



# A Review of Acute Aortic Syndrome: Pathophysiology, Risk Factors, Diagnosis, and Management

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## Abstract

**Background:** Acute Aortic Syndrome (AAS) encompasses a group of severe aortic pathologies, including Acute Aortic Dissection (AAD), Intramural Hematoma (IMH), and Penetrating Aortic Ulcer (PAU). These conditions share overlapping clinical presentations but arise from distinct pathological mechanisms and can coexist. The urgency of AAS is underscored by the rapid increase in mortality rates, particularly in type A dissections, with early intervention being crucial for patient survival. This review explores the pathophysiology, clinical features, and management strategies of AAS. **Methods:** A comprehensive review of the current literature on AAS was conducted, analyzing studies on etiology, risk factors, diagnostic imaging, and treatment outcomes. Emphasis was placed on recent advances in diagnostic techniques such as computed tomography (CT) angiography and the management of AAS subtypes based on clinical guidelines. **Results:** AAS pathologies originate from different mechanisms: AAD results from an intimal tear

and is often complicated by cystic medial necrosis, while IMH occurs due to the rupture of vasa vasorum, and PAU arises from atherosclerotic plaque erosion. Risk factors for AAS include hypertension, smoking, genetic conditions, and trauma. Imaging studies, especially CT angiography, play a critical role in diagnosis, with echocardiography and magnetic resonance imaging providing supplementary insights. Surgical intervention is essential for type A AAD, while type B and IMH cases are generally managed with medical therapy or endovascular repair. PAU cases often require surgical repair if symptomatic. **Conclusions:** Understanding the distinct pathophysiological mechanisms and timely recognition of AAS subtypes are critical for improving patient outcomes. Early diagnosis and personalized treatment strategies are key in managing this life-threatening condition. Continued advancements in imaging technologies and a better understanding of underlying genetic factors will aid in enhancing diagnosis and optimizing treatment protocols.

**Keywords:** Acute aortic dissection, intramural hematoma, penetrating aortic ulcer, diagnosis, treatment

**Significance** | This study determined no significant link between serum Vitamin D levels and COVID-19 mortality, except with LDH.

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## 1. Introduction

The term acute aortic syndrome (AAS) was first introduced in 1998 by Vilacosta et al. to describe a spectrum of severe and life-

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threatening aortic pathologies, including acute aortic dissection (AAD), intramural hematoma (IMH), and penetrating aortic ulcer (PAU) (Vilacosta et al., 1998). Despite overlapping clinical presentations, these conditions are distinct pathological entities with the potential to coexist or evolve into one another. For example, approximately 12% of individuals with AAD also present with IMH or PAU at diagnosis (Akin et al., 2012). From a diagnostic and surgical perspective, AAS is categorized based on the involvement of the ascending aorta, with Stanford type A involving the ascending aorta and Stanford type B sparing it (Vilacosta et al., 2021).

The urgency of early diagnosis and intervention is underscored by the time-sensitive nature of AAS. Mortality rates in type A dissections increase by approximately 2% per hour during the initial 48 hours (Harris et al., 2011). IMH, a variant of aortic dissection, lacks an intimal tear and is characterized by localized hemorrhage within the aortic wall (Leone et al., 2018). PAU, in contrast, involves the ulceration of atherosclerotic plaques, penetrating through the internal elastic lamina into the media layer. While many cases of PAU remain asymptomatic, progression to AAS or aortic rupture is possible in some patients (Oderich et al., 2019).

The etiology of AAS encompasses diverse mechanisms and shared risk factors. AAD, which constitutes 70–80% of all AAS cases, is precipitated by an intimal tear, often secondary to cystic medial necrosis or degeneration, exacerbated by hypertension and mechanical stress (Nienaber & Clough, 2015). In IMH, rupture of the vasa vasorum or localized thrombosis is thought to initiate hemorrhage, although the exact mechanisms remain debated (Mulligan-Kehoe, 2010; Song, 2004). PAU arises from atherosclerotic plaque erosion, which can progress to rupture or dissection in severe cases (Vilacosta et al., 1998).

Risk factors for AAS include hypertension, smoking, hyperlipidemia, and genetic conditions like Marfan and Ehlers-Danlos syndromes. Autoimmune diseases, infections, trauma, and iatrogenic injuries also contribute to its pathogenesis (Akutsu, 2019). Advanced imaging modalities, such as computed tomography angiography, are critical for timely diagnosis and management, providing detailed insights into the extent and severity of the pathology (Erbel et al., 2014). Pharmacological therapy focuses on controlling blood pressure and alleviating symptoms, while surgical intervention is often necessary for type A dissections or cases with imminent rupture risk (Lombardi et al., 2020).

