Non-occlusive mesenteric ischemia. CT findings and most frequent risk factors in 70 patients.

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Learning objectives

The purpose of our educational exhibit is to:

- Review 70 cases of non-occlusive mesenteric ischemia (NOMI).
- Describe the most frequent risk factors.
- Depict the most common CT findings.
Background

With the increase in average life expectancy, acute bowel ischemia represents one of the most threatening abdominal conditions in elderly patients. Acute bowel ischemia may involve the small or large bowel, be segmental or diffuse, and be only partial mural (meaning that it involves only the mucosa and submucosa, with or without parts of the muscularis) or transmural (meaning that it leads to continuous necrosis of all bowel wall layers). Ischemic colitis, as an example of only partial mural and superficial colonic ischemia, is the most common type of colitis in patients older than 50 years and is often self-limiting, whereas acute bowel infarction (accounting for approximately 1% of all cases of acute abdomen) has a higher annual mortality rate than colon cancer.

Non-occlusive mesenteric ischemia (NOMI) comprises all forms of mesenteric ischemia without occlusion of the mesenteric arteries and accounts for 20-30% of all cases of acute mesenteric ischemia. The clinicopathologic diagnostic criteria of NOMI include no occlusion of the mesenteric artery or vein in the area of bowel necrosis, the presence of ischemic and necrotic spots and segments that are distributed over a wide area in a non-consecutive manner, and histopathological findings including hemorrhagic and necrotic changes, as well as small veins without fibrin plugs.

NOMI is commonly caused by decreased cardiac output resulting in splanchnic hypoperfusion. It generally affects patients over 50 years of age suffering from ischemic heart disease, congestive heart failure, renal and peripheral artery disease and patients following cardiac/vascular surgery.

During a 50% decrease in superior mesenteric artery (SMA) blood flow, the initial response of the mesenteric circulation is autoregulatory vasodilatation, which results in a decrease in mesenteric vascular resistance and an increase in collateral mesenteric blood flow through the celiac artery. After several hours of diminished SMA blood flow, autoregulatory systems are overridden and mesenteric arterial resistance begins to rise as a result of active mesenteric vasoconstriction. If the 50% occlusion of the SMA is released as soon as mesenteric vasoconstriction begins, this vasoconstriction is readily reversible. If, however, mesenteric vasoconstriction is allowed to continue for 30 min or longer, it persists even after blood flow through the SMA is restored.

NOMI is a condition characterized by high morbidity and mortality rates. Reasons for low survival rates are advanced age and the difficulty of diagnosis because the symptoms of NOMI are nonspecific, and 20-25% of NOMI patients do not have abdominal pain, which makes the diagnosis of NOMI more difficult. Tolerance to ischemia of the intestine
is limited and becomes critical after 3-6 h, therefore, an early diagnostic workup and immediate therapy is essential for a successful outcome.
Findings and procedure details

We analyzed retrospectively clinical and imaging data of 70 patients with the diagnosis of non-occlusive mesenteric ischemia from our hospital from January 1st 2008 to August 31st 2015.

RISK FACTORS AND CLINICAL DATA

NOMI is commonly caused by a decrease in cardiac output that results in splanchnic hypoperfusion. Reported causes of mesenteric vasospasm are myocardial infarction, congestive heart failure, renal or hepatic disease, digitalis, various forms of shock, septicemia, dehydration and hypotension following dialysis, and heart and major abdominal surgery.

In our study, searching the clinical history of the 70 patients, we found the following risk factors probably associated with NOMI:

- Congestive heart failure (43%).
- Chronic renal failure (39%).
- Ischemic heart disease (33%).
- Cardiac arrhythmia (27%).
- Peripheral artery disease (19%).
- Recent heart / vascular surgery (3% - 2 cases).

