CASE REPORT

Acute phlegmonous gastritis: A case report

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1 INTRODUCTION

Acute phlegmonous gastritis (APG) is a rare and often fatal disease. Symptoms can be non-specific. Patients typically present with abdominal pain, nausea, vomiting, fever, and signs of infection.1 The most common pathogens related to APG described in the literature are the Streptococcus species.1,3 Although the pathogenesis is not completely known, predisposing factors—including mucosal injury, immunocompromised state, chronic alcohol use, underlying gastric malignancy, and a history of gastritis—have been hypothesized as contributing to this condition.2 However, approximately half of patients who develop APG were previously healthy.3 Because of its rarity, best practices for treatment are poorly understood, making management decisions difficult. We present a case of APG affecting an afebrile healthy adult that resolved without complications with conservative management.

2 CASE REPORT

A 44-year-old healthy female presented to the emergency department (ED) with burning epigastric abdominal pain that had worsened in intensity over the previous 2 days, with associated nausea and anorexia. She reported feeling well until 2 days prior, when she began having pain that she initially felt in her midback. This pain migrated to her epigastrium and worsened in severity over the next 36 hours. She felt warm and had chills at home, but she had no documented fever. Despite having severe nausea and anorexia, she had no episodes of vomiting. She denied other symptoms, including chest pain, headache, shortness of breath, cough, rhinorrhea, diarrhea, genitourinary symptoms, hematochezia, and rash. The patient denied any history of trauma.

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or triggering factors, including ingestion of spicy food, alcohol use, prior bouts of gastritis, or recent use of non-steroidal inflammatory drugs. Before the onset of her symptoms, the patient was in good health. Her past medical history included psoriasis (requiring only topical medications) and a remote stress fracture of her right femoral neck that had healed with conservative management. Her history was negative for recent surgeries. Other than a topical corticosteroid for psoriasis, the patient reported taking no home medications or supplements. Social history was negative for tobacco use, illicit drug use, and alcohol abuse. The patient reported being married with 2 children at home, and she was working full time as a physician. She had not traveled recently.

On presentation to the ED, the patient appeared uncomfortable but non-toxic. Her initial vital signs were temperature 98.6°F; heart rate 63 beats per minute; respiratory rate 16 breaths per minute; blood pressure 151/94 mm Hg; and pulse oximetry 99% on room air. The patient was alert and fully oriented. Her physical examination was pertinent for significant tenderness to palpation over the epigastric region of her abdomen, without abdominal rigidity, rebound, or guarding. An initial evaluation, including complete blood count with differential, serum lipase, comprehensive chemistry panel with liver function tests, polymerase chain reaction testing for COVID-19, electrocardiogram, chest radiography, and a bedside point-of-care ultrasound of the abdominal right upper quadrant, was unremarkable with no acute abnormalities. Given the severity and persistence of the patient’s epigastric pain and nausea, computed tomography (CT) imaging of the abdomen and pelvis with intravenous contrast was obtained, revealing mucosal enhancement with significant submucosal edema at the gastric body and edema within the soft tissues along the lesser curvature of the stomach, concerning for APG (Figure 1). Evaluation with upper endoscopy to exclude underlying neoplasm was recommended. After the patient’s case and imaging study were reviewed with the general surgery and gastroenterology physicians at the community hospital where she presented, she was transferred to an ICU at a nearby tertiary center for further care, given the potentially aggressive course of APG and high concern that the patient could acutely decompensate. Broad-spectrum antibiotic therapy with intravenous piperacillin-tazobactam and fluconazole was initiated before transfer; the patient was also started on intravenous fluids and was directed not to take any fluids or food by mouth.

Broad-spectrum antibiotic therapy with intravenous piperacillin-tazobactam and fluconazole—along with pantoprazole—were continued throughout her hospital course in the tertiary care center ICU. She remained afebrile and hemodynamically stable. Her epigastric pain and nausea improved rapidly after initiation of intravenous antibiotic therapy. By the patient’s second hospital day, her epigastric discomfort was minimal, and her nausea had resolved. Repeat CT imaging of the abdomen and pelvis—obtained approximately 48 hours after the patient’s initial imaging study—showed a significant interval decrease in gastric body submucosal edema compared to that seen on her prior examination (Figure 2).

