Rare disease

Portal venous gas: is it a premorbid indicator?

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Summary

A 72-year-old Caucasian man presented with subtle and insidious symptoms of pain in the left iliac fossa and vomiting. The first clinical impression was of diverticulitis but he did not improve with conservative management. An initial contrast tomography (CT) scan demonstrated no diagnostic features. Four days later, a repeat CT scan demonstrated intramural gas in the ascending colon and in the intrahepatic portal venous radicles. There was a thrombus in the superior mesenteric artery with infarction of the caecum and ascending colon. An urgent right hemicolectomy was carried out. Although there was resolution of the thrombus and air in the portal system, the patient died 8 days later from intestinal haemorrhage.

BACKGROUND

Hepatic portal venous gas (HPVG) is a rare sign. If demonstrated, the diagnosis of ischaemic colitis should be considered and aggressive surgical management is indicated if appropriate.

CASE PRESENTATION

A 72-year-old Caucasian man presented to the emergency department with sharp central abdominal pain, which had been worsening over the past 14 days. The pain radiated to the left flank and had been associated with nausea and bilious vomiting over the past 5 days.

His co-morbidities included hypertension, ischaemic heart disease, previous coronary angioplasties, transient ischaemic attacks, type 2 diabetes, hypercholesterolaemia and diverticulosis. He was a non-smoker, non-drinker, lived alone and was independently mobile.

Examination of the cardiovascular system was unremarkable and respiratory examination revealed only slightly reduced air entry in the right basal region. His abdomen was soft and non-distended. There were no palpable masses or herniae and he was tender in the right hypochondrium and left iliac fossa without peritonism. Digital rectal examination was normal with no blood or mucus.

INVESTIGATIONS

Laboratory findings upon admission included normal full blood count and urea and electrolytes. Blood chemistry tests showed bilirubin levels of 35 µmol/l, alkaline phosphatase 132 U/l, γ-glutamyl transferase 348 U/l and C reactive protein 328 mg/l. An initial ECG, CT scan of the abdomen, pelvis and head, and MR cholangiopancreatography showed no significant findings.

A repeat CT of the abdomen after 4 days showed significant dilatation of the large bowel with intramural gas in the ascending colon (figure 1A), gas in the intrahepatic portal venous radicles (figure 1C) and infarction of the

caecum and ascending colon with thrombotic occlusion of the superior mesenteric artery.

DIFFERENTIAL DIAGNOSIS

Diverticulitis, cholangitis, acute cholecystitis, stroke and mesenteric ischaemia.

TREATMENT

Initially, on admission, his management was supportive, with fluid resuscitation, analgesia and intravenous antibiotics. After 4 days when his clinical condition had not improved, a repeat CT was performed. The findings of HPVG and pneumatosis intestinalis, when taken with the clinical picture, suggested mesenteric ischaemia. Therefore, the patient was taken to theatre for urgent laparotomy.

At operation, the ileum and ascending colon were necrotic up to the level of the hepatic flexure. A right hemicolectomy was performed. Histological examination of excised ileum, caecum and colon showed acute ischaemic enterocolitis. The patient was treated with antibiotics, analgesia and anticoagulants. He was extubated less than 24 h postoperatively and made a good initial recovery. Five days postoperatively, a repeat CT showed complete resolution of the portal pyaemia and a reduced thrombus size seen in the superior mesenteric vessel. The ileo-colic anastomosis was intact.

OUTCOME AND FOLLOW-UP

Unfortunately, at 7 days postoperatively, the patient had a catastrophic lower gastrointestinal bleed and despite aggressive resuscitation he died the following day. At post-mortem, the cause of death was shown to be intestinal bleed secondary to ischaemia.

DISCUSSION

The first case of HPVG in infants with necrotising enterocolitis was described by Wolfs and Evans in 1955.¹ The first reported adult case of HPVG was seen in the small bowel

Figure 1 (A) Coronal CT image showing marked dilatation of the large bowel with intramural gas collections within the ascending colon and caecum (arrows). (B) Coronal CT image showing air in the intramural intestinal wall and portal venous gas. (C) Axial CT image showing gas in the intrahepatic portal veins.

ischaemia due to superior mesenteric artery thrombosis.² Since then, around 100 cases of HPVG related to a variety of causes have been reported. Advances in imaging have led to an increased rate of diagnosis of HPVG caused by a number of pathologies (table 1). Mortality varies according to the cause; bowel ischaemia is the most common cause of HPVG and is associated with the highest mortality (75–90%).^{3–4}

The pathogenesis of HPVG is unknown but two hypotheses have been postulated. An increase in intraluminal pressure along with disruption of intestinal mucosa may cause luminal air to enter the capillary veins.⁵ Alternatively, pneumatosis intestinalis may be microbial in origin; analysis of the gas reveals hydrogen, which strongly supports a microbial cause.⁶

The initial management is related to the underlying cause. A conservative approach with close monitoring and intravenous antibiotics is the initial plan of action for most causes. A laparotomy is the second-line of management if unresponsive. Surgical intervention with laparotomy is the first-line management for peritonitis, bowel perforation and bowel ischaemia.

The prognosis of HPVG is variable dependent on the cause. A mortality of 100% within 48 h is expected if seen on CT and the cause is an ischaemic bowel.⁷ Histologically, the case presented had areas of mucosal, submucosal and entire bowel wall ischaemia, which is a predictor of a high mortality. The relevant specific CT findings were consistent with pneumatosis intestinalis, bowel dilatation and superior mesenteric artery ischaemia. Other findings may include bowel wall thickening, mesenteric oedema and mesenteric venous embolism. Clinically, the patient may have a history of cardiac arrhythmia, hypercoagulable state, recent operation presenting with bowel distension and metabolic acidosis.

The presence of ischaemic colitis and HPVG is considered a premonitory state. The high mortality is explained by the widespread systemic effect with irreversible shock from bowel ischaemia. The prognosis remains very poor and is unaltered by the improved radiological findings. There should be a high index of suspicion in older patients with co-morbidities (eg, diabetes, ischaemic heart disease). Hence, prompt recognition and diagnosis combined with early aggressive management is vital to patient survival.

Table 1 Recognised causes of hepatic portal venous gas (portal pyaemia).^{3–4 8–12}

Vascular	
	Bowel ischaemia
	Blood loss
	Superior mesenteric artery syndrome
Inflammatory	
	Inflammatory bowel disease
	Diverticulitis
	Cholangitis
	Acute pancreatitis
	Acute appendicitis
Infective	
	Infective colitis
	Abdominal abscess
Neoplastic	
	Colonic carcinoma
	Gynaecological malignancy
Iatrogenic	
	Barium enema
	Endoscopic retrograde cholangiopancreatography
	Upper gastrointestinal endoscopy
	Colonoscopy
	Percutaneous endoscopic gastrostomy tube placement
	Intestinal ileus
	Laparoscopy
Traumatic	
	Blunt abdominal trauma
Other	
	Mechanical small bowel obstruction
	Gastric dilatation
	Volvulus
	Gastric ulcer
	Liver transplantation

Learning points

- The importance of recognising portal venous gas on imaging.
- Its relevance in making a diagnosis of ischaemic colitis.
- The need for aggressive treatment of the underlying cause.
- High mortality in patients with HPVG associated with ischaemic colitis.

Competing interests None.

Patient consent Obtained.

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