

Endovascular stenting of the ascending aorta for visceral malperfusion in a patient with type A aortic dissection

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ABSTRACT

A type A aortic dissection is a challenging condition for both cardiothoracic and vascular surgeons. Although open surgery remains the gold standard, there is considerable interest in the use of endovascular techniques for patients who present with malperfusion. We present the case of an unstable 55-year-old man with visceral malperfusion from a type A dissection who was stabilized using an endovascular technique as a bridge to open surgery. A bare metal thoracic endograft was used in the ascending aorta to rapidly restore perfusion. This hybrid approach to the problem of malperfusion in type A dissection could be useful for these patients with complicated cases. (*J Vasc Surg Cases Innov Tech* 2023;9:101341.)

Keywords: Dissection; Endovascular; Malperfusion; TEVAR

Traditionally, the mainstay treatment of a type A dissection has been open surgery; however, even in high-volume surgical centers, mortality has been reported at 13% to 25%.^{1,2} Patients who present with visceral malperfusion have an even poorer prognosis with higher rates of mortality of >60%.^{3,4}

With the advent of endovascular surgery, the approach to treating dissections has recently changed. For type B aortic dissections, thoracic endovascular aneurysm repair (TEVAR) has become popular for treating symptomatic patients and might prevent future aneurysmal degeneration. For patients who present with visceral malperfusion, TEVAR can change the long-term survival to be similar to that for patients with an uncomplicated dissection.⁵

Limited advancement has occurred with the endovascular technique for type A aortic dissection outside of trials and physician-sponsored investigational device exemptions. Few off-the-shelf Food and Drug Administration–approved endovascular options are available. The aortic branch vessels preclude coverage with a covered graft and require open repair for definitive management. In case reports in which an endovascular technique has been used as a bridge to open repair, the question of the appropriate timing for definitive open surgery remains, especially for those who present with visceral malperfusion.⁶

In the present report, we demonstrate an endovascular approach to stabilize a young patient presenting with a

type A aortic dissection complicated by cerebral, peripheral, and visceral malperfusion before his open surgery. True lumen expansion allowed for resolution of the malperfusion and served as a bridge to definitive open repair. The patient provided verbal informed consent for the report of his case details and imaging studies.

CASE REPORT

The patient was a 55-year-old man who presented with severe chest pain radiating to his back and syncope. In the emergency department, the patient was severely hypotensive, tachycardic, and confused. He had absent radial and pedal pulses, concerning for malperfusion of both upper and lower extremities, with significant motor and sensory deficits of his lower extremities. Emergent computed tomography angiography of his chest, abdomen, and pelvis demonstrated a type A aortic dissection extending from his aortic root, involving all the major arch vessels, and continuing down into the left common iliac artery (Fig 1). The arch vessels demonstrated minimal flow into the left internal carotid and bilateral subclavian arteries. The infrarenal aorta had no flow beginning just proximal to the aortic bifurcation and without reconstitution. His visceral vessels appeared opacified with diminished flow. The laboratory test results were remarkable for a pH of 7.32 and lactate of 10.1 mmol/L.

The initial plan was open repair; however, given the patient's hemodynamic instability, end-organ ischemia, and profound lactic acidosis, the vascular surgery team was asked about potential hybrid options that might help alleviate the systemic hypoperfusion. We initially planned to place a thoracic endograft to the level of the left subclavian artery. However, because the tear was in the ascending aorta and the flow to the great vessels was compromised, we decided to place a bare metal stent into the ascending aorta and across the arch. We hoped this would realign the orifices of the great vessel. The patient was brought emergently to a hybrid operating room, anesthesia was administered, arterial lines were placed in both upper extremities, and unilateral open femoral access was obtained directly on the right side via a cutdown technique. No spinal drain was placed, as is our institutional practice. A pigtail catheter was advanced into the ascending arch, following which intravascular ultrasound confirmed the true lumen location of the wire. Initial angiography demonstrated malperfusion of the

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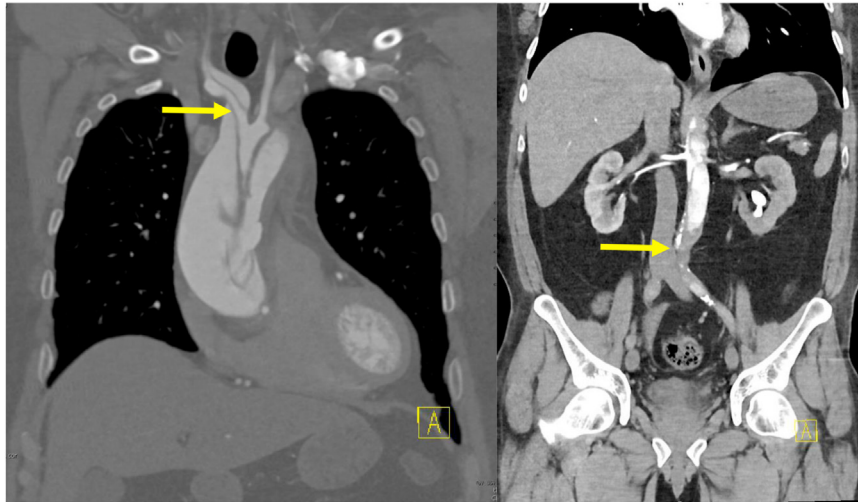


