

of TEVAR in patients with uATBAD, endovascular therapy should become the primary therapy for all patients with uATBAD with favorable anatomy who are not at the extreme ends of the age spectrum. It is time to move forward; this is not a complicated decision.

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Commentary: The 2-step strategy

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According to current guidelines, complicated type B aortic dissection (TBAD) requires endovascular therapy.¹ Nonetheless, the treatment of uncomplicated TBAD (uTBAD) remains controversial.¹ In this setting, thoracic endovascular aortic repair (TEVAR) has gained consensus over time; however, its recommendation remains in class IIa due to the lack of clinical trials.¹ Spinelli and colleagues² add evidence on the safety, efficacy, and usefulness of TEVAR in uTBAD. Analyzing Gore's Global Registry for Endovascular Aortic Treatment Registry, the authors report comparable outcomes between patients with complicated TBAD and uTBAD in terms of mortality, aortic complications, and reinterventions. The rationale of applying TEVAR in the treatment of TBAD is to seal the intimal tear, allowing the true lumen expansion and reducing the tension of the false lumen (FL) to promote its thrombosis. Moreover, endovascular aortic repair should reduce the risk of aortic remodeling and aortic-related complications (eg, aneurysm



Thoracic endovascular aortic repair.

CENTRAL MESSAGE

Stratification of uncomplicated type B aortic dissection and correct timing for TEVAR improves survival and prevents aortic-related adverse events.

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formation, rupture, and organ ischemia).³ Nevertheless, this procedure may be complicated by retrograde aortic dissection and the rate of reinterventions for endoleaks is around 24%.⁴ The treatment of uTBAD with TEVAR should be carefully evaluated according to patient-specific presentation, weighting benefits and risks of the procedure. Reviewing preoperative computed tomographic images of uTBAD patients, Sailer and colleagues⁵ identified 5 risk factors: connective disorders, circumferential extension of FL, the maximal aortic diameter, FL outflow, and intercostal branches involved. Based on these characteristics, they were able to stratify patients in low, intermediate, or high risk for adverse events. Moreover, the concavity of the distal arch and entry tears >10 mm were associated

with poor prognosis at either presentation or during hospitalization.^{6,7} Finally, Sato and colleagues⁸ reported that the shape of the true lumen was a predictor for aortic growth. Using these tools, a tailored approach can be hypothesized. In the study presented by Spinelli and colleagues,² it would have been of interest to stratify the risk of aortic adverse events of uTBAD patients receiving TEVAR. It is well known that optimal anti-impulse therapy (OMT) is effective in controlling symptoms and is associated with an intrahospital survival of 90%.³ However, survival decreases to 64% and 29% in intermediate and high-risk patients, respectively.⁹

We recommend early TEVAR treatment in high-risk patients, whereas low-risk patients may follow a delayed 2-step strategy consisting of OMT first and TEVAR second.¹⁰ OMT induces partial FL thrombosis, reduces aortic wall tension, and allows tissues thickening as result of inflammation. All of these factors are less likely to have aortic procedure-related complications.^{11,12} However, chronicization reduces the tissue elasticity, lowering the possibility of effectively reducing the FL.^{11,12} In light of these considerations, stratification and timing are essential to maximize TEVAR benefit and reduce postoperative complications. Despite this, the Interventional Stent Treatment Acute Dissection trial and Acute Dissection: Stent Graft or Best Medical Therapy trial failed to show lower mortality with TEVAR over OMT, and TEVAR was associated with higher rate of FL thrombosis and reduced aortic remodeling at follow-up.^{13,14} Because a number of patients with uTBAD will develop postdissection aneurysms requiring surgery, we can conclude that OMT alone remains a suboptimal treatment and elective TEVAR may play an important role in preventing aortic wall remodeling.

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