



# Emphysematous liver abscess with hepatic portal venous gas

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A 59-year-old man with type 2 diabetes mellitus presented to our emergency department with a 3-day history of chills, fever, malaise, and poor dietary intake. Physical examination revealed a body temperature of 38.5 °C, pulse rate of 73 beats/min, respiratory rate of 16 breaths/min, and blood pressure of 133/71 mmHg. Neither chest nor abdominal pain was noted. Laboratory examinations yielded a white blood cell count of 6980 cells/mm<sup>3</sup> with 80% neutrophils, a platelet cell count of 11,000/mm<sup>3</sup>, C-reactive protein level of 40.7 mg/dL (reference range: <0.3 mg/dL), blood glucose level of 534 mg/dL, and glycosylated hemoglobin level of 11.8%. A chest radiograph revealed obscure gas formation in the right subphrenic area. Computed tomography (CT) of the abdomen revealed a huge emphysematous liver abscess (ELA; size, 12 × 6 cm<sup>2</sup>) in the right lobe of the liver, along with hepatic portal venous gas (HPVG) in the left lobe of the liver (Fig. 1). No other abdominal abnormality was noted. Antimicrobial therapy with a third-generation cephalosporin was initiated. A pigtail catheter was inserted for drainage the ELA. Both blood and drainage pus cultures revealed pan-drug-susceptible *Klebsiella pneumoniae* with identical antimicrobial susceptibility. Antiamoebic antibody test yielded negative result. The ELA gradually reduced in size after treatment, and HPVG disappeared on hospital day 10. However, right pleural effusion occurred, which progressively loculated over the following days. Video-assisted

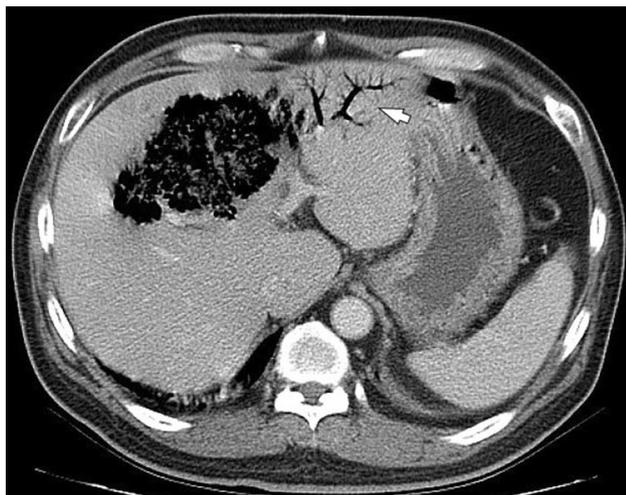
thoracoscopic surgical decortication was performed on hospital day 16. Pleural empyema pus culture was positive for *K. pneumoniae*. A third-generation cephalosporin was administered for 4 weeks. The patient was discharged in a favorable condition on hospital day 28.

HPVG is a radiologic sign that appears when gas accumulates in the portal venous system. It presents a pattern of branching radiolucency extending almost to the periphery of the liver [1]. CT and ultrasound are more sensitive to HPVG than is conventional radiography. Although ultrasound is highly sensitive for HPVG detection and follow-up and can be used as an initial screening, its further use is limited by its interoperator variability being relative high [2]. By contrast, CT not only eases HPVG detection, but also enables identification of its underlying cause in the abdomen [3]. Therefore, CT is the most suitable diagnostic tool for HPVG [3]. More than half of HPVG cases occur in patients with intestinal necrosis, which has a 75% mortality rate [1, 2, 4]. By contrast, HPVG is rarely associated with ulcerative colitis (8%), Crohn disease (4%), complications of endoscopic procedures (4%), cholangitis (2%), and pancreatitis (2%), all of which require only conservative management and do not cause death [1, 4]. HPVG is also associated with intraabdominal abscesses (6%) including those in the pelvis, retroperitoneum, and subphrenic area; such abscesses have a 30% mortality rate [1]. Regarding intraabdominal abscesses, this case report is the first to describe *K. pneumoniae* FLA as a cause of HPVG. Surgical intervention is recommended in cases of intestinal necrosis and intraabdominal abscess, whereas conservative management is advised for patients without ischemic conditions [4]. The overall mortality rate is 39%, but it varies depending on the underlying disease [4]. The prognosis is related to the underlying disease and is not influenced by the presence of HPVG [2, 4]. The predisposing factors of HPVG include (1) mucosal damage due to intestinal necrosis, (2) bowel distention caused by increased internal intestinal pressure including endoscopic procedures, and (3) sepsis due to gas-forming bacteremia [1, 4]. In this case, the possible mechanism of underlying gas formation in the portal venous system may involve *K.*

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**Fig. 1** CT scan of the abdomen indicates a huge gas-forming pyogenic liver abscess in the right liver lobe and branching gas densities in the left liver lobe (arrow)

*pneumonia* bacteremia. HPVG is not a specific diagnostic sign, but a diagnostic clue in patients with acute abdominal conditions, as mentioned previously [4]. As seen in the current case, HPVG disappeared after the ELA resolved. Thus, the approach for treating patient with HPVG should involve the direct treatment of the underlying disease. ELA accounts for 7–24% of all pyogenic liver abscess cases [5]. In patients with diabetes mellitus, *K. pneumoniae* infection is the main cause of ELA development [5]. In this case, the complication of right-sided pleural empyema occurred during hospitalization. The mechanism of this pleural empyema was likely associated with transdiaphragmatic extension of ELA or pleural metastasis infection of the invasive liver abscess syndrome caused by *K. pneumoniae*. Effective antimicrobial therapy, efficient blood sugar control, and an adequate drainage are essential for ELA treatment.

## Compliance with ethical standards

**Conflict of interest** The authors declare that they have no conflict of interest.

**Statement of human and animal rights** All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

**Informed consent** Informed consent was obtained.

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