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SPECIAL EDITION

Controversies in Surgery for Thoracic Aorta

How should we manage type B aortic dissections?

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Abstract Dissection of the descending aorta is a serious and potentially lethal event. Treatment options consist of medical therapy, open surgical replacement of the affected aorta and thoracic endovascular repair. In acute cases, medical treatment is started initially. When complicated, endovascular repair is generally considered as first choice treatment, except for connective tissue disorders where open surgery remains the standard. In stable, uncomplicated patients with risk factors for future aortic growth pre-emptive endovascular repair should be considered in the sub-acute phase of the dissection. The treatment strategy in chronic dissections is somewhat debated. Long-term results and aortic remodeling of endovascular repair are disappointing and open surgery remains the standard.

Keywords Type B aortic dissection · Endovascular surgery · Thoracic aorta · Aortic remodeling

Introduction

Dissection of the descending aorta, with the primary entry tear distal to the left subclavian artery, is a serious and potentially lethal event. Advancement in diagnostic and endovascular techniques in the last two decades have made this a highly debated and complex issue, with guidelines and recommendations continuously evolving. Nowadays, dissection of the descending aorta consists of a whole array of phenotypes and subgroups, of which clinical presentation and surgical management are variable and tailored specifically towards patients. Endovascular treatment in acute type B dissection, for instance, has shifted from a complication-specific indication to a pre-emptive approach in selected cases.

This article will give an overview of the current literature, define the different subgroups and its different treatment modalities and define an evidence-based approach on how to treat dissection of the descending aorta.

Classification

Traditionally aortic dissection is categorized according to anatomic location of the dissection. Stanford type A for a dissection involving the ascending aorta and type B for the descending aorta [1]. Debakey further classified this according to the location of the intimal tear and the extent of dissection. Type IIIa involves only the descending thoracic aorta, while in type IIIb the dissection extends below the diaphragm [2]. Furthermore, a simple time-related and clinical classification exists: acute (less than 2 weeks) versus chronic and complicated versus uncomplicated, respectively. Uncomplicated dissection has become a misnomer, because of newly identified subgroups who are susceptible for late (aortic related) morbidity and mortality. These classifications were valuable in the open surgical era, where the nature of the dissection, the role of aortic remodeling and long-term outcomes were less clear and the choice between surgical or conservative therapy had to be made.

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However, due to the widespread adaption of endovascular therapy, these classifications are somewhat clinically and surgically redundant.

The classification in time is important for two reasons. First off, historically Crawford had reported an autopsy series wherein 74% of deaths of both type A and B dissections occurred within 2 weeks, mostly due to rupture, aortic insufficiency and branch vessel obstruction [3]. The IRAD had highlighted that mortality and complications occur more frequently in the acute and subacute phase of dissection [4]. In 2013, a consecutive series of TEVAR (thoracic endovascular aortic repair) in complicated type B dissections showed that one in six patients had a life-threatening complication more than 2 weeks after onset of symptoms [5]. In the group defined as 'subacute' (15–90 days), 50% of complications were rapid aortic dilatation, whereas in the first 2 weeks rupture was the most important complication.

Second, the transition period wherein the dissection flap stabilizes and matures and the aorta retains its plasticity needs to be considered. It is well established that the dissection flap becomes more rigid and less mobile as time passes by and favorable aortic remodeling with true lumen expansion and false lumen thrombosis is less likely to occur after endovascular treatment of chronic dissections [6, 7]. Studies suggested that increased flap stability reduces complication rates after TEVAR, but could reduce success rate of aortic remodeling [8, 9]. For example, an early intervention would benefit aortic remodeling but the acutely inflamed aorta is also at high risk for rupture or retrograde dissection. As described by Desai et al., retrograde dissection was seen in 6.8% of patients treated in the early phase, compared with 0% in the subacute group (treated more than 2 weeks after the event) [10]. A recent retrospective analysis of 135 patients treated with TEVAR showed significantly lower procedural time, more conventional femoral access and fewer blood loss in the acute and sub-acute group compared with the chronic group [11].

