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RESEARCH ARTICLE

Navigating Contemporary Strategies in Endovascular Management of Descending Thoracic Aortic Dissection: A Comprehensive Review of the Literature

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ABSTRACT

Introduction: Acute aortic dissection (AD) is the deadliest form of acute aortic syndromes (AAS). Mortality is significant; around 80% in the highest-risk individuals. The challenge in managing AD is pronounced, especially with the high-risk open surgical approach. Endovascular aortic stent grafts offer a less invasive alternative. We retrospectively reviewed literature from 2004 to 2023 on type B AD endovascular management and focused on the newest patient series since the last meta-analysis (after January 2011).

Materials and Methods: Identified from PubMed Central were two consensus papers, two randomized controlled trials, two historical articles, five meta-analyses, 31 clinical trials, and 50 review papers in English. Nineteen patient series from January 2011 onward were included, excluding non-English publications, duplicate reports and papers dealing only with type A AD or only with true thoracic aortic aneurysms (TAA).

Results: Among 4235 patients in 19 publications, 69.2% had type B AD. Thoracic endovascular aortic repair (TEVAR) was performed in 90.5% of patients, with a 5.9% 30-day mortality rate. Complications included type I-II (and sometimes III) endoleaks (11.1%), stroke (5.5%), and paraplegia or paraparesis (3.2%). The need for re-intervention was around 17.7%, and the mean follow-up time was 36.4 (±20.7) months.

Conclusion: This review highlights TEVAR's technical success, potential advantages, complications, and survival rates for type B AD patients with its long-term efficacy still undetermined.

Keywords: endovascular repair, TEVAR, type B aortic dissection, descending thoracic aortic dissection, aortic disease, stent-graft, acute aortic syndrome.

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INTRODUCTION

Background

Annually, 43,000 to 47,000 Americans succumb to aortic and branch diseases, a rising trend. Thoracic aortic conditions increasingly find remedy in stent-grafting. Acute aortic dissection (AD), the deadliest among acute aortic syndromes (AAS), occurs at a minimum rate of 30 cases per million yearly. Untreated acute type A AD bears a 1% per hour mortality, with 80% expected fatalities within two weeks. Acute type B AD, though less fatal, poses significant risks: 10% mortality at 30 days and about 80% in high-risk cases. Renowned figures like Laennec, Maunoir, and the surgical breakthrough by DeBakey, Cooley, and Creech in 1954 contribute to understanding AD's historical context. Key milestones include the 1996 establishment of the International Registry of Acute Aortic Dissection (IRAD) and the May 1999 NEJM issue, which introduced endovascular management for acute type B AD, marking the era of endovascular AD treatment. [1,15]

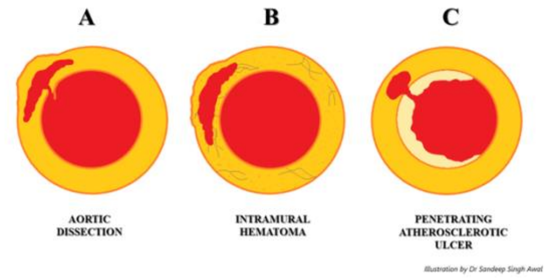
Definitions

Acute Aortic Syndromes (AAS)

Acute aortic syndromes (AAS) involve diverse and life-threatening conditions like AD, intramural hematomas (IMH), penetrating atherosclerotic ulcers (PAU) with or without aortic rupture, and aortic trauma causing laceration. In each case, the aorta's media layer is disrupted, causing bleeding (IMH), layer separation (AD), or transmural wall rupture (ruptured PAU or trauma). (Figure 1). [2,3,4]

Aortic dissection (AD)

AD involving the separation of aortic wall layers, is a serious cardiovascular condition. Acute cases can have immediate lethal complications, while chronic dissection leads to aneurysm formation. This results from rapid blood flow from the aortic lumen into the media through intima ruptures, forming a false lumen. True and false lumens are separated by a fine tissue membrane, consisting of the intima and 70% of the media. The false lumen's aortic wall is thin and weak, comprising 30% of the media and the aortic adventitia. [5]



