Prediction of bowel necrosis in patients with hepatic portal venous gas detected by computed tomography

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ABSTRACT *Purpose:* The aim of this study is to clarify the factors linked to bowel necrosis in patients with acute abdomen who had hepatic portal venous gas (HPVG) on computed tomography (CT) scans. *Methods:* Over the past 6 years, 22 patients with HPVG detected by CT scan were treated. Medical records from these patients were reviewed to identify signs associated with bowel necrosis. Of the 22 patients with gas in the portal venous system seen on a CT cross-section, nine patients had bowel necrosis. The patients were divided into two groups for comparison: Necrotic bowel group (n=9) and Non-necrotic bowel group (n=13). *Results:* Signs of peritoneal irritation were present in all patients of the bowel necrotic group, but in only one of the bowel non-necrotic group (p=0.00002). Age, sex, and laboratory findings were not associated with bowel necrosis. On CT findings, pneumatosis intestinalis (PI) was significantly associated with bowel necrosis (p=0.00732). *Conclusion:* Signs of peritoneal irritation and PI were strongly associated with bowel necrosis in acute abdomen patients with HPVG.

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Introduction

Wolfe and Evans¹⁾ provided the first radiographic description of hepatic portal venous gas (HPVG). It has since been described in association with a variety of pathologic conditions. Liebmann et al²⁾ stated that the causes of HPVG were (1) mucosal damage, (2) bowel distention, and (3) sepsis (bacterial translocation). According to a later study, two widely accepted theories are used to explain HPVG. One theory states that HPVG results from the direct invasion of bowel gas into the intraluminal venula through an ulcer or necrotic section of intestine^{3,4)}. The other theory states that sepsis is caused by a gas-forming infection⁵⁾. Historically, the most common cause of HPVG

was bowel ischemia, having a mortality rate of 75%²⁾. Since computed tomography (CT) was introduced, the diagnosis rate of HPVG has risen, and as early diagnosis of HPVG became possible, the mortality rate has dropped. Recent studies have dealt with various non-ischemic conditions and even with incidental findings of HPVG, some of which can be cured with conservative management^{6,7)}. Thus, the diagnosis of bowel necrosis in patients with HPVG is critically important in determining the appropriate treatment strategy.

The aim of this study is to clarify the factors linked to bowel necrosis in patients with acute abdomen who had HPVG on CT scans.

Methods

Between January 2003 and December 2008, 54,676 patients underwent helical CT scans. Intravenous contrast medium (Iopamiron 370, Nippon Schering) was used in all cases, except in 5,639 patients who had renal failure or a known hypersensitivity to iodine. Working with a database of medical records, of the 90 patients with HPVG on CT, 62 cases were excluded from this study because their

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Fig. 1. Flow chart of patients with hepatic portal venous gas on computed tomography scan.

HPVG: hepatic portal venous gas, CPA: cardiopulmonary arrest

Fig. 2. Hepatic portal venous gas in bilateral lobe of the liver (white arrow) detected on computed tomography scan.

Fig. 3. Pneumatosis intestinalis (black arrow) and mesenteric venous gas (white arrow) detected on computed tomography scan.

scans were performed to detect the causes of cardiopulmonary arrest (CPA), and 6 additional cases were excluded because the patients died in the emergency department without definite diagnoses. The remaining 22 patients were divided into two groups, necrotic bowel group and non-necrotic bowel group. (Fig. 1 is a flow chart showing the numbers of patients who were included and excluded, and their outcomes).

Two experienced radiologists, blindly reviewed the CT images from each of the 22 patients. The criterion for portal venous gas is a branching radiolucency extending to within 2 cm of the liver capsule (Fig. 2), as seen on a CT scan without contrast medium⁸⁾. CT scans in this study were also examined for the presence of pneumatosis intestinalis (PI)(Fig. 3), ascites, mesenteric venous gas (Fig. 3), the distribution of HPVG (left, right, or bilateral lobes

Table 1. Clinical comparison with or without bowel necrosis.

		Necrotic bowel group (n=9)	Non-necrotic bowel group (n=13)	p-value
Clinical history	Age (> 80)	6 (67%)	7 (54%)	0.67399
	Sex (Male)	3 (33%)	9 (69%)	0.19195
	Peritoneal irritation (+)	9 (100%)	1 (8%)	0.00002
	Abdominal pain as a chief complaint	8 (89%)	9 (69%)	0.36022
Laboratory data	WBC (≥ 9545) * 【3506 ~ 9545】	4 (44%)	7 (54%)	0.99999
	CRP (≥ 2.0 mg/dl) ** 【2.0 ~ 15.0】	4 (44%)	7 (54%)	0.99999
	pH (≤ 7.35) * 【7.35 ~ 7.45】	2/8 (25%)	1/11 (9%)	0.54592
	BE. (≤ -2.3 mEq/l) * 【-2.3 ~ 2.3】	5/8 (63%)	4/11 (36%)	0.36985
	Lactate (≥ 16 mg/dl) * 【4 ~ 16】	5/7 (71%)	5/11 (45%)	0.36652
CT findings	Pneumatosis intestinalis (+)	7 (78%)	2 (15%)	0.00732
	Ascites (+)	5 (56%)	6 (46%)	0.99999
	Mesenteric venous gas (+)	6 (67%)	6 (46%)	0.41486
	Distribution of HPVG (bilateral lobes)	8 (89%)	9 (69%)	0.36022
	Diffuse contrast enhancement of the intestine	4/6 (60%)	4/11(36%)	0.33484

