Shimane J. Med. Sci., Vol.42 pp.31-36, 2025

Balancing Nutrition and Ischemia of Small Intestine: A Fatal Case of Non-Occlusive Mesenteric Ischemia Caused by Enteral Nutrition Following Esophagectomy

Takeshi MATSUBARA, Shunsuke KAJI, Hikota HAYASHI, Yoko SENAHA, Hiroki OKAMURA, Keisuke INOUE, Kazunari ISHITOBI, Takahito TANIURA, Kiyoe TAKAI, Tetsu YAMAMOTO, Masaaki HIDAKA

Department of Digestive and General surgery, Faculty of Medicine, Shimane University, Izumo, Shimane 693-8501, Japan

（Received January 14, 2024; Accepted January 21, 2024; Published online July 25, 2025）

Introduction: Non-occlusive mesenteric ischemia（NOMI）is a rare but potentially fatal complication of postoperative enteral nutrition, reported following surgeries for gastrointestinal cancers, including esophageal cancer. ***Case presentation:*** A 77-year-old male underwent thoracoscopic-assisted subtotal esophagectomy. Enteral nutrition was initiated gradually and initially well-tolerated. On postoperative day（POD）5, the patient developed reduced urine output, mild abdominal distension, and diarrhea, which temporarily improved. However, on POD 6, he experienced cardiopulmonary arrest. Imaging revealed portal vein and intestinal emphysema as NOMI. Emergency surgery revealed extensive intestinal necrosis due to vascular spasm. Despite the intervention, the patient succumbed to multiple organ failure on POD 7. ***Conclusion:*** NOMI seems to be exacerbated by high-osmolarity enteral nutrition and bacterial overgrowth. Typical early symptoms of NOMI did not exist, and diagnosis was challenging. This case highlights the need for careful monitoring during enteral nutrition in high-risk patients and emphasizes the importance of early detection and individualized management to prevent NOMI-related complications.

Keywords: NOMI, enteral feeding

INTRODUCTION

Non-occlusive mesenteric ischemia（NOMI）is a rare but potentially fatal postoperative complication that can arise during enteral nutrition following esophageal cancer surgery. Although early enteral nutrition is crucial for preserving intestinal integrity and reducing the risk of infectious complications, it can sometimes lead to intestinal ischemia.［1］［2］［3］The reported incidence of postoperative NOMI varies between 0.1% and 3.5%, depending on the underlying condition and type of surgical intervention.［4］Despite significant advancements in postoperative care, the exact mechanisms, risk factors, and effective preventive measures for NOMI remain poorly understood, posing challenges for clinical management.

This case highlights the delicate balance between the benefits and risks of early enteral nutrition and underscores the need for further research to optimize postoperative care strategies. Key areas of focus include the development of predictive biomarkers for early detection of NOMI, evaluating optimal enteral nutrition formulations and administration rates, and establishing standardized monitoring protocols to

Corresponding author: Takeshi MATSUBARA, M.D., Ph.D.
Department of Digestive and General Surgery, Faculty of Medicine, Shimane University, 89-1 Enya-cho, Izumo, Shimane 693-8501, Japan
Email: nanadai@med.shimane-u.ac.jp

identify and address ischemic events at their earliest stages.

CASE REPORT

A 77-year-old male with a history of hypertension and chronic obstructive pulmonary disease（COPD）was independent in daily life and had a performance status（PS）of 0 before surgery. He was diagnosed with early-stage esophageal cancer and underwent endoscopic submucosal dissection（ESD）over five years ago. One year before this admission, an enlarged lymph node in the abdominal cavity was detected, raising suspicion of recurrence. Following detailed evaluations, a thoracoscopic-assisted subtotal esophagectomy with two-field lymph node dissection was planned.（Figure 1, 2）Feeding jejunostomy was also created concurrently at 15 cm from the Treitz's ligament.

The surgery lasted approximately seven hours, with an estimated blood loss of 340 ml. The patient's vital signs remained stable throughout the procedure. Postoperatively, he was transferred to the intensive care unit（ICU）for circulatory support. His condition stabilized, and all medications were discontinued the following day.

