

Pneumomediastinum, stomach wall and hepatic portal vein gas secondary to partial necrosis of the stomach wall

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ABSTRACT

The combination of pneumomediastinum, gastric wall gas and hepatic portal vein gas is a challenging clinical problem. Although different causes of the individual gas sign have been reported in the literature, the cause of a triad of these signs in a single patient is less clear, and represents an extremely rare condition. A 65-year-old man presented with severe lower chest and epigastric pain of a few hours' duration. Initial assessment confirmed epigastric tenderness. Computed tomography showed pneumomediastinum, air in the stomach wall, hepatic portal vein gas and bowel dilatation. Small bowel and right colon dilatation was confirmed at laparotomy. The patient was treated subsequently with antibiotics to cover Gram-positive and Gram-negative bacteria, and anaerobes. The patient was discharged in good general condition on the 12th postoperative day. In conclusion, the triad of pneumomediastinum, gastric wall gas and hepatic portal vein gas is an extremely rare condition and associated with gastric necrosis.

Keywords: gastric wall gas, hepatic portal vein gas, pneumomediastinum, stomach wall necrosis

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INTRODUCTION

Pneumomediastinum is a rare condition with an incidence of 1/33,000, and can be caused by the perforation of the respiratory or gastrointestinal tracts.⁽¹⁾ The main causes of gastric emphysema, however, are infections and non-infectious conditions.⁽²⁾ Hepatic portal vein gas (HPVG), on the other hand, has a different aetiology and may suggest gastrointestinal ischaemia.⁽³⁾ The presentation is varied, from a mild benign course to a severe lethal condition. In the majority of cases, the diagnosis is based on clinical and radiological findings. Treatment largely depends on the presentation and on radiological signs. Although a combination of two of the three signs of pneumomediastinum, gastric emphysema and HPVG has

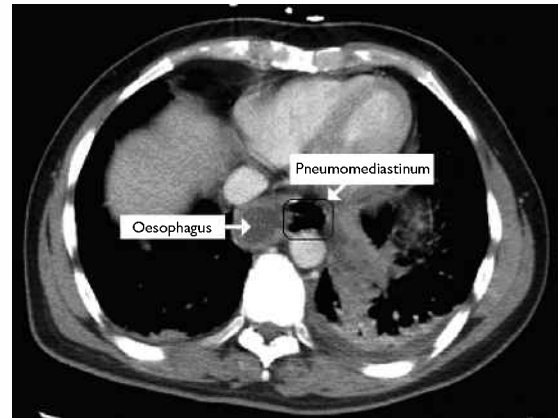


Fig. 1 Axial CT image of the thorax shows a pneumomediastinum.

been reported in the literature, the presence of these three signs in a single patient is very rare.

CASE REPORT

A 65-year-old man presented to the accident and emergency unit at 4 am with severe, constant lower chest and epigastric pain of a few hours' duration, and associated nausea, retching and vomiting. The pain did not radiate anywhere, and there were no special aggravating factors. It was partially relieved after 10 mg of intravenous morphine. He had no experience of such pain before, no recent gastroscopy or biopsy, and no significant medical or surgical problems. He was living alone, had no psychiatric problem, and was a non-smoker who occasionally drank alcohol. On examination, he was fully conscious, oriented but apprehensive. He was not dehydrated, anaemic or jaundiced. The initial assessment indicated a pulse rate of 110/min, blood pressure of 95/55 mmHg, a normal temperature and saturation of 94% on air. Chest examination was normal, while abdominal examination revealed a slightly distended, but soft abdomen with epigastric tenderness and no guarding or rigidity. There was no mass or organomegaly, and bowel sounds were sluggish. Rectal examination was unremarkable. Blood tests and arterial blood gas analysis revealed a normal haemoglobin 15 g/L, leucocytosis $14.8 \times 10^9/L$, C-reactive protein 48 mg/L, urea 7.3 mmol/L,

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Fig. 2 Axial CT images show hepatic portal vein gas (horizontal arrows) and stomach wall gas (vertical arrows).

