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Nonocclusive mesenteric ischemia associated with postoperative jejunal tube feeding: Indicators for clinical management

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Abstract

Postoperative nutrition via a jejunal tube after major abdominal surgery is usually well tolerated. However, some patients develop nonocclusive mesenteric ischemia (NOMI). This morbid complication has a grave prognosis with a mortality rate of 41% to 100%. Early symptoms are nonspecific, and no treatment guideline is available. We reviewed cases of NOMI at our institution and cases described in the literature to identify factors that impact the clinical course. Among five patients, three had no necrosis and one had segmental necrosis and perforation. These patients recovered with limited resection and decompression of the bowel and abdominal compartment. In one patient with extended bowel necrosis at the time of re-laparotomy, NOMI progressed and the patient died of multiple organ failure. The extent of small bowel necrosis at the time of re-laparotomy is a relevant prognostic factor. Therefore, early diagnosis and treatment of NOMI can improve the prognosis. Clinical symptoms of abdominal distension, cramps and high reflux plus paraclinical signs of leukocytosis, hypotension and computed tomography findings of a distended small bowel with pneumatosis intestinalis and portal venous gas can help to establish the diagnosis. We herein introduce an algorithm for the diagnosis and management of NOMI associated with jejunal tube feeding.

Keywords

Postoperative tube feeding, mesenteric ischemia, pneumatosis intestinalis, bowel necrosis, diagnostic algorithm, treatment algorithm

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Introduction

Enteral feeding after major upper gastrointestinal procedures reduces catabolic stress and improves immunological function and wound healing because of stimulation of enterocyte growth, stabilization of the mucosal barrier and a decrease in bacterial translocation.[1–4] Septic complications occur less frequently than with total parenteral nutrition.[5] Enteral feeding is generally well tolerated in the early postoperative course. However, symptoms of intestinal dysfunction secondary to jejunal tube feeding frequently occur (up to 40% of cases) and include diarrhea, nausea, abdominal distension and cramping.[6] Discontinuation of tube feeding in most patients diminishes these symptoms.

Some patients' condition progresses to a syndrome of abdominal distension, hypotension and nonocclusive mesenteric ischemia (NOMI). Thompson[7] reported the first case of small bowel necrosis in a 20-year-old man after jejunal catheter feeding in 1983. The literature contains 43 recorded cases of NOMI following abdominal surgery. The reported incidence of this condition varies from 0.14% to 3.50% in retrospective studies.[8] The typical finding at laparotomy is small bowel necrosis starting distal to the feeding tube site without occlusion of the central mesenteric vessels. Bowel necrosis requires resection to various extents and, in some cases, repeated resections. The reported mortality rate ranges from 41% to 100%.[8]

Despite the reported high mortality rates, the extent of bowel necrosis and the prognosis differ in the published reports; this corresponds with the experience in our clinic. Because no guideline has been established for the management of tube feeding-associated NOMI, we reviewed our own cases and previously reported cases in the literature to identify factors that affect the clinical course. Our overall goal was to create an algorithm for the clinical management of NOMI associated with jejunal tube feeding.

Patients and methods

Patient 1

A 61-year-old man with gastroesophageal reflux disease and cachexia was diagnosed with advanced gastric cancer and underwent multivisceral resection with gastrectomy, D2 lymphadenectomy and left pancreatosplenectomy. He also underwent placement of a jejunostomy catheter (Flocare Jejunokath; Pfrimmer Nutricia GmbH, Erlangen, Germany). Tube feeding started on postoperative day (POD) 1 with Fresubin Energy Fibre (Fresenius Kabi Deutschland GmbH, Langenhagen, Germany) at a rate of 20 mL/h. On POD 3, the patient developed acute abdominal pain, distension and high reflux via the nasogastric tube. Laboratory examination showed leukocytosis and an elevated C-reactive-protein concentration but no metabolic acidosis. No hypotension was seen. An abdominal computed tomography (CT) scan showed paralytic ileus, pneumatosis intestinalis (PI) and portal venous gas (PVG) (Figure 1). Emergency re-laparotomy was performed, and ischemia of several small bowel loops without necrosis was found. There was no strangulation of the mesentery or occlusion of the visceral arteries. We decompressed the congested small bowel via jejunotomy; resection was not necessary. The postoperative period was uneventful, and the patient was discharged on POD 15.

