

# Hepatic Portal Gas in Adults

## Review of the Literature and Presentation of a Consecutive Series of 11 Cases

Nicolas Peloponissios, MD; Nermin Halkic, MD; Mauro Pugnale, MD; Philippe Jornod, MD; Petter Nordback, MD; Alain Meyer, MD; Michel Gillet, MD

**Background:** Hepatic portal venous gas (HPVG) in adults is a rare entity. The underlying pathologic condition is usually an intestinal ischemia, but it has been reported in association with a variety of conditions.

**Hypothesis:** Miscomprehension of the underlying pathologic conditions has led to some confusion in the literature concerning its etiology, diagnostic methods, and clinical consequences.

**Setting:** Centre Hospitalier et Universitaire Vaudois, Lausanne, Switzerland, and District Hospital of Morges, Morges, Switzerland.

**Method:** Between February 1, 1995, and May 30, 2000, eleven cases of HPVG were treated. These cases were retrospectively reviewed, together with a review of the literature to define the clinical significance of HPVG, the role of the computed tomographic scan, and the necessity of performing systematic emergency exploratory surgery.

**Results:** Two groups have to be distinguished—those who have HPVG with associated pneumatosis intestinalis and those who have HPVG without associated pneumatosis intestinalis. When associated with pneumatosis intestinalis, the cause is usually an intestinal ischemia and in a symptomatic patient it justifies systematic exploratory surgery. An abdominal computed tomographic scan including lung window settings to better identify air in the bowel wall will distinguish between these 2 groups. Pneumatosis intestinalis and HPVG due to bowel ischemia do not allow prediction of the severity of bowel wall damage.

**Conclusions:** The cause of HPVG without pneumatosis intestinalis is variable. Good knowledge of the possible causes combined with the clinical picture and the abdominal computed tomographic scan is required to correctly identify the underlying cause of HPVG and to avoid unnecessary surgery.

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From the Departments of Surgery (Drs Peloponissios, Halkic, Nordback, and Gillet), Radiology (Dr Pugnale), and Gastroenterology (Dr Jornod), Centre Hospitalier et Universitaire Vaudois, Lausanne, Switzerland; and the Department of Surgery, District Hospital of Morges, Morges, Switzerland (Dr Meyer).

**H**EPATIC PORTAL venous gas (HPVG) was first reported in neonates in 1955 by Wolfe and Nevins as cited by Liebman et al.<sup>1(p281)</sup> In adults, it has since been described in association with a variety of pathologic conditions (**Table 1**) that include intestinal ischemia and necrosis (75% of the cases), ulcerative colitis (8% of the cases), and intra-abdominal abscess (6% of the cases).<sup>1,47</sup> Patients with hepatic portal venous gas have a global survival rate of less than 25%<sup>1,2</sup> owing to the severity of the underlying pathologic condition that most commonly is intestinal necrosis.<sup>1</sup> This is a review of the literature and a retrospective study of 11 reported cases of HPVG that presented between February 1, 1995, and May 30, 2000, at either the Centre Hospitalier et Universitaire Vaudois, Lausanne, Switzerland, or the District Hospital of Morges, Morges,

Switzerland. The aim of the study was to clarify the significance and treatment of HPVG, emphasizing the role of performing early exploratory surgery.

### METHODS

Our case series included 6 men and 5 women whose mean age was 66 years (age range, 40-84 years). In 6 of the 11 patients the diagnosis of the underlying disease was confirmed by either surgery or autopsy (extensive intestinal ischemia, 3 patients; segmental bowel ischemia, 2 patients; and giant gastric ulcer, 1 patient). One case of pylephlebitis following diverticulitis was confirmed on computed tomographic (CT) scan. In 4 patients intestinal ischemia was suspected but was never confirmed. The diagnosis of HPVG in 10 patients was confirmed by CT scans, although the diagnosis was already suggested after ultrasonography or standard abdominal radiographs in 6 patients. In 9 cases of HPVG, CT scans revealed an associated pneumatosis intestinalis (PI). The 2 other cases of HPVG were not as-

sociated with PI and neither of these cases was related to intestinal ischemia. Eight of 11 patients died within 48 hours after presentation. Of the 5 patients who underwent surgery, 2 (40%) survived. These results are summarized in **Table 2**.

### COMMENT

Hepatic portal venous gas can be due to gas under pressure in the bowel lumen or to an alteration of the mucosa, allowing the gas to enter the portal system through the mesenteric veins.<sup>1</sup> These 2 mechanisms were demonstrated by Shaw et al<sup>49</sup> in 1967 using a dog model. Based on these observations, we can hypothesize that any pathologic changes in the mucosa, for example, Crohn disease, may lead to alteration of vessel walls and the surrounding tissue, preventing them from collapsing. Thus, it allows an easier entry for air, as already hypothesized by Katzgraber et al<sup>50</sup> in a gastric ulcer. A third mechanism is gas-forming bacteria in an intra-abdominal abscess<sup>1,3,4,43</sup> with or without a related pylephlebitis<sup>3-5,51</sup> as observed in our patient 4.

