

Phlegmonous Gastritis Refractory to Antibiotics with Subsequent Improvement on Empiric Quadruple Therapy

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ABSTRACT

Phlegmonous gastritis is a rare infection of the gastric wall that presents with nonspecific symptoms and can rapidly progress with high mortality. We report a 59-year-old woman with two days of intractable nausea, vomiting, and inability to tolerate oral intake while on chronic proton-pump inhibitor therapy. She had leukocytosis, diffuse gastric wall thickening on computed tomography, and severe gastric mucosal abnormalities with antral sparing on endoscopy. Biopsy showed acute purulent gastritis but was negative for *Helicobacter pylori*. Given the unclear etiology and incomplete response to initial broad-spectrum antibiotics, empiric *H. pylori* quadruple therapy was initiated. The patient subsequently demonstrated clinical improvement without requiring surgical intervention.

KEYWORDS: Phlegmonous Gastritis; *Helicobacter pylori*; Endoscopy

INTRODUCTION

Phlegmonous gastritis (PG), also known as purulent gastritis, is a rare but potentially life-threatening bacterial infection of the gastric wall, marked by localized or diffuse suppurative inflammation of the submucosa and muscularis layers. Without prompt treatment, it can rapidly progress to necrosis, perforation, or septic shock.¹ PG typically presents with nonspecific symptoms, including epigastric pain, fever, nausea, and systemic signs of infection. Endoscopy, imaging, and histopathology remain the mainstay of diagnosis, while treatment consists of broad-spectrum intravenous antibiotics, with surgery reserved for severe or refractory cases.² Risk factors include mucosal injury (e.g., endoscopic procedures, peptic ulcer disease), immunosuppression, malignancy, and chronic alcohol use.²⁻⁴ *Streptococcus* spp. are the most common causative organisms. Although rare, *Helicobacter pylori*-associated PG has also been documented.^{5,6} We present a case of acute PG initially unresponsive to broad-spectrum antibiotics that improved following empiric quadruple therapy, with *H. pylori* considered a possible but unconfirmed contributing factor.

CASE DESCRIPTION

A 57-year-old woman presented to the hospital with two days of intractable nausea, vomiting, left-sided abdominal pain, and inability to tolerate oral intake. Her past medical history was significant for relapsing-remitting multiple sclerosis, for which she was not receiving disease-modifying therapy, and generalized anxiety disorder. She had no significant family history. Her home medications included a daily proton pump inhibitor (PPI). She endorsed chronic constipation and denied diarrhea, fever, chills, sick contacts, or change in her dietary habits, including any unusual food items.

On presentation, she was tachycardic with heart rates up to 127 beats per minute. Laboratory evaluation revealed leukocytosis with a white blood cell count of $20.2 \times 10^9/L$ (reference range $4-10 \times 10^9/L$) and an elevated lactate level of 2.1 mmol/L. Computed tomography (CT) of the abdomen demonstrated diffuse wall thickening of the gastric body, suggestive of gastritis [Figure 1].

The patient underwent Esophagogastroduodenoscopy (EGD) on hospital day 2, which revealed severe, unusual-appearing gastric mucosa with antral sparing [Figures 2A,B].

Figure 1. Contrast-enhanced computed tomography (CT) of the abdomen and pelvis demonstrating diffuse wall thickening and edema of the gastric body (yellow arrow).



Figure 2A. Initial esophagogastroduodenoscopy demonstrating diffuse severe erythema of the gastric body.

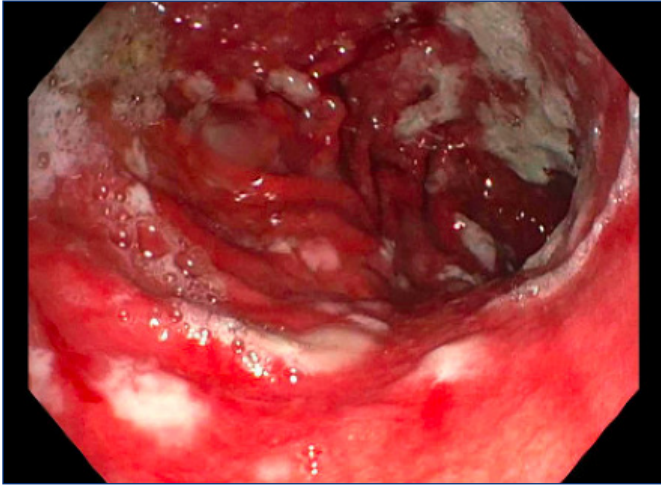
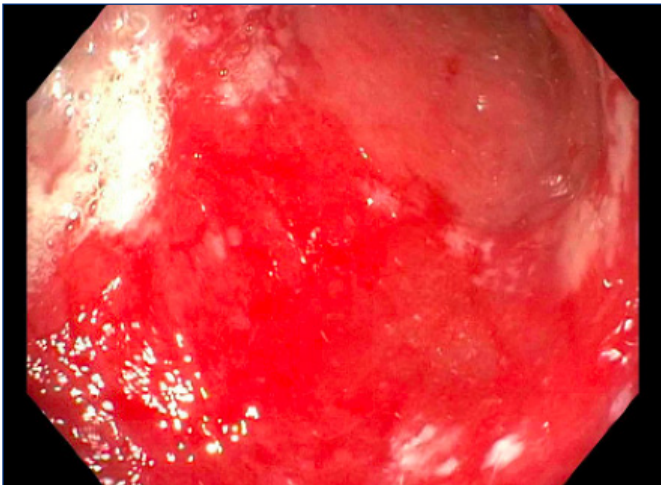


