Archives of Current Medical Research
Arch Curr Med Res 2025; 6(3): 345-349

Received: 06 May 2025
Revised: 08 May 2025
Accepted: 25 June 2025
DOI: 10.47482/acmr.1693051

CASE REPORT

Isolated Total Gastric Necrosis Following Coronary Artery Bypass: A Rare and Life-Threatening Complication

Bilal Turan[1] M. Emre Alptekin[1] Sadık Yağız Kurşun[2] Hava Coşgun[2] İlknur Küçük[2] Elvin Sataf[2] Gökçe Şenocak[2] Öykü Çelebi[2] Serdar Acar[1]

1 Süleyman Demirel University, Faculty of Medicine, Department of General Surgery, Isparta, Türkiye
2 Süleyman Demirel University, Faculty of Medicine, Faculty of Medicine, Isparta, Türkiye

Abstract

Acute gastric necrosis is an exceptionally rare condition, attributed to the stomach's rich vascular supply. While often linked to gastric dilatation or mesenteric ischemia, isolated total gastric necrosis without bowel involvement is highly unusual. A 73-year-old male presented with acute abdomen on postoperative day 10 following coronary artery bypass grafting (CABG). He was on systemic anticoagulation. Laboratory findings revealed leukocytosis, renal dysfunction, and coagulopathy. CT imaging showed free air around the stomach. Emergency laparotomy revealed isolated total gastric necrosis without gastric dilatation or small bowel involvement. Total gastrectomy with Roux-en-Y esophagojejunostomy was performed. Histopathology showed full-thickness necrosis with ischemic and inflammatory changes but no vascular occlusion or NSAID-induced damage. The patient recovered well and was discharged on postoperative day 24. Despite robust collateral circulation, gastric ischemia can occur under certain systemic conditions such as recent cardiac surgery, anticoagulation, and cardiovascular compromise. In our case, the absence of other ischemic organ damage makes this presentation unique. Early signs are often nonspecific, and diagnosis is frequently delayed, contributing to high mortality. Isolated gastric necrosis, though rare, should be considered in post-CABG patients with acute abdominal symptoms. Early recognition and surgical intervention are vital for survival.

Keywords: Acute gastric necrosis, coronary artery bypass, gastric ischemia, total gastrectomy.

Corresponding Author:
Bilal Turan MD, Süleyman Demirel University, Faculty of Medicine, Department of General Surgery, Isparta, Türkiye
E-mail: bturan117@gmail.com

INTRODUCTION

Acute gastric necrosis is an uncommon clinical entity, largely attributed to the extensive and redundant vascular network supplying the stomach. Various underlying causes have been identified in the literature, including acute gastric dilatation (AGD), mesenteric arterial thrombosis, and AGD triggered by excessive food intake in the context of conditions such as psychogenic polyphagia, bulimia, or Prader-Willi syndrome. Additional reported etiologies include intrathoracic gastric herniation, gastric volvulus, acute necrotizing gastritis, ingestion of corrosive substances, therapeutic embolization procedures, and certain postoperative complications. (1–3).

Here, we aim to present the clinical features and literature review of a 73-year-old patient who underwent coronary artery bypass grafting (CABG), was receiving anticoagulant therapy, and was referred to our clinic with acute abdomen on postoperative day 10, during which exploratory laparotomy revealed isolated total gastric necrosis. This case report was prepared and presented as part of a third-year medical student graduation project to contribute to the current literature on this rare condition.

CASE REPORT

A 73-year-old male patient had undergone coronary artery bypass grafting (CABG) by the cardiovascular surgery department 10 days prior. He was on systemic anticoagulation and had been discharged two days before presenting to our center. Due to deteriorating general condition, he sought care at an external facility. Upon evaluation, the presence of free intraperitoneal air was noted, and a diagnosis of gastrointestinal perforation was considered. The patient was referred to our hospital for further management.

He reported progressive worsening of diffuse abdominal pain over the past 24 to 48 hours, accompanied by abdominal distension, anorexia, chills, and night sweats. He denied nausea, vomiting, or documented fever. His smoking history was significant for 80 pack-years, and he currently smoked half a pack per day.

