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Case Report

Intramural gastric air in an acutely ill patient with endoscopic and surgical correlation

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ABSTRACT

Intramural gastric air is a rare but potentially life-threatening imaging finding. We present the case of a 56-year-old female patient with a history of schizophrenia, who presented with multiple episodes of vomiting and altered mental status. CT scan revealed intramural gastric air and portal venous gas. The patient was treated conservatively with a favorable outcome. Gastric emphysema and emphysematous gastritis should be considered in the differential diagnosis of air in the gastric wall. It is critical to distinguish between these 2 entities as their clinical course and management differ significantly, although radiographic findings may mimic each other.

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Case description

A 56-year-old woman presented to the Emergency Department (ED) with a severely altered mental status and multiple episodes of vomiting. Her medical history included schizophrenia with episodes of foreign bodies' ingestion (food boluses, bones, pins). Clinical examination revealed epigastric tenderness without peritonism. During the physical examination the patient became comatose and required in-

tubation. Subsequently, she underwent a full-body Computed Tomography (CT) scan while blood samples were obtained for biochemistry, hematology and toxicology analysis. Blood investigations revealed markedly elevated blood glucose (681 mg/dL) and deranged renal function with a blood urea of 56 mg/dL and creatinine of 2 mg/dL. Her white cell count was 10,790 cells/ μ L and hemoglobin was 10, 7 g/dL with normal coagulation parameters. Moreover, the inflammatory markers were only mildly raised. Toxicological tests were negative. Her cranial and thoracic CT scan was also unre-

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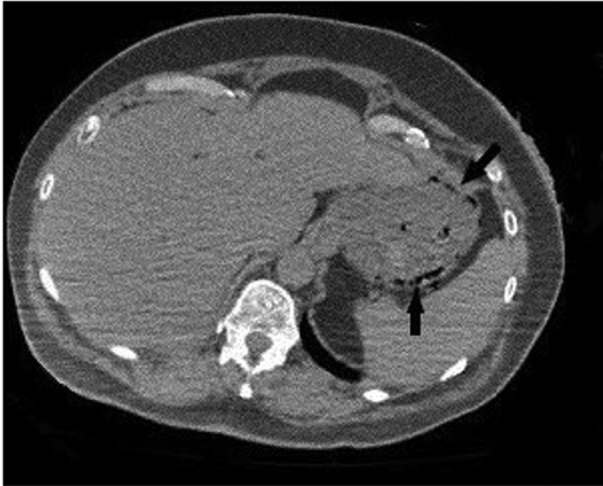


Fig. 1 – Nonenhanced axial CT scan of the abdomen demonstrates air bubbles within the gastric wall (arrows).

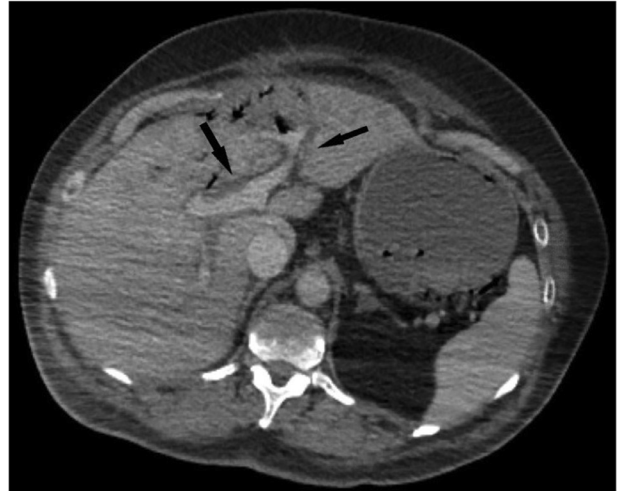


Fig. 3 – Contrast-enhanced axial CT scan of the abdomen in the portal venous phase shows periportal oedema (black arrows).