However, AAS represents a spectrum of life-threatening aortic conditions requiring swift diagnosis and coordinated multidisciplinary management. Understanding the pathophysiology, risk factors, and clinical presentation of each entity within the syndrome is vital to improving patient outcomes.

## 2. Etiology of Acute Aortic Syndrome

Acute Aortic Syndrome (AAS) is an umbrella term encompassing three primary pathologies: acute aortic dissection (AAD), intramural hematoma (IMH), and penetrating aortic ulcer (PAU) (Figure 1). These conditions share overlapping clinical presentations, etiologies, and risk factors but arise from distinct pathophysiological mechanisms (Halushka et al., 2016).

### 2.1 Acute Aortic Dissection (AAD)

AAD constitutes 70–80% of all AAS cases, primarily originating from a tear in the aortic intima. This tear is often preceded by cystic medial necrosis or medial degeneration, which compromises the structural integrity of the aorta (Nienaber & Clough, 2015). Elevated blood pressure and mechanical shearing forces contribute to the development of a false lumen, as blood dissects the intima-media layer from the outer wall (Vilacosta & San Román, 2001). The dissection may propagate proximally or distally, with type A dissections involving the ascending aorta and type B dissections occurring distal to the innominate artery (Lombardi et al., 2020). These classifications align with the guidelines of the Society for Vascular Surgery and the Society of Thoracic Surgeons (Lombardi et al., 2020).

### 2.2 Intramural Hematoma (IMH)

The etiology of IMH remains debated. The leading hypothesis attributes its origin to spontaneous rupture of the vasa vasorum, the microvascular network supplying the outer aortic wall layers (Mulligan-Kehoe, 2010). Another mechanism involves intramural thrombosis, which arises from stagnant blood following an intimal microtear without a reentry tear (Song, 2004). Trauma, such as vascular catheter insertion, may also contribute to IMH development (Oderich et al., 2019).

### 2.3 Penetrating Aortic Ulcer (PAU)

PAU is characterized by the erosion of atherosclerotic plaques, which disrupt the intima and penetrate outward through the aortic wall layers. This condition can progress to AAD or IMH and, in severe cases, result in rupture or visceral ischemia (Vilacosta et al., 1998). PAU is strongly associated with widespread and severe atherosclerosis, often accompanied by hypertension, coronary artery disease, and chronic obstructive pulmonary disease (Oderich et al., 2019).

### 2.4 Risk Factors and Predisposing Conditions

Common predisposing factors for AAS include hypertension, smoking, hyperlipidemia, and cocaine use. Genetic conditions such as Marfan syndrome, Ehlers-Danlos syndrome, and Turner syndrome, alongside congenital anomalies like bicuspid aortic valve and coarctation of the aorta, significantly increase the risk (Akutsu, 2019). Additionally, vascular inflammation (e.g., giant cell arteritis), infections (e.g., syphilis), trauma, and iatrogenic injuries from catheterization or aortic surgery are important contributors (Akutsu, 2019; Harris et al., 2011).

### 3. Epidemiology

The incidence of AAS varies by subtype and population. AAD has an estimated incidence of 2.6 to 7.2 per 100,000 person-years, with 65% of cases occurring in males, typically in their seventh decade (Hagan et al., 2000). Predisposing factors for AAD differ between age groups; hypertension and atherosclerosis are predominant in older patients, whereas younger individuals more commonly have connective tissue disorders or congenital anomalies (Erbel et al., 2014). IMH accounts for 5–25% of AAS cases, with an incidence of approximately 1.2 per 100,000 person-years, and is more common in individuals in their 80s (Park et al., 2008). PAU, representing 2–7% of AAS cases, has an average incidence of 2.1 per 100,000 person-years and is frequently associated with concurrent aortic aneurysms in 42–61% of cases (Bossone & Eagle, 2021).

Understanding the distinct pathophysiological mechanisms and shared risk factors of AAD, IMH, and PAU is critical for the accurate diagnosis and effective management of AAS. Early identification of predisposing conditions and timely intervention are essential to mitigate the significant morbidity and mortality associated with these life-threatening conditions.

