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CASE REPORT

A case report of emphysematous gastritis in a diabetic patient: favorable outcome with conservative measures

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Emphysematous gastritis (EG) is a rare cause of abdominal pain, which should be differentiated from gastric emphysema. It is hypothesized to result from air-producing microorganisms in patients with underlying predisposing factors. Because of the non-specific presentation of EG, it is diagnosed radiographically. CT scan is the diagnostic modality of choice that typically reveals irregular, mottled appearance of the air in the thickened gastric wall and in the portal vein in the liver. We report a rare case of EG in a male with a history of diabetes mellitus who presented to the emergency department with diarrhea, nausea, vomiting, and epigastric pain. On examination, he was hypotensive and had mild tenderness in the epigastrium. Laboratory tests revealed leukocytosis, elevated lactate, anion gap metabolic acidosis, and acute kidney injury. A non-contrast CT abdomen revealed findings consistent with EG. Even though mortality rate in access of 60% have been reported without prompt surgical intervention in EG, recent literature suggests favorable prognosis with conservative measures in patients without an overt surgical indication. Our patient was also managed conservatively with IV antibiotics and gradual advancement of diet and had complete resolution of symptoms over the ensuing few days. The factors that correlate with a poor prognosis include elevated serum lactate, serum creatinine, and concomitant pneumatosis in small bowel and colon.

Keywords: *emphysematous gastritis*; *epigastric pain*; *lactate*; *metabolic acidosis*; *CT scan*; *mortality*

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Emphysematous gastritis (EG) is a rare cause of abdominal pain, with less than 40 reported cases in English literature through 2012 (1). It has a non-specific presentation and is diagnosed via radiography in the appropriate clinical setting in patients with underlying predisposing factors. As EG can be potentially life-threatening, the physician should be aware of the appropriate management. We present a case of a diabetic male who had gastrointestinal symptoms from EG and rapid resolution of symptoms with only conservative management.

Case report

A 61-year-old male was presented to the emergency department with complains of diarrhea without any hematochezia or melena, which started 24-hours prior. He subsequently developed nausea, foul-smelling belching, and multiple episodes of vomiting without hematemesis. He also complained of stabbing epigastric pain without radiation. Review of systems was positive for intermittent chills and dizziness, but he denied any fever or headache. He did not report any recent travel, sick contacts, or unusual food intake. He had a 20-year history of ulcerative colitis (UC) status post total procto-colectomy with ileal–anal anastomosis 14 years prior. His UC had been in remission since that time with four bowel movements per day on average. His other pertinent medical history included diabetes mellitus, hypercholesterolemia, and hypertension. His home medications included sitagliptin–metformin 50–500 mg twice daily, atorvastatin 10 mg daily, enalapril 20 mg daily, and metoprolol tartrate 100 mg twice daily. He denied any excessive consumption of non-steroidal anti-inflammatory drugs (NSAIDs), smoking, or alcohol abuse.

On examination, the patient was hypotensive with a blood pressure of 80/56 mmHg while his pulse was 60, respiratory rate 16, and temperature 36°C (309.1 K). His abdomen was soft, non-distended, and mildly tender in the epigastrium without any rebound tenderness or guarding. Laboratory tests revealed leukocytosis (white blood cell count of 20.9×10^9/L), elevated lactate (3 mEq/L, reference range 0.5–2.2 mEq/L), and an anion gap metabolic

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acidosis with bicarbonate of 14.6 mEq/L (reference range 24–31 mEq/L) and gap of 14 mEq/L (reference range 4–12 mEq/L). Sodium (135 mEq/L, reference range 135–153 mEq/L) and potassium (3.9 mEq/L, reference range 3.5–5.3 mEq/L) were normal. He also had acute kidney injury with a creatinine of 299 μmol/L (reference range 53–106 μmol/L). A non-contrast CT of the chest, abdomen, and pelvis revealed findings suspicious for emphysema in the wall of the stomach with a small amount of air in peri-gastric veins (Fig. 1). Portal venous gas within the left lobe of the liver was also visualized (Fig. 2). Also, a non-specific left adrenal nodule measuring 24.2 mm × 18.9 mm was visualized. As the patient's blood pressure and diabetes had been under good control (HbA1c of 7.5) with normal sodium and potassium on labs, further work-ups for primary hyperaldosteronism, pheochromocytoma, or Cushing syndrome were not considered.

