GASTRIC PNEUMATOSIS AS A MANIFESTATION OF ISCHEMIC INFARCTION OF THE STOMACH

Report of a case and review of the literature

Bassam ABBOUD1, Wassim MCHAYLEH1, Ghassan SLEILATY1, César YAGHI2

Abstract • Background: Gastric pneumatosis is a very rare entity, and of the hollow viscera, the stomach is the least often reported site for intramural gas.

Objective: To describe a patient with gas in the gastric wall associated with pneumoperitoneum and diffuse ischemic changes of the small and large bowels.

Patient: Old man with past history of hemodialysis for renal failure and a diffuse vascular atherosclerosis presenting with an acute abdomen. The CT scan of the abdomen revealed a gastric pneumatosis with pneumoperitoneum and complete thrombosis of the coeliac trunk. Laparotomy showed gastric infarction with peritonitis and diffuse changes through the gastrointestinal tract. A therapeutic abstention was decided in agreement with the family.

Results: The patient developed septic shock under broad spectrum antibiotic coverage and died after 12 hours of multiple organs failure.

Conclusion: Surgeons, internists and gastroenterologists should be aware of the possibility of gastric pneumatosis in some cases. Underlying disease are important to clarify in order to apply the adequate treatment.

Introduction

Gastric pneumatosis is defined by the presence of gas in the gastric wall. This is a rare entity, and of the hollow viscera, the stomach is the least often reported site for intramural gas [1-9]. Although uncommon, the endoscopic, histologic [1], radiologic [8], and percutaneous ultrasound [4, 10] findings of this condition are well described. In the literature, cases of gastric pneumatosis secondary to diffuse intestinal hypoperfusion are extremely rare.

We report herein a case of intramural gas in the gastric wall with pneumoperitoneum and diffuse ischemic changes of the small and large bowels and review of the literature.

Case

Mr. A. B., an 85-year-old white man, nondiabetic, with a 12 years history of renal failure treated with hemodialysis three times weekly, presented with an episode of abdominal pain of 3 days duration with nausea and fatigue. The patient had a one month history of abdominal discomfort and nausea especially postprandial.

Physical examination revealed an elderly male with temperature 35°C, heart rate 98/mn, respirations 18/mn, blood pressure 110/60 mmHg. Physical exam revealed tenderness, guarding and rebound tenderness on abdominal examination. There was no evidence of organomegaly. Rectal examination was normal and stool occult blood was negative.

Complete blood count demonstrated a white blood count of 9,200, hemoglobin 14.1 g/dl, hematocrit 42.2%, WBC 9,200 with 67% neutrophils, 2% lymphocytes, 11% monocytes, 10% eosinophils, and 1% basophils. Platelets were normal at 270,000.

Continuous intravenous broad spectrum antibiotic coverage and died after 12 hours of multiorgan failure.
cell count of 37,000/mm³, with a differential of 83% polymorphonuclear cells. Hemoglobin was 12 g/dl, and the platelet count was 245,000/mm³. Sodium was 142 meq/l, potassium 5.7 meq/l, bicarbonate was 12 meq/l. Liver enzymes, albumin, and the remaining serum chemistries were unremarkable. A contrast enhanced CT scan of the abdomen (Fig. 1) showed a gastric pneumatosis with pneumoperitoneum, associated with complete thrombosis of the coeliac trunk. No other abnormalities were found in the CT scan especially in the small and large bowels.

The patient was admitted to the medicine intensive care unit (MICU), where an upper endoscopy was performed and showed a gastric necrosis of the lesser curvature and diffuse erythema and swelling of the remaining gastric mucosa. In agreement with the family, an emergency surgery was decided because of the deterioration despite optimal medical treatment, the involvement of a large portion of the stomach and suspicion of gastric infarction and perforation. During the intervention we found purulent liquid around the stomach; a specimen was cultured. The presence of air in the gastric wall is a very rare condition. In children, this is associated with pyloric stenosis, gastric malrotation, annular pancreas, cardiac surgery and incorrect positioning of feeding catheters [11-13]. In adults, instrumentation-related injury, gastric outlet obstruction by gastric, duodenal or pancreatic malignancies, and bowel ischemia or infarction account for the majority of cases [7, 13].

Four theories have been proposed to explain the development of gas within gastric wall [13]. In the bacterial theory, gas-forming aerobic colonic bacilli generate the intramucosal gas seen in emphysematous gastritis [14]. In the mechanical theory, air is thought to enter the gastric wall due to increased mural pressure, possibly caused by air insufflation during endoscopy or intestinal obstruction [13]. In the mucosal damage theory, air enters the gastric wall through disrupted mucosa [10, 13]. This may account for gastric air associated with a penetrating gastric ulcer. In the pulmonary disease theory, alveolar air dissecting down the mediastinum into the gastric wall in patients with severe asthma or emphysema results in the presence of gastric wall air [13].

Historically, gas within the stomach wall has been categorized by most authors under three headings: (1) gastric emphysema, (2) emphysematous gastritis, (3) pneumatosis cystoides intestinalis.

