Chronic Dissections: Are Endovascular Options Viable?

The success of TEVAR in the treatment of chronic dissection aneurysms depends on the extent of disease.

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Over the past decade, thoracic endovascular aortic repair (TEVAR) has been established as the gold standard in the treatment of patients with acute complicated type B dissection.[1] Despite the lack of level 1 evidence, the benefits of TEVAR in the life-threatening setting of type B dissection with rupture or malperfusion are evident when compared to open repair. Uncomplicated type B dissection is traditionally managed medically with aggressive antihypertensive treatment. With time, approximately half of the patients with chronic dissection will develop an expansion of the false lumen with thoracic or thoracoabdominal false lumen aneurysm, which is the main challenge of dissections in the chronic setting.[1]

Indications for surgical or endovascular treatment of chronic dissection aneurysm include dilatation of the aorta to > 5.5 cm, symptomatic dilatation, and rapid expansion defined as > 1 cm/year. Open surgical treatment is associated with significant mortality and morbidity. This is why endovascular repair has been advocated as a potential treatment option. TEVAR aims to cover the primary entry tear in the descending aorta, resulting in a reduction of flow and pressure in the false lumen and formation of false lumen thrombosis (Figure 1). Although TEVAR for treating chronic type B dissection has been reported in numerous single-center and registry-based analyses, the benefit of this treatment is still often debated. The presence of a rigid chronic dissection flap and multiple chronic reentries often located distal to the treated thoracic aorta could have a negative impact on outcome. For patients with dissection extending into the abdominal aorta,

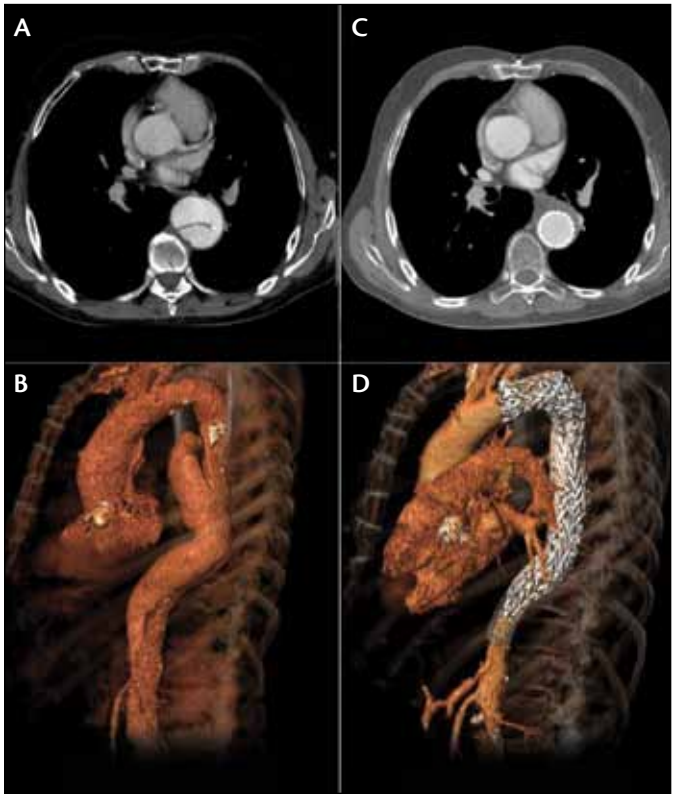

Figure 1. Chronic type B dissection with false lumen expansion treated with TEVAR (A). The primary entry tear is located at the mid-descending thoracic aorta (B), resulting in a type IIIB aortic dissection extending into the abdominal aorta (C). Two years after TEVAR, the aorta has remodeled, the true lumen has expanded, and the false lumen has reduced in size (D). TEVAR was performed from the left subclavian artery to the level of the celiac trunk. This patient later had infrarenal EVAR performed to cover the distal entry tear in the abdominal aorta and iliacs.

