Editorial

Endovascular treatment for chronic type B aortic dissection: current opinions

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First described in the 18th century at the autopsy report of King George II, aortic dissection remains the most common emergency and catastrophic disease of the aorta, associated with a high preadmission and in-hospital mortality. Aortic dissection occurs in about 3–4 per 100,000 persons per year. Men are twice than women. Although less frequently affected by aortic dissection, women are significantly older than men and had a worse prognosis (1,2).

Different classifications have been proposed. The most used is the Stanford classification based on the involvement of the ascending aorta (type A) or involvement of the distal aorta alone distally to the origin of the left subclavian artery without implication of the ascending aorta (type B). Sixty % of dissection are classified as type A aortic dissection (TAAD), the 40% as type B (TBAD) (1-4).

Type A and type B aortic dissections can be considered two faces of the same problem with a different natural history, clinical outcome, surgical indications, timing for treatment and results.

Despite advances in medical therapy, the mortality for patients with TAAD remains too high. About 33% of untreated patients with TAAD die within the first 24 hours, 50% die within 48 hours and 75–90% die within 30 days (3,4). Emergency surgical approach with ascending aorta replacement is the only option to improve the survival (3,4). As reported by International Registry of Acute Aortic Dissection (IRAD), early mortality in patients undergoing TAAD repair remains high, ranging from 17% to 26% (3,4).

As for TAAD the first cause of death in patients with TBAD is the aortic rupture followed by malperfusion syndrome due to significant ischemia of visceral organs. Nevertheless, type B aortic dissection has a lower incidence of spontaneous rupture resulting in the tendency to stabilize and became chronic (5,6). The natural history of type B dissection is not easy to define since data regarding patients treated with medical therapy are sparse. Update information can be deduced from the IRAD and The INvestigation of STEnt Grafts in Aortic Dissection (INSTEAD) trial (3,4,7).

Systemic hypertension, present in over 80% of patients, along with older age and atherosclerosis are the most important risks for the development of type B aortic dissection. More rarely connective tissue disorders can be considered risk factors for the development of TBAD (5,6).

According to the current classification based on time of onset of symptoms, type B aortic dissection can be defined as acute when symptoms onsetted within 14 days and chronic after 14 days. Additionally, dissections onsetted 2 to 6 weeks from pain are deemed subacute (1,3,4).

About clinical presentation it can be classified as complicated or uncomplicated since clinical outcome and prognosis differs significantly. Complicated is defined by one of the following symptoms: refractory pain and/or hypertension despite full medication, early aortic dilatation or signs of rupture or impending rupture (i.e., haemothorax, increasing periaortic and mediastinal haematoma), malperfusion syndrome involving visceral, renal, or extremity or retrograde aortic dissection. When patients are hemodynamically stable, asymptomatic without pain or malperfusion signs, the dissection is defined as uncomplicated (4,8).

In-hospital mortality of untreated patients with complicated dissection, remains high, approximately of 50% (8-10). At admission approximately 25–30% of patients presenting with TBAD are complicated by malperfusion syndrome or hemodynamic instability, resulting in a high risk of early death. Immediate treatment is imperative (11).

On the contrary, uncomplicated dissection has a relatively benign clinical course. Early survival of these patients treated with optimal medical management alone is high as 90%. with a survival of approximately 80% within five years (8,10).

Management of type B aortic dissection remains still a clinical challenge. The initial management goal is to prevent aortic rupture and propagation of the dissection and symptoms. Medical therapy remains the standard of care, aimed at lowering the blood pressure, heart rate and pain, to reduce hemodynamic stress on the damaged aortic wall. Following initial stabilization most patients will require long-term antihypertensive treatment including β-blockers and calcium-channel blockers (1,2,4,8).

For patients with chronic uncomplicated dissection, medical therapy is the treatment of choice to prevent aortic expansion, rupture and/or recurrent dissection. Blood pressure control can reduce re-dissection by two-thirds. Despite appropriate medical therapy approximately 20–40% of patients in the long-term outcome will require a secondary operation for aortic aneurysmal degeneration (12,13).

