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AN UNDER-RECOGNISED RAPIDLY FATAL CONDITION: PHLEGMONOUS GASTRITIS

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ABSTRACT

Phlegmonous gastritis is a rare condition, characterised by an infectious process in the gastric wall. There is an association with haemolytic *Streptococcus* infection in approximately 70% of cases, less frequently with other infectious agents such as *Staphylococcus aureus*, *Pneumococcus* and *Enterococcus*. Diagnosis is often delayed due to non-specific symptoms, such as abdominal pain, nausea, vomiting and fever. Abdominal computed tomography (CT) typically reveals thickening of the gastric wall, low-intensity areas within the gastric wall and gas accumulation. The therapeutic approach includes broad-spectrum antibiotics, and surgical resection of the gastric area involved in complicated cases.

The authors present the clinical case of a 58-year-old male admitted to the hospital with headache, vomiting, abdominal pain and fever. Laboratory evaluation showed increased inflammatory parameters. An abdominal CT scan showed markedly diffuse parietal thickening of the stomach with increased mucosal enhancement and densification of perigastric fat. There was rapid progression to septic shock and the patient was admitted to the intensive care unit. An urgent upper gastrointestinal endoscopy revealed diffuse oedema of the gastric mucosal with no signs of tumour infiltration, confirming the diagnosis of phlegmonous gastritis. Broad-spectrum antibiotic therapy was started, and the patient underwent an urgent total gastrectomy. Despite the interventions carried out, refractory shock with multi-organ dysfunction occurred, resulting in death. Histopathologic findings in the gastrectomy specimen were compatible with phlegmonous gastritis.

The clinical case presented demonstrates the need for high clinical suspicion for an early diagnosis of phlegmonous gastritis, especially in patients with gastrointestinal symptoms and clinical severity, for early treatment and improvement of the prognosis.

KEYWORDS

Phlegmonous gastritis, haemolytic Streptococcus, septic shock, gastrectomy

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LEARNING POINTS

- Phlegmonous gastritis is a rare and deadly infectious disease of the gastric wall, mainly occurring in the submucosa of the stomach.
- Important diagnostic tools include CT imaging and gastroscopy. However, endoscopic findings may be varied and non-specific, making early diagnosis difficult.
- Conservative treatment using antibiotics may not work and urgent surgery may be needed.

INTRODUCTION

Phlegmonous gastritis (PG) is a rare and potentially fatal infectious disease of the gastric wall, affecting primarily the submucosa of the stomach[1,2]. Since 1862, when it was first described, fewer than 1,000 cases have been reported in the literature. The mortality rate nowadays is approximately 50%. PG is more frequent among men, between 30 and 70 years old. The exact pathophysiology of the disease is poorly understood; there is a possible association between PG and prior history of gastritis, gastric malignancy or achlorhydria, previous gastric invasive procedures, alcoholism and immunosuppression. Haemolytic *Streptococcus* is the causative agent in approximately 70% of cases, followed by *Staphylococcus aureus*, *Pneumococcus* and *Enterococcus*[2,3]. Clinical manifestations of PG are non-specific, as patients often present with abdominal pain, nausea, vomiting and fever. This frequently leads to delays in diagnosis, with rapid progression of the disease, ultimately ending up in necrosis of the gastric wall, peritonitis, septic shock and death[1]. Due to its rarity, standardised treatment protocols for the management of this condition have not been established yet, therefore treatment decisions are difficult and might involve total gastrectomy.

We report a case of PG that rapidly progressed to septic shock with multi-organ failure and death, despite antibiotic treatment and total gastrectomy.

