Eur Radiol (2002) 12:1179–1187
DOI 10.1007/s00330-001-1220-2

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Non-occlusive mesenteric ischemia: etiology, diagnosis, and interventional therapy

Received: 3 September 2001
Accepted: 24 September 2001
Published online: 21 December 2001

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Abstract Non-occlusive mesenteric ischemia (NOMI) compromises all forms of mesenteric ischemia with patent mesenteric arteries. It generally affects patients over 50 years of age suffering from myocardial infarction, congestive heart failure, aortic insufficiency, renal or hepatic disease and patients following cardiac surgery. Non-occlusive disease accounts for 20–30% of all cases of acute mesenteric ischemia with a mortality rate of the order of 50%. Acute abdominal pain may be the only early presenting symptom of mesenteric ischemia. Non-invasive imaging modalities, such as CT, MRI, and ultrasound, are able to evaluate the aorta and the origins of splanchnic arteries. Despite the technical evolution of those methods, selective angiography of mesenteric arteries is still the gold standard in diagnosing peripheral splanchnic vessel disease. In early non-occlusive mesenteric ischemia, as opposed to occlusive disease, there is no surgical therapy. It is known that mesenteric vasospasm persists even after correction of the precipitating event. Vasospasm frequently responds to direct intra-arterial vasodilator therapy, which is the only treatment that has been shown to be effective.

Keywords Non-occlusive mesenteric ischemia · Intra-arterial DSA · CT · MRI · Vasodilator therapy

Introduction

Non-occlusive mesenteric ischemia (NOMI) comprises all forms of mesenteric ischemia without occlusion of the mesenteric arteries. Non-occlusive mesenteric ischemia is commonly caused by decreased cardiac output resulting in splanchnic hypoperfusion. It generally affects patients over 50 years of age suffering from myocardial infarction, congestive heart failure, aortic insufficiency, and renal or hepatic disease. Non-occlusive mesenteric ischemia accounts for 20–30% of all cases of acute mesenteric ischemia [1, 2, 3, 4]. We review the etiology, pathophysiology, diagnosis, and interventional therapy of non-occlusive mesenteric ischemia based on the literature and combined personal experience of the authors.

Morbidity and mortality

Non-occlusive mesenteric ischemia is a condition characterized by high morbidity and mortality rates. In the past 20 years a decrease in mortality from over 80 to approximately 50% has been reported [5, 6, 7]. Reasons for low survival rates are advanced age and the commonly long delay between the onset of symptoms and initiation of treatment. An early diagnostic workup and immediate therapy is essential for a successful outcome. Tolerance to ischemia of the intestine is limited and becomes critical after 3–6 h [8]. Since NOMI follows severe microvascular vasoconstriction, angiography is the only diagnostic modality which reliably establishes an early diagnosis. In the early stage of NOMI, the intestinal mucous membrane is not yet necrotic so that reperfusion as opposed to surgery is the primary therapeutic option. Ta-

Table 1 Overview of the mortality of patients with acute mesenteric ischemia

Reference	No. of patients	NOMI (%)	Mortality (%)
[6]	62 (NOMI only)	100	58
[8]	141	13	71
[13]	113	12	77
[14]	62	18	53
[2]	42	42	43
[3]	30	26	50
[7]	38	5	53
[4]	186		80
[17]	27	41	52

ble 1 gives an overview of the mortality of acute mesenteric ischemia on the basis of the literature.

Etiology of NOMI

Hypoperfusion of peripheral mesenteric arteries can be caused by different mechanisms. Cardiovascular and drug related factors are discussed [6]. The risk of developing NOMI increases with age. Pharmacological interactions in patients suffering from chronic diseases requiring a variety of drugs are cofactors of mesenteric vasoconstriction. Bruch et al. [2] examined 42 patients the majority of whom also presented with cardiac problems. It has been hypothesized that the apparent coincidence of NOMI and cardiac disease is based on a low cardiac output leading to peripheral hypoperfusion. This in turn leads to an activation of the sympathetic system resulting in an increased cardiac output and also further vasoconstriction of peripheral mesenteric vessels [4].

