Where Do We Stand on Truly Uncomplicated TBAD? It's Complicated

The management of uTBAD remains controversial—mostly due to difficulty identifying patients with truly uTBAD.

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Acute aortic dissection remains one of the most complex and life-threatening aortic disorders, with type B aortic dissection (TBAD) accounting for approximately one-third of dissections. Historically, the management and prognosis of TBAD patients depended on complications at the time of initial diagnosis. Based on the most recent 2022 American College of Cardiology (ACC)/American Heart Association (AHA) guideline for the diagnosis and management of aortic disease, TBAD is defined as complicated TBAD (cTBAD) if rupture or malperfusion exists and if there is an extension of dissection; progressive enlargement of the true lumen, false lumen (FL), or both in the acute phase; intractable pain; and uncontrolled hypertension (HTN).[1] The guidelines include a recommendation for intervention in addition to optimal medical treatment (OMT). If the previously mentioned complications do not exist, dissection is defined as uncomplicated TBAD (uTBAD), and OMT is recommended as initial management for reducing heart rate and blood pressure to decrease aortic wall stress.[1]

Treatment of cTBAD has evolved over the past 2 decades, as thoracic endovascular aortic repair (TEVAR) has become the standard of care due to improved outcomes compared to open surgical repair.[2-5] However, the management of uTBAD remains controversial, mostly due to the difficulty of identifying patients with truly uTBAD.

NATURAL HISTORY OF uTBAD

The number of patients presenting with uTBAD varies from 60% to 75% of all patients with acute TBAD.[6-8] Most patients with uTBAD are treated medically with blood pressure and anti-impulse control, with 3% to 10% early mortality. Nearly 40% of uTBAD patients will require a late intervention, mostly due to aneurysmal degeneration, with long-term survival of 50% to 60%.[7,9-15] With this natural history and the significant improvement of outcomes of patients with cTBAD treated with TEVAR, there has become an increased interest in TEVAR for uTBAD.[3,5,16]

ROLE OF TEVAR IN uTBAD

The INSTEAD trial randomized 140 patients with subacute uTBAD into cohorts of elective TEVAR with OMT to OMT alone and found no significant difference in mortality at 2 years between the groups; however, the TEVAR group was associated with aortic remodeling and FL thrombosis.[16] When they extended the follow-up to 5 years in the INSTEAD XL trial, they found significant improvement in aortic-related mortality and improved aortic remodeling in the TEVAR plus OMT group compared to OMT alone.[17]

The ADSORB trial compared TEVAR plus OMT to OMT in uTBAD.[18] It showed no difference in early mortality between the groups, with one death in the TEVAR

plus OMT group at 1-year follow-up. ADSORB also demonstrated significantly improved aortic remodeling, with FL thrombosis and FL diameter decrease in the TEVAR plus OMT group compared to OMT alone.[18] One major criticism of those studies is the lack of data on high-risk features.

"HIGH-RISK" FEATURES OF uTBAD

In recent years, many have tried to identify features of uTBAD that would predict the development of late complications. Despite the inconsistency and difficulty in reproducibility of those results, the 2022 ACC/AHA guideline gave a weak recommendation that TEVAR may be considered if any of the following high-risk factors are present: maximal aortic diameter > 40 mm; FL diameter > 20 to 22 mm; entry tear > 10 mm; entry tear on the lesser curvature; increase in total aortic diameter of > 5 mm between serial imaging studies; bloody pleural effusion; imaging-only evidence of malperfusion; refractory HTN despite more than three different classes of antihypertensive medications at maximal recommended or tolerated doses; and refractory pain persisting > 12 hours despite maximal recommended or tolerated doses and need for readmission.[1]

Does this mean that the truly uTBAD are those that lack any of the above high-risk factors? Can we make this decision based on moderate-quality data? Can we determine organ malperfusion based solely on static cross-sectional imaging? What would happen if we started treating all those patients with TEVAR? Will we prevent future aortic degeneration?