Hepatic portal venous gas can occur alone or in association with PI (**Figure**). When associated with PI, the origin seems to be intestinal ischemia, as observed in our case series. Hepatic portal venous gas is not predictive of its severity when caused by intestinal ischemia<sup>1</sup> and has even been observed with reversible ischemia.<sup>1,52,53</sup> In

this series, 2 cases of HPVG and PI were observed with segmentary bowel ischemia (patients 9 and 10).

Associated portal and inferior or superior vena cava gas has already been described<sup>53-55</sup> and also has been observed in patient 1 of our case series. In 1 of the 3 reported cases described, air could clearly be observed in the superior and inferior mesenteric, hemorrhoidal, internal iliac, and left colic veins and inferior vena cava. Mallens et al<sup>53</sup> hypothesized that the important quantity of gas caused temporary obstruction of the portal sinusoids with portosystemic shunting. In 1 of the cases described by Kriegshauser et al,<sup>54</sup> the gas was observed with real-time sonography passing through the liver from the portal veins to the systemic circulation. In the third case the association between HPVG and air in the vena cava was observed in a fulminant sepsis and was attributed to a gas-forming organism causing portal and systemic venous gas.<sup>55</sup>

Transient cases of HPVG without clinical consequence have been observed in numerous cases (inflammatory bowel disease, acute gastric dilatation, blunt abdominal trauma, jejunostomy catheter insertion, and other isolated cases).<sup>45,47,53</sup> Benign HPVG has even been observed, but exceptionally, in association with colonic intramural air.<sup>56</sup>

Abdominal radiographs can detect large quantities of HPVG or intestinal intramural gas, but its accuracy in demonstrating them is inferior to ultrasonographic or CT scans, both of which allow an earlier detection of small quantities of gas in the portal tract.<sup>6,44,57</sup> The finding of HPVG on standard radiographs is suggestive of a poor prognosis and is commonly associated with bowel infarction (patients 7 and 11).<sup>6</sup> A radiograph is more revealing in cases of HPVG when taken with the patient lying on his or her left side.<sup>1</sup>

At echography HPVG appears as numerous small hyperechogenic images with inconstant acoustic shadows.<sup>6,58,59</sup> Gas in the portal venous system is carried by the centrifugal flow of blood in the periphery of the liver, appearing to extend to within 2 cm of the hepatic capsule. Inversely, gas in the biliary tract moves with the centripetal flow of bile, thus appearing more centrally in the liver. Ultrasonography gives additional real-time information, allowing the observation of the hepatofugal or hepatopetal gas displacement.<sup>59</sup> With concomitant portal hypertension, gas bubbles may be slower to reach the liver periphery, making the diagnosis difficult.<sup>7</sup> A history of

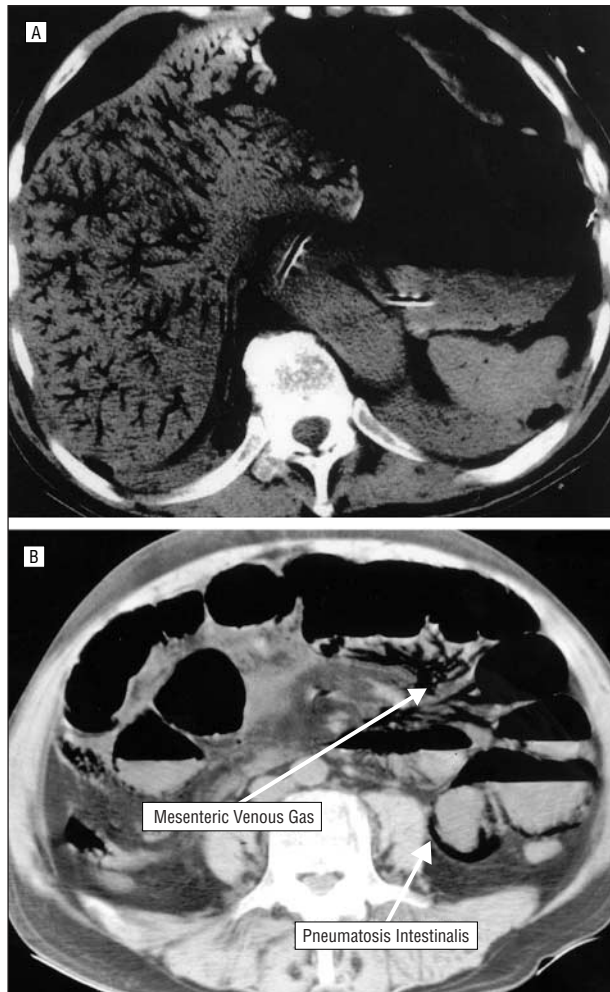
**Table 1. Uncommon Pathologic Conditions Associated With Hepatic Portal Venous Gas**

