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Review Article

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Approach to aortic dissection in light of the American College of Cardiology (ACC) and American Heart Association (AHA) 2022 Guidelines

Abstract
The blood necessary for sustaining life passes into the aorta through the pump function of the left ventricle and is distributed to the systemic arterial bed via the aorta and its branches. In an average lifetime, about 200 million liters of blood, the basis of life, are delivered to the organs via the aorta and its branches Acute Aortic Syndromes, especially Aortic Dissection, can impair this vital function of the aorta. Aortic Dissection (AD) is not one of the most common diseases encountered by physicians in daily clinical practice, but it is frequently associated with life threatening complications. It has a high mortality rate of 1% to 2%/h if it is not diagnosed quickly and appropriate treatment is not started immediately. However, this diagnosis is not always easy to make. The main reason for this is the lack of a very specific symptom that would allow physicians, especially emergency physicians, cardiologists and cardiovascular surgeons, who encounter AD more frequently, to easily suspect AD and its course with nonspecific symptoms. In this review, we will evaluate new developments in the diagnosis and treatment of AD in the light of the American College of Cardiology (ACC) and American Heart Association (AHA) 2022.

Keywords: American College of Cardiology, American Heart Association, acute aortic syndromes, acute aortic dissection, diagnosis and treatment of aortic dissection

INTRODUCTION

Acute aortic syndrome (AAS) describes the acute presentation of patients with characteristic “aortic pain” caused by one of several life threatening thoracic aortic pathologies. These include aortic dissection, intramural haematoma, penetrating atherosclerotic ulcer, aneurysmal leak, and traumatic transection. (1). Predisposing factors such as hypertension, atherosclerosis, or certain connective tissue diseases create a favorable environment for the development of AAS by causing degeneration in the media layer of the aortic wall (2,8). In addition to AAS, AD requires the formation of an entry site in the aortic wall for blood to penetrate into the media layer. The key factor for this is the shearing stress created by high-pressure blood flow in the aortic wall (3).

Through the tear in the aortic intima caused by shearing stress, high-pressure blood flows into the media layer and forms a flap. Through a second tear (re-entry) in the aortic intima, blood returns to the aortic lumen. As a result, a new lumen is formed within the media layer of the aortic wall (false lumen) (4). The progression

of the false lumen in an antegrade or retrograde manner can lead to a series of life- threatening complications such as acute aortic insufficiency, myocardial ischemia, cardiac tamponade, acute stroke, malperfusion syndromes, or aortic rupture (5,9).

AD has a high mortality rate, ranging from 1% to 2% per hour (6,10). In untreated cases, the mortality rate can increase to 33% by the end of the first 24 hours, 50% by the end of the first 48 hours, and up to 75% by the end of the first week (7).

In this article, we will evaluate the current approaches to the diagnosis and treatment of AD based on the 2022 guidelines for the diagnosis and treatment of aortic diseases by the American College of Cardiology (ACC) and the American Heart Association (AHA).

Definition

AD occurs when there is an intimal tear in the aortic wall that allows blood to pass into the media. At the intimal tear, high-pressure blood filling the medial layer splits the medial layer longitudinally to form a flap in both the antegrade and retrograde directions. Blood traveling through the medial layer returns to the lumen through a second tear in the aortic wall. This creates a new lumen in the aorta that allows blood flow (false lumen). The length and extension of this lumen and the loss of strength in the aortic wall are the main factors determining the clinical outcome (11).

There are two important parameters used in AD classification. The first one is based on the time from the onset of symptoms until the diagnosis is made and the other one is based on the localization of the intimal tear and the extension of the false lumen, i.e. it is an anatomical classification.

Traditionally, the disease was divided into two main groups based on this time elapsed ("acute" during the first 2 weeks and "chronic" after the second week). However, the International Registry of Acute Aortic Dissection (IRAD) research group proposes a time-dependent division of AD into 4 temporal subtypes to guide the decision on the timing of potential intervention and contribute to improving prognosis. (Table 1) Because the shorter the time from symptom onset to diagnosis, the better the prognosis and the most contemporary temporal classification improves prognosis and guides decision making about the timing and types of potential intervention (12).

Table 1. Classification of aortic dissection according to time to diagnosis

Classification of aortic dissection chronicity based on the 2020 SVS/STS reporting standards

Type	Time since onset of symptoms
Hyperacute	<24/h
Acute	2-7 d
Subacute	8-30 d
Chronic	>30 d

Anatomical classification based on the localization of the intimal tear and the extension of the false lumen is as important as temporal classification and is one of the main factors in determining treatment protocols. Anatomical classification is traditionally done in two different ways. Historically, the first is the DeBakey classification defined in 1965. The second is the Stanford classification defined in 1970 (13). The Stanford classification is the most commonly used by clinicians (14). The DeBakey classification is used to provide more anatomical detail, while the Stanford classification is simpler. In the Stanford classification, ADs are divided into two groups as A and B according to the anatomical location of the false lumen, whereas in the DeBakey classification, ADs are divided into three groups as types 1, 2 and 3.

