

Hepatic portal venous gas complicating septic thrombophlebitis of the superior mesenteric vein

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Hepatic portal venous gas is a rare radiological finding with a wide spectrum of underlying pathologies. We describe a case of hepatic portal venous gas due to septic thrombophlebitis of the superior mesenteric vein. The clinical management of portomesenteric venous gas and the importance of computed tomography in delineating its underlying causes are discussed.

Introduction

Portal venous gas is a rare radiological finding first described in infants with intra-abdominal catastrophes in 1955,¹ and subsequently reported in adults in 1960.² In the early reports hepatic portal venous gas was usually related to mesenteric ischaemia associated with extensive bowel necrosis and a fatal outcome. Because of the high mortality rate, the presence of hepatic portal venous gas was considered an ominous sign and urgent laparotomy was recommended.³ However, recent reports have shown that portal venous gas can be due to a range of disease processes, some of which do not necessarily require surgical intervention.^{4,5} We report a rare case of hepatic portal venous gas secondary to septic thrombophlebitis of the superior mesenteric vein. The patient was successfully treated with antibiotics and anticoagulants. The contribution of the radiological findings toward making a prompt diagnosis and in guiding further management of portal venous gas is emphasised.

Case report

In March 2006, a 52-year-old man presented with a history of fever, periumbilical pain, and tea-coloured urine for 3 days. He was a heavy alcohol drinker but had no history of acute pancreatitis or hepatitis. He had been in good health prior to this illness. A physical examination upon admission showed an icteric patient with a fever of 38.5°C and mild diffuse abdominal tenderness, but no rebound or guarding suggestive of peritonitis. His liver was non-tender and mildly enlarged, 4 cm below the costal margin. The bowel sound was normal and he was haemodynamically stable. Initial investigations showed an abnormal, cholestatic liver function pattern with a bilirubin level of 93 (reference range, 2-23) µmol/L, alkaline phosphatase of 225 (reference range, 53-128) IU/L, gamma-glutamyl transpeptidase of 329 (reference range, 11-50) IU/L, and alanine transaminase of 37 (reference level, <41) IU/L. A full blood count showed a mild neutrophilia of 8.2×10^9 /L (reference range, $1.8-7.5 \times 10^9$ /L) and a marked thrombocytopenia of 20×10^9 /L (reference range, $150-500 \times 10^9$ /L). A blood film showed thrombocytopenia with occasional large platelet forms. The coagulation profile was impaired with a prothrombin time of 12.3 (reference range, 9.9-11.9) seconds and an activated partial thromboplastin time of 40.4 (reference range, 26.7-35.5) seconds. The serum fibrinogen level was raised to 6.6 (reference range, 2.0-4.0) g/L and the D-dimer was 0.5-1.0 (reference level, <0.5) µg/mL. The serum amylase and lactate levels were normal all along. Arterial blood gases revealed no metabolic acidosis. The overall picture was compatible with disseminated intravascular coagulation due to sepsis.

Abdominal and chest X-rays showed normal bowel shadows with no free gas under the diaphragm. Urgent ultrasonography revealed multiple reverberation artefacts at the subcapsular region of the dome of the left lobe, and between the middle and right hepatic veins, compatible with gas collections. The biliary tree was not dilated and no aerobilia or ductal stones were identified. Plain computed tomography of the abdomen showed peripheral branching tubular gas in the portal vein and gas along the superior mesenteric vein (Figs 1a and 2a). Computed tomography with contrast showed a filling defect along the superior mesenteric vein which extended cranially to reach the lower portal vein near the confluence (Fig 1b). The visible bowel loops showed colonic diverticuli at the hepatic flexure without obvious inflammation or pericolonic abscesses (Fig 2b). There was no abnormal bowel dilatation, intramural gas, nor

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肝門靜脈呈現氣體併發上腸系膜靜脈細菌感染血栓性靜脈炎

肝門靜脈呈現氣體是很罕有的放射學情況，有多種潛在的病理原因。本文描述一宗因上腸系膜靜脈細菌感染血栓性靜脈炎引致肝門靜脈呈現氣體的病例，並討論門腸系膜靜脈呈現氣體的臨床治療，和以電腦掃描找出其潛藏病因的重要性。

