Sarcina ventriculi

Review of the Literature

Mohamed Rizwan Haroon Al Rasheed, MBBS; Carmencita G. Senseng, MD

• ***Sarcina ventriculi*** **is an increasingly common gram-positive coccus, recognized in gastric biopsies, particularly of patients with delayed gastric emptying. It occurs most commonly in adult women and can be identified easily by its characteristic morphologic features, such as basophilic staining, cuboid shape, tetrad arrangement, red blood cell–sized packets, flattened cell walls, and refractile nature on light microscopy. Although the pathogenesis of the organism is debated, it has been implicated in cases of gastric perforation, emphysematous gastritis, and peritonitis as well as occurring in the background of gastric adenocarcinomas. This review of the literature discusses the clinical features, endoscopy findings, histopathology, ancillary studies, microbiology, pathogenesis, differential diagnosis, treatment, and prognosis of this bacterium based on 19 published cases.**

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Sarcina ventriculi is a gram-positive, anaerobic coccus that can grow in acidic environments,[1] with a carbohydrate fermentative metabolism as its sole energy source.[2] It was first identified as a human pathogen by Goodsir[3] in 1842, which was followed by a few early reports of its presence in the human stomach.[4] However, since then, there have been only 19 cases of *S ventriculi* (Table 1) reported in the English literature, to our knowledge, and almost all of those cases were reported within the past 5 years. Importantly, *S ventriculi* accompanies delayed gastric emptying[5,6] and is thought to cause emphysematous gastritis[7] and perforation.[8] This review of the literature discusses the clinical features, endoscopic findings, histopathology, ancillary studies, microbiology, pathogenesis, differential diagnosis, treatment, and prognosis of this rare, but increasingly common, bacterium based on 19 published cases.

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From the Department of Pathology, University of Illinois, Chicago (Dr Haroon Al Rasheed); and the Department of Pathology, John H. Stroger Jr Hospital of Cook County, Chicago (Dr Senseng).

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Reprints: Mohamed Rizwan Haroon Al Rasheed, MBBS, Department of Pathology, University of Illinois, 840 S Wood St, Ste 130 CSN, Chicago, IL 60612 (email: mharoon@uic.edu or mohamedrizwan@gmail.com).

CLINICAL FEATURES

Sarcina ventriculi occurs mainly in adults (Table 2) but has been identified in an age range from 3 to 73 years. It occurs more often in women than it does in men, with 13 of the reported 19 cases (68%) in women, resulting in a female to male ratio of slightly more than 2:1. Although the race of the patient was not documented in most articles, there is at least one report identifying patients as Hispanic,[8] white,[9] and African American.[10] More than one-half of the patients (11 of 19; 58%) had a history of gastrointestinal surgery, gastroparesis, and/or gastric outlet obstruction. *S ventriculi* is also reported to present in association with acute, life-threatening complications, such as gastric perforation[8] and emphysematous gastritis.[7] In addition, it has been identified in the stomach of patients with gastric adenocarcinoma and pancreatic adenocarcinoma.[5]

The earliest clinical observations of patients with this organism led to the term *sarcinous vomit*, which described a characteristic, obstinate, frothy vomit in patients with chronic disease of the stomach.[6] Clinically, as shown in Table 2, most patients present with gastrointestinal symptoms, such as nausea, vomiting, and/or abdominal pain.[5,11] However, the patients may be asymptomatic, and the bacterium may present as an incidental finding in a gastric biopsy[12] or with more worrying symptoms, such as melena (3 cases; 15.8%), hematemesis (2 cases; 10.5%), and weight loss. During endoscopy, the most consistent feature is the presence of food bezoar (9 cases; 47.4%) because of the delayed gastric emptying, which is often accompanied by an inflamed and/or ulcerated gastric mucosa (12 cases; 63.2%).

