

Penetrating Aortic Ulcer and Intramural Hematoma

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Abstract Acute aortic syndromes include a variety of overlapping clinical and anatomic diseases. Penetrating aortic ulcer (PAU), intramural hematoma (IMH) and aortic dissection can occur as isolated processes or can be found in association. All these entities are potentially life threatening, so prompt diagnosis and treatment is of paramount importance. PAU and IMH lesions in the Stanford Type A distribution often require urgent open surgical repair. Lesions in the Stanford Type B distribution may be managed medically in the absence of symptoms or progression; however, a low threshold for endovascular or surgical treatment should be maintained. This review summarizes the clinical presentation, epidemiology, diagnosis, indications for treatment and endovascular strategies in patients with PAU or IMH.

Keywords Penetrating aortic ulcer · Intramural hematoma · Acute aortic syndrome

Introduction

Acute aortic syndrome (AAS) is characterized by disruption of the aortic media to a varying degree (Fig. 1). The incidence of AAS is estimated on 2.6–3.5 cases per 100,000 person/year [1]. There is some clinical overlap between penetrating aortic ulcer (PAU), intramural hematoma (IMH) and aortic dissection. One in eight patients with acute aortic dissection has a PAU or IMH [1]. The diagnosis is largely based on imaging. Risk factors include hypertension, male gender, tobacco use, atherosclerosis, prior aortic operations, catheter-based interventions, bicuspid aortic valve and connective tissue disorders. Trauma to the aorta with intimal laceration may also be considered as AAS with similar clinical characteristics.

Penetrating Aortic Ulcer

PAU is a focal disruption in the arterial intima and elastic lamina extending into the media. This likely results from erosion of an atherosclerotic plaque and inflammatory changes in the aortic wall [2–5]. While the elastic and muscular portions of the wall lose strength, the wall calcification and inflammation are thought to hamper the progression of hemorrhage in isolated PAU, leading to a very focal dissection [1, 6]. The clinical course of PAUs is variable; these can remain stable, enlarge or progress to IMH, dissection, pseudo-aneurysm or even aortic rupture if the ulcer breaks through the adventitia (Fig. 2).

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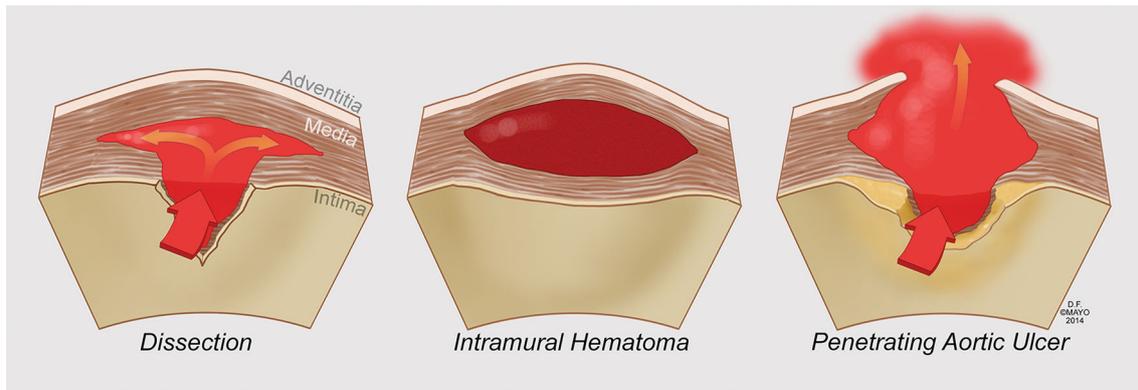
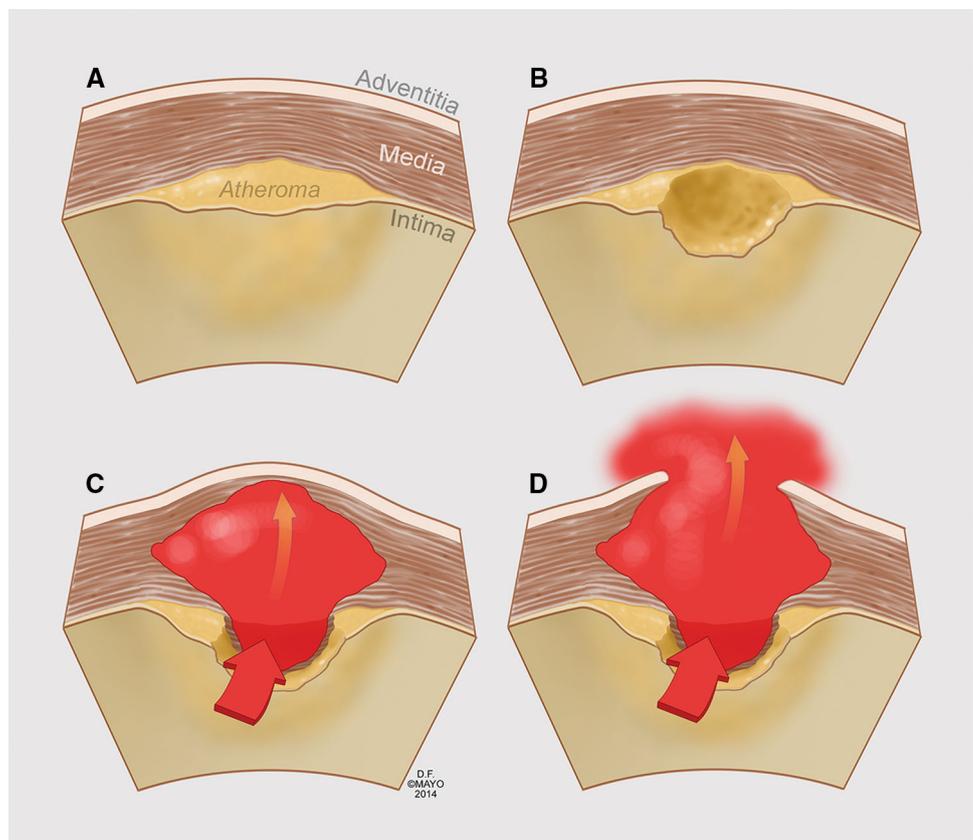


