

# Chronic Gastric Ischemia Leading to Gastric Perforation

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## ABSTRACT

A 69-year-old man with diabetes, peripheral vascular disease, and hypertension presented with 3 months of diffuse abdominal pain that worsened with meals, weight loss, and dysphagia. Esophagogastroduodenoscopy and computed tomography revealed findings consistent with chronic gastric ischemia secondary to atherosclerosis. Gastric ischemia eventually led to perforation. We discuss causes, symptoms, diagnosis, and management of gastric ischemia, an underdiagnosed and potentially fatal condition that requires urgent diagnosis and treatment.

## INTRODUCTION

Chronic gastric ischemia is an uncommon condition that is often misdiagnosed as more benign entities such as gastritis or peptic ulcer disease. Classic symptoms are nonspecific and include postprandial abdominal pain, nausea, vomiting, weight loss, and gastrointestinal bleeding. The substantial collateral blood supply of the stomach is protective against this rare entity. Overall, chronic gastric ischemia due to atherosclerosis has an exceptionally poor prognosis as a result of its insidious presentation, which leads to delays in diagnosis and intervention.

## CASE REPORT

A 69-year-old man with history of type 2 diabetes, hypertension, peripheral arterial disease, and femoral popliteal bypass presented for evaluation of 2 months of recurrent abdominal pain, 11.25-kg weight loss, nausea, and vomiting. Two weeks prior to admission, he was admitted to an outside hospital for these symptoms. Work-up at the time included an esophagogastroduodenoscopy (EGD) showing a “blue stomach” and abdominal computed tomography (CT) revealing gastric wall emphysema. He was able to tolerate a minimal diet and was discharged to a rehabilitation facility, where his abdominal symptoms worsened. He was brought to our hospital for further evaluation.

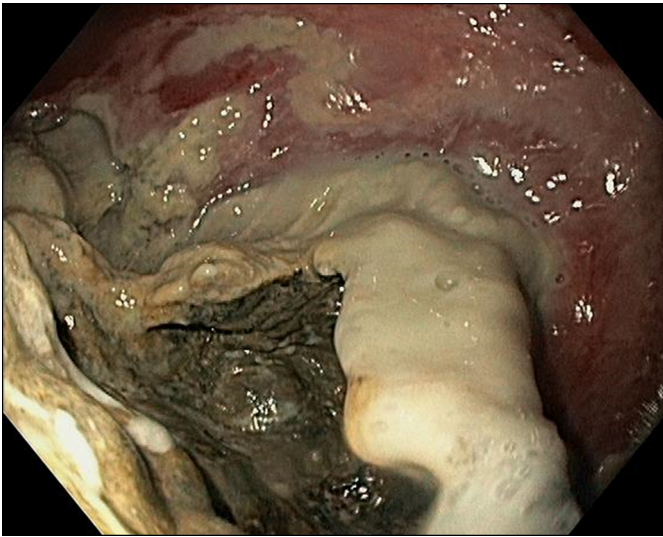
On admission, the patient complained of constant mild abdominal pain in the right upper quadrant, nausea with retching, and non-bloody emesis. He also complained of episodic intense epigastric pain and dysphagia associated with meals. On examination, the patient was alert and fully oriented, afebrile, and hemodynamically stable. There is no history of hypotension, procedure-related or otherwise, throughout his hospitalization. His abdomen was soft, with tenderness to deep palpation only at the right upper quadrant. No abdominal bruit was appreciated. The remainder of the physical exam was unremarkable. Laboratory findings were significant for leukocytosis 21,500 cells/ $\mu$ L with 73% neutrophils, normal lipase and liver function tests, and normal lactate 1.4 mmol/L. Abdominal CT with intravenous (IV) contrast showed no evidence of acute gastric ischemia, with resolution of prior gastric emphysema.

