Failure Modes, Complications, and Limitations of Aortic Dissection Treatment

Understanding, predicting, and managing both procedure- and device-based issues.

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Aortic dissections are classified by site (Stanford type A, ascending aorta; type B, descending aorta) and chronicity (acute if onset of symptoms < 2 weeks, chronic if > 2 weeks). The acute group can further be subdivided into complicated and noncomplicated subgroups. Complications, and therefore indications for intervention, include rupture, expanding aneurysms, visceral and lower limb malperfusion, refractory pain, hypertension, and false aneurysm formation.

Endovascular intervention is the preferred treatment option in the acute complicated group, but in the acute uncomplicated group, endovascular treatment is debatable.[1] In patients with chronic presentations, endovascular treatment is gaining acceptance. However, due to the need for recurrent reinterventions, open surgery in this group remains the most common practice in good surgical candidates.[2,3]

Acute uncomplicated type B thoracic dissections are initially managed medically. An expert multidisciplinary panel in the treatment of type B aortic dissection showed that for acute uncomplicated type B aortic dissection, the pooled early mortality rate was 6.4% with medical treatment, 10.2% with TEVAR, and 17.5% with open surgery.[4] Further studies are comparing medical management with endovascular stent grafting, such as in the ADSORB trial.[5]

Acute complicated type B thoracic dissections are best managed by endovascular stent graft treatment, and a survival advantage has been shown compared to open surgery in this group.[4]

Patients with chronic type B thoracic dissections can be managed medically with or without surgery or by endovascular stent graft treatment. The 5-year survival rate with medical therapy alone is only 60% to 80%, due to the progression of the disease and the development of complications in many patients. Progressive aortic dilatation occurs in 59% of medically treated patients, with a mean expansion rate of 1.7 ± 7 mm/year.[4] For patients who received interventional treatment in the first instance, the pooled early mortality rate was 6.6% with TEVAR and 8% with open surgery. Long-term data from the INSTEAD trial, a randomized trial of best medical management with or without endovascular stent grafting, have suggested a significant benefit of TEVAR between 2 and 5 years after intervention, including all-cause mortality (0% vs 16.9%), aorta-specific mortality (0% vs 16.9%), and progression (4.1% vs 28.1%).[6]

DISSECTIONS AT A GLANCE

- Acute uncomplicated type B thoracic dissections should first be treated with medical management.
- Acute complicated type B thoracic dissections are best managed by endovascular stent graft treatment.
- Chronic type B thoracic dissections can be managed medically, with surgery, or by stent graft treatment when there is expansion of the false lumen.
- Type A aortic dissections should be treated with open surgery.

Chronic type B aortic dissection should be managed with optimum medical therapy and close image monitoring, with strategic endovascular intervention when required. Recurrence of symptoms, aortic aneurysmal dilation (> 55 mm), or a yearly increase of > 4 mm after the acute phase are predictors of adverse outcome and need for delayed aortic repair.[4]

The therapeutic advantage of TEVAR can only be maintained by ensuring a low complication rate, and it is therefore important to review the reasons for failure, complications, and limitations of thoracic stent grafting.

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FAILURE MODES, COMPLICATIONS, AND LIMITATIONS

Failure can be defined as progressive aortic dilatation or development of further symptoms and can relate to the number and location of re-entry sites across the dissection membrane.[7] These allow reperfusion of the false lumen, resulting in progressive aortic dilatation, although from the false lumen, visceral branches and the lower limbs may remain perfused. The movement of the dissection flap can also create a dynamic obstruction at the origin of the visceral vessels.

When sealing the primary entry and re-entry sites with the stent graft in the true lumen, potential complications can arise. These are due to the extent of the thoracic aorta that is required to be covered, leading to reduced anterior spinal artery flow from covered intercostal branches and resulting in symptoms of spinal cord ischemia. Bare stents have been used below the covered proximal stent, which covers the primary entry site, and these can be extended distally (PETTICOAT [Provisional Extension to Induce Complete Attachment] procedure).[8] This allows spinal perfusion to be maintained but fails to stop dilatation of the abdominal-segment false lumen in the midterm—although it increases the true lumen volume and reduces total aortic volume in the thoracic segment.[9] If the distal stent graft is too short, it fails to address the lower multiple re-entry sites. The distal end of the stent graft can create another distal re-entry by eroding the dissection membrane, leading to reperfusion of the false lumen and aneurysmal enlargement.[10]

