Aortic remodeling and competitive flow after surgical treatment of aortic dissection

Shin Mei Chan, BS,a Anand Brahmandam, MD,b Jonathan A. Cardella, MD,b John Elefteriades, MD,c John F. Setaro, MD,c Abeel A. Mangi, MD, MBA, and Cassius Iyad Ochoa Chaar, MD, MS,b New Haven, Conn

ABSTRACT
Aortic remodeling after dissection is poorly understood and remains a focus of current research. In the present report, we have described the cases of two patients with acute lower extremity ischemia related to malperfusion from aortic dissection treated with extra-anatomic axillobifemoral bypass. During long-term follow-up, aortic remodeling led to reinstatement of flow through the native aorta. This resulted in competitive flow, leading to complete thrombosis of the extra-anatomic conduits. These cases highlight the occurrence of spontaneous aortic recanalization and subsequent competitive flow, two vascular phenomena that are not well understood but can significantly affect patient outcomes. (J Vasc Surg Cases Innov Tech 2021;7:404-7.)

Keywords: Acute limb ischemia; Aortic dissection; Axillobifemoral bypass; Extra-anatomic bypass; Malperfusion; Recanalization

Aortic dissection can compromise perfusion to essential organs and can manifest as malperfusion. Spontaneous aortic recanalization after aortic occlusion from dissection is a phenomenon that has not been extensively reported. In the present report, we have described aortic remodeling and competitive flow in two patients who had presented with malperfusion from aortic dissection. The patients were treated with emergent extra-anatomic axillobifemoral bypass. Long-term follow-up of these patients showed spontaneous recanalization of the aorta, leading to restoration of flow through the native vessel. Subsequently, competitive flow led to thrombosis of the extra-anatomic grafts. This series of events is not well understood. The patients provided written informed consent for the report of their case details.

CASE REPORT
Patient 1. A 64-year-old man had presented to the emergency department with excruciating back pain and hypotension. His medical history was notable for essential hypertension and claudication or rest pain. On presentation, his systolic blood pressure was 178 mm Hg. A computed tomography angiogram (CTA) revealed an acute type A aortic dissection extending to zone 9. He underwent emergent root-sparing, open, ascending aortic, and hemiarch replacement with a 28-mm Hemashield graft (Boston Scientific, Marlborough, Mass) and aortic valve replacement with a 23-mm Carpentier-Edwards biological valve (Edwards Lifesciences, Irvine, Calif) under deep hypothermic circulatory arrest.

The patient returned to the intensive care unit for recovery, where pedal Doppler signals were not detected. Emergent CTA revealed collapse of the true lumen (zones 5-8) and complete aortic occlusion (zone 9), resulting in bilateral lower extremity ischemia (Fig 1). A left axillobifemoral bypass with an 8-mm ringed Gore-Tex graft (WL Gore & Associates, Flagstaff, Ariz) and bilateral four-compartment fasciotomies were performed. The remainder of his postoperative recovery was notable for respiratory failure necessitating reintubation and cholecystitis-related sepsis. After recovery, the patient was discharged on postoperative day 25.

At the 1-year follow-up, the patient was ambulating well without claudication, and surveillance CTA showed gradual restoration of flow in the native aorta (Fig 2). At 1.5 years, CTA demonstrated spontaneous recanalization of the formerly occluded infrarenal aortic segment and layering thrombus within the axillobifemoral bypass graft, which was also demonstrated on duplex ultrasound (Fig 2). Despite the layering thrombus in the bypass graft, normal velocities and waveforms were found on duplex ultrasound before complete thrombosis, which had occurred after 3 years. At 5 years, the patient demonstrated continued aortic remodeling with an increase in the maximal aortic diameter (41 ± 37 mm vs 37 ± 36 mm initially; Fig 3). At the last follow-up, his ankle brachial index (ABI) was normal, and he continued to lead an active lifestyle without claudication or rest pain.