Patient 2

A 74-year-old man developed Boerhaave's syndrome after prostatectomy. As the primary treatment, he underwent esophagectomy without anastomosis for a long esophageal perforation. As the secondary treatment, a gastric tube was formed for reconstruction and cervical anastomosis.

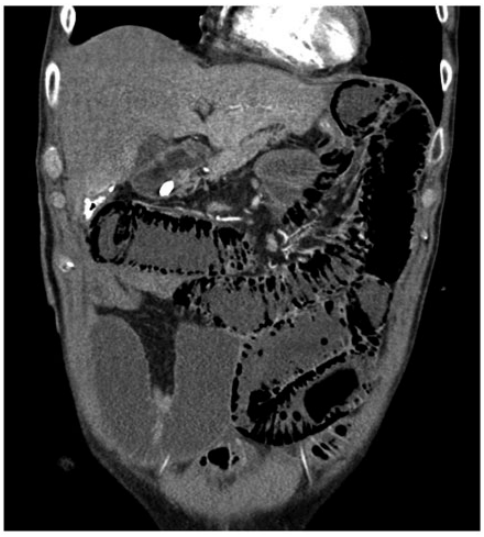

Figure 1. Computed tomography findings. Small bowel distension, pneumatosis intestinalis and portal venous gas are evident.

A jejunostomy catheter was then placed (Flocare Jejunokath; Pfrimmer Nutricia GmbH). Jejunostomy feeding started on POD 3 with Fresubin Energy Fibre (Fresenius Kabi Deutschland GmbH) at a rate of 40 mL/h, which was successively increased to 80 mL/h. Diagnostic gastroscopy was performed on POD 12 for investigation of asymptomatic leukocytosis, and an ischemic lesion was found in the gastric tube for esophageal reconstruction.

A CT scan showed anastomotic leakage and small bowel dilation with PI. During an emergency reoperation, the ischemic proximal gastric tube was resected and cervical esophagostomy was performed. Segmental NOMI of the small bowel without necrosis was also confirmed (Figure 2). We performed an enterotomy and decompression of the small bowel without resection. The patient recovered and was discharged on POD 31.

Patient 3

A 72-year-old woman underwent gastrectomy with D2 lymphadenectomy for gastric cancer. Enteral feeding via a nasojejunal tube (Freka Trelumina FR 16/9; Fresenius Kabi Deutschland GmbH) started on POD 1 with Biosorb (Pfrimmer Nutricia GmbH) at a rate of 20 mL/h. On POD 2, suspicious intra-abdominal drainage fluid was detected, and re-laparotomy was performed for repair of a possibly insufficient anastomosis. The only pathological finding was a lymphatic fistula after lymphadenectomy; there was no evidence of insufficient anastomosis. During tube feeding at a rate of 70 mL/h on POD 4, the patient developed painful abdominal cramps, progressive abdominal distension and hypotension. The laboratory examination results showed leukocytosis but no metabolic acidosis. An abdominal CT scan showed PI, PVG and small bowel distension. Emergency re-laparotomy was performed, and patchy ischemia along the full length of the small bowel without necrosis was found (Figure 3). The mesentery exhibited venous congestion. There was no evidence of intestinal strangulation or central mesenteric arterial occlusion. We decompressed the small bowel and released the abdominal compartment by placing Vicryl mesh (Johnson & Johnson Medical GmbH, Norderstedt, Germany) for closure of the abdomen. The patient recovered and was discharged from the hospital on POD 18.

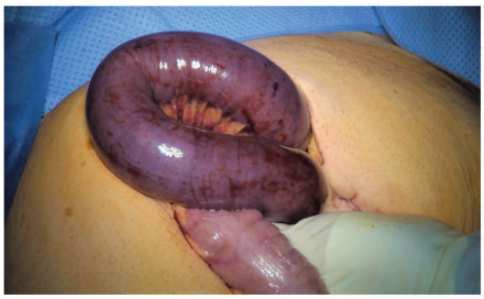

Figure 2. Segmental ischemia without necrosis.

