Acute Complicated Type B Aortic Dissection: Do Alternative Strategies Versus Central Aortic Repair Make Sense?

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Abstract

Current guidelines dictate emergency repair for an acute complicated type B aortic dissection (TBAD). Surgical approaches for the treatment of acute complicated TBAD can be divided into open and endovascular. The endovascular approach is further divided into central aortic repair and alternative endovascular techniques. Central repair includes endoluminal aortic stent graft repair, such as thoracic endovascular aortic aneurysm repair and provisional extension to induce complete attachment, extended provisional extension to induce complete attachment and stent-assisted balloon-induced intimal disruption and re-lamination in aortic dissection repair techniques. Alternative endovascular techniques include reno-visceral stenting, endovascular aortic membrane fenestration and targeted false lumen thrombosis. This review discusses and compares the various endovascular approaches to repair of acute complicated TBAD, focusing on central versus alternative endovascular techniques. We also discuss indications for technique selection, focusing on the acute management of complicated TBAD.

Keywords

Type B aortic dissection, complicated acute type B aortic dissection, TEVAR, stent graft, aortic fenestration, reno-visceral stenting

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Acute aortic dissection accounts for the most common acute aortic syndrome requiring surgical repair, with an incidence of 15 per 100 people per year.[1] Based on the Stanford classification, described in 1970, acute aortic dissection is divided into types A and B.[2] Type A dissection includes all dissection of the ascending thoracic aorta, whereas type B includes all dissection distal to the left subclavian artery. Depending on the clinical severity of the disease, type B aortic dissection (TBAD) has a wide spectrum of treatments and outcomes.[3]

TBAD is divided into acute (≤14 days), subacute (>14 days and ≤3 months) and chronic (>3 months) based on the onset of clinical symptoms.[1] Acute TBAD accounts for approximately one-third of all aortic dissections and can present as uncomplicated or complicated disease. Complicated disease is characterised by the presence of at least one of the following: aortic rupture; malperfusion syndrome (i.e. end-organ ischaemia secondary to aortic branch compromise leading to visceral, renal, spinal or lower extremity ischaemia); recalcitrant hypertension despite best medical therapy; recurrent or refractory pain; and/or early disease progression.[1,4,5]

Approximately 25–40% of cases of TBAD are complicated, and 10–20% of all patients with complicated TBAD present with malperfusion syndrome. The severity of the malperfusion syndrome is correlated with early mortality.[6–8] Branch vessel involvement or malperfusion is an independent risk factor for early mortality.[4,9] Data from the International Registry of Aortic Dissection (IRAD) study revealed an in-hospital mortality rate of 13% for acute TBAD, with most deaths occurring during the first week after presentation.[10] Aortic rupture and visceral ischaemia were the most common causes of death in patients who presented with TBAD.[11]

Current guidelines dictate emergency repair for an acute complicated TBAD.[12,13] Surgical approaches for the treatment of acute complicated TBAD can be divided into open and endovascular. The endovascular approach is further divided into central and alternative endovascular techniques. Central repair includes endoluminal aortic stent graft repair, such as thoracic endovascular aortic aneurysm repair (TEVAR). The alternative endovascular techniques include aortic membrane fenestration and stenting of the side branches.[8] This review discusses and compares the various endovascular approaches to repair acute complicated TBAD, focusing on central versus alternative endovascular techniques.

Central Aortic Repair

In central aortic repair for acute complicated aortic dissection, the main goal is to address the entry tear. Replacing the proximal descending thoracic aorta (in the case of open aortic repair) or covering the entry tear with a stent graft (in the case of endovascular repair) will re-equilibrate the perfusion pressure between the true and false lumens. Malperfusion syndrome occurs because of static or dynamic obstruction of a branch vessel, or a combination of both. Although static obstruction is a fixed obstruction of the true lumen, dynamic obstruction may arise due to intermittent occlusion of the branch vessel by protrusion of the flap or false lumen into the ostium of the vessel during the cardiac cycle. Dynamic

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obstruction will typically require a central aortic repair, whereas static obstruction of the branch vessels may require more focused treatments, such as stenting and fenestration techniques.[8,14,15]

Historically, acute complicated TBAD was managed with open surgical repair, which was associated with high perioperative morbidity and mortality. Swee and Dake reported the results of the first TEVAR operations performed for the treatment of acute complicated TBAD in 1999.[16] TEVAR was technically successful in all cases (n=19), with complete thrombosis of the false lumen in 79% of patients and partial false lumen thrombosis in 21% of patients within 6 months.[16] Resolution of branch vessel ischaemia was observed in 79% of obstructed branches, and the 30-day mortality was 16%.[16] Since that initial report, many studies have shown the superiority of TEVAR to both open surgery and optimal medical therapy for complicated TBAD.[17,18] In this review, we focus on the endovascular approach to acute complicated TBAD.

