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## Mesenteric ischemia: A case report highlighting the importance of early detection and intervention

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### Abstract

This case report emphasizes the importance of early detection and intervention in mesenteric ischemia. We present a 42-year-old patient with a history of splenectomy who presented with hemodynamic instability, severe epigastric pain, vomiting, and no bowel movement or gas for three days, along with an unspecified fever. Laboratory tests showed abnormal values, and an abdominal CT scan revealed dysmorphic liver, portal cavernoma, peritoneal effusion, and superior mesenteric vein thrombosis. Surgical intervention was necessary, and the patient underwent exploratory laparotomy, which revealed necrosis of the small intestine that was resected. The patient experienced complications such as pulmonary embolism, and a second surgery was performed, but it was negative. The patient was discharged with follow-up care in the internal medicine department.

This case report highlights the importance of recognizing the signs and symptoms of mesenteric ischemia and prompt intervention to improve patient outcomes.

**Keywords:** Entero-mesenteric ischemia, peritonitis, general surgery, surgical emergencies

### Introduction

IEM (Mesenteric ischemia) is a medical-surgical, absolute, digestive, and vascular emergency. When not fatal, ischemia causes considerable sequelae due to extensive small bowel resection, and necrosis is a decisive stage. If left untreated, the patient may die within a few hours, whereas prompt action will either allow the intestine to recover or result in very limited resection. Early diagnosis and monitoring of individuals at risk remain the challenge for all physicians. In the presence of any abdominal pain, mesenteric ischemia should be considered until proven otherwise.

### Case presentation

Patient aged 42, medical history includes splenectomy performed 20 years ago for an undocumented pathology. Upon admission, the patient presented with hemodynamic instability, tachycardia, respiratory rate of 17, and oxygen saturation of 97% on room air. Blood pressure was 8/6. The patient complains of severe epigastric pain, bilious vomiting, and no bowel movement or gas for three days. These symptoms occurred in the context of an unspecified fever. Upon examination, the patient had generalized sensitive abdominal, and abdominal rigidity.

Laboratory tests showed a hemoglobin level of 10, white blood cell count of 24,000, lactate level of 250, and C-reactive protein level of 400. Abdominal CT scan revealed a dysmorphic liver with uneven contours, hypertrophy of segment I, and atrophy of segment V. Portal cavernoma was present, as well as significant peritoneal effusion with signs of digestive distress and superior mesenteric vein thrombosis.

Surgical intervention was indicated, and the patient underwent exploratory laparotomy. Necrosis of the small intestine was present 10 cm from the duodenojejunal angle and extended for 70 cm. The colon and small intestine were distended without any area of caliber disparity. Resection of the necrotic portion of the small intestine was performed with end-to-end anastomosis. The patient was admitted to the intensive care unit on norepinephrine at a rate of 20 and later developed pulmonary embolism. The patient was readmitted to the operating room due to suspicion of small intestine necrosis, but the laparotomy was negative.



**Fig 1:** Bowel segment resection

Upon readmission to the surgical ward nine days after the first surgery, the patient had positive bowel sounds and stool passage, a soft abdomen, and drainage of 30cc of serohematic fluid. The patient was on Lovenox 0.4 mg twice a day and was maintained on this medication after discharge. The patient received consultation and follow-up care in the internal medicine department for further investigation

### Discussion

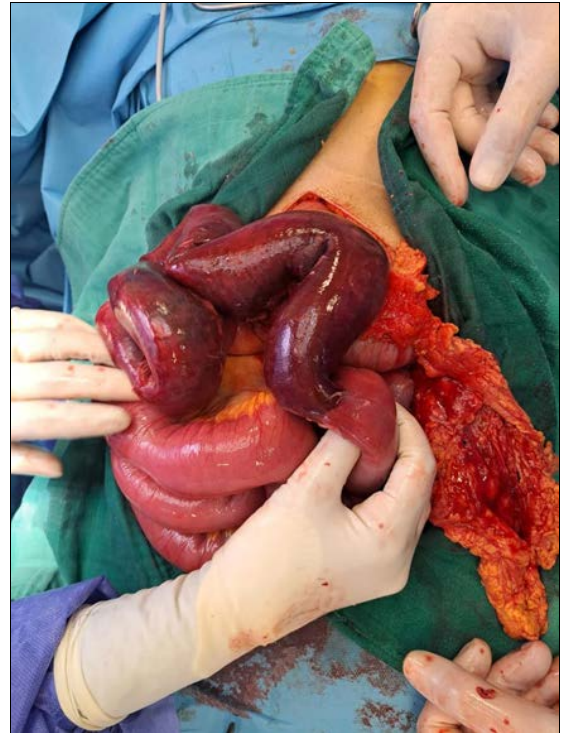
Venous mesenteric ischemia represents a rare symptomatic entity, occurring in approximately 1 in 5000-15000 admissions. Venous ischemia accounts for 6-9% of cases of mesenteric ischemia [4].