Patient 2. A 62-year-old man had presented to the emergency department from an unaffiliated hospital with severe lower back pain. His pertinent medical history included...
smoking, hypertension, hyperlipidemia, previous chronic type B5-9 aortic dissection, and a known small infrarenal abdominal aortic aneurysm. Before presentation, the patient had discontinued his blood pressure medications. He had presented with a systolic blood pressure of 180 mm Hg. A CTA demonstrated a new, acute type B5-7 dissection tear with a narrow true lumen. The flap extended into the celiac axis, superior mesenteric artery, and right renal artery. Anti-impulse therapy was initiated for medical management.

The following night, the patient developed acute bilateral lower extremity ischemia associated with sensory and motor impairment on clinical examination. He had presented with a systolic blood pressure of 180 mm Hg. A CTA demonstrated a new, acute type B5-7 dissection tear with a narrow true lumen. The flap extended into the celiac axis, superior mesenteric artery, and right renal artery. Anti-impulse therapy was initiated for medical management.

The following night, the patient developed acute bilateral lower extremity ischemia associated with sensory and motor impairment on clinical examination. He had presented with a systolic blood pressure of 180 mm Hg. A CTA demonstrated a new, acute type B5-7 dissection tear with a narrow true lumen. The flap extended into the celiac axis, superior mesenteric artery, and right renal artery. Anti-impulse therapy was initiated for medical management.

A 9-month postoperative surveillance CTA demonstrated recanalization of the dissected lumen (zone 9), accompanied by thrombosis of the femoral–femoral limb of the axillofemoral bypass. At this point, the patient’s ABIs were normal. At 3 years, complete thrombosis of the axillofemoral graft had occurred with persistent inline flow through the aorta, and the patient has continued to lead an active lifestyle without claudication.

**DISCUSSION**

We have demonstrated the rare event of spontaneous aortic recanalization after occlusion in the setting of aortic dissection. Previously, Taguchi et al. reported spontaneous recanalization of an occluded descending thoracic aorta after an acute type B aortic dissection in an octogenarian with malperfusion who had been medically treated. Within 5 days, the aorta had spontaneously recanalized, and she had not experienced any sequela.1 DeAnda et al.3 reported spontaneous recanalization of an occluded aorta 6 months after axillofemoral bypass, which remained patent at 2 years of follow-up. In their
Finally, Choinski et al. reported an embolic occlusion of the infrarenal aorta, which had spontaneously recanalized 6 months after an axillofemoral bypass. Similarly, the bypass graft had occluded from competitive flow after recanalization of the aorta. The present report adds to the limited data of spontaneous recanalization of the aorta for a longer period than previously described.

Occlusion of the axillofemoral graft in both patients had likely resulted from competitive flow after aortic remodeling. Aortic remodeling has largely been described after thoracic endovascular repair (TEVAR), whereby reinstitution of inline flow through the true lumen leads to eventual thrombosis of the false lumen, stabilizing the aorta. In the present report, the dissections were not treated with TEVAR but, rather, with initial anti-impulse therapy. In patient 1, fresh aortic arch anastomoses and complete distal aortic occlusion precluded safe performance of TEVAR. In patient 2, TEVAR was precluded by the presence of a complex dissection with multiple tears and a distal aortic occlusion. In these patients, the specific mechanism of remodeling and the period required to reestablish inline aortic flow remained unclear.

In TEVAR-treated patients, aortic remodeling is often measured by true and false lumen measurements using CTA or magnetic resonance angiography; however, the surveillance methods used have been highly inconsistent. Based on the current Society for Vascular Surgery guidelines for surveillance of lower extremity revascularization using extra-anatomic bypass, we would recommend surveillance of bypass grafts using duplex ultrasound and ABI measurements at 3, 6, and 12 months after treatment, followed by 6 to 12 months thereafter.