Fig 1. Preoperative imaging study showing dissection extending into innominate artery (**Left**) and occlusion of distal aortic bifurcation (**Right**).

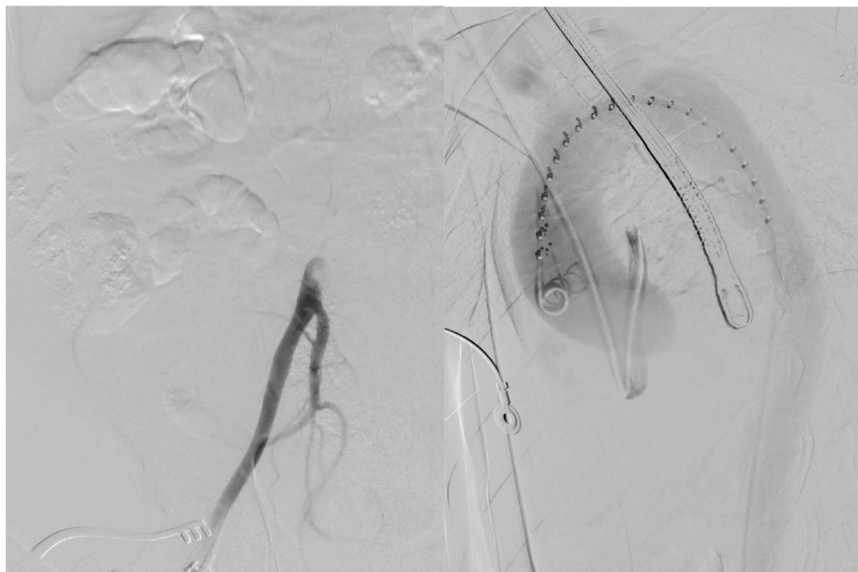


Fig 2. Initial angiography via femoral access demonstrating dissection flap occluding flow (**Left**) and malperfusion to the great vessels of the arch (**Right**).

patient's cerebral and visceral arteries (Fig 2). The initial computed tomography angiogram showed the coronary vessels were preserved.

Intravascular ultrasound demonstrated a compressed true lumen that was nearly obliterated. The ascending aorta was aneurysmal and measured >50 mm in diameter, but the arch was normal in caliber. A covered stent was not an option because the ascending aorta was short and no landing zone was present. At this point, we debated between placing an endograft at the level of the left subclavian artery vs placing a bare metal dissection stent across the arch. The dissection stent comes in two diameters, 36 mm and 46 mm. A 36-mm \times

180-mm Cook Zenith dissection endovascular stent (Cook Medical Inc) was chosen because the arch was normal in size. The goal was to place the dissection stent to realign the great vessels. The intravascular ultrasound images were critical for evaluating the aortic tear and helping to identify the area of the ascending aorta without the dissection, where the proximal aspect of the graft would be best suited for deployment. The dissection stent was advanced up into the ascending arch and deployed, extending from just distal to the aortic valve across the origins of the head vessels and down to the proximal descending aorta. The stent was deployed in the presence of hypotension; thus, no additional maneuvers were used. The

