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REVIEW /*Gastrointestinal imaging*

Acute mesenteric ischemia: A critical role for the radiologist

P. Copin[a,b], **M. Zins**[c], **A. Nuzzo**[b,d], **Y. Purcell**[a], **S. Beranger-Gibert**[c], **L. Maggiori**[b,e], **O. Corcos**[d], **V. Vilgrain**[a,b,f], **M. Ronot**[a,b,f,*]

[a] *Department of Radiology, University Hospitals Paris Nord Val-de-Seine, Beaujon, 92110 Clichy, Hauts-de-Seine, France*
[b] *University Paris Diderot, Sorbonne Paris Cité, 75018 Paris, France*
[c] *Department of Radiology, Groupe Hospitalier Paris Saint-Joseph, 75014 Paris, France*
[d] *Department of Gastroenterology, University Hospitals Paris Nord Val-de-Seine, Beaujon, 92110 Clichy, Hauts-de-Seine, France*
[e] *Department of Colorectal surgery, University Hospitals Paris Nord Val-de-Seine, Beaujon, 92110 Clichy, Hauts-de-Seine, France*
[f] *Inserm U1149, centre de recherche biomédicale Bichat-Beaujon, CRB3, 92110 Paris, France*

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Abstract Acute mesenteric ischemia is defined as an inadequate blood supply to the gastrointestinal tract resulting in ischemic and inflammatory injury that may progress to necrosis of the bowel wall. Prognosis is poor with a mortality rate greater than 95% without treatment, dropping to around 70% when surgical treatment is performed. Contrast-enhanced computed tomography (CT) has become the cornerstone of the diagnosis by showing features of vascular disorders (occlusion and/or insufficient blood supply) and features of intestinal ischemic injury. CT should be performed as rapidly as possible. Imaging-based patient management is required, and multimodal and multidisciplinary management should be introduced. The treatment involves multidisciplinary management by gastroenterologists, vascular and digestive surgeons, cardiologists, intensivists, and diagnostic and interventional radiologists. Based on our experience at a dedicated mesenteric stroke center, this article gives an overview of the diagnosis of acute mesenteric ischemia. The goal of this review is to improve the understanding of the imaging-based diagnosis to further improve the management of this life-threatening condition.

* Corresponding author. Department of Radiology, Beaujon Hospital, AP—HP, 100, boulevard du Général-Leclerc, 92118 Clichy, France.
E-mail address: maxime.ronot@aphp.fr (M. Ronot).

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Acute mesenteric ischemia (AMI) is defined as the inadequate blood supply to the gastro-intestinal tract resulting in ischemic and inflammatory injuries that may progress to necrosis of the bowel wall. Vascular insufficiency can occur as a result of arterial embolism, arterial thrombosis, mesenteric venous thrombosis, or be non-occlusive [1]. Prognosis is poor with a 95% mortality rate without treatment, dropping to around 70% when surgical treatment is performed [2]. Survivors often have a poor functional outcome because of short bowel syndrome or malnutrition [3]. Early diagnosis of AMI is clinically difficult because symptoms are non-specific. As there are no readily available laboratory tests to accurately diagnose AMI, contrast-enhanced computed tomography (CT) has become the cornerstone of the diagnosis. A meta-analysis has reported a pooled sensitivity of 93.3%, and a pooled specificity of 95.9% for diagnosing primary AMI in symptomatic patients with multidetector CT [4], suggesting that it should be used as the first-line imaging technique.

The treatment of AMI is complex and is based on three complementary pillars including prevention of further aggravation, revascularization of non-necrotic bowel, and resection of necrotic bowel. Imaging-based patient management is required, and multimodal and multidisciplinary management should be the rule. As the prognosis is poor, treatment should start promptly.

This article provides an overview of the diagnostic process of AMI, with a focus on the central role of the radiologist for an early diagnosis.