Digitalis is an additional risk factor for developing NOMI. It induces vasoconstriction and thus an increased resistance in peripheral splanchnic vessels [10]. Alkaloids constitute another substance group causing smooth muscle contraction of the arteriolar wall. Ergotamine is one of the most potent vasoconstrictors in this group, which plays an important pathogenic role in NOMI [6, 9]. A combination of glycosides and diuretics is frequently administered to patients with congestive heart failure. The increased renal blood flow caused by furosemide leads to a diminished mesenteric perfusion. This is probably due to the furosemide-related activation of the renin–angiotensin–aldosterone system with subsequently increased levels of angiotensin II [3, 11].

Other causes of mesenteric vasospasms are various forms of shock, septicemia, dehydration and hypotension following dialysis and heart surgery or major abdominal surgery [1].

The frequent concomitance of pancreatitis in NOMI is explained by the proximity of the superior mesenteric artery (SMA) and the celiac plexus to the pancreas. The inflammation of the pancreas induces vasomotor activation [3].

Splanchnic ischemia

Mesenteric vasoconstriction may be seen as early as 10 min after the onset of hypotension. The impairment of bowel perfusion may be severe up to a point where blood flow is virtually not measurable [11]. A decrease in O_2 uptake below demands results in an increased production of lactate by the bowel as a result of anaerobic glycolysis within cells. Splanchnic ischemia also increases xanthinoxidase in the small bowel mucosa. Xanthinoxidase transforms hypoxanthine to uric acid. The formation of free radicals occurs when the supply of oxygen is renewed after reperfusion. This in turn damages the cytomembrane and cell edema ensues with cellular decay. The small bowel mucosa reacts most sensitively to these changes which gradually affect the entire intestinal wall. This tissue damage leads to bacterial translocation with multi-organ failure [1, 11, 12].

Clinical signs of NOMI

Immediate angiography in patients with suspected acute mesenteric ischemia allows an early diagnosis, which has led to an improved survival. Nevertheless, clinical signs of NOMI are not very specific. Early symptoms are frequently absent so that acute abdominal pain may be the only presenting symptom of mesenteric ischemia. In 1996 Howard et al. [13] described the diagnostic dilemma of NOMI. In this retrospective study which included 113 patients with acute mesenteric ischemia, 12% of the patients were shown to have NOMI and 77% of these patients complained of abdominal pain. Physical signs, such as abdominal distension, abdominal tenderness and muscular defense, hypotension, fever, decreased bowel sounds, nausea, sickness, diarrhea, and anorexia, were observed with decreasing frequency [12]. Blood markers were also non-specific and only helpful subsequent to therapy. Following intestinal dehydration, the hematocrit increases [2]. Anaerobic glycolysis during hypoxia leads to lactic acidosis [11]. Damaged tissue cells release various enzymes, among them lactate dehydrogenase and creatinine kinase. Blood leukocytosis can be regarded as a non-specific associated reaction. Both, an experimental study on rabbits and the examination of a study population in a clinical setting [2], showed a close correlation of the beta-galactosidase-activity with NOMI. This increased activity could be found as early as 90 min after the onset of ischemia.

Differential diagnoses of splanchnic ischemia

Surgery is not a therapeutic option in the early stage of NOMI as opposed to arterial and venous splanchnic thrombosis or arterial embolism of the superior mesen-

teric artery [8, 9, 11]. In recent years, perfusion therapy using vasoactive substances has gained an important role in the successful treatment of NOMI. This emphasizes the importance of differentiating between NOMI and embolic or atherosclerotic causes of ischemia. The differential diagnoses include the following causes of splanchnic ischemia: splanchnic atherosclerosis; splanchnic arterial thrombosis and embolism; splanchnic small vessel disease; splanchnic venous thrombosis; septic shock; hypovolemic shock; hemorrhagic shock; anaphylactic shock; and strenuous physical exercise [9, 11].