#### 3.1 Pathophysiology

Acute aortic syndrome (AAS) is primarily driven by atherosclerotic disease and hypertension, though other factors such as trauma, infections, and medial pathology may also play significant roles (Akin et al., 2012). The pathophysiological process frequently begins with an intimal tear caused by the shearing forces of blood flow or disruption of the aortic media, leading to the separation of the aortic wall layers (Vilacosta et al., 2021). This separation results in the creation of a true lumen and a false lumen. The true lumen, lined by intima, remains connected to undissected segments of the aorta, while the false lumen is delineated by an "intimal flap." Over time, blood flow through the false lumen can cause an aortic aneurysm (Baliyan et al., 2018).

Intramural hematoma (IMH), traditionally attributed to the rupture of the vasa vasorum, is now recognized as having overlapping mechanisms with aortic dissection (AD), including microintimal tears. The absence of a large reentrant tear distinguishes IMH, as this prevents the patency of the false lumen (Song, 2004). Conversely, penetrating aortic ulcers (PAUs) result from atherosclerotic plaques invading the intimal layer, progressively eroding toward the adventitia. Depending on their progression, PAUs can evolve into IMH, AD, or pseudoaneurysms, highlighting their shared pathological underpinnings (Oderich et al., 2019).

#### 3.2 Histopathology

The hallmark histopathological feature of AAD is medial degeneration, characterized by elastic fiber fragmentation,

thinning, and the accumulation of mucoid extracellular matrix (Leone et al., 2018). Intimal thinning is also notable, with dissections commonly occurring near the vasa vasorum in the outer media. Individuals with connective tissue disorders or aortic aneurysms typically exhibit more pronounced medial degeneration, which serves as a final common pathway in various forms of AAS (Halushka et al., 2016).

IMH is histologically distinct from AD, primarily involving restricted intramural blood flow. The hematoma resides within the media and can either resolve or progress to AD (Park et al., 2008). While vasa vasorum rupture remains a leading hypothesis for IMH, some studies propose that IMH may represent an AD with a thrombosed false lumen, further blurring the distinction between these entities (Macura et al., 2003).

PAUs primarily develop in the descending thoracic aorta within the context of severe atherosclerosis (Vilacosta et al., 1998). Histopathological findings typically include localized intramedial bleeding and the formation of an ulcerative crater, which may act as an entry point for dissection. Calcification and associated inflammation often confine hemorrhages to localized areas, but severe cases can result in adventitial penetration, leading to aortic rupture or pseudoaneurysm formation (Nienaber & Clough, 2015).

### 4. History and Physical Examination

Severe aortic pain is the most defining clinical symptom of AAS, often described as "tearing," "ripping," or "pulsating" (Vilacosta & San Román, 2001). Data from the International Registry of Acute Aortic Dissection (IRAD) indicates that sharp pain is the most common presenting complaint, though approximately 4.5% of cases are asymptomatic and identified incidentally (Harris et al., 2011). Neurological symptoms such as syncope may also occur, signifying impaired blood flow to the central or peripheral nervous system (Gaul et al., 2008).

Involvement of the ascending aorta is particularly concerning due to potential complications, including myocardial ischemia, aortic regurgitation, or pericardial effusion. Hypertension is the most prevalent risk factor for AAS, observed in 86% of cases in a prospective study by Landenhed et al. (Rogers et al., 2011). Despite its strong association, the Aortic Dissection Detection Risk Score does not prioritize hypertension as a critical predisposing factor (Bima et al., 2020).

#### 4.1 Clinical Implications

Understanding the pathophysiology and histopathology of AAS is essential for early diagnosis and effective management. The intricate interplay between atherosclerosis, medial degeneration, and vasa vasorum rupture underscores the need for advanced imaging modalities and biomarkers to delineate these entities accurately. Furthermore, integrating patient history, physical examination findings, and risk factor assessment, including

hypertension, can guide clinical decision-making and improve outcomes for patients presenting with AAS symptoms.

#### 4.2 Evaluation of Acute Aortic Syndrome (AAS)

Evaluation of AAS necessitates a systematic approach combining clinical history, physical examination, and diagnostic testing. Non-imaging and imaging-based investigations are pivotal in confirming the diagnosis and assessing the severity of the condition.

