

Gastric Perforation Due to Ischemic Necrosis: Radiological Diagnosis and Emergency Surgical Management. Case Report

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ABSTRACT

Gastric necrosis is an extremely rare clinical entity due to the stomach's rich vascularization, which usually protects it from ischemia (1,2). When it presents as acute gastric perforation, it constitutes a devastating surgical emergency with high mortality, often exceeding 50–60% even with early surgical management, and reaching up to 80% in patients presenting with septic shock (2,3). We report the case of a 53-year-old male patient with multiple comorbidities and previous surgical history, who presented with acute abdominal pain, leukocytosis, and radiological findings suggestive of hollow viscus perforation. Computed tomography revealed abundant pneumoperitoneum and free fluid, which led to emergency exploratory laparotomy. Intraoperatively, transmural ischemic necrosis with perforation of the anterior wall of the stomach from the fundus to the antrum was identified, without evidence of other ischemic lesions (6,16). Segmental gastric resection and primary closure were performed; however, the patient died in the early postoperative period. This case underscores the importance of early clinical suspicion, prompt radiological diagnosis, and immediate surgical intervention (2,13). Despite progress in diagnostic and therapeutic techniques, perforated gastric necrosis remains a condition with extremely high lethality, often requiring damage control surgical strategies in patients with hemodynamic instability (16).

KEYWORDS: Anterior gastric perforation, gastric necrosis, acute abdomen, exploratory laparotomy, gastrectomy, damage control surgery, case report.

ABBREVIATIONS: CT (Computed Tomography), NPRS (Numeric Pain Rating Scale)

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INTRODUCTION

Gastric necrosis is an exceedingly rare and severe pathology, largely due to the stomach's rich vascular supply, which protects it from ischemia even following the ligation of major arterial trunks (1,2). Only a limited number of cases have been reported in the literature, generally associated with predisposing factors such as vascular events (systemic hypoperfusion secondary to shock, thrombosis, or arterial

embolism of the celiac trunk) [1,2]; mechanical factors (gastric volvulus, intrathoracic diaphragmatic hernia, massive gastric distension or dilation due to pyloric obstruction or ileus, post-fundoplication “gas-bloat” syndrome) [5,16]; eating disorders (binge eating in bulimia nervosa or psychogenic polyphagia causing acute dilation) [8,9]; toxic causes (ingestion of caustic substances or other corrosive agents) [11]; and infectious causes (necrotizing gastritis,

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particularly in immunocompromised patients) [6]. Iatrogenic etiologies have also been described, such as gastric necrosis as a late postoperative complication following antireflux fundoplication or adjustable gastric banding in bariatric surgery (5,7). However, in some patients no clear precipitating cause can be identified, and cases of idiopathic gastric ischemia have been reported where no underlying mechanism was found (10).

Clinically, gastric necrosis often presents with nonspecific symptoms and may be easily misinterpreted as other causes of acute abdomen (3). It typically manifests with severe abdominal pain, vomiting, and signs of peritoneal irritation in the event of perforation, alongside systemic features of inflammatory response or septic shock in advanced stages. Given the potential for fulminant progression, early diagnosis is critical for improving outcomes—particularly before perforation occurs. In this context, imaging plays a central role (12,13).

Chest or abdominal radiography may demonstrate pneumoperitoneum (subdiaphragmatic free air) in cases of perforation, quickly guiding suspicion toward hollow viscus injury. However, abdominal computed tomography (CT) is the imaging modality of choice for evaluating suspected visceral perforation, given its high sensitivity for detecting small amounts of extraluminal air and for localizing the site of perforation (12,13). CT not only confirms gastric perforation through direct findings (wall defect, contrast leak) or indirect signs (pneumoperitoneum, free fluid, wall thickening, or mucosal enhancement loss), but may also provide clues about the underlying etiology (13). One particularly relevant radiological finding is gastric pneumatosis (air within the gastric wall), especially when associated with portal venous gas, as it is highly suggestive of transmural gastric ischemia. Indeed, CT imaging in patients with ischemic gastric necrosis has shown wall thinning, absence of contrast enhancement, pneumatosis, and even the presence of portal venous gas (4).