In the Stanford classification, if the ascending aorta is involved, it is called type A AD, and if the descending aorta is not involved, it is called type B AD. In the DeBakey classification, Type 1 involves the ascending aorta, arch aorta and descending aorta, Type 2 involves only the ascending aorta and Type 3 involves only the descending aorta (distal to the left subclavian artery) (13,14) (Figure 1).

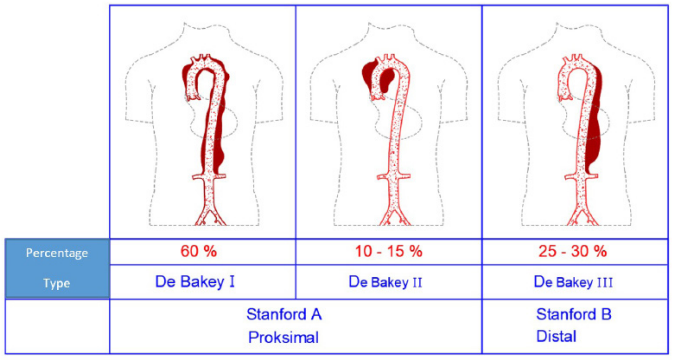

Figure 1. Anatomic classification of aortic discussion

However, in daily clinical practice, there were cases where both of these anatomical classifications were insufficient. In response, the European Association for Cardio-Thoracic Surgery and the European Society for Vascular Surgery published an expert consensus document in 2019. Accordingly, a third category of AD called "non-A non-B dissection" was defined. The aim was to classify patients whose proximal dissection flap originated from the aortic arch (15).

Most recently, in 2022, the Association for Cardio-Thoracic Surgery and the European Society for Vascular Surgery proposed a completely new classification scheme that defined the anatomy of AD in more detail.

Accordingly, dissections were classified according to the location of intimal tears and the degree of proximal and distal progression of the dissection process (16).

The purpose of making these classifications in great detail is to provide the most appropriate patient with the ideal treatment method.

Because the placement of the dissection flap is one of the most influential factors on mortality and morbidity in AD patients. For this reason, this new anatomical classification in the ACC/AHA 2022 guidelines is presented in addition to the historical anatomical classifications (Figure 2).

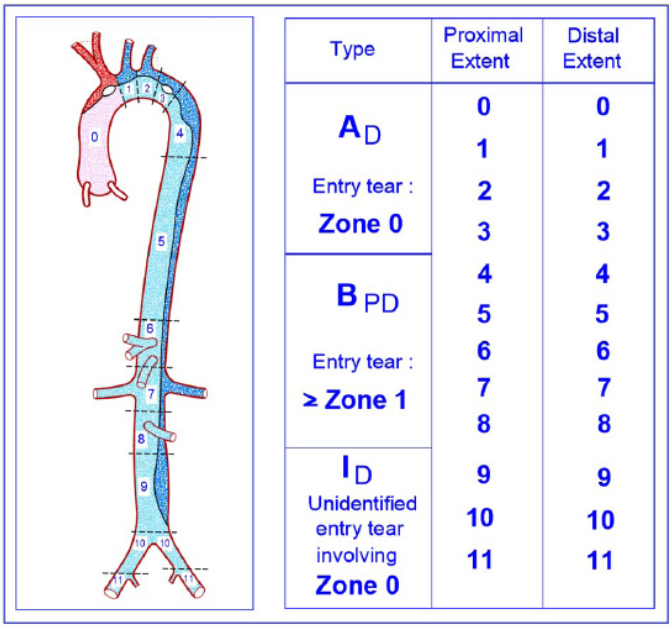

Figure 2. Anatomic classification of aortic dissection

Epidemiology

According to US data, the incidence of AD is 2.5-3 cases/100,000/year, but in some studies it is as high as 7.2 cases/100,000/year (1). AD cases increase significantly with advanced age and two-thirds of cases are men. Most cases of dissection occur between the ages of 50 and 70 years, although it occurs at a younger age in patients with connective tissue diseases (17). The average age is 63 years in men and 67 years in women (17,18).

The etiologic and risk factors of AD include age, gender, dyslipidemia, elevated apolipoprotein A, arterial hypertension, aortic stenosis, aortic coarctation, aortic aneurysm, connective tissue diseases, and a wide variety of acute and chronic factors leading to aortic wall stress. (cocaine use, weight lifting, trauma, infectious and inflammatory vasculitis) (18). The common feature of etiologic and risk factors is that they weaken the aortic wall and reduce its elasticity, and the most common etiologic factor is hypertension (17,19).

Mechanical and Degenerative Factors in the Development of Aortic Dissection

Mechanical and degenerative factors involved in the development of AD either cause structural weakening of the aortic wall by causing deterioration in the elements that make up the vessel wall or damage to the intima by increasing shear stress.

