

Combination of antibiotics, gastric lavage and nasojejunal feeding—an effective alternative for the management of acute phlegmonous gastritis: a case report

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Abstract

Background: Phlegmonous gastritis is a rare bacterial infection of the gastric wall, characterised by purulent inflammation of the gastric mucosa, submucosa and muscularis layers. Phlegmonous gastritis has a high mortality rate, even with correct diagnosis and antimicrobial therapy.

Case presentation: A 22-year-old man presented for acute epigastric pain associated with aqueous diarrhoea, vomiting and sustained fever. Abdominal computed tomography showed diffuse oedema and thickened gastric wall, increased number and size of abdominal lymph nodes and the absence of pneumoperitoneum. Fibregastroscopy revealed oedematous, ridged and thickened gastric mucosa with abundant purulent secretion, especially in the antrum, consistent with phlegmonous gastritis, which was confirmed by histological evaluation of gastric biopsies. Cultures of the tissue biopsies and purulent secretion were positive for *Enterococcus cecorum*. He was treated with sensitive antibiotics according to the antibiogram, and importantly, with continuous gastric lavage and individualised nutritional support therapy. He eventually recovered well and was discharged with no abdominal symptoms.

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Conclusions: Our case indicates that early diagnosis and immediate treatment are crucial to achieve positive outcomes. The combination of sensitive antibiotics, gastric lavage and early enteral nutrition via nasojejunal feeding might be an effective alternative for the comprehensive treatment of acute phlegmonous gastritis.

Keywords

Phlegmonous gastritis, gastric lavage, antibiotics, nasojejunal feeding, histopathology, nutritional support

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Introduction

Phlegmonous gastritis is an uncommon condition with a high mortality rate, even if treated timely. It is a rare acute bacterial infection, characterised by purulent inflammation of the gastric wall involving the mucosa, submucosa and muscularis layers.¹ Phlegmonous gastritis can rapidly become fatal if untreated; therefore, prompt diagnosis and management are required. The mortality rate remains high (27%–40%), even with a correct diagnosis and antimicrobial therapy.² *Streptococcus pyogenes* is the most common causative organism, although many other microorganisms have been identified. The aetiology of phlegmonous gastritis is largely unknown. Immunosuppression and undergoing invasive gastric procedures have been considered risk factors, but many patients do not present with known risk factors.³ We report a case of phlegmonous gastritis that occurred after ingesting contaminated food and which was cured by a combination of antibiotics, gastric lavage and nasojejunal feeding.

Case report

A 22-year-old man consulted our emergency department for acute epigastric pain that

began after eating contaminated food (Chinese hot pot), with a duration of 2 days with a 3-hour evolution. The patient had experienced chronic gastritis, without regular treatment. The abdominal pain was associated with aqueous diarrhoea, vomiting and an episode of haematemesis, accompanied by a sustained fever > 39°C (armpit). On admission, the patient presented with general deterioration, weakness, pallor and profuse sweating. Vital signs on admission were as follows: temperature: 39.5°C, blood pressure (BP): 106/65 mmHg, heart rate (HR): 113/minute and respiratory rate (RR): 20/minute. His abdomen was soft but painful with pressure applied to the epigastric area, where he also showed signs of peritoneal irritation. Blood test results indicated impaired kidney function (creatinine level: 168.0 µmol/L), leucocytosis with left neutrophil shift (leucocytes: 9.51×10^9 /L with 82% neutrophils), C-reactive protein: 106.30 mg/L, procalcitonin: 15.3 pmol/L, interleukin-6: 27.020 pg/mL and mild metabolic acidosis (pH: 7.32, pCO₂: 32 mmHg, HCO₃⁻ 20 mmol/L and base excess: 3 mmol/L). Electrocardiography revealed sinus tachycardia; abdominal and thoracic X-rays showed no abnormalities. The patient received fluid resuscitation and antipyretic therapy (ibuprofen). Abdominal

computed tomography (CT) showed diffuse oedema and thickening of the gastric wall, increased number and size of the abdominal lymph nodes and absence of pneumoperitoneum (Figure 1). The cause of the abdominal pain was unclear; therefore, he was admitted to the department of gastrointestinal surgery for further treatment.