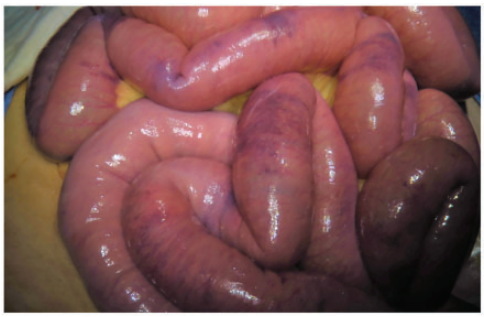

Figure 3. Full-length patchy ischemia without necrosis.

Patient 4

A 62-year-old woman with pancreatic head cancer infiltrating the transverse mesocolon underwent pylorus-preserving duodenopancreatectomy with en bloc right hemicolectomy. Enteral feeding started on POD 1 using an intraoperatively placed nasojejunal tube (Freka Trelumina FR 16/9; Fresenius Kabi Deutschland GmbH). On POD 6, during tube feeding with Biosorb (Pfrimmer Nutricia GmbH) at a rate of 50 mL/h, the patient complained of abdominal pain and nausea. An abdominal CT scan showed PI in a distended small bowel loop. The patient's symptoms were relieved after stopping the tube feeding, and her laboratory examination results were normal. Exploratory re-laparotomy was not indicated. During the following days, the patient developed hypotension and recurrent episodes of abdominal pain. Laboratory examination showed a continuously increasing C-reactive protein concentration. A control CT scan revealed free gas and fluid in the abdominal cavity.

The patient underwent emergency re-laparotomy on POD 10. Intra-abdominal exploration showed that all anastomoses were sufficient; however, a perforation of the jejunum caused by segmental ischemia of the small bowel was found. No further evidence of actual small bowel ischemia was found at the time of re-laparotomy. We resected the perforated segment and restored the intestinal continuity. The patient recovered and was discharged on POD 21.

Patient 5

A 65-year-old man with squamous cell carcinoma of the distal esophagus had a medical history of alcohol-induced liver cirrhosis (Child class A) and cachexia. He refused radiochemotherapy and insisted on surgical treatment. For improvement of his nutritional status, a percutaneous jejunostomy catheter (Flocare Jejunokath; Pfrimmer Nutricia GmbH) was placed, and high-caloric enteral feeding (Nutrison Multifibre 1500 kcal/d; Pfrimmer Nutricia GmbH) was prescribed for 6 weeks. He then underwent Ivor Lewis esophagectomy.

Jejunostomy tube feeding was restarted immediately after the operation with Fresubin Energy Fibre (Fresenius Kabi Deutschland GmbH) at a rate of 20 mL/h. This rate was gradually increased to 80 mL/h. On POD 5, re-intubation and invasive ventilation were required for pulmonary dysfunction. A CT scan and gastroscopy showed an anastomotic leak, and endoluminal vacuum-assisted therapy (Endo-SPONGE; B. Braun Melsungen AG, Melsungen, Germany) was initiated. On POD 7, the patient additionally developed abdominal distension, leukocytosis and hypotension. Another CT scan revealed small bowel dilation, PI and free peritoneal gas. Emergency re-laparotomy revealed extended small bowel ischemia with two necrotic segments of about 30 cm each, and one segment was perforated (Figure 4). We performed discontinuity resection of the necrotic small bowel segments and placed a temporary abdominal dressing (ABThera; KCI Medizinprodukte GmbH, Wiesbaden, Germany). At the staged re-exploration on the following day,

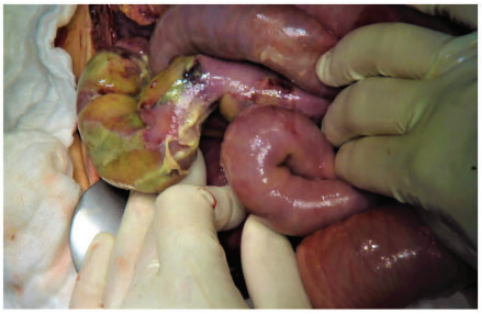

Figure 4. Ischemia with necrotic segments and perforation.

we found that the necrosis had progressed to the whole small bowel, and the right hemicolon and gastric tube had become ischemic. Thus, we had no further surgical options. The patient died of multiple organ failure on POD 10.