Fig. 1 Acute aortic syndromes can have overlapping features that include dissection, penetrating aortic ulcer and intramural hematoma. These three conditions can occur with overlapping clinical,

radiological and anatomic features. *Used with permission of Mayo Foundation for Medical Education and Research. All rights reserved*

Fig. 2 Progression of penetrating aortic ulcers starting with atheroma (A), plaque disruption (B), ulcer (C) and rupture of the aortic wall (D) leading to formation of a contained hematoma, pseudoaneurysm or frank aortic rupture. *Used with permission of Mayo Foundation for Medical Education and Research. All rights reserved*



Epidemiology

The first descriptions of PAU are from Shennan [7]. However, it was not until 1986 that Stanson and colleagues from the Mayo Clinic recognized PAU as a unique condition [5]. The true incidence of PAUs is unknown as some are asymptomatic and found only incidentally on imaging. Studies have estimated that isolated PAUs occur in 2.3–7.6% of cases of AAS [3, 8]. Typically, PAUs occur in the setting of advanced atherosclerosis and severe intimal

disease. Therefore, patients with PAUs are more commonly older and have more cardiovascular risk factors and diffuse atherosclerosis compared to those with aortic dissection, who are younger and often only have hypertension as comorbidity [9, 10]. PAUs can also be seen in younger patients, but often in the setting of a connective tissue disorder [11]. Men are more commonly affected than women.

PAUs can be found throughout the aorta but are most common in the descending thoracic aorta (Fig. 3 and

Fig. 3 Seventy-eight-year-old male patient with large penetrating aortic ulcer in the proximal thoracic aorta. The planned proximal landing zone for endovascular repair (Figs. 4 and 5) is outlined with red line

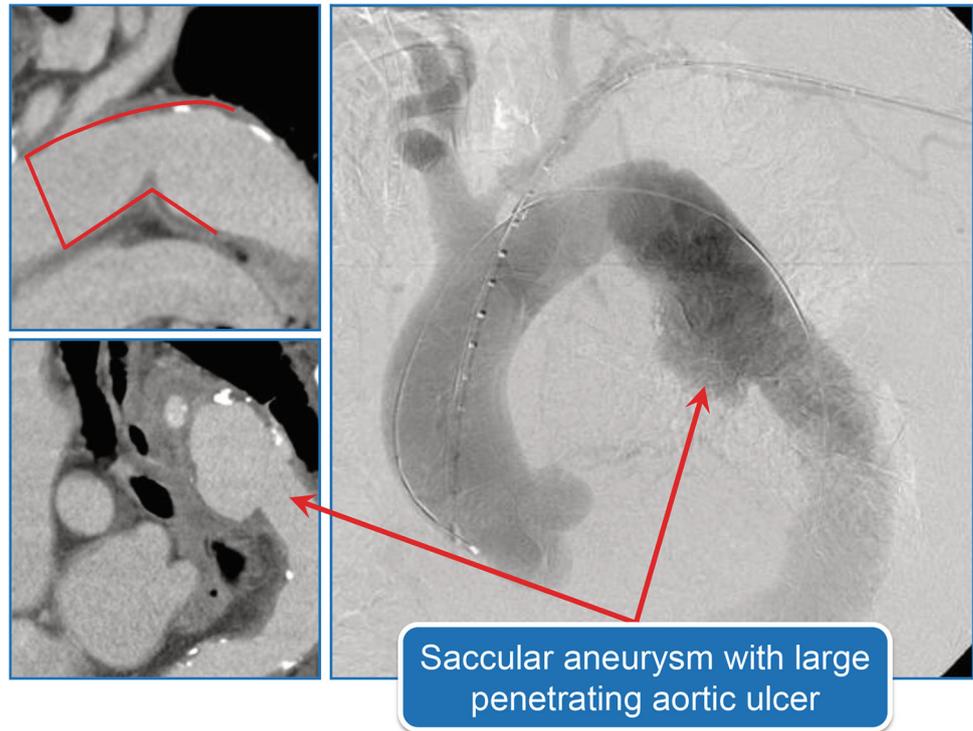


Table 1 Early outcomes of contemporary clinical series of endovascular repair of penetrating aortic ulcers

References	N	PAU location			Adjunctive debranching	30-day mortality	Stroke	Spinal cord ischemia
		Thoracic	Abdominal	Both				
Schoder et al. [65]	8	100%	0	0	0	0	0	13%
Kos et al. [66]	10	100%	0	0	–	0	0	10%
Tsuji et al. [67]	4	0	100%	0	0	0	0	0
Eggebrecht et al. [68]	10	80%	20%	0	0	0	0	0
Demers et al. [69]	26	100%	0	0	4%	12%	4%	0
Brinster et al. [70]	21	100%	0	0	0	0	0	0
Eggebrecht et al. [71]	22	73%	9%	18%	0	0	5%	0
Piffaretti et al. [72]	13	0	100%	0	0	0	0	0
Dalainas et al. [73]	18	89%	11%	0	0	0	0	0
Geisbusch et al. [61]	48	71%	25%	4%	–	6.2%	4%	0
Botta et al. [62]	18	100%	0	0	5%	11%	0	0
D'Souza et al. [74]	20	100%	0	0	5%	0	0	0
Clough et al. [75]	3	100%	0	0	–	0	–	–
Palombo et al. [76]	16	81%	19%	0	25%	6%	0	6%
Czerny et al. [77]	72	96%	1%	3%	35%	4%	3%	1%
Mestres et al. [78]	22	100%	0	0	27%	5%	0	5%
Georgiadis et al. [79]	19	0	100%	0	0	5%	0	0
János et al. [43]	63	86%	0	14%	5%	8%	0	0

Table 1). Nathan and colleagues reported the largest series to date of 388 PAUs in 315 patients [12]. The ulcer was located in the descending thoracic aorta in 62%, the

abdominal aorta in 31% and in the aortic arch in 7%. Reports of PAUs in the ascending are less frequent, likely because this area of the aorta is less affected by

