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Emphysematous gastritis associated with ulcerative esophagitis

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Abstract. – Emphysematous gastritis is a rare form of gastritis characterized by gaseous dissection of the stomach wall usually secondary to invasion by gas forming organisms, corrosive ingestion, trauma, or gastric infarction. We report a unique case of emphysematous gastritis secondary to ulcerative esophagitis without an underlying history of trauma, ischemia, or ingestion of corrosive agents. The patient responded well to conservative management. Emphysematous gastritis is a deadly infectious disease. Early recognition and management are crucial as there is no specific approach to treatment despite advances in medical science.

Key Words:

Gastritis, Gastric emphysema, Emphysematous gastritis, Ulcerative esophagitis, Therapy.

Case Report

A 66-year-old Caucasian male with a past medical history of chronic gastroesophageal reflux disease and Type II diabetes mellitus presented to the Emergency Room with worsening heartburn associated with diffuse epigastric pain, nausea, brown-colored emesis, and non-bloody diarrhea. The patient denied fevers, chills, weight loss, or other gastrointestinal symptoms. His abdominal examination was consistent with diffuse epigastric tenderness without distention or peritoneal signs. Bowel sounds were audible. His liver and spleen were not palpable. Laboratory evaluation was significant for a white blood cell count of 19,000 cells per cubic millimeter. A diagnosis of emphysematous gastritis was made with computed tomography scan (CT) of the abdomen, which showed a linear gas pattern along the lesser curvature of the stomach and mural thickening of the distal esophagus (Figures 1 and 2). An esophagogastroduodenoscopy (EGD) revealed circumferential ulcerative esophagitis involving the distal third of the esophagus (Figure 3). The patient was managed conservatively with intravenous fluids, intravenous ciprofloxacin, metronidazole, and proton-pump inhibitor (PPI) therapy. Blood cultures remained negative for any pathological organism. The patient's symptoms began to resolve and his clinical condition improved. On hospital day 5 the patient was discharged home on twice daily oral PPI therapy. A repeat EGD showed complete healing of the esophagitis. The patient was well on a return visit six weeks later.

Discusssion

The presence of air in the wall of hollow organs is an unusual finding, and the stomach is the least common site for intramural gas[1]. Air in the stomach wall has been described in the literature as gastric emphysema (or gastric pneumatosis) and emphysematous gastritis. In gastric emphysema, as a result of disruption of gastric mucosa, air dissects into the stomach wall either from the mediastinum, peritoneum, or esophageal lumen. On the other hand, emphysematous gastritis is a lethal variant caused by invasion of the gastric wall by gas forming organisms and carries a very poor prognosis. Emphysematous gastritis was first described in 1889 by Frankel, who strongly believed infection to be its etiology[2]. Its radiographic appearance was first described by Weens in 1946[3].

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The stomach wall is well protected from bacterial invasion due to its abundant blood supply, acidic pH, and tight junctions connecting adjacent cells[4]. Four theories have been proposed for the development of intramural gastric gas. Bacterial theory indicates that gas forming organism including *Clostridium welchii, Escherichia coli, Streptococcus, Bacillus subtilis*, and *Bacillus proteus*, invade the gastric mucosa and form gas in the stomach wall. The prognosis is usually fatal despite aggressive therapy with broad spectrum antibiotics and surgical intervention. Mechanical theory suggests that air enters the gastric wall due to an increase in intramural pressure secondary to intestinal obstruction or an endoscopic procedure. According to the mucosal damage theory conditions such as a gastric ulcer cause mucosal disruption that results in air entering the stomach wall. The pulmonary disease theory suggests that patients with longstanding chronic obstructive pulmonary disease (COLD) could rupture their emphysematous bulla resulting in air entry the mediastinum with resultant dissection along the great vessels to the retroperitoneum[5].

In our case, breach of the esophageal mucosa secondary to ulcerative esophagitis appeared to be the primary mode for the development of emphysematous gastritis. Current evidence suggests that corrosive ingestion (37%) and alcohol abuse (22%) are the most common risk factors for the development of emphysematous gastritis. Systemic immunocompromised conditions, use of immunosuppressive agents, and diabetes mellitus can also be risk factors for the development of intramural gas. Other risk factors include recent abdominal surgery, gastroenteritis, and non-steroidal anti-inflammatory drug (NSAID) use[6]. An earlier case of emphysematous gastritis was associated with excessive ingestion of carbonated beverages such as soft drinks[7]. In addition to the gas forming bacteria,

