



# Experience in curing refractory gastrointestinal bleeding due to type A aortic dissection combined with mesenteric artery malperfusion: a case report

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**Abstract:** Aortic dissection (AD) is a cardiovascular emergency that seriously endangers human health. It has acute onset, dangerous condition and many complications. The mortality without treatment is very high, and the mortality within 24 hours is 25%. AD combined with mesenteric artery malperfusion has an incidence of only 5%, but a hospital mortality rate of up to 33–100%. Mesenteric artery malperfusion increases the mortality of acute AD by 3–4 times. Even after complete revascularization, ischemia/reperfusion injury still leads to frequent postoperative deaths. In this paper, we describe the case of a 60-year-old man with type A aortic dissection and mesenteric artery malperfusion who developed refractory gastrointestinal bleeding postoperatively. He was cured after conservative, interventional, and surgical hemostasis, finally recovered and discharged. This case provides a certain reference value for clinical treatment of such diseases. Aortic dissection combined with mesenteric artery malperfusion is a significant surgical challenge. Ischemia/reperfusion injury may still occur after thoracotomy, even when the blood supply is normalized. Immediate surgery is recommended for preventing death from acute AD, but the strategy should be modified according to the specific symptoms and ischemic severity. In addition, interventional/surgical treatment should be performed more actively in patients with refractory gastrointestinal bleeding after cardiac surgery and a poor response to conservative treatment.

**Keywords:** Aortic dissection; mesenteric artery malperfusion; gastrointestinal bleeding; case report

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## Introduction

Aortic dissection (AD) combined with mesenteric artery malperfusion has an incidence of only 5%, but a hospital mortality rate of up to 33–100%. Mesenteric artery insufficiency increases the mortality of acute AD by 3–4 times. Even after complete revascularization, ischemia/reperfusion injury still leads to frequent postoperative deaths. In this paper, we report our experience in successfully treating a case of refractory gastrointestinal bleeding induced by type A AD combined with mesenteric artery malperfusion. We present the following case in

accordance with the CARE reporting checklist (available at <https://dx.doi.org/10.21037/apm-21-2173>).

## Case presentation

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written

consent is available for review by the editorial office of this journal.

A 60-year-old man presented to the emergency department with sudden chest and back pain for over 3 hours. Physical examinations showed 95/52 mmHg blood pressure, 24 beats/min respiration, 95 beats/min heart rate, and 90% pulse oxygen saturation (SpO<sub>2</sub>). The patient presented with drowsiness, a cold right lower limb, and an unpalpable dorsalis pedis artery. No abdominal tenderness was examined. Computed tomography (CT) images of the great vessels revealed the existence of type A AD and massive hemopericardium (*Figure 1*). Laboratory examinations revealed the following: alanine aminotransferase (ALT), 899 U/L; aspartate aminotransferase (AST), 2,884 U/L; total bilirubin (TBil), 39.3 μmol/L; direct bilirubin (DBil), 15.1 μmol/L; indirect bilirubin (I-Bil), 24.9 μmol/L; and creatinine (Cre), 239 μmol/L. The patient was diagnosed with type A AD, cardiac tamponade, multi-organ hypoperfusion, and severe (grade 3) hypertension. The patient underwent ascending aorta replacement + aortic root repair + elephant trunk stent placement by deep hypothermic cardiopulmonary bypass (CPB). During the CPB (181 min), aortic cross-clamp time was performed for 100 min, and deep hypothermic circulatory arrest (DHCA) for 20 min. Antegrade selective cerebral perfusion (ASCP) was maintained for 20 min.

The patient fell into a moderate coma postoperatively. CT images of the head on the 3<sup>rd</sup> day (Day 3) did not reveal intracranial organic changes, suggesting hypoxic-ischemic encephalopathy (HIE). The patient also developed acute renal insufficiency and hepatic encephalopathy, and was symptomatically treated with hemodialysis and medications to protect the liver and lower bilirubin. His consciousness became clear on Day 5, and the tracheal cannula was extubated on Day 7 with the assistance of noninvasive ventilation. Four days later (Day 11), the patient was re-intubated with mechanical ventilation for hypercapnia. Debridement and suturing were performed for a chest incision infection examined on Day 13.