In the case of our patient, venous ischemia resulted from an extension of the portal thrombus to the superior mesenteric networks, likely due to cirrhotic liver. A porto-portal compensation system then formed to compensate for the interruption of portal flow, thereby masking the clinical and biological manifestations of hepatic distress.

Intestinal infarction secondary to mesenteric thrombosis may be favored by arterial vasospasm and/or risk factors for atherosclerosis. The normal blood flow rate for the small intestine is 75ml/min/100 g per minute, whereas for the colon it is 35ml/min/100 g per minute. A sudden decrease in blood flow to < 30 ml/min/100 g constitutes a critical threshold beyond which adaptive processes can no longer maintain sufficient tissue oxygenation, leading to cellular anoxia. At this stage, cells trigger anaerobic metabolism, and anaerobic glycolysis leads to the production of two intracellular toxic substances: lactate and calcium.

At this stage, the measurement of lactate in the blood is a good indicator of the severity of ischemia. In the case of our patient, the lactate levels were 400, indicating significant consumption and intracellular acidosis leading to cellular necrosis and destruction of cellular homeostasis. In most cases of splanchnic ischemia, arterial lactate levels remain normal despite an increase in lactate production by the intestine. This dissociation is due to the hepatic metabolic capacity of lactate. Thus,

systemic lactic acidosis, a classic sign of severity of intestinal ischemia, is a late phenomenon that indicates severe ischemia.



**Fig 2:** Ischemic bowel segment

Necrosis progresses through several successive stages, infiltrating the different layers. Involvement of the submucosa induces edematous and hemorrhagic infiltration, clinically manifested by bloody diarrhea at this stage. These lesions occur between 3-6 hours of ischemia and are reversible after revascularization at this stage. If necrosis continues transmurally, the stage of infarction occurs between 6-24 hours post-ischemia, and the lesions are irreversible [1, 4].

The ultimate stage is peritonitis due to perforation resulting from bacterial infection caused by necrosis and perforation of the digestive tissue. Generally, this stage occurs > 24 hours post-ischemia with clinical manifestations ranging from abdominal pain to generalized contraction, cessation of bowel movements and gas, and vomiting, as was the case with our patient.

Without treatment, the progression leads to septic shock and death within 48 hours.

The thrombosis of the superior mesenteric vein refers more to an ischemia secondary to an abdominal pathology such as venous stasis caused by cirrhosis of the liver or congestive heart failure, which could correspond to the case of our patient. Thrombosis of the distal venous branches refers more to a thrombopathy. It should be noted that 21-49% of venous ischemias are idiopathic in origin.

Venous-origin digestive ischemia produces a gradual transition between healthy structures and necrotic structures, unlike arterial ischemia, which presents clear boundaries due to the cessation of arterial supply. It should be noted that the ileum and jejunum are by far the regions most affected during ischemia.

In the absence of appropriate treatment, superior mesenteric artery (SMA) occlusion is constantly fatal. Despite appropriate treatment, mortality ranges from 40% (venous ischemia) to over 80% (arterial ischemia). The main prognostic factors are the timeliness of treatment, the mechanism of ischemia, the age and general health of the patient. Late diagnosis at the stage of necrosis and peritonitis explains the high mortality rate and the

prevalence of short bowel syndrome among survivors. Three elements are associated with the presence of irreversible necrosis: the presence of one or more organ failures, serum lactate levels greater than 2 mmol/L, and intestinal dilation greater than 25 mm. The rate of intestinal necrosis increases from 3% in the absence of these factors to 38%, 89%, and 100% in the presence of one, two, or three factors, respectively. These elements allow for the definition of the two stages of the disease [3, 2].

### Conclusion

The mesenteric ischemia represents a vital emergency that is still widely unknown. The key issue is to identify the early signs of reversible ischemia and thus stop the progression towards irreversible and potentially fatal necrosis.

To further develop this conclusion, it is important to emphasize the importance of early detection and intervention in cases of mesenteric ischemia. This condition occurs when blood flow to the intestines is decreased or stopped, leading to damage and potential death of the affected tissue. Prompt diagnosis and treatment can help prevent complications such as bowel infarction and sepsis, which can be life-threatening.

Furthermore, healthcare providers should be aware of the risk factors and potential causes of mesenteric ischemia, such as atherosclerosis, embolism, and thrombosis. Early intervention may involve measures such as blood thinners, surgical revascularization, or removal of any obstructions.

Overall, recognizing the signs and symptoms of mesenteric ischemia and acting quickly can help improve outcomes for patients and potentially save lives.

### Conflict of Interest

Not available

### Financial Support

Not available

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#### How to Cite This Article

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