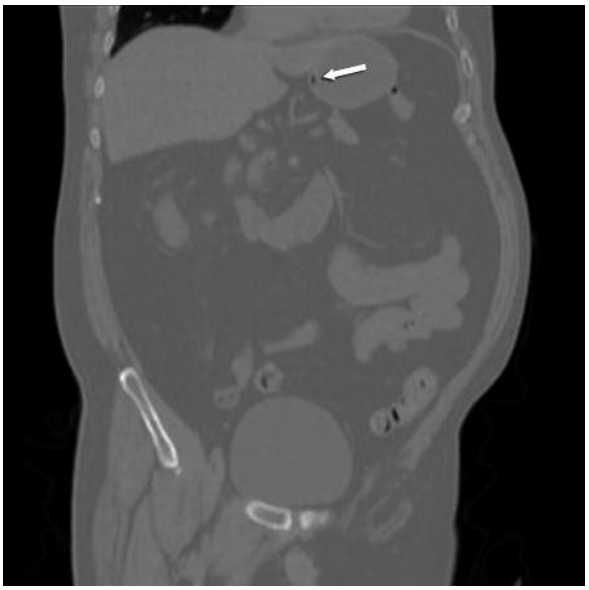

Figure 2. There is fluid distension of the stomach with minimal mural thickening and air bubbles visible.

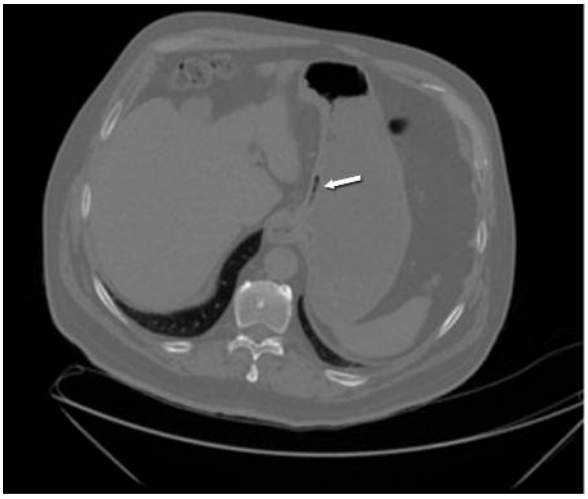

Figure 1. There is fluid distension of the stomach with linear gas noted along the lesser curvature.

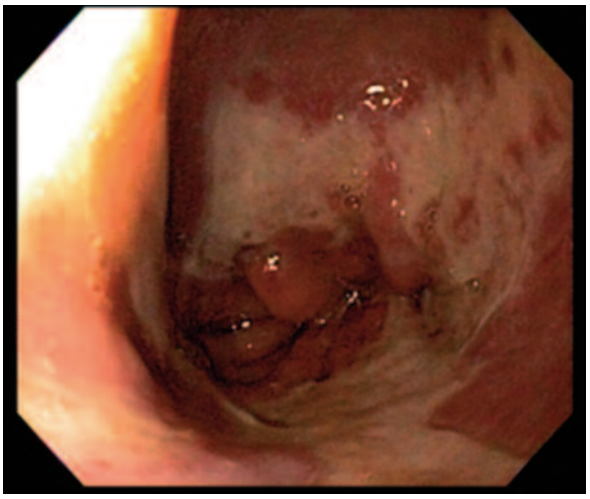

Figure 3. Esophagogastroduodenoscopy showing ulcerative esophagitis involving the distal third of the esophagus.

chiefly coliform and *Clostridium welchii*, other organisms that have been implicated in the development of emphysematous gastritis include *Candida, Mucormycosis*[8].

Clinically, emphysematous gastritis can present as an acute abdomen with symptoms that include abdominal pain, nausea, vomiting, diarrhea, or constipation and often haematemesis. More subtle presentations can be seen in patients with diabetes mellitus, renal failure, or systemic immunocompromised conditions[4].

The diagnosis of emphysematous gastritis can be made through clinical presentation and characteristic findings on imaging procedures. Two radiological patterns have been described for intramural gas in the stomach. Linear lucency along the greater curvature is present in gastric emphysema, and a cystic mottled pattern is usually associated with emphysematous gastritis. However, these patterns are not sufficiently specific to distinguish between these two clinical conditions. The CT scan is not only more sensitive and specific but also describes the exact location of gas in the abdominal cavity[5]. Characteristic findings for emphysematous gastritis on the CT include thickened gastric mucosal fold with cystic pockets of air in the gastric wall, pneumoperitoneum, and air in the portal vein[1]. Further, EGD has been used as a diagnostic procedure, but its sensitivity has not yet been established. Endoscopic findings consist of erythematous, erosive, necrotic mucosa and a thickened gastric mucosal wall[5]. Emphysematous gastritis can have a grave prognosis with an associated mortality rate as high as 60%. Other non-lethal complications include gastric stricture rates of up to 25% [2]. Early recognition, use of broad spectrum antibiotics, and conservative measures are key factors in managing this often fatal disease. There is no specific approach that has been established for management to date. Surgery is reserved for gastric infarction, development of peritonitis, perforation, or failure of medical management[1,5].

Conclusion

To our knowledge, our case of emphysematous gastritis secondary to ulcerative esophagitis without the underlying predisposing factors of infection, trauma, ischemia, or infarction is unique. We believe that our patient's ulcerative esophagitis resulted in mucosal breach and extension of air into the stomach. The patient was treated successfully with medical management with no complications on follow up.

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