The most common risk factor encountered was congestive heart failure, affecting 30 patients. Its association with NOMI is well known given that in these cases small changes in blood volume or heart rate can lead to an exacerbation of their heart failure and therefore a decrease in cardiac output, which may lead to NOMI. Ischemic heart disease and cardiac arrhythmia are likely to be related to congestive heart failure. A fact to be taken into account is that 18 patients had a history of atrial fibrillation. Atrial fibrillation is usually associated with acute mesenteric ischemia (AMI) secondary to arterial embolism, but perhaps in NOMI patients atrial fibrillation is just a finding secondary to cardiac structural abnormalities in the context of a congestive heart failure (e.g. dilation of the left atrium in mitral valve stenosis or insufficiency).

Chronic renal failure and more frequently patients in hemodialysis (HD) are at greater risk of suffering NOMI. In our study, 27 patients had chronic renal failure 12 of whom had diabetes mellitus and 5 were in hemodialysis. Previous studies showed that the incidence of mesenteric ischemia in HD patients ranges from 0.3 to 1.9% per patient-year. Acute NOMI tends to occur in patients who are on HD for a long time and who
are advanced in age. Intradialysis and/or chronic hypotension are also associated with mesenteric ischemia.

Atherosclerosis is the leading cause of peripheral arterial disease of the extremities in patients over 40 years of age with the highest incidence in the sixth and seventh decades of life. Peripheral artery disease has as one of its main features the presence of calcified atherosclerotic plaques along the vessels, not only affecting the extremities but also the abdominal descending aorta and its splanchnic branches (celiac artery and superior and inferior mesenteric arteries). This feature along with stenosis of the branches origin may lead more easily to splanchnic hypoperfusion (Fig. 1).

Acute mesenteric infarction after extracorporeal circulation is an infrequent but catastrophic event that constitutes 5% to 27% of all intraabdominal complications occurring on an active cardiac service. Although thrombotic and emboli mesenteric ischemia occurs, NOMI is the cause in 25% to 75%.

According to previous reports, digitalis is an additional risk factor for NOMI, perhaps because it induces vasoconstriction and thus increases resistance in peripheral splanchnic vessels. In this study, no patient was being treated with digitalis.

Age is also a risk factor of NOMI, mainly because all the aforementioned risk factors are more frequent at an older age. In our study, the mean age of the patients was 79,6.

IMAGING FINDINGS

Siegelman et al. addressed angiographic criteria for the diagnosis of mesenteric vasospasm:

1. Narrowing of the origins of multiple branches of the SMA.
2. Alternate dilatation and narrowing of the intestinal branches, the so-called string of sausages sign.
3. Spasms of the mesenteric arcades.
4. Impaired filling of intramural vessels.

Catheter angiography, while previously considered to be the standard for making an imaging diagnosis of mesenteric ischemia, is not available in many centers and takes time to arrange and perform. Computed tomography (CT) has been reported to represent an excellent diagnostic tool for the early and accurate detection and localization of acute mesenteric ischemic changes. Moreover, with maximum intensity projection (MIP) reconstructions in the CT angiographic phase it is possible to obtain images very simmilar to the conventional angiography, with the typical Siegelman criteria (Fig. 2). In addition,
unlike catheter angiography, CT findings make it possible to rule out other disorders in attempts to reach a differential diagnosis in patients with mesenteric ischemia.

CT Protocol

In our study, all patients underwent multidetector computed tomography (MDCT) scanning on a 16- or a 64-detector.

They received 110 mL of non-ionic intravenous contrast agent at a rate of 4 mL/sec. Initially, a 2 mm slice CT angiographic phase acquisition was performed with a region of interest (ROI) placed in the abdominal descending aorta (above the celiac trunk) with a delay of 10 seconds when the Hounsfield units (HU) reached the threshold of 150. After this, a conventional 2 mm slice portal phase acquisition was obtained 70 seconds after injection of the contrast agent began.

CT Findings

The angiographic CT phase ruled out occlusion of the main visceral arteries (Fig. 3), the celiac trunk and the superior and inferior mesenteric arteries, as well as the main visceral veins, therefore excluding arterial or venous thrombosis as the cause of the mesenteric ischemia.