Given her significant clinical and radiographic improvement, the patient was allowed to begin a clear liquid diet, which she tolerated well. She was discharged home with directions to complete a 2-week course of oral amoxicillin-clavulanate and fluconazole, along with oral pantoprazole twice daily. She was scheduled to return for an esophagogastroduodenoscopy (EGD) procedure 2 weeks after hospital discharge rather than undergoing EGD during her hospitalization, given the gastroenterology team’s concern that she was at higher
FIGURE 2  Repeat computed tomography (CT) study of the abdomen and pelvis with intravenous contrast on patient’s second day of hospitalization

risk for EGD-related complications during the acute phase of her illness. During the patient’s posthospital course, her symptoms continued to steadily improve; she was able to advance to her regular full diet within 48 hours after discharge from the hospital, without recurrence of abdominal pain or nausea. An EGD study performed 2 weeks after hospital discharge found a single localized 3 millimeter erosion in the prepyloric region of the stomach but no other abnormalities. Immuno-histochemistry for Helicobacter pylori was negative for microorganisms. At both 6-month and 1-year follow-up, the patient was healthy and denied any recurrent episodes of epigastric pain, back pain, nausea, or fever. She had not required any acute medical care or hospitalizations in the year after her hospitalization for APG.

3  |  DISCUSSION

APG is a rare and often fatal condition characterized by invasive bacterial infection of the gastric wall. An average of one case annually has been reported in the literature over the past century.1–3 Although advances in antibiotic therapy have decreased the mortality rate, several patients with APG have been diagnosed after gastrectomy or on autopsy. Prior idiopathic cases have been reported.

APG has been reported in all age groups, most commonly in adults in their 50s to 70s, with a 2:1 male-to-female ratio.1 Although the etiology is not fully understood, bacterial invasion of the gastric wall into the submucosa seems to be the primary pathophysiologic mechanism. Streptococcus species have been determined to be the causative agent in the majority of cases, although Staphylococcus species, Escherichia coli, Pneumococcus, Enterococcus, Candida, and other organisms indigenous to the gastrointestinal tract have been reported.1–3 The presence of gastric ulcers, chronic gastritis, and underlying gastric malignancy has been implicated as risk factors for this disease.1,2 Chronic alcohol abuse, immunocompromised states, malnutrition, and advanced age have also been reported as associated factors.1–3 Idiopathic cases in previously healthy individuals have been described among the relatively small number of cases reported in the literature. Early diagnosis and prompt treatment are critical to achieving positive outcomes.

Typical clinical manifestations of APG include acute abdominal pain, fever, chills, nausea, and vomiting.1–3 Hematemesis and/or purulent emesis may occur in severe cases.3 Owing to the rare nature of APG and its non-specific clinical features, early diagnosis can be difficult. The differential diagnosis for APG is quite broad and includes conditions that occur much more commonly: biliary disease, pancreatitis, infectious gastroenteritis, peptic ulcer disease, non-phlegmonous gastritis, esophagitis, esophageal or gastric perforation, and referred gastrointestinal symptoms due to cardiac pathology (ie, myocardial infarction, pericarditis, myocarditis). For patients with significant acute epigastric pain and infectious symptoms (fever, chills, nausea, vomiting) in whom the more common aforementioned diagnoses have been excluded, APG (and CT imaging to evaluate for the disorder) should be considered. This condition can develop and progress quickly, often with resulting gastric wall necrosis, perforation, and peritonitis.1–3 The extremely high mortality rate associated with APG may be due in part to the fact that the diagnosis is often delayed or missed. Before the advent of antibiotics, the mortality rate exceeded 90% for this condition; with antibiotic therapy, the mortality rate has decreased to around 48%.3 For patients in whom antibiotic therapy is ineffective, surgical intervention is required.

Abdominal CT is helpful for both prompt diagnosis of APG and detecting associated complications. Characteristic CT findings of this condition include thickening of the gastric wall, low-intensity areas within the gastric wall (indicative of abscess), and gas accumulation.1,3

4  |  CONCLUSION

APG is a rare condition associated with high morbidity and mortality rates. Although this condition most commonly affects patients with underlying chronic conditions, it can also affect healthy individuals.3 Although fever has been a key presenting feature in previously reported cases of APG, the case we report involved a healthy female presenting with severe epigastric pain, nausea, and anorexia, without documented fever (although subjective fevers at home were reported), leukocytosis, or other signs of infection. A high index of suspicion is required to make this uncommon diagnosis in a timely manner. Because
of the high mortality rate for this disorder, early recognition and treatment are key to achieving an optimal prognosis for affected patients. Despite prior case reports of APG requiring operative management, our patient’s symptoms improved rapidly with conservative management, which included initiation of broad-spectrum parenteral antibiotics early in her course.

CONFLICT OF INTEREST
The authors declare no conflict of interest.

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