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EDITORIAL

Hepatic portal venous gas: Physiopathology, etiology, prognosis and treatment

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Abstract

Hepatic portal venous gas (HPVG), an ominous radiologic sign, is associated in some cases with a severe underlying abdominal disease requiring urgent operative intervention. HPVG has been reported with increasing frequency in medical literature and usually accompanies severe or lethal conditions. The diagnosis of HPVG is usually made by plain abdominal radiography, sonography, color Doppler flow imaging or computed tomography (CT) scan. Currently, the increased use of CT scan and ultrasound in the inpatient setting allows early and highly sensitive detection of such severe illnesses and also the recognition of an increasing number of benign and non-life threatening causes of HPVG. HPVG is not by itself a surgical indication and the treatment depends mainly on the underlying disease. The prognosis is related to the pathology itself and is not influenced by the presence of HPVG. Based on a review of the literature, we discuss in this paper the pathophysiology, risk factors, radiographic findings, management, and prognosis of pathologies associated with HPVG.

Key words: Hepatic portal venous gas; Bowel ischemia/necrosis; Diverticulitis; Gastric pathologies; Ulcerative colitis; Abdominal computed tomography scan; Crohn's disease; Liver transplantation; Chemotherapy

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INTRODUCTION

Hepatic portal venous gas (HPVG), an ominous radiologic sign, was first described by Wolfe and Evens in infants with necrotizing enterocolitis (NEC)[1,2]. HPVG is associated with numerous underlying abdominal diseases, ranging from benign causes to potentially lethal diseases that require prompt surgical intervention[3-6].

The mechanism for the appearance of gas in the portal vein is not well understood. The proposed factors predisposing the portal venous system to the accumulation of gas include the following: (1) escape of gas produced by gas-forming organisms in the bowel lumen or in an abscess which then circulate into the liver or (2) the presence of gas-forming organisms in the portal venous system with passage of gas into the circulation[7].

The diagnosis of HPVG is usually made by plain abdominal radiography, sonography, color Doppler flow imaging, or computed tomography (CT) scan. The characteristic finding on abdominal plain film is a branching radiolucency extending to within 2 cm beneath the liver capsule. This is because of the centrifugal flow of portal venous blood, which carries portal venous gas peripherally, in contrast to biliary gas, which tends to collect centrally as a result of the centripetal movement of bile. Although HPVG may be diagnosed by conventional radiography, detection is difficult and it is easily overlooked[8]. Sonography, color Doppler flow imaging, and CT scan have been reported to be superior to abdominal radiographs in identifying HPVG. Sonography coupled to Doppler is very sensitive for HPVG detection and its follow-up, and can be useful as an initial screening examination[9,10]. However, use of ultrasound is limited because of its high inter-operator variability and lack of availability at all times. The typical ultrasonographic features of HPVG are (1) highly echogenic particles, flowing within the portal vein, or (2) poorly defined, highly echogenic patches within the hepatic parenchyma, which are most apparent in the non-dependent part[11].

Color Doppler flow imaging shows hyperechogenic foci moving within the lumen of the portal vein, producing sharp bidirectional spikes superimposed on the normal monophasic portal vein wave pattern. The CT scan has a high sensitivity for detection of HPVG and can detect the underlying pathology[12,13]. On scanographic images, HPVG is characteristically associated with peripheral gas lucencies, which branch out and are noted even within the last 2 cm beneath the liver capsule. This peripheral gas distribution is related to the direction of blood flow into the liver. It is crucial to differentiate it from pneumobilia, which is centrally located because of the biliary anatomy and the direction of bile flow[14,15]. Also, a CT scan can disclose gas in the bowel wall (pneumatosis intestinalis) and in the extrahepatic portal vein or its splanchnic vasculature.

The underlying clinical events associated with HPVG might be important factors contributing to patient survival and prognosis. Liebman *et al*[7] reported that HPVG is associated most commonly with bowel necrosis (72%), followed by ulcerative colitis (8%), intra-abdominal abscess (6%), small bowel obstruction (3%) and gastric ulcer (3%). This explains the high mortality rate (56%-90%) reported in association with HPVG[16,17]. Another factor affecting the outcome of these patients is the coexistence of a long-term chronic disease, such as chronic renal failure, diabetes mellitus or hypertension[13], which decreases immune functions and alters the intestinal microbial flora.

However, currently, the increased use of CT scan and ultrasound in the inpatient setting allows early and highly sensitive detection of such severe illnesses[16,18-20] and recognition of an increasing number of benign and non-life threatening causes of HPVG[20,21]. The prognosis is related to the pathology itself and is not influenced by the presence of HPVG[12].