Sonography

Ultrasound is a widespread, easy-to-handle screening tool for patients presenting with abdominal disorders. Combining B-mode ultrasound and duplex sonography is a standard initial non-invasive method to visualize the aorta and larger splanchnic arteries. In addition, several functional parameters of splanchnic blood flow can be obtained [18]. Duplex sonography is not able to reliably evaluate small splanchnic vessels or measure small bowel mucosal perfusion. In addition, the use of ultrasound is limited by the lack of patient compliance in critically ill patients, or obscured by distended bowel loops. Other deficits are problems in locating the vessel origin in approximately 10% of patients [19] and the poor correlation between flow parameters and the severity of ischemia [20, 21].

Sonomorphological findings, such as distended bowel loops, hypoechoic thickening of bowel wall by edema, decreased peristalsis or ileus, peritoneal fluid collections, and intramural gas collections, are not specific. Fluid collections and intramural gas are considered signs of severe necrosis of the mucosa and bowel wall.

If ultrasound is initially performed in patients with suspected mesenteric ischemia, a complete obstruction of the proximal mesenteric artery may be diagnosed. However, ultrasound cannot reliably exclude more peripherally located mesenteric ischemia; therefore, those patients should be referred to angiography even if the roots of the mesenteric arteries are patent.

Endoluminal laser Doppler flow measurements

Laser Doppler flowmetry involves a monochromatic laser beam focused on the gastrointestinal mucosa. Moving objects, thereby changing the light spectrum, diffuses the light. The change in spectrum is linearly related to mucosal perfusion. Major problems associated with its clinical application are the difficulty in avoiding movement artifacts, large measurement variability [22], and a large overlap between subjects with normal and abnormal mucosal perfusion. Since a small catheter is required to apply laser light, this method is not feasible in the middle and distal portions of the small bowel.

Plain films

Plain-film radiographs of the abdomen (anteroposterior and lateral decubitus views) are traditionally among the first modalities requested in patients presenting with acute abdominal disorders. The role of plain-film imaging is to exclude an intestinal or gastric perforation, ileus, and other causes (e.g., large abdominal masses, retroperitoneal hematoma) of acute abdominal pain. In mesenteric ischemia, abdominal plain films may show different changes depending on the duration and, less importantly, on the location of mesenteric artery occlusion. Probst et al. [23] classified radiological features as non-specific, appointing, or typical. Variable, well-defined intraluminal gas collections without thickening of the bowel wall were considered as non-specific, distended small and large bowel loops with bowel wall edema, and air–fluid levels as appointing. Intramural gas collections or gas bubbles in the portal vein and its branches are pathognomonic for intestinal ischemia. Beyer and Köster [24] suggested a similar classification but regarded bowel wall thickening and separation of bowel loops as specific. The limitation of plain-film radiographs is that up to 12–18 h after the onset of symptoms, only non-specific signs are present [23]. Furthermore, positive findings on plain films are present in only 20–60% of cases. Appointing signs are non-specific, since distended bowel loops with bowel wall edema may also be caused by many other entities such as intramural bleeding, inflammatory processes, or tumor [25]. Specific signs are nearly always due to necrosis of the bowel mucosa. Intramural gas collections in inflammatory bowel disease or following trauma are rare [26]. A normal abdominal plain film in a patient suffering from pain, which is out of proportion to physical findings, is suggestive of early mesenteric ischemia and should prompt consideration of diagnostic angiography.

Enteroclysma and contrast enema

Conventional diagnostic tools to assess the lumina of small or large bowel (enteroclysma using barium suspension and dilution or a double-contrast barium enema) are of no use in diagnosing acute mesenteric ischemia. In the chronic course of the disease, signs of ischemic damage of the bowel wall can be found [27]. They include mucosal ulcers, the loss of haustral or mucosal folds, and segmental stenoses.

