HEPATIC PORTAL VENOUS gas (HPVG) was first described in abdominal plain radiographs in 1955 by Wolfe and Evans in 6 neonates who died secondary to necrotic bowels, followed by reports of HPVG in 5 adults who died and the first reported survivor in 1965. Liebman and colleagues analyzed all cases of HPVG reported in the literature by 1978 and found an oft-cited mortality rate of 75%, thereby codifying the link between HPVG and risk of imminent death and the corresponding maxim that HPVG demands laparotomy.

Hepatic portal venous gas is a rare radiologic finding, with only 182 cases documented in the literature by 2001. Retrospective reviews of computed tomographic (CT) scans identified 17 cases in 14 000 at 1 academic medical center and 11 in 19 000 at another. Hepatic portal venous gas is defined radiologically as tubular areas of decreased attenuation in the liver periphery. This definition was derived from the work of Sisk, who injected radiologic contrast into the portal vein and detected it in the liver periphery, within 2 cm of the capsule. Proof of the localization of HPVG to the portal vein and detected it in the liver periphery, within 2 cm of the capsule. Proof of the localization of HPVG to the portal vein and detected it in the liver periphery, within 2 cm of the capsule. Proof of the localization of HPVG to the portal vein and detected it in the liver periphery, within 2 cm of the capsule.
Portal sinusoids came from Wiot and Felson, who clamped all hepatic vessels during an autopsy, injected barium into the portal circulation, and demonstrated mixture of the gas and contrast. Portal venous gas can be distinguished from aerobilia, an indication of gallstone ileus, where air is found centrally in the biliary tree, and from pneumoperitoneum, where gas is found outside the liver capsule, due to perforation of a hollow viscus.

The left lobe of the liver is predisposed to develop HPVG, possibly because of peculiarities in hepatic venous anatomy. Males and females are equally likely to develop HPVG. In approximately 50% of reported cases, HPVG presents with pneumatosis intestinalis (PI), gas within the intestinal wall. It is generally presumed that PI ascends from the draining venous mesentery and condenses in the portal venous system; therefore, PI and HPVG represent progressive steps in a single process.

Experimental support for this sequence is scarce, although air injected into the submucosa or mesenteric veins of dog intestines was observed in the portal venous system. The left lobe of the liver is predisposed to develop HPVG, possibly because of peculiarities in hepatic venous anatomy. Males and females are equally likely to develop HPVG. In approximately 50% of reported cases, HPVG presents with pneumatosis intestinalis (PI), gas within the intestinal wall. It is generally presumed that PI ascends from the draining venous mesentery and condenses in the portal venous system; therefore, PI and HPVG represent progressive steps in a single process.

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Remarkably, in several early works, surgeons reported air bubbles flowing in the mesenteric veins of patients with preoperative HPVG. In 1 case, the surgeons transilluminated the mesentery and described the veins as “resembling the bubbles of gas seen in certain neon light signs.” In another, the surgeons noted “intravascular gas seen in all the mesenteric and portal veins” with “a large amount of frothy air bubbles” in a tear in the liver capsule. Modern ultrasonography studies have visualized air emboli moving through the hepatic portal system in real time in patients with HPVG.

**REPORT OF CASES**

**CASE 1**

A 63-year-old woman presented to the emergency department complaining of constipation and bilious vomiting. She denied bowel movements over the preceding 7 days and had developed escalating diffuse abdominal pain, bloating, and vomiting. During a prior episode of abdominal discomfort months earlier, CT examination discovered a lung mass, and she was diagnosed with stage IIIB non–small cell lung carcinoma, for which she initiated treatment days prior. Her vital signs were within normal limits, but her abdomen was tense and rigid. Laboratory analysis was notable for leukocytosis. A plain abdominal radiograph demonstrated diffuse gaseous distention of the small and large bowel, and HPVG was visible. A contrast-enhanced abdominal CT confirmed diffuse gaseous distention of the small bowel and colon with pneumatosis of the colon and portal and mesenteric venous gas. In addition, free peritoneal air was present, consistent with hollow viscus perforation. Unfortunately, within hours of the CT scan, the patient died in shock. The primary cause of her gastrointestinal disease was never elucidated.

