Retrograde stenting of the superior mesenteric artery is the procedure of choice for dissection of the aorta with mesenteric compromise

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ABSTRACT
Acute mesenteric ischemia secondary to aortic dissection in type A and type B is a true vascular surgical emergency. Presentation can be subtle or dramatic, and time to revascularization is limited before irreversible changes occur. The literature recognizes the catastrophic consequences of acute superior mesenteric artery occlusion and the need for urgent revascularization, often before central aortic repair in type A. There is no optimal revascularization technique described in this scenario. We present a case of type A aortic dissection with acute dynamic and static superior mesenteric artery occlusion and describe treatment that resulted in successful salvage of the bowel and the patient’s life. (J Vasc Surg Cases and Innovative Techniques 2019;5:431-4.)

Keywords: Mesenteric ischemia; Aortic dissection; Type A; Revascularization; Superior mesenteric artery; Laparotomy

CASE REPORT
A 36-year-old woman presented to an outside hospital with sudden onset of severe abdominal pain, vomiting, and bowel evacuation. Physical examination demonstrated extreme hypertension; abdominal pain disproportionate to physical findings, which revealed mild tenderness in all quadrants without rebound tenderness; no evidence of lactic acidosis; and marginally elevated white blood cell count. Lower extremity pulse examination revealed diminished femoral and popliteal pulses and absent dorsalis pedis and posterior tibial pulses. Biphasic Doppler signals were heard over both dorsalis pedis and posterior tibial vessels. Renal function appeared intact as evidenced by satisfactory urine output and serum creatinine concentration. Computed tomography angiography (CTA) revealed type A aortic dissection (TAAD) with nearly total obliteration of the true lumen of the thoracoabdominal aorta with occlusion of the origin of the superior mesenteric artery (SMA; Fig 1). To further demonstrate the obstruction, Fig 2 shows dynamic and static SMA obstruction.

Measures to control blood pressure and anti-impulse therapy were initiated at the outside facility, and the patient was transferred to our tertiary medical center. At the time of our assessment, 24 hours had elapsed since symptom onset. The patient remained hemodynamically stable; she had a preoperative lactate level of 1.9 mmol/L and an intraoperative white blood cell count of 19.62 × 10⁹/L (only available white blood cell count for this admission), and she reported no chest pain. The complaint of abdominal pain was out of proportion to physical findings. CTA revealed a nearly total occlusion of the true lumen of the thoracoabdominal aorta and SMA. The celiac, SMA, and right renal artery arose from the true lumen of the aorta, and the proximal SMA was thrombosed.

We decided that restoration of mesenteric flow was the priority before TAAD repair. At the time of initial laparotomy, the small bowel was gray in appearance but viable. No pulse in the SMA at the root mesentery was felt; in addition, there was an absence of Doppler signals at the mesenteric border of the small bowel. There was no evidence of mesenteric venous thrombosis. The SMA was exposed at the root of the mesentery, and a distinct transition from the thrombosed vessel proximally to a patent vessel in the mid-SMA was seen. The SMA was accessed in the patent segment of the vessel under direct vision after isolating the SMA through laparotomy. To prevent distal embolization, the SMA was accessed in a segment that was free of thrombus as evidenced by the external appearance of the vessel.

Revascularization of the SMA by retrograde stenting is demonstrated in Fig 3. The SMA was accessed in the patent segment and a 7F sheath placed. The patient was then systemically anticoagulated with intravenous heparin at 100 units per kilogram of body weight. The size of the stent was based on the physician’s clinical judgment and the size of the vessel relative to the 7F sheath placed in the SMA. A Glidewire (Terumo Interventional Systems, Somerset, NJ) was advanced under fluoroscopy guidance and entered the true lumen of the dissected aorta. The course of the wire could be followed in the true lumen proximally in the descending thoracic aorta, which followed the
same course as had been noted on CTA. An 8×57-mm Express LD (Boston Scientific, Marlborough, Mass) balloon-expandable stent was placed from the origin of the SMA to the patent segment of the vessel. Immediate restoration of pulsatile flow to the bowel was achieved; it turned a normal pink color and had active peristalsis.

