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OPEN ACCESS

Journal of Surgery

Chao WS, et al. J Surg 5: 1299.

DOI: 10.29011/2575-9760.001299

Research Article

Appropriate Clinical Management in Patients with Acute Abdomen and Hepatic Portal Vein Gas

Wen-Shan Chao[1], Cheng-Chung Wu[2,5*], Chih-Chiang Hung[3,4], Chuan-Hsun Chang[5]

[1]Division of General Surgery, Tung's Taichung Metro Harbor Hospital, Taichung, Taiwan

[2]Department of Surgery, Taichung Veterans General Hospital, Taichung, Taiwan

[3]Division of Breast Surgery, Department of Surgery, Taichung Veterans General Hospital, Taichung, Taiwan,

[4]Department of Applied Cosmetology, College of Human Science and Social Innovation, Hungkuang University, Taichung, Taiwan

[5]Department of Surgery, Cheng-Hsing general hospital, Taipei Taiwan

[*]**Corresponding author:** Cheng-Chung Wu, Department of Surgery, Taichung Veterans General Hospital, 1650 Taiwan Boulevard Sect. 4, Taichung 40705, R.O.C, Taiwan

Citation: Chao WS, Wu CC, Hung CC, Chang CH (2020) Appropriate Clinical Management in Patients with Acute Abdomen and Hepatic Portal Vein Gas (HPVG). J Surg 5: 1299. DOI: 10.29011/2575-9760.001299

Received Date: 17 March, 2020; **Accepted Date:** 27 March, 2020; **Published Date:** 31 March, 2020

Abstract

Background: Hepatic Portal Venous Gas (HPVG) is a well-known, rare, but ominous radiological sign in patients with acute abdomen. The outcomes of the patients with HPVG is uncertain. The purpose of this study is to review our experiences and establish an appropriate clinical decision in patients with acute abdomen and HPVG.

Methods: We retrospective searched the patient with the key words: HPVG, bowel ischemia, acute abdomen at tertiary care academic medical centers. 20 patients were obtained from February 2008 to March 2018. The early and long-term outcomes were studies and analyzed.

Results: The patients included eleven males and nine females, with an age range of 40-87 years old. Sixteen patients underwent surgical intervention, and 6 of them died of severe ischemic bowel. The other four patients did not have surgery, 2 of them died of moribund status secondary to severe bowel ischemia; the other 2 survivors received conservative treatment because of equivocal clinical presentation. Preoperative shock was the only impact factor for mortality in this series. No recurrent HPVG in the twelve survivors after followed-up period 12-120 months.

Conclusion: HPVG is an episodic presentation in acute abdomen but not an indication for surgical intervention. The mortality rate in patients with both HPVG and bowel ischemia was high but sometimes with equivocal presentation. Thus, for patients with acute abdomen, negative laboratory findings and physical examinations (PE), and CT-proven presence of portal venous gas still need for further investigations.

Keywords: Acute abdomen; Bowel ischemia, Hepatic portal venous gas

Introduction

Hepatic Portal Venous Gas (HPVG) is a well-known, rare, but ominous radiological sign, which indicates a critical clinical condition with a mortality rate of over 75% [1]. The major cause of HPVG was intestinal mucosa injury combined with overgrowth of gas-producing bacteria [1-4]. The composition of portal gas has been analyzed and found to have a high CO_2 content [5]. The mortality rate fell to 29% after following the widespread use of Computed Tomography (CT) [6], which has a higher sensitivity for the detection of HPVG than plain abdominal radiography [6-8]. The presentation of patients is varied and the diagnosis of the underlying problem depends mainly on the clinical symptoms and CT finding [7-9]. The common causes of HPVG are bowel ischemia, inflammation and/or infection of the Gastrointestinal Tract (GIT), and GIT cancer [1,9]. Because of the uncertainty of this radiological sign, the variety of underlying diseases, and the

high potential mortality rate, the clinical management should be comprehensive. In this study, we present our clinical experience, describe the outcomes of 20 patients with long-term follow up period 12-120 months, and provide a review of the literature on HPVG in acute abdomen.

