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Paper

Hepatic portal venous gas – three non-fatal cases and review of the literature

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ABSTRACT

Background: Hepatic portal venous gas is a rare imaging finding most commonly associated with intestinal ischaemia and high mortality. Increased use of advanced imaging techniques has resulted in increased reporting and recognition of hepatic portal venous gas. Advanced imaging can also recognise the many associated pathologies which have variable management strategies and prognoses.

Methods: We report 3 non-fatal cases and review the pathogenesis, aetiology, diagnosis, management and prognosis of hepatic portal venous gas.

Conclusion: Once considered an indication for urgent surgery, hepatic portal venous gas is a rare imaging finding. More recently, HPVG has been recognised to be associated with various benign causes many of which may be treated non-operatively. However, intestinal ischaemia remains the most common cause and the most important to exclude. CT is the diagnostic modality of choice. The underlying cause determines the treatment strategy and outcome.

BACKGROUND

Hepatic portal venous gas (HPVG) is a rare imaging finding first described in 1955 in neonatal necrotising enterocolitis[1]. A subsequent review of adult cases concluded that this was an ominous finding – usually indicating intestinal ischaemia, necessitating urgent laparotomy and mortality of 75%[2]. In more recent decades, in particular with the advent of computed tomography (CT), HPVG has been increasingly recognised. It has been associated with various abdominal pathologies and a lower overall mortality than previously reported. We report 3 non-fatal cases and review the pathogenesis, aetiology, diagnosis, management and prognosis of HPVG.

METHOD

The clinical notes and imaging of 3 non-fatal cases of HPVG recently diagnosed in our hospital were reviewed. PubMed, PubMed Central and BioMed Central databases were searched using the terms 'hepatic portal venous gas' and 'portal venous gas'. A literature review of articles in the English language regarding HPVG in adults was conducted. References cited in articles were also reviewed and 45 relevant articles were selected.

CASE 1: HPVG WITH ACUTE PANCREATITIS AND GASTROINTESTINAL DILATATION

A 76-year-old man was admitted with upper abdominal pain radiating through to the back and vomiting. He had a history of alcohol excess, liver cirrhosis and ischaemic heart disease. Physical examination revealed tachycardia and right hypochondrial tenderness. Laboratory data showed deranged liver function and hyperamylasaemia of 984 U/l (25-125 U/l). A diagnosis of acute pancreatitis was made and abdominal ultrasonography was performed to exclude gallstones. This showed gallbladder sludge, a distended stomach and gas bubbling through the hepatic portal veins (Figure 1). CT demonstrated gross fluid distension of the oesophagus, stomach, duodenum and proximal jejunal loops. Gas was seen within the mesenteric and hepatic portal veins (Figure 2). No biliary or pancreatic abnormality was identified.

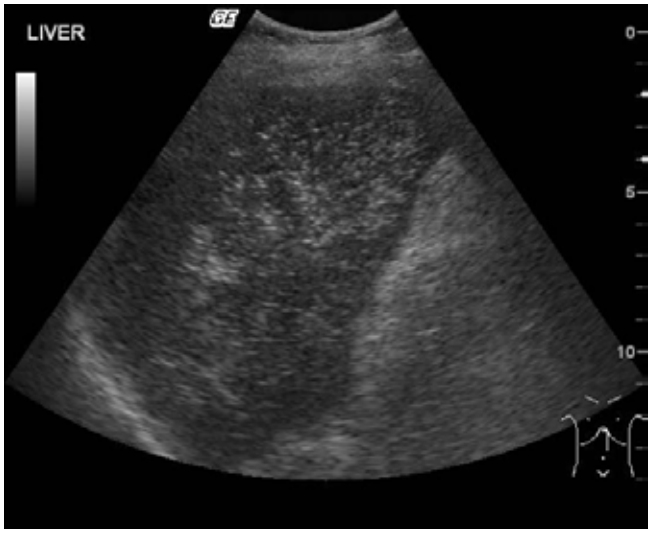

Fig 1. Multiple non-shadowing echogenic foci consistent with intrahepatic portal venous gas.

