Acute mesenteric ischemia : Classification, evaluation and therapy

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Abstract

Mortality rates of acute mesenteric ischemia still range between 60 and 100%. Unfortunately, retrospective series have not shown any significant improvement in mortality in the past decades. With approximately 50%, superior mesenteric artery (SMA) embolism is the most common form of acute mesenteric ischemia, followed by SMA thrombosis (~25%), nonocclusive mesenteric ischemia (~20%) and mesenteric venous thrombosis (~5%). Clinical presentation may be unspecific, but is often characterised by an initial discrepancy between severe subjective pain and relatively unspectacular findings on physical examination. The key to a better outcome (and the main problem in clinical practice) is early diagnosis. Up to now, helas, there are no simple and noninvasive diagnostic tests of sufficient sensitivity and specificity. Thus, angiography remains the cornerstone of diagnosis and should be performed early in all patients with a risk profile and a clinical presentation suspicious of AMI. The initial therapeutic step in all patients with AMI is resuscitation and a stabilization of circulation. If an advanced stage of ischemia is suspected, broad spectrum antibiotics have to be given. Nonocclusive mesenteric ischemia without signs of peritoneal infarction may be managed by pharmacological vasodilation, and vasodilators are also considered as a valuable supportive treatment option in patients with obstructive mesenteric ischemia. Patients with mesenteric venous thrombosis have to be treated by immediate anticoagulation, followed by laparotomy if peritoneal signs are present. Standard treatment for patients with obstructive mesenteric arterial syndromes is a laparotomy with embolectomy or revascularization and, if indicated, resection of infarcted bowel. – . This review will give an overview on the different forms of mesenteric ischemia and then focus on the diagnosis and on generally recommended forms of treatment. (Acta gastroenterol. belg., **2002**, 65, **220-225**).

Key words : mesenteric ischemia, nonocclusive mesenteric ischemia, angiography, mesenteric embolism, mesenteric thrombosis, mesenteric venous thrombosis.

Introduction

The clinical setting of acute mesenteric ischemia is characterised by the combination of a rather rare, difficult diagnosis, high fatality rates and the need for rapid and aggressive diagnostic and therapeutic interventions in often enough elderly and multimorbid patients. Thus, clinical management of patients with acute mesenteric ischemia is a challenging task and requires much experience and possibly dangerous decisions from the doctor in charge. In fact, mortality rates are still as high as 60 to 100% (1-4), and there seems to be very little progress in the last decades as far as this poor outcome is concerned. In a series of 102 patients of a Scottish teaching hospital published in 1987 (5), there was an overall mortality of 92%. In the same hospital, a very recently published retrospective series still showed similarly appalling results : Only 32% of patients were accurately diagnosed before operation or death, and 81% of patients died (3).

Furthermore, management of mesenteric ischemia is complicated by the fact that most of our knowledge is based on descriptive studies, "experience" and expert reports. There are no randomized controlled trials and only few non-randomized controlled trials and collaborator case-controlled trials with historical controls (6). Thus, from the viewpoint of "evidence based medicine", management of acute mesenteric ischemia is still a quite unsatisfactory field – and, considering the enormous difficulty to establish studies of this relatively infrequent and difficult to diagnose disease entity, it is quite probable that we will have to live with this uncertainty for the next years.

This review will give an overview on the different forms of mesenteric ischemia, then focus on the diagnosis and on accepted forms of treatment, mainly in accordance with the recently published official recommendations of the American Gastroenterological Association (6,7).

Classification and clinical presentation of acute mesenteric ischemia

Acute mesenteric ischemia may be caused by an impairment of arterial or venous mesenteric circulation. Fig. 1 gives an overview of the different forms of mesenteric ischemia. The vessel most commonly involved is the superior mesenteric artery (SMA). With an estimated proportion of 50%, SMA embolism is the most common form of acute mesenteric ischemia, followed by SMA thrombosis (~25%), non-occlusive mesenteric ischemia (NOMI, ~20%) and mesenteric venous thrombosis (~5%) (2,4).