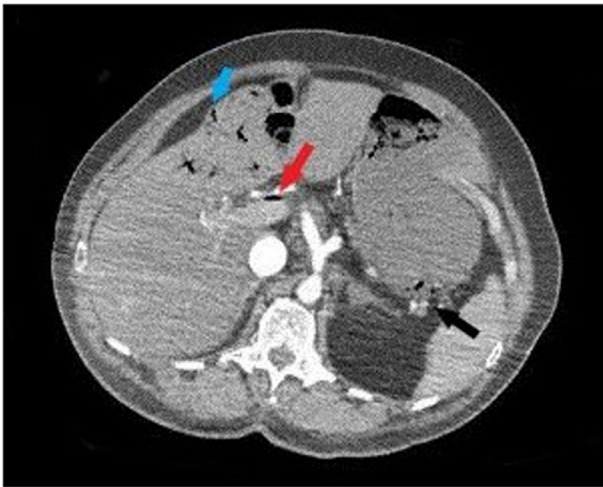


Fig. 2 – Contrast-enhanced axial CT scan of the abdomen demonstrates hepatic portal venous gas (red arrow) along with linear air in the intrahepatic portal branches (blue arrow). Also note the layering of air bubbles along the posterior wall (black arrow) of stomach.



Fig. 4 – Contrast-enhanced axial CT scan of the abdomen in the arterial phase shows focal narrowing of the proximal celiac trunk with poststenotic dilatation of its branches (black arrow).

markable. Abdominal contrast-enhanced CT scan revealed a distended stomach with air-fluid levels and linear streaks of air mainly in the posterior gastric wall along with locules of gas in small gastric veins but no wall thickening or pneumoperitoneum (Fig. 1). Also, hepatic portal venous gas and periportal oedema were present (Figs. 2 and 3). Additionally, focal narrowing of the proximal celiac artery and poststenotic dilatation were demonstrated (Fig. 4). Emergency upper gastrointestinal endoscopy was performed, revealing gastropathy along with superficial ulcers due to foreign bodies' ingestion. Considering the possibility of gastric ischemia the patient underwent an exploratory laparotomy. Operative findings showed no visible evidence of gastric ischemia or necrosis. The patient's sudden decrease in the level of

consciousness was attributed to diabetic ketoacidosis, as laboratory investigations revealed not only hyperglycemia, but also acidosis and ketonuria. In order to further stabilize the patient hemodynamically and respiratorily, she was admitted to the Intensive Care Unit (ICU) and commenced conservative treatment including crystalloid fluids, insulin, and broad-spectrum antibiotics. Repeat upper gastrointestinal endoscopy showed normal gastric mucosa with negative respective biopsy results. Follow-up CT scan of the abdomen showed resolution of intramural gastric air and portal venous gas with no signs of celiac artery dissection or occlusion (Fig. 5). The patient was discharged from the ICU 2 weeks later.

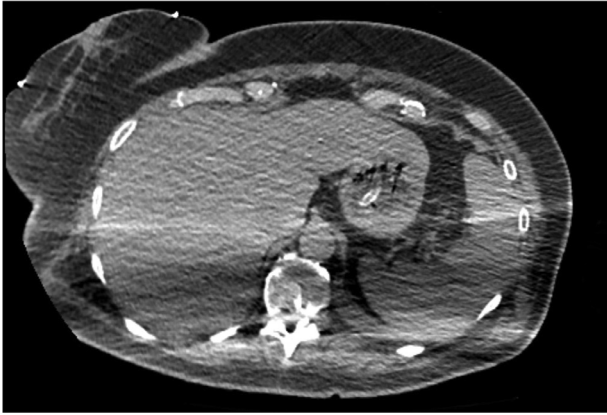


Fig. 5 – Follow up nonenhanced axial CT scan of the abdomen shows resolution of hepatic portal venous gas and air within the stomach wall. A nasogastric tube is also present.

Discussion

Gastric pneumatosis refers to the presence of air within the gastric wall and may be a result of various possible conditions which can be classified into 2 major categories, gastric emphysema and emphysematous gastritis. Abdominal radiography can be used as the initial imaging modality as it may reveal the presence of air within the gastric wall [1]. However, CT has an inherent superiority when compared to plain radiography as it can detect even a minimal amount of air within the wall of the stomach and is the imaging method of choice for distinguishing between the above entities [1].

