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CASE REPORT | ESOPHAGUS

Concomitant *Sarcina*-Associated Erosive Esophagitis and Refractory *Helicobacter pylori* Gastritis

Matthew Chan, DO[1], Deepanshu Jain, MD[2], Corrado Minimo, MD[3], and Alison Platt, DO[2]

[1]*Department of Internal Medicine, Albert Einstein Medical Center, Philadelphia, PA*
[2]*Division of Gastroenterology and Hepatology, Albert Einstein Medical Center, Philadelphia, PA*
[3]*Department of Pathology and Laboratory Medicine, Albert Einstein Medical Center, Philadelphia, PA*

ABSTRACT

For unclear reasons, there has been an increasing number of reported cases of *Sarcina* infections in the gastrointestinal tract over the past several years. Associated clinical conditions with the infection most commonly include delayed gastric emptying from diabetes mellitus, a history of previous gastrointestinal surgery, and ulcer disease. The precise pathogenetic role of *Sarcina* infection in humans remains unclear. Because of the ubiquitous environmental presence of *Sarcina* and limited previously reported clinical cases, the link between symptoms along with endoscopic findings to *Sarcina* can be associative at best. When found in the upper GI tract, the decision to treat along with the chosen regimen remains debatable. *Sarcina*, however, has rarely been seen in the esophagus. We report the third case of *Sarcina* of the esophagus associated with *Helicobacter pylori* gastritis.

INTRODUCTION

Sarcina is a Gram-positive, nonmotile, anaerobic coccus that relies exclusively on the fermentative metabolism which was first identified in the gastric contents of human beings by John Goodsir in 1842.[1,2] Since then, its precise pathogenetic role in human beings still remains unclear because clinical observations have ranged from asymptomatic to life-threatening complications, such as gastric perforation and emphysematous gastritis.[2,3] To date, less than 30 cases of *Sarcina*-associated gastrointestinal pathology have been published in the literature, with most of them presenting with symptoms of abdominal pain, nausea, vomiting, and delayed gastric emptying.[4] Our review of literature yielded a total of 7 case reports that reported 8 patients with *Sarcina* on esophageal biopsies.[5–11] Two of those had concomitant *Helicobacter pylori* gastritis.[11] We report the third case of *Sarcina* of the esophagus associated with *H. pylori* gastritis.

CASE REPORT

A 70-year-old woman presented to the gastroenterology outpatient clinic with an increase in abdominal discomfort, heartburn, postprandial nausea, and intermittent episodes of nonbilious, nonbloody vomiting over the past several months. She had previously been taking omeprazole daily for reflux symptoms without relief and naproxen daily for joint pain. She underwent an esophagogastroduodenoscopy that showed erythematous mucosa in the distal esophagus, gastropathy, and a hiatal hernia (Figure 1). Targeted esophageal and gastric mucosal biopsies showed erosive esophagitis with many colonies of *Sarcina* species and chronic gastritis with *H. pylori* infection, respectively (Figure 2). The diagnosis of *Sarcina* was made with hematoxylin-eosin stain and Gram stain.

The patient was prescribed tetracycline, metronidazole, and bismuth subsalicylate for 2 weeks, and her omeprazole increased to twice daily for treatment of both infections. After completing the treatment regimen, the patient reported having little improvement in symptoms. Repeat esophagogastroduodenoscopy showed normal-appearing mucosa of the esophagus, stomach, and duodenum. Esophageal biopsies showed normal squamous mucosa. Gastric biopsies showed persistent *H. pylori* gastritis. She was started on a salvage treatment regimen of levofloxacin 500 mg daily, amoxicillin 1 g twice daily, and omeprazole 20 mg twice daily for a total of 14

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Correspondence: Matthew Chan, DO (chanmatt@einstein.edu).

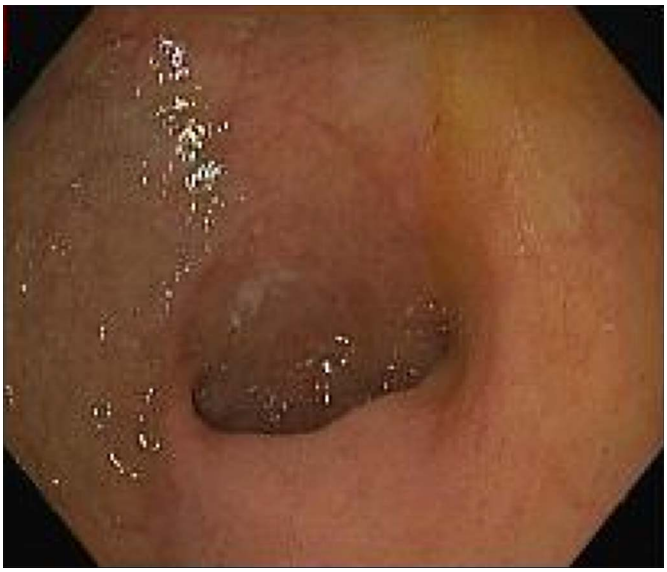

Figure 1. Esophagogastroduodenoscopy showing gastric erosions.

days. Repeat testing was noted for persistent *H. pylori* infection. At this point, we planned to await culture and antibiotic sensitivity report before considering retreatment.

