Endovascular Management of Acute Aortic Syndromes

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ABSTRACT

The term “acute aortic syndrome” (AAS) refers to a spectrum of life-threatening thoracic aortic pathologies including intramural hematoma, penetrating atherosclerotic ulcer, and aortic dissection. Clinically, patients often present with characteristic aortic pain. AAS often leads to aortic rupture. Therefore, recognition of this condition, its prompt diagnosis, and timely treatment is crucial to obtain clinical success and improved overall survival. The management of AAS, however, remains a therapeutic challenge. Endovascular strategies have gained wide acceptance and now represent a minimally invasive alternative to traditional open surgery. Several studies have shown endovascular repair of varying thoracic aortic pathologies to be technically feasible with fewer complications than open surgery. In this review, the authors discuss AAS pathology and its management, with particular attention to the current role of endovascular aortic repair and its treatment.

KEYWORDS: Penetrating atherosclerotic ulcer, intramural hematoma, aortic dissection

Objectives: Upon completion of this article, the reader should be able to explain the clinical presentation and imaging findings of acute aortic dissection, penetrating atherosclerotic ulcer, and intramural hematoma, and the role for endovascular management in their treatment.

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The term “acute aortic syndrome” (AAS) was first introduced into the literature in 1998¹ and further characterized in 2001 by Villacosta et al² to describe a variety of acute aortic pathologies. These include aortic dissection, intramural hematoma (IMH), and penetrating atherosclerotic ulcer (PAU).

The clinical entities that give rise to AAS are distinct with varying pathophysiologic mechanisms that precipitate their appearance and therefore distinguish their radiologic findings. However, it is clear that there is

considerable overlap. Furthermore, the progression from one pathologic process to another has been documented. That is to say, some patients with IMH may evolve into an aortic dissection whereas others with PAU are accompanied by some degree of IMH; furthermore, penetrating ulcers may act as the entry site for aortic dissection. It is clear that while these acute aortic pathologies appear mostly separate, in some patients one may precede the other and often times may coexist (Fig. 1).

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Figure 1 Type A intramural hematoma (IMH) with penetrating atherosclerotic ulcer (PAU). (A) A noncontrast axial computed tomography (CT) image of the ascending aorta demonstrates a high-attenuation crescent representing intramural hematoma. (B) Contrast-enhanced axial CT scan at the same level confirms the presence of IMH. (C) A coexisting penetrating atherosclerotic ulcer is noted in the aortic arch.

These entities also share a common classification system. Although the Stanford Classification was originally applied to aortic dissection, it now defines AAS pathologies according to location, which broadly correlates with its management. By definition, type A pathologies affect the ascending aorta and aortic arch and type B pathologies affect the descending aorta defined as those distal to the origin of the left subclavian artery.

AAS is characterized by “aortic pain” and a history of underlying severe hypertension is frequently present. Aortic pain has been described as a severe, sudden onset of tearing or ripping migratory chest pain. Chest pain that radiates to the neck, throat, or jaw typically indicates the aortic segment involved is the ascending aorta; pain located in the back or abdomen suggests involvement of the descending aorta. Additional diagnostic clues may be the presence of a murmur of aortic regurgitation and pulse differentials.

The need to consider and promptly diagnose acute chest pain or acute thoracic pathology as an AAS is clear. Although AAS has a relatively lower annual incidence compared with acute coronary syndrome (0.5 vs 3.0 per 100,000), AAS has an inordinately high rate of mortality.³ In fact, AAS is the most frequently fatal condition in the spectrum of patients with chest pain.⁴ Therefore, though acute myocardial infarction must be excluded, other clinical entities such as AAS should be

considered. Furthermore, with the aging population and improved imaging techniques at our disposal, this entity is becoming a more commonly encountered clinical phenomenon.

The advent of improved endovascular techniques and devices has facilitated recent advances in the management of thoracic aortic pathology. Many of the patients who are at risk for developing AAS are poor surgical candidates and conservative medical management alone is often inadequate. Minimally invasive alternatives such as endovascular thoracic aortic repair are increasingly being undertaken. The aim of this review is to provide an overview of AAS pathology, its management, and in particular to assess the current role of endovascular thoracic aortic repair in its treatment.

