Hepatic–Portal Venous Gas in Adults:

Etiology, Pathophysiology and Clinical Significance

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The roentgenographic finding of hepatic–portal venous gas (HPVG) has been reported extensively in the pediatric and radiology literature. The surgical implications and clinical significance have yet to be fully defined. This study reviews the 60 reported cases in the literature and adds four new cases. HPVG appears as a branching radiolucency extending to within 2 cm of the liver capsule. HPVG is associated with necrotic bowel (72%), ulcerative colitis (8%), intra-abdominal abscess (6%), small bowel obstruction (3%), and gastric ulcer (3%). Mucosal damage, bowel distention and sepsis predispose to HPVG. The current mortality rate of 75% represents an improvement from previous experience. Analysis of survivors indicates that the finding of HPVG requires urgent surgical exploration except when it is observed in patients with stable ulcerative colitis.

THE OCCURRENCE OF GAS in the portal venous system of infants dying of abdominal catastrophes was first noted in 1955.[37] Later this finding was reported in an adult, critically ill with small bowel infarction.[30] Since that time hepatic–portal venous gas (HPVG) has been reported with increasing frequency in the radiology[11,19,27,31] and pediatric literature.[1,3,34] However, the surgical implications and clinical significance of this finding in the adult have yet to be fully defined.

This paper reviews the 60 reported cases of adults with HPVG and adds four cases of our own. The etiology, pathophysiology and clinical significance of HPVG are discussed.

Case Reports

R.G. (UH-734946) A 65-year-old white man with a long history of chronic obstructive pulmonary disease and arteriosclerotic peripheral vascular disease necessitating a right above-the-knee amputation was admitted to University Hospital with three day history of nausea, vomiting and abdominal pain. Physical examination revealed rales bilaterally and a grossly distended abdomen with decreased bowel sounds. There was no abdominal tenderness. A large right inguinal hernia was noted. There was no tenderness over the hernia which was partially reducible. Plain x-ray films of the abdomen revealed distended large and small bowel. Hepatic portal venous gas (HPVG) was present but noted only in retrospect.

The patient was treated with nasogastric suction and intravenous fluids. He failed to improve and developed progressive respiratory failure. Repeat x-ray examination of the abdomen failed to reveal evidence of HPVG. Forty-eight hours after admission the patient underwent exploratory laparotomy. A large portion of the ileum was found incarcerated in a right indirect inguinal hernia. The bowel was dusky but there was no gangrene or perforation and a resection was not performed. The patient's postoperative course was stormy, complicated by respiratory failure and wound sepsis, but he recovered and was discharged on his ninety-sixth postoperative day.

G. McP. (UH-551522). A 69-year-old white woman, two months postsigmoid resection for adenocarcinoma with evidence of metastatic disease, underwent a four and one-half week course of radiotherapy consisting of 4500 rads and was then admitted to University Hospital with abdominal pain and diarrhea. Her physical examination revealed mild abdominal distention with no tenderness. Suddenly on the second hospital day the patient became hypotensive with abdominal pain and bloody diarrhea. X-rays at this time revealed an extensive pattern of HPVG and intramural intestinal gas. The patient died four hours after this x-ray.

J.C. (UH-551838). A 58-year-old white man with a history of myocardopathy and mitral regurgitation was admitted to an outlying hospital in congestive heart failure with evidence of a pulmonary embolism. He sustained a cardiopulmonary arrest from which he was resuscitated but remained semi-comatose. He was transferred to University Hospital. Soon after admission the patient developed right-sided abdominal pain and became hypotensive. Plain x-rays of the abdomen revealed HPVG with evidence of intramural intestinal gas. The patient underwent urgent laparotomy which revealed a necrotic cecum and ascending colon. The superior mesenteric artery was patent. A right colectomy was performed with the

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Supported in part by the Department of the Army Contract DA 17-76-C-6034 and The National Institute of Health Contract #2-RO1-GM-173-66-07-CO2.

Submitted for publication: June 22, 1977.

