

AORTIC DISSECTION: CLINICAL AND SURGICAL MANAGEMENT

DISSECÇÃO DA AORTA: MANEJO CLÍNICO E CIRÚRGICO

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ABSTRACT

Aortic dissection is a dramatic condition whose early accurate diagnosis is fundamental for patient survival. Within the context of acute chest pain in the emergency room, its diagnosis can be overlooked, requiring a high level of suspicion to be performed in a timely manner. The availability of imaging methods has contributed to a faster diagnosis. The initial management goal is to control pain and blood pressure, mainly through the use of intravenous beta-blockers. This strategy decreases shear stress on the aortic wall, minimizing the progression of delamination. Identifying the location of the dissected aortic segment is crucial, as this will impact on the treatment and prognosis. Patients with uncomplicated Stanford type B dissection may receive pharmaceutical treatment alone, while acute type A dissection is a surgical emergency. In relation to surgery, the benefit of the "Frozen Elephant Trunk" technique has been discussed, which corrects a greater area of compromised aorta and may benefit patients with distal ischemia, despite adding greater complexity and increasing the risk of neurological complications. For type B dissections, endovascular repair has been widely used, and several experts have also suggested this approach for uncomplicated cases, as recent studies have described the influence of the treatment on a ortic remodeling and consequently, on survival.

Keywords: Aneurysm, Dissecting; Aorta; Aortic Diseases.

RESUMO

A dissecção da aorta é uma condição grave cujo diagnóstico preciso precoce é fundamental para a sobrevida dos pacientes. Dentro do contexto da dor torácica aguda no setor de emergência, seu diagnóstico pode passar despercebido, o que exige um alto índice de suspeição para ser realizado em tempo hábil. A disponibilidade dos métodos de imagem têm contribuído para a prontidão desse diagnóstico. Os objetivos iniciais do tratamento consistem no controle da dor e da pressão arterial através, principalmente, do uso de betabloqueadores endovenosos. Tais medidas diminuem o stress na parede da aorta, minimizando a propagação da delaminação. A identificação da localização do segmento de aorta dissecado é crucial, pois impacta no tratamento e no prognóstico. Pacientes com dissecção tipo B de Stanford e sem complicações podem receber tratamento medicamentoso exclusivo, enquanto que a dissecção aguda tipo A de Stanford é uma emergência cirúrgica. Em relação à cirurgia, têm-se discutido o benefício da técnica do Frozen Elephant Trunk, a qual corrige uma maior extensão de aorta comprometida, podendo beneficiar pacientes com isquemia distal, apesar de apresentar maior complexidade e aumentar o risco de complicações neurológicas. Para as dissecções tipo B, o reparo endovascular tem sido amplamente utilizado e vários especialistas têm sugerido essa abordagem também para os casos não complicados, pois estudos recentes descrevem a influência do tratamento no remodelamento aórtico e, consequentemente, na sobrevida.

Descritores: Aneurisma Dissecante; Aorta; Doenças da Aorta.

INTRODUCTION

Aortic dissection is a dramatic condition for which an early and accurate diagnosis is essential for patient survival.

The classic aortic dissection can be defined as a middle layer delamination caused by blood influx through an entrance orifice in the intima layer that creates a false lumen of varying extent along the vessel. Other entities like intramural

hematoma and penetrating aortic ulcer have clinical characteristics similar to classical dissection and are responsible for acute aortic syndromes. These conditions are called atypical aortic dissections and may also have fatal outcomes if not properly diagnosed and treated.

The International Registry of Acute Aortic Dissection (IRAD) reported that, despite advances in aortic dissection

propaedeutic, disease-related mortality rates remain high at 25–30% in acute events.¹ Dissection-associated death can be caused by rupture of the vessel into the pericardial sac, causing cardiac tamponade, by rupture to other cavities (thorax, retroperitoneum), acute aortic insufficiency when the aortic valve is affected, coronary ostium obstruction, or ischemia of the target organ due to obstruction of the aortic branch. Therefore, a high index of suspicion is essential for treating cardiovascular emergencies.