Intra-abdominal or retroperitoneal abscess <sup>1-16</sup>
Inflammatory bowel disease
After diagnostic studies <sup>17-27</sup>
Without diagnostic studies <sup>28-30</sup>
Diverticulosis after barium enema <sup>27,31</sup>
Biliary infection <sup>32,33</sup>
Gastric ulcer <sup>6,34,35</sup>
Gastric dilatation <sup>36-38</sup>
Duodenal perforation <sup>39</sup>
Blunt abdominal trauma without intestinal perforation <sup>40-42</sup>
Ileus from anticholinergic adverse effects <sup>43</sup>
Acute necrotizing pancreatitis <sup>1</sup>
After hepatic or cardiac transplantation <sup>44-46</sup>
After placement of a jejunostomy tube <sup>47</sup>
Transgastric endoscopic ultrasound-guided biopsy <sup>48</sup>

**Table 2. Outcome of Patients With Hepatic Portal Venous Gas**

Patient No.	Cause of the Hepatic Portal Venous Gas	PI	Surgery	Outcome
1	Suspected intestinal ischemia	+	-	Death
2	Suspected intestinal ischemia	+	-	Death
3	Suspected intestinal ischemia	+	-	Death
4	Pylephlebitis following diverticulitis	-	-	Alive
5	Large perforated gastric ulcer	-	+	Alive
6	Diffuse arterial intestinal ischemia	+	+	Death
7	Diffuse arterial intestinal ischemia	-	+	Death
8	Suspected intestinal ischemia	+	-	Death
9	Segmentary intestinal ischemia (distal ileon)	+	-	Death
10	Focal colonic necrosis of unknown cause	+	+	Alive
11	Diffuse arterial intestinal ischemia	?	+	Death

Abbreviations: PI, pneumatosis intestinalis; -, absent or no; +, present or yes.



A, Computed tomographic scan showing a huge volume of hepatic portal venous gas throughout the whole liver, extending to below the hepatic capsule. B, Computed tomographic scan showing an important pneumatosis intestinalis and the presence of air in the mesenteric venous branches.

biliodigestive anastomosis, endoscopic papillotomy, biliary endoprosthesis, or choledointestinal fistula are certainly of importance in the differential diagnosis.<sup>28</sup>

With its excellent spatial and contrast resolution, a CT scan provides a conclusive diagnosis in most cases. Furthermore, associated necrotic bowel signs are more specific on CT scan than with ultrasonography. In necrosis, the gas generally produces a bubbly image whereas with innocuous causes, it can be linear or clusterlike, larger and more spherical.<sup>60</sup> However, these signs are not specific and do not allow a clear-cut diagnosis. The standard window setting for an abdominal CT scan may overlook even extensive gas, particularly when using restricted windows. Thus, the use of complementary lung window settings is necessary when PI is suspected.<sup>61,62</sup> Nevertheless, in all of our cases, PI was clearly observed with standard window settings.

## CONCLUSIONS

The recognition of HPVG in adults indicates, in most cases, a life-threatening, acute abdominal process. Hepatic portal venous gas as well as PI are radiological clues

and not diagnoses. Two groups have to be distinguished—those who have HPVG with associated PI and those who have HPVG without associated PI. When associated with PI, the cause of HPVG is usually an intestinal ischemia and in a symptomatic patient it justifies performing systematic exploratory surgery. An abdominal CT scan, including lung window settings to better identify air in the bowel wall, will distinguish between these 2 groups. Pneumatosis intestinalis and HPVG due to bowel ischemia do not allow prediction of the severity of bowel wall damage. The finding of HPVG on standard radiographs is suggestive of a poor prognosis and is commonly associated with bowel infarction. The cause of HPVG without PI can vary between innocuous and life-threatening causes. Both the patient's history and the clinical findings have to be considered to exclude a benign cause of HPVG and, thus, avoid unnecessary laparotomy. In doubtful cases, a prompt laparoscopic exploratory procedure is mandatory to exclude a surgically treatable disease.

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Corresponding author and reprints: Nicolas Peloponissios, MD, Department of Surgery, Centre Hospitalier et Universitaire Vaudois, Rue du Bugnon 46, 1011 Lausanne, Switzerland (e-mail: nicolas.peloponissios@hospvd.ch).

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