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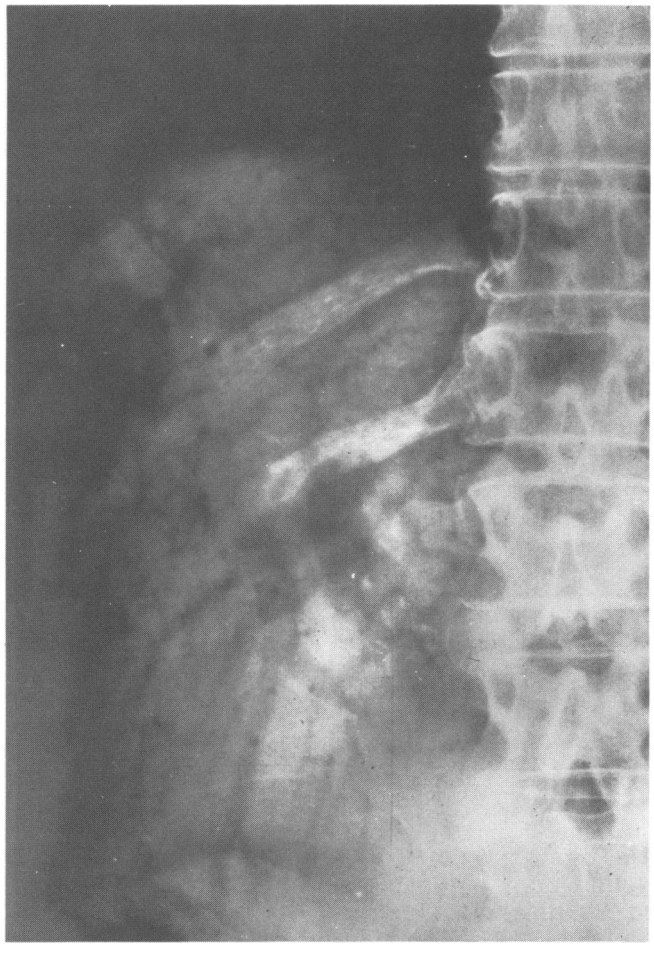

FIG. 1. Plain abdominal roentgenogram of G. McP. (Table 1, Case #62) showing extensive HPVG. Note characteristic branching radiolucencies extending to periphery of liver.

patient experiencing an intraoperative cardiac arrest from which he was resuscitated. His postoperative course was complicated by renal failure, respiratory failure and extreme bilirubinemia despite modestly elevated or normal liver enzymes.

W.V. (HGH-356395). A 23-year-old white man with a history of chronic ulcerative colitis felt lightheaded and experienced a shaking chill during a routine barium enema examination. X-ray revealed HPVG. The patient was admitted to Harbor General Hospital and treated with antibiotics. Urine and blood cultures were negative and the patient made an uneventful recovery.

Radiographic Finding

The radiographic pattern of HPVG must be differentiated from that seen with biliary tract gas. The pattern of HPVG has been described as a branching radiolucency extending nearly to the liver capsule (Fig. 1). Susman and Senturia[30] state that gas in the biliary tract is carried toward the porta hepatis by the centripetal flow of bile and therefore appears central in the liver while HPVG travels peripherally, enhanced by the centrifugal flow of blood. Sisk[27] analyzed T tube cholangiograms and found no instance in which contrast material came within 2 cm of the liver capsule, while all normal portal venograms demonstrated venous radicles extending almost to the liver periphery. The radiographic criteria for HPVG is a branching radiolucency extending to within 2 cm of the liver capsule. X-rays are most revealing of HPVG when taken with the patient in the left lateral decubitus position.

HPVG has been described in the literature as portal vein gas, pneumoportogram or gas embolization of the portal vein, *etc.* We have used the term hepatic–portal venous gas or HPVG to describe this entity. Gas in the portal venous system can only be clearly noted as a radiolucency in the intrahepatic portal radicles of the liver, thus the rationale for use of the descriptive term "hepatic" in HPVG.

Intramural bowel gas is a commonly associated finding with HPVG and is secondary to necrotic bowel. It is characterized by a thin, continuous, crescentic radiolucency within distended bowel wall.[22] Intramural gas is not a necessary accompaniment of HPVG even in the presence of bowel infarction.