Aetiology-Epidemiology

Historically, syphilis was often associated with Aortic Acute Syndromes (AAS), but today, various factors contribute to AAS. [4]

AD primarily results from hypertension and aortic media degeneration, seen in 80% of AD patients. Hypertension induces mechanical and metabolic strain on the aortic wall, leading to media degeneration. The aorta, with a 3-layered structure (intima, media, and adventitia), exhibits media degeneration in most AD cases. Genetic conditions (Marfan syndrome, Turner syndrome, Noonan syndrome, Ehlers-Danlos - especially type IV, and Loeys–Dietz syndrome) predispose individuals to AD. Non-syndromic mutations, notably in the SMC actin gene (ACTA2), associate with thoracic aneurysms. Additional risk factors encompass smoking, pregnancy, labor, traumatic aorta rupture, aortic trauma during open-heart surgery, phaeochromocytoma, lupus erythematosus, and illicit drug abuse. Identified risk factors by the International Registry of Acute Aortic Dissection (IRAD) include male sex, age in the 60s and 70s, hypertension, prior cardiac surgery (especially aortic valve repair), bicuspid aortic valve, and a history of Marfan syndrome. [1,4,5,12,17-18]

Population-based studies estimate an incidence of 2 to 3.5 cases per 100,000 person-years, with an increasing trend, reaching 16 per 100,000 male individuals per year in the USA. [7,13-16]

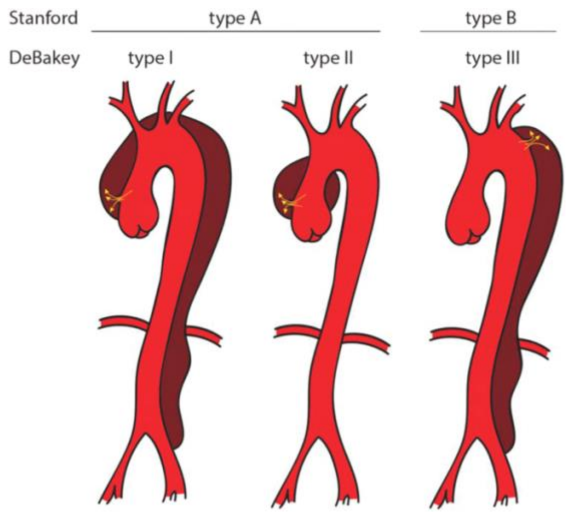
Pathogenesis

AD, predominantly caused by a transverse tear in the aortic wall (95%), affects the intima and most of the media, covering almost half of the aortic perimeter. This tear leads to blood entering the aortic wall, forming a false lumen that can extend to the aortic valve, causing complications such as aortic valve regurgitation, coronary artery occlusion, or rupture towards the right heart cavities, superior vena cava, or pulmonary artery trunk. Frequently, the false lumen compresses the true lumen, causing collapse and resulting in ischemic complications in about 30% of cases. [1,5]

In atherosclerosis, the inflammatory response to media thrombus can lead to necrosis, apoptosis of smooth muscle cells (SMCs), and elastic tissue degeneration, increasing the risk of medial rupture. FDG uptake on positron emission tomographic imaging often heralds this risk. Inflammatory disorders like periarteritis nodosa, Takayashu's syndrome, or Behcet's syndrome elevate the risk of Acute Aortic Syndromes (AAS). [4,12] Whereas the proximal thoracic aorta is the site of the entry point (65% ascending aorta, 10% aortic arch, 20% proximal descending aorta, 5% distal descending thoracic and abdominal aorta), secondary or re-entry tears or fenestrations can occur distally in the thoracic aorta or in the abdominal aorta or iliac arteries. [1,5]

Classification

Acute AD is clinically diagnosed within 14 days of symptom onset. Beyond this period, AD is considered chronic, but this classification is often inaccurate. Two widely used anatomical classifications include the DeBakey classification (type I affecting the entire aorta, type II affecting the ascending aorta, and type III involving the distal part) and the simpler Stanford classification (type A affecting the ascending aorta and type B affecting the rest). Type B constitutes 33% of acute AD cases, beginning after the left subclavian artery. (Figure 2). [1,5]