The tracheal cannula was re-extubated on Day 15. Melena was delivered on Day 17 (2,500–3,000 mL/day), and hemoglobin (Hb) was reduced to 62 g/dL. Symptomatic and supportive treatments, including gastric acid secretion inhibition, hemostasis, fluid infusion, as well as supplementation of fibrinogens, coagulation factors, and hemoglobin, did not achieve a satisfactory outcome. Gastroscopy findings showed multiple active ulcers in the descending part of the duodenal bulb, and extensive

bleeding in the intestinal wall below the descending part (*Figure 2*). The patient was received with conservative treatment, considering his wide range of lesions and coagulation dysfunction. However, the efficacy of 8-day conservative treatment was unsatisfactory, and the patient was then treated with interventional embolization for hemostasis on Day 25.

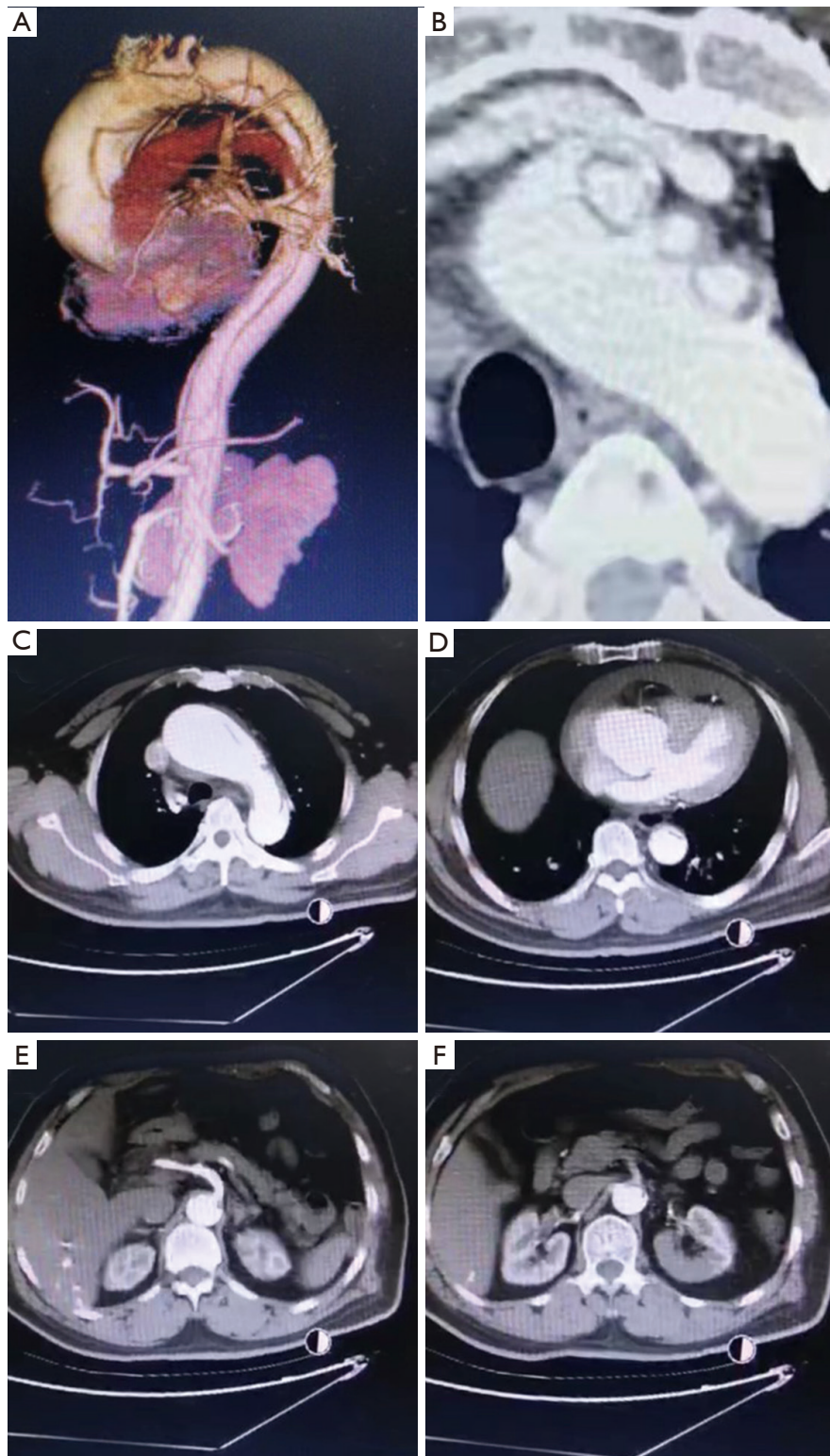
During the first round of interventional embolization, the bleeding branch of the gastroduodenal artery was identified and clotted by coil embolization (*Figure 3A–3C*). However, melena appeared on the next day (Day 26) (3,000 mL/day). We performed the second round of coil embolization, due to the bleed from the branches of the superior mesenteric artery that communicated with the arteria pancreaticoduodenalis (*Figure 3D–3G*). Ten days later (Day 36), melena appeared in a large quantity, and emergency bedside gastroscopy showed the bleeding of the descending duodenal bulb. Hemostasis under gastroscope failed. As a result, the patient was treated with partial gastrectomy + Roux-en-Y (Billroth II reconstruction) + surgical suture ligation of the bleeding duodenal ulcer. Subsequently, the patient recovered and was discharged. CT images of the aorta during follow-up did not show internal leakage, and true-lumen blood supply of the celiac trunk and the mesenteric artery were observed (*Figure 4*). The whole course of the disease is briefly depicted in *Figure 5*.