Empirical antibiotic treatment with cefminox and levofloxacin was administered



Figure 1. Abdominal computed tomography (CT) showing diffuse oedema and thickening of the gastric wall.

considering the possibility of a serious bacterial infection. However, the patient's epigastric pain and fever persisted. We then performed fibregastroscopy out on the fifth day after the onset of symptoms, and the results showed oedematous, ridged and thickened gastric mucosa with abundant purulent secretion, especially in the antrum, consistent with phlegmonous gastritis (Figure 2). Histological evaluation of gastric biopsies showed acute inflammation of the submucosa, with exudation and necrosis, which confirmed the diagnosis of phlegmonous gastritis (Figure 3). Bacterial cultures of the tissue biopsies and purulent secretion were positive for *Enterococcus faecium*. The antibiotic treatment was modified according to the antibiogram, introducing cefoperazone and tazobactam, despite negative haemoculture.

In addition to optimising the antibiotics, our patient received gastrointestinal decompression and individualised nutritional support therapy. According to the fibregastroscopy findings, continuous gastric lavage was performed to remove the

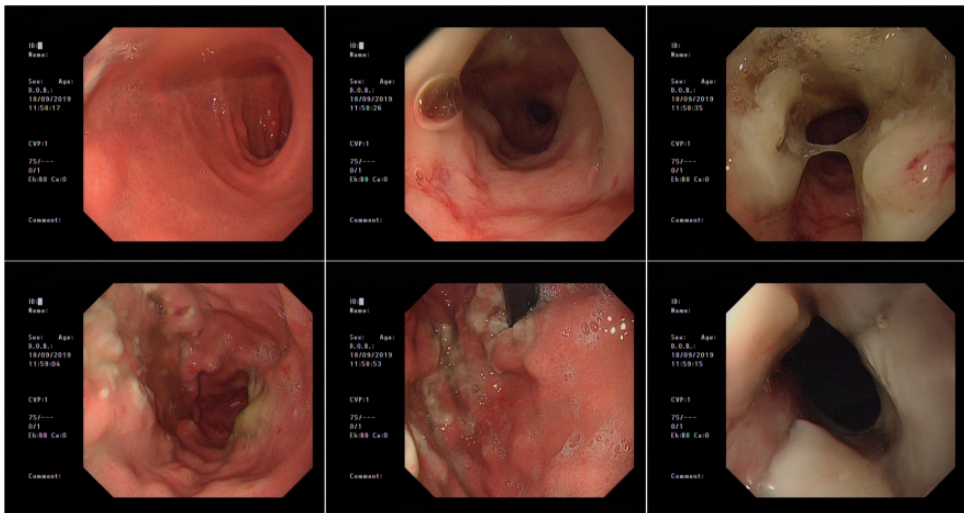


Figure 2. Fibregastroscopy showing oedematous, ridged and thickened gastric mucosa, with a large amount of purulent secretion in the stomach.

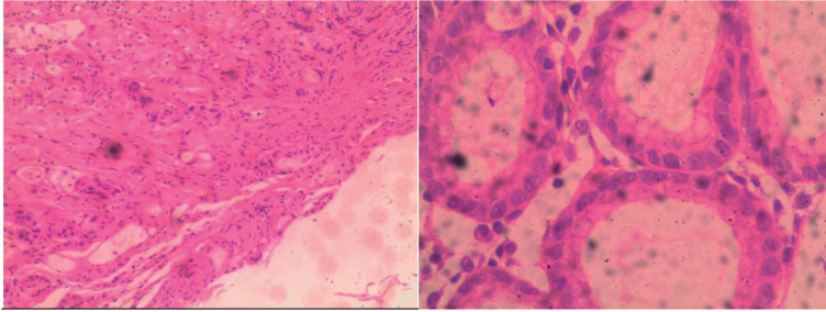


Figure 3. Histology (haematoxylin & eosin (H&E) staining $\times 40$) of the gastric tissue biopsies showing acute inflammation in the submucosa, with exudation and necrosis.

purulent secretion and proinflammatory mediators adherent to the mucosa. Specifically, 250 mL of 37°C to 40°C normal saline was slowly injected through a gastric tube, and then aspirated with gastrointestinal decompression. Gastric lavage was repeated every 6 hours, 4 times per day, until the returned gastric juice was clear. Meanwhile, the patient was supported with total parenteral nutrition (TPN). With this treatment, his epigastric symptoms improved, and infection was controlled, as evidenced by body temperature normalisation, and leucocytosis and neutrophil down-regulation. However, after 1 week of continuous lavage and TPN, the patient presented with abnormal liver function, indicated by increased alanine aminotransferase (ALT) and aspartate transferase (AST) concentrations, which was interpreted as parenteral nutrition-associated liver disease (PNALD). Oral feeding was not yet recommended as the stomach contents were still slightly purulent. To avoid long-term TPN and its complications, we placed a nasojejunal tube under radiological guidance, to provide nutritional support. Therefore, he received partial enteral feeding plus supplementary parenteral nutrition and was gradually transferred to total enteral nutrition.

