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Intramural Stomach Gas with Hepatic Portal Venous Gas Indicating Spontaneous Stomach Necrosis

Igor Petrovic [1], Maja Sremac [2], Dario Grbavac [1], Goran Pavlek [1], Ante Bogut [3], Branko Bakula [3], Renata Romic [4], Ivan Romic [1,*]

1. University Hospital Centre Zagreb, Department of Surgery, Croatia
2. University Hospital Centre Zagreb, Department of Gastroenterology, Croatia
3. University Hospital Sveti Duh, Department of Surgery, Croatia
4. Health Centre Zagreb, Family Medicine, Croatia

* **Corresponding Author:**
Ivan Romic , MD
Address : University Hospital Centre Zagreb, Kispaticeva 4, Croatia

Tel : + 385238-8888
Fax : + 3851-238-8888
Email : i.romic@gmail.com

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ABSTRACT

Intramural gas in the stomach associated with hepatic portal venous gas is a rare entity, which suggests ischemic or infectious pathology of the stomach. We report a case of a 73-year-old man who presented with epigastric pain and nausea of 6 hours duration followed by hematemesis. The patient had pale skin, anemia, and a diffusively tender and distended abdomen. Abdominal radiography and computed tomography (CT) revealed gas in thickened gastric wall and gas in intrahepatic portal vein branches. Surgery was indicated, which consisted of partial gastrectomy with Roux en esophago-jejunal anastomosis. Postoperative course was uneventful, and pathohistological analysis indicated stomach wall necrosis with emphysametous gastritis (EG). The patient was free of symptoms at 2 years follow-up. Intramural gas in the stomach should always be meticulously investigated to differentiate between emphysematous gastritis and gastric emphysema, as this would direct the therapeutic approach to be adopted.

KEYWORDS:
Stomach rupture; Necrosis; Liver diseases

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INTRODUCTION

Stomach necrosis is an uncommon clinical entity, which is usually a result of a specific underlying pathology. In most cases, this is seen in the postoperative period after surgical devascularization of the stomach or as a consequence of tumors/ulcers or ischemic infarction of the stomach (occlusive or non-occlusive). However, as we describe in our case here, sometimes no exact cause can be found, which suggests that emphysematous gastritis may have a role in the pathophysiology of unexplained gastric necrosis. Intramural gas may be a sign of such necrotic changes and it should always be meticulously investigated in order to differentiate between emphysematous gastritis and gastric emphysema, which require different diagnostic and therapeutic approach.

CASE REPORT

A 73-year-old man presented with epigastric pain and nausea of 6 hours duration followed by hematemesis for the past 2 hours. The patient was

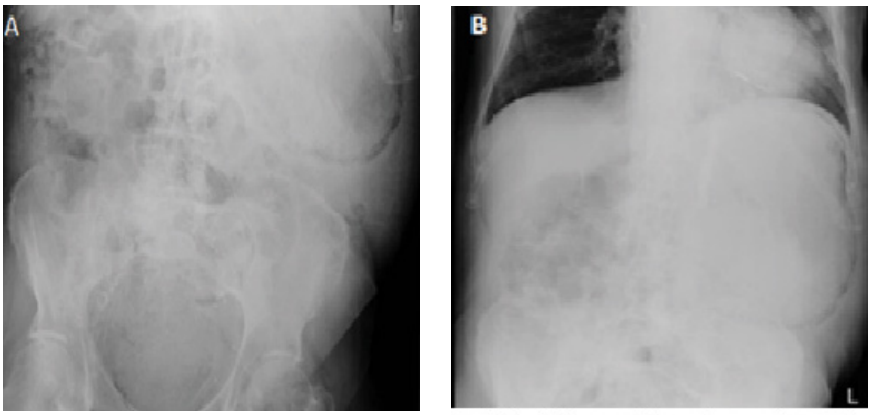

Fig.1: Abdominal radiographs showing: (A) linear gas pattern in upper right abdomen; (B) gas is seen in a projection of greater stomach curvature

a smoker, had a pacemaker because of arteriovenous (AV) block (II degree), and had no other significant comorbidities. On physical examination, the patient had pale skin, blood pressure 155/70 mm Hg, heart rate 85 beats/min with capillary oxygen saturation of 92%. The abdomen was diffusively tender and distended, but no clear rigidity was found. Hemoglobin was 90 mg/dL, white blood count was 16×109/L, and C-reactive protein 122 mg/L.

