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REVIEW ARTICLE

Acute aortic dissection: An update

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Abstract The aorta, which has a complex intrinsic biology and sophisticated mechanical properties for conducting the blood ejected from the left ventricle to the rest of the systemic arterial bed, is the largest and strongest artery in the body. It carries roughly 200 million liters of blood in an average lifetime. Any process that undermines the architecture threatens the structure, stability, and functionality of the aorta. In this regard, acute aortic dissection (AAD) requires special attention because it is the most catastrophic acute illness of the aorta; it has high morbidity and mortality because of potentially fatal complications. AAD has, therefore, become an important topic of recent research, and knowledge about this disease has improved during the past few years. Up-to-date knowledge about the natural history, epidemiology, presentation, physiopathology, evolution, management, follow-up, and long-term outcomes of AAD are summarized in this review.

Introduction

Acute aortic dissection (AAD) is part of the acute aortic syndrome (AAS) [1,2]; it is an age-dependent, life-threatening cardiovascular disease associated with high morbidity and mortality due to potentially fatal complications [3–13]. On the basis of autopsy data, AAD is believed to be the most common form of immediately life threatening aortic pathology, or aortic catastrophy [14]. If untreated, AAD has a mortality of 33% within the first 24 hours and rises to 50% mortality by the first 48 hours [4,9,12]. The mortality rate approaches 75% in patients with undiagnosed ascending AAD by the second week after onset, and after 3 months without appropriate treatment, the mortality rises to 90% [11].

Epidemiology of the aortic dissection

There are many reports on the incidence of AAD [14–18]. However, the true incidence is unknown because an undetermined number of undiagnosed or misdiagnosed cases result in sudden death and do not undergo an autopsy

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[9,15,19]. Population-based studies have estimated the incidence of AAD to be about three cases per 100,000 people per year [8]. It is known that three out of 1000 patients presenting in the emergency room with chest or back pain, or both, have an aortic dissection. The prevalence of aortic dissection after autopsy is 1—3% [9,14]. The incidence of AAD after major cardiac surgery is 0.03—0.1%, with the highest incidence occurring in patients who have undergone an aortic valve replacement (0.5—1.0%). Iatrogenic AAD has an incidence of 0.12—0.16% [20].

Pathophysiology

Any condition that weakens the composition of the elastic or muscle fibers, or both, of the aortic media predisposes a person to aortic dissection [13]. Local factors determine the site of the tear, which is usually in the right lateral wall of the ascending aorta or the descending thoracic aorta close to the ligamentum arteriosum because this is where the points of greatest hydraulic stress in the aorta are [21]. A loss in the balance between degradation and reconstruction of the aortic wall is due to a decrease in inhibitors and an increase in proteases within the aortic wall, which leads to the destruction of extracellular matrix and the death of vascular smooth muscle cells [22]. This is the histological hallmark of the dissected aorta wall and it is known as cystic medial degeneration.

Although similar in presentation, the physiopathology mechanism of each AAS is not the same. In AAD, a tear in the intima leads to an inflow of blood all along the aortic media, which separates the intima and creates a false lumen [3]. Aortic intramural hematoma is a distinct type of aortic dissection, which is believed to be initiated by bleeding from the vasa vasorum into the media without intimal disruption and blood flow; however, this can be the initial step in the evolution of further aortic dissection [23]. An atherosclerotic lesion that penetrates the elastic layer of the aortic wall and creates a hematoma within the medial layer of the aorta is defined as a penetrating atherosclerotic ulcer [1].

Classification of aortic dissection

Three major systems are used to classify AADs: (1) DeBakey I, II, and III; (2) Stanford types A and B; and (3) "proximal" and "distal" aortic dissection. These three systems use the anatomical location of the intimal tear in the aortic wall and the length of the false lumen to make the classification of the aortic dissection. These classifications apply in the same way for all AASs.

The DeBakey type I classification is defined as a dissection with an intimal tear in the ascending aorta and the dissected false lumen continuing to the entire aorta. In DeBakey type II, only the ascending aorta is compromised, and in DeBakey type III, the intimal tear is located in the descending aorta, without the involvement of the arch and the ascending aorta. There is a sub-classification of the DeBakey III classification [24]: type IIIa, which is when the dissection is localized only in the thoracic aorta; and type IIIb, which is when the dissection reaches the abdominal aorta.

In the Stanford classification, the involvement of the ascending aorta is used for categorizing regardless of the anatomical location of the intimal tear. The Stanford type A dissection includes any dissection that compromises the ascending aorta (DeBakey I and II), and the Stanford type B dissection includes only the descending aorta (DeBakey III) [19].