Table 2 Late outcomes of contemporary clinical series of endovascular repair of penetrating aortic ulcers

References	N	Endoleak	Reintervention	Related mortality	Follow-up (months)
Schoder et al. [65]	8	13%	0	13%	14
Kos et al. [66]	10	20%	–	10%	9
Tsuji et al. [67]	4	0	0	0	14
Eggebrecht et al. [68]	10	10%	20%	0	24
Demers et al. [69]	26	14%	4%	4%	51
Brinster et al. [70]	21	0	0	0	14
Eggebrecht et al. [71]	22	5%	9%	0	27
Piffaretti et al. [72]	13	0	0	0	24
Dalainas et al. [73]	18	6%	0	0	41
Geisbusch et al. [61]	48	23%	8%	–	31
Botta et al. [62]	18	17%	11%	0	22
D'Souza et al. [74]	20	15%	5%	0	24
Clough et al. [75]	3	–	–	0	36
Palombo et al. [76]	16	6%	0	13%	16
Czerny et al. [77]	72	4%	1%	1%	42
Mestres et al. [78]	22	14%	14%	5%	53
Georgiadis et al. [79]	19	5%	11%	0	33
Jánosi et al. [43]	63	6%	19%	0	45

atherosclerotic disease [13–15]. PAUs in the ascending aorta are more frequently associated with acute complications such as IMH or rupture [16]. PAUs may be solitary or found in more than one location in the aorta [17].

While many PAUs are found incidentally, some are acutely symptomatic. In the study by Nathan and colleagues, 18% of patients presented with chest or abdominal pain [12]. The pain associated with PAUs is thought to be due to the rapid stretching of the aortic adventitia resulting in stimulation of the aortic nerve plexus [18, 19]. Thoracic PAUs are more likely to be symptomatic than those found in the abdominal aorta [12]. Rarely, PAUs develop thrombi on the surface of the ulcer, which may result in distal embolization. These are more frequently abdominal PAUs rather than thoracic [5, 20, 21].

PAUs vary in size with reports showing depths ranging from 4 to 30 mm and diameters ranging from 2 to 25 mm [12, 14]. Frequently, PAUs are associated with saccular aneurysms or pseudo-aneurysms and with IMHs. IMHs with associated PAUs portend worse outcome than isolated IMHs [22]. Studies have shown that a subset of PAUs progress over time [10, 12]. Larger ulcer diameter, greater ulcer depth and increasing pleural effusion are associated with progressive disease [22]. Symptomatic PAUs are also more likely to show radiographic progression [12]. The rupture rate for symptomatic PAUs is higher than that for aortic dissection, with some studies reporting rupture rates as high as 38% [3, 16]. Clinical data on late outcomes are limited to a few reports (Table 2), but endovascular repair has been established as the first line of treatment in patients who meet anatomic criteria for stent-grafts (Figs. 4 and 5).

Rate of endoleaks and re-interventions are similar to those reported for TEVAR for other indications, ranging from 0 to 13% and 0 to 20%, respectively (Table 2).

Intramural Hematoma

IMH, described first by Krukenberg in 1920, is defined as bleeding into the arterial wall from the vasa vasorum or from small tears in the intima of the aorta (Fig. 1) [23]. The resulting hemorrhage propagates along the media of the artery for a variable distance, but instead of forming two channels of free flowing blood as in an aortic dissection, the blood in the space between arterial layers clots to become a hematoma. While it was initially thought that IMHs, by definition, occurred without a tear in the arterial intima, some studies have suggested that small intimal tears do occur (Fig. 6) which are undetectable by classic imaging studies [24, 25]. The resulting hematoma can lead to infarction of the aortic wall, which may contribute to the development of a later aortic dissection or aortic aneurysm [26].

Epidemiology

IMHs account for 5–20% of the cases presenting with AAS with incidence approaching 30–40% in Asian studies [22, 27, 28]. Like patients with PAUs, those with IMHs are typically older than patients with aortic dissection [26, 29]. Patients are more likely to be male and more likely to have chest pain than those with aortic dissection [29]. Patients

Fig. 4 Artists representation of patient illustrated in Fig. 3 before endovascular repair (A) and after stent-graft coverage with a chimney graft into the left subclavian artery (B). Used with permission of Mayo Foundation for Medical Education and Research. All rights reserved

