

Single Case

Emphysematous Gastritis Associated with *Sarcina ventriculi*

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Keywords

Emphysematous gastritis · *Sarcina ventriculi* · Gastric emphysema

Abstract

Emphysematous gastritis is a rare and lethal gastrointestinal emergency if not promptly identified and managed. In rare cases, emphysematous gastritis is associated with *Sarcina ventriculi* infection, usually in patients with delayed gastric emptying. Here we report a lethal case of *S. ventriculi*-associated emphysematous gastritis in the absence of delayed gastric emptying in which the diagnosis was confirmed postmortem. This case provides an opportunity to review the clinical presentation, pathophysiology, and management of emphysematous gastritis so that the condition can be promptly diagnosed and managed to prevent significant morbidity and mortality.

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Introduction

Emphysematous gastritis is a rare and usually lethal gastrointestinal emergency, with an estimated mortality rate approaching 60% [1]. The condition is associated with chronic alcohol consumption, recent abdominal surgery, leukemia, lymphoma, renal failure, diabetes mellitus, gastric wall corrosion from acid or alkali poisoning, acute pancreatitis, disseminated strongyloidiasis, adenocarcinoma, phytobezoar consumption, and long-term treatment with

steroids or antibiotics [2]. Infections are an important but less common cause of emphysematous gastritis and are generally present in patients with predisposing conditions that delay gastric emptying. The infections most frequently associated with emphysematous gastritis include the gas-producing bacteria *Clostridium perfringens*, *Enterobacter* spp., *Klebsiella pneumoniae*, *Staphylococcus aureus*, *Streptococcus pneumoniae*, *Escherichia coli*, *Pseudomonas aeruginosa*, *Proteus* spp., and sometimes fungi such as *Mucormycosis* spp. and *Candida glabrata*, *krusei*, and *albicans* [3]. Emphysematous gastritis can also be caused by *Sarcina ventriculi*, a Gram-negative, gas-producing bacterium that has very rarely been described in association with emphysematous gastritis in the absence of delayed gastric emptying.

Here we present a rare but fatal case of emphysematous gastritis confirmed after post-mortem review in a patient without diabetic gastroparesis but with a preceding *S. ventriculi* infection. Since this condition is uncommon and lethal in most cases, we use this opportunity to review its clinical presentation, pathophysiology, and management so that gastroenterologists and other clinicians can quickly identify the condition, promptly intervene, and prevent significant morbidity and mortality.

Case Description

An 86-year-old woman with a history of hypertension and type II diabetes mellitus presented to the emergency department with a 3-day history of diffuse stabbing abdominal pain, nausea, vomiting, loss of appetite, and diarrhea described as black by her family. At presentation, she was afebrile and normotensive but appeared confused. There was generalized abdominal tenderness without guarding, rigidity, or rebound tenderness. She had a white cell count of 33×10^3 cells/ μ L (normal $4.8\text{--}10.8 \times 10^3$), serum creatinine 1.76 mg/dL (normal 0.8–2 mg/dL), high anion gap metabolic acidosis with serum bicarbonate 11 mmol/L (normal 24–31 mmol/L), an anion gap of 33 mmol/L (normal 5–15 mmol/L), beta-hydroxybutyrate 2.71 mmol/L (normal 0.02–0.28 mmol/L), and a lactic acid of 4.6 mmol/L (normal 0.5–2.2 mmol/L). An abdominal CT scan revealed air in the stomach wall and portal vein, air distending the stomach, and extensive prominent small bowel looping (Fig. 1). Intravenous fluids, vancomycin, piperacillin-tazobactam, and pantoprazole were started for the presumed diagnosis of emphysematous gastritis. The patient then had massive hematemesis, after which she rapidly became hemodynamically unstable. Despite resuscitation efforts, the patient died. A postmortem review confirmed the underlying diagnosis of emphysematous gastritis (Fig. 2) in the presence of bacterial overgrowth by *S. ventriculi* (Fig. 3a, b).