HISTOPATHOLOGY

Classically, *S ventriculi* is identified by light microscopy in a gastric biopsy specimen with the following characteristic morphologic features: (1) basophilic staining with hematoxylin-eosin[11,12]; (2) cuboid shape[13]; (3) 1.8 to 3 μm individual size,[5] or packets approximately the size of a red blood cell[11]; (4) a tetrad packet arrangement[14] (Figure, A), which is a result of the replication occurring in at least 2 planes of growth[5]; (5) flattening of the cell walls in areas of contact with adjacent cells[8]; and (6) its refractile nature, which can mimic vegetable matter.[5]

The organisms are generally located near the mucosal surface in the gastric mucin (Figure, B), rather than in the gastric pits, and are not invasive.[11] The histologic features of the gastric mucosa can vary, ranging from being fairly unremarkable (3 cases, 15.8%) in some patients[15,16] to

Table 1. Clinical Features, Endoscopic Findings, Histopathology, Treatment, and Follow-up of 19 Cases of *Sarcina ventriculi* Reported in the Literature

Source, y	Age, y/Sex	Clinical	Endoscopy
Tolentino et al,[8] 2003	14/M	Abd pain, distension, gastric perforation, peritonitis, and necrosis; PMH: bowel resection	N/A
	50/M	N, V, hematemesis, melena, weight loss	Esophagitis, hiatal hernia, duodenal mass
Laass et al,[7] 2010	3/F	Anorexia, V, hematemesis, abd distension; x-ray: dilated stomach with intramural air, GOO	Necrotic stomach distended by blood and air
Lam-Himlin et al,[5] 2011	58/F	Abd pain, V; PMH: GOO	Gastritis, pyloric mass with obstruction and bezoar
	44/F	Postprandial dyspepsia; PMH: type I DM with GP	Gastric ulcer, pyloric polyps, and food bezoar
	36/M	N, V, with epigastric pain; PMH: narcotic-related GP and GOO	Food bezoar
	12/F	Dysphagia; PMH: esophageal atresia after gastric pull-through with anastomotic narrowing	Stricture at anastomosis, food bezoar
	46/F	Painful epigastric spasms; h/o pylorus-sparing pancreatico-duodenectomy for pancreatic adenocarcinoma	Bile, food bezoar
Ratuapli et al,[22] 2013	73/M	Chronic iron deficiency anemia; PMH: refractory gastric ulcers treated by Billroth II antrectomy and truncal vagotomy	Diffuse gastric erythema, 2 polyps at anastomosis site, food bezoar
Sauter et al,[11] 2013	12/M	V, epigastric pain refractory to PPIs; serum positive for *H pylori*	Erosive esophagitis, gastritis, edematous and tight pylorus
	16/F	V, epigastric pain; PMH: GERD	Erosive esophagitis, gastritis, edematous pylorus, food bezoar
Kulkarni et al,[16] 2013	34/F	Veterinarian with epigastric pain, chronic intermittent diarrhea	Normal esophagus, stomach, and duodenum
DiMaio et al,[27] 2014	37/F	Intermittent epigastric pain, N, anorexia × 2 wk, abdominal distension; PMH: cystic fibrosis	Severe erythema, linear erosions in posterior wall of gastric antrum
Kumar et al,[15] 2014	3/M	V, chronic diarrhea; PMH: hep A	Normal gastric mucosa, mild grooving in D2
Bhagat et al,[28] 2015	55/F	V, abd pain; CT: diffuse thickening of pyloric antrum	Deep ulcer in pylorus
Medlicott and Adams,[26] 2015	53/F	Epigastric pain, regurgitation, V; PMH: gastric banding, gastric ulcer, GERD, DM, hiatus hernia	Food bezoar, polyps, healed ulcer
Berry et al,[9] 2015	65/F	Melena, diarrhea, weakness; PMH: laparoscopic banding	Pouch, restriction with ulcers
Sopha et al,[10] 2015	32/F	Headache, dyspnea, melena; PMH: gastric banding	Ulcer at gastric cardia
Haroon al Rasheed et al,[12] 2016	57/F	Asymptomatic; PMH: type 2 DM, *H pylori*, chronic active gastritis, multiple ulcers	Scarred and eroded before prepyloric ulcer, food bezoar