In this paper, we discuss the pathophysiology, risk factors, management, and prognosis of pathologies associated with HPVG.

NEC

NEC is a disease of premature neonates, with 90% of cases occurring in infants born before 36 wk gestational age. In 10% of cases it occurs in full-term infants who have comorbidities predisposing them to decreased mesenteric perfusion[22]. This pathognomonic radiographic sign can be missed in extremely low birth weight (less than 1000 g) neonates, despite the gravity of the illness, because of absence of enteral feeding.

NEC is a multifactorial disease process resulting from the association of a hypoxic ischemic injury of the immature gastrointestinal tract and alterations in the microbiological intestinal flora[1]. Hypoperfusion in preterm infants has many etiologies (Table 1). It induces blood to shunt away from the bowels towards critical organs which may cause alterations in the mucosal barrier. These alterations, in combination with pathogenic microbiological intestinal flora proliferation exaggerated by feeding and antibacterial use, result in mucosal and/or transmural necrosis.

Table 1 Pathologies associated with hypoperfusion in neonates

Etiologies of hypoperfusion in preterm infants
Patent ductus arteriosus
Sepsis
Polycythemia
In utero cocaine exposure
Peri- or postnatal asphyxia
Respiratory distress syndrome
Congenital heart disease
Umbilical catheters use and exchange transfusion

The bacterial translocation and production of hydrogen gas into the bowel wall cause pneumatosis intestinalis[1] which can be seen radiographically as linear or circular lucencies within the intestinal wall[23]. As a consequence, the gas can embolize from the bowel wall through the mesenteric veins to the portal venous system and the non-dependent parts of the liver, particularly the left lobe and anterior segment of the right lobe.

Abdominal sonography is very specific and sensitive for early detection of portal and hepatic gas thus allowing early diagnosis and efficacious treatment of NEC. The micro-bubbles of gas appear as highly echogenic particles flowing within the portal vein and highly echogenic patches within the hepatic parenchyma. At a more advanced stage, HPVG can be seen on abdominal radiographs. Furthermore, HPVG may only be a transitional sign or can persist for longer than 2 d[24].

HPVG alone is not an indication for surgery[24], since 47% of infants with NEC and HPVG survive without operative procedure. Surgery is indicated when infants do not improve significantly despite medical treatment for several days, as well as when radiographs show persistent fixed dilated bowel loops or pneumoperitoneum which are, respectively, signs of bowel necrosis and perforation. In infants with a birth weight > 1500 g, laparotomy and resection of the necrotic intestine is generally the preferred approach. In very low birth weight infants < 1500 g, and unstable neonates, surgery is associated with a high rate of mortality and morbidity[22]. In this case, peritoneal drainage can be indicated as a definitive procedure or as part of the resuscitation phase prior to definitive laparotomy.

HPVG has been associated, in some studies, with a poorer survival rate. In their prospective study, Sharma *et al*[24] found that infants with HPVG were 1.4 times more likely to have severe NEC, but mortality rates did not differ from those of infants without HPVG.

HPVG is associated with severe lesions of the bowel wall and intra-mural gas that leads to muscular disruption and secondary stricture formation in up to 20% of all patients with NEC[22,23].

BOWEL ISCHEMIA

Bowel ischemia and/or infarction is a common and

Table 2 Etiologies of bowel ischemia

Etiologies	
Thromboembolism	Atherosclerosis
	Arterial dissection
	Aortic surgery
	Neoplasm, inflammatory or infectious causes
Vasculitis	Producing occlusion of large, medium and small arteries
Segmental mediolytic arteriopathy	Characterized by a non inflammatory arteriopathy causing lysis of adult visceral arteries
Bowel obstruction	Distension of proximal bowel loops resulting in venous congestion
	Strangulation of mesenteric vessels
Abdominal trauma	Blunt abdominal trauma causing intestinal stenosis with late chronic presentation
	Penetrating trauma with direct injury to the major mesenteric vessels
Neoplasms	Invasion of the major mesenteric vessels by the tumor
	Over distension and fecal material stagnation above an obstacle
Abdominal inflammatory conditions	Mesenteric, portal and splenic vein thrombosis
Chemotherapy, drugs and corrosive injury	Vasoconstriction
	Hypotension
	Thromboembolism
	Liquefaction necrosis (Alkalis)
	Coagulative necrosis (Acids)
Radiation	Obliteration of small arterioles producing a progressive occlusive vasculitis

dangerous abdominal condition, especially in elderly patients[25,26]. It is associated with a high mortality rate that ranges from 75% to 90% of cases[15].

