

Review

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# Endovascular repair for acute aortic syndrome involving the descending aorta

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## Abstract

This review paper delves into the acute aortic syndromes, with a particular focus on those affecting the descending thoracic aorta, including acute type B aortic dissection (aTBAD), intramural hematoma (IMH), penetrating aortic ulcer (PAU), blunt traumatic thoracic aortic injury (bTAI), and ruptured aneurysm of the descending thoracic aorta (rDTA). These conditions present with sudden-onset symptoms such as severe chest or back pain, necessitating immediate medical attention. While traditional open surgical repair was historically the mainstay of treatment, advancements in endovascular techniques have revolutionized management approaches. Endovascular treatment offers advantages such as reduced operative time, blood loss, and hospital stay, making it a safer option for high-risk patients. However, it is crucial to carefully evaluate patients for endovascular suitability, considering the potential complications and risks associated with these techniques. This paper aims to provide an updated overview of acute aortic syndromes involving the descending thoracic aorta, analyze available therapeutic options, and review contemporary treatment modalities, shedding light on the technical aspects and considerations guiding clinical decision-making in this complex and life-threatening scenario.

**Keywords:** Acute aortic syndromes, aortic dissection, intramural hematoma, penetrating aortic ulcers, aortic trauma, ruptured aortic aneurysms

## INTRODUCTION

Acute aortic syndromes (AAS) involving the descending thoracic aorta are potentially life-threatening



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disorders that encompass a diverse range of patients with similar clinical features but different underlying diseases, including aortic rupture, aortic dissection (AD), intramural hematoma (IMH), and ulcerative aortic lesions (PAU)<sup>[1]</sup>. Despite their distinctive nature, these conditions are grouped together due to the presence of comparable symptoms, and distinguishing between these pathological scenarios can sometimes be challenging<sup>[2,3]</sup>.

The primary clinical manifestation of AAS typically involves intense, abrupt chest and/or back pain. Nevertheless, individuals affected by AAS may display a diverse array of symptoms including abdominal pain, episodes of fainting, and diminished or absent pulse in the legs or feet. AAS presents a substantial threat to life, with mortality rates potentially soaring to 40%-50%; hence, timely identification of AAS patients is paramount, as is the ability to differentiate between AAS and other conditions sharing similar clinical features but associated with lower morbidity and mortality rates<sup>[1-4]</sup>.

The management of AAS involves a comprehensive approach encompassing medical, surgical, and endovascular interventions. Over the past three decades, the introduction of endovascular techniques has fundamentally altered the treatment landscape for acute aortic syndromes affecting the descending thoracic aorta. This period has witnessed a notable shift away from open surgical approaches, with a steady rise in the utilization of complete endovascular management. This review article will discuss the various pathologies included in the AAS group and explore the current advancements in endovasculartherapies. Description, results, and considerations regarding the contemporary treatment of the acute aortic syndromes have been liberally extrapolated by the authors of this review article from position papers and from the updated literature in this field.

#### **aTBAD - acute type B aortic dissection**

Aortic dissection can be a devastating event that leads to a diverse spectrum of clinical presentations; the particular impacts encountered by individuals are directly correlated with the distribution and magnitude of aortic and branch vessel involvement.

Classification systems for thoracic aortic dissection play a crucial role in facilitating rapid and accurate communication among caregivers for describing aortic diseases, thereby aiding in triage, treatment, and prognostic assessments. From an anatomical perspective, early experiences revealed distinct differences between patients with dissection of the ascending aorta, which carries a worse immediate prognosis, and those with dissection of the descending aorta<sup>[5-8]</sup>. Recognizing this differentiation, two commonly referenced classification systems, namely the DeBakey and Stanford classifications, emphasized the importance of distinguishing between these two types of dissection<sup>[6,9]</sup>. These schemes have been widely adopted and are generally well understood, particularly in the context of determining whether open surgical repair or conservative medical therapy is necessary. However, it is important to note that these classification systems were developed before the advent of advanced diagnostic imaging techniques and during a time of limited therapeutic options. Historically, these systems relied on identifying the anatomical location of intimal entry tears and the longitudinal extent of the dissection flap. The original DeBakey classification categorized aortic dissection based on anatomical features, while the more widely embraced Stanford classification simplified this approach. However, the Stanford classification lacks characterization of the distal extent of dissection, and neither system addresses aortic dissections originating in the arch. Recognizing these shortcomings, novel classification systems have recently emerged to delineate aortic arch involvement more precisely, and to provide a more nuanced description of the condition.

Similarly, in the past, aortic dissections were divided into acute (occurring within 2 weeks of the onset of symptoms) and chronic (occurring more than 2 weeks after symptom onset). Additionally, acute type B aortic dissections (aTBAD) were traditionally classified as complicated or uncomplicated; major complications included impending rupture and malperfusion. Distinguishing between complicated and uncomplicated aTBAD holds significance in terms of prognosis, with reported in-hospital mortality rates of 18%-50% for complicated cases compared to only 1.2%-10% for uncomplicated cases<sup>[10-12]</sup>. Aortic rupture is associated with high mortality regardless of the treatment type, while visceral ischemia, present in 7%-8% of aTBAD cases, is another devastating complication and the leading cause of death, accounting for 15% of mortality<sup>[13-15]</sup>. Therefore, timely diagnosis is crucial to improve survival chances.

In terms of clinical management, traditional strategies involving surgical intervention and medical therapy dictate distinct approaches for complicated and uncomplicated aTBAD cases. However, advancements in our understanding of the disease and the emergence of endovascular techniques have sparked a re-evaluation and debate regarding all aspects of the previously used TBAD classification. This includes a rethinking of what constitutes an “uncomplicated” patient with aTBAD. As a result, numerous position papers have been published, and new classifications have recently been proposed to better evaluate therapeutic options and predict outcomes. The Society for Vascular Surgery/Society of Thoracic Surgeons (SVS/STS) has put forth a different temporal classification for aortic dissection, which is outlined in [Table 1](#)<sup>[16]</sup>.

The TEM classification, introduced by the group of Czerny, provides a detailed description of aortic dissection based on the Type of dissection (T), the location of the Entry tear (E), and the presence of Malperfusion (M). This classification is outlined in [Table 2](#)<sup>[17]</sup>. In addition to the widely recognized Type A and Type B dissections, the TEM classification introduces a distinct category known as “non-A-non-B”. This category describes a dissection with involvement of the proximal aortic arch but without extension into the ascending aorta.

Recently, Lombardi *et al.* introduced a new classification in the SVS reporting standards for TBAD. This classification considers clinical signs and radiographic features to divide patients with aTBAD into three categories: uncomplicated, complicated, and high-risk. The details of this classification are outlined in [Table 3](#)<sup>[16]</sup>.

The new classification proposed by Lombardi *et al.* incorporates the clinical presentation of patients. In this classification, uncomplicated patients are characterized by the absence of rupture and/or end-organ malperfusion. On the other hand, complicated patients are those who exhibit rupture and/or malperfusion affecting organs such as the kidneys, viscera, limbs, or spinal cord. It should be noted that aTBAD without immediate rupture or malperfusion may still pose a significant risk of subsequent complications. Factors such as aortic diameter exceeding 40 mm, diameter of the proximal entry tear exceeding 1 cm, as well as clinical features like persistent pain and hypertension, are now recognized as potential causes of late complications. As a result, a new group has been introduced in the classification called “high-risk” patients, who exhibit these specific characteristics<sup>[16]</sup> [[Figure 1](#)].

A significant update introduced by the new classification proposed by Lombardi *et al.* is the incorporation of a modified anatomical classification. This classification considers the location of the proximal entry tear, as well as the proximal and distal extensions of the dissection. While this classification offers exceptional accuracy in describing the anatomical characteristics of the dissection, its adoption in urgent or emergent settings remains limited due to the preference for simpler classifications<sup>[16]</sup>.

**Table 1. SVS/STS chronicity classification for AD<sup>[16]</sup>**

Chronicity classification	Time from symptoms onset
Hyperacute	< 24 h
Acute	1-14 days
Subacute	15-90 days
Chronic	> 90 days

**Table 2. The TEM classification of AD<sup>[17]</sup>**

<b>T - type</b>	A	Involvement of the ascending aorta with or without extension into the aortic arch and the descending aorta
	B	Involvement of the descending aorta without extension into the aortic arch or the ascending aorta
	non-A-non-B	Involvement of the aortic arch with or without extension into the descending aorta, and without extension into the ascending aorta
<b>E - entry</b>	E0	No detectable entry tear
	E1	Entry tear in the ascending aorta
	E2	Entry tear between the innominate artery (proximal edge) and the left subclavian artery (LSA) (distal edge)
	E3	Entry tear distal to the LSA
<b>M - malperfusion*</b>	M0	No malperfusion
	M1	Dissection of at least one main coronary artery
	M2	Dissection of at least one supra-aortic vessel
	M3	Dissection of at least one visceral, renal or iliac vessel

\*Malperfusion is also described with plus (+) or minus (-): (+) if the malperfusion is associated with clinical symptoms, and (-) if it is not.