CASE DESCRIPTION

A 58-year-old Caucasian male was admitted to the emergency department with persistent vomiting, diarrhoea and abdominal pain, lasting around twelve hours. No history of relevant previous illness was reported, except obesity and hypertension. On admission he was febrile, diaphoretic, hypertensive and tachycardic, with polypnea and desaturation, and diffuse abdominal pain. The laboratory evaluation showed a severe increase in inflammation parameters and acute kidney injury, and arterial blood gas analysis showed hypoxemia and hyperlacticaemia. A chest radiograph revealed a reticular bilateral pattern suggestive of pulmonary congestion. A thoracic, abdominal and pelvic computed tomography (CT) scan with intravenous contrast was performed *(Fig. 1)* and revealed markedly diffuse parietal thickening of the stomach with increased mucosal enhancement and densification of perigastric fat, especially across the greater curvature. There was also low enhancement of the submucosal and muscular layers, with permeability of gastric, short gastric and gastroepiploic arteries. Blood cultures were collected, and the patient was started on intravenous piperacillin-tazobactam but developed septic shock and was admitted to the intensive care unit. An urgent upper gastrointestinal endoscopy revealed gastric mucosal diffuse oedema with no signs of tumour infiltration *(Fig. 2)*.

The constellation of signs and symptoms presented, associated with imaging and endoscopic findings, favoured the diagnosis of PG. The antibiotic regimen was changed to IV meropenem. Due to progressive clinical worsening,

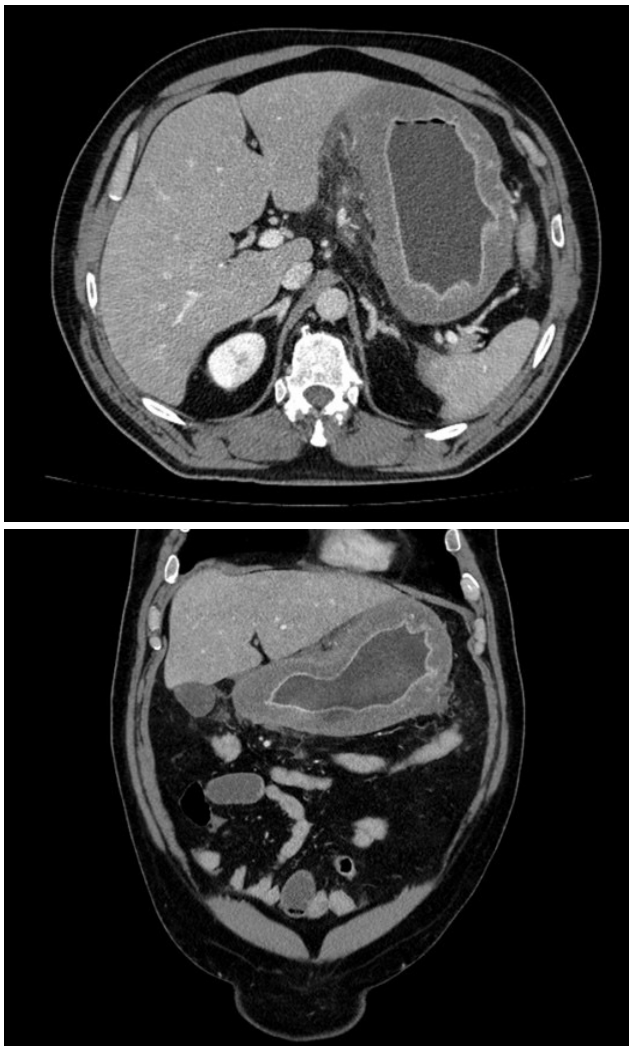

Figure 1. Abdominal-pelvic CT scan documenting markedly diffuse parietal thickening of the stomach with increased mucosal enhancement and densification of peri gastric fat and decreased homogeneous enhancement of the submucosal and muscular layers.

