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Hepatic portal venous gas associated with ischemic colitis: a case report

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Abstract

Background Cases of Hepatic portal venous gas (HPVG) have been associated with high mortality rates and frequently require emergency exploratory laparotomy. However, the widespread utilization of computed tomography (CT) scans has revealed that HPVG is often connected to benign conditions, as demonstrated by numerous studies. Given the intricate nature of the underlying causes of HPVG, there remains a lack of consensus regarding the necessity of emergency surgical exploration for patients with HPVG.

Case Report An octogenarian female patient was admitted to the emergency department due to abdominal pain, accompanied by symptoms of nausea and vomiting. A CT scan of the abdomen and pelvis revealed a significant presence of radiolucency in the peripheral branching of the liver, indicating the existence of portal venous gas. Subsequently, the patient was transferred to the Emergency Intensive Care Unit for further management. Colonoscopy of the patient reveals features consistent with ischemic colitis, characterized by mucosal congestion, edema, erosion, ulcers, with some ulcers covered by pseudomembranes. After undergoing a series of conservative treatments, the patient's condition improved, as confirmed by a follow-up CT scan of the abdomen and pelvis conducted 8 days later, which showed complete absorption of the gas. Consequently, the patient was discharged from the hospital.

Conclusions The management of HPVG should take into account the pathophysiology and clinical manifestation, and should be tailored towards addressing the root cause. The selection of surgical or conservative intervention should be guided by the underlying etiology, while the prognosis and outcome of HPVG are contingent upon the underlying cause.

Keywords Hepatic portal venous gas, Colon ulcers, Case reports

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Background

Hepatic portal venous gas (HPVG) refers to the presence of gas in the portal vein, as well as its intrahepatic branches and branches of the gastrointestinal tract [1]. The first report of HPVG in infants with necrotizing enterocolitis was made by Wolfe and Evans in 1955 [2]. HPVG is a rare clinical finding that is associated with a high mortality rate and requires prompt clinical attention. In cases of perspective, it is suggested that gas-producing bacteria within the portal vein system generate gas during infections, such as bacterial enteritis [3]. It often coexists with gas within the intrahepatic biliary channels, which is typically located in the central liver parenchyma [4].

Previous studies have reported a high fatality rate of HPVG high to 75% in 1978, with ischemic bowel disease being the main underlying condition. However, with the widespread use of abdominal CT, HPVG has been found to be associated with clinically benign illness, leading to a decrease in the overall fatality rate to 39% [5]. Subsequent investigations have established a strong association between HPVG cases and elevated mortality rates, frequently necessitating emergency exploratory laparotomy. Furthermore, the increased utilization of CT scans has revealed that HPVG is frequently observed in benign conditions, such as ulcerative colitis [6], Crohn's disease [7], enteritis [8, 9], cholangitis [10], gastric ulcer [5], following endoscopic procedure [8, 11]. Additionally,, it has been observed that HPVG may appear in healthy young patients without any clear and definite pathogenesis [1]. Given the complex nature of the underlying causes of HPVG, there is still a lack of consensus regarding the necessity of emergency surgical exploration for patients with HPVG [12]. The presence of HPVG in conjunction with shock, abdominal effusion, and peritonitis frequently indicates the presence of internal intestinal ischemia-necrosis within the abdominal cavity. This condition is associated with a poor prognosis and high mortality rates, highlighting the necessity of considering proactive surgical interventions.

Table 1 Blood laboratory reports and normal reference values

	reports	normal ranges
pH	7.546	7.350–7.450
PCO2	13.6mmHg	35-45mmHg
HCO3-	18.4 mmol/L	22-27mmol/L
BE	−13mmol/L	±3mmol/L
WBC	8.8×10^9/L	(3.5–9.5)× 109/L
Neutrophil ratio	86.20%	(50–75) %
lymphocyte ratio	7.50%	(20–40)%
Hemoglobin	134 g/L	(120–160)g/L
blood platelet count	177×10^9/L	(100–300)%
NT-pro BNP	773.20 pg/mL	<125pg/mL
hsTnI	0.02 ng/mL	<0.01262ng/mL
albumin	32.50 g/L	(40–55)g/L
glutamic oxalacetic transaminase	69.65 U/L	(15–40)U/L
serum creatinine	148.70umol/L	(57–97)umol/L
interleukin-6	600.36 pg/mL	<7umol/L
procalcitonin	0.793 ng/L	<0.065ng/L
fibrinogen degrdtion products	13.26ug/L	<5ug/L
D-Dimer	2.93ug/L	<0.05ug/L

