ACG CASE REPORTS JOURNAL

IMAGE | ESOPHAGUS

Sarcina ventriculi as an Unknown Culprit for Esophageal Stricturing

Jennifer Behzadi, MD[1], Rohan M. Modi, MD[2], Kashika Goyal, BS[3], Wei Chen, MD, PhD[4], and Sheryl Pfeil, MD[1]

[1]*Division of Gastroenterology, Hepatology and Nutrition, The Ohio State University Wexner Medical Center, Columbus, OH*
[2]*Department of Internal Medicine, The Ohio State University Wexner Medical Center, Columbus, OH*
[3]*The Ohio State University College of Medicine, Columbus, OH*
[4]*Department of Pathology, The Ohio State University Wexner Medical Center, Columbus, OH*

CASE REPORT

A 65-year-old woman with metastatic breast cancer and a history of Schatzki ring presented with 2 months of progressive dysphagia. Imaging with x-ray fluoroscopy revealed an irregularly short segment stricture at the mid to distal esophagus. Esophagogastroduodenoscopy (EGD) showed a benign-appearing stenosis with a single 7-mm nodule at the gastroesophageal junction and non-bleeding gastric ulcers in the setting of residual food in the stomach (Figure 1). She underwent subsequent dilation 6 days later under fluoroscopy with a Savary dilator up to 12 mm, and she was medically managed with a proton pump inhibitor. Esophageal and gastric biopsies revealed acute and chronic inflammation with rare large tetrads of bacterial cocci, morphologically consistent with *Sarcina ventriculi* (Figure 2). In light of *S. ventriculi* presence, she was treated with 7 days of metronidazole and ciprofloxacin. An esophageal stent was placed 1 week after initial dilation failed to improve symptoms. Unfortunately, her hospital course was complicated by hemorrhagic gastropathy secondary to therapeutic heparin in the setting of a pulmonary embolism, which resulted in stent removal 2 weeks after initial placement. Ultimately, the patient was transitioned to palliative comfort care due to overall poor prognosis.

S. ventriculi is a well-described pathogen in veterinary medicine as a Gram-positive organism with the microscopic appearance of spherical cells organized as tetrads. Human presentation has varied widely, from gastrointestinal symptoms of nausea, vomiting, and epigastric pain to emphysematous gastritis complicated by gastric perforation.[1,2] EGD findings have also differed, but common themes have emerged, including luminal findings of retained food residue and mucosal findings of erosions and gastric ulcers.[3] Similar to our case, previous treatment regimens for *S. ventriculi* infection have included antibiotics and proton pump inhibitors.[4] This case is unique in that most reported cases of *S. ventriculi* pathology have been gastric in nature with very few described in the esophagus. Compared to previously reported esophageal *S. ventriculi* pathology, our findings are in the setting of stasis secondary to esophageal strictures.

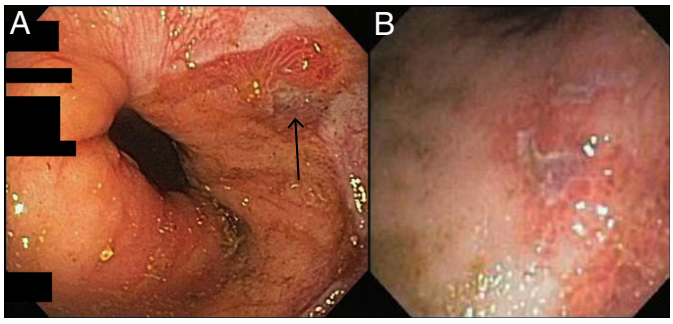

Figure 1. EGD findings of (A) severe esophageal stenosis with a single 7-mm nodule (arrow) at the gastroesophageal junction, and (B) non-bleeding superficial gastric ulcers in the body and antrum.

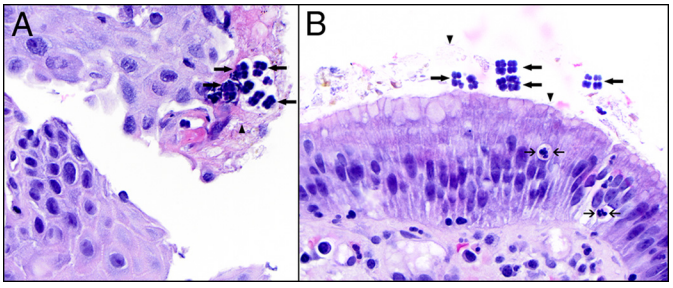

Figure 2. Hematoxylin and eosin stain (magnification 600x). (A) Esophageal and (B) gastric biopsy showing purple *Sarcina ventriculi* cocci in characteristic tetrads (long arrows) that are larger in comparison to the luminal bacilli (arrowheads). There is mild acute inflammation demonstrated by the intraepithelial neutrophils (short arrows), in the adjacent gastric epithelium.

ACG Case Rep J 2017;4:e118. doi:10.14309/crj.2017.118. Published online: November 8, 2017.

Correspondence: Jennifer Behzadi, 395 W 12th Ave, 2nd Floor, Columbus, OH 43210 (jennifer.behzadi@osumc.edu).

Studies of the esophageal microbiome do not identify *S. ventriculi* in patients with normal histology, which suggests that, in contrast to gastric sites, the organism is more likely to be a pathogen.[5] However, it is important to recognize that *S. ventriculi* may not be a causative agent in the setting of esophageal stricture and may instead be a benign pathogen. Moving forward, it will be important to further assess the importance of *S. ventriculi* in the pathogenesis of esophageal diseases.

DISCLOSURES

Author contributions: J. Behzadi and R. Modi wrote manuscript. K. Goyal and S. Pfeil edited the manuscript. W. Chen provided the pathological images. J. Behzadi is the article guarantor.

Financial disclosure: None to report.

Informed consent could not be obtained for this case report, as attempts to contact the patient's next of kin were unsuccessful.

Previous presentation: This case was presented in part at the World Congress of Gastroenterology at ACG2017; October 13–18, 2017; Orlando, Florida.

Received June 29, 2017; Accepted September 12, 2017

REFERENCES

1. Laass MW, Pargac N, Fischer R, et al. Emphysematous gastritis caused by *Sarcina ventriculi*. *Gastrointest Endosc*. 2010;72:1101–3.
2. Lam-Himlin D, Tsiatis AC, Montgomery E, et al. *Sarcina* organisms in the gastrointestinal tract: A clinicopathologic and molecular study. *Am J Surg Pathol*. 2011;35:1700–5.
3. Smith SMB-LJ, Chen W. *Sarcina* species-associated gastrointestinal disease. *AJCP Case Reports*. 2016;MB16:1–10.
4. Ratuapli SK, Lam-Himlin DM, Heigh RI. *Sarcina ventriculi* of the stomach: A case report. *World J Gastroenterol*. 2013;19:2282–5.
5. Carrigan S, Grin A, Al-Haddad S, et al. Emphysematous oesophagitis associated with *Sarcina* organisms in a patient receiving anti-inflammatory therapy. *Histopathology*. 2015;67(2):270.