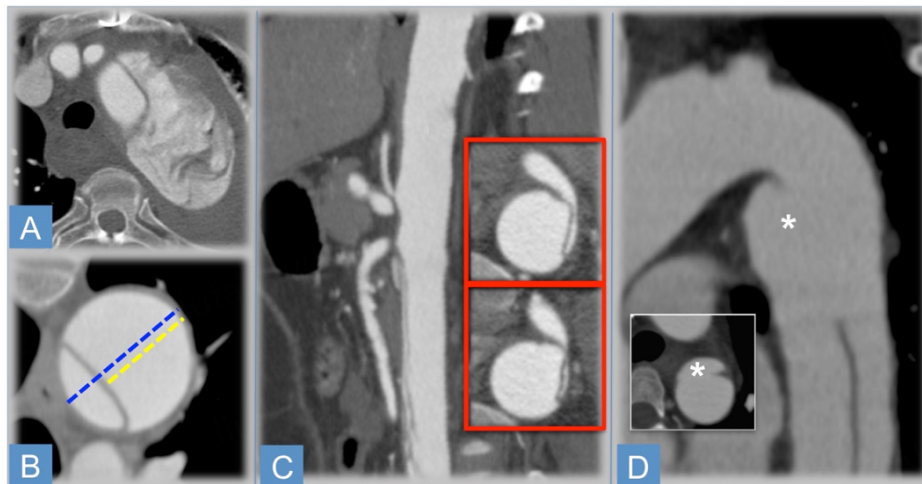
**Table 3. SVS/STS acuity classification for AD<sup>[16]</sup>**

<b>Uncomplicated</b>	No rupture
	No malperfusion
	No high-risk features
<b>High-risk</b>	Refractory pain
	Refractory hypertension
	Bloody pleural effusion
	Aortic diameter > 40 mm
	Radiographic only malperfusion
	Readmission
	Entry tear: lesser curve location
<b>Complicated</b>	False lumen diameter > 22 mm
	Rupture
	Malperfusion

In this dynamic and evolving landscape, the primary goals of treatment for patients with aTBAD are to ensure or restore organ perfusion and prevent aortic rupture. The consensus is that patients with uncomplicated aTBAD can be managed conservatively, while those with complicated aTBAD require immediate non-conservative interventions. However, the issue of whether to pursue invasive preventive treatment for uncomplicated patients deemed “high risk” to mitigate the potential for late complications remains a subject of debate. Different perspectives exist on this matter within the medical community.

## MANAGEMENT

Medical management for patients with aTBAD focuses on pain relief and reducing systolic blood pressure



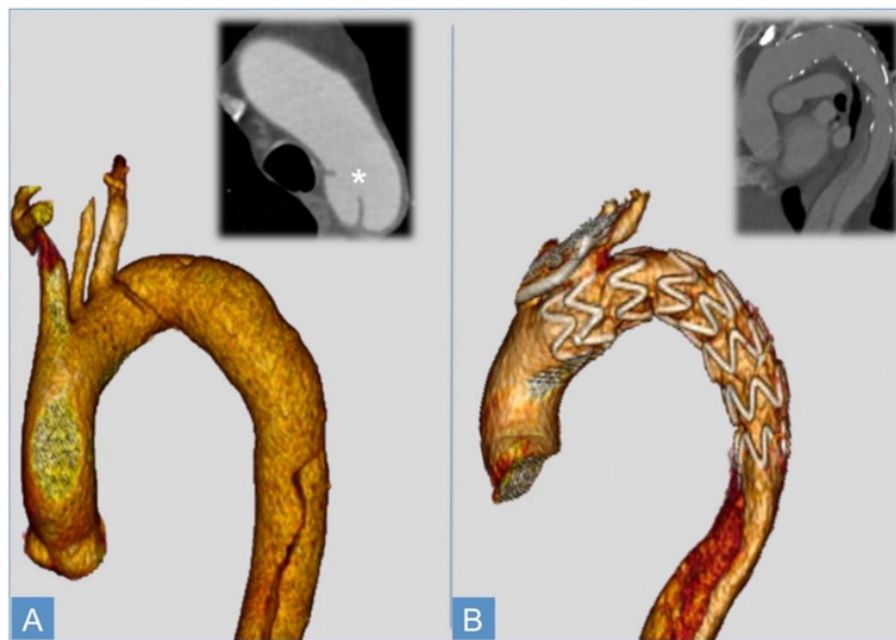
**Figure 1.** Radiographic characteristics of “high-risk” patients in accordance with the SVS reporting standards for TBAD<sup>[16]</sup>. (A) Presence of hemorrhagic pleural effusion. (B) Diameter of the false lumen exceeding 22 mm and total aortic diameter (TL + FL) exceeding 40 mm. (C) Radiographic evidence of malperfusion. (D) Inner curvature location of the entry tear with a tear diameter larger than 1 cm (\*).

to a range below 100–120 mmHg while maintaining a low heart rate. The use of narcotics,  $\beta$  blockers, diuretics, calcium blockers, and angiotensin-converting enzyme inhibitors is common in this context<sup>[18]</sup>.

In the case of complicated aTBAD, a non-conservative approach is typically required and should be viewed as a life-saving procedure. In the past, the surgical approach involving thoracotomy and surgical replacement of the thoracic aorta was considered the standard, but it carried high rates of morbidity and mortality. However, with the advancements in endovascular techniques, thoracic endovascular aortic repair (TEVAR) has progressively replaced the surgical approach and is now regarded as the first-line treatment for patients with complicated aTBAD<sup>[19,20]</sup>. Although preoperative planning primarily relies on preoperative Angio-CT scans, intraoperative tools such as intravascular ultrasound (IVUS) can provide supplementary information during the procedures.

In individuals experiencing complicated aTBAD, the foremost aim of TEVAR is to address the proximal entry tear, excluding completely the ruptured segment in case of rupture, and restore proper blood flow to affected organs in cases of malperfusion. By excluding the dissected aortic segment with TEVAR, there is a potential for inducing thrombosis within the false lumen (FL) and even facilitating remodeling of the aorta<sup>[21]</sup>. Encouraging outcomes have been reported in patients with complicated aTBAD who have undergone TEVAR<sup>[22,23]</sup> [Figure 2].

However, TEVAR alone may not always be sufficient to address associated malperfusion, and it often fails to completely exclude the FL, leading to potential late dilatation<sup>[24]</sup>. In cases of renal and visceral static malperfusion, additional stenting of branch vessels may be necessary<sup>[25]</sup>. Another option for persistent dynamic malperfusion after TEVAR is the Provisional Extension To Induce Complete Attachment Technique (PETTICOAT), which involves using a proximal stent graft along with distal expansion of the true lumen (TL) using bare metal stents<sup>[26]</sup>. However, a limitation of this technique is the ongoing perfusion of the FL, with a notable incidence of late aortic dilatation<sup>[27]</sup>.

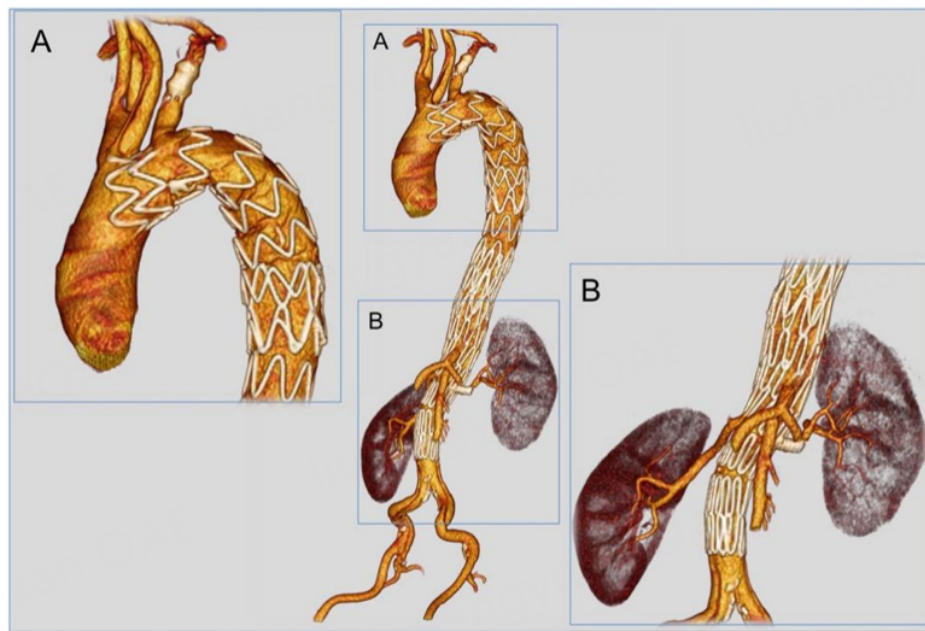


**Figure 2.** (A) Acute Type B aortic dissection depicted in the 3D volume rendering of an angio-CT scan. The axial scan highlights the proximal entry tear (\*). (B) Postoperative 3D volume rendering and multiplanar reconstruction after treatment. The patient underwent an endovascular approach, specifically TEVAR, to cover the proximal entry tear, which involved intentional coverage of the left subclavian artery (LSA).