DISCUSSION

Sarcina has been well documented in the veterinary literature as being a causative organism of gastric dilatation and death in sheep, goats, cats, and horses.[12,13] The bacteria have been found in the soil, air, and stagnant water, and even in the feces of human beings who have been noted to eat primarily a plant-based diet.[14] However, there is currently no clear understanding of whether it has a precise mechanism for causing direct disease in human beings. Most cases of infection have been seen in patients with a history of gastric outlet obstruction, diabetic gastroparesis, pyloric stenosis, and gastrointestinal surgeries.[4,14] It has been believed that these pre-existing conditions serve as a prime environment for rapid growth of the anaerobic organism, which in turn produces extensive carbon dioxide through fermentative metabolism that leads to patients reporting symptoms of bloating and abdominal discomfort.[2]

Sarcina has also been implicated in human cases of gastric perforation, emphysematous gastritis, and gastric adenocarcinomas.[15] However, there have been reports of asymptomatic patients with *Sarcina* found incidentally on gastric biopsies.[13] In our case, *Sarcina* was found on esophageal biopsy, and not on gastric biopsy. Based on our review of the literature, there have been 8 patients noted to have *Sarcina* identified on esophageal biopsies.[5–11] Common presenting symptoms included epigastric pain, dysphagia, and vomiting. Of the 8 cases, 3 had endoscopic findings of erosive esophagitis.[9,11] Of the 8 cases, 2 had concomitant *H. pylori* gastritis.[11] Of the 8 cases, 2 had concomitant alternative esophageal etiologies that included *Candida* and Cytomegalovirus infection.[5,7] Of the 8 cases, only 2 were provided treatment for *Sarcina*.[8,9]

In our case, minimal improvement in patient symptoms despite successful *Sarcina* eradication and persistent *H. pylori* infection suggests that although there was endoscopic and histologic evidence of esophagitis, most patient symptoms were likely due to *H. pylori* or noninfective etiology. This finding supports those who favor *Sarcina* to be a benign pathogen in the GI tract. Furthermore, there is currently no consensus on a standard regimen or duration for the treatment of *Sarcina* infection. Because of its implication in cases of life-threatening complications, such as gastric perforation and emphysematous gastritis, some have decided to treat patients with a regimen consisting of antibiotics, a proton-pump inhibitor, and a prokinetic.[12] In our case, our patient was treated with a similar regimen that covered for both *Sarcina* and *H. pylori* infections. Although persistent *H. pylori* infection was noted on repeat biopsy, *Sarcina* was not. This result illustrates endoscopic and histological improvement with treatment, but our patient did not improve symptomatically. It is unclear whether *H. pylori* gastritis in any way can be associated with *Sarcina* esophagitis, but this case certainly raises a question.

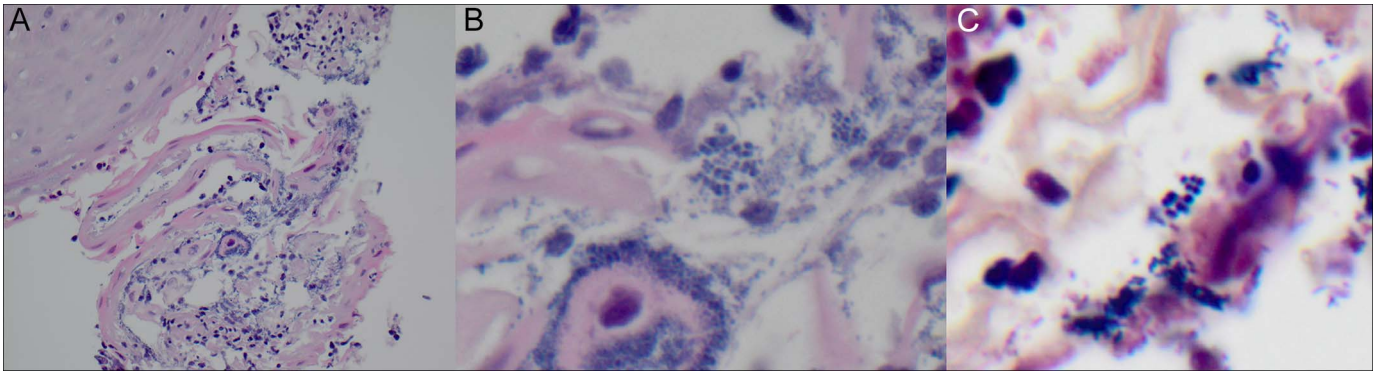

Figure 2. Biopsy showing (A) squamous mucosa with acute inflammation and reactive cellular changes that includes parakeratosis and fibrinopurulent exudate indicative of esophageal erosion (hematoxylin and eosin stain, 40× magnification) and (B) polymorphous population of cocci, the largest in tetrads characteristic of *Sarcina* organisms (hematoxylin and eosin stain, 60× magnification) and Gram stain showing Gram-positive cocci (60× magnification).

In conclusion, our case is the third-ever report of concomitant *Sarcina* esophagitis and *H. pylori* gastritis. In the absence of known risk factors for *Sarcina* infection of the upper GI tract such as gastric outlet obstruction or gastroparesis, the possibility that active *H. pylori* infection is a risk factor for *Sarcina* infection cannot be refuted. Whether *Sarcina* has a direct pathogenetic role in gastric and esophageal diseases along with it warranting antibiotic treatment remains unknown. Few prefer to treat it because of the risk of associated life-threatening complications.

DISCLOSURES

Author contributions: M. Chan wrote the manuscript. D. Jain and A. Platt edited the manuscript. C. Minimo provided the pathology images. M. Chan is the article guarantor.

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Informed consent was obtained for this case report.

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