Patients and Methods

A retrospective review was conducted from February 2008 to March 2018. The following key words were used to search for relevant cases in the literature: hepatic portal venous gas, bowel ischemia, and acute abdomen. Findings were confirmed by re-review of the original CT to determine the presence of HPVG. A total of 20 patients were identified, and their long-term outcomes, spanning a period ranging from 12 to 120 months, were recorded.

After completing the analysis of radiographic findings, a chart review of the 20 patients with HPVG was then conducted. Characteristics of patients, underlying diseases, presenting signs and symptoms, other imaging findings, presence of shock, defined as systolic blood pressure less than 90 mmHg [10], surgical versus nonsurgical management (Table 1), clinical course, and long-term outcomes were also reviewed.

Case #/ Age/sex	Underlying disease	Abd. S/S	Shock	Other CT findings	DPL	Final Diagnosis	Treatment
Survived							
1/71/F	Uremia s/p H/D, HTN, DM, Cervical cancer s/p radiation therapy complicating with radiation enteritis	Yes	No	PI with PE, MG, SBD	Nil	Bowel ischemia	Segmental resection of small bowel
2/87/M	HTN	Yes	No	PI	Nil	Enteritis	Conservative
3/45/M	CAD-1 s/p PCI (2008), HTN	Yes	No	PI	Nil	Enteritis	Exploratory laparotomy
4/84/M	CAD,HTN,DM, CHF (LVEF:41)	Yes	No	PI with PE, MG	Nil	Bowel ischemia	Segmental resection of small bowel
5/85/M	DM, HTN	Yes , Fever, bloody stool	No	T-colon wall thickening	WBC: 802	Entero-colitis	Conservative
6/80/M	Arrhythmia	Yes	No	Free air, ascites	Nil	PPU	Gastrojejunostomy+ Gastric partition
7/40/F	s/p myotomy (2014)	Yes	Yes	Bowel distension with PI	Nil	SBO	Lysis
8/87/F	Arrhythmia, DM, HTN	Yes	No	PE	Nil	Cholecyst-itis	cholecystectomy
9/63/M	Idiopathic lung fibrosis with chronic respiratory failure	Yes	No	PI with PE Diffuse bowel distension	Nil	Enteritis (ileitis)	Exploratory laparotomy
10/68/M	COPD	Yes	No	PI with PE, SBD, ascites	Nil	Ruptured appendic-itis	Appendectomy and drainage of interloop abscess

11/65/M	Tonguecancer, cT2N1, stage 3 s/p neoadjuvant, DM	Yes	Yes	thickening small bowel PI and PE	Nil	Bowel ischemia with perforation and acute GI bleeding	Segmental resection of small bowel
12/80/M	COPD, DM , CKD stage 3, CAD-3 s/p CABG, CHF	Yes	Yes	Diffuse thickening of bowel, PI and PE	Nil	Bowel ischemia	Segmental resection of small bowel
Expired							
13/75/F	Uremia	Yes	Yes	PI with PE, MG	Nil	Bowel ischemia	Segmental resection of small bowel
14/77/F	CAD, DM Stroke, Uremia, HTN	Yes	Yes	PI with PE, MG	WBC: 7400	Bowel ischemia	Segmental resection of small bowel
15/45/F	PSS	Yes	No	PI with PE, MG	Nil	Bowel ischemia	Exploratory laparotomy (open and closed)
16/87/M	CAD-III, HTN, DM	Yes	Yes	PI with PE, MG	Nil	Bowel ischemia	Refused treatment.
17/71/F	Uremia, DM	Yes	Yes	PI with PE, MG	Nil	Bowel ischemia	Right hemicolectomy
18/68/F	Uremia , DM	Yes	Yes	PI, Free air	Nil	Bowel ischemia	Right hemicolectomy
19/82/M	DM, HTN, CKD, dementia,	Yes	No	PI with PE at proximal jejunum	Nil	Bowel ischemia	Refused treatment
20/72/F	Critical aortic stenosis,	Yes	Yes	PI	Nil	Bowel ischemia	1.Small intestine resection of small bowel 2. enterostomy,

Abd. S/S = abdominal symptom/sign; S/P = status post; H/D = hemodialysis; CAD = Coronary artery disease; CHF = chronic heart failure; CKD = Chronic kidney disease; HTN = Hypertension; DM = Diabetes;

COPD = chronic obstructive pulmonary disease; Obst. = obstruction; MG = mesenteric gas;

PI = pneumatosis intestinalis; PE = poor enhancement of intestine; PSS = Progressive systemic sclerosis;

SBD = small bowel dilatation; SBO = small bowel obstruction

Table 1: Characteristics of patients demonstrating hepatic portal venous gas in acute abdomen.

