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ORIGINAL ARTICLE

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Detection of hepatic portal venous gas: its clinical impact and outcome

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Abstract The clinical impact and outcome of a rare radiographic finding of hepatic portal venous gas (HPVG) as well as the effectiveness of computed tomography (CT), CT scanogram, and conventional radiography in the detection of HPVG were retrospectively analyzed. CT scans, CT scanogram, and plain film radiographs of 11 patients with HPVG were reviewed and compared with their medical records and surgical and pathology reports. Eight of the 11 patients underwent plain film radiographs 1 day before or after the CT scan. HPVG was detected at CT in all 11 patients, on CT scanogram in three (3 of 11, 27.3%), and on plain films in one (one of eight, 12.5%). In nine of 11 patients (81.8%), CT revealed an associated pneumatosis intestinalis. In six of the 11 patients (54.6%), acute mesenteric ischemia was the underlying disease for HPVG. Seven patients (63.6%) underwent emergency exploratory laparotomy. The mortality rate for HPVG alone was 27.3% (3 of 11) and for HPVG related to mesenteric bowel disease 50% (three of six). Acute mesenteric ischemia is the most common cause of HPVG, which continues to have a predictably higher mortality. CT is superior to CT scanograms and radiographs in the detection of HPVG and its underlying diseases and, therefore, should be used as the primary diagnostic tool.

Keywords Computed tomography · Portal venous gas · Mesenteric infarction

Introduction

Hepatic portal venous gas (HPVG) is a rare but important radiographic finding whose pathogenesis is still not fully understood. HPVG is thought to be caused by mesenteric ischemia, but it can also occur under nonischemic conditions which may not require emergency exploratory laparotomy [1–3].

In 1955, Wolfe and Evans [4] described the first cases of HPVG in infants with necrotizing enterocolitis on plain films. Five years later, Susman and Senturia [5] reported the first adult case associated with small bowel infarction. Gas in the portal vein was initially detected on plain radiographs of the abdomen and was associated at that time with a mortality rate of 75% [6]. Since the introduction of computed tomography (CT), the clinical outcome of HPVG has improved and its mortality rate has dropped to 29% due to earlier diagnosis of the disease [7]. Recent studies have dealt with various nonischemic conditions and even with incidental findings of HPVG, some of which could be cured with conservative management alone [3, 8–14].

This study was designed to determine the value of CT, CT scanogram, and conventional radiography in the detection of hepatic portal venous gas, its impact on clinical management, and the outcome of the finding of HPVG.

Materials and methods

All data on the 19,500 abdominal CT scans performed at our institute between January 1997 and March 2005 and stored in our computer workflow management system (Centricity RIS, GE, USA) were searched for the following key words: portal venous gas or air, hepatic gas or air, portal gas or air, and pneumatosis intestinalis. Only patients older than 18 years were included in the study. The database search disclosed ten patients who presented with HPVG on abdominal CT scan. A further patient who presented before January 1997 with a history of HPVG was retrieved from our teaching files. Because abdominal CT scans were performed in the context of patient care and were evaluated in retrospect, no institutional review board approval or patient informed consent according to the guidelines of our institution had to be obtained.

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Of the 11 patients thus selected, six were female and five were male; they ranged in age from 47 to 88 years (mean age 65 years). In all patients, HPVG was detected by CT. All patients received oral contrast material for abdominal CT scanning; nine were given additional intravenous (IV) contrast material. Eight of the 11 patients underwent conventional radiographs of the full abdomen 24 h before or after the CT examination.

The sensitivities of CT scanograms and conventional radiographs for HPVG and pneumatosis intestinalis using CT as reference standard were calculated from 2×2 contingency tables. All radiographic exams were reviewed by two radiologists, one a board-certified radiologist, the other an experienced radiology resident, both with a special interest in abdominal imaging as a consensus reading. For abdominal CT images, a soft-tissue window was used (center 40, width 400). In four patients, an additional lung window (center 650; width 1,500) was available. The images of these four patients were reviewed on a PACS workstation. The studies of the other nine patients were read off film.