Associated Clinical and Pathologic Findings

HPVG occurs in a variety of clinical settings and pathological conditions at a mean age of 58 years (Table 1). There is no difference in male–female incidence. Necrotic bowel is associated with HPVG in over two-thirds of the cases (Table 2). However, bowel necrosis need not be present and even in the patients with necrotic bowel, HPVG can offer no information concerning the extent of the necrosis. The infarcted segment may vary from almost total small bowel destruction (Table 1, case #38,43), to a limited area of necrosis (Table 1, case #5,63). In some instances HPVG is associated with apparent reversible ischemia (Table 1, case #45,61).

In adults, one iatrogenic cause of HPVG has been identified. This is a barium enema examination (with or without air contrast) in patients with ulcerative colitis.

Intra-abdominal sepsis is also associated with HPVG. Interestingly there are no reports of HPVG in patients with ascending cholangitis, acute suppurative cholecystitis, liver abscess, appendicitis, or diverticulitis.

Pathophysiology

The factors that predispose to HPVG are: 1) mucosal damage as seen with infarcted bowel, ulcerative colitis, or peptic ulcer disease (86%), 2) bowel distention revealed by x-ray (85%); and 3) sepsis defined by positive blood cultures, dead bowel, spreading celluli-

TABLE 1. *Clinical and Pathological Features of HPVG*

Case No.	Reference No.	Age	Sex	Clinical Findings & Course	Survival After Dx	Pathology	Septic Source	Distention	Mucosal Damage
1	30	77	M	Diarrhea, blood in rectum, atrial fibrillation. Nasogastric suction produced "coffee grounds." Op:* Exploration.	24 hrs	Superior mesenteric artery thrombosis gangrene SB†	Yes DB‡	++	Yes
2	27	19	F	Mental defective. Abdominal distention. Nasogastric suction produced bloody fluid. Op: Exploration.	<4 hrs	Acute hemorrhagic pancreatitis. Dilated stomach, duodenum.	No	+++	No
3	27	81	F	Vomiting, distended, hypoactive bowel sounds. Op: Resect. SB.	7 days	Incarcerated ileum & appendix in obturator fossa. Purulent peritonitis, gangrene SB. Acute cholecystitis, centrilobar necrosis.	Yes	+++	Yes
4	27	84	M	Sore foot. Chest pain. Crampy Abd. pain, nausea. UGI was normal. After 1 wk. became worse, distention.	<4 hrs	Bronchopneumonia. ASCVD. Jejunal necrosis. Dilatation of aorta. Ca of prostate.	Yes DB	++	Yes
5	36	63	M	Abdominal pain, distention, diarrhea. Rectal exam revealed watery, bloody stool. Op: Resect. SB.	7 days	Distal colon & SB showed areas of necrosis. Prepyloric ulcer penetrating to pancreas.	Yes DB	++	Yes
6	36	35	M	Diabetic ketoacidosis.	8 hrs	SB distention. No ulceration or necrosis.	No	++	No
7	36	52	F	4-day hx. of abdominal pain, diarrhea, vomiting. Diabetic.	4 hrs	SMA§ occlusion; SB areas of necrosis.	Yes DB	++	Yes
8	2	60	M	Abdominal pain.	6 hrs	Gangrene SB.	Yes DB	?	Yes
9	2	85	F	Abdominal pain; shock.	6 hrs	Necrosis duodenum to anus.	Yes DB	?	Yes
10	14	54	F	Vomiting, diarrhea. Hx. of CHF, cardiomyopathy. Op: Exploration.	<24 hrs	SMA; embolism from mural cardiac thrombus. Gangrene SB and ascending colon.	Yes DB	0	Yes
11	14	69	M	Abdominal pain. Atrial fibrillation.	<24 hrs	Gangrene SB; SMA thrombosis.	Yes DB	+	Yes
12	29	75	F	Vomiting, distention, diarrhea. Abd. pain 3 days PTA; Rectal-black stool. Hx. of aortic stenosis & myocardial infarction.	<8 hrs	SMA embolism. SB gangrene.	Yes DB	++	Yes
13	29	68	F	Rheumatoid arthritis. Crampy abd. pain, vomiting.	24 hrs	Mesenteric vein thrombosis; 1.5 m ileum gangrenous	Yes DB	++	Yes
14	29	84	M	Lower abd. pain; vomited. Op: Exploration.	<24 hrs	Mesenteric artery occlusion.	Yes DB	0	Yes
15	17	30	F	4½ yrs. after SB & prox. colon resection, secondary to malrotation—abdominal pain, bloody NG drainage.	2 days	Areas of necrosis and ulceration of colon.	Yes DB	0	Yes
16	17	82	F	Epigastric pain, nausea, vomiting—3 wks. Stool was + occult blood.	34 hrs	SB and colon necrosis.	Yes DB	++	Yes
17	6	69	M	Abd. pain, shock. Macroglobulinemia.	2 hrs	Necrotic SB.	Yes DB	+	Yes
18	16	48	M	Double-contrast Ba enema. Crampy abd. pain followed w. distention, emesis.	Survived	Free peritoneal gas and HPVG	Yes	+	Yes
19	20	?	?	?	?	Mesenteric thrombosis.	Yes DB	?	Yes
20	20	?	?	?	?	Mesenteric thrombosis.	Yes DB	?	Yes
21	20	?	?	?	?	Mesenteric thrombosis.	Yes DB	?	Yes
22	20	?	?	?	?	Strangulation, obstruction.	Yes DB	+	Yes