On postoperative day（POD）5, the patient was transferred from the ICU to a general ward. Enteral nutrition was initiated by ELENTAL®（761 mOsm/L）on POD 2 at a rate of 10 mL/hour, increasing by 10 mL/hour per day using PEPTAMEN® AF（440 mOsm/L）, following a standardized protocol designed to minimize gastrointestinal stress while providing adequate nutritional support. This approach aligns with current guidelines recommending gradual escalation to assess patient tolerance and mitigate complications such as intestinal ischemia. The patient initially tolerated feeding well, with no immediate gastrointestinal complications observed.

On POD 5, the patient exhibited reduced urine output, mild abdominal distension, and diarrhea at midnight. These symptoms improved with medication, and his vital signs remained stable. However, early on the morning of POD 6, the patient developed impaired consciousness and suffered a cardiopulmonary arrest. Return of spontaneous circulation（ROSC）was achieved following prompt cardiopulmonary resuscitation. Display the blood test findings in chronological order.（Table 1）A CT scan revealed portal vein and intestinal emphysema, leading to a diagnosis of non-occlusive mesenteric ischemia（NOMI）.（Figure 3, 4）

Thus, an emergency operation was performed to resect necrotic intestinal segments nearly 300 cm. Extensive intestinal necrosis was observed, predominantly from jejunum to ileum. Although no significant obstruction of the mesenteric artery was detected, vascular spasm and blood flow insufficiency were evident, mainly distal to the site of the enterostomy as indicated by the preoperative CT im-

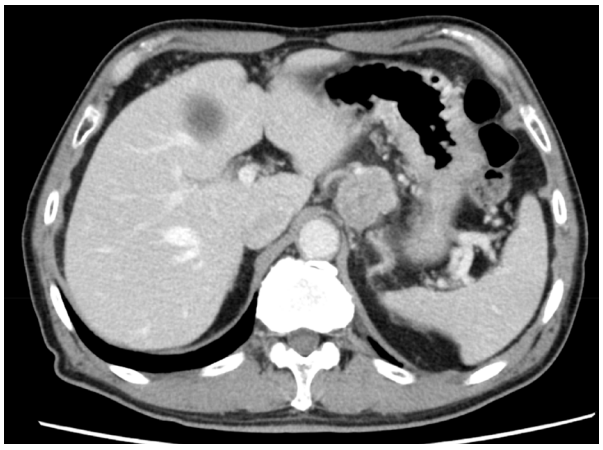

Figure 1. An enlarged lymph node was observed on the lesser curvature of the gastric cardia, and squamous cell carcinoma（SCC）was diagnosed by fine-needle aspiration（FNA）.

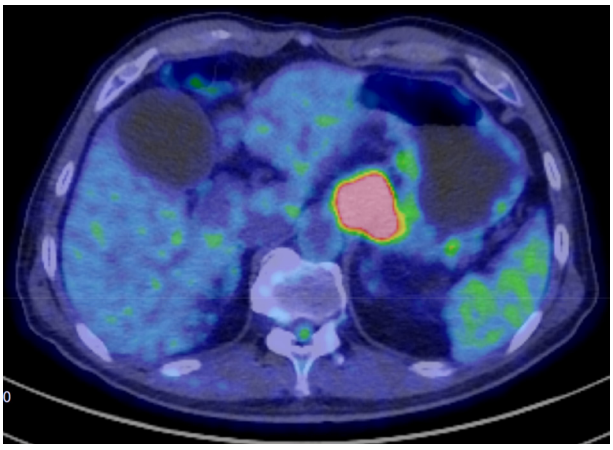

Figure 2. The enlarged lymph nodes showed high fluorodeoxyglucose（FDG）uptake.