In current clinical practice open or endovascular surgery is reserved for patients with acute TBAD who fail aggressive medical therapy or present complications such as impending rupture or faster aneurysmal expansion of the aorta, visceral or limb malperfusion syndrome, resistant systemic hypertension and recurrent or intractable pain (12.14). In chronic dissection, aortic aneurysmal degeneration with a diameter >5.5 cm or a rapid expansion of the aortic diameter exceeding 5 mm in 6 months or saccular aneurysm protruding ≥2 cm beyond the aortic wall or increased diameter of false lumen with a collapsed true lumen or recurrent symptoms of a new extension of the aortic dissection are indications for surgical treatment (12,14).

In the past, the only life-saving treatment for acute complicated TBAD or chronic recurrent complicated TBAD was conventional open surgery, currently reserved for patients with connective tissue disorders or unsuitable for endovascular surgery or patients in whom endovascular treatment has failed. In patients with aneurysmatic dilatation or rupture of descending thoracic aorta, current strategies for conventional surgery consist of open repair of the descending thoracic aorta with a surgical graft replacement through a posterolateral thoracotomy, with partial or total cardiopulmonary bypass, whereas in patients with visceral or limb ischemia is indicated an aortic fenestration and/or an extra-anatomical bypass. The aim is to replace or repair the dissected or ruptured descending thoracic aorta, restoring peripheral and visceral perfusion (8). Thanks to the progress in surgical technique and anesthesiologist management, the results of conventional open surgery for acute type B aortic dissection, had improved in the last decade, although they remain unsatisfactory with a high mortality ranged from 29% to more than 60% for patients presenting with malperfusion signs or aortic rupture and a high morbidity as a paraplegia rate of 30–36% (8,15,16). Postoperative complications affect from 40% to 80% of patients. Only a few single-center series have reported more favorable results (8,16,17).

For chronic TBAD patients, conventional open surgery is currently reserved for patients with anatomy unsuitable for endovascular treatment or patients with connective tissue disease or patients scheduled for hybrid repair due to inadequate stent graft landing zones (14,17). In chronic type B aortic dissection, with thoraco-abdominal aortic aneurysmatic degeneration and visceral artery involvement, open surgical repair of the thoraco-abdominal aorta through a thoraco-phreno-laparotomy with total cardiopulmonary bypass and deep hypothermic circulatory arrest, is indicated. Open repair is required in about 30% of patients with complicated chronic type B aortic dissection and is associated with higher procedural morbidity and mortality (3,4,18). Recently IRAD review shows that open surgical operative mortality has improved in more recent years, possibly due to improvement in patient's selection and referring to high-volume centers of excellence (18). In recent studies reporting the outcome of patients with chronic type B aortic dissection, who underwent open surgical repair in expert hands, the in-hospital mortality was approximately 9.5–10% with an incidence of neurologic complications of 11% (6% stroke) and severe postoperative complications occurred in about 15% of patients at follow-up reintervention was required in 14% of patients (17,19,20).

Over the last decade thoracic endovascular aortic repair (TEVAR) emerged as a safe and valid alternative to conventional open surgery and has established as the preferred strategy for the treatment of the disease of

the descending thoracic aorta both in acute or chronic complicated TBAD (8). Mean goal of the treatment is the entry tear coverage in the descending thoracic aorta, restoring the distally blood flow in the true lumen with reperfusion of visceral organs or limbs. The false lumen subsequently depressurizes allowing to partial or complete false lumen thrombosis, preventing dissection progression or aortic rupture and increasing the blood flow in the true lumen. Decreasing and regression of the false lumen with remodeling of the aorta is now called "reverse aortic remodeling" (8,17). TEVAR has proven to be superior to the medical therapy alone and versus open surgery with a decrease of the 30-day mortality from 29% to 3–11% (15). As reported by same meta-analyses, mortality of TEVAR for acute complicated dissection ranged from 7.3% to 11.5% with a risk of paraplegia ranging from 1.9% to 6.3%. In patients presenting with visceral malperfusion, although visceral vascular patency is excellent after TEVAR, 30-day mortality remains high (ranging from 30% to 60%) as well as aortic related complications at follow-up (8,15,21).