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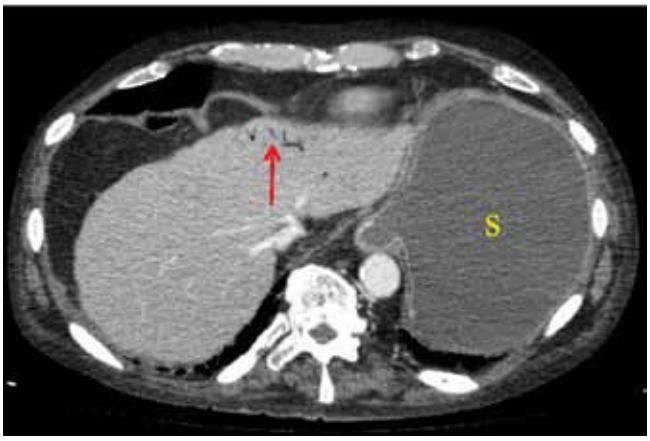

Fig 2. Branching low-attenuation areas within 2cm of the left hepatic lobe capsule in keeping with HPVG (red). Markedly distended and fluid-filled stomach (S).

The patient was treated with nasogastric decompression, intravenous fluids and analgesia. A gastrografin meal and follow-through at 6 days showed a normal calibre stomach and small bowel. His symptoms resolved and he was discharged after 9 days.

CASE 2: HPVG WITH ABDOMINAL HAEMATOMA AND GASTRIC DILATATION

A 44-year-old female underwent an elective Roux-en-Y hepaticojejunostomy for a benign biliary stricture. Four days post-operatively she developed right-sided abdominal pain and vomiting. On examination she was found to be pale, tachycardic and hypotensive. Laboratory investigations revealed: haemoglobin of 7.2 g/dl (11.5-16.5 g/dl), leukocytosis of 35.2x10^9/l (4.0-10.0 x10^9/l) and elevated C-reactive protein of 459mg/l (1-10mg/l). Two units of packed red cells were transfused and intravenous antibiotics were commenced.

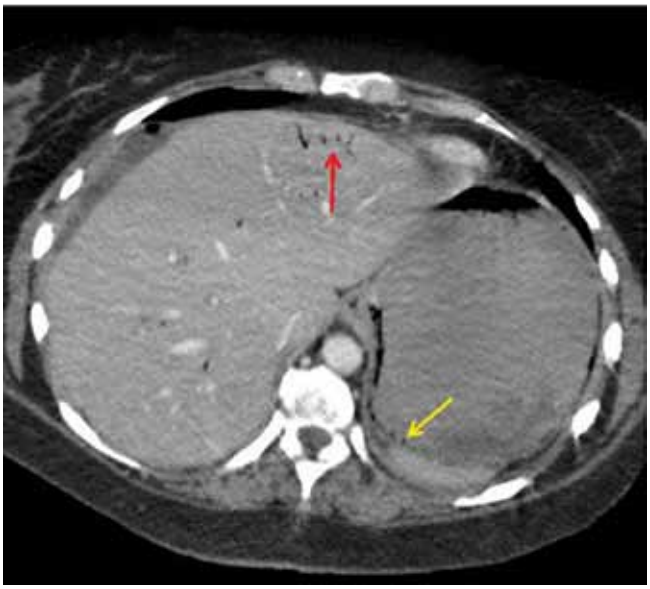

Fig 3.
Intra-abdominal haematoma (H). Linear gas collections (yellow) within the gastric wall consistent with gastric pneumatosis. HPVG in the anterior periphery of the left lobe (red) .Gas within the medial gastric wall (yellow).

Abdominal CT showed a large right-sided intra-peritoneal haematoma. The stomach was markedly distended and gas was seen within the wall of the stomach and oesophagus. Gas was identified peripherally within both lobes of the liver (Figure 3).

