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Acute Massive Idiopathic Gastric Ischemia*



Isquemia gástrica masiva aguda de causa idiopática

The stomach is one of the most vascularized abdominal viscera in the organism. For this reason, gastric ischemia is considered a rare condition. There are known causes of gastric ischemia associated with toxic, mechanical factors, vascular damages or infections. Nevertheless, sometimes there is no clear trigger. Massive idiopathic gastric ischemia is an extremely infrequent entity with very few cases described in the medical literature.¹

We present a case of patient with massive idiopathic gastric ischemia.

A 61-year-old woman with a past medical history of hipertension and mastectomy with axillary lymphadenectomy due to a lobular carcinoma of the breast three years ago, presented to the emergency department for abdominal pain of 48 h evolution. On clinical examination, the patient had hypothermia (34.7 °C), a blood pressure of 96/50 mmHg, pallor, obnubilation and tachycardia (150 min⁻¹). Marked rebound tenderness was found on abdominal examination. Laboratory values showed 940 leukocytes/mm³ (88.3% neutrophils), a Creactive protein level of 48 mg/dl, a procalcitonin level of 48 mg/dl and a venous pH was 7.30. After initial evaluation and resuscitation, the patient underwent a thoracic and abdominal CT scans that identified bilateral pleural effusion, free intra-abdominal fluid and mucosal edema in the gastric antrum and gastric body. Gastroscopy revealed ischemic mucosa in the cardia. Through a midline laparotomy, a massive gastric necrosis was found (Fig. 1) and total gastrectomy was performed. During the procedure, the patient was hemodynamically unstable and required vasoactive drugs at high doses. For these reasons, primary anastomosis was not performed and an esophagostomy and a feeding jejunostomy

were created. Pathologic examination revealed an extensive necrosis, with focal transmural involvement that caused subtotal mucosal hemorrhage, severe congestion of the entire wall and acute necrotizing inflammation that extended throughout the submucosa, reaching muscle and focally subserosa and serosa. After surgery, the patient was admitted to the intensive care unit. However, the septic shock was refractory to the measures and the patient died at 14 hours postoperatively. The family rejected autopsy.

The most frequent causes of gastric ischemia are volvulus, intrathoracic herniation of the stomach, and massive gastric dilatation due to mechanical factors such as intestinal obstruction, pyloric stenosis or atonicity of the stomach caused by anorexia nervosa and electrolyte imbalance. On the other hand, infectious causes (necrotizing gastritis) have been reported, generally involving immunocompromised patients (diabetes, AIDS, neoplasia). The intake of toxins such as caustics can also cause necrosis in the stomach. In our case, mechanical obstruction and vascular compromise have been ruled out at CT findings. In the same way, the intake of toxins was discarded through a meticulous anamnesis. Bacterial growth was not evident in the analysis of the piece as the cause of the disease. For these reasons, the etiology of this massive gastric ischemia was considered idiopathic.

Generally, the most common clinical manifestations are abdominal pain, emesis or those derived from a complication, such as intestinal obstruction or perforation. Physical findings can be abdominal distension, tympanism and tenderness.³

Currently, therapeutic options in gastric ischemia include nasogastric tuve, to decompress the stomach, and immediate surgery. When we suspect a gastric ischemia, surgical therapy

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Fig. 1 – Massive gastric ischemia showing edema and necrosis.

must be emergent and aggressive because mortality is elevated for delayed diagnosis.³ Gastric resection with gastrojejunal or esophajejunal anastomosis is the procedure of choice if the patient's hemodynamic conditions allow it. Our patient was hemodynamic instability and, for this, anastomosis was rejected in the first time. In any case, despite early surgery, the mortality estimated in a patient with massive acute gastric ischemia is greater than 50%–60%.²

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