

Treatment Paradigms for Type B Aortic Dissection–Related Malperfusion and Rupture

Assessing the pathophysiology and endovascular treatments for complicated type B aortic dissection with malperfusion or rupture.

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Type B aortic dissection (TBAD) is characterized by an intimal tear occurring distal to the left subclavian artery and propagating for variable distances down the thoracic or thoracoabdominal/iliac arteries. Generally speaking, TBADs can be uncomplicated or complicated, the latter of which are further subdivided into those causing malperfusion or associated with rupture. Complicated TBADs (cTBADs) should undergo urgent repair, with most benefiting from thoracic endovascular aortic repair (TEVAR). Indeed, TEVAR has been shown to be superior to open surgical repair of cTBAD with improved mortality rates and reduced morbidity compared with open surgical cohorts.¹⁻³ This article details the pathophysiology and treatments for each of these types of cTBADs.

MALPERFUSION

Malperfusion is characterized by end-organ or limb ischemia due to propagation of the intimal tear affecting blood flow to the viscera or lower extremities. In all cases, some degree of true lumen compression within the aorta exists. In 25% to 50% of cases, branch vessel compromise exists.⁴ The most common form of malperfusion is called *dynamic obstruction*, where the dissection flap does not extend into the branch vessel but where the true lumen becomes compressed and there is resultant compromised flow into the branch vessel emanating from the true lumen. The dissection septum may even prolapse into the orifice of the branch vessel during cardiac systole, but importantly, in dynamic

obstruction, the tear does not involve the branch vessel. In contrast, *static obstruction* is characterized by flap extension into the branch vessel. When this occurs, the origin and more distal segment of the branch vessel can become narrowed. In long-standing cases of static obstruction, thrombus may develop past the area of obstruction, which often requires thrombectomy or other mechanical means to remove the occlusive thrombus.

Distinguishing between dynamic and static obstruction, as well as early and late static obstruction, is critical to successful treatment of malperfusion due to branch vessel compromise. Although there are nuances that can exist (at times, the dissection flap can extend into the branch vessel or the flap can autofenestrate, eliminating the potential of a compressed true lumen in the branch vessel), these general categories provide guidance in treating TBAD with malperfusion. Thus, careful review of axial imaging, taking note of whether branch vessels originate from the true or false lumen, and careful measurements of intended landing zones are critically important for optimizing the success of the procedure.

Treatment

The majority (> 80%) of malperfusions occur due to dynamic obstruction.⁵ In these cases, coverage of the primary entry tear, effectively repressurizing the true lumen, restores perfusion to those branch vessels exhibiting signs of compromised perfusion. The main

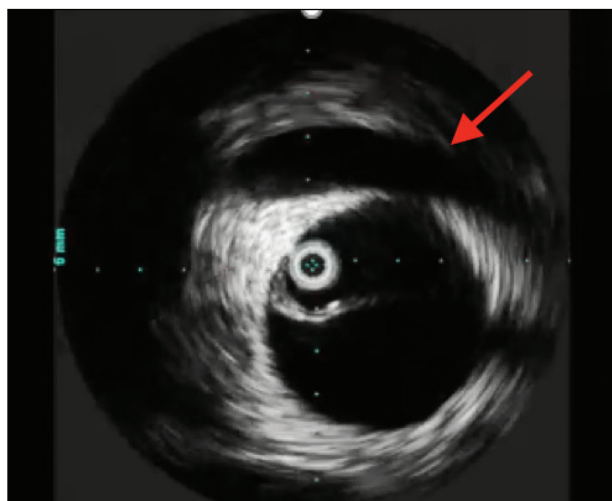


Figure 1. IVUS with left renal vein crossing anteriorly (arrow). Note the IVUS catheter in the true lumen.

femoral access side is chosen by evaluating which is less impacted by the dissection and/or lends the most direct access to the true lumen. Because the entry tear is either in zones 2 or 3, some degree of coverage of the left subclavian artery is often required. The thoracic stent graft should never be oversized beyond 10% when placed in an acutely dissected aorta, as further oversizing has been associated with the dreaded complication of retrograde type A dissection (RTAD).⁶ Although proximal bare stents have not been shown to be associated with RTAD,⁶ the proximal stent should never be ballooned as this could precipitate retrograde dissection in an acutely inflamed, fragile aorta. Intravascular ultrasound (IVUS) should be used as an adjunct to ensure that wires and catheters are in their intended locations in the true lumen prior to device deployment. The left renal vein crossing anteriorly over the aorta is helpful for orientation through the visceral segment (Figure 1).