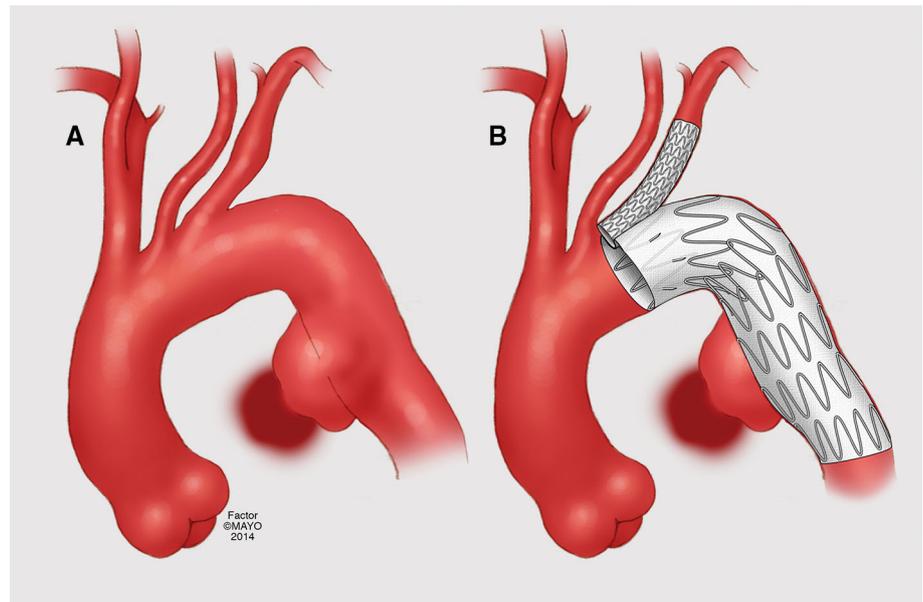
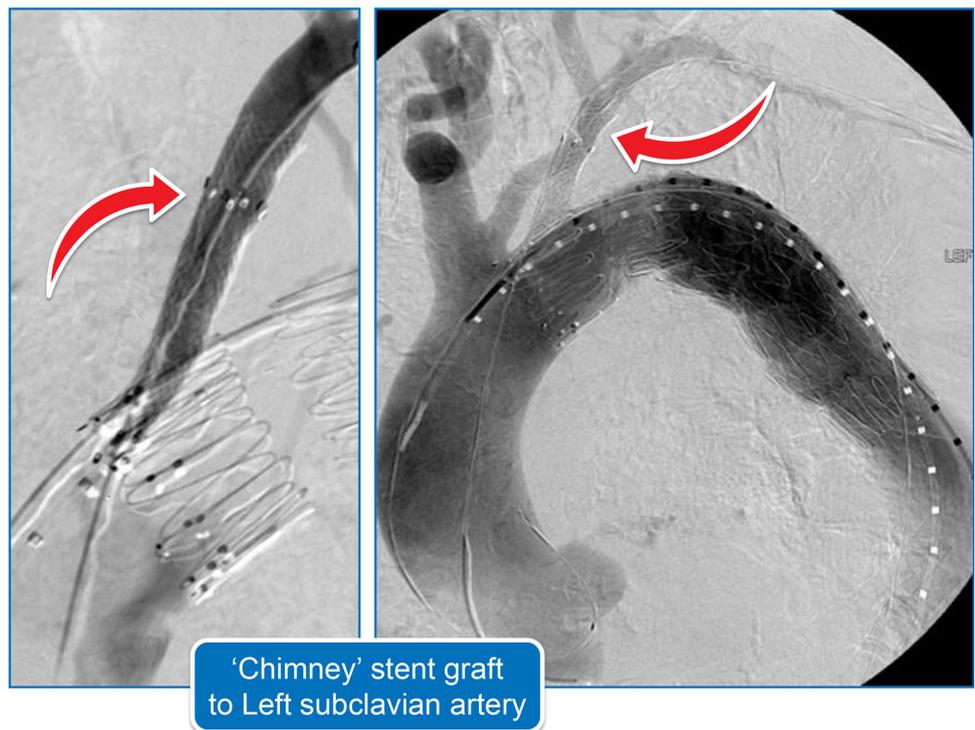


Fig. 5 Completion angiography of patient illustrated in Figs. 3 and 4 demonstrates patency of chimney and thoracic stent-grafts with no evidence of endoleak



with IMHs also have a lower risk of end-organ malperfusion or lower extremity ischemia although this has been described in literature [29, 30]. Like PAUs, IMHs are most frequently found in the descending thoracic aorta and are less likely to extend to the abdominal aorta. IMHs can also be seen in the ascending aorta, especially in Asian populations, or may extend to the ascending aorta in a retrograde fashion from the descending aorta [31]. However, the

entry tear is often invisible in IMH and the direction of expansion of the hematoma is difficult to define without sequential imaging. Type A IMHs involve the ascending aorta or the aortic arch (30% and 10% of cases, respectively), and Type B hematomas are restricted distal to the left subclavian artery (LSA) (60–70% of cases) [32].

The IMH has a variable course (Tables 3 and 4). It may regress or reabsorb in up to 100% of patients that undergo

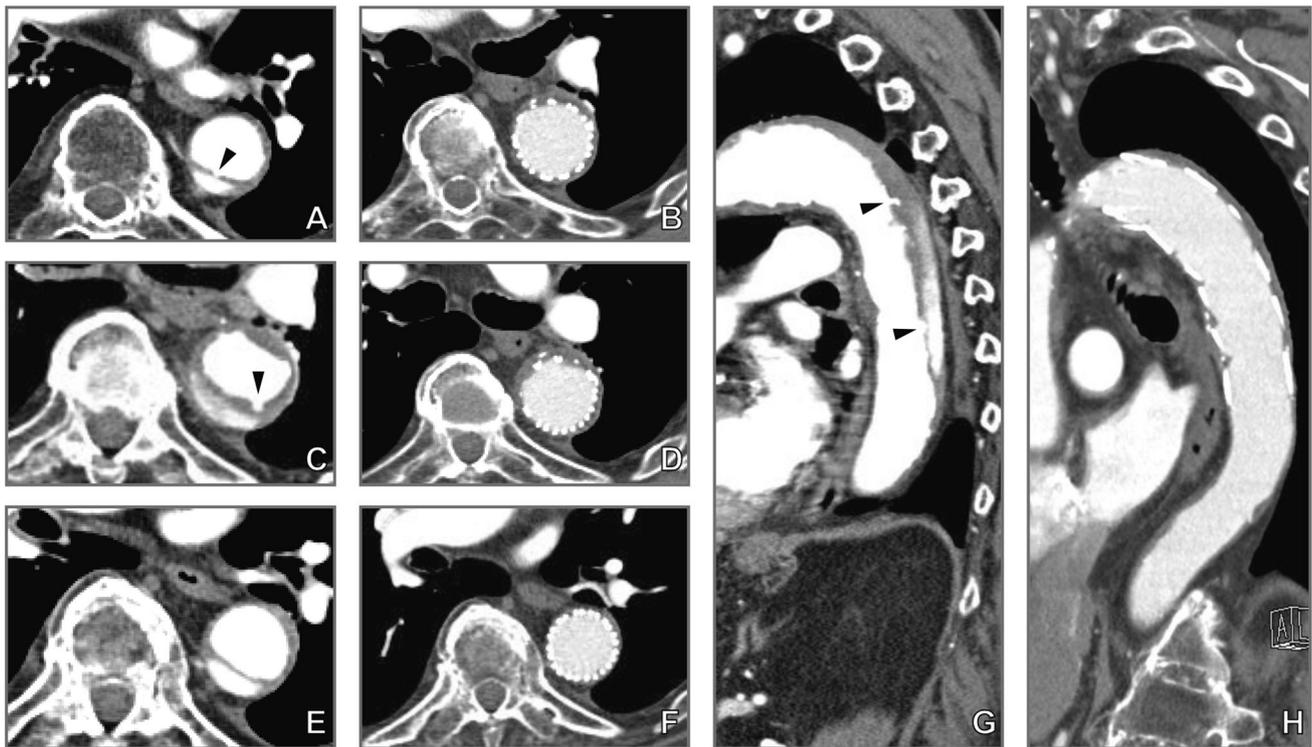


Fig. 6 Preoperative (A, C, E, G) and postoperative (B, D, F, H) computed tomography angiography after endovascular of complicated penetrating aortic ulcer with acute intramural hematoma and