The term "proximal" aortic dissection is reserved for dissections proximal to the origin of the left subclavian artery, regardless of the involvement of any segments distal to this anatomical point. "Distal" aortic dissection is reserved for dissections distal to the origin of the left subclavian artery and not involving the aorta proximal to that point [25]. In addition, an aortic dissection less than 2 weeks from symptom onset is defined as acute, and one more than 2 weeks from symptom onset is defined as chronic [13]. The term subacute is reserved for aortic dissections from 15 to 60 days after onset and describes the transition between the acute to the chronic phase [26].

Demography and characteristics

The peak incidence of aortic dissection occurs between the 6^{th} and 7^{th} decades of life [4,12,13,19] at a mean age of 61 years. Geographic differences or international heterogeneity in clinical presentation, diagnostic frequency, and clinical outcomes have been described; men are more often affected than women (68% vs. 32%) [27—29]. Aortic dissection is unusual in patients under 40 years old [30], for whom AAD is usually related to Marfan syndrome or bicuspid aortic valve, etc. Patients with AAD may present with a history of aortic surgery or the comorbidity of a larger aortic diameter.

AAD exhibits circadian and seasonal variations [31—33]. It usually occurs from early morning to midday, with a peak at between 8:00 AM and 9:00 AM There is no significant variation for the day of the week, but the frequency of AAD is higher during winter, both in cold and in temperate climates, with a peak in January.

Cocaine use is a risk factor for aortic dissection [34,35]. This etiology is more common in young, black, and hypertensive men, and the dissection more often appears earlier in the descending aorta [9,34,35]. AAD is an extremely rare complication in pregnancy [27,36]; it typically occurs in the third trimester or in the early postpartum period, is usually thoracic, and the proximal tear most frequently occurs in the ascending aorta [4,37]. Blunt trauma can cause dissection of the aorta at the level of the aortic isthmus, but this is uncommon [4,38]. Iatrogenic trauma is more commonly associated with true AAD after cardiac intervention (0.03—0.1% occurring after major cardiac surgery [4]), and intravascular or cardiac catheterization procedures. This incidence rises to between 0.5% and 1.0% in patients undergoing aortic valve replacement [20]. Type A iatrogenic AAD is most common after cardiac surgical intervention, whereas type B is most frequent after cardiac catheterization [39].

Genetic disorders and aortic dissection

Genetic defects in matrix protein synthesis are well known to predispose people to aortic dissection. Cystic medial

necrosis of the proximal aorta with dissections is associated with a number of conditions, such as Turner syndrome, Loeys—Dietz syndrome, Marfan syndrome, Noonan syndrome, Ehlers—Danlos syndrome type IV, osteogenesis imperfecta, homocystinuria, adult polycystic kidney disease, bicuspid aortic valve, coarctation of the aorta, and familial forms of thoracic aneurysm and dissection [4,5,13,36,40]. Up to 20% of patients with AAD have a family history of thoracic aortic aneurysms and dissection [36].

Clinical presentation

A routine clinical examination is not enough to rule out aortic dissection [41]. A high level of suspicion is the most important key in identifying a patient with an AAD. On initial examination, the diagnosis of AAD is missed in up to 38% of patients, and the diagnosis is first established on a post-mortem examination in up to 28% of patients [9].

The most common symptom at presentation is a sudden onset of severe chest pain, with or without back pain, radiating to the neck or shoulders. Nevertheless, in about 10% of the patients presenting with AAD, chest pain is absent at physical examination [18,42]. Hypotension or shock, or both, at presentation are more common in patients with type A dissection, whereas hypertension is more common in patients with type B dissection [8,18,36,43—45]. Other clinical findings include fever, diaphoresis, absence of pulse or cerebrovascular manifestations, acute abdominal pain, neurological deficit, paraplegia, aortic regurgitation related to cardiac failure, cardiac tamponade, and syncope [46]. An abrupt onset of pain and lack of hypertension at presentation is more common in patients less than 40 years old [30]. In contrast, hypotension is more common in patients who are over 70 years old [42,44].

Signs and symptoms

Pain is the most common initial symptom of AAD [18,45]. In type A aortic dissection, pain is more commonly localized in the anterior chest, and in type B, pain is more frequently localized in the back or in the abdomen [18,45]. A pulse deficit is found in fewer than 20% of all patients [18]. In patients with proximal dissection, a diastolic murmur is indicative of aortic regurgitation [47]. Paraplegia might be seen when critical intercostal arteries are separated from the aortic lumen [19]. Jugular venous distention, distant heart sounds and pulsus paradoxus are indicative of a more ominous extension and demand rapid diagnostic confirmation [4,19]. In about 7% of the cases, the ostium of a coronary artery may be involved in causing, with or without coronary malperfusion or even resulting in an acute myocardial infraction [13,41,48]. Pulse deficits, the murmur of aortic regurgitation, and neurological manifestations are more characteristic of proximal than distal dissection [13].

