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REVIEW

Nonocclusive Mesenteric Ischemia: A Review for Interventional Radiologists

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Abstract:
Nonocclusive mesenteric ischemia (NOMI) is a condition characterized by segmental or discontinuous mesenteric ischemia and intestinal necrosis without an organic obstruction in the mesenteric vessels. Diagnosis is challenging, and early intervention is crucial for improving patient outcomes. Various factors such as background factors, symptoms, biomarkers, and imaging techniques contribute to the diagnosis. Ensuring an early diagnosis and prompt treatment is of paramount importance. Although studies reported on the effectiveness of intra-arterial vasodilator infusion therapy as an endovascular treatment, its future role remains uncertain. Therefore, this review primarily aimed to provide a comprehensive summary of the advancements in the current state of NOMI management, with a specific emphasis on the implementation of endovascular therapy.

Keywords:
NOMI, nonocclusive mesenteric ischemia, intra-arterial infusion therapy, contrast-enhanced CT

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Introduction

Nonocclusive mesenteric ischemia (NOMI), initially documented by Ende in 1958 [1], is characterized by segmental or discontinuous mesenteric ischemia and intestinal necrosis without an organic obstruction in the mesenteric vessels. Owing to the difficulty of early diagnosis, even with the recent intensive care advances, coupled with the high mortality rate surpassing 50%, NOMI has a poor prognosis [2].

NOMI predominantly manifests in patients afflicted by shock, especially after cardiac surgery, those with low-output heart failure, and those with various acute critical illnesses all requiring high-dose vasopressor therapy [3, 4]. Accurate diagnosis and better survival rates for patients with complex NOMI rely on understanding current diagnostic methods, distinguishing NOMI from other causes of acute intestinal ischemia (AII), such as bowel obstruction, and reviewing effective endovascular and surgical treatments. This article outlines the current status of NOMI management, diagnosis, and the actual practice of arterial infusion therapy as an endovascular treatment.

Pathophysiology

Although the exact pathophysiology of NOMI remains uncertain, the primary mechanism is thought to be a severe reduction in intestinal blood flow or an imbalance between supply and demand [5-7]. When systemic hypoperfusion persists because of various factors, AII occurs as a result of endogenous vasopressin and angiotensin secretion to maintain cerebral and cardiac blood flow. Furthermore, vasoconstrictors administered as therapeutic agents contribute to mesenteric vasoconstriction [7, 8]. Thus, mucosal damage ensures at an early stage and is caused by reduced intestinal blood flow. Impaired intestinal mucosal barrier function then facilitates bacterial translocation, endothelial dysfunction, and ischemia-reperfusion injury, accompanied by local cytokine production. These progressive processes gradually culminate in full-thickness intestinal necrosis [8, 9]. As shown in an animal experimental study by Chiu et al. [6], the onset speed and severity of mucosal lesions correlate with decreased blood flow. Therefore, an early multidisciplinary therapeutic intervention for AII has been reported to improve life outcomes [10]. Conversely, NOMI poses par-

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ticular challenges due to the prevalence of poor general conditions in many patients, concurrent use of multiple medications, diagnostic difficulties, delayed treatment, and unclear demarcation between the affected and normal areas. As a result, the treatment often involves extensive bowel resection and NOMI is associated with a higher mortality rate than AII caused by organic mesenteric vascular obstruction [5, 9].

Diagnosis

Risk factors and symptoms

The typical risk factors for patients developing NOMI include advanced age, dialysis, cardiac disease, its postoperative period, medications (digitalis, catecholamines, or diuretics), decreased circulating plasma volume (burns, pancreatitis, dehydration, or bleeding), diabetes, and sepsis, especially in patients on maintenance dialysis and those who underwent cardiac surgery [1-8, 11, 12]. Conversely, NOMI symptoms are generally nonspecific, making the diagnosis difficult. The common findings of AII include severe abdominal pain, diarrhea, upper and lower gastrointestinal bleeding, and vomiting, regardless of whether it is caused by NOMI or other etiologies [5], whereas specific findings may suggest the presence of NOMI, including ileus, increased abdominal circumference, increased intra-abdominal pressure, and stomach reflux. However, NOMI diagnosis can be challenging because many patients with NOMI are typically under intensive care and may be sedated. This can make it difficult to obtain abdominal findings and can contribute to the overall complexity of the diagnosis [5, 7, 8].

