Emphysematous Gastritis with Air in Portal Venous System

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Abstract
Emphysematous gastritis is a rare but severe form of phlegmonous gastritis caused by gastric mucosal disruption and infection of stomach wall by gas-forming bacteria. Ingestion of corrosive substances is the most common predisposing factor, followed by alcohol abuse, abdominal surgery, diabetes and immunosuppression. Patients usually present with abdominal pain, vomiting, diarrhea, constipation and/or gastro-intestinal hemorrhage. Characteristic radiological findings include presence of gas in the gastric wall. Management of this condition includes broad-spectrum antibiotics and supportive therapy. Outcome of emphysematous gastritis is frequently fatal due to septic shock and multi-organ failure. We report a case of a 65 years old male who presented with fever and upper abdominal tenderness. He had history of uncontrolled diabetes and chronic alcohol intake. Radiological investigations revealed air within the gastric wall, portal vein, liver and spleen. Despite initial improvement with conservative management, patient succumbed due to sepsis and shock.

Introduction
Emphysematous gastritis (EG) is a rare disease characterized by the presence of gas in the gastric wall. Common predisposing factors include ingestion of corrosive substances, alcohol abuse, abdominal surgery, diabetes and immunosuppression.¹⁻³ Common presenting symptoms include abdominal pain, vomiting, diarrhea/constipation and gastro-intestinal hemorrhage. Management with broad-spectrum antibiotics remains the mainstay of initial therapy. We report a case of a 65 years old male having uncontrolled diabetes and history of chronic alcohol intake who presented with fever and upper abdominal tenderness and was diagnosed to have EG.

Case Report
A 65 years old male presented with history of high grade fever since 2 days. He had been suffering from type 2 diabetes mellitus since 3 years (recently uncontrolled). There was history of significant amount (> 100 gm/day) of alcohol intake almost daily since last 20 years until recently. There was no h/o pain abdomen, gastro-intestinal bleed, or jaundice. There was no h/o corrosive ingestion, recent gastroenteritis or abdominal surgery. Examination revealed abdominal distension, epigastric tenderness and decreased bowel sounds. His blood pressure was 100/70 mm Hg, pulse rate-100/min, respiratory rate-18/min and temperature-100.2° F. Investigations revealed: Hemoglobin-10.1 g/dl, Total leucocyte count-2.9 x10³/mm³, platelets-80,000/mm³, urea-100 mg/dl, creatinine-4.1 mg/dl, Aspartate Transaminase-758 IU/ml, Alanine Transaminase-275 IU/ml. Amylase/lipase were within normal limits. Abdominal ultrasonography was normal except multiple echogenic foci with dirty acoustic shadowing in both lobes of liver s/o air in portal vein (PV) radicals. CT abdomen revealed specks of air in dependant part of stomach wall, along with thickening of proximal duodenum with air in its wall (Figure 1). Specks of air were also seen in PV, Superior mesenteric vein (SMV) and liver parenchyma (likely to be in PV radicals). During the hospital stay, patient developed upper gastro-intestinal (UGI) bleed. UGI endoscopy revealed large superficial ulcers in distal gastric body and antrum. Patient was managed conservatively with broad spectrum antibiotics and other conservative measures. Despite initial improvement, patient succumbed within one week due to overwhelming sepsis and hypotension.

Discussion
EG is an extremely rare form of acute phlegmonous gastritis caused by a diffuse infection of stomach wall by gas-forming bacteria. The differential diagnosis includes gastric emphysema occurring due to invasion of gas (barotrauma) into gastric wall, as a result of acute occlusion of gastric emptying or complications following endoscopic procedures.

The first review on EG by Moosvi et al¹ included 27 cases derived from the literature from 1889 until 1990. Another review by Tsuan-Hao et al⁴ described 15 cases from 1990 to 2005. A further of about 10 case reports have...
been published in world literature since then, while none has been reported from India.

Under physiological conditions, stomach wall is well protected from bacterial infection by the close connection between cells, an acid pH and good blood supply. Factors damaging these protective mechanisms leading to EG are: ingestion of corrosives (37%), overindulgence in alcohol (21%) and abdominal surgery/gastroenteritis in about 15% cases. In our patient, Bacillus subtilis, Bacteroides sp., and Clostridium perfringens. In addition, diabetics may be prone to develop EG due to intestinal circulatory disturbances due to diabetic microangiopathy and predisposition to microbial infection due to hyperglycaemia. Rheumatic diseases, treatment with corticosteroids/ cytotoxins, and peritoneal dialysis are other predisposing factors. Altered gastric mucosa due to various insults is invaded by gas-forming bacteria, possibly due to local spread of infection or hematogenous dissemination from a remote focus such as otitis. Most frequently isolated microorganisms are streptococci, enterobacteriaceae, Escherichia coli, Pseudomonas aeruginosa, Clostridium perfringens and Staphylococcus aureus; rarely, Klebsiella pneumoniae, Candida albicans, Proteus sp., Bacteroides sp., Bacillus subtilis and other clostridia. In our patient, diabetes mellitus and heavy amount of alcohol ingestion were the factors probably compromising protective function of stomach wall leading to EG.

Generally, clinical presentation is dramatic. Most frequent symptoms reported are abdominal pain, nausea and vomiting, diarrhea/constipation and hemorrhage from digestive tract. The pathognomic sign is vomiting of necrotic mucosa in the shape of gastric wall cast. In the further course, an acute abdomen usually develops. In diabetics, immuno-compromised or renal failure patients, clinical picture may be less dramatic. X-ray abdomen in patients with EG reveals thickened gastric wall, stratified due to presence of gas. Gas in the wall is in form of irregular bubbles or spots and remains in place despite changing body position or absorption through the gastric tube. In contrast, in gastric emphysema, a more linear distribution of gas in gastric wall is characteristic. USG finding in patients with EG reveal thickened mucosal folds and gas in gastric wall as longitudinal hyperechoic densifications. USG also allows the diagnosis of a pneumoperitoneum in suspected GI tract perforation and gas in the origin of PV. Computed tomography (CT) offers better sensitivity and specificity in confirming the diagnosis. It allows the detection of smaller quantities of gas and visualizes the thickened gastroplication more precisely. It also confirms a possible presence of gas in the origin of the PV, a possible retroperitoneal gas dissection, abscesses or gas accumulation outside GI lumen. Endoscopy in EG reveals erythematous, erosive and hemorrhagic-inflammatory, at times already necrotic, changes of gastric mucosa.

Our patient presented with fever and epigastric tenderness. Bilio-pancreatic disease was ruled out with biochemical tests and ultrasound. Because of the presence of air in portal vein seen on ultrasound and presence of underlying risk factors, possibility of EG was kept and CT abdomen was done which confirmed the diagnosis.

The outcome of EG is frequently fatal due to septic shock and multiorgan failure. The acute phase requires empirical parenteral antibiotic therapy with a broad-spectrum antibiotics. Stomach resection is usually not indicated in the acute phase as active infection slows down or prevents wound healing. Possible complications are leakage of the anastomosis, occurrence of fistulae and strictures. Despite meticulous treatment, prognosis for EG patients is poor. Mortality rate varies between 60%-100 %. Survivors often have gastrenteritis (up to 21%) requiring surgical treatment, and complications in the form of fistulae may occur. Our patient was managed conservatively with broad spectrum antibiotics and other conservative measures. Despite initial improvement, patient succumbed due to sepsis and shock. He could not be subjected to surgery because of poor medical condition.

Conclusion

The possibility of EG should be considered in patients presenting with upper gastro-intestinal symptoms, especially when underlying risk-factors are present so that prompt management can be initiated.

References