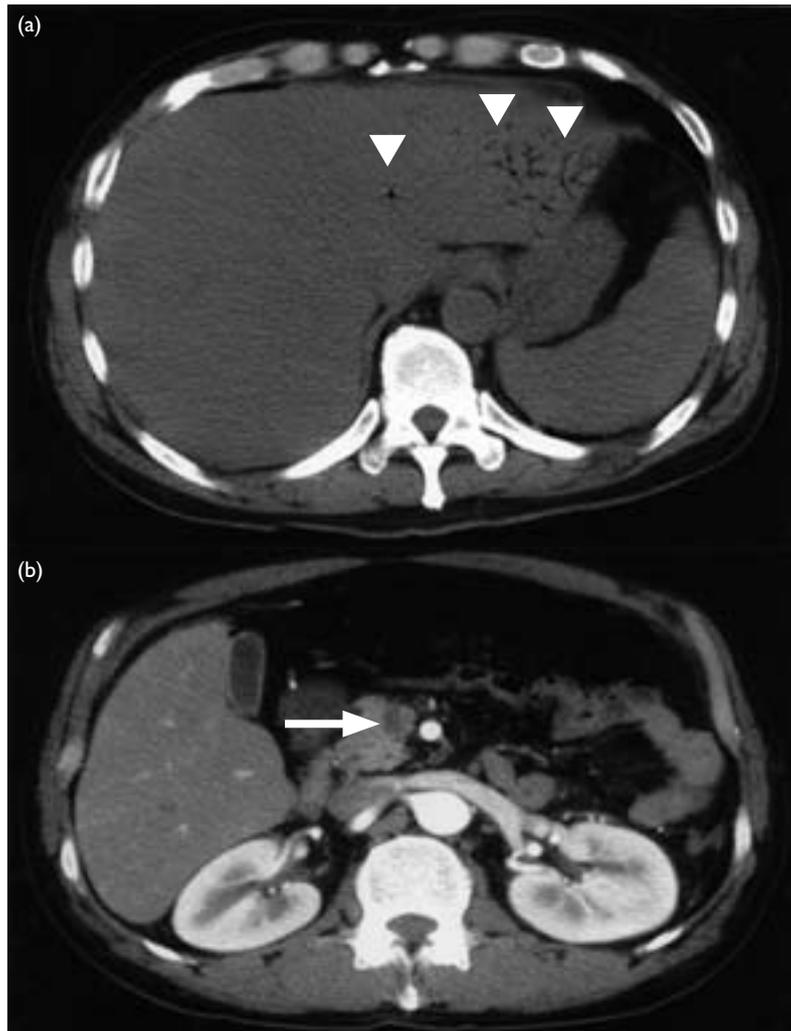


FIG 1. (a) Plain computed tomography of the abdomen demonstrates gas in the portal vein with the typical appearance of branching gas densities over the periphery of the liver (arrowheads). (b) Contrast-enhanced computed tomography shows a thrombus in the superior mesenteric vein (arrow)

thickened bowel wall. The pancreas appeared normal and no intra-abdominal collection was identified. The patient was treated conservatively with intravenous triple antibiotics including cefuroxime, ampicillin, and metronidazole. Subsequently, blood cultures grew *Bacteroides fragilis*. The diagnosis was hepatic portal

venous gas due to septic thrombophlebitis of the superior mesenteric vein. He was put on low-molecular-weight heparin followed by warfarin once his platelet count had returned to normal 1 week after initial presentation. The international normalised ratio was kept at around 2.0. Colonoscopy was deferred for 4 weeks as acute diverticulitis is a contra-indication for the procedure. Colonoscopy later confirmed the presence of multiple diverticuli over the hepatic flexure, and found no features of ischaemic bowel disease or colonic tumours. The patient's liver function tests and full blood counts returned to normal. Two months later, computed tomography of the abdomen showed complete resolution of the portal venous gas and superior mesenteric venous thrombosis. Six months later, all prothrombotic screening tests including anti-phospholipid syndrome, Factor V Leiden, protein C, protein S, and anti-thrombin III were normal after stopping warfarin; hence anti-coagulation was discontinued.

