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Case report

# Phlegmonous Gastritis in a Cirrhotic Patient with a Gastrointestinal Stromal Tumor

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## Abstract

Phlegmonous gastritis is a rare disease with high mortality, developing from a pyogenic infection originating from gastric cancer or ulcers. Identified risk factors include immunosuppressive therapy and endoscopic procedures. Early diagnosis and intensive medical treatment are crucial, although emergency surgery may be required in some cases. We present the case of a 63-year-old woman with a history of liver cirrhosis and a gastrointestinal stromal tumor (GIST), who was admitted to the emergency department with secondary bacterial peritonitis following phlegmonous gastritis. In the subsequent days, the patient experienced a poor clinical course and a fatal outcome.

## Keywords

Gastritis, liver cirrhosis, peritonitis, gastrointestinal bleeding.

## INTRODUCTION

Phlegmonous gastritis is a rare and often fatal form of acute gastritis, with a mortality rate approaching 50%. It is characterized by a pyogenic infection of the gastric submucosa and muscularis propria, typically sparing the mucosal layer[1–5].

This condition is most commonly associated with *Streptococcus pyogenes*, though other microorganisms such as *Staphylococcus spp., Klebsiella pneumoniae, Escherichia coli, Haemophilus influenzae, Proteus,* and *Clostridium spp.* have also been implicated[4,6]. Clinical presentation is typically non-specific and includes abdominal pain, fever, nausea, vomiting, and signs of systemic infection[3].

We present the case of a 63-year-old woman with hepatic cirrhosis and a gastric gastrointestinal stromal tumor (GIST) who developed secondary bacterial peritonitis associated with phlegmonous gastritis, with *S. pyogenes* isolated in ascitic fluid and blood cultures.

## CASE REPORT

The patient was a 63-year-old woman with hepatic cirrhosis due to metabolic dysfunction-associated steatotic liver disease (MASLD), previously classified as Child-Pugh B, without any history of acute decompensation. She presented to the emergency department with severe abdominal pain, rated at 9/10 in intensity. An upper digestive endoscopy performed three weeks earlier (**Figure 1**) revealed three medium-sized esophageal varices and a 23 x 26 mm ulcerated subepithelial lesion located in the gastric fundus.

Endoscopic ultrasound identified endosonographic features consistent with a gastric GIST (**Figure 2**). Fourteen days prior to admission, she experienced abdominal pain and ascites, followed by episodes of fever and disorientation five days before her arrival at the hospital.

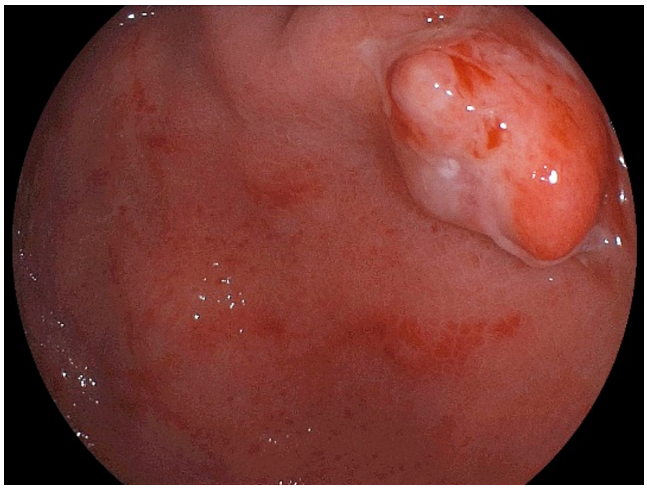

**Figure 1.** Upper digestive endoscopy showing an ulcerated gastric subepithelial lesion. Source: Gastroenterology Department, Hospital Cayetano Heredia.