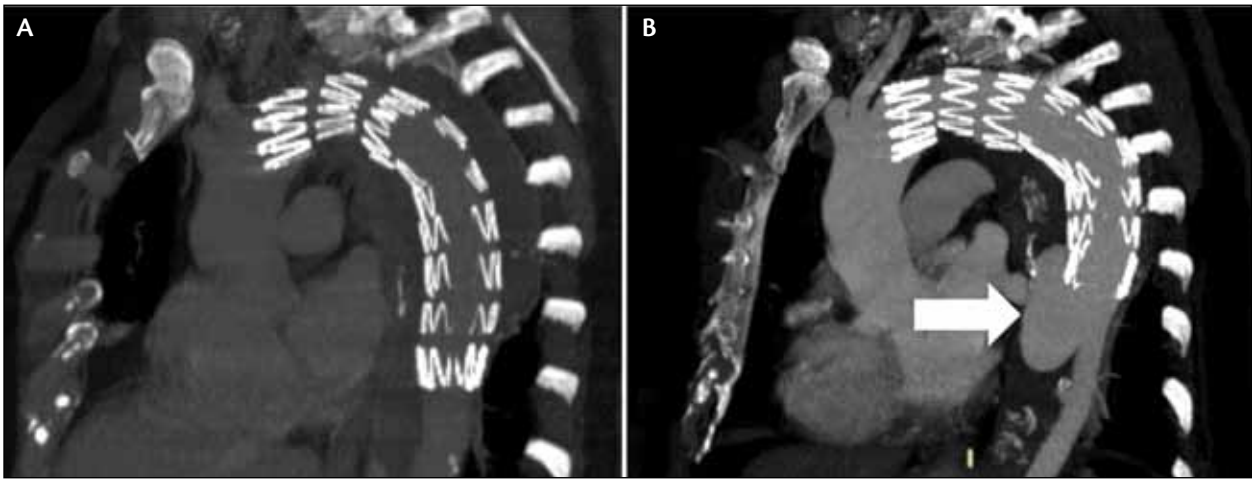

Figure 2. This patient with a chronic type B dissection was treated with TEVAR with an endograft covering the primary entry tear (A). The graft was sized according to the diameter of the aorta at the level of the left subclavian artery. The distal end of the graft is constrained by the reduced diameter of the true lumen. At 4-year follow-up, a new entry has occurred at the distal end of the stent graft (B; white arrow), due to erosion of the intimal flap. Distal stent graft extension was planned, but the patient died from aortic rupture 1 week before the planned intervention.

TEVAR is seldom a definitive treatment, and the continued perfusion of the distal lumen could create further problems.

SHORT-TERM OUTCOMES OF TEVAR FOR CHRONIC DISSECTION

In a review of endovascular treatment of chronic type B dissection, Thrumurthy et al analyzed 17 studies on TEVAR for chronic dissection, identifying 567 reported cases.[2] The studies included patients treated over a 15-year period, with varying chronicity, symptoms, and extension of disease. Technical success (defined as closure of the primary entry tear without endoleak, conversion, or death) was 90%. The reported 30-day mortality rate was 3.2%, with a paraplegia rate of 0.4%. These results compare favorably with open repair, which has a reported 7% to 11% mortality and 6% to 12% paraplegia risk.[3-5]

Paraplegia and retrograde dissection are two of the most dreaded complications after TEVAR for type B dissection. Retrograde dissection occurs in 1% to 4% of patients and is more prevalent when the proximal landing zone is in the arch.[6] Ballooning of the proximal stent graft should be avoided in TEVAR treatment of all type B dissections due to an increased risk of retrograde dissection in patients with fragile aorta. For the same reason, some authors advise against active proximal fixation utilizing hooks or barbs. Spinal cord ischemia rates are reportedly lower after TEVAR than after open repair, but this complication remains a concern and has been reported in up to 10% of patients treated with TEVAR for chronic dissection.[7] To reduce the risk of spinal cord ischemia, revascularization of the left subclavian artery is often recommended in the elective setting, especially in patients with thoracoabdominal extension of the dissection who may require extensive aortic coverage over time.[8] Cerebrospinal fluid drainage is a useful perioperative adjunct to reduce the risk of spinal cord ischemia when extensive descending thoracic aortic coverage is performed.[9]

AORTIC REMODELING

In the long term, the success of TEVAR for chronic dissection depends on remodeling of the dissected aorta to reduce the risk of rupture. In the acute setting, TEVAR results in early significant aortic remodeling with expansion of the true lumen and reduction or even obliteration of the false lumen.[9,10] The potential for significant remodeling also remains in the subacute phase of chronic dissections, as shown in the INSTEAD-XL trial.[11] In this trial, the median time from dissection to randomization was 39 days; 90% of the patients achieved false lumen thrombosis at the thoracic level, and morphological evidence of remodeling was present in 79% of the cases.

