

Successfully superior mesenteric artery stenting in operated type A aortic dissection complicated with delayed mesenteric malperfusion

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Abstract

Here, we describe a case of a 61-year-old male patient with acute type A aortic dissection involving the ascending aorta, aortic arch, descending aorta, and the abdominal aorta down to the iliac bifurcation with evidence of left common iliac artery occlusion. Computed tomography angiography revealed progressive dissection into the superior mesenteric artery and left renal artery with no clinical signs of mesenteric ischemia. Emergent ascending aortic reconstruction of the dissected aorta relieves the leg ischemia. On a postoperative day 9, the evolution was complicated by massive right hemothorax. Although the patient was hemodynamically stable after obtaining hemostasis, the patient developed paralytic ileus with a high elevated lactate level. Visceral malperfusion was not detected by exploratory laparotomy. Emergency abdominal aortic angiography revealed superior mesenteric artery intermittent occlusion, successfully treated by stenting implantation.

Keywords

Mesenteric malperfusion, acute type A aortic dissection, superior mesenteric artery stenting

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Introduction

Acute type A aortic dissection (AAD) is a potentially life-threatening condition; the incidence is three to four cases per 100,000 patients per year.¹ AAD complicated with malperfusion syndrome is defined as the loss of blood supply to a vital organ caused by loss of branch arterial perfusion secondary to the dissection.² Traditionally, dissections involving the ascending aorta and aortic arch have been managed entirely by surgery. This management has changed over the past years due to improvements in adjunctive endovascular techniques with the introduction of branched endograft, especially for the treatment of visceral malperfusion. Visceral malperfusion caused by AAD occurring in 3.7% of patients, but it is associated with a huge hospital mortality rate of over 60%.³ Superior mesenteric artery (SMA) branch vessel compromise by dissection with bowel malperfusion is a life-threatening complication of type AAD. Sometimes, the diagnosis of acute mesenteric ischemia in patients with AAD can be challenging because, at presentation, abdominal pain

is a nonspecific symptom. Patients may be pain-free, and often, mesenteric malperfusion may be associated with pain determined by an additional malperfused vascular bed. The pathophysiologic mechanism of branch vessel affected by dissection dictates endovascular treatment options in AAD complicated with visceral malperfusion. In static obstruction, the dissection flap enters the branch vessel with the propagation of the false lumen, and a self-expanding stent deployed in the true lumen is a preferred approach. In dynamic obstruction, the dissection plane spares the branch origin, impeding flow to the branch vessel, and aortic fenestration combined with a self-expanding stent deployed in the true lumen is the therapeutic option of choice.⁴ The

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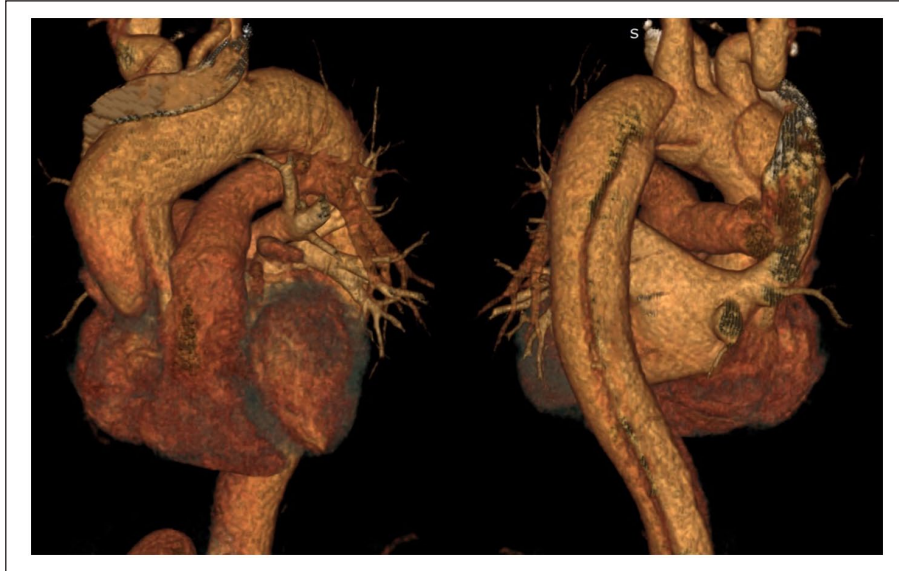


Figure 1. Preoperative reconstruction of contrast-enhanced angio-CT 3D showing the ascending aorta, aortic arch, and descending aorta dissection.

interdisciplinary treatment of mesenteric ischemia improves survival if carried out in time.⁵ Earlier detection and prompt restoration of mesenteric blood flow are the only pathways that will significantly improve the outcome to reduce the overall mortality associated with AAD and mesenteric malperfusion syndrome.⁶