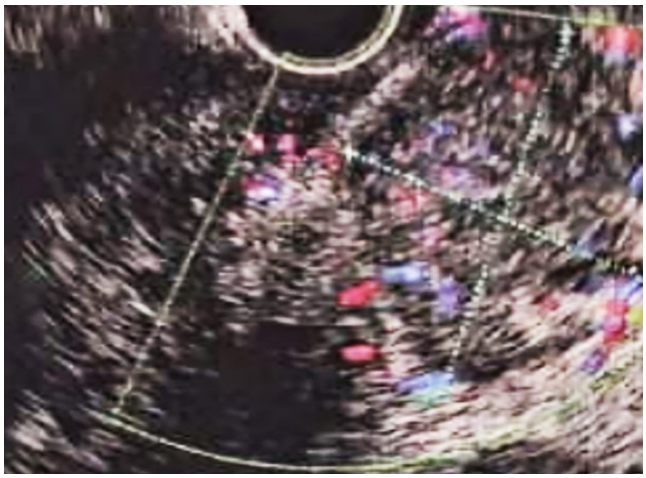

**Figure 2.** Endoscopic ultrasound showing a heterogeneous lesion in the fourth echolayer, measuring 23 x 26 mm, consistent with a GIST. Source: Gastroenterology Department, Hospital Cayetano Heredia.

On admission, the patient presented with tachycardia, normal blood pressure, and jaundice. Abdominal evaluation revealed shifting dullness and tenderness in the epigastric and mesogastric regions. Laboratory tests showed leukocytosis (14.61 cells/mm$^3$) with a left shift, thrombocytopenia, mildly elevated transaminases, elevated C-reactive protein (192 mg/L), and severe hypoalbuminemia (2.3 g/dL). Analysis of the ascitic fluid met Runyon's criteria[7], with protein levels of 2 g/dL, glucose at 47 mg/dL, and lactate dehydrogenase levels exceeding the upper limit of serum reference values, confirming a diagnosis of secondary bacterial peritonitis. Treatment with meropenem was initiated.

An abdominal computed tomography (CT) scan (**Figure 3**) revealed thickening of the gastric wall up to 13 mm, with peripheral mucosal and serosal enhancement and heterogeneous content within the thickened wall, without evidence of pneumatosis. A few days later, *S. pyogenes* was isolated in both the ascitic fluid and blood cultures. The final diagnosis was phlegmonous gastritis secondary to an ulcerated and infected gastric GIST. Broad-spectrum antibiotic therapy was continued; however, the patient's clinical course worsened, and she died five days after admission.

## DISCUSSION

Phlegmonous gastritis is a form of acute gastritis caused by the invasion of the gastric wall by pyogenic organisms[8]. The diffuse type is more frequently reported than the localized (antral) type, the latter having a mortality rate of up to 54%[4,9–11].

It is essential to distinguish among three closely related nosological entities: gastric emphysema, phlegmonous gastritis, and emphysematous gastritis. Gastric emphysema refers to the presence of gas within the gastric wall following mucosal disruption and typically has a favorable prognosis[12], such as after endoscopic therapy. Phlegmonous gastritis involves the invasion of the gastric wall by pyogenic organisms, without the presence of gas in the gastric wall. In contrast, emphysematous gastritis is a rare, severe form of phlegmonous gastritis, characterized by suppurative inflammation of the gastric wall with gas present[8,10], and it frequently progresses to perforation[13]. Primary cases are often associated with an ulcer or neoplastic lesion, as in our case. However, secondary cases can arise from systemic infections, and in some instances, no identifiable origin is found (idiopathic cases)[14].

There are multiple risk factors, including conditions associated with immunosuppressive states (viral infections, diabetes *mellitus*, autoimmune diseases, chronic kidney disease, hepatic cirrhosis, leukemia, alcoholism, chemotherapy-induced neutropenia, malnutrition, chronic corticosteroid use) and those directly related to the stomach (mucosal injury, cancer, therapeutic endoscopic procedures). Our patient presented with Child-Pugh C cirrhosis during this hospitalization (previously Child-Pugh B) and an ulcerated gastric GIST larger than 2 cm, as well as a recent endoscopic ultrasound performed in the preceding days[3,5].

