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Acute Mesenteric Ischemia: Basic concepts, Diagnosis, Treatment Options and Outcomes

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ABSTRACT

Acute mesenteric ischemia (AMI) is a group of diseases in which blood flow to the intestine is interrupted by occlusive or nonocclusive causes that include mesenteric arterial embolism, mesenteric arterial thrombosis, and mesenteric venous thrombosis. AMI risk factors include advanced age, Caucasian race, history of systemic arterial hypertension, arterial fibrillation, chronic kidney disease, and congestive heart failure. The clinical triad is characterized by the sudden onset of severe abdominal pain accompanied by gastrointestinal symptoms in the presence of an identified embolic source. Diagnosis is commonly made using computed tomography angiography, where the obstruction is observed directly in occlusive causes. Treatment involves fluid resuscitation and restoring mesenteric artery circulation using endovascular or surgical procedures. The mortality rate varies depending on the patient's clinical history and treatment approach, with a higher risk associated with open surgery. Lifelong anticoagulation or antiplatelet therapy may be necessary to prevent recurrence. Follow-up evaluations are recommended to monitor for restenosis and to prevent acute myocardial infarction.

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INTRODUCTION

Acute mesenteric ischemia (AMI) is caused by a decrease in upper mesenteric artery-dependent blood perfusion, which supplies the third and fourth portions of the duodenum, jejunum, ileum, and colon proximal to the splenic curvature. The risk of ischemia is highest at the splenic flexion, ileocecal junction, and rectosigmoid junction. [(1)] The leading causes of mesenteric ischemia are embolism or thrombosis of the superior mesenteric artery and mesenteric venous thrombosis. [(2)]

According to the *World Journal of Emergency Surgery*, mesenteric ischemia is a group of diseases in which blood flow is interrupted to irrigate parts of the intestine and can originate from occlusive or nonocclusive causes. The primary etiology can be divided into mesenteric arterial embolism (50%), mesenteric arterial thrombosis (15 - 25%), or mesenteric venous thrombosis (5 - 15%). [(3)]

The incidence of acute mesenteric ischemia ranges between 5.3 and 8.4 cases per 100,000 inhabitants, with a prevalence of up to 0.1%. The risk of developing this condition increases

with age. Most patients develop the disease between the ages of 60 and 70, with an average age of 67. Women account for more than 70% of cases of mesenteric ischemia, but most of them are chronic, whereas acute mesenteric ischemia affects men and women equally. [4]

Demographic risk factors for AMI include advanced age, female gender, and Caucasian race. Additionally, systemic arterial hypertension, atrial fibrillation, chronic kidney disease, congestive heart failure, peripheral vascular disease, chronic obstructive pulmonary disease, coronary artery disease, smoking, hyperlipidemia, and diabetes are associated. [6]

In 20% of cases, thrombosis in the superior mesenteric artery is a complication of atherosclerotic occlusion of the mesenteric vessels. This can lead to localized atherosclerosis in the coronary and cerebral arteries and peripheral vascular disease. [7]

PHYSIOPATHOLOGY

Mesenteric arterial embolism.

Around 50% of cases of intestinal mesenteric artery ischemia occur because of acute embolism of the superior mesenteric artery. The embolus can originate from the left atrium (e.g., atrial fibrillation), left ventricle (e.g., ventricular dysfunction with diminished ejection fraction), heart valves (e.g., endocarditis), or, less frequently, an atherosclerotic aorta. [3]

The embolus is lodged in the superior mesenteric artery due to its large diameter and low angle. Usually, emboli lodge between 3-10 cm below the origin of the superior mesenteric artery, which can affect the jejunum and proximal colon. [3]

Mesenteric arterial thrombosis.

Thrombosis of the superior mesenteric artery is associated with chronic atherosclerotic diseases leading to stenosis. Thrombosis usually occurs at the origin of the visceral arteries. An underlying plaque of the superior mesenteric artery eventually progresses to a critical stenosis, forming collateral vessels. If the ileocolic artery is involved, this will result in necrosis of the proximal colon. [3]

Acute nonocclusive mesenteric ischemia.

Acute nonocclusive mesenteric ischemia is caused by vasoconstriction of the superior mesenteric artery associated with a low flow of the splenic circulation. Insufficient blood flow will affect the proximal colon due to the involvement of the ileocolic artery. These patients usually have a severe pre-existing disease (e.g., heart failure), which sepsis can aggravate. Hypovolemia and the use of vasoconstrictors may precipitate an acute nonocclusive mesenteric ischemia. [3]

Mesenteric venous thrombosis.

