



Phlegmonous Gastritis Associated with Gastric Stasis and Immunosuppression: A Case Series

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Abstract

Background: Phlegmonous gastritis is a rare condition caused by bacterial infection of the gastric wall. Largely regarded as an acute process with poor prognosis, published reports describe a wide variation in its clinical presentation. In this study, we describe five cases of phlegmonous gastritis seen at a single large academic referral center that demonstrates an association with immunosuppression and gastric stasis.

Case Presentation: Two Caucasian females and three Caucasian males, ages 35 to 78 years, were identified with histologic and/or endoscopic characteristics of phlegmonous gastritis. Abdominal pain, nausea, and vomiting were the most common presenting symptoms. Two patients underwent outpatient evaluation, while the three other patients required hospital admission. On endoscopy, superficial gastric ulcerations, mucosal friability, and diffuse exudate were noted. Endoscopic biopsies showed evidence of severe active and suppurative gastritis. At diagnosis, three patients were immunosuppressed whose comorbidities included rheumatoid arthritis on abatacept, amyloidosis status-post stem cell transplantation, and Crohn's disease on leflunomide therapy. The remaining two patients had evidence of gastric stasis due to gastroparesis and gastric outlet obstruction respectively. In all cases, symptomatic resolution was achieved with medical management including broad-spectrum antibiotics and proton pump inhibitor.

Conclusion: The rarity of phlegmonous gastritis and its diverse presentation represent a diagnostic challenge. Improved recognition of the factors, such as immunosuppression, which predispose to this condition is crucial for timely management and may provide further insight to understanding its pathophysiologic mechanisms. Our case series further demonstrates that gastric stasis and inflammatory bowel disease may be under-recognized risk factors and should be areas of future study.

Keywords: Phlegmonous gastritis; Gastric stasis; Immunosuppression; Inflammatory bowel disease

Abbreviations

PhG: Phlegmonous Gastritis; CT: Computed Tomography; EUD: Endoscopic Ultrasound; IBD: Inflammatory Bowel Disease

Background

PhG is a rare clinical condition associated with bacterial infiltration of the gastric wall. A variety of microorganisms have been associated with this infectious process. It appears to predominately affect older patients and men [1]. Several predisposing factors have been reported including immunosuppression, autoimmune diseases, mucosal damage, and malignancy [1-6].

Though generally considered an acutely fulminant condition, the clinical presentation of PhG ranges from epigastric pain [4] to acute abdomen [1]. Presently, PhG is a clinical diagnosis supported by radiologic, endoscopic, and histologic evidence. Computed tomography (CT) [1,4] and endoscopic ultrasound (EUS) [1,2] are particularly helpful in assessing for gastric wall gangrene, emphysema, necrosis or intramural hemorrhage [2,4,7]. The endoscopic features of PhG are diverse and may include thickening of the gastric mucosa, ulceration, erosion, edema [1,4] violaceous and inflamed gastric mucosa with fibrino-purulent exudate [3]. Histological presentation of PhG includes necrosis, neutrophil and plasma cell infiltrates, granulation tissue, intramural hemorrhage,

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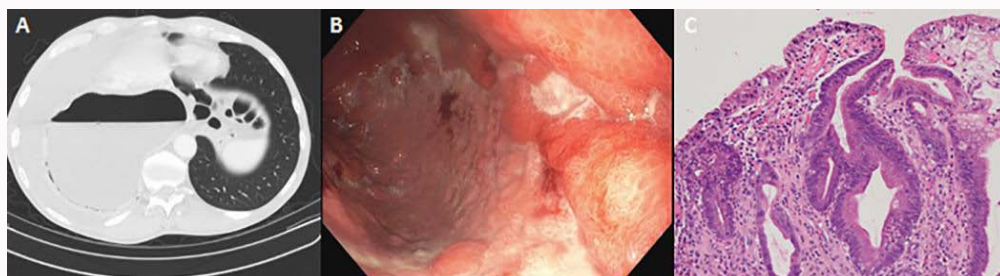


Figure 1: Dilation and pneumatosis of gastric conduit on abdominal CT (A). EGD demonstrated numerous discrete and confluent gastric ulcerations (B). Histological changes of acute inflammation and ulceration on biopsy (C).

or thrombosis of submucosa blood vessels [4,5].

Given its rarity, much of the current understanding of PhG is derived from case reports while its underlying pathogenesis remains unknown. We describe the experience of a large tertiary referral center with PhG as illustrated by five cases.