Natural history

In the acute phase of aortic dissection (AD), the formation of a dissecting aneurysm and aortic rupture poses risks, resulting in 75% of documented deaths. Survival improves in the chronic phase, with complications being most common in ascending aorta dissections. Without surgery, survival for acute type A or I or II is around 10% in the first year. Type B AD patients have better survival, reaching 90% in the first month and approximately 50% in the first year, with most deaths occurring in the initial 30 days. [5]

Presentation

Chest discomfort, irrespective of age, gender, or other complaints, is a frequent hospital presentation. Patients are often admitted to rule out a myocardial infarction, despite coronary disease being more common. Unfortunately, almost 40% of acute aortic dissection cases are initially overlooked during presentation. [4,6]

Acute aortic dissection (AD) manifests with diverse symptoms, including chest and/or back pain, often mimicking conditions like pulmonary embolism, pneumothorax, pneumonia, pericarditis, musculoskeletal pain, or stroke. It can also mimic or cause coronary ischemia, necessitating prompt differential diagnosis. Thrombolysis, beneficial in acute myocardial infarction, is contraindicated in AD. In an IRAD study of 464 AD patients, 95.5% reported pain at presentation. [5,7] Pain was abrupt in onset in nearly 85% and severe in 90%. Others have shown that a ripping, tearing or migratory quality to the pain can differentiate those with acute AD from those without in a cohort of patients presenting with a complaint of chest pain. [8,9] Less frequently, acute AD can be painless, or silent, on presentation. The incidence of painless acute AD is estimated to be 5%–15%. [6,7]

In acute type B aortic dissection, a typical presentation involves a man in his 60s or 70s arriving at the emergency department with sudden, severe chest pain. Hypertension is common, but low blood pressure can occur if the dissection has ruptured. Physical findings may include pulse deficit, blood pressure discrepancies, and possibly a diastolic heart murmur due to aortic valve regurgitation (50% of cases). Paraplegia or paraparesis (2% of cases) may be detected, caused by intercostal or lumbar arterial occlusion. [1,5]

Diagnosis

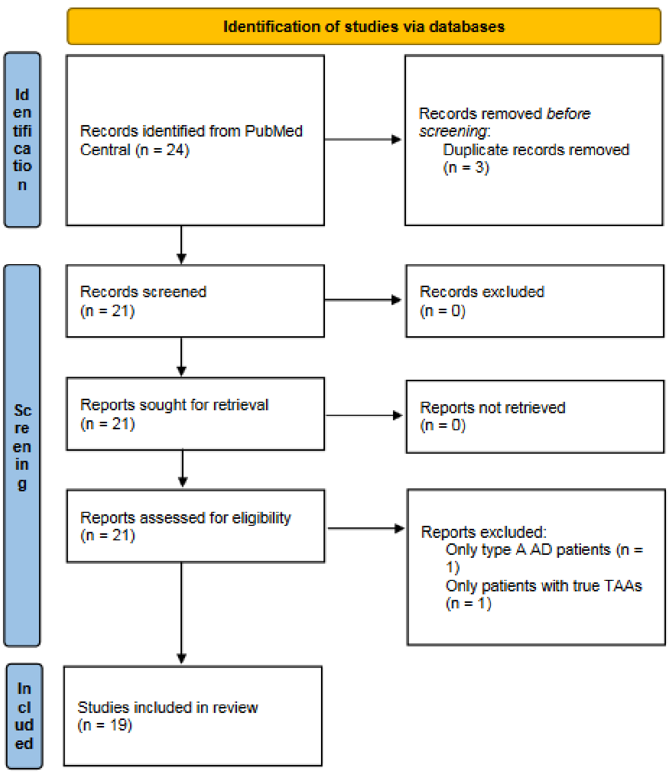
For diagnosing acute aortic dissection (AD), a highly intuitive physician is crucial. Suspecting AD is warranted in any patient with acute chest pain lacking electrocardiographic ischemic findings. Chest x-rays offer limited diagnostic value, potentially revealing mediastinal expansion and left pleural effusion. Trans-thoracic echocardiography (TTE) may indicate pericardial effusion, aortic valve regurgitation, or dissection at the ascending aorta. Trans-esophageal echocardiography (TEE) is highly sensitive and specific for diagnosing AD in the ascending or descending thoracic aorta. CT-angiogram, magnetic resonance angiography (MRA), and digital subtractive angiography (DSA) are gold standards, with CT-angiogram being the best screening method for size information. MRA provides detailed data on dissection extent and false lumen content, while DSA offers information on blood flow, aortic branch patency, and aortic valve regurgitation presence. [4,5]