Thoracic Endovascular Aortic Aneurysm Repair

Although controversial at first, TEVAR has gained acceptance in the past 15 years as a safe and effective treatment for acute complicated TBAD.[19–21] Since Swee and Dake published their data on TEVAR for the treatment of TBAD in 1999, this technology has revolutionised the management of TBAD.[15] TEVAR has a significantly lower in-hospital mortality rate than traditional open repair.[13,18,22,23]

TEVAR promotes remodelling of the aorta in cases of acute TBAD by closure of the entry tear and inducing false lumen thrombosis, which will result in true lumen expansion.[24–26] This is particularly important because continued false lumen patency is strongly associated with late aneurysmal degeneration of the aorta and late mortality.[24,27] It has been shown that the strongest predictors of aneurysmal degeneration are an initial false lumen diameter ≥22 mm, a maximum aortic diameter of ≥40 mm on initial presentation, an initial entry tear of ≥10 mm and a patent or partially thrombosed false lumen.[5]

Complete thrombosis of the false lumen is uncommonly seen after TEVAR, particularly in the untreated aortic segment.[28,29] Studies looking at risk factors contributing to incomplete thrombosis of the false lumen identify visceral branches originating partially or fully from the false lumen, re-entry tears and maximum diameter of false lumen as predictors for incomplete thrombosis of the false lumen after TEVAR.[30] To mitigate the relatively high rates of persistent false lumen patency observed after TEVAR for acute TBAD, various alternative novel central aortic repair techniques have been introduced.

Provisional Extension to Induce Complete Attachment

The Provisional Extension to Induce Complete Attachment (PETTICOAT) technique has been introduced in recent years to promote true lumen expansion and favourable aortic remodelling. PETTICOAT is an endovascular technique for the treatment of acute and subacute TBAD using a self-expandable bare stent in the visceral aorta following TEVAR. This stabilises the distal collapsed intimal flap and restores blood flow to the reno-visceral vascular beds.[31] The use of bare-metal stents to promote true lumen expansion following TEVAR was first proposed by Mossop et al. in 2005.[32] Since then, the PETTICOAT technique has evolved rapidly due to its favourable short- and mid-term outcomes.[33–35] Early results show increased true lumen volume, decreased false lumen volume and increased rates of complete false lumen thrombosis at 5 years compared with TEVAR alone.[33–35] However, remodelling is not typically achieved in the abdominal aorta using PETTICOAT.[36,37]

The STABLE I feasibility study and STABLE II pivotal study are two prospective non-randomised multicentre studies conducted to examine the efficacy of the PETTICOAT technique in TBAD.[37,38] STABLE I was designed as a prospective multicentre clinical trial for the endovascular treatment of complicated TBAD using a composite device design that enrolled 40 patients with TBAD (60% acute, 15% subacute and 25% chronic).[37] Most patients (77.5%) in the trial presented with complicated TBAD in the form of impending aortic rupture or branch vessel malperfusion. The 30-day mortality was 4.7%. Freedom from all-cause mortality was 88.3% at 1 year and 84.7% at 2 years. At 1 and 2 years, 80.3% and 73.9% of patients had a stable or reduced diameter of the thoracic aorta, respectively.[37] Favourable aortic remodelling was seen during the follow-up period, with complete thrombosis of the thoracic false lumen occurring in 31% of patients at 12 months.[38,39]

The STABLE II clinical trial is a prospective non-randomised multicentre study that examined the efficacy of the PETTICOAT technique in 73 patients with acute complicated TBAD.[38] In STABLE II, 27% of patients presented with aortic rupture and 78% presented with malperfusion. The 30-day mortality was 6.8%, and freedom from all-cause mortality was 80.3% at 1 year. At 12 months, complete or partial thrombosis of the false lumen was seen in all patients. These results revealed favourable clinical and anatomic outcomes in acute TBAD using PETTICOAT.[38]

Extended Provisional Extension to Induce Complete Attachment Technique

The extended PETTICOAT (e-PETTICOAT) technique was introduced as an alternative to the original PETTICOAT to promote aortic remodelling and to reduce late aortic-related complications. The e-PETTICOAT technique is an extension of the standard PETTICOAT technique, with the addition of covered stent grafts (or bilateral kissing iliac stent grafts) placed in the infrarenal aorta extending down to the iliac bifurcation.[34] Distally, this adds radial force to the abdominal bare-metal stent to re-enforce the infrarenal portion of the bare-metal stent, cover distal re-entry tears and relaminate the membrane in the common iliac arteries. This, in turn, promotes false lumen thrombosis and aortic remodelling.[40]