On clinical evaluation, the patient appeared severely ill—somnolent, poorly responsive, and hemodynamically unstable with a blood pressure of 85/45 mmHg, tachycardia at 112 beats per minute, and oxygen saturation reduced to 85%. Abdominal examination revealed mild distension and diffuse tenderness. Although guarding and rebound were suspected, their presence was not conclusively confirmed. Laboratory analysis indicated marked leukocytosis (WBC: 32,100/mm^3 with 95.6% neutrophils), anemia (hemoglobin 9.4 g/dL), and thrombocytosis (platelet count 514,000/mm^3). Renal parameters were deranged, with elevated BUN (62.4 mg/dL) and creatinine (2.8 mg/dL), suggesting acute kidney injury. Coagulopathy was evident, with an INR of 1.8. Arterial blood gas analysis showed metabolic acidosis: pH 7.152, bicarbonate 16.8 mEq/L, and PCO_2 49.2 mmHg. Serum lactate was elevated at 3.2 mEq/L, indicative of systemic hypoperfusion.

A computed tomography (CT) scan performed at the referring hospital prior to surgical evaluation showed: “Minimal free fluid in the perisplenic area. Presence of gas around the liver and stomach. Gastrointestinal stromal tumor (GIST) perforation should be considered until proven otherwise.” An initial clinical impression suggested intestinal necrosis resulting from mesenteric ischemia. Following stabilization with crystalloids and fresh frozen plasma, the patient underwent an urgent exploratory laparotomy.

Approximately 1500 mL of purulent fluid was encountered in the peritoneal cavity and was aspirated. Exploration revealed isolated total gastric necrosis. There was no marked gastric dilatation. Adhesions were noted between bowel loops, which were lysed via adhesiolysis. Interloop abscesses were drained. The esophagus, small intestine, colon, and rectum appeared viable. The abdominal cavity was irrigated with saline. Upon opening the gastrocolic ligament, additional necrosis was identified on the posterior surface of the stomach. The entire posterior gastric wall was necrotic and paper-thin in appearance. No pathology was observed outside the totally necrotic stomach. A decision was made to perform total gastrectomy. Total gastrectomy with Roux-en-Y esophagojejunostomy was performed.

Pathological examination of the surgical specimen revealed a large, full-thickness total gastrectomy specimen involving the antrum and distal body (Figure 1).