Clinical evaluation begins with electrocardiography (ECG), which aids in distinguishing cardiac causes of chest pain, such as myocardial infarction. While AAS and myocardial infarction may coexist, they require distinct management strategies. Data from the International Registry of Acute Aortic Dissection (IRAD) indicate that 31% of patients with type A dissection presented with normal ECG findings, while the remaining patients exhibited changes ranging from nonspecific ST- and T-wave abnormalities to patterns consistent with acute myocardial infarction (Boone et al., 2002). Blood tests also contribute to the diagnostic process by ruling out differential diagnoses or providing supportive evidence for AAS. Commonly utilized tests include complete blood count, blood urea nitrogen and electrolyte levels, C-reactive protein, liver function tests, D-dimer, troponin, creatinine kinase, and arterial blood gas analysis, including lactate and glucose levels. The choice of tests is often guided by clinical presentation and resource availability.

Imaging studies are central to diagnosing AAS, with various modalities offering complementary insights. Chest radiography is frequently the initial imaging test performed, with a sensitivity of up to 64% and a specificity of 86%. Radiographic signs indicative of AAS include mediastinal widening, aortic kinking, tracheal deviation, and a double-density appearance of the aortic shadow (Nienaber et al., 2004). However, computed tomography (CT) angiography is considered the gold standard for AAS diagnosis due to its superior sensitivity, exceeding 95%, and specificity, ranging from 87% to 100% (Yamashiro et al., 2015; Cigarroa et al., 1993). This modality is widely available in emergency departments and provides rapid, noninvasive imaging that is less dependent on operator expertise compared to ultrasound techniques.

Echocardiography, including transthoracic (TTE) and transesophageal (TEE) approaches, also plays a role in diagnosing AAS. While TTE can identify proximal dissections and their complications, its effectiveness is limited for visualizing the entire aorta. In contrast, TEE offers closer proximity to the aorta and achieves a sensitivity of 99% and a specificity of 89% (Erbel et al., 2001). Despite these advantages, TEE is more invasive, operator-dependent, and less accessible in emergency settings compared to CT angiography. Magnetic resonance imaging (MRI) is rarely employed for acute AAS evaluation due to its limited availability in emergency scenarios and the life-threatening nature of the condition. Nevertheless, MRI is the most sensitive and specific

modality for diagnosing all types of AAS and may be used when initial investigations yield inconclusive results (Lindsay et al., 1999).

#### 5. Treatment and Management of AAS

Acute aortic syndrome (AAS) is categorized based on the involvement of the ascending or descending aorta. Conditions affecting the ascending aorta are surgical emergencies, whereas static AAS involving the descending aorta is often managed conservatively. However, descending aorta cases that actively progress, cause organ or limb malperfusion, result in unmanageable pain, or pose a rupture risk require urgent surgical intervention (Hiratzka et al., 2010). Initial management aims to lower systolic blood pressure below 120 mm Hg and reduce the rate of blood pressure change (dp/dt) to halt dissection or prevent rupture. Intravenous  $\beta$ -blockers, such as labetalol, are the cornerstone of medical therapy, with nondihydropyridine calcium channel blockers serving as alternatives. Vasodilators may also be used in combination with these treatments (Table 1)

Acute aortic dissection (AAD) involving the ascending aorta is traditionally managed with an open surgical approach. The primary objective is to eliminate the false lumen by closing or excising the intimal tear and, if necessary, reinforcing the aortic wall with synthetic grafts. Aortic valve insufficiency and coronary artery damage, often caused by proximal extension, may require resuspension or replacement of the valve. While endovascular repair has been attempted for type A dissections, its application remains limited due to sparse evidence and challenges in addressing dissection involving the aortic valve and root (Gleason et al., 2014). Acute type B dissection is categorized into complicated and uncomplicated cases. Complicated type B dissection, seen in approximately 25% of patients, is characterized by hemodynamic instability, organ or limb malperfusion, unrelenting chest pain, uncontrollable hypertension, or progressive imaging findings (Dake et al., 1999). For these cases, endovascular stenting with synthetic grafts combined with aggressive medical therapy offers the most favorable outcomes. Conversely, uncomplicated type B dissection has traditionally been managed with medical therapy alone. Prophylactic endovascular treatment to prevent progression remains under debate, as conclusive evidence from large-scale, long-term randomized controlled trials is lacking.