Biomarkers

No specific biomarkers were observed in patients with NOMI. However, an increased lactate level indicates tissue ischemia, and increased AST, LDH, CPK, and D-dimer levels might be useful as auxiliary diagnoses for NOMI [5, 8]. Conversely, these biomarkers are often altered in critically ill patients with or without NOMI, resulting in lower sensitivity [13]. Intestinal fatty acid-binding protein (I-FABP) is specifically distributed in the small intestinal mucosa and is rapidly released into the bloodstream upon injury. Recently, I-FABP was found useful for the early diagnosis NOMI [14-18]. Matsumoto et al. reported that I-FABP demonstrated sensitivity of 76%, specificity of 80.3%, and an area under the curve of 80.5% to diagnose AII, including 15 patients with NOMI. These findings indicated that I-FABP performed better than other biomarkers in terms of diagnostic accuracy [15]. Sekino et al. measured daily plasma I-FABP levels in ICU patients with septic shock and reported a high incidence of NOMI with I-FABP levels of >19.0 ng/mL [16]. Furthermore, urinary I-FABP levels were found to improve the early AII diagnosis [18]. Conversely, I-FABP is found to be also elevated in various forms of nonvascular intestinal ischemia, such as acute enterocolitis, Crohn's disease, and simple bowel obstruction [15]. A subset of patients with NOMI has been suggested to exhibit lower I-FABP levels, potentially resulting from enterocyte depletion within the ischemic and/or necrotic bowel [16]. Moreover, I-FABP was reported to be significantly higher in patients with chronic kidney disease than in those with normal renal function [19]. Although these results require further validation, they suggest that serum or urinary I-FABP may contribute not only to the early diagnosis of NOMI but also to the determination of the timing of therapeutic intervention, such as surgery or intra-arterial infusion therapy, as well as that of treatment strategy and effectiveness.

Imaging

Historically, angiography has been the "gold standard" for the diagnosis of NOMI, but with the recent development of multidetector-row computed tomography (MDCT), dynamic CT is currently playing a central role, particularly in Japan. Abdominal plain X-rays, abdominal ultrasound, and contrast MRI are used as needed for support. Herein, we discuss angiographic and contrast-enhanced CT findings of NOMI.

Angiography

There have been numerous reports on angiographic findings in patients with NOMI. The following angiographic findings suggest mesenteric vasoconstriction, as demonstrated by Siegelman et al. in 1974 [20], which are still considered important nowadays [21]: (1) narrowing at the origins of the major branches of the superior mesenteric artery (SMA); (2) irregularities in intestinal branches with segments of narrowing, zones of beading, and abnormal tapering; (3) spasm of the intestinal arcades; and (4) impaired filling of intramural vessels. These findings may be localized and difficult to be assessed accurately. Moreover, individual findings are not specific to NOMI and may differ depending on external factors such as catheter position and/or contrast medium administration. Therefore, a study argued that angiography is not the tool of choice for diagnosing NOMI [13]. However, there are attempts to standardize and quantify angiographic findings, predict prognosis using angiographic findings, and evaluate the treatment outcomes. For instance, Minko et al. [22] standardized angiographic findings by developing a scoring system (**Table 1**). Their study reported high sensitivity of 85.7%, specificity of 71.4%, and receiver operating characteristic curve of 0.851 with a cutoff value of 3.5, indicating a high probability of predicting the perioperative prognosis. Conversely, other reports proposed that quantitative values such as peak density and time to peak can be calculated by analyzing angiographic images on a workstation using two-dimensional perfusion angiography (2D-PA), which was found to be useful for evaluating the treatment outcome of infusion therapy and for predicting the prognosis [4, 23]. Moreover, further utilization of this technique is expected in the future.