## Discussion

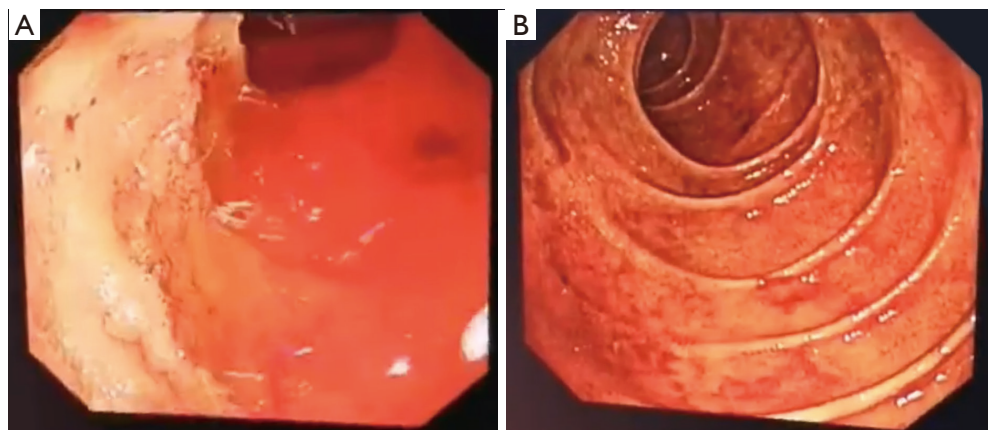
Among the diverse but interrelated reasons for gastrointestinal complications after cardiac surgery, visceral hypoperfusion is considered to be the main one. Despite the low incidence of gastrointestinal complications after cardiac surgery (0.5–5.5%), they may lead to a mortality rate of as high as 87% (1).

Considering its high rates of hospital mortality (33–100%), gastrointestinal bleeding after AD caused by mesenteric artery malperfusion should be particularly concerning. Mesenteric artery malperfusion markedly increases the mortality of acute AD by 3–4 times (2,3), and ischemia/reperfusion injury also leads to the high postoperative mortality, even after complete revascularization (4), due to ischemia-reperfusion injury, intestinal flora displacement will occur, which will lead to serious postoperative infection, acidosis, and even septic shock, resulting in multiple organ failure and eventually death.

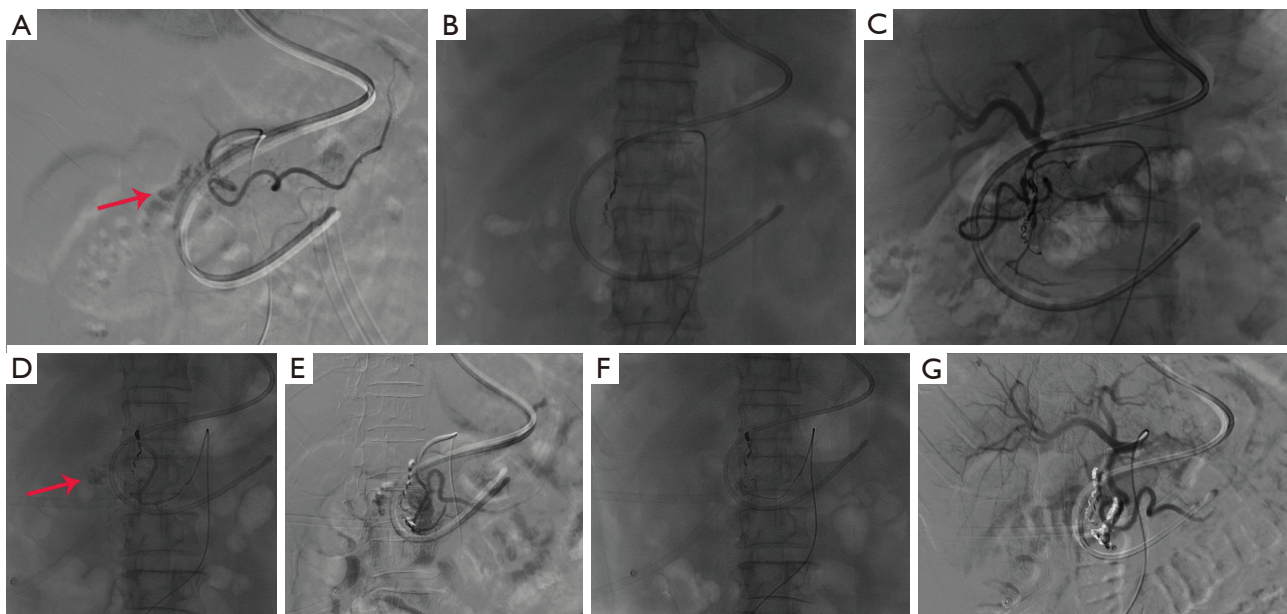
Currently, there is a lack of specific diagnostic indicators and means for dissection complicated with mesenteric