The patient improved gradually, and he was discharged with no abdominal

symptoms after 24 days of hospitalisation. Leucocytosis, neutrophil count and liver and kidney function normalised. Effective treatment of the phlegmonous gastritis was confirmed by repeat fibregastroscopy before discharge. The patient was very satisfied with the treatment he received throughout the duration of his hospital stay. After discharge, gradually introducing a normal diet and gastric ulcer treatment were strongly recommended. We also recommended that the patient undergo gastroscopy and biopsy examination 3 months after discharge, which was performed. To our great relief, fibregastroscopy showed healing of the gastric antral ulcer (Figure 4), and biopsy showed chronic non-atrophic inflammation of the submucosa, without intestinal metaplasia (Figure 5). Additionally, the patient regained normal gastric function, without complaints of abdominal pain, acid regurgitation or belching.

Discussion

Phlegmonous gastritis is a rare entity first mentioned in 1862 by Cruveilhier; less than 500 cases have been reported in the literature.⁴ The associated pathology involves purulent gastric wall inflammation caused by bacterial infection. According to

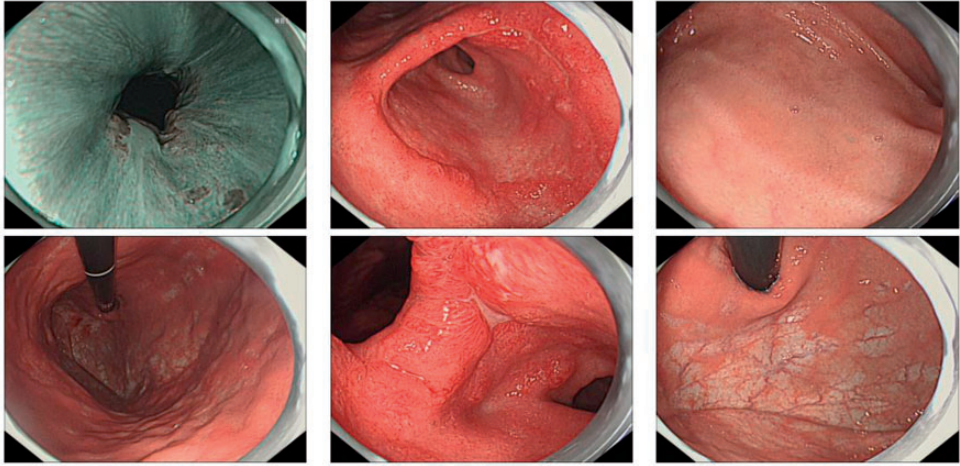


Figure 4. Fibregastroscopy 3 months after discharge showing healing of the gastric antral ulcer.

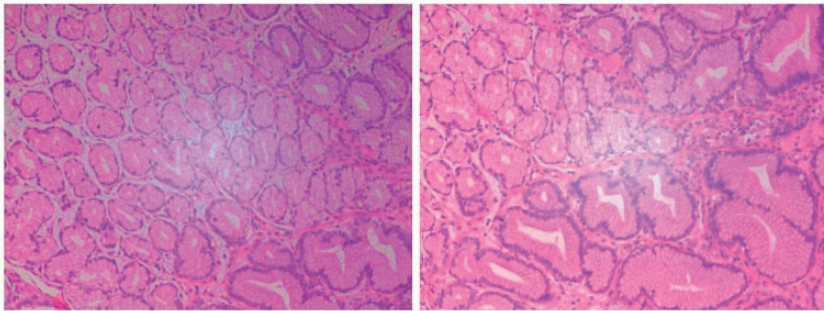


Figure 5. Histology (haematoxylin & eosin (H&E) staining $\times 40$) of gastric tissue biopsies 3 months after discharge showing chronic non-atrophic inflammation of the submucosa, without intestinal metaplasia.

previous reports, *Streptococcus pyogenes* was isolated in approximately 70% of cases; however, other microorganisms have also been identified as aetiologic agents, such as *Staphylococcus*, *Pneumococcus* and *Enterococcus*, and approximately 17% of cases were attributed to polymicrobial infection.⁵ Of particular concern, two previous case studies reported *Enterococcus* as the pathogenic bacteria in the gastric wall; *E. fecalis* and *E. faecium*, respectively.^{3,6} However, there are few reported cases similar to our isolated case caused by *E. cecorum*.

