ACG CASE REPORTS JOURNAL

CASE REPORT | STOMACH

# Emphysematous Gastritis in a Patient with Untreated Cyclic Vomiting Syndrome

Sara L. Robinson, MD, MS[1], Brett W. Sadowski, MD[2], Christa Eickhoff, MD[3], Edward Mitre, MD[3], and Patrick E. Young, MD, FACP, FACG, FASGE[2]

*[1]Department of Medicine, Division of General Internal Medicine, Walter Reed National Military Medical Center, Bethesda, MD*
*[2]Department of Medicine, Division of Gastroenterology, Walter Reed National Military Medical Center, Bethesda, MD*
*[3]Department of Medicine, Division of Infectious Diseases, Walter Reed National Military Medical Center, Bethesda, MD*

## ABSTRACT

Emphysematous gastritis (EG) is an uncommon and potentially fatal disease characterized by gastric pneumatosis in the setting of infection. While this disease has been described in the literature, it has not previously been identified as a potential complication of cyclic vomiting syndrome. We describe a patient with a history of cyclic vomiting syndrome who presented acutely ill and was found to have radiographic, endoscopic, and histologic evidence of EG. This case illustrates how an untreated functional bowel disorder can lead to severe and potentially fatal complications.

## INTRODUCTION

Emphysematous gastritis (EG) is a rare diagnosis characterized by air in the stomach wall due to infection of the gastric mucosa. While associated with multiple etiologies including malignancy, infarction, and gastric outlet obstruction, it is rarely described in association with retching and vomiting alone.[1] Cyclic vomiting syndrome is a functional gastroduodenal disorder characterized by episodes of nausea and vomiting, and it is typically managed supportively during acute episodes.[2] Prophylactic therapy may also be considered, especially in those with comorbid migraine headaches. This case highlights the importance of recognizing and treating cyclic vomiting syndrome because uncontrolled symptoms can lead to repeated trauma to the stomach mucosa and increase the risk for a potentially lethal infection.

## CASE REPORT

A 62-year old Caucasian woman with a history of chronic migraine headaches, bipolar disorder, and recurrent episodes of severe emesis over several years presented with syncope after one day of severe nausea and vomiting. She reported more than 15 episodes of non-bloody, non-bilious emesis that were associated with nausea and an antecedent migraine, which she treated with sumatriptan. She also reported subjective fever at home. She initially sought medical attention due to a head laceration sustained after experiencing a syncopal event during an episode of retching and vomiting. Computed tomography (CT) of the head demonstrated no acute intracranial pathology.

Her physical exam was notable for mild tachycardia with a heart rate of 103 beats per minute and no other vital sign abnormalities. The physical exam was remarkable for diffuse abdominal tenderness without rebound or peritoneal signs and a 3-cm well-approximated head laceration. Laboratory evaluation was notable for leukocytosis

*ACG Case Rep J* 2018;5:e90. doi:10.14309/crj.2018.90. Published online: December 5, 2018.

Correspondence: Sara L. Robinson, MD, MS, Walter Reed National Military Medical Center, 8901 Rockville Pike, Bethesda, MD 20889 (sara.loftin.robinson@gmail.com).

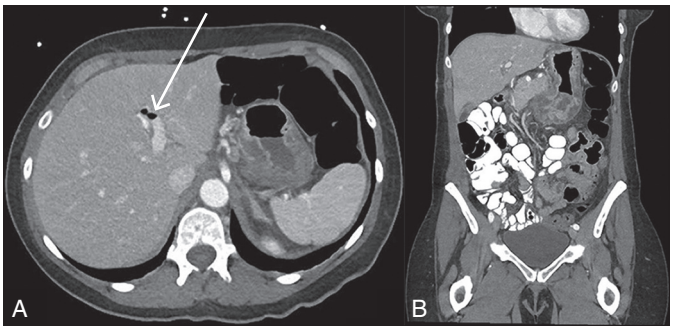


**Figure 1.** Computed tomography imaging of abdomen and pelvis (A) axial and (B) coronal image showing gastric wall thickening and mucosal hyperenhancement with associated portal venous gas and gastric pneumatosis.

(19,000 cells/$\mu$L) with 12% band cells and elevated lactic acid (3.3 mmol/L). Other studies including erythrocyte sedimentation rate, C-reactive protein, and blood and urine cultures were unremarkable. An abdominal CT scan demonstrated circumferential gastric wall thickening and mucosal hyperenhancement, gastric pneumatosis, and portal venous gas concerning for EG (Figure 1). On the basis of these imaging findings, the patient was initially given broad empiric antimicrobial therapy with intravenous ciprofloxacin and metronidazole to cover typical enteric bacteria as well as liposomal amphotericin B in case she had gastrointestinal mucormycosis.[3] An esophagogastroduodenoscopy showed extensive necrotic mucosa in the stomach (Figure 2). Gastric biopsies showed suppurative gastritis with ulceration and necroinflammatory debris (Figure 3) with no fungal elements. No obstructing lesions were found, and the anatomy of the esophagus, stomach, and examined duodenum was normal without malrotation. She was managed conservatively with broad-spectrum antibiotic therapy until de-escalation to ampicillin-sulbactam after tissue cultures grew *Streptococcus mitis* and normal flora. Early surgical consultation recommended non-operative management, given the patient's clinical stability. Over a week of observation, the patient's symptoms resolved, and interval cross-sectional imaging and endoscopy demonstrated marked improvement (Figure 4). At discharge, the patient was started on prophylactic migraine

