

Sonographic findings of hepatic venous gas in association with spontaneous rupture of a *Klebsiella pneumoniae* liver abscess: a case report

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Abstract

Hepatic venous gas (HVG) is a very rare ultrasonic finding, and it is defined as abnormal accumulation of gas in the hepatic venous system. Various diseases can cause HVG, and femoral venous catheter is the most common cause. We, herein, present the case of a 79-year-old female patient with HVG that was caused by spontaneous rupture of a *Klebsiella pneumoniae* liver abscess. This was first found by bedside ultrasonography. On the basis of the blood culture results, imipenem–cilastatin and cefoperazone sulbactam were administered and the effect was acceptable. After 41 days of antibacterial and symptomatic treatment in the hospital, the patient had recovered well and was discharged. All of the previous reports on HVG have been summarized by thoroughly reviewing the previous published work. Overall, this is the first patient with HVG in association with spontaneous rupture of a *K. pneumoniae* liver abscess, and it might provide insights for future studies regarding the treatment of this disease.

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Keywords

Hepatic venous gas, liver abscess rupture, *Klebsiella pneumoniae*, bedside ultrasound, spontaneous, treatment, femoral venous catheter

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Introduction

Hepatic venous gas (HVG) is a rare radiological and ultrasonic finding, and it is defined as the abnormal accumulation of gas in the hepatic vein. Several cases with HVG have been reported using ultrasonography (US) and computed tomography (CT). Various conditions and diseases can cause HVG including femoral venous catheterization, emphysematous pyelonephritis, trauma, bowel infarction, and liver abscess.¹⁻⁸ However, the presence of HVG in association with spontaneous rupture of a liver abscess caused by *Klebsiella pneumoniae* has never been reported.

We, herein, report the case of a patient with HVG and a liver abscess caused by *K. pneumoniae* who underwent 41 days of anti-bacterial and symptomatic treatment in the hospital after it ruptured. She recovered well. We also provided a brief review on the topic.

Case report

A 79-year-old Chinese woman with a 5-day history of fever, malaise, anorexia, and progression to confusion and unconsciousness 3 days after the onset of symptoms was admitted to the emergency department at the Peking University International Hospital. She had a history of diabetes (type 2 diabetes) for 23 years, hypertension, and atrial fibrillation. She has previously undergone cholecystectomy, appendectomy, and other operations, and had no

intestinal diseases such as diverticulitis or colorectal cancer.

The patient had pyrexia (maximum temperature [T_{max}], 39°C). Noninvasive mechanical ventilation was provided. Her vital signs (blood pressure [BP], 120/90 mmHg) were normal, but her heart rate (HR) showed tachycardia at 155 beats per minute (bpm). The patient's attitude was indifferent at the hospital visit. Physical examination revealed decreased breath sounds, soft abdomen, and no peripheral edema. Laboratory test results revealed increased procalcitonin level (31 ng/mL), high white blood cell count ($12.21 \times 10^9/L$), anemia (hemoglobin, 93 g/L), low platelet cell count ($22 \times 10^9/L$), and elevated N-terminal pro-B-type natriuretic peptide (NT-pro-BNP) levels (14100 pg/mL).

Abdominal US radiograph (Philips CX50, C5-1 Curved Array, Philips, Seattle, WA, USA) showed a non-homogeneous echo zone of about $6.1 \times 5.3 \times 4.2$ cm in the left lobe of the liver (Figure 1), which showed many tiny high-amplitude hyperechoic foci that were compatible with gas bubbles. Gas bubbles were also observed in the adjacent hepatic vein (Figure 2a, b). The intravascular bubbles were passed into the inferior vena cava and then entered the right atrium (Figure 2c). US examination showed no obvious abnormal hepatic artery perfusion, and there were no obvious abnormalities in echocardiography. During the CT scan, the patient was unable to hold her breath due to unconsciousness, resulting in poor CT

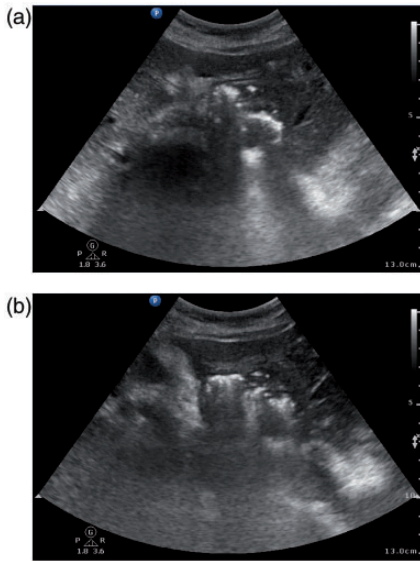


Figure 1. Sonogram of the left hepatic lobe showing many tiny high-amplitude hyperechoic foci that are compatible with gas bubbles of about $6.1 \times 5.3 \times 4.2$ cm (a, b).