**Figure 1** Preoperative vascular CT. (A) Involvement into the ascending aorta, arch, and descending aorta; (B) involvement of the anonymous artery, left common carotid artery, and left subclavian artery; (C) involvement into the arch; (D) medium amount of pericardial effusion; (E) blood supply of the celiac trunk by a small-sized true-lumen; (F) blood supply of the superior mesenteric artery by a small-sized true-lumen.



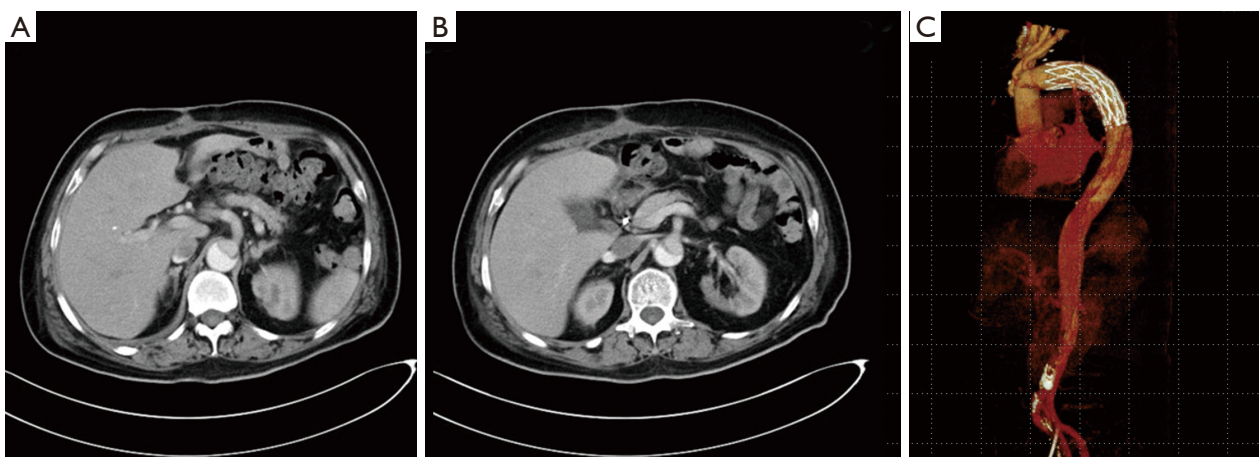
**Figure 2** Results of gastroscopy. (A) Multiple ulcers and bleeding in the descending part of the duodenal bulb; (B) extensive bleeding below the descending part of the duodenal bulb.



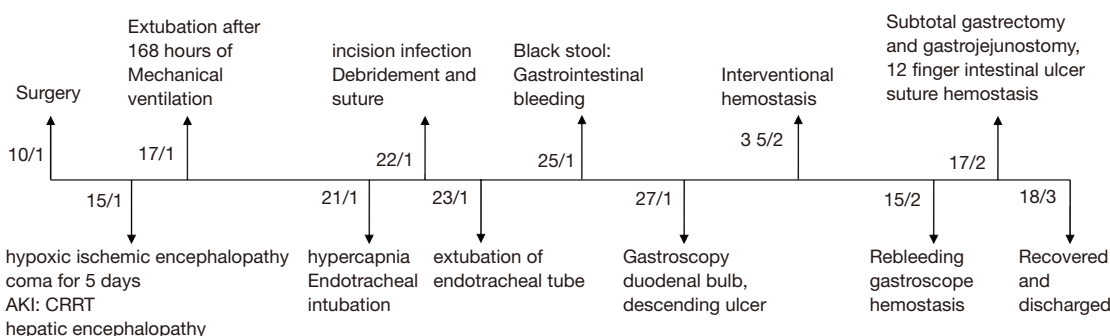
**Figure 3** Findings of 2 interventional operations. (A-C) First interventional embolization: (A) bleeding branch of the gastroduodenal artery is indicated by the red arrow in the angiography; (B) coil embolization of the bleeding artery; (C) no bleeding after coil embolization. (D-G) Second interventional embolization: (D) bleeding from the branches of the superior mesenteric artery communicating with the arteria pancreaticoduodenalis is indicated by the red arrow in the angiography; (E) embolization of the upper segment of the bleeding artery; (F) embolization of the lower segment of the bleeding artery; (G) no bleeding post-embolization.