PATHOLOGY

Aortic dissection is characterized by the existence of an intimal medial entrance tear, and in the majority of cases, a reentrance tear. Intramural hematoma is characterized by the presence of a hemorrhage into the aortic media, which leads to the development of a false lumen. The entrance and reentrance tears seen in classic aortic dissection are absent. Aortic ulcers usually penetrate through the intima into the media. A causal relation has been proposed linking these three conditions. The

Figure 2 Type B intramural hematoma (IMH) evolves to dissection. (A) An axial computed tomography scan shows a type B IMH. (B) Follow-up imaging 4 months later shows IMH evolution to aortic dissection.

presence of an ulcer may precipitate both an intramural hemorrhage and a classic dissection, a condition that usually occurs in a severely atherosclerotic aorta. An intramural hematoma, in turn, may precipitate a dissection (Fig. 2).

Aortic Dissection

Aortic dissection involves a tear in the intimal lining of the aorta allowing blood to flow within the layers of the media. The commonly referred to intimal flap in the aortic dissection is actually a misnomer as the tissue flap is composed mainly of aortic media that is delaminated from the aortic wall; hence, it is more appropriately referred to as an intimomedial tear. The proportion of aortic media that remains at the outer wall of the false channel differs from patient to patient. The more media involvement in the intimomedial flap, the thinner the external wall of the false lumen, and the more likely it will be to rupture.⁵ False channel rupture is the most common mechanism of death in patients with aortic dissection. Additionally, the false lumen may compress the true lumen (Fig. 3A–D) or extend into branch arteries creating malperfusion states. The entrance can occur at any point throughout the aorta, although is most frequently found in areas of greatest hydraulic stress such as the right lateral wall of the ascending aorta or in the proximal segment of descending thoracic aorta. Additionally, it is not uncommon for patients with aortic dissection to have a reentrance tear and several communicating points between the true and false lumen throughout the descending thoracic aorta and abdominal aorta.

It is estimated that dissections of the descending thoracic aorta occur with an incidence of 2–3.5 cases per 100,000 with up to 14,000 reported new cases in the United States each year.^{6–8} As previously discussed, aortic dissections are classified by location with type A

pathologies involving the ascending aorta and aortic arch and type B pathologies involving the descending thoracic aorta. Dissections can also be classified temporally into acute and chronic phases. The acute phase is defined as < 14 days from the onset of symptoms. This was based on early studies of aortic dissections demonstrating an increased associated mortality occurring within the first 2 weeks.⁹ Additionally, dissections are typically classified as complicated or uncomplicated. Complicated dissections involve those with malperfusion, rupture or impending rupture, refractory hypertension, or continued pain.¹⁰ Approximately 30% of type B dissections are complicated at initial presentation.¹¹ Others have advocated stratification of uncomplicated type B dissections as those with or without high risk features.¹²

Patients with type A dissections in the International Registry of Aortic Dissection (IRAD) database had an overall in-hospital mortality of 27% for patients treated surgically and 56% for those treated medically.¹³ This underscores the current treatment paradigm where all patients with type A dissections undergo emergent surgical repair regardless of whether the dissection is complicated or not. Conversely, patients with type B dissection treated medically have markedly reduced early mortality in the range of 10–17%.^{3,8,14} The most common cause of death in these patients was rupture closely followed by symptomatic branch vessel involvement with malperfusion.

Intramural Hematoma

IMH is thought to be present in 10–30% of patients with AAS.^{5,15} IMH is considered a variant of classic aortic dissection where blood collects within the aortic media without the presence of an intimal flap (Fig. 4).¹⁶ The false lumen is created by rupture of the vasa vasorum that lies in the media or from hemorrhage within an atherosclerotic plaque.¹⁷ It is important to note that

IMH may not only be attributed to spontaneous rupture of a “sick” vasa vasorum, but may also be the result of traumatic rupture of “healthy” vasa vasorum during blunt aortic trauma.⁵ Additionally, the hematoma, while often intramedial in nature, may extend through the outer medial layer into the subadventitial space representing a contained aortic rupture. Conversely, the intramedial hemorrhage may also extend toward the aortic lumen and progress to dissection. Complications of IMH are common with reported progression to complete dissec-

tion in 28–47% of cases and early aneurysm formation or contained rupture in 20–45% of patients.¹⁸ However, some patients may show spontaneous resolution of IMH under medical treatment.