Close attention was paid during evaluation of CT scans to the following:

1. Presence or absence of HPVG. On abdominal CT scans, the amount of HPVG was graded semiquantitatively as:
   - small, + (in the periphery of the liver)
   - moderate, ++ (in the periphery and some in the central portion of the liver)
   - large, +++ (in the peripheral and central portion of the liver)
2. Anatomic location of the HPVG (left or right liver lobe, central or peripheral)

Table 1 Clinical and radiological data of 11 patients with HPVG

Case #/age/sex	Risk factors and underlying disease	CT	CT scanogram	Plain X-ray	Treatment	Clinical outcome
Ischemic						
1/72/F	AF, sepsis, intestinal ischemia	R +, L +++, periph. and cent. PI, occlusion of SMA	HPVG	PI	Surgery	Died POD 1
2/78/M	Sepsis, intestinal ischemia	R ++, L +, periph. PI, intestinal obstruction	No HPVG	No HPVG	Surgery	Survived
3/88/M	Esophageal carcinoma, intestinal ischemia	R +, L ++, periph. PI	No HPVG	PI	Refused surgery, conservative management	Died 2 days after CT scan
4/65/F	AF, intestinal ischemia	R ++, L +++, periph. and cent. PI, intestinal obstruction	No HPVG	No HPVG	Surgery	Died POD 1
5/57/F	Lung cancer, intestinal ischemia	R +, L ++, periph. and cent. PI, intestinal obstruction, occlusion of SMA	No HPVG	N/A	Surgery	DOC at 2 months
6/54/M	Diabetes mellitus, intestinal ischemia	R +, L +++, periph. and cent. PI, pneumoperitoneum	HPVG	N/A	Surgery	Survived
Nonischemic						
7/50/F	COPD	R +, L +, periph. PI, intestinal obstruction, pneumoperitoneum	No HPVG	Intestinal obstruction	Conservative management	Survived
8/78/M	AF, sepsis, small bowel obstruction caused by adhesions	R +, L +++, periph. and cent. PI, intestinal obstruction	HPVG	HPVG, PI, intestinal obstruction	Surgery	Survived
9/56/M	Inguinal hernia	R +, L +, periph. intestinal obstruction	No HPVG	N/A	Conservative management	Survived
10/47/F	Uterine sarcoma	R +, L +, periph. peritoneal carcinomatosis	No HPVG	No HPVG	Conservative management	DOC 3 days
11/73/F	AF, abdominal aortic aneurysm	R +, L ++, periph. PI, mesenteric venous gas	No HPVG	No HPVG	Surgery, no intestinal ischemia found	DOC 2 months

AF Atrial fibrillation, *R* right liver lobe, *L* left liver lobe, *PI* pneumatosis intestinalis, *POD* postoperative day, *DOC* death of other causes, *COPD* chronic obstructive pulmonary disease, *SMA* superior mesenteric artery, *cent.* central, *periph.* peripheral, *N/A* not applicable, *HPVG* hepatic portal venous gas

3. Presence or absence of occlusion of the splanchnic vasculature
4. Presence or absence of pneumatosis intestinalis on CT scans and plain films
5. Other relevant findings (e.g., mesenteric ischemia, bowel obstruction, tumor)

All medical records with follow-ups of up to 3 years and the surgical and pathologic reports of the patients were reviewed.

Results

The 11 patients with HPVG were divided into two groups, one comprised of six patients with mesenteric ischemia, the other of five patients without ischemia (Tables 1 and 2). Of the six patients with mesenteric ischemia [confirmed either by surgery (n=5) or autopsy (n=1)], all had pneumatosis intestinalis on CT scan. Contrast-enhanced CT revealed obstruction of the superior mesenteric artery in two of these patients (cases #1 and 5), but CT could not confirm the underlying etiology of the ischemia in the other four patients. All patients required an emergency laparotomy. In one patient with intestinal ischemia (case #3), the family objected to surgical intervention due to the patient's advanced age and poor clinical condition. The patient died 2 days after the CT scan. Three of the six patients died within 48 h after presentation, one later died of another cause (cardiogenic shock). The remaining two patients had an uneventful recovery and survived.