Computed tomography

Computed tomography as another non-invasive method is able to depict the abdominal aorta, the origins of the splanchnic arteries, their central parts, and first-order branches in diagnostic quality. The examination time is short, and several other causes of acute abdominal pain other than mesenteric ischemia can be ruled out. When focusing on the mesenteric vessels, the examination has to be performed as a spiral- or multislice CT, using a thin collimation and a thin and overlapping reconstruction interval. For spiral CT, a collimation of 3–5 mm with a pitch of 1.5 and a reconstruction interval of 1–2 mm is sufficient. Vascular enhancement is achieved by administration of iodinated contrast agents in bolus technique (2–4 ml/s) [28]. Bolus tracking or the measurement of individual circulation time should be used to achieve optimal arterial enhancement. In patients with vague abdominal symptoms a protocol including a collimation of 5–8 mm, a pitch of 1.5, and an increment of 4–8 mm following the administration of an iodinated contrast agent (100–150 ml at 2–4 ml/s) is regarded as sufficient. Scan delay may be set at 70–100 s, thus providing good enhancement also within abdominal veins and parenchymal organs [29].

To evaluate the origins of splanchnic arteries and to differentiate between high-grade stenosis and an occlusion, multiplanar reformatting of data is necessary. Maximum intensity projection (MIP) of SMA branches requires time-consuming editing of the axial images. Even when using all available technical amenities, imaging of small branches of the mesenteric arteries or of mucosal perfusion by CT is not possible. Whether multislice CT (MSCT) and novel software solutions, including the automatic segmentation of single slices, will lead to a significant improvement remains to be investigated.

Central occlusions of the SMA and thrombosis of mesenteric veins is diagnosed by CT with a sensitivity of approximately 85%. Computed tomography can reveal rare causes of mesenteric ischemia such as a dissection of the abdominal aorta. Other signs found in CT are thickening of bowel wall, absence of bowel wall enhancement, intramural hemorrhage, focal or diffuse intraperitoneal fluid collections, intestinal pneumatosis, portal venous gas collections, edematous wall thickening, or inhomogeneous contrast enhancement of the mucosa (target phenomenon) in venous thrombosis [30, 31]. These unspecific signs are present in only 20–60% of patients. Mesenteric ischemia cannot be excluded by CT. In patients at risk and with suspected NOMI, CT should not be used as a first-line test and patients should be referred to angiography without any unnecessary time delay.

Magnetic resonance imaging

Similar to CT, MRI as a non-invasive method is able to depict the abdominal vasculature. With the introduction of contrast-enhanced MR angiography (CE MRA) it became feasible to generate high-quality and high-resolution images of the mesenteric vasculature. The short scan time diminishes peristalsis-related artifacts and eliminates both respiratory motion artifacts as well as misregistration artifacts due to inconsistent breath-holds [32]. Most radiologists prefer the coronal plane for the primary acquisition because it gives the best coverage of the renal arteries and the iliac arteries next to depicting the mesenteric vasculature, thus providing a “road map” of the major abdominal vessels. Most authors advocate a dose of 0.1–0.2 mmol Gd/kg body weight. The MIPs in all directions are obtained, and even oblique projections may be necessary. To evaluate small vessels, it is essential to review the raw data sets. Because of limitations in spatial resolution and despite multiplanar imaging possibilities, only proximal large and medium-sized arteries can sufficiently and reliably be imaged with CE MRA [33]. Further technical improvements are to be expected with faster gradients, improved coil technology, and parallel acquisition techniques.

In addition to anatomical detail, MRI provides information about visceral function. Li et al. [34] evaluated a cine-2D phase contrast (PC) technique to determine changes in blood flow of the SMA. The same authors used T2 relaxation times of blood in the SMV to determine the degree of oxygen extraction by the bowel (in vivo MR oximetry) [35].

In comparison with CT and ultrasound, MRA is more time-consuming and, similar to other non-invasive methods, there is no therapeutic option; therefore, MRA is no feasible diagnostic tool in critically ill patients or patients with severe acute abdominal disorders, including patients with suspected mesenteric ischemia. In patients with chronic disorders or suspected alteration of mesenteric blood flow CE MRA and functional MR imaging are diagnostic tests almost comparable to diagnostic angiography.