Abbreviations: Abd, abdominal; CT, computed tomography; D2, second part of the duodenum; DM, diabetes mellitus; GERD, gastroesophageal reflux disease; GOO, gastric outlet obstruction; GP, gastroplasty; hep A, hepatitis A; h/o, history of; *H pylori*, *Helicobacter pylori*; N, nausea; N/A, not applicable; PMH, past medical history; PPI, proton pump inhibitor; V, vomiting.

displaying diffuse, acute hemorrhagic gastritis with ulceration in others.[8] Given that there are no consistently associated histologic features in the gastric mucosa, a high level of suspicion is necessary, particularly when examining gastric biopsies from patients with a history of gastrointestinal surgery or delayed gastric emptying.

Regarding its relationship to *Helicobacter pylori*, only Sauter et al[11] identified the concurrent presence of *H pylori* and *S ventriculi* in the same specimen, whereas Haroon Al Rasheed et al[12] reported the presence of *S ventriculi* after treatment of *H pylori*, raising the interesting possibility of *S ventriculi* and *H pylori* being mutually exclusive. However, case reports have identified *S ventriculi* as occurring concurrent with other organisms, particularly *Candida* spp (2 cases, 10.5%).[7]

ANCILLARY STUDIES

Sarcina ventriculi is typically diagnosed with a hematoxylin-eosin stain and, if needed, with a Gram stain, which stains strongly positive. However, the organism can also be confirmed at a molecular level by polymerase chain reaction and sequencing of the 16S ribosomal RNA (rRNA) gene and pyruvate decarboxylase gene. The 16S rRNA gene is highly conserved within the different species of bacteria and is, therefore, commonly used to classify and identify organisms. The 16S rRNA gene of each bacterial species is unique, so

Table 1. Extended

Histopathology	Treatment	Follow-up
Diffuse, acute, hemorrhagic gastritis with perforation, ulceration, necrosis, and *Sarcina*	Gentamicin and metronidazole × 12 d	Symptomatic improvement
Chronic, superficial gastritis and ulcer with *Sarcina*	N/A	N/A
Chronic, active gastritis, *Sarcina*, and intramural air	Imipenem and fluconazole × 2 wk and omeprazole	Fully recovered; normal mucosa and no *Sarcina*
Chronic, active gastritis and *Sarcina*	Partial gastrectomy.	Treated for gastric adenocarcinoma
Nonmalignant gastric ulcer with *Sarcina*, gastric hyperplastic polyps	Omeprazole, ranitidine, metoclopramide × 5 mo	Symptomatic improvement
Normal gastric mucosa and *Sarcina*	Jejunostomy tube for malnutrition	No *Sarcina*
Reflux esophagitis and *Sarcina*	N/A	N/A
Chronic active duodenitis and *Sarcina*	N/A	Continued spasms
Diffuse gastritis, gastric ulcer, and *Sarcina*; no *H pylori*	Metronidazole and ciprofloxacin × 1 wk and sucralfate	Improved gastric erythema; no food bezoar or *Sarcina*
Active erosive esophagitis and ulcer bed; chronic, active *H pylori* gastritis and duodenitis; *Sarcina* in esophagus and stomach	N/A	N/A
	N/A	N/A
Normal gastric mucosa with *Sarcina*; intraepithelial eosinophils in esophagus and lymphocytosis with villous blunting in duodenum	Ciprofloxacin and metronidazole × 1 wk	Identical histology but no *Sarcina*
Moderate chronic gastritis and superficial mucosal hemorrhage with *Sarcina* and *Candida*	Omeprazole.	Symptomatic improvement
Normal duodenal mucosa with *Sarcina* and *Giardia*	N/A	N/A
Gastric adenocarcinoma, ulcer, and *Sarcina*	N/A	N/A
Mild, chronic gastritis with *Sarcina*	Motilium and metronidazole	Persistent polyps and reactive gastropathy; no *Sarcina*
Sarcina	PPIs	Stable
Sarcina	Metronidazole, fluoroquinolone, and PPI × 4 wk	Persistent ulcer with perforation during endoscopy
Sarcina, ulcer bed, and reactive gastropathy	N/A	N/A