Diagnostic tests

When evaluating patients with a potential aortic dissection, laboratory testing plays a minor role because currently there is no sensitive or specific diagnostic laboratory test [19]. However, new tests that may confirm AAD, such as those that measure soluble elastin fragments, smooth muscle myosin heavy chain, acute-phase reactants such as the white blood cell count, high-sensitivity C-reactive protein, fibrinogen and D-dimer, are being developed [4]. An electrocardiogram (ECG) trail can be performed during the evaluation of a patient suspected of having aortic dissection. The ECG findings include non-specific ST-segments or T-waves. However, the ECG trail can be normal in almost a third of patients [4].

Diagnostic images

A dissection flap between two separate lumens is a definitive finding in diagnosing an aortic dissection, and this can be established by echocardiography, computed tomography (CT), magnetic resonance imaging (MRI), or aortography [19,36]. A chest X-ray can be used to evaluate patients with potential AAD [42]; however, a sensitivity of 64% and a specificity of 86% limits its use [49]. The most common abnormality in chest X-ray imaging is a widening aortic silhouette [13]; an abnormal cardiac contour, the "calcium sign", and a left pleural effusion are other common signs [4,13,19,40]. In almost 12% of patients who present to the emergency room, the chest radiograph is unremarkable [13].

With a sensitivity of 100% and a specificity of 98% [50], contrast-enhanced helical CT is currently the most commonly used imaging modality for evaluating patients with a potential aortic dissection. Transesophageal echocardiography (TEE) appears to be the second most used diagnostic tool in the diagnosis of aortic dissection [51]; it has a sensitivity of 99% and a specificity of 89% [52]. TEE has the advantage that it can be used in an emergency setting in a hemodynamically compromised patient [49]. Due to the low sensitivity (77—80%) and specificity (93—96%) of transthoracic echocardiography [19], it is of limited value for diagnosing AAD. MRI is the most accurate diagnostic modality, with a sensitivity of 100% and a specificity of 94% [52]. However, MRI is rarely used as the initial imaging technique because it is not applicable for hemodynamically unstable patients, it is incompatible with implanted metal devices, its availability is limited, it takes time to process the images, and there are often monitoring difficulties during examination [50,51].

Management

Case series and registries, consensus documents, systematic reviews, and local expertise results are the basis of the clinical pathways in the treatment of AAD [8]. The first step when treating suspected AAD is admitting the patient into an intensive care unit for hemodynamic monitoring [53]. Medical therapy should be begun as soon as possible to decrease the left ventricular dP/dt max, systolic blood pressure, and heart rate until a definitive diagnosis is obtained. Intravenous morphine should be used to treat pain [36]. Anxiety can be reduced with intravenous anxiolytics [4,13]. The target systolic blood pressure, unless hypotension is present, should be 100—110 mmHg, with

a target heart rate of between 50 and 60 beats per minute. Intravenous β-blockers are the initial agent of choice when treating suspected AAD because they reduce dP/dt max [36]. Short-acting β-blockers such as propranolol, labetalol, or esmolol are preferred over long-acting β-blockers because they are easier to control and can be rapidly discontinued if surgery is planned [13]. Sodium nitroprusside should never be used alone because it can induce an increase in dP/dt max that contributes to the propagation of the dissection. Therefore, it is necessary to give β-blockers before nitroprusside [4,13,53]. Patients with acute or chronic renal insufficiency should be treated with intravenous fenoldopam instead of sodium nitroprusside [54]. If β-blockers are contraindicated, intravenous calcium channel antagonists, such as diltiazem and verapamil, are an option for patients with bronchospasm. For patients with refractory hypertension, the intravenous angiotensin-converting enzyme inhibitor enalapril is the best option [4,13].

Cardiac tamponade or aortic rupture can cause hypotension, but before installing the treatment for hypotension, pseudohypotension must be ruled out. Rapid volume expansion should be administered [4,13]. If refractory hypotension results, vasopressors, such as norepinephrine (Levophed) or phenylephrine (Neo-Synephrine), are preferred to maintain organ perfusion. Dopamine should be reserved for improving renal perfusion and used only at a very low dose because it may increase dP/dt max [4,13]. Only in patients with cardiac tamponade and shock complicating aortic dissection is pericardiocentesis used; it may be successful in patients who have pulseless electric activity or shock while waiting for a definitive surgical repair [55].