Contrast-enhanced Computed Tomography (CT)

With the recent development of MDCT technology, the ability to visualize the intestinal tract, mesentery, and mesenteric arteriovenous vessels has improved. As a result, dy-

Table 1. Simplified NOMI Score Based on Minko et al.[22].

Criteria/Score	Description		
Vessel morphology	SMA trunk	SMA branches	Mesenteric arcades
0	Normal	Normal	Normal
1	Normal	Slightly constricted	Slightly constricted
	Slightly constricted	Normal	Normal
	Slightly constricted	Slightly constricted	Slightly constricted
2	Partly constricted	Constricted	Constricted
3	Completely constricted and partly not visible		
Contrast media-reflex into the Aorta	*Flow rate: 5 ml/sec, amount: total 25 ml		
0	No contrast medium reflux into the aorta		
1	Some contrast medium reflux into the aorta		
2	Sever contrast medium reflux with complete aortogram		
Time to portal vein filling			
0	≦ 8 sec		
1	< 8 to ≦ 12 sec		
2	> 12 sec		

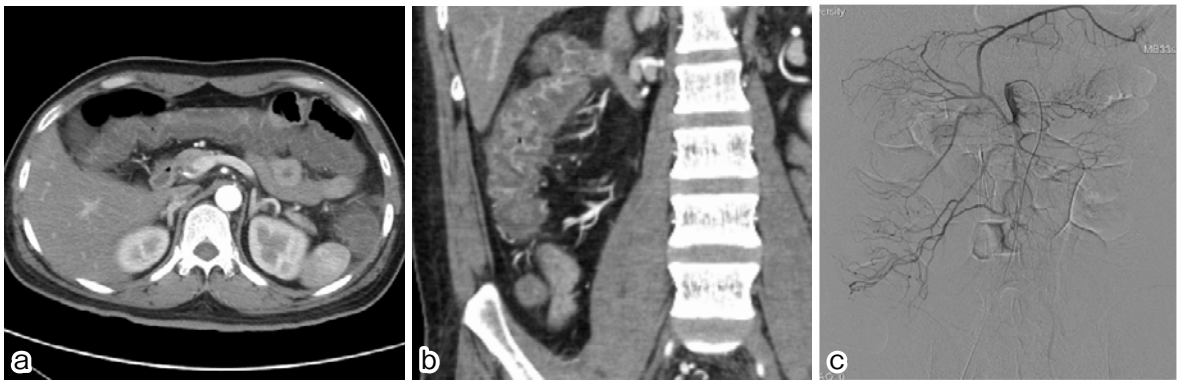

Figure 1. A male in his 30s with NOMI.
These figures were reproduced from reference 42. The authors received approval from the Japanese Society of Interventional Radiology (JSIR).
a, b: Axial (a) and coronal (b) contrast-enhanced CT images showed edematous bowel wall changes.
c: Angiography of the superior mesenteric artery showed poor bowel enhancement.

namic CT plays a very important role in the diagnosis of NOMI. However, the interpretation and evaluation of the vascular findings, particularly the SMA, are of utmost importance. Therefore, thin-slice images acquired during the arterial phase and reconstructed images (including the maximum intensity projection images) should be thoroughly evaluated. These techniques are used to (1) rule out differential diseases, (2) identify ischemic changes in the intestinal tract and mesentery, and (3) confirm abnormal findings in the SMA and its major branches. In particular, paralytic ileus, strangulated bowel obstruction, SMA and superior mesenteric vein (SMV) thrombosis, and dissection of the SMA (localized dissection or spread of aortic dissection) are important as differential diseases. Ischemic changes in the intestinal tract are confirmed by edematous thickening and poor enhancement of the intestinal wall, intestinal fluid retention with intestinal dilatation, emphysema in the intestinal wall and portal vein, and increased intestinal wall density on non-contrast-enhanced CT (reflecting intestinal wall necrosis) (**Fig. 1** and **2**). All of these findings are important for the diagnosis of intestinal ischemia as well as NOMI. However, these unspecific findings are present in only approximately 60% of patients with NOMI, but the absence of these findings does not rule out NOMI [21]. Furthermore, a recent report highlighted that intestinal ischemia resulting from NOMI can potentially affect not only the small intestines but also the stomach and colon. The prognosis becomes notably unfavorable when necrosis occurs, particularly in the more proximal regions of the gastrointestinal tract [24]. Therefore, screening for intestinal ischemia should be performed not only in the superior mesenteric region but also in the whole intestinal tract. Furthermore, it

