Portomesenteric venous gas in acute small bowel infarction associated with acalculous gangrenous cholecystitis

Aéroportie et aéromésentérie compliquant un infarctus mésentérique associé à une cholécystite gangrenée alithiasique

Hepatic portal venous gas (HPVG), an ominous radiologic sign, was first described by Wolf and Evans in 1955 in infants with necrotizing enterocolitis [1–5]. The mechanism inducing this gas in the portal vein is not well understood. It has numerous causes, predominantly mesenteric infarction, which is associated with poor prognosis [3,5]. Abdominal computed tomography (CT) has become the key imaging procedure used to diagnose acute bowel ischemia and to detect faint signs of portomesenteric venous gas [6–9]. The radiologic presentation has some particularities that physicians must be aware of to avoid delaying treatment. We report a case of extensive portomesenteric gas due to acute small bowel infarction associated with acalculous gangrenous cholecystitis and discuss the pathogenesis and radiologic presentation of this rare entity.

Case report

A 77-year-old man presented to the emergency department complaining of abdominal fullness and bilious vomiting. He reported that he had had no bowel movements over the preceding 7 days and had developed escalating, diffuse abdominal pain, bloating, and vomiting. His medical history included complete atrioventricular block, treated by a permanent pacemaker, as well as hypertension and congestive heart failure.

Physical examination revealed a body temperature of 37 °C, blood pressure of 80/45 mm Hg, a pulse rate of 125 beats/ min, and a respiratory rate of 25 cycles/min. The abdomen was distended and tympanitic, with no bowel sounds, rebound, or guarding. A simple umbilical hernia was also observed. The rectal examination was normal.

Laboratory studies revealed a hemoglobin value of 13.9 g/dL, a white blood cell count of 27,400 cells/µL, 90.1% of them neutrophils, and a prothrombin time of 17.4 seconds. His creatinine level was 150 µmol/L and his C-reactive protein level 171 mg/L. Arterial blood gas analysis showed a pH of 7.51, pCO₂ of 28.9 mm Hg, pO₂ of 65.4 mm Hg, HCO₃ of 23.4 mm Hg, and pO₂/FI of 115.

A contrast-enhanced abdominal CT showed multiple branching radiolucencies in the periphery of the liver (figure 1), large amounts of gas in the portal (figure 2a) and superior mesenteric veins (figure 2b) as well as its distal ramifications (figure 2c), and pneumatosis intestinalis at the mid-ileum (figure 2d). Abdominal CT also revealed circumferential thickening of the gallbladder wall and formation of a contiguous hepatic abscess (figure 2a). These findings strongly suggested intestinal infarction and acalculous cholecystitis.

Emergency surgery followed. Laparotomy showed extensive necrotic change along the Treitz ligament, from 1.60 to 2.35 m (figure 3), associated with acalculous gangrenous cholecystitis and a small abscess in the gallbladder bed. Pulsation of the superior mesenteric artery was detected. Thus, cholecystectomy and resection of 75 cm of necrotic small intestine were performed and an ileal stoma constructed.

The patient died 48 hours after surgery.

Discussion

HPVG is a rare radiologic finding. Retrospective reviews of CT scans identified 17 cases in 14,000 at one university medical center and 11 in 19,000 at another [7]. Its pathogenesis is not fully understood. Mechanical and bacterial origins have been proposed [1–7,10,11]. The former presumably involves the escape of gas due to increased pressure in the bowel lumen, with an ulcer, fissure, or mucosal tear serving as a portal for it to enter the intramural venous plexus and then the liver. Mucosal damage and bowel distention are important factors in this theory. About 85% of patients with HPVG have mucosal ulceration with bowel distention and increased intramural pressure. The second theory postulates that gas-forming bacteria, mainly the Clostridium type, enter the submucosa through mucosal tears and produce gas inside the intestinal wall and then enter the portal system, with or without related pyle-phlebitis. HPVG occurs in different clinical scenarios. The commonest cause is bowel necrosis (43%) [1,4,7,11,12]. In this case report, we described an association between an intestinal infarction with acalculous gangrenous cholecystitis not previously described in the literature. HPVG may occur secondary to an infection in the region drained by the portal system or contiguous to the portal vein, such as diverticulitis, appendicitis, or an intra-abdominal abscess. It can be found in some non-infectious conditions, some of which have been cured with...
**Figure 1**
Multiple branching radiolucencies in the periphery of the liver

**Figure 2**
a: large amounts of gas in the portal vein (arrow 1), circumferential wall thickening of the gallbladder (arrow 2) and hepatic abscess (arrow 3); b: gas in the superior mesenteric vein (SMV); c: gas in the distal ramification of SMV; d: pneumatosis intestinalis at the midileum

**Figure 3**
Operative finding: extensive necrotic change from 1 m 60 to 2 m 35 of the Treitz ligament
conservative management alone; these include bowel obstruction, hemorrhagic pancreatitis, gastric ulcer, blunt abdominal trauma, ulcerative colitis, Crohn disease, and complications of endoscopic procedures [1–3,7,9,11–14].

