Treatment of Uncomplicated Type B Aortic Dissection
The Devil Is in the Details...Or Is It?
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In a retrospective cohort study of 7105 patients with acute uncomplicated type B aortic dissection (uTBAD) occurring between 2011 and 2018, Weissler et al used inpatient claims data from the US Centers for Medicare & Medicaid Services (CMS) database to examine whether there was any benefit of thoracic endovascular aortic repair (TEVAR) in uTBAD. The benefit of TEVAR in uTBAD would be to achieve positive aortic remodeling, whereby the true lumen expands, the false lumen regresses (mostly or completely), and aneurysmal degeneration is minimized, thereby decreasing the risk of aortic-related morbidity and mortality. However, TEVAR is not without its risks, including stroke, paralysis, vascular access-related complication, retrograde aortic dissection, and stent-induced new entry (SINE) that may lead to malperfusion acutely or aneurysmal degeneration in the short term and mid term.

The study found no association of TEVAR in uTBAD with improved survival or decreased aortic interventions or aorta-related or cardiovascular hospitalizations in a propensity-weighted analysis with a median follow-up of 2.55 years. Several other important findings included higher 5-year mortality (45%) compared with other studies and the fact that only 16% of patients who were initially medically managed required later aortic intervention. Incidence of stroke was higher after initial TEVAR compared with medically treated patients. Paralysis from TEVAR was rare.

The study is well performed and includes important covariates studied in a multivariable logistic regression model. Another strength is its large cohort and 1-year, 2-year, and 5-year follow-up data, which contrasts with 2 small randomized clinical trials with only 140 and 61 patients in the INSTEAD trial and ADSORB trial, respectively, as well as other reports from single centers or registries.

However, is the devil in the details? First, the study defined intervention as occurring within 30 days of hospitalization for acute uTBAD. The more recently accepted definition for phases of acute aortic dissection has changed such that the acute period encompasses 14 days, a subacute phase encompasses 15 to 90 days, and the chronic period encompasses greater than 90 days, adopted because of evidence demonstrating differing morbidity and mortality to TEVAR depending on timing of intervention. That means this study included TEVAR for both acute and subacute uTBAD and did not include TEVAR for subacute TBAD performed between 31 and 90 days, which may partially explain why the incidence of initial intervention was only 16% (eg, missed interventions after a follow-up computed tomography scan and clinic visit beyond the 30-day mark but still performed within the subacute period).

Second, while the authors were able to tease out uTBAD using International Classification of Diseases, Ninth Revision (ICD-9) and ICD-10 codes, the CMS database lacks granularity regarding anatomic features of the dissection. For example, which patients underwent TEVAR for uTBAD with high-risk features, such as refractory hypertension, aortic diameter greater than 40 mm, false lumen diameter greater than 22 mm, radiographic-only malperfusion, or entry tear on the lesser curve? Currently, the Society of Thoracic Surgeons/American Association for Thoracic Surgery clinical practice guidelines on the management of TBAD give TEVAR for uTBAD with high-risk features a Class of Recommendation IIb, Level of Evidence B-NR. This recommendation is based on single-center, clinical trial, and registry data reporting high incidence of aneurysmal degeneration (more than 70%) and mortality (25% to 30% at 3 to 5 years). However, if the study by Weissler et al could not differentiate between the presence or absence of high-risk features, the findings might have favored TEVAR in the overall cohort (from bias because of benefit in uTBAD with high-risk features), which was not the case.

The CMS database also does not contain details of the endovascular intervention. Was the proximal extent of the TEVAR in zone 2 or zone 3 (ie, what was the quality of the aorta at the proximal landing zone and how were the left subclavian and left vertebral arteries managed if the proximal landing zone was in zone 2, which might have affected incidence of stroke)? Was the initial extent of coverage short (15 to 20 cm) just to treat the primary entry tear, with the plan to extend to the celiac later if the distal descending aorta grew—or was the entire descending thoracic aorta treated? Was there adjunctive treatment of the distal landing zone to promote false lumen thrombosis and positive aortic remodeling—or was there persistent retrograde false lumen flow? And which patients developed SINE that led to future interventions?

We still do not understand how aortic properties at the proximal landing zone, the dissection septum, and the disrupted mediaval layer influence the optimal timing of TEVAR in acute TBAD. If there is hematoma (whether intramural, periadventitial, or periaortic) near the subclavian artery or more proximally, which is not uncommon in acute TBAD, perhaps waiting until the hematoma resolves would decrease risk of intimal injury from deploying an oversized device exerting constant radial force on the tissue. For DeBakey type IIIb TBAD, the distal extent of the stent graft will land in the true lumen and abut the fragile dissection septum for a portion of the device circumference. The septum can remain mobile for some time but undergoes changes that lead to thickening and immobility (hence, decreased success with TEVAR in chronic TBAD). When is the appropriate time to allow for expansion of the mobile septum but avoid SINE? The angle of the aortic arch, the tortuosity of the aorta, the maximum diameter of the most an-
eurysmal segment, location and size of re-entry tears or fenestrations, and branch vessel supply of the false lumen all render TEVAR for TBAD more complicated than for degenerative aneurysm disease. Lastly, how do the lamellar units of the disrupted medial layer heal after TEVAR, or do they ever? Unfortunately, the opportunity to obtain aortic tissue for histologic study of the false lumen from a specimen obtained at the time of TEVAR explant is infrequent. There is more we need to understand to optimize this endovascular treatment that remains much less invasive than open descending thoracic aortic replacement.

Interestingly, Weissler et al report geographic variation in TEVAR for uTBAD (most frequent in the South, lowest in the Northeast) and a significant association between higher center TEVAR volume and higher rates of performing initial TEVAR. For acute type A aortic dissection, literature over the past decade has suggested improved outcomes when repair is performed at high-volume centers by experienced surgeons, as detailed in the American Association for Thoracic Surgery expert consensus document for the management of acute type A dissection. Perhaps this will also be demonstrated with TEVAR for TBAD, especially for high-risk patients.

Maybe the details matter, maybe they do not. Currently, the lack of high-quality data from large randomized clinical trials limits the ability to make strong recommendations for optimal timing for TEVAR or routine TEVAR in uTBAD, with or without high-risk features, to prevent long-term aortic-related complications. The study by Weissler et al provides support that a continued measured approach to TEVAR in uTBAD is warranted until more compelling evidence of benefit is available. With the use of TEVAR in complicated TBAD well-established and more clinical equipoise among centers and physicians in providing both optimal medical management and endovascular intervention for TBAD, it is time for a large prospective randomized clinical trial to settle this decade-old debate the right way.

ARTICLE INFORMATION

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REFERENCES


