

What are the options for treating chronic type B dissections with aneurysmal enlargement?



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Aneurysmal degeneration in patients with chronic type B dissection (cTBAD) is a challenging dilemma, yet endovascular solutions are not without their own challenges. Open reconstruction in young patients via thoracoabdominal repair should always be given strong consideration, but in the frail, elderly population, this is usually not a good solution. When considering thoracic endovascular aortic repair (TEVAR) for cTBAD, we preach patience with the understanding that the exclusion of vulnerable aorta by thrombosing the false lumen may require several staged procedures.

The anatomic limitations can be vast and complicated with this pathology, and we should also know our own limitations with what we can do with endografts and stents. Anatomically, we are sometimes surprised by the reexpansion of a “small” true lumen when challenged by an endograft. Sometimes, diameters of 10 to 15 mm can demonstrate significant remodeling with both tapered and nontapered systems. However, when evaluating a true lumen that is < 10 mm with a thick intimal septum, one should strongly consider other options.

A key difference between acute and chronic dissections is that in a chronic dissection, false lumen dependence is common for end-organ perfusion in one or more visceral vessels. Because the goal would be to thrombose the false lumen in this setting, the treat-



Figure 1. Reestablishing continuity of flow to the left renal artery from the true lumen was accomplished successfully with the deployment of a bridging covered stent.

ment strategy usually involves first reestablishing continuity of the visceral vessels dependent on false lumen flow back to the true lumen. This can be achieved with bridging the end-organ vessel with a covered stent delivered through the true lumen (Figure 1). Aggressive entry and reentry tear coverage can then commence, excluding the aneurysm and minimizing false lumen flow. Commonly, you will find codependence between true and false lumens with cTBAD, and a covered stent via the true lumen can be efficacious here as well.

TEVAR is meant to cover the primary entry tear and distal thoracic reentry tears. Therefore, with cTBAD, extensive coverage of the thoracic aorta to the celiac is usually the norm with minimal (5%–10%) oversizing. Don't be discouraged when the postoperative CT angiogram (CTA) shows the false lumen is equally opacified compared to the first CT scan. Numerous reentry tears are not obvious during your first procedure, and leaving an obvious reentry tear uncovered can be advantageous moving forward.

Indeed, the false lumen is rarely thrombosed after the first intervention, even if one does extensive false lumen exclusion with covered stents and endografts. Therefore, leaving an obvious reentry tear alone is not going to set you back in your ultimate goal. I frequently and purposefully do this (preferably in the iliac artery) to provide myself easy access to the false lumen for subsequent procedures. Leaving this access allows me to perform direct interventions within the false lumen. A number of options exist, such as coiling outflow vessels, gelfoam-thrombin slurry, plugs, etc. Usually, these strategies are employed during your second procedure, once you are certain no further communication exists between the false lumen and visceral vessels. If there is still the question of flow from the false lumen to a visceral vessel, these embolic adjuncts can be catastrophic, so be sure. Complete closure of that final reentry tear should be delayed until you are satisfied with your false lumen treatment.

Finally, there is no shame in achieving only partial false lumen thrombosis. As long as the largest vulnerable area is thrombosed, one can cautiously anticipate that protection from rupture has been conferred, provided there is no further expansion. Additionally, we have seen regression of false lumen size with minimizing or decreasing false lumen flow. These strategies require the utmost attention to follow-up with a routine plan of systematic measurement performed by the operating surgeon. I usually get a 30-day, 3-month, and 6-month CTA followed by a yearly surveillance CTA if false lumen flow persists. If false lumen flow exists only in the abdominal aorta, duplex ultrasonography can be reliable.

TEVAR can make a significant impact in the treatment of chronic type B aortic dissections for aneurysmal degeneration. The key to treatment is to ensure adequate visceral flow through the true lumen, exclude entry and reentry tears with covered stents and endografts, and directly thrombose the false lumen if necessary. This modality can be performed safely as long as patients have suitable anatomy and meet criteria for repair.



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Acute type A and B dissection are very serious conditions that seem to occur more and more in our hypertensive population. Whereas acute type A dissection is usually treated immediately by open surgery, acute type B dissection can be treated either conservatively or by endovascular means.

Whatever the type and the treatment, these patients need to be followed for life, as a number of them will develop aneurysmal degeneration of the whole aorta. Indeed, the dissection rarely stops at the level of the thoracic aorta, and the resulting aneurysmal degeneration is to be considered a thoracoabdominal aneurysm.