Table 1. Blood Test Results During Follow-Up

	POD4	POD5	POD6（post-emergency）		
				Normal range	Unit
WBC	9.4	7.42	18.34	3.30 ～ 8.60	$x10^3/\mu L$
Neutrophils	83.3	81.6	83.8	40.0 ～ 75.0	%
RBC	3.74	3.97	4.51	4.35 ～ 5.55	$x10^6/\mu L$
Hb	11	11.8	13	13.7 ～ 16.8	g/dL
Ht	34	36.9	43.2	40.7 ～ 50.1	%
Plt	115	141	208	158 ～ 348	$x10^3/\mu L$
Alb	2.9	3	2.8	4.1 ～ 5.1	g/dL
T-Bil	1.2	1.2	1.6	0.4 ～ 1.5	mg/dL
AST	25	48	88	13 ～ 30	U/L
ALT	19	41	95	10 ～ 42	U/L
LDH	183	205	370	124 ～ 222	U/L
CK	230	102	90	59 ～ 248	U/L
Amy	24	32	167	44 ～ 132	U/L
BUN	13.8	12.1	41.1	8.0 ～ 20.0	mg/dL
Crea	0.59	0.56	2.09	0.65 ～ 1.07	mg/dL
Na	137	137	144	138 ～ 145	mmol/L
K	4.1	4	4.6	3.6 ～ 4.8	mmol/L
Cl	107	104	101	101 ～ 108	mmol/L
Ca	7.9	8.3	9	8.8 ～ 10.1	mg/dL
CRP	11.27	10.67	7.08	<0.14	mg/dL
PT			45.9	70 ～ 130	%
APTT			26.3	24.0 ～ 34.0	sec
INR			1.59	0.90 ～ 1.10	
Fib			471	200 ～ 400	mg/dL
D-dimer			21.6	≦1.0	μg/mL
Blood Gas Analysis					
pH			6.864	7.35 ～ 7.45	
$PaCO_2$			55.9	35.0 ～ 48.0	mmHg
PaO_2			96.1	83.0 ～ 108.0	mmHg
HCO_3^-			10.1	21.0 ～ 28.0	mmol/L
BE			-23.4	-3 ～ 3	mmol/L
Lactate			18	0.5 ～ 1.5	mmol/L

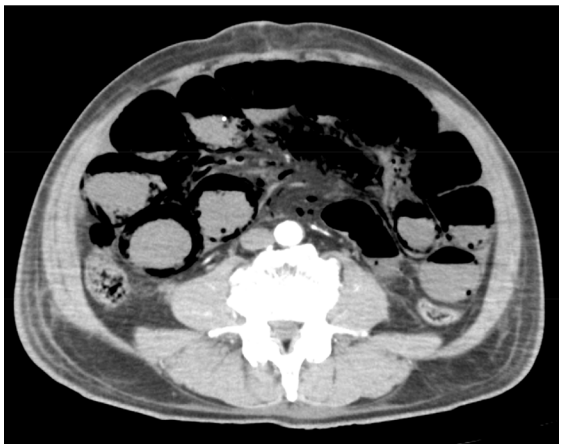

Figure 3. The small intestine exhibited widespread decreased blood flow and thinning of the intestinal wall.

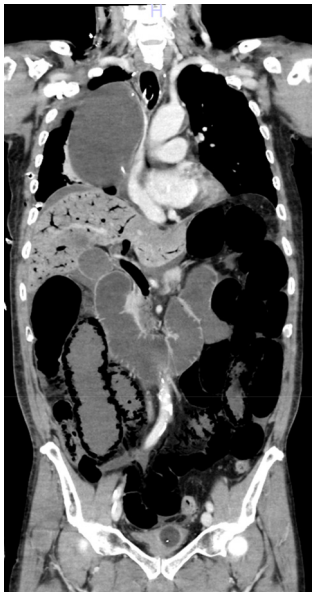

Figure 4. Blood flow to the intestinal tract is preserved up to the vicinity of the Treitz ligament, but decreased blood flow is observed in the intestinal wall distal to the enterostomy insertion site. In addition, intrahepatic portal venous gas is present, although ascites is limited to the liver surface.

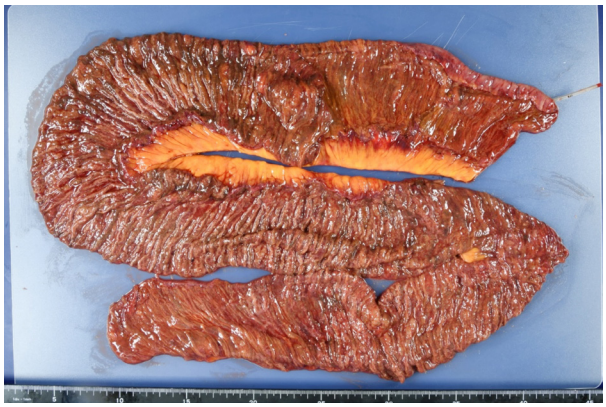

Figure 5. Macroscopically, the mucosa appeared black in color with edema from the enterostomy insertion site onward.

ages. (Figure 3, 5) Intraoperative findings revealed pale, ischemic intestinal segments with a poor capillary refill, confirming compromised mesenteric perfusion. Despite surgical intervention, the patient's condition deteriorated, progressing to multiple organ failure. He passed away on POD 7 in the ICU.

