

EDITORIAL



Managing acute mesenteric ischaemia

Annika Reintam Blaser^{1,2*} , Craig M. Coopersmith³  and Stefan Acosta⁴ 

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Acute mesenteric ischaemia (AMI) is a condition where different etiopathogenetic pathways may lead to necrosis of the bowel and thereby to lethal outcome. Despite the common threat of intestinal necrosis, different subtypes of AMI (occlusive arterial, occlusive venous and non-occlusive) have variable incidence, are managed differently and lead to different outcomes [1]. In this article, we summarize recent evidence related to diagnosis, management and outcomes of AMI.

Timely diagnosis

Diagnosis of AMI is complex due to different causative mechanisms and nonspecific clinical and laboratory features. In addition, patients with AMI are seen by different specialties, many of whom have insufficient experience with this infrequently encountered condition. AMI is diagnosed in 0.04–0.07% of adult hospital admissions, with a highly variable proportion (0–50%) of non-occlusive mesenteric ischaemia (NOMI) observed in different hospitals [1–4]. The variable incidence most likely indicates differences in awareness. Accordingly, it is imperative for intensivists to be aware of AMI—especially NOMI—as this condition is often underrecognized and underdiagnosed, and the intensive care unit (ICU) team often plays a key role in diagnosing and managing AMI. Clinical factors such as atrial fibrillation and acute abdominal pain in mesenteric arterial embolism or previous venous thromboembolism in mesenteric venous thrombosis can heighten suspicion for AMI (Fig. 1) [5]. Although the median age is around 70 years, AMI may occur in patients of any age [1].

Elevated lactate can support suspicion of AMI, whereas normal lactate should never be used to exclude AMI [1].

Appropriately raised suspicion should trigger an immediate referral for computed tomography (CT)-angiography, with possibility of AMI specifically mentioned for the radiologist, increasing chances for timely diagnosis [1, 6]. Importantly, increased creatinine level and risk of contrast-induced renal failure should never lead to omission of intravenous contrast in patients suspected of having AMI as timely diagnosis may allow early revascularization in acute superior mesenteric artery (SMA) occlusion with avoidance of bowel resection [7, 8]. In cases of NOMI, vascular findings on CT-angiography are less distinct. Notably, NOMI is not necessarily related to underlying atherosclerosis. Bowel findings on CT-scan are important to assist in clinical decisions regarding necessity of laparotomy [9].

Management

The ultimate target of management of AMI is to restore perfusion of the bowel before irreversible bowel damage has occurred. Therefore, immediate revascularization in acute SMA occlusion should have priority in management. However, studies demonstrate that patients commonly arrive to hospital 24 h after onset of symptoms [1, 3] and often already have irreversible bowel damage, requiring immediate laparotomy. Despite the need for bowel resection, the importance of revascularization should not be forgotten and should be attempted where applicable [10]. Stenting of the SMA for occlusive proximal arterial thrombotic AMI, aspiration embolectomy for embolic SMA occlusion, and thrombolysis with or without adjunctive endovascular therapy in patients with occlusive arterial AMI without peritonitis are good options for immediate interventional revascularization. Endovascular therapy only has a minor role in venous occlusion or NOMI.

Bowel resection is necessary in case of transmural necrosis [10]. However, the magnitude of necrosis may not be immediately obvious during operation, especially since ischaemia is more extensive at the mucosa side than

*Correspondence: annika.reintam.blaser@ut.ee

¹ Department of Anaesthesiology and Intensive Care, University of Tartu, Tartu, Estonia

