomas are ominous precursors of aortic dissection and require urgent treatment. They are not detected by angiography but by echocardiography, computed tomography, magnetic resonance imaging, or a combination of these tests. This stresses the importance of pursuing investigations in any patient with suggestive symptoms until such pathologies are definitely ruled in or out.[9]

Treatment

Immediate appropriate treatment on any suspected or confirmed aortic dissection has improved outcome to a current overall in-hospital mortality below 30%.[13,17] In old series, the death rate of untreated aortic dissection was 1 to 2% per hour, with less than 10% of patients surviving 72 hours.[18] The risk of fatal rupture of the ascending aorta or aortic arch is extremely high (around 90%) in Type A dissection, and requires immediate replacement of the aorta. This risk is lower in Type B dissection, unless a periaortic or left pleural haematoma is present, and can be controlled by reducing aortic stress medically. Aortic stress is reduced by lowering blood pressure to just maintain adequate renal, cerebral, and myocardial perfusion, and by reducing the pulse upstroke and heart rate. β-adrenergic blockers are especially suitable for this purpose.

Surgery in type A dissection consists of replacing the ascending aorta (and aortic arch in young patients or when the entry intimal tear is there), reconstructing the aortic root to restore aortic valve competence, and directing blood flow preferentially into the true lumen distally. A diseased or non-reconstructible aortic root or aortic valve needs to be replaced with the ascending aorta as well. Nowadays, the operative mortality in Type A dissection is between 10 and 25%.[5,13] Resecting the entry site of dissection and connecting the prosthetic tube to the aortic wall after split layers have been glued together reduces perfusion of the false lumen (complete thromboses of the false lumen occurs in 5 to 25% of patients after surgery). This may increase perfusion into aortic branches that were compressed by a tense false lumen and may slow subsequent dilatation of the false channel. Although the benefit of surgery is undisputed for essentially all cases of Type A dissections, it is less well established for Type B dissections.[5,13] Rupture of the aorta is less likely and 70% of cases progress to a chronic form with medical treatment. Resection of the descending aorta carries a significant mortality and morbidity. Paraplegia and renal failure are devastating complications that may develop from acute ischemia during aortic crossclamping.[14] Experience, refinement in surgical techniques such as support for the distal circulation by extracorporeal circulation, and improvement of biomaterials (use of soft prostheses and extracorporeal circuits requiring low doses of anticoagulants) have reduced mortality in uncomplicated Type B dissection to under 10%.[19,20] These achievements, however, are no better than those obtained with non-operative management. Medical therapy alone is hence indicated in Type B dissections with a favourable outlook (the majority of cases) and in patients with severe comorbidity,[13,19] and surgery in Type B dissections with a less favourable outlook. This includes cases of increased diameter of the aorta (many surgeons have empirically set the limit to 5 cm), progression of the dissection, malperfusion of a distal aortic branch, uncontrollable hypertension, and persistent pain.[12,14,19]

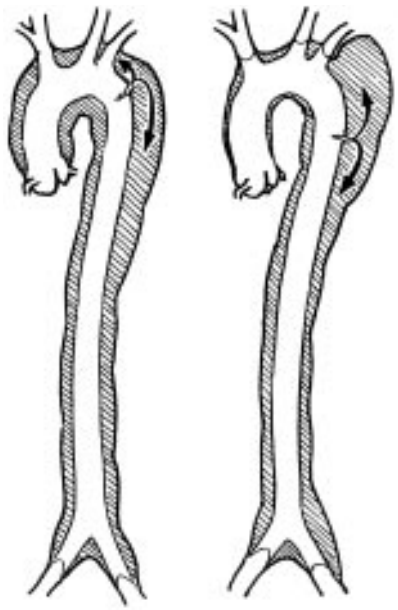

Figure 2: **Other forms of aortic dissection**
Significant involvement of the ascending aorta (left) requires replacement of the ascending aorta and aortic arch. In the absence of dilatation of the aorta, pericardial effusion, and aortic insufficiency (right), medical treatment may be more appropriate.

