The impact of thoracic endovascular aortic repair on long-term survival in type B aortic dissection: response to editorial

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We appreciate the interest and comments of Karkos *et al.* in reviewing our paper entitled: "*The Impact of Thoracic Endovascular Aortic Repair on Long-Term Survival in Type B Aortic Dissection*" recently published in the *Annals of Thoracic Surgery*.

Thoracic endovascular aortic repair (TEVAR) has revolutionized the treatment paradigm for patients presenting with acute type B aortic dissections (TBADs) and is now well-established as the strategy of choice in the management of complicated TBADs. Endovascular therapy covers the proximal entry tear and remodels the dissected aorta by expanding the true lumen and shrinking or obliterating the false lumen. The management of uncomplicated TBADs—traditionally treated with optimal medical therapy (OMT)—is less clear. However, as prior reports and our current analysis have demonstrated (1-5), almost half of these patients fail OMT over time and undergo open surgery or TEVAR in the chronic phase. Long-term outcomes of OMT are similarly poor with 58.9% overall survival and 30.9% intervention-free survival at 10 years (5).

Our results support INSTEAD trial data demonstrating improved mid-term outcomes with TEVAR compared to OMT (6). Earlier intervention is potentially advantageous as the acute dissection flap is more pliable, providing the best chance of complete remodeling. Given these findings, there has been increased interest in expanding the role of TEVAR to the treatment of uncomplicated TBADs. However, TEVAR is not a risk-free procedure and carries peri-procedural risks of retrograde type A dissection, stroke, spinal cord ischemia, and acute kidney injury (7). Therefore, it should only be utilized in those patients who will ultimately fail OMT in the chronic phase.

As our reviewers astutely point out, the issue remains in characterizing the high-risk features that predict failure of medical management and identifying which patients would benefit from early TEVAR. There remains an overall lack of understanding and a great deal of controversy regarding the pathophysiology of TBAD and aneurysmal degeneration, and this is an active area of ongoing investigation at our institution. Specifically, we are assessing the high risk radiographic predictors that promote failure of medical management in the chronic phase of uncomplicated TBAD including the role of aortic size, false lumen status, and entry tear site and size. Additionally, understanding the tissue-level biomolecular and biomechanical factors that facilitate favorable aortic remodeling in the acute phase of TBAD is a focus of future efforts. The ability to select high-risk patients and target therapy and timing of intervention would optimize current therapy as well as dramatically impact the long-term survival data we have presented.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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