Although endovascular surgery remains the preferred approach over surgical repair in acute complicated type B aortic dissection, recently here is on-going debate regarding TEVAR role in chronic type B aortic dissection. Critics of TEVAR have supported that a thickened intimal flap in the chronic setting does not reapproximate to the native wall as readily as acute dissections (17). On the contrary a meta-analysis reported by Thrumurthy and coauthors, analyzing 17 studies on 567 patients, showed that the rates of complete false lumen thrombosis and reverse aortic remodeling in patients with chronic type B dissection treated by TEVAR had a median of 86% (range, 38–100%) (22). The mortality rate was 3.2%, with a low paraplegia rate of 0.4%. The VIRTUE registry, a prospective, non-randomized, multicenter European clinical registry, reported no early death after TEVAR for chronic TBAD, with a spinal ischemia rate of 3.8% (23). About late results of TEVAR for chronic TBAD, the Medtronic Thoracic Endovascular Registry (MOTHER) database reports the data collection of 195 patients, with chronic dissection undergone endovascular treatment, followed for an average of 6 years. Overall aortic-related mortality was very low, below 3% within the first 6 years (24).

Endovascular surgery is associated with low procedural morbidity and mortality rates, but these results are partially tempered by higher rates of descending aortic reinterventions during follow-up. In the meta-analysis reported by Thrumurthy the incidence of reinterventions range from 0 to 60% with an average of about 23.1% versus 14.3 % for open surgery (22). Half of the reinterventions were due to stent graft related complications that will almost certainly decrease in frequency over time because of improvements in device design, as well as increased operator experience.

Persistent False lumen patency in chronic type B aortic dissection predicts poor outcome showing a high risk of complications, sudden death, and need for surgery, whereas complete thrombosis of the false lumen has beneficial prognostic value (4,25). Reports from IRAD shows that a partially thrombosed false lumen is associated with increased surgical mortality, and among patients with acute TBAD who are discharged alive, those presenting with a partially thrombosed false lumen had increased mortality (4,25). Recent studies have shown that complete false lumen thrombosis and remodeling of the aorta, are achieved in a median of 86% (ranging from 38% to 100%) of patients with chronic TBAD treated with TEAVR (22). In their study Tolenaar and coworkers report a false lumen thrombosis rate of 32% lower compared to 40–90% reported in other studies (25). Via coverage of the entry tear and directing blood-flow to the true, stent graft placement can induce false lumen thrombosis, promoting reverse aortic remodeling and preventing late expansion and malperfusion. Their analysis shows that a false lumen thrombosis at 1 year was predominantly obtained in patients with partial false lumen thrombosis at presentation and was relative to entry tear location and false lumen diameter and extension. On the contrary patients with a patent false lumen at presentation and branch vessel involvement are less likely to develop false lumen thrombosis and potential have a worse outcome. These findings suggest that these patients may require a more extensive procedure (25).

In our experience, from March 2001 to September 2017, 129 patients were treated for TAAD, 81 for acute (within 14 days) and 48 for chronic dissection (after 14 days). The overall 30-day mortality was 3.9% (5 patients): 3 (3.7%) patients with acute TBAD e 2 (4.2%) with chronic TBAD. All deaths were the result of preoperative malperfusion syndrome. There were no neurological complications or paraplegia. At long-term follow-up, ranging from 1 to 15 years, aortic related mortality was 6.4% and 8.1% for acute and chronic dissection respectively. A secondary endovascular or conventional procedure was required in 25 patients (20.1%), 15.6% and 23.8% for acute and chronic TBAD respectively. There were no statistically significant differences between groups.

However, based on revised literature, TEVAR is a feasible and safe procedure for chronic type B aortic dissection too, with acceptable early and late morbidities and mortality though associated with a high incidence of reinterventions. It should not be performed blindly, considering potential early and late complications, including retrograde type A aortic dissection, endoleaks, spinal cord ischaemia and intimal flap injury due to prosthesis.

Open repair, reserved for patients with connective tissue disease or unsuitable for TEVAR, is predictably associated with higher procedural morbidity and mortality rate. Overall late survival is similar, and a lower rate of secondary descending aortic reinterventions is required.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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