

Current status of endovascular management of complicated acute type B aortic dissection

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Acute type B dissection is a catastrophic condition. Up to 20% of these patients develop severe complications such as rupture or branch vessel malperfusion. These challenging patients are usually triaged to surgical or endovascular management. The surgical approach has been the gold standard for treatment of complicated type B dissection. However, the management has been challenging even in centers of excellence. For these reasons, thoracic endovascular aortic repair of acute aortic dissection has gained increased interest as an initial treatment option. The goal of this therapy is to exclude the primary entry site, obliterate the false lumen, prevent aortic rupture, and relieve visceral and lower body malperfusion. The aim of this study is to review the contemporary outcome of patients undergoing endovascular treatment for complicated acute type B aortic dissection.

Diseases of the aorta are among the most feared conditions encountered in the emergency department. The term 'acute aortic syndrome' refers to a group of catastrophic diseases that affect the aorta. These include aneurysmal aortic rupture, intramural aortic hematoma, penetrating atherosclerotic ulcer, traumatic aortic transection and acute aortic dissection (AAD). AAD is not an infrequent clinical entity; with more than 20 cases per million per year worldwide, AAD is actually the most common catastrophe of the aorta [1–3]. It is a time-sensitive and rapidly fatal disease.

The classic clinical presentation of type B AAD is that of a male patient aged between 60 and 70 years with a history of hypertension, who presents to the emergency department with the complaint of abrupt onset of severe, sharp, ripping chest and/or back pain. Patients appear in distress with pain and sweating, and appear anxious as in shock. Blood pressure may be normal or elevated. Physical examination may reveal a pulse deficit or focal neurological findings. Definitive imaging methods usually include a computed tomography (CT) angiography. Critically ill patients will need definitive airway support, fluid resuscitation, blood pressure and heart rate control, and emergent open surgical or endovascular repair.

The majority of patients with uncomplicated disease may be treated conservatively with anti-impulsive and antihypertensive therapy [4]. However, up to 20% of these patients may present or develop severe complications, such as rupture, impending rupture, unrelenting pain

or branch-vessel malperfusion [5]. These challenging patients are usually triaged to surgical or endovascular management.

Etiology & pathogenesis

The heart serves as a positive displacement pump to supply blood to organs in humans. For every cardiac cycle, the left ventricle pushes (displaces) a fixed amount of blood into the aorta, which serves not only as a conduit but also as a modulator and damper to convert the pulsatile blood flow into an even reservoir of blood supply to the tissue. The aortic wall is made up of three layers: the inner layer of intima, the middle layer of media and the outer layer of adventitia.

Classic AAD begins with a tear in the intimal layer. In younger patients, elastic tissue degeneration observed in connective tissue disorders and pregnancy is the most common cause. In patients not affected by connective tissue disorders, media degeneration appears to be primarily related to the wear-and-tear of aging and that induced by arterial hypertension [6,7]. This explains the observation that type B aortic dissection is more commonly observed in the elderly population and in hypertensive patients [8,9]. It has been postulated that the smooth muscle cell degeneration these patients have may be due to some pathologic process or just aging [10]. Other contributory factors include bicuspid aortic valve, aortic coarctation and giant cell arteritis [6].

Once the intimal tear is formed, blood under pulsatile pressure subsequently forces the tear to open and dissects along the media layers in

Keywords

- aorta ■ complication
- dissection ■ endovascular
- management ■ outcome

the diseased aortic wall, forming a false lumen (FL). The dissection can propagate distally, proximally or both along the extent of the aorta. In the majority of cases (>90%), as the dissection propagates, it also causes one or more secondary tears in the intimomedial layer, allowing blood to flow back to the true lumen of the aorta. While the primary entry site can be in the thoracic aorta, the secondary site can be in the abdominal aorta. AAD can be catastrophic because the dissecting FL can rupture, causing rapid exsanguination and death. The FL can cause dynamic or anatomic (static) obstruction of the arterial branches coming off the aorta, causing serious and potentially lethal complications. In type A AAD, these include stroke, acute myocardial infarction, pericardial tamponade or aortic valve insufficiency. In type B AAD, potential complications include intestinal ischemia, renal failure, paraplegia and other malperfusion syndromes.

