Aortic Dissection

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Abstract
Aortic dissections are catastrophic vascular emergencies, and early recognition and appropriate interventions can be crucial to survival. Research has changed the way aortic dissections are managed over the past two decades and will continue to contribute to the evolution of treatment modalities. Early treatment for uncomplicated type B dissections still remains controversial but certain characteristics may benefit from early intervention.

Keywords
Aortic dissection, complicated dissection, TEVAR, thoracic endovascular aortic repair, type B aortic dissection, uncomplicated dissection

The first classification of aortic dissection was made by Debakey et al. in 1965.¹ The widely accepted Stanford classification was coined by Daily et al. 5 years later; in this classification type B signifies that the intimal tear is distal to the left subclavian artery (similar to Debakey type 3).² The basis of these classifications was predicated on the vastly different outcomes and treatment modalities for the different presentations. Additionally, the timeline from onset of symptoms can divide presentation into acute (<2 weeks), subacute (2 weeks to 3 months) and chronic (>3 months). It was recognised early on that medical management of type B aortic dissection (TBAD) resulted in good initial results with a review of published literature suggesting a 5-year mortality of 75–88%.³,⁴ TBAD can quickly become catastrophic – in-hospital mortality is as high as 10–14%.⁵,⁶

Presentation and Diagnosis
The classic presentation of TBAD is an abrupt onset of severe sharp tearing or ripping pain in the chest or back. Pain is the presentation in 70–72.4% of cases of TBAD, with people most often experiencing pain in the back or occasionally the abdomen. Hypertension is the second most common presentation according to the International Registry of Aortic Dissection (IRAD) for TBAD, occurring in 66–70.1% of cases.⁵,⁷ Hypertension is also a predictor for complications, with blood pressure control representing an important goal for management. Early high inpatient mortality represents a subset of TBAD due to malperfusion and/or rupture.⁸ However, this may only represent the tip of the iceberg because estimates from autopsies suggest that 20–30% cases of TBAD do not make it to hospital.⁹

Malperfusion may be the presenting feature and occurs in up to 10% of TBAD cases.⁸ End organ dysfunction must be recognised early and is indicative of impending organ failure. This may be static or dynamic for flow, which can be corroborated by laboratory findings and imaging. Compromised spinal, visceral, renal or iliac blood flow may result in paralysis, paraplegia, lower limb ischaemia, abdominal pain or diarrhoea with raised liver function, amylase, lactate, urea, creatinine and coupled with absence (thrombosis) or delay in contrast enhancement in the target organ.

The role of contrast enhanced imaging cannot be over emphasised as it gives useful information about the predictors of failure, malperfusion, pre-treatment sizing and – most importantly – aortic rupture. The diagnosis of impending rupture as defined as haemorrhagic pleural effusion or expanding peri-aortic haematoma can only be made with imaging.⁹ Historically, retrograde aortography was considered the gold standard for diagnosis; fortunately, this is only used during interventions. CT angiography (CTA) has become widely available with rapid image acquisition. CTA has a 93.8% sensitivity and a specificity of 87.1% which compares well with other modalities such as MRI or transoesophageal echocardiogram for acute aortic emergencies.¹⁰ MRI has sensitivity rates of 95–100% but is limited by its long examination time and availability.¹¹ Transoesophageal echocardiogram for TBAD is limited by the need for general anaesthesia and it loses sensitivity in the aortic arch which is in the vicinity of the proximal tear. Intravascular ultrasound is now considered the standard intervention for TBAD.

Natural History and Aortic Remodelling
In 80% of TBAD cases, the intimal tear is in a posterior-lateral aspect to the aorta, a few centimetres distal to the left subclavian artery with the trajectory of the false lumen taking various configurations but most commonly involving the left renal artery.¹² Debakey et al. first reported that up to 40% of TBAD will degenerate over time.¹³ Since the founding of IRAD in 1995, there is a better understanding of the long-term outcomes. Five-year analysis suggests a better long-term survival in thoracic endovascular repair versus best medical management for...
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TBAD shown by the IRAD registry. The ideology behind this is that aortic-related pathology will develop with time and the patients will eventually succumb to it. Only 41% of TBAD remain intervention-free at 6 years follow-up with 65% of the aortic-related interventions related to aneurysmal degeneration. Note that this is highly predicated on time, as these benefits were not realised at the 2-year follow-up. A significant reduction in all-cause mortality and aortic-specific mortality at 5 years was shown by the first randomised trial for uncomplicated TBAD with thoracic endovascular repair of the aorta (TEVAR) versus best medical management.13

There is a correlation with false lumen patency and progression to aneurysmal progression in TBAD. Tsai et al. showed mortality post-discharge based on thrombosis of false lumen was 13.7% in complete thrombosis, 31.6% in partial thrombosis and 22.6% in complete thrombosis.14 The concept of aortic remodelling with inducing false lumen thrombosis by sealing the aortic tear with TEVAR and redirecting flow to the true lumen has the potential to reduce complications. Additionally, Tollenar et al. showed patients with fully patent false lumen at presentation and branch vessel involvement are less likely to develop thrombosis and may benefit from TEVAR.15 This was corroborated in the Investigation of Stent Grafts in Aortic Dissection (INSTEAD) trial with positive remodelling showing expansion of the true lumen from a mean of 19.4 mm to 32.4 mm at 2 years and shrunken false lumen from a mean of 29.3–8.6 mm at 2 years in subacute to early chronic, uncomplicated TBAD. This was maintained at 5 years and was reproducible at different levels of the aorta.15

The real-world experience from the VIRTUE Registry – a prospective, non-randomised, multicentre European registry – shows promisingly similar results for complicated TBAD.20 The true lumen increased across all levels of aorta involved with reduction in the false lumen after TEVAR but interestingly, this reduction was more marked in the acute and subacute groups. This adds insight into the plasticity of the intermembrane, suggesting the more acute the dissection, the more likely it is that aortic remodelling will be successful. The majority of the changes in the lumen size occurs by 6 months.

