Should Clinically Asymptomatic Patients With Acute Type B Dissections and Radiographic Malperfusion Be Treated?

An expert panel discusses when endovascular treatment is appropriate to help improve symptoms and survival.



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Approximately one-quarter of patients who present with acute type B aortic dissection (TBAD) go on to develop complications. The visceral and renal arteries are most commonly compromised when complica-

tions do occur. The time from development of an acute TBAD to malperfusion can be highly variable. With increasing use of high-quality, three-dimensional (3D) CT angiography, radiologic evidence of malperfusion can be more apparent. This does not always translate into clinical evidence of malperfusion. However, when radiologic evidence of malperfusion exists, this can be a preceding sign of complications. Younger patients who have more physiologic reserve may not manifest symptoms until malperfusion has significantly progressed. Once complications develop, the risk of mortality significantly increases. We believe it is appropriate in select cases to use tailored endovascular therapy in patients with acute TBAD and radiographic features that suggest either impending or subclinical malperfusion.

Although both the ADSORB and INSTEAD trials included patients with uncomplicated TBAD, they did not demonstrate any short-term improvement in survival as compared with medical management. However, both trials were underpowered, were performed in the earlier phase of application of endovascular therapy, and did not take into account the nuances of successful stent grafting. Stent grafting is not without complications, which can occur in up to half of patients. Both trials suggested aortic remodeling and increased false lumen thrombosis in the short term, and this was also demonstrated in the INSTEAD-XL trial.

Recent advances in endovascular stenting, including use of bare-metal stents to expand the true lumen, may help obviate the need for visceral and renal stenting in some patients with malperfusion, making endovascular therapy for acute TBAD more accessible to cardiovascular specialists across the world.



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Acute TBAD accounts for 25% to 40% of all aortic dissections; however, there is still debate on the criteria that determine a "complicated" dissection and when to intervene in the absence of malperfusion syndrome or hemodynamic instability. Since its first introduction by Wheat and colleagues, 1 optimal medical treatment (OMT) has been the first-line therapy in managing patients acutely and has significantly improved in-hospital mortality. However, contemporary data sets from the INSTEAD trial show that up to 12% of patients fail OMT within the first 14 days of treatment, with up to 41% requiring intervention within 6 years.² This suggests that OMT may only be a temporary measure and that more can be done to improve outcomes. The insights from these contemporary data, combined with the advent of endovascular technologies and more advanced imaging modalities, may provide greater justification for intervention in this subset of patients.

The most commonly identified presentation of acute TBAD is severe hypertension. Although this usually subsides, a retrospective analysis by Durham et al identified that the majority of TBAD patients (65.8%) were undergoing some form of antihypertensive therapy at the time of initial presentation, with 43.6% on a multiple-drug regimen.³ It is also well known that those who remain hypertensive are at an increased risk of adverse events. So, although OMT is important acutely, it is not always easily obtainable. We often see radiologic evidence of malperfusion of one or both renal arteries. The question

proposed, however, is whether radiologic evidence of malperfusion in an asymptomatic patient is an indication to intervene. We personally do not believe that this entity exists. If it does, we truly would manage with medical therapy alone. The clinical impact of endorgan function—whether it is a visceral, renal, or lower extremity branch vessel—may be far less pronounced at initial presentation, and intervention can be delayed until the subacute phase. However, we believe most of these patients are actually more symptomatic than we believe. According to the interdisciplinary expert consensus document on the management of type B aortic dissection, and must be recognized early through both radiographic and laboratory means.

Access to and use of high-resolution imaging modalities (multidetector CT, MRI, intravascular ultrasound [IVUS]) is essential in identifying patients at high risk for end-organ or limb malperfusion after presenting with acute TBAD. Although adequate in the initial identification of the dissection, CT angiography may underestimate the true extent and effect of malperfusion because it only presents a static image of the dissection. The use of dynamic MRA or IVUS can more substantially visualize the flap motion, which in turn allows for identification of possible intermittent ischemia to the end organs. Such ischemia, while not clinically relevant at the time, can result in eventual damage to end-organ function and more importantly to overall survival. Celiac perfusion via collateral flow, such as the gastroduodenal artery, can support function; however, mesenteric ischemia may result in increased release of bacteria, toxins, and vasoactive mediators, myocardial depression, systemic inflammatory response, sepsis, bowel ischemia, and eventually possible bowel necrosis. In the case of the renal arteries, the ischemia itself greatly decreases the effectiveness of any attempts to medically manage hypertension.

