Gastric *Sarcina ventriculi*: incidental or pathologic?

Andrew C. Berry\(^a\), Shivtaj Mann\(^b\), Rahman Nakshabendi\(^c\), Ozdemir Kanar\(^d\), Lorna Cruz\(^d\)

University of South Alabama; Nova Southeastern University College of Osteopathic Medicine; University of Florida College of Medicine; West Palm Hospital, USA

A 65-year-old Caucasian female with a history of lap-band procedure ten years ago presented with generalized weakness. For the past two weeks, the patient had been fatigued, sleeping 12-20 h a day and having minimal strength for her activities of daily living. For three days she had had dark stools and diarrhea and denied having a history of gastroparesis. She denied tobacco, alcohol, or illicit drug use. She was taking omeprazole and clopidogrel daily. Her vitals were unremarkable. She was mildly tender to palpation on the lower left abdomen and mildly distended. Labs were unremarkable except hemoglobin 10.4 g/dL and hematocrit 32.2%. Stool for occult blood was positive. The patient underwent esophagogastroduodenoscopy (EGD) with biopsy of the gastric wall (Fig. 1A). Three days later, a subsequent colonoscopy was performed due to bright red blood per rectum and persistent symptomatic anemia.

*Sarcina ventriculi* (*S. ventriculi*) is a gram-positive anaerobic bacterium uncommonly found in the feces and blood of healthy humans, with only nine cases isolated from human gastric specimens [1]. Hematoxylin and eosin staining showed structures occurring in tetrads with molding and flattening of the cell borders (Fig. 1B, C, D), suggestive of gastric *S. ventriculi*. This represents only the second of ten cases that presented without typical gastrointestinal symptoms and the first without gastrointestinal symptoms and evidence of gastroparesis. *S. ventriculi* has been implicated as a potential cause of gastric ulcers, emphysematous gastritis, and gastric perforation [1]. To date, two cases of *S. ventriculi* have been associated with life threatening illness from emphysematous gastritis [2]. Its limited association with severe illness warrants pre-emptive treatment with anti-ulcer therapy and antibiotics.

We suspect that both the ulcerations on EGD (Fig. 1A) and colonoscopy findings of an arteriovenous malformation, diverticulosis, and internal hemorrhoids contributed to the patient’s chronic symptomatic anemia. Since our patient’s condition did not appear life-threatening, she was treated with proton pump inhibitors, was hemodynamically stabilized, and was discharged without antibiotic treatment. It is important to assess whether gastric *S. ventriculi* is an innocent bystander or whether it assumes a pathologic role to help guide treatment decisions.

References