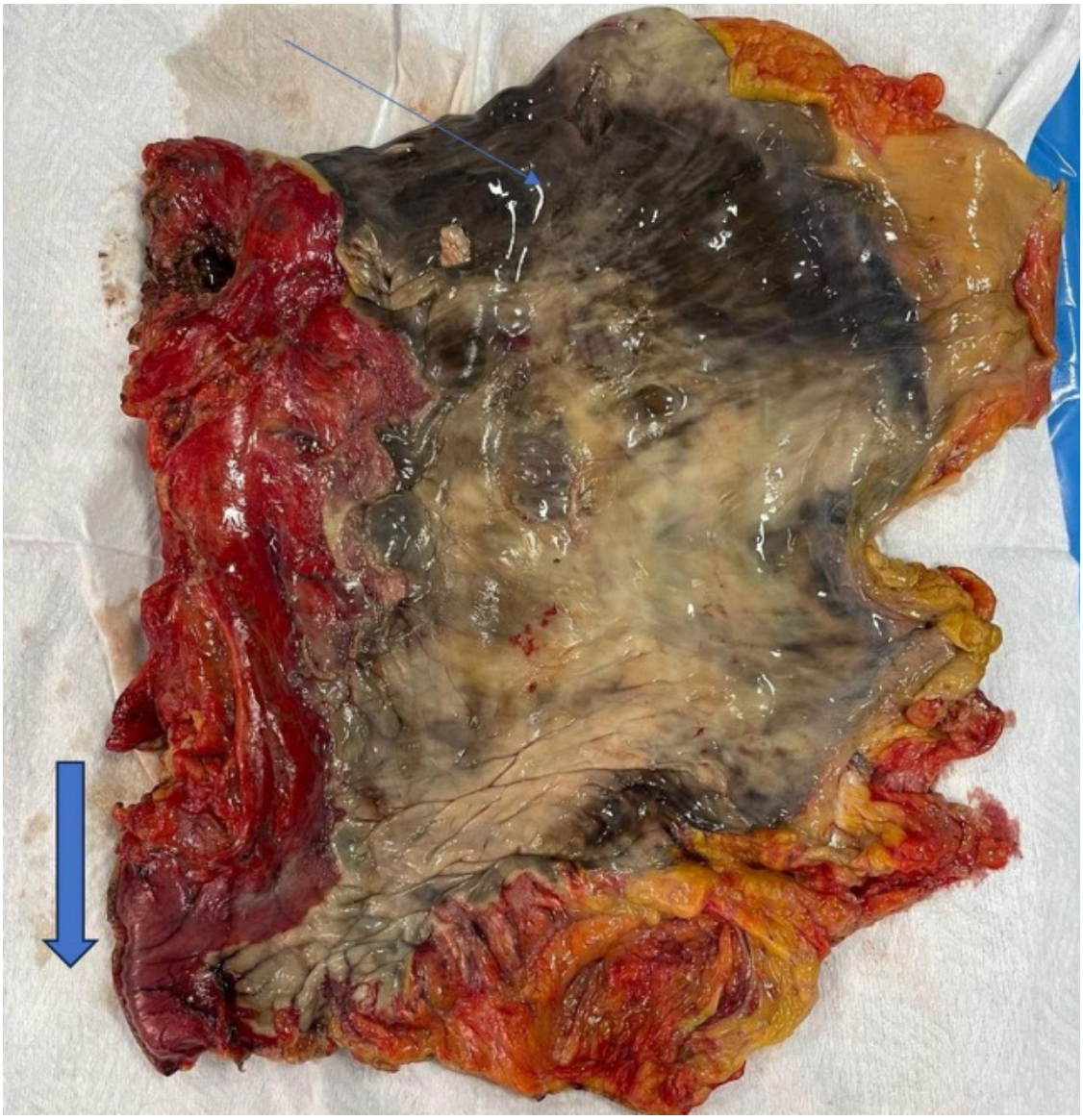

Figure 1: Macroscopic view of the resected stomach showing extensive transmural gastric necrosis. The specimen demonstrates diffuse black-gray discoloration and full-thickness necrosis involving both anterior and posterior gastric walls. The thin arrow indicates the cardia, while the thick arrow marks the pylorus. The posterior gastric wall appears paper-thin and friable, consistent with advanced ischemic changes. No signs of tumor or obstructive pathology are present. Surrounding omental tissue shows inflammation and hyperemia.

Histologically, there was extensive transmural involvement of the gastric wall characterized by abscess formation and necrotizing active chronic inflammation, ischemic changes, hyperemia in the omentum, fresh hemorrhage, focal abscesses with active chronic inflammation, and purulent peritonitis. There were no signs of reactive gastropathy or vascular lesions typically associated with nonsteroidal anti-inflammatory drug (NSAID) use.

During the postoperative course, the patient's acute renal failure resolved. Red blood cell suspension was administered, and anticoagulant therapy was initiated. By postoperative day 8, the patient regained consciousness. Nutritional support was provided through a nasogastric tube using enteral feeding and total parenteral nutrition.

The patient was later transferred to the ward and, on postoperative day 24, was discharged in good general condition, tolerating both oral diet and enteral nutrition, with stable vital signs and adequate urine output. No complications were observed during subsequent outpatient follow-ups

DISCUSSION

Acute gastric necrosis is considered rare due to the stomach's abundant vascular supply, which consists of five major arterial sources—the right and left gastric, right and left gastroepiploic, and short gastric arteries—as well as numerous minor and collateral vessels (4). Cadaveric studies have demonstrated that full-thickness vascular perfusion of the gastric wall can be achieved even with a single patent major artery. Experimental animal models have also shown that gastric necrosis may develop only after ligation of the right and left gastric arteries, the right and left gastroepiploic arteries, and up to 80% of the collateral circulation (5,6).

Several potential causes of this condition have been outlined in the literature, with the most frequently cited including AGD, thrombosis of mesenteric arteries, and AGD triggered by excessive food intake in association with psychogenic disorders such as polyphagia, bulimia, and Prader-Willi syndrome. Other recognized contributors include intrathoracic gastric herniation, volvulus, necrotizing gastritis, ingestion of corrosive agents, therapeutic vascular embolization, and complications arising in the postoperative period. (1–3).