Case history

A 61-year-old male patient was admitted to our clinic with intense posterior thoracic pain with sudden onset and a lipothymic episode. From the patient's medical history, we mention significant cardiovascular risk factors: systemic hypertension, obesity, dyslipidemia, aneurysm of the ascending aorta, 2 weeks after remitted SARS-CoV-2 infection. On admission, the patient was stable with a systemic blood pressure of 160/90 mmHg, pulse rate 61 bpm, oxygen saturation levels 98%, and at physical examination, no left femoral pulse with intermittent pain without other clinical signs of visceral or peripheral ischemia. The electrocardiogram (ECG) showed normal sinus rhythm with 60 bpm, without signs of myocardial ischemia. Transthoracic echocardiography (TTE) found a left ventricular ejection fraction of 50%, a calcified aortic tricuspid valve with a mild aortic valve insufficiency, with the following two-dimensional (2D) diameters: valsalva sinuses = 42 mm, ascending aorta = 44 mm, an intimal flap on the ascending aorta starts at the level of the right coronary artery ostium and also a pericardial effusion of 19 mm anterior to the right ventricle. Doppler ultrasound assessment of supra-aortic and femoral vessels revealed normal supra-aortic vessels without any evidence of dissection and the extension of the dissection flap at the level of the left common femoral artery and left superficial

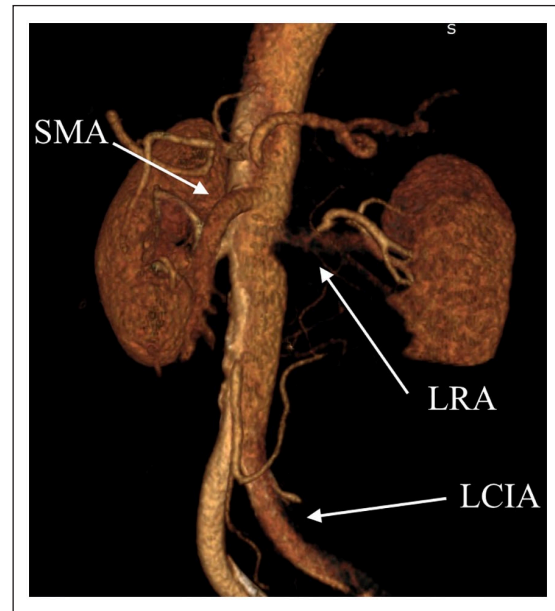


Figure 2. Preoperative reconstruction of contrast-enhanced angio-CT 3D showing LCIA occlusion, SMA dissection, and LRA occlusion.

SMA: superior mesenteric artery; LCIA: left common iliac artery; LRA: left renal artery.

femoral artery. Three-dimensional (3D) computed tomography angiography (CTA) (Figure 1) revealed the presence of dissection flap starting on the ascending aorta, aortic arch, and descending aorta extended to the left iliac artery, with occlusion of the left common iliac artery (LCIA), the dissection flap was also extended on the SMA and left renal artery (LRA) (Figure 2). Laboratory evaluation was notable

creatinine 2.09 mg/dL. Based on clinical and paraclinical findings, the patient was diagnosed with AAD, and he was transferred to the operating room for surgical treatment. Besides the patient's standard monitoring for cardiovascular surgery, we used specific monitoring like near-infrared spectroscopy (NIRS) for reflecting cerebral oxygenation of the frontal cortex to detect any potential cerebral malperfusion during circulatory arrest. The surgery was performed by median sternotomy, and the right axillary artery was dissected and isolated through a right subclavicular incision. Cardiopulmonary bypass was established between the right axillary artery and the right atrium. The ascending aorta was cross-clamped. After transversally opening ascending aorta, myocardial protection was achieved by directly infused a single dose of Custodiol cardioplegia. We removed the entire dissected ascending aorta and hemiaortic arch on the circulatory arrest when the patient's rectal temperature reached 28°C, under antegrade selective cerebral protection. Valve leaflets and Valsalva sinuses were considered to be conservable. We performed the ascending aorta and the proximal aortic arch replacement with a No. 30 INTERGARD Woven Vascular Graft (Figure 3). After uneventful weaning from cardiopulmonary bypass, peripheral pulses were present on both inferior limbs distally. The initial postoperative evolution was uneventful. On the ninth postoperative day, the patient's general status deteriorated, Hb decreased by 3 g from 10 to 7 mg/dL, and X-ray revealed full opacity on the left hemithorax. The patient was transferred to the operating room (OR), where hemostasis control was achieved. A rapid increase of seric lactic acid values was noted, and the patient's hemodynamic status began to deteriorate. Therefore, an emergency exploratory laparotomy was performed, which revealed no intestinal modifications consistent for acute mesenteric ischemia. After a multidisciplinary team consensus, we decided that the most appropriate treatment option for the patient was the percutaneous catheterization attempt of the SMA. The right common femoral artery was accessed, and a 6-French vascular sheath was placed. A 4.0 JR guide catheter and DG pigtail catheter were advanced into the abdominal aorta under fluoroscopic guidance. The pressure

