Acute mesenteric ischemia: How to diagnose it

Poster No.: C-2897
Congress: ECR 2019
Type: Educational Exhibit
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Keywords: Obstruction / Occlusion, Ischaemia / Infarction, Embolism / Thrombosis, Education, CT-Angiography, CT, Gastrointestinal tract, Emergency, Abdomen
DOI: 10.26044/ecr2019/C-2897

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Learning objectives

Acute mesenteric ischemia (AMI) is an important abdominal emergency because of its high mortality (50-90%) and therefore requires an early diagnosis and treatment.

The aim of this review is to learn the physiopathology and imaging findings of acute mesenteric ischemia, in order to be able to make a correct diagnosis early in the course of the disease, and avoid its serious complications.
Background

ANATOMY

Irrigation of the intestinal tract depends on three branches of the abdominal aorta: the celiac trunk, the superior mesenteric artery (SMA) and the inferior mesenteric artery (IMA).

The first and second portions of the duodenum are irrigated by the gastroduodenal artery, which most commonly arises from the common hepatic artery of the celiac trunk.

The third portion of the duodenum receives arterial blood from the inferior pancreaticoduodenal branches of the SMA.

The jejunum, ileum and caecum are irrigated by the AMS, as well as the ascending colon (by the right colic artery) and the transverse colon (by the middle colic artery).

Finally, the descending colon, the sigma and the upper portion of the rectum are irrigated by the IMA and its branches the left colic artery, sigmoid branches and superior hemorrhoidal artery respectively.

ETIOLOGY

AMI occurs because of an inadequate blood supply to the bowel, due to many different causes, that can be divided into occlusive or not occlusive.

In the first group, the occlusion can be either arterial (the most frequent of both) or venous; for the former, the most frequent cause is thromboembolism (40-50% of the cases), followed by thrombosis (20%), dissection (5%), arteriosclerosis, postsurgical complications, vasculitis and fibrous dysplasia, while the latter is mainly caused by venous thrombosis, hypercoagulability states, infiltrative tumors, inflammation or abdominal infections.

For the second group, non-occlusive reduced intestinal blood flow may be caused by systemic low blood flow due to systemic shock, cardiac failure, dehydration, stress, drugs or pheochromocytoma, or it can be caused by mechanical causes, such as bowel strangulation or marked abdominal distension. An inadequate blood supply to the bowel
may also occur after irradiation, trauma, immunodepression, chemotherapy, tumors or abdominal inflammation like pancreatitis, appendicitis, diverticulitis and peritonitis…

FISIOPATHOLOGY

The first layer of the bowel wall that is affected by ischemia is the mucosa. At this point, the lesion is reversible and can cure without significant consequences.

If the ischemia continues, the necrosis will affect the submucosa and the muscular layer, and in this case, residual fibrosis may develop after the ischemic process is resolved.

Lastly, the necrosis process may reach the serosa, affecting the whole intestinal wall (transmural necrosis). This is an emergency, which requires urgent surgery.

CLINICAL AND ANALYTICAL FINDINGS

The prevalence of AMI increases with age so it should be suspected on elderly patients with cardiovascular risk factor (arteriosclerosis, arrhythmia, DVT, etc.).

Symptoms and signs are often nonspecific, such as acute abdominal pain, abdominal distension, gastrointestinal bleeding (resulting in anemia), leukocytosis with neutrophilia, increased D-dimer… The elevation of alkaline phosphatase, LDH, amylase or lactic acidosis is indicative of established intestinal necrosis.
Findings and procedure details

Abdominal X-ray and ultrasound can be useful to suspect AMI within the correct clinical context, but the absence of abnormal findings does not exclude the diagnosis. Typical X-ray findings include bowel dilatation, portomesenteric gas, pneumatosis intestinalis and pneumoperitoneum, whereas ultrasound findings include parietal thickening of a long segment of intestine with diminished or absent Doppler flow.