Among inherited connective tissue disorders, Marfan syndrome, Loeys-Dietz syndrome and Ehlers Danlos syndrome cause weakening of the aortic wall. In Marfan syndrome, there is a heterozygous mutation in the FBN1 gene encoding the extracellular matrix protein fibrillin-1, while in Loeys-Dietz syndrome there is a mutation in the components of the TGF-β signaling pathway, (TGF-β plays a key role in cellular events such as cell proliferation and differentiation) In Ehlers Danlos syndrome, there is a mutation in the genes encoding collagen fibrils (20).

The role of atherosclerosis, which involves the vessel wall and is characterized by chronic inflammation, in the development of AD is due to the triggering of immunoinflammatory mechanisms. Activated immunoinflammatory mechanisms cause weakening of the structural elements of the aortic wall (21).

The main effect of biomechanical factors such as hypertension, bicuspid aortic valve, aortic stenosis, aortic coarctation, aortic aneurysm on the development of AD is intimal damage and weakening of the vessel wall caused by prolonged high pulsatile pressure and increased shear stress (22,23).

Hypertension is also one of the most important risk factors for the development of medial degeneration. This, in turn, can lead to weakening of the aortic wall, leading to the development of AD (24).

The adverse effect of aging on the development of AD is due to an increase in collagen in the vessel wall, loss of elastin fibrin, varying degrees of smooth muscle cell changes and loss of vascular function. These adverse changes weaken the vessel wall and predispose older patients to the development of AD (25).

Histopatology

Both mechanical and degenerative factors involved in the development of AD lead to a series of histopathologic changes. The Association for European Cardiovascular Pathology (AECVP)/Society for Cardiovascular Pathology (SCVP) (2016) diagnostic criteria are currently used to define these histopathologic changes (26).

Regardless of which etiologic cause AD develops, including connective tissue diseases, the last common histopathologic finding in all of them is medial degeneration (26). The main medial degeneration findings are as follows (3,6).

1. Mucoid deposition occurs in the extracellular matrix leaving intralamellar or translamellar spaces. This is why it is called "cystic degeneration" (27) (The main component of mucin deposition is chondroitin-6-sulfate).
2. Fragmentation, thinning and irregularity of elastic fibers. Loss of elastic fibers leads to further enlargement of the translamellar spaces, which reduces the elasticity of the aorta.
3. Nuclear loss and irregularities are seen in smooth muscle cells. The main cause of nuclear loss in smooth muscle cells is smooth muscle cell necrosis.

4. Fibrosis of the collagen fibers occurs. This is the least common finding in degenerative aortic disease.

These findings are usually present together and are classified as mild/moderate/severe depending on the severity of the degenerative lesions (3).

Diagnostic Approach (Imaging, Laboratory Tests)

The most common symptoms and signs of aortic dissection are shown in Table 2.

Physicians face some difficulties in diagnosing AD. These are factors such as the fact that AD is a very rare disease, the lack of specific signs and symptoms of AD, the fact that patients may present to the physician with very different nonspecific complaints, and the fact that tests that are frequently used in daily clinical practice such as direct chest X-ray, electro cardiography (ECG), and laboratory tests are used in differential diagnosis rather than diagnosing AD (2,28).

Another reason for the difficulty in making the diagnosis is that the definitive diagnosis is only possible with advanced radiological imaging that can only be performed in certain centers (28,29) (computed tomography (CT), magnetic resonance imaging (MRI) and transesophageal echocardiography (TEE)).

A plain chest X-ray is neither sufficiently sensitive nor specific for the diagnosis of AAS. However, it is a frequently used imaging modality in daily clinical practice. Some radiographic findings on plain chest X-ray may lead us to suspect aortic dissection (30) (Table 3).

Table 2. Most common symptoms and findings in aortic dissection

Signs and symptoms of AD	
Clinical signs and symptoms	**Cause**
Asymmetric blood pressure (>20 mm Hg) between limbs	Compromise of branch artery flow
Bowel ischemia or gastrointestinal bleed	Malperfusion of the celiac or superior mesenteric artery
Oliguria or hematuria (gross)	Malperfusion of 1 or both renal arteries
Dysphagia	Compression of the esophagus
Dyspnea	Compression of trachea or bronchus, congestive heart failure from aortic regurgitation, or cardiac tamponade
Hemoptysis	Vascular rupture into lung parenchyma
Horner's syndrome	Compression of sympathetic chain
Myocardial ischemia or myocardial infarction	Coronary artery involvement by dissection or compression by aneurysm
New murmur of aortic regurgitation	Incomplete aortic valve closure secondary to leaflet tethering by the dilated aorta or cusp prolapse because of dissection into the aortic root
Paraplegia	Spinal malperfusion attributable intercostal artery involvement
Lower extremity ischemia	Malperfusion of iliac artery
Superior vena cava syndrome	Compression of the superior vena cava
Shock	Cardiac tamponade, hemothorax,frank aortic rupture, acute severe aortic regurgitation, severe myocardial ischemia
Shortness of breath	Pericardial effusion, congestive heart failure from acute severe aortic regurgitation, or hemothorax
Syncope	Carotid artery involvement or cardiac tamponade