Abdominal radiography revealed soft tissue shadow with lamellar gas in the projection of gastric wall over the length of 15 cm along the greater curvature (Figure 1). CT showed thickened stomach wall over the area of 20 cm with gas seen mainly in intramural venous branches (Figure 2). Gas was also seen in intrahepatic portal vein branches. Gastroscopy showed no tumor but an area of stomach necrosis in the distal part of the corpus, which involved 1/3 of the circumference. Urgent surgery was indicated, and we found an ischemic stomach body with necrotic areas along the greater curvature (Figure 3).

Partial gastrectomy was done with Roux en esophago-jejunal anastomosis. The postoperative course was uneventful, and the patient was discharged on the 7th postoperative day. The patient was free of symptoms with unremarkable gastroscopy findings during the one-year follow-up period. Pathohistological analysis showed signs of ischemic transmural necrosis in the greater curvature with edematous and inflamed smaller curvature. No sign of ulcer, tumor, or mucosal laceration was found, and such pathological finding was consistent with the diagnosis of emphysematous gastritis. Microbiological analysis of the specimen detected polymicrobial infection (*Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, and *Bacteroides fragilis*).

DISCUSSION

Emphysematous gastritis (EG) is a rare but potentially life-threatening type of phlegmonous infectious gastritis caused by pathogenic gas-forming bacteria. It may present with an array of signs and symptoms, and some studies suggest high mortality rates, as high as 62%.1-2 As the disease progresses, patients usually become septic, have severe abdominal pain, nausea and hematemesis. Gastric emphysema (GE) occurs in patients with organic lesions in the upper gastrointestinal (GI) tract and is most commonly due to physical or bariatric trauma from nasogastric or endoscopic intubation.[3] GE does not benefit from surgery unless there are complications related to intubation or underlying disorders, such as malignancy. In contrast, EG arises from a septic focus in the gastric wall, is more common in patients with significant and immunocompromising comorbid conditions, and benefits from early and aggressive resuscitation and observation in a critical care environment.

EG is usually associated with risk factors such as alcoholism, immunosuppression, abdominal surgery, ingestion of corrosive substances, diabetes, gastric infarction, and tumors of the stomach.[4]

Less commonly reported causes include: invasive mycotic infections, anorexia nervosa, neonatal emphysematous gastritis, and acute gastric volvulus. Pathogens commonly involved are E*scherichia coli*, *Streptococcus*, *Enterobacter*, *Clostridium*, and *Pseudomonas aeruginosa*.[5,6] Early diagnosis and initiation of conservative treatment with bowel

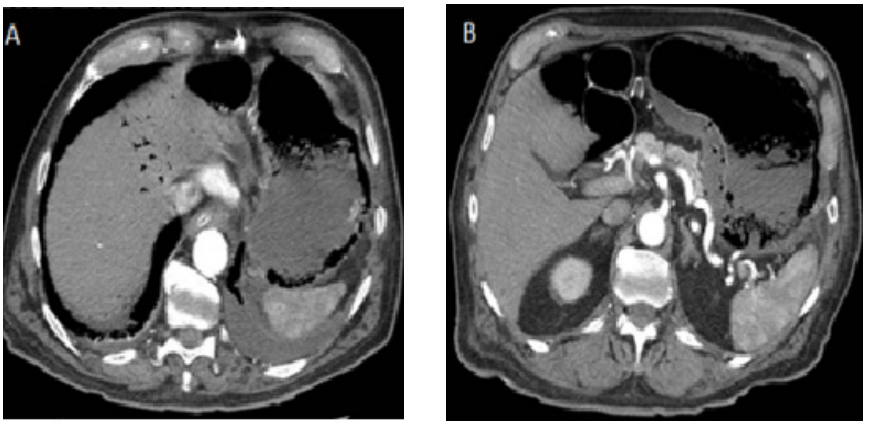

Fig. 2: **Abdominal tomogram showing: (A) gas in intrahepatic portal vein branches; (B) gas bubbles within stomach wall along the lesser curvature**