Discussion

NOMI associated with postoperative enteral tube feeding is considered to be a type of demand ischemia of the small bowel. Several hypotheses and contributing factors have been described, including large fluid shifts caused by the hyperosmolar load of feeding, gut distension and microvascular sludging.[6,8–11] Bacterial overgrowth associated with tube feeding can lead to gut distension, local vasospasm and hypoperfusion.[12,13] Systemic hypotension increases the risk of mucosal necrosis but can also be observed secondary to ischemic bowel wall damage.[12] However, most patients described in the literature did not develop major episodes of hypotension prior to the occurrence of NOMI.[8] Additionally, comorbidities accounting for low mesenteric flow, such as atherosclerotic disease, diabetes, or congestive heart failure, were not consistently present in the published cases.[8] Therefore, a multifactorial pathogenesis of NOMI associated with jejunal tube feeding must be assumed.

How the operation itself has an impact on the occurrence of NOMI remains unclear. One of our patients tolerated tube feeding well for 6 weeks before the operation but developed NOMI as tube feeding was continued postoperatively. Starting tube feeding at a low rate with only a gradual increase (<20 ml/h in 24 hours) seems to reduce morbidity and mortality.[6,9,17]

Two of our patients received enteral feeding via a nasojejunal tube. In most published cases, the patients received tube feeding via a jejunostomy catheter. The type of tube is not likely to cause differences in the occurrence of NOMI because the same organ is affected. Spalding et al.[16] reported no difference in the incidence of NOMI between needle catheter jejunostomy and open jejunostomy. Data on tube feeding via a nasojejunal tube are not available; thus, we cannot exclude the possibility of effects of this application on the occurrence of NOMI.

Tube feeding-associated NOMI is often difficult to identify because early clinical findings are nonspecific and similar to frequently seen functional complaints.[8,17,20] Furthermore, no factors have been proven to identify patients at risk for NOMI.[8] Corresponding to the clinical signs reported in the literature, our patients developed a painful distended abdomen, nausea and paralytic ileus with high output from the nasogastric channel of the tube. Paraclinical findings supporting the diagnosis in our patients were leukocytosis, hypotension and an increased C-reactive protein concentration (Table 1). Hypotension has been described as a rather late poor prognostic sign that is correlated with mortality if vasopressor treatment is required.[8,12,17] Three of our patients developed hypotension requiring vasopressor therapy. Lactic acidosis was not present in our patients. CT scans revealed small bowel distension with wall thickening and PI without obstruction of the mesenteric vessels in all

Table 1. Patient characteristics.