The acute presentation of TBAD requires the assessment of many variables in order to determine the best course of treatment. Medical management alone may not be optimal for every patient, and in those with radiographic malperfusion, additional treatment should be considered. Survivability curves for both IRAD and INSTEAD-XL trials support the premise for endovascular intervention for TBAD with adjunctive OMT.

^{1.} Wheat MW Jr, Palmer RF, Bartley TD, Seelman RC. Treatment of dissecting aneurysms of the aorta without surgery. J Thorac Cardiovasc Surg. 1965;50:364–373.

^{2.} Nienaber CA, Rousseau H, Eggebrect 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.

^{3.} 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.

^{4.} Fattori R, Cao R, De Rango P, et al. Interdisciplinary expert consensus document on management of type B aortic dissection. J Am Coll Cardiol. 2013;61:1661–1678.



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What this question essentially asks is whether patients with silent clinically asymptomatic ischemia should be considered complicated cases of type B dissection and be treated similar to patients with obvious malperfusion syndromes. The community agrees and guidelines strongly recommend treating malperfusion and ischemia efficiently (with the use of stent graft reconstruction or scaffolding of the true lumen) by redirecting flow to the true lumen, thereby improving symptoms and survival.¹

At present, there are no signs or data to support the idea that symptoms alone are crucial for treatment; rather, all available data show that evidence of malperfusion is a signal of danger.^{2,3} For instance, in IRAD experience, it became clear that asymptomatic dissection can actually cause more harm with worse outcomes than dissection with classic symptoms²; chances are that both diagnosis and progressive worsening will be missed. In other words, the lack of symptoms (ie, pain or discomfort) does not negate malperfusion to vital organs such as a healthy kidney or segments in an intact bowel that may not yet be symptomatic but hypoperfused on radiographic images. You are basically waiting for damage to occur, as seen by progressive loss of parenchyma in the absence of symptoms.

As a general principle in cardiovascular medicine, symptoms alone have never been the sole reason for curative or restorative treatment. Consider silent myocardial ischemia, collateral flow jeopardy with single-sided carotid obstruction, or peripheral artery occlusion that lacks symptoms of claudication at rest. If we could apply a stress test to single-sided kidney hypoperfusion on imaging, or if a kidney could cry, tissue malperfusion would not be overheard, and a nihilistic "academic" discussion by superskeptics would have no audience.

Beyond the recognition of silent, asymptomatic, but undoubtedly existing malperfusion, the element of preemptive treatment prior to organ damage needs to be discussed and understood.³ Why wait in a high-risk

scenario for damage to manifest if you can identify the thread and prevent it? Or why leave a healthy organ at risk if you can save it?

It all boils down to a proper risk-benefit analysis that balances the risk of an endovascular procedure with the benefit and prospect to save an organ or prevent further disease progression and late complications. However, a safe environment, expertise, and skills are essential and required for intervention. Regionalized care provided by specialized centers of excellence with multidisciplinary teams exposed to a high caseload is likely to provide the highest standard of care. With such standards at hand, it would be a low standard of care to leave silent malperfusion untreated.

- 1. Fattori R, Cao R, De Rango P, et al. Interdisciplinary expert consensus document on management of type B aortic dissection. J Am Coll Cardiol. 2013;61:1661–1678.
- 2. Fattori R, Montgomery D, Lovato L, et al. Survival after endovascular therapy in patients with type B aortic dissection: a report from the International Registry of Acute Aortic Dissection (IRAD). JACC Cardiovasc Interv. 2013;6:876–887
- 3. 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.