image quality. Abdominal CT showed irregularly mixed slightly lower-density lesions in the left lobe of the liver, which showed gas density inside with an unclear boundary (Figure 2d). A small gas density in the adjacent liver parenchyma was observed, and the radiologist strongly suspected that the lesion was an infectious entity. A CT report on the same day from another hospital indicated a ruptured liver abscess and intrahepatic bile duct pneumatosis. Blood cultures of *K. pneumoniae* that were sensitive to imipenem were grown (minimal inhibitory concentration [MIC] <1). On the basis of the bedside US, CT examination, and blood culture results, the patient was diagnosed with a ruptured *K. pneumoniae* liver abscess leading to HVG.

The patient was administered anti-infective treatment with imipenem and cilastatin (Merck Sharp & Dohme Corp., Elkton, VA, USA) at an intravenous dose of 1 g every 8 hours for 14 consecutive days.

The infection index showed subsequent improvement. The treatment was then changed to cefoperazone sulbactam (Pfizer Pharmaceutical Co., Ltd., Dalian, China), which was administered as an intravenous infusion of 3 g once every 8 hours for 5 consecutive days. The patient was administered other general supportive measures. No specific treatment was provided for HVG formation. The patient showed gradual improvement with this treatment regimen. Two days after admission, bedside abdominal US examination revealed no further gas in the hepatic vein and the liver abscess, and a small liquefaction zone appeared in the abscess area. During hospitalization, the patient underwent four subsequent follow-up US examinations. The abscess in the left lobe of the liver gradually shrank and disappeared and no other abscess formation was found. The patient was discharged from the hospital after undergoing 41 days of anti-inflammatory and symptomatic treatment.

Discussion

HVG is a rare condition that is observed during routine US scans, and only a few cases have been reported to date. The first case report of HVG was found by US in a patient with emphysematous pyelonephritis. James et al.⁶ revealed that the intravascular bubbles entered the inferior vena cava through the right renal vein and traveled into the hepatic vein, causing a rare occurrence that represents a transient phenomenon. Galbois et al.⁹ analyzed 235 CT scans of patients in intensive care units and found that 5% of CT scans showed HVG, and all patients with HVG had undergone femoral venous catheterization. Fahrner et al.¹ found that HVG might occur as a transient incidental finding after blunt abdominal trauma. In addition, there are reports of gas embolism in the hepatic vein during laparoscopic liver surgery^{10,11} Tichauer et al.⁷