Figure 2B. Initial esophagogastroduodenoscopy demonstrating overlying inflammatory exudates within the gastric body mucosa.



Histopathologic examination of biopsy specimens showed diffuse acute purulent gastritis with surface ulceration and erosion, crypt abscesses, and dense neutrophilic infiltration. Special stains were negative for *H. pylori*, fungal organisms, viral inclusions, and malignant cells. Blood cultures remained negative. She was initially treated with intravenous ceftriaxone 2g every 24 hours, intravenous metronidazole 500mg every 8 hours, intravenous pantoprazole 40mg twice daily, and intravenous famotidine 40mg nightly.

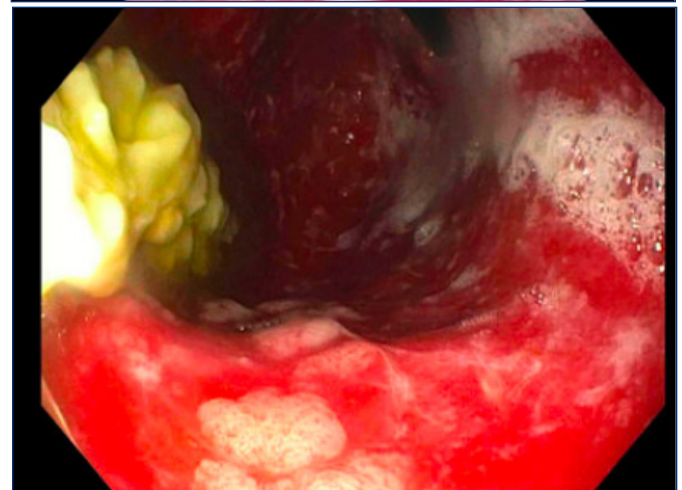
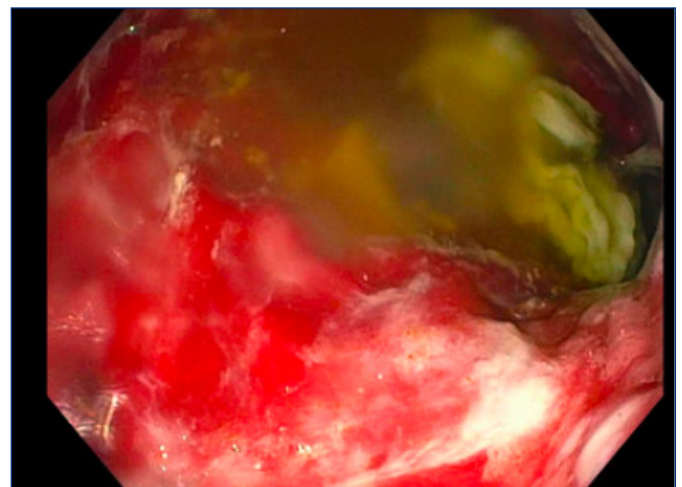
Given her prior use of a PPI, the possibility of a false-negative *H. pylori* result was considered. On hospital day 6, she was empirically started on quadruple therapy, consisting of clarithromycin, metronidazole, amoxicillin, and a PPI for a planned duration of 14 days, and ceftriaxone was discontinued. A stool antigen test for *H. pylori* subsequently returned negative.

Despite treatment, the patient continued to experience abdominal pain requiring opioid analgesia, along with persistent leukocytosis and tachycardia. A repeat EGD was performed on hospital day 8, demonstrating persistent acute gastritis characterized by intense erythema and exudates involving the gastric fundus and body, with an abrupt transition to normal-appearing mucosa in the antrum [Figures 3A,B]. Repeat biopsy specimens were again negative for *H. pylori*, viral inclusions, and malignancy. Sucralfate was initiated on hospital day 8 for additional symptom management.

By hospital day 13, the patient's white blood cell count had normalized to $9.7 \times 10^9/L$, and she was tolerating an oral diet. She was subsequently discharged home.

At outpatient follow-up approximately two weeks later, she had completed the full course of empiric *H. pylori* therapy. She reported persistent but mild nausea and epigastric discomfort, rated as 2 out of 10 in severity, for which she was advised to resume sucralfate as needed. A repeat outpatient EGD was planned for 2–3 months to assess for endoscopic resolution. However, the patient was lost to follow-up.