Table 3. Aortic dissection on flat chest x-ray findings

Signs of aortic dissection on chest x-ray findings
a. Mediastinal widening
b. Disruption of the normally distinct contour of the aortic knob
c. "Calcium sign," which appears as a separation of the intimal calcification from the aortic wall of >5 mm
d. Double density appearance within the aorta
e. Tracheal deviation to the right
f. Deviation of the nasogastric tube to the right

Diagnostic Tools in Aortic Dissection

High-quality images of the aorta and its branches are needed for rapid and accurate diagnosis of AD and determination of the most appropriate treatment option (31,32). In addition to clearly demonstrating the anatomy of the aorta and its branches in all plans, the extent of the pathologic condition, the loss of function in the organs and the complications that occur should be fully defined (2). Thus, it will be possible to choose the most appropriate treatment for the patient in the shortest time possible. Considering that the diagnosis and treatment of AD is a race against time, it is clear that such diagnosis and treatment planning will make a positive contribution to mortality and morbidity.

The diagnostic tools that can be used in AD are Computed Tomographic Angiography (CTA), Magnetic Resonance Angiography (MRA), Transthoracic Echocardiography (TTE), Transesophageal Echocardiography (TEE) and Aortography (AG). The choice of which of these diagnostic tools to use

depends on the diagnostic performance of the diagnostic tools in AD as well as on patient-related factors and the physical capacity of the healthcare institution (2). Table 4 shows a comparison of the diagnostic tools that can be used in AD.

As seen in Table 4, although the sensitivity, specificity and diagnostic reliability of CTA, MRA and TTE, which are commonly used diagnostic tools in the diagnosis of AD, are high, CTA is currently the first preferred imaging method in the diagnosis of AD. This is due to the technical success of CTA in the diagnosis of AD, its widespread availability in emergency departments at all hours, and its rapid and easy application. CTA not only provides a rapid and accurate diagnosis of AD, but also reveals all aspects of pathologic changes in the aorta and its branches. In addition, it provides detailed information about complications such as malperfusion, pericardial effusion, hemopericardium, periaortic or mediastinal hematoma and pleural effusion (2).

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Table 4. Comparison of diagnostic modalites used in aortic dissection

Diagnostic performance of aortic imaging modalities					
Method	**BTA**	**MRA**	**TTE**	**TÖE**	**AG**
Easy usability	+++	++	+++	++	+
Sensitivity	++	+++	++	++	++
Specificity	++	+++	+++	+++	++
Diagnostic reliability	+++	+++	+	+++	++
Mobility	-	-	+++	+++	+++
Acquisition speed	+++	+	++	++	++
Spatial resolution	+++	++	++	+++	++
Three-dimensional data provisioning	+++	++	+	+	+
Evaluation of aortic branches	+++	+++	++	+	-
Capillaries and ventricles evaluation of function	+	+	+++	+++	-
Bedside use	-	-	++	++	-
Aortic wall imaging	+++	+++	+	++	-
Mural thrombus	+++	+++	+	+++	+
Identifying the coronaries involved	-	+	+	++	+++
Identifying the mediastinal hematoma	+++	+++	++	++	-
Identifying the Pericardial fluid	++	++	++	+++	-
Cost	++	+++	+	+	+++
Radiation	+++	++	-	-	+++
Impact on the kidney	+++	++	-	-	+++

BTA: computed tomographic angiography, MRA: magnetic resonance angiography, TTE: transthoracic echocardiography, TEÖ: transesophageal echocardiography, AG: aorta radiograph, +++=excellent results, ++=good results, +=bad results, -=not available

Obtaining high quality images of the aorta and its branches, whether by CTA or other advanced imaging modalities, and clearly demonstrating the pathologic change is necessary but not sufficient for the patient to receive the most appropriate treatment. In order to reach the optimal treatment method for the patient in AD, the myocardial performance, the morphology and function of all heart valves, especially the aortic valve, and the end-organ perfusion status of the patient must be clearly demonstrated (33). Such an approach is only possible if all available diagnostic tools, especially echocardiography, are used in a complementary manner and the findings are supported by laboratory tests. Such a multidisciplinary approach will increase the chances of rapid and accurate diagnosis

and contribute positively to the success of treatment.

Since the diagnosis and treatment of AD is a race against time, experts have tried to develop algorithms to be used in patients with suspected AD in order to accelerate this process. Two of these algorithms were recommended in the ACC/AHA 2022 guidelines (2).

In the first one, the patient is given 1 point for each risk category in the aortic dissection detection risk score (AAD-RS). The risk categories are classified according to examination findings, symptoms and the patient's medical history (Table 5). According to the AAD-RS, patients are divided into three groups according to the sum of their scores. Patients with a score of zero are considered to be in the low risk group for ADD-RS, those with a score of 1 are considered to be in the moderate risk group, and patients with a score of 2 to 3 are considered to be in the high risk group. If patients are in the low and moderate risk group, they are tested for D-Dimer. If D-Dimer is <500 ng/mL, the diagnosis of AD can be excluded without the need for further radiological examination for AD. However, if D-Dimer is >500 ng/mL, further imaging methods are recommended. If patients are in the high-risk group, they are referred directly to advanced imaging centers (2,34).