Of the group of five patients with nonischemic causes for HPVG, three (cases #7–9) exhibited bowel obstruction on both clinical exam and radiographic imaging. All of these patients survived, with only one having to undergo surgical decompression of the small bowel. One of the five patients (case #10) presented with a uterine sarcoma. The abdominal CT scan revealed a huge intraperitoneal tumor combined with carcinomatosis, HPVG, and pneumatosis intestinalis. The patient died after 3 days of conservative management. One patient (case #11) presented with acute abdominal symptoms and an abdominal CT scan showing HPVG, pneumatosis intestinalis, and an abdominal aortic aneurysm. At explorative laparotomy, the surgeon could find no infarcted bowel and no clear cause for HPVG. Two months after the surgery, the patient died secondary to multiorgan failure.

Three of the six patients with mesenteric ischemia died within 48 h after presentation of HPVG; three died of another disease some time later (multiorgan failure and cardiogenic shock). The remaining five patients survived to eventual discharge. The overall mortality rate for HPVG in our study population was 27.3% (3 of 11) and for those with HPVG related to mesenteric ischemia, 50% (three of six).

The diagnosis of HPVG was based on CT examination in all of the 11 patients. Of the eight patients who underwent both CT scan and plain film, just one (case #8) had HPVG detected on plain film (12.5%, one of eight). In this case, HPVG also occurred on the CT scanogram and a large amount of gas (+++) was present in the left liver lobe (Fig. 3). In three patients (cases #1, 6, and 8), HPVG could be identified on the CT scanogram (Figs. 1, 2, and 3). In these cases, large amounts of HPVG (+++) occurred in the left liver lobe and at the level of the portal vein bifurcation. Neither the CT scanograms nor the radiographs demonstrated HPVG in the case of small (+) or moderate (++) volume of gas on the CT scans.

CT showed a left lobe predilection in six patients. When the volume of gas was substantial (+++), it was encountered in both liver lobes and in the central portion of the liver (n=4). Smaller volumes of gas (+) were detected mainly in the periphery of the liver (n=6) (Fig. 4a). Two patients (cases #1 and 4) with a large volume of portal gas (+++) and one patient (case #3) with a moderate amount (++) died soon after presentation. Large and moderate volumes of gas were also seen in patients who survived (n=5) (Table 3).

The CT scans of nine patients (81.8%, 9/11) revealed an associated pneumatosis intestinalis (Fig. 3c). In correlation with CT images, radiographs had a sensitivity of 37.5% (3 of 8) for the detection of pneumatosis intestinalis. In two patients (case #10 and 11), both CT and radiograph were negative for pneumatosis intestinalis.

Table 2 Distribution of the amount of gas, pneumatosis intestinalis, and death in the ischemic and nonischemic groups

	Ischemic cause for HPVG	Nonischemic cause for HPVG
Amount of gas		
+	/	3/5 (60%)
++	3/6 (50%)	1/5 (20%)
+++	3/6 (50%)	1/5 (20%)
Intestinal pneumatosis (on CT scan)	6/6 (100%)	3/5 (60%)
Death (due to all causes)	4/6 (66.6%)	2/5 (40%)
Death (due to intestinal ischemia)	3/6 (50%)	/

Discussion

Although the presence of gas in the portomesenteric venous circulation is an important radiographic finding, its precise pathogenic mechanism is still debated on, with multiple explanations being offered. The major predisposing factor for development of hepatic portal venous gas is damage to the intestinal mucosa combined with bowel