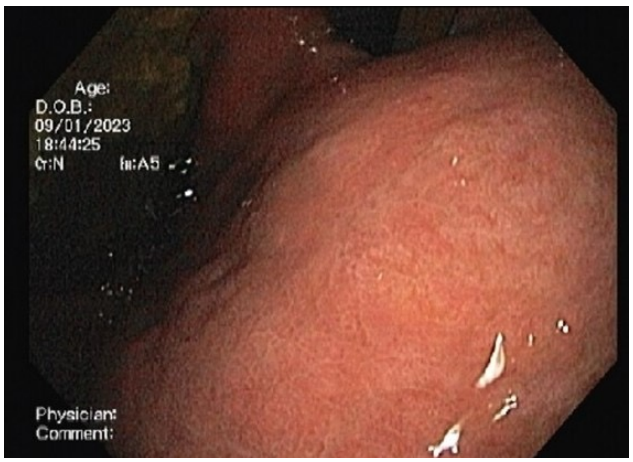

Figure 2. Upper gastrointestinal endoscopy revealing gastric mucosal diffuse oedema with no signs of tumour infiltration.

an urgent total gastrectomy was performed. Despite the interventions carried out, the patient developed refractory septic shock with multi-organ dysfunction and died within the first 24 hours after admission to the intensive care unit.

Histopathologic findings in the gastrectomy specimen were compatible with PG *(Fig. 3)*.

These showed intense oedema of submucosal and muscular layers, inflammatory infiltrate predominantly by neutrophils, extensive areas of necrosis and abscess formation, intense vascular congestion of lamina propria and gastric mucosal with preserved architecture, and non-atrophic chronic gastritis with mild activity. Gastric biopsies obtained with upper gastrointestinal endoscopy revealed intense atrophic chronic gastritis with moderate activity and presence of lymphoid aggregates, vascular congestion, oedema and foci of complete intestinal metaplasia. *Helicobacter pylori* was identified by immunohistochemistry, with the presence of multiple bacilli. Finally, the results from blood and gastric specimen cultures were all positive for the presence of *Streptococcus pyogenes*.

DISCUSSION

Phlegmonous gastritis (PG) is a rare and often fatal condition, frequently difficult to diagnose in time. It can affect healthy individuals in 50% of cases[4]. Here, we report on the case of a patient with PG, with no apparent risk factors, who presented to the emergency department with non-specific gastrointestinal symptoms. Clinical presentation in PG is usually ill-defined, with abdominal pain, vomiting, diarrhoea and fever, leading to multiple differential diagnosis and delayed treatment. Purulent emesis might be considered pathognomonic, although it rarely occurs in clinical practice[5]. Differential diagnosis includes conditions with disparate treatment and prognosis such as viral gastroenteritis, mucosal-associated lymphoid tissue lymphoma, *H. pylori* gastritis, pancreatitis and cholecystitis[6].

Important diagnostic tools include imaging evaluation. Abdominal CT typically reveals thickening of the gastric wall, low-intensity areas within the gastric wall (indicative of an abscess) and gas accumulation in emphysematous cases, with esophagogastroduodenoscopy usually being superior to CT in assessing the extent of gastric wall thickness and excluding underlying malignancy[5]. Endoscopic findings include oedematous and thickened gastric mucosa with occasional purulent discharge, superficial ulcerations, fibrinopurulent exudates lining the stomach and loss of rugae with stomach necrosis[2,7]. However, due to the variability of endoscopic findings and non-specific imaging characteristics, definitive diagnosis is usually obtained with gastrectomy histopathology or with autopsy. Inflammation mainly involves the submucosa, and typical pathologic findings include infiltration with neutrophils and plasma cells, therefore, biopsies involving only the mucosa may be normal or present with unspecific findings of gastritis[3]. In advanced cases, intramural haemorrhage, necrosis, thrombosis of the submucosal blood vessels and abscess formation might be present[7].