Famularo et al performed a systematic review assessing medium- and long-term outcomes after TEVAR for the treatment of TBAD.[19] They showed that patients after TEVAR still had a significant risk for developing aneurysmal degeneration in the thoracic aorta, which was similar to the risk in patients treated with OMT. In addition, the risk for aneurysmal degeneration in the abdominal aorta was shown to be higher than in the thoracic aorta.[19] All this shows that we are still far from fully understanding this complex disease.

RISK PREDICTORS FOR DELAYED COMPLICATIONS IN PATIENTS WITH uTBAD: WHAT ARE WE MISSING?

Currently, the recommended initial imaging is CT, with MRI and echocardiography as alternatives.[1] Our protocol must evolve to include more technologies that can examine flow dynamics, such as dynamic CT, computational fluid dynamics, and four-dimensional flow MRI. An increasing number of publications correlate different flow patterns in the FL to different risk levels for delayed complications.[20-24] Including dynamic imaging studies in evaluating uTBAD will help overcome the complexity and heterogeneity of the disease. In addition, incorporating those technologies during follow-up with all aortic dissection patients will allow us to understand the changes in the flow and pressure in the FL and true lumen over time as the acute dissection becomes chronic, and even more after an intervention and placement of a stent graft or performing graft replacement of the aorta.

The World Health Organization describes social determinants of health (SDOH) as the conditions in which people are born, grow, work, live, and age, as well as the wider set of forces and systems shaping the conditions of daily life. These nonclinical and nonbiologic factors, including HTN control, can profoundly impact health outcomes.

Factors associated with uncontrolled HTN include medication costs, health care access, medication complexity, patient beliefs and perceptions, educational achievement, socioeconomic status, depression and demoralization, perceived racism and discrimination, social networks and support, physician prescribing practices, and neighborhood segregation.[25-32] HTN is the major clinical risk factor for dissection and death due to aortic dissection. In fact, up to 10% of patients with hypertensive emergencies develop aortic dissection—and > 70% of patients admitted to the hospital due to aortic dissection have HTN.[33] Studies have demonstrated the importance of controlling HTN and heart rate in preventing delayed aortic complications and improving outcomes in patients with aortic dissection.[34-37] Yet, none of those SDOH factors are considered in most studies investigating the long-term outcomes of uTBAD.

CONCLUSION

Where do we stand on truly uTBAD? Unfortunately, we are back to square one. We still don't know who are the truly uTBAD patients. We must be able to define our patients into acute cTBAD, delayed cTBAD, and truly uTBAD. Our task should be to focus on identifying the patients who will develop delayed cTBAD, which requires a large, randomized trial comparing TEVAR plus OMT to OMT alone. To do this, we must first reproduce and validate some of the high-risk features that are currently suggested as indications for intervention in uTBAD. More importantly, we must rethink the data that we collect. This means that, in addition to the usual clinical, demographic, and radiologic data, we need to add new imaging technologies that can provide information about flow dynamics, dynamic behavior of the dissection flap, and wall stress measurements.

We must then incorporate SDOH data and try to understand how health care insecurities influence the outcomes of patients with uTBAD and ensure that our patients are compliant with OMT and document the effectiveness of that management as well as identify the barriers to compliance if they exist.

If we can do these things, our future predictive risk models for identifying delayed cTBAD will look different. Future interventions might require more than surgical interventions—they could require building a safety net for our patients to ensure they have access to long-term follow-up, adequate blood pressure control, and chronic aortic dissection treatment. Only then will we know where we stand with truly uTBAD and, more importantly, if they can still be defined that way. ■