Pt.	Diagnosis	Procedure	Osmolality (mOsm/kg)	Days on feeding	Symptoms	Laboratory results*	CT findings	Intraoperative diagnosis	Outcome
1	Advanced gastric cancer	Multivisceral resection with gastrectomy and left pancreatosplenectomy	430	3	Abdominal distension, painful abdominal cramps, high reflux	WBC: 13.2 CRP: 146	Pneumatosis intestinalis, portal venous gas, and distended, paralytic small bowel	Distension of small bowel with segmental ischemia without necrosis	Discharged on POD 15
2	Boerhaave's syndrome	Esophagectomy with secondary reconstruction	430	12	Abdominal distension	WBC: 16.8 CRP: 153	Pneumatosis intestinalis and distended, paralytic small bowel	Distension of small bowel with segmental ischemia and necrosis of gastric tube	Discharged on POD 31
3	Gastric cancer	Gastrectomy	440	4	Abdominal distension, painful abdominal cramps, hypotension	WBC: 14.7 CRP: 138	Pneumatosis intestinalis, portal venous gas, and distended, paralytic small bowel	Distension of small bowel with ischemia without necrosis	Discharged on POD 18
4	Cancer of pancreatic head with infiltration of transverse mesocolon	Pylorus-preserving duodenopancreatectomy and right hemicolectomy	440	6	Painful abdominal cramps, nausea, hypotension	WBC: 9.9 CRP: 229	Free peritoneal gas, pneumatosis intestinalis, and distended, paralytic small bowel	Segmental ischemia and circumscribed necrosis with perforation	Discharged on POD 21
5	Squamous cell carcinoma of the distal esophagus	Ivor Lewis esophagectomy	430	7	Abdominal distension, hypotension	WBC: 17.3 CRP: 257	Free peritoneal gas, pneumatosis intestinalis, and distended, paralytic small bowel	Extended small bowel ischemia with two necrotic segments of about 30 cm each, with a covered perforation	Died on POD 10

Pt., patient; CT, computed tomography; WBC, white blood cells; CRP, C-reactive protein; POD, postoperative day;
*WBC in Gpt/L, CRP in mg/L.

of our patients (Figure 1). PVG was found in two patients. Free abdominal gas was also detected in two patients (Table 1). These findings are consistent with most published cases.[8,20]

The impact of PI in the context of NOMI is controversial. Some reports have described PI in patients with complete remission of symptoms after discontinuation of enteral feeding.[14,15] Furthermore, some authors have described successful conservative therapy for NOMI in patients with PI and massive PVG.[18] This is remarkable because the latter is commonly considered to be suggestive of transmural bowel infarction.[19] In one of our patients with PI, surgical exploration was not performed because the clinical symptoms were relieved after tube feeding was discontinued. Later in the course, however, the patient underwent an emergency operation for ischemic perforation. Based on the clinical and paraclinical findings seen in our patients, our experience indicates that PI is a sign that necessitates exploratory surgery.

The intraoperative findings during emergency laparotomy in our patients varied from ischemia without evident necrosis (Figure 3) to patchy necrosis with perforation and segmental necrosis of different extents (Figure 4). The operative strategy in our patients involved resection of the necrotic bowel segment, decompression of the congested small bowel and decompression of the abdominal compartment if necessary. We found a correlation between the extent of necrosis and the prognosis. All patients with limited necrosis at the time of laparotomy recovered with a short time after the re-intervention.

Reports of conservative treatment of tube feeding-associated NOMI are rare. Discontinuation of tube feeding is recommended if clinical signs of NOMI develop. However, the effect of the latter on the extent of bowel necrosis and the prognosis is unclear.[8] Kurita et al.[18] described medical treatment of two patients with papaverine and prostaglandin E1. Conservative management was chosen for these patients, even with the presence of PI and PVG, because their vital signs were stable and acute peritoneal signs were absent. The patients underwent close clinical and CT follow-up to monitor their recovery.[18] Because NOMI is reversible until a certain time point, conservative therapy can be an option early in the course. The challenge in this setting is to determine the presence of bowel necrosis, which can be fatal. However, our knowledge regarding the effects of conservative treatment remains preliminary because there are only two documented cases in the literature to date.

The extent of bowel necrosis seems to have a relevant impact on the prognosis; therefore, early diagnosis and surgery are crucial to increase the chance of a good outcome.[8,17,20] In our series, early intervention resulted in limited necrosis in most patients and a survival rate of 80%. For patients with nonspecific clinical signs, however, early diagnosis can be challenging. With reference to the literature and our cases, we suggest the following diagnostic and treatment algorithm for patients receiving postoperative enteral tube feeding.

1. If patients develop abdominal cramps, distension, nausea and high nasogastric tube output:
   - enteral feeding must be stopped
   - awareness of a potential diagnosis of NOMI is important for further clinical evaluation
2. If complaints persist or leukocytosis and/or hypotension develops:
   - a CT scan should be performed
   - procedure-related postoperative complications should be excluded
3. In case of radiologic findings of PI or PVG and/or free peritoneal gas:
   - emergency re-laparotomy is indicated

Table 2. Reported cases of tube feeding-associated NOMI following abdominal surgery.