An alternative treatment option is the Stent-Assisted Balloon-Induced Intimal Disruption and Relamination in Aortic Dissection Repair (STABILISE) technique<sup>[28]</sup>. This technique involves deploying a proximal covered stent-graft to cover the proximal dissection entry tear, with a graft oversizing of less than 10% compared to the non-dissected proximal landing zone. A second distal covered stent-graft may be deployed in the descending thoracic aorta, landing just above the origin of the celiac trunk with significant overlap with the proximal component. Additional aortic bare metal stents are then deployed distal to the covered stent-grafts, covering the dissected thoracic and abdominal aortic segments, with at least one stent overlapping proximally. The diameter of the bare stents is chosen to be equal to or greater than the total aortic diameter (TL + FL) at that level. A compliant balloon is used to selectively dilate only the covered stent-grafts, inducing rupture of the intimal lamella and achieving aortic relamination in the descending thoracic segment and complete obliteration of the FL without the risk of over-dilatation. Non-compliant balloon dilation is then used to dilate the aortic bare metal stents, not exceeding the total aortic diameter (TL + FL) at that level, to induce rupture of the intimal lamella and achieve relamination at the distal level, minimizing the risk of aortic rupture. Balloon dilations are continuously monitored radiologically and, when possible, using transesophageal echocardiography (TEE). If any vessels arise from the FL, they are catheterized before balloon dilation from the TL through the fenestrations in the lamella. After completing the STABILISE technique, a bare or covered stent may be deployed to optimize alignment between the fenestration in the lamella and the target vessel<sup>[28,29]</sup> [Figure 3].

While several authors have reported favorable results with the STABILISE technique, it falls outside the approved instructions for the use of bare metal stents, and larger sample sizes and longer follow-ups are needed to establish the safety and effectiveness of this approach. Therefore, in 2018, an international, multicenter, non-randomized observational registry was initiated to gather data on patients with acute/subacute Type B AD treated using the STABILISE technique, and this registry is still ongoing.

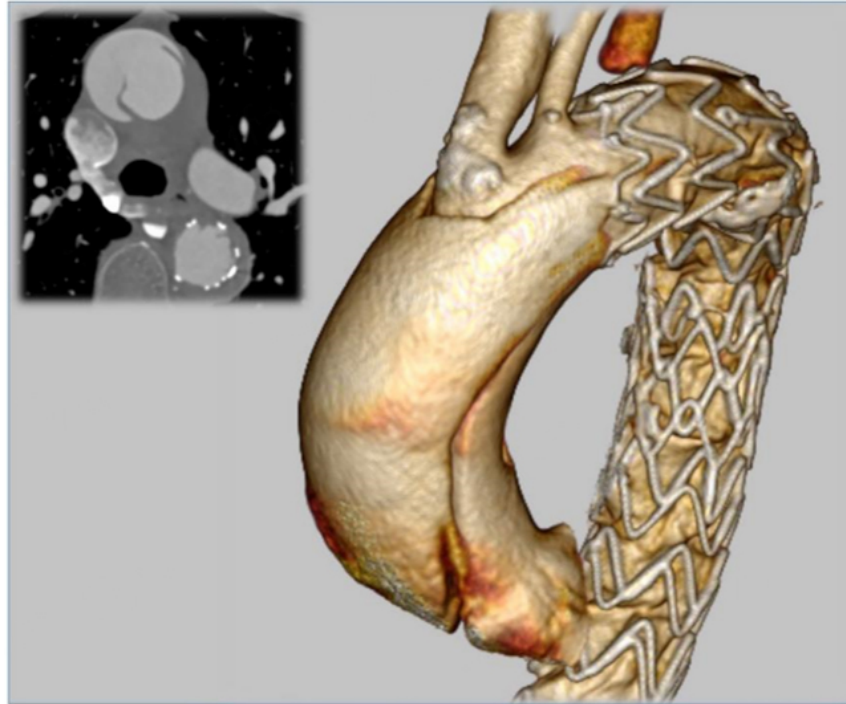




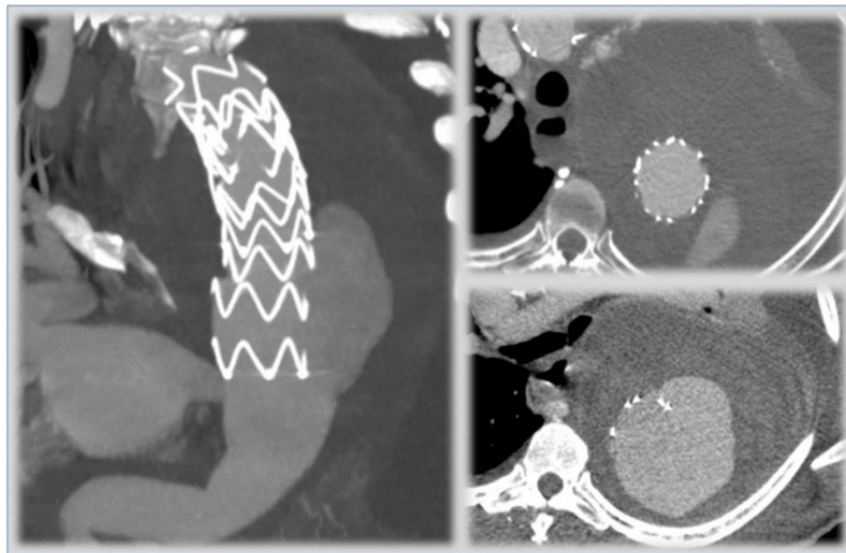
**Figure 3.** Angio-CT (3D volume rendering) after applying the STABILISE technique to treat aTBAD. (A) A stent-graft was deployed, intentionally covering the LSA. Additionally, a left carotid-to-subclavian bypass was performed, along with endovascular exclusion of the proximal LSA using a plug. (B) Distal to the stent-graft, two additional bare-metal aortic stents were deployed and expanded to recreate a “single-channelled” aorta. The left renal artery was also stented.

Additional techniques have been proposed to achieve complete occlusion of the FL during endovascular treatment for aortic dissection (AD), such as the Knickerbocker and Candy-Plug techniques<sup>[30,31]</sup>. The Knickerbocker technique entails the placement of an oversized tubular stent-graft within the true lumen, followed by the rupture of the dissection membrane using a compliant balloon positioned at the midpoint of the graft. This action facilitates the expansion of the graft's midsection into the false lumen, effectively halting retrograde flow. Conversely, the Candy-plug technique involves deploying a modified occlusive thoracic stent-graft into the false lumen to block distal backflow. Initially, this method involved adapting standard off-the-shelf thoracic devices, but now, manufacturers produce custom-made devices tailored to individual patient needs. In critical scenarios like rupture, where complete exclusion of the false lumen is imperative and unattainable through standard TEVAR or TEVAR with systems for false lumen exclusion, a more comprehensive approach using branched and fenestrated devices (F/B-EVAR) may be considered. However, employing F/B-EVAR in acute cases presents challenges, including a heightened risk of spinal cord ischemia due to extended aortic exclusion. Moreover, technical hurdles may arise from the narrow true lumen and associated complexities in visceral bridging. Additionally, the unavailability of custom-made devices in acute settings restricts treatment options to off-the-shelf devices only. Consequently, the application of F/B-EVAR in treating acute type B aortic dissections is constrained.

However, it is important to note that certain complications may arise following thoracic endovascular aortic repair (TEVAR) in patients with aTBAD, including retrograde Type A AD (RAD) and stent-graft induced new entry tears (SINE)<sup>[11]</sup> [Figures 4 and 5]. Therefore, meticulous procedural planning is crucial, considering factors such as the type of graft (radial force, proximal barbs), appropriate oversizing, and adequate aortic length coverage. Given the specific frailty of the aortic wall in aTBAD patients, a reduced proximal oversizing of less than 10%, stent-grafts with low radial force and without barbs are generally preferred in this context, while extensive ballooning of the landing zones is discouraged<sup>[32]</sup>.