A prospective single-centre study looking at efficacy and mid-term aortic remodelling in 17 patients with acute complicated TBAD treated with the e-PETTICOAT technique reported 100% technical success, with complete resolution of malperfusion syndrome in all patients.[34] No mortality occurred within the first 30 days after the procedure. Favourable aortic remodelling was observed in all cases, with partial false lumen thrombosis reported in 76% of patients. In addition, no paraplegia, stroke, kidney failure or new visceral branch occlusion was reported at follow-up (range 24–42 months).[34,40]

Stent-assisted Balloon-induced Intimal Disruption and Re-lamination in Aortic Dissection Repair

In 2012, Hofferberth et al. proposed a different version of the PETTICOAT technique, consisting of ballooning of the true lumen of the aorta inside the stent graft as well as the distally deployed bare stents to rupture the lamella.[33] This technique is known as stent-assisted balloon-induced intimal disruption and relamination in aortic dissection repair (STABILISE).[31] The goal of the STABILISE technique is to create full expansion of the stent in a single-channel aorta. Hofferberth et al. reported outcomes for 11 patients (seven with type A aortic dissection and four with acute type B aortic dissection) who underwent the STABILISE technique. There were no intraprocedural complications reported, 30-day mortality was 9% (n=1) and complete false lumen thrombosis was achieved in 90% of patients.[33]

Despite these positive aortic remodelling results, the technique has not gained widespread use. This could be due, in part, to the inherent risk of aortic rupture with ballooning of a dissected aorta.

Endovascular Alternative Aortic Repair Techniques

There are several alternative (non-central) endovascular techniques available for the treatment of acute TBAD with malperfusion syndrome, including endovascular fenestration and stenting of branch vessels. The concept of an alternative repair technique is to stabilise dynamic or mixed dissection flaps affecting the reno-visceral segment of the aorta, as well as static obstructions of branch vessels. One major limitation of central aortic repair is the inability to address static malperfusion syndrome because static obstruction often requires stent grafting to ensure patency.[3] In these situations, a combination approach using a central aortic repair strategy with an adjunct alternative repair strategy may be warranted. Compared with TEVAR, alternative techniques have lower risks of retrograde type A aortic dissection, spinal cord ischaemia and paraplegia.[41] There are no limitations in aortic diameter, an appropriate proximal landing zone or adequate access to accommodate stent graft delivery systems. In addition, TEVAR stent grafts are at risk of infection in patients who present with bowel ischaemia/necrotic bowel due to malperfusion syndrome or other causes of sepsis, a risk that is much less common with alternative repair strategies.[42] For these reasons, it is important to study alternative aortic repair techniques for acute TBAD and to be able to apply them in selected patients.

Reno-visceral Stenting

Ideal management of visceral branches in the setting of acute TBAD with malperfusion is controversial. This is due, in part, to the scarcity of data on the natural history of branch vessels as it relates to the dissection flap after TEVAR. However, reno-visceral stenting for acute complicated TBAD is a feasible and sometimes necessary technique for resolving persistent dynamic and static malperfusion.

There are limited data on the outcomes of isolated reno-visceral stenting for acute complicated TBAD, probably because most physicians use this approach as an adjunct technique along with central aortic repair. In a single-centre experience, Barnes et al. examined 165 patients with aortic dissection who presented with malperfusion (115 patients with acute dissection and 50 with chronic dissection).[43] Renal malperfusion was confirmed with systolic gradient between the aortic root and renal hilum in 90 patients, 71 of whom underwent endovascular therapy including isolated renal artery stenting (n=31), proximal aortic fenestration with and without aortic stenting (n=24) and/or both renal and aortic intervention (n=16). Barnes et al. reported one stent thrombosis requiring thrombolysis and one type III endoleak.[43] The 30-day mortality in the intervention cohort was 21%.[43] Based on these outcomes, Barnes et al. argued that malperfusion of the renal artery, as established by aortorenal pressure gradients, can be relieved by the deployment of a self-expanding stent.[43] It is important to note that these results are not categorised based on the type of dissection the patient presented with or the chronicity of disease.

