Diagnostic dilemma of gastric intramural air

A Misro, H Sheth

Ealing Hospital NHS Trust, UK

ABSTRACT

Emphysematous gastritis and gastric emphysema remain the two most important differential diagnoses of intramural gastric air bubbles, both differing vastly in their clinical presentation, radiographic findings, management and prognosis. This report discusses a case of gastric emphysema along with the importance of reaching an accurate clinical diagnosis early in the disease course.

KEYWORDS

Emphysema – Gastric – Ischaemia – Gastritis – Portal – Vein – Gas

Accepted 5 December 2013; published online XXX

CORRESPONDENCE TO

Aswini Misro, E: draswini@gmail.com

Case History

An 81-year-old man presented with a short history of sudden onset severe epigastric pain. His past medical history included ischaemic heart disease, hypertension, glaucoma and recurrent postprandial abdominal pain. He was on the following medications: calcium and vitamin D₃, antihypertensive medication and glaucoma eye drops. He did not report use of alcohol, smoking or illicit drugs. His vital signs were within normal range apart from mild tachycardia. Abdominal examination showed epigastric tenderness without guarding, rigidity or peritonism. There were active bowel sounds. There was no lump, distension or organomegaly. His hernia orifices, renal angles and genitalia were within normal limits. Other systems were normal.

Blood examination showed deranged liver and renal function tests along with lactic metabolic acidosis and a very high amylase level (2,197u/l). White cell count ($10.7 \times 10^9/l$) and C-reactive protein (9mg/l) were mildly raised. Electrocardiography, urine examination, chest and abdominal x-ray showed no abnormality. The initial diagnosis was acute pancreatitis. Computed tomography (CT) of the abdomen and pelvis showed pockets of air layers in the posterior gastric wall (Figs 1 and 2) with a calcified plaque at the origin of the coeliac axis suggestive of gastric ischaemia (Fig 3). There was no evidence of thickened gastric wall or any pancreatic pathology. Hepatic portal venous gas was also present (Figs 1 and 2).

With a clinical diagnosis of gastric emphysema with hepatic portal venous gas secondary to acute on chronic gastric ischaemia, the patient was treated with antibiotics and proton pump inhibitors. Every possible care was taken to rule out emphysematous gastritis. After discussion with gastrointestinal surgeons at a tertiary care centre, transfer for possible

surgery was arranged provisionally as a contingency plan. His tachycardia settled by the third day. His symptoms, abdominal signs and blood picture improved with time. CT carried out three days later showed resolution of the hepatic portal venous gas and gastric air bubbles (Fig 4). Upper gastrointestinal endoscopy six weeks later showed normal gastric mucosa without any evidence of ischaemia.

Discussion

Emphysematous gastritis and gastric emphysema remain two important differential diagnoses of intramural gastric air. They differ in their clinical presentation, radiographic findings, management and prognosis. It is therefore essential to reach an accurate clinical diagnosis early in the disease course.

Gastric emphysema or gastric cystic pneumatosis, defined as air in the gastric wall without an underlying infection, is a rare radiological finding. It was first described by Brouardel in 1895 and only 42 cases have been described in the literature. Similar findings in the bowel are termed pneumatosis intestinalis.

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: (i) raised intragastric pressure (ie gastric outlet obstruction); (ii) trauma (eg at endoscopy and instrumentation); (iii) severe vomiting; (iv) dissection of air from the mediastinum (eg chronic obstructive pulmonary disease or pneumothorax); and (v) ischaemia.¹ Gastric ischaemia has been reported as aetiology for gastric emphysema and hepatic portal venous gas. Most patients usually have mild or no symptoms apart from those patients presenting with gastric ischaemia, who may present with an acute abdomen. The prognosis of this

Figure 1 Hepatic portal venous gas (arrows) in non-contrast computed tomography

Figure 3 Calcified plaque (arrow) at the origin of coeliac axis

condition is excellent.² The treatment is directed towards the precipitating aetiology.

In contrast, emphysematous gastritis is an acute infection of the stomach wall with gas forming organisms associated with systematic toxicity. *Escherichia coli*, *Streptococcus* spp, *Enterobacter* spp and *Pseudomonas aeruginosa* are the usual causative agents. Predisposing factors for this condition are gastroduodenal surgery, ingestion of corrosive materials, gastroenteritis or gastrointestinal infarction, alcohol abuse, abdominal surgery, diabetes and immunosuppression.³ Around 40 cases have been reported.²

Emphysematous gastritis usually has a fulminant course with a mortality rate of 60% and gastric strictures are as common as 25%.^{3,5} It must therefore be differentiated as

soon as possible from gastric emphysema to avoid adverse outcomes and to direct appropriate management. Patients with emphysematous gastritis usually present with severe abdominal pain, nausea, vomiting, haematemesis, low grade fevers and tachycardia.^{3,4}

Surgery should be avoided during the acute phase of emphysematous gastritis in the absence of bowel perforation, necrosis or ischaemia owing to friability of the mucosa and the delay in healing of the sutured margins.² There have been reports of successful conservative treatment of this condition without any operative intervention.⁴ A gastrectomy is often necessary when expectant medical management fails.

Figure 2 Layering of air bubbles along the posterior wall (arrow) of stomach suggestive of gastric ischaemia

Figure 4 Resolution of hepatic portal venous gas and gastric ischaemic change after three days of conservative treatment

Late surgical intervention is preferable because of the tissue friability, as mentioned above.

The differentiation of these two clinical conditions relies on the patient's clinical presentation, assessment of predisposing conditions and CT. In this case, the patient did not have any obvious predisposing factor for emphysematous gastritis. Recurrent postprandial abdominal pain in this patient, who had longstanding ischaemic heart disease and hypertension, makes gastric ischaemia more likely, which is further corroborated by a calcified plaque at the origin of the coeliac axis.

An acute abdomen, a high serum amylase level and lactic metabolic acidosis, which was most likely secondary to gastric ischaemia and dehydration, exacerbated the diagnostic dilemma in the beginning. CT did not reveal any obvious stomach wall thickening. There were pockets of air layering posteriorly in the fundus, in keeping with gastric emphysema. Moreover, the inflammatory markers were only mildly raised. The non-toxic presentation, smooth recovery with expectant treatment, and resolution of the gastric and portal venous gas in three days further substantiates the diagnosis of gastric emphysema secondary to gastric ischaemia.

Conclusions

Intramural gastric air is an uncommon but important radiological sign to recognise as it may represent either of two conditions: emphysematous gastritis or gastric emphysema. These differ in their clinical presentation, radiographic findings, management and prognosis.

References

1. Kussin SZ, Henry C, Navarro C *et al.* Gas within the wall of the stomach: report of a case and review of the literature. *Dig Dis Sci* 1982; **27**: 949–954.
2. van Mook WN, van der Geest S, Goessens ML *et al.* Gas within the wall of the stomach due to emphysematous gastritis: case report and review. *Eur J Gastroenterol Hepatol* 2002; **14**: 1,155–1,160.
3. Yalamanchili M, Cady W. Emphysematous gastritis in a hemodialysis patient. *South Med J* 2003; **96**: 84–88.
4. Paul M, John S, Menon MC *et al.* Successful medical management of emphysematous gastritis with concomitant portal venous air: a case report. *J Med Case Rep* 2010; **4**: 140.
5. Hadas-Halpren I, Hiller R, Guberman D. Emphysematous gastritis secondary to ingestion of large amounts of Coca Cola. *Am J Gastroenterol* 1993; **88**: 127–129.