Surgical treatment

After the successful initiation of medical treatment, further management is based primarily on three factors: type of dissection (type A or type B); false lumen characteristics (classic dissection or intramural hematoma); and hemodynamic status of the patient. For type A AAD, surgery is almost always mandatory; the therapeutic aim is to resect the intimal tear, which can be done by replacing the ascending aorta or by replacing the proximal or total aortic arch using the stented elephant trunk procedure. Deciding which aortic segment will be replaced is based on the location of the intimal tear. Furthermore, the following techniques have improved both early and late surgical outcomes in recent years: (1) open distal anastomosis to avoid aortic cross clamping; (2) antegrade systemic recirculation after distal anastomosis; (3) biological glue to preserve the native aortic valve when indicated; (4) cerebral protection with selective antegrade cerebral perfusion (SACP) or retrograde cerebral perfusion; and (5) postoperatively receiving a false lumen examination with periodical imaging studies [56]. When repairing an AAD, brain preservation is still one of the major challenges during surgery, and failure to do so is the best predictor of hospital mortality [57]. Cook et al. [58] showed that a systemic temperature of below 22°C may not be necessary and may be associated with a higher incidence of neurological injury when SACP is used during deep hypothermic arrest. Salazar et al. [59] supported the conclusion that systemic circulatory arrest with SACP at moderate hypothermia can be safely performed. Notably, surviving the initial surgical repair does not guarantee freedom from subsequent aortic events, because much of the thoraco–abdominal aorta frequently remains dissected and at risk of forming a dissecting aneurysm in the late phase [60–63]. This problem was partially resolved by a recent and more aggressive approach to surgically repairing AAD, which utilized a total aortic arch replacement combined with the stented elephant trunk procedure and yielded a low incidence of reoperation [64]. By contrast, the best treatment for the octogenarian patient is still unclear and needs further research. However, a minimally invasive approach, such as the less-invasive quick replacement with moderate hypothermic circulatory arrest followed by aggressive rapid rewarming, appears to be an option when treating this group of patients [56].

For type B AAD, the initial therapy is medical, and surgery or endovascular repair is reserved and indicated for patients who develop complications, such as persistent symptoms (despite medical treatment), a rapidly expanding false lumen, impending or frank aortic rupture, or major-organ malperfusion that cannot be resolved by percutaneous therapy [65]. Despite significant improvements in anesthesia, surgical techniques, and postoperative care, emergency open surgical repair of type B AAD continues to be associated with a significant mortality risk (as high as 25–50% in many reports) and only a few papers have reported more favorable results [65–69].

The role of thoracic endovascular aortic repair (TEVAR) in treating uncomplicated type B AAD remains controversial; however, TEVAR has emerged as a new therapeutic option in the treatment of type B AAD associated with life-threatening complications. In the International Registry of Acute Aortic Dissection, the less invasive nature of endovascular treatment seems to provide better short-term outcome in terms of mortality and associated complications than open surgical repair [68]. Recently, Zeeshan et al. [70] also supported the conclusion that TEVAR for complicated type B AAD is associated with superior early outcomes and improved midterm survival compared with conventional open surgical therapy. Nevertheless, a longer follow-up is necessary to assess late outcomes and establish the best treatment strategy [68,70].

Follow-up

Regardless of the type of dissection and treatment, at 10 years, 15–30% of patients require new surgery for threatening conditions, including aortic dilatation and rupture, progressive aortic regurgitation, organ malperfusion, and irreversible ischemia [4]. Thus, all patients, regardless of the initial therapeutic strategy used, need extremely close follow-up visits, including long-term medical therapy with β-blockers and serial imaging. According to the 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM Guidelines for the Diagnosis and Management of Patients with Thoracic Aortic Disease [42], CT imaging or MRI of the aorta should be carried out at 1, 3, 6, and 12 months post-dissection

and, if the patient is stable, annually thereafter so that any threatening enlargement can be detected in its early stages and in a timely fashion. Reoperation or endovascular stenting is needed if the maximum diameter of the dissected aorta is $\geq$55 mm, there is a rapid enlargement of dissected aorta ($>$10 mm/year), rupture of dissected aorta, aortic valve insufficiency, or aortic root dilatation [71,72].

Outcome

Further dilatation and subsequent rupture of the distal aorta are the most common causes of death after surgery for AAD [62,73]. Predictors of late aortic growth, further dilatation, and rupture include, male gender [60], age of $<$60 years, and type B aortic dissection [61], Marfan syndrome [62], a postoperative initial aortic diameter of $\geq$40 mm [60,74,75], the presence of blood in the false lumen [76], a large false lumen [77], the size and location of the intimal tear [78], elevated systolic blood pressure [78], elevated pulse pressure [79], a completely patent false lumen [61,63,80], and a partially patent false lumen [75,78,81].

Patients with a completely thrombosed false lumen have a better outcome during the follow-up period [42,61,82]. However, whether a completely patent false lumen is or is not an independent risk factor for a faster and a higher growth rate, and whether it has a worse outcome than does a partially patent false lumen has recently been debated [63,73,75,80,81]. Further investigation is needed to clarify this issue.

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