Angiography

Despite the technical evolution of non-invasive methods, such as CT, MR, and ultrasound, angiography, typically performed as digital subtraction angiography (DSA), is still the gold standard in diagnosing peripheral splanchnic vessel disease. Technically, following an overview aortogram (anteroposterior and lateral), selective angiograms of the splanchnic vessels are obtained following the intravenous or intra-arterial injection of 20–40 mg scopolamine (for technical details see Table 2) [36, 37]. Patients with suspected mesenteric ischemia should be

Table 2 Technical details for performing aortograms and selective angiograms of splanchnic vessels. *SMA* superior mesenteric artery; *IMA* inferior mesenteric artery

	Catheter	Amount of contrast media (ml)	Flow rate (ml)
Abdominal aorta	Pigtail	20–30	10–15
SMA	Cobra Sidewinder J-curved	20–40	4–6
IMA	Cobra Sidewinder	8–15	3–5
Celiac trunk	Cobra Sidewinder J-curved	20–30	5–8

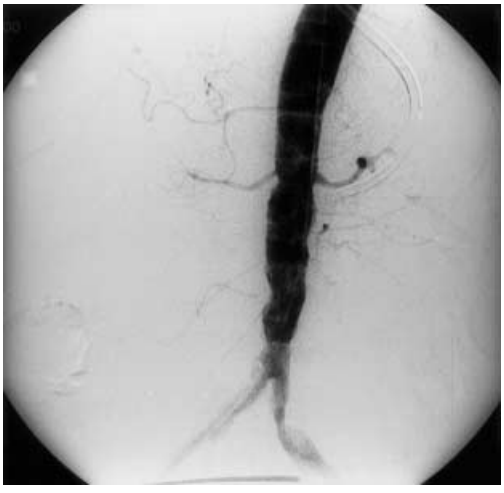

Fig. 1 Severe non-occlusive mesenteric ischemia, anteroposterior (AP) aortogram. The AP aortogram (25 ml, 15 ml/s) shows vasospasms with extreme vessel narrowing of the entire mesenteric vasculature and hepatic artery, renal arteries, and lumbar arteries

referred to angiography during the early clinical course. If occlusive disease (either embolic or thrombotic) is identified at the superior mesenteric arterial origin, no further angiographic work-up is required. If NOMI is diagnosed using the criteria listed below, the catheter tip should remain in stable position within the superior mesenteric artery for subsequent vasodilator therapy.

Biplanar aortography shows the origins of the celiac trunk and the superior mesenteric and inferior mesenteric arteries. Occlusive disease or stenoses of central parts of those vessels can be identified. In case of severe NOMI vasoconstriction is not only present in branches of the SMA, but also in branches of the celiac trunk and in the inferior mesenteric artery (IMA). Even the renal arteries may be involved (Figs. 1, 2). Having proven these findings, selective angiography of the SMA is only necessary to document the status prior to vasodilator therapy.

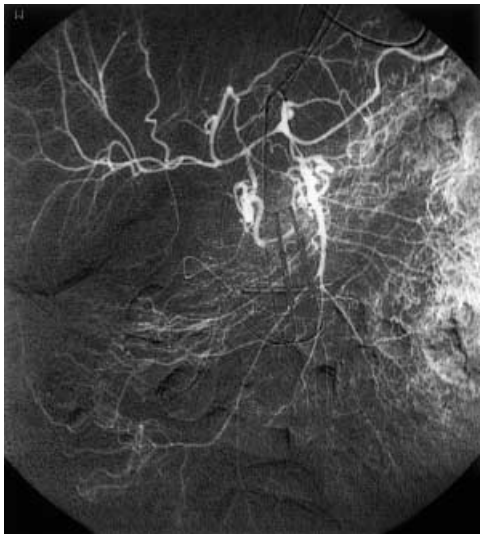

Fig. 2 Severe non-occlusive mesenteric ischemia, superior mesenteric artery (SMA) angiogram. The selective SMA angiogram (30 ml, 5 ml/s) demonstrates narrowing of all SMA branches and is also seen with the hepatic artery and splenic artery, which are contrasted by retrograde flow because of a celiac trunk stenosis. Intramural bowel vessels within the ileum and ascending colon are not contrasted due to severe ischemia