In patients in shock with a strong suspicion of ascending aortic dissection (AD), initial use of trans-thoracic echocardiography (TTE) is reasonable before urgent surgery, followed by trans-esophageal echocardiography (TEE) before sternotomy. While TTE and TEE are vital for bedside assessment, they lack the anatomical detail needed for planning endovascular interventions. Intravascular ultrasound may become a useful addition to endovascular procedures, but its primary diagnostic value is limited. [4]

Treatment

Upon AD presentation, all patients receive medication to alleviate pain and prevent dissection expansion, typically vasodilators and β-blockers. Intravenous β-blockers maintain heart rate at 60-70 bpm, while sodium nitroprusside achieves vasodilation, lowering systolic blood pressure. This controls acute chest pain. Diuresis above 25ml/h is crucial. After symptom control, diagnostic procedures like CT-angiogram, DSA, or trans-esophageal echocardiogram follow. The treatment type (conservative or surgical/endovascular) is determined by diagnostic findings, including primary entry point identification, AD extent, and complications. Acute type A AD mandates immediate surgical therapy, preventing aortic rupture and AD expansion. Surgery involves ascending aorta substitution with a prosthesis. Uncomplicated type B AD initiates conservative therapy with antihypertensive medication; surgery is reserved for complications. Emergency techniques for complicated cases include stent-grafting, aortic graft replacement, flap fenestration, catheter reperfusion, or surgical bypass. [15]

The mentioned surgeries for acute aortic dissection (AD) carry high morbidity (35-75%) and up to 80% mortality, particularly with bowel ischemia. Endovascular options like TEVAR and/or EVAR, often complemented by fenestration or transposition of aortic branches or the Chimney technique, are considered. "Hybrid" or "composite" operations, and TEVAR, are preferred for complicated acute type B ADs. (Figure 3) Stent grafting for descending thoracic aorta dissections is established but lacks settled indications due to limited randomized trials. Despite encouraging results, there's insufficient level 1 evidence supporting long-term overall survival improvement with TEVAR compared to traditional treatment. [5, 32-37]



Multidisciplinary consensus on management of type B AD

An expert panel developed treatment algorithms for type B aortic dissection based on a consensus approach. Data from 63 studies (2006-2012) included 1,548 patients with medical treatment, 1,706 with open surgery, and 3,457 with thoracic endovascular repair (TEVAR). For acute type B dissection, early mortality was 6.4% with medical treatment, 10.2% with TEVAR, and 17.5% with open surgery, mostly for complications. Subacute type B dissection had a 2.8% early mortality with TEVAR. In chronic cases, 5-year survival was 60-80% with medical therapy, while TEVAR and open surgery had 6.6% and 8.0% early mortality, respectively. The panel acknowledged the consensus document's reliance on non-robust evidence and emphasized cautious interpretation due to heterogeneous literature results. [38]

MATERIAL AND METHODS

Purpose of the present study was a retrospective review of the existing literature from the years 2004-2014 on management of descending thoracic aortic dissection, with a focus on meta-analyses and reviews of endovascular treatment of type B dissection of the descending thoracic aorta. We then searched for the newest patient series published after the publication of the most recent meta-analysis (after January 2011) and then performed a review of those results.

In accordance with the PRISMA guidelines (Preferred Reporting Items for Systematic Reviews and Meta-Analyses), this systematic review was conducted to ensure transparency and completeness in reporting. The PRISMA checklist [2020 version] [39] was followed throughout the review process to enhance the quality and rigor of the study (Figure 4).

Endovascular techniques are already a reality in the management of complicated (even in select cases of uncomplicated) type B AD. With the introduction of modern, more evolved synthetic stent grafts, they are also gaining ground in the treatment of carefully selected patients with acute type A AD, although their technical efficacy and safety are yet to be proven, and mortality seems to be comparable to that of open surgical therapy.

