愛知医科大学医学会雑誌 (J. Aichi Med. Univ. Assoc.)
Vol. 43, No. 2, pp. 35–40 (June 15, 2015)

症　　例

Exploratory Laparotomy by Laparoscopy Proved to be Effective for the Emergency Treatment of a Case of Simultaneous Inferior Vena Cava and Hepatic Portal Vein Gas Related to Ischemic Colitis

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A 64-year-old male presented with acute abdominal pain during in-hospital treatment for chronic heart failure due to dilated cardiomyopathy. A computed tomography (CT) scan demonstrated simultaneous inferior vena cava and hepatic portal vein gas, ascites, and intramural emphysema of small intestine. Physical examination showed only mild tenderness without peritoneal signs. We conducted an emergent exploratory laparotomy by laparoscopy, which is considered less invasive than open surgery. Intestinal necrosis was not detected, although serous ascites and intramural emphysema were identified. Abdominal pain was relieved by fasting and administration of antibiotics. Abdominal CT scan 3 days after the laparoscopy showed disappearance of inferior vena cava and portal vein gas, and wall thickening of the ileum and ascending colon. A colonoscopy 6 days after laparoscopy revealed mucosal erosion and ulceration of terminal ileum without necrosis. Histological diagnosis of this ulceration was ischemic colitis. His general status improved gradually and the diet was resumed on the 3rd postoperative day. Here we report a case of simultaneous inferior vena cava and hepatic portal vein gas related to ischemic colitis.

Key words: hepatic portal vein gas, inferior vena cava gas, ischemic colitis, laparoscopy

INTRODUCTION

Hepatic portal vein gas (HPVG), commonly detected by computed tomography (CT), is an ominous sign that has resulted in approximately 29% mortality in spite of various kinds of treatment[1]. Also, because patients with a history of vascular surgery or bowel obstruction may have a higher mortality rate, careful clinical evaluation is mandatory for that population. We experienced a case of ischemic colitis with inferior vena cava gas (IVCG), which may be a more serious condition than HPVG. This case was treated conservatively with success, and may indicated the value of urgent laparoscopy prior to definitive open surgery, especially in patients with serious comorbidities.

CASE REPORT

A 64-year-old male under medical treatment

Received March 12, 2015; Accepted May 1, 2015

Table 1. Laboratory data when patient complained of abdominal pain

WBC (×10/μg)	4600	Amylase (IU/l)	57
RBC (×10/μg)	436	Na (mEq/l)	138
Hemoglobin (g/dl)	11.9	K (mEq/l)	4.4
Hematocrit (%)	36.6	Cl (mEq/l)	99
MCV (fl)	83.8	BUN (mg/dl)	21.7
Platelet (×10)	14.5	Creatinine (mg/dl)	1.24
Total protein (g/dl)	6.8	CRP (mg/dl)	0.1
Albumin (g/dl)	3.8	ABG (room air)	
Total-bilirubin (mg/dl)	1.36	pH	7.415
Direct-bilirubin (mg/dl)	0.58	pCO_2 (mmHg)	42.6
AST (IU/l)	13	pO_2 (mmHg)	50.9
ALT (IU/l)	9	HCO_3 (mmol/L)	26.7
LDH (IU/l)	264	Base excess (mmol/L)	1.9
CPK (IU/l)	96	Lactate (mg/dl)	10.9

WBC: white blood cells; RBC: red blood cells;
MCV: mean corpuscular volume; AST: aspartate aminotransferase;
ALT: alanine aminotransferase; LDH lactate dehydrogenase;
CPK: creatine phosphokinase; BUN: Blood urea nitrogen;
CRP: C-reactive protein