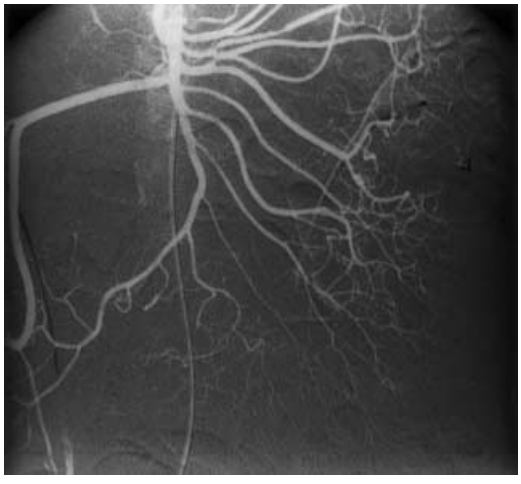

Fig. 3 Non-occlusive mesenteric ischemia of proximal SMA. The selective SMA angiogram (30 ml, 5 ml/s) shows proximal narrowing of the jejunal arteries, alternate dilatation and narrowing of some jejunal arteries, and a vasoconstriction of ileal arteries and the ileocolic artery

Siegelmann et al. [38] described four reliable arteriographic criteria for the diagnosis of mesenteric vasospasm:

1. Narrowing of the origins of multiple branches of the SMA (Fig. 3)
2. Alternate dilatation and narrowing of the intestinal branches, the so-called string of sausages sign (Figs. 3, 4)

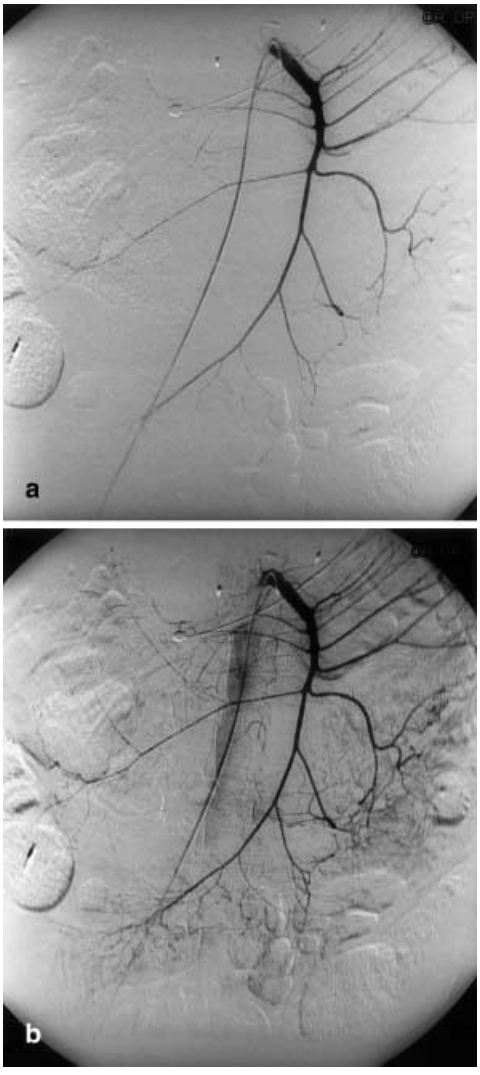

Fig. 4a, b Severe non-occlusive mesenteric ischemia of distal SMA. The selective SMA angiogram (25 ml, 5 ml/s) during an **a** earlier and **b** later arterial phase demonstrates a vasoconstriction of the distal SMA and all of its branches, including spasm of mesenteric arcades. Some distal jejunal and ileal branches show alternate dilatation and narrowing. Impaired and delayed filling of intramural vessels of the jejunum and ileum with absent contrast within intramural vessels of the ascending colon is also seen. Note reflux into the aorta