Bowel ischemia is produced by insufficient blood flow to or from the intestines. It may have an acute or chronic setting depending on the underlying disorder (Table 2). The extent of bowel ischemia in the bowel wall is divided into three stages[26]: Stage Ⅰ: the ischemic lesions are confined to the mucosa and are reversible (known as reversible ischemic enteritis); Stage Ⅱ: characterized by necrosis of the mucosal and submucosal tissues, which may lead to fibrotic stricture development; Stage Ⅲ: the entire wall is affected by ischemia. It is associated with a high mortality rate.

Intestinal ischemia results in damage to the mucosal barrier which, in association with over-distension of the bowel loops and gas-forming bacterial proliferation, leads to gas moving from the intestinal lumen to the mesenteric veins and flowing through it to the portal system and hepatic parenchyma.

Bowel ischemia is the primary etiology of HPVG (70% of cases) and when associated, they are related to transmural necrosis in 91% of cases and to a high mortality rate (85% of patients)[15]. These facts signify that HPVG is an absolute indication for surgery in the context of mesenteric ischemia.

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[23].

Currently, the multidetector row CT angiography (MDCT) has become the first choice for HPVG detection and determination of the underlying process[20]. MDCT sensitivity has been markedly increasing over time from a low of 39% to a current high of 82%, and it has reached a similarly high sensitivity in diagnosing acute bowel ischemia as that of angiography.

HPVG is often associated with pneumatosis intestinalis, posing a grave prognosis, especially in the ischemic intestine[27-31]. The CT scan alone cannot predict which patients are experiencing true intestinal ischemia and which simply have benign pneumatosis. The presence of HPVG does not provide any information concerning the extent of bowel necrosis. In all cases, CT findings should be correlated with the clinical signs and with laboratory parameters to reach a high sensitivity and specificity level for intestinal necrosis. When HPVG associated with ischemic bowel disease is encountered, coexisting other abdominal conditions should be considered pre- and intraoperatively. Intestinal resection is performed when bowel necrosis is found on laparotomy. Nowadays, with the development of highly advanced imaging techniques, potentially severe pathologies, such as bowel ischemia, are diagnosed at much earlier stages, allowing prompt treatment and significantly reducing mortality rates.

DIVERTICULITIS

Hepatic portal venous gas is a rare complication of diverticulitis[16,32]. However, Sellner *et al*[16] found that complicated diverticulitis is the second most frequently reported cause of HPVG, which can be due to two mechanisms: The first is a septic thrombophlebitis of the inferior mesenteric vein complicated by gas-forming pathogens. The second is a direct communication between the intestinal lumen and the portomesenteric vein system. This is caused by intramesocolic intestinal perforation, dissecting between the peritoneal leaflets of the mesocolon and creating access to mesocolic veins[33].

Patients with mesocolic abscess have better prognosis than patients with septic pylephlebitis[16].

Diverticulitis associated with HPVG necessitates a selective surgical approach after adequate reanimation with intravenous fluids and antibiotics[34]. However, Nobili *et al*[35] suggested that if medical conservative therapy is effective and the clinical status improves, the surgery could be delayed.

In one case, Negro *et al*[36] reported a patient with sigmoid diverticulitis who developed a massive embolism of the intra- and extra-hepatic portal systems due to an enterovascular fistula, and who was treated with fistula embolization and subsequent sigmoidectomy.

GASTRIC PATHOLOGIES

HPVG has been reported in association with either gastric dilatation or gastric emphysema[37,38]. The treatment is conservative or surgical depending on the underlying process.

Gastroparesis is a frequent complication after a polytraumatic event, leading to gastric emphysema and subsequent HPVG[37]. In these cases, gastric decompression with a naso-gastric tube, nil by mouth and observation is sufficient treatment.

Furthermore, HPVG has been described in a patient with hypertrophic pyloric stenosis[39,40], and in a patient with peptic ulcer[41]. A gastric volvulus in a diaphragmatic hernia, without necrosis, was also reported in one patient[42]. These observations prove that raised intra-luminal pressure results in gastric pneumatosis and HPVG in the absence of bowel ischemia and gas-forming organism proliferation.

Several cases of HPVG have been described after accidental ingestion of caustics, hydrogen peroxide and chronic toluene inhalation[30,43], which lead to mucosal ulcerations and acute gastric distension by oxygen production. Generally, the patient will be stable and can be managed by symptomatic treatment. In cases of massive gas embolization to the portal venous system, and the presence of cardiac and neurological symptoms, hyperbaric oxygen should be used and can be a successful treatment[44].

