Have High-Risk Criteria Influenced the Management of Type B Aortic Dissection?

Applying the new framework for patients with uncomplicated TBAD and high-risk features to improve decision-making and ongoing research efforts.

By Robert A. Larson, MD

There are few areas in vascular surgery that remain as contentious as the management of acute type B aortic dissection (TBAD). It has been almost 20 years since the first reported endovascular repair for TBAD, yet questions related to which patients benefit from surgical intervention and the timing of intervention remain without definitive answers. Although it is now clear that patients presenting with complications, such as rupture and malperfusion syndromes, do better with surgical treatment with thoracic endovascular aortic repair (TEVAR) when feasible, patients who initially present as "uncomplicated" have had a much more varied course. Investigators over the past 2 decades have shown that TBAD is a dynamic disease process with myriad factors associated with worse outcomes, and disease severity does not fit into a simple dichotomy of "complicated" and "uncomplicated."

DECISION-MAKING FOR TBAD CONTINUES TO EVOLVE

Acute TBAD is recognized as a heterogeneous disease. It has been shown that the majority of uncomplicated TBAD (uTBAD) patients, up to 60% in the International Registry of Aortic Dissection (IRAD),[1] treated with optimal medical therapy (OMT) develop aneurysmal degeneration. Is this really an uncomplicated process, at least in the long run? Short- and medium-term benefits of TEVAR have been shown in the INSTEAD and ADSORB randomized clinical trials, which demonstrated improved aortic remodeling and false lumen thrombosis during postoperative follow-up. Several recent retrospective studies have shown that uTBAD patients can undergo TEVAR with a low rate of early complications and favorable midterm outcomes.[2] Nevertheless, the general consensus and current guidelines still recommend OMT for uTBAD.

With increased understanding of the natural history of the disease and improved endovascular technology, there has been an increase in the use of TEVAR since the late 1990s, most notably in complicated cases. Trimarchi et al demonstrated in the IRAD registry that the use of TEVAR increased from 19.1% of cases from 1996 to 2007 to 37.2% from 2013 to 2022.[3] This was associated with an in-hospital mortality decrease from 10.7% to 6.1%. In the earliest group, 46% of patients presented as a complicated TBAD, compared to 30% in the latest group. Although the utility of TEVAR in TBAD is now well-established, the choice for each individual patient remains less clear.

IDENTIFYING HIGH-RISK uTBAD

Can a high-risk subgroup of uTBAD patients be identified? From the early days of surgical intervention for

TBAD, there has been a search for markers to identify patients at high risk for future complications. Several physiologic and radiographic findings at the time of presentation have been linked to worse outcomes; among these are aortic diameter > 40 mm, entry tear on the inner curve of the aorta, primary entry tear > 10 mm in length, false lumen diameter > 22 mm, Marui fusiform index > 0.64, and partial false lumen thrombosis. The recognition of these high-risk factors led to proposals for a more nuanced clinical stratification in TBAD.[4-6]

In an effort to standardize the reporting of TBAD cases and provide a framework that supports clinical research, the Society for Vascular Surgery (SVS) and Society of Thoracic Surgeons (STS) developed a consensus statement outlining the definitions to be used when reporting aortic dissections. In addition to a new system for documenting the type and extent of dissections, there are also new categories regarding the chronologic stage and acuity of the patient.[7] The historical concepts of uncomplicated and complicated dissections are largely unchanged, but there is now a new category of TBAD: *TBAD with high-risk features* (HRF). These patients present without acute complications but have anatomic or physiologic features that increase the risk of developing complications. The risk factors are those that have long been shown to portend worse outcomes, as noted previously. In addition to the anatomic factors that are found on imaging on presentation (eg, CTA), there are the physiologic factors of refractory pain and refractory hypertension despite OMT. Refractory is defined as lasting > 12 hours. Also, any readmission within 30 days for a dissection-related diagnosis is considered an HRF for documentation purposes.

MOVING FORWARD USING THE HRF CRITERIA

Part of the difficulty in reaching a consensus on how best to treat uTBAD and TBAD with HRF patients is that most of the relevant clinical research has come from registries, retrospective reviews, and single-center experiences. The heterogeneity of the definitions and associated data have limited the ability to compare or combine study outcomes. The new reporting standards set forth a common framework that will make interpreting the outcomes of future research more effective.

Studies using the new reporting standards are starting to appear, and there are useful insights to be gleaned from them. Herajärvi et al retrospectively reviewed 162 uTBAD patients at their institution, 114 of whom presented with the HRFs of refractory hypertension, aortic diameter > 40 mm, lesser curve entry tear, false lumen > 22 mm, and imaging-diagnosed malperfusion.[8] All patients received OMT, and 13 of the TBAD patients with HRF underwent TEVAR at a mean of 8.5 days, primarily for aneurysmal degeneration. At a mean follow-up of 5.1 years, aneurysmal degeneration occurred in 8.3% of the uTBAD patients and 27% of the HRF TBAD group. Within 6 months of presentation, about 20% of TBAD patients with HRF required an aortic intervention, whereas none of the uTBAD patients required an aortic intervention. At 10 years, the overall survival in the uTBAD group was 71% compared to 60% in the HRF TBAD group. Not all of the SVS/STS HRF could be identified retrospectively, but the HRF TBAD group clearly fared worse over time.