We traditionally carry out aortic stent graft repair down to the diaphragm, as this effectively improves hemodynamics through the aorta and spares important intercostal arteries that exist above the celiac axis. In instances of visceral malperfusion, a steep lateral oblique arteriogram is useful to evaluate the takeoff of the celiac and superior mesenteric artery (SMA). It can also evaluate the SMA along the length of the vessel, which is important in long-standing cases of static obstruction where more distal dissection extension or thrombus can be present. The standard anterior-posterior angiographic view can be used to ensure that flow through the celiac axis is unencumbered to the proper hepatic artery and that there is good flow into at least one of the renal vessels. Additional branch stenting,

ensuring that the proximal aspect of the stent extends a few millimeters into the aorta and true lumen, should be performed if continued branch vessel stenosis or occlusion is noted. In instances of lower extremity ischemia, aortoiliac arteriography should be performed to evaluate the need for further iliac stenting. Continued end-organ ischemia (ie, SMA thrombus distal to the stented portion or continued limb ischemia distal to the iliac stent) can be rectified with direct exposure and thrombectomy as needed.

Unique Clinical Scenarios

In the case of complete aortic occlusion as a complication of TBAD, coverage of the primary entry tear is critical, and entry into the true lumen either via the femoral artery or the axillary artery should be performed. Difficulty entering the true lumen from below can be facilitated by femoral cutdown and arteriotomy with direct access of the true lumen aided by visualization. Alternatively, stent graft delivery from the right axillary artery can be performed as well. After thoracic stent graft deployment, arteriography should be performed to ensure that there is adequate flow through the visceral vessels as well as iliac arteries, with additional stenting performed as needed.

Malperfusion due to dissection extending down the aortoiliac segment has also been approached endovascularly and takes advantage of the fact that there is often a fenestration in the distal abdominal aorta. After gaining wire access from the true lumen in one groin and the false lumen in the other groin, the wire is snared and transfemoral access across the dissection septum is achieved. A balloon is used to open the fenestration, which allows for pressure equalization between the true and false lumen.⁷

Another method that has been described is one in which the wire is snared from the true lumen and the wire is pulled down, effectively enlarging the fenestration, analogous to a “cheese wire.” Advocates of this method have described it as creating a seal zone in the distal abdominal aorta with further stenting applied as needed to prevent migration of the friable dissection flap distally.⁸ Open surgical fenestration has been described as well, with septectomy performed in the visceral aorta to relieve malperfusion and suture repair of the aorta buttressed with Teflon felt. With continued leg malperfusion due to compromised inflow, consideration should be given to extra-anatomic femoral-femoral or right axillary to femoral-to-femoral artery bypass as needed.

Although these fenestration techniques have been successfully reported, the long-term consequences of

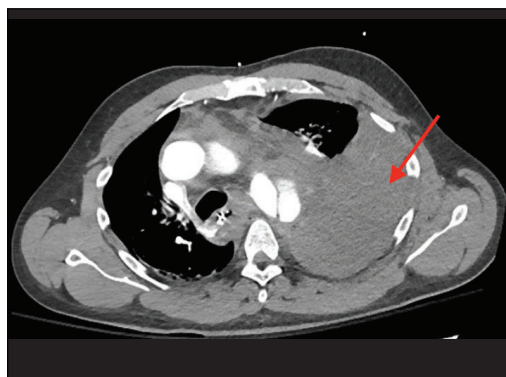


Figure 2. Ruptured type B dissection with large hemothorax (arrow).