CLASSIFICATION

An accurate classification of aortic dissection is extremely important to defining the treatment strategy and prognosis. Considering the timing of symptom presentation, dissection is defined as acute when the period of pain onset is \leq 14 days and chronic when the first symptom occurred >14 days prior, with fatal complications occurring more frequently in the first few symptomatic hours or days.

The two major anatomical classifications used are the Stanford and DeBakey classifications.² The Stanford classification defines dissection as type A when it affects the ascending aorta regardless of the entry orifice location and the distal extension of the involved aorta. Dissections that begin distal to the left subclavian artery are defined as type B. The DeBakey classification categorizes dissections according to the extent and involved segment of the aorta. In type I, the intimal rupture occurs in the ascending aorta and the delamination progresses distally to at least the aortic arch. In type II, the dissection originates and is restricted to the ascending aorta. In type III, the dissection begins in the descending aorta and progresses distally. Atypical aortic dissections can also be classified similarly. Figure 1 illustrates the two classifications of aortic dissection.

EPIDEMIOLOGY

The incidence of acute aortic dissection in the general population is an estimated 2.6–3.5 cases/100,000 people/year.³ A review from the IRAD of 464 patients reported a mean age of 63 years at clinical presentation with a significant predominance in men (65%).^{1,4} Regarding the affected aortic

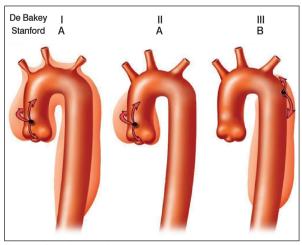


Figure 1. Classification of thoracic aortic dissections.

segment, dissections of the ascending aorta are twice as prevalent as dissections of the descending aorta.⁵

ETHIOPATHOGENESIS AND PHYSIOPATHOLOGY

The etiology of aortic dissection can be degenerative, associated with genetically mediated collagen disorders, or related to traumatic acceleration and deceleration or iatrogenic instrumentation injuries.

Cystic medial necrosis is the common pathological substrate, being considered a prerequisite for intimal rupture or medial hemorrhage. In fact, 50–65% of intimal lesions are found in the ascending aorta near the sinotubular junction.¹

RISK FACTORS

The investigation of high-risk conditions frequently associated with aortic dissection is extremely relevant since it contributes to the rapid identification of patients with a high probability of developing the disease. Systemic arterial hypertension is the main predisposing factor for acute aortic dissection. According to the IRAD review, 72% of patients had a history of hypertension. Situations of abrupt blood pressure increases in cocaine or crack users or during excessive weight lifting have been associated with the occurrence of dissection.

Genetically mediated collagen disorders such as Marfan, Loeys-Dietz, and Ehlers-Danlos syndromes are also important risk factors. A published IRAD review reported that 50% of cases of Marfan syndrome are diagnosed in patients < 40 years of age.⁶

The presence of a bicuspid aortic valve increases the risk of aortic dissection. This predisposition seems to be caused by a genetic disorder of the aortic wall that mainly affects the ascending aorta and/or artic root, even in patients with normal aortic valve function.⁷

The association with coarctation of the aorta and bovine arch may also be related to a greater risk of dissection. Previous aortic aneurysm is also a risk factor, and the risk of delamination is proportional to the largest aortic diameter. This risk becomes more significant when the ascending aortic aneurysm exceeds 5.5 cm.8 Previous cardiac surgery, especially related to the aortic valve or the aorta itself, is also a risk factor for aortic dissection, as whell as a history of cardiovascular catheterization for diagnostic examination (coronary angiography) or therapeutic intervention (coronary angioplasty, transcatheter aortic valve implantation, or endovascular treatment of the aorta). A family history of aortic disease is an important predisposing factor since the genetic non-syndromic etiology is already well recognized. Other risk conditions include coarctation of the aorta, Turner syndrome, and inflammatory diseases with vasculitis, more commonly giant cell arteritis and Takayasu's arteritis.9 Pregnancy and delivery are independent risk factors for aortic dissection, but the presence of other additional conditions (Marfan syndrome, bicuspid aortic valve) increases risk prediction.