CT portal venous phase was evaluated for evidence of bowel wall thickening (defined as a wall thickness of more than 3 mm, Fig. 4), mucosal enhancement (Fig. 5), focal lack of bowel wall enhancement (Fig. 6), bowel dilatation (Fig. 7), mesenteric stranding (Fig. 8), ascites (Fig. 9), solid organ infarction (Fig. 10), free intraperitoneal air (Fig. 11), pneumatosis intestinalis (Fig. 12), superior mesenteric or portal venous gas (Fig. 13).

In our study, all 70 patients had CT evidence of mesenteric ischemia in both small bowel and/or colon. Small bowel ischemia usually presents as lack of bowel wall enhancement, dilation and in more advanced cases, pneumatosis intestinalis and portal venous gas. In our study, 37 patients had evidence of small bowel ischemia, the jejunum was affected in 19 cases (Fig. 14), the ileum in 32 (Fig. 15) and both jejunum and ileum in 14 patients (Fig. 16). Therefore, the primary location of small bowel ischemia was the ileum.

Hyperemia and hyperperfusion of an ischemic bowel segment may be diffuse or primarily involve the mucosa and submucosa; in the latter conditions, hyperemia and hyperperfusion may cause a typical target sign in addition to the surrounding mural
edema. Contrarily, hyperenhancement of the bowel wall in shock bowel indicates neither hyperemia nor hyperperfusion but typically corresponds to prolonged enhancement of the bowel wall due to reduced arterial perfusion after vasospasm of the mesenteric arteries caused by the effects of angiotensin II or after reduced venous outflow due to contraction of mesenteric veins caused by the effects of adrenaline and noradrenaline.

On the other hand, **large bowel non-occlusive ischemia** normally appears as ischemic colitis. Wall thickening, fat stranding, and abnormal wall enhancement are the most frequent findings on CT (Fig. 17). There are two putatively high-risk areas for ischemic colitis known as "watersheds" in the literature; the sigmoid-rectal junction at the distal limit of inferior mesenteric artery perfusion (Fig. 18), and Griffith's point at the splenic flexure (Fig. 19), representing the junction between blood supplies, from the superior mesenteric artery and the inferior mesenteric artery. In our study, the primary location of large bowel ischemia was the left colon (23 cases, Fig. 20), whereas the right colon was affected in 10 cases and both left and right colon was detected in 13 patients (Fig. 21).

**Pneumatosis intestinalis** refers to gas within the wall of the bowel and represents a mucosal injury secondary, in these cases, to mucosal infarction and leads to leakage of gas within the bowel wall (Fig. 22). We found pneumatosis intestinalis in 19 cases (27%). **Portal venous gas** is the accumulation of gas in the portal vein and its branches, usually representing a progression of the pneumatosis intestinalis gas to the mesenteric veins and finally to the portal venous system (Fig. 23). Must be distinguished from aerobilia (Fig. 24), as the latter is typically seen as linear branching air within the liver, most prominent in central large calibre ducts as flow of bile pushes gas toward the hilum, in contrast to portal venous gas where peripheral small calibre branching air is usually seen due to flow of blood out from the hilum. In our study, portal venous gas was present in 18 patients.

Complications such as **bowel perforation** was detected in our study in 4 cases (Fig. 25). In addition, **solid organ infarction** is also a common CT finding when analyzing NOMI. We found out that 6 patients had areas of lack of enhancement in a wedge-shape distribution in the liver (Fig. 26), 6 cases affecting the spleen (Fig. 27) and in 5 patients bilateral renal infarctions were detected (Fig. 28). In one case emphysematous cholecystitis (Fig. 29) was detected on CT and surgery and pathology findings confirmed the diagnosis of gallbladder wall ischemia.