DISCUSSION

NOMI is characterized by mesenteric artery spasms and reduced intestinal blood flow, leading to ischemia. High-osmolarity enteral nutrition solutions are believed to exacerbate mucosal stress and compromise blood flow. [5] [6] [7] [8] Other contributing factors include bacterial overgrowth, mucosal barrier damage, vascular endothelial injury, and the production of inflammatory cytokines, which collectively aggravate ischemia and complicate its clinical course. [9] [10]

In this case, the rapid progression of intestinal ischemia following the initiation of enteral nutrition underscores the need for heightened vigilance during the early postoperative period. Early nonspecific symptoms, such as mild abdominal discomfort, distension, and decreased urine output, were not adequately recognized or addressed, as they were attributed to the expected effects of enteral nutrition and the normal postoperative course. [11] These signs may have been precursors to the catastrophic events that ensued. Enhanced monitoring protocols and earlier interventions might have altered the outcome.

Despite its rarity, NOMI remains a life-threatening complication of early postoperative enteral feeding, with an incidence of feeding-related bowel necrosis ranging from 1.2% to 1.7%. [8] The onset of NOMI has been reported to occur 1 to 17 days (median 6 days) after the start of enteral nutrition after esophagectomy [12], and post-operative NOMI is reported to occur around 1 to 12 days. [11] [13] The fatality rate for NOMI is extremely high, reaching 41-100% in the reported range. [11]

NOMI's pathogenesis and clinical characteristics are poorly understood due to its rarity. Previous studies have proposed the following mechanisms for feeding-related NOMI: [11]

1: The absorption of intraluminal nutrients may inadvertently increase the energy demands of metabolically stressed enterocytes, promoting splanchnic blood flow. In the context of hypoperfusion or inadequate resuscitation, this increased energy demand, coupled with a diminished oxygen supply, exacerbates mesenteric ischemia.
2: Enteral feeding in the setting of ileus can facilitate bacterial overgrowth, accumulating intraluminal gas and toxins that compromise mucosal integrity.

Nutritional management also warrants closer scrutiny. In this case, the hyperosmolar nutritional solution may have contributed to intestinal ischemia by imposing additional stress on the intestinal mucosa. Tailored nutritional plans with lower initial infusion rates, gradual escalation, and close monitoring are essential, particularly for elderly patients or those at high risk of circulatory failure. These recommendations are consistent with guidelines from the European Society for Clinical Nutrition and Metabolism (ESPEN), emphasizing individualized approaches to minimize complications and improve patient outcomes. [14]

In our clinical experience with NOMI, enteral feeding was initiated at a low rate and incrementally increased while carefully monitoring for feeding intolerance. Nevertheless, abdominal distension occurred unexpectedly without prior warning signs. Additionally, diarrhea suggested a potential gastrointestinal motility disorder, and we should either reduce the infusion rate or temporarily suspend feeding. Early diagnosis is critical to prevent bowel necrosis and perforation, but this remains challeng-

ing for two reasons: [15]

1: Mild gastrointestinal symptoms, such as abdominal pain and distension, are relatively common in patients receiving enteral nutrition post-esophagectomy, potentially masking early signs of NOMI.
2: Vital signs often remain within normal limits during the early stages of NOMI, as observed in our cases.

The lack of effective multidisciplinary collaboration also emerged as a critical issue. Improved communication among physicians, nurses, and dietitians could have facilitated earlier recognition of subtle symptoms such as decreased urine output and abdominal discomfort. A team-based approach is essential to optimize outcomes in high-risk cases.

CONCLUSION

Non-occlusive mesenteric ischemia (NOMI) is a rare but life-threatening complication of postoperative enteral nutrition, particularly challenging to diagnose early due to its nonspecific symptoms and the stability of vital signs in its initial stages. This case underscores the critical importance of gradual feeding escalation, tailored nutritional protocols, and close monitoring of gastrointestinal and hemodynamic parameters to minimize risk. Multidisciplinary collaboration among physicians, nurses, and dietitians is pivotal in identifying subtle signs early and implementing timely interventions. To improve outcomes, further research is needed to establish predictive biomarkers, optimize feeding strategies, and standardize monitoring protocols to prevent and manage NOMI.

