BACKGROUND

Diabetic patients are prone to several bacterial infections and among them emphysematous ones are the most fulminant and dreaded. It is not unusual for one to encounter a case of emphysematous cholecystitis or pyelonephritis in individuals with poor glycemic control. Other than for Helicobacter pylori, the harsh acidic milieu of the stomach is an unfavorable environment for bacterial replication. Also, the good vascular supply of the stomach does not predispose to ischemic insults. We present here a rare case of emphysematous gastritis in a young diabetic female. There are about 60 case reports in the literature for this rare entity with diabetes as the most common facilitating condition. There is usually an insult which leads to mucosal injury (alcohol abuse, corrosive ingestion, NSAID drugs) or ischemia (hypotension, atherosclerosis, vasopressors).

CASE SUMMARY

A 24-year-old AA female with PMH of diabetes, exocrine pancreatic insufficiency, and severe malnutrition presented to the ER with severe upper abdominal pain for 3 days associated with nausea & vomiting. She had been receiving treatment with fluconazole and omeprazole following a recent hospitalization, when esophageal candidiasis was diagnosed. On exam there was severe tenderness in epigastrium and left hypochondrium. Laboratory findings were significant for neutrophilic leukocytosis (WBC: 30,000/cu.mm). A CT abdomen revealed moderate to severe gastric wall thickening with intramural air. An upper endoscopy (EGD) showed severe mucosal changes with necrosis in the gastric body and antrum, with a biopsy showing clusters of intense inflammation, bacteria, and pigment with no identifiable gastric mucosa. She was treated with cefepime and metronidazole and was kept NPO with TPN for nutrition. She responded well to treatment; her repeat CT done 6 days later showed resolution of the gastric wall thickening with no gas. Enteral feedings were subsequently resumed, with continued improvement.

DISCUSSION

Given the inherently hostile environment of the stomach to most bacterial species, it is not surprising to see that additional factors must be present to lower its resistance to infection. Apart from the already stated ones, acid suppression is also a major aide. One case report identified stasis as a risk factor. In 60% of reported cases, no organism was identified, and of the remaining, half were polymicrobial. Gram positive bacteria were more frequent than gram negative, with the most common being: Streptococcus, E.coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, Enterococcus spp., and Staph spp.

Candida spp. were implicated in a fair number of cases as well (42%)(2,3,4,5). Sarcina ventriculi is also uniquely associated with EG(6). In the older literature a mortality as high as 60% has been reported(2). In our patient, her poor glycemic control probably played a major role, and coupled with proton-pump inhibitors, protein calorie malnutrition and gastroparesis, a perfect environment was created for such a catastrophic infection. Though no microbiological diagnosis could be made, with early recognition and prompt institution of broad-spectrum antibiotics, the patient made a full recovery.

REFERENCES