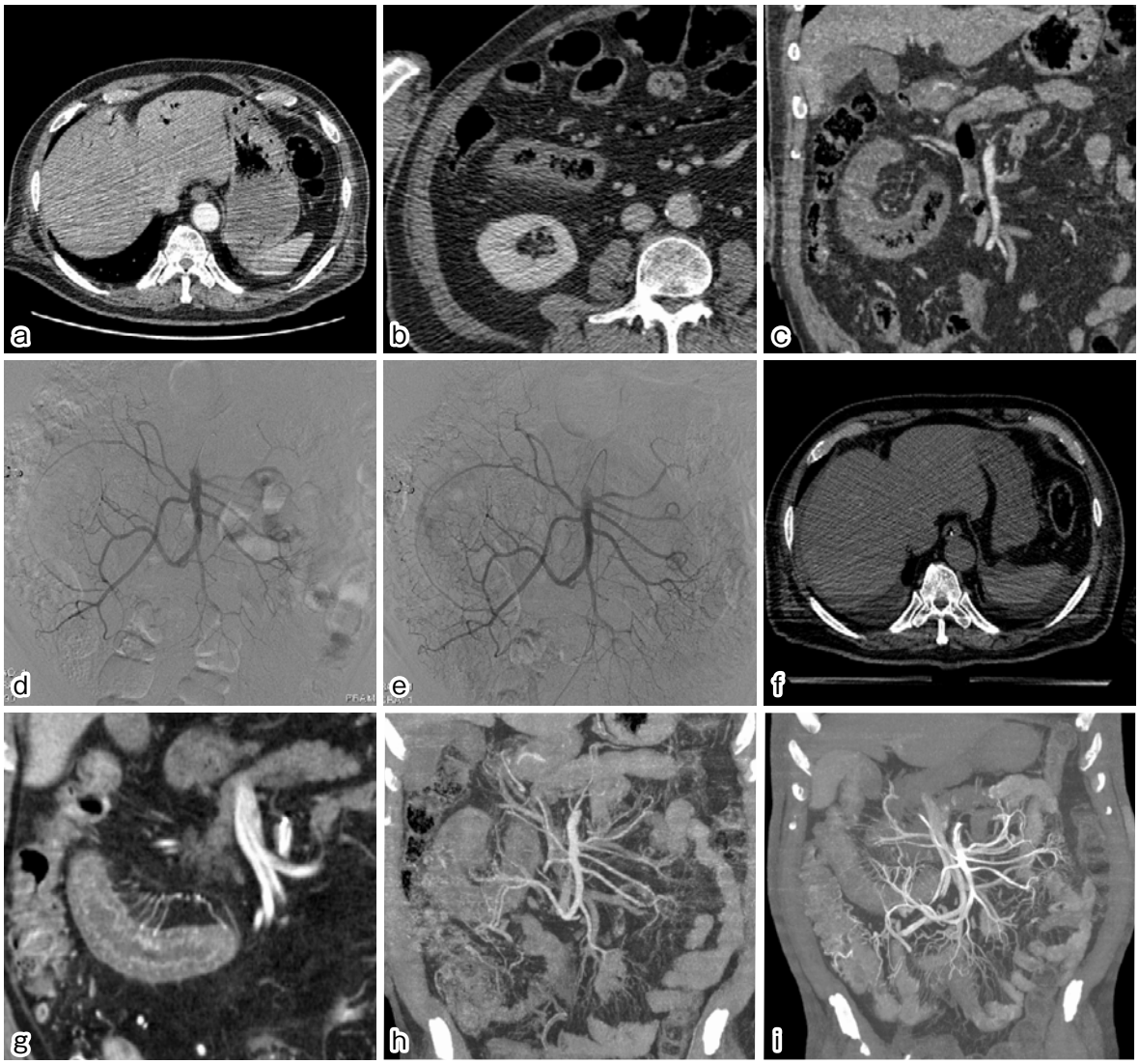

Figure 2. A male in his 60s with NOMI.

These figures were reproduced from reference 42. The authors received approval from the Japanese Society of Interventional Radiology (JSIR).

a: An axial contrast-enhanced CT image revealed portal venous gas.

b, c: Axial (b) and coronal (c) contrast-enhanced CT images showed poor mucosal enhancement and edematous bowel wall changes.

d, e: Angiography of superior mesenteric artery pre- (d) and post (e) intra-arterial vasodilator infusion therapy. Preangiography (d) showed irregularities in the intestinal branches and arcade spasms with no contrast within the intramural vessels of the bowel. Postangiography (e) showed markedly improved intramural vessel filling.

f, g: Axial (f) and coronal (g) CT during superior mesenteric arteriogram images showed the disappearance of portal venous gas and improved visualization of intramural vessels.

h, i: Pre- (h) and posttreatment (i) coronal CT images demonstrate improved intramural vessel visualization.

has been reported [25] that a higher prevalence and higher number of organ infracts were significantly different in patients receiving vasoconstrictor agents (VCAs) compared with those not receiving VCAs. Moreover, attention should be given to other organs. In the assessment of abnormal vascular findings, several studies from Japan have indicated that the four diagnostic indicators mentioned above [20] can be evaluated with comparable efficacy to angiography [26, 27]. These reports have also revealed that the SMA and SMV diameters are lower