**CASE 2**

A 56-year-old man presented to the emergency department complaining of crampy abdominal pain with diarrhea, nausea, and vomiting over the preceding 5 days. He described several episodes of melena and admitted to having lost 30 lb over preceding months. He denied hemoptysis, fever, chills, or night sweats. He admitted to frequent use of ibuprofen to treat chronic lower back pain. Vital signs were stable, and on examination, his abdomen was soft with active bowel sounds and no rebound or guarding. Rectal examination results were positive for occult blood. Serum lactate level was not elevated. An abdominal CT imaging study was performed, and the results supported a diagnosis of nonsteroidal anti-inflammatory drug–induced gastritis, with a mild pneumatosis of the gastric wall and HPVG, raising concern of a perforation. Surgical and gastroenterologic services were consulted, but, given the absence of peritonitis, it was decided to treat conservatively. On the fourth hospital day, he underwent an upper gastrointestinal tract series, revealing a 40-mm, nonbleeding, cratered gastric ulcer in the cardia. The patient was discharged after 2 weeks with significant clinical improvement.

**COMMENT**

**RECENT EVIDENCE**

In the half century since HPVG was first described, it has been reported in many nonfatal conditions, such as Crohn...
disease,\textsuperscript{19} ulcerative colitis,\textsuperscript{20,21} graft-vs-host disease,\textsuperscript{22} bowel obstruction, pseudo-obstruction,\textsuperscript{23} bacterial abscesses,\textsuperscript{22,24-28} diverticulitis,\textsuperscript{3} paralytic ileus,\textsuperscript{29} suppurative cholangitis,\textsuperscript{30} and colovenous fistulae.\textsuperscript{31} Hepatic portal venous gas has been described in a number of nonsurgical conditions, including cystic fibrosis,\textsuperscript{32} seizures,\textsuperscript{33} and colchicine toxicity,\textsuperscript{34} although secondary effects, such as ileus, cannot be excluded. Frequently, there is no immediate risk of mortality, for example, in patients presenting with inflammatory bowel disease and HPVG.\textsuperscript{35,36} Finally, a substantial literature exists on iatrogenic HPVG, with HPVG observed in patients after laparoscopy,\textsuperscript{37} and endoscopic retrograde colangiopancreatography,\textsuperscript{38-41} as well as other endoscopic procedures,\textsuperscript{42,43} gastric dilatation,\textsuperscript{44} liver transplantation,\textsuperscript{47} radiofrequency tumor ablation,\textsubscript{48} arterial catheterization,\textsuperscript{49} and enema.\textsuperscript{50,51} As early as 1971, higher survival rates were recognized in iatrogenic HPVG-associated illness compared with natural pathologies,\textsuperscript{14} and in 1986, experts were already urging surgeons to avoid laparotomy in patients without toxic reaction with iatrogenic HPVG.\textsuperscript{34} In a recent survey of HPVG literature, Kinoshita and colleagues\textsuperscript{6} reported 39% mortality among all 182 cases reported by 2001. Although smaller case series cite both lower\textsuperscript{7,8} and higher mortality rates for HPVG-associated disease,\textsuperscript{13,55,56} these studies included fewer than 20 patients each. This is obviously a significant reduction from the 75% mortality seen in 1978, itself an “improvement” over earlier estimates.\textsuperscript{5} The observed reduction in mortality was driven by an increase in the proportion of nonfatal conditions reported with HPVG and a corresponding decrease in the proportion of HPVG associated with mesenteric ischemia. Bowel necrosis accounted for 72% of diagnoses in the Liebman et al survey\textsuperscript{3} in 1978, but only 43% of the diagnoses in
reports of HPVG-positive patients surveyed by Kinoshita et al\(^6\) in 2001, although the mortality in these patients remained high (75%, n=79). Kinoshita et al found that the mortality of patients with HPVG with Crohn disease, ulcerative colitis, intraabdominal tumors, cholangitis, pancreatitis, and nonfulminant hepatitis was 0% (n=28). A variety of conditions present intermediate mortality rates: 30% in patients with abscesses (n=20), 25% with gastric ulcers (n=7), and 21% with digestive tract dilatation (n=21). Collectively, the fraction of HPVG cases associated with diseases other than ischemic or necrotic bowel rose from 30%\(^5\) to 51%\(^6\) when the 2 studies were compared.