As management depends highly on the initial presentation, the distinction between complicated and uncomplicated is important. In a retrospective contemporary study of 442 patients, uncomplicated dissections had an early mortality of 2.6%, whereas complicated dissections treated either medically, surgically or with TEVAR had an overall early mortality of 16.1%. These results are comparable with earlier studies [12]. This study also showed a 10-year survival of uncomplicated medically treated cases of 66.7% as compared to 48.2–48.5% in complicated medically or surgically treated cases [13]. The Penn classification (Table 1), already validated for type A dissections, subdivides these complications [14].

While the adoption of endovascular therapies have been rapidly expanding, trials have been reported with different classification systems, definitions and heterogeneous study populations with variability in endpoints as a result. The DEFINE investigators proposed a set of definitions to create uniformity in reporting [15].

As an extension thereof, a classification that includes all critical factors was created, so communications between emergency doctors, radiologists, cardiologists, intensivists and surgeons can be clear and uniform and a well-founded decision on treatment can be made. The DISSECT classification includes five factors: duration (timing of dissection), intimal tear, size of aorta, segmental extent of involvement, clinical complications and thrombosis of false lumen (Table 2) [16].

In the future, an even more detailed classification can be expected once biochemical markers and hemodynamical parameters of dissection are further explored and the distinction between complicated and uncomplicated will change to stable versus unstable. Uncomplicated patients at risk for late aortic problems will be identified and will be candidates for pre-emptive TEVAR.

Description of treatment modalities

Best medical treatment (BMT)

The treatment of descending aortic dissections in the acute phase is aimed at preventing complications. First by limiting extension of the false lumen and preventing branch-vessel malperfusion and second by reducing hemodynamic stress on the aorta and preventing expansion or rupture. Decreasing aortic wall stress and the force of left ventricular ejection can be achieved by rate control and pressure control. Intravenous beta-blockers are a first choice, as recommended by most guidelines, with a target pressure of 100–120 mmHg systolic and a target rate of <60 bpm (class I recommendation, grade C evidence) [17, 18]. As an adjunct, or in patients who do not tolerate beta-blockers, other intravenous antihypertensives can be used, for instance calcium channel blockers or vasodilators such as sodium nitroprusside or nitrates. These should be used with caution to avoid reflex tachycardia. In the acute phase, intensive monitoring with electrocardiogram, invasive blood pressure, central venous pressure, urinary output and arterial blood gas sampling are deemed necessary. Pain needs to be appropriately addressed with opiate analgesics.

As the IRAD database has shown, refractory pain is an important prognostic factor and should be taken very seriously as this can be a harbinger of impending rupture [12].

Once stabilized, a transition to oral antihypertensive agents follows, preferably beta-blockers and calcium channel blockers combined as these are associated with improved

Table 1 The Penn-classification of complications in acute dissection

Class A (uncomplicated)	Absence of branch-vessel ischemia or circulatory compromise
	Type 1: high risk for future complications
	Type 2: low risk for future complications
Class B (complicated)	Branch-vessel malperfusion with visceral, renal, lower-extremity, and/or spinal cord hypoperfusion
Class C (complicated)	Circulatory compromise
	Type 1: aortic rupture with hemorrhage outside the aortic wall, with/without cardiac arrest, shock and hemothorax
	Type 2: threatened aortic rupture typically heralded by refractory pain and/or hypoperfusion
Class BC (complicated)	Branch-vessel malperfusion combined with circulatory compromise

Table 2 The DISSECT classification

Duration	Acute: <2 weeks from initial onset of symptoms
	Subacute: 2 weeks–3 months
	Chronic: >3 months
Intimal tear (primary location)	Ascending aorta
	Aortic arch
	Descending aorta
	Abdominal aorta
	Unknown
Size of the aorta	Based on maximum trans-aortic diameter measured by center-line analysis in millimeters at any level within the dissected segment
Segmental extent of aortic involvement	Ranging from ascending aorta to iliac vessels and everything in between
Clinical complications	Complicated: aortic valve involvement, tamponade, rupture, branch vessel malperfusion (anatomic and clinical), progression of aortic involvement, uncontrollable hypertension, rapid expansion (>10 mm in the first 2 weeks)
	Uncomplicated
Thrombosis of aortic false lumen	Patent false lumen (evidence of flow or contrast) or
	Complete thrombosis within the following segments (ascending, arch, descending, abdomen) and
	Partial thrombosis within the following segments (ascending, arch, descending, abdomen)

survival and reduced aortic growth [19–21]. A tight heart-rate control may be very important; as shown by Kodama et al. [22] a heart rate of <60 bpm is associated with significantly fewer aortic events and less surgical requirement.