Definitions

Aortic dissection is categorized as acute or chronic depending on the onset of symptoms or dissection. If the symptoms or dissection occur within the last 14 days prior to presentation to the emergency department, the dissection is termed AAD. If the symptoms or dissection occur outside of the 14 days, the dissection is termed chronic aortic dissection (CAD). AAD is further classified depending on the location of the dissection. In the DeBaakey classification, type I refers to dissections that propagate from the ascending aorta, extend to the aortic arch and, commonly, beyond the arch distally. Type II refers to dissections that are confined to the ascending portion of the aorta. Type III dissections are limited to the descending aorta. Type III also includes dissections that start in the descending aorta that extend proximally to the arch and ascending aorta. In the more commonly used Stanford classification system (FIGURE 1), type A refers to dissections that involve the ascending and transverse aorta regardless of the site of origin; type B refers to dissections that involve only the descending aorta. Rarely, dissections that start in the descending aorta can also extend proximally into the aortic arch and the ascending aorta. This is a special case of type B dissection, and should be termed as a retro-A dissection.

The term 'complicated' type B AAD is used for patients who present with, or during the hospital course develop, the following symptoms: rupture, malperfusion syndromes, refractory pain

or rapid aortic expansion. This is an important distinction as patients with complicated type B AAD usually require immediate endovascular or open surgical intervention.

Management

At triage, patients who presents with symptoms consistent with type B AAD should be quickly examined by a physician. Prompt diagnosis and treatment of patients with suspected AAD is essential for improved outcome. Pain should be treated with appropriate analgesics. Heart rate and hypertension should be aggressively controlled with β -blockers [4]. An arterial vasodilator without the reflex tachycardia (e.g., nicardipine) can be added as second-line drugs for refractory hypertension. β -blockers are the first-line drugs because they control the maximal force of left ventricular contraction (dP/dt) in addition to controlling heart rate and blood pressure. However, the role of β -blockers in reducing malperfusion is unknown. Targeted heart rate and systolic blood pressure should be less than 60 beats/min and less than 100 mmHg, respectively. This reduction in heart rate has been demonstrated to reduce further dissection, branch vessel malperfusion and weakening of the aortic wall [11]. Common β -blockers include labetalol, esmolol, metoprolol and atenolol. In patients with potential intolerance to β -blockers (e.g., patients with asthma and heart failure), a test dose of a short-acting β -blocker, such as esmolol, should be tried. If β -blockers cannot be used, calcium channel blockers (e.g., nicardipine, clevidipine or diltiazem) might be alternatives to control blood pressure in these patients. Hydralazine and sodium nitroprusside have been popularized for medical management of type B AAD. Although these agents reduce blood pressure, they increase the maximal force of left ventricular contraction (dP/dt) and cause reflex tachycardia, and are thus contraindicated in patients with AAD. These agents are still used in many centers, but β -blockers and calcium channel blockers should be preferred.

Hemodynamically unstable patients with type B AAD have complicated AAD. They are either in hemorrhagic shock or septic/metabolic shock from visceral or limb malperfusion. They should be endotracheally intubated and fluid resuscitated with crystalloid, Packed red blood cells or other colloid. In these patients, a transesophageal echocardiogram or CT angiogram are the preferred diagnostic tools, depending on the hospital setting. If the diagnosis of a

complicated type B AAD is confirmed, patients should be immediately brought to an operating room with c-arm fluoroscopy or ideally a hybrid operating room for endovascular and/or surgical treatment options. Endovascular or surgical options may be offered to hemodynamically stable patients with a complicated type B AAD.

The surgical approach has traditionally been the gold standard for treatment of complicated acute type B dissection. This includes left thoracotomy and replacement of the dissected aorta using Dacron graft. This can be carried out using simple clamp-and-go technique, left heart bypass, complete cardiopulmonary bypass or hypothermic circulatory arrest. However, surgical management of complicated type B AAD remains challenging. This also pertains in obtaining adequate homeostasis at anastomotic sites.