Table 3 Correlation between the amount of HPVG on CT scan and mortality

Amount of HPVG	+	++	+++
Survived	3	3	2
Death due to intestinal ischemia	/	1	2

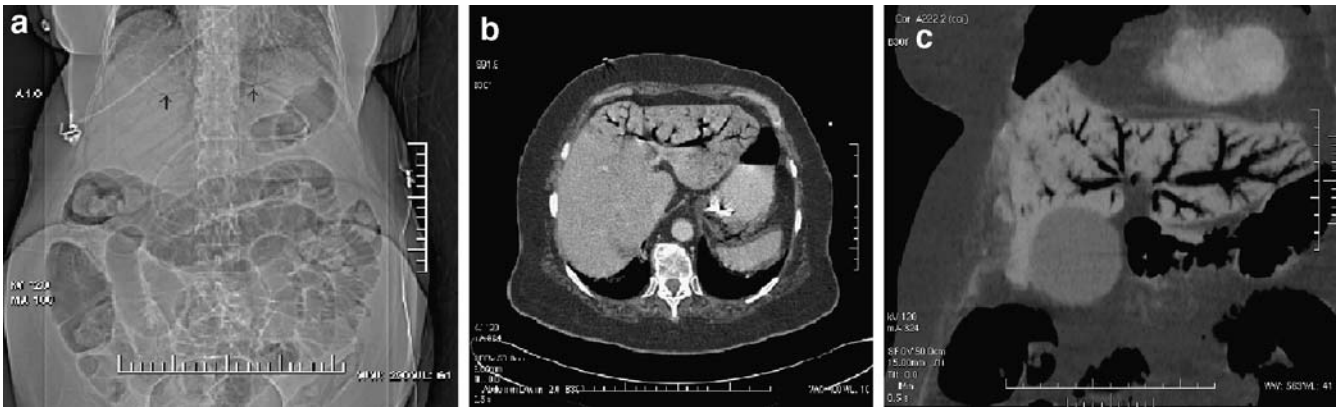

Fig. 1 A 72-year-old woman (case #1) with atrial fibrillation and sepsis. **a** CT scanogram demonstrates multiple branching tubular lucencies in the liver (*arrows*). **b** CT scan during the portal venous phase shows a large volume of hepatic portal venous gas distributed mainly in the left liver lobe. **c** Minimum intensity projection of coronal CT reformation shows branched areas of decreased attenuation in the left ventral hepatic lobe

distension or bacterial gas production. Most authors agree that the formation of HPVG is a multiplex incident in which two or all three of these conditions are present. Approximately 15% of HPVG cases, however, are idiopathic [1].

Damage to the intestinal mucosa facilitates the passage of intraluminal gas into the portomesenteric system. The most common causes of mucosal injury are bowel ischemia with mucosal ulceration, perforating gastric ulcer, and inflammatory bowel disease [1]. Minimal mucosal disruption may also result from increased intraluminal pressure secondary to bowel distention, which is associated with intestinal obstruction, blunt trauma, and iatrogenic intestinal dilatation (e.g., endoscopy, barium enema, and sclerotherapy) [1, 15]. Gas-producing bacteria may reach the intramural compartments through direct invasion of the wall [15]. The presence of bacteria may subsequently alter the intraluminal gas content and cause septicemia in branches of the mesenteric and portal veins (pylephlebitis) [15–17].

HPVG can be detected by conventional radiography and CT [1, 7, 18]. The radiographic pattern for HPVG has been described as a tubular lucency branching from the porta hepatis to the liver capsule (Fig. 3a) [6]. The gas travels peripherally in the portal vein consequent to the centrifugal flow of blood. The predominant location is the left liver lobe if the patient is in a supine position. The appearance of HPVG on CT scans resembles that of plain films: branching lucencies of decreased attenuation in the hepatic periphery (Fig. 4). HPVG must be differentiated from gas in the biliary tracts (pneumobilia), which tends to move with the centripetal flow of bile toward the hilum.