Case Presentation

Case 1

A 63-year-old Caucasian male with remote esophageal gastric junction adenocarcinoma status-post Ivor Lewis gastrectomy and pyloromyotomy presented to the hospital with one week of epigastric pain and vomiting. On admission, abdominal CT demonstrated dilation and pneumatosis of the gastric conduit (Figure 1A). An esophagoduodenoscopy (EGD) showed numerous discrete and confluent gastric ulcerations (Figure 1B); biopsies from these regions showed acute inflammation and ulceration (Figure 1C). As these clinical features were highly suggestive of acute PhG, the patient was initiated on 7-day course of piperacillin/tazobactam and proton pump inhibitor. Symptoms gradually improved with medical therapy, and he was discharged home.

Case 2

A 65-year-old Caucasian male with systemic AL amyloidosis presented with acute rectal bleeding and hematemesis 13 days after undergoing autologous cell transplantation. An EGD showed friable gastric mucosa with adherent green-black exudate covering the entire gastric fundus. Biopsies demonstrated severe acute gastritis consistent with PhG. A CT abdomen and pelvis showed fluid density material with trapped pockets of gas within the gastric lumen adjacent to the wall of the fundus. The patient was initiated on vancomycin and piperacillin/tazobactam, and repeat EGD 2 weeks later demonstrated mucosal healing. The patient's hospital course was complicated by manifestations of systemic amyloidosis and eventually the patient passed away due to amyloidosis-related complications in the intensive care unit.

Case 3

A 78-year-old Caucasian female, with a history of rheumatoid arthritis on immunosuppressive therapy with rituximab and abatacept, was referred for outpatient evaluation of acute on chronic postprandial abdominal pain and weight loss. An EGD demonstrated a few, large non-bleeding erosions in the gastric antrum (Figure 2A). Biopsies of the ulcerated regions demonstrated severe active chronic gastritis, histologically consistent with PhG (Figure 2B). A CT enterography study was also significant for multiple thick-walled enhancing strictures involving the proximal and mid-ileum with proximal small bowel dilation. The patient was initiated on a 10-day

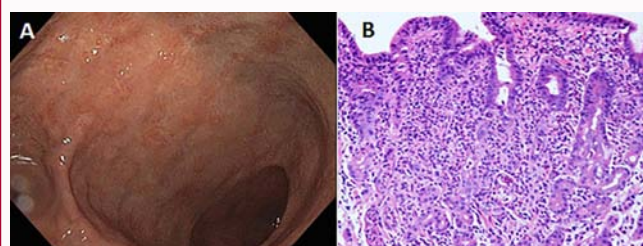


Figure 2: Large erosions in gastric antrum on EGD (A). Severe active chronic gastritis, consistent with PhG, was present on histology (B).

course of clindamycin and ciprofloxacin which led to improvement in her postprandial symptoms. An exploratory laparotomy was later performed with resection of the ileum. Pathologic review of the resected specimen was consistent with Crohn's disease.

Case 4

A 74-year-old Caucasian male with idiopathic recurrent small-bowel obstruction was admitted to the hospital with a several week history of generalized malaise and melena. On presentation, an EGD was performed and was notable for retained food and generalized friable appearance to the gastric mucosa. Corresponding gastric biopsies were consistent with acute suppurative gastritis representative of PhG (Figure 3). The patient was started on levofloxacin and was discharged home following clinical improvement. Several days after discharge, the patient was readmitted with acute nausea, vomiting, and diarrhea. An abdominal x-ray showed multiple distended loops of small bowel and sigmoid decompression, consistent with small-bowel/proximal colonic obstruction. Following a course of medical management including bowel rest, the patient's symptoms resolved.

Case 5

A 35-year-old Caucasian female with Crohn's disease on leflunomide therapy presented to clinic with chronic nausea and

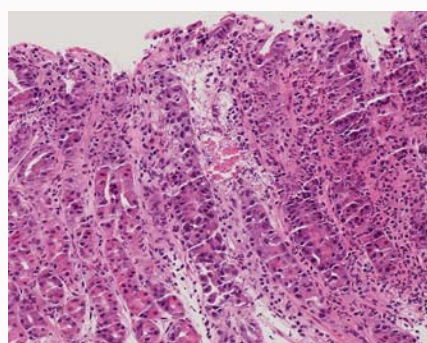


Figure 3: Acute suppurative gastritis consistent with PhG.