Multiphase CT of the abdomen and pelvis is the imaging modality of choice in the assessment of patients with suspected AMI. An arterial phase is done triggered automatically by a real-time bolus tracking technique with the ROI in the origin of the abdominal aorta when the aorta reaches >100 HU. The portal venous phase is done 30 seconds after the arterial phase finishes. A phase without contrast and the use of oral contrast are optional.

Typical findings in the CT for the diagnosis of AMI are bowel dilatation, abnormal bowel wall enhancement, thickening or thinning of the bowel wall, intramural bowel gas (pneumatosis intestinalis), gas in the mesenteric vein or in the portal vein (pneumatosis portalis), ascites and mesenteric edema. The perforation of the wall may cause pneumoperitoneum.

In some cases, the cause can be identified and the occlusion of the mesenteric vessels or the portal vein can be seen.

Examples of patients with acute mesentric ischemia due to different causes:

CASE 1: Venous thrombosis. 69-year-old woman with abdominal pain during 3 weeks, which got worse in the last 5 days accompanied with diarrhea and leukocytosis. (Fig. 1, 2, 3 and 4)

CASE 2: Bowel obstruction and dilatation secondary to colon cancer. 73-year-old with abdominal pain and distension for the last 2 days accompanied with nausea and vomiting. Slight leukocytosis with neutrophilia and increased CRP and lactate levels. (Fig. 5 and 6)

CASE 3: Hypovolemic shock. 80-year-old patient with systemic shock and abdominal pain. (Fig. 7 and 8)
CASE 4: **Arterial thrombosis.** 81-year-old man with a history of a cardioembolic stroke who presents abdominal pain, leukocytosis, increased levels of CRP and alkaline phosphatase after an episode of atrial fibrillation. (Fig. 9, 10, 11 and 12)

CASE 5: **Arterial vasoconstriction due to drugs (noradrenaline).** 48-year-old woman admitted to the hospital with respiratory insufficiency and fever of 40º. After a neuroleptic syndrome she is relocated to the Intensive Care Unit in a state of coma. A blood test shows leukocytosis and increased levels of LDH and alkaline phosphatase. (Fig. 13 and 14)
Fig. 1: Coronal image. Thrombosis of the superior mesenteric vein (arrow).

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Fig. 2: Axial image. Thrombosis of the superior mesenteric vein (arrow).

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**Fig. 3:** Portal vein thrombosis (asterisk) and ascites.

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Fig. 4: Thickening and decreased enhancement of the intestinal walls as signs of ischemia. Ascites.

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**Fig. 5:** Colon cancer (arrow) which causes obstruction and dilatation of the ascending colon and ileum.

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Fig. 6: Intramural bowel gas (pneumatosis intestinalis).

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Fig. 7: Collapsed inferior vena cava and high enhancement of the adrenal glands (arrow) as signs of systemic shock.

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**Fig. 8:** Pneumatosis portalis (red arrow), pneumoperitoneum (green arrow) and ascites.

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Fig. 9: Coronal image. Thrombosis of the superior mesenteric artery (arrow).

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**Fig. 10:** Axial image. Thrombosis of the superior mesenteric artery (arrow).

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**Fig. 11:** Hyper-enhancement of the wall of a segment of bowel.
**Fig. 12:** Thickening of the intestinal wall.

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**Fig. 13:** Pneumatosis intestinalis (green arrow) and gas in the mesenteric veins (red arrow).

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Fig. 14: Bowel dilatation, pneumatosis intestinalis and portomesenteric vein gas.

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Conclusion

AMI is an abdominal emergency and it is important for radiologists to be familiar with the image manifestations in order to make a quick diagnosis and treatment because it has a high mortality rate.

It occurs because of an inadequate blood supply to the bowel, due to many different causes, that can be divided into occlusive (arterial or venous) or not occlusive (hypoperfusion, vasoconstriction, etc...).

It is important to suspect this pathology after a clinical evaluation and blood test (abdominal pain, raised lactate, metabolic acidosis...) in order to decide the best image technic.

Multiphase CT of the abdomen and pelvis is the imaging modality of choice for the diagnosis of AMI. Its findings may not be specific, but often the combination of them within the clinical context are at least suggestive of the diagnose.
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