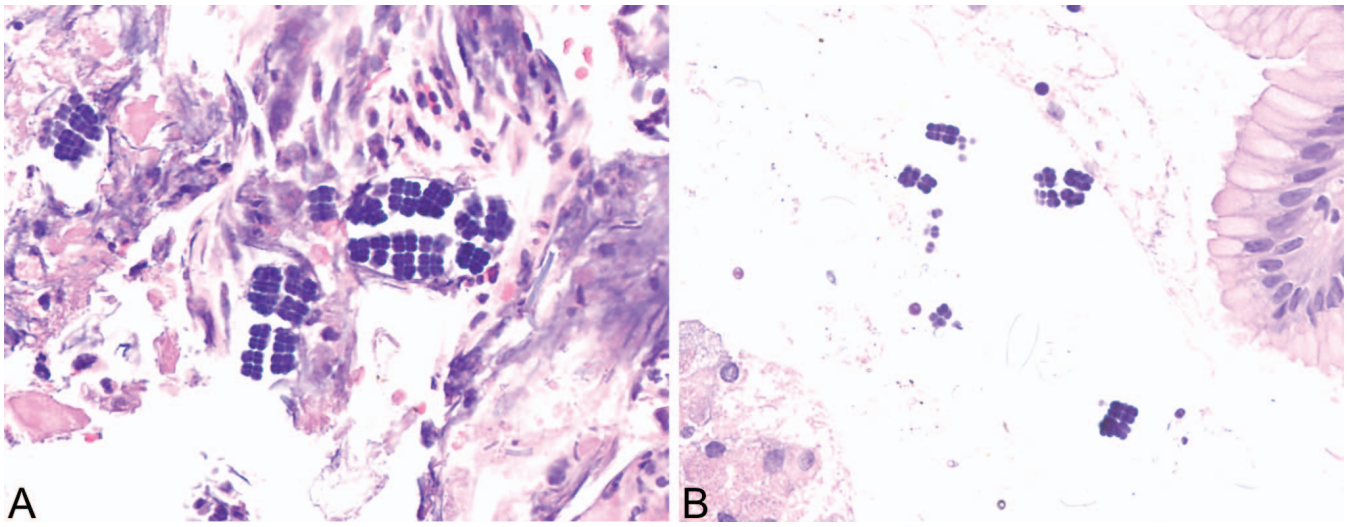
once it is sequenced, the 16S rRNA gene can be compared with validated gene-sequence databases to identify the specific bacterium. In addition, the pyruvate decarboxylase protein is involved in a rare metabolic pathway present in only a few bacterial species, including *S ventriculi*.[5]

These findings were demonstrated by Lam-Himlin et al,[5] who designed 2 sets of primers using sequence data from GenBank to target proximal, mid, and terminal regions of the 16S rRNA gene (AF110272.1) and the pyruvate decarboxylase gene (AF354297.1) of *S ventriculi*. According to the article,[5] *S ventriculi* was identified as *present* only with histologic evidence, a positive polymerase chain reaction result, and sequencing for both regions of pyruvate decarboxylase and at least one region of 16S rRNA.[5] Similarly, polymerase chain reaction and DNA sequencing for the pyruvate decarboxylase gene was used by Sauter et al[11] for positive identification of *S ventriculi*. Both studies successfully provided molecular confirmation in nearly all the histologically suspected cases and provided support for an accurate diagnosis being based on histology alone.