For the above mentioned reasons and since endovascular experience is yet limited in type A AD, we decided to limit the present study only in studying patients treated with TEVAR for type B AD, alone or compared with open surgery or the best medical therapy.

During our first search of the literature (2004-2014), using "Meta-analysis" or "Review" as filters, 2 multi-disciplinary consensus papers, 2 randomized controlled trials, 2 historical articles, 5 meta-analyses, 31 clinical trials, and 50 review papers published in English between 1st January 2004 and 1st January 2014 were identified in PubMed Central. [40-87].

In our second search of the most recent literature (1 January 2011 – 31 October 2023), using "Clinical Trial" as a filter, 24 series [19-29, 88-100] were identified in PubMed Central. MESH headings included: "endovascular (treatment OR therapy OR management OR repair OR TEVAR) for (type B OR descending thoracic) aortic dissection". In the context of this scientific article, only the latest versions of studies have been considered for inclusion, with a deliberate exclusion of any prior publications or iterations of the same studies [27,29,92] to ensure the incorporation of the most current and up-to-date findings in the analysis. Publications in Chinese, Spanish, Russian, or other languages than English were excluded. All series included had more than 25 patients enrolled. 2 among them also included patients with type A AD. Series studying only patients with type A AD [95] or only with true TAAs [96] were excluded.

Statistical analysis

Rates of events were calculated as the number of events divided by the number of treated patients with available data. In Table 1, results are presented as mean ± 1 standard deviation or median and range, when appropriate. AD was classified according to the Stanford classification [30]. Dissection was considered an acute event if it occurred within the first 14 days [31] from onset of symptoms, whereas it was considered chronic beyond 14 days. A third, subacute phase is considered in some publications as an intermediate phase between the first two, ranging from 2-8 weeks after initial onset of AD symptoms. [20,32]

RESULTS AND DISCUSSION

There were 4235 patients in the 19 publications [19-29, 88-100] that were analyzed. Out of them, 2930 had type B AD (69.2%), 729 (17.2%) had type A AD, 140 (3.3%) had true thoracic aortic aneurysms (TAA), 29 (0.7%) had IMH, 33 (0.8%) had PAU and 6 (0.1%) had traumatic aortic rupture. 73.0% of them were men and 27.0% were women. Their average age was 62.8 (±6.5) years. In 769 cases the AD was acute (58.0%), in 97 subacute (7.3%) and there were 461 chronic cases (34.7%). In 3 publications there was no reference to the phase of the AD. In 607 patients, the AD was uncomplicated (24.3%), while in the majority (1893, 75.7%) it was complicated. In 1 publication, there was no reference if the AD was uncomplicated or complicated.

Thoracic endovascular aortic repair (TEVAR) was performed in 2652 patients (90.5%) with type B AD. The average procedural duration was 178.7±111.4 minutes, while the technical success reached 98.8%. Various stent-grafts were used, with a higher preference in Medtronic stent-grafts (Talent, Valiant). Pooled 30-day mortality was 5.9%. The most often complications described were type I-II (and in some cases III) endoleaks (11.1%), stroke (5.5%) and paraplegia or paraparesis (3.2%). Need for re-intervention was approximately 17.7%, while the time for re-intervention ranged from 2-5 years. Finally, the mean follow-up time was 36.4 (±20.7) months, and patients' survival (or cumulative freedom from late adverse events) rates were 90.3%, 87.7%, and 77.5% after 1, 2-3, and 4-5 years, respectively, after TEVAR.

Comparing the above data from our analysis with the recently published data in meta-analyses [32,40,41,63], having enrolled patients with almost the same demographics, there seem to be some differences. For example, the pooled early (30-day) mortality in all AD cases (acute, subacute, chronic) is higher when compared with previously published results from Europe and China (5.9% compared to 5.3% and 2.3%, respectively). Complication rates seem to be also much higher. Stroke seems to have occurred in 2-times or even 12-times the number of patients (5.5%, compared to 2.9% (European data) and 0.5% (Chinese data), respectively), while paraplegia or paraparesis presents 4-times or even 30-times more frequently (3.2%, compared with 0.8% (European data) and 0.1% (Chinese data), respectively). Procedural / technical success is similar (98.8%, compared with 98.5% (European data) and 89.4% (Chinese data), respectively).

