Covering all the bases

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The traditional treatment of uncomplicated Stanford type B aortic dissections (TBADs) has been optimal medical management, including anti-impulse therapy. Late complications from medical management alone include aneurysmal dilatation with or without rupture and late death. Late deaths in patients with TBADs treated medically who die suddenly support the concern about rupture. Successful remodeling and obliteration of the false lumen are key to inducing healing and realignment of the aortic wall layers, stabilizing the previously dissected aorta and decreasing the risk of rupture.

The introduction of thoracic aortic stent grafts allowed successful thoracic endovascular aortic repair with improved results relative to open repair, and the natural evolution was to adapt this technology to thoracic aortic dissections. There are now data to support coverage of the proximal primary intimal tear of TBADs with stent grafts in addition to optimal medical management. The INSTEAD-XL (Investigation of Stent Grafts in Aortic Dissection) and ADSORB (Acute Dissection: Stent Graft or Best Medical Therapy) trials have shown improved outcomes for patients treated early with stent grafts to allow for aortic remodeling and avoiding late complications. Both trials included patients initially treated with medical management who later required stent grafts for late complications.

Despite early coverage of the dissection entry point, there are TBADs that fail to remodel because of continued flow in the false lumen, placing patients at risk for late aneurysmal dilatation. In this issue of the Journal, Liu and colleagues provide us with further contributions to our understanding of aortic enlargement despite early treatment of TBADs with stent grafts. Their study supports the idea that the number of false-lumen branches is a predictor of aortic enlargement along the stent graft. Similar to the type II endoleak in endovascular aneurysm repair, these branches allow continuous flow in the false lumen, making it more difficult for the false lumen to thrombose.

There are, however, several issues to consider. There can be multiple entry and reentry points in the dissection flap. The study of Liu and colleagues does not clearly specify the number of stents used and the length of the aorta covered. If there is concern that the increased number of branches impedes false-lumen thrombosis, longer lengths of the descending thoracic aorta could be covered, but at the expense of potentially increasing the risk of spinal cord ischemia. Their group A patients (those with ≥8 thoracic false-lumen branches) also had a statistically greater number of patent abdominal false-lumen branches, which have previously been shown to be a negative prognostic factor because of subsequent persistence of flow in the false lumen. Thus it is unclear whether it is the thoracic, abdominal, or a combination of both types of patent false-lumen branches that are critical in maintaining patency of the false lumen.

Identification of the false-lumen branches may be difficult. Dynamic magnetic resonance imaging allows better visualization of branches and provides for accurate measurement of aortic diameter than does the static image from computed tomography. This may allow more optimal selection of the stent size and length, leading to better apposition of the stent to the aortic wall at the proximal entry point.

Finally, Liu and colleagues treated a mixture of acute and chronic TBADs in patients with either complicated or uncomplicated dissections, in essence 4 subgroups. Although their results do support the utility of thoracic false-lumen branches as a prognostic indicator, their sample size is too small to suggest that is there a subgroup of
patients who might not benefit from thoracic endovascular aortic repair. In their own experience, they aggressively use thoracic endovascular aortic repair even for uncomplicated TBADs. Perhaps their own data may provide a moment of pause before empirically treating all TBADs the same.

As the literature begins to support the coverage of entry point in aortic dissections, we are starting to see a paradigm shift in the treatment of uncomplicated TBAD from optimal medical management to treatment with the addition of stent grafting. The goal is to allow aortic remodeling with the thrombosis of the false lumen. Factors that may contribute to persistent flow in the false lumen include increased number of false-lumen branches, late timing of the repair after the dissection flap has remodeled, and inappropriate sizing of the stent graft measured from static computed tomographic imaging rather than dynamic magnetic resonance angiography. More work is needed, but this is an exciting time as we determine the optimal treatment guidelines for uncomplicated TBAD.

References