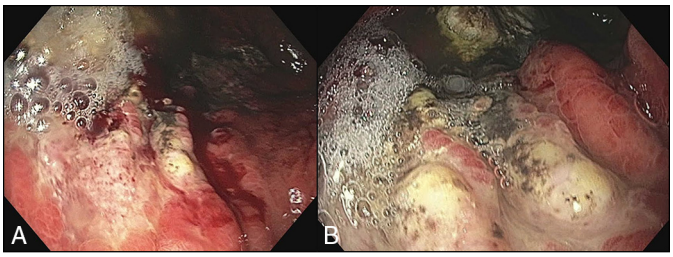


**Figure 2.** Endoscopy showing extensive necrotic tissue in the body of the stomach.

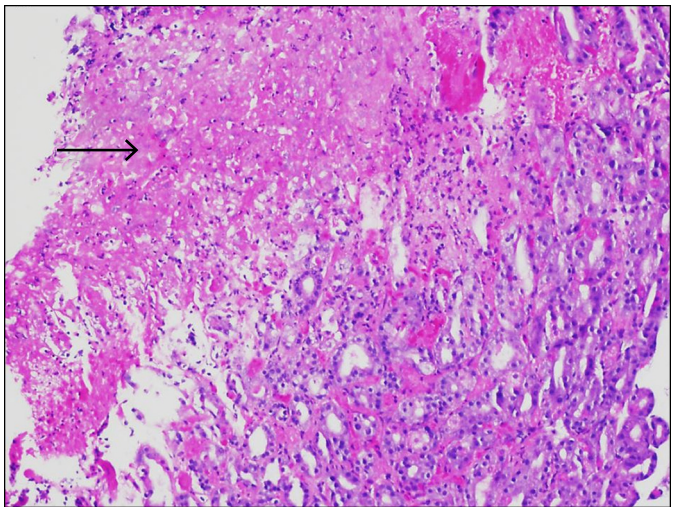

**Figure 3.** Histopathology showing suppurative gastritis with an area of acute inflammation (arrow), fibrin and necroinflammatory debris. No stainable organisms were identified.

medication. She has not had a recurrence of vomiting episodes as of 1 year later.

## DISCUSSION

EG is a highly lethal infectious disease of the gastric mucosa associated with gastric pneumatosis and portal venous gas. Predisposing factors for EG include diseases that damage the integrity of the gastric mucosa and malignancy, caustic ingestion, malrotation, bezoar formation, hernias, abdominal surgery, and nonsteroidal anti-inflammatory drug use. A small number of cases of EG following an episode of vomiting have been reported.[4,5] We suspect that our patient developed EG as a complication of previously unrecognized cyclic vomiting

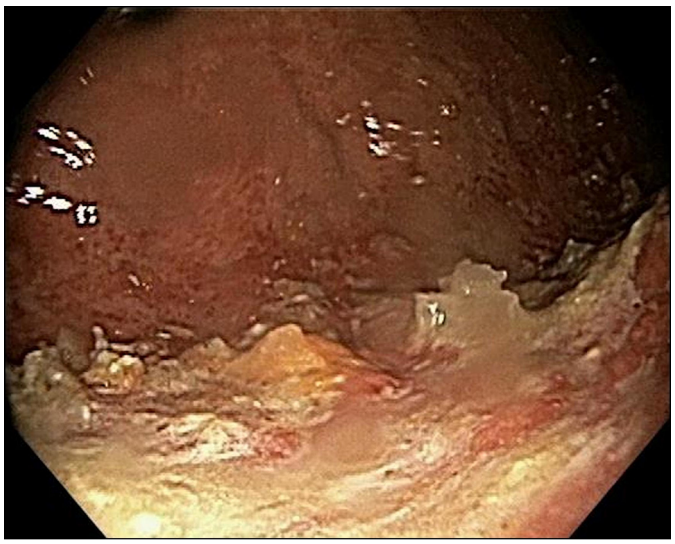

**Figure 4.** Endoscopy on the ninth day of admission showing resolution of necrosis in the body of the stomach.

syndrome (CVS). Patients with EG typically present with severe abdominal pain, nausea, and vomiting. Causative infectious organisms include Gram-positive bacteria, such as *Sarcina* and *Streptococcus* sp., Gram-negative and anaerobic bacteria, as well as fungal infections, especially in the setting of immune compromise.[1,3,6,7]