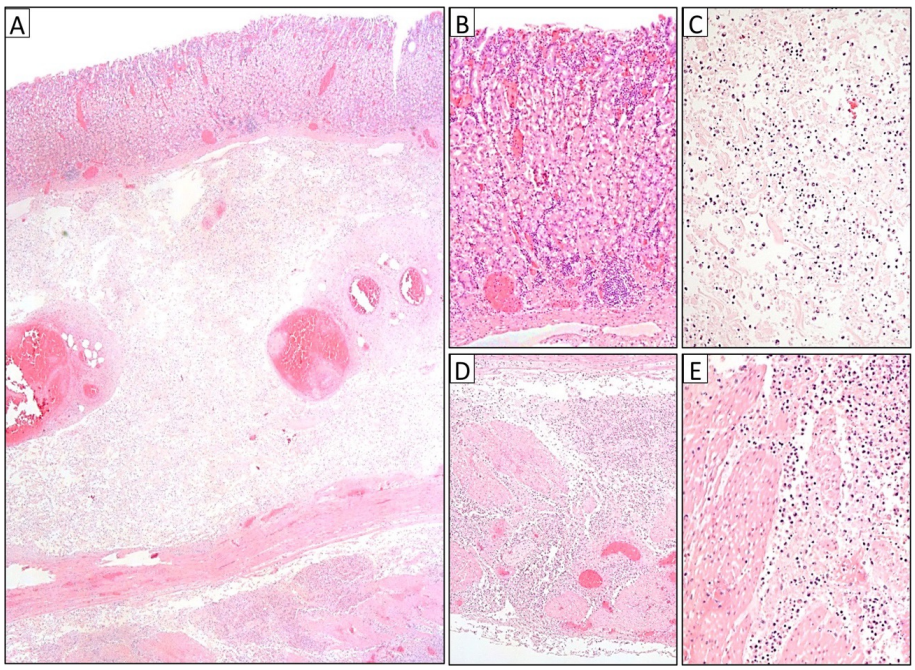

Figure 3. Histological sections of the gastric wall (haematoxylin and eosin stain). In the fundic gastric submucosa, there is (A) marked vascular congestion and ectasia and (B) mucosa with moderate inactive chronic gastritis, without architectural distortion or glandular atrophy, within an (C) oedematous stroma that is expanded by active chronical inflammation with granulocytes; within the muscularis propria layer, there is a (D) severe active chronical inflammation which dissociates the smooth muscle bundles and reaches the subserosa, with (E) abscess formation.

Culture of gastric aspirate or tissue, or blood cultures, reveal the pathogens involved, with β-haemolytic *Streptococcus* group A being the most common, identified in 70% of the cases, with other less frequent pathogens being *Enterobacteriaceae*, *Staphylococcus* spp., *Haemophilus influenzae* and endogenous bacteria of the oral cavity[5]. Although the pathophysiology of the disease is unknown, plausible mechanisms include direct invasion through gastric lesions from ingestion of infected respiratory tract secretions or gastric stasis that might promote bacterial growth, blood-borne spread (from bacterial endocarditis, erysipelas, tooth extraction, staphylococcal osteomyelitis and toxic shock syndrome)[3].

The recommended therapeutic approach is prompt initiation of broad-spectrum antibiotics and surgical resection of the involved gastric area in refractory and complicated cases[2]. However, there are no guidelines to direct antibiotic length or further management beyond clinical improvement. More studies and reports are needed to establish appropriate guidelines to identify and manage this disease.

Despite the high diagnosis suspicion within 24 hours after the first clinical manifestations and the prompt initiation of broad-spectrum antibiotic therapy and total gastrectomy, the patient developed refractory septic shock with multi-organ dysfunction and died. This is in line with the rapid progression of the disease leading to high morbidity and mortality, even after medical and surgical treatment.

Our case stands out as our patient had no apparent risk factors or other PG-related comorbidities but presented with a non-diagnosed and untreated *H. pylori* infection, that was found in the gastric specimen biopsy. To our knowledge, there is only one other case of PG with concomitant *H. pylori* infection reported in the literature[6].

CONCLUSION

We present a case of PG caused by *Streptococcus pyogenes* in a male patient with unspecific clinical manifestations and no apparent risk factors. CT allowed the detection of suggestive findings of PG, which along with upper gastrointestinal endoscopy findings and the typical rapidly worsening course, led to the initiation of broad-spectrum antibiotics. Nevertheless, the patient developed shock with multiple organ failure and underwent total gastrectomy and demise.

This case highlights that, despite being rare, PG is an entity that every doctor, especially those who work in the emergency department and the intensive care unit, should be aware of, to rapidly recognise and treat it.

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