Table 5. Aortic dissection detection risk score

Aortic Dissection Detection Risk Score (ADD-RS)		
High-risk conditions	**High-risk pain features**	**High-risk examination features**
(Chest, back, or abdominal pain described as)		
Marfan syndrome or other connective tissue disease	Abrupt onset	Pulse deficit or systolic blood pressure differential
Family history of aortic disease	Severe in intensity	Focal neurologic deficit (with pain)
Known aortic valve disease	Tearing/fragmentation sensation	Murmur of aortic regurgitation (new, with pain)
Recent aortic manipulation	Ripping or tearing in quality	Hypotension or shock state
Known thoracic aortic aneurysm		

For each risk category, 1 point is given if there is ≥1 risk factor. As a result, the total ADD-RS will range from 0 to 3. An ADD-RS of 0 points is low risk; 1 point is moderate risk; and 2 to 3 points is high risk

The second proposed algorithm is the simplified aortic score (Aorta simplified score (AORTAs) (Table 6). Patients with a total score between 0 and 1 are considered to have a low probability of aortic dissection, whereas patients with a total score ≥2 are considered to have a high probability. After this point, patients are evaluated in the same way as ADD-RS (5,35,36). However, it should never be forgotten that a low D-Dimer level does not completely rule out the diagnosis of AD, nor does a high D-Dimer level allow a definitive diagnosis.

Tablo 6. Aorta simplified score

Aorta simplified score (AORTAs) pretest probability assessment score	
Clinical item	**Points**
Hypotension/shock	2
Aneurysm	1
Pulse deficit	1
Neurologic deficit	1
Severe pain	1
Sudden-onset pain	1

The patient is given the number of points corresponding to each clinical item present in the patient's clinic. The points are summed and a total of 0 to 1 points indicates a low probability of aortic dissection, where a total of ≥2 points indicates a high probability).

Treatment of Aortic Dissection

The most fundamental way to prevent mortality and morbidity in AD is to provide patients with the most appropriate treatment in the shortest possible time. The appropriate treatment protocol in AD ranges from emergency surgical intervention to endovascular methods and even hybrid techniques combining surgical intervention and endovascular methods (2).

It is extremely important that patients with suspected AD are followed up with an accurate and effective emergency medical treatment while necessary radiologic and laboratory tests are performed. A correct and effective emergency medical treatment facilitates the diagnostic process in patients, as well as preventing complications that may occur and increasing the chance of success of permanent treatment (2).

Emergency medical treatment includes aggressive heart rate control, aggressive blood pressure control and pain control. The main goal of emergency medical treatment is to reduce aortic wall stress. Intravenous beta- blockers (esmolol, metoprolol and labetalol) are the most effective treatment to reduce heart rate, while intravenous vasodilators (nicardipine, sodium nitroprusside) are the first-line agents to control blood pressure. Intravenous beta-blockers are the first-line agents for controlling heart rate, but if there is a contraindication to the use of beta-blockers (heart block or bradycardia) or if the patient cannot tolerate beta-blockers, then intravenous non- dihydropyridine calcium channel blockers (e.g. verapamil or diltiazem) should be preferred (2,37).

Although there is no clear consensus on the ideal heart rate and blood pressure values in patients with AD, it is recommended to maintain a heart rate of 60 to 80 beats/min and systolic blood pressure as close as possible to 120 mm Hg without impairing end-organ perfusion (2,38).

The pain that occurs in patients with AD negatively affects heart rate and blood pressure as it causes the release of catecholamines. Therefore, pain treatment is very important in controlling the patient's blood pressure and heart rate. Intravenous opiates

should be primarily preferred for pain treatment (2).

Long-term use of beta-blockers, angiotensin converting enzyme inhibitors (ACEs) or angiotensin receptor blockers (ARBs) for blood pressure control in patients with AD, regardless of the modalities used, has very positive effects on the long-term success of treatment (2).

Regardless of the method by which AD is treated, the primary goals of therapy are to prevent (or treat) aortic rupture, prevent retrograde spread of dissection toward the aortic root, prevent antegrade spread of dissection distally to undissected segments, and mitigate or, if possible, prevent malperfusion syndromes (2).

The decision on which treatment option is appropriate for which patient is based on the type of AD, its anatomical location, the signs and symptoms of the patient, the presence or absence of complications and the specific features of the anatomy of the patient's aorta and its branches, the capacity of the center where the patient receives treatment, the experience of the surgical team, and the result of the stent-graft feasibility study (2).

Patients with Type A aortic dissection, aortic rupture or end-organ malperfusion should be treated with urgent surgical intervention. Because in the presence of these three clinical pictures, medical treatment has a low chance of success. When medical treatment and surgical treatment were compared in terms of mortality rates in such cases, mortality rates of patients treated with medical treatment alone were found to be 2 to 3 times higher (2). IRAD data showed that from 1995 to 2013, the mortality rate of surgical treatment decreased from 25% to 18%, while the mortality rate of medical treatment alone remained unchanged over the years at 57% (39).