than those of normal ones, which was aligned with findings from Western countries [28-30]. Their results are summarized in **Table 2**. Thus, these results may serve as important objective indices in the CT diagnosis of NOMI. Another study indicated that not only the SMA, SMV, celiac artery, inferior mesenteric artery, and inferior vena cava diameters but also the CT value changes of the intestinal wall have been suggested to be associated with prognosis [30]. Notably, the combination of the inferior vena cava diameter and CT value alterations between the late arterial phase and unenhanced scan of the intestinal wall has demonstrated a remarkable ability to predict the prognosis with a high accuracy level.

Table 2. SMA and SMV Diameters.

Reference	SMA diameter in NOMI patients		SMA diameter in controls		SMV diameter in NOMI patients		SMV diameter in controls	
	Mean diameter (mm) (range)	Number	Mean diameter (mm) (range)	Number	Mean diameter (mm) (range)	Number	Mean diameter (mm) (range)	Number
Woodhams et al [26] (2010)	3.4 ± 1.1 (2-4)	4	6.0 ± 1.5 (4-8)	13	N.L	N.L	N.L	N.L
Nakamura et al [27] (2013)	6.0 (median) (3.5-8.0)	11	7.6 (median) (5.5-9.0)	44	7.5 (median) (5.0-9.0)	11	11.0 (median) (8.0-14.0)	44
Pérez-García et al [28] (2018)*	5.39 ± 1.21 (N.L)	55	7.32 ± 1.21 (N.L)	55	N.L	N.L	N.L	N.L
Kammerer et al [29] (2018)	5.9 ± 1.4 (N.L)	28	N.L	N.L	9.2 ± 2.9 (N.L)	28	N.L	N.L
Bagnacci et al [30] (2022)**	4.1 ± 1.1 (N.L)	43	4.9 ± 1.2 (N.L)	41	8.5 ± 2.8 (N.L)	43	9.9 ± 2.4 (N.L)	41

N.L: No listed, PGE1: prostaglandin E1

*Control is a previous CT images in same patients.