After aortic remodeling, thrombosis of the grafts was likely due to competitive flow, whereby recanalization of the native vessel leads to increased flow, creating a pressure differential between two conduits. Subsequent resistance in the smaller prosthetic conduit and the larger native vessel leads to failure of the smaller conduit. The phenomenon has primarily been evaluated in the context of coronary artery bypass grafting failure. Berger et al. demonstrated that internal mammary artery grafts were more likely to occlude when the native vessels were only low-to-moderately stenosed owing to competitive flow. Another study suggested that the addition of multiple bypass grafts to a stenosed vessel could introduce more competitive flow, increasing graft failure. Competitive flow has been less studied in larger conduits, such as axillofemoral grafts and larger vessels.
such as the aorta. A better understanding of this sequela might improve patient selection and expectations during follow-up.

CONCLUSIONS

Spontaneous recanalization can occur after an acute aortic dissection and occlusion. Extra-anatomic bypass is effective for treatment of acute limb ischemia, and competitive flow after aortic recanalization can lead to thrombosis. Further research to better understand aortic remodeling is needed.

REFERENCES

1. Taguchi T, Saito S, Monta O, Ryugo M, Asada S, Yamada S, et al. Spontaneous recanalization of totally occluded descending aorta due to acute aortic dissection. Ann Thorac Surg 2019;108:e123.
2. Choinski K, Wood E, Korayem AH, Safir SR, Nakazawa KR, Tadros RO. Spontaneous recanalization of a total occlusion of an infrarenal abdominal aorta after left axillary-bifemoral bypass. J Vasc Surg Cases Innov Tech 2020;6:195-8.
3. DeAnda A, Kasirajan V, Henry D, Myers SI. Complete regression of an intramural hematoma of the aorta after distal reperfusion. J Vasc Surg 2005;42:149-52.
4. Watanabe Y, Shimamura K, Yoshida T, Daimon T, Shirakawa Y, Torikai K et al. Aortic remodeling as a prognostic factor for late aortic events after thoracic endovascular aortic repair in type B aortic dissection with patent false lumen. J Endovasc Ther 2014;21:517-25.
5. Patterson BO, Cobb RJ, Karihkesalingam A, Holt PJ, Hinchliffe RJ, Loftus JM, et al. A systematic review of aortic remodeling after endovascular repair of type B aortic dissection: methods and outcomes. Ann Thorac Surg 2014;97:588-95.
6. Leshnower BG, Duwayri YM, Chen EP, Li C, Zehner CA, Binongo JN, et al. Aortic remodeling after endovascular repair of complicated acute type B aortic dissection. Ann Thorac Surg 2017;103:1878-85.
7. Zierler RE, Jordan WD, Lai BK, Mussa F, Leers S, Fulton J, et al. The Society for Vascular Surgery practice guidelines on follow-up after vascular surgery arterial procedures. J Vasc Surg 2018;68:256-84.
8. Swillens A, De Witte M, Nordgaard H, Lövstakken L, Van Loo D, Trachet B, et al. Effect of the degree of LAD stenosis on competitive flow and flow field characteristics in LIMA-to-LAD bypass surgery. Med Biol Eng Comput 2012;50:839-49.
9. Pagni S, Storey J, Ballen J, Montgomery W, Qaqish NK, Etoch S, et al. Factors affecting internal mammary artery graft survival: how is competitive flow from a patent native coronary vessel a risk factor? J Surg Res 1997;71:172-8.
10. Berger A, McCarthy PA, Siebert U, Carlier S, Wijns W, Heyndrickx C, et al. Long-term patency of internal mammary artery bypass grafts: relationship with preoperative severity of the native coronary artery stenosis. Circulation 2004;110(Suppl 1):II36-40.
11. Harskamp RE, Alexander JH, Ferguson TB, Hager R, Mack MJ, Englum B, et al. Frequency and predictors of internal mammary artery graft failure and subsequent clinical outcomes. Circulation 2016;133:131-8.

Submitted Feb 23, 2021; accepted May 6, 2021.