in the abdominal aorta was measured in order to check the position in the true lumen. The contrast agent was injected via the pigtail catheter, and it revealed a dissection flap on the first portion of SMA (Figure 4) with blood flow limitation and a dissection flap with occlusion on the left renal artery. The decision for stent implantation was made. Systemic 5000 IU of unfractionated heparin was given. We used a Herculink Elite® stent (Abbott Vascular, Santa Clara, CA, USA) 7.0 × 15 mm, 135 mm (stent available in our clinic current use for renal artery stenting). The stent was carefully implanted at the ostium of the SMA, with minimal aortic protrusion (Figure 5). Proximal landing zones were expanded with a stent graft balloon catheter to improve the results. Blood flow improvement is maintained in the SMA, with minimal persistent dissection flap in the distal segments with minimal blood flow restriction. Angio-seal vascular closure device for the right femoral artery was placed with excellent hemostasis. We did not consider it was necessary to stenting the left renal artery. After the procedure, the patient's evolution was favorable; the plasma lactate level decreased to within the normal range by the end of the next day, with

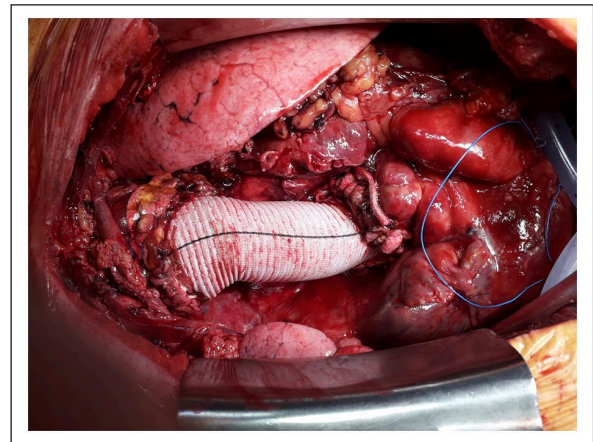


Figure 3. Intraoperative view of complete replacement of the ascending aorta and aortic hemiaortic arch with No. 30 INTERGARD Woven Vascular Graft.

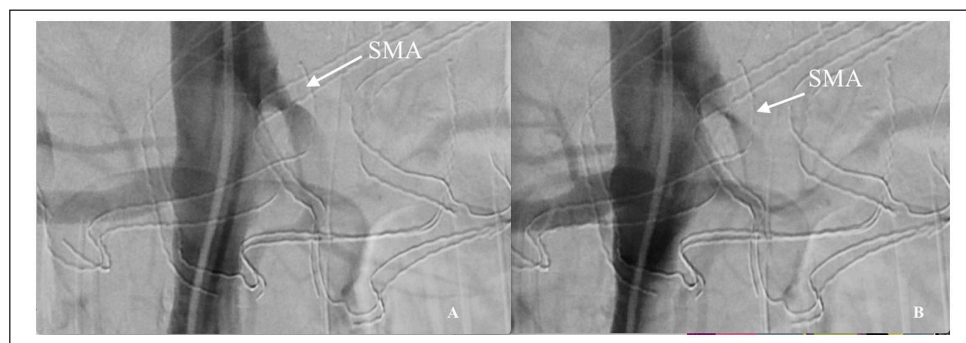


Figure 4. Angiography with evidence of the SMA obstruction by dissection fold. SMA: superior mesenteric artery.

complete remission of abdominal distension and pain, and the patient was discharged from the hospital on the 20 days after SMA stent placement. Follow-up computed tomography (CT) examination at 2 months showed the correct position of the stent and patency of the SMA (Figure 6).

Discussion

AAD is a life-threatening emergency that requires emergent surgery, and it can be complicated by visceral malperfusion in 16%–33% of cases and by peripheral vascular ischemic symptoms in approximately 30% of patients.^{7,8} Malperfusion syndrome can affect any vascular bed being the most challenging associated with a three- to four-fold increase in



Figure 5. Postprocedural angiography of the SMA after stent placement stabilizes the dissection fold and improved peripheral blood flow.

