Delayed intestinal stenosis of nonocclusive mesenteric ischemia after autologous blood collection: A case report

Takahiro Arima *, Takashi Omura, Koji Hattori, Ken Kawamoto, Yuji Koba
Higashiyamato Hospital, Department of Surgery, 1-13-12 Nangai, Higashiyamato, Tokyo, 207-0014, Japan

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A B S T R A C T
INTRODUCTION: Nonocclusive mesenteric ischemia (NOMI) has been reported to be associated with high mortality. Early diagnosis of NOMI and prompt restoration of the intestinal blood flow is necessary in order to achieve a favorable outcome.

PRESENTATION OF CASE: We present the case of a patient who developed NOMI after autologous blood collection and was treated by selective infusion of the superior mesenteric artery with papaverine, intestinal decompression using a long intestinal tube, the administration of antibiotics, and fluid replacement. Although this non-surgical management was successful, 8 weeks after the ischemic event, segmental bowel resection was necessary because of repeated intestinal obstruction caused by bowel stricture.

DISCUSSION: Autologous blood collection might be a risk factor of NOMI. In addition, the possibility of delayed intestinal stenosis remains, even if bowel necrosis and surgical resection were avoided with non-surgical management including vasodilator therapy.

CONCLUSION: Rapid diagnosis and intervention are essential to minimize intestinal ischemia.

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1. Introduction

Nonocclusive mesenteric ischemia (NOMI) is relatively rare but often fatal. The overall incidence of autopsy-verified fatal NOMI was 2.0/100,000 person years [1]. It accounts for about 20–30% of all the cases of acute mesenteric ischemia, with a mortality rate of about 50% [2–4]. NOMI is caused by the reduction of blood flow in the mesenteric circulation. The risk factors include heart failure, hemodialysis, septic shock, recent cardiopulmonary bypass, and several medications such as furosemide, digitalis, and vasoconstrictive agents. Rapid infusion of vasodilators via the superior mesenteric artery (SMA) is the only decisive intervention. We report a case of NOMI after autologous blood collection, developing delayed intestinal stenosis following vasodilator therapy.

1.1. Presentation of case

A 76-year-old man was admitted to the hospital with fever and right lower quadrant pain after autologous blood collection, as preparation for nephrectomy scheduled in two weeks to resect a renal cancer. He had temporal hypotension (67/33 mmHg) about 40 min after the collection of autologous blood (400 ml). Although his vital signs were stabilized with fluid replacement in approximately 30 min, abdominal pain remained severe, requiring analgesic agents several times. Laboratory data showed no specific changes on admission and the inflammatory markers on day 3 were the following: WBC 14,020/μl and CRP 35.02 mg/dl.

Contrast-enhanced multi-detector computed tomography (CT) on day 3 demonstrated thickening of the small bowel wall, poor staining of the wall, and intramural emphysema (Fig. 1). In addition, angiography revealed diffuse spasms in the branches of the proximal SMA (Fig. 2A). With a diagnosis of NOMI, we started continuous infusion of papaverine (2.5 mg/h) with a bolus of prostaglandin E1 (5 μg) through the intra-arterial catheter placed in the SMA and continued until day 8. Angiography on day 6 demonstrated an improvement of the circulation of SMA (Fig. 2B). Decompression using a long intestinal tube was also continued from day 5 to day 14. Piperacillin/tazobactam (4.5 g) was administered three times per day from day 2–day 9. Non-surgical management was successful resulting in discharge of the patient on day 21.

However, the patient was readmitted because of intestinal obstruction on day 35. Conservative treatment was successful and he was discharged on day 43. The intestinal obstruction occurred again on day 49. During this time, conservative treatment including decompression by a long intestinal tube was unsuccessful. CT revealed a perforation of the small bowel associated with the intestinal obstruction. On day 56, an emergency operation (right hemicolectomy, and partial resection of 60 cm of the strictured and inflammatory distal ileum) was carried out.

According to pathological examination, two sites of stricture were found in the ileum, which were adhering to each other causing obstruction (Fig. 3). Moreover, in the stricture sites of the ileum, severe thickening was found with fibrosis in the sub-mucosal layer (Fig. 4A). An ulcer formation was also observed in the stricture.
site leading to perforation (Fig. 4B). Post-operatively, complications of gastric ulcer and liver abscess arose, and the hospital stay was extended to day 109.

2. Discussion

The following two points are the subject of this case report. First, autologous blood collection can be a risk factor of NOMI. Second, delayed intestinal stenosis after the recovery from NOMI may require surgical intervention in some cases.

Low cardiac output states have been reported to lead to NOMI developing [1–3,5]. Taking blood for autologous blood transfusion may cause volume depletion and low cardiac output, which is similar to the condition of removing water during hemodialysis. In fact, temporal hypotension was found while collecting autologous blood in this case. It is known that the temporal decreases in the SMA blood flow for only several hours can cause mesenteric vaso-
Due to the thickening of the wall, a severe stricture of the ileum was found in two sites which were adhering to each other causing bowel obstruction.