Hepatic portal venous gas therefore remains an ominous sign in the specific context of bowel ischemia or necrosis. Hepatic portal venous gas has been identified as a risk factor for surgical intervention and increased mortality\(^7\) and the degree of bowel ischemia may be correlated with the likelihood of HPVG or PI.\(^6,63\) Experimental occlusion of the mesenteric arteries of dogs resulting in infarction also results in HPVG, supporting mucosal ischemia as playing a mechanistic role.\(^5,58\) Two reports describe postmortem HPVG after cardiopulmonary resuscitation,\(^39,60\) linking ischemia and HPVG, as cardiac output during cardiopulmonary resuscitation is poor.\(^61\) It is presumed that ischemic insult or frank necrosis results in mucosal disruption, although this mechanism has not yet been proven.

We propose that the increase in benign HPVG-associated conditions is due to the adoption of CT scanning. The original HPVG literature of the 1950s and 1960s was based on plain radiographs,\(^1,2\) primarily left lateral decubitus views.\(^5\) However, CT is superior for detection of intra-abdominal gas, demonstrated in studies of pneumoperitoneum. Increased sensitivity with CT has made it possible to detect mild HPVG, while reliance on plain radiography captures only scenarios wherein a large volume of gas accumulates.\(^8,62,63\) In addition, remarkable increases in the volume of patients undergoing advanced imaging techniques over time have been demonstrated,\(^64\) increasing the prevalence of HPVG. Digital CT images also provide an opportunity to manipulate the images for ideal viewing, and many authors note that a “lung-window” CT setting permits easy identification of both HPVG and PI,\(^8,69\) although other settings are also advised.\(^7\)

**PATHOPHYSIOLOGY**

There is no evidence available to date to identify the nature of the gas observed in imaging studies. The leading hypotheses are (1) microbe-derived gas production and (2) absorbed intraluminal air.

No clear experimental or natural data describe how gas production secondary to microbial metabolism results in HPVG.\(^65\) Bacteremic liver metastases can result in in situ gas production,\(^24,25\) but this is rare. Septic phlebitis can result in gaseous accumulations in the portal system, or gas generated in abscesses subjacent to inflamed mesentery could enter the vasculature,\(^12,22,26,27,66\) although few data support these models. Regardless of anatomical route, microbe-derived gases would be hypothesized to be molecularly and atomically distinct from swallowed intraluminal air. Indeed, the cystic gas of pneumatosis cystoides intestinalis has been shown to be hydrogen gas, strongly supporting a bacteriologic etiology for this distinct pathology.\(^67\) and similar analyses of HPVG would be convincing support for a microbial origin.

The majority of patients in both the Liebman et al\(^3\) and Kinoshita et al\(^6\) studies demonstrated bowel disrupted mucosa, increased intraluminal pressure. It is hypothesized that luminal air enters the capillary veins either by an impaired epithelial barrier or by increased intraluminal pressure. Indeed, in a large number of “natural experiments,” HPVG has been demonstrated in patients with mucosa disrupted by inflammatory bowel disease and intraluminal pressures increased by enema\(^9,20,22,68,69\) or colonoscopy.\(^21,70\) Pneumatosis intestinalis was generated experimentally in cadavers with ulcerated mucosa by application of intraluminal air pressure.\(^14,71\) Shaw et al\(^31\) were able to chemically reproduce these effects in intact dog intestines using hydrogen peroxide enemas, wherein hydrogen peroxide bypassed the epithelium and released oxygen gas on interacting with intracellular catalase enzymes or iron, causing oxygenation of the affected tissues and the formation of bubbles in the mucosa, draining mesentery, and portal veins.