Full author information is available at the end of the article

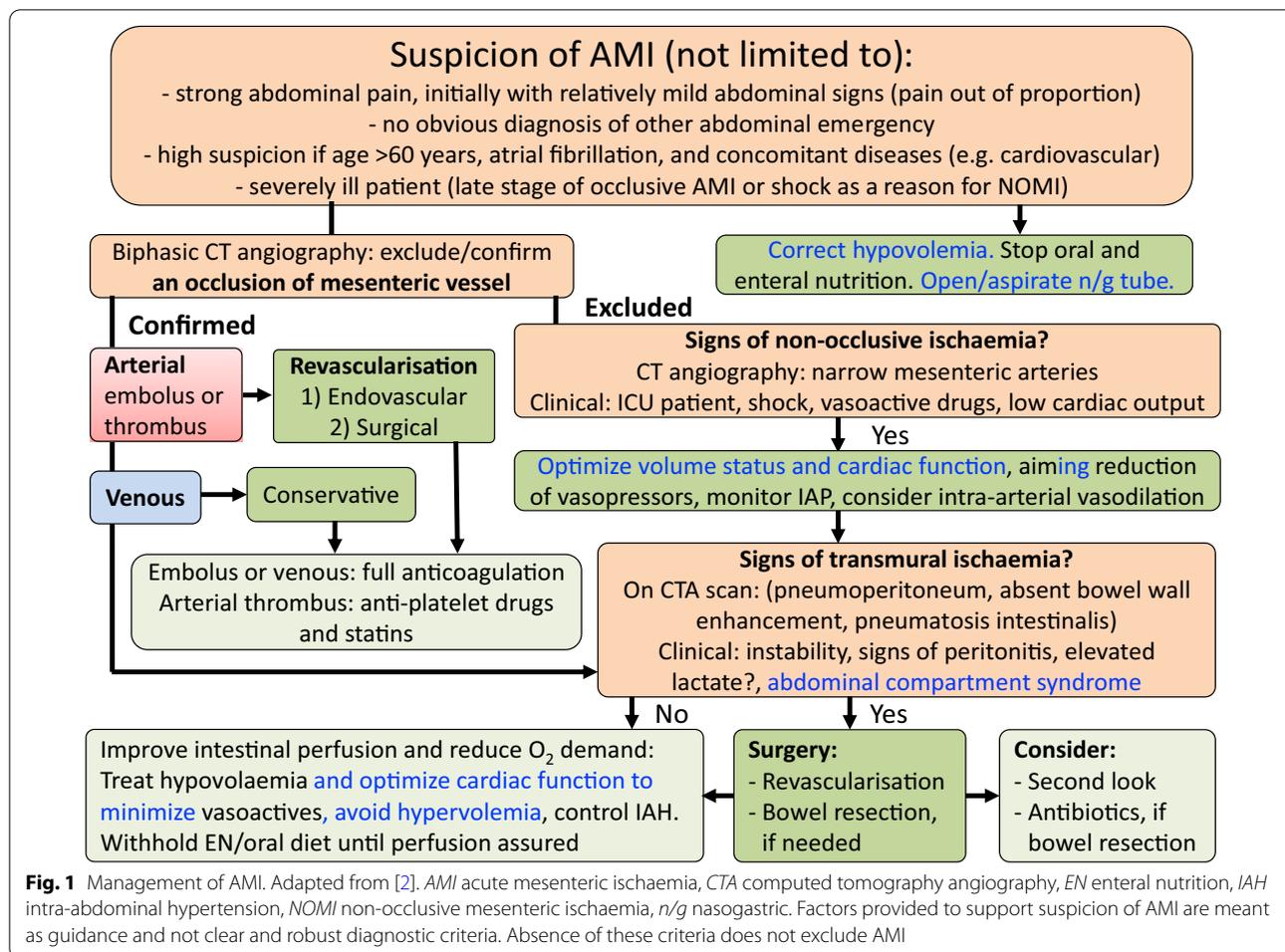


Fig. 1 Management of AMI. Adapted from [2]. AMI acute mesenteric ischaemia, CTA computed tomography angiography, EN enteral nutrition, IAH intra-abdominal hypertension, NOMI non-occlusive mesenteric ischaemia, n/g nasogastric. Factors provided to support suspicion of AMI are meant as guidance and not clear and robust diagnostic criteria. Absence of these criteria does not exclude AMI

the serosa side. As such, pre-planned second look with delayed abdominal closure has been suggested as a preferred strategy, although the benefit from this approach has not been proven.