Causes of vascular insufficiency

Arterial obstruction accounts for 60—85% of AMI [5]. It has an embolic origin in 40 to 50% of patients, and is due to thrombosis of a preexisting stenosis of the mesenteric artery in 20 to 35% of patients. Arterial dissection or vasculitis occurs in less than 5% of patients [5]. Patients are usually men with cardiovascular risk factors in the fifth decade.

Mesenteric venous thrombosis represents about 5—15% of AMI. Patients are usually younger (4th decade) and more frequently women. Mesenteric vein obstruction is usually due to clotting, and less likely, to malignant invasion of the portal vein. In most instances, a systemic condition is identified, such as prothrombotic states (myeloproliferative disorders, prothrombin gene mutation or antiphospholipid syndrome, cancer or any inflammatory/infectious condition of the bowel). Some patients also present with local causes, such as pancreatitis, or cirrhosis [6].

Lastly, AMI can be related to non-occlusive mesenteric ischemia. This etiology is still poorly understood and represents one of the most challenging presentations. It occurs during systemic or regional low flow states, i.e. shock, hemorrhage, surgery, dialysis, hypovolemia, cardiogenic injuries, vasoactive treatments, intoxications or intense exercise. Low-flow states activate the renin-angiotensin-aldosterone pathway that accentuates splanchnic vasoconstriction, which decreases regional blood flow under critical threshold [7].

Clinical and biological features

AMI is an extreme diagnostic and therapeutic emergency. As there are no strong clinical and biological findings, any sudden (''vascular''), unusual, intense abdominal pain should be suspicious of AMI. The pain can follow a period of mesenteric angina, often misdiagnosed. Since cardiovascular or thromboembolic associated comorbidities can be overlooked, especially in younger patients, such abdominal pain requires emergency multiphasic abdominal CT angiography [8]. Other associated signs (vomiting, diarrhea, digestive hemorrhage, hyperleukocytosis, lactic acidosis...) are inconsistent and/or too late and have low diagnostic value [3]. In a retrospective study of 221 patients followed in our intestinal failure unit for post-AMI short bowel syndrome, the initial history of these patients showed that they presented to the emergency room early with no peritoneal signs, no organ failure and normal serum lactate levels in 85%, 77%, 57% of cases, respectively [9]. Unrecognized at this stage, the diagnosis was carried out later, explaining that 184/221 (83%) of the patients required intestinal resection for necrosis. Thus, if most AMI patients seem to present at an early stage when physical examination is poor and biological tests falsely reassuring, diagnosis is reached later. As a result, AMI is characterized by two clinical pictures, early and late, defined respectively by the absence of any criterion or the presence of one of the following criteria of severity: (1) organ failure, (2) serum lactate levels > 2 mmol/L and (3) surgical complication (perforation, peritonitis, intestinal necrosis) [10]. These latter signs usually suggest the presence of irreversible transmural intestinal necrosis requiring surgical evaluation and resection [11]. Conversely, clinicians should not be reassured in the absence of any signs of clinical or biological severity. Indeed, diagnosis and treatment are even more urgent at an early stage when AMI is potentially completely reversible [11]. Left undiagnosed and/or untreated, early AMI progresses to late AMI, and subsequent complications and death.

Diagnostic imaging

AMI requires prompt diagnosis as well as imaging-based patient management. Diagnosis and staging are essential in the management of AMI, and for treatment planning. Several imaging examinations can be performed, but the cornerstone of the diagnosis is contrast-enhanced CT [12,13]. Its excellent reported sensitivity and specificity [4] suggest that it may be used as the first-line imaging technique. A recent study focused on the impact of contrast-enhanced multidetector CT on the survival in patients with acute superior mesenteric artery occlusion; in-hospitality mortality rate was 42% for patients who underwent contrast-enhanced multidetector CT, versus 71% for patients not examined with CT [14]. Another issue is radiologist expertise, as CT examinations require comprehensive evaluation of the GI tract and abdominal vasculature. This has been shown to have an important impact on CT performance [15]. Besides the