**NOMI patients means patients with negative outcome, and control means patients with positive outcome.

Treatment

First, the occurrence of NOMI is generally associated with an underlying disease. As a result, the initial crucial step in the treatment involves addressing the underlying condition. However, it is worth noticing that if VCAs are used in the treatment process, discontinuing these drugs should be considered before starting the NOMI treatment. Specific treatments for NOMI can be broadly classified into two categories: pharmacotherapy with vasodilators to maintain mesenteric vascular blood flow and surgical therapy for intestinal necrosis.

Nonsurgical therapy: practice of pharmacotherapy and intra-arterial infusion

Pharmacotherapy

As summarized in a review by Trompeter et al. in 2002 [21], vasodilators have been widely used because tolazoline use was first reported in 1967 [31]. Subsequent reports [11, 32-34] have highlighted the efficacy of specific vasodilators administered via intra-arterial infusion for NOMI treatment [35]. Conversely, Mitsuyoshi et al. reported the usefulness of high-dose continuous intravenous prostaglandin E1 (PGE1) infusion (0.01-0.03 μg/kg/min) [36]. This method gained significant recognition in Japan as a treatment that can be performed quickly and easily because angiography is no longer performed as a diagnostic procedure as NOMI diagnosis has become possible using the MDCT technique. All guidelines that mention NOMI in English [37-40] and that have been identified so far recommend an intra-arterial infusion of vasodilators as a treatment for NOMI. Only the Appropriateness Criteria [37] by the American College of Radiology mention that a high-dose PGE1 continuous intravenous infusion can be as effective as an intra-arterial vasodilator infusion. In any case, intra-arterial infusion, or continuous high-dose PGE1 intravenous infusion is based on a small number of retrospective studies. Moreover, no randomized control trial (RCT) data are currently available on these therapies. Additionally, the risks of side effects, including vascular injury with intra-arterial infusion, hypotension with high-dose intravenous PGE1 infusion, and bleeding due to the inhibition of platelet aggregation with PGE1, have not been adequately assessed. It is anticipated that future studies will change the status of these treatments.

Angiography and Intra-arterial Infusion Therapy

As noted above, many studies have reported intra-arterial infusion therapy (IAIT), but the technical aspects and drug dosage widely varied. Although a report of basic experiments showing that the effects of each drug on the human SMA are comparable [41], no reports compared the actual clinical efficacy. **Table 3** summarizes recent representative past reports for reference. The procedure is performed using a 4-Fr catheter in many institutes worldwide. However, some papers report the use of Cobra, Simmons, and Loop-type catheters [42]. It can be presumed that they are using a catheter that they are familiar with for selecting the SMA. Contrast administration for SMA angiography is performed using a contrast agent dose of 300-370 mgI iodine preparation at an injection rate of 4-6 mL/s and a contrast agent volume of 20-40 mL.

IAIT commonly uses PGE1 or papaverine hydrochloride alone or in combination. Regarding the dosage, several studies have reported continuous administration with papaverine hydrochloride at 30-60 mg/h or continuous administration with papaverine hydrochloride at 30-60 mg/h after bolus administration of PGE1 (5-10 μg). Rittgerodt et al. [4] reported the use of a diagnostic sheath and catheter after fixing directly to the skin for continuous infusion. Conversely, another institute changed the diagnostic catheter to a heparin-coated Cobra-type catheter and then fixed it in place [42].

Table 3. Intra-arterial Infusion Therapy.