Intramural hematoma (IMH), despite having lower mortality than AAD, is treated similarly due to its risk of progression to dissection, aneurysm formation, or rupture. While aneurysms in the ascending aorta often require stenting via an endovascular approach, lesions in the descending aorta may be managed through watchful waiting, optimal medical therapy, or surgical intervention (Nienaber et al., 2016). Stable IMH of the descending aorta is managed medically, while unstable cases necessitate endovascular management. IMH progression and regression complicate stent sizing and increase the

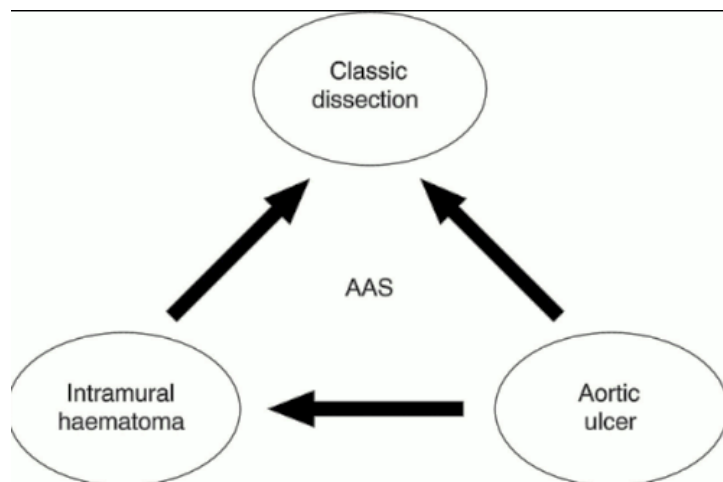


Figure 1. Acute Aortic Syndrome

Table 1. Management and Treatment of Acute Aortic Syndromes (AAS)

| Category                              | Description  | Management Approach  | Notes   |
|---------------------------------------|--|--|---|
| <b>Acute Aortic Syndrome (AAS)</b>    | Categorized by involvement of ascending or descending aorta.                 | <ul style="list-style-type: none"> <li>- <b>Ascending aorta:</b> Surgical emergency.</li> <li>- <b>Descending aorta (static):</b> Often managed conservatively.</li> </ul>               | Descending cases with progression, malperfusion, unmanageable pain, or rupture risk require urgent surgical intervention.   |
| <b>Initial Medical Management</b>     | Aims to lower systolic BP (<120 mm Hg) and reduce rate of BP change (dP/dt). | <ul style="list-style-type: none"> <li>- <b>Drugs:</b> Intravenous β-blockers (e.g., labetalol).</li> <li>- <b>Alternatives:</b> Nondihydropyridine calcium channel blockers.</li> </ul> | Vasodilators may be used in combination with the above treatments.  |
| <b>Acute Aortic Dissection (AAD)</b>  | Involves intimal tear with false lumen formation.                            | <ul style="list-style-type: none"> <li>- <b>Type A (Ascending aorta):</b> Open surgical approach.</li> <li>- <b>Type B (Descending aorta):</b> Medical therapy or stenting.</li> </ul>   | Type A: Open surgery focuses on closing the intimal tear, reinforcing the wall, and addressing complications (e.g., valve resuspension).<br>Type B: Stenting with synthetic grafts is preferred in complicated cases. |
| <b>Intramural Hematoma (IMH)</b>      | Risk of progression to dissection, aneurysm, or rupture.                     | <ul style="list-style-type: none"> <li>- <b>Ascending aorta:</b> Often requires stenting (endovascular approach).</li> <li>- <b>Descending aorta:</b> Medical or surgical.</li> </ul>    | Stable IMH of the descending aorta is managed medically. Unstable cases require endovascular management, but complications (e.g., stent sizing, endoleaks) are common.  |
| <b>Penetrating Aortic Ulcer (PAU)</b> | Focal aortic wall erosion that may progress to aneurysm or rupture.          | <ul style="list-style-type: none"> <li>- <b>Asymptomatic PAU:</b> Managed medically.</li> <li>- <b>Symptomatic PAU:</b> Surgical repair or endovascular stent grafts.</li> </ul>         | Endovascular repair is preferred in older patients with comorbidities, as it reduces mortality rates significantly.   |
| <b>Acute Type B Dissection</b>        | Divided into complicated and uncomplicated cases.                            | <ul style="list-style-type: none"> <li>- <b>Complicated:</b> Endovascular stenting with aggressive medical therapy.</li> <li>- <b>Uncomplicated:</b> Managed medically.</li> </ul>       | Prophylactic endovascular treatment is debated due to limited evidence from long-term randomized controlled trials.   |

risk of type I endoleaks. Isolated asymptomatic penetrating aortic ulcer (PAU) can be treated medically, but symptomatic cases require surgical repair due to a higher likelihood of progression to aneurysm, pseudoaneurysm, or rupture. Endovascular repair with stent grafts is often preferred for patients with PAU due to advanced age and comorbidities, as it significantly reduces mortality rates (Czerny et al., 2012).