Discussion

S. ventriculi, a Gram-negative obligate anaerobe that ferments carbohydrates to produce carbon dioxide and ethanol as by-products, was first reported as an animal infection and also in the feces of healthy humans consuming a predominantly vegetarian diet [4]. However, since these initial reports, there have been increasing reports of *Sarcina* spp. in association with peritonitis after gastrointestinal viscous perforation and, rarely, emphysematous gastritis [5, 6].

Emphysematous gastritis is very rare, so its exact pathophysiology remains unclear. However, it is thought that a preexisting gastric ulcer serves as a nidus for bacterial infection, overgrowth, and penetration into the gastric wall [6]. After penetration, the organisms produce

gas. In the case of *S. ventriculi*, bacterial overgrowth is promoted by underlying conditions associated with delayed gastric emptying such as diabetic gastroparesis, pyloric stenosis, gastric surgery, slipped gastric banding, scarring, or obstructing masses, because gastric content retention and decreased gastric outflow provide time for the organisms to grow and penetrate the gastric wall [7]. Furthermore, overgrowth of *Sarcina* spp. is promoted by this organism's ability to survive and grow even at pH 1, essentially evading the natural defense mechanism of the stomach's acidic environment [8, 9].

Identifying the symptoms of emphysematous gastritis can be challenging and hence catastrophic when missed. The most frequent symptoms include abdominal pain, nausea, vomiting, and, in some cases, fever and chills. These symptoms can cause clinical confusion since they are nonspecific and there is considerable overlap with the symptoms of gastroenteritis. However, the presence of hematemesis, melena, leukocytosis, portal vein gas, lactic acidosis, and hemodynamic instability or hypotension arouse suspicion and support the diagnosis of emphysematous gastritis [10, 11]. Diagnostic testing for emphysematous gastritis involves plain abdominal radiographs or abdominal CT, which may show air within the gastric wall and portal vein [2]. Clinically, gas within the stomach wall can be categorized into two groups: emphysematous gastritis and gastric emphysema, a relatively benign condition caused by air entering the gastric wall, usually following trauma to the gastric mucosa. This distinction is critical, since patients with gastric emphysema are generally asymptomatic and the air in the stomach wall is generally considered benign, in contrast to emphysematous gastritis, which is never benign [10, 11].

The management of emphysematous gastritis is challenging because patients can quickly deteriorate and become hemodynamically unstable, as seen in this case. The most ominous sign in these patients is the radiographic finding of portal vein gas caused either by intraluminal air or air from the bacterium [12, 13]. This sign generally represents the worst prognostic marker in these patients, with an estimated mortality exceeding 75% when present [13]. Despite a lack of consensus guidelines for the treatment of emphysematous gastritis, a therapeutic approach based on disease severity has been devised in a few case reports [10, 14, 15]. Prompt intervention with intravenous antibiotics, analgesia, total parenteral nutrition, and proton pump inhibitors should be attempted. There are some reports in the literature of gastrectomy as a potential treatment option; however, patient age and comorbidities should be weighed accordingly in discussions regarding the risks and benefits of invasive procedures because of the high mortality rate associated with this condition [16]. Though the role of surgery during the acute phase remains unclear, indications for emergency surgery include clinical deterioration despite medical treatment, involvement of a large portion of or the entire stomach, and gastric infarction or perforation [16, 17]. Other complications such as development of strictures may occur in approximately 25% of patients as late findings during healing and are considered absolute indications for surgery [18].

In summary, we report a case of emphysematous gastritis in association with *S. ventriculi* in the absence of the most common risk factors including delayed gastric emptying. The underlying etiology was unclear. Although treatment was started promptly and death was probably inevitable, this case serves as a reminder to gastroenterologists and other clinicians that a high degree of suspicion for emphysematous gastritis should be maintained when patients present with abdominal pain, hematemesis, and/or melena. Imaging that shows gas within the wall of the stomach and portal vein must be acted on immediately so that appropriate interventions can be initiated to prevent mortality. Clinicians should also be aware that *S. ventriculi* can present in patients in the absence of delayed gastric emptying. Due to the rarity of

this condition and the lack of clear consensus guidelines, future reports may be useful to identify cases that may benefit from less invasive therapies such as endoscopy to spare patients from surgery.