1. Isselbacher EM, Preventza O, Black JH 3rd, et al. 2022 ACC/AHA guideline for the diagnosis and management of aortic disease: a report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines. Circulation. 2022;146:e334-e482. doi: 10.1161/CIR.0000000000001106
2. Dake MD, Kato N, Mitchell RS, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. N Engl J Med. 1999;340:1546-1552. doi: 10.1056/NEJM199905203402004
3. Fattori R, Tsai TT, Myrmel T, et al. Complicated acute type B dissection: is surgery still the best option? A report from the International Registry of Acute Aortic Dissection. JACC Cardiovasc Interv. 2008;1:395-402. doi: 10.1016/j.jcin.2008.04.009
4. Nienaber CA, Kische S, Ince H, et al. Thoracic endovascular aneurysm repair for complicated type B aortic dissection. J Vasc Surg. 2011;54:1529-1533. doi: 10.1016/j.jvs.2011.06.099
5. Hanna JM, Andersen ND, Ganapathi AM, et al. Five-year results for endovascular repair of acute complicated type B aortic dissection. J Vasc Surg. 2014;59:96-106. doi: 10.1016/j.jvs.2013.07.001
6. Cooper M, Hickes C, Ratchford EV, et al. Diagnosis and treatment of uncomplicated type B aortic dissection. Vasc Med. 2016;21:547-552. doi: 10.1177/1358863X16643601
7. Afifi RO, Sandhu HK, Leake SS, et al. Outcomes of patients with acute type B (DeBakey III) aortic dissection a 13-year, single-center experience. Circulation. 2015;132:748-754. doi: 10.1161/CIRCULATIONAHA.115.015302
8. 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
9. Schwartz S, Durham C, Clouse WD, et al. Predictors of late aortic intervention in patients with medically treated type B aortic dissection. J Vasc Surg. 2018;67:78-84. doi: 10.1016/j.jvs.2017.05.128
10. DeBakey ME, McCollum CH, Crawford ES, et al. Dissection and dissecting aneurysms of the aorta: twenty-year follow-up of five hundred twenty-seven patients treated surgically. Surgery. 1982;92:1118-34.
11. Elefteriades JA, Hartleroad J, Gusberg RJ, et al. Long-term experience with descending aortic dissection: the complication-specific approach. Ann Thorac Surg. 1992;53:11-21. doi: 10.1016/0003-4975(92)90752-p
12. Marui A, Mochizuki T, Mitsui N, et al. Toward the best treatment for uncomplicated patients with type B acute aortic dissection: a consideration for sound surgical indication. Circulation. 1999;100(19 Suppl):II275-80. doi: 10.1161/01.cir.100.suppl_2.ii-275
13. Juvonen T, Ergin MA, Galla JD, et al. Risk factors for rupture of chronic type B dissections. J Thorac Cardiovasc Surg. 1999;117:776-786. doi: 10.1016/S0022-5223(99)70299-0
14. Durham CA, Cambria RP, Wang LJ, et al. The natural history of medically managed acute type B aortic dissection. J Vasc Surg. 2015;61:1192-1198. doi: 10.1016/j.jvs.2014.12.038
15. Tsai TT, Fattori R, Trimarchi S, et al. Long-term survival in patients presenting with type B acute aortic dissection: insights from the International Registry of Acute Aortic Dissection. Circulation. 2006;114:2226-2231. doi: 10.1161/CIRCULATIONAHA.106.622340
16. Nienaber CA, Rousseau H, Eggebrecht H, et al. Randomized comparison of strategies for type B aortic dissection: the INvestigation of STEnt Grafts in Aortic Dissection (INSTEAD) trial. Circulation. 2009;120:2519-2528. doi: 10.1161/CIRCULATIONAHA.109.886408
17. Nienaber CA, Kische S, Rousseau H, et al. Endovascular repair of type B aortic dissection: long-term results of the randomized investigation of stent grafts in aortic dissection trial. Circ Cardiovasc Interv. 2013;6:407-416. doi: 10.1161/CIRCINTERVENTIONS.113.000463
18. Brunkwall J, Lammer J, Verhoeven, Taylor P. ADSORB: a study on the efficacy of endovascular grafting in uncomplicated acute dissection of the descending aorta. Eur J Vasc Endovasc Surg. 2012;44:31-36. doi: 10.1016/j.ejvs.2012.03.023
19. Famularo M, Meyermann K, Lombardi JV. Aneurysmal degeneration of type B aortic dissections after thoracic endovascular aortic repair: a systematic review. J Vasc Surg. 2017;66:924-930. doi: 10.1016/j.jvs.2017.06.067