Reference	Catheters		SMA angiography		Vasodilator	Dosage
	Size (Fr)	Type	Flow late (ml/sec)	Amount (ml)		
Koltz et al [33] (2001)	5	N.L	N.L	N.L	Papaverine	Infusion 0.7 mg/kg/h (~60 mg/h)
Trompeter et al [21] (2002)	N.L	Cobra Sidewinder J-curved	4-6	20-40	PGE1	Bolus of 20 μg infusion 2.5-5 μg/h for Max 3 days
Watanabe et al [11] (2008)	N.L	N.L	N.L	N.L	PGE1	Infusion 500 μg for 30 min
Minko et al [22] (2014)	4	Cobra	5	25	Levosimendan	N.L
Bomberg et al [34] (2016)	4	Cobra	N.L	N.L	Iloprost	Infusion 4 ng/kg/min
Becker et al [23] (2020)	4	Cobra Sidewinder	4	24	PGE1	Bolus of 20 μg for 10 min
Winzer et al [35] (2020)	2.7/4/5	Cobra SIM1	N.L	N.L	Papaverine	infusion 50 mg/h for Max 1 hour 25 mg/h following 6 hours
Kawada et al [42] (2022)	4/5	GHC-A Cobra	4	20	PGE1 Papaverine	Bolus of 5 μg Infusion 40 mg/h
Rittgerodt et al [4] (2022)	4	Cobra2	N.L	N.L	PGE1	Bolus of 20 μg for 10 min Infusion 60-80 μg/24 h

N.L: No listed, PGE1: prostaglandin E1

Surgical therapy

Bowel resection of NOMI is indicated when the intestinal tract is necrotic. However, it is desirable to avoid short bowel syndrome as much as possible because wide bowel resection is life threatening and affects the quality of life. Therefore, resection should be as short as possible, but the actual decision for surgical intervention is difficult to make and the extent of resection is not easy to determine. Furthermore, intestinal ischemia occurs on the mucosal surface. Thus, observation from the serosal surface by laparotomy may not be sufficient for evaluation. A second-look surgery may be useful to address this problem [43]. Although few reports have been published to diagnose NOMI, the indocyanine green (ICG) fluorescence method, which has been used to evaluate intraoperative organ blood flow [44] and lymphatic flow [45], should be applied to identify ischemic sites in the intestinal tract [46, 47]. Interestingly, the use of the ICG fluorescence method has identified intestinal necrosis that was indistinguishable by the naked eye [48], which might be a promising tool.

In recent years, various hybrid therapies have been performed in hybrid operating rooms and they are being utilized in the abdominal region [49]. In the future, the aforementioned indexes may be used to influence strategies, such as performing arterial infusion in areas at a high risk of necrosis.

Treatment outcome

There are no RCTs comparing different treatments for NOMI, and thus, established treatment strategies are lacking. Consequently, discussions regarding treatment outcomes are confined to case series from individual institutions.

As for the results of the IAIT treatment, Winzer et al. reported the treatment outcomes for 66 patients with NOMI [35]. Notably, their findings demonstrated an enhancement in 30-day mortality: 96.8% for 31 patients under conservative treatment, in contrast to 65.7% for 35 patients treated with IAIT. Similarly, Mitsuyoshi et al. [36] demonstrated that among 22 patients with NOMI who received intravenous PGE1 infusion therapy (IVIT) at their institution, 13 conventionally managed patients experienced a mortality rate of 69.2%. By contrast, nine patients who underwent early treatment with IVIT demonstrated a significantly better outcome, with a mortality rate of 11.1%.

Regarding surgical treatment outcomes, Nakamura et al. reported the results of their second-look operation strategy [50]. Among 30 patients with NOMI, the mortality rate was 33.3% for nine cases managed via conventional surgical treatment whereas it was 28.6% for 21 patients treated with second-look operation strategy. Therefore, although the efficacy of individual treatments has been documented, direct comparisons between the different treatments are complicated because of the absence of standardized diagnostic criteria. Furthermore, the potential to combine these treatments for improved efficacy remains unexplored as a detailed evaluation is still in progress.

Conclusion

This review provides an overview of NOMI's disease concept, diagnostic methods, and treatment options, along with a summary of the current status and practical aspects of endovascular therapy. Owing to the lack of precise diagnostic tools at present, NOMI remains a disease where clinical sus-

picion, early diagnosis, and prompt treatment initiation significantly influence the prognosis. Further accumulation of research is necessary to standardize the diagnosis and establish treatment algorithms.

Conflict of Interest: None

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