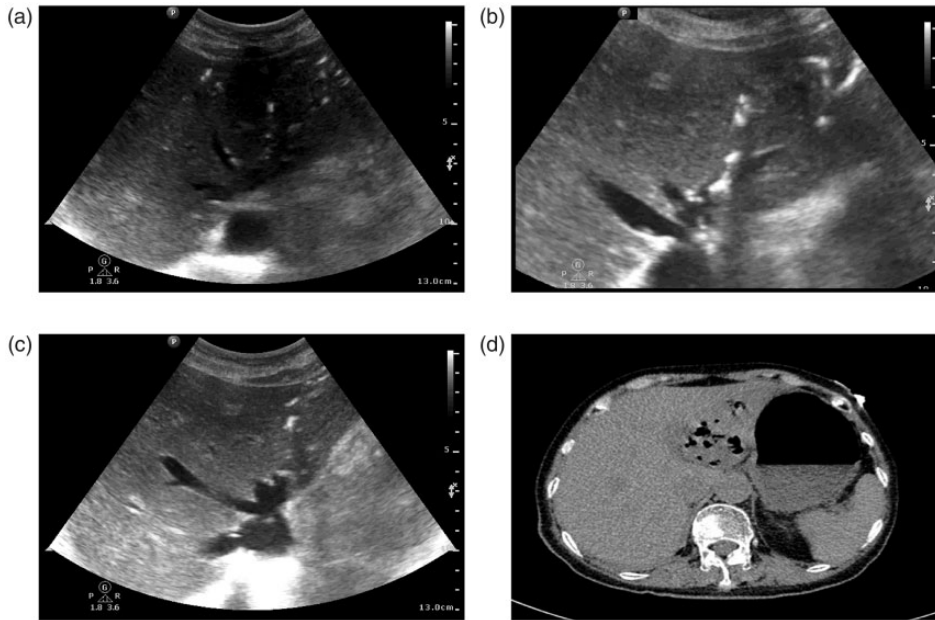


Figure 2. Sonogram of the left hepatic lobe showing multiple small hyperechoic foci of gas in the left hepatic vein (a, b). Multiple small hyperechoic foci of gas in the inferior vena cava (arrow) and liver parenchyma (arrowhead) (b, c). CT images showed irregularly mixed with slightly lower-density lesions in the left lobe of the liver, and the appearance of gas density inside the lesions. The boundary of the lesion was unclear (d). Gas bubbles were oscillating in the hepatic vein and then flowed into the vena cava (real-time ultrasound examination).

CT, computed tomography.

reported a case of HVG that was associated with hepatic abscess, in which the pathogen was unclear, and HVG was confirmed by CT examination. We, herein, reported the case of a patient with HVG associated with spontaneous rupture of liver abscess caused by *K. pneumoniae*, in whom HVG was first confirmed using bedside US examination.

K. pneumoniae is a gram-negative bacterium, and it is the most common pathogenic microorganism that causes gas-forming liver abscesses in patients with diabetes mellitus. High levels of glucose might act as a favorable environment for microorganisms to form gas *via* acid fermentation of glucose compared with normal glucose levels.¹² The liver has a dual source blood supply, which includes sterile arterial blood from the

hepatic artery and venous blood from the gut. Transient bacteremia in the portal system is not uncommon, and the intestinal lumen is the most likely origin of the *K. pneumoniae* isolate that seeds the liver, thus causing an abscess.¹³ Several studies from Taiwan have reported that the incidence of *K. pneumoniae*-related liver abscess rupture ranged from 1.2% to 5.7%.^{12,13} Although it is an uncommon condition, it increases patient morbidity and mortality.¹⁴ The risk factors of spontaneous rupture of liver abscess in HVG patients included liver cirrhosis, large abscess size (≥ 6 cm), gas-forming abscess, and other septic metastases with pyogenic liver abscess.¹⁵

In this report, the sonographer first made an accurate diagnosis of hepatic

abscess rupture combined with HVG and guided further treatment. Because of abscess ruptured in our patient, hospitalization time, antibiotic use, percutaneous/surgical drainage rate, and incidence of metastatic infections were higher than in those with a non-ruptured liver abscess.¹⁴ US has revealed a specific sign for diagnosing HVG, which is the rhythmic movement of gas bubbles in the hepatic vein that, in turn, is related to the heart rate. The visualized rhythmic movement of gas bubbles assists in making a differential diagnosis between HVG and other hepatic venous gases such as portal venous gas and gas in the liver abscess.^{8,9} We also found that the rhythmic movement of large quantities of gas bubbles in the hepatic vein was considered to be evidence of a liver abscess rupture. Because the hepatic venous pressure is higher than the inferior vena cava pressure, these bubbles entered the inferior vena cava and the right atrium. This provides accurate evidence about the invasive *K. pneumoniae* liver abscess syndrome that is caused by blood dissemination, and it includes complications of extrahepatic infections such as endophthalmitis, central nervous system infection, and necrotizing fasciitis.

Both CT and US have high a sensitivity for detecting HVG. Bedside US examination provides the additional advantages of real-time interpretation, organ-targeted imaging, portability, and accessibility.³ In our case, the CT image quality remained poor owing to patient mismatch, and the gas entering the hepatic vein after abscess rupture was mistaken for gas accumulation in the intrahepatic bile duct. However, an accurate diagnosis was made using bedside US on the first attempt. HVG may not have been found by CT because the abscess continued to release a small amount of gas, and the duration of gas release remained short. No HVG was found during the second US examination.

HVG might occur from multiple etiologies. It is necessary and urgent to immediately identify the underlying cause and provide the appropriate treatment.^{5,16} If the gas in the hepatic vein was secondary to a liver infection, then an empirical antibiotic should be used immediately. If treatment is delayed, the bacteria may continue to spread.¹⁶ In our case, imipenem and cilastatin injection for 15 days and cefoperazone sulbactam for 4 days were administered. After symptomatic treatment for 41 days, the patient was discharged.