Twelve of the 20 patients survived and were discharged, while 8 died (mortality rate was 40%). We analyzed their presenting signs and symptoms (including vital signs and peritoneal sign, which were examined by an experienced general surgeon), abdomen CT findings, and laboratory findings. Laboratory examinations included white blood cell count (WBC), serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), international normalized ratio (INR), total bilirubin, albumin, creatinine (Cr.), lactic dehydrogenase (LDH), creating kinase (CK), C-reactive protein (CRP), blood gas (pH), and amylase (Table 2).

	Survive (n=12)		Expire (n=8)		*p* value
Age	75.5	(63.5, 84.75)	73.5	(68.8, 80.8)	0.969
Gender (%)					0.065
F	3	(25.0%)	6	(75.0%)	
M	9	(75.0%)	2	(25.0%)	
Cardiopulmonary (%)	10	(83.3%)	8	(100.0%)	0.495
OP performed (%)	10	(83.3%)	6	(75.0%)	1.000
Physical examination					
Pain (%)	11	(91.7%)	7	(100.0%)	1.000
Vomit (%)	8.0	(66.7%)	4.0	(57.1%)	1.000
Peritoneal signs (%)	11.0	(91.7%)	6.0	(100.0%)	1.000
Shock (%)	1.0	(8.3%)	5.0	(83.3%)	**0.004****
Systolic BP (mmHg)	111.0	(94.0, 135.0)	84.0	(76.3, 107.3)	0.078
HR (/min)	89.0	(71.0, 122.0)	108.5	(96.0, 124.3)	0.315
BT (C)	36.0	(35.7, 37.3)	35.9	(35.3, 37.3)	0.391
RR (/min)	20.0	(18.0, 21.0)	22.0	(17.5, 26.0)	0.411
Image finding					
Ascites (%)	6.0	(54.5%)	2.0	(40.0%)	1.000
Free air (%)	2.0	(18.2%)	0.0	(0.0%)	0.515
Gastroduodenal dilatation (%)	4.0	(40.0%)	3.0	(50.0%)	1.000
Small intestinal dilatation (%)	6.0	(60.0%)	6.0	(100.0%)	0.234
Large intestinal dilatation (%)	1.0	(10.0%)	1.0	(16.7%)	1.000
Poor enhancement of the bowel (%)	5.0	(50.0%)	5.0	(83.3%)	0.307
Intestinal pneumatosis (%)	9.0	(90.0%)	6.0	(100.0%)	1.000
Mesenteric pneumatosis (%)	5.0	(50.0%)	6.0	(100.0%)	0.093
PVG (%)	12	(100.0%)	8	(100.0%)	--
PI (%)	8	(66.7%)	5	(62.5%)	1.000
Laboratory					
INR (0.85-1.15)	1.15	(1.1, 1.3)	1.98	(1.1, 7.3)	0.281
CK (U/L)	84.5	(43.0, 159.5)	96.0	(26.3, 221.3)	1.000
WBC (M:3900-10600; F:3500-11000)					0.117
Normal	4	(33.3%)	0	(0.0%)	
Abnormal	8	(66.7%)	8	(100.0%)	
CRP (mg/dL)(< 0.3 mg/dL)					0.400
Normal	0	(0.0%)	1	(12.5%)	
Abnormal	12	(100.0%)	7	(87.5%)	
pH (7.35-7.45)					0.642
Normal	3	(25.0%)	3	(37.5%)	