Acute Type A aortic dissection lacks a consensus among aortic surgery specialists regarding the limits and methods of surgical treatment. However, there are key determinants in establishing the surgical technique and boundaries, including the patient's overall health status and hemodynamic stability, the location and extent of the tear, and the presence of complications such as malperfusion and valve pathologies (2).

Based on these factors, the boundaries of the surgical procedure are determined by the aortic surgery team during the preoperative period. These boundaries can range from aortic root and valve replacement to hemiarch replacement or even complete replacement, including hybrid techniques involving endovascular methods. It should be noted that Type A aortic dissection is a highly dynamic pathology, and significant changes in the dimensions of the tear can occur during the preoperative period. Therefore, making decisions regarding the limits and methods of surgical treatment during the operation itself is the most appropriate approach (40).

In surgical treatments limited to the ascending aorta and aortic arch, the fate of the remaining dissection flap in the descending aorta used to be a significant concern. Consequently, hybrid techniques that combine conventional surgical techniques involving the replacement of the ascending aorta or aortic arch with the frozen elephant trunk (FET) technique have emerged in recent years. This method involves simultaneously replacing the ascending aorta or aortic arch and extending a prosthetic graft into the descending aorta. Studies on the reliability and applicability of the FET technique have shown favorable results in terms of aortic remodeling, false lumen thrombosis, and organ malperfusion in patients with acute Type A aortic dissection. Furthermore, the FET technique has demonstrated positive outcomes in reducing complications and improving survival rates in these patients (41).

Factors that increase the mortality rates of surgical treatment include the presence of shock, cardiac tamponade, myocardium ischemia, neurological and visceral malperfusion (2).

Similar to surgical treatment strategies for acute Type A aortic dissection, there is no consensus among aortic surgeons regarding cannulation techniques and antegrade/retrograde cerebral perfusion strategies. Various cannulation techniques have been defined for use during aortic surgery with cardiopulmonary bypass. These different cannulation techniques have different anatomical characteristics and flow patterns within the aorta. Currently, many aortic surgery teams primarily use one of three cannulation techniques: femoral artery cannulation, axillary artery cannulation, or central aortic cannulation. Unfortunately, there is a lack of comprehensive randomized studies comparing these three cannulation techniques. However, a few retrospective studies have compared axillary artery cannulation to femoral artery cannulation. These studies have suggested that right axillary artery cannulation is associated with better clinical outcomes compared to femoral artery cannulation, although the data are extremely limited. (42).

It is not possible to assert that any perfusion strategy is perfect or complication-free based on the limited and controversial results from these few studies in the literature. Nevertheless, based on the consistent results of many retrospective studies, antegrade perfusion strategy is preferred over retrograde perfusion strategy by aortic surgeons (43).

The natural course of the residual dissected flap and the dissected aortic segment remaining after surgical treatment of Type A aortic dissection is a significant concern. The persistence or enlargement of the false lumen in the dissected aortic segment can lead to various complications, primarily ischemic events. Therefore, regardless of the method used for treatment, close monitoring of the residual dissected aortic segment is crucial in the postoperative follow-up of Type A aortic dissection patients. The problems caused by the remaining aortic segment depend on the patient's current pathological condition and the type of complication that arises, and treatment strategies are developed accordingly (44).

Among the factors that increase the mortality rates of surgical treatment are shock, cardiac tamponade, meridional ischemia, presence of neurologic and visceral malperfusion (2). In

the absence of findings such as aortic rupture, branch artery occlusion and malperfusion, extension of dissection, aortic enlargement, intractable pain and uncontrolled hypertension in Type B aortic dissection, medical treatment should be the first choice in Type B dissection cases. The morbidity and mortality rates are high in patients presenting with complicated acute type B aortic dissection or reverting to a complicated form during their follow-up. The first treatment option for these patients is surgery, endovascular or one of the hybrid methods. However, endovascular methods are now the first-choice treatment in the treatment of complicated type B aortic dissection. It is uccessfully applied in many centers with low morbidity and mortality rates (2,45). However, the positive contribution of early endovascular intervention to the long-term survival rates of patients, rather than long-term medical treatment and follow-up, has not been clearly demonstrated (45).

CONCLUSION

Despite advances in imaging methods, surgical and interventional techniques in recent years, AD continues to be associated with significant morbidity and mortality. Clinical practice guidelines for the diagnosis and treatment of AD are regularly updated and published in order to alleviate the difficulties that may be encountered during clinical practice and to reduce complication rates.

In the recently published 2022 ACC/AHA guideline, innovations in the diagnosis and treatment of AD were presented in all aspects. In particular, it was emphasized that the increase in hourly morbidity and mortality is due to diagnostic delays rather than treatment strategies. In order to prevent diagnostic delays, the importance of a multidisciplinary approach was emphasized.