Predictors of disease progression and mortality include recurrent or persistent pain, the presence of a concomitant PAU, involvement of the ascending aorta and maximum aortic diameter > 50 mm.^{5,19,20} Although the aortic diameter itself is the main determinant of wall stress, IMH places increased stress on the wall of the

Figure 3 Type B aortic dissection treated with a thoracic stent graft with subsequent false lumen remodeling. (A,B) Select axial images CT at the level of the main and right pulmonary arteries shows a type B dissection with compression of the true lumen. (C,D) Sagittal oblique reformatted and volume-rendered imaging of the thoracic aorta shows the origin of the aortic dissection just distal to the left subclavian artery extending throughout the descending thoracic aorta. Marked compression of the true lumen is noted.

Figure 3 (Continued) (E–G) Follow-up CT imaging 5 days following thoracic stent-graft placement shows lack of false lumen blood flow at the site of primary intimal-medial tear; however, false lumen flow is noted in the distal descending thoracic aorta. (H–J) Follow-up CT imaging 20 months following thoracic stent-graft placement demonstrates improved true lumen patency throughout the descending thoracic aorta with no flow and subsequent remodeling of the false lumen.

aorta. Therefore, the presence of IMH in an already dilated aorta would be expected to carry a greater risk of dissection and rupture. Predictors of resolution or improved prognosis include younger age, aortic diameters < 45 mm, and thickness of the hematoma < 10 mm.^{21,22}

Penetrating Atherosclerotic Ulcer

PAU has been found to be in 2.3–7.6% of patients with AAS.¹ PAU is defined by an ulceration of an aortic atherosclerotic plaque, which penetrates the internal elastic lamina into the aortic media.²³ These patients therefore tend to be older with greater cardiovascular comorbidity. PAUs are usually focal lesions and may be found anywhere in the aorta; however, they are most commonly located in the descending thoracic aorta

(Fig. 5). This may reflect the increased presence of atheromatous disease in this location. PAUs, though focal, may be single or multiple, and the excavated “crater” of these ulcerated lesions may contain debris, cholesterol, and thrombotic material. Progressive intimal erosion can lead to blood entering the media. This may precipitate an aortic dissection or formation of IMH. Further penetration to the adventitia may lead to pseudoaneurysm formation with contained or even free rupture.^{23–26} Not surprisingly therefore, PAU has the highest rate of aortic rupture up to 42% when compared with IMH or aortic dissection.²⁷ This underscores the aggressive surgical approach many groups have taken in treatment of PAU. Even so, other groups have reported success with a nonoperative expectant approach.^{28,29} Still, it appears that symptomatic patients presenting

Figure 4 Type B intramural hematoma (IMH) resolving with best medical therapy. Type B IMH was found in a patient with previously untreated hypertension. (A–C) Select axial computed tomography images show a moderate-sized type B IMH. There were no complicating features and the patient was treated with best medical therapy. (D–F) Follow-up CT imaging 8 months later demonstrates residual type B IMH that has significantly improved in appearance.

with PAU have a worse prognosis than asymptomatic patients with incidentally found PAU. Additional high risk features include lesion size ≥ 20 mm or depth > 10 mm.²² The natural history of PAUs is unknown. However, many authors have documented progression from PAU to aneurysmal dilatation,³⁰ ulcer extension into the adventitia to form a pseudoaneurysm,¹ or an entrance point to aortic dissection.^{31,32}

MEDICAL MANAGEMENT

Medical management in patients with AAS consists of close intensive care unit monitoring with tight regulation of blood pressure, heart rate, and pain control. The overall goal is to decrease aortic wall shear stress. General guidelines to achieve this goal include a heart rate < 60 beats per minute and systolic blood pressure < 100 – 120 mm Hg or mean arterial pressure < 60 – 76 mm Hg. Beta blockers are a staple of optimal medical management. However, additional hypertensive agents

are also often required. Regardless of dissection location, all patients in whom there is a strong suspicion of aortic dissection should be immediately placed on antihypertensive therapy to limit progression of dissection.^{33,34}