Although several reports have described abdominal CT's superior sensitivity for detection of HPVG than plain film radiography, to our knowledge, no single study has directly compared these two modalities [7, 13, 14]. One major purpose of the present study, therefore, was to directly compare the ability of CT scanogram and plain film radiographs to identify HPVG. In our study, CT was found to be superior to plain radiography at detecting HPVG in patients with smaller volumes of gas. Radiographs showed

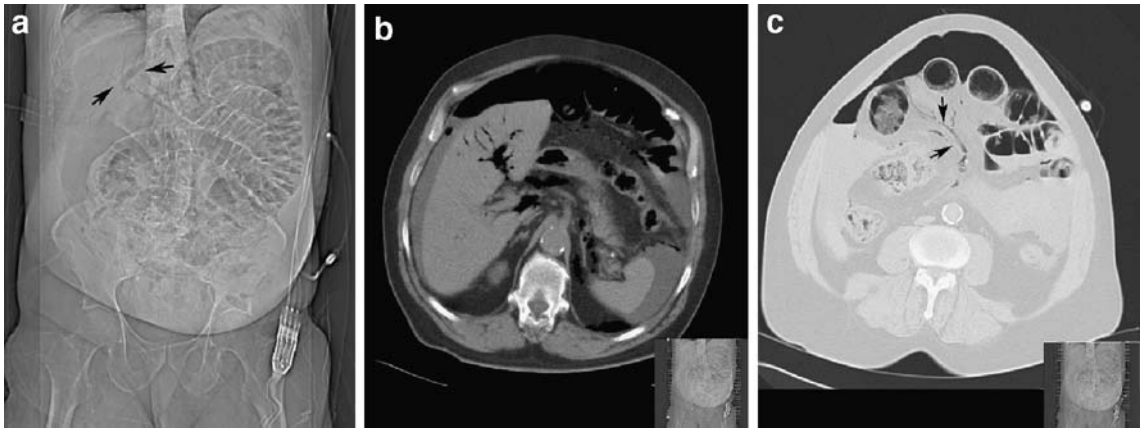

Fig. 2 A 54-year-old man (case #6) with diabetes mellitus. **a** CT scanogram demonstrates multiple branching tubular lucencies in the left liver lobe and at the level of the portal vein bifurcation (*arrows*). **b** Unenhanced CT scan reveals a large collection of portal gas in the left liver lobe (central portion and in the periphery of the liver). **c** Abdominal CT scan (lung window) shows pneumoperitoneum, pneumatosis intestinalis, and gas within the branches of the superior mesenteric vein (*arrows*)