**TREATMENT AND PROGNOSIS**

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. This phenomenon of persistent prolonged vasoconstriction plays an important role in the development and maintenance
of NOMI ischemia and may also complicate mesenteric revascularization. The exact mechanism leading to the persistence of vasospasm is unknown, but it frequently responds to direct intra-arterial vasodilator infusion (conservative treatment), referring patients to laparotomy when there is no reaction to vasodilator infusion or if serum markers or CT findings suggest necrosis (e.g. complete lack of wall enhancement or pneumatosis intestinalis / portal venous gas) or peritonitis (Fig. 30).

In our study:

- 36 (51%) patients were treated conservatively.
- 34 (49%) underwent urgent abdominal surgery.

Of the 36 treated conservatively, 19 patients, in spite surgery being the first therapeutic option, were rejected for surgical treatment because of multiple factors, mainly advanced age or signs of very extensive ischemia. All these 19 cases died within 6 days. Therefore in 17 patients conservative treatment was the first therapeutical option with high survival rates and 9 of them underwent a colonoscopy that confirmed the diagnostic suspicion of ischemic colitis.

34 patients underwent urgent abdominal surgery, where in most cases resection of large segments of ischemic bowel was performed. 4 patients died within the next 3 days, and the other 30 cases survived at least until hospital discharge. Histopathologic detection of hemorrhagic and necrotic changes is required for definite diagnosis of NOMI and all of them had the histological confirmation of mesenteric ischemia.

Another fact to be taken into account is that the mean time between the completion of CT and surgery was 5 hours, with a median of 3 hours. Surgical intervention is necessary if there is intestinal necrosis or frank perforation or when there is clinical deterioration over a 12 to 24-hour period despite intensive medical support, as evidenced by persistent or worsening metabolic acidosis. In these cases, the sooner the surgery is performed, the better the long-term results.
**Fig. 1:** 68-year-old female. Sagittal maximum intensity projection (MIP) contrast-enhanced CT (CECT) at angiographic phase shows atheromatous calcium plaques in the abdominal aorta (yellow arrow) and in the origin of the celiac trunk and superior mesenteric artery (red arrows). Note the nasogastric tube (green arrow).

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Fig. 2: Female, 55 years old. Coronal MIP CECT at angiographic phase shows permeability of the celiac trunk (green arrow) and the superior mesenteric artery (red arrow). Note the narrowing of the origins of multiple jejunal branches of the SMA and subtle dilatation and narrowing (yellow arrows), similar to the string of sausages sign.

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**Fig. 3:** 65-year-old male. Coronal volume rendering (VR) CECT at angiographic phase demonstrates permeability of the celiac trunk (yellow arrow), superior mesenteric artery (red arrow) and inferior mesenteric artery (blue arrow). Abdominal aorta calcified plaques are seen (green arrow).

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**Fig. 4:** 77-year-old female with left ischemic colitis. Axial CECT at portal venous phase shows a hypodense splenic flexure wall thickening with the 'target sign' (yellow arrows). Ischemic colitis was confirmed in colonoscopy.

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Fig. 5: Male, 60 years old with diffuse NOMI. Coronal MPR CECT at portal venous phase shows a mucosal hyperenhancement affecting the stomach (red arrow), jejunum (yellow arrow) and ascending colon (green arrow).

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Fig. 6: 79-year-old male. Axial CECT at portal venous phase demonstrates a lack of bowel wall enhancement of a jejunum loop (red arrow) compared with the normal wall enhancement (yellow arrow).

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Fig. 7: Male, 73 years old. Coronal MPR CECT at portal venous phase shows a diffuse bowel dilation and mural thickening (red arrow). Note the lack of wall enhancement in the pelvic ileum (yellow arrow).

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Fig. 8: 83-year-old male. Axial CECT at portal venous phase depicts a splenic flexure mesocolon fat stranding (yellow arrow). Colonic splenic flexure wall thickening is seen (red arrow). Colonoscopy confirmed the diagnosis of ischemic colitis.

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**Fig. 9:** 68-year-old female with extensive NOMI. Axial CECT at portal venous phase shows a large amount of peritoneal fluid at perihepatic and perisplenic locations (yellow arrows). A focal loss of parenchymal enhancement is seen in the spleen, with ill-defined borders, consistent with a splenic infarction (red arrow).