Endovascular Aortic Fenestration

Aortic fenestration is an alternative endovascular technique to address TBAD with malperfusion syndrome, and has been shown in some studies to be an effective modality in addressing both dynamic and static malperfusion of aortic branch vessels.[44] Proponents of this technique argue that the key pathophysiological reason for the aneurysmal degeneration of a dissected aorta is the presence of two lumens, with increased tensile force on the false lumen. Aortic fenestration creates a haemodynamic equilibrium between the two channels, which, in theory, reduces the pressure and thus lowers the risk of aneurysmal degeneration of the aorta over time.[45]

Aortic fenestration is typically performed from the smaller (usually true) lumen to the larger (usually false) lumen. The technique should be performed with the assistance of intravascular ultrasound to fully visualise the relationship between the true and false lumen. A fenestration is created near a compromised aortic branch vessel using a curved hollow metal needle, such as a Rösch–Uchida needle. After the needle and a stiff wire are advanced from the true to the false lumen, a 5-Fr catheter is advanced. Confirmation of the position across the membrane is performed by contrast injection. Subsequently, an angioplasty balloon (15–25 mm) is used to create a large fenestration tear.[46] This will allow for equalisation of pressure between the true and false lumens. Fenestrations close to the target vessel can lead to local redirection of blood flow to the ischaemic end organ.[16]

There are a number of alternative techniques to aortic fenestration that do not require the use of an endovascular needle or balloon angioplasty. The scissor technique is a fenestration strategy whereby stiff guidewires are introduced in each lumen from a single femoral access, and a single long sheath is advanced over the two wires, thus dividing the membrane over this distance. Those familiar with the use of this technique have reported both clean longitudinal tears (the ideal result) and circumferential separation of the flap from the aortic wall with aorto-aortic intussusception (not ideal).[47] 'Cheese wiring' is another novel fenestration technique that involves snaring a wire from the true to false lumen via bilateral femoral access, followed by application of downward traction on the wires to create a longitudinal tear in the dissection membrane. Once the tear is completed, an endograft is placed in the thoracic aorta.[48]

Given the lack of large prospective studies and paucity of long-term follow-up data on the use of the endovascular aortic fenestration technique, it is difficult to establish a reliable account of outcomes. Slonim et al. reported outcomes for 40 patients with malperfusion (10 type A aortic dissection and 30 type B aortic dissection); 14 patients were treated with stenting of either the true or false lumen combined with balloon fenestration of the intimal flap, 24 patients were treated with stenting alone and two patients were treated with fenestration alone.[44] Overall technical success, as defined by restoration of flow to the ischaemic territory, was achieved in 93% of patients, and the 30-day mortality was 25%.[44] In another study, Petal et al. analysed 69 patients presenting with acute TBAD with malperfusion.[49] All patients were treated with a combination of flap fenestration and/or true lumen or branch vessel stenting when appropriate. The overall technical success rate for flow restoration was 95.7%, and the 30-day mortality was 17% (five patients died from false lumen rupture).[49] Freedom from aortic rupture or open repair at 1, 5 and 8 years was 80.2%, 67.7% and 54.2%, respectively.[49] Although the authors of both studies argue that this technique is effective in the treatment of complicated TBAD, there were no comparison groups using more traditional approaches, so the frequency by which this technique should be used is not currently clear.

Targeted False Lumen Thrombosis

Various techniques have been proposed to promote false lumen thrombosis in TBAD. The 'knickerbocker technique' involves relining the true lumen in the descending thoracic aorta with an oversized endograft, followed by controlled rupture of the septal wall using a large, compliant balloon.[50] The 'candy plug' technique involves occlusion of a large false

lumen with an endograft to create an hourglass or 'candy plug' configuration. This is then followed by an Amplatzer vascular plug (Abbott) at the waist to occlude flow. However, this can result in narrowing of the true lumen.[51,52]

The 'cork in the bottleneck' technique was described in 2013 by Loubert et al. in two patients and involves the placement of a Greenfield filter or an occluder device in the false lumen, followed by detachable balloons and thrombin.[53] In this way, retrograde flow into the thoracic aorta is blocked. False lumen embolisation with coils, plugs, onyx and glue has also been described in the literature to promote false lumen thrombosis.[54] In this technique, the false lumen is cannulated through a known fenestration, and the false lumen is subsequently embolised with coils.[52]

In a systemic review evaluating outcomes associated with targeted false lumen thrombosis techniques, 101 patients with type A aortic dissection (n=40) or complicated TBAD (n=61) were included; 79% of the patients in the complicated TBAD cohort had a previous endovascular repair.[54] The technical success rate was reported at 100%, with 0% 30-day mortality. During the follow-up period (range 2–63 months), late mortality was 7.1%, and false lumen thrombosis was achieved in 62% of patients.[54]