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Statement of Ethics

This study was completed in compliance with guidelines for human studies and is ethically in accordance with the World Medical Association Declaration of Helsinki. The patient's next of kin gave written informed consent to publish this case and images.

Disclosure Statement

The author has no conflicts of interest to declare. He declares no relevant funding sources.

Author Contributions

Dr. K. Singh wrote and prepared this paper.

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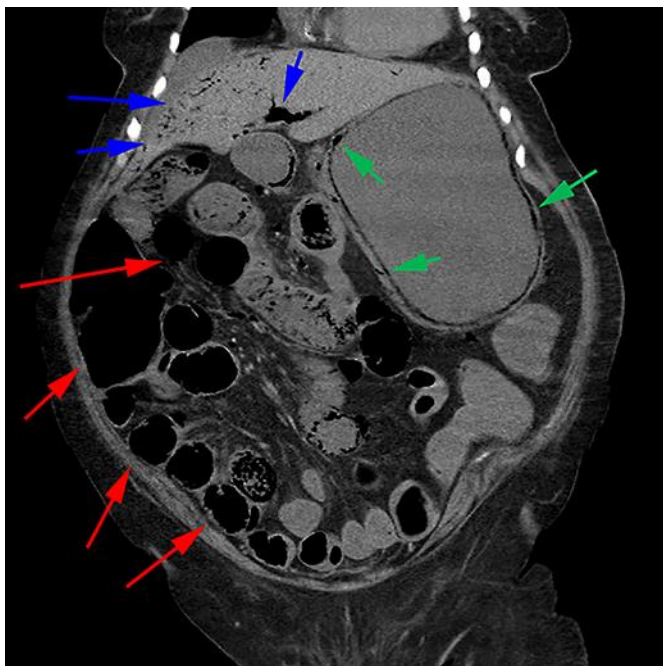


Fig. 1. Abdominal CT scan revealing marked air within the gastric wall (green arrows), extensive prominent looping of the small bowel (red arrows), and air within the portal vein (blue arrows).



Fig. 2. Gross specimen of the stomach retrieved from autopsy showing areas of hyperemia and necrosis suggestive of emphysematous gastritis.

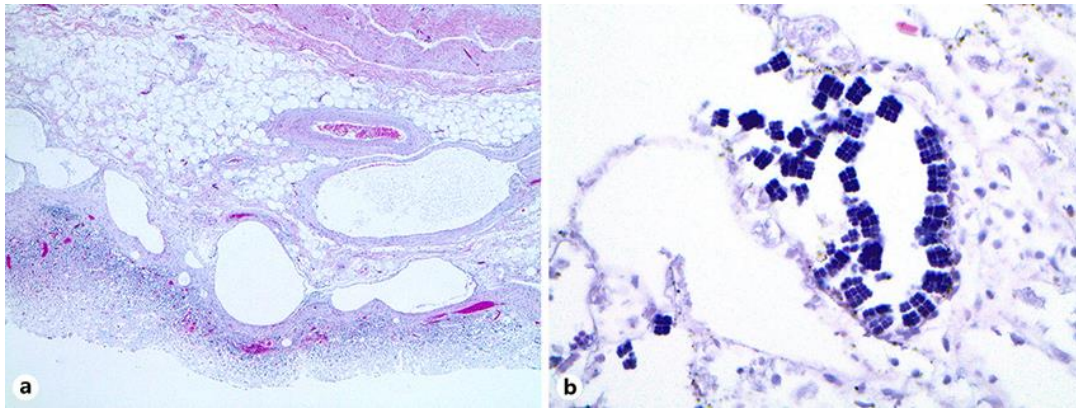


Fig. 3. Photomicrographs of the stomach stained with hematoxylin and eosin. **a** 4× magnification showing air sacs within the stomach suggestive of emphysematous gastritis. **b** 40× magnification of the stomach specimen demonstrating the characteristic features of bacterial overgrowth by *S. ventriculi*, namely basophilic, cuboid-shaped organisms in a tetrad formation.