Obstruction of a side branch of the aorta occurs when the false lumen is blind (not decompressed by a distal intimal tear) and compresses the true lumen (figure 1). Restoration of perfusion can be obtained by a proximal intervention in which the entry site of the dissection is resected and blood flow directed through the true lumen.[15,20] Because of possible re-entries along the intimal septum, decompression of the true lumen may not be obtained for remote abdominal branches. Surgical

	Sensitivity	Specificity	Major assets	Major limitations
Angiography	80–90%	90–95%	Shows entry site Shows aortic branches Shows dependency of aortic branches	Time and personnel Nephrotoxic agents Misses intramural hematoma
Conventional CT	65–85%		Easiness and rapidity	Misses entry site
Ultrafast CT scan	80–100%	95–100%		Partial analysis of aortic branches
MRI	95–100%	95–100%	Precise flow analysis Shows aortic branches No contrast material	Transfer of patient Acquisition time and surveillance High cost
Echocardiography (transthoracic and transoesophageal)	95–100%	85–90%	Rapidity Bedside examination Assesses aortic regurgitation Assesses pericardial effusion Assesses myocardial function	Misses the distal ascending aorta Misses branch involvement Misses entry site Requires expertise Uncomfortable on the awake patient

MRI=magnetic resonance imaging.

Table 2: **Major characteristics of common diagnostic tests**

resection of the intimal septum (surgical fenestration) in the abdominal aorta to create a wide communication between lumens restores adequate perfusion. In ill patients, extra-anatomic bypass, which avoids surgery on the aorta and crossclamping, is another option to perfuse an ischaemic organ or limb.[16,21] Percutaneous insertion of endovascular stents to expand the compressed true lumen of an aortic branch and percutaneous fenestration of the intimal septum to equalise perfusion pressure in the lumens has been explored by a few teams mastering these techniques and has shown satisfactory preliminary results.[22]

Long-term management

The overall survival rate of patients with aortic dissection leaving hospital is currently around 80 and 40% at 5 and 10 years, respectively, and is not influenced by the type of dissection.[5,13,19,23] As expected, the diseased aorta is responsible for many late deaths (20–40%), but associated cardiovascular diseases are also other important causes of death (30–50%).[13,19] Rupture of the aorta complicates continuous dilatation of the false lumen or retrograde extension of Type B dissection, and accounts for 20% of late deaths. Complete thrombosis and, to a lesser extent, reduced flow in the false lumen decreases the risk of subsequent aortic events and improves survival.[24,25] In a series of patients with a repaired type A dissection, the rates of survival and freedom from aortic events were 95 and 84% at 5 years, respectively, when the false lumen was thrombosed, and 76 and 63% when it was patent.[24]

Whatever treatment has been given initially, patients with aortic dissection need continued medical treatment and close surveillance. Residual aortic disease deteriorates into threatening conditions requiring surgery in 15 to 30% of patients at 10 years.[5,26] Dilatation of the dissected aorta with the risk of rupture, progressive reduction of organ perfusion with the risk of dysfunction and irreversible ischaemia, and development of severe aortic insufficiency with the risk of left ventricle failure are the most common.[5,21,23,26] Early surgical treatment is the sole approach to these conditions that can improve survival by preventing the occurrence of these complications.[20,26,27] Finally, due to its high prevalence and impact on outcome, any sign of cardiac or cerebrovascular atherosclerosis should also be pursued and treated vigorously.[13,19]

Surveillance of the aorta should start during initial hospitalisation and then between every 3 months to 1 year depending on the size and rate of increase of the diameter of the aorta.[23,26,27] Ultrafast computed tomogram scan and magnetic resonance imaging can map the entire aorta and are most suitable for serial follow-up (table 2). A retained aortic valve, even a competent one, needs repeated echocardiographic evaluation, because insufficiency may progress or appear if dissection recurs in the aortic root. Angiography is indicated mainly to evaluate perfusion of aortic branches.

Patients with Marfan syndrome have a diminished life expectancy. Dissection of the aorta is the most common cause of death, and once it has occurred, survival is severely jeopardised by deterioration of the false lumen. Because dissection usually starts in a dilated ascending aorta, prophylactic replacement of this segment is considered. The procedure (called the Bentall-deBono operation) entails resection of the aortic valve, aortic root and ascending aorta, and carries on operative mortality of less than 5% if performed electively by an expert surgeon.[28] This mortality and the mechanical valve-related complications warrant replacement on a young and symptomless patient only if the risk of dissection appears high. Until possible genotypes of increased risk are identified, two parameters help with the decision: the size (and rate of increase) of the ascending aorta and a family history of aortic dissection. The procedure must be considered when the diameter of the aorta reaches 6 cm at the sinus portion or 5 cm above, especially in high-risk families, patients with marked aortic regurgitation and women planning pregnancy.[29] The other patients will benefit from prophylactic β-adrenergic blockade which has been proved to reduce the rate of aortic enlargement and the incidence of cardiac and aortic events.[30]

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