There are only few available meta-analyses on endovascular stent-graft placement in type B AD, an emerging treatment option for this potentially lethal disease. Our review, although it is not a prospective and randomized comparison with other treatment strategies (conservative medical treatment, open surgery) and even if it is limited only in the most recently published data (2011-2023), it provides an insight into the technical success, potential advantages and complications, as well as survival rate for patients treated with TEVAR for type B AD. It should, nevertheless, be viewed in the light of its limitations, exactly like all other reviews published before. The relatively low percentage of studies with data available for some valuable parameters might further lower patient representation and increase selection bias. In some of the analyzed publications further considerations arise from ambiguous definitions. This could also lead to decreased statistical power and lower reliability of our results. Last but not least, one must consider that so far, patient observation after EVAR is limited in short- and mid-term follow-up. Long-term outcomes need to be evaluated.

Table 1. Detailed overview over the analyzed patient series

First author	Month/Year	Total patients (n)	Patients with type B AD (n)	Patients treated with TEVAR (n)	Median age (years) (±SD)
Jing [100]	2020	73	73	73	56.8 (±13.3)
Lin [99]	2020	84	84	84	53.6 (±10.6)
Bavaria [98]	2020	50	50	50	57.2 (±12.3)
Werlin [97]	2019	162	15	162	73.0 (±8.0)
Beach [94]	2017	200	28	200	72.6 (±3.1)
Cambria [93]	2015	50	50	50	57.1 (±13.0)
Bell [91]	2015	72	8	48	70.0 (±18.0)
Brunkwall [90]	2014	61	61	30	63.5 (±11.5)
VIRTUE Registry Investigators (Heijmen) [89]	2014	100	100	100	60.1 (±12.0)
Lombardi [88]	2014	86	86	86	58.7 (±13.3)
Zahn [19]	2013	191	104	191	n/a
Steuer [20]	2013	124	124	124	68
Wilkinson [21]	2013	454	454	49	70.1 (±12.3)
Qin [22]	2013	193	193	152	n/a
Jia [23]	2013	303	303	208	53.6 (±20.3)
Li [24]	2012	1812	1086	825	n/a
Heijmen [25]	2012	100	42	100	64.6 (±12.0)
Ehrlich [26]	2013	29	29	29	61.0
Zipfel [28]	2011	91	41	91	65.0
		4235	**2930**	**2652**	**62.8 (±6.5)**

Table 1. (continued)

Male Sex (%)	Female Sex (%)	Stent-graft(s)	Acute/Subacute/ Chronic AD (n/n/n) (%)	Uncomplicated/ Complicated AD (n/n) (%)	Procedural success (%)
75.0	25.0	Microport Medical Castor	50/0/23	73/0	97.0
88.1	11.9	Various	84/0/0	0/84	100.0
80.0	20.0	Valiant Captivia	50/0/0	0/50	100.0
74.0	26.0	Cook multibranched modular	0/0/15	0/15	100.0
54.0	46.0	Cook TX1, TX2	0/0/28	0/28	100.0
74.0	26.0	Gore TAG	50/0/0	0/50	100.0
68.1	31.9	Various	3/0/5	8/0	98.6
78.7	21.3	Gore TAG	61/0/0	61/0	100.0
76.0	24.0	Medtronic Valiant	50/24/26	n/a	98.0
73.3	26.7	Cook TX2	55/31/0	0/86	100.0
n/a	n/a	Various	n/a	0/104	92.1
65.0	35.0	Various	102/22/0	0/124	n/a
57.1	42.9	Gore TAG, Medtronic Talent or Valiant, Cook TX	53/20/0	0/73	100.0
n/a	n/a	Various	152/0/0	41/152	n/a
72.3	27.7	Various	0/0/303	95/208	100.0
n/a	n/a	Various	n/a	261/825	n/a
81.0	19.0	Valiant Captivia	n/a	0/42	100.0
75.9	24.1	Medtronic Valiant	29/0/0	0/29	100.0
75.8	24.2	Relay	30/0/61	68/23	95.0
73.0	**27.0**		**769/97/461 (58.0/7.3/34.7)**	**607/1893 (24.3/75.7)**	**98.8**