**Figure 4.** Angio-CT (3D volume rendering) of a patient experiencing retrograde aortic dissection following TEVAR for aTBAD. The dissection originates clearly from the proximal stent and involves the ascending aorta.



**Figure 5.** Angio-CT scan of a patient with stent-graft-induced new entry tear (SINE) after TEVAR for TBAD. The SINE originates from the distal portion of the stent-graft, resulting in reperfusion of the false lumen and its enlargement.

In the case of uncomplicated aTBAD, particularly in the “high-risk” patient group, the use of pre-emptive TEVAR as a preventive measure to reduce the risk of late complications, instead of relying solely on medical therapy, is an intuitive approach. However, further research and randomized trials are necessary to assess its efficacy.



Randomized trials, such as the INSTEAD-XL study, have reported higher rates of thrombosis in the FL during follow-up in uncomplicated patients who underwent TEVAR compared to those who received medical therapy alone. Additionally, there was a decrease in aortic-related mortality in the TEVAR group<sup>[33]</sup>. Despite these findings, the use of pre-emptive TEVAR in uncomplicated aTBAD remains a subject of debate and controversy.

### **IMH - intramural hematoma**

Intramural hematoma (IMH) is characterized by the presence of blood within the aortic wall. It is believed to occur as a result of ruptured vasa vasorum within the tunica media or as a consequence of aortic dissection (AD) with thrombosis of the FL and without a detectable entry tear<sup>[34,35]</sup>. IMH has been described in the literature as a precursor or even a subtype of AD, indicating a close relationship between the two conditions<sup>[36,37]</sup>.

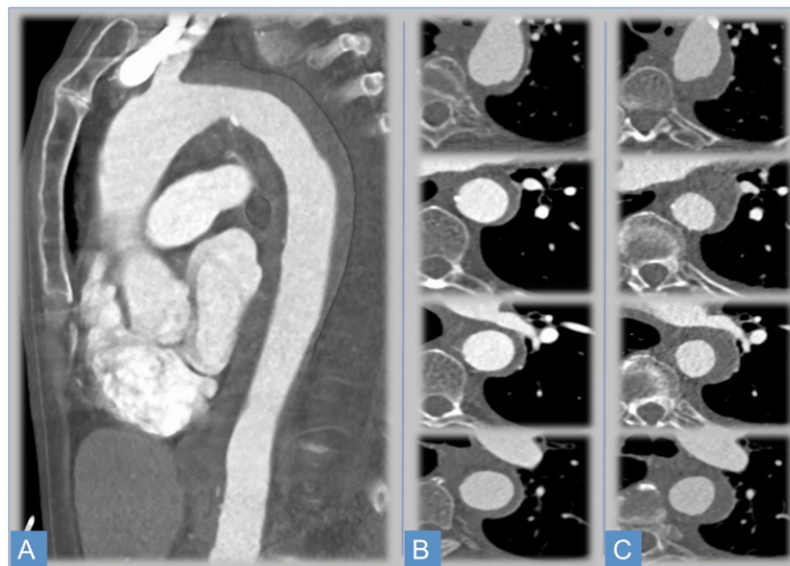
Similar to AD, IMH is also classified using the Stanford classification system. Type A IMH refers to cases where the hematoma is localized in the ascending aorta, while type B IMH involves the aortic arch and/or the descending thoracic aorta (DTA) without the involvement of the ascending aorta.

### *Management*

In cases of Intramural Hematoma (IMH), it is possible to refer to the guidelines for aortic dissection (AD) for the corresponding segment of the aorta. Type A IMH is considered a cardio-thoracic emergency and requires prompt open ascending aortic repair<sup>[38,39]</sup>. On the other hand, Type B IMH can be either uncomplicated or complicated. A complicated IMH is characterized by refractory chest and/or back pain, progressive hematoma expansion, pleural effusion, or aortic rupture<sup>[36,37]</sup>.

Patients with uncomplicated Type B IMH are typically managed medically, following a similar approach as with aTBAD. However, in cases of complicated Type B IMH, a non-conservative management strategy is recommended. Currently, endovascular repair with thoracic endovascular aortic repair (TEVAR) is preferred over open surgery for patients with favorable anatomy<sup>[40]</sup>. Many authors suggest complete coverage of the IMH lesion with the stent-graft, ensuring an ideal > 20 mm proximal and distal landing zone of healthy aorta. A maximal oversize of 10% is commonly advocated<sup>[41-44]</sup>. However, some authors propose a less extensive aortic coverage, with incomplete distal coverage of IMH, particularly in patients with large patent critical intercostal arteries<sup>[42]</sup>. IMH usually do not extend below the diaphragmatic aorta, and adjunctive procedures for visceral branch involvement may be less necessary compared to AD. It is important to note that patients with IMH exhibit a similar aortic frailty as seen in AD, and complications such as retrograde Type A AD (RAD) and stent-graft induced new entry tears (SINE) may also occur in this setting. Therefore, a cautious approach is advised during procedural planning, including reduced oversizing, avoidance of ballooning, and the use of stent-grafts without barbs and low radial force<sup>[36,37]</sup>.

Notably, medical therapy alone has shown high failure rates in the treatment of IMH, with only 10% of patients experiencing regression. IMH frequently evolves into classic AD (28%-47%) or aortic rupture (20%-45%)<sup>[42]</sup>. Several radiological signs have been identified as potential predictors of failure of medical therapy, such as IMH thickness > 8 mm and aortic diameter > 55 mm. In these cases, many authors recommend considering endovascular management, even in the absence of complications<sup>[42,45]</sup>. Close imaging surveillance using magnetic resonance imaging (MRI) or computed tomography (CT) is therefore recommended [Figure 6].



**Figure 6.** Imaging of a patient with intramural hematoma (IMH). (A) Multiplanar reconstruction revealing the longitudinal extension of the IMH from the LSA to the visceral aorta. (B) Axial images taken a few hours after the initial onset of thoracic and back pain. (C) Subsequent imaging after 48 hours documenting a progressive thickening of the IMH.

Recent updates concerning the management of patients with intramural hematoma (IMH) have been outlined in the 2022 ACC/AHA guidelines, emphasizing the significance of “high-risk features” associated with IMH<sup>[46]</sup>.

#### PAU - penetrating aortic ulcer

Penetrating Aortic Ulcer (PAU) refers to an ulcerating atherosclerotic lesion that starts in the intima and extends through the internal elastic lamina into the media of the aortic wall. It is classified as a degenerative aortic disease and is more commonly observed in elderly patients<sup>[47]</sup>. In the early stages, the lesion is limited to the intima and often remains asymptomatic. However, as it progresses into the media, it can cause thoracic pain. It is important to note that a significant number of patients with PAU may remain asymptomatic, and these lesions are frequently discovered incidentally.

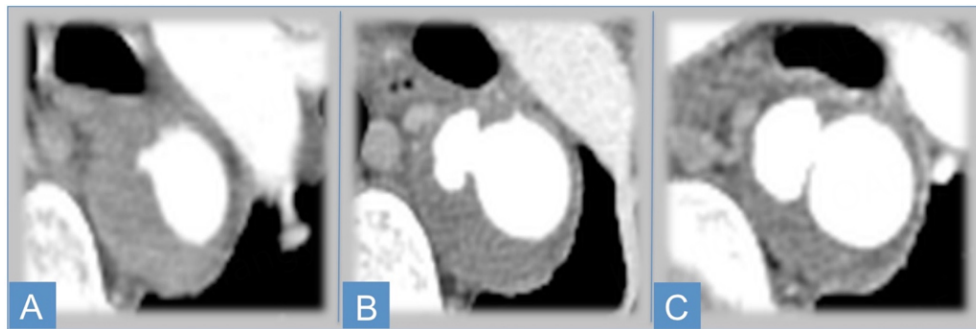
The natural history and evolution of PAU are not yet fully understood, but there is a general consensus that the risk of rupture is high<sup>[48]</sup> [Figure 7]. Rupture of a PAU can lead to life-threatening complications, such as aortic dissection or intramural hematoma. Therefore, prompt diagnosis and appropriate management are essential in patients with suspected or confirmed PAU.

#### Management

PAUs can be classified according to the Stanford classification system, similar to aortic dissections. Type A PAUs involve the ascending aorta, while type B PAUs involve the aortic arch and/or the DTA.

For patients with type A PAUs, open surgical repair is generally recommended due to the involvement of the ascending aorta. This approach allows for direct access to the lesion and provides the opportunity for complete excision or reconstruction.