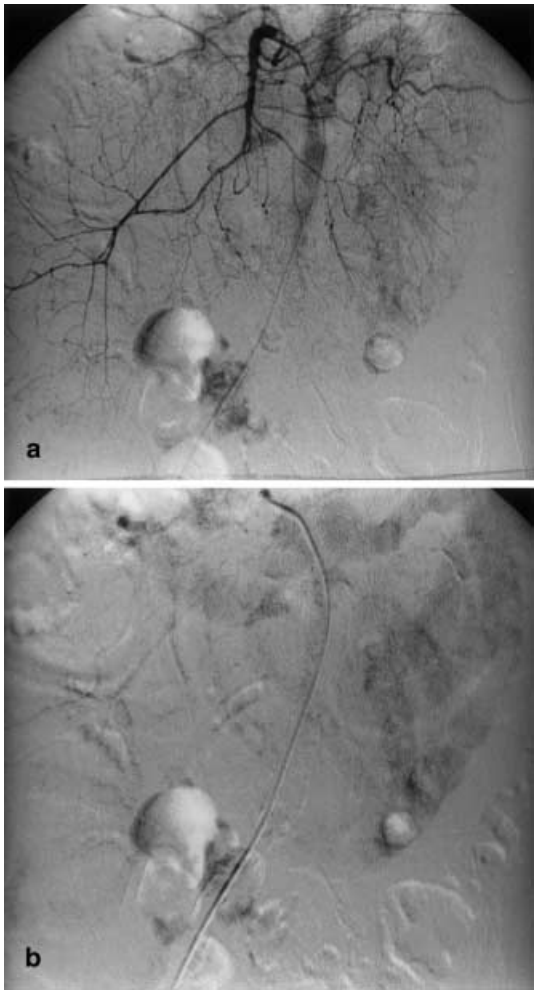

Fig. 5a, b Severe non-occlusive mesenteric ischemia. The selective SMA angiograms (30 ml, 5 ml/s) during a late **a** arterial phase and **b** venous phase exhibit vasoconstriction of all SMA branches with alternate dilatation and narrowing of small branches and impaired filling of intramural vessels and reflux into the aorta (**a**). Jejunal bowel loops show capillary filling during the venous phase (**b**) with absent contrast within mesenteric veins

3. Spasms of the mesenteric arcades (Fig. 4)
4. Impaired filling of intramural vessels (Fig. 5)

Other angiographic findings in NOMI are vasoconstriction of the distal trunk of the SMA and its branches, occlusion of distal parts of segmental arteries and intramural vessels, reflux of contrast into the abdominal aorta, and spread out segmental arteries due to distension of bowel loops [36, 38]. Impaired or missing filling of intramural vessels leads to delayed or impaired contrast in mesenteric veins (Fig. 5). Bruch et al. [2] regarded this a pathognomonic feature.

Such angiographic findings can vary from complete involvement of all mesenteric branches to a mere involvement of single branches. Only one of those signs needs to be present to make the diagnosis; however, none of them is absolutely specific for NOMI. Narrowing or alternate dilatation and narrowing of the intestinal branches can be caused by arteriosclerosis as well and then often presents as more eccentric narrowing. Delayed filling of the mesenteric veins must not be mistaken for mesenteric vein thrombosis.

Clark and Gallant [17] added diminished mesenteric blood flow as a criterion for diagnosing NOMI, using the "spillover" technique for quantitation. This method is re-

Table 3 Vasodilator therapy

Reference	Vasodilator	Dosage	Control
[41]	Tolazolin		
[43]	Phenoxybenzamin	Bolus of 0.2 mg/kg Infusion 0.7 mg/kg for 1 h	Angiography after 1 h
[3]	Prostaglandin E1	Bolus of 20 µg Infusion 60 µg/24 h for max. 72 h	Angiography after bolus and depending on clinical findings
[42]	Papaverine	30–60 mg/h max. 4 h	Angiography depending on clinical findings
[9]	Laevodosine	2 ml bolus, infusion 2.4 ml/h	Angiography after bolus

liable only if the estimated mesenteric blood flow is determined in relation to cardiac output. Moreover, there must be a defined position of the catheter tip in the SMA.