PATHOGENESIS

Sarcina ventriculi is a gram-positive, nonmotile, chemoorganotrophic, anaerobic coccus with an exclusive carbohydrate fermentative metabolism. The main products of carbohydrate fermentation are ethanol, acetaldehyde, carbon dioxide, and hydrogen.[8]

Sarcina ventriculi was first isolated in pure culture from the stomach in 1911, using strict anaerobic techniques.[17] *Sarcina*



A, Sarcina *organisms arranged in tetrad packets in the background of gastric mucin and inflammatory cells in a patient with gastric outlet obstruction secondary to recurrent gastric ulcers. B, Basophilic-stained and cuboid-shaped* Sarcina *organisms adjacent to normal-appearing foveolar gastric mucosa in a patient with diabetic gastroparesis (hematoxylin-eosin, original magnification ×400 [A and B]).*

ventriculi has also been reported in the feces of healthy humans, particularly those with vegetarian diets.[18] The organism can normally be found in the soil and air, where it can survive for years by forming spores at alkaline pH.[1] *Sarcina* species, whose natural habitat is the soil, is probably ingested with soil particles present in the food.[13] Various reports in veterinary literature have implicated the *Sarcina* species in the development of gastric dilatation[19] and death in livestock, cats, and horses.[20–22]

There are conflicting reports on whether this organism is found in healthy human stomachs. Some authors favored this theory,[23] whereas others, such as Canale-Parola,[13] suggest that, although it was originally observed in stomach contents, this organism is not found in the healthy human stomach. Growth of *S ventriculi* takes place in the human stomach because of delayed gastric emptying from pathologic conditions such as diabetic gastroparesis, gastric surgeries, scarring, pyloric stenosis,[13] or an obstructing mass. With these diseases, the acidic pH of the stomach, and the presence of carbohydrates and other growth nutrients contained in food, *S ventriculi* thrives and multiplies rapidly.[13] Only 2 cases (10.5%) had severe complications, such as emphysematous gastritis and peritonitis, and both patients had underlying ulcers. Therefore, Lam-Himlin et al[5] suggested that a preexisting mucosal defect provided the nidus for emphysematous gastritis to develop, rather than direct invasion of *Sarcina* species into the gastric wall. Although the role of *S ventriculi* as the mechanism of mucosal injuries in these cases is not clear, the local accumulation of acetaldehyde and ethanol formed from carbohydrate fermentation by the organism could induce stomach and duodenal injuries, similar to the acetaldehyde-induced mucosal injuries in acute alcohol ingestion. Furthermore, the carbon dioxide production from glucose fermentation and pyruvate metabolism results in abdominal distention in some of these patients.[8]

DIFFERENTIAL DIAGNOSIS

The tetrad or packet-forming arrangement is not unique to *Sarcina* organisms, and the main differential diagnosis upon light microscopy is with *Micrococcus* species, which is also a gram-positive coccus that occurs in tetrads or packets.[13] However, a few features are helpful in differentiating the 2 organisms: histologically, at 0.5 μm, the *Micrococcus* species are considerably smaller than the *Sarcina* bacterium and, unlike the *Sarcina* species, the *Micrococcus* species tends to form tightly packed clusters.[5] In addition, *Micrococcus* bacterium yields positive catalase results,[24] is aerobic, and does not form spores, in contrast to *S ventriculi*, which is catalase-negative, anaerobic, and spore forming.[13]

Other differential diagnoses for *S ventriculi* are *Sarcina maxima* and *Staphylococcus* species. The most helpful feature in light microscopy to differentiate *S ventriculi* from *S maxima* is the thick extracellular layer present on the outer surface wall of *S ventriculi*, which is not observed in *S maxima*. This layer generally measures 150 to 200 nm in thickness and is composed of mostly of cellulose or a closely related compound.[13] The thick layer is the result of the refractile nature of the cell wall, which has caused *S ventriculi* to be mistaken for a fungus or vegetable matter.[10] *Sarcina* bacteria can be differentiated from *Staphylococcus* species, even though both organisms are gram positive, because *Staphylococcus* bacteria, at approximately 1 μm in diameter, is much smaller and is arranged in characteristic grapelike clusters, rather than a tetrad pattern.[25]