In the case of complicated type B PAUs, which include symptomatic PAUs, PAUs with signs of aortic rupture, or those with a diameter greater than 20 mm and/or depth greater than 10 mm, a non-conservative



**Figure 7.** PAU (Penetrating Aortic Ulcer) may exhibit rapid progression. (A) Initial imaging conducted shortly after the sudden onset of thoracic pain showing an ulcerating lesion on the aortic wall at the level of the descending thoracic aorta. Subsequent angio-CT scans performed at 48 h (B) and 72 h (C) demonstrate progressive expansion of the ulcerating aortic lesion.

management approach is indicated. The current guidelines, such as the 2017 European Society for Vascular Surgery guidelines, recommend endovascular repair with TEVAR when anatomically feasible<sup>[49-51]</sup>.

During the endovascular procedure, an appropriate oversizing of the stent graft is generally applied, similar to the management of degenerative aortic aneurysms. The degree of oversizing usually falls within the range of 10% to 20%, depending on individual patient factors and the characteristics of the PAU.

The management of asymptomatic patients with type B PAUs is still a topic of debate. Currently, conservative management through pharmacological treatment and close follow-up with CT imaging is often employed. However, recent studies have reported higher morbidity and mortality rates in symptomatic patients treated with TEVAR compared to asymptomatic patients<sup>[52]</sup>. Further research is needed to better understand the optimal management strategy for asymptomatic PAUs.

#### **bTAI - blunt traumatic thoracic aortic injury**

Although many authors do not classify blunt traumatic aortic injuries (bTAI) as AAS, this review encompasses them to provide a comprehensive overview of diseases involving the descending thoracic aorta with acute onset. bTAI typically occurs as a result of deceleration accidents, impact collisions, or vertical falls, which subject the aorta to shear and stretch forces. These injuries are associated with high mortality rates, ranging from 80% to 90%<sup>[53-55]</sup>.

The classification proposed by Azizzadeh *et al.* categorizes bTAI into four grades based on the extent of the injury:

1. Grade I: Intimal tear - This refers to a tear in the innermost layer of the aortic wall, the intima.
2. Grade II: Intramural hematoma - In this grade, blood accumulates within the aortic wall, causing separation and disruption of the layers.
3. Grade III: Pseudoaneurysm - A pseudoaneurysm is a contained rupture of the aortic wall, forming a sac filled with blood.
4. Grade IV: Rupture - This grade indicates a complete rupture of the aortic wall, leading to uncontrolled bleeding.

These grades represent a spectrum of severity, with Grade I being the least severe and Grade IV being the most severe and life-threatening<sup>[56]</sup>. Early recognition and appropriate management are crucial in bTAI cases due to the high mortality rates associated with these injuries. Prompt diagnosis is necessary to improve the patient's chances of survival.

### *Management*

Endovascular repair with TEVAR has become the preferred treatment approach for patients with acute bTAI. TEVAR has shown lower early and late morbidity and mortality rates compared to open surgical repair<sup>[57,58]</sup>. However, there are certain considerations to keep in mind during the endovascular management of bTAI. One critical aspect is the selection of the appropriate stent-graft. In patients with severe hypotension, standard 10%-20% stent-graft oversizing based on preoperative CT imaging may not be sufficient, increasing the risk of type I endoleak. Additionally, patients with bTAI are often at a young age, and have aortic diameter variations during the cardiac cycle, and angled aortic arches, which can result in proximal inadequate apposition of the stent-graft (bird's beak effect) and subsequent potential complications, such as graft collapse<sup>[59-61]</sup>. Careful procedure planning is necessary to address these challenges and avoid complications. In this scenario, intra-vascular ultrasound (IVUS) can be a valuable adjunctive tool during the procedure, providing real-time measurements of the aorta before and after stent-graft deployment<sup>[62]</sup>.

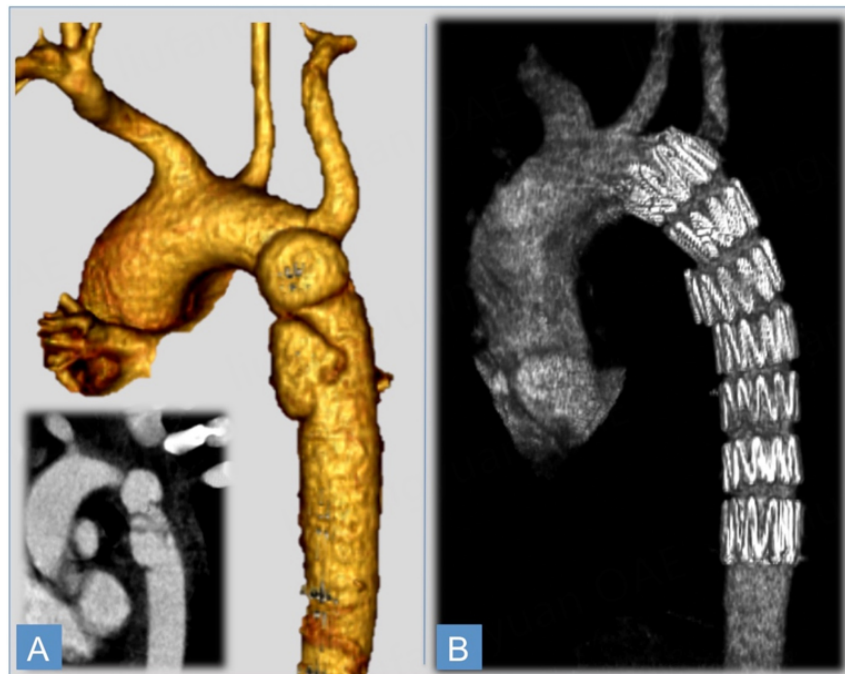
To achieve an adequate proximal sealing zone, intentional coverage of the left subclavian artery (LSA) may be necessary, especially in patients with isthmus aortic lesions. The RESCUE trial, which evaluated TEVAR in patients with bTAI, reported that among the treated patients, some required complete LSA coverage, while others required partial LSA coverage. The trial demonstrated that these patients did not experience strokes or spinal cord injuries, and only a small number required LSA revascularization<sup>[63]</sup>.

It is important for the treatment team to carefully assess the individual patient's anatomy, hemodynamic status, and potential risks in order to plan the most appropriate and effective endovascular repair strategy for bTAI [Figure 8].

The indications for treatment and the timing of intervention in patients with blunt traumatic thoracic aortic injuries (bTAI) are still debated. Recent evidence suggests a more conservative approach for patients with lower grades of aortic injuries, specifically grade I and II<sup>[59,64]</sup>. A study published by the Aortic Trauma Foundation Global Registry supports the use of medical therapy alone as the definitive treatment in patients with grade I and II bTAI. The registry reported low overall intervention rates and no aortic-related deaths in this group of patients<sup>[65]</sup>.

Regarding the timing of intervention, a recent study analyzed the association between mortality and the timing of repair. The study found that patients who underwent repair after 24 h had better survival outcomes compared to those who underwent earlier intervention. This suggests that a delay in intervention may be beneficial in terms of mortality<sup>[66]</sup>.

It is important to consider individual patient factors, such as the severity of the aortic injury, hemodynamic stability, associated injuries, and the overall clinical status, when determining the appropriate treatment approach and timing for bTAI. A multidisciplinary approach involving vascular surgeons, trauma surgeons, and other specialists is crucial in making these decisions and optimizing patient outcomes.



**Figure 8.** (A) Angio-CT scan of a patient with blunt traumatic aortic injury (bTAI) and aortic rupture at the isthmus level. (B) The patient underwent TEVAR, which involved excluding the false aneurysm and intentionally covering the LSA.

#### **rDTA - ruptured aneurysm of the descending thoracic aorta**

A ruptured descending thoracic aortic aneurysm (rDTA) is a serious and potentially life-threatening condition. If left untreated, it can lead to high mortality rates. Endovascular repair with TEVAR has emerged as the preferred treatment approach for rDTA, when feasible. TEVAR involves the placement of a stent graft within the diseased segment of the aorta, providing a new pathway for blood flow and sealing off the rupture. This minimally invasive technique has shown favorable outcomes in terms of 30-day morbidity and mortality rates compared to open surgical repair [Figure 9]<sup>[67]</sup>.