Patients with *acute embolic obstruction* of the SMA characteristically are admitted with severe abdominal pain of sudden onset, generally located periumbilically or in the right umbilical fossa. Typically, this severe pain appears to be out of proportion to the rather innocent physical examination which may reveal a soft abdomen with an only discrete tenderness on palpation. Patients may complain about nausea, vomiting or diarrhea. A special effort should be made to take a detailed history

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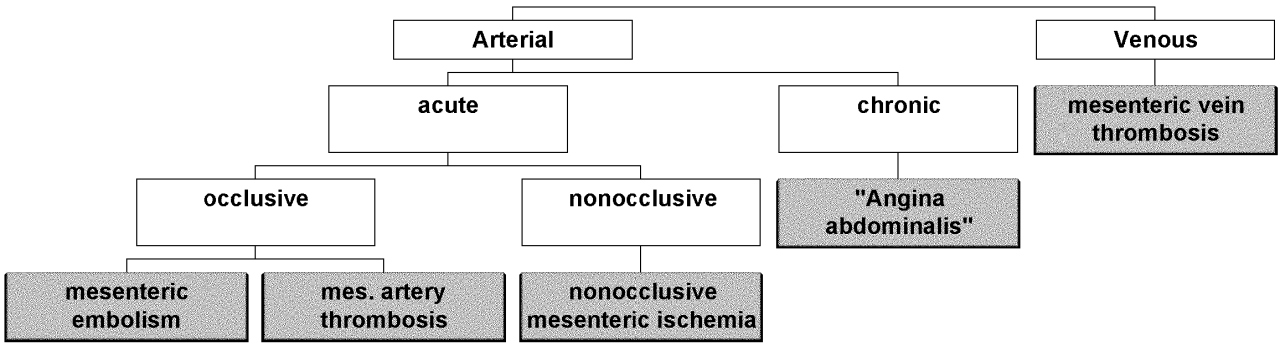

Fig. 1. — Subsets of mesenteric ischemia (adapted from (4))

on cardiac diseases such as previous myocardial infarction, arrhythmias or previous arterial and venous embolism and to note any signs of cardiac valve disease or arrhythmias.

Mesenterial arterial thrombosis is usually a complication of preexistent stenosing atherosclerosis of visceral arteries. Due to the formation of collaterals, symptoms of mesenteric infarction tend to develop more subacutely compared to mesenteric arterial embolism. There may be a history of postprandial abdominal pain and weight loss, suggestive of chronic mesenteric ischemia.

Non-occlusive mesenteric ischemia (NOMI) is probably still a rather underdiagnosed and underestimated condition. It is defined best by exclusion : It is *not* caused by atherosclerosis, arterial or venous thrombosis, embolism or vasculitis. Other terms which have been used to describe this condition such as functional mesenteric infarction, spastic mesenteric insufficiency or low flow syndrome give an impression of the suggested underlying pathophysiological mechanisms. NOMI is the result of a seriously diminished blood supply of the intestine produced by a systemic low flow syndrome with secondary mesenteric vasoconstriction. These changes in mesenteric hemodynamics may be caused by heart failure, hypotension or hypovolemia ; besides, secondary vasoconstriction may be augmented by the use of vasoconstrictive drugs such as catecholamines, vasopressin, digoxin, ß-blockers or even cocaine (8-10). Diagnosis of NOMI may be very difficult, as patients very often will already have been transmitted to the intensive care unit for other reasons and the clinical presentation may be dominated by the underlying disease.There is usually gradually increasing abdominal pain, but patients may as well complain about a discontinuous or colicky abdominal discomfort and other more or less unspecific gastrointestinal symptoms. If intestinal gangrene has occured, peritoneal symptoms and signs and possibly septicemia will prevail.