**Gastric emphysema** defines situations where gas collects in the gastric wall without associated infection [1, 7]. This condition has been reported secondary to increased luminal pressure mainly related to gastric outlet syndrome [2] as a carcinoma of the stomach, an intestinal volvulus, small bowel obstruction, forceful vomiting and gallstones. Bowel subjected to distension due to obstruction can go through several stages prior to transmural perforation. In 1880 Heschl described this process as diastatic rupture. This condition is usually self resolving and has a benign clinical picture. Mortality of gastric emphysema is less than 30% than other cases [8].

**Emphysematous gastritis** [15-33] is an infectious gastritis. It is characterized by the presence of gas within the wall of the stomach and associated with systemic toxicity. This is a rare condition, with only 12 well documented cases by 1982, and only 42 cases described in the English literature by 2003. In this situation, the gastric folds are frequently abnormal because of the primary infectious process involving the gastric mucosa. The patient is invariably toxic, with sudden onset of chills, fever, abdominal pain, bloody fowl smelling emesis. It can mimic an acute abdomen. Laboratory findings include severe leucocytosis and metabolic acidosis; alkaline phosphatase may be high due to sepsis. Amylase can be increased in some cases of gastric infarction. Factors that may predispose an individual to this disorder include ingestion of caustic or corrosive substances, alcohol intoxication, ischemia and/or infarction, previous gastrointestinal surgery and parasites such as strongyloides; other predisposing factors include diabetes and immunosuppression. Causative infectious agents include: *E. Coli*, *Enterobacter*, *Clostridium* sp., *Pseudomonas aeruginosa*, *Bacillus* sp., *Aerobacter aerogenes* and *Staphylococcus aureus* [23]. The reported overall mortality ranges from 60 to 80% compared to less than 30% in cases not associated with infectious etiology [1, 15, 33]. Therapy in these
cases consists in antibiotics covering gram-negative and anaerobic microorganisms, on the basis of gastric aspirations and blood cultures, as well as vigorous support of fluid, electrolytes, and acid-base homeostasis. Surgery should be deferred if the patient’s condition stabilizes under adequate therapy; it cannot be recommended in the acute phase. Indication for emergency surgery includes deterioration despite optimal medical treatment, the involvement of a large portion of the stomach, the presence of peritonitis and gastric infarction. Specimen should be cultured and might be helpful in the postoperative management [15]. In our present case, ischemia and infarction of the wall of the stomach led to infection and the presence of air in the gastric wall.

**Pneumatosis cystoides intestinalis**: This entity describes the collection of gas in the walls of the gastrointestinal tract. Most cases involve the terminal ileum, cecum, splenic flexure, and descending colon in that order. The gastric wall is the least common site. The etiologic factors of this situation are neoplastic, infectious, chemical, and mechanical [2]. Clinical presentation of the case is variable and may include signs of systemic toxicity especially for the infectious situations. Treatment can be either supportive or invasive [1].

Classification of these entities is very unclear, but is essential to allow physicians to choose investigations, treat and prognosticate on the basis of shared information about such cases. Finally, gastric pneumatosis is not a disease, it’s a finding that should be investigated based on the patient’s clinical situation, and his past and presenting history.

The diagnosis of gastric wall air is usually made radiographically. Two radiological patterns of gastric intramural air have been described. The linear lucency pattern is usually associated with gastric emphysema and the cystic, mottled pattern is usually associated with emphysematous gastritis, a much more serious condition. However, these patterns are not specific enough to distinguish between these two clinical entities. Computerized tomography is the test of choice because it evaluates the entire abdominal cavity [8, 32-34]. Radiographically, computed tomography is the best diagnosis procedure allowing to show numerous mottled gas bubbles (in emphysematous gastritis) mainly among the greater curvature of a thickened gastric wall which differs from the radiographic findings of non-infectious gastric pneumatosis [32]. Endoscopic findings in patients with gastric emphysema or emphysematous gastritis have been described. These include submucosal gas bubbles, necroinflammatory changes, and erosions; in some cases, the mucosa appears normal. Endoscopic biopsy may reveal numerous empty spaces in the lamina propria [13, 15, 33]. Endoscopic ultrasonography [13] clearly demonstrates the presence of linear band of air in the submucosal layer. Although the presence of air in the gastric wall can be easily diagnosed by non-invasive tests such as ultrasonography or computerized tomography, endoscopic ultrasonography allows better visualization of the gastric wall and is useful in differentiating between gastric emphysema and emphysematous gastritis.

Cases of gastric pneumatosis associated with gastric ischemia and/or infarction are described as case reports in the literature, but there are no documented cases of gastric pneumatosis associated with intestinal hypoperfusion. The case we are reporting may be the first case described.

**CONCLUSION**

Surgeons, internists and gastroenterologists should be aware of the possibility of gastric pneumatosis in some cases. Underlying disease are important to clarify in order to apply the adequate treatment.

**REFERENCES**

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