First author (year)[Ref]	Diagnosis	Procedure	Osmolality (mOsm/kg)	Days on feeding	Intraoperative diagnosis	Outcome
Schunn (1995)[12]	Gastric cancer	Total gastrectomy	490	4	Entire small bowel necrosis	Survival
	Gastric cancer	Distal gastrectomy	367	9	Bowel necrosis distal to the jejunostomy	Survival
	Pancreatic cancer	Pancreaticoduodenectomy	310	7	Bowel necrosis distal to the jejunostomy	Death
	Pancreatic cancer	Jejunostomy	300	4	Bowel necrosis distal to the jejunostomy	Death
Rai (1996)[21]	Pancreatic cancer	Pancreaticoduodenectomy	300	14	Bowel necrosis distal to the jejunostomy	Death
Lawlor (1998)[22]	Esophageal cancer	Esophagectomy	375	3	Entire small bowel necrosis	Survival
	Barrett esophagus	Transhiatal esophagectomy	300	5	Patchy necrosis of small bowel, beginning 10 cm proximal to the jejunostomy insertion site and extending to the cecum	Survival
	Colon cancer	Gastrojejunostomy	300	6	Bowel necrosis distal to the jejunostomy	Survival
Jorba (2000)[23]	Distal common bile duct carcinoma	Pancreaticoduodenectomy	ND	6	Bowel necrosis distal to the jejunostomy	Death
Halkic (2005)[24]	Ampullary cancer	Pancreaticoduodenectomy	ND	7	Bowel necrosis with perforation distal to the jejunostomy	Survival
Thaler (2005)[25]	Pancreatic cancer	Pancreaticoduodenectomy	ND	4	Bowel necrosis distal to the jejunostomy	Survival
	Chronic pancreatitis	Pancreaticoduodenectomy	ND	3	Bowel necrosis distal to the jejunostomy	Survival
Messiner (2005)[26]	Pancreatic cancer	Pancreaticoduodenectomy	ND	ND	Bowel necrosis	Survival
	Pancreatic cancer	Pancreaticoduodenectomy	ND	ND	Bowel necrosis	Death
	Ampullary cancer	Pancreaticoduodenectomy	ND	ND	Bowel necrosis	Death
Spalding (2006)[16]	Gastric cancer	Total gastrectomy	300	3	Proximal jejunal necrosis	Death
	Gastric cancer	Total gastrectomy	300	4	Entire small bowel necrosis	Death
	Gastric lymphoma	Total gastrectomy	300	17	Entire small bowel necrosis	Death
	Ampullary cancer	Pancreaticoduodenectomy	300	4	Entire small bowel and right colon necrosis	Death
	IPMN	Pancreaticoduodenectomy	300	6	Partial small bowel ischemia	Survival
	Pancreatic cancer	Pancreaticoduodenectomy	460	12	Entire small bowel necrosis	Death
Melis (2006)[8]	Esophageal cancer	Esophagectomy	460	6	Bowel necrosis distal to the jejunostomy	Death
Sarap (2010)[27]	Gastric cancer	Distal gastrectomy	ND	2	Entire small bowel and right colon necrosis	Death
Qureshi (2010)[28]	Esophageal cancer	Esophagectomy	ND	3	Entire small bowel and large bowel necrosis	Death

(continued)