TABLE 1. (*Continued*)

Case No.	Reference No.	Age	Sex	Clinical Findings & Course	Survival After Dx	Pathology	Septic Source	Distention	Mucosal Damage
23	20	?	?	?	?	Perforated ulcer with necrosis.	Yes DB	?	Yes
24	18	71	M	Abd. pain, vomiting.	2 hrs	Necrosis duodenum to lower ileum.	Yes DB	+	Yes
25	21	79	F	Epigastric pain, vomiting. Op: Exploration.	<7 hrs	Infarction SB and LB.	Yes DB	+	Yes
26	24	61	F	Crampy, abd. pain. Op: Small bowel resection.	<36 hrs	Volvulus SB; perforated duodenum; Patchy necrosis entire SB.	Yes DB	+	Yes
27	24	72	F	? myocardial infarction. Nasogastric suction produced red blood. Op: Exploration.	<6 hrs	Necrosis from esophagus to distal sigmoid.	Yes DB	+++	Yes
28	8	44	F	Vomiting. Ingestion of hydrochloric acid.	<6 hrs	Necrotizing gastritis and proximal enteritis.	No	0	Yes
29	8	57	M	Ingestion of antifreeze. Nasogastric suction and rectal exam showed red blood.	<6 hrs	Necrotic gastrointestinal mucosa.	No	0	Yes
30	19	58	F	Epigastric pain, nausea & vomiting; hematemesis, hematocolpia. Op: Small bowel resection.	Survived	SB gangrene 125 cm., secondary to band obstruction.	Yes DB	+	Yes
31	5	13	M	Mentally retarded—cerebral palsy. Shock: distended abdomen.	Survived	None	No	++	No
32	13	49	F	Ca of cervix with recto-vaginal fistula, treated with loop colostomy. Op: Lyse adhesion; abscess drained.	18 days	Pelvic abscess. No ischemia.	Yes Pelvic abscess	++	No
33	7	60	F	Megaloblastic anemia. Nausea, abd. cramps, bloody diarrhea. Op: Small bowel resection.	Survived	Gangrene SB; SMA thrombosis.	Yes DB	++	Yes
34	7	42	F	UGI bleed; gastric ulcer. Op: Subtotal gastrectomy.	Survived	Gastric ulcer	No	0	Yes
35	9	58	M	Burns. Bloody nasogastric suction.	6 hrs	Infarction—stomach, duodenum, ileum, cecum, pancreas. Portal vein thrombosis, SMA & celiac embolism.	Yes DB	?	Yes
36	9	64	M	Hypertensive. Aortic abd. aneurysm resected. Abd. pain, hematemesis.	1 day	Hemorrhagic exudate—esophagus, stomach, SB. Myocardial infarction.	No	?	Yes
37	9	47	M	Abdominal pain, fever. Op: Drain abscess.	10 days	Multiple colonic fistulas. Retroperitoneal abscess.	Yes Retroperitoneal abscess	?	Yes
38	9	39	M	GSW right thigh. Abd. pain. Nasogastric suction produced blood. Op: Exploration.	1 day	Necrosis duodenum to anus.	Yes DB	?	Yes
39	35	35	M	Ulcerative colitis, asthma. Ba enema.	Survived	None	No	+	Yes
40	33	?	?	Bowel infarction.	?	SMA thrombosis.	Yes DB	?	Yes
41	33	?	?	Bowel infarction.	?	SMA thrombosis	Yes DB	?	Yes
42	33	?	?	Bowel infarction.	?	?	Yes DB	?	Yes
43	4	34	M	Hematemesis, extreme abd. distention, hematochezia. Op: Exploration.	24 hrs	Stomach—cecum necrotic, duodenal ulcer.	Yes DB	+++	Yes
44	4	58	M	Abd. pain, congestive heart failure, ascites.	Survived	None	No	?	No
45	4	72	F	Vomiting. Op: Small bowel resection.	Survived	Band obstruction ileum; no necrosis.	No	—	No