Case presentation

An 81-year-old woman was admitted to emergency department due to unexplained abdominal pain, discomfort, nausea and vomiting that had persisted for 6 h. The pain was described as persistent dull pain, primarily located in the upper and lower left abdomen. Prior to the onset of symptoms, the patient had been in good health without any known chronic diseases. Her medical history included hypertension and cholecystectomy. The patient had not been taking any specific medications. Upon admission, the patient's vital signs were as follows: temperature of 36.6 degrees Celsius, heart rate of 60 beats per minute, respiratory rate of 25 breaths per minute, and blood pressure of 161/87 mmHg. Physical examination revealed tenderness and softness in the abdomen, with no rebound tenderness. Blood gas analysis showed that following results: pH:7.546, PCO2 of 13.6mmHg, HCO3 of 18.4 mmol/L, BE of −13 mmol/L. Routine blood test displayed the following findings:: WBC of 8.8×10^9/L, Neutrophil ratio of 86.2%, lymphocyte ratio of 7.50%, Hemoglobin of 134 g/L, blood platelet count of 177×10^9/L. N-terminal probrain natriuretic peptide(NT-pro BNP) was measured at 773.20 pg/mL, High Sensitivity Cardiac Troponin-T(hsTnI) at 0.02 ng/mL, albumin at 32.50 g/L, glutamic oxalacetic transaminase at 69.65 U/L, serum creatinine at 148.70umol/L, interleukin-6 at 600.36 pg/mL, procalcitonin at 0.793 ng/L, fibrinogen degrdtion products at 13.26ug/L, D-Dimer at 2.93ug/L. Routine urine test revealed the presence of white blood cell(3+), ketone body(1+). While stool for routine indicated a occult blood test positive for occult blood. See Table 1 for complete details of Blood laboratory reports and normal reference values.

We further conducted routine CT examination on the patient. A computed tomography (CT) scan of the abdomen and pelvis showed a significant presence of radiolucency in the peripheral branching of the liver, indicating the presence of portal venous gas (Fig. 1a). As a result, the patient was hospitalized to the Emergency intensive Care Unit and a nasogastric tube was intubated for stomach decompress. Due to the high mortality associated with hepatic portal venous gas (HPVG), we consulted surgeon who recommended an emergency laparoscopic exploratory procedure. However, considering the patient's age and the potential surgical risks, the patient's relatives of patient declined for surgery.

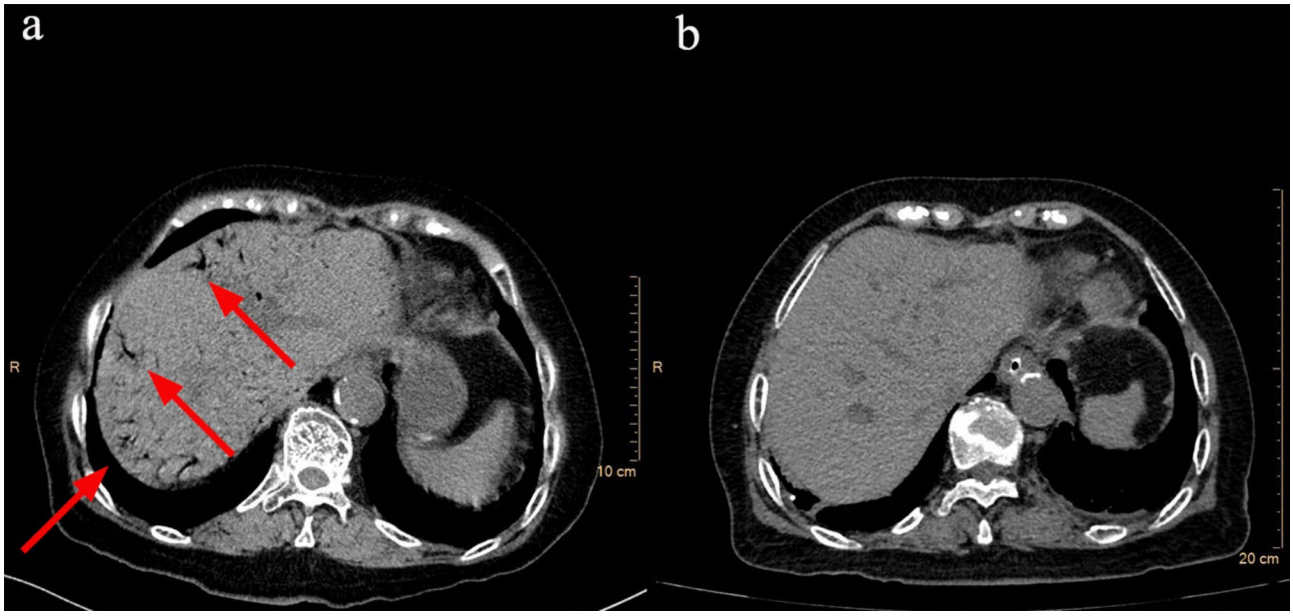

Fig. 1 CT scans of hepatic portal venous gas. **a** showed the hepatic portal venous gas and the pneumatosis on the first day of the patient's hospital stay, Abdominal CT scan revealed multiple branching gas shadows within the liver of the patient, as indicated by the red arrows. **b** showed the same view of the patient's abdomen CT after 8 days later, which showed complete absorption of the gas.