The patient was started on intravenous fluids, piperacillin/tazobactam, and an intravenous infusion of pantoprazole while being admitted to the intensive care unit. He experienced rapid resolution of symptoms over the ensuing days. Admission stool studies were negative for *Clostridium difficile*, *Salmonella* spp., *Shigella* spp., *Campylobacter* spp., or *Escherichia coli*. His diet was advanced gradually, which he tolerated well. His renal function also markedly improved and returned to baseline with intravenous fluid resuscitation. Follow-up CT of the abdomen and pelvis with contrast 3 days after presentation revealed resolution of the EG. He was discharged home on a regular diet, oral pantoprazole 40 mg twice daily, and ciprofloxacin and metronidazole to complete a 10-day course of antibiotics. He was also advised to follow up with gastroenterology in 3–4 weeks for upper endoscopy. He was also recommended a follow-up CT abdomen in 6 months for his incidental adrenal nodule, which confirmed the stability of the nodule.

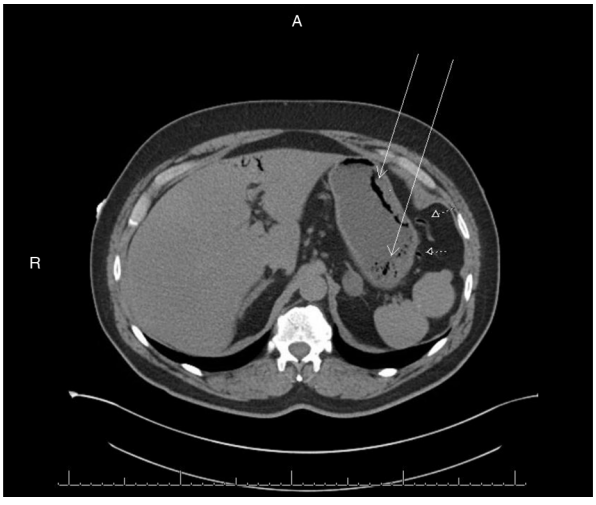

Fig. 1. A non-contrast CT abdomen (axial) revealing gastric intramural air suspicious for gastric emphysema (white arrows) with a small amount of air in peri-gastric veins (dotted arrows).

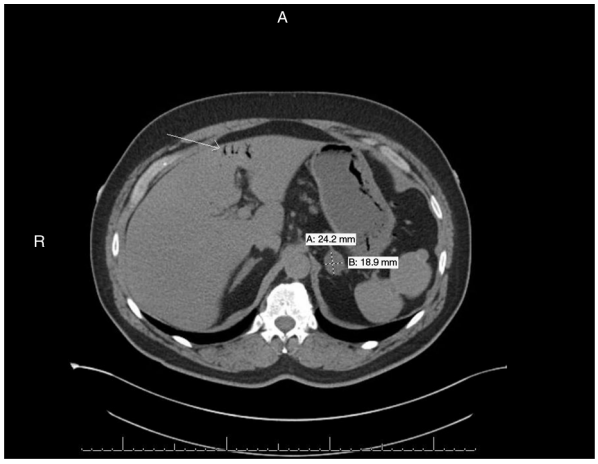

Fig. 2. A non-contrast CT abdomen (axial view) showing portal venous gas within the left lobe of the liver (white arrow). Also, a non-specific left adrenal nodule measuring 24.2 mm × 18.9 mm is visualized.