CLINICAL STATUS

Symptoms and signs of acute aortic dissection depend on its extent and the affected cardiovascular structures. It generally starts with sudden-onset severe anterior chest pain, stabbing, or tearing, which forces the patient to seek medical attention within minutes or hours. In patients with type B dissection, back pain is more frequently reported, with possible irradiation to the thoracic or abdominal region.

Cases can be asymptomatic with few clinical manifestations. They are generally related to older patients with a history of diabetes or previous diagnosis of aneurysm with initial clinical manifestation of syncope or stroke.¹⁰

Compromised perfusion with target organ ischemia is common and caused by occlusion or extrinsic compression of the aortic branch by the false lumen. The presence of decreased or absent flow in peripheral arteries manifests clinically as a pulse deficit or decrease, as well as considerable variation in the systemic arterial pressure (>20 mmHg) compared to that in the contralateral limb. Although it is a characteristic finding, it is present in only 9–30% of cases.¹¹

Although most patients are hypertensive during an acute event, they can have hypotension with hemodynamic instability as a result of acute aortic insufficiency, cardiac tamponade, hemorrhage, myocardial ischemia, or compression of the true vessel lumen.

Acute aortic valve insufficiency due to proximal extension of the delamination is the main cardiac complication of type A dissection. Part Root dilatation and misalignment or valve leaflet collapse are mechanisms responsible for valvar dysfunction, which manifests as a new murmur at physical examination and may progress to cardiogenic shock. Aortic insufficiency affects about 50–60% of patients with aortic dissection involving the ascending portion.

In addition to aortic insufficiency, cardiac failure may be due to myocardial ischemia or cardiac tamponade. Ischemic insult is explained by the extrinsic compression of the coronary ostia or delamination progression to the ostium (up to 15% of cases). Cardiac tamponade can be caused by fluid transudation from the thin wall of the false lumen or aortic rupture directly into the pericardial sac. In this case, the patient is hemodynamically instable and presents decreased arterial oxygen saturation in the dorsal decubitus, dyspnea, or chest discomfort and muffled sounds at auscultation.¹⁴

Neurological deficits may occur due to poor cerebral or spinal perfusion or extrinsic nerve compression. Syncope occurs in 5–10% of patients and usually indicates supra-aortic branch involvement or cardiac tamponade.¹⁴

DIAGNOSIS

Aortic dissection should be suspected when high-risk clinical signs and symptoms are identified, which will guide the correct choice of complementary imaging tests required to confirm the diagnosis since these tests can show the dissection layer that separates the true and false lumens. Several studies have been published with the objective of identifying the clinical characteristic of a high predictive value for diagnosing aortic dissection. Rogers et al. ¹⁵ published the IRAD results and proposed a high sensitivity (Aortic Dissection Detection Risk Score: ADD-RS) to identify the disease based on the presence of one or more of the following groups:

 High-risk conditions such as Marfan syndrome, family history of aortic disease, previous diagnosis of aortic valve disease or aortic aneurysm, and previous manipulation of the aorta including cardiac surgery;

- History of chest or abdominal pain of abrupt onset or high intensity ("tearing");
- Physical examination showing perfusion deficits such as pulse or pressure discrepancy, focal neurological deficit, or diastolic aortic murmur associated with hypotension.
- The presence of one or more markers within groups provides a score of one (of a maximum of three) when there is at least one marker in each group.

As for the use of serum markers to detect aortic dissection, only D-dimer seems useful in this scenario. However, as it is a non-specific indicator of intravascular coagulation, it may increase in other conditions as well (e.g., myocardial infarction, pulmonary thromboembolism). In any case, it seems a useful tool for identifying patients without dissection. A result <500 ng/mL has high predictive value for excluding this diagnosis. ¹⁶

Since clinical examinations and laboratory tests are insufficient for complete diagnostic elucidation, imaging tests are essential. Findings on chest radiography may be the first clue. Mediastinal enlargement and changes in the aortic contour, the main alterations observed, are present in up to 80% of cases. Thowever, due to its limited sensitivity, especially for type B dissections, additional imaging tests are essential to confirm the diagnosis.