The concept of antiimpulse pharmacologic management is the mainstay of medical therapy in patients with AAS. However, the ideal medical management following initial discharge is not as well defined. It would seem that following the same antiimpulse regimen used to treat patients acutely would have continued long-term benefit. Several reports advocate the use of β blockers,³⁵ tight heart rate control,³⁶ and alternative antihypertensive agents such as angiotensin converting enzyme inhibitors.³⁷ Although β blockade and tight heart rate control help diminish overall aortic wall shear stress, there is evidence that angiotensin converting enzyme inhibitors have unique vascular remodeling properties.³⁸ Finally, it is important to note that because medical therapy alone does not stop blood flow to the false lumen, a significant number of patients who survive

Figure 5 Type B penetrating atherosclerotic ulcer (PAU) treated with thoracic stent graft. (A) Axial computed tomography (CT) image shows a penetrating ulcer in the descending thoracic aorta. (B) Volume-rendered imaging demonstrates the focal nature of this disease in the distal descending thoracic aorta. (C) Angiographic image of the PAU during planned endovascular treatment. (D) Postendograft placement angiography shows successful exclusion of previously shown distal type B PAU.

the acute phase develop late aneurysmal degeneration of the false lumen in 1–5 years.^{39,40}

SURGICAL MANAGEMENT

A discussion of potential endovascular treatments of AAS would not be appropriate without a brief discussion of the current surgical management. This highlights

opportunities for improved morbidity and mortality and places proper context for the role of our newer less invasive treatment alternatives.

Aortic Dissection

The treatment paradigm for aortic dissections dates back to the work in the late 1960s and early 1970s

demonstrating the disparate outcomes between dissections involving the ascending aorta and those isolated to the descending thoracic aorta.³⁸ The standard management of type A aortic dissections since that time has been surgical repair of the ascending aorta with additional repair of the aortic arch, aortic root, or aortic valve as necessary.⁴¹ Traditionally, operative repair of type A aortic dissections has been limited to the ascending aorta. More recently, endovascular techniques have been added as adjuncts to the surgical management of type A dissections. For dissections extending into the descending thoracic aorta, placement of a stent graft into the unrepaired descending aorta can reduce the complications related to the residual dissected vessel.^{42,43} Others have advocated endovascular treatment of type A dissections that extend in a retrograde fashion (the so-called retro-A dissection) from a dissection originating in the descending thoracic aorta by covering the primary entry site tear. Additionally, some groups propose endovascular treatment of malperfusion states in patients with type A dissections before operative repair of the ascending aorta.^{44,45} Branched endografts may allow for endovascular repair of classic type A dissections in the future, but no suitable devices are currently available.

Given the low in-hospital mortality rates for uncomplicated type B dissections managed medically, early surgical repair has generally been reserved for patients with complicated type B dissections. A type B dissection is considered complicated when there is rupture or impending rupture, pain or hypertension refractory to medical therapy, progression of the dissection despite optimal medical therapy, and visceral or limb ischemia. Surgical repair is also indicated in cases of aneurysmal degeneration of the dissected segments of the descending aorta to prevent late aortic rupture. Surgical repair for aneurysmal disease usually involves a left posterolateral thoracotomy with retroperitoneal exposure of the abdominal aorta if necessary to replace the entire length of dilated aorta. Reimplantation of intercostal or lumbar arteries can be performed if necessary during an open surgical procedure. Surgical fenestration can be performed if the entire dissected segment of the aorta is not removed.⁴⁶ Surgical treatments of malperfusion states include surgical bypass of the ischemic vascular beds as well as surgical fenestration. However, mortality rates for operative repair in the setting of complicated type B dissections remain high.^{11,47}