The role of ACE inhibitors is less clear. They have shown some benefit in the long term in the Marfan population and in some case series [23], however, in the IRAD registry no beneficial effect on survival was noted [19].

In the long-term smoking cessation, management of lipid profile with statins, control of hypertension (<140/90 mmHg) and rate control are important. Best medical treatment can at best *delay* the rate of aortic expansion and aortic-related events rather than inducing aortic remodeling and fails to prevent late aortic complications.

Results

Medically treated patients have a 90–100% in-hospital survival and a 5-year survival of 50–87%, illustrating the efficacy of current medical therapy [4, 11, 13, 24–27].

However, the conservatively treated dissected aorta will develop aneurysmatic dilatation or aortic rupture over the years. About 20–55% of conservatively treated patients develop an aneurysm after 5 years [4, 11, 27, 28]. This can be attributed to incomplete aortic remodeling.

The concept of post-dissection aortic remodeling is important as actors such as intimal tear size, number and location, true lumen versus false lumen ratio and false lumen hemodynamics have an impact on prognosis [20, 29]. Complete false lumen thrombosis (FLT) has consistently shown to be a negative predictor for aortic growth [28, 30–32].

Partial FLT, although debated, is considered a predictor of mortality and aortic growth [33, 34]. As learned from the INSTEAD and ADSORB trials, the morphological evolution in medically treated patients is less than ideal.

In ADSORB, only 3% of medically treated patients had complete FLT after 1 year. INSTEAD showed complete FLT after 2 years in only 19.4% as compared to 91.3% in patients treated with TEVAR [25, 35].

Open surgical treatment (OST)

Open repair of dissection of the descending aorta consists of replacing the aortic segment containing the primary entry tear, removal of the aneurysmatic part and redirect blood flow into the true lumen to correct malperfusion. Malperfusion, present in 10% of complicated cases, has been categorized into dynamic and static branch compromise. In dynamic obstruction, the true lumen is severely narrowed and the false lumen pushes the intimal wall into the vessel. Around 80% of perfusion deficits are dynamic and can be solved by replacing the aortic part with the primary entry tear or by covering this segment as is done in TEVAR [36]. In static branch compromise, the dissection flap extends into the ostium of the branch and a surgical or endovascular fenestration (with additional stenting) is required.

Through a left posterolateral thoracotomy in the fourth or fifth intercostal space, the proximal descending aorta is cross-clamped and the affected thoracic or thoracoabdominal aorta replaced. Prevention of visceral, renal and spinal cord ischemia is achieved by several protective measures. Distal aortic perfusion with left-heart bypass and sequential aortic clamping combined with cerebrospinal fluid drainage, intrathecal papaverine, moderate hypothermia and maximal reimplantation of intercostal branches T7–L1 are the adjuncts used in our center, as previously described [37]. Total extracorporeal circulation and deep hypothermic arrest can be used when cross-clamping is difficult to achieve.

In contrast to TEVAR, no anatomical constraints are encountered. However, this technique does not eliminate risk of aneurysmal degeneration of the distal uncorrected false lumen.

Currently, absolute indications for open repair are an acute complicated dissection not suitable for endovascular repair and patients with connective tissue disorders. This subpopulation has been excluded from most trials as the reliability of the stent graft in these fragile aortas has been questioned. A recent review of endovascular repair in Marfan syndrome showed acceptable morbidity and mortality but high reintervention rates and disappointing aortic remodeling [17, 38].

Results

Contemporary results of open surgery in the acute and chronic phase of dissection are important to consider, as numerous improvements in perioperative and operative techniques have been made. These results can act as a baseline to compare newer therapies such as TEVAR or hybrid procedures. Open surgery in the acute phase is only performed in complicated cases with limited indications, so only a few contemporary studies exist, as reviewed by Fattori et al. [39]. Early mortality is estimated at 17.5%, a stroke rate of 5.9% and paraplegia in 3.3%. In 2015, Afifi et al. published a retrospective series of 52 patients with early mortality of 11.5%, a stroke rate of 1.9% and paraplegia in 1.9% [13].