focal dissection. Note on axial and coronal views the small penetrating ulcers (black arrowheads). Complete remodeling occurred after stent-graft placement (B, D, F, H)

endovascular treatment. In 28–47% of patients, IMH progresses to acute aortic dissection. Aneurysm formation or contained ruptures have been seen in 20–45% of patients [22]. Predictors of complications are recurrent or persistent pain despite adequate medical treatment, presence of PAU or ulcer like projections in the involved segment, ascending aortic involvement, associated organ ischemia, aortic diameter ≥ 50 mm, enlarging aortic diameter, progressive maximum aortic wall thickness (> 11 mm), recurrent pleural effusion and difficult blood pressure control [22, 31, 33–35]. Younger age, hematoma thickness of less than 1 cm and aortic diameter of less than 4–4.5 cm have been shown to be associated with favorable outcome based on retrospective studies but have not been reproduced in others [22, 27, 35–37]. IMHs in the Stanford Type A distribution have classically been treated by urgent surgical repair due to concerns about progression to dissection or rupture. Von Konolitsch reported an early mortality rate of 55% in patients with Type A IMH managed medically in comparison with 8% of patients managed surgically [33]. An analysis of the International Registry of Acute Aortic Dissections (IRAD) registry reported that the in-hospital mortality rate for IMHs was 7%. Aortic enlargement or aneurysm formation was less common in isolated IMH as compared to classic aortic dissection (39 vs. 61%) [29].

Diagnosis

Patients often present with non-specific chest or back pain. The location of the pain initially correlates with the site of initial aortic disruption but can change as the pathology progresses along the length of the aorta. If aortic branches are involved, the pain can radiate [38]. Pain may also correlate with the location of end-organ malperfusion. An electrocardiogram (ECG) and chest X-ray are recommended in all patients during the initial evaluation. The ECG is abnormal in approximately 60% of patients with aortic dissection, but this is non-specific [39]. The ECG results in isolated IMHs and PAUs have not been reported, while the chest X-ray may show a widened mediastinum, displaced aortic calcifications or a pleural effusion, in 20% of patients with AAS the chest X-ray appears normal [39]. Patients should have a troponin level obtained to rule out acute coronary syndrome. Some studies have shown a correlation between D-dimer level of greater than 500 $\mu\text{g/L}$ and the severity and extent of the AAS, but this is non-specific and should not be utilized to make the diagnosis [40–43].

PAUs and IMHs are most commonly diagnosed with a contrast-enhanced computed tomography angiogram (CTA, Fig. 7). A CTA can evaluate the entire aorta and can localize the site of the intimal flap, presence of

Table 3 Early outcomes of contemporary clinical series of endovascular repair of intramural hematomas

References	N	IMH Location			Adjunctive debranching	30-day mortality	Stroke	Spinal cord ischemia
		Thoracic (%)	Abdominal	Both				
Grimm et al. [63]	8	100	0	0	0	0	0	0
Monnin-Bares et al. [80]	15	100	0	0	0	0	0	0
Manning et al. [81]	7	100	0	0	0	29%	14%	0
Li et al. [82]	33	100	0	0	0	0	0	0
Lavingia et al. [83]	44	100	0	0	20%	5%	0	5%
Bischoff et al. [84]	28	43	0	57%	18%	4%	0	11%

Table 4 Late outcomes of contemporary clinical series of endovascular repair of intramural hematomas

References	N	Endoleak	Decrease in hematoma	Re-intervention	Related mortality	Follow-up (months)
Grimm et al. [63]	8	0	100%	0	0	16
Monnin-Bares et al. [80]	15	7%	100%	13%	7%	21
Manning et al. [81]	7	–	–	0	0	31
Li et al. [82]	33	0	100%	0	0	28
Lavingia et al. [83]	44	–	100%	11%	0	26
Bischoff et al. [84]	28	–	100%	21%	0	32

hemopericardium and hemothorax as well as extent of hematoma and presence of any aortic fenestrations. The scan should be carried from at least 3 cm above the aortic arch to the level of the femoral vessels to completely assess the entire aorta and its main branches. In order to accurately measure the aortic annulus, the aortic sinus and the proximal ascending aorta, an ECG-gated CTA is highly recommended [44].

CTA features of PAUs are an aortic out-pouching or crater in the setting of intimal calcification and severe atherosclerotic disease. These can be associated with IMH [45, 46]. The edges of the ulcer are typically irregular or jagged. However, it may be difficult to determine the extent of intimal penetration, particularly in the setting of extensive, calcific atherosclerotic plaque. IMH is characterized by a smooth, high-attenuation crescent-shaped thickening of part of the aortic wall measuring greater than 5 mm in diameter and that extends in a longitudinal, non-spiral fashion. In an isolated IMH, no intimal tear is identified and no two-lumen flow is seen. IMH may be more difficult to diagnose than a PAU or aortic dissection. The IRAD showed that initial imaging of IMH may be negative in more than 12% of patients, requiring repeat imaging at a later time [26]. IMH can also be mistaken for a mural thrombus or a dissection with a thrombosed false lumen. IMH is also often associated with a peri-aortic hematoma, which is blood outside the aortic adventitia. The presence

of a peri-aortic hematoma and pleural effusion can make it difficult to differentiate between the outer wall of the IMH and rupture of the aortic adventitia [29]. While the presence of a peri-aortic hematoma is a poor prognostic sign and has been associated with the risk of rupture, the presence of pleural effusion may represent reactive fluid collection and is not associated with poor outcomes unless it increases in size.

