

Case Report

Critical Malperfusion Caused by Central Aortic Repair for Acute Aortic Dissection: A Case Report

Yuki Mochida, MD, Hiroyuki Morinaga, MD, Yusuke Shimizu, MD, Takaaki Sakamoto, MD, Yasuhiko Miyakuni, MD, Yasuhiko Kaita, MD, Takehiko Tarui, MD, PhD, and Yoshihiro Yamaguchi, MD, PhD

We encountered a case of hepatic malperfusion resulting from central repair for Stanford type A acute aortic dissection (AAD). A 78-year-old woman had AAD, for which ascending aortic repair was performed. Hepatic malperfusion developed 3 days postoperatively. The superior mesenteric and celiac arteries were occluded by a false lumen (FL). We believed that the surgery caused a change in the blood flow in FL. Percutaneous transluminal angioplasty and stenting of the superior mesenteric artery were performed, and the patient's condition improved. Thus, intervention for the branched artery should be performed prior to central repair, depending on the type of malperfusion.

Keywords: malperfusion, central aortic repair, acute aortic dissection

Introduction

The mortality rates associated with Stanford type A acute aortic dissection (AAD) have remarkably decreased in recent years owing to advances in surgical and anesthetic techniques.¹⁾ However, organ malperfusion complications due to AAD are still associated with high mortality rates, and there are no definite management strategies.²⁻⁴⁾

Central aortic repair as an intervention may disrupt blood flow owing to a false lumen (FL) and cause organ ischemia. We encountered a case in which the interruption

of the superior mesenteric artery (SMA) and celiac artery (CEA) by central aortic repair caused malperfusion 3 days postoperatively.

Case Report

A 78-year-old woman experienced sudden onset of stomach ache and vomiting and developed AAD. She was taken to another hospital by an ambulance. The diagnosis was confirmed using enhanced computed tomography (CT) at the hospital. Although CT revealed that the proximal CEA and SMA diverged from the FL of the aorta (Fig. 1), it was considered that there was no malperfusion because of enhanced CEA and SMA. Hence, central aortic repair via ascending aortic replacement was performed. On admission at this hospital, blood tests revealed aspartate transaminase (AST) levels of 115 U/l and alanine transaminase (ALT) levels of 80 U/l. Thereafter, there were no signs of ischemia, and the general condition of the patient improved. CEA and SMA were monitored as a preventive measure, but no additional treatment was administered. Tracheal extubation was performed on postoperative day 2. However, abdominal discomfort reappeared on postoperative day 3. AST and ALT levels were 7,089 and

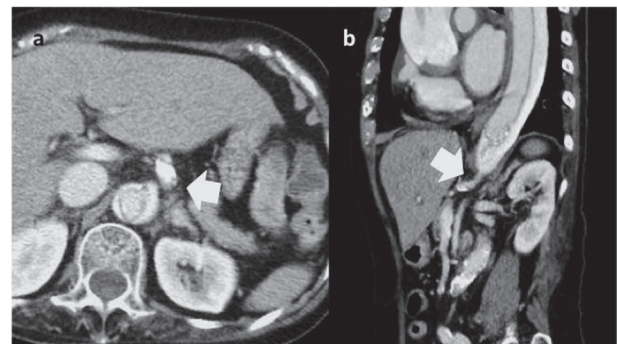


Fig. 1 (a) Preoperative computed tomography (CT) showing acute aortic dissection and the branch of celiac artery (CEA) from false lumen (FL) in the aorta (arrow). (b) Sagittal view of CT showing the existing blood flow of FL and CEA (arrow).

Department of Trauma and Critical Care Medicine, Kyorin University School of Medicine, Mitaka, Tokyo, Japan

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Corresponding author: Yuki Mochida, MD. Department of Trauma and Critical Care Medicine, Kyorin University School of Medicine, 6-20-2 Shinkawa, Mitaka, Tokyo 181-8611, Japan
Tel: +81-422-47-5511, Fax: +81-422-42-4866
E-mail: yukimochida99@yahoo.co.jp


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Fig. 2 (a) Computed tomography (CT) showing no enhancement of false lumen in the aorta and ischemia of the liver. (b) Sagittal view of CT showing obstruction of superior mesenteric artery (arrow) and celiac artery (arrowhead).