Therapy

In early non-occlusive mesenteric ischemia without mucosal necrosis as opposed to occlusive disease there is no surgical therapy. It is known that mesenteric vasospasm persists even after correction of the precipitating event [39]. This phenomenon of persistent prolonged vasoconstriction plays an important role in the development and maintenance of NOMI ischemia and may also complicate mesenteric revascularization [40]. The exact mechanism leading to the persistence of vasospasm is unknown, but it frequently responds to direct intra-arterial vasodilator infusion. Aakhus and Braband [41] described the infusion of tolazoline in 1967; Boley et al. [42] used papaverine in a protocol for the diagnosis and treatment of patients with acute mesenteric ischemia in 1977. In this study, any patient at risk of developing acute abdominal pain was started on an aggressive protocol. If plain-film radiography did not reveal a cause for their pain, angiography was performed and vasodilator therapy was instituted. Laparotomy was reserved for patients with acute arterial thrombosis, mesenteric arterial embolism, or persistent symptoms, despite intra-arterial papaverine. Using this approach, Boley and coworkers [39] reported a mortality rate of only 40% in their subset of patients with NOMI. Other authors who used papaverine or other vasodilators reported similar results. An overview to different vasodilator protocols is given in Table 3.

Presently, early angiography is performed in patients at risk where mesenteric ischemia is suspected and no signs of sepsis or peritonitis are present. If NOMI is diagnosed, vasodilator therapy will start, papaverine and prostaglandin being the vasodilators most commonly used. Angiographic control and monitoring of serum markers is performed, referring patients to laparotomy when there is no reaction to vasodilator infusion or if serum markers suggest necrosis or peritonitis. Intraoperative examination of bowel vitality using Doppler sonography or fluorescine injection and inspection with a Wood's lamp is occasionally performed [44]. Vasodilator therapy is continued until release of vasospasm or for a maximum of 72 h (Fig. 6) [3, 6]. We prefer infusion of

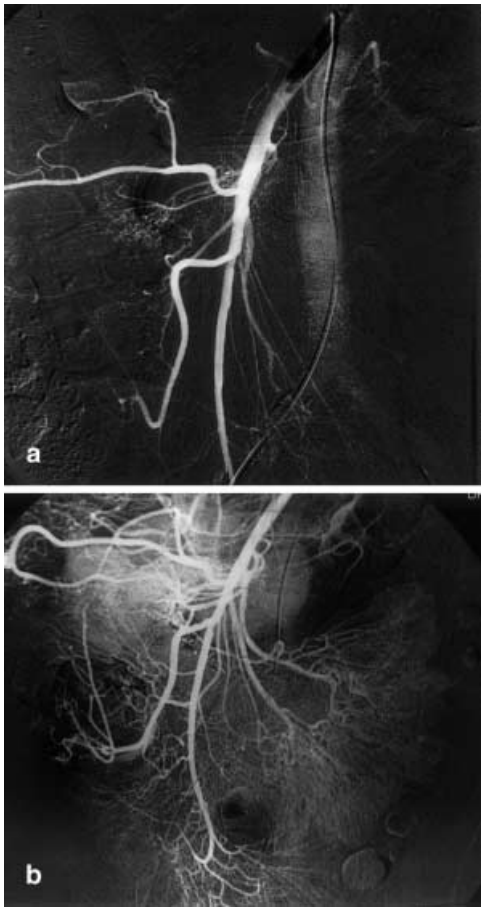

Fig. 6a, b Severe non-occlusive mesenteric ischemia: treatment. Selective angiograms of the SMA (25 ml, 5 ml/s) are obtained **a** before and **b** following infusion of prostaglandin for 72 h. Initial angiography shows extreme vasoconstriction of jejunal and ileal small bowel branches with alternate dilatation and narrowing of right colic branches and reflux into the aorta (**a**). Post-treatment angiography demonstrates a residual vasoconstriction of some jejunal and ileal branches with markedly improved filling of intramural vessels and absent reflux into the aorta (**b**)

prostaglandin with administration of an initial bolus of 20 µg and subsequent infusion of 2.5–5 µg/h for a maximum of 3 days according to Bruch et al. [3]. Angiographic control may be performed after initial bolus administration and if no improvement is observed following a 24-h interval.

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