In summary, a combination of high clinical suspicion in a patient with risk factors and radiologic signs (pneumoperitoneum, pneumatosis, gastric wall edema, etc.) should prompt timely diagnosis of gastric necrosis, given the rarity of the condition but its markedly high mortality if left untreated (2,3).

CASE REPORT

We present the case of a 53-year-old male patient with a history of systemic arterial hypertension and type 2 diabetes mellitus, both under adequate control, as well as sequelae of a cerebrovascular accident in 2024, which resulted in left-sided hemiparesis. His surgical history included an exploratory laparotomy for ascending colon perforation with loop colostomy, followed by a second procedure involving colostomy remodeling with right hemicolectomy, primary closure of the transverse colon, and terminal ileostomy

performed in June 2025. The current condition began in July 2025, when the patient presented to a secondary-level hospital with sudden-onset diffuse abdominal pain rated 10/10 on the Numeric Pain Rating Scale (NPRS), without radiation, associated with nausea but no vomiting. He reported no bowel movements in the past seven days and denied passage of flatus at the time of evaluation. He was admitted for diagnostic workup.

Laboratory tests revealed leukocytosis ($15,100/\text{mm}^3$) with neutrophilia ($13,800/\text{mm}^3$), hemoglobin of 11 g/dL, and arterial blood gas analysis showing an acid-base imbalance: pH 7.48, pCO_2 34 mmHg, pO_2 106 mmHg, lactate 1.5 mmol/L, HCO_3^- 25.3 mmol/L, and base deficit of -6.89 . Chest X-ray showed subdiaphragmatic free air, which was further evaluated with a non-contrast abdominal and pelvic CT scan. The CT revealed free intraperitoneal air suggesting hollow viscus perforation (Figures 1–4).

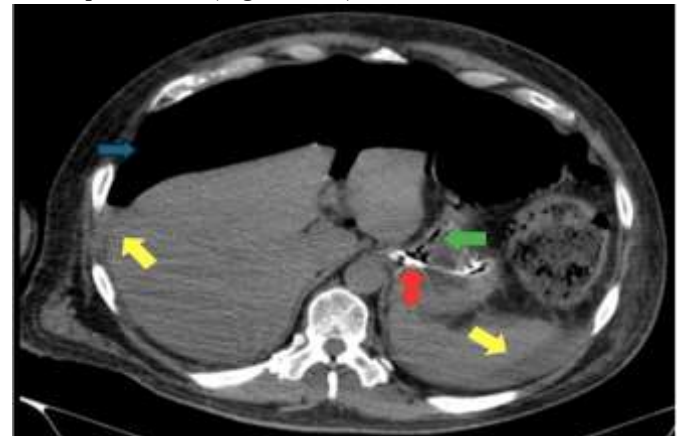


FIGURE 1. Non-contrast axial CT at the gastric level showing abundant free air in the abdominal cavity (blue arrow), free air near the lesser curvature of the stomach (green arrow), and intraluminal nasogastric tube (red arrow), with scant perihepatic and perisplenic free fluid (yellow arrows).

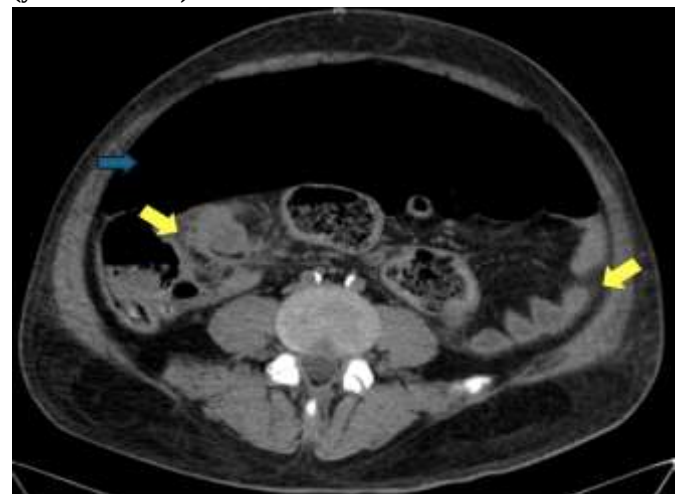


FIGURE 2. Non-contrast axial CT at the level of the common iliac arteries showing abundant free intraperitoneal air (blue arrow), with minimal free fluid between bowel loops and in both paracolic gutters (yellow arrows).