Computed tomography angiography (CTA) should be the test of choice for hemodynamically stable patients. The diagnosis is basically performed by showing a vessel flap separating the true and false lumens, ¹⁸ with the possibility of showing associated complications (Figure 2). The reported sensitivity and specificity of CTA for diagnosing acute aortic dissection are 83–95% and 87–100%, respectively. ¹⁹ In addition to the diagnosis itself, CTA allows a complete anatomical evaluation, including potential involvement of the aortic branches, which are fundamental to the planning of surgical and endovascular treatments. Its main disadvantages are the use of ionizing radiation, contrast nephrotoxicity, and its inability to evaluate aortic valve function. ²⁰

Magnetic resonance angiography is an alternative test to CTA in patients where the diagnosis is suspected later because it is highly accurate for the diagnosis of aortic dissection.²¹ It has the advantage of not exposing the patient to ionizing radiation or requiring the administration of iodinated contrast. The disadvantage is that it



Figure 2. Tomographic angiography of the thoracic aorta showing a dissection flap in the ascending and descending aorta (Stanford type A).

is a time-consuming test not commonly available in the emergency department.

For hemodynamically unstable patients, the most commonly recommended test is transesophageal echocardiography (Figure 3), which provides a rapid diagnosis and can be easily performed at bedside in the emergency room with a sensitivity of 98% and specificity of 63–96%.²² This test can also evaluate aortic valve function and the presence of pericardial effusion. The disadvantage is the need for esophageal intubation, which may require a degree of sedation that may further hinder the patient's hemodynamic state and is an operator-dependent exam.

CLINICAL TREATMENT

The clinical management of acute aortic dissection is based on pain, heart rate, and blood pressure control, since such procedures may decrease the rate of ventricular contraction and shear stress on the aortic wall, minimizing the trend in dissection progression.²³

Morphine should be used to control pain. The reduction of systolic blood pressure should be initiated with intravenous beta-blockers followed by intravenous vaso-dilators as necessary. The systolic blood pressure should be reduced to the lowest tolerated level, usually 100–120 mmHg,²⁴ and the heart rate should be around 60 beats per minute. To achieve such goals, the initial medication of choice is esmolol because of its short half-life. Labetalol can also be used and is easily administered. Diltiazem and verapamil are alternatives for patients who do not tolerate beta-blockers.²⁵

If the systolic blood pressure remains high after beta-blocker administration, nitroprusside can be included. However, it should not be used before heart rate control is achieved since isolated vasodilation may induce the reflex activation of the sympathetic nervous system and result in increased ventricular contraction strength and stress on the aortic wall.²⁶

The rapid identification of the location of the dissected aorta segment is vital and must be determined simultaneously with the diagnosis since it impacts the initial management and prognosis. Patients with type B dissection and no target organ complications can be exclusively treated with drug



Figure 3. Transesophageal echocardiography showing the dissection layer of the ascending aorta.

therapy, whereas acute dissection involving the ascending aorta (Stanford type A) is a surgical emergency.²⁷

SURGICAL TREATMENT

Surgical treatment is the approach of choice for ascending aortic dissection due to its high mortality rate (1–2% per hour in the first 24–48 hours).²⁸ Although the operative mortality rate remains high (7–36% in reference centers),²⁹ the 1-month survival rate is 10% for clinical treatment only and 70% for surgical treatment. Patients with other acute aortic syndromes affecting the ascending aorta, such as intramural hematoma and aortic penetrating ulcer, should also be referred for surgery.

The basic principle of the surgical approach is blood redirection into the true lumen through excision of the aorta segment containing the intimal lesion and correction with a prosthetic vascular graft.³⁰

In most cases of aortic valve insufficiency associated with aortic dissection, the valve is normal and can be preserved and the insufficiency is corrected through the suspension of the commissural pillars associated with ascending aorta replacement.³¹ Patients with unrepairable valve leaflet abnormalities may require aortic valve replacement.