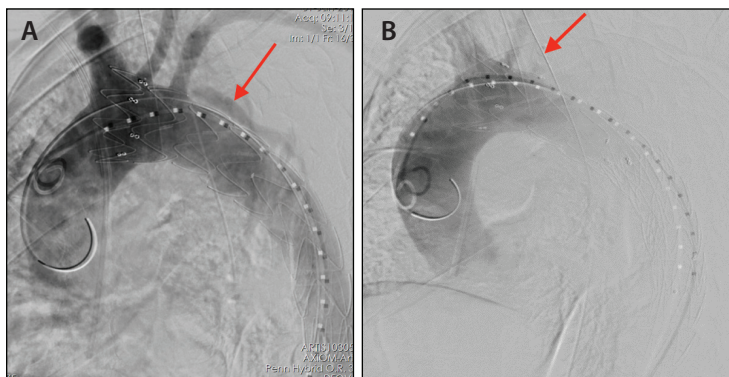


Figure 3. TEVAR with coverage of the left subclavian artery and continued false lumen filling (arrow) (A). TEVAR and Amplatzer plug placement (arrow) with diminished false lumen filling (B).

equal pressurization of the false lumen remain unclear, with one study reporting an increase in total aortic diameter at follow-up.⁹ In our experience, thoracic stent grafting effectively expands the true lumen and only in rare cases does additional branch vessel stenting need to be performed for malperfusion. The need for endovascular or open surgical fenestration has not been borne out in our practice.

RUPTURE

Rupture complicating a TBAD is rare and is due to rapid expansion of the weakened false lumen wall. Patients presenting with a rupture of their TBAD are often in extremis and have an associated large left hemothorax (Figure 2). TEVAR is an appropriate initial treatment strategy for these patients, as it allows them to be stabilized before a more definitive open surgical repair, if needed. It is important to review axial imaging to ensure that an adequate diameter stent graft is placed because these patients may be hypotensive and thus have a relatively undersized aortic diameter at the time of CTA. Placement of an Amplatzer Vascular Plug (Abbott Vascular) or ligation of the left subclavian artery is required to prevent backbleeding from the left subclavian artery (Figure 3).¹⁰ The presence of distal reentry tears can be an ongoing source of bleeding and these need to be addressed with further stent graft extension and/or branch vessel stents, extending from the true lumen into the vessel as necessary. Although TEVAR is effective at stabilizing the situation in most cases, definitive surgical repair should be performed for ongoing bleeding. This typically involves some form of arch replacement with preservation of a portion of the thoracic stent graft as a form of frozen elephant trunk.

CONCLUSION

Although malperfusion and rupture are catastrophic complications of TBAD, the majority of patients can be effectively managed with TEVAR, with or without adjunctive endovascular techniques. Open surgical repair may be required after performing TEVAR for rupture in the presence of ongoing bleeding. ■

1. Cambria RP, Crawford RS, Cho JS, et al. A multicenter clinical trial of endovascular stent graft repair of acute catastrophes of the descending thoracic aorta. *J Vasc Surg.* 2009;50:1255-1264.
2. Bavaria JE, Brinkman WT, Hughes GC, et al. Outcomes of thoracic endovascular aortic repair in acute type B aortic dissection: results from the Valiant United States investigational device exemption study. *Ann Thorac Surg.* 2015;100:802-808; discussion 808-809.
3. Fattori R, Tsai TT, Myrmmel T, et al. Complicated acute type B dissection: is surgery still the best option?: a report from the International Registry of Acute Aortic Dissection. *JACC Cardiovasc Interv.* 2008;1:395-402.
4. Cambria RP, Brewster DC, Gertler J, et al. Vascular complications associated with spontaneous aortic dissection. *J Vasc Surg.* 1988;7:199-209.
5. Williams DM, Lee DY, Hamilton BH, et al. The dissected aorta: percutaneous treatment of ischemic complications—principles and results. *J Vasc Interv Radiol.* 1997;8:605-625.
6. Canaud L, Ozdemir BA, Patterson BO, et al. Retrograde aortic dissection after thoracic endovascular aortic repair. *Ann Surg.* 2014;260:389-395.
7. Slonim SM, Nyman U, Semba CP, et al. Aortic dissection: percutaneous management of ischemic complications with endovascular stents and balloon fenestration. *J Vasc Surg.* 1996;23:241-251; discussion 251-253.
8. Clair DG. Aortic dissection with branch vessel occlusion: percutaneous treatment with fenestration and stenting. *Semin Vasc Surg.* 2002;15:116-121.
9. Beregi JP, Haulon S, Otal P, et al. Endovascular treatment of acute complications associated with aortic dissection: midterm results from a multicenter study. *J Endovasc Ther.* 2003;10:486-493.
10. Trimarchi S, Segreti S, Grassi V, et al. Emergent treatment of aortic rupture in acute type B dissection. *Ann Cardiothorac Surg.* 2014;3:319-324.

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