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Case Report

Gastric pneumatosis in immunocompromised patients: A report of 2 cases and comprehensive literature review ☆

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ABSTRACT

Gastric pneumatosis (GP) is a rare finding. It can be seen with both gastric emphysema (GE) and emphysematous gastritis (EG); however, both conditions present similarly and differentiating between the 2 is difficult radiographically. Moreover, the treatment is vastly different for both conditions, in which treatment for GE is focused on supportive care while treatment for EG may even involve gastrectomy. Making the distinction between GE and EG is crucial because GE has a benign clinical course, while EG carries significant mortality. Early endoscopy may be a useful tool in differentiating between the 2 conditions and to guide further management. Herein, we present a case series of 2 immunocompromised patients who presented with symptoms and radiographic evidence consistent with gastric pneumatosis. We found that early endoscopy assisted in risk stratification and helped guide our management strategy. We recommend consideration of endoscopic evaluation as part of ritualized evaluation of patients presenting with gastric pneumatosis.

Introduction

Gastric pneumatosis (GP), or air in the gastric wall, is a relatively uncommon diagnosis. The etiologies of GP can be characterized by gastric causes, such as bowel ischemia, gastritis or caustic ingestion versus extra-gastric causes, such as volvulus, malignancy, superior mesenteric artery syndrome, to name a few [1,2]. Regardless of etiology, the primary mechanism is a breach in the integrity of the gastric wall [1–4].

GP can be further classified as gastric emphysema (GE) or emphysematous gastritis (EG) [4,5]. These are 2 similar conditions, with very different radiographic findings and prognosis [4,6]. GE occurs when there is air within the gastric wall in the absence of an underlying infection and is clinically benign [6]. It is, however a very rare diagnosis with around only 40

Abbreviations: GP, gastric pneumatosis; GE, gastric emphysema; EG, emphysematous gastritis; HIV, human immunodeficiency virus; DM, diabetes mellitus; ESRD, end-stage renal disease; CT, computerized tomography; EGD, esophagogastroduodenoscopy; MRI, magnetic resonance image.

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cases previous described [4]. Conversely, EG is a sinister diagnosis which carries a high mortality rate [4–6]. EG occurs when there are gas-forming organisms within the gastric wall, most commonly from infectious spread of *Escherichia coli*, *Streptococcus* species, *Enterobacter* species, *Pseudomonas aeruginosa*, and *Clostridium perfringens* [4,5]. EG is also uncommon because the acidic pH, mucosal barrier and abundant gastric vasculature are often successful in preventing these types of infections within the stomach [5,7]. Several models have proposed theories underlying the pathogenesis despite these protective mechanisms, including the Bacterial Theory, which postulates that gas-forming bacteria result in an accumulation of air in the stomach, the Mechanical Theory which states that gas enters the stomach as a result of increased luminal pressures or direct trauma or the Mucosal Damage Theory, which attributes gas build-up to damaged mucosa and submucosa in the setting of inflammation and ischemia. [7] Other theories, such as the Pulmonary Disease Theory attributes this accumulation to damage to the alveoli [7]. Both EG and GE may lead to gastric ischemia [4,5]. Abdominal surgeries, ingestion of corrosive chemicals and objects, alcohol abuse and gastrointestinal infections and ischemia predispose patients to both conditions, however the presence of underlying diabetes or immunosuppression are only additional risk factors for GE [4,5].

Clinically, both EG and GE present similarly on a spectrum ranging from symptom and laboratory evidence concerning for infection to non-specific symptoms of upper abdominal pain, abdominal distension, nausea, vomiting, hematemesis, diarrhea or melena [6]. Often the presentation is not concerning for an acute abdomen, which decreases the suspicion for diagnosis [6]. Moreover, the diagnoses may be masked in patients with underlying comorbidities, such as diabetes, human immunodeficiency virus (HIV), cirrhosis or kidney failure, which ultimately delays the diagnosis and management [8].

The focus of treatment in EG includes correction of acid-base and/or electrolyte abnormalities, fluid resuscitation, as well as administration of intravenous antimicrobial therapy [5,6]. Definitive surgical treatment with gastrectomy may be necessary when expectant medical management fails, however surgical treatment should be avoided in the absence of ischemic bowel or necrosis due to the high risk of bowel perforation [4]. Thus, definitive treatment can be delayed until the patient is clinically stable and the mucosa is not as friable [4]. The mortality rate for EG is between 60% and 80%, regardless of early intervention [4,5]. But this differs drastically from GE, which runs a benign clinical course and spontaneously resolves [5,6]. It has been shown that when serum lactic acid levels exceed 2.0 mmol/L at the time of diagnosis in GP, the mortality rate is greater than 80% [8].

Herein we present 2 immunocompromised patients who were diagnosed radiographically with GP, but were definitively diagnosed with GE or EG after endoscopic evaluation, which ultimately aided in risk stratification and early intervention.