* 【standard value of our hospital】 ** 【value of middle-class inflammation】

of the liver), and for the decline in contrast enhancement of the bowel wall.

Each of the 22 patients' medical records and follow-up imaging studies was reviewed to determine the clinical course. Clinical data (age, sex), symptoms (abdominal pain as a chief complaint), physical examination (signs of peritoneal irritation), laboratory data (WBC, CRP, pH, BE., Lactate), surgical, and pathologic records were also reviewed. In this study, peritoneal irritation was confirmed by at least 2 surgeons at the time of CT, and was considered present if a patient had rebound tenderness upon abdominal exam. The definition of bowel necrosis is of irreversible bowel ischemia as surgical findings, and pathologically, the necrosis having extended into the submucosa.

Statistics

We analyzed the associations between categorical clinico-pathologic variables and bowel necrosis using Chi-square tests. P-values were calculated by Fisher's exact test to determine which variables were significant predictors of bowel necrosis.

Results

Twenty-two patients were included in this study (12 male and 10 female). Mean age was 77years old with the range from 53 to 94.

Of 22 patients, nine had bowel necrosis (necrotic bowel group) and thirteen did not have bowel necrosis (non-necrotic bowel group). Eight patients in the bowel necrotic group underwent resection of gangrenous bowel. All of them survived to be discharged. The other patient died of duodenal perforation due to necrosis, which was revealed by autopsy.

Of the thirteen patients in the non-necrotic bowel group, five had open looped bowel obstruction. Their condition was improved by an ileus tube or stomach tube. The cause of their HPVG was conjectured to be bowel distention and fugitive mucosal damage. Two patients had an intraperitoneal abscess. Bacterial translocation was considered to be the likely cause of HPVG, because blood cultures were positive. Another patient who recovered without surgical management had been hospitalized for pulmonary suppuration, with HPVG being detected incidentally on CT. Blood cultures were not performed on this patient, but the most likely cause was bacterial translocation, judging from the circumstances. In another patient hospitalized for hypoglycemic coma, paralytic ileus, and pneumonia, *Clostridium perfringens* was detected in the blood culture. The cause of his HPVG was thought to be bowel distention and bacterial translocation. The other three patients were hospitalized for ischemic enteritis. They recovered from intravenous fluids, and the cause of their HPVG was thought to be fugitive mucosal damage. One patient underwent a laparotomy, but no bowel necrosis was observed. Though his presentation and abdominal symptoms were

unclear due to active schizophrenia, PI and signs of peritoneal irritation were considered positive. Laparotomy revealed only paralytic ileus. Interestingly, 6 months after this procedure, he had a partial enterectomy due to necrosis of the small intestine without a closed-loop bowel obstruction. At this time, his intestine had pathological ischemic change. Consequently, the cause of HPVG in initial presentation was thought to be reversible bowel ischemia such as non-occlusive mesenteric ischemia (NOMI).

Predictors of Bowel Necrosis

Table 1 summarizes several variables and their distribution among patients with and without bowel necrosis. Age, sex and the presence of abdominal pain did not correlate with bowel necrosis. Of the nine patients in the bowel necrotic group, all of them presented with signs of peritoneal irritation. However, only one patient in the bowel non-necrotic group presented with these signs ($p=0.00002$).

Among the laboratory data, there were no significant predictive factors of bowel necrosis.

PI was present in seven of the nine patients (78%) in the bowel necrotic group, but only two patients (15%) in the bowel non-necrotic group had PI ($p=0.00732$). The presence of ascites, mesenteric venous gas, decline of contrast enhancement in the bowel wall, and distribution of HPVG did not correlate significantly with bowel necrosis.

Discussion

HPVG in adults is a rare clinical occurrence and most published studies of it have been case presentations. Peloponissios et al⁹, stated that HPVG may be found in a variety of pathological conditions, including intestinal ischemia and necrosis (75%), ulcerative colitis (8%), and intra-abdominal abscess (6%)^{2,10}. Patients with HPVG have a survival rate of less than 25% owing to the severity of the underlying conditions². Thus, the diagnosis of bowel necrosis in the patients with HPVG is critically important in choosing a treatment strategy.