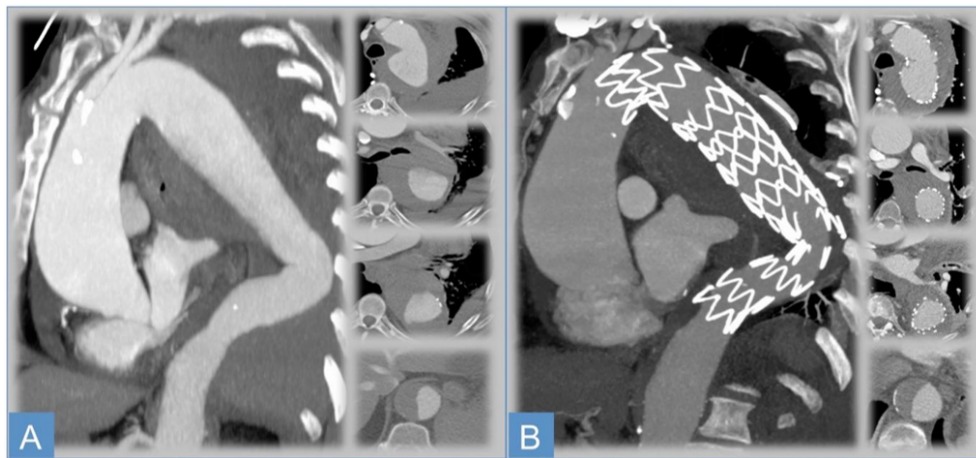
TEVAR offers several advantages over open surgery; however, it is important to note that not all rDTA cases are suitable for TEVAR, as the anatomical characteristics of the rupture and the patient's overall condition must be carefully evaluated, and thus, in some cases, open surgical repair may still be necessary.

#### *Management*

Some of the most important considerations regarding the endovascular management of rDTA may be summarized in the following key points: Stent-Graft Oversizing: Avoiding stent-graft oversizing of less than 15%-20% is crucial, especially in patients with hypotensive shock, to prevent potential under-sizing and ensure optimal sealing<sup>[68]</sup>.

Proximal Extension: In certain scenarios, achieving endovascular exclusion of the aneurysm may necessitate proximal extension with coverage of the left subclavian artery (LSA). However, the decision to cover the LSA remains contentious, and the necessity for revascularization should be meticulously assessed on a case-by-case basis<sup>[69]</sup>.





**Figure 9.** (A) Angio-CT scan of a patient with ruptured dissecting thoracic aneurysm (rDTA) and extensive hemothorax. (B) The patient received TEVAR treatment to exclude the aneurysm. Intentional coverage of the LSA was performed to achieve proper proximal sealing.

**Distal Landing Zone and Visceral Vessels:** When the aneurysm extension involves visceral vessels, distal extension with coverage of the celiac trunk (CT) may be required for endovascular exclusion. This approach enables patient stabilization and deferral of more intricate procedures. While the safety and efficacy of CT coverage during thoracic endovascular aortic repair (TEVAR) have been demonstrated, a thorough evaluation of collateral circulation is imperative. In instances where collateral circulation appears insufficient during temporary CT occlusion, additional techniques such as chimney grafts, periscopes, or physician-modified stent-grafts may be considered<sup>[70,71]</sup>.

**Off-the-Shelf Multibranched Stent-Grafts:** In urgent or emergent situations where custom-made devices are unavailable, off-the-shelf multibranched stent-grafts may serve as a viable option for patients with rDTA involving the thoraco-abdominal aorta. However, this approach necessitates a more intricate endovascular procedure, sufficient material availability, and expertise. Studies have indicated acceptable morbidity and mortality rates with off-the-shelf multibranched stent-grafts, even for ruptured aortic aneurysms<sup>[72]</sup>.

**Individualized Decision-Making:** The management of rDTA demands personalized decision-making based on the patient's clinical status, anatomical considerations, resource availability, and the proficiency of the medical team.

### **Multidisciplinary team-led management**

Recent evidence underscores the indispensable role of multidisciplinary team-led management in the treatment of acute aortic syndromes involving the descending thoracic aorta. Collaborative efforts among cardiovascular surgeons, interventional radiologists, cardiologists, anesthesiologists, and other specialists are paramount for optimizing patient outcomes<sup>[73]</sup>. This approach allows for comprehensive evaluation, precise diagnosis, and tailored treatment strategies. Studies have shown that multidisciplinary teams lead to more accurate and timely diagnoses, reducing the risk of misdiagnosis or delayed intervention. Moreover, the complexity of acute aortic syndromes often necessitates a multifaceted treatment approach, incorporating different techniques. Multidisciplinary teams facilitate seamless coordination between different specialties, ensuring that patients receive the most appropriate and effective therapies. Furthermore, they enable ongoing monitoring and follow-up, essential for long-term management and prevention of recurrence. Overall, the recent evidence highlights the crucial role of multidisciplinary team-led management in

providing comprehensive, patient-centered care for acute aortic syndromes involving the descending thoracic aorta, ultimately improving outcomes, and reducing morbidity and mortality associated with these life-threatening conditions.

### **Brief general considerations about the endovascular approach in patients with AAS**

TEVAR, particularly in cases involving extensive aortic coverage, carries an inherent risk of spinal cord ischemia (SCI) stemming from the occlusion of a high number of intercostal arteries. When treating patients with AAS, in urgent or emergent settings, the incidence of this severe complication may escalate compared to elective procedures. Factors contributing to this heightened risk in acute cases include hemodynamic instability, longer aortic coverage for effective disease exclusion, non-staged procedures, and limitations in the usage of the available adjuncts for spinal cord protection. While many risk factors are non-modifiable preoperatively, it is crucial to emphasize vigilant patient monitoring and prompt application of available tools to prevent or mitigate this life-threatening event as swiftly as possible<sup>[74-77]</sup>.

Another important aspect to consider is the incidence of genetically triggered aortic disorders in patients with AAS, as these conditions inherently elevate the risks of aTBAD, IMH, and aortic rupture in general. While open surgical approaches may offer superior long-term outcomes in patients with collagenopathies, TEVAR presents advantages in urgent or emergent scenarios due to its reduced invasive nature. Consequently, TEVAR is commonly accepted, albeit sometimes viewed as a bridging procedure for stabilizing patients. For individuals with genetically triggered aortic disorders undergoing endovascular treatment, rigorous follow-up is imperative to promptly detect, and address, potential complications, including those requiring surgical intervention<sup>[78]</sup>.

## **CONCLUSIONS**

The advent of endovascular treatments has marked a significant advancement in the management of acute aortic syndromes involving the descending thoracic aorta, often demonstrating superior outcomes compared to traditional open surgical repair. Nonetheless, it is imperative to acknowledge the inherent risks and potential complications associated with endovascular techniques, despite their overarching benefits. Consequently, a meticulous patient selection process is essential to discern the optimal candidates for endovascular intervention versus those better suited for medical therapy alone.

Vascular surgeons play a pivotal role in this decision-making process, requiring a nuanced understanding of available therapeutic modalities and the ability to tailor treatment strategies to individual patient profiles and disease presentations. As the landscape of acute aortic syndrome management continues to evolve, ongoing research efforts and technological advancements are paramount to further enhancing patient outcomes and addressing the complexities inherent in these conditions.

Through continuous learning and refinement of techniques, the medical community can progressively confront and mitigate the challenges posed by acute aortic syndromes, thereby rendering them less formidable adversaries. Therefore, it is incumbent upon medical professionals to remain abreast of the latest advancements, guidelines, and evidence-based practices in AAS management to deliver optimal care to patients.

## **DECLARATIONS**

### **Authors' contributions**

Made substantial contributions to the conception and design of the study and performed data analysis and interpretation: Rinaldi E, Mascia D, Campesi C, Pizzutilli AC, Melissano G

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All authors declared that there are no conflicts of interest.