Table 1. (continued)

Procedural duration (min) (±SD or range)	Endoleaks type I-III (%)	Stroke (%)	Paraplegia (%)	Reintervention (in 2-5 years, %)	30-day mortality (%)
128.2 (±66.8)	0.0	0.0	0.0	n/a	1.4
n/a	4.7	1.2	n/a	1.2	1.2
142.9 (±125.6)	8.0	8.0	2.0	14.0	8.0
404 (±116)	16.0	5.0	5.0	74.0	0.0
152 (102-201)	28.0	3.5	4.0	25.0	6.5
n/a	2.0	18.0	8.0	18.0	8.0
n/a	16.7	4.2	1.4	13.9	11.1
n/a	n/a	0.0	0.0	n/a	0.0
138 (±81)	12.0	9.0	4.0	27.0	8.0
n/a	n/a	8.2	1.2	24.4	4.7
107 (±122)	8.5	3.9	1.7	3.3	5.6
n/a	n/a	4.8	2.4	n/a	7.3
n/a	n/a	6.1	6.1	20.0	10.2
n/a	n/a	n/a	n/a	n/a	n/a
n/a	n/a	n/a	0,9	4.3	2.9
n/a	n/a	n/a	n/a	n/a	2.2
n/a	16.7	4.0	1.0	1.0	4.0
n/a	13.8	10.3	n/a	3.4	17.0
(42-540)	7.0	2.2	5.5	n/a	8.0
178.7 (±111.4)	**11.1**	**5.5**	**3.2**	**17.7**	**5.9**

Table 1. (continued)

Mean follow-up (months) (±SD or range)	Cumulative freedom from late adverse events or survival in 1 year (%)	Cumulative freedom from late adverse events or survival in 2-3 years (%)	Cumulative freedom from late adverse events or survival in 4-5 years (%)
61.0 (48-72)	95.0	n/a	93.2
60.0	97.6	95.2	92.9
60.0	86.0	n/a	83.0
13.2 (9.6-48.0)	89.5	n/a	n/a
57.6 (±39.6)	85.8	n/a	55.6
24.2 (±10.2)	88.0	85.0	n/a
62.0 (±34.5)	88.0	n/a	63.0
12.0	100.0	n/a	n/a
36.0	86.0	84.7	n/a
24.0	88.3	84.7	n/a
24.5 (±27.7)	n/a	n/a	n/a
n/a	n/a	n/a	n/a
27.7 (±34.9)	n/a	n/a	n/a
n/a	97.0	89.0	67.0
28.5 (±16.3)	n/a	91.6	88.1
n/a	n/a	n/a	n/a
2.3 (±1.2)	n/a	n/a	n/a
53.0 (±41.0)	82.0	n/a	77.0
n/a	n/a	84.0	n/a
36.4 (±20.7)	**90.3**	**87.7**	**77.5**

CONCLUSIONS

AAS describes the acute presentation of patients with characteristic "aortic pain" caused by one of several life threatening thoracic aortic pathologies. These include AD, IMH, PAU, aneurysmal leak, and traumatic transection/rupture of the aorta. AAS heralds imminent aortic rupture. Highlighting acute aortic pathology as an AAS is therefore important to encourage prompt recognition of this condition and avoid any diagnostic delays. The management of AAS remains a therapeutic challenge. The traditional surgical approach to acute type B (descending thoracic) aortic pathology is unsatisfactory with high morbidity and mortality. Endovascular aortic stent grafts now represent an alternative minimally invasive approach in these patients who are often poor surgical candidates. Studies show endovascular repair to be technically feasible with fewer complications. Long term results are awaited to assess its true efficacy.

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AUTHORS' CONTRIBUTIONS

The participation of each author corresponds to the criteria of authorship and contributorship emphasized in the Recommendations for the Conduct, Reporting, Editing, and Publication of Scholarly Work in Medical Journals of the International Committee of Medical Journal Editors. Indeed, all the authors have actively participated in the redaction and revision of the manuscript and provided approval for this final revised version.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest in this case.

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