

# A Rare Case of Chronic Gastric Ischemia with Gastric Ulceration and Anemia

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## Keywords

*Ischemia, Atherosclerosis, Gastric Ulceration, Gastrointestinal Bleeding, Abdominal Pain, Mesenteric*

## Abstract

We present the patient case of a 42-year-old woman with clinically significant abdominal pain and repeated episodes of gastrointestinal bleeding, who was ultimately diagnosed with chronic gastric ischemia with recurrent gastric ulcers. Current clinical literature suggests that gastric ischemia is more common than previously thought. A higher degree of suspicion for such relatively rare presentation may be required in the clinical setting.

## Background

Gastrointestinal mesenteric ischemia is a reduction in intestinal blood flow, either acutely or secondary to chronic hypoperfusion of the small intestine, and typically presents in patients with multivessel mesenteric stenosis or occlusion. Because of the redundant blood supply of the stomach, gastric ischemia with ulceration is a rare manifestation of mesenteric ischemia.

## Objective

To highlight the multidimensional presentations of chronic gastric ischemia and to emphasize the need for a higher index of suspicion, when patients present with recurrent gastric ulcerations in relevant and related vascular territories.

## Case Report

A 66-year-old woman initially presented to our health care center with symptoms of abdominal pain, passage of black tarry stools, and early satiety. She was admitted for acute anemia and suspected lower gastrointestinal bleeding. The first esophagogastroduodenoscopy (EGD) showed several nonbleeding ulcers and pale-appearing mucosa. Meanwhile, stigmata of recent bleeding were evident. A 19-mm ulcer was seen on the greater curvature of the stomach, as were 2 additional ulcers at the antrum and 1 ulcer on the lesser curvature of the stomach. She was taken under treatment with proton-pump inhibitors with plans to repeat EGD and to follow-up her biopsy results as an outpatient.

Two months later, she was readmitted with symptoms of severe diarrhea and early satiety. CT scans of the abdomen showed severe atherosclerosis of the mesenteric vessels and sigmoid colitis. The second EGD revealed moderately erythematous mucosa throughout the stomach with no ulceration. Biopsies were negative for *Helicobacter pylori*, celiac disease and malignancy. Several nonbleeding angioectasias were seen in the greater and lesser curvatures of the stomach. A colonoscopy revealed nonbleeding, ulcerated mucosa in the ascending colon. She was discharged to outpatient follow-up.

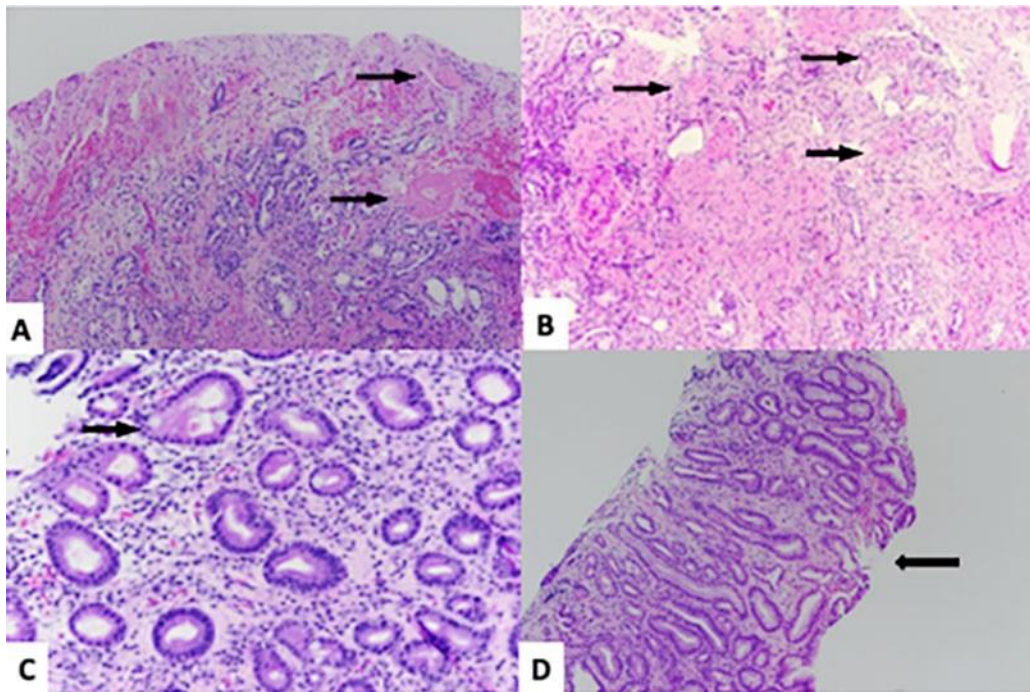
Five months after the initial presentation, she again presented with melena and one episode of hematemesis. The third EGD showed recurrent ulcers in the gastric body, antrum and prepyloric region. After consulting with the vascular surgery department, a mesenteric artery duplex ultrasound was performed to investigate chronic ischemia as the source of recurrent gastric ulceration. Duplex ultrasound showed significant stenosis in superior mesenteric (SMA) and inferior mesenteric (IMA) arteries of more than 70%. The patient subsequently had mesenteric angiography, which showed high-grade focal stenosis at the origin of the SMA, which was confirmed with intravascular ultrasound, as well as stenosis of the IMA and heavy calcification in the origin of the celiac artery (CA). The SMA was treated with balloon angioplasty and a covered metal stent. Dual antiplatelet therapy was continued after the patient was discharged.