Intraluminal and microbial origins for HPVG are not mutually exclusive. Rather, it is possible that these are separate pathways by which patients can arrive at the radiologic finding of HPVG. In support of this, sepsis alone was observed in 2 of 64 patients with HPVG in the Liebman et al study,\(^3\) and 26 of 182 patients in the Kinoshita et al study had an infectious etiology in the absence of other bowel disease.\(^6\) These data suggest that a microbial origin for HPVG may therefore represent an independent mechanism in a minority of patients with HPVG, unrelated to that seen in necrotic bowel.

As noted earlier, HPVG has also been detected by ultrasonography,\(^18,26,47,72-75\) where the HPVG appears as hyperechoic foci in the background of the liver parenchyma. Ultrasonography has the advantages of low cost, bedside imaging, and a lack of radiation exposure to the patient. It is possible that ultrasonography may prove even more sensitive than CT,\(^74,75\) although this requires formal analysis. An even more limited literature exists describing magnetic resonance imaging–based identification of HPVG.\(^76\)

**CONCLUSIONS AND RECOMMENDATIONS**

While HPVG was clearly an ominous radiologic finding in previous decades, today it is a puzzling finding that may confound patient management (Table). The development of CT has created more opportunities to visualize gas in the portal system, revealing a host of benign conditions. The main conclusion offered by this review is that radiologic detection of HPVG by CT should not determine clinical or surgical management per se, rather disease severity should. To this effect, a management algorithm is proposed in Figure 4 and is summarized by the mnemonic “ABC.” Urgent lapa-
Patients with more equivocal presentation and HPVG—mucosal disruption, bowel distention, abscesses, or gastric ulcers, as examples—should be monitored intensely with a reduced threshold for surgical correction under appropriate conditions ("be careful"). These patients may be at risk for mortality as high as 20% to 30%, based on the severity of ischemia and clinical outcome. 

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Nelson and colleagues describe an important and potentially life-threatening clinical condition—HPVG. They present a clearly written and very educational overview.

According to the given algorithm, the finding of HPVG in abdominal radiographs should lead directly to an emergency laparotomy. This assumption is based on the results of historical reports, in particular the review of the literature by Liebman et al with a mortality rate of 75% in patients with HPVG detected on abdominal radiographs published in 1978. It is questionable if this guideline is still applicable because in most departments a CT scan is available in the emergency setting and will be included in the diagnostic workup for most patients before an emergency laparotomy should be performed.

The given “ABCs” are in concordance with other treatment guidelines in patients with HPVG. It is important to discriminate patients with radiologic and clinical findings of HPVG if intestinal ischemia or infarction is the underlying disease in order to select the appropriate patients to undergo an emergency laparotomy. The given “ABCs of management” can be used as a mnemonic trick in this rare but important clinical situation; however, more distinct treatment pathways have been published in the literature recently. In flowcharts presented by Hou et al and Iannitti et al, more detailed clinical recommendations are given regarding the diagnostic workup, the necessity of immediate surgical intervention based on radiological and clinical findings, and the nonsurgical treatment (antibiotics, endoscopy, drainage) options.

As stated in the present article, the finding of HPVG by CT “should not determine clinical or surgical management per se.” In emergency situations with a critically ill patient (acute abdomen) and the finding of HPVG, emergency surgery is still mandatory. But in all other conditions, in particular in cases where bowel ischemia can be excluded, a conservative management of patients with HPVG might also be appropriate.

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