Thoracic endovascular aortic repair for chronic distal aortic dissection: The jury is still out

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It is no surprise that after the successful application of endovascular therapy to treat atherosclerotic descending thoracic aortic aneurysms, thoracic endovascular aortic repair (TEVAR) is now being used for distal aortic dissection. TEVAR has revolutionized the treatment of patients with acute DeBakey III aortic dissection complicated by malperfusion or rupture and is universally acknowledged as the standard of care.¹ However, the role of TEVAR in the treatment of chronic distal aortic dissection has yet to be elucidated.

The article by Dr Roselli² eloquently outlines the pertinent issues currently surrounding open versus endovascular therapy for the treatment of chronic, complicated (ie, aneurysmal degeneration) distal aortic dissection. Open replacement of dissected, aneurysmal segments of the descending thoracic and abdominal aorta remains the gold standard and can be accomplished with mortality ranging from 2% to 8% in high-volume centers.³⁻⁵ These procedures are durable with 5-year survival of 70% to 79% and 5-year freedom from reintervention rates of 85% to 92%. However, these procedures are highly morbid and carry significant risks of adverse renal, respiratory, and neurologic events.

The minimally invasive TEVAR procedure is typically performed via a transfemoral approach that can be accomplished via a small groin incision or totally percutaneous access. The technical goal of TEVAR for chronic aortic dissection is to cover the primary intimal tear and all distal aortic fenestrations down to the celiac axis. The success of the procedure is based on the long-term capacity of the endograft to promote (1) true lumen expansion, (2) false lumen thrombosis and shrinkage, and (3) stabilization or a reduction in total aortic diameter. These changes are broadly categorized as “reverse aortic remodeling.” TEVAR has been demonstrated to be a safe procedure for chronic complicated distal aortic dissection with an operative mortality of

less than 5%. TEVAR also has been shown to significantly reduce the renal and neurologic complications associated with open descending/thoracoabdominal aortic replacement and eliminate the pulmonary complications by avoiding a thoracotomy. As with all endografting procedures, the trade-off for the reduction in perioperative morbidity and mortality is a higher reintervention rate compared with open descending aortic replacement.⁶⁻⁹

If endograft coverage of all intimal tears is feasible, as in DeBakey IIIa dissections, TEVAR is highly effective in remodeling the aorta and reducing total aortic diameter. However, the majority of chronic distal aortic dissections extend through the visceral aortic segment to the aortic bifurcation (residual DeBakey I or DeBakey IIIb). As Dr Roselli² astutely emphasizes, these are the challenging patients in whom the efficacy of TEVAR is unclear. Reverse remodeling in these patients is inconsistent because of the presence of distal aortic fenestrations. These distal communications may result in persistent false lumen perfusion and the prevention of false lumen thrombosis and shrinkage. Indeed, the series by Kang and colleagues⁶ from Cleveland Clinic report a 78% success of total false lumen thrombosis in patients with IIIa dissections, but only a 13% total false lumen thrombosis with IIIb dissections. Similar results have been reported by several other high-volume aortic centers.⁷⁻⁹

A recent report from Hughes and colleagues⁹ demonstrated aneurysm sac regression greater than 1 cm in 89% of patients with chronic DeBakey IIIb dissections with aneurysmal degeneration limited to the descending thoracic aorta alone. These encouraging results highlight the concept that TEVAR will be highly efficacious in select cohorts of patients with chronic dissection who have favorable anatomy (ie, DeBakey IIIa, limited distal fenestrations). The current challenge is to identify which anatomic variants of distal aortic dissection possess “favorable anatomy.” Unfortunately, the existing data are both limited and immature, and therefore unable to answer the question.

The “jury is still out” regarding the role of TEVAR for chronic aortic dissection extending distal to the celiac axis. We share Dr Roselli’s² opinion that TEVAR should be viewed as a complementary strategy, rather than a replacement for open surgery. TEVAR for chronic dissection is in its infancy, and outcomes will only improve as experience accumulates and “dissection-specific” devices become available. However, we believe that age, comorbidities, and anatomy will continue to serve as the primary determinants of whether patients receive open or endovascular

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therapy for the treatment of complicated chronic distal aortic dissection.

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