

ACUTE MESENTERIC ISCHEMIA: CASE STUDY

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Abstract: Introduction: Acute mesenteric ischemia is a frequently fatal surgical emergency, largely due to delayed diagnosis. Despite advances in therapy and supportive measures, the disease still persists with a high degree of morbidity and mortality, with mortality rates reaching 70% of cases. The nonspecific physical examination, uncertainty of diagnosis, the magnitude of surgeries and possible complications have contributed to limit the experience and justify the failure of treatment. Discussion: Mesenteric ischemia can be caused by arterial or venous obstruction. In the first hours, there are no signs of peritoneal irritation, distension or fever. Radiological signs are often suggestive only in advanced stages, when they reveal wall edema, abnormal separation of the loops, intestinal pneumatosis or presence of gas in the portal system. Most patients have at least some part of the intestine frankly necrotic at the time of diagnosis. It is advisable to avoid resection of a bowel with questionable viability at the time of the first surgery. A second revision surgery “second look” can be performed, ideally 18-48 hours after the first procedure. Wide resections and discontinuity of the colon in the intestinal transit favor the establishment of short bowel syndrome, characterized by diarrhea, weight loss, dehydration, hydroelectrolytic and malabsorptive disorders. Advances in diagnostic methods, combined with advances in maintaining nutritional status, have a positive impact on the prognosis and quality of life of the patient. Conclusion: Despite the enormous advances in diagnostic methods and knowledge of its pathophysiology, the diagnosis of intestinal ischemia remains eminently clinical. The high mortality rates may be related to the difficulty in early diagnosis of intestinal ischemia, and to the lack of specificity of abdominal pain and complementary tests available. It is likely that the incidence of intestinal ischemia is higher

than previously recognized.

Keywords: Mesenteric ischemia; Short bowel syndrome; Enterectomy; Second look

INTRODUCTION

Acute mesenteric ischemia is a frequently fatal surgical emergency, largely due to the delay in making a diagnosis. Despite advances in treatment and support measures, the disease still has a high level of morbidity and mortality, with mortality rates reaching 70% of cases.

The relative absence of characteristic physical signs, the uncertainty of diagnosis, the magnitude of the operations and possible complications have contributed to limiting the experience and justifying the failure of treatment.

CASE REPORT

Patient A.V.S.C., female, 49 years old, long-time smoker, with a report of intermittent abdominal pain for 1 month, associated with nausea and vomiting, sought emergency care at the HFA with worsening of pain and abdominal distension. On examination, she presented tachypnea, tachycardia and signs of peritoneal irritation. The laboratory showed significant leukocytosis with left shift.

Computed tomography showed gastric distension, thickening and distension of small bowel loops, with formation of an air-fluid level and presence of free fluid in the cavity.

She underwent exploratory laparotomy, showing an extensive segment of necrotic ileum affecting the ileocecal valve (Figures 1 and 2). Enterectomy, ileotyphlectomy and primary anastomosis were performed, with a small bowel remaining of approximately 170 cm. On the seventh postoperative day, anastomotic descent was evidenced, requiring re-approach and creation of a terminal ileostomy.

Patient presented, in the immediate postoperative period, high fluid output through the ostomy, requiring hydration, electrolyte replacement, parenteral and enteral nutrition and supplementation. High doses of loperamide associated with codeine were used. The patient was discharged after 45 days, accepting an exclusive oral diet and maintaining his weight.



Figure 1: Identified ileal segment and ileocecal valve with necrosis.



Figure 2: Surgical specimen containing segment of necrotic ileum.

DISCUSSION

Mesenteric ischemia is caused by arterial or venous obstruction. The etiology of arterial obstruction can be embolism, thrombosis, low blood flow, extrinsic compression and vasospasm induced by vasoactive drugs. Studies have shown that 35 to 50% of patients who die from intestinal ischemia have a history of chronic ischemia of a transient

nature, secondary to atherosclerotic disease of the mesenteric arteries.