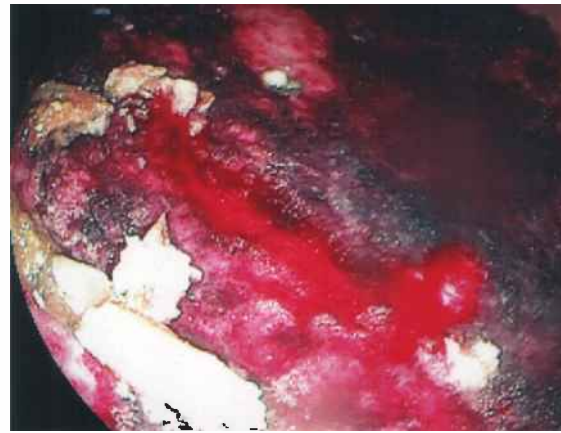


Fig. 3 Gastroscope photograph shows necrosis of the fundic mucosa.

creatinine 90 $\mu\text{mol/L}$, a pH 7.1 and a base excess of -11.5 mEq/L.

The abdominal radiograph showed non-specific small bowel dilatation, and chest radiograph was normal. Urgent contrast-enhanced computed tomography (CT) showed a pneumomediastinum, air in the stomach wall with radiological signs of inflammation and gastric fundus thickening, HPVG and bowel dilatation without the features of an obstructing lesion. The mesenteric angiographical phase confirmed no evidence of vascular occlusion or bowel ischaemia. Gastrografin study was performed to rule out oesophageal perforation, and showed no leak of the contrast.

Small bowel and right colon dilatation was confirmed at laparotomy. However, the examination of the bowel indicated no evidence of ischaemia or mechanical obstruction. The diaphragm was normal, and there was no evidence of stomach volvulus. The liver was normal, and there was no free peritoneal fluid. Exploration of the lesser sac showed neither stomach nor pancreatic gross abnormality. Peroperative gastroscope was performed and showed a 7 cm \times 5 cm patch of necrosis and inflammation of the fundic mucosa, which was

biopsied. Necrotic sloughs, non-specific ulceration and inflammation were reported on histological examination. No growth of bacteria was isolated from the gastric aspirate or biopsy. The patient was treated with antibiotics (augmentin 1.2 g thrice daily, and metronidazole 500 mg thrice daily) and omeprazole 40 mg/day. He was nursed in the intensive care unit for a week, and then discharged to the surgical ward. Gastroscope, performed ten days later, showed normal gastric mucosa. He was discharged in good general condition on the 12th postoperative day. Follow-up at three weeks, three months and six months were unremarkable.

DISCUSSION

This was a challenging clinical scenario for diagnosis and management. The patient presented with features suggestive of upper gastrointestinal pathology. Early diagnosis was crucial as pathology, such as a perforated oesophagus, can be treated within the golden period. CT, however, revealed dilated small bowel loops, pneumomediastinum, HPVG and air in the stomach wall. These three unusual gas signs posed a question of what the likely causes and immediate management options were. Pneumomediastinum is caused by gastrointestinal or respiratory pathology. It is an uncommon entity, first described almost 400 years ago.⁽⁴⁾ Oesophageal perforation is the main gastrointestinal cause. On the other hand, the alveolar leak of air could be secondary to bronchial asthma, vomiting and retching, chest injury, mechanical ventilation and trauma to the duodenum, colon and rectum. It can occur also after diagnostic and therapeutic chest procedures, such as mediastinoscopy and chest drain insertion. Chest pain and dyspnoea are the main symptoms of pneumomediastinum, and the surgical emphysema can be detected in about 65% of cases.⁽⁵⁾ The primary factors that favour the development of this

pathological entity are intestinal wall alterations, bowel distension and sepsis; while pneumothorax, surgical emphysema and pneumopericardium are the possible consequences of pneumomediastinum.