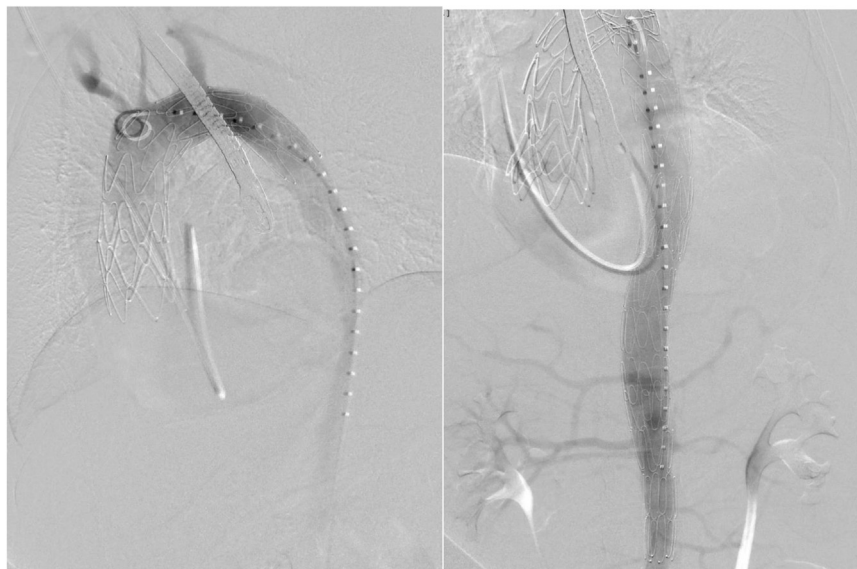


Fig 3. Angiography after repair showing improvement in flow to the innominate, left carotid, and left subclavian arteries (**Left**) and restoration of flow to the visceral vessels (**Right**).

hypotension allowed for relatively precise deployment distal to the coronary ostia and across the arch. This reapproximated the ostia of the branch vessels with the dissection flap and restored flow to the cerebral circulation and upper extremities, which was confirmed via the bilateral arterial lines. The previous stent deployment was then extended further using a 38-mm × 167-mm Cook Alpha thoracic endovascular graft (Cook Medical Inc), which was deployed just distal to the left subclavian artery. The rationale for using the endograft was to provide enough radial force for true lumen expansion, stabilize the dissection, and prevent migration. A second 36-mm × 180-mm Cook Zenith dissection endovascular stent (Cook Medical Inc) was placed to the level of the aortic bifurcation. The patient's vital signs subsequently improved, with a return of palpable pulses in all four extremities (Fig 3). The initial dissection extended into the great vessels with complete occlusion of the distal left common carotid artery. Dedicated imaging was not performed due to the presence of this acute dissection; however, angiography from the arch demonstrated patency of the carotid vasculature. Completion angiography demonstrated improved flow to the patient's visceral vessels and no evidence of thrombus. The patient was then transferred to the intensive care unit for resuscitation and observation.

The patient had significant lactic acidosis, and concern existed for organ malperfusion. However, due to the hemodynamic compromise at presentation, we planned on resuscitation with a low threshold for a return to the operating room to assess for mesenteric or extremity ischemia. The general surgery team was asked to evaluate the patient and be available on standby. During the next 24 hours, the patient improved, with resolution of the lactic acidosis and no concern for extremity reperfusion injury. The next day, on weaning from sedation, he was noted to be neurologically intact. He was extubated, and,

again, a thorough physical examination revealed no neurologic deficits, normalization of metabolic derangements, and complete resolution of his chest and back pain. The initial TEVAR was performed on a Friday afternoon. In consultation with the cardiac surgery team, we chose to perform definitive repair on a weekday. On postoperative day 3, he returned to the operating room for open repair of his proximal aorta. The patient underwent zone II arch replacement, debranching of the innominate artery and left common carotid artery with a bifurcated graft using selective antegrade perfusion and deep hypothermic circulatory arrest, and aortic root replacement. The bare metal stent was removed with relative ease in its entirety. The ascending graft was sewn to the dissected portion of the native aorta. Additional distal dissection was aborted due to the posterior location of the subclavian artery and concern for not being able to safely access the vessel for left subclavian artery bypass. As such, a segment of dissected aorta was left between the ascending graft and the TEVAR graft. The remainder of his hospital course was unremarkable, and he was discharged home on postoperative day 10 after his index TEVAR. Computed tomography imaging at 2 months demonstrated an intact aortic arch repair with adequate perfusion of the bilateral upper and lower extremities and visceral arteries (Figs 4 and 5).

DISCUSSION

Type A aortic dissection is a potentially devastating condition associated with significant morbidity and mortality. We present the case of a 55-year-old man with an acute type A dissection involving significant malperfusion who was stabilized successfully using endovascular techniques as a bridge to definitive open repair.

Endovascular approaches to treat type A dissection have previously been reported, although in a limited