In arterial embolism, the main embolic sources are mural thrombi of the cardiac cavities associated with myocardial infarction and atrial fibrillation. Arterial thrombosis occurs in a previous atherosclerotic plaque, which can be of degenerative or, more rarely, inflammatory origin. Venous occlusion can be caused mainly by venous thrombosis, infectious and inflammatory processes and coagulation alterations. Venous thrombosis occurs in 10% of intestinal ischemia. In non-occlusive intestinal ischemia, there is no evidence of arterial or venous mechanical occlusion, but rather a decrease in intestinal blood perfusion due to severe splanchnic vasoconstriction. It accounts for 20 to 30% of acute mesenteric ischemia and affects patients with severe heart disease who are taking digitalis and who are often hospitalized due to recent worsening of their heart disease or a serious complication (infection, trauma or major surgery). In acute ischemia, $\frac{2}{3}$ of patients are women and are generally over 70 years old. The first response of the intestine to ischemia is intense vasospasm, which explains the intense pain followed by nausea (25-30%), vomiting (50%), diarrhea and occult rectal or gastric blood (25%). Initially, the physical examination is relatively poor, with an innocent abdomen. In the first hours, there are no signs of peritoneal irritation, distension or fever. The pain, as in chronic ischemia, is usually located in the epigastric or periumbilical region and is typically disproportionate to the physical examination findings. Laboratory tests show leukocytosis invariably above 20,000, an increase in CPK-MB within 6 hours of the onset of the process, and metabolic acidosis, amylase and lipase levels only increase in the more advanced stages.

The complementary tests that can aid in

the diagnosis are: complete blood count, simple radiography, duplex mapping, angiography, computed tomography and angioresonance, of which panoramic or selective angiography is the gold standard. Intense pain, gas, abdominal distension, the patient's immobility and lack of cooperation make the use of imaging tests very difficult, in most cases making an accurate diagnosis impossible. Often, radiological signs are only suggestive in advanced stages, when they reveal wall edema, abnormal separation of the loops, presence of gas in the walls of the intestine (intestinal pneumatosis) or in the portal system and dilation of the small intestine loops and transverse colon.

The treatment of arterial ischemia consists of recognizing the lesion as early as possible and reestablishing blood flow. Revascularization of the intestine must be performed within 6 to 8 hours after the onset of symptoms, when the ischemia is reversible, through embolectomy or thromboendarterectomy and revascularization through aorto-mesenteric shunts. Although endovascular therapy by catheter can restore arterial blood supply to the ischemic intestine, most patients with acute ischemia have at least some part of the intestine that is frankly necrotic at the time of diagnosis. These patients still require laparotomy, even when arterial circulation is successfully restored by endovascular techniques.

In the late stages of massive infarction, revascularization is not indicated. In addition to the small possibility of minimizing resection, it favors reperfusion syndrome, with reabsorption of substances produced by cell lysis and bacterial contamination capable of producing a decrease in plasma volume, electrolyte disturbances, intestinal hemorrhage, and circulatory collapse. Therefore, candidates for thrombolytic therapy will constitute only a small percentage of the

total number of cases, and early diagnosis is important to select those patients with no peritoneal signs on physical examination and no indications of bowel infarction on abdominal radiographs or computed tomography.

Delayed diagnosis and failure to restore arterial blood flow early favor wide resections of bowel segments. It is advisable to avoid resection of bowel with questionable viability at the time of the first operation. A second-look revision surgery is often performed after revascularization for acute bowel ischemia. The second-look operation is ideally performed 18-48 hours after the first procedure. Any remaining nonviable bowel is resected, and the integrity of vascular repairs and bowel anastomoses is reassessed.

To determine bowel viability can be difficult. Unnecessary resections lead to short bowel syndrome. Conversely, insufficient resections require repeat operations. Postoperative progression of ischemia is a reality, and is more common when resection is the only treatment instituted.

Short bowel syndrome is characterized by diarrhea, fluid and electrolyte abnormalities,

malabsorption and weight loss. It causes inconveniences in parenteral and enteral nutrition and can progress to sepsis and death. Recent advances and improvements in propaedeutic methods, combined with advances in maintaining the nutritional status and the hydroelectrolytic and acid-base balance of patients, are improving the prognosis of this disease somewhat, which depends on early diagnosis.

In the postoperative period of arterial and venous thrombosis, patients must be kept on anticoagulation for a prolonged period to avoid the risk of recurrence.

CONCLUSION

Despite the enormous advances in diagnostic methods and knowledge of its pathophysiology, the diagnosis of intestinal ischemia remains predominantly clinical.

The high mortality rates may be related to the difficulty in early diagnosis of intestinal ischemia and the lack of specificity of abdominal pain and complementary tests available. It is likely that the incidence of intestinal ischemia is higher than previously thought.

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