Open repair for chronic dissections has excellent results with early mortality rates of 5.6–21%, stroke rates of 0–13.3% and paraplegia in 0–16.4% [40, 41]. Open repair remains an extremely durable solution as reintervention rates are low (5.8–29%) [40]. In a retrospective series of 240 patients, there was a 10-year freedom for reintervention on the operated aortic segment of 94.3% and a 10-year freedom from reintervention on any aortic segment of 89.3% [42]. In a small series, Andersen et al. reported 0% reinterventions at 34 months after open surgery compared to 24% after TEVAR, of which half of these reinterventions were required to treat stent graft complications [43].

Thoracic endovascular aortic repair (TEVAR)

The goal of endovascular repair of the dissected aorta is to cover the primary entry tear and obliterate blood flow into the false lumen and as such achieve expansion of the true lumen and thrombosis and eventually collapse the false lumen. As described above, this concept of aortic remodeling has an impact on long-term prognosis.

Bilateral access of the common femoral arteries is acquired and introduction of a stiff wire in the true lumen to the level of the ascending aorta is made. The proximal landing zone is identified with aortography and transesophageal ultrasound (TEE).

The landing zone is a segment of non-dissected, non-aneurysmatic aorta of approximately 2 cm length (device-specific) where the stent is deployed. When the origin of the left subclavian artery (zone 2) is intentionally covered, vertebrobasilar insufficiency and subclavian steal should be considered in certain cases, although debated there is a tendency towards selective surgical revascularization. Patients with a large left vertebral artery or a right hypoplastic vertebral artery, a patent LIMA bypass or an incomplete circle of Willis will certainly need an extra-anatomic bypass, amongst others [44]. Furthermore, a recent study showed significantly fewer endoleaks and improved FL thrombosis after embolization of the covered left subclavian artery [47]. Perioperative TEE is also useful for ensuring true lumen cannulation, arch vessel patency and assessing the number of entry tears. Adequate sizing of the stent graft is important as a little oversizing is required to achieve sufficient radial force and adequate endoseal. The rate of oversizing is debated as this is associated with complications such as creation of stent graft-induced new entry (SINE) and retrograde dissections towards the

ascending aorta especially in the acute setting [45]. As learned from the MOTHER-registry retrograde, dissection was highly associated with oversizing (OR 1.14 per 1% increase in oversizing above 9%, $P < 0.0001$) [46]. Post-deployment dilatation of the stent graft is generally avoided in the acute setting for similar reasons although it has been advocated to ensure a better seal at the landing zones.

Fanelli et al. saw no difference in endoleaks and retrograde dissection comparing a group with and without post-deployment dilatation although a significantly higher rate of FL thrombosis was seen in the dilated group [47]. The length of the stent graft is determined by adequate coverage of the entry tear but avoiding too extensive coverage of the thoracic aorta as this brings an increased risk of ischemic spinal cord injuries. In chronic dissections, longer stents may be necessary.

A distal extension with bare metal stenting may provide structural stability in the true lumen and promote FL thrombosis while maintaining intercostal flow (PETTICOAT technique) [48].

Technical contra-indications for TEVAR can be classified as access related, such as calcified peripheral arteries or small, tortuous vessels, or anatomical, such as connective tissue disorders and inappropriate landing zones.

Results

As previously reviewed, the short-term results of TEVAR are excellent [26]. In acute complicated patients, the pooled early mortality rate is 10.2%, stroke occurs in 4.9% and spinal cord injuries were seen in 4.2%. In chronic dissections, electively treated with TEVAR, pooled early mortality is 6.6%, a stroke rate of 1.9% and spinal cord injuries occur in 1.5%.

Numerous studies have shown excellent aortic remodeling after TEVAR in the acute and subacute phase of dissection. As compared to BMT reduction in aortic diameter, true lumen expansion and false lumen thrombosis are significant [25, 49]. Complete false lumen thrombosis is seen in 80–90% of patients.