For these reasons, thoracic endovascular aortic repair (TEVAR) of AAD has gained increased interest as an initial treatment option for these patients with complicated type B AAD [12–19]. The goal of this therapy is to exclude the primary entry site, obliterate the FL, prevent aortic rupture, and relieve visceral and lower body malperfusion. The endovascular treatment of patients with complicated type B aortic dissection is complex and requires algorithmic approach. The patients are divided into a ‘malperfusion’ or ‘rupture’ pathway based on physical examination, laboratory results and CT scan findings. During the procedure, the true lumen is accessed and the access is verified using intravascular ultrasound. The size of the stent graft is chosen based on the diameter of the healthy proximal aorta. The stent grafts are oversized by a maximal 10% and there is no ballooning of the stent grafts after deployment. In cases of (impending) aortic rupture, the entire descending thoracic aorta is covered using stent graft. This is necessary as frequently the location of the tear is not localized on imaging. In patients with malperfusion, the proximal entry site that has been localized with intravascular ultrasound or contrast angiogram is covered with stent graft. Subsequently, the branch vessels are reassessed for malperfusion. This can be carried out using angiograms and intravascular ultrasound interrogations, as well interluminal pressure measurements. If further branch vessel malperfusion is found, these arteries are stented. More details on endovascular techniques used for TEVAR have been elucidated previously [20–24].

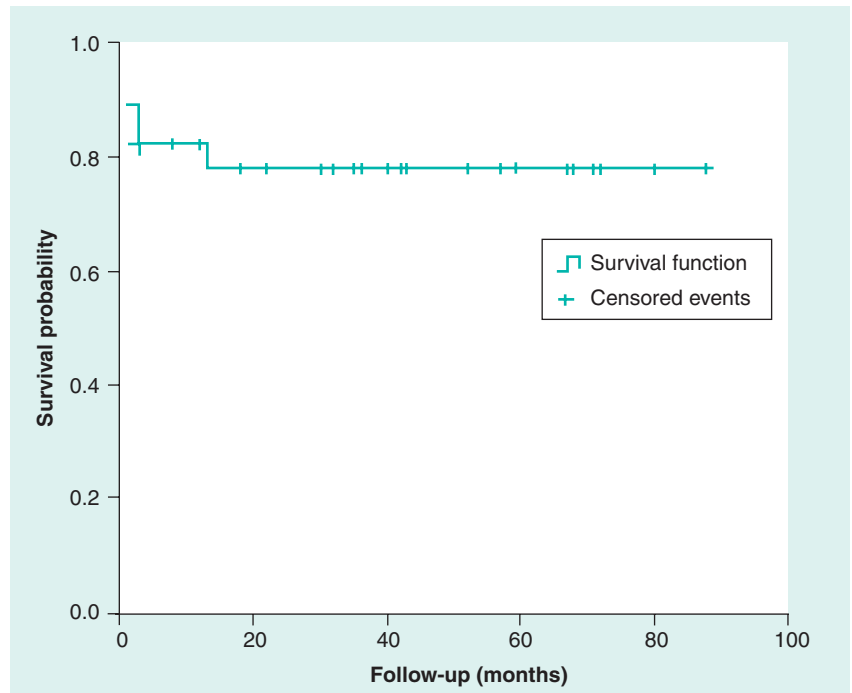


Figure 1. Kaplan–Meier survival curve. Lines indicate at least one censored patient due to length of the follow-up.

Outcome

The short-term prognosis for patients with type B AAD is better than those with type A, and the medical management of type B AAD is associated with less mortality compared with the open surgical intervention approach. Overall, 89% of patients with type B AAD survive to hospital discharge (although the in-hospital survival rates were as low as 29% for the highest risk group, 64% for the intermediate and 97% for the lowest risk group) [2]. Aortic rupture, shock and malperfusion are the most important in-hospital risks leading to poor outcome in up to 20% of these patients [5]. Of the survivors, 80% will develop aneurysmal dilatation of the FL, requiring cardiothoracic surgical intervention in a third of cases [4,5,12–15]. Therefore, although medical management of patients with type B AAD is the standard of care at the moment, it is still associated with significant morbidity and mortality in patients with uncomplicated type B AAD.

