Delayed Intestinal Ischemia after Surgery for Type A Acute Aortic Dissection

We report a rare case of delayed intestinal ischemia after total arch replacement for type A acute aortic dissection. At the onset of acute aortic dissection, computed tomography (CT) angiography revealed celiac trunk occlusion and progressive dissection into the superior mesenteric artery without stenosis. However, following total arch replacement, visceral malperfusion was not detected by exploratory laparotomy. On postoperative day 12, the patient developed paralytic ileus without an elevated lactate level. CT angiography revealed new superior mesenteric artery stenosis by a thrombosed false lumen with persistent celiac trunk occlusion. Endovascular treatment including stent implantation resolved intestinal ischemia.

Keywords: aortic dissection, endovascular treatment, intestinal ischemia

Introduction

Mesenteric malperfusion caused by type A acute aortic dissection (AAD) is rare, occurring in 3.7% of patients, but it is associated with a tremendously high hospital mortality rate of over 60%, which may be related to the difficulty of accurate diagnosis and prompt treatment. Delayed mesenteric malperfusion after the surgical repair of type A AAD is very rare, and its clinical course is unclear. We herein describe a case of delayed intestinal ischemia caused by a newly narrowed superior mesenteric artery secondary to a thrombosed false lumen and persistent occlusion of the celiac trunk after total arch replacement for complex type A AAD with peripheral malperfusion that was successfully treated with endovascular treatment.

Case Report

A 56-year-old male with a history of smoking and untreated hypertension was referred to our hospital for treatment of AAD. His primary complaint was intense back pain, but he also developed pale lower limbs with bilaterally absent femoral artery pulsation and clouded consciousness when he arrived at our hospital. There was no history of connective tissue disorders. Computed tomography (CT) angiography revealed a type A AAD with the entry located in the anterior wall of the aortic arch, an enlarged ascending aorta (48 mm), and pericardial effusion (Fig. 1A). Aortic dissection spread widely, from the ascending aorta to the left common iliac artery and the brachiocephalic artery, which caused malperfusion, including occlusions of the celiac trunk (Fig. 1B, solid arrow) and bilateral common iliac artery (Fig. 1C, broken arrow). The blood supply of the superior mesenteric artery originated from both the true and false lumens without stenosis (Fig. 2A, solid arrow), and that of the inferior mesenteric artery originated from the true lumen. Laboratory tests showed metabolic acidosis with an elevated plasma lactate of 49 mg/dL. We diagnosed type A AAD with peripheral malperfusion that required emergency surgery.

We performed a total arch replacement through a median sternotomy. Cardiopulmonary bypass was established with an arterial cannula in the cardiac apex and a two-stage venous cannula through the right atrial appendage because the malperfusion included the iliac artery and the brachiocephalic artery. The total arch replacement, using the elephant trunk technique with a four-branched artificial vascular graft (Triplex® 26 mm, Terumo Corporation, Tokyo, Japan) was executed under moderate hypothermic circulatory arrest at 28°C with selective antegrade cerebral perfusion. Then, dissected wall of aortic root was fixed with BioGlue® (CryoLife Inc., NW Kennesaw, GA, USA), and the replacement of the ascending aorta was performed. After uneventful weaning from cardiopulmonary bypass, we performed an exploratory laparotomy to identify visceral ischemia because of the occlusion of the celiac trunk and the elevated plasma lactate. On exploratory laparotomy, intestinal ischemia was not visually detected. Moreover, limb

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ischemia had immediately improved, with palpable arteries of the lower limbs. Therefore, we did not see a need for additional surgical intervention.

After the operation, the plasma lactate level dramatically decreased to within the normal range by the next day, and the metabolic acidosis improved. However, on postoperative day 12, the patient developed paralytic ileus with abdominal pain, watery diarrhea (no melena), and sepsis. CT angiography revealed a newly narrowed superior mesenteric artery secondary to a thrombosed false lumen (Fig. 2B, solid arrow) as well as persistent occlusion of the celiac trunk. We diagnosed intestinal ischemia caused by the newly developed superior mesenteric artery stenosis. The plasma lactate level remained normal, so after improving the septic state, we performed percutaneous stent implantation of the celiac trunk (8 mm × 17 mm Express® LD stent, Boston Scientific Corporate, Marlborough, MA, USA) and superior mesenteric artery (10 mm × 40 mm Cordis S.M.A.R.T.® Control® Self-Expanding Nitinol Stent, Johnson & Johnson, New Brunswick, NJ, USA) through the right common femoral artery and the left brachial artery on postoperative day 20. We simultaneously executed thoracic endovascular aortic repair via the right common femoral artery, using a Zenith TX2® endovascular graft (ZTG-2P-32-140-PF and ESBE-26-80-T-JP, Cook Medical Incorporated, Bloomington, IN, USA) to close the entry site in the descending aorta and secure the true lumen. We matched the size of the true lumen because the elephant trunk was slightly collapsed and the diameter of descending aorta gradually increased. During the procedure, intravascular ultrasound was helpful to identify the true lumen, celiac artery, and superior mesenteric artery. After endovascular treatment, the paralytic ileus immediately improved. Postoperative CT angiography showed good patency of the celiac trunk and superior mesenteric artery (Fig. 2C, solid arrow, and Fig. 3, broken arrow). As of 1 year postoperatively, the patient has been doing well without any symptoms derived from occlusion of the aortic branches.

Discussion

Mesenteric malperfusion in type A AAD usually occurs simultaneously with onset of the AAD. Delayed intestinal ischemia after the central aortic repair is very rare. This patient developed intestinal ischemia caused by a newly narrowed superior mesenteric artery secondary to a thrombosed false lumen with persistent occlusion of the celiac trunk on postoperative day 12 following central aortic repair for type A AAD with peripheral malperfusion. In type A AAD, central aortic repair improves visceral ischemia in some patients but not in others. Recently Shiya and colleagues reported that, in type A AAD with branch-type malperfusion and progressive dissection into the visceral arteries, central aortic repair had minimal effect on the visceral ischemia.2) Our patient also had branch-type malperfusion of the celiac trunk and superior mesenteric artery.
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Delayed intestinal ischemia of aortic dissection was associated with high mortality in such cases. Shiiya and colleagues recommend that the choice of surgical versus percutaneous intervention in cases with visceral malperfusion should be made based on the perfusion pattern of the visceral arteries. For example, a central aortic operation or fenestration should be selected for aortic-type malperfusion (dynamic obstruction) defined by occlusion of the vessel origin by a dissection flap within the aortic lumen. On the other hand, stenting or bypass grafting should be selected for branch-type malperfusion (static obstruction) defined as an occluded or narrowed visceral arteries by directly progressive dissection into the visceral arteries.

Conclusion

We obtained good results with endovascular treatment, which included stent implantation and stent grafting, in our case of delayed intestinal ischemia due to an occluded or narrowed true lumen secondary to a thrombosed false lumen, a branch-type malperfusion. Although some patients with intestinal necrosis may require resection via laparotomy, endovascular treatment may be useful for mesenteric malperfusion that accompanies aortic dissection.

Disclosure Statement

The authors have no conflicts of interest to declare.

References