Author contribution

The contributions of the authors are as follows:

Author TM: Managed the patient case, collected and analyzed the case data, and drafted the initial manuscript. Author SK, HH, YS, TO, KI, TT, KT, TY: Conducted the literature review and verified the academic accuracy of the case report. Author MH: Reviewed and revised the manuscript, prepared the figures and tables, and approved the final draft. All authors reviewed and approved the final manuscript and consented to its publication.

Conflict of interest

The authors declare no conflicts of interest related to this study.

REFERENCES

1) Moore EE, Jones TN. Benefits of immediate jejunostomy feeding after major abdominal trauma – a prospective, randomized study. *J Trauma*. 1986;26:874-81. doi: 10.1097/00005373-198610000-00003.
2) Deitch EA, Winterton J, Li M, Berg R. The gut as a portal of entry for bacteremia. Role of protein malnutrition. *Ann Surg*. 1987;205:681-92. doi: 10.1097/00000658-198706000-00010.
3) Bell SJ, Borlase BC. Feeding jejunostomy for post operative nutritional support. *JPEN J Parenter Enteral Nutr*. 1992;16:395-6. doi: 10.1177/0148607192016004395.
4) Melis M, Fichera A, Ferguson MK. Bowel necrosis associated with early jejunal tube feeding. A complication of postoperative enteral nutrition. *Arch Surg*. 2006;141:701-4. doi: 10.1001/archsurg.141.7.701.
5) Gaddy MC, Max MH, Schwab CW, Kauder D. Small bowel ischemia: a consequence of feeding jejunostomy? *South Med J*. 1986;79:180-2. doi: 10.1097/00007611-198602000-00011.
6) Cha CJ, Randall HT. Osmolality of liquid and defined formula diets: the effect of hydrolysis by pancreatic enzymes. *JPEN J Parenter Enteral Nutr*. 1981;5:7-10. doi: 10.1177/014860718100500107.
7) Worthington KJ, Cuschieri A. Activation and release of proteolytic kinin-forming enzymes from rat jejunal loops perfused with hyperosmolar glucose solutions. *Gut*. 1977;18:279-83. doi: 10.1136/gut.18.4.279.
8) Schunn CD, Daly JM. Small bowel necrosis associated with postoperative jejunal tube feeding. *J Am Coll Surg*. 1995;180:410-6.
9) Baldwin BA, Zagoren AJ, Rose N. Bacterial contamination of continuously infused enteral alimentation with needle catheter jejunostomy--clinical implications. *JPEN J Parenter Enteral Nutr*. 1984;8:30-3. doi: 10.1177/014860718400800130.
10) Smith-Choban P, Max MH. Feeding jejunos-

tomy: a small bowel stress test? *Am J Surg*. 1988;155:112-7. doi: 10.1016/s0002-9610(88)80267-8.

11) Marvin RG, McKinley BA, McQuiggan M, *et al*. Nonocclusive bowel necrosis occurring in critically ill trauma patients receiving enteral nutrition manifests no reliable clinical signs for early detection. *Am J Surg*. 2000;179:7-12. doi: 10.1016/s0002-9610(99)00261-5.

12) Kurita D, Fujita T, Horikiri Y, *et al*. Non-occlusive mesenteric ischemia associated with enteral feeding after esophagectomy for esophageal cancer: report of two cases and review of the literature. *Surg Case Rep*. 2019;5(1):36. doi:10.1186/s40792-019-0580-2.

13) Wani ML, Ahangar AG, Lone GN, *et al*. Feeding jejunostomy: does the benefit outweigh the risk (a retrospective study from a single centre). *Int J Surg*. 2010;8:387-390. doi: 10.1016/j.ijsu.2010.05.009.

14) Sethuraman SA, Dhar VK, Habib DA, *et al*. Tube feed necrosis after major gastrointestinal oncologic surgery: institutional lessons and a review of the literature. *J Gastrointest Surg*. 2017;21:2075-82. doi: 10.1007/s11605-017-3593-9.

15) Kurita D, Fujita T, Horikiri Y, Sato T, Fujiwara H, Daiko H. Non-occlusive mesenteric ischemia associated with enteral feeding after esophagectomy for esophageal cancer: report of two cases and review of the literature. *Surg Case Rep*. 2019;5:36. doi: 10.1186/s40792-019-0580-2.