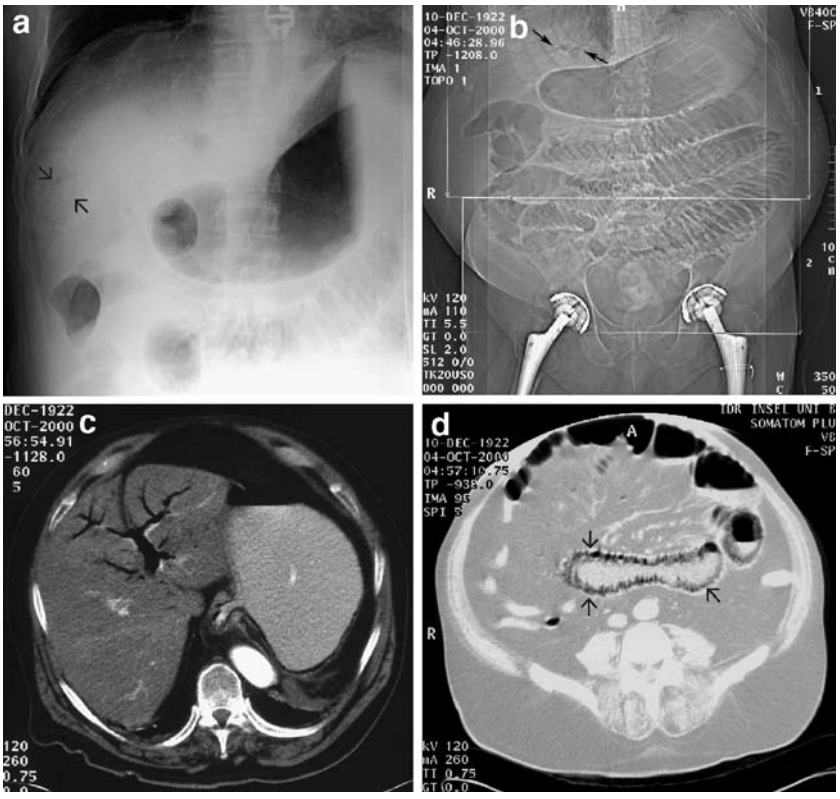

Fig. 3 A 78-year-old man (case #8) with small bowel obstruction caused by adhesions. **a** Plain abdominal radiography demonstrates HPVG in the periphery of liver (*arrows*). **b** CT scanogram shows tubular branching lucencies in the left liver lobe (*arrows*). **c** Contrast-enhanced CT scan during arterial phase reveals a large collection of portal gas in the left liver lobe. **d** Contrast-enhanced CT scan (lung window) demonstrates intramural bowel gas (*black arrows*)

portal venous gas in only one (12.5%) of eight patients in whom the corresponding CT scans revealed its presence. However, in three out of 11 patients (27.3%), HPVG was identified on the CT scanogram. All those patients presented with a substantial amount of gas at the time of the CT scan. The slightly higher sensitivity of CT sonograms compared to plain films for the detection of HPVG may be explained by the different examination times. CT scan and scanogram were acquired at the same time whereas abdominal plain films were obtained within a wider time frame. The whole process of the elaboration and absorption of the portal venous gas which predominantly consists of carbon dioxide and oxygen is dynamic. Continual replenishment of gas through the mesenteric system into the portal venous system should be present to enable the diagnosis of HPVG. At the time the plain film radiographs were assessed, the portal venous may already have been absorbed partially. Furthermore, a CT scanogram may be more valuable than a standard plain film because, usually, the whole liver is imaged whereas on plain films of the abdomen, especially on those obtained as a kidney, ureter, or bladder film, parts of the liver may be excluded. An accumulation of portal venous gas may consequently be situated above the upper margin of the film and may be excluded from the view.

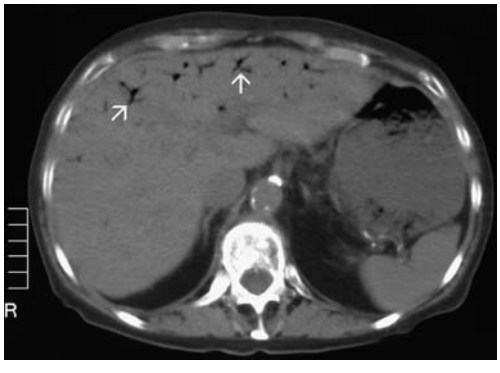

Fig. 4 A 73-year-old women (case # 11) with an abdominal aortic aneurysm and atrial fibrillation. Unenhanced CT scan shows linear gas collections in peripheral portal venous branches (*arrows*) extending close to the hepatic capsule

HPVG can occur alone or in association with pneumatosis intestinalis. When associated with pneumatosis intestinalis, it usually indicates the presence of mesenteric ischemia [18]. All of our patients with mesenteric infarction showed pneumatosis intestinalis on the CT scan. However, the coexistence of HPVG and pneumatosis intestinalis unrelated to mesenteric ischemia is also reported [6, 13, 19]. In our study, three patients had both findings in nonischemic conditions. In a recent paper, Wiesner et al. [18] noted that, in cases of intestinal ischemia, CT findings of pneumatosis intestinalis and HPVG do not generally allow prediction of transmural bowel infarction because they may also be observed in patients with only partial ischemic bowel wall damage. Moreover, no direct correlation between the amount of HPVG and mortality was found in our patients (Table 3), which accords with the study of Faberman and Mayo-Smith [7]. The presence of HPVG should, therefore, not be regarded as a direct predictor of mortality.