In chronic dissection, results are more heterogeneous but remodeling is consistently less favorable. Some argue the dissection flap has become too rigid and more fenestrations between the true and false lumen exist, for which multiple or lengthier stent grafts will be needed [7]. Behavior of the false lumen in the untreated visceral portion of the aorta is somewhat unpredictable in these cases.

Differences between limited (type IIIa) and extensive dissections (type IIIb) have been noted by Kang et al. [50]. In type IIIa chronic dissections, 78% had a complete FLT compared to only 13% in type IIIb.

In a small retrospective series for chronic type IIIb dissections, complete aortic remodeling was observed when all visceral vessels originated from the true lumen and there were fewer than three residual distal fenestrations, possibly identifying a subgroup suitable for successful TEVAR in chronic cases [51].

In all, there seems to exist a therapeutic window, situated between 2 weeks and 3 months, in which TEVAR delivers maximal aortic remodeling and gives a minimal risk of complications.

As seen in the INSTEAD-XL trial, 5-years aortic-related mortality in uncomplicated dissections is significantly lower for TEVAR than for BMT (6.9 vs. 19.3%, $p = 0.045$) and pre-emptive TEVAR turns beneficial at 5 years of follow-up (NNT = 13).

Aortic-related mortality after TEVAR is low despite a higher need for reinterventions. 29% of patients treated with TEVAR in chronic dissections and 54% of acute dissections needed a reintervention within 6 years, as seen in the MOTHER database, although no association between reintervention rate and mortality is noted [52]. Andersen et al. saw a 32% reintervention rate within 3 years after TEVAR in chronic dissections. 7% (5/75) of chronic dissections treated with TEVAR required conversion to open or hybrid surgery [43]. This is similar to results of the VIRTUE-registry wherein chronic dissection reintervention rates are around 30% as compared to roughly 20% in acute and subacute dissections after 3 years. The most common reinterventions were stent graft extensions [53]. Causes for reinterventions are endoleaks, with persistent false-lumen patency and subsequent aortic growth, and stent graft-induced new entry tears (SINE). A recent study comparing TEVAR in acute versus chronic dissections showed SINE in 5.9 and 32.3%, respectively. Independent predictors for SINE were distal oversizing of the graft and TEVAR in chronic dissections [54].

Conclusion

A standardized approach for the treatment of descending aorta dissections should be developed, considering the initial clinical presentation together with the radiological features of dissection and correlating these with age and comorbidities.

One in four type B dissections will present with complications (as described above, Table 1).

In hemodynamically unstable patients, emergency TEVAR acts as a first choice, and can even be considered in patients with connective tissue disorders, as a bridge to open surgery in elective conditions [38].

Hemodynamically stable but symptomatic patients should be initially treated medically in the hyperacute phase. If these patients remain stable but symptomatic (refractory hypertension, pain, aortic growth,

malperfusion), TEVAR should be considered as a first-line treatment in the acute phase, unless contra-indicated. When stabilized, these patients should be considered for pre-emptive TEVAR, considering favorable anatomy and risk factors for future aortic growth. The factors currently seen as predictors for aortic growth are a large primary entry tear, location of the entry tear at the concavity, a large aortic diameter (>40 mm), a large false lumen diameter, or partial false lumen thrombosis. More research is needed to adequately determine predictive risk factors for future aortic complications and to define the optimal cohort for pre-emptive TEVAR. Current guidelines still recommend best medical treatment and follow-up in uncomplicated dissections, although we believe that waiting until criteria for surgery or TEVAR are met, seems to jeopardize the prognosis of some patients. By identifying and treating these patients in the early stage of dissection, maximal aortic remodeling is obtained and these patients are prevented to evolve into the group of chronic dissections. Clinical follow-up and serial imaging is advised 3 months after the acute event, 6 months, 12 months and yearly thereafter. Irrespective of intervention, best medical treatment should always be initiated.

In patients with large or symptomatic chronic dissections and aneurysmal degeneration, open surgery remains the golden standard, especially in younger patients with good life expectancy and limited comorbidities. TEVAR or fenestrated and branched endovascular repair can be considered in selected patients (elderly frail patients) with suitable anatomy. So-called less invasive options such as hybrid repair are being studied but the physiological impact of these interventions should not be underestimated.

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