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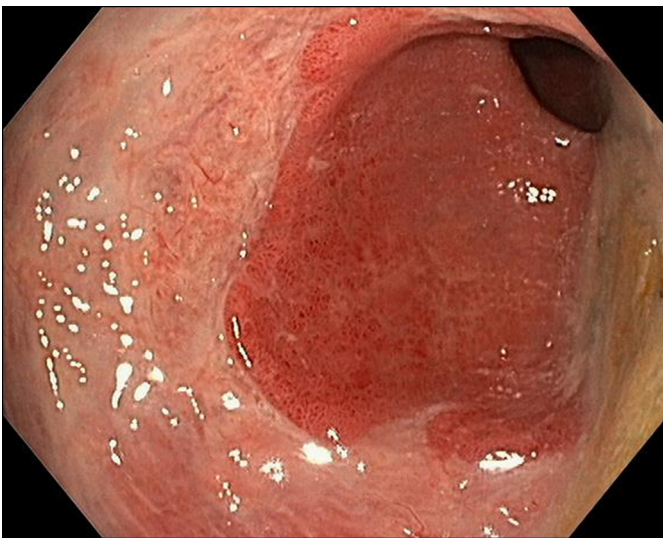
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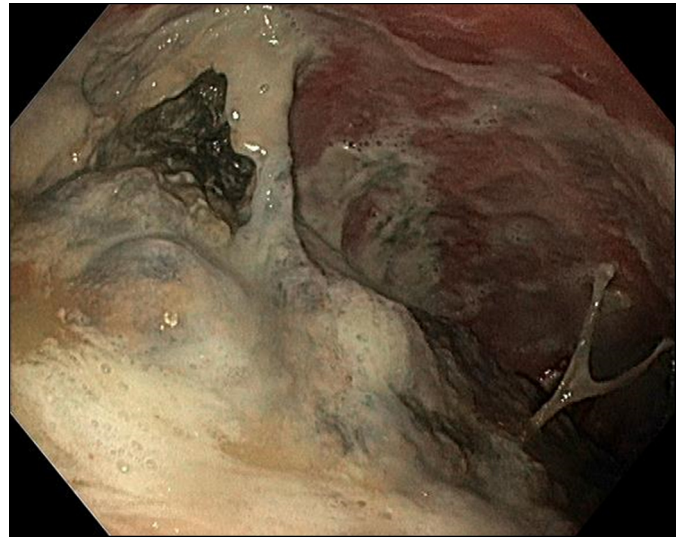
**Figure 1.** Upper endoscopy on hospital day 2 visualized a deep central ulceration in the proximal body and fundus of the stomach.

EGD on hospital day 2 revealed a normal esophagus and duodenum, excavated lesions, and a large contiguous area of pale scarring with deep central ulceration in the proximal body and fundus of the stomach (Figure 1). Normal gastric mucosa with a sharp demarcation was found in the antrum and cardia (Figure 2). Multiple gastric biopsies obtained from the scarred proximal body and fundus, including the large central ulcer, revealed ulceration and necrosis.

Despite IV pantoprazole twice daily, IV piperacillin-tazobactam, and sucralfate, he continued to have nausea and retching, eventually progressing to hematemesis on hospital day 5 with concurrent worsening leukocytosis.

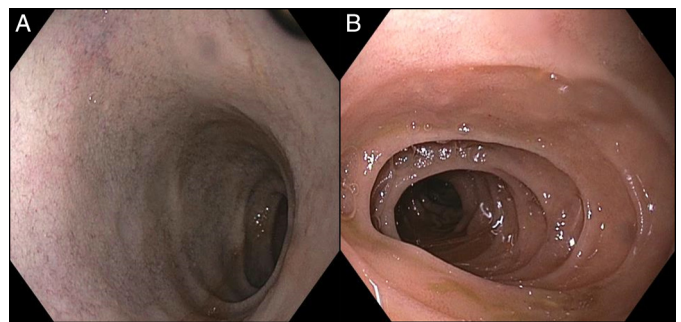


**Figure 2.** Upper endoscopy also noted a large contiguous area of pale scarring throughout the stomach body with a sharp demarcation to normal mucosa in the antrum.



**Figure 3.** Repeat upper endoscopy on hospital day 6 visualized the necrotic ulcer in the body and fundus, which had become more extensive.