The clinical presentation is non-specific but often involves sepsis, upper abdominal pain, peritonitis, and purulent

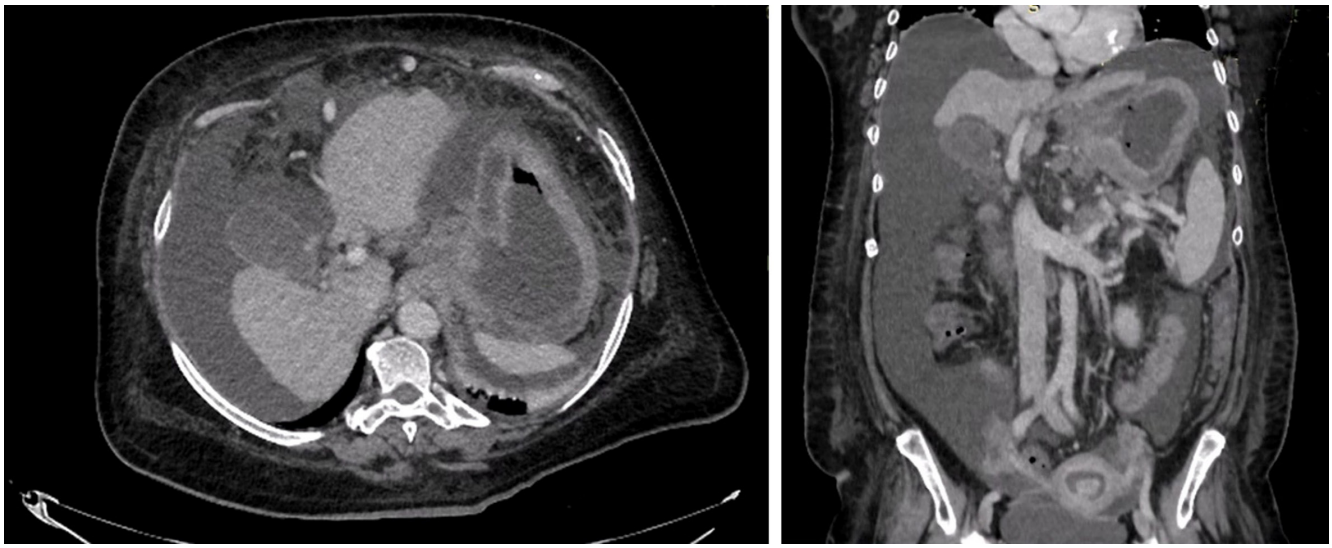

**Figure 3.** Abdominal computed tomography showing gastric wall thickening with peripheral mural enhancement. Source: Gastroenterology Department, Hospital Cayetano Heredia.

ascites[1]. Other manifestations include nausea, vomiting, and hematemesis. Since it poses a diagnostic challenge, computed tomography (CT) is a valuable tool, with key findings including gastric wall thickening and abscesses[4,15,16].

Upper gastrointestinal endoscopy may reveal mucosal erythema and edema, while endoscopic ultrasound is the most effective diagnostic test, as it allows for the identification of submucosal edema and thickening. Rapid initiation of antibiotic therapy after diagnosis—or even in cases of high clinical suspicion—is recommended, as delayed treatment is associated with a high mortality rate, approaching 92%[6,14].

The frequency of ascites or secondary bacterial peritonitis in phlegmonous gastritis is unknown. Severe cases may have unfavorable outcomes due to perforation[17]. In such situations, urgent surgical intervention is necessary, and studies have reported a significantly lower mortality rate following surgery[14,18].

Our patient did not initially undergo surgical treatment due to the absence of signs of perforation and the potential postoperative complications in a patient with Child-Pugh C cirrhosis. Subsequently, as her clinical condition deteriorated, her family declined surgical intervention. In such cases, we believe that treatment decisions should be individualized for each patient.

## CONCLUSIONS

Phlegmonous gastritis is a rare condition with a high mortality rate, necessitating a high index of diagnostic suspicion. Computed tomography and endoscopic ultrasound are crucial for early diagnosis. Early antibiotic therapy is associated with a better prognosis. Surgical management should be considered in cases of poor clinical evolution, diffuse forms of the disease, or perforation.

### Conflict of Interest

The authors declare no conflicts of interest.


### Funding Sources

The authors declare that no external funding sources were used.


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