HPVG is usually diagnosed by plain abdominal radiography, CT, or ultrasonography. Of these imaging methods, CT is the most sensitive and specific for detecting HPVG and for demonstrating associated intra-abdominal disorders and coexisting abnormal air [3,4,14,15]. On tomographic images, HPVG appears as branching radiolucencies or hypodense areas within 2 cm of the liver capsule. This peripheral gas distribution is related to the centrifugal flow of blood into the liver. This radiographic aspect predominates in the left lobe because of its ventral location [1–7,9,12,14,16].

It is crucial to differentiate radiographically intrahepatic portal vein gas from air in the biliary tree (pneumobilia): because their therapeutic implications differ greatly, their misinterpretation can be catastrophic. Gas in the biliary tree is prevented from migrating peripherally and, due to the centripetal flow of bile, tends to collect in the large bile ducts at the hilum. It is also important to consider and rule out a history of endoscopic papillotomy, after biliodigestive anastomosis, cholecodochocystostomy, or biliary endoprosthesis [1,2,5,6,8,16,17]. With its excellent spatial and contrast resolution, a CT scan provides not only a conclusive diagnosis but its cause in most cases. Bowel necrosis is the primary cause of HPVG, accounting for 72% of diagnoses in the 1978 survey by Liebman et al., but only 43% of the diagnoses in reports of patients with HPVG surveyed by Kinoshita et al. in 2001 [1]. Abdominal CT with contrast has a sensitivity of 64% and a specificity of 92% in the diagnosis of intestinal ischemia [4]. The most specific ischemic changes visualized by CT are [4,6–10,17] intramural gas, bowel wall thickening greater than 3 mm, marked or absent enhancement of the bowel wall, dilation of the small bowel and colon, mesenteric fat stranding, ascites, and mesenteric arterial or venous thromboembolism. In cases of intestinal necrosis, gas usually produces an image of microbubbles or a band-like pneumatisos, while the images for patients who can safely be treated nonsurgically may be linear or clustered, larger and more spherical.

The most specific findings for acute bowel ischemia on CT are intramural gas and gas in the splanchnic and portal vasculature [17,18], but these have been reported in only 3–14% cases of acute bowel ischemia [8]. In the case reported here, CT showed multiple branching radioluencies in the periphery of the liver, large amounts of gas from the distal ramification of the superior mesenteric vein to the portal vein, and diffuse wall thickening over the ileum with pneumatisos intestinals.

HPVG has also been detected by ultrasonography. The typical features are highly echogenic particles, flowing within the portal vein, or poorly defined, highly echogenic patches within the background of the liver parenchyma, most apparent in the non-dependent part [5,12]. The high inter-operator variability of ultrasonography limits its usefulness.

Conventional radiography can find HPVG. However, its sensitivity is lower than that of CT or ultrasound, and it cannot discriminate air in the biliary tree from air in the intrahepatic portal vein [12–14]. The literature describing magnetic resonance imaging for diagnosing HPVG is still more limited [2]. The management of portal venous gas related to intestinal infarction includes appropriate antibiotics and an immediate laparotomy for the treatment of the primary septic source [2,11]. Therapeutic delay may lead to dissemination of the gas and systemic embolism [19]. Even with emergency surgery, prognosis is poor. The high mortality rate ranges from 75 to 90% [4–6,12,15]. Liebman et al. analyzed all cases of HPVG reported in the literature by 1978 and found an oft-cited mortality rate of 75% [2,7]. Kinoshita et al. reviewed the clinical data from 182 cases of this entity in adults from the literature and found mortality was highest for patients with HPVG associated with bowel necrosis (75%) [1].

Some factors are reported to predict a high rate of mortality, especially treatment delay and extent of bowel necrosis [1,3,4,7,11,14,16]. Mortality is reported to differ substantially between patients in whom transmuralinfarction involves only one bowel segment (14%) and those in whom the infarction involves two (71%) or three bowel segments (100%) [18]. The infarcted bowel segment in our patient covered 75 cm, a factor that undoubtedly explains his death, at least in part. Abdominal radiographs are not sensitive for early stage bowel ischemia detection, but they predict bowel infarction and a poor prognosis when intramural gas and HPVG are seen [5,10]. A mortality rate of 84% is reported for patients with portomesenteric venous gas on radiographs [18]. The presence of HPVG provides no information about the extent of intestinal necrosis [5,15], and has even been observed with reversible ischemia [6]. Thus, it is not itself a predictor of mortality [1].

**Conclusion**

Portomesenteric venous gas is a nonspecific radiological sign. Its causes are various, but bowel necrosis is the most common. Abdominal CT allows a diagnosis with a high degree of sensitivity and specificity and identifies the underlying cause. Urgent laparotomy is mandatory, to remove the infarcted intestine without delay to improve the prognosis. The mortality rate remains high and is related mainly to the extent of bowel necrosis.

**Conflict of interest statement:** none

**References**


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