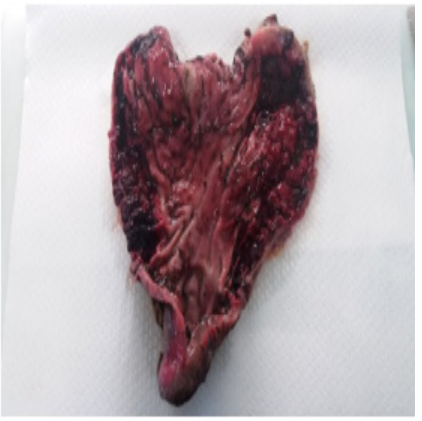

Fig. 3: **Surgical specimen showing stomach wall necrosis**

rest, intravenous fluids, and broad-spectrum antibiotics is imperative for an effective outcome that may be sufficient in milder cases. Late presentation and advanced disease may need exploratory laparotomy and total/partial gastrectomy due to transmural ischemia and peritonitis.[7]

Radiologically, in addition to the stomach wall, gas may be seen in other organs, in the portal vein, or in its branches and literature reviews show that hepatic portal gas indicates more severe disease and is associated with higher mortality.[8-9] Hepatic portal gas is most commonly associated with bowel necrosis, followed by ulcerative colitis, intra-abdominal abscess, small bowel obstruction, and gastric pathology (ulcer, tumor, or obstruction).[10]

Radiological patterns may help to differentiate EG from GE. While the former is associated with the cystic mottled, bubbly patterns, and signs of stomach wall gangrene, the latter is manifested by linear lucency along the greater curvature of the stomach.[11]

Both emphysematous gastritis and gastric emphysema are sporadically described mostly as case reports or small case series, which suggests a very low incidence of this clinical scenario. Our comprehensive literature review revealed only one systemic review, which identified 75 cases (both GE and EG are calculated) between 1980-2012.[12] This analysis demonstrated the main features of every single case, and 39 cases of EG were compared with 36 cases of GE. In 35 cases of EG, the etiology of stomach wall pneumatosis was identified, and in one case, the exact cause was unknown. Most frequent etiological factors were malignant stomach or small bowel obstruction followed by acute gastric dilatation and gastrostomy complications. Therefore, idiopathic (spontaneous) EG, as seen in our case, is extremely rare, and possible factors that lead to hypoperfusion of such highly vascularized organs should be investigated.

Our proposed etiology, in this case, was the hypoperfusion of the stomach due to periods of hypotension as a result of heart disease associated with advanced atherosclerosis of visceral blood vessels. The patient was a smoker, had heart arrhythmias and anemia, which all may have contributed to the decreased gastric blood supply, development of ischemic stomach wall necrosis, and secondary polymicrobial infection. The thrombotic or embolic incident is also a possible factor in the pathogenesis of EG, although we did not find evidence of left gastric artery embolism on CT scan nor intraoperatively.

EG has varied clinical presentations and may pose both diagnostic and therapeutic challenges to the attending physicians, especially when it is necessary to discriminate between GE and EG. Unlike GE that has a more benign clinical course, EG requires surgical intervention, but the type and

timing for surgery still remain controversial. Conservative management of EG has been described in the literature, but only after full-thickness necrosis and septic complications is ruled out.

In conclusion, surgeons, gastroenterologists, and radiologists should be aware of the causes and clinical course of gas in the stomach wall and should strive to differentiate GE from EG preoperatively, which will result in the more appropriate selection of treatment approaches.

ETHICAL APPROVAL

There is nothing to be declared.

CONFLICT OF INTEREST

The authors declare no conflict of interest related to this work.

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