TABLE 1. (*Continued*)

Case No.	Reference No.	Age	Sex	Clinical Findings & Course	Survival After Dx	Pathology	Septic Source	Distention	Mucosal Damage
46	4	?	?	?	?	Enteric necrosis.	Yes DB	?	Yes
47	4	?	?	?	?	Enteric necrosis.	Yes DB	?	Yes
48	4	?	?	?	?	Enteric necrosis.	Yes DB	?	Yes
49	4	?	?	?	?	Enteric necrosis.	Yes DB	?	Yes
50	4	?	?	?	?	Enteric necrosis.	Yes DB	?	Yes
51	4	?	?	?	?	Enteric necrosis.	Yes DB	?	Yes
52	4	?	?	?	?	Enteric necrosis.	Yes DB	?	Yes
53	4	?	?	?	4 days	Necrosis of SB.	Yes DB	?	Yes
54	23	?	?	?	?	?	Yes DB	?	Yes
55	15	44	F	2 episodes of HPVG: Biopsy 3 days prior to air contrast Ba enema; 12 mos. later post-evacuation film during Ba enema.	Survived	Ulcerative colitis.	No	+	Yes
56	26	70	M	2 days asymptomatic melena, hematemesis. Op: 70–80% gastrectomy.	Survived	Gastric ulcer	No	+++	Yes
57	28	79	M	Ulcerative colitis. 4 yrs. previously seen with pain & fever.	Survived	Ulcerative colitis.	No	++	Yes
58	12	58	F	Shock, melena, alcoholism. Op: 1) Negative exploration; 2) Abscess drained 3 wks. later.	Survived	Perforated diverticulum of sigmoid with abscess.	Yes Blood culture	+	No
59	32	56	F	Total gastrectomy for Z.E.; linear tear, esophagus. Op: Drain abscess.	Survived	Abscesses—Right lower quadrant and Right subdiaphragmatic.	Yes Perforated Esophagus	+	Yes
60	10	40	F	3rd post-op. day—Abd. hysterectomy & unilateral salpingo-oophorectomy.	12 hrs	Patchy necrosis entire SB.	Yes DB	+	Yes
61	R.G.	65	M	Abd. pain, nausea & vomiting, shock. Op: No resection.	Survived	70% SB. No necrosis.	No	++	No
62	G. McP.	69	F	Radiation vasculitis, abd. pain, diarrhea	12 hrs	Radiation vasculitis.	Yes DB	++	Yes
63	J.C.	58	M	Abd. pain, Myocardopathy. Op: Resection right colon.	Survived	Necrotic cecum.	Yes	++	Yes
64	W.V.	23	M	Ulcerative colitis—Ba enema.	Survived	Ulcerative colitis.	No	++	Yes

* Op = Operation. † SB = Small bowel. ‡ DB = Dead bowel. § SMA = Superior mesenteric artery.

tis or abscess (88%). Many patients show two of these findings (50%) and some show three (35%). Only one patient in this series failed to exhibit at least one of these factors (Table 1, Case #45). For example, case #1 had superior mesenteric artery thrombosis with bowel distention, peritonitis and bowel necrosis, *i.e.*, all factors frequently associated with HPVG. Case #34 showed no distension and negative blood cultures but had a penetrating lesser curvature gastric ulcer (mucosal damage) and evidence of HPVG. Case #37 had HPVG associated with retroperitoneal abscesses as a complication of pancreatitis and no evidence of mucosal damage or bowel distention. Case #2 showed evidence of HPVG and extreme abdominal distention. No signs of sepsis or mucosal injury were present.