Mesenteric venous thrombosis is usually listed as one of the forms of mesenteric ischemia although, of course, pathophysiology of venous obstruction differs significantly from arterial mesenteric ischemia. Mesenteric venous thrombosis may show an extremely wide clinical spectrum, reaching from the completely asymptomatic patient who is diagnosed by chance to an acute, severe and lifethreatening disease. Half of the patients present with peritonitic signs and temperatures of more than 38° (11). Predisposing factors for mesenteric venous thrombosis include all states of hypercoagulability, portal hypertension, portal vein thrombosis, abdominal inflammations and a history of previous surgery or abdominal trauma (12).

Table 1. — **Relation between early diagnosis of acute mesenteric ischemia and survival (adapted from (6))**

		Mortality (%)	
Reference	No. of patients	Symptoms < 24 hrs	Symptoms > 24 hrs
Vellar (16)	52	54	95
Boley (13)	47	57	73
Inderbitzi (14)	83	0	88
Ritz (15)	141	44	92

Evaluation of acute mesenteric ischemia

There is general agreement that the main key to a better survival rate of AMI is early diagnosis, i.e. diagnosis before intestinal infarction has occured. Table 1 lists some retrospective studies (13-16) which clearly document the markedly improved outcome if diagnosis had been made within 24 hours of the onset of symptoms. At the same time, early diagnosis is the most difficult problem in clinical practice.

The recently published guidelines of the American Gastroenterological Association (AGA) recommend that all patients at risk who have abdominal pain severe enough to call to the attention of a physician and whose pain persists for more than 2 to 3 hours should be evaluated. Patients at risk have been identified as those older than 50 years with congestive heart failure, cardiac arrhythmias, recent myocardial infarction, hypovolemia, hypotension or sepsis. Besides, cardiac surgery and dialysis (for NOMI), a history of previous arterial

emboli, vasculitis, deep vein thrombosis, different hypercoagulable states or chronic postprandial pain have been defined as risk factors. However, as patients younger than 50 years and without any defined risk factors have been reported in series with AMI, the AGA-guidelines conclude that in any patient who has the classic early finding of severe abdominal pain out of proportion to the physical findings the diagnosis of AMI should be considered and pursued (6).

A sensitive and specific *biochemical marker* for intestinal ischemia, such as creatinkinase or troponin T for myocardial infarction, would, of course, help enormously in the struggle for early diagnosis, and huge efforts have been spent in the investigation of such serum markers. So far, however, expectations have not been fulfilled. In an extensive review on this subject, Kurland and colleagues (17) concluded that so far no single enzyme or combination of enzymes has proved to be sensitive or specific enough to enable the diagnosis of mesenteric ischaemia to be made sufficiently early to improve morbidity and mortality rates. Markers tested include creatinine kinase isoenzymes, lactate dehydrogenase, intestinal isoenzyme of alkaline phosphatase, diamine oxidase, hexosaminidase, asparate transferase, serum inorganic phosphate, serum D(-)-lactate or, very recently, D-dimers (18). However, elevations in most of these serum markers occur only after transmural bowel infarction has developed or only in part of the patients with mesenteric ischemia. The assays of some of the most promising experimental markers, such as intestinal fatty acid binding protein (19), currently take more than 12 hours and thus are of no practical value (6). Unspecific laboratory signs such as leukocytosis, neutrophilia or metabolic acidosis may be present. The determination of serum lactate is widely used if mesenteric ischemia is suspected (20,21), but, again, this parameter may lack specificity and sensitivity, and a normal lactate must not be interpreted as a valid exclusion criterion for mesenteric ischemia.

B-scan ultrasonography may reveal a thickening of bowel walls, signs of ileus or subileus, intraperitoneal fluid or even air in the portal tract as a sign of massive intestinal necrosis. As most of these findings are unspecific, however, the main aim of B-scan ultrasonography is to exclude other abdominal diseases such as an aortic aneurysm. *Duplex ultrasonography* has been reported to be highly specific for the identification of occlusions or severe stenoses of the splanchnic vessels and to have a sensitivity of 70-89% (6,22). However, ultrasound evaluation may be very difficult in patients with acute mesenteric ischemia due to significant meteorism, and it is not helpful in diagnosing emboli beyond the proximal main vessels or in NOMI.

