Treatment for malperfusion syndrome in acute type A and B aortic dissection: A long-term analysis

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Aortic dissection remains the most frequent and lethal complication of thoracic aortic disease, and although advances in care have demonstrated improved outcomes for patients with acute aortic dissection, malperfusion remains a significant adverse risk factor for survival for both acute type A and B aortic dissection.¹⁻⁴ Malperfusion syndrome is defined as the loss of blood supply to a vital organ caused by branch arterial obstruction secondary to the dissection. The result is end-organ ischemia, with possible malfunction and infarction if not corrected in a timely fashion. Malperfusion can be classified as both dynamic and static, depending on the anatomic obstruction. Dynamic obstruction is secondary to the motion of the intimal flap within the aortic lumen resulting from hemodynamic forces. The intimal flap may obstruct the orifice of a branch vessel to a vital organ, causing ischemia with potential end-organ dysfunction and infarction. As a dynamic phenomenon, malperfusion may vary depending on changes in blood pressure and hemodynamic forces, and in many cases can be adequately managed with medical therapy: controlling blood pressure and hemodynamics. If this fails, intervention is needed. Static malperfusion is defined as dissection of the branch vessel with obstruction of the true lumen. This form of obstruction cannot be corrected with medical management and will require interventional correction.^{5,6}

Approximately one third of patients with type A dissection manifest malperfusion syndrome.⁷ If the malperfusion is dynamic, restoration of true aortic luminal flow by eliminating the tear and replacing the ascending aorta will often correct the secondary malperfusion.⁸ With delayed diagnosis of dissection, however, established end-organ ischemia and clinical deterioration are typical, and the conventional strategy of immediate ascending aortic repair may be suboptimal. In these situations, alternative management strategies should be considered.

In 1997, we described our early results for the strategy of operative delay for those patients presenting with acute type A dissection, malperfusion, and ischemic end-organ dysfunction. In that study, a historical cohort of patients presenting with ischemic end-organ dysfunction from malperfusion taken directly for open repair was compared with a cohort managed with initial percutaneous fenestration, selective branch vessel stenting, and delayed operative repair after resolution of the perfusion injury. In contrast with an in-hospital mortality of 89% for the historical group, those patients who underwent operative delay had an overall 25% mortality, including a 15% mortality from rupture.⁹

These early results led to a consistent strategy for all patients presenting with acute type A dissection with significant end-organ ischemia from malperfusion at our institution: restoring end-organ perfusion before operation. After 10 years, we reviewed this strategy to determine the outcomes and the validity of the approach. Over that time period, 196 patients with acute type A dissection presented to the University of Michigan Hospital, and 70 were diagnosed with malperfusion and end-organ dysfunction. These patients underwent the strategy of percutaneous end-organ reperfusion and medical stabilization, followed by surgical repair. There was a 95% success rate in opening obstructed vessels percutaneously. Twenty-three patients (38%) died before surgical repair, 12 (19%) of rupture and 11 (19%) of malperfusion complications. This was in stark contrast with the 126 patients who did not have malperfusion or end-organ dysfunction, whose early operative mortality was 9.5%. Cumulative survival analysis showed that the median survival for the uncomplicated patients was 96 months, whereas for the malperfused it was 54 months ($P < .001$).

An analysis was also undertaken to determine whether malperfusion of any particular vascular bed was an independent correlate of mortality: Mesenteric malperfusion was statistically significant. We then analyzed the outcomes for the uncomplicated patients versus the malperfused patients who survived the percutaneous protocol and went on to operative repair. This analysis was to determine whether corrected malperfusion syndrome would have an adverse impact on surgical outcomes once the syndrome had resolved. The analysis compared death, stroke, and need for dialysis or tracheostomy between the 2 groups and found no statistically significant difference. Cumulative survival curves also showed no statistical significance, with uncomplicated patients having a median of 96 months of survival versus 81 months for the malperfused patients who proceeded to surgery.¹⁰ It was concluded that presentation of

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acute type A dissection with malperfusion and end-organ dysfunction is an important adverse factor for long-term survival, particularly in the setting of mesenteric malperfusion. The strategy of immediate reperfusion, stabilization, and planned operation still carries a significant risk for early mortality (38%). Those patients who do survive the initial malperfusion and undergo repair have no adverse effect and have a similar operative and late survival when compared with those presenting with uncomplicated dissection.