In our opinion, the risk of rupture associated with this thoracoabdominal aneurysm is the main indication to treat these patients. It is very rare to see late malperfusion problems. If one accepts that the main indication is the treatment of the thoracoabdominal aneurysm with complete exclusion as a goal, a number of options are excluded. We personally do not believe in standard thoracic stent grafting without a perfect distal seal in these patients. Even if there is no flow in the false lumen, pressure transmission will usually result in ongoing growth of the aneurysm and keep the patient at risk for rupture.

Open repair is the gold standard but represents a very difficult procedure (open thoracophrenolaparotomy with left-left bypass, or even two staged procedures including arch repair with [frozen] elephant trunk and later a thoracophrenolaparotomy). Not many centers can offer this procedure with acceptable outcomes, and our educated guess is that many postdissection aneurysms are therefore left alone, or to rephrase, not offered treatment.

An endovascular option with custom-made stent grafts featuring fenestrations and/or branches was not considered for many years, although more and more patients with arteriosclerotic thoracoabdominal aneurysms were offered an endovascular option. The reasons were obvious to all centers using advanced endovascular techniques. First, the usually very small true lumen was regarded as a contraindication. Second, the fact that visceral branches of the aorta could originate from both the true and the false lumen was regarded as a major technical hurdle. Third, both proximal and distal access as well as landing zones were not always available or ideal.

A few years ago, we attempted to treat a postdissection aneurysm with a fenestrated/branched stent graft. It was possible to stent all visceral vessels, and the outcome was fine on CTA. This initial experience pushed us to pursue this experience, and others followed. Although both the planning and technical execution proved more difficult than in arteriosclerotic thoracoabdominal aneurysms, overall results were acceptable. Publications from our center alone or in combination with Regensburg and the Cleveland Clinic have demonstrated the feasibility of the technique. The two major concerns were addressed with success. It was shown that working in a small true lumen was possible and that the dissection flap could gradually be moved away. Second, moving from one lumen to the other was the least of our problems. Almost every single time, it was no problem to find a way through the dissection membrane, suggesting that there are more entry and re-entry tears than we can identify with imaging techniques. This also suggests that simple thoracic stent grafting is not efficient in postdissection thoracoabdominal aneurysms. Only twice did we need to perforate the dissection flap with the back of a wire through a guiding sheath or even with a transjugular intrahepatic portosystemic shunt needle.

In my opinion, dissection patients need better follow-up after their acute dissection. A significant number of them should have treatment for their postdissection aneurysmal enlargement. Open repair is certainly the first option when patients are young and healthy enough to undergo such a major procedure. We are still at an early stage with the use of fenestrated and/or branched stent grafts to treat this specific type of pathology, but our results demonstrate the potential of this technique, and it is far less invasive than open surgery and therefore applicable to more patients. In view of the nature of the disease, both open and advanced endovascular repair of postdissection thoracoabdominal aneurysms should be performed in high-volume, experienced centers.



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Answering this question for chronic dissection is a challenge because it is a disease process that can occur in a wide demographic of individuals. The younger patient with dissection requires far different consideration compared with the octogenarian, and the pres-

ence of a known or suspected connective tissue disorder or an untreated proximal aneurysm further complicates the discussion. These are pertinent issues because they carry implications for the development of aneurysms in contiguous aortic territories over time, and they will have a bearing on the durability of the repair.

For younger patients or those with known connective tissue disorders, I think the standard of care is open thoracoabdominal repair. This should be done at an aortic center with a high volume of experience and with access to cardiopulmonary bypass, neurologic monitoring, and a strong critical care service with experience with the postoperative coagulopathy that is frequently seen. In these cases, the maximum aortic diameter that serves as the threshold for intervention is controversial. For most older patients, I would advocate 6 cm as a trigger for operative intervention. However, for patients with Marfan syndrome or disorders of the TGF-beta signaling pathway, the threshold is likely smaller, although there is no clear evidence to guide this decision. For the young patient with no diagnosed connective tissue disorder or for the patient with a strong family history, I may also consider a smaller threshold after weighing all the risks and a long discussion with the patient.

In older patients or those with considerable comorbidities, I think that endovascular repair is a reasonable option. Although we don't know the durability of this approach, it allows for a staged approach to the treatment, which may decrease the risk of spinal cord ischemia and also makes the entire repair easier to tolerate. Fenestrated devices are preferred over branched devices because of the often-restricted true lumen that poses a challenge to access branch arteries, as well as forcing branch stents into configurations that may impact their long-term durability. Patients with a particularly restrictive true lumen provide another reason to stage the repair: placement of a proximal thoracic stent may help dilate the true lumen during the period of manufacturing delay.