Similar as for ultrasonography, the main reason for *plain abdominal roentgenograms* is to exclude other abdominal pathology. Highly suggestive, but rather uncommon and usually late signs include pneumatosis intestinalis, pneumoperitoneum, portal vein pneumatosis or mural "thumb printing", multiple round smooth soft-tissue densities projecting into the air-filled intestinal lumen which are elicited by submucosal edema or hemorrhage (23). *Computed tomography (CT)* carries the same problem of either unspecific or late signs of arterial mesenteric ischemia, although a recent study on the value of helical CT in mesenteric ischemia is somewhat more optimistic (24). The identification of mesenteric venous obstruction by CT, however, is generally regarded as reliable and considered as the diagnostic test of choice (12,25). Sensitivity of CT for mesenteric venous thrombosis lies between 90 and 100% (12,26). Thus, although the general use of CT scan for patients suspected to have any form of acute mesenteric ischemia is not supported in the literature, the use of a contrast-enhanced CT scan as the initial imaging study in patients with abdominal pain *and* a history of deep vein thrombosis or thrombophlebitis or a family history of a hypercoagulable state has been suggested (6,7). First studies of *magnetic resonance imaging angiography* have been performed and a high sensitivity and specificity were found for severe stenoses or occlusions of the proximal vessels (27) ; however, experience with and availability of this method are still too limited to recommend it in clinical practice.

Thus, the most reliable and definitive diagnostic method for arterial mesenterial ischemia remains *selective mesenteric angiography*. It allows an early diagnosis before infarction has occured, the etiologic classification of ischemia and the exact localisation of the obstruction. Besides, it may give valuable information for surgical treatment (embolectomy, revascularisation) or even be the first step for medical treatment by topic pharmacological vasodilation. The main arguments that have been brought forward against early routine angiography are the difficulties in performing angiography in critically ill patients, the large number of negative results if angiography is used in all patients considered as possibly suffering from mesenteric ischemia and, most importantly, the possible critical delay in surgical treatment if angiography is not readily available (6). Inspite of these drawbacks, the high sensitivity and specificity of angiography in a number of reported series with routine angiography and the decreased mortality rates in these series have prompted the American Gastroenterological Association to recommend an abdominal angiogram in all patients with a possible mesenteric ischemia, no peritoneal findings and no history of deep venous thrombosis or a familial hypercoagulable state (7) (fig. 2). The need for an angiogram in a patient with suspected acute mesenteric ischemia and peritoneal signs is still controversial. While supporters of angiography claim that a definitive diagnosis, the means to administer intraarterial vasodilators, the provision of a "roadmap" for revascularization procedures and the access for serial postoperative angiographic studies justify the procedure (6), others (including the