This etiology is attributed to the components of the *Virchow* triad: stagnant blood flow, hypercoagulability, and endothelial damage. An inflammatory process around the superior mesenteric vein caused by pancreatitis or inflammatory bowel disease, surgical trauma such as a splenectomy, or bariatric surgery can lead to thrombosis and AMI. [3]

CLINICAL PRESENTATION

Embolism of the mesenteric artery causes acute, diffuse, and severe abdominal pain. [Table 1]. This pain is part of the clinical triad, which includes gastrointestinal symptoms (nausea, vomiting, and diarrhea) in the presence of an identified embolic source. The disease presentation depends on the location and size of the obstruction. The clinical onset can be divided into two phases. In the initial phase, the abdomen will be soft and painless, and upon auscultation, hyperactive intestinal sounds may be heard; patients will also experience a slight temporary improvement followed by later progress towards peritonitis, as palpation pain occurs when the entire intestinal wall becomes ischemic. In the advanced stage (12 hours after onset), compression sensitivity (Blumberg's sign) and stiffness will indicate a bowel wall infarction. In this phase, the intestinal noises will be

Table 1. Acute mesenteric ischemia manifestations presented at hospital admission

CLINICAL FINDINGS	FREQUENCY (%)
Abdominal pain	100 %
Diarrhea or vomiting	84 %
Previous episode of embolism	33 %
Hematoquezia	25 %
Elevation of lactate in plasma	90 %
Leukocytocis	65 %
Metabolic acidosis	60 %

hypoactive or absent. The patient may develop hematochezia, fever, and shock. [8,9]

Different clinical characteristics can be observed based on the classification of the AMI. Mesenteric arterial embolism presents with sudden pain, a pain-free interval 6 - 12 hours after onset, nausea, vomiting, diarrhea, and rectorrhagia in up to 15% of the cases. Gradual postprandial pain, nausea, and diarrhea occur in mesenteric arterial thrombosis. Mesenteric venous thrombosis presents a non-specific abdominal pain of subacute and periumbilical onset; only 9% of patients will have symptoms of less than 24 hours. Nonocclusive mesenteric ischemia will present with increased abdominal pain, gradual or acute, periumbilical, and constant colic. Patients will present with metabolic acidosis and hypotension. [10]

DIAGNOSIS

There are no serological tests available for early detection of AMI. However, specific indicators can be observed in laboratory studies. These indicators include neutrophilic leukocytosis, hemoconcentration, elevated lactic acid levels, metabolic acidosis, increased amylase levels, as well as elevated lactate dehydrogenase (LDH) and aspartate aminotransferase (AST). Notably, increased levels of lactic acid may also be associated with decreased intravascular volume and renal lesions. [8] The liver can metabolize large amounts of lactate, and elevated serum levels manifest when ischemia progresses to transmural infarction. [11] Diagnosis may be supported by a history of atrial fibrillation or a recent embolic or ischemic event. [8]

On the other hand, different radiological techniques can be used to diagnose intestinal abnormalities. For instance, simple abdominal radiography can help identify intestinal dilation, and in advanced cases, edema of the intestinal wall, intraperitoneal free gas, portal venous gas, and intestinal pneumatosis may be observed. Doppler ultrasound allows visualization of the celiac trunk and the superior mesenteric artery, with a high specificity within 92 - 100% but a lower sensitivity of 70-89%. [11]

Multidetector computed tomography with intravenous iodine contrast is the preferred imaging technique for diagnosis due to its high sensitivity and specificity, which are close to 100% based on studies. [11,12] It also allows us to rule out other differential diagnoses. [Table 2]. [11, 13]

Computed tomography angiography is helpful for diagnosis, with a sensitivity of 85-98% and specificity of 91-100%. Obstruction is observed directly in occlusive processes such as embolism or thrombosis. However, in nonocclusive mesenteric ischemia, the diagnosis is supported by four findings: 1. Narrowing originating from the superior mesenteric artery, 2. Irregularities in its branches, 3. Spasms of the vascular arch, 4. Reduction in the filling of intramural vessels. [8]

	FINDINGS	ETIOLOGY
Intestinal wall	• Narrowing of the wall • Wall thickening	Arterial isquemia
Bowel attenuation on a CT scan without contrast	• High density (due to bleeding)	All ischemia
Caption of contrast at the intestine	• None	All ischemia
	• Higher contrast caption	Reperfusion, venous ischemia, early bowel obstruction
	• Prolonged contrast caption	Intestinal obstruction (due to a reduction in arterial perfusion and venous flow)
Pneumatosis	• Air in the intestinal wall	All ischemia
Mesenteric trabeculation	• Increased attenuation of mesenteric fat	Reperfusion, venous ischemia
Diminished superior mesenteric vein	• The diameter of the superior mesenteric vein is lower than that of the superior mesenteric artery	Arterial isquemia
Others	• Ascitis	Reperfusion, venous ischemia

Table 2. Tomographic findings in mesenteric ischemia.