Ischemic damage to the bowel may vary in correlation with the duration and the severity of ischemia. When the blood flow is reduced to <50% of normal baseline, injuries may develop in the gastrointestinal tract [9]. Fibrosis was observed in the sub-mucosal layer on microscopy. This is a typical finding in the injured bowel associated with ischemia at the chronic stage [10]. Severe ischemia may cause transmural necrosis or gangrene, and in some cases it results in fibrosis and stricture, or persistence of the ulcers [10,11]. The healing process of the intestine needs a longer time in the case of severe damage [11]. In our case, the radiological intervention was performed about 48 h after the onset of symptoms and the intestinal obstruction occurred in about 5 weeks. If the intervention had been performed earlier, ischemic damage might have been less and delayed stricture might have been avoided. There is also one report regarding delayed intestinal stricture after NOMI in which the intestinal obstruction occurs approximately 8 weeks after the ischemic event [12]. Therefore, close follow-up is necessary to detect the possible development of intestinal obstruction caused by delayed intestinal stricture when non-surgical management is successful.

constriction persisting even after the recovery of blood flow [2,3]. Early recognition of NOMI occurring is very important to achieve a favorable outcome. Therefore, when examining a patient with abdominal pain during and after autologous blood collection, NOMI should be one of the differential diagnoses.

The treatment of NOMI consists of fluid resuscitation, bowel decompression, the administration of antibiotics for bacterial translocation, removal of inciting medications and correction of underlying causes of hypoperfusion. In 1977, Boley demonstrated that the selective injection of papaverine into the SMA could relieve mesenteric vasoconstriction [3]. In addition, several reports have suggested the benefit of vasodilator therapy [2,3,5–8] and one study concluded that selective mesenteric angiography with continuous vasodilator infusion was a simple, fast, and effective diagnostic and therapeutic tool to reduce the need for laparotomy [5]. Appropriate dosage levels for this therapy are not well established and in this case a lower dose than optimal was administered. However, the therapy was effective resulting in an improvement of the clinical condition and the SMA circulation. Randomized controlled studies are necessary to confirm the efficacy of endovascular treatment.
3. Conclusion

Autologous blood collection can be a contributing factor of NOMI. In addition, there is a risk of delayed intestinal stenosis associated with ischemia, even if bowel necrosis and surgical resection could be avoided with non-surgical management including vasodilator therapy. Rapid diagnosis and prompt intervention are essential to minimize intestinal ischemia and to reduce mortality and morbidity.

Conflicts of interest statement

All authors declare that they have no conflicts of interest.

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Ethics approval

In our institution, the need to obtain ethics approval for case reports is waived. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Guarantor

Not applicable.

Authors’ contributions

TA drafted the manuscript and assisted in all of the operative procedures described. TO is the chief of the department and contributed supportive information. KH, KK, and YK are members of the attending staff of the department who discussed the treatment. All of the authors approved publication of the manuscript.

References

[1] S. Acosta, M. Ogren, N.H. Sternby, D. Bergqvist, M.J. Björck, Fatal nonocclusive mesenteric ischemia: population-based incidence and risk factors, J. Intern. Med. 259 (2006) 305–313.
[2] M. Trompeter, T. Braza, T. Vestring, R. Reimer, Non-occlusive mesenteric ischemia: etiology, diagnosis, and interventional therapy, Eur. Radiol. 12 (2002) 1179–1187.
[3] S.J. Boley, S. Sprayregan, S.S. Siegelman, F.J. Veith, Initial results from an aggressive roentgenological and surgical approach to acute mesenteric ischemia, Surgery 82 (1977) 848–855.
[4] S.A. Käser, T.C. Müller, A. Guggemos, U. Nitsche, C. Späth, C.A. Maurer, K.P. Janssen, J. Kleeff, H. Friess, D. Wilhelm, F.G. Bader, Outcome after surgery for acute right-sided colonic ischemia without feasible vascular intervention: a single center experience of 58 patients over 6 years, BMC Surg. 15 (2015) 31.
[5] S. Klotz, T. Vestring, J. Rötker, C. Schmidt, H.H. Scheld, C. Schmid, Diagnosis and treatment of nonocclusive mesenteric ischemia after open heart surgery, Ann. Thorac. Surg. 72 (2001) 1583–1586.
[6] A. Mitsuyoshi, K. Ohbana, N. Shinkura, T. Ito, M. Zaima, Survival in nonocclusive mesenteric ischemia: early diagnosis by multidetector row computed tomography and early treatment with continuous intravenous high-dose prostaglandin E1, Ann. Surg. 246 (2007) 229–235.
[7] T. Kazui, M. Yamasaki, K. Abe, S. Watanabe, K. Kawazoe, Non-occlusive mesenteric ischemia: a potentially lethal complication after cardiovascular surgery: report of two cases, Ann. Thorac. Cardiovasc. Surg. 18 (2012) 56–60.
[8] P.L. Kozuch, I.J. Brandt, Review article: diagnosis and management of mesenteric ischemia with an emphasis on pharmacotherapy, Aliment. Pharmacol. Ther. 21 (2005) 201–215.
[9] J.J. Kolkin, P.B. Mensink, Non-occlusive mesenteric ischemia: a common disorder in gastroenterology and intensive care, Best Pract. Res. Clin. Gastroenterol. 17 (2003) 457–473.
[10] S. Mitsudo, I.J. Brandt, Pathology of intestinal ischemia, Surg. Clin. North Am. 72 (1992) 43–63.
[11] D.J. Glotzer, A.H. Villegas, S. Anekamaya, R.S. Shaw, Healing of the intestine in experimental bowel infarction, Ann. Surg. 155 (1962) 183–190.
[12] S. Maetzawa, M. Fujita, T. Sato, S. Kushimoto, Delayed intestinal stricture following non-resectional treatment for non-occlusive mesenteric ischemia associated with hepatic portal venous gas: a case report, BMC Surg. 15 (2015) 37.