### **5.1 Differential Diagnosis**

Acute aortic syndrome (AAS) can be challenging to diagnose due to its clinical overlap with other conditions, including acute aortic dissection (AAD), intramural hematoma (IMH), penetrating aortic ulcer (PAU), thoracic and abdominal aortic aneurysms, traumatic aortic injury, myocardial infarction, and pulmonary embolism. Comprehensive clinical evaluation and judicious use of diagnostic imaging are essential for differentiating AAS from these entities and ensuring appropriate management (Akin et al., 2012; Vilacosta et al., 2021).

### **5.2 Staging and Classification**

The classification of AAD and IMH relies predominantly on the DeBakey and Stanford systems, which provide insights into the origin and extent of the dissection. The DeBakey classification categorizes dissections into three types: type I originates in the ascending aorta and extends distally, sometimes beyond the arch; type II involves only the ascending aorta; and type III is confined to the descending aorta (Vilacosta et al., 2021). The Stanford classification simplifies this into type A, which includes any dissection involving the ascending aorta, and type B, which excludes the ascending aorta (Hagan et al., 2000). These classifications are pivotal for determining the prognosis and guiding treatment strategies.

### **5.3 Prognosis**

AAD is associated with high mortality rates, particularly in type A dissections. Without surgical intervention, type A dissections result in mortality rates of approximately 24% within 24 hours, 44% by seven days, and 49% within two weeks. Surgical management significantly improves outcomes, reducing mortality to 10% within the first day, 16% by seven days, and 20% within two weeks (Nienaber & Clough, 2015; Harris et al., 2011). Type B dissections have a comparatively better prognosis, with mortality rates around 10% within 30 days; however, complicated cases can exceed 25% mortality (Moulakakis et al., 2014). IMH complicates diagnosis and management, as up to 30% of patients with AAD may also have IMH. While spontaneous resolution occurs in 10% of IMH cases, progression to dissection can occur in 47% of patients. Surgical management of type A IMH yields better outcomes, with a mortality rate of 14% compared to 36% for medical treatment. For type B IMH, medical and surgical outcomes are comparable, with mortality rates of 14% and 20%, respectively (Alomari et al., 2014).

### **5.4 Complications**

AAS may lead to various complications, including aortic aneurysm formation, rupture, end-organ ischemia, limb ischemia, aortic valve dysfunction, haemopericardium, pleural effusion, coronary artery dissection, stroke, myocardial infarction, and death. Endoleaks, a complication of endovascular stenting, are categorized into five types based on leakage origin. Type I endoleaks involve inadequate sealing at the attachment sites, type II arise from collateral vessel flow, type III result from graft defects, type IV are attributed to graft porosity, and type V (endotension) involves aneurysm sac expansion without visible leakage. Types I and III require urgent intervention, while type II endoleaks are typically monitored and treated selectively, particularly when sac growth exceeds 5 mm. Most type II endoleaks resolve spontaneously, with a rupture risk of less than 1%. Types IV and V are rare and usually managed conservatively (Oderich et al., 2019; Lombardi et al., 2020).

### **5.5 Patient Education**

Effective patient education is a cornerstone in reducing the risks and complications associated with aortic syndromes. Smoking cessation is pivotal, as tobacco use substantially heightens the risk of vascular diseases, including acute aortic syndromes (AAS) (Hagan et al., 2000; Vilacosta et al., 2021). Patients should be educated on the critical role of avoiding tobacco and offered support through cessation programs. Managing hypertension is equally vital, given its contribution to aortic wall stress, which can lead to dissection or rupture (Erbel et al., 2014). Patients should be encouraged to monitor blood pressure regularly and adhere to prescribed antihypertensive therapies.

Additionally, patients must avoid recreational drug use, especially cocaine, due to its strong correlation with acute increases in aortic pressure, precipitating dissection events (Carrel et al., 2023). Clinicians should inform patients about the associated dangers and facilitate access to drug rehabilitation programs as necessary. Lifestyle and dietary modifications play a complementary role in mitigating risks. A diet low in sodium and rich in fruits, vegetables, and whole grains, combined with regular physical activity, promotes vascular health and reduces cardiovascular risk (Nienaber et al., 2015). Patients should also strive to maintain a healthy weight and manage other modifiable risk factors such as diabetes and dyslipidemia (Bossone & Eagle, 2021).