INFLAMMATORY BOWEL DISEASES

Kinoshita *et al*[3] reported that in 182 cases of HPVG, 4% were associated with ulcerative colitis, and 4% with Crohn's disease.

HPVG, in patients presenting inflammatory bowel disease, can be caused by mucosal damage alone or in combination with bowel distension, sepsis, invasion by gas-producing bacteria[45,46], or after colonoscopy, upper gastrointestinal barium examination, barium enema or blunt abdominal trauma[47]. Therefore, a finding of HPVG associated with Crohn's disease does not mandate surgical intervention especially in the absence of peritoneal signs or free intraperitoneal gas[48].

LIVER TRANSPLANTATION

Hepatic portal venous gas is a common finding on Doppler sonography in the early postoperative period after liver transplantation. Chezmar *et al*[9] concluded in their study that HPVG alone, in the absence of bowel necrosis, intra-abdominal abscess, small-bowel obstruction or sepsis, is a transient finding without clinical significance. Furthermore, there was no correlation between the finding of portal venous air and transplant rejection, the need for retransplantation, the cause of hepatic failure, the type of biliary anastomosis, ventilator dependence, or subsequent death.

In pediatric liver transplant recipients, Wallot *et al*[49] suggested that the detection of HPVG beyond the early postoperative period may be a sign of intra-abdominal post-transplant lymphoproliferative disease, leading to the loss of bowel wall integrity.

CHEMOTHERAPY

Two cases of HPVG after chemotherapy have been reported. In the first case, Kung *et al*[50] described HPVG to be secondary to pneumatosis intestinalis in a patient who received irinotecan and cisplatin. On laparotomy, the colon and the small bowel were normal, and the patient was managed supportively with success. Gastrointestinal toxicity is a common side effect of irinotecan[51] which may be exacerbated by the adjunction of cisplatin leading to mucosal ulceration, bowel distension and gas-forming anaerobic bacterial proliferation.

In the second case, Zalinski *et al*[52] reported localized HPVG in the right liver after complete colorectal cancer liver metastasis necrosis in a patient receiving a treatment of oxaliplatin and cetuximab. Infection of the necrotized metastasis was promoted by the tumor which subsequently turned into a liver abscess, and fistulized to the right portal vein.

One case of HPVG was reported after chemoradiation therapy for an advanced esophageal cancer[53].

OTHER CONDITIONS

HPVG has been reported in association with acute pancreatitis[54,55], obstructive pyelonephritis after extracorporeal shock wave lithotripsy[56], acute appendicitis[57], cholangitis[20], gastro-jejunal anastomotic leak after laparoscopic gastric bypass[58], uterine gangrene[31], and percutaneous endoscopic gastrostomy tube placement[59]. HPVG has also been seen in the presence of a jejunal feeding tube[60], following esophageal variceal band ligation[61], gastrointestinal perforation with amyloidosis[62], with severe hyperglycemic shock[63], and in superior mesenteric artery syndrome[64-66].

These conditions may lead to bowel hypoperfusion and subsequent intestinal ischemia, or may induce an ileus and intestinal distention with mucosal damage and bacterial proliferation producing pneumatosis intestinalis and HPVG. In these cases, surgical treatment after adequate medical resuscitation is indicated depending on the underlying process.

Occasionally, in cases of abdominal blunt trauma, the increased intra-luminal pressure causes mucosal tears within the intestine, which allows gas to enter submucosal veins and flow to the hepatic portal vein[19]. However, a severe blunt abdominal trauma may lead to intestinal necrosis and eventual bowel rupture with peritonitis. Thus, when HPVG after abdominal trauma is associated with free intra-abdominal gas, pneumatosis intestinalis and signs of peritonitis, surgical intervention is mandatory.

CONCLUSION

A radiologic finding of HPVG does not necessarily indicate a severe underlying pathology. It can be seen in relatively benign situations such as following endoscopic procedures and gastric dilatation which only necessitate conservative therapy. Traditionally, HPVG was considered as being an indicator of bad prognosis and as being associated with a particularly high mortality rate. Nowadays, with the development of highly advanced

imaging techniques, potentially severe pathologies, such as bowel ischemia, are diagnosed at much earlier stages, allowing prompt treatment and significantly reducing mortality rates. HPVG is not by itself a surgical indication and the treatment depends mainly on the underlying disease. The prognosis is related to the pathology itself and is not influenced by the presence of HPVG.

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