Abnormal	9	(75.0%)	5	(62.5%)	
BE	-0.9	(-6.5, -0.1)	-2.7	(-9.1, 0.3)	0.253
T-BiL (mg/dL)	0.7	(0.4, 0.9)	0.6	(0.4, 3.6)	0.906
AST (U/L)(8 - 38)					0.070
Normal	7	(58.3%)	1	(12.5%)	
Abnormal	5	(41.7%)	7	(87.5%)	
ALT (U/L)(M:10-50,F:10-35)					0.167
Normal	9	(75.0%)	3	(37.5%)	
Abnormal	3	(25.0%)	5	(62.5%)	
LDH (U/L)(120-240)					0.242
Normal	3	(25.0%)	0	(0.0%)	
Abnormal	9	(75.0%)	8	(100.0%)	
Amylase(20-140)					1.000
Normal	6	(50.0%)	4	(50.0%)	
Abnormal	6	(50.0%)	4	(50.0%)	
Albumin(3.5-5.0)					0.242
Normal	3	(25.0%)	0	(0.0%)	
Abnormal	9	(75.0%)	8	(100.0%)	
Lactate(3 - 12 mg/dL)					--
Normal	0	(0.0%)	0	(0.0%)	
Abnormal	12	(100.0%)	8	(100.0%)	
Cr.(0.7-1.4)					0.495
Normal	2	(16.7%)	0	(0.0%)	
Abnormal	10	(83.3%)	8	(100.0%)	
Chi-square test, Mann-Whitney U test, Median (IQR). *p<0.05, **p<0.01					

Table 2: (N=20).

Statistical Analyses

The clinicopathological data, as well as the clinical presentation and laboratory data of the survival and non-survival groups were compared. The continuous variables are presented as median (range) and were compared using Mann-Whitney U test. Parameters were compared using Chi-square test as appropriate. *P* values <0.05 were considered statistically significant.

Results

The reviewed cases consisted of eleven males and nine females, who ranged in age from 40 to 87 years old (mean age, 71.5 years). The characteristics of the patients are shown in Table 1. Nineteen patients presented with abdominal pain, while one patient suffered from tarry stool and fever. Ten of the 16 patients had bowel ischemia, 1 patient had ruptured appendicitis, 1 patient had adhesive intestinal obstruction, 1 patient had duodenal ulcer with perforation, 2 patients had enteritis and one patient had cholecystitis. In the survival group, ten patients received surgical intervention and two of twelve received conservative treatment because of very old age (more than 85 years) and clinically were less likely to have bowel ischemia and gangrene. One of the patients received diagnostic peritoneal lavage (DPL) with RBC: 8276/cumm and WBC: 802/cumm. Non-surgical treatment was performed and these two patients were discharged four to six days later. Follow-up CT scan two months later disclosed a reduction in HPVG. Four of the twelve patients underwent surgical interventions to establish the cause of acute abdomen. Another four patients had enteritis and two of them received unnecessary exploratory laparotomy, with a post-operative diagnosis of enteritis. The remaining four patients were diagnosed with bowel ischemia and underwent

resection of small bowel (resection length of 60-160 cm) and were discharged smoothly. In the non-survival group, all of the patients were diagnosed with bowel ischemia; seven of the eight patients presented in the emergency room (ER) with shock. In five of the eight, CT scan findings showed pneumatosis intestinalis with intestinal poor enhancement and mesenteric gas without statistical significance. Two patients did not receive surgical intervention because of poor prognostic condition.

The overall mortality rate was 40%. The mortality rate related to bowel ischemia status post-operation was 60%. For the survival group, we determined the long-term outcomes ranging from 12 months to 120 months (median, 72 months). Among the non-surgical patients, one eventually died of pneumonia 48 months later. For the surgical patients, although they all survived the surgery, one expired 24 months later due to complications of hemodialysis, one 71-year-old patient died of urosepsis after 26 months, one 87-year-old patient died of transitional cell carcinoma with brain metastasis 67 months later, one 80-year-old patient died 4 months later due to stroke, and one suffered from aspiration pneumonia with respiratory failure and was transferred to the respiratory care ward, and eventually died of pneumonia 120 months after discharge. Among the remaining patients, none were found to have HPVG on follow-up CT scans and none were re-admitted for recurrence of acute abdomen.