Fattori *et al.* analyzed the impact on survival of different treatment strategies in 571 patients with type B AAD enrolled from 1996 to 2005 in the International Registry of AAD [25]. A total of 390 patients (68.3%) were treated medically, 59 (10.3%) with standard open surgery and 66 (11.6%) with an endovascular approach. Patients who underwent emergency endovascular or open surgery were younger (mean age:

58.8 years; $p < 0.001$) than their counterparts treated conservatively, and had male preponderance and hypertension in 76.9%. Patients submitted to surgery presented with a wider aortic diameter than patients treated by interventional techniques or by medical therapy (5.36 ± 1.7 cm vs 4.62 ± 1.4 cm vs 4.47 ± 1.4 cm; $p = 0.003$). In-hospital complications occurred in 20% of patients subjected to endovascular technique and in 40% of patients after open surgical repair. In-hospital mortality was significantly higher after open surgery (33.9%) than after endovascular treatment (10.6%; $p = 0.002$). After propensity and multivariable adjustment, open surgical repair was associated with an independent increased risk of in-hospital mortality (odds ratio: 3.41; 95% CI: 1.00–11.67; $p = 0.05$). Therefore, this registry supports increased utilization of endovascular stent grafts for patients with type B aortic dissection, especially in the cohort with complicated type B AAD.

For complicated type B AAD, surgical intervention was the gold standard. However, the surgical management has been challenging even in centers of excellence [12–14]. Most recent results of surgical management of complicated type B AAD were presented by Coselli and coworkers [15]. The in-hospital mortality was 22.4%, while almost two-thirds of survivors experienced at least one complication [15]. Mortality is higher in patients presenting with bowel ischemia due to dissection and obstruction of the superior mesenteric artery. Therefore, in experienced centers TEVAR has become the preferred approach to this difficult patient cohort.

The authors recently reviewed their data on 28 consecutive patients with complicated type B AAD who underwent TEVAR between August 1999 and July 2007. Indications for emergency endografting were rupture in four patients (14%), severe lower body malperfusion in eight patients (29%), visceral/renal malperfusion in seven patients (25%), persistent chest pain despite proper anti-impulsive therapy in five patients (18%), uncontrollable hypertension in one patient (4%) and acute dilatation of FL with impending rupture in three patients (11%). A total of three patients (11%) died early. A further three patients died during follow-up of nonaorta-related causes. Overall survival was 82 and 78% at 1 and 5 years' follow-up, respectively. The aorta-related mortality was 10% for the entire follow-up period. Complete thrombosis of the FL in the thoracic aorta was achieved in 22 members (85%) of the surviving cohort, and partial thrombosis was achieved

in the remainder. The rate of treatment failure according to the Stanford criteria was 18% at 5 years' follow-up [19]. Mean follow-up was 36 months, and follow-up was complete in 28 patients (100%).

Patel *et al.* reported their results of endovascular approach to restore end-organ perfusion in cases of type B AAD with malperfusion [26]. A total of 69 patients were treated with a combination of flap fenestration, true lumen or branch vessel stenting where appropriate. Identified malperfused vascular beds included spinal cord (five patients), mesenteric (40 patients), renal (51 patients) and lower extremity (47 patients). Major morbidity included required dialysis (11 patients), stroke (three patients), paralysis (two patients) and 30-day mortality ($n = 12$; 17.4%). Mean Kaplan–Meier survival was 84.3 months. Although late mortality was associated with age ($p < 0.0001$), neither the type nor the number of malperfused vascular beds correlated with vital status at last follow-up ($p < 0.4$). Freedom from aortic rupture or open repair at 1, 5 and 8 years was 80.2, 67.7 and 54.2%, respectively.

Verhoye *et al.* reported data on 16 patients with complicated type B AAD who underwent endovascular stent-grafting within 48 h of presentation [27]. Complications included contained rupture, hemothorax, refractory chest pain, and severe visceral or lower limb ischemia. Early mortality was $25 \pm 11\%$ (70% CI), with no late deaths. No new neurologic complications occurred. According to the latest scan, four patients (25%) had complete thrombosis of the FL; the lumen was partially thrombosed in six patients (38%). Distal aortic diameter was increased in only one patient. Actuarial survival at 1 and 5 years was $73 \pm 11\%$; freedom from treatment failure (including aortic rupture, device fault, reintervention, aortic death or sudden, unexplained late death) was $67 \pm 14\%$ at 5 years. With follow-up for 9 years, endovascular stent-grafting for patients with complicated type B AAD conferred benefit.