Naso-gastric decompression and urgent laparotomy were performed. The haematoma was evacuated, haemostasis achieved and a further drain inserted. The stomach did not appear ischaemic. The naso-gastric tube was removed 2 days later and a further CT at 4 days showed resolution of the gastric distension, pneumatosis and HPVG. The patient was discharged 6 days later.

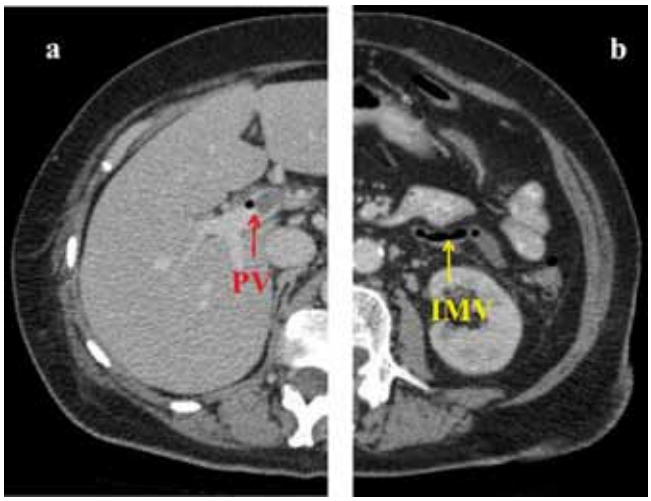

Fig 4.
a. Gas and thrombus in the portal vein (PV)
b. Gas in the inferior mesenteric vein (IMV)

CASE 3: HPVG WITH ACUTE DIVERTICULITIS, INTRA-ABDOMINAL ABSCESS & SEPTIC THROMBOPHLEBITIS

A 67-year-old female presented with lower abdominal pain, vomiting and rigors. She had a past history of superior mesenteric venous thrombosis 9 years previously resulting in mesenteric infarction necessitating ileo-caecal resection followed by anti-coagulation for 6 months. Pulse rate was 128/min, blood pressure 103/59mmHg and temperature 37.8°C. She had left iliac fossa tenderness on examination. Laboratory data revealed a white cell count of 11.7x10^9/l (4.0-10.0 x10^9/l) and C-reactive protein of 363 mg/l (1-10 mg/l). CT of the abdomen and pelvis identified sigmoid diverticulitis with an adjacent 3.2cm gas and fluid-filled collection consistent with a diverticular abscess. Inferior mesenteric, splenic and portal venous gas was evident (Figure 4). There was also peripheral intra-hepatic portal venous gas (Figure 5). A filling defect was seen within the inferior mesenteric and extra-hepatic portal veins in keeping with thrombus and a diagnosis of septic thrombophlebitis. Blood cultures were positive for Pseudomonas stutzeri and Streptococcus milleri.

She was treated with therapeutic low-molecular weight heparin and intravenous meropenum and gentamicin. Her symptoms resolved, inflammatory markers improved and she was discharged 2 weeks later on lifelong oral anticoagulation.

PATHOGENESIS

The mechanism of HPVG is not fully understood. Theories include: (i) migration of swallowed gas via mural capillaries into the portal venous circulation due to high gastrointestinal luminal pressure. This is the most likely mechanism in Cases 1 and 2 where marked gastric dilatation appear to have been precipitated by pancreatitis and intra-abdominal bleeding respectively; (ii) disruption of intestinal mucosa with passage of gas into the mesenteric venous system from gas-forming organisms within the bowel lumen; (iii) the presence gas-forming bacteria either from an abscess or porto-mesenteric pyelophlebitis[2-5]. In Case 3, where there was diverticulitis with an associated abscess, both theories (ii) and (iii) could explain the development of portal venous gas. In many cases these factors appear to contribute in combination[6]. Gas within the portal vein then passes centrifugally via the intra-hepatic portal veins to the hepatic periphery.