On postoperative day 1 (POD 1), the patient returned to the operating room for a second-look laparotomy. The bowel and SMA pulse were normal, and the abdomen was closed. On POD 2, the patient underwent ascending aorta and proximal hemiarch replacement with resuspension of the aortic valve for TAAD. Cardiopulmonary bypass (CPB) was initiated through a 10-mm conduit sewn to the right axillary artery. Cerebral perfusion on bypass was satisfactory by cerebral oximetry, remaining unchanged before and after establishment of CPB. Flow in the common femoral artery dropped significantly on CPB, suggesting some degree of limb malperfusion. This was corrected after central aortic reconstruction.

The subsequent postoperative course was uneventful except for abdominal pain on POD 1. The abdominal pain prompted another CTA study, which confirmed a widely patent stented SMA with normal bowel perfusion. She was discharged a week later in good condition. Her course at home during the following 3 months has been smooth, with no evidence of mesenteric ischemia. The patient gave consent to publication of this report.

**Fig 1.** Computed tomography angiography (CTA) imaging on initial assessment of the patient. A, Dissection in the aortic arch. B, Severely narrowed true lumen in the descending thoracic aorta (coronal). C, Dissection extending into the superior mesenteric artery (SMA).

**Fig 2.** Computed tomography angiography (CTA) imaging of both dynamic and static obstruction of the superior mesenteric artery (SMA).
DISCUSSION
Timing of aortic repair in the setting of visceral malperfusion remains a matter of some controversy. Approximately 30% of patients presenting with TAAD and ∼20% of patients presenting with type B aortic dissection (TBAD) suffer from malperfusion syndrome. The incidence of mesenteric ischemia is ∼5.0% to 7.0% in TAAD and TBAD, respectively. Visceral malperfusion is usually associated with renal and limb malperfusion. In individual studies, the incidence of visceral ischemia in complicated TBAD is between 13.8% and 25.0%. In-hospital mortality rates specifically from visceral ischemia range between 1.9% and 11.0%. The International Registry of Acute Aortic Dissection reported an incidence of visceral ischemia in 7.1% of patients with TBAD, with highly significant difference in in-hospital mortality rate of 30.8% in patients with visceral ischemia vs 9.1% without.

Thoracic endovascular aortic repair has largely replaced open fenestration procedures, which were previously used in malperfusion syndrome resolution. The goal of thoracic endovascular aortic repair is to seal the proximal entry tear, to recapture the true lumen, and to promote false lumen thrombosis. Therefore, the procedure overcomes dynamic obstruction of the branch vessels. When static obstruction of the branch vessels is additionally present, focal stenting or surgical revascularization techniques must be used to relieve ischemia around the branch vessel. Dramatic improvement in outcomes has been reported with endovascular interventions compared with open repair for malperfusion syndromes.

Fann et al reported a 31% incidence of malperfusion with a 5% incidence of visceral ischemia in a series of patients with TAAD. Operative mortality was 43% in patients with visceral ischemia. The International Registry of Acute Aortic Dissection reported an incidence of 3.7% of mesenteric ischemia in >1800 patients presenting with TAAD. A mortality rate of 63% vs 23.8% has been reported after central aortic repair in TAAD patients with visceral malperfusion vs those without. For this case, the physicians made the decision to restore mesenteric flow first because of the severe and advanced condition of mesenteric ischemia and the significantly increased risk for overall negative outcomes. In the opinion of the cardiac surgeon involved, in the time that it would take to repair the TAAD, the patient would suffer infarction of the bowel from SMA occlusion and probably die. In situations in which the clinical manifestation of mesenteric ischemia is not at an advanced stage, it would be reasonable to perform repair of the TAAD first and to observe whether this resolves the associated problem of mesenteric ischemia.

Retrograde stenting of the SMA was independently introduced by a single institution and subsequently validated as a treatment option by other reports, including the present author. The distinct advantage is being able to inspect bowel viability and rapidly re-establish mesenteric perfusion. In the absence of prosthetic material for bypass, the risk of graft infection is eliminated, particularly for compromised bowels. The short distance between the access point in the SMA and the point of obstruction gives greater maneuverability and the wire enters the true lumen, which is eccentrically...
compromised. The fact that the SMA almost always arises from the true lumen of the aorta speaks to the success of the intervention. In addition, the stent’s ability to traverse the length of the obstruction can overcome the component of static obstruction that may be present, as was the case in our patient.

CONCLUSIONS

We believe from our experience and with the support of other reports that retrograde stenting of the SMA is the preferred approach for patients with visceral malperfusion in the setting of TAAD and TBAD, where there is both dynamic and static obstruction of the SMA.

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