Occasionally, after endovascular repair, the false lumen remains perfused via the divided lumen of visceral branches, intercostal, or lumbar arteries. This is a dilemma because the conventional treatment of type II endoleaks would advocate branch embolization, which is not possible in two of the three mentioned sources. In this case, I have followed the practice that intervention should only occur in case of growth, and in that case, placement of thromboembolic devices such as plugs or coils in the false lumen is far preferable to glue.

What's most important is that chronic dissection requires a tailored therapy, so a one-size-fits-all

approach is unlikely to work in the majority of cases. Careful consideration of the patient, his or her clinical scenario, and the possibility of connective tissue disorder lurking under the surface are all important for a successful outcome.



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The current standard of care for treating uncomplicated type B aortic dissections and for management of extensive dissections treated by proximal aortic repair is medical therapy. Medical therapy is effective in more than 80% of patients. However, long-term outcomes in patients who live long enough for their dissections to reach the chronic phase leave much to be desired. It has been estimated that more 50% of patients with uncomplicated dissections develop aneurysmal dilatation either requiring repair or leading to rupture.

Endovascular repair in this setting aims to decompress false lumen perfusion. This is accomplished by covering the proximal tear, which promotes aortic remodeling by causing false lumen thrombosis and true lumen expansion. For chronic aortic dissections, the typical strategy consists of endovascular coverage from a suitable proximal landing zone (typically in the distal arch or proximal descending thoracic aorta) to the level of the celiac axis.

Although the results of TEVAR for chronic aortic dissections have shown promise with respect to morbidity and mortality rates, morphological changes in the aorta have been less favorable compared to those achieved in the acute phase. Thrombosis of the false lumen is highly variable (50%–100%), averaging > 70% in most reports. TEVAR for chronic aortic dissections has been shown to effectively decrease maximal aortic diameter in the stent grafted aorta, but distal aortic segments tend to continue to increase in patients with extensive dissections. Overall, approximately 15% to 30% of patients experience aortic enlargement after TEVAR, and 4% to 34% require open or endovascular reinterventions for endoleak, sac enlargement, or rupture.

Endovascular strategies to deal with continued sac enlargement after TEVAR for chronic dissections include selecting one of the following.

False Lumen Exclusion

False lumen exclusion can be accomplished with placement of coils or plugs in the false lumen, in general at the distal level of the stent graft. Large 0.035-inch coils or plugs can be utilized. Kolbel and associates described a technique of using a “candy plug” that conforms to the false lumen, thereby providing a more reliable exclusion. I have used specially manufactured plugs by Cook Medical Inc. (Kolbel, personal communication) with good results in selected patients.

Alternatively, the Hamburg group described the “knickerbocker” technique of relining the true lumen in the descending thoracic aorta with an oversized thoracic tubular endograft, followed by controlled rupture of the dissection membrane using a large compliant balloon within the graft’s midsection. This allows expansion of the stent graft into the false lumen, resulting in occlusion. Preliminary results in three patients showed successful exclusion, but clinical data on routine false lumen exclusion are lacking.

Occlusion of Reentrance Sites

Patients with isolated reentrance sites are the exception and not the rule. However, if an isolated reentrance site can be accurately located and excluded by placement of a covered stent or plug, this may be indicated. One limitation is that placement of renal stents may interfere with definitive total endovascular repair using fenestrated, branched, or parallel stent grafts as described in the following sections.

Completion Endovascular Repair

Aortic remodeling with endovascular repair can only be consistently predicted by placement of the stent graft in normal aorta with complete coverage of intimal tears. Because most patients have multiple reentrance tears across the visceral segment, treatment of aortic enlargement after TEVAR for aortic dissection remains a challenge. Hybrid procedures have been used with mixed results.

The application of fenestrated and branched endografts to patients with chronic dissection has been limited because of unfavorable anatomical features, namely the narrow true lumen, variable vessel anatomy with origin from false lumen, thick intimal flap with multiple mature fenestrations, lack of distal seal zones and friable aortic tissue, which is more prone to retrograde aortic arch dissections.

Finally, we have performed an increasing number of fenestrated and branched endograft procedures for chronic dissections. I believe this will continue to have increasing application and will ultimately be the preferred technique to deal with this problem. ■