Table 2. Continued

First author (year)[Ref]	Diagnosis	Procedure	Osmolality (mOsm/kg)	Days on feeding	Intraoperative diagnosis	Outcome
Gwon (2012)[29]	Liver mass	Laparotomy, RFA	ND	15	Small bowel necrosis, mucosal infarction, and congestion of the entire intestine	Death
	Mid common bile duct carcinoma	Bile duct resection and cholecystectomy	ND	10	Hemorrhagic necrosis of mucosal layer and congestion of submucosa	Survival
Al-Taan (2017)[20]	Gastric cancer	Total gastrectomy	ND	13	Bowel necrosis distal to the jejunostomy	Survival
	Gastric cancer	Total gastrectomy	ND	7	Bowel necrosis distal to the jejunostomy	Death
	Gastric cancer	Total gastrectomy	ND	6	Bowel necrosis distal to the jejunostomy	Survival
	Gastric cancer	Total gastrectomy	ND	7	Bowel necrosis distal to the jejunostomy	Survival
	Gastric cancer	Total gastrectomy	ND	5	Bowel necrosis distal to the jejunostomy	Survival
	Gastric cancer	Total gastrectomy	ND	4	Bowel necrosis distal to the jejunostomy	Survival
Sethuraman (2017)[17]	Pancreatic cancer	Pancreaticoduodenectomy	ND	ND	Patchy antimesenteric transmural necrosis distal to the jejunostomy site	Death
	Insulinoma	Total pancreatectomy	375	3	Bowel necrosis distal to the jejunostomy	Death
	Ampullary cancer	Pancreaticoduodenectomy	620	2	Bowel necrosis distal to the jejunostomy	Survival
	Pancreatic cancer	Total pancreatectomy	600	9	Bowel necrosis distal to the jejunostomy	Survival
	Pancreatic cancer	Pancreaticoduodenectomy	620	10	Bowel necrosis distal to the jejunostomy	Death
	Esophageal cancer	Esophagectomy	350	3	Bowel necrosis distal to the jejunostomy	Survival
	Pancreatic cancer	Pancreaticoduodenectomy	350	5	Bowel necrosis distal to the jejunostomy	Survival
	Esophageal cancer	Esophagectomy	350	14	Bowel necrosis distal to the jejunostomy	Survival
Nakagawa (2018)[30]	Gastric cancer	Distal gastrectomy	ND	4	Small bowel ischemia without necrosis	Survival
Kurita (2019)[18]	Esophageal cancer	Esophagectomy	700	4	No operation	Survival
	Esophageal cancer	Esophagectomy	400	6	No operation	Survival

NOMI, nonocclusive mesenteric ischemia; IPMN, intraductal papillary mucinous neoplasm; RFA, radiofrequency ablation; ND, no data available.

In case of small bowel distension with wall thickening and free fluid, but no PI:

- conservative therapy (papaverine, prostaglandin E1) can be considered for stable patients
- close clinical and CT follow-up is necessary for patients without signs of peritonitis

4. Emergency re-laparotomy should involve resection of the necrotic bowel segments, decompression of the distended small bowel, and, if necessary, decompressions of the abdominal compartment by either mesh interposition or temporary (vacuum-assisted) abdominal dressing. The clinician should also consider the need for staged re-laparotomy for lavage as well as possible further resection and re-anastomosis.

The suggested algorithm is based on reports of a limited number of patients in the literature (Table 2). These previously described patients underwent various operations, were administered various feeding formulas and had different comorbidities. Data from a larger and more homogeneous cohort of patients are desirable but rather unlikely to be obtained because of the low incidence of NOMI. Whether sequential therapy of vasodilatory treatment and surgery can help to reduce the extent of necrosis and improve patients' outcomes requires further study.

Conclusion

NOMI is a rare but serious complication of postoperative enteral tube feeding and is associated with a grave prognosis and high mortality. The extent of small bowel necrosis at the time of re-laparotomy seems to have a relevant impact on the prognosis. Therefore, early diagnosis including CT and re-laparotomy should be the main focus for these patients. Early symptoms are nonspecific, and the clinical management is often based on subjective criteria. We have herein introduced an algorithm used in our clinic to evaluate the clinical findings, laboratory results and radiological investigations to establish the diagnosis of NOMI and determine the need for re-laparotomy. Operative procedures in patients with NOMI should focus on resection of necrotic bowel segments. In the early stage of NOMI without evident necrosis, decompression of the congested small bowel and the abdominal compartment can facilitate recovery and improve the prognosis.

Informed consent was obtained from all individual participants included in the study. All patients were treated by routine surgery, and ethics approval was not necessary.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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