In another study, Potter et al looked at uTBAD patients who presented with the HRF of rapid growth/aortic diameter > 40 mm, refractory pain, and refractory hypertension using the Vascular Quality Initiative (VQI) TEVAR and Complex EVAR registry data set.[9] Among the 811 patients identified, they found no difference in in-hospital, 30-day, or 1-year mortality based on the number of HRF at presentation. Patients undergoing early TEVAR (0-2 days) had a significantly higher risk of stroke (11%) compared to those treated from 3 to 6 days (4.1%), 7 to 14 days (4.1%), and 15 to 90 days (2.7%). A multivariable analysis found that TEVAR during the subacute phase (15-90 days) was associated with a 62% reduction in the odds of in-hospital and 30-day mortality. Unfortunately, the VQI does not yet fully implement the recording of all the new HRF, and so some high-risk patients were likely not identified. This and several previous studies have shown that the timing of TEVAR in TBAD is another critical decision affecting short-term outcomes. As with HRF, there has been heterogeneity in how the time to surgery data have been reported. To address this, the new SVS/STS guideline divides the chronicity of TBAD into four categories: hyperacute (< 24 hours), acute (1-14 days), subacute (15-90 days), and chronic (> 90 days).

CONCLUSION

How has the recognition of high-risk criteria in TBAD changed management of HRF patients? There has been a slow but steady trend toward being more open to TEVAR in this subgroup in the literature, especially over the past decade. It is unlikely that the new reporting standard and classification system will change the way TBAD patients are treated. However, the new framework will help improve our understanding of this complicated disease process through ongoing research.

(Continued on page 69)

(Continued from page 60)

At this point, there appears to be ample evidence to suggest that TBAD patients who present with HRF may benefit from a TEVAR to reduce the risk of late complications. The timing of intervention appears to favor the subacute phase.[10] Given the lack of high-quality data to support intervention, every case must be evaluated individually, and the patient's comorbidities and other risk factors must be considered. For the foreseeable future, there will be no cookbook algorithm for the treatment of TBAD patients with HRF, and solid clinical judgment will continue to guide therapy. For each case, one must ask if the benefits of TEVAR (improved remodeling, reduced dissection progression, reduced aorta-related mortality) outweigh the risks (retrograde type A dissection, stroke, spinal cord ischemia, access complications, cost). It is obvious that high-quality level 1 data from randomized clinical trials are needed to move us forward, and the new reporting standards are well positioned to support these efforts. ■

1. Jonker FH, Trimarchi S, Rampoldi V, et al. Aortic expansion after acute type B aortic dissection. Ann Thorac Surg. 2012;94:1223-1229. doi: 10.1016/j.athoracsur.2012.05.040
2. Lou X, Chen EP, Duwayri YM, et al. Early results of thoracic endovascular aortic repair for the management of acute uncomplicated type B aortic dissection. Semin Thorac Cardiovasc Surg. 2023;35:289-297. doi: 10.1053/j.semtcvs.2021.12.014
3. Trimarchi S, Gleason TG, Brinster DR, et al. Trends in management and outcomes of type B aortic dissection: a report from the International Registry of Aortic Dissection. Eur J Vasc Endovasc Surg. Published online May 16, 2023. doi: 10.1016/j.ejvs.2023.05.015
4. Dake MD, Thompson M, van Sambeek M, et al. DISSECT: a new mnemonic-based approach to the categorization of aortic dissection. Eur J Vasc Endovasc Surg. 2013;46:175-190. doi: 10.1016/j.ejvs.2013.04.029
5. Augoustides JG, Szeto WY, Woo EY, et al. The complications of uncomplicated acute type-B dissection: the introduction of the Penn classification. J Cardiothorac Vasc Anesth. 2012;26:1139-1144. doi: 10.1053/j.jvca.2012.06.024
6. Chen S, Larion S, Ahanchi SS, et al. A novel anatomic severity grading score for acute type B aortic dissections and correlation to aortic reinterventions after thoracic endovascular aortic repair. J Cardiothorac Surg. 2017;12:39. doi: 10.1186/s13019-017-0590-8
7. Lombardi JV, Hughes GC, Appoo JJ, et al. Society for Vascular Surgery (SVS) and Society of Thoracic Surgeons (STS) reporting standards for type B aortic dissections. J Vasc Surg. 2020;71:723-747. doi: 10.1016/j.jvs.2019.11.013
8. Herajärvi J, Mustonen C, Kesavuori R, et al. Impact of high-risk features on outcome of acute type B aortic dissection. JTCVS Open 2023;13:20-31. doi: 10.1016/j.xjon.2023.01.005
9. Potter HA, Ding L, Han SM, et al. Impact of high-risk features and timing of repair for acute type B aortic dissections. J Vasc Surg. 2022;76:364-371 e3. doi: 10.1016/j.jvs.2022.03.030
10. Zhao W, Yang Y, Wu Z, et al. Endovascular repair of acute vs. subacute uncomplicated type B aortic dissection: a systematic review and meta-analysis. Front Cardiovasc Med. 2023;10:1189750. doi: 10.3389/fcvm.2023.1189750

Robert A. Larson, MD
Associate Professor of Surgery
Associate Chair for Quality and Patient Safety
VCU Health
Richmond, Virginia
robert.larson@vcuhealth.org
Disclosures: None.