Discussion

Gas in the hepatic portal venous system can be due to either gas under pressure in the bowel lumen or an alteration of the bowel mucosa allowing gas to enter the portal system through the mesenteric veins. Severe intra-abdominal sepsis involving gas-producing bacteria also results in portomesenteric venous gas.⁶ Hepatic portal venous gas is not a specific disease entity, but merely a diagnostic clue in patients with acute abdominal pathology. It is not, by itself, a predictor of mortality. Rather, the mortality risk correlates with the underlying cause of portal venous gas.⁴ The most commonly reported pathologies associated with portal venous gas include mesenteric ischaemia, post-procedural complications, Crohn's disease, and intra-abdominal abscesses. It is also associated with colon cancer, gastric ulceration, acute pancreatitis, and sigmoid diverticulitis.⁴⁻⁶ It is important to differentiate life-threatening mesenteric ischaemia from other more benign non-ischaemic causes. Patients with portal venous gas should undergo a complete history and physical examination to ascertain the underlying diseases. Computed tomography not only helps detect small amounts of portal venous gas, making it possible to demonstrate portomesenteric venous gas in the earlier stage of the disease, but also elucidates the underlying pathology and guides the subsequent clinical management. Urgent laparotomy is only mandatory in patients in whom intestinal ischaemia or infarction is suspected on the basis of radiological and clinical findings.

In our patient, portal venous gas was first detected by ultrasonography and later confirmed by computed tomography. Intrahepatic portal venous gas should be differentiated from aerobilia. In the latter condition, the air is usually located centrally around the confluence of the common hepatic duct. In contrast, collections of portal venous gas are smaller, more numerous, and

are seen in the liver periphery. The findings of gas and thrombus in the superior mesenteric vein together with positive blood cultures supported the diagnosis of septic thrombophlebitis. The likelihood of mesenteric ischaemia in our patient was low as he had neither predisposing factors, peritoneal signs, nor metabolic acidosis. Besides, his bowel shadows were normal on the abdominal X-ray. Computed tomography of the abdomen revealed no occlusion of the splanchnic vasculature, bowel distention, bowel wall thickening, pneumatosis intestinalis, or mesenteric oedema suggestive of bowel ischaemia. Acute pancreatitis was ruled out by the normal serum amylase level together with a normal pancreas seen on computed tomography.

Septic thrombophlebitis of the portomesenteric vein is a rare but serious complication of an intra-abdominal inflammatory process. The clinical features of thrombophlebitis are generally non-specific. Patients usually develop fever, chills, and right upper quadrant tenderness. Hepatomegaly and jaundice may be present. Impaired liver function tests and leukocytosis are common. Most frequently, the diagnosis of thrombophlebitis has not been suspected, and a thrombosed portal vein is unexpectedly found on computed tomography.⁷ The common aetiologies include diverticulitis,⁸ appendicitis,⁹ pancreatitis,¹⁰ cholecystitis, and cholangitis.⁶ Septic thrombophlebitis has been associated with portal venous gas in patients with diverticulitis, ischaemic bowel disease and Crohn's disease.^{11,12} Patients with pre-existing thrombosis of the portal venous system due to a hypercoagulable state may also develop thrombophlebitis if a septic focus sets in. In our patient, diverticulitis was the likely underlying source of septic thrombophlebitis. Although computed tomography only revealed multiple colonic diverticuli without pericolonic abscesses or bowel wall thickening, the thrombotic branch of the superior mesenteric vein was the venous drainage of the affected portion of the colon. Furthermore, no other source of intra-abdominal sepsis was identified and prothrombotic screening tests were all negative.

Septic mesenteric thrombophlebitis following diverticulitis can be treated successfully with intravenous antibiotics if there are no other complications.¹³ For patients with other complications of diverticulitis, for instance, perforation, abscess,^{14,15} as well as septic shock, or poor response to medical therapy,¹⁶ surgery is indicated. The role of anticoagulation is to prevent thrombus propagation into the enteric system and hence reduce risk of recurrence and mesenteric ischaemia.¹⁷ Patients with thrombophlebitis and a hypercoagulable state due to malignancy or clotting factor deficiencies should be anti-coagulated. However, the benefit of anticoagulation in patients with normal clotting function remains controversial. Baril et al¹⁰ showed that none of the patients with pylephlebitis and a normal clotting profile developed ischaemic bowels. Our patient had a negative prothrombotic screen after stopping warfarin,