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**REFERENCES**

1. Vilacosta I, Aragoncillo P, Cañadas V, Román JAS, Ferreirós J, Rodríguez E. Acute aortic syndrome: a new look at an old conundrum. *Postgrad Med J* 2010;86:52-61. [DOI](#)
2. Ganaha F, Miller DC, Sugimoto K, et al. Prognosis of aortic intramural hematoma with and without penetrating atherosclerotic ulcer: a clinical and radiological analysis. *Circulation* 2002;106:342-8. [DOI](#)
3. Evangelista A, Czerny M, Nienaber C, et al. Interdisciplinary expert consensus on management of type B intramural haematoma and penetrating aortic ulcer. *Eur J Cardiothorac Surg* 2015;47:209-17. [DOI](#)
4. Bossone E, LaBounty TM, Eagle KA. Acute aortic syndromes: diagnosis and management, an update. *Eur Heart J* 2018;39:739-49d. [DOI](#) [PubMed](#)
5. Austen WG, Desanctis RW. Surgical treatment of dissecting aneurysm of the thoracic aorta. *N Engl J Med* 1965;272:1314-7. [DOI](#) [PubMed](#)
6. DeBakey ME, Henley WS, Cooley DA, Morris GC, Crawford ES, Beall AC. Surgical management of dissecting aneurysms of the aorta. *J Thorac Cardiovasc Surg* 1965;49:130-49. [DOI](#)
7. Beckwith J, Muller WH Jr, Warren WD. Problems in the surgical management of acute dissecting aneurysm of the aorta. *Ann Surg* 1956;144:530-48. [DOI](#) [PubMed](#) [PMC](#)
8. Jr MW, Palmer RF, Bartley TD, Seelman RC. Treatment of dissecting aneurysms of the aorta without surgery. *J Thorac Cardiovasc Surg* 1965;50:364-73. [DOI](#)
9. Daily PO, Trueblood HW, Stinson EB, Wuerflein RD, Shumway NE. Management of acute aortic dissections. *Ann Thorac Surg* 1970;10:237-47. [DOI](#) [PubMed](#)
10. Estrera AL, Miller CC 3rd, Safi HJ, et al. Outcomes of medical management of acute type B aortic dissection. *Circulation* 2006;114:1384-9. [DOI](#)
11. Howard C, Sheridan J, Picca L, et al. TEVAR for complicated and uncomplicated type B aortic dissection-Systematic review and meta-analysis. *J Card Surg* 2021;36:3820-30. [DOI](#)
12. Tsai TT, Trimarchi S, Nienaber CA. Acute aortic dissection: perspectives from the international registry of acute aortic dissection (IRAD). *Eur J Vasc Endovasc Surg* 2009;37:149-59. [DOI](#) [PubMed](#)
13. MacKenzie KS, LeGuillan MP, Steinmetz OK, Montreuil B. Management trends and early mortality rates for acute type B aortic dissection: a 10-year single-institution experience. *Ann Vasc Surg* 2004;18:158-66. [DOI](#) [PubMed](#)
14. Jonker FH, Patel HJ, Upchurch GR, et al. Acute type B aortic dissection complicated by visceral ischemia. *J Thorac Cardiovasc Surg* 2015;149:1081-6.e1. [DOI](#)
15. Hagan PG, Nienaber CA, Isselbacher EM, et al. The international registry of acute aortic dissection (IRAD): new insights into an old disease. *JAMA* 2000;283:897-903. [DOI](#)
16. Lombardi JV, Hughes GC, Appoo JJ, 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. [DOI](#)
17. Sievers HH, Rylski B, Czerny M, et al. Aortic dissection reconsidered: type, entry site, malperfusion classification adding clarity and enabling outcome prediction. *Interact Cardiovasc Thorac Surg* 2020;30:451-7. [DOI](#)
18. Estrera AL, Miller CC, Goodrick J, et al. Update on outcomes of acute type B aortic dissection. *Ann Thorac Surg* 2007;83:S842-5;discussion S846. [DOI](#)

19. Dake MD, Kato N, Mitchell RS, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. *N Engl J Med* 1999;340:1546-52. DOI
20. Zhang MH, Du X, Guo W, Liu XP, Jia X, Ge YY. Early and midterm outcomes of thoracic endovascular aortic repair (TEVAR) for acute and chronic complicated type B aortic dissection. *Medicine* 2017;96:e7183. DOI PubMed PMC
21. Alfson DB, Ham SW. Type B aortic dissections: current guidelines for treatment. *Cardiol Clin* 2017;35:387-410. DOI PubMed
22. Tsai TT, Fattori R, Trimarchi S, et al; International Registry of Acute Aortic Dissection. Long-term survival in patients presenting with type B acute aortic dissection: insights from the international registry of acute aortic dissection. *Circulation* 2006;114:2226-31. DOI
23. Harky A, Chan JSK, Wong CHM, Francis N, Grafton-Clarke C, Bashir M. Systematic review and meta-analysis of acute type B thoracic aortic dissection, open, or endovascular repair. *J Vasc Surg* 2019;69:1599-609.e2. DOI
24. Parker JD, Golledge J. Outcome of endovascular treatment of acute type B aortic dissection. *Ann Thorac Surg* 2008;86:1707-12. DOI PubMed
25. Elshra A, Kölbel T, Panuccio G, Rohlfßs F, Debus ES, Tsilimparis N. Endovascular therapy for nonischemic vs ischemic complicated acute type B aortic dissection. *J Endovasc Ther* 2020;27:145-52. DOI PubMed
26. Bertoglio L, Rinaldi E, Melissano G, Chiesa R. The PETTICOAT concept for endovascular treatment of type B aortic dissection. *J Cardiovasc Surg* 2019;60:91-9. DOI PubMed
27. Mascia D, Rinaldi E, Salvati S, et al. Thoracic endovascular aortic repair with additional distal bare stents in type B aortic dissection does not prevent long-term aneurysmal degeneration. *J Endovasc Ther* 2021;28:425-33. DOI
28. Kahlberg A, Mascia D, Bertoglio L, et al. New technical approach for type B dissection: from the PETTICOAT to the STABILISE concept. *J Cardiovasc Surg* 2019;60:281-8. DOI
29. Melissano G, Bertoglio L, Rinaldi E, et al. Satisfactory short-term outcomes of the STABILISE technique for type B aortic dissection. *J Vasc Surg* 2018;68:966-75. DOI
30. Rohlfßs F, Spanos K, Tsilimparis N, Debus ES, Kölbel T. Techniques and outcomes of false lumen embolization in chronic type B aortic dissection. *J Cardiovasc Surg* 2018;59:784-8. DOI PubMed
31. Yap KH, Tham YC, Tay KH, et al. Homemade candy plug using a zenith alpha thoracic stent-graft for false lumen distal occlusion in acute-on-chronic type B aortic dissection. *J Endovasc Ther* 2019;26:732-5. DOI
32. Burdess A, Mani K, Tegler G, Wanhainen A. Stent-graft induced new entry tears after type B aortic dissection: how to treat and how to prevent? *J Cardiovasc Surg* 2018;59:789-96. DOI PubMed
33. Nienaber CA, Kische S, Rousseau H, et al; INSTEAD-XL trial. Endovascular repair of type B aortic dissection: long-term results of the randomized investigation of stent grafts in aortic dissection trial. *Circ Cardiovasc Interv* 2013;6:407-16. DOI
34. Mukohara N. Intramural hematoma - contradiction to the theory of rupture of the vasa vasorum at onset. *Ann Thorac Cardiovasc Surg* 2014;20:949-50. DOI
35. Gutschow SE, Walker CM, Martínez-Jiménez S, Rosado-de-Christenson ML, Stowell J, Kunin JR. Emerging concepts in intramural hematoma imaging. *Radiographics* 2016;36:660-74. DOI PubMed
36. Uchida K, Imoto K, Karube N, et al. Intramural haematoma should be referred to as thrombosed-type aortic dissection. *Eur J Cardiothorac Surg* 2013;44:366-9. DOI
37. Song JK. Update in acute aortic syndrome: intramural hematoma and incomplete dissection as new disease entities. *J Cardiol* 2014;64:153-61. DOI PubMed
38. Chou AS, Ziganshin BA, Charilaou P, Tranquilli M, Rizzo JA, Elefteriades JA. Long-term behavior of aortic intramural hematomas and penetrating ulcers. *J Thorac Cardiovasc Surg* 2016;151:361-73.e1. DOI PubMed
39. Erbel R, Aboyans V, Boileau C, et al; ESC Committee for Practice Guidelines. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The task force for the diagnosis and treatment of aortic diseases of the European society of cardiology (ESC). *Eur Heart J* 2014;35:2873-926. DOI
40. Ince H, Nienaber CA. Diagnosis and management of patients with aortic dissection. *Heart* 2007;93:266-70. DOI PubMed PMC
41. Chen Q, Jiang D, Kuang F, Shan Z. The evolution of treatments for uncomplicated type B intramural hematoma patients. *J Card Surg* 2020;35:580-90. DOI
42. Mesar T, Lin MJ, Kabir I, Dexter DJ, Rathore A, Panneton JM. Medical therapy in type B aortic intramural hematoma is associated with a high failure rate. *J Vasc Surg* 2020;71:1088-96. DOI
43. Ye K, Qin J, Yin M, Jiang M, Li W, Lu X. Acute intramural hematoma of the descending aorta treated with stent graft repair is associated with a better prognosis. *J Vasc Interv Radiol* 2017;28:1446-53.e2. DOI
44. Bischoff MS, Meisenbacher K, Wehrmeister M, Böckler D, Kotelis D. Treatment indications for and outcome of endovascular repair of type B intramural aortic hematoma. *J Vasc Surg* 2016;64:1569-79.e2. DOI PubMed
45. Evangelista A, Maldonado G, Moral S, et al. Intramural hematoma and penetrating ulcer in the descending aorta: differences and similarities. *Ann Cardiothorac Surg* 2019;8:456-70. DOI PubMed PMC
46. Isselbacher EM, Preventza O, Hamilton Black J 3rd, et al; Peer Review Committee Members. 2022 ACC/AHA guideline for the diagnosis and management of aortic disease: a report of the american heart association/american college of cardiology joint committee on clinical practice guidelines. *Circulation* 2022;146:e334-482. DOI
47. Wada H, Sakata N, Tashiro T. Clinicopathological study on penetrating atherosclerotic ulcers and aortic dissection: distinct pattern of development of initial event. *Heart Vessels* 2016;31:1855-61. DOI
48. Bischoff MS, Geisbüsch P, Peters AS, Hyhlik-Dürr A, Böckler D. Penetrating aortic ulcer: defining risks and therapeutic strategies.