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Fig. 10: Female, 68 years old. Axial CECT at portal venous phase demonstrates multiple focal areas of lack of parenchymal enhancement, with wedge-shape and ill-defined borders affecting the liver (red arrow) and spleen (yellow arrow), consistent with parenchymal infarctions.

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Fig. 11: 81-year-old male with proximal ileum ischemia. Coronal MPR CECT at portal venous phase shows extraluminal air following the mesenteric vessels (yellow arrows), indicating bowel perforation.

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**Fig. 12:** Male, 81 years old. Sagittal MPR CECT at portal venous phase depicts an ileum loop dilatation, mesenteric stranding and pneumatosis intestinalis (yellow arrows).

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Fig. 13: 85-year-old female. Axial CECT at portal venous phase shows gas within the portal vessels consistent with portal venous gas (yellow arrows). Note the lack of mural enhancement, dilatation and pneumatosis intestinalis in the splenic flexure of the colon (red arrow).

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**Fig. 14:** 81-year-old female. Sagittal MPR CECT at portal venous phase demonstrates a jejunum loop ischemia. Lack of wall enhancement (yellow arrow) and pneumatosis intestinalis (red arrow) are seen.

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**Fig. 15:** Female, 73 years old. Axial CECT at portal venous phase shows lack of mural enhancement and pneumatosis intestinalis in a pelvic ileum loop (yellow arrow) indicating ischemia. Note the gas in the mesenteric veins (red arrow).

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Fig. 16: 81-year-old female. Coronal MPR CECT at the portal venous phase depicts an extensive NOMI affecting the jejunum (yellow arrow) and the ileum (red arrows), as we can see lack of mural enhancement, dilated loops, mesenteric stranding and pneumatosis intestinalis. There is also a liver infarct (green arrow).

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**Fig. 17:** Female, 77 years old with left ischemic colitis. Axial CECT at portal venous phase demonstrates an engorgement of the colonic splenic flexure with edematous mural thickening (yellow arrow), left mesocolon fat stranding (red arrow) and mucosal hyperenhancement (green arrow). Ischemic colitis was confirmed in colonoscopy.

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Fig. 18: Female, 88 years old. Sagittal MPR CECT at portal venous phase shows findings consistent with sigmoid-rectal junction colitis. There is wall thickening and mucosal hyperenhancement (red arrow), free fluid in the presacral space (yellow arrow) and mesosigmoid fat stranding (green arrow). Colonoscopy confirmed the suspected diagnosis of ischemic colitis.

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Fig. 19: Female, 74 years old. Axial CECT at portal venous phase demonstrates a splenic flexure colitis with edematous mural thickening (red arrow), pericolonic fat stranding (green arrow) and mucosal hyperenhancement (yellow arrow). Ischemic colitis was confirmed afterwards in colonoscopy.

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Fig. 20: 72-year-old female. Coronal MPR CECT at portal venous phase shows an extensive left colitis, affecting the whole descending colon (yellow arrows). Ischemic colitis was confirmed in colonoscopy.

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**Fig. 21:** Female, 86 years old. Sagittal MPR CECT at portal venous phase shows findings consistent with right colitis. There is wall thickening and mucosal hyperenhancement (red arrow), free fluid in the posterior pararenal space (yellow arrow) and pericolonic fat stranding (green arrow). Again, colonoscopy confirmed the diagnosis of ischemic colitis.

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Fig. 22: 66-year-old female with non-occlusive mesenteric ischemia affecting the ileum. Coronal MPR CECT at portal venous phase demonstrates an ileum loop mural abnormalities: paper-thin wall with lack of enhancement (red arrow) as well as intramural bowel gas consistent with pneumatosis intestinalis (yellow arrow). These findings are very suggestive of mesenteric ischemia.