Szeto *et al.* examined the results of TEVAR in 35 patients with complicated type B AAD with rupture ($n = 18$) or malperfusion syndrome ($n = 17$) [28]. Technical success (coverage of the primary tear site) was achieved in 34 patients (97.1%). Coverage of the left subclavian artery was required in 25 patients (71.4%). The 30-day mortality was 2.8% and the 1-year survival was $93.4 \pm 4.6\%$. Complications included permanent renal failure (2.8%), stroke (2.8%), spinal

cord ischemia (transient [5.7%] and permanent [2.8%]) and vascular access (14.2%). The mean intensive care unit and hospital stay were 4.7 ± 2.6 and 16.7 ± 12.0 days, respectively.

These reports demonstrate that endovascular repair of complicated type B AAD is associated with relatively low morbidity and mortality and has emerged as the surgical therapy of choice for complicated type B aortic dissection.

Various published reports have demonstrated that TEVAR may have reasonable results for CAD. With the cut-off of 14 days, some physicians are concerned about the arbitrary nature of this classification. This has contributed to an additional term 'subacute' dissection that involves patients with dissection up to 3 months. The authors suspect the endovascular treatment in patients with subacute and chronic dissection may not have as favorable results in the long-term follow-up. This is due to thickened dissection flap and the inability of complete exclusion of the FL.

Guanggi *et al.* compared outcomes between type B AAD (group A; $n = 72$) and type B CAD (group B; $n = 49$) in 121 patients [29]. The procedure success rates were 88.9 and 77.6%; the rates of postoperative endoleak were 11.1 and 22.4%; the 30-day mortality rates were 1.4 and 8.2%; and the 30-day stroke rates were 4.2 and 2.0% for groups A and B, respectively. No postoperative spinal cord ischemia was observed. The mean follow-up periods for groups A and B were 14.4 ± 11.0 months and 22.1 ± 20.8 months, respectively. Late mortality was 1.5% in group A and 4.8% in group B. In group A, the rates of complete FL thrombosis at 1 month, 1 year and 2 years postoperatively were 32.3, 51.4 and 53.8%, respectively, and in group B were 26.2, 44.8 and 50.0%, respectively. They demonstrated that endovascular repair is feasible for both acute and chronic dissections; however, better results were demonstrated for acute dissections.

Alves *et al.* reported their results with 106 patients exclusively with complicated type B AAD or type B CAD who were treated with TEVAR [30]. Technical success was achieved for 99% of patients, and the clinical success rate was 83% (exclusion of the FL, no early death or surgical conversion). In-hospital death occurred in five patients, two of which were after surgical conversion. Three patients required urgent surgical conversion. Neurologic complications occurred in five patients (one case of paraplegia). The average time of follow-up was 35.9 ± 28.5 months. During follow-up, 37% of

patients who were initially successfully treated reached a failure criterion (new endovascular or surgical intervention in the same aortic segment or death due to aortic or unknown cause). The Kaplan–Meier curve showed late survival rates higher than 80% in 2 years. Patients with both type B AAD and type B CAD had excellent initial results with TEVAR.

Jing *et al.* also compared endovascular treatment for type B AAD ($n = 32$) and type B CAD ($n = 35$) [31]. Compared with patients in the chronic group, those in the acute group had higher percentages of pleural effusion (15.6 vs 0; $p = 0.02$) and visceral/leg ischemia (21.9 vs 2.9%; $p = 0.02$). Procedure-related complications, including endoleak and postimplantation syndrome occurred more frequently in the acute group than in the chronic group (21.9 vs 2.9% and 31.3 vs 8.6%, respectively; $p = 0.02$ and $p = 0.02$). Kaplan–Meier analysis showed no significant difference in survival rate at 4 years between the two groups (86.4 vs 92.3%; $p = 0.42$ by Log-rank test). However, the 4-year event-free survival rate was higher in patients with chronic dissection than in patients with acute dissection (96.2 vs 73.9%; $p = 0.02$ by Log-rank test). They demonstrated that endovascular repair with stent-graft was safe and effective for the treatment of both AAD and CAD. However, both immediate and long-term major complications occurred more frequently in patients with acute dissection than in those with chronic dissection.