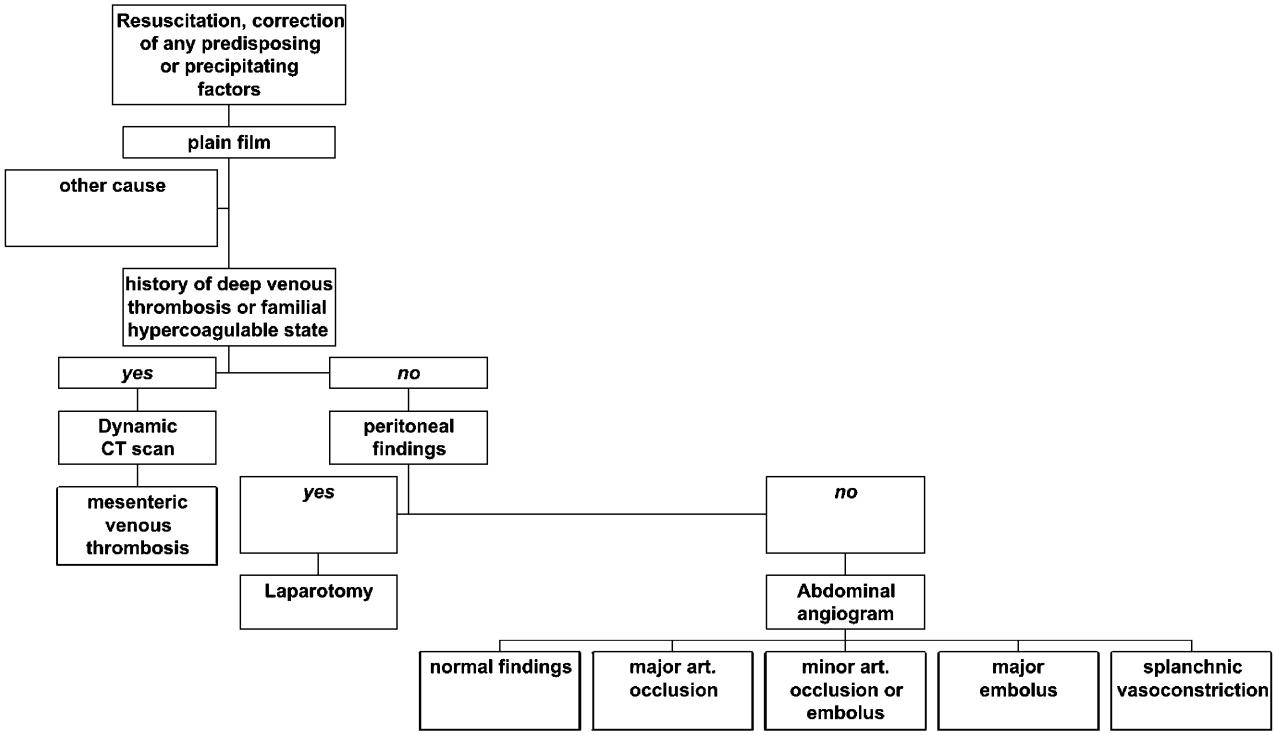

Fig. 2. — Evaluation of patients with suspected acute mesenteric ischemia (adapted from (7))

official recommendation of the AGA) recommend to procede directly to laparotomy.

Treatment of acute mesenteric ischemia

General treatment

The initial step in all patients with suspected acute mesenterial ischemia (before any further invasive diagnostic modalities !) is resuscitation with the replacement of lost fluid, correction of acid-base and electrolyte disturbances and, if necessary, optimization of cardiac output and treatment of hemodynamically relevant arrhythmias. Drugs acting as vasoconstrictors (vasopressin, digitalis) have to be discontinued. For hemodynamic monitoring, a Swan-Ganz catheter is recommended, if not possible, at least a central venous line. A nasogastric tube should be placed for gastric decompression. In patients with hypotensive or hypovolemic shock, there will always be mesenteric vasoconstriction ; thus, if there is a strong suspicion of NOMI, an angiography before correction of hypovolemia or hypotension will not be of diagnostic help. If an advanced stage of ischemia is suspected, broad spectrum antibiotics (e.g. a broad spectrum penicillin or a third generation cephalosporine in combination with metronidazole) have to be given, as ischemia will predispose to bacterial translocation and sepsis.

SMA embolus

If signs of peritonitis are present, exploratory laparotomy is mandatory, and embolectomy with resection of any infarcted bowel should be performed. If there are no peritoneal signs and angiography has demonstrated SMA embolism, the standard treatment still remains surgical embolectomy. In this setting, however, treatment with thrombolytic agents has been used with some success in a few reported cases and may be thought of as an alternative treatment option, especially if the embolus is only partially occluding, in one of the branches of the SMA or in the main SMA distal to the origin of the ileocolic artery (6,7). Repeat angiograms are necessary in theses cases.

Furthermore, infusion of papaverine has been used as the sole therapy in highly selected cases with minor or major emboli, although experience with this technique is still limited. Papaverine is a potent phosphodiesterase-inhibitor, leads to an increase in cAMP and thus mediates vasodilation. It is applied selectively into the SMA via the angiographic catheter usually as a continuous infusion of 30 to 60 mg/hr. During the infusion of papaverine, a continuous monitoring of heart rate, heart rhythm and blood pressure on an intensive care unit has to be guaranteed.