STAGING

Mesenteric ischemia is classified into three stages based on the extent of involvement of the intestinal wall. Stage I is characterized by necrosis, erosions, ulcerations, edema, and localized hemorrhage in the mucosa. It is considered a reversible disease and can be cured spontaneously without sequelae. In Stage II, necrosis is already extending to the submucosal and muscular layers, which can lead to fibrotic

stenosis. Finally, in stage III, necrosis affects all layers of the intestinal wall (transmural necrosis) and has a high mortality rate. [11]

TREATMENT

Treatment begins with immediate fluid resuscitation, administering isotonic crystalloids, and correcting electrolyte imbalances. Before considering surgical intervention, it is advisable to monitor diuresis and maintain control of blood pressure to ensure optimal parameters. The main goal of management is to restore circulation in the mesenteric artery using endovascular approaches or surgical procedures, all before irreversible intestinal damage is established. When transmural intestinal necrosis occurs, intestinal revascularization may reduce tissue damage and promote recovery. [14]

Optimal treatment involves both surgical and endovascular options. When patients present with peritonitis, elevated lactate levels, metabolic acidosis, and advanced signs of intestinal ischemia on computed tomography, exploratory laparotomy is considered the preferred treatment. The objectives of this procedure are to restore blood flow in the mesenteric artery, remove non-viable intestinal tissue, and preserve the still functional intestine. In patients who are stable with transmural intestinal necrosis, revascularization is performed before definitive bowel surgery, as the extent of irreversible bowel injury may be difficult to assess before blood flow restoration. [14]

*Embolectomy***.**

After laparotomy, the superior mesenteric artery can be accessed by making a horizontal incision in the peritoneum at the base of the transverse mesocolon. The artery is located to the superior mesenteric branch's left and is divided into several branches. It is important to isolate these branches using blood vessel loops to prevent hemorrhage. A transverse arteriotomy is performed through the superior mesenteric artery, and the embolectomy is performed with a balloon catheter of 3 or 4 F. After the embolism is removed, the pulsating flow of blood must be observed. The arteriotomy is then closed using prolene sutures, ensuring proper closure. [8]

Endovascular aspiration thrombectomy.

This procedure is available for patients who do not exhibit signs of peritonitis and can be performed through either brachial or femoral access routes. However, using the femoral artery to access the superior mesenteric artery is more favorable. The artery is catheterized during the procedure, and a 0.035 fully hydrophilic guide wire is passed through it. Once the wire is in place, an introductory sheath of 6 or 7 F of 60 cm with a removable bucket or an 8-9 F guide catheter is passed into the superior mesenteric artery. The catheter is designed to remove blood clots using manual or electric vacuum aspiration with a 20ml locking syringe, followed by angiography. Risks of endovascular aspiration thrombectomy include the spread of distal thrombus to the mesentery's peripheral and smaller arterial branches. Careful examination of the patient for any clinical deterioration is mandatory after successful aspiration of the embolus. [8]

Local intraarterial thrombolysis.

This treatment option involves minimal invasion and is suitable for elderly patients with multiple comorbidities. It can complement incomplete aspiration embolectomy after endovascular aspiration thrombectomy or as the primary treatment for patients who do not have peritonitis or advanced intestinal ischemia. A multi-sided hole infusion catheter is inserted into the main ileocolic trunk of the superior mesenteric artery, and a thrombolytic agent is administered in continuous infusion. Thrombolytic agents include recombinant tissue plasminogen activator (tPA) infused at a rate of 0.5 to 1 mg/h or urokinase (120,000 IU/h); progress is angiographically evaluated every 12 hours or otherwise depending on the patient's clinical status. [8]

Anterograde recanalization and superior mesenteric artery stent.