Selection of the Endovascular Technique

Since the adaptation of endovascular repair, TEVAR has become the first-line therapy for the treatment of acute complicated TBAD. There is robust experience in the literature supporting favourable aortic remodelling with TEVAR in the acute and subacute phases.[55] However, the likelihood of achieving a successful endovascular repair is contingent on favourable anatomy, including suitable proximal and distal landing zone lengths (15–25 mm proximally and 20–30 mm distally), a healthy and non-tortuous aortic segment without a heavy calcium or thrombus burden and appropriately sized access vessels. There are a number of other patient factors, including age, frailty and comorbidities (including the presence of connective tissue disorder), that also play a role in operative decision-making. With stent grafts ranging from 21 to 46 mm for TEVAR, it is possible to treat aortic diameters from 16 to 42 mm. Larger or smaller aortas are not typically candidates for endovascular central aortic repair. Extreme angulation of the landing zone in the aortic arch is another limitation to TEVAR because it can lead to incomplete endograft opposition, known as a 'bird-beak' configuration. This will, in turn, lead to type I endoleak, and secondary interventions may occur in up to 15% of patients.[56] Barring such anatomical limitations, TEVAR should be used as first-line therapy in acute complicated TBAD.

Extension of TEVAR with bare-metal stent implantation beyond the thoracoabdominal aortic junction using the PETTICOAT and e-PETTICOAT techniques has shown promising short- and mid-term results in reducing malperfusion-related mortality and promoting aortic remodelling.[34,36] Given these positive preliminary results, this technique should be considered as optional first-line therapy in patients presenting with TBAD and malperfusion. Certainly, in cases of persistent malperfusion despite coverage of the entry tear with a stent graft, the PETTICOAT or e-PETTICOAT techniques should be used in an effort to expand the true lumen and promote reno-visceral perfusion via flap stabilisation. However, larger cohort and prospective randomised studies are needed to confirm the long-term results of this technique.

The role of reno-visceral stenting in the treatment of acute complicated TBAD is less clear, as noted above. We place self-expanding stents in branch vessels of patients presenting with complicated acute TBAD in two instances: persistent malperfusion despite TEVAR with PETTICOAT/e-PETTICOAT; and malperfusion in the setting of inappropriate anatomy for TEVAR. Similarly, aortic fenestration is reserved for extreme instances in which malperfusion is present and central aortic repair is not an option. Although the fenestration techniques have been successfully reported in small studies, the long-term effects are largely unknown.

In general, alternative endovascular techniques are often described in more morbid populations with poor prognoses. Although they should be in a surgeon's armamentarium, the learning curve related to these techniques should also be taken into consideration. Hence, complex alternative aortic repair techniques should be used sparingly and in experienced centres.

Long-term Non-aortic Cardiovascular Complications of Thoracic Endovascular Aortic Aneurysm Repair

There is a paucity of data regarding the physiological changes induced by the placement of thoracic endografts. However, emerging clinical evidence has demonstrated that aortic stent grafting can have adverse long-term cardiovascular outcomes.[56] Endograft-induced arterial stiffening can result in increased left ventricular stroke work and mass.[57,58] In a retrospective population-based study evaluating 133 patients with acute aortic syndromes, there was a 2.6-fold increase in the rate of non-aortic cardiovascular deaths compared with the general population.[59] As a result, the effects of TEVAR on aortic compliance and central haemodynamics require long-term cardiovascular management in patients in the postoperative period.[60] Further studies are required to improve endograft design to minimise compliance mismatch with the native aorta.

Conclusion

The management of acute complicated TBAD requires various considerations, and the treatment approach should be catered to the clinical situation. Central aortic repair for the treatment of acute complicated TBAD is associated with favourable mid- and long-term outcomes, as supported by large multicentre studies.[12,34,36,40] TEVAR has become the preferred therapeutic modality in most specialised centres. More recently, endovascular repair with distal bare-metal stenting to treat the thoracoabdominal aorta following TEVAR (i.e. PETTICOAT, e-PETTICOAT techniques) has gained acceptance due to the favourable aortic remodelling and stabilisation of disease progression compared with TEVAR alone.[34,36] Although novel alternative techniques to address malperfusion and a persistent false lumen in TBAD are feasible, data regarding their long-term safety and efficacy are scarce. As a result, alternative strategies for the treatment of acute complicated TBAD should be used on a case-by-case basis depending on the patient's specific clinical presentation and anatomy, and the surgeon's experience. ❑

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