TREATMENT AND PROGNOSIS

The recent increase in reports of *Sarcina* organisms supports the observation by Sopha et al[10] underscoring the need to define a standard regimen for its treatment. Current reports indicate successful eradication of the organism, with treatment usually consisting of metronidazole in combination with another antibiotic[8,16] and/or a gastrointestinal agent.[22,26] However, as evident in Table 1, there is no consensus on the type and duration of treatment. Medlicott and Adams[26] outlined a treatment approach based on the clinical status of the patient. If the patient is clinically stable and healthy, they suggested it was reasonable to forego treatment because the organism can occur commensally. If, however, the patient is symptomatic, particularly with prominent dysphagia or substernal burning, treatment with a proton pump inhibitor and a prokinetic was indicated. If

Table 2. Clinical and Histologic Presentation of *Sarcina ventriculi* in the Literature

Characteristics	Cases, No. (%), n = 19
Sex	
F	13 (68.4)
M	6 (31.6)
Age, y	
0–10	2 (10.5)
11–17	4 (21)
18–64	11 (57.9)
≥65	2 (10.5)
Clinical presentation	
Abdominal pain	11 (57.9)
Nausea and vomiting	9 (47.4)
Abdominal distension, diarrhea, melena	3 (15.8) each
Asymptomatic, dysphagia, or hematemesis	2 (10.5) each
Other (weight loss, anorexia, regurgitation, weakness)	6 (31.6)
Delayed gastric emptying	11 (57.9)
Gastrointestinal surgery or gastric outlet obstruction	7 (36.8) each
Gastroparesis	4 (21)
Endoscopy/intraoperative	
Food bezoar	9 (47.4)
Gastric ulcer, gastritis	4 (21) each
Esophagitis	3 (15.8)
Edematous/tight pylorus, gastric perforation, healed/scarred gastric ulcer, mass, necrosis, or normal	2 (10.5) each
Other (hiatal hernia, polyp, duodenal groove, bile, or stricture)	6 (31.6)
Histopathology, in addition to *Sarcina* organisms	
Gastric ulcer	8 (42)
Chronic active gastritis	5 (26.3)
Chronic inactive gastritis	4 (21)
Duodenitis, esophagitis	4 (21) each
Unspecified gastric findings	5 (26.3)
Normal	3 (15.8)
Gastric adenocarcinoma	2 (10.5)
Hyperplastic polyp, intramural air, necrosis, reactive gastropathy	1 (5.3) each
Other concurrent organisms	6 (31.6)
Candida	2 (10.5)
Giardia, Helicobacter pylori, Staphylococcus, or mixed gram-positive and gram-negative flora	1 (5.3) each

there is evidence of associated gastric erosion or similar acute conditions, eradication of the organism with antibiotics was recommended.[26] However, there is little to no data on the efficacy of the various treatment regimens. The treatment provided in the few reports with follow-up data (n = 12; 63%) are listed in Table 1, with subsequent improvement in symptoms and histology,[5,22] and the organism was never reidentified in any of the cases.

CONCLUSIONS

In conclusion, *S ventriculi* is an increasingly common, gram-positive bacterium, seen predominantly in patients with delayed gastric emptying. Presence of *S ventriculi* has been associated with cases of gastric perforation and emphysematous gastritis, and although the pathogenicity of this organism is not entirely certain, its recognition in an endoscopic biopsy raises important diagnostic and therapeutic considerations.[27] Because *Sarcina* organisms are difficult to grow on cultures in general laboratories and molecular methods of confirmation are not available in many parts of the world, histopathologic examination for the classic morphologic features remains a key to the diagnosis until specific microbiologic diagnostic methods become available.

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