Bowel ischemia and mesenteric infarction are the most common causes of HPVG associated with prompt surgical intervention and an unfavorable clinical outcome. The

greater use of CT and ultrasound has improved the radiologists' sensitivity for detection of HPVG, especially in earlier stages [7, 18]. Furthermore, the increasing use of multislice CT in emergency situations has also led to increased detection of HPVG associated with various nonischemic conditions [7, 14, 18]. The HPVG in 45.4% of our patients had a nonischemic etiology, including intestinal obstruction (n=3), abdominal tumor (n=1), and one idiopathic case. In recent years, numerous papers, most of them case reports, have described other nonischemic causes of HPVG, including traumatic and/or iatrogenic events, Crohn's disease, ulcerative colitis, diverticulitis, severe pancreatitis, and even child abuse [3, 8–12]. The majority of these cases had a favorable outcome and many did not require surgical intervention. When portal venous gas is detected, therefore, the radiologist—in collaboration with the emergency room physician—must distinguish between life-threatening mesenteric ischemia and more benign nonischemic causes.

To identify the disease process underlying HPVG, an abdominal contrast-enhanced CT scan should be performed before therapy. A complementary lung window is recommended for identification of gas in the bowel wall (Figs. 2c and 3c) [14]. Faberman and Mayo-Smith [7] propose widening of the window width and centering of the window level closer to fat (to make the difference in density between fat and gas more evident) when the question of portal venous or mesenteric gas arises. The CT findings of bowel ischemia include arterial occlusion, venous thrombosis, bowel distension secondary to elevated air–fluid levels, bowel wall thickening, intense or absent mucosal enhancement, intestinal pneumatosis, portomesenteric vein gas, and ascites [1, 20]. Acute bowel ischemia can mimic various intestinal diseases because it can affect the small and/or large bowel and may be segmental or focal, diffuse, or localized as well as superficial or transmural [21]. Due to the difficult, nonspecific radiological manifestation of this disease entity, a robust clinical approach must be pursued to improve its detection and diagnosis.

An understanding of the pathogenesis of acute mesenteric ischemia with its various causes and clinical manifestations is essential to avoid unnecessary exploratory laparotomy. Surgical intervention, however, may sometimes be mandatory in patients with nonischemic conditions such as intestinal obstruction, traumatic injury, or severe inflammatory disease [2]. In the present study, surgery was performed in two patients with nonischemic causes for HPVG.

HPVG detected on radiographs was once considered to be an "ominous finding", with an overall mortality rate of 75% [6]. As HPVG is now usually detected at CT and not on radiographs, it is detected in a less advanced stage with consequent improved prognosis. In a large study of 17 patients, HPVG detected at CT was found to be caused by both ischemic and nonischemic conditions and had an overall mortality rate of 29% [7]. The 11 patients with HPVG in our study had a slightly lower mortality of 27.3%. Even so, the clinical outcome of our patients with HPVG caused exclusively by intestinal ischemia (n=6) was less favorable, with a mortality of 50%. Two recent investigations on HPVG reported similar mortality rates of 43% (n=7) and 56% (n=17) in patients with bowel wall ischemia [18, 22]. Thus, the presence of portomesenteric venous gas in patients with bowel ischemia remains ominous compared to nonischemic causes of HPVG.

The limitations of this study are its relatively small patient sample and its retrospective character. HPVG is a rare radiographic finding with not more than 182 reported cases in the literature up to 2001 [23]. Furthermore, the fact that the plain film radiographs were not assessed at the same time as the CT scan could create a bias against the plain film's efficiency. Our results do, however, point to the continued need for a further prospective study.

In conclusion, HPVG is an important radiological finding associated with many pathological processes and not to be regarded solely as a prognostic factor. As shown here, CT is more sensitive than plain film radiographs and CT scanograms in the detection of HPVG and its underlying diseases and, therefore, merits use as the diagnostic tool of choice. With regard to clinical outcome, two groups of patients with HPVG can be defined: patients with ischemic and patients with nonischemic causes, with the former group having a predictably higher mortality. Patients with a radiographic finding of HPVG should be given a detailed history review and physical examination to determine their underlying condition and provide a sound basis for exploratory laparotomy.

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