Intramural Hematoma

In general, the surgical treatment of IMH parallels the treatment of aortic dissection. However, given the less well-established natural history of IMH, optimal surgical management remains a matter of debate. Consensus favors operative repair of type A intramural hematoma

with excision of the affected segment and replacement with a surgical graft. However, rates of surgical repair for type A IMH are lower than the corresponding rates for type A dissections. Surgical treatment for type B IMH is usually reserved for aneurysmal degeneration of the aorta, persistent pain or hypertension, or organ/limb ischemia. Additional high risk features that may indicate treatment of IMH include an aortic diameter > 50 mm, a hematoma thickness > 11 mm, or pleural/pericardial fluid. The surgical techniques employed for repair of IMH parallel those for aortic dissections described above.^{18,21,48–50}

Penetrating Atherosclerotic Ulcer

Similar to IMH, the optimal treatment of PAUs remains a matter of debate given its lower prevalence and less well-understood natural history compared with traditional aortic dissection. Some series have reported rates of rupture as high as 38%⁵¹; others have described more successful management of PAUs with medical management as compared with surgical therapy. High risk features include lesion size of ≥ 20 mm or depth > 10 mm.²² When operative repair is required, graft replacement of the entire involved aorta is performed. It is of particular importance to remove the aortic segment including the PAU in the setting of concomitant dissection or intramural hematoma.²¹

ENDOVASCULAR MANAGEMENT

The introduction of endovascular techniques for the treatment of acute aortic syndromes has the potential to significantly alter the risk/benefit ratios for medical therapy versus repair. Although no prospective randomized trials comparing endovascular techniques to surgical therapy have been conducted, multiple reports have demonstrated high technical success rates and low rates of morbidity and mortality with endovascular techniques employed to treat acute aortic syndromes.⁵²

Aortic Dissection

Options available to the interventionalist treating aortic dissections include branch vessel stenting, aortic fenestration, aortic endograft placement, or a combination of these techniques. Selection of the appropriate treatment method is based on the indication for the intervention, dissection anatomy, and flow dynamics within the true and false lumens. Of immediate concern in treating complicated aortic dissection is revascularization of ischemic vascular beds in malperfusion syndromes. Branch vessel obstruction can be related to either static or dynamic obstructions, as classified by Williams et al.⁵³ Static obstruction refers to propagation of the dissection

or associated thrombus into the branch vessel itself, with restriction of flow through the perfusing lumen. Dynamic obstruction refers to intermittent obstruction of a branch vessel origin by the aortic dissection flap itself, as well as to obstruction of one aortic lumen (usually the true lumen) by the other lumen above a branch vessel origin. An additional goal in the treatment of aortic dissection includes protecting the patient from further aortic-related complications such as delayed rupture or aneurysm formation.

FENESTRATION

The goal of endovascular aortic fenestration is to treat malperfusion syndromes by equalization of pressures between the true and false lumens, relieving the dynamic aortic component of obstruction caused by the dissection. In some cases, adjunctive aortic stenting may be used to maintain expansion of the true lumen. Branch vessel stenting is then used to treat the static obstruction of branch vessels caused by the dissection. These techniques are thus widely applicable in treating malperfusion symptoms caused by both static and dynamic obstructions, and can be applied no matter the location of the primary tear. However, application of fenestration and branch stenting techniques is time consuming, technically demanding, and often requires complex intraprocedural decision making. Additionally, in preserving flow through the false lumen the fenestration procedure does not address other potential complications of the dissection, namely aneurysm formation and late rupture.

Static obstruction caused by extension of the dissection flap or hematoma into branch vessels is treated with placement of stents within the branch vessel from the perfusing lumen. Branch vessel stenting should be performed after treatment of dynamic aortic obstruction (either by fenestration or stent graft placement) if the two conditions coexist to optimize vessel inflow. Superior mesenteric obstructions should be treated before renal or iliac lesions, given the bowels decreased tolerance for ischemia compared with the kidney or lower extremities. Extension of the trailing stent edge further into the aortic lumen than used for treatment of ostial atherosclerotic lesions is advocated.⁵³ The aorta should be reassessed after treatment of large branch vessel obstructions, as improving outflow from one lumen can alter the flow dynamics to such a degree that a new dynamic obstruction within the aorta can occur. Some authors have advocated preferentially using the false lumen as the inflow for an ischemic vascular bed with branch vessel obstruction to avoid such a result.⁴⁷