FIGURE 3. Non-contrast coronal CT demonstrating bilateral subdiaphragmatic free air (blue arrows), free air near the lesser curvature of the stomach (green arrow), intraluminal nasogastric tube (red arrow), and minimal perihepatic and bilateral paracolic gutter free fluid (yellow arrows).

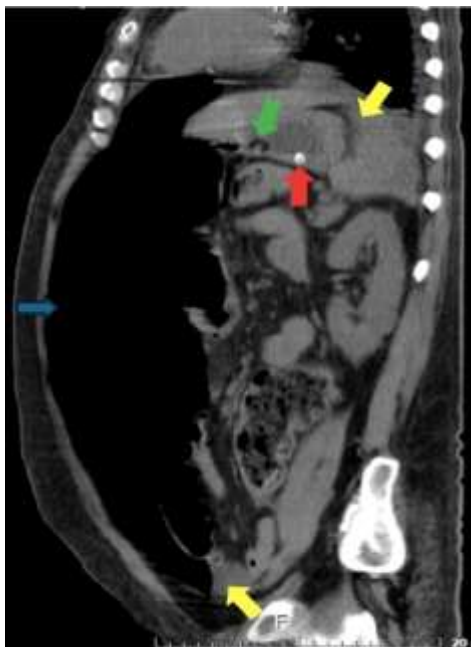


FIGURE 4. Non-contrast sagittal CT view showing abundant intraperitoneal free air (blue arrow), free air near the lesser curvature of the stomach (green arrow), intraluminal nasogastric tube (red arrow), and minimal free fluid in the perihepatic space and pelvic cavity (yellow arrows).

Given the clinical findings of an acute abdomen and imaging evidence of free air and fluid in the abdominal cavity, the patient underwent emergency exploratory laparotomy. Intraoperatively, multiple loose and firm adhesions were found, along with abundant free fluid in all four abdominal quadrants and formation of interloop abscesses.

A systematic quadrant-by-quadrant exploration revealed ischemic necrosis and perforation of the anterior wall of the stomach, extending from the fundus to the antrum. No other ischemic lesions were observed. The gastric circulation appeared preserved, with no evidence of thrombosis or vascular ligation (Figures 5 and 6).

A resection of the ischemic gastric segment was performed with edge revitalization, followed by a two-layer primary closure. Methylene blue testing showed no evidence of leakage. The patient was transferred to postoperative observation; unfortunately, he died 14 hours after the surgical procedure.



FIGURE 5. Intraoperative photograph showing the anterior gastric wall with evidence of necrosis and perforation measuring approximately 12 × 8 cm, with ischemic edges.

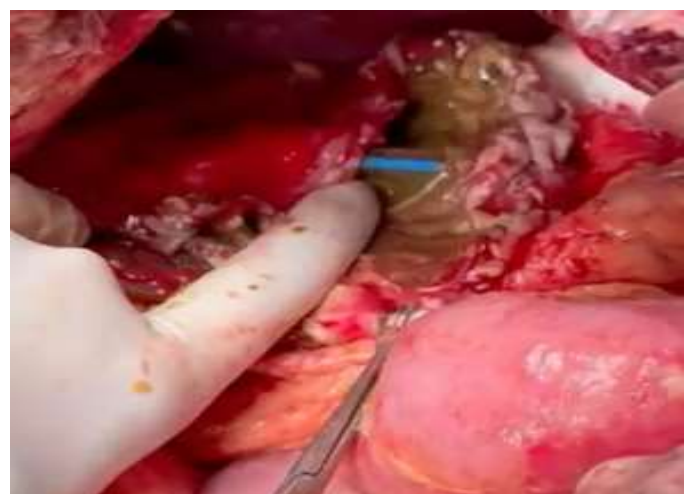


FIGURE 6. Intraoperative photograph showing perforation and necrosis of the anterior gastric wall with visible gastric lumen and presence of a nasogastric tube; ischemic margins are evident.