On the other hand, acute mesenteric ischaemia is the most common cause of HPVG, which continues to have a predictably higher mortality.⁽⁶⁾ For HPVG alone, the mortality could reach 27.3%, and for HPVG related to mesenteric bowel disease, it ranges from 50% to 75%.^(7,8) Portomesenteric vein gas is idiopathic in approximately 15% of cases.⁽⁹⁾ The other non-mesenteric ischaemia cases relate to abdominal trauma, gastric biopsy, paracolic abscess, sepsis, cancer, barium enema, colonoscopy, inflammatory bowel disease and simple intestinal or gastric distension.⁽¹⁰⁾ Knowledge of these conditions may help to avoid misinterpretation of CT findings, inappropriate clinical uncertainty and unnecessary surgery in certain cases.⁽¹¹⁾ More interestingly, the presence of HPVG may suggest the occurrence of a serious lesion in the abdominal cavity. Therefore, appropriate treatment should be performed immediately.⁽¹²⁾

The third sign of air in the stomach wall can be caused by non-infectious conditions, such as gastric distension and vomiting, pulmonary disease, instrumentation of the stomach, gastroscopy and biopsy, and obstructing lesions of the antrum and pylorus.⁽¹³⁾ Emphysematous gastritis due to infection is an extremely rare condition. This clinical entity was first described by Fraenkel in 1889.⁽¹⁴⁾ The most commonly-involved microorganisms are streptococci, *Escherichia coli*, *Pseudomonas aeruginosa*, *Clostridium perfringens* and *Staphylococcus aureus*, in addition to mucormycosis.⁽¹⁵⁾ Diagnosis is made by the demonstration of both intramural and portal venous gas on radiological studies.⁽¹⁶⁾

Laparotomy was decided upon because of the clinical picture, arterial blood gas acidosis, and CT suggestion of stomach fundus pathology (inflammation) and dilated bowel loops. The main concern was whether this was a case of gastrointestinal ischaemia or perforation. CT findings of pneumatosis intestinalis and porto-mesenteric venous gas due to bowel ischaemia, do not generally allow prediction of transmural bowel infarction, because they may be observed in patients with only partial ischaemic bowel wall damage.⁽¹⁷⁾ Therefore, whenever HPVG is detected on CT, urgent exploratory laparotomy is only mandatory in a patient with whom intestinal ischaemia or infarction is suspected, on the basis of radiological and clinical findings.⁽¹⁸⁾ Hence, it was dangerous to assume that this was a benign entity of pneumomediastinum, gastric emphysema and HPVG. The endoscopy examination in this case contributed to a complete assessment and

management plan. The aetiology and pathophysiology of the pneumomediastinum and HPVG may be less clear in our case. We think the main pathology was the gastric necrosis. While the associated bowel dilatation is a causative factor for portomesenteric gas,⁽¹⁰⁾ the cause of the HPVG and pneumomediastinum, may be explained by the association of the gastric necrosis, violent vomiting and retching, as reported in the literature.^(4,5)

The history, clinical examination and investigations should provide the evidence of possible diagnosis to set an immediate, safe and curative management plan. The main lesson of this case report was to highlight the clinical features, and hence heighten the index of suspicion to this diagnosis. Practically, negative laparotomy in this patient has excluded a catastrophic bowel accident and ensured that the only pathology was gastric fundus necrosis; whether it was due to infection or not; to account for presentation and three gas signs in this patient. Failure to isolate infective organisms may not be enough to exclude infection as the patient had already started on antibiotics at the emergency unit. An infective gastric necrosis is another possibility. Although accurate diagnosis is important, the immediate, crucial action is to rule out a correctable intra-abdominal pathology, which we have done, to achieve a safe outcome for our patient. In conclusion, the combination of pneumomediastinum, gastric emphysema and HPVG is associated with partial gastric wall necrosis. The immediate work-up diagnosis is very important so as to plan the appropriate management, and to exclude or avoid delay in identifying and repairing for a possible surgical catastrophe.

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