It is important to distinguish EG from gastric emphysema, which is a noninfectious condition in which air has entered the gastric mucosa due to increased intraluminal pressure. On imaging studies, EG exhibits small pockets of air throughout the stomach wall associated with thickened gastric walls, whereas gastric emphysema often appears as thin, linear lucencies in the gastric mucosa.[8] An additional diagnosis to consider in cases of gastric pneumatosis and portal venous gas is gastric ischemia. In our case, it was felt that infectious EG was the dominant pathophysiology but that the trauma of emesis and retching could have also induced some element of gastric ischemia, which may have contributed to the patient's clinical and histologic findings.[9] Our patient was diagnosed with EG based on the severity and acuity of symptoms, gastric wall thickening, diffuse air in the gastric wall, and severe inflammation observed on endoscopy and histology which responded to antibiotics.

While management of EG has been traditionally centered around surgery, reports of cases caused by vomiting alone have demonstrated the effectiveness of conservative therapy.[1,6] As was seen in this case, aggressive early antimicrobial therapy and supportive care may be considered in clinically stable patients. The mortality rate of EG has been estimated to be as high as 60% in past literature, but in more recent years the mortality rate has declined to 33%. While still a very lethal diagnosis, the improvement in mortality in recent years has been attributed to the increasing trend toward endoscopy and medical management.[10]

CVS is characterized by 3 or more brief acute episodes of intense vomiting separated by asymptomatic periods and has a prevalence of between 3–14% in patients with chronic emesis. CVS patients often have a personal or family history of migraine headaches. Classified as a functional gastrointestinal disorder according to the Rome IV criteria, CVS may be linked to periods of stress, sleep deprivation, or a patient's menstrual cycle.[2] While generally benign, patients affected by this disorder often undergo endoscopic evaluation to rule out alternative causes.[11] Even during acute episodes, patients most frequently have a benign course that responds well to supportive therapy. However, our case illustrates that CVS exacerbations are not always free of complications. The successful use of preventative medical therapy against migraine headaches to prevent CVS attacks has been previously described.[12] The clinical severity and extent of the mucosal damage in this case would support the use of migraine-prevention strategies as was done in this patient, who experienced no recurrence of illness as of 1 year later.

## DISCLOSURES

Author contributions: All authors contributed equally to this manuscript. SL Robinson is the article guarantor.

Financial disclosure: None to report.

Informed consent was obtained for this case report.

Received May 15, 2018; Accepted September 11, 2018

## REFERENCES

1. Matsushima K, Won EJ, Tangel MR, Enomoto LM, Avella DM, Soybel DI. Emphysematous gastritis and gastric emphysema: Similar radiographic findings, distinct clinical entities. *World J Surg*. 2015;39:1008–17.
2. Stanghellini V, Chan FK, Hasler WL, Malagelada JR, Suzuki H, Tack J, Talley NJ. Gastroduodenal disorders. *Gastroenterology*. 2016;150:1380–92.
3. Lee SH, Son YG, Sohn SS, Ryu SW. Successful treatment of invasive gastric mucormycosis in a patient with alcoholic liver cirrhosis: A case report. *Exp Ther Med*. 2014;8:401–4.
4. Al-Jundi W, Shebl A. Emphysematous gastritis: Case report and literature review. *Int J Surg*. 2008;6:e63–e66.
5. Parikh MP, Sherid M, Ganipisetti V, Gopalakrishnan V, Habib M, Tripathi M. Vomiting-induced gastric emphysema and hepatoportal venous gas: A case report and review of the literature. *Case Rep Med*. 2015.
6. Paul M, John S, Menon M, Golewale N, Weiss SL, Murthy UK. Successful medical management of emphysematous gastritis with concomitant portal venous air: A case report. *J Med Case Rep*. 2010;4:140.
7. Al Rasheed MRH, Senseng CG. *Sarcina ventriculi:* Review of the literature. *Arch Pathol Lab Med*. 2016;140:1441–5.
8. Viswanath S, Jain AKC. Emphysematous gastritis: A rare diagnosis with unique presentation. *OA Case Reports*. 2014;3(2):11.
9. Tang S, Daram SR, Wu R, Bhaijee F. Pathogenesis, diagnosis, and management of gastric ischemia. *Clin Gastroenterol Hepatol*. 2014;12:246–52.
10. Watson A, Bul V, Staudacher J, Carroll R, Yazici C. The predictors of mortality and secular changes in management strategies in emphysematous gastritis. *Clin Res Hepatol Gastroenterol*. 2017;41(1):e1–e7.
11. Bhandari S, Venkatesan T. Clinical characteristics, comorbidities and hospital outcomes in hospitalizations with cyclic vomiting syndrome: A nationwide analysis. *Dig Dis Sci*. 2017;62:2035–44.
12. Redon S, Maureau C, Guedj E, Donnet A. Cyclic vomiting syndrome in adults and children: A hypothesis. *Headache*. 2017;57:943–51.