Management

Medical management guidelines recommend a target systolic blood pressure of 100–120 mmHg with heart rate <60 BPM.21 Beta-blockers should be the first-line treatment and should be used in preference to vasodilators unless contraindicated. It is not unusual for patients to need multiple medications for blood pressure control. Continuous monitoring in an intensive setting is preferred because up to 10–12% of these patients will have complications that occur during the initial encounter.

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Details of stent grafting for a complicated TBAD was first published in 1999.22,23 This was the start of endovascular treatment of TBAD, which resulted in TEVAR and it was initially only used in cases that were not amenable for open surgery. The definition of complication was standardised by the interdisciplinary expert group to include malperfusion, persistent or uncontrolled hypertension despite full medical therapy, and rupture or impending rupture.24 Open repair has traditionally offered the best chance of survival in complicated TBAD with the necessity for thoracotomy and left heart bypass adding to its risk of mortality. A high 30-day mortality of 19% and combined neurological complication of 9.8% in a meta-analysis by Moulakakis et al. made this a daunting procedure.25 This is compared to mortality in TEVAR of 10.2% and combined neurological complications at 9.1%.26 There are now several measures to improve neurological outcomes. Long-term outcomes of TEVAR now include 5-year freedom from aortic-related interventions of 45–77% and survival ranging from 62 to 100%. This changing landscape of treatment has led an expert consensus to recommend TEVAR as the first-line treatment for complicated TBAD. Other modalities for treatment, such as fenestrations, have fallen out of favour.

Interventions may be multiple; in addition to TEVAR, it may be necessary to ensure flow to the renal, visceral, iliac and left subclavian artery with stenting, fenestration or open surgical adjuncts. Complicated TBAD may also include increasing size, progression of dissection or refractory pain. Trimarchi et al. reviewed the IRAD data and found that recurrent pain or refractory hypertension appeared to be clinical features associated with increased inpatient mortality when managed medically (35.6% versus 1.5%).27

The real-world data from IRAD is encouraging. TEVAR confers a similar mortality benefit compared with medical management despite TEVAR being more suitable for the treatment of complicated TBAD.16 This led to a few TEVAR trials that were specific for acute complicated TBAD. The GORE TAG 08-01 study used the new conformable design placed in 50 patients, resulting in an 8% mortality, 18% stroke rate and 6% paraplegia rate at 30 days, and 88% survival at 1 year; there was a reduced mean false lumen area and increased true lumen diameter up to 3 years post-implantation.28 The Zenith TX2 was a ‘pathology-specific’ graft with a combination of proximal covered stent and distal uncovered bare metal stent. It was implanted in 86 patients with acute and subacute complicated TBAD. There was stability or an increase in the true lumen size and a reduction of the false lumen in the thoracic aorta with a 30-day mortality of 4.7%, 7% stroke rate and 88.3% freedom from all-cause mortality at 1 year.29 However, the false lumen of the abdominal aorta continued to expand. The Valiant Captiva was implanted in 50 patients for acute complicated TBAD with 8% mortality, 6% stroke rate and 6% spinal ischaemia at 30 days; however, 47% of the patients had serious adverse events by 1 year. The true lumen was stable or increased in 93.1%, the false lumen was stable or decreased in 44% with partial or complete thrombosis in 91%.30

Overall, this represents the past two decades of a new alignment of treatment of stent graft treatment for complicated TBAD, with trials still ongoing. Continued efforts are needed to lower mortality and neurological complications in this catastrophic disease that would otherwise be fatal. Fenestrated/branched devices may have a role for the future. Open repair is superior selectivity and only reserved for a minority of cases without proximal seal with rupture or impending rupture at the intimal tear in a patient who can tolerate an open procedure. Short segment proximal descending aortic replacement with left ventricular bypass would be the most appropriate procedure.

Acute Uncomplicated Type B Dissection

There is mounting evidence that uncomplicated TBAD will become aneurysmal even with best medical therapy and will be responsible for up to 30% of late mortality combined with up to 66% of late aortic interventions.31,32 Controversy exists over the decision to intervene or to watch and wait. It is without question that those who develop aneurysmal disease and require intervention have a much higher mortality.
Aortic dissection is a serious medical condition that involves the splitting of the aorta's inner lining, which can lead to complications such as rupture or stringent. The disease is typically characterized by a sudden onset of chest or back pain, or occasionally, abdominal pain. The exact cause of aortic dissection remains unclear, but it is associated with stress, physical activity, and sometimes congenital anomalies or degenerative changes in the aortic wall.

The treatment of aortic dissection depends on the location and severity of the dissection. It may involve medical management, such as optimal medical therapy combined with anticoagulant therapy, or endovascular repair, where a stent graft is used to seal off the area of dissection and prevent further extension of the false lumen.

Endovascular repair is a minimally invasive procedure that has become increasingly popular over the past few years. The procedure involves the insertion of a stent graft through a small incision in the leg. The stent graft is then deployed to the area of the aorta that requires treatment, and it is shaped to fit the natural curvature of the aorta.

The results of the INSTEAD trial, a randomised study comparing endovascular repair (AdSORB) to medical therapy, showed significant improvements in survival outcomes. The study also highlighted the importance of early diagnosis and intervention in the management of aortic dissection.

In conclusion, the management of aortic dissection continues to evolve, with ongoing research aimed at improving diagnostic techniques and developing new treatment options. Endovascular repair remains an important tool in the management of aortic dissection, and further research is needed to identify predictors of better outcomes and improve patient outcomes.

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