Figures 3A and 3B. Repeat esophagogastroduodenoscopy demonstrating persistent erythema and inflammation of the gastric body.



DISCUSSION

PG is an elusive diagnosis that can rapidly progress to clinical deterioration if not recognized early. Although only several thousand cases have been documented worldwide, it remains a rare but highly fatal condition, and this case attempts to fill the current void in literature on this rare diagnosis.⁷ Historically, mortality rates approached 90% but have declined to below 50% with antimicrobial therapy.^{6,8,9}

The pathophysiology of PG involves bacterial infiltration of the gastric wall, typically beginning in the submucosa via direct mucosal invasion, hematogenous or lymphatic spread, or ingestion of pathogenic organisms.^{10,11} Once established, infection can rapidly extend through the gastric wall, including the serosa. Based on the extent of involvement, PG is classified as either localized or diffuse, with the diffuse form associated with significantly higher mortality rates.¹² In this case, endoscopic findings were most consistent with the diffuse pattern.

Streptococcus spp. are the most common causative organisms, though polymicrobial infections involving gram-negative bacilli and anaerobes have also been reported.¹³⁻¹⁵ Although rare, *H. pylori*-associated PG has also been documented.^{5,6}

Our case presents with classic features as reported in the literature, including acute abdominal pain, systemic inflammatory response, and marked leukocytosis.^{6,10,16} Our patient was relatively young, without significant comorbidities or immunosuppression, and demonstrated clinical improvement following empiric *H. pylori*-directed therapy in addition to initial broad-spectrum antibiotics, thereby avoiding the need for surgical intervention. Although mucosal injury is a recognized risk factor, endoscopy remains essential for definitive diagnosis and longitudinal disease assessment.

Given the rarity of PG and the lack of standardized treatment guidelines, management is often individualized. Among the two documented cases of possible *H. pylori*-associated PG, one case from Portugal progressed to refractory shock with multiorgan dysfunction and resulted in death,⁶ while the other was successfully treated with medical therapy alone.⁵ In cases refractory to initial broad-spectrum antibiotic therapy, empiric 14-day quadruple therapy may be considered, particularly when *H. pylori* cannot be reliably excluded and diagnostic uncertainty persists. While this regimen primarily targets *H. pylori* and includes agents with activity against some anaerobic and gram-positive organisms, it does not provide comprehensive coverage for typical pathogens implicated in PG. Its use in this setting may be considered an adjunctive or alternative empiric approach when response to standard therapy is inadequate. This may be particularly relevant in patients receiving acid suppression, where diagnostic testing for *H. pylori* may be less reliable.

This case reinforces the critical role of early diagnosis and endoscopic evaluation in the management of this highly

morbid condition. It also suggests a possible role for *H. pylori* in select cases, although causality remains unproven. A broad infectious differential should be maintained. Unfortunately, the patient was lost to follow-up, underscoring loss of outpatient continuity as a significant barrier to care, and emphasizing the need for coordinated transitions between inpatient management and outpatient primary care and gastroenterology follow-up to ensure complete disease resolution and organism identification.¹⁷

Our case has several limitations that preclude establishing *H. pylori* as the definitive cause of PG. Histopathologic staining and stool antigen testing were negative, arguing against active infection, and although chronic PPI use may reduce diagnostic sensitivity, this does not provide confirmatory evidence. The absence of microbiologic confirmation further limits identification of the causative pathogen, and alternative etiologies such as *Streptococcus* species or a polymicrobial process remain plausible. In addition, the temporal association between initiation of quadruple therapy and clinical improvement does not establish causality and may reflect a delayed response to prior antibiotics, cumulative antimicrobial coverage, or mucosal healing with acid suppression. Finally, loss to follow-up precluded serologic evaluation and repeat testing, which may have provided further clarification of infection status. These limitations highlight the diagnostic challenges of PG and support maintaining a broad infectious differential even when a specific organism is suspected.

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Conflicts of interest statement: The authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed.

Prior Presentation: This case was previously presented at the Annual Scientific Meeting of the American College of Gastroenterology (ACG) 2024, held in Philadelphia, Pennsylvania, USA, on October 28, 2024. <https://acg2024.eventscribe.net/fsPopup.asp?PosterID=683656&mode=posterInfo>

Patient Consent Statement: Repeated and documented attempts were made to contact the patient and the patient's designated emergency contact to obtain written informed consent for publication. Despite exhaustive efforts to reach the patient, next of kin, and/or parent or guardian, informed consent could not be obtained, as the patient was lost to follow-up. No directly or indirectly identifiable patient information is included in this manuscript. In accordance with ethical standards, the authors believe that publication of this case poses minimal risk to patient privacy. This case also underscores the clinical importance of robust follow-up mechanisms to reduce loss to follow-up in routine medical care.

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