Case 1

A 58-year-old female presented to the hospital with left-sided hip pain. She had an extensive past medical history of Stage IV, Metastatic-Squamous Cell Lung Carcinoma, ESRD on hemodialysis after renal transplant 10 years prior, systolic heart failure, hypertension, dyslipidemia and insulin-dependent Type-II DM. Initial physical exam was only remarkable for decreased range of motion of the left hip with focal tenderness. The patient was diagnosed with a left proximal femur effusion and admitted for hemiarthroplasty. Due to the patient's inability to ambulate, she was medically optimized prior to surgery and experienced an uneventful operative course. On post-operative day, 1 patient was noted to have a drop in her hemoglobin from 12.2 g/dl to 8.1 g/dl with evidence of hemodynamic compromise. She was transfused one unit of packed red blood cells and transferred to the medical intensive care unit for closer monitoring.

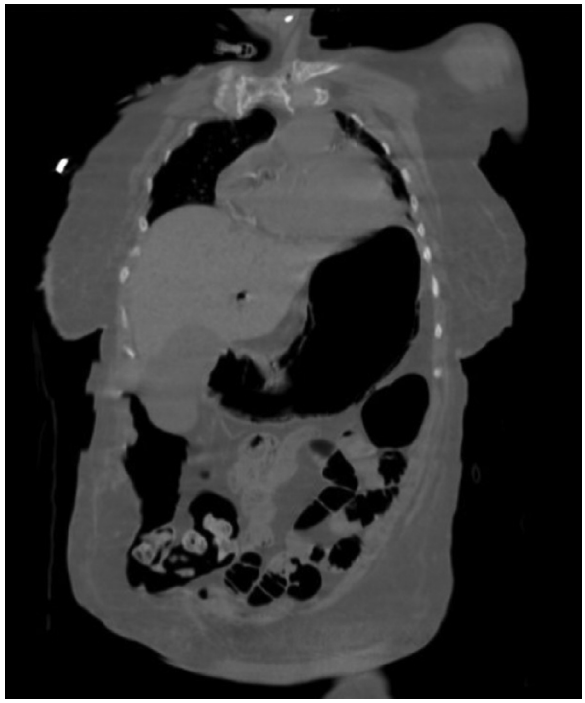

Fig. 1 – Coronal computerized tomography from Case 1, demonstrating portal venous gas with pneumatosis of the stomach and the adjacent duodenum.

A CT of the chest, abdomen and pelvis with intravenous contrast revealed portal venous gas with pneumatosis of the stomach and the adjacent duodenum (Fig. 1). At this time, the patient had epigastric and left-upper quadrant tenderness upon deep palpation. She underwent an EGD to evaluate the integrity of the gastric mucosa. The EGD was consistent with a diffusely ulcerated, edematous hemorrhagic gastric mucosa sparing the pre-pyloric region. Yellow exudate was visualized overlying the hemorrhagic mucosa, without evidence of active bleeding. Surgical consultation was placed for gastrectomy, however given the patient's comorbidities and hemodynamic instability she was deemed a poor surgical candidate. Despite resuscitative efforts, the patient continued to decline, requiring vasopressor support and intubation. Interval imaging with CT angiogram revealed re-demonstration of the gastric pneumatosis and a widely patent portal vein without evidence of

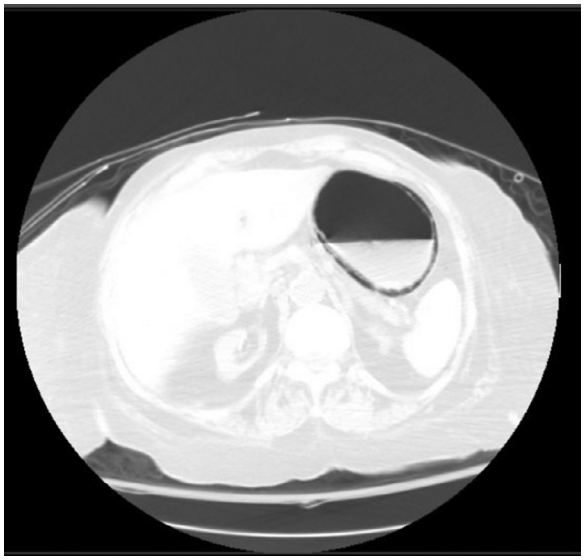

Fig. 2 – Sagittal computerized tomography from Case 2, demonstrating a severely distended stomach with air-bubbles along the anti-dependent wall of the stomach, indicative of gastric pneumatosis.

dissection or large vessel occlusion. Five days after admission, the patient experienced ventricular fibrillation-cardiac arrest and unfortunately expired.