Conclusion

In brief, the management and outcome of complicated type B AAD is challenging. Surgical management has a higher rate of complications compared with endovascular repair. The data on TEVAR demonstrates favorable early- and mid-term results for patients undergoing TEVAR for complicated type B AAD. Although long-term durability of TEVAR for complicated type B AAD is yet to be established, we conclude that TEVAR is the therapy of choice for patients with complicated type B AAD, and that a paradigm shift is warranted from open surgical treatment to TEVAR in these undesirable surgical candidates.

Future perspective

Endovascular management of complicated type B AAD is complex and challenging. It has become an emerging alternative to the open repair in many centers, since initial feasibility and safety was demonstrated by the Stanford

group [17]. The indications for endovascular therapy for complicated type B AAD have yet to be determined. At present, the purest indications for TEVAR in the setting of type B AAD are malperfusion and rupture. These critically ill patients have very high open surgical mortality even in centers of excellence [12,15]. Although validation studies comparing surgical therapy to TEVAR are missing, the endovascular approach has been increasingly used for these indications [32]. Intractable pain is thought to be a symptom of progression of dissection, possible impending rupture or malperfusion, necessitating endovascular intervention [15,19,32,33]. Uncontrollable hypertension is a less common problem with modern antihypertensive and anti-impulsive strategies [34,35]. However, dissection into renal arteries may cause poorly or uncontrollable arterial hypertension due to an uncoupled renin–angiotensin–aldosterone system. Although these patients may not develop increased creatinine levels and frank renal ischemia, many authors postulate surgical intervention or TEVAR to reduce the chance of rupture or early aneurysmal degeneration in the proximal descending thoracic aorta [15,19,34]. As increased follow-up and experience has been gained in TEVAR in various patients with complicated type B AAD, there will be a refinement of patient selection criteria and enhance

physician judgment along with the evolution of more sophisticated, small-caliber, flexible and pathology-specific TEVAR devices and deployment systems.

If the results of TEVAR are found to be non-inferior to maximal medical therapy, this may be a superior option for the patients, as 80% will develop aneurysmal dilatation of the FL, requiring cardi thoracic surgical intervention in a third of cases [35–37]. The Investigation of Stent Grafts in Patients with Type B Aortic Dissection (INSTEAD) trial was designed to address this question, but was associated with methodological flaws by including patients with type B CAD [38]. However, it did help us understand that earlier intervention in these patients may be beneficial. With more refined clinical trials, there will be a push towards endovascular treatment of uncomplicated type AAD.

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The authors have no relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties.

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Executive summary

Introduction

- Complicated type B acute aortic dissection (AAD) belongs to the group of the acute aortic syndromes.
- The typical patient is a male patient aged between 60 and 70 years with a history of hypertension presenting with severe acute chest and back pain.

Etiology & pathogenesis

- Aortic dissection is initiated with a tear in the intimal layer.
- The dissection propagates in the media layer, producing multiple intimal tears.

Definitions

- A dissection is defined as acute if the symptoms occur less than 14 days prior to presentation to the emergency department.
- Stanford type B aortic dissection is defined as a dissection in the descending thoracic aorta.
- Complicated type B aortic dissection is defined as a dissection with impending or frank rupture, branch vessel malperfusion, unrelenting pain and hypertension.

Management

- Anti-impulsive therapy is the standard of care in uncomplicated type B AAD.
- Surgical management used to be the golden standard for the treatment of complicated type B AAD.
- Endovascular repair is currently the preferred method of treatment in experienced centers.

Outcome

- Medical management of uncomplicated type B aortic dissection has an acceptable early mortality.
- Complicated type B aortic dissection has better outcome if treated with stent-grafts compared with open surgery.

Future perspective

- Indications for stent-grafting for complicated type B AAD may change as more papers are published.
- Uncomplicated type B AAD may be treated with stent-grafting, if proven to be superior to medical therapy.

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