In patients treated with TEVAR for an established false lumen aneurysm after dissection, the remodeling of the aorta is less prominent.[10] Reduction of the aortic diameter relies on rerouting of flow into the true lumen and reduction of pressure in the false lumen, which is signified by false lumen thrombosis on follow-up. The reported rate of complete false lumen thrombosis after TEVAR for chronic dissection varies between reports, from 38% to 100%.[2] This variation could potentially depend on case mix and the extent of disease, as well as mode of assessment of patency

of the false lumen.[12] Complete false lumen thrombosis is much more likely in patients with dissection limited to the thoracic aorta (DeBakey type IIIA) compared to when the dissection extends to the abdominal aorta (type IIIB).[6,9] Additional selective coverage of a distal reentry may facilitate thrombosis of the false lumen distally to the aortic stent graft (eg, by stent grafting a renal artery perfused by the false lumen).

As in EVAR treatment of infrarenal aortic aneurysms, reduction of the aortic diameter serves as a surrogate marker for successful intervention in TEVAR for chronic dissection. Diameter reduction of the aorta occurs mainly in patients with extensive false lumen thrombosis. In a study by Scali et al, patients with complete false lumen thrombosis had a mean reduction of aortic diameter of 8 mm over follow-up, compared to no reduction in patients with partial thrombosis.[7] In another study, only 5% of the patients with complete false lumen thrombosis experienced expansion of the aortic diameter over follow-up, compared to 40% of those with a patent false lumen at the level of the stent graft.[6]

LONG-TERM OUTCOME

Over time, a significant number of patients treated with TEVAR for chronic dissection require reintervention.[2] The rate of reintervention is estimated at 30% at 3 years[6,7] and includes proximal and distal stent graft extensions as well as interventions to aortic branches or conversion to open repair. The reintervention rate is higher in patients with extensive dissection involving the thoracoabdominal aorta.[9] Although thrombosis of the false lumen is a positive factor for long-term outcome, late failures of TEVAR may occur with rupture of the intimal flap, resulting in pressurization of the false lumen with risk for catastrophic outcome (Figure 2). Thus, regular imaging follow-up with CT or MR angiogram remains mandatory for these patients, as well as optimal medical treatment with aggressive antihypertensive medication.

Long-term survival rates after TEVAR vary significantly between studies, from 60% to 100% at 4 years.[2] This variation in outcome could be related to differences in case selection. Although there are no direct comparative studies between open and endovascular repair for chronic dissection, retrospective reports indicate similar long-term outcome despite lower perioperative mortality and morbidity rates for TEVAR.[5]

Long-term outcome after TEVAR is related to thrombosis of the false lumen and aortic remodeling. In an analysis of 58 patients treated with TEVAR for chronic dissection, the 3-year survival rate was 89% for patients with aortic remodeling, defined as > 0.5-cm reduction of the aortic diameter, compared to 54% in those with lack of remodeling.[6] In the same group of patients, aortic remodeling depended on the extent of false lumen thrombosis, with significant aortic diameter reduction during follow-up in patients with total thrombosis of the false lumen or thrombosis at least along the full length of the stent graft.[6] As total false lumen thrombosis is more prevalent in patients with type IIIA dissection, the long-term outcome of TEVAR for chronic dissection is superior in this group of patients.

CONCLUSION

The success of TEVAR for treatment of chronic type B dissection depends on the extent of disease. In the short term, TEVAR offers superior outcome when compared to open surgical repair, with a significantly lower mortality rate and lower risk for spinal ischemia. Thrombosis of the false lumen and aortic remodeling are surrogate markers for success and predict long-term outcome. Remodeling is less prominent than in the acute dissections and occurs more often in patients with type IIIA dissection limited to the thoracic aorta who have a superior long-term outcome. Late failures do occur, and reintervention rates remain high; thus, TEVAR cannot be regarded as a definitive treatment for patients with chronic dissection, and vigilant follow-up imaging remains mandatory. ■

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