Repeat EGD on hospital day 6 revealed that the necrotic ulcer had become more extensive, covering the body and fundus of the stomach (Figure 3). In addition, mucosal pallor and lack of vascularity extended to the duodenal bulb while the second part of the duodenum appeared normal (Figure 4). CT angiography (CTA) revealed atheromatous changes with significant narrowing at both the celiac trunk and superior mesenteric artery (SMA) origins (Figure 5). Subsequently, an urgent mesenteric angiogram revealed complete occlusion of both the celiac trunk and the SMA with patent inferior mesenteric artery and unsuccessful cannulation of the SMA. The patient developed lactic acidosis and required intubation after the procedure. His lactate, for the first time, was elevated and peaked at 7 mmol/L. Given the extent of vascular occlusion, angioplasty was not a viable option, which left mesenteric bypass as the only therapeutic intervention. An emergent exploratory laparotomy revealed a gastric perforation along the greater curvature of the stomach, corresponding to the area of



**Figure 4.** Repeat upper endoscopy also showed (A) pale blue mucosa in the duodenal bulb contrasting with (B) normal mucosa in the second part of the duodenum.



**Figure 5.** CTA in sagittal cross-section shows high-grade stenosis (arrow) of the celiac trunk and superior mesenteric artery origins at the aorta.

deep ulceration seen previously on EGD. Repair of the gastric perforation and right renal artery to the common hepatic artery bypass using the great saphenous vein were performed. The postoperative course was complicated by mesenteric ischemia requiring small bowel resection, with pathology showing loss of villous architecture, acute hemorrhage, and granulation tissue compatible with bowel ischemia. He developed septic shock and died of multi-organ failure on hospital day 35.

## DISCUSSION

Gastric ischemia is a rare entity due to the rich vascular supply of this organ. There are five major arterial sources: the right and left gastric, right and left gastroepiploic, and the

short gastric arteries. Collateral arterial sources also form an extensive blood supply. In live animal models, 95% of the arterial supply to the stomach may be ligated without injury to the mucosa.<sup>1</sup> Gastric ischemia can present as acute or chronic abdominal pain. Acute gastric ischemia results from etiologies such as systemic hypotension, gastric volvulus, disseminated thromboembolism, and vasculitis. As described in our patient, however, chronic gastric ischemia secondary to atherosclerosis can have an insidious presentation that can result in delayed diagnosis and intervention.<sup>1</sup>

Gastric ischemia is often misdiagnosed. It is prudent to consider gastric ischemia in patients with gastric ulcers that fail to respond to conservative therapy and proton pump inhibitors, as well as in patients with other signs of vasculopathy for whom common causes have been ruled out. Risk factors include diabetes, history of smoking, hypertension, and atherosclerotic disease. The initial diagnostic work-up includes endoscopy and imaging studies, especially CTA. Importantly, diagnostic endoscopy is safe in gastric ischemia, even in the presence of gastric pneumatosis.<sup>2</sup> In urgent cases, interventional angiography can be both diagnostic and therapeutic without obtaining a CTA first. As described in our patient, endoscopic findings of large gastric ulcerations in the fundus are common, due to impairment of the gastric mucosal barrier with accumulation of gastric acids in a fundal pocket when lying supine.<sup>2</sup> Intravascular stenting remains the recommended intervention for chronic mesenteric ischemia.<sup>3</sup>

In cases of non-healing gastric ulcers that are negative for *Helicobacter pylori* and do not respond to conservative therapy with bowel rest and IV proton pump inhibitor therapy, it is imperative that mesenteric ischemia is considered in the differential as early vascular intervention is life-saving, particularly in patients with atherosclerotic vascular disease.<sup>4</sup> Gastric ischemia is an underdiagnosed condition and has a poor prognosis. Early diagnosis and intervention have the potential to significantly reduce morbidity and mortality.

## DISCLOSURES

Author contributions: E. Lundsmith performed the literature search. E. Lundsmith and M. Zheng wrote the manuscript. P. McCue provided the pathology slide and description. B. Niu provided the endoscopic picture, revised the manuscript, and is the article guarantor.

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Informed consent was obtained for this case report.

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