Use of vasodilators in NOMI has shown potential [11], but diagnostic criteria and selection of patients is likely to play a role. Available scarce evidence does not allow clear guidance on indications and timing of intra-arterial mesenteric or systemic infusion of vasodilators, and concomitant vasoactive systemic management.

In the absence of any single variable that has appropriate precision to drive decision-making, we suggest actively assessing lactate, intra-abdominal pressure and organ dysfunction (e.g. circulatory, renal) in addition to clinical assessment of the abdomen to determine whether patients with NOMI require surgery.

In all subtypes of AMI, correction of hypovolemia and minimization of vasoconstriction through optimizing volume status and cardiac function is essential. Specific vasopressors or doses leading to or worsening NOMI are not clear and are likely highly dependent on concomitant

volume status. Accepting lower systemic blood pressure could potentially be dangerous in these patients, as they often also have intra-abdominal hypertension. In addition, oral intake and enteral nutrition should be immediately stopped due to the risk of increased oxygen demand of already ischaemic bowel [12].

The use of anticoagulation and antiplatelet therapy depends on the subtype of AMI (Fig. 1), with therapeutic anticoagulation being the main treatment for venous occlusive AMI. Antibiotic therapy should be administered to all patients with peritonitis undergoing bowel resection, whereas no guidance exists in patients without need of bowel resection [10, 13].

Although time to diagnosis and intervention is very important in the management of AMI, severity of bowel damage and selection of an optimal management strategy cannot be made based on time alone as time since symptom onset or from admission to diagnosis does not inherently alter outcome and has been shown to be similar in patients allocated to palliative compared to active treatment [1, 3]. Moreover, patients referred to a specialist

unit had the longest time from symptoms to treatment while simultaneously having the best outcome [1] with the caveat that they had lower disease severity and less laboratory abnormalities at admission. In addition, treatment success is influenced by the presence of collaterals and subtype of AMI.

Outcome

Mortality of AMI is globally still very high, with approximately 50% survival [1, 2, 4]. However, most patients receiving active treatment survive beyond their hospitalization [1, 3]. One-year survival greater than 50% has been reported with early revascularization [14]. Patients with AMI developing intestinal necrosis without receiving active treatment inevitably die, whereas many patients undergoing an extensive bowel resection survive, although this may be the price of having permanent short bowel syndrome and/or an intestinal stoma. Identifying cases in which active intervention should be denied due to lack of efficacy remains to be clarified.

Mortality is highest for NOMI and lowest for mesenteric venous thrombosis [1, 2, 4, 15]. Importantly, subtypes of AMI can be difficult to distinguish, with uncertainty occurring in more than 10% of cases, indicating the need for more clear nomenclature and guidance in diagnosis from a multidisciplinary point of view [1]. In addition, there is an ongoing debate about whether bowel ischaemia due to mechanical causes such as strangulating bowel obstruction should be considered as a subtype of AMI.

It is believed that accurate serum/plasma biomarkers (ongoing/planned studies NCT05194527, NCT06212921) for early diagnosis of AMI are necessary to substantially improve outcomes. At the same time, consensus on clinically useful definitions (including defining “suspicion”) of AMI and its subtypes is warranted.

Take-home message

A key to improve management of AMI is awareness, allowing adequately raised and communicated suspicion followed by early diagnosis and treatment. CT with intravenous contrast enhancement and imaging in the arterial phase followed by early revascularization in case of arterial occlusion carries the potential to rescue the bowel, whereas early systemic treatment optimizing volume status and organ perfusion is important for all subtypes of AMI, but most crucial for NOMI. Selection of initial management requires a multidisciplinary approach and cannot be based on time alone.

Author details

¹ Department of Anaesthesiology and Intensive Care, University of Tartu, Tartu, Estonia. ² Intensive Care Unit, Lucerne Cantonal Hospital, Lucerne, Switzerland.

³ Department of Surgery and Emory Critical Care Center, Emory University, Atlanta, GA, USA. ⁴ Department of Clinical Sciences, Lund University, Malmö, Sweden.

Declarations

Conflicts of interest

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