On account of stool for routine showed occult blood test positive, and black tarry stool after admission to hospital, we made electronic gastroscope and electronic colonoscope. Gastroscopy revealed reflux esophagitis (LA-B), chronic atrophic gastritis C1 (Fig. 2). Gastroscopy showed a relatively rough gastric mucosa with a coarse granular or nodular appearance (Fig. 2). Colonoscopy found multiple mucosal congestion, edema, erosion, ulcers, with some ulcers covered by pseudomembranes in the ascending colon (Fig. 3). Based on the combination of colonoscopy findings, the patient's symptoms of abdominal pain, and rectal bleeding, we are considering a final diagnosis of ischemic colitis. Following, she was given cefoperazone sulbactam sodium 3.0 g IVGTT Q12H anti-infection, omeprazole sodium 40 mg IVGTT QD protect gastric mucosa, gastrointestinal decompression, rehydration, Glutamine Capsules 0.5 g PO TID, combined Bacillus Subtilis and Enterococcus Faecium Granules with Multivitamines PO TID. After undergoing a series of conservative treatments, the patient's condition improved, as confirmed by a follow-up CT scan of the abdomen and pelvis conducted 8 days later, which showed complete absorption of the gas (Fig. 1b). The patient also didn't have any symptoms of abdominal pain. Then the patient was transferred out of the emergency intensive care unit for further treatment. After receiving nutritional support, the patient was discharged with a better health condition.

Discussion

HPVG represents a critical clinical emergency for surgeons, often indicating serious infection and potentially leading to septic shock within a very short time [13]. The exact mechanisms underlying HPVG formation are still not fully understood, but there are three possible mechanisms. One theory suggests that gas escapes into the submucosa and blood vessels through the damaged intestinal mucosa, subsequently entering the portal vein system via blood flow, as seen in cases of mesenteric ischemia [14] and bowel obstruction [15]. In cases of perspective, it has been observed that gas-producing bacteria within the portal vein system generate gas during the course of infections, such as bacterial enteritis [8]. Furthermore, there are cases that exhibit a combination of these mechanisms, such as HPVG following colonic endoscopic submucosal dissection (ESD) with positive blood culture for Escherichia coli [16]. However, there have been instances, such as in the case of Idiopathic Hepatic Portal Venous Gas in a Healthy Young Man [1], where these explanations fail to provide a satisfactory understanding.

In this particular case, although the patient exhibited indications of inflammation based on white blood cell count, interleukin-6, and procalcitonin levels, there were no accompanying symptoms of chills and fever, which led us to forego conducting a blood culture. Furthermore, colonoscopy revealed the presence of multiple erosive ulcers and bleeding in the ascending colon, supporting the hypothesis that gas escaped into the blood vessels by the damaged intestinal mucosa.