Endovascular treatment of atherosclerotic superior mesenteric artery occlusion is often performed after the removal of the clot by aspiration or thrombolysis. Percutaneous transluminal angioplasty with stenting may be achieved through femoral or brachial access points. Once the surgeon gains access to the ileocolic artery with a stable 0.035-inch cable, an introductory sheath is inserted beyond the atherosclerotic lesion. A balloon stent is deployed at the occlusion site, followed by sheath retraction to expose the stent. The lesion is then treated with a 7/8 mm diameter stent. The result is confirmed by repeat angiography. [8]

Bypass from the superior mesenteric artery.

When an endovascular approach is not possible in the context of atherosclerotic occlusion, or if the embolectomy fails to restore the inflow, surgical bypass revascularization may be performed. Options for approach include access from the external iliac artery, common iliac artery, or infrarenal aorta. Shunt to the superior mesenteric artery alone in AMI is preferred over multivessel shunt graft. Most authors prefer an autologous vein graft in the AMI due to fear of peritoneal contamination and subsequent graft infection. All patients undergoing laparotomy for suspected AMI should be prepared to include both lower extremities of the knee. This allows for possible collection of saphenous or femoral veins if necessary. [8]

OUTCOMES

The European Society of Vascular Surgery conducted a study that showed an overall mortality rate of 30 days after endovascular revascularization of 17.2%, compared with 38.5% after open surgery. Five years after endovascular revascularization and open surgery, survival was 40% and 30%, respectively. [3]

Perioperative mortality in patients undergoing revascularization ranges from 44 - 90%. The outcome is not

as favorable for patients with chronic mesenteric ischemia. Recurrence is very common, leading to a poor prognosis. [15] Nonocclusive mesenteric ischemia is associated with high mortality as it affects older adults with comorbidities. [8]

On the other hand, patients above 60 years increased the mortality rate with a relative risk ratio of 3.0. A recent surgery history increased the death rate by 2.4 times. Bowel resection in the first or second intervention reduced the mortality rate with a relative risk ratio of 0.5. [16]

Patients who underwent extensive bowel resection usually developed short bowel syndrome, significantly increasing their mortality; they require a long-term total parenteral feeding or a small intestine transplant. Likewise, digestive continuity should be restored with hormonal therapy to optimize absorption function and achieve nutritional autonomy. Multiple studies have analyzed that using a GLP-2 analog significantly reduced total parenteral nutrition dependence and improved quality of life in these patients. [3,15]

Most patients treated with AMI will require lifelong anticoagulants or antiplatelet therapy to prevent the disease from coming back. In patients who have received an endovascular stent, clopidogrel is given for six months, and acetylsalicylic acid as a lifelong maintenance treatment**.** **[3]** It is crucial to conduct follow-up evaluations to assess the restenosis of the stent or graft. This is important because acute myocardial infarction after mesenteric revascularization accounts for 6-8% of late deaths. *The Vascular Surgery Society* suggests conducting duplex ultrasonography one month, six months, and twelve months after the procedure, followed by annual evaluations. [3]

CONCLUSION

Acute mesenteric ischemia is a disease characterized mainly by vascular compromise of the superior mesenteric artery. Given the suspected diagnosis, it is important to know the epidemiology and the multiple risk factors that predispose to this clinical condition and to perform a thorough physical examination, because the symptoms are usually non-specific in many cases. In addition, we must know the diagnostic tools available in our environment to make a timely diagnosis (abdominal computed tomography, angiography), and know the therapeutic options offered in our environment for rapid approach and management from the emergency medical service. All of this will result in an increase in the survival of affected patients, with a possible decrease in post-surgical complications or endovascular treatment, offering a better quality of life as a result.

REFERENCES

I. Cano Matías A, Marenco de la Cuadra B, Sánchez Ramírez M, Retamar Gentil M, Pérez Margallo E, Oliva Mompeán F, et al. Isquemia mesentérica aguda: un desafío aún no resuelto. *Cir Andal*. 2019;30(1):57-65.

II. Fernández S., Lázaro P., Rodríguez P., Sánchez Y., Sanz E., Nitza J. & González A. (2018). Diagnóstico y tratamiento de la isquemia mesentérica aguda por oclusión vascular. *Correo Científico Médico*, *22*(3), 514-529. http://scielo.sld.cu/scielo.php?script=sci_arttext&pid=S1560-43812018000300013&lng=es&tlng=es.