We believe it is important that the distinction is clearly made between malperfusion and malperfusion with ischemia and end-organ dysfunction. Those patients with malperfusion but no significant adverse end-organ effects are best treated with immediate surgical repair. An acute type A dissection is still a surgical emergency, and patients without end-organ ischemia—both with and without malperfusion—have an equal operative risk, which is significantly lower than patients with malperfusion and end-organ dysfunction.

The International Registry of Acute Dissection recently published data noting an in-hospital mortality of 13% for all patients with acute type B dissection. Branch vessel involvement or malperfusion was an independent predictor of early death, with an odds ratio of 2.9 ($P = .002$).⁴ The options for patients with type B malperfusion are open surgical repair, percutaneous fenestration, and bare-metal stenting to create a reentry tear and establish reperfusion, and thoracic endograft placement (TEVAR) to cover the primary tear. Publications show that even with the latest surgical techniques and accumulated experience, the early mortality for those requiring operative repair ranges from 18% to 36%.^{4,11} The International Registry of Acute Dissection published a 32% mortality.⁴

It was in this setting that we and others described a percutaneous endovascular solution of fenestration and stenting to relieve end-organ ischemia from branch vessel compromise as a potential method to improve early outcomes.^{5,6} Thoracic endografting was soon proposed as an alternative solution to open repair or percutaneous fenestration as a means to resolve acute type B dissection with malperfusion.¹² With this approach, the entry tear is sealed by TEVAR and the dynamic obstruction is relieved. Associated static obstruction is not treated by TEVAR and requires open revascularization or branch vessel stenting. The benefits of TEVAR in this setting include not only the exclusion of the entry tear and relief of dynamic obstruction but also promotion of thrombosis of the false lumen, with subsequent beneficial aortic remodeling, possibly reducing the need for subsequent future operations or rupture.

The demonstration of beneficial aortic remodeling after TEVAR for aortic dissection with malperfusion was first defined by Dake and colleagues,¹² and later verified by Rodriguez and the group from Arizona.¹³ Duebener and colleagues,¹⁴ from Germany, reported results in 10 patients with type B dissection and malperfusion in whom they had

90% success excluding the entry tear and reestablishing perfusion. However, they had a 20% mortality rate and a 30% complication rate that required crossover to open surgery. They concluded that TEVAR is a promising form of therapy, but that refinements in stent design and application are needed to improve the prognosis for these very sick patients.¹⁴ Szeto and colleagues,¹⁵ from the University of Pennsylvania, described the use of TEVAR for type B complicated dissections in 35 patients. Seventeen of these patients were treated for malperfusion: They reported no operative mortality in this group and 94% 1-year survival. However, long-term data in this study were lacking, and the incidence of neurologic complications in the entire series exceeded 10%.¹⁵ Feezor and colleagues,¹⁶ from Gainesville, reported on the use of TEVAR for 33 patients with acute complicated type B dissection. The 30-day in-hospital mortality for their group was 21%, with 15% having permanent spinal cord complications, 12% having renal failure requiring dialysis, and a 12% incidence of stroke. They concluded that TEVAR for complicated type B dissection was associated with significant mortality and morbidity, and that the overall role for treatment should be better defined and compared with other surgical and interventional options.¹⁶

We reported long-term results for percutaneous fenestration with bare metal stenting to recreate a reentry tear and establish reperfusion in patients with acute type B dissection with malperfusion.¹⁷ A cohort of 69 patients was studied, with a technical success rate of 96%. There was a 17% mortality rate in this group: 7% dying of false lumen rupture and 10% dying of malperfusion complications. The incidence of stroke was 4.3%, and 14% required temporary dialysis, which resolved. There was no paraplegia or spinal cord injury caused by the procedure, and no retrograde aortic dissection. Kaplan–Meier survival curves showed a 1-year survival of 76%; 5-year survival was 65%, and 8-year survival was 55%. The freedom from open repair or rupture was 80% at 1 year, 68% at 5 years, and 55% at 8 years, with a median time of treatment failure at 80 months. The important limitation of this approach is its inability to treat the risk of aortic rupture.

An acute type B dissection complicated by malperfusion with end-organ dysfunction is an important adverse factor for early and late survival. The strategies of percutaneous therapy seem to improve survival when compared with historical reports of both medical and surgical intervention. TEVAR is an attractive percutaneous technique because it eliminates the entry tear, reexpands the true lumen, allows remodeling, and potentially markedly reduces the risk of rupture. However, neurologic complications, trauma to the aortic wall resulting in dissection, or rupture occur in significant numbers, and increased experience and long-term data are needed to assess this therapy. The long-term data from fenestration and stenting can be used for comparison to evaluate TEVAR as a primary therapeutic modality in complicated acute type B dissection.

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