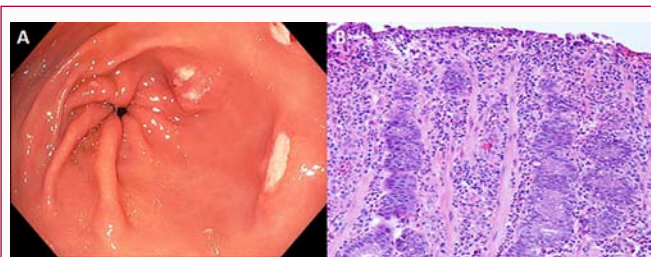


Figure 4: Antral superficial ulceration and erosions were present on EGD (A). Histology consistent with severe and active chronic gastritis (B).

epigastric pain. An EGD demonstrated superficial ulceration and erosions in the gastric antrum (Figure 4A); histologic review of the corresponding biopsies showed severe active chronic gastritis with negative *Helicobacter pylori* immunostaining (Figure 4B). The patient was initially treated with high dose pantoprazole but due to refractory abdominal pain, an EGD was repeated and demonstrated persistent chronic gastritis and antral ulceration with consistent histologic findings for PhG. The patient was then initiated on a 10 day course of levofloxacin and metronidazole. A repeat EGD 1 month after antibiotics showed resolving gastric ulcers/erosions but histologic persistence of severe active chronic gastritis. Due to slow resolution, the patient is presently completing a 3 month course of azithromycin for persistent PhG.

Discussion

In our series, three patients were immunosuppressed at the time of PhG diagnosis while gastric stasis was also a likely factor in three cases. Our patients developed gastric stasis due to gastric outlet obstruction and gastroparesis. To our knowledge, there has only been one case report detailing an association between PhG related and gastric outlet obstruction [8]. Gastroparesis has not been previously associated with PhG. Interestingly, two patients also had an underlying diagnosis of inflammatory bowel disease (IBD) and we are unaware of prior reports detailing an association between these conditions.

Antibiotics are presently the mainstay therapy for PhG. In one review of 77 PhG cases, successful management entailed medical therapy, including antibiotics, in 52 cases (67.5%), surgical resection in 21 cases (27.3%), and surgical drainage in 3 cases (3.9%) [1]. One report has suggested that PhG-related mortality was highest when *Streptococcus* species are the etiology of infection (53%) [7]. While not performed in our case series, we recommend endoscopy-facilitated bacterial cultures to guide antibiotic selection.

Conclusion

In summary, our series corroborates a diverse range of clinical and radiologic features seen among patients with PhG — underscoring the challenge of establishing this clinical diagnosis. Gastric stasis, in addition to immunosuppression, was a common risk factor in our series which may foster further insight to understanding the pathophysiology of PhG. IBD may also be associated with PhG, although our case series was limited by the small sample size.

Author Involvement with Manuscript

Xin Zhang and Bradley Anderson – Design, acquisition of data, and drafting of manuscript; Thomas Smyrk and Sunanda Kane – Drafting of manuscript and critical revision of the manuscript for important intellectual content.

References

1. Nomura K, Iizuka T, Yamashita S, Kuribayashi Y, Toba T, Yamada A, et al. Phlegmonous gastritis secondary to superior mesenteric artery syndrome. *SAGE Open Med Case Rep.* 2015;3:2050313X15596651.
2. Kato K, Tominaga K, Sugimori S, Nagami Y, Kamata N, Yamagami H. Successful treatment of early-diagnosed primary phlegmonous gastritis. *Intern Med.* 2015;54:2863-6.
3. Kim GY, Ward J, Hennessey B, Peji J, Godell C, Desta H. Phlegmonous gastritis: case report and review. *Gastrointest Endosc.* 2005;61:168-74.
4. Min SY, Kim YH, Park WS. Acute phlegmonous gastritis complicated by delayed perforation. *World J Gastroenterol.* 2014;20:3383-7.
5. Sahnan K, Davis BJ, Bagenal J, Cullen S, Appleton S. Acute gastric necrosis after routine oesophagogastrroduodenoscopy with therapeutic argon plasma coagulation. *Ann R Coll Surg Engl.* 2013;95:e99-101.
6. Kim KH, Kim CG, Kim YW, Moon H, Choi JE, Cho SJ. Phlegmonous gastritis with early gastric cancer. *J Gastric Cancer.* 2016;16:195-9.
7. Morimoto M, Tamura S, Hayakawa T, Yamanishi H, Nakamoto C, Nakamoto H, et al. Phlegmonous gastritis associated with group A streptococcal toxic shock syndrome. *Intern Med.* 2014;53:2639-42.
8. Park CW, Kim A, Cha SW, Jung SH, Yang HW, Lee YJ, et al. A case of phlegmonous gastritis associated with marked gastric distension. *Gut Liver.* 2010;4:415-8.