While the inner wall of an IMH is regular and smooth just deep to the bright, echo-dense intima, mural thrombi are irregular and located within the aortic lumen [47]. The calcification of the aortic wall is also displaced toward the center of the aorta, which is characteristic of both the IMH and the aortic dissection. However, aortic dissections often have a spiral formation, while IMHs are more longitudinal with a constant circumferential relationship with the aortic wall [11]. If a non-contrast CT scan is obtained, an IMH may be detected by its higher attenuation compared to intraluminal blood with Hounsfield units of 40–70. The combination of a non-enhanced and a contrast-enhanced acquisition yields good sensitivity (as high as 96%) for the detection of IMH [32]. A relatively new concept denominated ‘triple rule-out’ utilizes an ECG-gated multi-slice contrast-enhanced CT to evaluate patients with acute chest pain in the emergency department for the presence of coronary artery disease, pulmonary embolism or AAS [32, 48, 49].

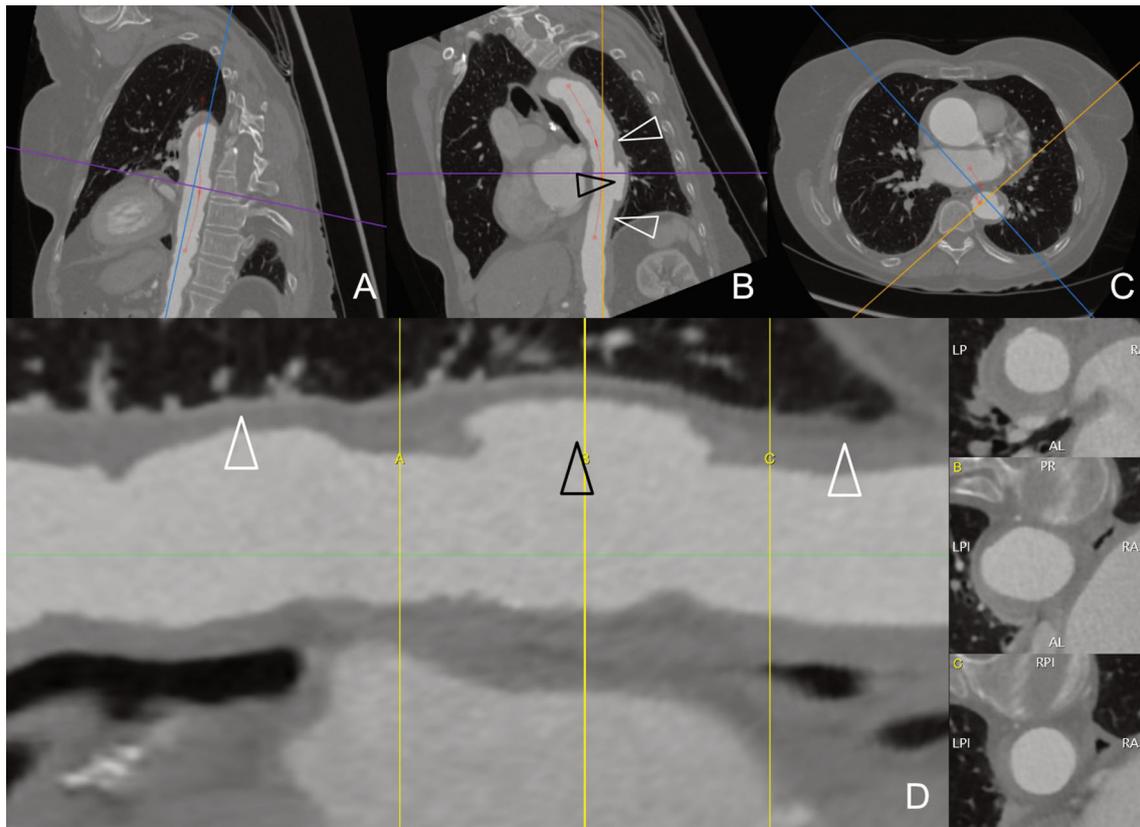


Fig. 7 Oblique sagittal (A), coronal (B), axial (C) and centerline of flow (D) computed tomography angiography reconstructions of an intramural hematoma (white arrowheads) with penetrating aortic ulcer (black arrowhead) in the descending aorta

Magnetic resonance imaging (MRI) is highly sensitive and specific for AASs. While non-contrast MRI can show the presence of an intimal flap, contrast-enhanced magnetic resonance angiography studies are needed for further define the pathology. MRI may detect small IMHs, which are not seen on CTA. Early in the evolution of IMH, the hematoma shows iso-intensity on T1 imaging and hyper-intensity on T2 imaging. However, after the first 1–2 days, oxyhemoglobin is converted to methemoglobin leading to a hyper-intense signal in both T1 and T2 MRI images, making MRI a valuable tool for assessing age of the IMH. This contrasts with mural thrombus which is hypo-intense or iso-intense in both T1 and T2 images [47]. Specific MRI techniques may also help differentiate IMH from atherosclerotic wall thickening, mural thrombus and thrombosed dissection when CT fails to do so [32, 44, 48]. In the setting of PAUs, MRI results are similar to those seen on CTA but is less affected by the presence of calcifications in the aortic wall. The utility of MRI is severely limited in the acute setting of aortic pathology. Nevertheless, MRI remains an excellent choice to follow PAUs or IMHs over the long term.

Transesophageal echocardiography (TEE) is useful especially in the ascending aorta and is highly sensitive at

identifying dissection in this region of the aorta [50]. TEE has more limited visualization of the proximal aortic arch due to bronchial air. An IMH in the thoracic aorta can be identified by TEE, but it can be difficult to differentiate the IMH from a mural thrombus. TEE can also visualize PAUs and can provide information on ulcer morphology. The study can be done at the bedside in the intensive care unit in a patient too unstable to transport to radiology [44, 51]. Transthoracic echocardiography (TTE) has more limited utility in AAS. However, it remains an important test of cardiac function and can identify aortic valve insufficiency and pericardial effusion, which may be associated with ascending aortic pathology. In the appropriate clinical scenario with a high index of suspicion, a positive TTE is sufficient to diagnose a Type A dissection, unstable PAU or IMH of the ascending aorta.