The presence of delamination involving the sinus of Valsalva or aortic root dilation requires reconstruction of the aortic root with aortic valve replacement through the use of a valve tube. In addition, the hemiarch approach is recommended by most specialists since it enables more complete repair and does not seem to be associated with increased operative mortality.³²

Distal extension of aortic repair is controversial because the replacement of the ascending aorta and hemiarch effectively corrects the entry orifice; however, a large extension of the aorta remains distally untreated, which may impair patients with visceral ischemic complications who may have a reentry orifice in the descending aorta. These patients could benefit from the Frozen Elephant Trunk technique based on the replacement of the ascending aorta and aortic arch with a tubular vascular graft integrated with the endoprosthesis positioned in the descending aorta (Figure 4). Although it

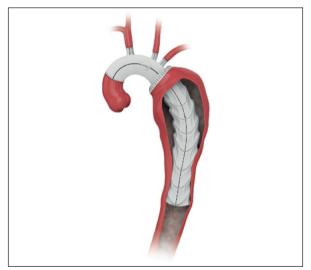


Figure 4. Frozen Elephant Trunk technique with replacement of the ascending aorta and aortic arch associated with endoprosthesis implantation in the descending aorta.

corrects a greater extension of the involved aorta in a single surgical session, this technique adds greater complexity and risk of neurological complications³³ and should be used only in younger patients, when there is dilatation or reentry orifices in the aortic arch or descending aorta and when there is suspicion of distal ischemia.³⁴

Recommending surgery for patients with neurological deficit or who are in a coma is also controversial. Although related to a worse prognosis, recovery has been demonstrated, especially when the time between symptom onset and operating room arrival is <5 hours. ³⁵ It is worth mentioning that even though age > 70 years is a predictor of a worse outcome, advanced age alone should not be considered a surgical contraindication. ³⁶

Dissections confined to the descending aorta (Stanford type B) should initially be treated with drug treatment alone. Immediate interventional treatment with an endoprosthesis should be recommended only in cases of dissection-related complications (refractory pain or hypertension, rapid aortic enlargement, hemorrhage or controlled rupture, distal organ ischemia).^{27,28}

An IRAD series of 384 type B dissection patients managed with clinical treatment alone showed an in-hospital mortality rate of 10%.³⁷

Thus, immediate endovascular or surgical treatment should be indicated for patients who present with the aforementioned complications. Endovascular repair has been widely used as an alternative to conventional open surgery for treating type B dissection because its morbidity and mortality rates are significantly lower than those of conventional surgical treatment. This procedure consists of coating the diseased aorta with an endoprosthesis to occlude the intimal entrance orifice and expand the vessel's true lumen, reducing the blood flow into the false lumen and causing stasis, thrombosis, and aortic remodeling (Figure 5).³⁹ This treatment can only be used when the anatomy is favorable, i.e., large-caliber peripheral vascular access, adequate passage routes for the endoprosthesis, and a region of the aorta with sufficient extension and diameter to support it.

A meta-analysis of 39 studies involving 609 patients who underwent endovascular treatment for type B dissection

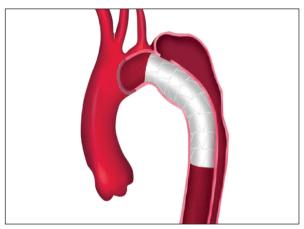


Figure 5. Correction of aortic dissection type B using an endoprosthesis.

reported success in 98% of procedures, in-hospital mortality of 5.2%, and a survival rate of 89% in a 2-year follow-up.⁴⁰ Several specialists have suggested endovascular treatment in cases of uncomplicated acute type B dissection, as it could influence aortic remodeling and consequently contribute to better late evolution of these patients.

The INvestigation of STEnt grafts in patients with Aortic Dissection (INSTEAD) study randomized 140 patients with uncomplicated subacute type B dissection and compared the endovascular and isolated drug strategies used therein. After a 2-year follow-up, no intergroup survival difference was noted (89% vs. 96%).⁴¹ However, in a 5-year follow-up (INSTEAD-XL), endovascular treatment was associated with a lower mortality rate related to aortic complications than clinical treatment alone,³⁸ which suggests that, for a selected group of patients, the use of an endovascular treatment in the acute phase can be indicated even without immediate complications.

CONFLICTS OF INTEREST

The author declares that he has no conflicts of interest in this work.

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