20. Evangelista A, Pineda V, Guala A, et al. False lumen flow assessment by magnetic resonance imaging and long-term outcomes in uncomplicated aortic dissection. J Am Coll Cardiol. 2022;79:2415-2427. doi: 10.1016/j.jacc.2022.04.017
21. Burris NS, Patel HJ, Hope MD. Retrograde flow in the false lumen: marker of a false lumen under stress? J Thorac Cardiovasc Surg. 2019;157:488-491. doi: 10.1016/j.jtcvs.2018.06.092
22. Dillon-Murphy D, Noorani A, Nordsletten D, Figueroa CA. Multi-modality image-based computational analysis of haemodynamics in aortic dissection. Biomech Model Mechanobiol. 2016;15:857-876. doi: 10.1007/s10237-015-0729-2
23. Rudenick PA, Segers P, Pineda V, et al. False lumen flow patterns and their relation with morphological and biomechanical characteristics of chronic aortic dissections. Computational model compared with magnetic resonance imaging measurements. PLoS One. 2017;12:e0170888. doi: 10.1371/journal.pone.0170888
24. Burris NS, Nordsletten DA, Sotelo JA, et al. False lumen ejection fraction predicts growth in type B aortic dissection: preliminary results. Eur J Cardiothorac Surg. 2020;57:896-903. doi: 10.1093/ejcts/ezz343
25. Schroeder K, Fahey T, Ebrahim S. Interventions for improving adherence to treatment in patients with high blood pressure in ambulatory settings. Cochrane Database Syst Rev. 2004;2004:CD004804. doi: 10.1002/14651858.CD004804
26. Briesacher BA, Gurwitz JH, Soumerai SB. Patients at-risk for cost-related medication nonadherence: a review of the literature. J Gen Intern Med. 2007;22:864-871. doi: 10.1007/s11606-007-0180-x
27. Cooper LA. A 41-year-old African American man with poorly controlled hypertension: review of patient and physician factors related to hypertension treatment adherence. JAMA. 2009;301:1260-1272. doi: 10.1001/jama.2009.358
28. Whelton PK, Einhorn PT, Muntner P, et al. Research needs to improve hypertension treatment and control in African Americans. Hypertension. 2016;68:1066-1072. doi: 10.1161/HYPERTENSIONAHA.116.07905
29. Boutin-Foster C, Offidani E, Kanna B, et al. Results from the trial using motivational interviewing, positive affect, and self-affirmation in African Americans with hypertension (TRIUMPH). Ethn Dis. 2016;26:51-60. doi: 10.18865/ed.26.1.51
30. Offidani E, Benasi G, Charlson ME, et al. Impact of depression and demoralization on blood pressure control in African Americans with hypertension: findings from the TRIUMPH Trial. J Racial Ethn Health Disparities. 2018;5:913-918. doi: 10.1007/s40615-017-0439-9
31. Ruppar TM, Dunbar-Jacob JM, Mehr DR, et al. Medication adherence interventions among hypertensive black adults: a systematic review and meta-analysis. J Hypertens. 2017;35:1145-1154. doi: 10.1097/HJH.0000000000001260
32. Johnson RL, Roter D, Powe NR, Cooper LA. Patient race/ethnicity and quality of patient-physician communication during medical visits. Am J Public Health. 2004;94:2084-2090. doi: 10.2105/ajph.94.12.2084
33. Papadopoulos DP, Sanidas EA, Viniou NA, et al. Cardiovascular hypertensive emergencies. Curr Hypertens Rep. 2015;17:5. doi: 10.1007/s11906-014-0515-z
34. 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
35. Suzuki T, Isselbacher EM, Nienaber CA, et al. Type-selective benefits of medications in treatment of acute aortic dissection (from the International Registry of Acute Aortic Dissection [IRAD]). Am J Cardiol. 2012;109:122-127. doi: 10.1016/j.amjcard.2011.08.012
36. Kumar KU, Zhao Q, Bai X, et al. Controlled heart rate and blood pressure reduce the life-threatening aortic events and increase survival in patients with type B aortic dissection: a single center experience. Int J Cardiol Heart Vasc. 2015;8:73-74. doi: 10.1016/j.ijcha.2015.05.008
37. Wang Z, Ge M, Chen T, et al. Impact of hypertension on short- and long-term survival of patients who underwent emergency surgery for type A acute aortic dissection. J Thorac Dis. 2020;12:6618-6628. doi: 10.21037/jtd-20-2336

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