STENT GRAFTS

Thoracic stent grafts were first utilized for the repair of aortic dissections slightly over a decade ago. Treatment

with covered stents is primarily aimed at sealing a primary tear within the thoracic aorta, decreasing flow through the tear into the false lumen and directing blood flow preferentially through the true lumen (Fig. 3H–J). The redirection of blood flow to the true lumen often relieves downstream dynamic obstructions. As opposed to fenestration, however, stent grafting can also lead to thrombosis and remodeling of the false lumen, which may decrease the risk of subsequent aortic rupture or late aneurysmal dilatation.⁵²

Stent grafts deployed for the treatment of aortic dissections should extend 20–40 mm beyond the primary intimal tear site in both the proximal and distal directions. Distal aortic tears are left uncovered and allowed to remain open for several reasons. First, the small tears will allow for continued perfusion of branch vessels supplied by the false lumen. Second, these distal tears are usually of sufficiently small size that they do not allow sufficient pressurization for true lumen obstruction or aortic rupture. Third, minimizing initial overall graft length decreases risk of paraplegia; if thrombosis of the false lumen does not occur additional graft segments can be inserted at a later time.^{48,54–56}

Stent graft placement can be limited in certain individuals by excessive aortic tortuosity, intramural thrombus, or calcification. Additionally, small femoral or iliac vessels can limit the use of aortic stent grafts in some patients. Another consideration is the location of the proximal landing zone. If the primary intimal tear is within 20 mm of the left subclavian artery origin, coverage of the origin by the stent graft may be necessary to ensure an adequate seal (Fig. 3G). The contralateral subclavian and vertebral arteries as well as the circle of Willis should be studied. Selective revascularization of the left subclavian artery when there is an incomplete circle of Willis or if the left vertebral artery is dominant has been advocated rather than universal revascularization, especially considering the urgent/emergent need for the aortic repair.

No prospective trial evaluating outcomes of patients with complicated dissections treated with open surgery versus endovascular repair has been or will likely be performed. However, retrospective analysis of patients within the IRAD registry undergoing endovascular or open repair of complicated type B dissections demonstrated lower in-hospital mortality (10.6% vs 33.9%) and neurologic/ischemic composite morbidity (20.8% vs 40.0%) in the endovascular group as compared with the open surgical cohort.¹¹ Given the paucity of long-term data, particularly regarding the use of endografts in acute aortic syndromes, caution is urged in employing endovascular treatment methods in young patients who are otherwise good operative candidates. Similarly, patients with connective tissue disorders may be unsuitable for endograft repair given the diffuse abnormality of their vasculature, with

increased predisposition to further dissection and aneurysmal dilation.⁴³

In addition, no trial comparing outcomes of complicated type B dissections treated with stent-graft procedures and fenestration has been performed. However, stent-graft procedures have gained popularity, likely in part due to the relative simplicity and speed of the procedure compared with fenestration, as well as increased familiarity with the techniques and devices used in their applications for treating aneurysmal disease. Though head-to-head comparisons are lacking, hemodynamic models suggest that decreasing flow into the false lumen is more effective in treating malperfusion states compared with fenestration.⁴⁷ Analysis of complicated type B dissections from the IRAD database reveals that malperfusion states were relieved in 9 of 18 patients treated with endovascular fenestration and 16 of 17 patients treated with stent graft.¹¹ Given the theoretical advantages of stent graft therapy, DiMusto et al suggested an algorithm for treatment of aortic dissections in which endograft therapy was used if a suitable entry tear was present, with fenestration reserved for cases where malperfusion is present and there is no entry tear amenable to endograft treatment.⁵²