- Herz 2011;36:498-504. [DOI PubMed](#)
49. Eggebrecht H, Herold U, Schmermund A, et al. Endovascular stent-graft treatment of penetrating aortic ulcer: results over a median follow-up of 27 months. *Am Heart J* 2006;151:530-6. [DOI](#)
  50. Jánosi RA, Gorla R, Tsagakis K, et al. Thoracic Endovascular repair of complicated penetrating aortic ulcer: an 11-year single-center experience. *J Endovasc Ther* 2016;23:150-9. [DOI](#)
  51. Riambau V, Böckler D, Brunkwall J, et al. Editor's choice - management of descending thoracic aorta diseases: clinical practice guidelines of the European society for vascular surgery (ESVS). *Eur J Vasc Endovasc Surg* 2017;53:4-52. [DOI](#)
  52. Rokosh RS, Rockman CB, Patel VI, et al. Thoracic endovascular aortic repair for symptomatic penetrating aortic ulcers and intramural hematomas is associated with poor outcomes. *J Vasc Surg* 2021;74:63-70.e1. [DOI](#)
  53. Gaffey AC, Zhang J, Saka E, et al. Natural history of nonoperative management of grade II Blunt Thoracic Aortic Injury. *Ann Vasc Surg* 2020;65:124-9. [DOI](#)
  54. Parmley LF, Mattingly TW, Manion WC, Jahnke EJ Jr. Nonpenetrating traumatic injury of the aorta. *Circulation* 1958;17:1086-101. [DOI PubMed](#)
  55. Crass JR, Cohen AM, Motta AO, Tomashefski JF Jr, Wiesen EJ. A proposed new mechanism of traumatic aortic rupture: the osseous pinch. *Radiology* 1990;176:645-9. [DOI PubMed](#)
  56. Azizzadeh A, Keyhani K, Miller CC 3rd, Coogan SM, Safi HJ, Estrera AL. Blunt traumatic aortic injury: initial experience with endovascular repair. *J Vasc Surg* 2009;49:1403-8. [DOI PubMed](#)
  57. der Zee CP, Vainas T, van Brussel FA, Tielliu IF, Zeebregts CJ, van der Laan MJ. Endovascular treatment of traumatic thoracic aortic lesions: a systematic review and meta-analysis. *J Cardiovasc Surg* 2019;60:100-10. [DOI PubMed](#)
  58. Patel HJ, Hemmilla MR, Williams DM, Diener AC, Deeb GM. Late outcomes following open and endovascular repair of blunt thoracic aortic injury. *J Vasc Surg* 2011;53:615-21. [DOI PubMed](#)
  59. Lee WA, Matsumura JS, Mitchell RS, et al. Endovascular repair of traumatic thoracic aortic injury: clinical practice guidelines of the society for vascular surgery. *J Vasc Surg* 2011;53:187-92. [DOI](#)
  60. Arbabi CN, DuBose J, Starnes BW, et al; Aortic Trauma Foundation Study Group. Outcomes of thoracic endovascular aortic repair in patients with concomitant blunt thoracic aortic injury and traumatic brain injury from the aortic trauma foundation global registry. *J Vasc Surg* 2022;75:930-8. [DOI](#)
  61. Melissano G, Civilini E, Rinaldi E, Chiesa R. Toward a better understanding of endograft collapse after thoracic endovascular aortic repair. *J Endovasc Ther* 2010;17:738-43. [DOI](#)
  62. Ceja-Rodriguez M, Realyvasquez A, Galante J, Pevec WC, Humphries M. Differences in aortic diameter measurements with intravascular ultrasound and computed tomography after blunt traumatic aortic injury. *Ann Vasc Surg* 2018;50:148-53. [DOI](#)
  63. Khojenezhad A, Donayre CE, Azizzadeh A, White R; RESCUE investigators. One-year results of thoracic endovascular aortic repair for blunt thoracic aortic injury (RESCUE trial). *J Thorac Cardiovasc Surg* 2015;149:155-61.e4. [DOI](#)
  64. DuBose JJ, Charlton-Ouw K, Starnes B, et al; AAST/Aortic Trauma Foundation Study Group. Do patients with minimal blunt thoracic aortic injury require thoracic endovascular repair? *J Trauma Acute Care Surg* 2021;90:384-7. [DOI](#)
  65. Arbabi CN, DuBose J, Charlton-Ouw K, et al; Aortic Trauma Foundation Study Group. Outcomes and practice patterns of medical management of blunt thoracic aortic injury from the aortic trauma foundation global registry. *J Vasc Surg* 2022;75:625-31. [DOI](#)
  66. Alarhayem AQ, Rasmussen TE, Farivar B, et al. Timing of repair of blunt thoracic aortic injuries in the thoracic endovascular aortic repair era. *J Vasc Surg* 2021;73:896-902. [DOI](#)
  67. Jonker FH, Trimarchi S, Verhagen HJ, Moll FL, Sumpio BE, Muhs BE. Meta-analysis of open versus endovascular repair for ruptured descending thoracic aortic aneurysm. *J Vasc Surg* 2010;51:1026-32. [DOI](#)
  68. Jonker FH, Verhagen HJ, Mojibian H, Davis KA, Moll FL, Muhs BE. Aortic endograft sizing in trauma patients with hemodynamic instability. *J Vasc Surg* 2010;52:39-44. [DOI](#)
  69. Janczak D, Ziomek A, Kobecki J, Malinowski M, Pormańczuk K, Chabowski M. Neurological complications after thoracic endovascular aortic repair. Does the left subclavian artery coverage without revascularization increase the risk of neurological complications in patients after thoracic endovascular aortic repair? *J Cardiothorac Surg* 2019;14:5. [DOI](#)
  70. Banno H, Ikeda S, Kawai Y, et al. Early and midterm outcomes of celiac artery coverage during thoracic endovascular aortic repair. *J Vasc Surg* 2020;72:1552-7. [DOI](#)
  71. Juszczak MT, Vezzosi M, Khan M, Mascaro J, Claridge M, Adam D. Endovascular repair of acute juxtarenal and thoracoabdominal aortic aneurysms with surgeon-modified fenestrated endografts. *J Vasc Surg* 2020;72:435-44. [DOI](#)
  72. Konstantinou N, Antonopoulos CN, Jerkku T, et al. Systematic review and meta-analysis of published studies on endovascular repair of thoracoabdominal aortic aneurysms with the t-Branch off-the-shelf multibranched endograft. *J Vasc Surg* 2020;72:716-25.e1. [DOI](#)
  73. Field ML, Kuduvali M, Oo A. Multidisciplinary team-led management of acute Type B aortic dissection in the United Kingdom? *J R Soc Med* 2011;104:53-8. [DOI](#)
  74. Moulakakis KG, Mylonas SN, Dalainas I, Kakisis J, Kotsis T, Liapis CD. Management of complicated and uncomplicated acute type B dissection. A systematic review and meta-analysis. *Ann Cardiothorac Surg* 2014;3:234-46. [DOI](#)
  75. Rinaldi E, Loschi D, Favia N, Santoro A, Chiesa R, Melissano G. Spinal cord ischemia in open and endovascular aortic repair. *Aorta* 2022;10:194-200. [DOI](#)
  76. Rinaldi E, Melloni A, Gallitto E, et al. Spinal cord ischemia after thoracoabdominal aortic aneurysms endovascular repair: from the italian multicenter fenestrated/branched endovascular aneurysm repair registry. *J Endovasc Ther* 2023;30:281-8. [DOI](#)



77. Melissano G, Bertoglio L, Mascia D, et al. Spinal cord ischemia is multifactorial: what is the best protocol? *J Cardiovasc Surg* 2016;57:191-201. [PubMed](#)
78. Steinmetz LM, Coselli JS. Endovascular repair in patients with marfan syndrome: concerns amid controversy. *Ann Vasc Surg* 2023;94:1-7. [DOI](#)