

Idiopathic gastric partial necrosis*

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ABSTRACT

We present the case of an elderly patient, who had orthopaedic surgery and suffered in the early post-operative period an idiopathic gastric partial necrosis. We reviewed the literature and it is an extraordinarily infrequent entity. Partial resections have already been described in case of limited necrosis, as reported in our case. Prompt diagnosis of idiopathic gastric necrosis and aggressive treatment are mandatory. Further awareness of this strange entity and its risk factors can lead to a quicker diagnosis and higher possibilities of survival for the patient.

Keywords: Gastric; Necrosis; Orthopedic Surgery

1. INTRODUCTION

Gastric partial necrosis is an extraordinarily infrequent entity with very few cases described in literature. It has been attributed to numerous aetiologies, such as lifestyle, underlying morbidities, acute necrotising inflammation, acute vascular insufficiency, post-operative complications, etc. [1].

The onset of symptoms is generally catastrophic and surgical therapy must be urgent, as a delay in the diagnosis or treatment in these types of pathologies causes a significant increase of morbimortality [1].

We describe the case of an elderly patient, who had orthopaedic surgery and suffered in the early postoperative period an idiopathic gastric partial necrosis. We reviewed the literature and it is the second case described of idiopathic gastric partial necrosis attributable to orthopedic surgery.

*Author contributions: González L. R. conceived of the study, drafted the manuscript and performed the intervention. Muínelo L. M and Pérez G. J. participated in the design of the study and performed the intervention. Muíños R. L., Ramírez R. L., Mohammed S. A., Rodríguez G. M., and Costa Buján J. A. performed the clinical research. All authors read and approved the final manuscript.

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2. CASE REPORT

An 82-year-old man with a history of severe chronic obstructive pulmonary disease and hypertension undergoes orthopedic surgery: total right hip replacement. 48 hours after the operation, he presents with distension and abdominal pain, so urgent blood tests are carried out showing acute renal failure, without leukocytosis or neutrophilia. Given the suspicion of intra-abdominal free gas in the simple abdominal X-rays, an urgent computed axial tomography (CAT) is made, showing subcapsular air and, in hepatic parenchyma predominant in left hepatic lobe, portal gas and diffuse dilation of small bowel loop together with gastric pneumatosis (**Figure 1**). The patient is transferred to the Service of General Surgery, where the yield of abundant amount of content in coffee ground through nasogastric tube is noticed (NG tube), so urgent upper gastrointestinal endoscopy (UGIE) is requested, showing severe ischemia of the gastric wall in the greater gastric curvature and gastric body. Given the clinical worsening of the patient, urgent surgery is decided, observing through midline laparotomy, ischemia of greater gastric curvature up to the angle of His (**Figure 2**), the rest of the stomach showing good vascularisation. Thus, it is decided to carry out an atypical gastric resection, making a sleeve gastrectomy at the expense of the lesser curvature.

The anatomopathological result showed transmural ischemia. The post-operative complications included right nosocomial pneumonia, with initial favourable development. On the sixth post-operative day evisceration is noticed, so urgent surgery is decided, observing anterior gastric perforation at antral level, possibly related to the NG tube position. No data of peritonitis, proceeding to the primary closure of the perforation and to the closure of the abdominal wall. The patient is sent to Resuscitation, being operated four days later due to new gastric perforation in the same antral area, making a new primary closure. After 24 hours, the patient passes away due to multiple organ failure.



Figure 1. Abdominal CT scan: showing subcapsular air and, in hepatic parenchyma predominant in left hepatic lobe, portal gas and diffuse dilation of small bowel loop together with gastric pneumatosis.



Figure 2. Ischemia of greater gastric curvature up to the angle of His.

3. DISCUSSION

Since the first case of acute gastric necrosis described in 1833 by Duplay, just a few cases of this infrequent pathology have been published, maybe mainly because of the rich gastric vascularization (right and left gastric artery, short gastric vessels and right and left gastroepiploic arteries) [1,2].

Numerous aetiologies of this pathology have been discussed, but this is the second case attributable to the aftermath of orthopedic surgery.

Gastric dilation might be very important, so as to provoke abdominal compartment syndrome [3]. When massive dilation occurs, ischemia is presumably due to venous insufficiency. Rupture can occur with intragastric pressures of more than 120 mmHg (corresponding to approximately 4 L of fluid. Distension and perforation can rapidly lead to neurogenic (vagal response) and subsequent septic shock. The repetitive massive consumption of food in the case of food disorders and electrolyte imbalances can lead to massive gastric distension. Mechanical factors can be implied in gastric dilation, like bowel obstruction or pyloric stenosis, and infectious

causes (necrotising gastritis) have been reported, generally involving immunocompromised patients (diabetes, AIDS, neoplasia). Physiopathologic theories have been debated in the past: one advocated upper esophageal sphincter relaxation (due to debilitation or anesthesia) with consequent aerophagia and gastric dilatation as a cause. Atonic theory (muscular atrophy during prolonged starvation does not support rapid refeeding), superior mesenteric artery syndrome (vascular compression of the third duodenal portion) or functional diseases caused from regional alterations (in pancreatitis, ulcer and other abdominal inflammations) are other supposed etiologies [1,4,5].

Clinically, emesis might be the initial symptom, but events can precipitate suddenly to shock. Physical findings are abdominal distension and tympanism, with tenderness and peritonitis in case of perforation. CT scan is useful in the diagnosis as it can demonstrate gastric distension, portal gas, gastric pneumatosis and pneumoperitoneum, as reported in our case. The UGIE can also help for the pre-operative diagnosis, but most times the diagnosis is made during surgery [1,6].

Detension with nasogastric tube is mandatory as the first therapeutical act, followed by immediate surgery in case of perforation. Necrosis might be partial, mostly in the lesser curve due to vascular supply). Total gastrectomy is the procedure of choice, but it requires time and stable hemodynamic conditions. Mortality after gastrectomy in acute gastric necrosis is very high, ranging from 50% to 80% [1,6].

Partial resections have already been described in case of limited necrosis, as reported in our case.

Unfortunately, a complicated post-operative period provoked the death of the patient despite the prompt diagnosis and surgery. In this patient, it is probably due to multifactorial causes (previous short fasting, hypertensive vascular disease, intubation, recent surgery, possible undiagnosed thromboembolic accident), but none of them stands alone as an evident reason for the catastrophic event.

Prompt diagnosis of idiopathic gastric necrosis and aggressive treatment are mandatory, but sometimes not sufficient to save our patient, like in our case, especially in older patients who can easily suffer irreversible multiple organ failure.

Further awareness of this strange entity and its risk factors can lead to a quicker diagnosis and higher possibilities of survival for the patient.

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