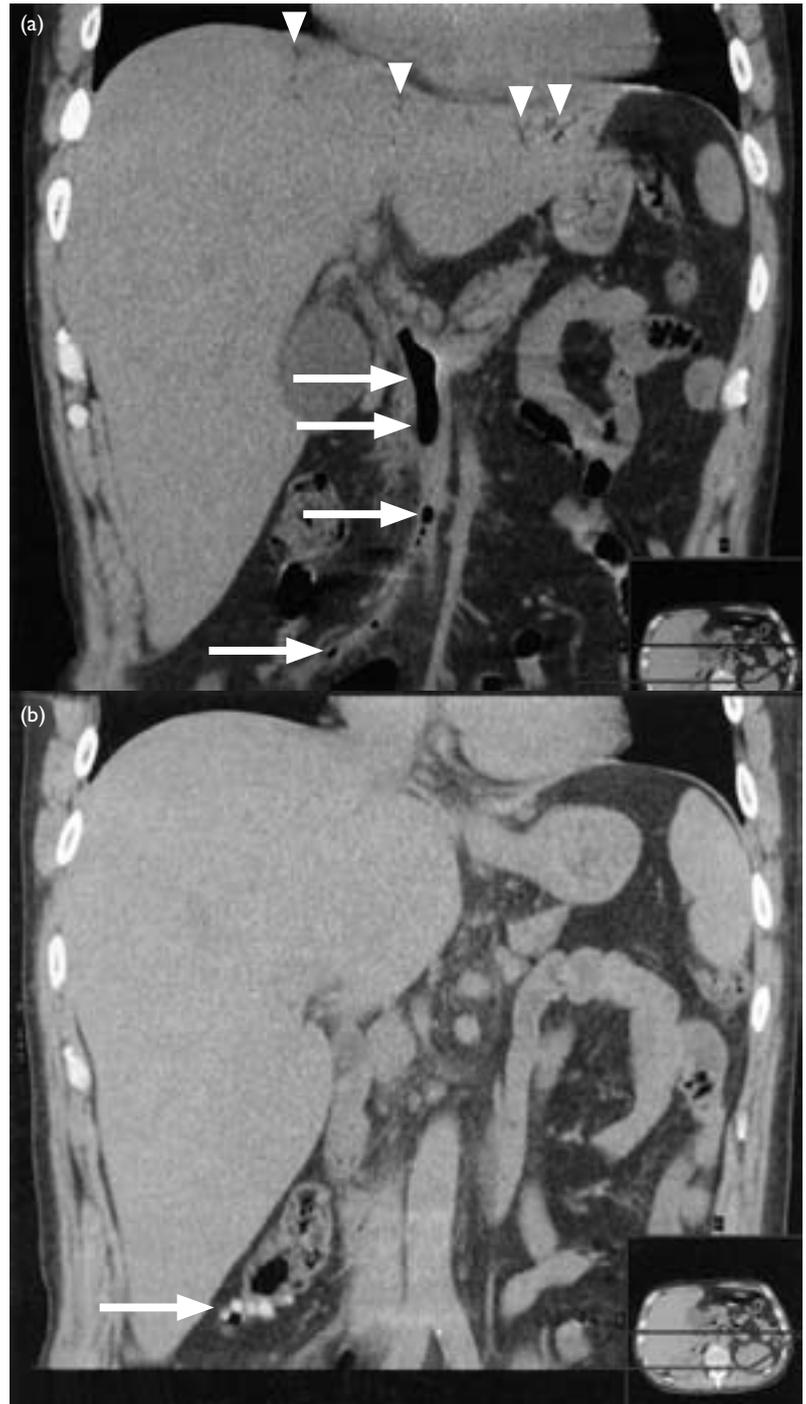


FIG 2. Reconstruction image of multi-slide computed tomography shows (a) gas in the superior mesenteric vein (arrows) extending into the hepatic portal vein (arrowheads), and (b) multiple diverticuli with faecaliths (arrow)

so anti-coagulation was discontinued.

In conclusion, hepatic portal venous gas is a rare radiological finding with a wide spectrum of underlying causes. Careful clinical assessment together with computed tomography is crucial for making an accurate diagnosis and initiating prompt treatment.

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