for chronic heart failure due to dilated cardiomyopathy had a chief complaint of acute abdominal pain. His medical history included atrial fibrillation, chronic renal failure, and diabetes mellitus. There was no history of abdominal surgery. Hemodialysis was not indicated. Physical examination showed normothermia (36.8℃), mild hypotension (84/70 mmHg), and mild tachycardia (Heart rate 101/min). Although mild tenderness in the lower abdomen was shown, no signs as symptoms of peritonitis were observed. Hematological examination showed a normal hemoglobin value and no leukocytosis (Table 1). Biochemical examination showed mild elevation of blood creatinine and blood urea nitrogen. The liver enzymes were not elevated. Arterial blood gas analysis revealed moderate hypoxemia without pH change. Abdominal CT showed ascites, left renal vein gas, IVCG, HPVG in the bilateral lobes, and intramural emphysema in the small intestine. Neither splenomegaly nor liver deformity suggesting cirrhosis were observed (Figure 1, 2). In addition, splenorenal venous shunts were detected. Blood flow of mesenteric and celiac artery was well maintained. To make a definitive diagnosis and evaluate bowel necrosis, an emergency exploratory laparoscopy, which is considered minimally invasive, was performed under general anesthesia. Serous ascites and intramural gas of small intestine were observed, but intestinal necrosis was not obvious by intra-abdominal surveillance using laparoscopy. The liver was of a normal appearance without cirrhosis. Bacterial culture of ascites was negative. An information drainage tube was placed in the abdominal cavity, and removed 24 hours after laparoscopy without alteration of drainage fluid. Conservative treatment with fasting and 4 day administration of antibiotics were indicated, after which abdominal pain subsided.

Abdominal CT reviewed 3 days after laparoscopy revealed disappearance of the IVCG and HPVG. Bowel wall thickness from the ileum to the ascending colon was detected (Figure 3). Colonoscopy 6 days after laparoscopy revealed the presence of mucosal erosion and ulceration

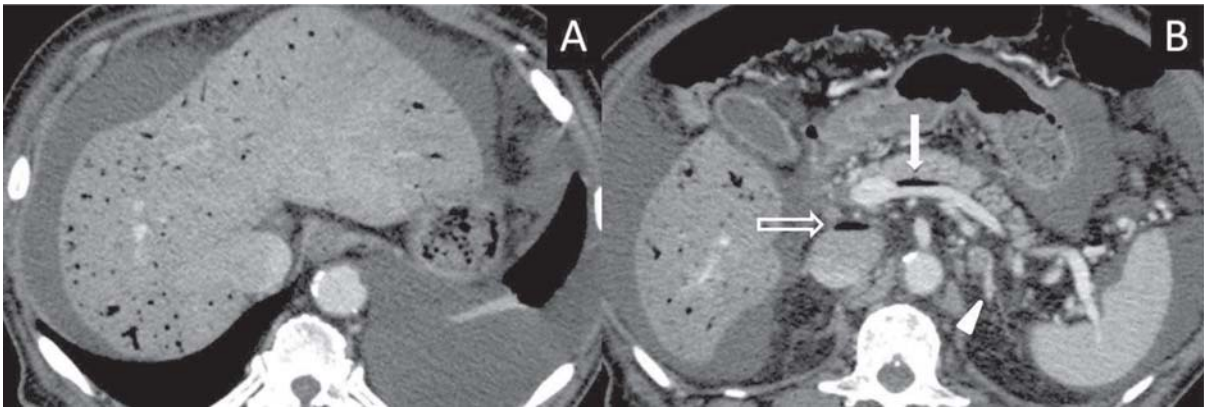

Figure 1. Abdominal CT scan showed ascites, hepatic portal vein gas (A), inferior vena cava gas (open arrow) and splenic vein gas (white arrow) and detected splenorenal shunts (white arrowhead) (B).

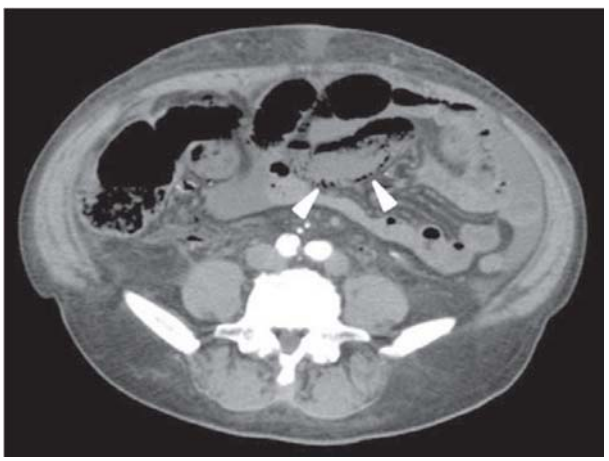

Figure 2. Abdominal CT scan on admission showed intramural gas in the small intestine (white arrowhead).

of the ileum 5 cm proximal from the Bauhin's valve, but obvious findings of bowel necrosis were absent (Figure 4). Histological findings from the colonoscopic biopsy were consistent with ischemic colitis.

