

# Emphysematous gastritis after acute pancreatitis

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**SUMMARY** A case of emphysematous gastritis associated with extensive gastric infarction after acute pancreatitis and acute renal failure is described. This complication was diagnosed on a plain abdominal radiograph and confirmed endoscopically. Extensive gastric and hepatic infarction was seen at necropsy.

Emphysematous gastritis presents characteristic radiological appearances.<sup>1</sup> These features and the endoscopic appearances are described and the possible aetiology in this case is discussed.

## Case report

A 41 year old Asian man was found to have acute haemorrhagic pancreatitis at laparotomy. Necrotic tissue was removed and peritoneal lavage carried out. The remaining pancreatic tissue, together with the spleen, was removed 48 hours later after which he required intermittent positive pressure ventilation and inotropic support with dopamine infusion (dosage 10 µg/kg/minute). Acute renal failure supervened and he was transferred to our care. Daily haemodialysis was started. Dopamine was reduced to 5 µg/kg/min and discontinued after 48 hours.

His subsequent course was stormy, including a cardiac arrest on day 6 and the development of adult respiratory distress syndrome. Dopamine was restarted on day 7 because of hypotension (dosage 5 µg/kg/min) but was replaced by dobutamine (dosage 3 µg/kg/min) because of digital ischaemia.

Despite treatment with gentamicin, metronidazole, and cefotaxime he remained clinically septicaemic with a white cell count which reached  $100 \times 10^9/\text{ml}$ . On day 26 he had a gastrointestinal

haemorrhage with two further bleeding episodes over the next 48 hours (requiring 11 units of blood). At gastroscopy he was found to have multiple gastric erosions and a large clot adherent to the posterior wall of the gastric fundus. Thereafter he required increasing inotropic support with dopamine and dobutamine to maintain an adequate cardiac output, though systolic pressures still fell at times to 60 mm/Hg. A plain abdominal radiograph on day 30 showed gas present within the walls of the stomach in a linear distribution (Fig. 1). A repeat gastroscopy confirmed the presence of gas bubbles within the mucosal lining (Fig. 2) and an extensive area of infarcted gastric mucosa was visualised. Culture of stomach aspirate grew *Klebsiella pneumoniae*, *Streptococcus faecalis* and *Bacteroides* species. The patient died two days later.

Post mortem examination confirmed the radiological and endoscopic findings. In addition there was a mass of necrotic tissue in the lesser sac, splenic bed and pancreatic bed. There was patchy necrosis of the left lobe of the liver and of the left hemidiaphragm.

## Discussion

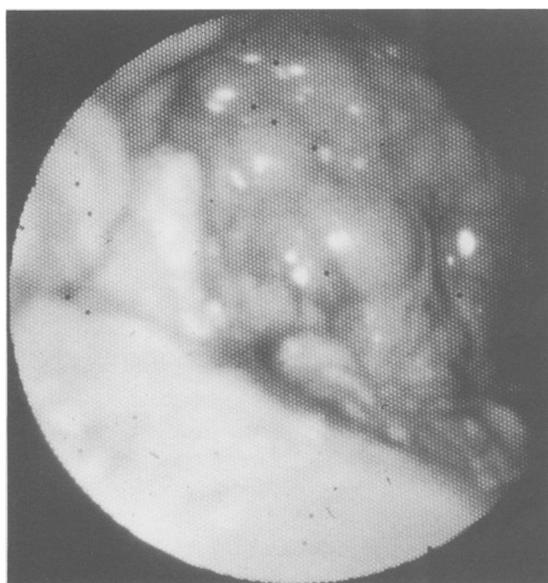
Gas in the wall of the stomach may either have entered from the outside or have been formed *in situ* within it, and these two conditions have been termed gastric emphysema and emphysematous gastritis respectively.<sup>2</sup> In this case the clinical setting, the radiological appearances, and the isolation of gas forming organisms from the gastric aspirate point to a

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**Fig. 1** Plain abdominal radiograph showing thickening of the wall of the stomach with bubbles, streaks of gas and mottled radiolucencies along the stomach contour.



**Fig. 2** Endoscopic appearances of the stomach showing bubbles of gas beneath the mucosa.

diagnosis of emphysematous gastritis consequent upon gastric necrosis. The endoscopic appearances of this rare condition, as presented here, are striking and equally as characteristic as the radiological appearances.

There are two main factors which may have been responsible for its development: continuing pancreatic bed sepsis and the use of inotropes. At necropsy he had continuing sepsis within the pancreatic bed and this was thought to have predisposed to thrombosis in the coeliac axis and its branches. Whether coeliac axis thrombosis occurred after the development of emphysematous gastritis as a result of the continuing pancreatic bed sepsis, or emphysematous gastritis followed coeliac axis thrombosis, it is not possible to say. At the time that intramural gas was first detected in the gastric mucosa the blood pressure was low and the patient was receiving both dopamine (12  $\mu\text{g}/\text{kg}/\text{min}$ ) and dobutamine (8  $\mu\text{g}/\text{kg}/\text{min}$ ). Alpha receptor stimulation appears to be the predominant factor in the regulation of gastric mucosal blood flow as shown by the elegant experiments of Hovendal and Bech.<sup>3</sup> Dopamine is known to have agonist activity at alpha receptors. Our patient had

already shown an unusual sensitivity to alpha agonism as shown by the onset of digital ischaemia at a dopamine dosage of 5 µg/kg/min. This, in conjunction with the reduced splanchnic blood flow mediated through alpha receptor stimulation and the clinical setting of a lowered blood pressure, may have further predisposed to the development of the complications described.

This patient developed emphysematous gastritis as a complication of acute pancreatitis. The radiological and endoscopic appearances of this unusual complication are illustrated. To our knowledge, there are no reports in the literature of gastric or intestinal infarction in response to dopamine infusion. Patients with acute haemorrhagic pancreatitis frequently require inotropic support and the contribution of pressor agents to both scenarios may be important. This

merits further investigation particularly when the continuing high mortality of this group of patients is considered.

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#### References

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