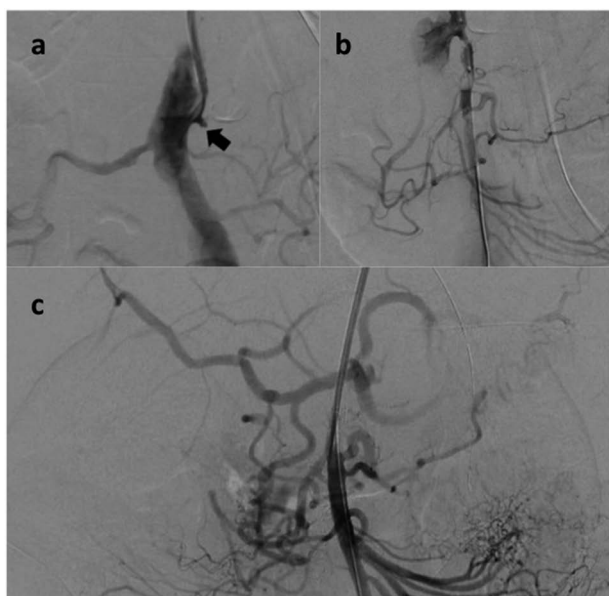


Fig. 3 (a) Angiography showing defect of superior mesenteric artery (SMA) (arrow). (b) Imaging after percutaneous transluminal angioplasty (PTA) of the proximal site of SMA. (c) SMA imaging showing branching of celiac artery through inferior pancreaticoduodenal artery.

4,060 U/I, respectively. Lactic acid levels also markedly increased, and enhanced CT showed obstruction of the proximal SMA and CEA and poor contrast enhancement of the liver (Fig. 2). Thus, the patient was transferred to our critical care center.

We suspected that hepatic malperfusion may have resulted from the obstruction of the proximal part of CEA and SMA. However, it was difficult to secure vascular access due to obesity. Therefore, the brachial artery was secured, and a 9-Fr vascular sheath was inserted. SMA arteriography was performed using a guiding catheter (Parent Plus60, Medikit, Tokyo, Japan). SMA arteriogram demonstrated proximal occlusion of SMA near the ostium of this artery (Fig. 3a). To restore the true lumen (TL) of SMA, a 0.016-inch guide wire (Asahi Intecc, Nagoya, Japan)

was inserted to the distal site and percutaneous transluminal angioplasty (PTA) was performed using a 5-mm balloon catheter (Boston Scientific, Marlborough, MA, USA) (Fig. 3b). Subsequent SMA arteriography showed improved blood flow, and CEA branches were perfused via the inferior pancreaticoduodenal artery (IPDA). Then, a 5 mm × 19 mm monorail premounted stent (Express, Boston Scientific) was placed in the stenosed SMA (Fig. 3c). Balloon dilation and stent placement were successfully performed without any complications.

Because CEA could not be approached, it was not unblocked. The suspected diagnosis was mesenteric malperfusion; therefore, an emergency laparotomy was performed. Ascites (light yellow) was confirmed during laparotomy. The color of the small intestine was normal, although few dark red portions, suggesting ischemia, were confirmed on the other side of the mesentery. Moreover, the ischemia was irreversible. There was no ischemia in the colon. The color of the liver changed to dark red, which indicated a change in the ischemic condition.

Chronic hepatic disease was also suspected as the hepatic surface had blunt margins. The emergency laparotomy was terminated without additional treatment for the ischemic findings to reduce the operative time and because CEA was being imaged through angiography.

Although angiography was performed again the day after the surgery due to persistent, high lactic acid levels, no blood flow issues were observed in SMA. Optimum blood flow in the portal vein was also confirmed using ultrasound, and hence, no additional treatment was performed, except conservative management.

Transfusion with fresh frozen plasma was continued to compensate for the liver function. AST and ALT levels increased to 17,010 and 5,470 U/I but reduced to 3,032 and 1,497 U/I after 3 days, respectively, indicating improvement. In addition, 8 days later, both the levels decreased to less than 100 U/I. Prothrombin time and international normalized ratio increased to a maximum of 6.04.

Long-term respiratory management was necessary due to massive transfusion. The patient was extubated on day 17 and was transferred to the previous hospital on day 38.

This report has been approved by our Institutional Review Board (approval number: 1278).

Discussion

Malperfusion is reported as a complication associated with Stanford type A AAD in 3.7% cases.³⁾ The mortality rate associated with malperfusion due to AAD is higher than that associated with AAD without malperfusion owing to the lack of a defined treatment strategy for malperfusion.¹⁻⁴⁾

There are two principal strategies that can be initially

performed: central aortic repair and revascularization of the artery.⁴⁻⁷⁾

Malperfusion may result from two types: aortic type and branch type.⁸⁾ The aortic type is caused by the retraction of TL by FL in the aorta. Therefore, the expansion of TL in the aorta is required. On the contrary, in branch-type malperfusion, the dissection progresses to the branch and the enlarged FL compresses TL in the branch artery. In the latter case, revascularization of the branch artery is necessary.

In the present case, although preoperative enhanced CT at the previous hospital revealed branch-type malperfusion of the proximal CEA and SMA, both were patent, and therefore, conservative management was chosen.