hypoperfusion before and after operation. The diagnosis is often based on clinical manifestations and auxiliary examinations. The clinical manifestations are mainly abdominal symptoms, such as abdominal pain, abdominal distention, muscle tension and bloody stool. It is reported that lactic acid and blood ammonia can be used as indicators of intestinal ischemia, as well as imaging examinations, such

as CT and color Doppler ultrasound. If diagnosis is needed, Angiography may be required to determine (5,6). And the therapeutic strategies for mesenteric artery malperfusion are controversial (4,7). Some scholars insist that open surgery for AD should be the prior, as opening the true-lumen collapse can significantly relieve celiac artery ischemia. Interventional therapy can be performed thereafter if celiac



**Figure 4** Postoperative reexamination. (A) True-lumen blood supply of the celiac trunk; (B) true-lumen blood supply of the superior mesenteric artery; (C) the three-dimensional image.



**Figure 5** Flow chart of the disease course.

artery ischemia cannot be relieved. Also, aortic rupture due to delayed surgery would cause more serious consequences. However, other opinions have proposed that the ischemic celiac artery should be treated initially by fenestration or stent implantation, which can significantly reduce mortality due to mesenteric artery malperfusion (by 50%). At present, a general consensus has been adopted that immediate surgery can effectively prevent death in patients with acute AD combined with mesenteric artery malperfusion, while patients with relatively stable AD (no ruptures or tamponades) combined with mesenteric artery malperfusion benefit more from a phased treatment of pre-recovery of organ implantation, followed by conventional thoracotomy. Aortic repair performed initially leads to a high mortality in patients with severe visceral malperfusion.

In the present case, thoracotomy for AD was performed first considering the appearance of cardiac tamponade. The

improved postoperative liver function and lack of clinical symptoms of abdominal distension indicated relieved abdominal organ malperfusion after thoracotomy. However, the patient had melena 15 days later, and gastrointestinal bleeding was considered due to reperfusion injury. Gastroscopy findings showed pale gastric mucosa, linear mucosal necrosis, multiple active ulcers, and extensive bleeding in the descending part of the duodenal bulb. The interventional embolization identified the bleeding branch of the gastroduodenal artery, which was clotted by coil embolization, while malperfusion of the celiac trunk and its branches were not detected. The patient had melena only 1 day after interventional embolization, due to bleeding from the branches of the superior mesenteric artery that communicated with the arteria pancreaticoduodenalis in the emergency gastroscopy. Interventional embolization of the upper and lower segments of bleeding arteries successfully

stopped the gastrointestinal bleeding. However, a massive gastrointestinal hemorrhage occurred 10 days after the second round of embolization. Considering the poor therapeutic efficacy, we performed a partial gastrectomy + Roux-en-Y (Billroth II reconstruction) + surgical suture ligation of the bleeding duodenal ulcer. The patient subsequently recovered and was discharged 1 month later.

## Conclusions

AD combined with mesenteric artery malperfusion is a significant surgical challenge. Ischemia/reperfusion injury may still occur after thoracotomy, even after the blood supply is normalized. Immediate surgery is recommended for preventing death from acute AD, but the strategy should be modified according to the specific symptoms and ischemic severity. In addition, interventional/surgical treatment should be performed more actively in patients with refractory gastrointestinal bleeding after cardiac surgery and a poor response to conservative treatment.

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## Footnote

*Reporting Checklist:* The authors have completed the CARE reporting checklist. Available at <https://dx.doi.org/10.21037/apm-21-2173>

*Conflicts of Interest:* All authors have completed the ICMJE uniform disclosure form (available at <https://dx.doi.org/10.21037/apm-21-2173>). The authors have no conflicts of interest to declare.

*Ethical Statement:* The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

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