How do you manage uncomplicated type B dissections?

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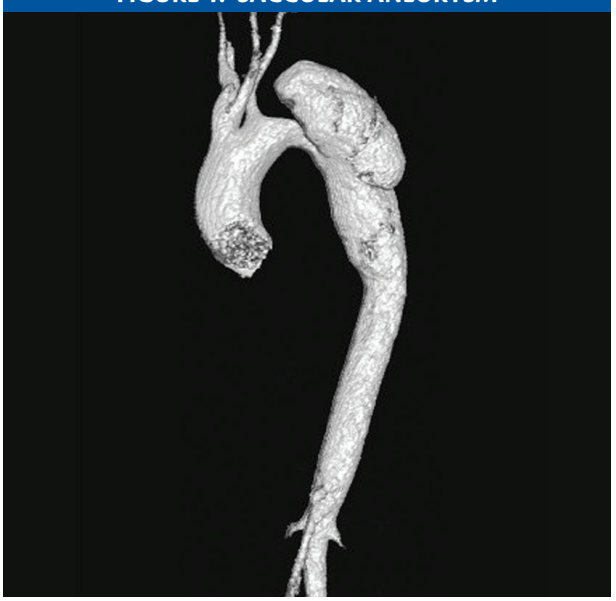
Uncomplicated type B thoracic aortic dissections are not a benign disease process. Although medical management with strict blood pressure control is beneficial in the short term, the primary late complication is aneurysm formation. In fact, nearly one-third of patients with type B dissections are at risk of significant late aneurysm development requiring surgical or endovascular treatment within the first 5 years after the initial event. Several radiographic findings may help predict individuals at risk for late aneurysm formation: maximum aortic diameter ≥ 40 mm, false lumen patency, false lumen diameter ≥ 22 mm, large proximal entry tear, and/or partial false lumen thrombosis. Moreover, improvements in the design and development of thoracic aortic stent graft devices have expanded the application beyond degenerative aortic aneurysms to now include treatment for acute complicated type B dissections and penetrating aortic ulcers.

FIGURE 1. SACCULAR ANEURYSM



Despite this, and given the lack of high-quality data, I remain conservative in my management of uncomplicated type B dissections. If there is no evidence of end-organ ischemia, pain, or rapid expansion, I will treat the vast majority of patients medically. If, however, there is a change in the clinical course of the patient, there exists a large associated saccular aneurysm (Figure 1), or if the associated aneurysm is ≥ 5.5 cm, consideration for early thoracic stent grafting is factored into the management.

In these circumstances, as in the complicated type B dissections, the procedure is performed with the main intent to cover the primary entry tear and any additional large fenestrations within the thoracic aorta to minimize false lumen flow and initiate false lumen thrombosis. I find procedural use of intravascular ultrasound to be extremely useful in assessing true lumen expansion and identifying associated fenestrations. Although it is tempting to expand the application of thoracic stent grafting for uncomplicated type B dissections, additional data are needed to support more widespread use.

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As of this time, there is no uniform strategy for treating uncomplicated type B dissections. Whereas with com-

plicated type B dissections, the benefits of intervention clearly outweigh the significant morbidity and mortality of medical therapy alone, with uncomplicated dissections, the risk/benefit ratio is still unclear. Clearly, the INSTEAD and ADSORB trials have demonstrated the potential benefit of thoracic endovascular aortic repair over time due to increased false lumen thrombosis and prevention of subsequent aneurysmal degeneration. Specifically, INSTEAD demonstrated reduced aortic mortality and reduced disease progression after 5 years. However, these benefits were small, which questions the utility of treating all patients with uncomplicated type B dissections, given that this treatment bears morbidity and mortality.

There is a fair amount of literature that stratifies these patients into those who are at increased risk for future aneurysmal degeneration. Many of these criteria are based on initial anatomic factors such as the patency and size of the false lumen. Using these criteria may help us better predict which patients will develop aortic degeneration over time and thus allow us to selectively treat those at highest risk. Ultimately, more data comparing intervention versus optimal medical therapy will help all of us determine who is best treated and who is best observed.

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We all hoped that the INSTEAD trial would answer the question posed with regard to the best management for patients with uncomplicated type B aortic dissection. But if anything, it has raised more questions than it has answered. It appeared that, for the first 2 years, endovascular intervention subjected patients to a significant perioperative risk, while patients on best medical therapy were largely unaffected in terms of aortic-related events. However, the longer-term results have shown that a cohort of patients on best medical therapy died from aortic-related events, which could in theory have been prevented by endovascular intervention at the time of presentation. Conversely, there is almost certainly a cohort of patients who will gain no benefit from endovascular repair. The key likely relates to the degree of aortic remodelling. We need to focus efforts into identifying the group that will benefit most (and least) from endovascular repair and optimize the timing and nature of the intervention itself.

So how do I currently balance that early perioperative risk of stroke, paraplegia, and death, with the knowledge that some patients might undergo unnecessary intervention? It has to be with an individualized approach. Aortic dissection is never simple, and we must move away from the traditional classification based on anatomical factors and duration of symptoms. There will also never be an answer to the simple question, "Is intervention better than conservative management?" Also, rather analogous to the debate regarding asymptomatic carotid interventions, if we are to achieve maximum clinical benefit in the group of uncomplicated patients, we must bring the risk of intervention to an absolute minimum.

I would have a low threshold for intervening in young patients, with even mild degrees of pain and/or hypertension. Especially in this group, I would try and wait 14 days before intervening. In the VIRTUE study, there was a significant reduction in perioperative morbidity and mortality associated with intervention in the subacute period (14–92 days), with results similar to those in the chronic group. The 3-year follow-up data, however, demonstrate degrees of aortic remodeling similar to acute dissection, with very low reintervention rates. It is feasible to hypothesize that waiting at least 14 days allows stabilization of the intimal tear. This potentially enables safer delivery of endografts into an otherwise very fragile aorta, and reduces the risk of retrograde type A dissection, which entails a 35% mortality rate.

I would keep intervention as simple as possible, to minimize the degree of wire and device manipulation in the aortic arch, again to reduce the risk of retrograde type A dissection. In terms of device choice, I use the device I am most familiar with. A recent review of the literature from my unit has shown the configuration of the stent does not influence the risk of retrograde type A dissection, including the presence of a bare proximal stent.

Furthermore, I strongly believe thoracic endografting in these patients should be performed by experienced endovascular practitioners, in high-volume aortic centres, with cardiothoracic surgery and intensive care facilities on site.

Going forward, we need to continue to question the classification and management of acute type B dissection. Further work is required to establish the subgroup of patients who may be most likely to benefit from early endografting, in particular with regard to an individual's estimation of risk and the role of detailed aortic imaging. In the meantime, I cannot justify intervention for all. I will continue to use careful clinical judgement alongside detailed counselling of each patient to guide the role and timing of intervention. ■