Discussion

Gas in the portal venous system was first reported in 1955 by Wolfe and Evans who used abdominal plain radiographs to describe necrotizing enterocolitis in neonates [11]. HPVG has subsequently been reported in adults and is usually associated with a poor prognosis [1,11]. The common causes of HPVG are bowel ischemia (61%), which accounts for most HPVG-associated mortality [9], inflammation and obstruction of the gastrointestinal tract (GIT)(25%), sepsis (6%), iatrogenic injury and trauma (3%), and GIT cancer (2%), according to Abdulzahra et al [9]. Some cases can be cured with conservative management. The major cause of HPVG was intestinal mucosa injury combined with overgrowth of aerogenic bacteria in the bowel [1-4]. Theoretically, fulminant disease may produce greater amount of HPV. Sebastian et al. reported that the amount of HPVG was not a risk factor [3], which was consistent with the findings of the present study, which did not reveal any association of HPVG amount with clinical outcomes. HPVG is mainly diagnosed by CT scan, which can help to establish the underlying pathology [1,4,7-9], such as air in the bowel wall (pneumatosis intestinalis), which is defined as the presence of extraluminal bowel gas that is confined within the bowel wall [12]. Surgical intervention is thus indicated when the possibility of ischemic bowel is high [2,9,13-16].

Hiroyuki et. al reported that the risk factors associated with bowel ischemia were low systolic BP (less than 108 mmHg), high LDH (more than 387 U/L), and pneumatosis intestinalis [17], and Umberto G et al. and Haim P et al. reported metabolic acidosis (pH < 7.3, HCO3 < 20) was the key laboratory finding [16,18]. However, in our analysis, no specific laboratory finding showed any associations, but presentation with shock was correlated with mortality. (p<0.005). We speculate that the considerable discrepancy between laboratory findings in the present study and those of previous investigations may be explained by the fact that all of the data in the present were collected from the ER. Nonetheless, our analysis revealed high LDH and profound metabolic acidosis in the Intensive Care Unit (ICU) in patients in the non-survival group postoperatively. One 84-year-old patient presented with fever and tarry stool without peritoneal sign. The patient was deemed to be at low risk of ischemic bowel disease, but HPVG and wall thickening of large intestine were noted on the CT scan. DPL was performed with negative findings. The patient was treated conservatively and discharged following an uneventful course. According to our clinical experience, patients may have weaker mucosal defense of the bowel wall for many reasons, such as old age, ongoing chemotherapy, more medical comorbidities, and probable prolonged inflammation/infection without seeking proper treatment, which may result in high penetration of bacterial invasion and HPVG formation. Thus, we recommend that an experienced surgeon perform Diagnostic Peritoneal Lavage (DPL), paracentesis, and/or repeated physical examination to manage the HPVG in the acute abdomen of low risk patients (Figures 1,2). Diagnostic Peritoneal Lavage (DPL) and paracentesis are invasive, rapid, and highly accurate tests for evaluating intraperitoneal hemorrhage or a ruptured hollow viscus [19-22].

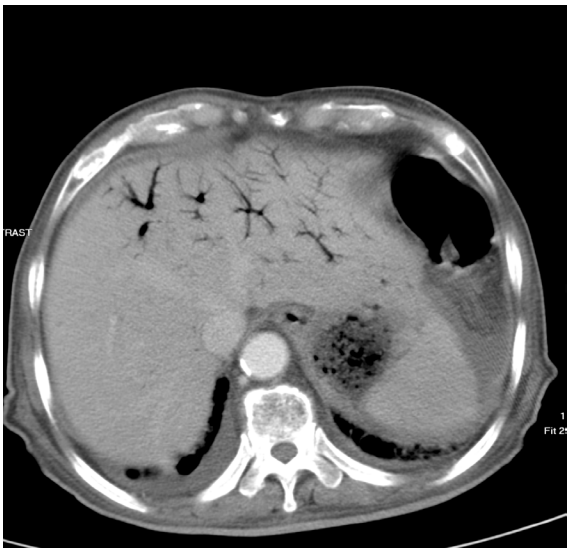


Figure 1a: Indicated pre-operative CT scan.