In 41 patients of this study a clinical history and physical examination were available. In 35 cases (85%) abdominal distention was noted. Twenty-two were observed to have extreme distention. It appears that increased intraluminal pressure contributes to the production of HPVG in many instances.

TABLE 2. *Pathologic Findings in 64 Patients with HPVG*

	No.
Necrotic Bowel (46 patients, 72%)	
Non-occlusive mesenteric infarction	24
Superior mesenteric artery thrombosis	11
Strangulated bowel secondary to adhesions, hernia, volvulus and malrotation	4
Superior mesenteric artery embolism	3
Toxic ingestion	2
Superior mesenteric vein thrombosis	2
Superior mesenteric vein thrombosis	1
Perforated ulcer with necrotic bowel	1
Ulcerative Colitis (5 patients, 8%)	
Secondary to enema	4
Spontaneous	1
Intra-Abdominal Abscess (4 patients, 6%)	
Perforated sigmoid diverticulum with abscess	1
Right pelvic and right subphrenic abscess	1
Pelvic abscess	1
Retroperitoneal	1
Bowel Obstruction, No Necrosis (2 patients, 3%)	
Adhesive band	1
Inguinal hernia	1
Gastric Ulcer	2
Acute Hemorrhagic Pancreatitis	1
Diabetic Ketoacidosis	1
Unknown Etiology	3
	64 TOTAL

Mucosal damage may be secondary to necrotic bowel, ulcerative colitis, or ulcer disease. Such injury provides a portal for intraluminal gas to enter the portal venous system. Further, the intestinal mucosal barrier to gas may be altered by mechanical distention alone without other specific injury. This has been determined by a hydrogen peroxide enema dog preparation in which mesenteric venous gas was produced.[25]

Sepsis alone without dead bowel is an infrequent cause of HPVG. In 6% of patients, HPVG was associated with an intra-abdominal abscess without either intestinal distention or mucosal damage. In these circumstances HPVG may have been induced by a gas forming organism.

Clinical Implication of HPVG

HPVG is not a specific disease entity but merely another diagnostic clue in patients suffering acute abdominal pathology. The approach to the patient with HPVG should be directed to the underlying disease.

HPVG may be more common than realized. The transient nature of HPVG is illustrated by our case reports as well as those of other authors. The portal gas composition has been analyzed and found to have a high CO_2 content.[36] This highly soluble gas would be expected to exist only briefly in the vascular system before being absorbed or removed by bulk flow, unless the gas production persisted.

As late as 1968, HPVG was viewed as an inevitable harbinger of death. The mortality rate was over 90% Analysis of the current series demonstrates the mortality to be 75% with 16 survivors. Excluding so-called iatrogenic cases, the mortality is 81%.

In reviewing the 16 survivors of this series we find nine patients who had an operation to resect dead bowel, relieve obstruction, treat bleeding ulcers, or drain sepsis. The five patients with ulcerative colitis all survived without surgery. Two patients without specific abdominal findings did not undergo operative exploration and survived.

The data indicate that most HPVG is associated with acute abdominal pathology. These patients require urgent surgical exploration. In contrast, patients with stable ulcerative colitis may develop HPVG after barium enema examinations, and require only supportive therapy and observation.

Summary

Hepatic portal venous gas (HPVG) presents a pattern of a branching radiolucency extending almost to the periphery of the liver. In the adult, this finding usually occurs in a variety of clinical situations: in patients seriously ill with necrotic bowel (72%), intra-abdominal abscess (6%), bowel obstruction (3%), bleeding gastric ulcers (3%), and following barium enemas in patients with stable ulcerative colitis (8%). Mucosal damage, bowel distention and sepsis are prominent conditions associated with HPVG. Virtually all patients have one or more of these factors. HPVG is often a transient finding which may be more common than realized. The mortality rate is 75%. Analysis of survivors indicates that urgent abdominal exploration is needed in all instances of HPVG with the notable exception of HPVG occurring in ulcerative colitis.

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