Many researchers advocate that prompt intervention should be performed for aortic branch obstruction after the diagnosis of malperfusion.^{5,8)}

If re-entry (the tear that causes blood to re-enter) is noted, then the compression of TL may become mild and blood flow from FL can continue.⁸⁾

On the contrary, in the absence of re-entry, if the entry site is excised by central aortic repair, then there would be no blood flow and thrombus in FL. It should be noted that central aortic repair may cause ischemia in case of no re-entry. In branch-type malperfusion, with the vessels branching from FL, it is presumed that the presence or absence of re-entry affects the course of the surgery. We believe that postoperative anesthesia management may mask the symptoms of ischemia. Therefore, interventions should be considered based on the clinical findings before central repair is performed. Even if central repair is chosen first, early, enhanced CT evaluation of malperfusion after the surgery should be performed.

SMA ischemia has a high mortality rate, and its most common complication is intestinal malperfusion.^{2,3)} In the present case, the relatively early intervention may have prevented intestinal malperfusion. Therefore, intervention against SMA ischemia should be prioritized, and laparotomy should be performed to confirm the presence or absence of intestinal malperfusion.⁵⁾ Single obstruction of the proximal site of CEA is usually not related to hepatic ischemia because SMA has a pathway to CEA through superior and inferior pancreaticoduodenal arteries.⁹⁾ Thus, intervention for CEA was not necessary in this case.

There are no reports stating that the occlusion of SMA and CEA causes liver ischemia.²⁾ Hepatic malperfusion is rare because the liver has a dual blood supply: from the hepatic artery and portal vein as well as collateral blood supply.¹⁰⁾ However, the occlusion of SMA and CEA can cause hepatic malperfusion due to reduced flow in the hepatic artery; in such a case, treatment is required to increase blood flow to the liver.

Conclusion

Central aortic repair can cause malperfusion as it alters the blood flow due to FL. Moreover, branch-type malperfusion without re-entry may require intervention after surgery. The patient should be carefully monitored, and the onset of malperfusion should not be missed.

Disclosure Statement

There are no conflict of interest to declare.

Additional Remarks

Informed consent was obtained from the patient for submission of this case report.

Author Contributions

Study conception: YM, HM

Data collection: YM, HM, YS

Analysis: YM, YK

Investigation: YM, YM, HM

Writing: YM

Critical review and revision: all authors

Final approval of the article: all authors

Accountability for all aspects of the work: all authors

References

- 1) Pape LA, Awais M, Woznicki EM, et al. Presentation, diagnosis, and outcomes of acute aortic dissection: 17-year trends from the international registry of acute aortic dissection. *J Am Coll Cardiol* 2015; **66**: 350-8.
- 2) Yagdi T, Atay Y, Engin C, et al. Impact of organ malperfusion on mortality and morbidity in acute type A aortic dissections. *J Card Surg* 2006; **21**: 363-9.
- 3) Di Eusanio M, Trimarchi S, Patel HJ, et al. Clinical presentation, management, and short-term outcome of patients with type A acute dissection complicated by mesenteric malperfusion: observations from the International Registry of Acute Aortic Dissection. *J Thorac Cardiovasc Surg* 2013; **145**: 385-90.e1.
- 4) Patel HJ, Williams DM, Dasika NL, et al. Operative delay for peripheral malperfusion syndrome in acute type A aortic dissection: a long-term analysis. *J Thorac Cardiovasc Surg* 2008; **135**: 1288-95; discussion, 1295-6.
- 5) Uchida K, Karube N, Kasama K, et al. Early reperfusion strategy improves the outcomes of surgery for type A acute aortic dissection with malperfusion. *J Thorac Cardiovasc Surg* 2018; **156**: 483-9.
- 6) Chiu P, Tsou S, Goldstone AB, et al. Immediate operation for acute type A aortic dissection complicated by visceral or peripheral malperfusion. *J Thorac Cardiovasc Surg* 2018; **156**: 18-24.e3.
- 7) Girardi LN, Krieger KH, Lee LY, et al. Management strate-

- gies for type A dissection complicated by peripheral vascular malperfusion. *Ann Thorac Surg* 2004; **77**: 1309-14; discussion, 1314.
- 8) Orihashi K. Mesenteric ischemia in acute aortic dissection. *Surg Today* 2016; **46**: 509-16.
- 9) Vaddineni SK, Taylor SM, Patterson MA, et al. Outcome after celiac artery coverage during endovascular thoracic aortic aneurysm repair: preliminary results. *J Vasc Surg* 2007; **45**: 467-71.
- 10) Carrol R. Infarction of the human liver. *J Clin Pathol* 1963; **16**: 133-6.