Spinal cord ischemia is a well-known complication, as mentioned previously. Subclavian artery patency is important, as it provides increased spinal perfusion from collateral branches. If the subclavian artery is overstented to gain an adequate seal of the primary aortic tear proximal to the subclavian artery, the use of carotid subclavian bypass allows this collateral supply to be maintained, but this has its own complications of stroke and arm ischemia.[11] The use of chimney grafts to maintain subclavian artery perfusion is another alternative. The length of aortic coverage and patency of the internal iliac circulation also affects the incidence of spinal cord ischemia. The use of spinal cerebrospinal fluid (CSF) drainage is routine and essential with extensive thoracic coverage[11] but can cause complications. Newer, automated CSF drainage (LiquoGuard, Möller Medical, Fulda, Germany) maintaining steady CSF pressure is more reliable than volume-monitored drainage.

Graft misplacement is another cause of failure in which the true lumen is not stented but the false lumen is,[12] although visceral perfusion can still be maintained.[13] Generally, in thoracic dissections, it is vital to stent the true lumen, which is smaller and compressed by the pressurized false lumen. Transesophageal echocardiography and angiography help to confirm the true lumen.

Infolding and graft occlusion are now less common (0.4%) and are related to proximal angulation and excessive graft oversizing.[14]

Aortic perforation is lethal and is initiated by a tear, which is caused by the proximal edge of the stent graft or barbs on the outer curve of the graft not being placed far enough proximally to mold around the aortic contour. The alignment across the arch, with the use of carotid subclavian bypass and placement of a left common carotid chimney parallel graft minimizes the outward force by the proximal stent or barbs.

Similarly, because the thoracic aorta is fragile in patients with dissection, it is important to pay careful attention to the proximal and distal native aortic diameter in order to avoid oversizing the stent graft. The use of tapered grafts to accommodate a smaller-diameter distal landing zone reduces the perforation of the distal dissection membrane. To this end, the proximal sealing zone should not be balloon molded, and the stent graft should not be oversized. Another technique to deploy the thoracic stents safely is to stack the thoracic grafts from below, deploying the smaller-diameter graft distally first.

It is important to calculate the length of the graft accurately using a straightened centerline adjusted to the outer curve and measuring the lumen diameter only,[15] as this is the line that the stent graft takes when deployed. If the first deployed proximal stent graft is too short (< 150 cm), it may cause difficulty in advancement and placement of

the second stent graft. This is due to the first short stent graft not having stability to align around the aortic arch; as a result, it may be prone to forward dislodgement, as the second stent graft will push it upward and outward in the arch.

Type 1a endoleak can be reduced by improved alignment of the proximal stent graft so as to avoid a "bird's beak" deformity, where the stent graft in the inner proximal thoracic curve protrudes into the lumen and is poorly aligned to the inner aortic wall.[16] This has mainly been achieved by advancements in graft material, particularly in terms of flexibility. Various strategies have been used to allow the proximal stent to conform to the aortic arch. The gap between the first and second stent has been enlarged, and the length of the first stent within the proximal stent graft has been reduced to allow better alignment to the curve of the thoracic arch. This is now seen in the new Zenith Alpha thoracic endovascular graft (Cook Medical, Bloomington, IN). Thoracic stent grafts have a trigger wire that restrains the graft from being opened until it is correctly positioned. The conformability among various devices has been studied experimentally, and the Valiant graft (Medtronic, Inc., Santa Rosa, CA) was more conformable in the higher degrees of angulation in the landing zone than Zenith TX2 with Pro-Form (Cook Medical), Conformable Gore TAG (Gore & Associates, Flagstaff, AZ), or Relay (Bolton Medical, Inc., Sunrise, FL). Multiple factors contribute to endoleak development in the treatment of dissections.[17] In one study, they were associated with coverage of the left subclavian artery (complex, $P < .001$), small radius of curvature (for type 1 endoleak and complex endoleaks, $P = .05$), and greatest length of unopposed proximal stent graft (complex, $P < .0001$).[18]

Retrograde dissection, which occurs in 1.3% to 6.8% of cases of type B thoracic dissection treated with endovascular grafting,[19] is also a complication related to the procedure without being device specific and may be prevented by avoidance of stent graft oversizing and the use of proximal stent grafts without barbs.[20] The barbs are not necessary to prevent migration, and they increase the trauma to the proximal intima adjacent to the stent graft; therefore, they are no longer found on any TEVAR devices. Similarly, bare rather than covered stents may have the same effect in an area where the aortic lining is unstable with periaortic hematoma contiguous to the dissection. However, there is no evidence to prove a causal relationship between the use of bare stents as a leading part of stent grafts and occurrence of retrograde dissections. Leading bare stents actually improve the alignment of stent grafts around the aortic arch. Retrograde dissection can cross the origins of the supra-aortic branches and extend to the coronary arteries and pericardium, with lethal consequences.