K. pneumoniae is a potential gas-forming microorganism that is associated with HVG. This could be confirmed by blood culture or by drainage culture. In diabetic patients with fever of unknown etiology, the possibility of hepatic abscess and HVG caused by *K. pneumoniae* should be considered. US examination is considered to be an effective way to diagnose and monitor HVG that is caused by the rupture of a liver abscess, and it provides a timely and convenient diagnosis.

Research ethics and patient consent

Review and approval of institutional review board were not required. Written informed consent was obtained from the participant.

Declaration of conflicting interest

The author(s) declare(s) that there is no conflict of interest.

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References

1. Fahrner R, Rauchfuss F, Scheuerlein H, et al. Posttraumatic venous gas in the liver - a case report and review of the current literature. *BMC Surg* 2018; 18: 14. DOI: 10.1186/s12893-018-0345-z.
2. Shah PA, Cunningham SC, Morgan TA, et al. Hepatic gas: widening spectrum of causes detected at CT and US in the interventional era. *Radiographics* 2011; 31: 1403–1413. DOI: 10.1148/rg.315095108.
3. Poley RN, Ho J and Gooding GA. Sonographic findings of systemic and hepatic venous gas associated with hemodialysis. *J Ultrasound Med* 2009; 28: 533–536. DOI: 10.7863/jum.2009.28.4.533.
4. Torzilli G, Cattaneo S, Lumachi V, et al. Ultrasonographic detection of portal branches and hepatic vein gas associated with mesenteric artery occlusion: a new finding correlated with patient prognosis? *Chir Ital* 2000; 52: 295–298.
5. Chen KW, Huang JJ, Wu MH, et al. Gas in hepatic veins: a rare and critical presentation of emphysematous pyelonephritis. *J Urol* 1994; 151: 125–126. DOI: 10.1016/s0022-5347(17)34889-9.
6. James PN, Chalmers AG and Fowler RC. Ultrasound demonstration of gas in hepatic veins. *Clin Radiol* 1989; 40: 429–430. DOI: 10.1016/s0009-9260(89)80151-5.
7. Tichauer M, Sakpal SV and Chamberlain RS. Right ventricular pneumocardia secondary to hepatic abscesses. *Case Rep Gastroenterol* 2010; 4: 498–501. DOI: 10.1159/000321158.
8. Piton G, Winiszewski H, Capellier G, et al. Echography of hepatic venous gas. The rhythmic movement. *Am J Respir Crit Care Med* 2019; 200: e44. DOI: 10.1164/rccm.201901-0087IM.
9. Galbois A, Cazejust J, Arrivé L, et al. Femoral venous catheter: a misleading cause of gas in the liver. *Crit Care Med* 2011; 39: 2447–2451. DOI: 10.1097/CCM.0b013e318225746d.
10. Otsuka Y, Katagiri T, Ishii J, et al. Gas embolism in laparoscopic hepatectomy: what is the optimal pneumoperitoneal pressure for laparoscopic major hepatectomy? *J Hepatobiliary Pancreat Sci* 2013; 20: 137–140. DOI: 10.1007/s00534-012-0556-0.
11. Kawahara T, Hagiwara M, Takahashi H, et al. Cerebral infarction by paradoxical gas embolism during laparoscopic liver resection with injury of the hepatic vessels in a patient without a right-to-left systemic shunt. *Am J Case Rep* 2017; 18: 687–691. DOI: 10.12659/ajcr.903777.
12. Chen SC, Tsai SJ, Chen CH, et al. Predictors of mortality in patients with pyogenic liver abscess. *Neth J Med* 2008; 66: 196–203.
13. Lee CH, Leu HS, Wu TS, et al. Risk factors for spontaneous rupture of liver abscess caused by *Klebsiella pneumoniae*. *Diagn Microbiol Infect Dis* 2005; 52: 79–84. DOI: 10.1016/j.diagmicrobio.2004.12.016.
14. Chou FF, Sheen-Chen SM and Lee TY. Rupture of pyogenic liver abscess. *Am J Gastroenterol* 1995; 90: 767–770.
15. Jun CH, Yoon JH, Wi JW, et al. Risk factors and clinical outcomes for spontaneous rupture of pyogenic liver abscess. *J Dig Dis* 2015; 16: 31–36. DOI: 10.1111/1751-2980.12209.
16. Foster SC, Schneider B and Seaman WB. Gas-containing pyogenic intrahepatic abscesses. *Radiology* 1970; 94: 613–618. DOI: 10.1148/94.3.613.