The patient's general condition gradually improved, and he was discharged 22 postoperative days after treatment of chronic heart failure. Postoperatively, the patient has not suffered from abdominal symptoms for 13 months.

DISCUSSION

In our case, ischemic colitis of the terminal

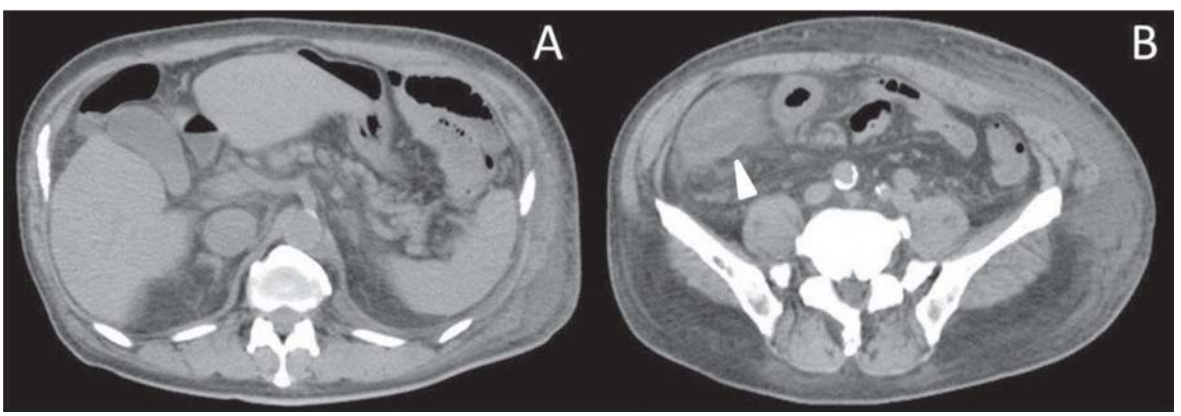

Figure 3. Abdominal CT scan 3 days after operation resalted in disappearance of inferior vena cava gas and hepatic portal vein gas (A), and thickness of the wall at the ileocecal bowel was revealed (white arrow) (B).

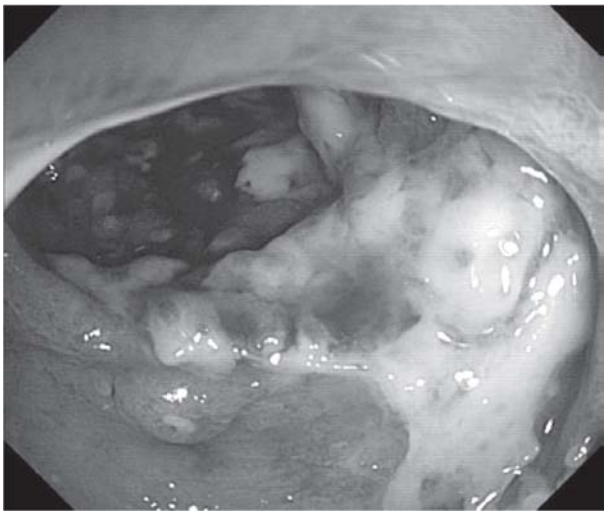

Figure 4. Results of colonoscopy showed ulceration from the ileum 5 cm proximal to Bauhin's valve to ascending colon, with definitive diagnosis ischemic colitis.

ileum to the ascending colon caused HPVG. To evaluate bowel conditions, we performed exploratory laparotomy by laparoscopy. Several cases of conservatively treated HPVG have been reported, but we should still regard HPVG as a critical condition with a poor prognosis, and pay special attention to the presence of bowel necrosis. Our patient was treated for chronic heart failure, and potentially had a restricted physical functional capacity. Thus, delay in making a diagnosis as to the cause of HPVG might lead to a poor prognosis even in a condition without peritoneal signs, as in this case. Accordingly, we conducted an emergent diagnostic laparoscopy, which is useful for making diagnoses and is less invasive than definitive open surgery[2)3)].