Deployment technique is important, and technical issues, such as the manner in which the graft releases and lies along the outer curvature of the distal aortic arch, may relate to the initial choice of the centerline that has been used to calculate the length and diameter of the stent graft preoperatively. Software to straighten the proposed line, which is more lateral to the centerline, allows this to be done accurately.[15]

In the actual deployment, attention should be paid to minimizing manipulations of the stent graft, as there is a 2% to 7% risk of stroke from embolization of aortic plaque into the carotid artery. The use of landmarks close to the aorta reduces parallax and allows precision placement. A useful marker in the best left anterior oblique projection ("open arch view") is the endotracheal tube, which is very close to the origin of the left carotid artery. This, when used with repeated angiography in the left anterior oblique projection, allows precise deployment of the proximal stent graft.

Chronic type B dissections should be treated with endovascular stent grafts when there is expansion of the false lumen, which remains pressurized. The length of dissection is a predictor of failure and the need for further intervention. De Bakey type IIIb dissections, which extend from the left subclavian artery to the common iliac arteries, have reduced morbidity compared to open surgical grafting and successfully show remodeling of the thoracic aorta when managed with endovascular stent grafts.[21]

If the stent graft is too short, this may cause erosion of the dissection membrane and a further distal reentry site and extension of the dissection, which requires further procedures to extend the stent graft with adjunctive fenestration for the celiac, superior mesenteric, and renal arteries and stenting of the iliac arteries. Intercostal backflow, seen as a type 2 endoleak, can pressurize the false lumen, and further aortic coverage or embolization of the intercostal branches may be necessary.[22] It is important to note that the extent of the false lumen thrombosis before treatment does not seem to have a significant effect on aneurysm growth in the dissected, nontreated segment, and according to some authors, involvement of the arch in type B dissections does not appear to affect medium-term mortality at 3 years.[22]

Type A aortic dissection is regarded as the final frontier of endovascular treatment.[23] Currently, open aortic surgery is used to treat these dissections, with endovascular management having only an adjunctive role. The replacement of the ascending aorta is used to correct the proximal entry tear, but in a subacute stabilized patient who has ongoing malperfusion of the visceral branches, there is an endovas-

cular option to place a bare stent in the true lumen distal to the replaced ascending segment to correct this.

In a patient with a chronic type A dissection who is in poor physiological condition, a proximal ascending aortic stent graft can be used, but this scenario is further limited by the site of the proximal entry in relation to the coronary arteries and the position of the innominate artery in relation to the sealing zone. Initial open ascending aortic replacement with a secondary endovascular arch branched device placed with branches to the supra-aortic arteries has also been used. Other options of supra-aortic vessel debranching facilitating a proximal landing zone for an arch or ascending thoracic endovascular stent have also been recorded.

OTHER ASPECTS OF CONTROLLING THE LIMITATIONS OF ENDOVASCULAR TREATMENT

Staging of the fenestrations is now useful to reduce the incidence of paraplegia. The principle is to limit the initial thoracic coverage, allowing the spinal perfusion to readjust via collaterals. If using a branched endograft, a perfusion branch can be left open by not completing the visceral vessel revascularization, and the branch can be reconnected at a later date.

Access for thoracic stent graft placement is planned based on the state of the iliac vessels in terms of stenotic disease. Lower-profile sheaths that facilitate this are increasingly available, and the use of surgically created access via the subclavian arteries proximally or via an iliac conduit facilitate the trackability of the device. A "through-and-through" wire is recommended, which is a wire in a protective sheath/catheter that runs from the groin to the subclavian access and stabilizes the graft to allow safe deployment and fenestration or branching. This is not used across an unstable aortic arch, but it can facilitate procedures after the initial proximal dissection is covered. In the context of minimizing embolization, a "through-and-through" wire also helps minimize thoracic graft movement during manipulation and stabilizes the thoracic endograft to allow cannulation for fenestration. Heparin should be administered and closely monitored to keep an activated clotting time of 200 to 250 seconds when manipulating close to the arch branch vessels.

The failure modes and complications are related, and the limitations of endovascular treatment are being reduced with recent device and procedural modification. Despite this, patient suitability, the type and presentation of the dissection, and the challenges posed by thoracic and aortic arch anatomy will continue to have the greatest influence on the eventual treatment of choice. ■

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