These interventions should be part of a comprehensive lifestyle modification strategy, underpinned by patient education. Such an approach not only lowers the likelihood of developing AAS but also improves long-term outcomes for individuals with a history of aortic disease. Multidisciplinary collaboration involving healthcare providers, patient educators, and support systems ensures patients are empowered with the knowledge and resources necessary to safeguard their health (Vilacosta et al., 2021).

### **5.6 Enhancing Healthcare Team Outcomes**

Rapid diagnosis is critical in managing AAS due to the high mortality rate associated with delayed treatment (Rogers et al., 2011). Emergency department teams are instrumental in expediting diagnosis and facilitating prompt specialist referrals. Initial evaluations should include vital sign monitoring, clinical assessment, and laboratory investigations, followed by imaging tailored to the patient's hemodynamic stability (Sommer et al., 1996).

Specialized aortic centers have demonstrated better patient outcomes, emphasizing the importance of centralizing care in high-volume facilities with expertise in aortic surgery (Leone et al., 2018). These centers reduce early mortality, decrease reoperation rates, and enhance long-term survival (Harris et al., 2011). Establishing an "aorta code" protocol can further mitigate delays in recognition and management. This pathway, operational 24/7, facilitates the rapid activation of emergency care from smaller hospitals and ensures swift transfer to specialized centers. The aorta code improves provider awareness, accelerates diagnosis, and ensures timely engagement of specialized surgeons, significantly enhancing clinical outcomes (Vilacosta et al., 2021).

## 6. Role of Pharmacists, Emergency Providers, and Nursing

Managing AAS requires a multidisciplinary approach, with pharmacists, emergency providers, and nurses playing pivotal roles in ensuring timely diagnosis, treatment, and optimized patient outcomes (Bima et al., 2020).

### 6.1 Pharmacists

Pharmacists are integral to the healthcare team, particularly in managing antihypertensive therapies critical for stabilizing patients with aortic dissection or other acute syndromes (Hagan et al., 2000). They monitor drug interactions, minimize adverse effects, and collaborate with physicians in selecting appropriate medications such as beta-blockers to reduce aortic wall stress. Additionally, they manage analgesic administration to alleviate sympathetic stimulation and prevent hemodynamic instability (Akutsu, 2019). Pharmacists also educate patients on long-term medication use, including antiplatelets and lipid-lowering agents, as part of secondary prevention strategies (Nienaber et al., 2015).

### 6.2. Emergency Providers

Emergency providers play a critical role as the first point of contact for patients presenting with acute aortic syndrome (AAS). Timely recognition of hallmark symptoms such as acute chest or back pain is crucial for initiating a diagnostic pathway that includes imaging studies, particularly computed tomography angiography (CTA) (Harris et al., 2011; Vilacosta et al., 2021). Emergency providers are responsible for rapid stabilization, including blood pressure and heart rate control, which is fundamental to reducing the risk of aortic rupture (Erbel & Nienaber, 2012; Nienaber & Powell, 2012). They must also triage patients efficiently and coordinate transfers

to specialized centers capable of surgical or endovascular interventions (Leone et al., 2018).

Emergency providers often activate specialized protocols, such as the "aorta code," to streamline communication and care pathways across multidisciplinary teams (Vilacosta et al., 2021). Additionally, they play a pivotal role in guiding patients and their families through critical, time-sensitive decisions, balancing the risks and benefits of medical versus surgical treatment options (Nienaber & Clough, 2015). Managing comorbid conditions, such as myocardial infarction or stroke, further underscores their indispensable role in AAS care (Gaul et al., 2008). Emergency providers also collaborate with specialists to determine the safest and most effective treatment strategies for patients with contraindications to specific therapies (Akin et al., 2012).

### 6.3 Nursing

Nurses are integral to both the acute and long-term management of AAS, providing patient-centered care at every stage. In emergency settings, they monitor vital signs, administer medications, and assist in stabilizing patients (Bima et al., 2020). Their ability to identify subtle changes in a patient's condition facilitates early interventions, reducing the risk of complications such as aortic rupture or organ ischemia (Halushka et al., 2016). In the postoperative or intensive care setting, nurses deliver continuous monitoring and support, managing pain, wound care, and the prevention of complications like infections or deep vein thrombosis (Leone et al., 2018).