Endovascular repair in addition to medical therapy is now accepted for the treatment of complicated type B aortic dissections. Uncertainty exists over the use of endovascular techniques in the treatment of uncomplicated acute aortic syndromes of the descending thoracic aorta. The risk/benefit profile has favored medical management of uncomplicated type B intramural hematomas and dissections compared with open surgical therapy. Yet rates of late aortic complications between 25–50% have been reported in patients treated with medical therapy alone.³⁸ It has been hypothesized that given the lower morbidity and mortality of endovascular repair, treatment of even uncomplicated dissections in the hope of reducing late aortic complications may be warranted. The INSTEAD (Investigation of Stent Grafts in Aortic Dissection) trial was designed to answer the question of whether aortic stent grafting in patients with subacute or chronic (>2 weeks from index dissection) uncomplicated type B dissections could reduce late aortic related morbidity and mortality.⁵⁷ Patients were randomized to optimal medical therapy versus optimal medical therapy plus endovascular treatment of the proximal aortic tear with a TalentTM stent graft (Medtronic Inc, Santa Rosa, CA). Patients treated with optimal medical therapy had overall and aortic-related death rates of 4.4% and 2.9% respectively, while the rates in the interventional group were 11.1 and 5.6%. The authors noted a lower than expected mortality rate among the optimal medical therapy group. However, significant aortic remodeling was noted in the intervention group, with 91% complete thrombosis at 2 years as

compared with 19% in the optimal medical therapy group ($P < 0.001$).

Although endovascular intervention failed to improve late outcomes compared with medical therapy in uncomplicated chronic type B dissections, results may differ in acute uncomplicated type B dissections. The dissection flap becomes thickened and develops an increasing number of fenestrations with time, decreasing the likelihood of thrombosis of the false lumen as a result of endograft coverage of the primary dissection tear.⁴³ One small case series showed increased success rates in endovascular treatment of acute dissections compared with chronic dissections.⁵⁸ However, a recent meta-analysis demonstrated a higher complication and mortality rate after thoracic endovascular aortic repair (TEVAR) in acute dissections compared with chronic dissections.⁵⁹ More definitive data on the role of endograft therapy for acute uncomplicated aortic dissections will be forthcoming as results from the ADSORB (European Study of Medical Management Versus TAG Device and Medical Management for Acute Uncomplicated Type B Dissection) trial become available. This randomized trial will compare best medical therapy to best medical therapy plus implantation of a GORE[®] TAG[®] endograft in patients with acute (<2 weeks of symptoms) uncomplicated (no evidence of malperfusion) type B dissections. Enrollment for this trial began in August 2008 and the study completion date is listed as December 2012.⁶⁰

Stratification of patients into type A versus type B, acute versus chronic, and complicated versus uncomplicated alone may not be sufficient to determine appropriate candidates for endovascular treatments. Recently, studies have demonstrated potential high risk features of uncomplicated type B dissections that may portend an increased risk of aneurysmal degeneration. Song et al studied 100 consecutive patients with aortic dissections. Forty-nine of these had type B dissections with mean computed tomography (CT) follow-up of 3 months. Twenty-eight percent of these patients developed aneurysmal degeneration (>6 cm). However, the overall initial aortic diameter was not an independent predictor of eventual aneurysmal dilatation. Interestingly, the false lumen diameter measured at the upper descending aorta was highly predictive. Those patients with a false lumen diameter of >22 mm were at significantly higher risk for aneurysmal formation (42% vs 5%, $P < 0.001$). This would suggest that patients with type B dissections, who are otherwise uncomplicated but have increased false lumen diameter, may represent a subset of patients in which early endovascular management may be appropriate.¹²

Intramural Hematoma

Several factors make endovascular stent grafting particularly well suited to the treatment of IMH. Patients who

present with IMH tend to be older and have more significant medical comorbidities than their counterparts presenting with traditional dissections. Additionally, the more focal nature of IMH compared with dissection allows for coverage of the entirety of the abnormal aorta in a higher percentage of case.⁵⁶

Two separate mechanisms have been postulated for IMH: rupture of the vasa vasorum leading to hemorrhage within the media, and extension of hemorrhage into the aortic wall from small atherosclerotic ulcers or intimal tears often located near the ligamentum arteriosum/distal arch. In cases associated with an intimal tear, the intramural hemorrhage can extend within the aorta in an antegrade or retrograde fashion. Some authors advise treating only IMH related to intimal tears in an endovascular fashion. Not surprisingly, these authors advocate using the shortest graft possible to safely cover the entry tear (100–170 mm) therefore, minimizing the risk of paraplegia.⁶¹ The mechanism of IMH formation has been used as a rationale by some to treat type A IMH with intimal tears within the descending aorta with descending thoracic endografts rather than the more conventional ascending aorta/arch replacement, though data for this treatment paradigm is limited.⁶² Other groups do not differentiate among the causes of IMH in patient selection, but advocate treatment of a longer segment of aorta (200–250 mm) to cover as much abnormal vessel as possible with the goal of preventing late complications by reducing wall stress.²²