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REFERENCES

1. Ahmad F, Cheshire N, Hamady M. Acute aortic syndrome: pathology and therapeutic strategies. Postgrad Med J. 2006;82:305-12.
2. Saito H, Hayashi H, Ueda T, Mine T, Kumita SI. Changes in aortic wall thickness at a site of entry tear on computed tomography before development of acute aortic dissection. Ann Vasc Dis. 2019;12:379-84.
3. Kim GB, Park KH, Kim SJ. Hemodynamics and wall shear stress of blood vessels in aortic coarctation with computational fluid dynamics simulation. Molecules. 2022;27:1403.
4. Levy D, Goyal A, Grigorova Y, Farci F, Le JK. Aortic Dissection. StatPearls Publishing. 2023.
5. Tsai TT, Nienaber CA, Eagle KA. Acute aortic syndromes. Circulation. 2005;112:3802-13.
6. Kuang J, Yang J, Wang Q, Yu C, Li Y, Fan R. A preoperative mortality risk assessment model for Stanford type A acute aortic dissection. BMC Cardiovasc Disord. 2020;20:508.
7. Valentine EA, Ochroch EA. Vascular Surgery. In: Essentials of cardiac anesthesia for noncardiac surgery. Elsevier, 2019. p. 313-39.
8. Bossone E, Eagle KA. Epidemiology and management of aortic disease: aortic aneurysms and acute aortic syndromes. Nat Rev Cardiol. 2021;18:331-48.
9. Vignaraja V, Thapar A, Dindyal S. Acute Aortic Syndrome. StatPearls Publishing. 2022.
10. Valentine RJ, Wind GG. Anatomic exposures in vascular surgery. 4th ed. Wolters Kluwer Health. 2020.
11. Shi Y, Zhu M, Chang Y, Qiao H, Liu Y. The risk of stanford type-A aortic dissection with different tear size and location: a numerical study. Biomed Eng Online. 2016;15:128.
12. Levy D, Goyal A, Grigorova Y, et al. Aortic Dissection. StatPearls Publishing. 2023.
13. Booher AM, Isselbacher EM, Nienaber CA, Trimarchi S, Evangelista A, Montgomery DG, et al.; IRAD Investigators. The IRAD classification system for characterizing survival after aortic dissection. Am J Med. 2013;126:730.e19-24.
14. Debakey ME, Henly WS, Cooley DA, Morris GC Jr, Crawford ES, Beall AC Jr. Surgical management of dissecting aneurysms of the aorta. J Thorac Cardiovasc Surg. 1965;49:130-49.
15. Daily PO, Trueblood HW, Stinson EB, Wuerflein RD, Shumway NE. Management of acute aortic dissections. Ann Thorac Surg. 1970;10:237-47.
16. Czerny M, Schmidli J, Adler S, van den Berg JC, Bertoglio L, Carrel T, et al.; EACTS/ESVS scientific document group. Current options and recommendations for the treatment of thoracic aortic pathologies involving the aortic arch: an expert consensus document of the European Association for Cardio-Thoracic surgery (EACTS) and the European Society for Vascular Surgery (ESVS). Eur J Cardiothorac Surg. 2019;55:133-62.
17. Lombardi JV, Hughes GC, Appoo JJ, Bavaria JE, Beck AW, Cambria RP, et al. Society for Vascular Surgery (SVS) and Society of Thoracic Surgeons (STS) reporting standards for type B aortic dissections. J Vasc Surg. 2020;71:723-47.
18. Nienaber C. Das akute Aortensyndrom. Deutsche medizinische Wochenschrift. 1946;11:752-6.
19. Sen I, Erben YM, Franco-Mesa C, DeMartino RR.

Epidemiology of aortic dissection. Semin Vasc Surg. 2021;34:10-7.