Currently, the initial evaluation of patients with suspected AASs that are hemodynamically stable includes ECG, troponin, creatinine, lactate, complete blood count, liver function tests, type and screen and CTA including the chest, abdomen, pelvis and extending to the common femoral arteries. Patients who are hemodynamically unstable should have ECG, chest X-ray, laboratory studies

and bedside TTE with consideration for a TEE, while preparing for operative intervention.

Treatment

Patients with Type A aortic dissection, rupture or end-organ malperfusion should primarily undergo immediate surgical intervention. The type of intervention (open or endovascular) is tailored on the basis of anatomic location, feasibility of stent-graft placement and clinical condition of the patient. For type A dissections, endovascular repair is possible in approximately 20–30% of the patients in highly specialized centers, but there is currently no widely available device for this indication. Patients with AAS who do not meet one of these criteria should be admitted to an intensive care unit. The first priority in treatment is to reduce the heart rate and blood pressure and therefore decrease the aortic wall stress and prevent progression.

Intravenous beta-blockers and calcium channel blockers are the first line of treatment with systolic blood pressure goals of less than 110 mmHg while preserving end-organ perfusion. The heart rate should be lowered to 60–80 beats per minute. Aggressive pain control is provided with intravenous narcotics to minimize the effects of the catecholamine release due to pain and resulting tachycardia and hypertension. Additional imaging studies are performed as necessary to better define the extent of the disease progression and associated comorbidities. Patients with stable hemodynamics, pain resolution and end-organ perfusion continue maximal medical therapy and undergo repeat CTA in 4–6 weeks. Those with ongoing pain, hemodynamic instability or signs of poor perfusion undergo immediate re-imaging and operative intervention.

Surgical and Endovascular Treatment Approaches

Ascending Aorta

Most experts agree that symptomatic PAUs or IMHs in the ascending aorta have a higher likelihood of progressing to dissection or rupture and should undergo surgical repair in an urgent or emergent basis [1, 52]. One series showed an early mortality rate of 55% in patients with Type A IMH managed medically versus 8% of patients managed surgically [33]. Hata reported 171 patients with acute Type A IMHs, of which 66 patients were treated medically. There was a 4% in-hospital mortality in the surgical group of which all deaths occurred in patients with complicated disease. In-hospital mortality was 26% in the medically managed group. In addition, five patients treated medically required emergency surgery because of cardiac tamponade,

five patients required mechanical ventilation as a result of heart failure or pneumonia, and four patients developed a stroke while hospitalized [53].

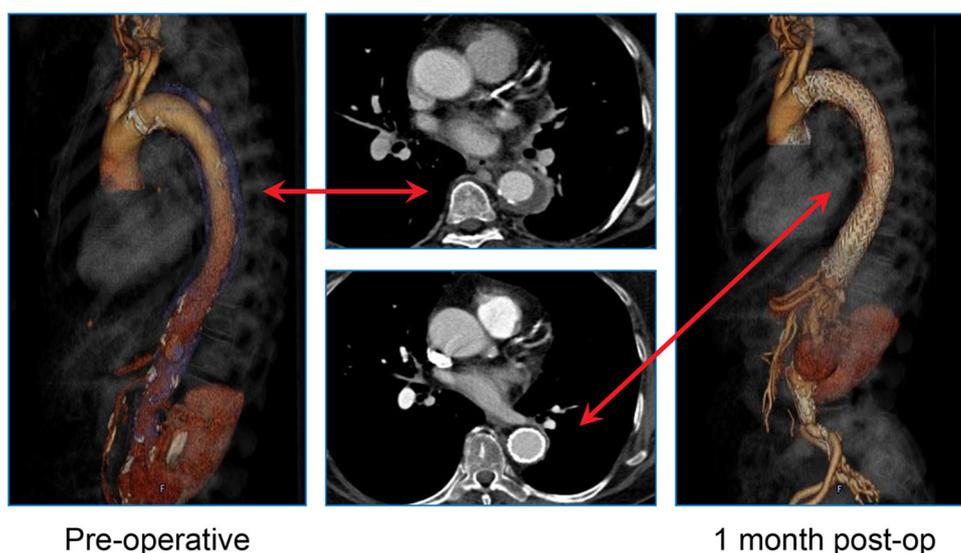
Treatment of uncomplicated Type A hematomas is somewhat controversial. Several studies have shown that Type A IMHs and aortic dissection with thrombosed false lumens may be successfully managed medically. Kaji reported in-hospital mortality of 7% and survival rates of 90% at 5 years after medical treatment of Type A IMH. However, 45% of patients developed disease progression to dissection or increases in size of the hematoma [54]. Watanabe presented 59 symptomatic patients with Type A acute IMHs and aortic dissections with thrombosed false lumens initially treated medically. Forty-six percent of patients developed aortic events including rupture in 2%, PAU enlargement in 19%, and recanalization of a thrombosed false lumen in 25%. All aortic events in this series occurred four or more days after presentation. Patient survival was 90% at 2-years [55]. Given the high complication rate with observation, open surgical repair remains the safest option, but Watanabe's results indicate that there is time to transfer stable patients to high volume centers for definitive surgical management. Optimal medical management with sequential imaging evaluation may be acceptable in patients with Type A IMH that are elderly or have severe comorbidities, especially in the absence of factors that predict complication [31].

Endovascular treatment for ascending aorta and arch is evolving. There are successful reports with devices been used outside the original indication for use, such as proximal cuffs, parallel grafts and surgeon modified devices [56–58]. Endovascular devices made specifically for the ascending aorta and the aortic arch are under investigation, and published data show acceptable results on the hands of experienced surgeons [51, 59]. Since open surgery provides good results, endovascular approach is currently being utilized for the treatment of patients that are deemed inoperable but have acceptable life expectancy and functional status.