One of the most controversial points of the management of SMA embolism is the question if all patients should receive papaverine before and after embolectomy to treat associated mesenteric vasoconstriction (which may persist even after the removal of the embolus). Although there are no hard data to support this approach and recent animal experiments doubt a positive effect of papaverine in segmental mesenteric vascular occlusion (28), the AGA recommends a continuous papaverine infusion pre- and postoperatively for major SMA

embolism (7), the main argument for this being the better results in (unrandomized, uncontrolled) series where papaverine was used.

SMA thrombosis

Treatment of choice is emergency surgical revascularisation. Again, continuous preoperative papaverine infusion is recommended. So far, there are only few reports on the use of thrombolysis (29,30) or angioplasty (31) in SMA thrombosis, and these methods still have to be considered experimental.

NOMI

Like in the other forms of mesenteric ischemia, there are no randomized studies for the management of NOMI. Nevertheless, there is a broad consensus on the strategies to follow, and a number of smaller series have reported good success and a relatively positive outcome with this strategy (6-8,32-38). In accordance with the pathophysiological concepts, early treatment is based on two major principles : 1. correction of predisposing and precipitating factors, and 2. effective treatment of mesenteric vasoconstriction. Thus, the measurements outlined above under "general treatment" are of imperative and pivotal importance in the treatment of NOMI. As soon as a diagnosis of NOMI is made angiographically, a continuous papaverine infusion into the SMA should be established. Patients with peritoneal signs have to undergo laparotomy while receiving papaverine before, during and after surgery. In patients without peritoneal signs, treatment can be limited to vasodilator infusions into the SMA. Repeat angiograms are usually performed at 24 hour intervals, and papaverine is continued until radiographic signs of vasoconstriction have disappeared and the patient`s symptoms have vanished.

Superior mesenteric venous thrombosis

As outlined above, the clinical presentation of superior mesenteric venous thrombosis may vary significantly. In asymptomatic patients in which SMV thrombosis has been found "by chance", either no therapy or a 3-6 months course of anticoagulation may be established ; there are no data to support one of these two options (6). In symptomatic patients with SMV thrombosis, anticoagulation should be started immediately. If peritoneal signs are present, laparotomy with, if possible, thrombectomy and, if indicated, resection of infarcted bowel should be performed, followed by heparine and papaverine. In the absence of peritoneal signs, an immediate institution of anticoagulant therapy followed by clinical observation may suffice. Heparine has been recommended for 7 to 10 days, followed by an oral regimen of Coumadin for 3 to 6 months, although these, again, are not evidence-based data but mere conventions (6), and other authorities tend to recommend a lifelong anticoagulation for all patients with an episode of acute mesenteric venous thrombosis (12)**.** Alternative treatment options comprise thrombolytic therapy (applied via the SMA, retrograde via the internal jugular vein or transhepatically via the portal vein) which has been successful in some cases but is still controversial because of the risks of hemorrhagic infarction.

Conclusion

The main fact a doctor confronted with a patient with possible acute mesenteric ischemia has to be aware of is that this patient will probably die if he *does* suffer from acute mesenteric ischemia and treatment is initiated too late. Thus, the early inclusion of acute mesenteric ischemia in the differential diagnosis and an aggressive diagnostic and therapeutic approach seem to be the only ways to improve the catastrophic outcome figures of this condition. Pathological laboratory parameters such as marked leukocytosis, a metabolic acidosis or an increased serum lactate level may be interpreted as an argument for the need of mesenteric angiography, the gold standard in the diagnosis of acute arterial mesenteric ischemia. The absence of these pathological laboratory parameters, however, cannot rule out mesenteric ischemia. In patients with a reasonable possibility for mesenteric ischemia the possible benefit of angiography by far outweighs the risks of this procedure. Hence, justification is needed for the angiographies that have been omitted – not for those that have been performed !

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