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**Fig. 23:** Female, 73 years old with NOMI. Axial CECT at portal venous phase depicts gas within the left portal veins (yellow arrow) consistent with portal venous gas and a large hepatic infarct seen as a hypodense area with ill-defined borders affecting hepatic segments III and IV (red arrow). Note the intramural stomach gas and lack of mucosal enhancement (green arrow). Stomach ischemia was confirmed at surgery.

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Fig. 24: LEFT: 75-year-old female with a history of pancreaticoduodenectomy (Whipple procedure). Axial CECT at portal venous phase demonstrates an accumulation of air in the biliary tree (yellow arrows) consistent with aerobilia. RIGHT: 88-year old female. Axial CECT at portal venous phase depicts the accumulation of gas in the portal vein branches (yellow arrows). Note the choledocolithiasis (red arrow) and jejunum mesenteric ischemia (green arrow).

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Fig. 25: 83-year-old male with ischemic colitis. Sagittal MPR CECT at portal venous phase shows inframesocolic (yellow arrow) and supramesocolic (red arrow) free...
peritoneal air (pneumoperitoneum), suggesting bowel wall perforation as an ischemic colitis complication.

**Fig. 26:** Female, 81 years old. Axial CECT at portal venous phase demonstrates a large wedge-shape lack of parenchymal enhancement in the anterior portion of the liver (yellow arrows) consistent with infarction. Note the portal venous gas (red arrow).

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Fig. 27: Female, 68 years old with extensive NOMI. Coronal MPR CECT at portal venous phase shows numerous hypodense lesions with ill-defined borders within the peripheral splenic parenchyma consistent with splenic infarcts (yellow arrows). Note the periesplenic (red arrow) and perihepatic fluid.

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Fig. 28: Female, 67 years old. Axial CECT at portal venous phase shows a wedge shape hypodense lesion within the lateral cortical parenchyma of the mid portion of the left kidney (yellow arrow). This CT appearance is very suggestive of renal infarct. Pericolonic fat stranding and lack of enhancement of the hepatic flexure of the colon is seen (red arrow).

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Fig. 29: 66-year-old male with diffuse mesenteric ischemia affecting both the small bowel and colon. Coronal MPR CECT at portal venous phase shows gas in the superior mesenteric vein (green arrow) and portal venous gas (red arrow). There is also gas in the wall of the gallbladder (yellow arrows) and without history of gallbladder manipulation, this finding is very suggestive of emphysematous cholecystitis. At surgery, mesenteric ischemia and gallbladder ischemia were confirmed.

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Fig. 30: 78-year-old male. LEFT: coronal MPR CECT at portal venous phase shows pneumatosis intestinalis and lack of mural enhancement of a large segment of the ileum (yellow arrows). Free peritoneal fluid is seen (red arrow). RIGHT: axial CECT at portal venous phase demonstrates portal venous gas (green arrow). Mesenteric ischemia was confirmed at surgery and resection of 95 cm of ileum was performed.

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Conclusion

Non-occlusive mesenteric ischemia (NOMI) is an acute mesenteric circulatory disorder that does not involve the organic occlusion of blood vessels. Its common cause is a decrease in cardiac output resulting in splanchnic hypoperfusion and it accounts for 20-30% of acute bowel ischemia.

In our study the main risk factors for developing NOMI were advanced age, congestive heart failure, chronic renal failure, ischemic heart disease, cardiac arrhythmia, peripheral artery disease and recent heart / vascular surgery.

70 patients were diagnosed with NOMI after performing an abdominopelvic CT with intravenous contrast and two phases, one angiographic to rule out occlusive mesenteric ischemia and afterwards a portal venous phase with the common CT findings of NOMI.

The most common location of NOMI was the ileum (32 cases), however, we identified more cases of ischemic colitis (46) than small bowel ischemia (37).

Approximately half of the patients underwent an urgent abdominal surgery and the other half received conservative treatment.

At the end of the day, because its symptoms are nonspecific and it frequently occurs in elderly patients or with postoperative consciousness disturbance, early diagnosis of NOMI is difficult. Within 3-6 h, intestinal ischemia becomes critical, and the morbidity rate is high.
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