Descending Aorta

Treatment guidelines for PAUs and IMHs involving the descending thoracic aorta are similar to what has been proposed for Type B aortic dissections. Patients with uncomplicated hematomas, incidental findings and those with small ulcers are treated medically. In symptomatic patients, time can be taken to maximize the medical condition and to plan the definitive repair, unless the patient is hemodynamically unstable or has evidence of rupture. As most patients with PAUs are typically of advanced age and have multiple medical comorbidities, care must be taken in selecting the appropriate treatment method. Refractory or

Fig. 8 Complete remodeling of the thoracic aorta after endovascular repair of acute intramural hematoma with stent-graft coverage



recurrent pain in the setting of PAU is one of the most important factors to consider when determining intervention [3]. Because of the high rate of progression in symptomatic PAUs, we recommend repair in patients who have persistent pain even if the blood pressure is controlled. In addition, the presence of saccular aneurysm or IMH in association with a hematoma portends worse prognosis and is an indication for repair. Other indications for intervention for type B IMHs are expansion of the hematoma despite medical therapy (rarely asymptomatic) and disruption of the intima with a CT demonstrating contrast enhancement. The 2017 European Society for Vascular Surgery guidelines on the management of thoracic aorta disease recommends that endovascular repair should be considered for complicated type B PAU or IMH (class IIa recommendation) [32].

Larger (diameter greater than 15–20 mm and neck greater than 10 mm) PAUs should also be considered for repair in select patients, based on some reports that indicate higher risk of disease progression [22, 43]. This recommendation has not been reproduced in other studies, and in our practice, if the ulcer is asymptomatic and has not enlarged, we continue to manage conservatively with serial imaging. If there is any sign of ulcer progression, enlargement or pain, repair is initiated.

As PAUs are typically focal, involve a relatively small portion of the aortic wall and occur in fragile patients, endovascular repair is typically considered the first line of treatment whenever possible. The same anatomic criteria for degenerative aneurysms are recommended, following strict recommendations for selection of healthy proximal and distal landing zones (Fig. 4). In-hospital mortality after endovascular repair is approximately 7–15% [60, 61]. Results of thoracic endovascular aortic repair (TEVAR) in

PAU patients compare favorably to larger clinical series of TEVAR performed for other indications, with a high rate of technical success, few neurologic complications and few requirements for additional procedures [60–62]. If primary endovascular repair is not possible or anatomically feasible, fenestrated procedures or hybrid procedures with debranching followed by endovascular repair should be considered. Open repair should be reserved for good risk patients unsuitable for endovascular interventions.

Grimm and colleagues identified small atherosclerotic plaque ruptures associated with IMHs using ECG-gated CT angiograms. One important aspect is that successful coverage of the original site of plaque rupture by the stent-graft is frequently associated with remodeling of the aorta and reabsorption of the IMH (Fig. 8), even if the hematoma is not entirely covered by the stent-graft and extends into the ascending aorta [63]. While it is unclear if all IMHs have such small ruptures in the intima, stent-grafts used for the treatment of IMHs should generally be longer than those used for PAUs in order to cover potential intimal disruption sites.

Patients who have extension of the aortic disease to the LSA requiring LSA coverage in TEVAR benefit from revascularization using carotid–subclavian bypass or transposition, parallel stent-graft, in situ fenestration or directional branch stent-graft [85]. Open surgical revascularization of the LSA is the most commonly utilized method. A systematic review including 2591 patients who underwent LSA coverage during TEVAR showed significantly lower perioperative stroke and spinal cord injury (SCI) rates in patients with LSA revascularization compared to those without [86]. Panneton and associates reported a series of 22 patients treated by in situ laser fenestration for LSA during emergency TEVAR with one

in-hospital death and one SCI [87]. Branched endografts remain investigational; the preliminary results of the Gore® Thoracic Branch Endoprosthesis showed 100% patency and zero stroke rate [88]. Parallel stent-graft (chimney) technique has also been utilized (Figs. 4 and 5) but remains limited by the risk of gutter endoleaks and lack of long-term data.

Treatment of acute patients deserves further consideration because of friable tissues and risk of retrograde aortic dissection or disruption. Care should be taken not to excessively oversize the device. The aortic lumen is slightly smaller during the acute phase of an IMH and then later enlarges as the hematoma resolves [64]. Balloon angioplasty should be avoided if at all possible because of risk of retrograde dissection. We and others recommend only performing balloon dilation of the stent-graft at areas of stent-graft overlap and avoiding balloon dilation at the proximal and distal landing zones to avoid disruption of the fragile aortic wall.

SCI is one of the most devastating complications of TEVAR. Although SCI is often a result of multiple insults, the most important risk factor is the extent of aortic coverage [89]. Standardized SCI prevention protocol should be applied in all high-risk patients undergoing TEVAR [90, 91]. These include permissive hypertension (mean arterial pressure \geq 80 mmHg) and selective cerebrospinal fluid drainage (baseline at 10 mmHg with maximum drainage of 20 ml/h). Neuromonitoring (somatosensory and motor evoked potentials or near-infrared spectroscopy) can be used to guide maneuvers designed to optimize spinal cord perfusion. Large sheaths are associated with limb and pelvic ischemia, which may aggravate SCI because of compromised collateral networks.

Conclusions

PAUs and IMHs are potentially life-threatening conditions which must be managed carefully in both the short and long-term in order to avoid complications. Lesions in the Stanford Type A distribution may need urgent open surgical repair. While lesions in the Stanford Type B distribution may be managed medically in the absence of symptoms or progression, a low threshold for surgical intervention should be maintained. Endovascular therapy should be the first line of treatment. Regardless of therapy, these patients require life-long follow-up.

Compliance with Ethical Standards

Conflict of interest Dr. Oderich has received consulting fees and grants from Cook Medical, W. L. Gore and GE Healthcare (all paid to Mayo Clinic with no personal income). The other authors have no conflict of interests to disclose.

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