Gastric emphysema is caused by a disruption in gastric mucosal integrity leading to the entry of air into the wall. There are various causes for this phenomenon including increased intragastric pressure, trauma, forceful vomiting, dissection of air from the mediastinum and ischemia [2]. The above usually points to a noninfectious source of intramural gastric air [3]. Gastric ischemia has also been reported as aetiology for gastric emphysema and hepatic portal venous gas [2]. Plain abdominal radiography will usually reveal marked gastric distension with radiolucencies (gas bubbles) outlining the gastric wall [4]. CT is the modality of choice and it typically demonstrates a linear and defined distribution of air within the wall of the stomach. Gastric distension may also be present, while signs of wall thickening and perforation are usually absent [1,6]. Patients have mild or no symptoms except for those experiencing gastric ischemia, who may present with an acute abdomen. Finally, gastric emphysema is usually self-limited and requires no intervention, but rather a conservative treatment, as the prognosis is excellent [5].

In contrast, emphysematous gastritis is a rare infection of the gastric wall secondary to gas forming organisms associated with systematic toxicity. *Streptococcus* species, *E.coli*, *Enterobacter* species, *P.aeruginosa* are the most common pathogens implicated in the pathogenesis of emphysematous gastritis [6]. Predisposing factors include gastroduodenal surgery, ingestion of corrosive materials, gastroenteritis or

gastrointestinal infarction, alcohol abuse, abdominal surgery, diabetes, and immunosuppression [5]. On abdominal X-ray often a contracted stomach can be identified along with frothy radiolucencies outlining its wall [4]. Characteristic CT findings include a curvilinear or mottled pattern of distribution of air within the gastric wall. CT may also reveal irregular gastric folds thickening due to underlying mucosal oedema [8]. Perigastric fat stranding and portal venous gas may also be identified [6]. As affected individuals often become toxic, urgent surgical intervention is needed. However, surgery should be avoided during the acute phase of emphysematous gastritis in the absence of bowel perforation, necrosis or ischemia because of mucosal friability and delayed healing of the sutured margins; therefore late surgical intervention should be preferred, if possible [9]. The overall prognosis is poor, with mortality rates reaching 60%-80% [7,9], whilst it is possible to lead to life-threatening complications, such as gastric perforation and pneumoperitoneum.

The differentiation of these clinical conditions relies on the patient's clinical presentation, predisposing conditions and characteristic imaging findings. In our case, the linear appearance of intramural gastric air and the gastric distension raised our suspicion towards the diagnosis of gastric emphysema. However, the portal venous gas and ingestion of small foreign bodies along with diabetes pointed out to the possibility of emphysematous gastritis. Furthermore, the narrowing of the celiac artery origin with poststenotic dilatation was suggestive of gastric ischemia. However, the presence of post-stenotic contrast enhancement makes this a less likely underlying cause.

The pathophysiologic mechanism of gastric pneumatosis in our patient is presumed to be the result of air entering the gastric wall through a small mucosal tear and increased gastric wall friability due to recurrent vomiting [10]. As a consequence, air within the wall of the stomach dissected through the tissue and reached the portal veins. Hepatic portal venous gas occurs when intraluminal or bacterial gas enters the portomesenteric circulation. Necrotic bowel wall from infection, inflammation, ischemia or highly increased intraluminal pressure such in our case, seem to favor the entry of air into the venous radicals [11].

Retrospectively, the recurrent episodes of vomiting, as a predisposing factor, the linear distribution of air within the gastric wall, the patient's mild elevation of inflammatory markers, the negative microbiology results, the lack of pathological intraoperative findings, the resolution of gastric intramural and portal venous gas following conservative management, and finally her uneventful recovery supports the diagnosis of self-limited gastric emphysema.

In conclusion, we present a case of gastric emphysema and portal venous gas induced by forceful vomiting where patient was successfully managed by conservative measures. Gastric emphysema and emphysematous gastritis present with overlapping imaging findings but differ in their clinical presentation, management, and prognosis. Currently, CT is the most accurate diagnostic exam. It is therefore essential for radiologists to be aware of the specific imaging characteristics of the above entities in order to reach an accurate diagnosis early in the disease and to achieve a favorable outcome.

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