Also, we reviewed the relevant literature describing IVCG and HPVG in terms of their relation to ischemic colitis. To characterize this rare situation, we searched the PUBMED databases using the keywords 'inferior vena cava gas,' and 'hepatic portal vein gas.' As of May 2014, there were 7 cases describing IVCG coexist with HPVG related to digestive disease (Table 2)[4~8)]. Kamikado et al.[5)] reported two potential routes of HPVG to the systemic veins: one is a drainage for hepatic vein, and the other is a portosystemic shunt. Kato et al.[9)] reported a potential mechanism of shunt blood flow from the portal vein to systemic veins through the liver parenchyma or extrahepatic collaterals in response to increased portal pressure. The Japan Society for Portal Hypertenson suggested congestive heart failure as a cause of portal hypertension[10)]. Although the cause of the splenorenal shunt in this case is unknown, it is considered that portal hypertension may be caused by high pressure of the right atrium with dilated cardiomyopathy. The important point is that IVCG was not necessarily related to the poor prognosis in contrast to the impressive CT findings in this case.

In 1955, Wolfe and Evans, in their first case series, described portomesenteric venous gas in six infants died who of intra-abdominal catastrophes[11)]. In 1960, Susman reported the first critically ill adult patient with small bowel infarction[12)]. Recent increases in the use of abdominal CT have led to increased detection of HPVG associated with various non-ischemic conditions[13)]. Liebmann et al.[14)] stated three potential causes of HPVG; mucosal damage, bowel distention, and sepsis (bacterial translocation). Kinoshita et al.[15)] reviewed 182 cases of HPVG, and found that the most common cause was bowel necrosis (43%), followed by digestive tract dilatation (12%), intraperitoneal abscess (11%), ulcerative colitis (4%), Crohn's disease (4%), complications of endoscopic procedures (4%), intraperitoneal tumor (3%), and others (15%). The mechanism of HPVG in terms of ischemic colitis may be considered as direct inflow of bowel gas into the intraluminal venula through an ulcer of the intestine[16)17)].

Table 2. Cases of systemic vein gas coexist with intrahepatic portal vein gas

Author	Age	Sex	Portal vein gas	Site of gas	Cause
Mallens	31	F	IHPV, left colic vein, IMV, SMV, hemorrhoidal veins	internal iliac vein, IVC	iatrogenic mucosal disruption
Kriegshauser	54	M	IHPV	hepatic vein, IVC	not available
	68	M	IHPV	prostatic venous plexus, common femoral veins	necrosis of small intestinal tumor
Wiot	63	M	IHPV, portal vein, mesenteric vein	hepatic veins, jugular veins, right atrium, right ventricle vessels at base of brain	bowel necrosis
	52	F	IHPV, mesenteric vein	jugular vein	bowel necrosis
Kamikado	92	F	IHPV, portal vein, splenic vein, SMV	hepatic vein, IVC, right atrium, right ventricle	alteration of colon mucosa
Zhang	50	M	SMV, splenic vein, portal vein, IMV, mesenteric vein	IVC	necrotizing enterocolitis
Present case	64	M	portal vein, SMV	IVC, left rental vein	ischemic colitis

Author	Age	Sex	Operation	Outcome	Route to systemic vein
Mallens	31	F	not operated	survived	via portosystemic shunt
Kriegshauser	54	M	not operated	dead	via liver enterovenous fistula caused by the tumor or polysurgery
	68	M	resection	survived	
Wiot	63	M	not operated	dead	not available
	52	F	not operated	dead	not available
Kamikado	92	F	not operated	survived	via liver
Zhang	50	M	not operated	dead	not available
Present case	64	M	exploratory laparotomy by laparoscopy	survived	via splenorenal shunt

IHPV: intrahepatic portal vein; IMV: inferior mesenteric vein; SMV: superior mesenteric vein; IVC: inferior vena cava

The existence of HPVG has not always indicated an exploratory laparotomy. However, it is necessary to make a precise diagnosis of the cause of HPVG. Emergency laparoscopy has a significant role in prevention due to the delay in obtaining a definitive diagnosis especially in patients with poor functional reserve due to several comorbidities.

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