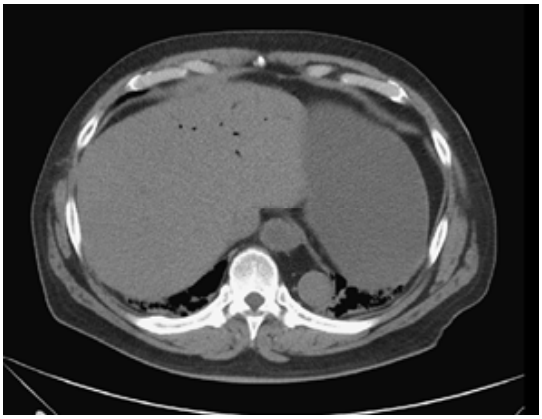
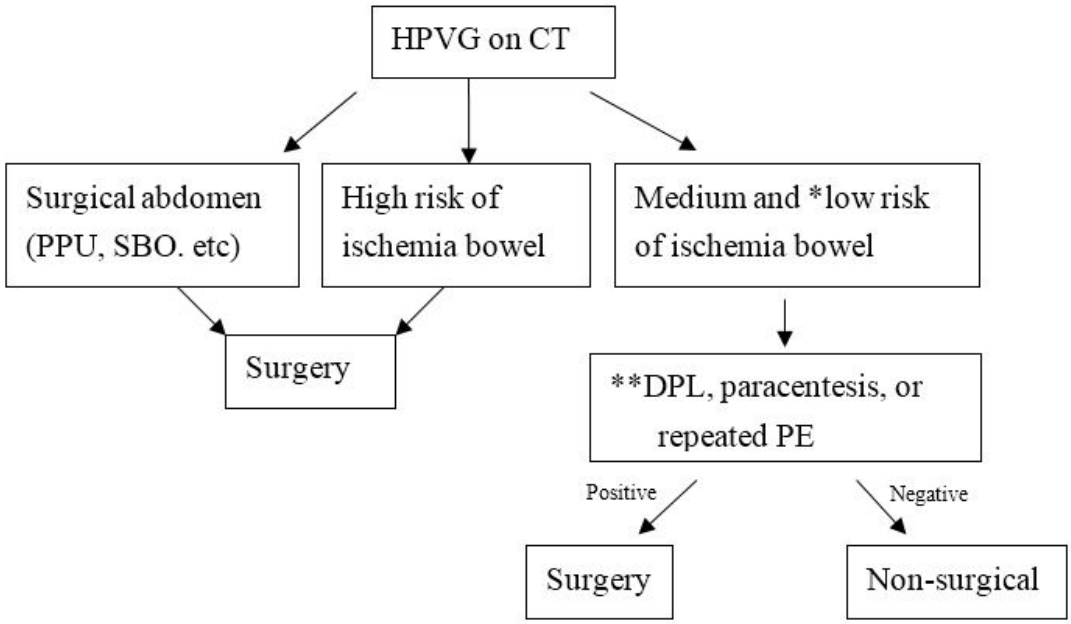


Figure 1b: Indicated post-operative CT scan (1.5 months after operation).



*Low risk: non-specific finding in Laboratory examination, presented with no shock status but with acute abdomen;

**diagnostic peritoneal lavage (DPL), physical examination (PE) by experienced surgeon.

Figure 2: Recommended approach for patients presenting with hepatic portal venous gas detected by computed tomography.

Conclusion

In our analysis, shock was the only prognostic parameter related to mortality ($p<0.005$). However, in some elder patients mild to moderate intra-abdominal infection and/or inflammation was associated with HPVG, which may be due at least in part to age-related weakening of the bowel mucosal defense. Hence, in patients presenting with acute abdomen under relatively stable condition and with HPVG + pneumatosis intestinalis seen on CT imaging, we recommend DPL, paracentesis, and repeated physical examination to better inform the clinical decision for surgical intervention. (Figure 2). A cute mesenteric ischemia should be highly suspected when HPVG and pneumatosis intestinal is are both evident on the CT image. Surgical intervention, such as exploratory or diagnostic laparoscopy, is strongly indicated for these high-risk patients [9,13-17]. (Figure 2).

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