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SCIENTIFIC LETTERS

Gastric emphysema and emphysematic gastritis, apparently similar entities with very different treatment☆

Enfisema gástrico y gastritis enfisematosa, entidades aparentemente similares con tratamiento muy diferente

To show the importance of the differential diagnosis between emphysematous gastritis (EG) and gastric emphysema (GE), we present two cases of gastric pneumatosis (GP) that required different therapeutic approaches.

A 34-year-old female with a history of endometriosis presented with abdominal pain and vomiting for 24 h. Her abdominal pain was accompanied by signs of peritonism and blood tests showed leukocytosis with neutrophilia. CT showed necrosis with gastric pneumatosis and portal pneumatosis (Fig. 1A and B). The acute abdomen and blood test results along with the radiological suspicion of emphysematous gastritis led to the decision for emergency surgery. As diagnostic laparotomy showed no evidence of intra-abdominal abnormalities, an intraoperative gastroscopy was performed, which revealed necrosis of the gastric mucosa without transmural involvement. No additional surgical procedures were performed. Endoscopic samples were taken for histological and microbiological analysis, with negative cultures and normal histology. The patient made satisfactory progress with broad-spectrum antibiotic therapy, and was discharged on day 7 after surgery. In the end, she was diagnosed with EG, with negative cultures and no clear precipitating cause.

A 25-year-old female with diabetes presented with 48 h of abdominal pain associated with copious vomiting. She had abdominal pain, but no peritonism or abnormal lab test results. CT showed gastric pneumatosis without portal pneumatosis (Fig. 1C). Gastric emphysema caused by increased intragastric pressure was suspected, and she was started on conservative medical treatment with fluid therapy, nil by mouth, nasogastric (NG) tube and proton pump inhibitors (PPI). The subsequent gastroscopy showed erosive gastritis, with normal histology and negative microbiology. The patient made good clinical progress and was discharged on day 3 with the diagnosis of GE.

EG and GE are two diseases with different aetiology, symptoms, treatment and prognosis. However, because of the similarity in the radiological findings, the differential diagnosis is complex. The presence of GP is typical in both conditions.[1–3]

EG is a serious infection induced by gas-producing microorganisms which invade the wall and cause inflammation, with the development of necrosis, abscesses and leukocyte invasion. Some of the typical causal germs are *Streptococcus* spp., *Enterobacter* spp., *Clostridium* spp., *S. aureus, P. aeruginosa* and *Candida* spp.[1] However, as with our patient, in a high percentage of cases it is not possible to isolate the germ responsible.

Risk factors for EG include ingestion of caustic substances, vomiting, gastric distension, cancer, recent surgery and treatment with NSAID, corticosteroids or chemotherapy. The common precipitating factor is gastric mucosal injury

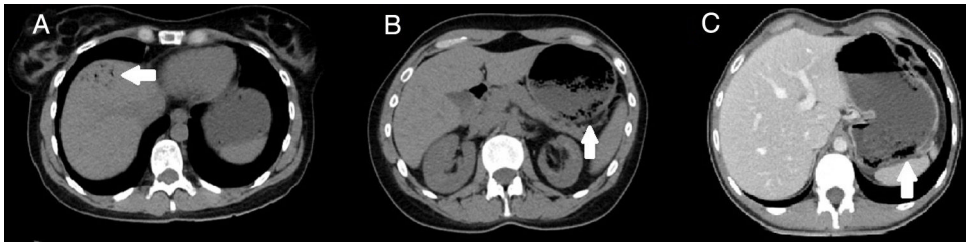

Figure 1 Unenhanced CT axial view showing gastric pneumatosis and portal pneumatosis (A and B). Portal phase showing gastric pneumatosis without portal pneumatosis (C).

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due to hyperpressure and subsequent colonisation by infectious agents.[2]

Meanwhile, GE is defined as the presence of gas in the stomach walls caused by disruption of the mucosa and subsequent dissection of air into the wall. Instrument-related iatrogenic trauma to the mucosa (by intubation or endoscopy) is the predominant cause.[2] In our case, the precipitating factor was the increase in endoluminal pressure associated with vomiting.

The initial symptoms in both conditions are nonspecific (nausea, vomiting, diarrhoea, etc.), which makes the diagnosis more complex. However, fever and systemic involvement tend to be more common in EG, while GE is generally asymptomatic or causes few symptoms.[1–3]

The presence of gastric pneumatosis on CT is a typical feature of both disorders, while gastric wall thickening and portal pneumatosis are findings suggestive of EG. Upper gastrointestinal endoscopy enables the gastric mucosa to be viewed and samples to be taken for histological and/or microbiological analysis.[3]

An accurate differential diagnosis is important due to the differences in treatment approach and outcome. EG has a high mortality rate and requires broad-spectrum antibiotic therapy (with coverage of Gram-negative, Gram-positive and anaerobic germs), fluid therapy, and sometimes emergency surgery (where conservative treatment fails or in cases of suspected complications, such as transmural necrosis or perforation). In our case, surgery was required at the outset due to a suspected complication. GE, in contrast, is generally self-limiting with conservative treatment (nil by mouth, gastric decompression with NG tube, PPI, fluid therapy and analgesia).[4,5]

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Conflict of interest

The authors declare that they have no conflicts of interest.

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Submucosal gland adenocarcinoma of the esophagus. A rare non-Barrett's associated tumor

Adenocarcinoma esofágico originado en glándulas submucosas. Un tumor raro no asociado a Barrett

Esophageal carcinoma is the 7th most frequent type of cancer worldwide, with 572.034 new cases in 2018. The most common histological subtype is the squamous cell carcinoma, although the incidence rate of esophageal adenocarcinoma (EAC) is rapidly increasing.[1] The vast majority of EAC arise from Barrett's esophagus located in the lower esophagus and the esophagogastric junction. Less frequently, EAC can develop from the submucosal gland/duct system or gastric heterotopia.[2,3]

Adenocarcinoma of the esophagus arising from the submucosal gland/duct system is a rare entity, with to our best knowledge, only seven reported cases of patients ranged between 43 and 74 years-old, presenting equally in both sexes.[2,3] Tumours were more frequently located in the middle and lower esophagus. Tumors may be more difficult to diagnose, since there is no visible precursor lesion at endoscopy, as in Barrett's esophagus or gastric heterotopia. Patients have been treated with surgery or endoscopic resection, with or without adjuvant therapy. Two patients died at 10 and 15 months. The rest were alive without disease between 18 months and 5 years follow-up.

We present a case of a 56-years old male who consulted with dysphagia. The upper digestive endoscopy revealed a distal esophageal stenosis covered with normal mucosa. (Fig. 1A), the Endoscopic Ultrasound (EUS) showed a hypoechogenic lesion within the esophageal wall invading the submucosa, the muscularis propria and reaching the adventitia. However, the mucosal layer was normal on EUS examination (Fig. 1B). EUS-FNA was performed and revealed an esophageal adenocarcinoma. Computed tomography scan (CT) showed concentric wall thickening (Fig. 1C). The patient received neoadjuvant chemotherapy with FLOT (docetaxel, oxaliplatin and fluorouracil/leucovorin) and an Ivor Lewis esophagectomy was performed. Gross examination revealed a normal mucosa with superficial erosion and no Barrett's esophagus (Fig. 1D). Transversal sections showed the tumor located within the wall thick-