Hepatic Portal Venous Gas Associated With Ischemic Colitis: A Case Report

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1. Abstract

1.1. 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.

1.2. Case Report:
An octogenarian female patient was admitted to the emergency department due to unexplained abdominal pain, discomfort, nausea and vomiting that had persisted for 6 hours. 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.

1.3. 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.

2. Keywords:
Hepatic portal venous gas, Ischemic Colitis, Case Reports

3. 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. HPVG is defined as the presence of branching radiolucency on radiograph (X-ray) or computed tomography (CT) scan, within 2 centimeters of the Glisson’ s capsule, particularly in the case of left lobe[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]. Subsequently, most HPVG cases have been linked to high mortality and often require 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].

4. 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 hours. 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 the following results: pH 7.546, PCO₂ of 13.6 mmHg, HCO₃ of 18.4 mmol/L, BE of -13 mmol/L. Routine blood test displayed the following findings: WBC of 8.8 x 10⁹/L, Neutrophil ratio of 86.2%, lymphocyte ratio of 7.50%, Hemoglobin of 134 g/L, blood platelet count of 177 x 10⁹/L.  N-terminal probrain natriuretic peptide (NT-pro BNP) was measured at 773.20 pg/mL, High Sensitivity Cardiac Troponin-T (hsTnT) at 0.02 ng/mL, albumin at 32.50 g/L, glutamic oxalacetic transaminase at 69.65 U/L, serum creatinine at 148.70μmol/L, interleukin-6 at 600.36 pg/mL, procalcitonin at 0.793 ng/L, fibrinogen degradation products at 13.26μg/L, D-Dimer at 2.93μg/L. Routine urine test revealed the presence of white blood cell (3+), ketone body (1+). While stool for routine indicated an occult blood test positive for occult blood.

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. On account of stool for routine showed occult blood test positive, and black tarry stool after admission to hospital, we made electronic gastroscopy and electronic colonoscopy. 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. However, due to the risk of colon perforation, a biopsy was not performed. 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 0.5g PO TID, combined Bacillus Subtilis and Enterococcus Faecium Granules with Multivitamines, Live 0.5g 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.

5. 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]. As seen in cases of perspective suggests that gas-producing bacteria within portal vein system generate gas during suffering infection, 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. However, it should be noted that a potential limitation of our case is that we did not perform a CT enterography to further evaluate the presence of intestinal ischemia and thrombosis.

Colonic ulcer refers to the inflammation of the mucosa and submucosa of the colon, which can manifest as localized coloboma or ulceration. It is commonly attributed to various factors such as infection, ischemia, inflammation, tumors, and drug usage. Notable diseases associated with colonic ulcer include ischemic colitis, ulcerative colitis, Crohn’s disease, colonic tumor, drug-induced enteritis, and infective enteritis[17]. 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 in the ascending colon, supporting the hypothesis that gas escapes into the blood vessels by the damaged intestinal mucosa. 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. It is also imperative to draw comparisons with ulcerative colitis and Crohn’s disease. Pathological biopsy remains the most valuable diagnostic tool; however, due to the patient’s severe ulceration with concurrent hemorrhage, performing a biopsy may pose an increased risk of perforation. Consequently, a complete biopsy was not performed in this instance.

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[18]. Consequently,
surgeon have typically recommended surgical intervention when HPVG is detected on imaging. 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[19]. 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[20]. 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]. Based on the patient’s medical history, the absence of previous abdominal pain and melena excludes ulcerative colitis and portal venous gas caused by gastric ulcers. Abdominal CT scan did not identify intestinal obstruction, and colonoscopy did not reveal any signs of obstruction or necrosis. The patient has also not undergone any recent invasive instrument examinations. Therefore, we hypothesize that the patient’s portal venous gas was a consequence of ischemic colitis. The patient, an elderly female, exhibited severe vascular calcification in multiple areas upon abdominal CT examination, which can lead to compromised blood supply to the intestines. Following treatment for ischemic colitis, the patient experienced alleviation of abdominal pain symptoms and no longer presented with melena. Moreover, a follow-up abdominal CT scan revealed the disappearance of portal venous gas.

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.

6. Conclusion:

Historically, HPVG cases have been linked to high mortality. With the widely used of CT, many studies have shown that HPVG was associate with various benign conditions. When it comes the treatment of HPVG, it is crucial to consider the disease process and clinical presentation, directing the approach towards addressing the underlying etiology. The choice between surgical or conservative treatment should be chosen based on the specific underlying cause, as the prognosis and outcome of HPVG are dependent on this factor.

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Availability of data and materials

The data are available from the corresponding author on reasonable request.

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