Our patient exhibited multiple risk factors for compromised gastric perfusion. He had known vasculopathy and had undergone coronary artery bypass grafting (CABG). He also had atrial fibrillation. Notably, there was no evidence of hepatic or small intestinal ischemia, which effectively excluded celiac artery thrombosis. The role of digitalis in contributing to mesenteric vasospasm has been previously reported (7), but our patient had no history of digitalis use.

Additionally, the patient had venous congestion due to congestive heart failure. The etiology of impaired gastric perfusion in our patient was likely multifactorial, arising from a combination of these conditions. Remarkably, despite near-total gastric ischemia, the esophagus, duodenum, and small intestines remained viable and healthy.

Because of its infrequent occurrence, gastric ischemia is typically diagnosed late. Initial clinical signs—such as mild epigastric discomfort, nausea, or diarrhea—may quickly escalate to severe conditions like peritonitis, septic shock, and ultimately, death. Radiological findings such as pneumoperitoneum, gastric wall air (pneumatosis), or gas within the portal venous system on CT imaging can be suggestive. Nonetheless, a definitive diagnosis is most often made intraoperatively or confirmed postmortem.

In patients with necrosis and perforation, mortality rates after delayed surgical intervention have been reported as high as 80% (2,8). Advanced cases with necrosis and perforation require surgical intervention (1).

Surgical treatment options include decompressive surgery, partial gastrectomy, total gastrectomy with esophagojejunostomy, total gastrectomy with cervical esophagostomy, and feeding jejunostomy (2,8). In our case, laparotomy revealed complete ischemia and necrosis of the stomach. Thus, a total gastrectomy and Roux-en-Y esophagojejunostomy were performed.

Our patient represents one of the rare survivors among the few cases reported in the literature, being discharged in good health on postoperative day 24.

Due to its rarity, the diagnosis of gastric ischemia may be overlooked or delayed. However, this condition should be considered in any patient presenting with abdominal symptoms who has recent risk factors for impaired gastric perfusion, such as a history of CABG surgery.

REFERENCES

1. Turan M, Sen M, Canbay E, Karadayi K, Yildiz E. Gastric necrosis and perforation caused by acute gastric dilatation: report of a case. Surg Today. 2003;33(4):302-4.
2. Gündeş E, Küçükkartallar T, Tekin A, Çakır M. Gastric necrosis and perforation caused by acute gastric dilatation. Ulus Cerrahi Derg. 2013;9;30(3):179-81.
3. Huang G, Jin Y. Total gastric necrosis: A case report and literature review. Niger J Clin Pract. 2017;20(5):645-46.
4. Brown JR, Derr JW. Arterial blood supply of human stomach. Br J Surg. 1952;64(5):616-21.
5. Schein M, Saadia R. Postoperative gastric ischaemia. Br J Surg. 1989;76(8):844-8.
6. SOMERVELL TH. Physiological gastrectomy. Br J Surg. 1945;33:146-52.
7. Bradbury AW, Brittenden J, McBride K, Ruckley CV. Mesenteric ischaemia: a multidisciplinary approach. Br J Surg. 1995;82(11):1446-59.
8. Todd SR, Marshall GT, Tyroch AH. Acute gastric dilatation revisited. Am Surg. 2000;66(8):709-10.

Abbreviations list

AGD: Acute Gastric Dilatation
CABG: Coronary Artery Bypass Grafting
WBC: White Blood Cell
BUN: Blood Urea Nitrogen
INR: International Normalized Ratio
PCO_2: Partial Pressure of Carbon Dioxide
CT: Computed Tomography
GIST: Gastrointestinal Stromal Tumor
NSAID: Nonsteroidal Anti-inflammatory Drug

Ethics approval and consent to participate.

Not applicable. This case report did not involve experimental interventions on humans or animals. Written informed consent for treatment was obtained from the patient according to institutional protocols.

Consent for publication

Written and verbal informed consent was obtained from the patient for the publication of this case report and accompanying images.

Availability of data and materials

Not available.

Funding

The authors received no financial support for the research and/or authorship of this article. There is no funding source.

Authors' contributions

BT: Surgery and clinical management. MEA, SYK, HC, İK, ES, GŞ, ÖÇ: data collection, literature review, and manuscript drafting. BT, SA: Critically revised. All authors read and approved the final manuscript.

Acknowledgements

None.