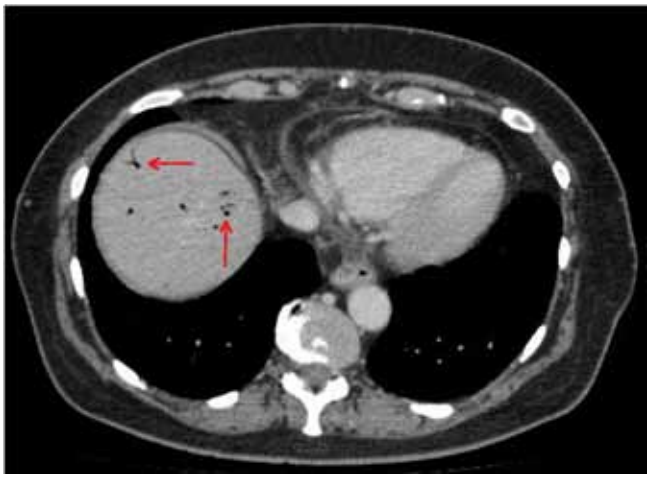

Fig 5. HPVG in the superior periphery of the right hepatic lobe.

AETIOLOGY

Mesenteric thrombosis with intestinal necrosis is still the most common underlying cause and the most important diagnosis to exclude. Recent studies have recognised other common pathologies associated with HPVG (Table 1) [3,6,7]. Many other causes have been reported and together account for approximately 15% of cases (Table 2).

DIAGNOSIS

HPVG is a rare imaging finding - just 28 cases were identified on review of 33,000 CT scans in 2 centres[33,34]. It is typically identified on plain x-ray, ultrasonography (US) or CT. The clinical presentation and examination findings are those of the underlying aetiology.

PLAIN RADIOGRAPHY

HPVG was originally described as a plain radiographical sign. It appears as branching radiolucencies extending to the liver periphery[2]. It may be detected in up to 12.5% of cases but requires the presence of large quantities of gas and is often a subtle finding[5,6,35]. A left lateral decubitus view increases sensitivity[2]. The presence of HPVG on plain x-ray has been considered a poor prognostic sign, usually associated with intestinal infarction[4,5]. Features of the underlying cause may also be evident such as marked pneumatosis intestinalis, gastro-intestinal oedema and dilatation or paucity of luminal gas.

Common causes of HPVG[3,4,7]

Intestinal necrosis	43-70%
Gastrointestinal dilatation	9-12%
Gastric dilatation	
Ileus	
Mechanical obstruction	
Pseudo-obstruction	
Gastrointestinal inflammation	8-16%
Diverticulitis	
Inflammatory bowel disease	
Sepsis	7-11%
Intra-peritoneal abscess	
Septic thrombophlebitis	
Endoscopic procedures	2-4%
Colonoscopy	
ERCP	
Percutaneous gastrostomy	
Ultrasonic biopsy	
Variceal banding/sclerotherapy	
Peptic ulcer disease	4%
Blunt trauma	3%
Abdomino-pelvic malignancy	2-3%
Oesphago-gastric carcinoma	
Colonic carcinoma	
Leiomyosarcoma	
Ovarian carcinoma	

US

On ultrasound scanning, HPVG appears as hyper-echoic, dot-like or streak-like foci flowing within the portal veins or liver parenchyma[7,36]. It is a rapid, low-cost, low-radiation method with comparable sensitivity and accuracy to CT[4]. Sensitivity may be increased if colour Doppler flow imaging is also utilised[37,38]. US also offers dynamic imaging of the centrifugal flow of portal gas to the hepatic periphery thus differentiating from biliary gas[39].