III. Bala, M., Catena, F., Kashuk, J., De Simone, B., Gomes, C. A., Weber, D., Sartelli, M., Coccolini, F., Kluger, Y., Abu-Zidan, F. M., Picetti, E., Ansaloni, L., Augustin, G., Biffl, W. L., Ceresoli, M., Chiara, O., Chiarugi, M., Coimbra, R., Cui, Y., … Moore, E. E. (2022). Acute mesenteric ischemia: updated guidelines of the World Society of Emergency Surgery. *World Journal of Emergency Surgery*, *17*(1). https://doi.org/10.1186/s13017-022-00443-x

IV. Molyneux, K., Beck-Esmay, J., Koyfman, A., & Long, B. (2023). High risk and low prevalence diseases: Mesenteric ischemia. *The American Journal of Emergency Medicine*, *65*, 154–161. https://doi.org/10.1016/j.ajem.2023.01.001

V. Rodríguez M., Oslanyer A., & Delgado C. (2022). Morbilidad y mortalidad por isquemia mesentérica aguda en el Hospital Universitario "Arnaldo Milián Castro". *Revista Cubana de Cirugía*, *61*(3), e1386. Epub 30 de noviembre de 2022. http://scielo.sld.cu/scielo.php?script=sci_arttext&pid=S0034-74932022000300007&lng=es&tlng=es.

VI. Chou, E. L., Wang, L. J., McLellan, R. M., Feldman, Z. M., Latz, C. A., LaMuraglia, G. M., Clouse, W. D., Eagleton, M. J., & Conrad, M. F. (2021). Evolution in the presentation, treatment, and outcomes of patients with acute mesenteric ischemia. *Annals of Vascular Surgery*, *74*, 53–62. https://doi.org/10.1016/j.avsg.2021.01.116

VII. Fernández S., Lázaro P., Rodríguez P., Sánchez Y., Sanz E., Nitza J. & González A. (2018). Diagnóstico y tratamiento de la isquemia mesentérica aguda por oclusión vascular. *Correo Científico Médico*, *22*(3), 514-529. http://scielo.sld.cu/scielo.php?script=sci_arttext&pid=S1560-43812018000300013&lng=es&tlng=es.

VIII. Gnanapandithan, K., & Feuerstadt, P. (2020). Review article: Mesenteric ischemia. *Current Gastroenterology Reports*, *22*(4). https://doi.org/10.1007/s11894-020-0754-x

IX. Ahmed, M. (2021). Ischemic bowel disease in 2021. *World journal of gastroenterology: WJG*, *27*(29), 4746–4762. https://doi.org/10.3748/wjg.v27.i29.4746

X. Navas-Campo, R., Moreno-Caballero, L., Ezponda Casajús, A., & Muñoz, D. I. (2020). Isquemia mesentérica aguda: Revisión de las principales

técnicas y signos radiológicos. *Radiologia*, *62*(5), 336–348. https://doi.org/10.1016/j.rx.2020.02.001

XI. Yikilmaz, A., Karahan, O. I., Senol, S., Tuna, I. S., & Akyildiz, H. Y. (2011). Value of multislice computed tomography in the diagnosis of acute mesenteric ischemia. *European Journal of Radiology*, *80*(2), 297–302. https://doi.org/10.1016/j.ejrad.2010.07.016

XII. Nakamura, Y., Kondo, S., Narita, K., Maeda, S., Fonseca, D., Honda, Y., Tani, C., Fukumoto, W., Mitani, H., Ishibashi, M., Chosa, K., Tatsugami, F., & Awai, K. (2023). Understanding CT imaging findings based on the underlying pathophysiology in patients with small bowel ischemia. *Japanese Journal of Radiology*, *41*(4), 353–366. https://doi.org/10.1007/s11604-022-01367-x

XIII. Lendzion, R. J., Frahm-Jensen, G., & Keck, J. (2022). Acute mesenteric ischemia. *Clinics in Colon and Rectal Surgery*, *35*(03), 227–236. https://doi.org/10.1055/s-0042-1743283

XIV. Oldenburg, W. A., Lau, L. L., Rodenberg, T. J., Edmonds, H. J., & Burger, C. D. (2004). Acute mesenteric ischemia: A clinical review. *Archives of Internal Medicine*, *164*(10), 1054. https://doi.org/10.1001/archinte.164.10.1054

XV. Park, W. M., Gloviczki, P., Cherry, K. J., Jr, Hallett, J. W., Jr, Bower, T. C., Panneton, J. M., Schleck, C., Ilstrup, D., Harmsen, W. S., & Noel, A. A. (2002). Contemporary management of acute mesenteric ischemia: Factors associated with survival. *Journal of Vascular Surgery*, *35*(3), 445–452. https://doi.org/10.1067/mva.2002.120373