mortality in AADs.⁹ In visceral malperfusion syndrome cases, particularly involving the SMA, central aortic repair improves visceral ischemia in some patients but not in others. Repair of ascending aorta followed by open or endovascular mesenteric revascularization has resulted in a 38%–75% mortality.^{3,10} However, the patients may be pain-free in up to 40% of cases.¹¹ Consequently, diagnosis is frequently made too late to save the patient.¹² Percutaneous management of mesenteric complications in patients with AAD includes balloon fenestration, aortic true lumen stent placement, branch vessel stent placement, or a combination of the last two.¹³ The patient's treatment approach must be uniquely formulated based on clinical signs and symptoms, the entire aortic anatomy, and the flap dissection's hemodynamics. The AAD approach requires emergent surgical repair of the ascending aorta before undergoing the endovascular procedure in patients with AAD with SMA dissected but with absent visceral malperfusion. Surgeons should consider correcting mesenteric malperfusion before undertaking open aortic repair in patients by stenting the SMA first, followed by the central repair or simultaneous repair, especially those with gross bowel necrosis at laparotomy, or serum lactate ≥ 6 mmol/L.^{14–16}

Emergent ascending aortic reconstruction of the dissected aorta performed by us, relieves in our case, the peripheral ischemic symptoms, with palpable distal arteries of both lower limbs (Morisaki reported a similar case).¹⁷ Shiiya and colleagues reported that in AAD with visceral malperfusion and progressive dissection into the SMA, the central aortic repair had minimal effect on the visceral ischemia.^{18,19} The occurrence of mesenteric ischemia on a ninth day coincided in our case with a massive shock bleeding and was a complex and challenging problem. Although hemostasis was

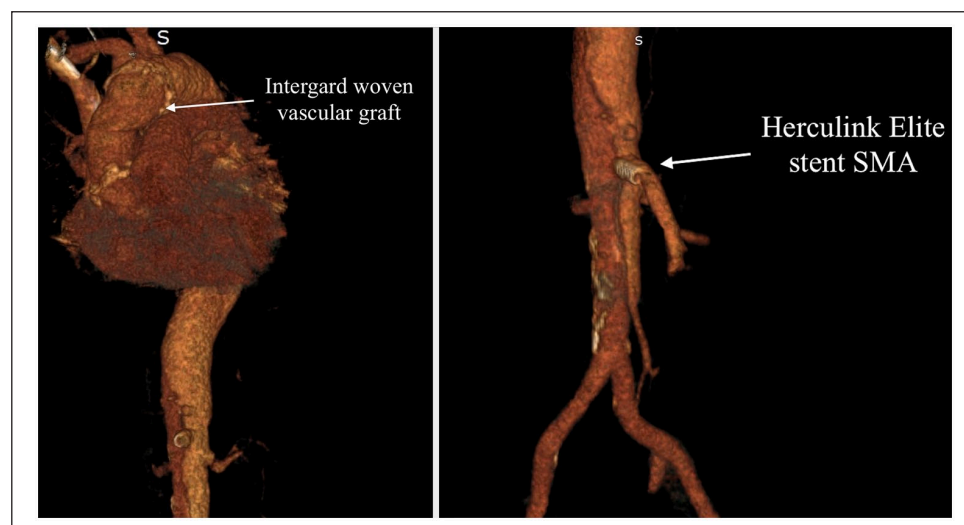


Figure 6. Postoperative reconstruction of contrast-enhanced angio-CT 3D at 2 months follow-up showing the INTERGARD Woven Vascular Graft in the ascending aorta, the correct position of the stent and patency of the SMA as well as the restoration of normal blood flow in the LCIA.

SMA: superior mesenteric artery; LCIA: left common iliac artery.

immediately done, increased lactate raised suspicion of mesenteric ischemia. An AAD can cause mesenteric ischemia by two mechanisms: first, by occluded or narrowed SMA arteries by directly progressive dissection into the vessel, and second, by occlusion of the vessel origin by a dissection flap within the aortic lumen.¹⁷ In our case, the hemorrhagic shock unstable hemodynamics of the dissection fold from SMA precipitating mesenteric ischemia and causing the increased lactate level. Emergent exploratory laparotomy revealed intestinal ischemia was not visually detected, permitting successful management of mesenteric ischemia by stenting the SMA. Plasma lactate level dramatically decreased within the normal range by the end of the next day, and the metabolic acidosis improved. Some authors recommend drug-eluting stent (DES) if vessel diameter is below 4–4.5 mm;²⁰ in our case, the SMA diameter was 7 mm. Complications associated with SMA endovascular treatment include thrombosis, dissection progression, and embolism that affect distal visceral vessels with the risk of acute mesenteric ischemia and death. Due to the high restenosis rate, all patients should be re-evaluated regularly at months 3, 6, and 12 and then every year, preferably by Doppler ultrasonography. However, the gold standard in the visualization of the visceral arteries is still mesenteric angiography.

Conclusion

Successful management of an AAD complicated with bowel malperfusion and delayed progression of SMA dissection can be obtained by central emergency operation, followed by endovascular stenting of the SMA at the first suspicion of mesenteric ischemia.

Declaration of conflicting interests

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Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

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Informed consent

Written informed consent was obtained from the patient for their anonymized information to be published in this article.

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