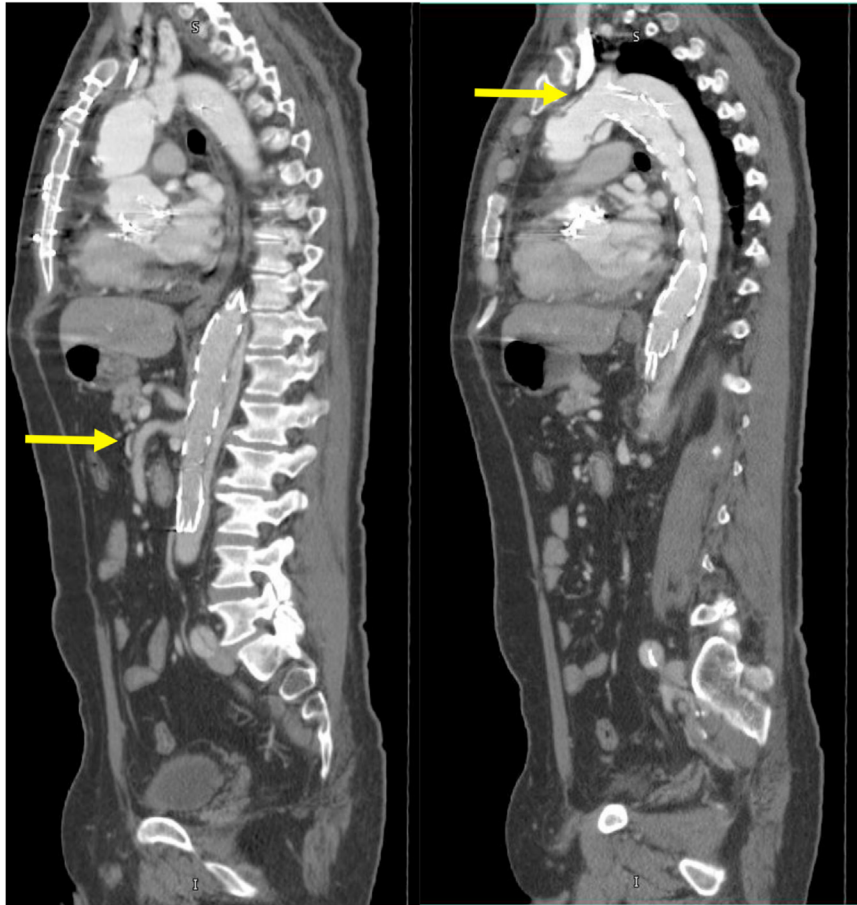


Fig 4. Computed tomography angiography at 1 month postoperatively showing perfusion of visceral vessels (*arrow; Left*) and origin of endovascular graft distal to left subclavian artery (*arrow*) after the proximal bare metal stent was removed during definitive open repair (**Right**).

fashion, ranging from an attempt at repair to as an adjunct for definitive open repair. These reports have been of patients whose dissection did not extend to the aortic root and who did not have symptoms of malperfusion.^{7,8} For patients who present with visceral malperfusion, endovascular fenestration is one technique that has been used to alleviate end organ ischemia before delayed open aortic arch replacement.⁹ One complication of this technique is the increased risk of aortic rupture as patients await their open repair.¹⁰ Patients treated with endovascular fenestration have been shown to achieve overall outcomes similar to those of patients who presented without malperfusion.¹¹

Data on staged endovascular to open repair is limited for type A dissections. Leshnowar et al¹² reported that for patients with malperfusion, initial treatment with TEVAR as a bridge to open repair resulted in improved outcomes for patients compared with other techniques. TEVAR first allowed patients to recover from their visceral malperfusion before undergoing delayed open repair.¹² In this technique, stents were not placed within the

ascending aorta and were only placed within 2 cm of the left subclavian artery.

In the present report, we describe an approach for stabilizing patients with type A dissection and presenting with multilevel malperfusion. Use of a bare metal stent allows for rapid alignment of the vessel orifices, thereby restoring perfusion, as evidenced in our patient. Furthermore, this approach could be preferred for cases such as local fenestrations, which run the risk of the septum unravelling unpredictably and covering the ostia of critical vessels. Placement of the stent and supporting it with an endovascular stent graft distal to the left subclavian artery allowed for rapid true lumen expansion and restoration of perfusion to the visceral vessels. The ascending aorta experiences significant forces, resulting in concern for graft dislodgement and “wind-socking.” A bare metal stent might not experience the same hemodynamics due to the absence of fabric that would otherwise cause the delivery system, as well as the graft, to be pushed down. It is unclear whether precise deployment could require additional maneuvers such as rapid ventricular



Fig 5. Computed tomography angiography at 1 month postoperatively showing adequate perfusion of bilateral common iliac arteries (arrow).

pacing or transvenous pacing, because our patient was hypotensive.

The described technique has some limitations. One is the availability of resources at community hospitals without access to a hybrid operating room or intravascular ultrasound. Precise placement of the proximal landing zone is imperative during the procedure. Additional risks of placing a bare metal stent in the arch are unknown because the stent was designed for use in type B dissections and in

conjunction with an endograft. Initially, a risk also exists of rupture during placement of the stent due to the thin wall of the damaged artery. Our choice of stent had the advantage of a lower radial force to prevent damaging the intima and further weakening the aortic wall.

CONCLUSIONS

Endovascular stenting using a combination of bare metal and covered stents to treat a type A aortic dissection with malperfusion could serve as a bridge to definitive open repair for high-risk patients who might not tolerate open repair as a first operation.

DISCLOSURES

None.

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