Bacterial invasion of the gastric wall can be caused by chronic gastritis, gastric ulcer, invasive diagnostic or therapeutic interventions involving the gastric mucosa and respiratory tract infection or other infection, especially with other risk factors.⁷ The cases we reviewed revealed that diabetes mellitus, human immunodeficiency virus (HIV) infection, long-term alcohol consumption and cancer were common risk factors for phlegmonous gastritis.^{8–11} The main reason that these risk factors are associated with phlegmonous gastritis is that patients with these conditions are usually

immunocompromised, which increases the likelihood of becoming infected by opportunistic pathogens. Our patient was a young man with no obvious risk factors; however, he had experienced chronic gastritis, which led to acute epigastric pain after ingesting contaminated food. Considering that *Enterococcus* is a gut commensal bacteria in humans that may become pathogenic and lead to septicemia under some circumstances,^{12,13} we presumed that the reason for our case might be that foods contaminated by *E. cecorum* entered the stomach, colonised the gastric wall and led to infection.

Phlegmonous gastritis symptoms develop rapidly. Abdominal pain, which begins in the epigastric region, sometimes generalises throughout the abdomen, commonly leading to nausea, vomiting, fever, diarrhoea and haematemesis.¹⁴⁻¹⁶ With its non-specific signs and symptoms, a high index of suspicion is required to diagnose phlegmonous gastritis, and it should be considered as a differential diagnosis in patients with acute abdomen. The differential diagnoses in our case were acute appendicitis, acute mesenteric lymphadenitis, peptic ulcer, lymphoma and gastric cancer. Abdominal CT, fibregastroscopy and histology were valuable in establishing the diagnosis. Additionally, culture of the biopsies provided pathogenic evidence for selecting the optimal antibiotic treatment.

Phlegmonous gastritis treatment has developed rapidly in recent decades, with advances in diagnostic techniques.¹⁷ Early diagnosis and prompt administration of antibiotics reduces patient mortality and prevents surgical intervention.¹⁷ Rapid progress in CT and fibregastroscopy has enhanced timely phlegmonous gastritis diagnosis, and parenteral antibiotics have become more common as the single treatment.¹⁸ However, approximately 40% of patients still required surgical treatment, in one study, such as surgical drainage or gastric resection, when antibiotics alone

failed to control the disease.⁵ Noteworthy, our patient was effectively treated using a combination of antibiotics, gastric lavage and nutritional support. First, fibregastroscopy showed a large amount of purulent secretion in the stomach adhering to the mucosal surface. For this reason, continuous gastric flushing with normal saline was recommended instead of surgical drainage. Second, although the stomach was not functioning normally, the gut remained functional. Therefore, to alleviate PNALD and to protect gut barrier function, nasojejunal feeding was initiated to achieve enteral nutritional support and prevent long-term TPN complications. As a result, the 24-day hospital stay in the present case was relatively shorter than that in most previous cases, with a range of 28 to 40 days.^{5,7,8} However, compared with parenteral nutrition, whether early nasojejunal feeding improves the disease prognosis requires further clinical studies.

In conclusion, phlegmonous gastritis is a rare entity that must be considered in the differential diagnoses of acute abdomen. Abdominal CT and endoscopy are essential for the diagnosis. *S. pyogenes* is most frequently isolated, but other microorganisms have also been identified as causal agents. The pathogenesis of *Enterococcus*-related gastritis deserves more attention and requires further study. The combination of antibiotics (according to sensitivity data), gastric lavage and early enteral nutrition via nasojejunal feeding might be an effective alternative for the comprehensive treatment of acute phlegmonous gastritis.

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Author contributions

JW, MW, and MX were responsible for managing the patient. TZ was the patient's supervising nurse. XZ and HH contributed to the collection

and analysis of the data. JW drafted the manuscript. All authors have critically revised the manuscript and agreed to be fully accountable for the integrity and accuracy of the work. All authors have read and approved the final manuscript.

Ethics statement

This case report was approved by the Ethical Committee of the Affiliated Hospital of Zunyi Medical University, China, in January, 2019.

Informed consent

Written informed consent was obtained from the patient to undergo the procedures in this report and to publish this report. Our case report complied with the CARE guidelines (<https://www.equator-network.org/reporting-guidelines/care>).

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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