

CASE REPORT

Gallbladder perforation in cholecystitis with liver abscess formation and septic thrombophlebitis of portal vein mimicking presentation of liver malignancy



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Summary Gallbladder perforation is a rare complication of cholecystitis. Similarly, septic thrombophlebitis of the portal vein, also called pylephlebitis, is another rare complication of intra-abdominal infections including cholecystitis. Both gallbladder perforation and pylephlebitis are associated with significantly higher morbidity and mortality. Herein, we report a patient with an atypical presentation of gallbladder perforation and liver abscess formation. A 68-year-old man suffered from malaise, poor appetite, and body weight loss for 1 month. Liver mass lesion and portal vein thrombosis were detected by ultrasound at a local clinic. He was referred to our institution under the tentative diagnosis of hepatocellular carcinoma. He underwent abdominal ultrasound and computed tomography examinations at our hospital. Cholecystitis with gallbladder perforation was highly suspected. Broad-spectrum antibiotics were administered immediately. Percutaneous transhepatic gallbladder drainage was performed in this case, and pigtail drainage for liver abscess was done later. The patient's condition—cholecystitis, liver abscess, and pylephlebitis (followed by ultrasound)—improved after treatment. Furthermore, the patient recovered his appetite and his body weight increased.

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Introduction

In 1934, Niemeier [1] classified gallbladder perforations (GBPs) into three types: Type I, acute perforation into the free peritoneal cavity; Type II, subacute perforation with abscess formation; and Type III, chronic perforation with cholecystoenteric fistula formation. In order to avoid heterogeneity with regard to data and to facilitate future research, Date et al [2] suggested that reporting types of GBP should be detailed according to the original Niemeier classification in future studies [2]. Thus, our case should be classified as type II, arising after intrahepatic GBP.

Patients with type II perforations are more complicated to manage, because of the atypical presentation of acute cholecystitis and a clinically insidious onset. Initially, a liver mass with portal vein thrombosis in the background of liver cirrhosis was detected by ultrasound in our case. The hepatocellular carcinoma was impressed according to the clinical presentation and imaging findings. The treatment between the infection and malignancy was different. An accurate and early diagnosis of GBP with liver abscess formation is important so as to start the treatment as soon as possible and thereby improve the prognosis.

Case report

A 68-year-old man had been previously healthy without any history of significant systemic diseases and regular medications. Three episodes of fever (up to 38.2°C) without chills developed for 1 day followed by malaise and poor appetite, with nausea. He did not consult a doctor until his weight dropped from 75 kg to 64.3 kg within 1 month. An

evaluation of the abdominal ultrasound at another hospital revealed that he had liver cirrhosis with splenomegaly, a space-occupying lesion of about 70 mm × 60 mm over the left lobe of the liver, and portal vein thrombosis. He was transferred to our hospital for treatment of a suspected hepatocellular carcinoma. The initial clinical findings at our hospital were as follows: pulse rate, 116 beats/minute; systolic blood pressure, 135 mmHg; diastolic blood pressure, 82 mmHg; temperature, 37.1°C. An abdominal examination revealed mild distention without abdominal tenderness. The laboratory findings were as follows: white blood cell count, 16,400/mm³; differential count, band 1% and neutrophil 91%; C-reactive protein, 6.68 mg/dL; platelet count, 215,000/cumm; prothrombin time, 14.1 seconds (control value 8.0–12.0 seconds); activated partial thromboplastin time, 28.8 seconds (control value 23.9–35.5 seconds); protein S, 67.0% (normal value >60%); protein C, 62.8% (normal value >70%). The liver profile was as follows: alanine aminotransferase, 55 IU/L; aspartate aminotransferase, 83 IU/L; alkaline phosphatase, 356 IU/L; gamma glutamyl transpeptidase, 213 IU/L; total bilirubin, 1.43 mg/dL; albumin, 2.2 mg/dL. He denied smoking and alcoholism. Hepatitis B surface antigen and antihepatitis C virus antibody were nonreactive, whereas the anti-HBc antibody was reactive. His Child–Pugh score was 7 (Class B).

The ultrasound (Fig. 1) showed multiple gallstones with heterogeneous contents and an irregular hypoechoic lesion near the gallbladder. Liver cirrhosis with mild ascites and splenomegaly was also noted. An esophagogastroduodenoscopy showed evidence of esophageal and gastric varices. A computed tomography (CT) scan (Fig. 2) revealed gallstones and marked gallbladder distention with wall

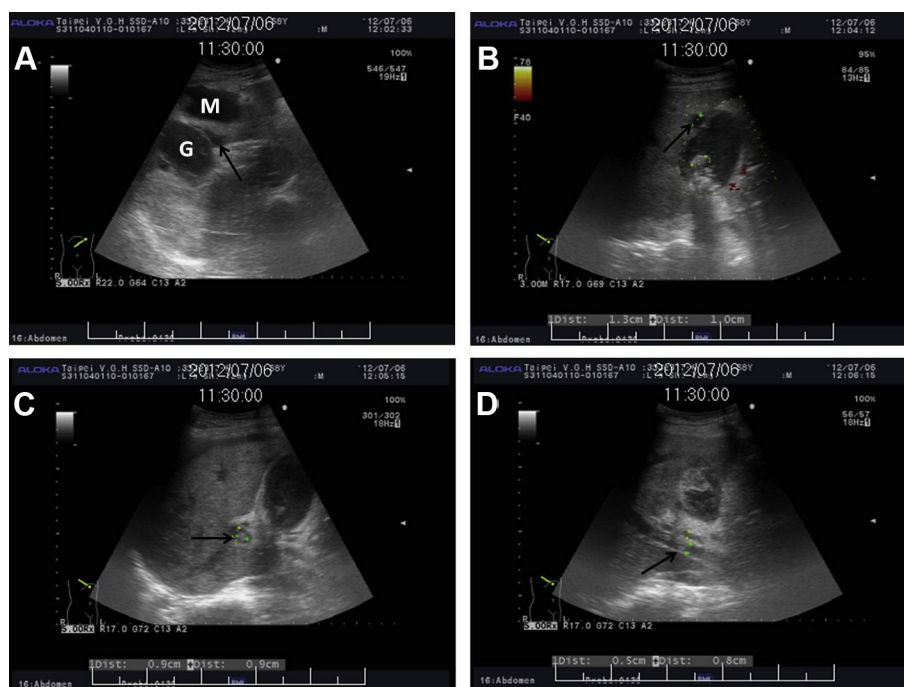


Figure 1 Abdominal ultrasound. (A) One irregular hypoechoic mass lesion (M) 5.5 cm × 6.6 cm in size near the gallbladder (G) and pericholecystic hypoechoic fluid (arrow). (B) Multiple gallstones with acoustic shadow and a small bulging lesion with gallbladder wall defect (arrow). (C, D) Heterogenic contents within the gallbladder and portal vein thrombus (arrow).

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