Beyond clinical tasks, nurses play a central role in patient education, emphasizing the importance of lifestyle modifications and medication adherence (Hagan et al., 2000). They guide patients through smoking cessation, dietary changes, and hypertension management strategies (Bossone & Eagle, 2021). Building trust and effective communication ensures patients and families are informed and motivated to follow prescribed care plans (Moulakakis et al., 2014). Furthermore, nurses facilitate interdisciplinary collaboration by updating other healthcare providers on patient progress and addressing emerging concerns (Akutsu, 2019).

### 6.4 Interdisciplinary Collaboration

Effective management of AAS relies on seamless interdisciplinary collaboration among emergency providers, nurses, and pharmacists. For example, pharmacists ensure the availability of critical medications and advise on appropriate dosing, while nurses administer these medications and monitor for adverse effects (Song, 2004; Rogers et al., 2011). Emergency providers depend on insights from both pharmacists and nurses to make informed decisions, particularly in high-pressure scenarios (Tsai et al., 2005). Standardized care protocols, such as the "aorta code," highlight the importance of clearly defined roles and responsibilities within the care team (Vilacosta & San Román, 2001). These protocols enhance

efficiency by reducing delays in diagnosis and treatment. For instance, while emergency providers initiate imaging and stabilization, nurses provide real-time updates on clinical status, and pharmacists ensure the timely availability of essential drugs (Oderich et al., 2019).

### 6.5 Enhancing Patient Outcomes

The combined efforts of emergency providers, nurses, and pharmacists significantly improve patient outcomes in both acute and long-term management of AAS. Timely diagnosis and intervention reduce mortality rates and enhance quality of life (Carrel et al., 2023). Furthermore, these professionals contribute to preventive care, helping minimize the risk of recurrence through patient education and consistent follow-up (Bossone & Eagle, 2021). By prioritizing early intervention and effective communication, they establish a care framework that emphasizes patient safety and well-being (Erbel et al., 2014).

The roles of emergency providers, nurses, and pharmacists are distinct yet interconnected, forming the backbone of multidisciplinary care in AAS management. Their dedication, expertise, and collaboration ensure comprehensive and timely treatment, ultimately advancing the standard of care for patients with life-threatening cardiovascular conditions.

### 7. Perspective

Acute aortic syndrome (AAS) presents a unique challenge in clinical medicine due to its highly variable presentation, complex pathophysiology, and the critical importance of early diagnosis and intervention. While the clinical features of AAS, such as chest pain, are common across its three main pathologies—acute aortic dissection (AAD), intramural hematoma (IMH), and penetrating aortic ulcer (PAU)—the underlying mechanisms and treatment strategies diverge significantly. The variability in presentation and risk factors, such as hypertension, smoking, and genetic disorders, adds layers of complexity to its diagnosis. This underscores the necessity for heightened clinical suspicion, particularly in high-risk populations, to ensure timely identification and intervention. Advances in imaging technology, particularly computed tomography (CT) angiography, have dramatically improved diagnostic accuracy, yet challenges persist in identifying early-stage AAS, especially in patients who present with atypical or mild symptoms. Understanding the pathophysiological differences between AAD, IMH, and PAU is paramount, as this informs management strategies that may range from conservative medical therapy to urgent surgical intervention.

### 8. Conclusion

In conclusion, acute aortic syndrome demands an integrated, multidisciplinary approach to care. Mortality rates associated with AAS are alarmingly high, particularly when diagnosis and

intervention are delayed. Rapid, accurate imaging plays a crucial role in guiding therapeutic decisions, while pharmacological and surgical interventions are tailored to the type and severity of the condition. The role of nursing interventions is indispensable, particularly in early detection, patient education, and managing the emotional and physical challenges of AAS. The prognosis is directly correlated with the speed of intervention; therefore, awareness and early recognition are critical to improving patient outcomes. Ultimately, a collaborative and timely approach involving clinicians, surgeons, and nurses is essential in managing AAS, emphasizing the need for continued research and refinement of diagnostic and treatment strategies to reduce mortality and enhance survival rates.

### Author contributions

All authors contributed equally to the conceptualization, design, data collection, analysis, and interpretation of the study. S.O.A., R.H.A., B.K.A., M.N.A., H.F.A., D.R.A., M.I.A., D.A.A., A.F.A., M.K.A., M.M.F.A., M.F.N.A., A.S.M.A., B.M.H.A., A.S.A.A., and A.I.A.A.F. were actively involved in drafting and revising the manuscript. All authors approved the final version for submission.

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### Competing financial interests

The authors have no conflict of interest.

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