Case 2

A 79-year-old male with a past medical history significant for peripheral arterial disease, DM, coronary artery disease, chronic obstructive pulmonary disease, seizure disorder, and benign prostatic hyperplasia presented to the hospital with generalized weakness and recent falls. He was hemodynamically stable on examination with a normal neurological workup. On physical exam, he was noted to have necrosis of the distal phalanx of the second toe of the right foot. A Magnetic Resonance Image (MRI) revealed osteomyelitis extending from the phalanges to the head of the metatarsal of the first and second digits of the right foot. He underwent amputation of the first and second toes to the metatarsal base, and was placed on broad-spectrum antibiotics. Further vascular workup revealed arterial insufficiency, requiring balloon angioplasty of the right profundal femoris artery.

On the sixth day, the patient had leukocytosis and nausea with mild distention on physical exam. At this time, he denied any pain and his abdomen was soft. A CT of the chest was performed showing a severely distended stomach with air-bubbles along the anti-dependent wall of the stomach, indicative of gastric pneumatosis (Fig. 2). He was transferred to the medical intensive care unit and a nasogastric tube was placed for decompression. He underwent an EGD, with findings of congestion, erythema and a hemorrhagic appearance with ulceration of the gastric fundus, body, and antrum. Biopsies were significant for mild chronic gastritis with foveolar hyperplasia, intestinal metaplasia, and negative *Helicobacter pylori* staining. This constellation of findings was suggestive of ischemic ulceration. The patient improved clinically and was discharged on long-term antibiotics for osteomyelitis and a proton pump inhibitor.

Discussion

Computerized tomography (CT) with contrast is the radiologic modality of choice, given the sensitivity and specificity in detecting intramural gas [2,5]. CT can also demonstrate portal venous gas, gastric wall thickening, and delineate the extent of the gastric wall extension more so than with plan radiography and barium studies [2,6]. GE gives the appearance of hypodense linear or curve fringe on the gastric wall and gastric distension in the absence of gastric wall thickening [6]. EG gives the appearance of a streaky and linear pattern of air along the gastric wall and gastric distension with gastric wall thickening [6]. These findings may be difficult to distinguish and thus imaging, regardless of modality is often unsuccessful at identifying the primary etiology to further direct management [2,6]. Identifying the primary etiology, as well as differentiating between EG and GE is also imperative for risk-stratification [5].

EGD has recently been utilized to further evaluate GP [9]. Endoscopically, GE gives the appearance of cobblestones, representing submucosal blebs of air within the stomach [5,6,9]. Early endoscopy can also identify necrosis, gastric ulcers and necrotic mucosal clots within the muscularis mucosa, indicative of EG [9].

Currently, there is no consensus on the role of upper endoscopy in the diagnosis and management of patients with GP; however, a systematic analysis demonstrated a reduction in overall mortality to 33% [9]. Between 44% and 60% of cases diagnosed with GP radiographically can be managed conservatively, but many patients are still taken for surgical exploration and treatment [7,9]. Surgical intervention carries high risk of complications, such as perforation, anastomotic leaks, fistulation, due to the friability of the mucosa [7,9]. Approximately 25% of patients will also develop strictures requiring endoscopic dilatation post-operatively [9]. Moreover, surgical exploration has been shown to be non-diagnostic in approximately 15% of cases, and is associated with a mortality rate of 40% in those with EG [7]. Thus, endoscopic evaluation can be useful in discerning EG from GE, identifying and treating the primary etiology, such as gastric ulcers, and reducing hospital length of stay, which has been shown to directly correlate with increased mortality [7,9]. Additionally, endoscopic evaluation and treatment may also prevent unnecessary surgical exploration in patients with GE, and/or inappropriate and risky surgery in patients with EG, further improving overall mortality and morbidity [9].

In our cases, early endoscopy did not reveal necrosis and patients were subsequently not taken for surgery. Although 1 of our 2 patients had clinical improvement following endoscopic evaluation, one met her demise from likely other factors rather than the diagnosis of GE. In both of our cases, early endoscopy assisted in risk-stratification and guided management appropriately. Imaging was suspicious for EG

while direct endoscopic evaluation was most consistent with GE. As both conditions present similarly and cannot be fully distinguished radiographically, we recommend early upper endoscopy to be incorporated as the standard of care.

Conclusion

Gastric emphysema should be distinguished from emphysematous gastritis endoscopically to guide management and risk-stratify patients who would benefit most from surgical exploration and definitive treatment.

Author contributions

Tagliaferri, A. and Melki, G. wrote the manuscript and performed the literary review. Cavanagh, Y., Mohamed, A., Grossman, M., and Baddoura, W. assisted in the collection of the patient's data and edited the final manuscript prior to submission. All authors are in agreement of this submission.

Data availability

Not applicable.

Patient consent

As this is a case report, consent was obtained for the purpose of this paper, in keeping with the journal's guidelines.

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