CT

With the increased use of abdominal CT scanning, HPVG has been more frequently diagnosed. Small volumes of gas can be detected and the application of 'lung-window' settings aids identification[4,5,34]. Gas is predominantly seen within the portal veins of the non-dependant left lobe and anterior right lobe[3,34,40]. Branching low-attenuation tubular areas are seen within 2cm of the hepatic capsule[6]. HPVG can be distinguished from intra-hepatic pneumobilia which is

detected centrally within the liver rather than extending to the peripheral parenchyma. It is highly sensitive and considered the gold standard imaging modality as it also offers the advantage of early detection of associated pathology[3,5,37]. In particular, dilatation and inflammation of the digestive tract, intra-peritoneal abscess and features of bowel ischaemia, such as pneumatosis intestinalis, may be demonstrated.

Other reported causes of HPVG (~15%)

Iatrogenic
- Barium enema[8]
- Cardiopulmonary resuscitation[9]
- Gastrojejunostomy leak[10]
- Haemodialysis[11,12]
- Hepatic artery embolisation[13]
- Intra-aortic balloon pulsation[14,15]
- Liver transplantation[16]
- Percutaneous liver biopsy[17]
- RF ablation of liver metastases[18]

Pancreaticobiliary
- Cholangitis[19,20]
- Pancreatitis[21]

Drugs
- Chemotherapy[22,23]
- Colchicine toxicity[24]

Miscellaneous
- Bronchopneumonia[25]
- Caustic ingestion[26]
- Cryptosporidium[27]
- Cystic fibrosis[28]
- Diabetic ketoacidosis[29]
- Emphysematous pyelonephritis[30-32]
- Hyperbaric decompression[33]
- Seizures[34]

MANAGEMENT AND PROGNOSIS

Early studies concluded that the diagnosis of HPVG was an ominous finding which necessitated urgent laparotomy. This was due to the association with bowel infarction and high mortality of at least 75%[2]. With advanced imaging techniques and the reporting of many non-life-threatening causes, subsequent authors questioned the need for immediate surgery[41-43]. More recent studies report an overall mortality of 29-39%[6,33,43]. The apparent decrease in mortality can be explained by the increased usage and sensitivity of CT. Early detection of HPVG and the identification of the precipitating diagnosis have facilitated timely and targeted treatment. Rather than being an indication for surgery, the presence of HPVG should be considered a diagnostic sign.

All three patients we present had clinical presentations indicating significant intra-abdominal pathology. The decision whether to operate or treat conservatively was based on careful correlation of the clinical, laboratory and radiological findings. In Case 2, with evidence of significant post-operative bleeding and possible gastric ischaemia, urgent laparotomy was indicated. Conservative management was adopted in the other cases. In Case 1, the history of excessive alcohol ingestion, pain radiating to the back, combined with hyperamylasaemia and the absence of gastrointestinal pneumatosis suggested a diagnosis of pancreatitis rather than mesenteric ischaemia. Conservative management with regular re-assessment was therefore indicated. The patient in Case 3 again was treated conservatively, primarily on clinico-radiological evidence of locally complicated diverticulitis rather than generalised peritonitis and the absence of CT signs of intestinal ischaemia. It is recognised that CT cannot definitively exclude or confirm intestinal ischaemia, however in the clinical context of these two cases, surgery was felt to be unjustified.

Management algorithms have been proposed[3,4]. Indications for immediate surgery include clinical and/or radiological signs of intestinal necrosis (75-85% mortality) and the presence of HPVG on plain x-ray (75% mortality)[5]. Close monitoring and a low threshold for surgery are advised in patients with gastro-intestinal distension, ulceration or abscess without peritonitis, as mortality approaches 20-30%[2,6]. Mortality in the remaining group of 'benign' aetiologies is extremely low. A conservative approach may be adopted in these cases[3,4]. This combines close observation, intravenous fluid, antibiotic therapy, and naso-gastric decompression when required.

CONCLUSION

Once considered almost pathognomic of intestinal necrosis, high mortality and an indication for urgent surgery, HPVG is a rare imaging finding. More recently, it has been associated with various non-fatal causes as demonstrated by the cases we have reported. However, intestinal ischaemia remains the most common cause. CT is the diagnostic modality of choice. The underlying cause of HPVG determines the treatment strategy and outcome.

The authors have no conflict of interest.

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