

WHEN GASTRIC INFLAMMATION TURNS DEADLY : INSIGHTS INTO ACUTE PHLEGMONOUS GASTRITIS - A CASE REPORT

Abstract

Acute phlegmonous gastritis is a rare and life-threatening bacterial infection of the stomach wall, characterized by rapidly progressing inflammation. Early diagnosis is crucial to prevent complications such as peritonitis, gastric necrosis or sepsis but the condition often presents with nonspecific symptoms like severe epigastric pain, fever, nausea and vomiting. Radiology plays a pivotal role in the diagnosis. We present the case of a 54 year old female who is a known case of type 2 diabetes mellitus, acid peptic disease on proton pump inhibitors admitted with acute abdominal pain and multiple episodes of vomiting.

Given the rarity (historically fewer than 100 cases have been documented) of acute phlegmonous gastritis, understanding its radiological features in the right clinical setting is essential for early identification and to impart timely treatment to improve outcomes.

Keywords: Acute phlegmonous gastritis, Streptococcus infection, gastric wall necrosis, peritonitis, sepsis.

Introduction

Acute phlegmonous gastritis (APG), also called phlegmonitis of the stomach, is a rare, life-threatening bacterial infection involving the submucosa and muscularis propria of the stomach. Patients typically present with nonspecific symptoms such as abdominal pain, fever, nausea, and vomiting. Streptococcus species are the most common pathogens.

The mortality rate remains high, up to 42%, largely due to diagnostic delays.

Case report

A 54-year-old female with a history of type 2 diabetes mellitus, hypothyroidism, and acid peptic disease on proton pump inhibitors presented to the emergency department with acute abdominal pain and multiple episodes of vomiting. She had reported heartburn and intermittent vomiting for

the past three days and underwent esophago-gastro-duodenoscopy (EGD) two days earlier, which revealed only mild antral gastritis.

On arrival, she was delirious. Vitals: temperature 98.6°F, heart rate 100 bpm, respiratory rate 18/min, blood pressure 70/50 mmHg on noradrenaline, and oxygen saturation 99% on room air. She had abdominal distension, diffuse tenderness with guarding, and sluggish bowel sounds.

Initial investigations revealed: WBC 11,800/mm³ (93% neutrophils), hemoglobin 10.3 g/dL, platelets 2 lakh/mm³, SGOT 331 IU/L, SGPT 151 IU/L, total bilirubin 3.1 mg/dL, creatinine 1.6 mg/dL, CRP 184.4 mg/L, and PT 18.3 seconds. Blood cultures grew beta-hemolytic streptococci.

Contrast-enhanced CT of the abdomen showed gastric luminal dilatation with diffuse wall thickening and submucosal hypodensity (edema). The fundus showed preserved mucosal enhancement, while the distal body and antrum had mucosal non-enhancement—findings consistent with APG and early ischemia.

Management and Outcome:

She was managed in the intensive care unit with intravenous fluids, vasopressors (noradrenaline and vasopressin), broad-spectrum antibiotics, nil per oral (NPO) status, nasogastric decompression, and hourly monitoring.

Following multidisciplinary team discussion, an emergency exploratory laparotomy was performed. Approximately 500 mL of seropurulent fluid was found in the supracolic compartment, with exudates tracking into the pelvis. The distal stomach wall was thickened, with the lesser omentum adherent to the anterior antrum. No perforation was observed.

Histopathology revealed fibrocollagenous tissue with extensive fibrin deposition and dense neutrophilic infiltration. Peritoneal cytology was negative for malignant cells. Pus culture showed no growth.

Postoperatively, the patient remained ventilated and on vasopressors. Echocardiogram showed no regional wall motion abnormalities. Two days later, she developed new-onset atrial fibrillation and hypotension. After cardioversion (150 J), she briefly returned to sinus rhythm but rapidly deteriorated into bradycardia and cardiac arrest. Resuscitation efforts were unsuccessful, and the patient expired.