20. Meester JAN, Verstraeten A, Schepers D, Alaerts M, Van Laer L, Loeys BL. Differences in manifestations of Marfan syndrome, Ehlers-Danlos syndrome, and Loeys-Dietz syndrome. Ann Cardiothorac Surg. 2017;6:582-94.
21. Tedgui A, Mallat Z. Cytokines in atherosclerosis: pathogenic and regulatory pathways. Physiol Rev. 2006;86:515-81.
22. Fatehi Hassanabad A, Garcia J, Verma S, White JA, Fedak PWM. Utilizing wall shear stress as a clinical biomarker for bicuspid valve-associated aortopathy. Curr Opin Cardiol. 2019;34:124-31.
23. Levy D, Goyal A, Grigorova Y, et al. (2022). Aortic Dissection. StatPearls [Internet]. Treasure Island. 2022.
24. Roberts WC. Frequency of systemic hypertension in various cardiovascular diseases. The American Journal of Cardiology, 1987;60:1E-8.
25. Gequelim GC, da Luz Veronez DA, Lenci Marques G, Tabushi CH, Loures Bueno RDR. Thoracic aorta thickness and histological changes with aging: an experimental rat model. J Geriatr Cardiol. 2019;16:580-4.
26. Halushka MK, Angelini A, Bartoloni G, Basso C, Batoroeva L, Bruneval P, et al. Consensus statement on surgical pathology of the aorta from the Society for Cardiovascular Pathology and the Association For European Cardiovascular Pathology: II. Noninflammatory degenerative diseases — nomenclature and diagnostic criteria. Cardiovascular Pathology. 2016;25:247-57.
27. Sawabe M. Vascular aging: From molecular mechanism to clinical significance: Vascular aging. Geriatr Gerontol Int. 2010;10:S213-20.
28. Evangelista A, Isselbacher EM, Bossone E, Gleason TG, Eusanio MD, Sechtem U, et al. Insights from the International Registry of Acute Aortic Dissection: A 20-year experience of collaborative clinical research. Circulation. 2018;137:1846-60.
29. Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey DE Jr, et al.;American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines; American Association for Thoracic Surgery; American College of Radiology; American Stroke Association; Society of Cardiovascular Anesthesiologists; Society for Cardiovascular Angiography and Interventions; Society of Interventional Radiology; Society of Thoracic Surgeons; Society for Vascular Medicine. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with thoracic aortic disease: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. Circulation. 2010;121:e266-369.
30. Bhave NM, Nienaber CA, Clough RE, Eagle KA. Multimodality imaging of thoracic aortic diseases in adults. JACC Cardiovasc Imaging. 2018;11:902-19.
31. Wu MY, Bang TJ, Restauri N, Chawla A, Khawaja RDA, Vargas D. Imaging acute aortic syndromes. Semin Roentgenol. 2022;57:335-44.
32. Grist TM, Rubin GD. Imaging of Acute Aortic Syndromes. In Hodler J, Kubik-Huch RA, Schulthess V, editors, Diseases of the Chest, Breast, Heart and Vessels 2019-2022: Diagnostic and Interventional Imaging. Springer. 2019.
33. Evangelista A, Maldonado G, Gruosso D, Gutiérrez L, Granato C, Villalva N, et al. The current role of echocardiography in acute aortic syndrome. Echo Res Pract. 2019;6:R53-63.
34. Nazerian P, Mueller C, Soeiro AM, Leidel BA, Salvadeo SAT, Giachino F, et al.; ADvISED Investigators. Diagnostic accuracy of the aortic dissection detection risk score plus D-dimer for acute aortic syndromes: The ADvISED prospective multicenter study. Circulation. 2018;137:250-8.
35. Ohle R, Anjum O, Bleeker H, McIsaac S. What is the specificity of the aortic dissection detection risk score in a low prevalence population?. Acad Emerg Med. 2019;26:632-8.
36. Rogers AM, Hermann LK, Booher AM, Nienaber CA, Williams DM, Kazerooni EA, et al.; IRAD Investigators. Sensitivity of the aortic dissection detection risk score, a novel guideline-based tool for identification of acute aortic dissection at initial presentation: results from the international registry of acute aortic dissection. Circulation. 2011;123:2213-8.
37. Tadros RO, Tang GHL, Barnes HJ, Mousavi I, Kovacic JC, Faries P, et al. Optimal treatment of uncomplicated type B aortic dissection: JACC review topic of the week. J Am Coll Cardiol. 2019;74:1494-504.
38. Suzuki T, Isselbacher EM, Nienaber CA, Pyeritz RE, Eagle KA, Tsai TT, et al.; IRAD Investigators. Type-selective benefits of medications in treatment of acute aortic dissection (from the International Registry of Acute Aortic Dissection [IRAD]). Am J Cardiol. 2012;109:122-7.
39. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D,

Karavite DJ, Russman PL, et al. The international registry of acute aortic dissection (IRAD): New insights into an old disease. JAMA. 2000;283:897-903.

40. Kuroda Y, Uchida T, Ohba E, Yamashita A, Nakai S, Kobayashi K, et al. Aortic remodelling effect of the frozen elephant trunk technique on Stanford type A acute aortic dissection. Interact Cardiovasc Thorac Surg. 2021;32:789-91.

41. Chivasso P, Mastrogiovanni G, Miele M, Bruno VD, Rosciano A, Montella AP, et al. Frozen elephant trunk technique in acute type A aortic dissection: Is it for all? Medicina (Kaunas). 2021;57:894.

42. Abe T, Usui A. The cannulation strategy in surgery for acute type A dissection. Gen Thorac Cardiovasc Surg. 2017;65:1-9.

43. Huang LC, Xu QC, Chen DZ, Dai XF, Chen LW. Combined femoral and axillary perfusion strategy for Stanford type a aortic dissection repair. J Cardiothorac Surg. 2020;15:326.

44. Neri E, Sani G, Massetti M, Frati G, Buklas D, Tassi R, et al. Residual dissection of the brachiocephalic arteries: Significance, management, and long-term outcome. J Thorac Cardiovasc Surg. 2004;128:303-12.

45. Xie E, Yang F, Liu Y, Xue L, Fan R, Xie N, et al. Timing and outcome of endovascular repair for uncomplicated type B aortic dissection. Eur J Vasc Endovasc Surg. 2021;61:788-97.