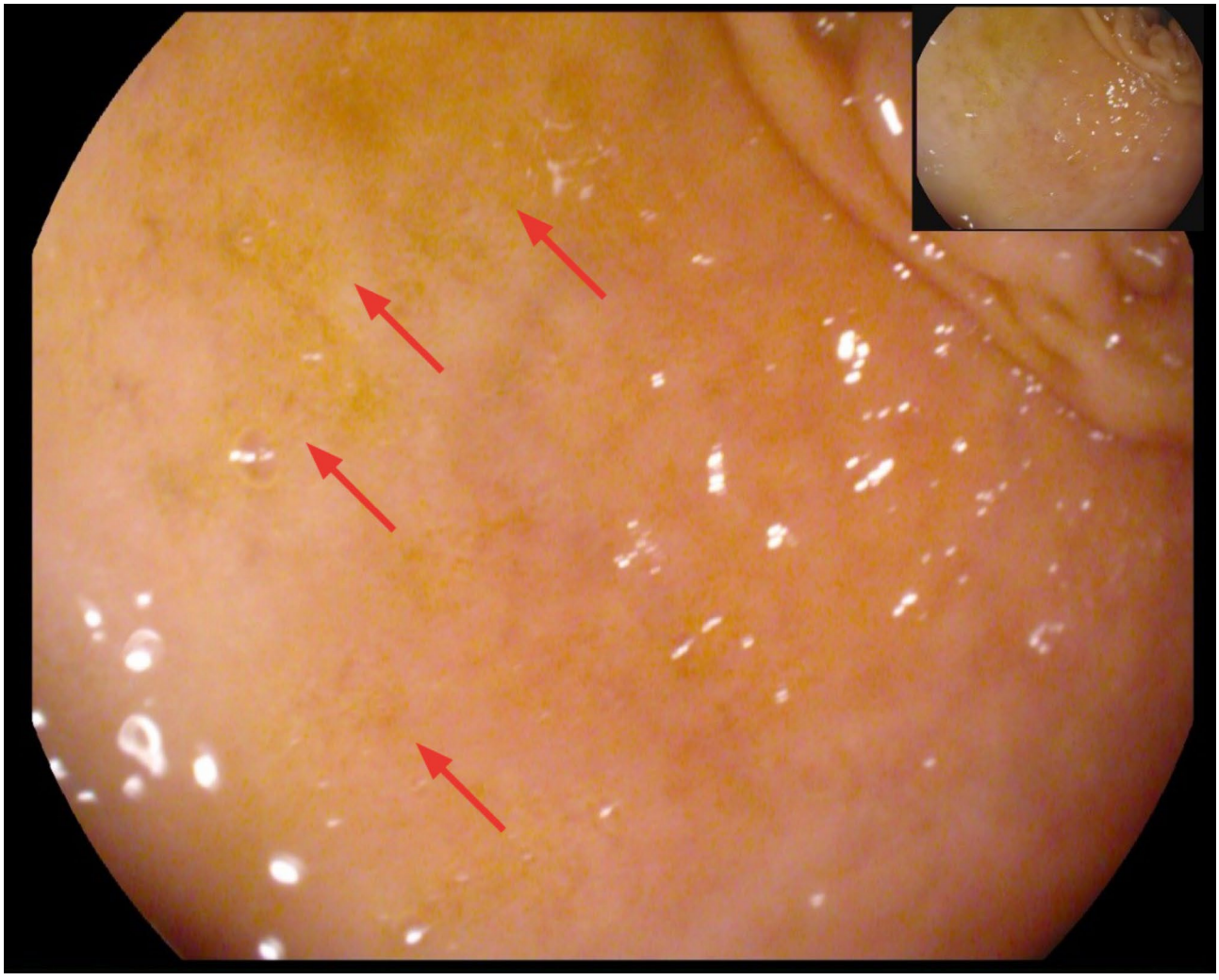

Fig. 2 Gastroscopy revealed chronic atrophic gastritis C1. Gastroscopy showed a relatively rough gastric mucosa with a coarse granular or nodular appearance, as indicated by the red arrows