Several studies have demonstrated that patients with PI and portomesenteric venous gas are likely to have bowel necrosis that requires emergency surgery. Lai et al¹¹, emphasized that the combination of portmesenteric venous gas and PI is strongly associated with transmural bowel infarction, especially band-like pneumatosis. Wiesner et al¹², found that 10 of 11 patients (91%) with portomesenteric

venous gas and PI had transmural bowel infarction. Kernagis et al¹³, also found that all patients with both findings had bowel necrosis. Peloponissios et al⁹, also supported this correlation after analysis of eleven patients with HPVG. Nevertheless, their series included four patients with suspected bowel ischemia who died without surgery or autopsy to confirm the causes of HPVG. Our study of patients with HPVG also supported the link between bowel necrosis and PI. We found that seven of nine patients (78%) in the bowel necrotic group had PI on CT, although only two (15%) in the bowel non-necrotic group had PI ($p=0.00732$). Therefore PI appears to be a reliable predictor for bowel necrosis in patients with HPVG.

Other CT findings, such as ascites, mesenteric venous gas, the decline of contrast enhancement in the bowel wall, and the distribution of HPVG were not significant predictors for bowel necrosis. The extent of gas in the portomesenteric vein (i.e., mesenteric venous gas and the distribution of gas in the hepatic lobes) was not a significant predictor for bowel necrosis. In a previous study, standard abdominal radiographs were able to show large quantities of HPVG and this finding was associated with a poor prognosis and bowel infarction^{9,14}. Further research is necessary to determine the predictive value of quantitative assessment of HPVG on CT scans.

In our study, the presence of abdominal pain as a chief complaint did not always indicate the bowel necrosis. Iannitti et al¹⁵, mentioned that abdominal symptoms were related to mortality. Even though the severity of abdominal symptoms may be difficult to assess and the evaluation of peritoneal irritation lacks objectivity, signs of peritoneal irritation were the most important predictor for bowel necrosis in our study. Based on our search of literature about HPVG, there were no reports describing the signs of peritoneal irritation. It is obvious that peritoneal irritation is important not only for bowel necrosis but for other diseases causing peritonitis. However, when peritoneal irritation and the presence of PI on CT scan are confirmed in addition to HPVG, and no obvious cause are apparent, emergency operation should be considered to evaluate bowel necrosis.

This is a retrospective study, so there are some biases which caused by the lack of objectivity. We should clarify more objective factors linked to bowel necrosis in patients who have HPVG on CT in the future study.

When necrotic intestine does not have pathologically distinctive findings such as thrombi, NOMI must be taken into consideration. NOMI is an acute mesenteric circulation disorder that is not caused by organic occlusion of

blood vessels. Early signs and symptoms are unclear, and the disease often advances to an irreversible stage before the diagnosis is made.

Conservative treatment of HPVG is acceptable on condition that i) vital signs are stable and there are no signs of peritoneal irritation. ii) primary disease can be speculated. iii) emergency surgery can be performed if necessary. Frequent evaluations of vital signs, laboratory data, radiographic data and physical findings must be performed with the treatment of the primary disease.

In our study, multivariable logistic-regression analysis was tried to identify the predictive factors for bowel necrosis in patients with HPVG more exactly. Because of the lack of sample size, we could not demonstrate the significant differences. The CPA patients and the patients who died without definite diagnoses were excluded. The relation between HPVG and bowel necrosis will be clearer by identifying the cause of death in these patients.

Conclusion

Clinical signs of peritoneal irritation and PI on CT scan might indicate the characteristics of the bowel necrosis in acute abdomen patients with HPVG. Emergency operation should be considered when these signs are present in patients with HPVG.

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原著論文

門脈ガス血症における腸管壊死の予測因子

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要旨 目的:この研究の目的は、CTで門脈内にガスを認めた急性腹症の患者における腸管壊死の予測因子を明らかにすることである。**方法:**過去6年間にCTで門脈ガスを認めた22名の患者が対象である。これらの患者の診療記録から、腸管壊死と関連のある因子を明らかにした。腹部断層CTにおいて肝内門脈系にガスを認めた22名のうち9名に腸管壊死があった。比較検討は腸管壊死グループ(n=9)と非腸管壊死群(n=13)に分けて行った。**結果:**腹膜刺激症状は腸管壊死群ではすべてに認められたが、非腸管壊死群では1例にしか認めなかった(n=0.00002)。年齢や性別、検査データは腸管壊死との関連はなかった。CT所見では、腸管気腫が腸管壊死と有意な相関があった(n=0.00732)。**結論:**門脈ガス血症がある急性腹症の患者において、腹膜刺激症状と腸管気腫が腸管壊死と有意な関連が認められた。

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