The patient was hospitalized for the fourth time for melena and anemia 5 months after initial admission. Mesenteric angiogram showed the SMA was patent. The fourth EGD revealed large areas of pale gastric mucosa with ischemic appearing geographic ulcers in the body of the stomach (Figure 1A-C).



**Figure 1.** Endoscopic finding suggestive of gastric ischemia. (A) Ischemic ulcer in the body of the stomach, (B) Pale gastric mucosa, and (C) A healing ulcer

Biopsies showed histologic evidence of gastric ischemia (Figure 2A-D). Treatment with additional stent placement in the celiac artery was discussed and ultimately deferred because of the high risks associated with further intervention.



**Figure 2.** Histologic findings of chronic gastric ischemia. (A) Mucosal ischemic changes with vascular thrombi (arrows). (B) Submucosal vascular thrombi (arrows). (C) Chronic inflammation and focal intestinal metaplasia (arrow pointing to goblet cells). (D) Healing erosion and fibrosis of lamina propria (arrow).

## Discussion

Mesenteric ischemia either acute or chronic is caused by blood flow that is insufficient to meet the metabolic demands of the visceral organs. The severity of ischemia and the type of organ involved depend on the affected vessel and the extent of collateral-vessel blood flow. More than 90% of cases of chronic mesenteric ischemia are related to progressive atherosclerotic disease that affects the origins of the visceral vessels. In 40 to 50% of cases mesenteric ischemia is associated with embolic occlusion, 20 to 35% cases are caused by thrombotic occlusion of the previously stenotic mesenteric vessel. Other causes include thrombophilia, vasculitis and low flow states. More than 70% of cases occur in females and the prevalence increases with age and in those with the history of hypertension, atherosclerosis, smoking and diabetes (1).

Gastric ischemia was first identified in 1995 with 4 cases noted in an autopsy series of 24000 patients (2). Chronic gastric ischemia can manifest as vague symptoms such as abdominal pain or angina, nausea, vomiting, diarrhea, and occult or overt GI bleeding. Chronic gastric is caused by the atherosclerosis of the mesenteric arteries, primarily of the CA and SMA. The CA is the major blood supply of the stomach and supplies the liver, spleen, pancreas, duodenal bulb and the descending duodenum proximal to the major papilla. The SMA supplies the duodenum distal to the major papilla, jejunum, ileum, ascending colon and the proximal two-thirds of the transverse colon. IMA supplies the distal one-third of the transverse colon, descending colon, sigmoid and rectum (3, 4).

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. The initial diagnostic work-up includes endoscopy and imaging studies (computed tomography angiography and selective mesenteric angiography) as in our patient case (5, 6). Endoscopic evaluation is a valuable tool in diagnosis, with ischemia suggested by findings of pale mucosa, loss of mucosal vascular pattern, or erosions/ulcers in atypical locations in the stomach, including the anterior or posterior wall (6, 7). Biopsy may confirm ischemic injury when performed early in presentation and help rule out other causes. A multidisciplinary approach with vascular surgery is essential in establishing a definitive diagnosis and for management because definitive treatment of ischemic ulcers may include vascular stenting.

In this patient case, the diagnosis of gastric ischemia was delayed because of vague presenting symptoms (diffuse abdominal pain, early satiety, nausea, diarrhea and intestinal bleeding). A higher degree of suspicion for gastric ischemia is needed in patients with existing vasculopathies and recurrent ulcers in regions supplied by celiac artery and superior mesenteric artery to facilitate early detection and management and minimize morbidity and mortality from this disease.

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