DISCUSSION

Gastric necrosis, as exemplified in our case, represents an extremely severe condition in emergency surgery scenarios. Despite its low incidence, the outcome is often fatal due to rapid progression toward perforation, peritonitis, and multiorgan failure (12). Literature reports indicate that mortality from ischemic gastric necrosis exceeds 50–60%, even when early surgical treatment is provided (2,5). In cases of delayed diagnosis or in patients presenting in septic shock, reported mortality rates reach as high as 80%. These data highlight the vital importance of early recognition and prompt, aggressive intervention (3,16).

In our patient, the initial clinical presentation was imprecise; however, radiological findings were determinant for diagnosis. The initial radiograph revealed subdiaphragmatic free air, immediately suggesting gastrointestinal perforation, and the abdominopelvic CT scan confirmed the presence of both free air and fluid in the abdominal cavity, along with findings suggestive of ischemia (gastric wall thickening and pneumatosis, associated with free fluid) (4,12). Multiple authors emphasize that multidetector CT not only localizes the perforation site in most cases but can also reveal associated signs pointing to the cause, such as evidence of mesenteric vascular thrombosis, hernia strangulation, bezoar presence, or gas infection signs in the gastric wall (12,13). In our case, early identification of ischemic gastric perforation enabled emergency surgical intervention, consistent with recommendations to manage any suspicion of acute gastric ischemia surgically and without delay (10).

During emergency surgery, the extent of gastric necrosis was confirmed. Similar cases described in the literature report surgical options ranging from limited resections (e.g., subtotal gastrectomy or gastric sleeve if necrosis mainly affects the greater curvature) to total gastrectomy with immediate reconstruction in relatively stable patients [6, 16]. However, in hemodynamically unstable patients or those with massive peritoneal contamination, some authors advocate for a damage control surgery strategy, involving resection of the necrotic stomach and deferring definitive reconstruction. Similarly, in our patient, an urgent gastric resection was required; due to the severity of the condition, it was decided not to perform a primary anastomosis during the same surgical act, ensuring adequate peritoneal lavage and drainage and planning transit reconstruction in a second stage. Although this strategy sacrifices initial gastrointestinal tract continuity, it prioritizes patient survival in critical situations (16).

Another aspect to consider is the relevance of aggressive hemodynamic resuscitation and preoperative optimization in these patients. Available evidence emphasizes that mortality from gastric necrosis relates not only to the extent of ischemic compromise and peritoneal contamination but also to the magnitude of the systemic inflammatory response. Therefore, it is recommended to initiate broad-spectrum antibiotics

early, strictly control volemia, and, if necessary, provide vasopressor support to maintain tissue perfusion before anesthetic induction. Additionally, it is important to highlight the scarcity of large case series on gastric necrosis, which limits the development of evidence-based management guidelines. Most available data come from isolated case reports and small retrospective series. This underscores the need to share clinical experiences such as ours, contributing to enriching the understanding of pathophysiology, risk factors, postoperative outcomes, and therapeutic strategies for such an infrequent pathology (12,16).

CONCLUSION

Gastric necrosis represents a challenging surgical emergency, with a rapid and potentially lethal clinical course. This case highlights the importance of considering ischemic gastric necrosis even in patients without clear antecedents of thrombotic events or recent bariatric interventions, as our patient presented no direct predisposing factors such as gastric volvulus, diaphragmatic hernia, or caustic ingestion, suggesting a possible multifactorial mechanism associated with chronic comorbidities and previously unidentified hypoperfusion status.

We emphasize the need for maintaining a high index of suspicion in patients with predisposing comorbidities and signs of acute abdomen, as well as the utility of timely radiological diagnosis to confirm perforation and assess stomach viability. Management must be prompt, multidisciplinary, and focused on controlling sepsis, resecting necrotic tissue, and stabilizing the patient to maximize survival chances. Despite advances in emergency surgery and intensive care, mortality remains high, underlining the necessity of documenting and disseminating such cases to strengthen collective knowledge and guide clinical decision-making in highly complex situations.

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