Discussion

Pneumatosis intestinalis refers to the presence of air within the bowel wall which can occur anywhere from the stomach to the rectum, with stomach being the least common site (2, 3). Gastric intramural air can be as a consequence of EG, which should be differentiated from a distant entity known as gastric emphysema (GE). It has been hypothesized that EG results from bacterial production of air. Various organisms that are usually responsible include *Enterobacter* spp., *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and *Candida albicans* (4). The predisposing factors reported in the literature include alcohol abuse, gastric surgery, recent gastroenteritis, corrosive ingestion, chronic consumption of NSAIDs/steroids, diabetes mellitus, COPD, and immunosuppression (5). However, GE, a more benign condition, results from air penetrating the wall from non-infectious etiologies, including raised intra-gastric pressures, dissection of air from the mediastinum, trauma, malignancy, inflammation, and ischemia (5, 6).

Clinical presentation of EG is usually non-specific and typically manifests as low-grade fever, chills, nausea, vomiting, mild-to-severe abdominal pain, diarrhea, hematochezia, or shock in severe cases (3). Abdominal examination can be benign or reveal signs of acute abdomen, including abdominal distension, epigastric tenderness, and decreased bowel sounds (7). Laboratory tests may show leukocytosis and metabolic acidosis from sepsis. On imaging, plain radiography can be helpful, but CT is the diagnostic modality of choice (2). CT in EG will identify a characteristic irregular, mottled appearance of the air in the thickened gastric wall and portal vein in the liver (3, 4). On the contrary, GE is

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typically asymptomatic or presents with mild symptoms with spontaneous resolution, except in those patients who have underlying gastric ischemia that may manifest as acute abdomen (5, 8). CT typically does not show thickening of the gastric wall in GE.

GE is usually self-limiting, whereas EG is a potentially life-threatening condition and mortality rates of 60–100% have been reported in the literature without prompt surgical intervention (3–5). However, in the absence of overt surgical indications like perforation or necrosis, there has been an increasing trend toward a conservative management with IV fluids, nasogastric tube placement, nutritional support, broad-spectrum antibiotics (covering enteric Gram-negatives and anaerobes) with contemplation for anti-fungal medications (1, 4, 5). The role of esophagogastroduodenoscopy (EGD) in gastric intramural air is unclear. Some experts avoid it acutely because of the risk of perforation while others recommend urgent EGD to rule out gastric ischemia that may need immediate laparotomy. In a retrospective study, factors associated with higher motility included elevated serum lactate, serum creatinine, and concomitant pneumatosis in small bowel and colon, warranting a more vigilant management of such cases (2). Gastric strictures and contractures as a long-term complication have been reported in over 20% of the cases (9).

The differentiation of EG and GE is generally based on clinical presentation, predisposing conditions, and CT (and not solely on radiography because of overlap in findings) (1, 5). Most patients with GE have little or no symptoms, except in cases of gastric ischemia (5). Our patient's rapid resolution of symptoms with conservative measures argues against gastric ischemia. The severity of his illness at presentation with leukocytosis, anion gap metabolic acidosis, and sepsis makes GE less likely and favors a diagnosis of EG. Furthermore, he also had predisposing factors for EG (diabetes and probable gastroenteritis) but none of the predisposing factors of GE (prior malignancy, trauma, or potential cause of raised intragastric pressure). Even though he had a high serum lactate and creatinine at presentation, both of which have been suggested to reflect poor outcomes, he improved remarkably well with conservative measures.

Conclusion

EG is a rare cause of abdominal pain, which is differentiated from GE, a more benign entity, based on the clinical presentation, predisposing factors, and radiological findings. It is associated with a high mortality; however, timely diagnosis and institution of conservative measures are associated with favorable outcomes. Surgical intervention may be needed in cases of life-threatening perforations or strictures that can develop afterward.

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Conflict of interest and funding

The authors declare that there is no conflict of interest regarding the publication of this paper.

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