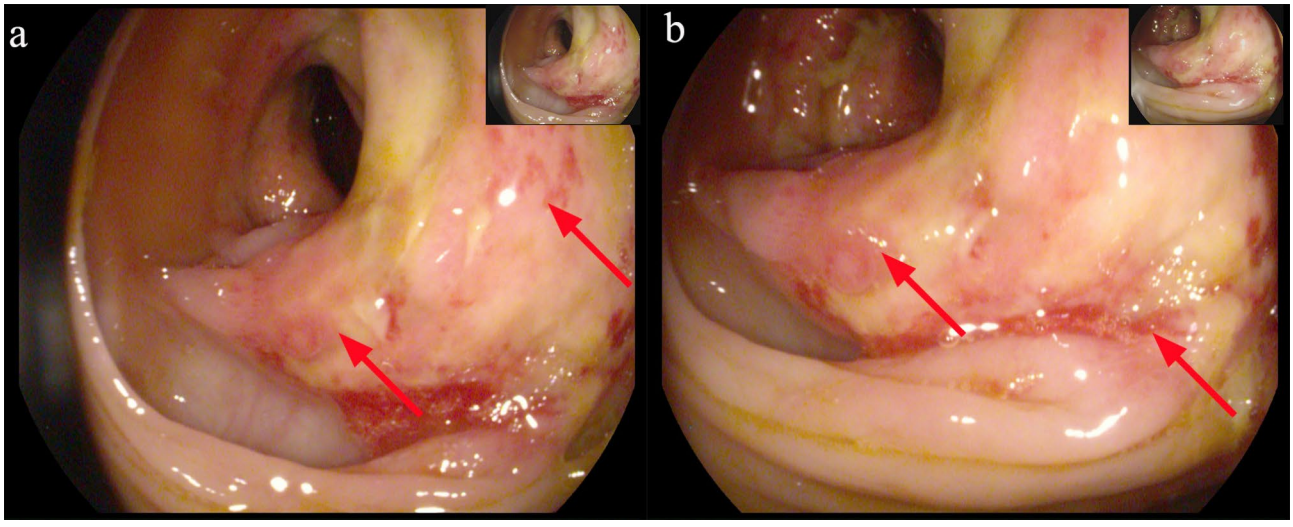

Fig. 3 Colonoscopy diagnosed ischemic colitis. Colonoscopy found multiple annual mucosal congestion(a), edema(a), erosion(a), ulcers(b), with some ulcers covered by pseudomembranes(b) in the ascending colon

In this particular case, the colonoscopy of the patient reveals features consistent with ischemic colitis, characterized by mucosal congestion, edema, erosion, ulcers, with some ulcers covered by pseudomembranes, and disappearance of the submucosal vascular network. The lesions demonstrate a segmental distribution. Therefore, taking into consideration the patient's presenting symptoms, clinical signs, and colonoscopic findings, we arrived at the final diagnosis of ischemic colitis. Treatment was initiated based on this diagnosis, resulting in a rapid improvement of the patient's condition.The predominant clinical presentations of ischemic colitis include paroxysmal abdominal pain, hematochezia, and pyrexia. The quantity of fecal blood is typically modest; however, the frequency is heightened, with grave instances precipitating intestinal wall necrosis, perforation, culminating in peritonitis and septic shock. Endoscopic examination reveals intestinal manifestations of ischemic bowel disease characterized by edema, erosion, vascular network obliteration, and a dark red or purplish-red mucosal appearance. Abdominal computed tomography (CT) imaging demonstrates mucosal edema and thickening at the affected site, occasionally accompanied by contrast medium peri-inflammatory extravasation. Surgical intervention should be contemplated in cases of ischemic colitis that are complicated by peritoneal irritation, intractable hemorrhage, abdominal effusion, and refractoriness to conservative pharmacotherapy.

Historically, the presence of HPVG was commonly found in patients with bowel necrosis has been associated with a poor outcome [13], and particularly in cases of intestinal ischemia and necrosis [17]. Consequently, surgeons have traditionally advocated surgical intervention upon detecting hepatic portal venous gas (HPVG) on imaging in the presence of abdominal effusion, peritonitis, and ileus. However, recent research has challenged the notion that surgical intervention is always necessary for HPVG. For instance, a case study reported that the successful recovery of male with tumor lysis syndrome and neutropenic sepsis who was treated conservatively for intrahepatic portal gas [18]. Similarly, an 82- year- old diagnosed with ileus, intestinal pneumatosis, and HPVG was managed conservatively and discharged, with HPVG believed to have been induced by mesenteric atherosclerosis and constipation [19]. In another case, a 17-year-old female who underwent an abdominal CT scan due to abdominal pain was found to have low-grade obstruction/ileus and HPVG. The patient responded well to anti-infective therapy, experienced clinical improvement, and was subsequently discharged [8].

Therefore, it is not necessary to immediately resort to surgery based solely on the imaging manifestation of HPVG. The treatment approach for HPVG should take into account the disease process and clinical presentation, focusing on addressing the underlying cause. We recommend performing a CT enterography to further investigate the underlying etiology. If there are no alarming signs and symptoms such as intestinal necrosis or ischemia, conservative management can be effective in treating HPVG. In cases where HPVG is suspected to be caused by invasive bacterial infection, blood culture and empiric antibiotic therapy may be beneficial. However, if CT enterography reveals alarming signs such as intestinal necrosis, ischemia, or intestinal obstruction, and physical examination indicates signs of peritoneal irritation, it is important to consider the need for laparoscopy in addressing HPVG.

Conclusion

Historically, HPVG has been associated with high mortality rates, but advances in CT imaging have revealed its links to various benign conditions. Treatment should focus on the underlying cause, guiding the choice between surgical and conservative approaches, as prognosis depends on this factor. Enhancements in radiology could further aid in accurately diagnosing the cause of HPVG.

Author contributions

Qianqian Zhou, Lian Lin and Junlong Gao wrote the main manuscript text and Hong Zhang revised manuscript. All authors reviewed the manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

This study was approved by the Ethics Committee of Gansu Provincial Hospital.

Competing interests

The authors declare that they have no competing interests, and all authors agreed to publish the manuscript.

Inform consent

A sentence confirming that informed consents (Consent to Participate and Consent to Publish) were obtained from all participants.

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