Discussions

Acute phlegmonous gastritis is a rare and life threatening disease involving acute infection of stomach arising from pyogenic bacteria and characterized by inflammatory process over the entire gastric mucosa, submucosa and muscularis propria.

It can affect any gastrointestinal site, but the stomach is most frequently involved. The pathogenesis of phlegmonous gastritis is unclear. Predisposing factors such as diabetes mellitus, immune suppression, gastric ulcers, excessive proton pump inhibitor use, iatrogenic: gastric surgery, invasive procedures such as upper gastrointestinal endoscopy and nasogastric tube insertion have been reported.

In our case, diabetes mellitus, proton pump inhibitor use and history of recent Esophago-Gastro-Duodenoscopy could be the predisposing factors.

Typical clinical manifestations of APG include acute abdominal pain, fever, chills, nausea, and vomiting. Hematemesis and/or purulent emesis may occur in severe cases. Owing to the rare nature of acute phlegmonous gastritis (APG) and its non-specific clinical features, early diagnosis can be difficult.

Streptococcus species have been found to be the most common causative organisms, accounting for about two-third of cases. This condition can develop and progress quickly, often with resulting gastric wall necrosis, perforation, peritonitis and septicemia. The extremely high mortality rate associated with APG may be due in part to the fact that the diagnosis is often delayed or missed.

APG therapy requires aggressive supportive care with powerful antimicrobial agents. For patients in whom antibiotic therapy is ineffective, surgical intervention/ partial or total gastrectomy is required.

Conclusion

APG is a rare condition associated with high morbidity and mortality rates. Although this condition most commonly affects patients with underlying chronic conditions, it can also affect healthy individuals.

Early diagnosis, treatment with broad spectrum antibiotics and adequate surgical intervention will likely provide the best outcome. Given the fast progression of this disease, early recognition and immediate action are crucial to achieve positive outcomes.

This case highlights the radiological findings in acute phlegmonous gastritis, focusing on the utility of various imaging modalities.

Teaching points

- 103
- 104 1) **Early Imaging is Crucial:** CT is essential in diagnosing APG, as clinical signs are often
105 nonspecific.
- 106 2) **Recognize Risk Factors:** Diabetes, PPI use, and recent endoscopic procedures increase
107 susceptibility.
- 108 3) **Multidisciplinary Management is Key:** Timely involvement of surgery, radiology, and
109 critical care can influence outcomes.
- 110 4) **Prognosis is Poor Without Early Intervention:** Mortality remains high, even with
111 aggressive treatment.

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113 **Acknowledgment**

114

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116 collaboration. We are also grateful to the patient's family for their support during this
117 challenging case.

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119 **References**

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131 **MCQs:**

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- 133 1. What is the most common causative organism in acute phlegmonous gastritis (APG)?
- 134 A) Escherichia coli
- 135 B) Streptococcus species

C) *Helicobacter pylori*

D) *Staphylococcus aureus*

Answer: B) *Streptococcus* species

2. Which imaging modality is most useful for diagnosing acute phlegmonous gastritis?

A) Ultrasound

B) Plain X-ray

C) Contrast-enhanced CT (CECT)

D) MRI

Answer: C) Contrast-enhanced CT (CECT)

3. Which of the following is NOT a known predisposing factor for APG?

A) Diabetes mellitus

B) Proton pump inhibitor use

C) Recent upper GI endoscopy

D) High-fiber diet

Answer: D) High-fiber diet

Figure legends:

Figure 1. Axial high-resolution computed tomography (CT) images of the abdomen taken on 22 August 2024.

(a) Gastric luminal dilatation with diffuse wall thickening, marked submucosal hypodensity (edema), and mucosal non-enhancement in the distal body and antrum (green arrow).

(b) Normal mucosal enhancement in the fundus (red arrow) without associated wall thickening.

Figure 2.(a) Coronal high-resolution contrast-enhanced CT image shows gastric luminal dilatation with diffuse wall thickening (outlined by yellow line), marked submucosal hypodensity (edema), and mucosal non-enhancement in the distal body and antrum. **(b)** Intraoperative photograph demonstrating a grossly thickened stomach wall at the antrum.

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