Penetrating Atherosclerotic Ulcer

Similar to IMH, PAU usually affects a more focal region of the aorta compared with aortic dissection. The age of patients at presentation tends to be older than either dissection or IMH, and patients tend to have more diffuse and severe atherosclerotic disease than in the other entities. Again, these factors make endovascular treatment options preferable to open surgery given the less invasive nature of the initial procedure and the increased inherent risks for open surgery in this population.²²

Given the focal nature of the abnormality in PAU, a single short stent graft can often be used to completely exclude the lesion (Fig. 5). Given the increased severity of atherosclerosis in PAU patients, the risk of a procedure-related access site or thromboembolic complications is higher as compared with patients with dissection or IMH. Therefore, minimal manipulation of the guidewire or delivery system in the arch is imperative. Additionally, consideration should be given to preferential access of the iliac arteries or the aorta from a retroperitoneal approach if the femoral arteries are diminutive or excessively diseased.^{22,63,64}

Data regarding the success of endografting in the treatment of PAU is mostly limited to single-center small case series. The available data were summarized by Eggebrecht et al in 2009, who reported high overall technical success rates (98%), and low rates of in-hospital mortality (7%), neurologic complications (4%), secondary procedures (5%), and subsequent aorta-related mortality (2%).²²

CONCLUSION

The optimal treatment of patients with AAS remains a challenging clinical dilemma. It is imperative to appreciate the spectrum of clinical, anatomic, and temporal manifestations within the pathology of AAS. Previous treatment algorithms stratify thoracic aortic pathologies strictly as type A versus type B. Subsequent management was dictated based on this classification with emergent surgical management indicated for type A pathologies and best medical therapy mandated for type B pathologies. The advent of minimally invasive endovascular techniques has the potential to shift the current treatment paradigm with treatment no longer limited to best medical therapy versus surgical repair alone. The current challenge is to determine the patient population best suited for endovascular treatment rather than best medical therapy alone. In some instances, endovascular management may replace surgical repair. In contrast to uncomplicated acute type A pathologies, uncomplicated type B pathologies have a relatively favorable early prognosis without surgical intervention: the rate of complication with surgery is markedly increased when compared with medical management alone. However, it is clear that procedural morbidity and mortality is reduced with endovascular repair when compared with surgical repair. Conversely, patients with complicated type B pathologies have a very high likelihood of mortality and require emergent surgical or endovascular treatment.

It is important to note that although endovascular treatments have demonstrated improved morbidity and mortality compared with surgical treatments, they are not without risk of significant complication. The most significant of which is paraplegia. The relative occurrence of this catastrophic event appears to be less common with endovascular repair versus surgical repair and chronic versus acute pathologies. We have proposed a potential treatment algorithm for AAS in the acute setting with consideration made for disease location and the presence of complicating or high risk features (Fig. 6). Further studies will help clarify the role for minimally invasive treatments while continued refinement of these endovascular devices will only continue to improve the procedural success.

Figure 6 Proposed algorithm for the treatment of acute aortic syndrome. Best medical therapy: close intensive care unit monitoring, target systolic blood pressure (SBP) 100–120 mm Hg, beta blockade if permissible, heart rate (HR) < 60, and pain adequately controlled. High risk features of aortic dissection: false lumen > 22 mm at aortic isthmus on initial presentation imaging. Retrograde type A (“Retro-A”) dissection: a dissection arising from a primary tear in the descending thoracic aorta with retrograde extension into the aortic arch. Complicating features in acute aortic syndrome (AAS): pain or hypertension refractory to medical therapy, rupture or impending rupture, visceral or limb ischemia. Initial short-interval and subsequent regular follow-up imaging is indicated in all patients with AAS who are treated medically and with endovascular therapies. IMH, Intramural hematoma; PAU, penetrating atherosclerotic ulcer.

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