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## Case Report

# Hemoperitoneum and sepsis from transhepatic gallbladder perforation of acute cholecystitis: A case report<sup>☆</sup>

Fumio Chikamori, MD<sup>a,\*</sup>, Sawaka Yukishige<sup>a</sup>, Koji Ueta, MD<sup>a</sup>, Haruka Takasugi<sup>a</sup>, Kai Mizobuchi<sup>a</sup>, Hisashi Matsuoka, MD<sup>a</sup>, Norihiro Hokimoto, MD<sup>a</sup>, Hiromichi Yamai, MD<sup>a</sup>, Kazuhisa Onishi, MD<sup>a</sup>, Nobuyuki Tanida, MD<sup>a</sup>, Nobumasa Hamaguchi, MD<sup>a</sup>, Takehiro Iwasaki<sup>b</sup>

<sup>a</sup>Department of Surgery, Japanese Red Cross Kochi Hospital, Kochi, Japan

<sup>b</sup>Department of Gastroenterology, Japanese Red Cross Kochi Hospital, Kochi, Japan

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## ABSTRACT

We report a case of hemoperitoneum and sepsis from transhepatic gallbladder perforation in an 87-year-old male with acute cholecystitis who had past history of endoscopic sphincterotomy for common bile duct stone. Contrast-enhanced computed tomography (CT) showed intrahepatic and subcapsular low density areas. A wall defect of gallbladder was seen in coronal and sagittal - sections at the liver bed. Fluids obtained through the paracentesis were hemorrhagic. Percutaneous transhepatic gallbladder drainage (PTGBD) was attempted. First cholangiography revealed an orifice of fistula. Further injection of contrast medium drained into the intrahepatic secondary abscess and intraperitoneal cavity confirming the diagnosis of transhepatic gallbladder perforation. We conclude that contrast-enhanced CT with coronal and sagittal - sections and cholangiography via PTGBD tube are useful to confirm diagnosis of transhepatic gallbladder perforation.

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## Introduction

Hemoperitoneum from transhepatic gallbladder perforation of acute cholecystitis is uncommon [1–5]. Preoperative diagnosis of transhepatic gallbladder perforation is very difficult

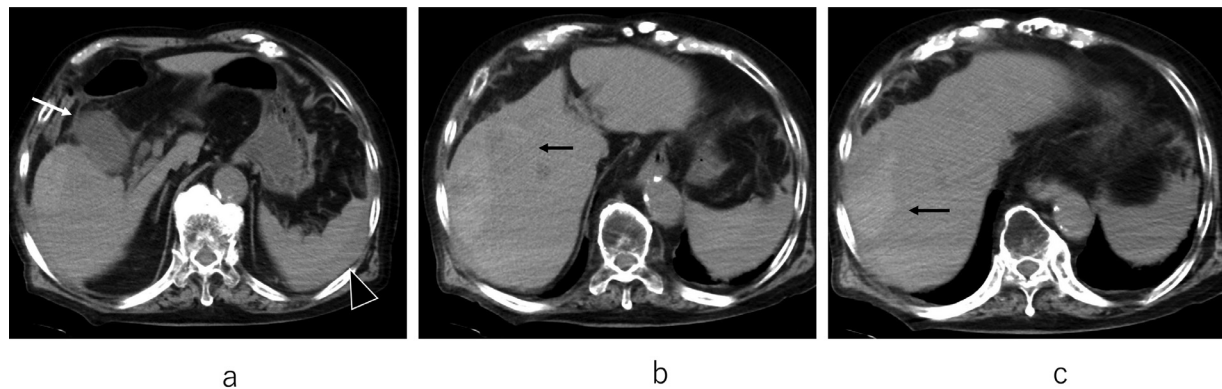
and rate of emergency laparotomy is high. We report a case of transhepatic gallbladder perforation with hemoperitoneum and sepsis, which was suspected by contrast-enhanced computed tomography (CT) with coronal and sagittal - sections and treated by interventional radiology.

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\* Corresponding author.

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**Fig. 1 – Plain CT. (a) Plain CT showed that swelling and wall thickening of the gallbladder were not detected (arrow). A small amount of free fluid around the spleen was present (arrow head). (b) Intrahepatic low density area was suspected but unclear (arrow). (c) Subcapsular high density indicated subcapsular hemorrhage (arrow).**

### Case report

An 87-year-old man was brought in by ambulance with complaints of fever of 38.0°C and general fatigue. He denied a history of trauma or falls. Past medical history showed endoscopic sphincterotomy for choledocholithiasis 10 months ago and percutaneous coronary intervention for ischemic heart disease 8 years ago. He took warfarin potassium of 2 mg and aspirin of 100 mg a day. Other medications were antidiabetic and diuretics.

On admission, the patient had a heart rate of 115 per minute, a blood pressure of 92/61 mmHg, and a respiratory rate of 13 per minute. His body temperature was 36.2°C. Glasgow coma scale was E4, V4, and M4. His height was 164 cm, body weight was 70 kg, and body mass index was 26.0 kg/m<sup>2</sup>. He complained of moderate abdominal pain and tenderness in the epigastrium.

Laboratory results showed a hemoglobin (Hb) of 9.6 g/dL (normal range, 13.5-17.4); a white blood cell count (WBC) of 23170/μL (3500-8000); a platelet count of 13.6 × 10<sup>4</sup>/μL (12.3-33.1); a total bilirubin of 1.0 mg/dL (0.3-1.3); an alanine transaminase of 843 IU/L (10-32); an aspartate aminotransferase level of 339 IU/L (5-27); lactate dehydrogenase of 1278 U/L (106-211); alkaline phosphatase of 947U/L (109-344); blood urea nitrogen of 40.5 mg/dL (8-20); creatinine of 3.06 mg/dL (0.36-1.06); prothrombin time of 18.3 seconds (9-12); prothrombin time % of 44% (70-130); international normalized ratio (INR) of 1.6 (1.0); activated partial thromboplastin time of 38.4 seconds (20-35); procalcitonin (PCT) of 251 ng/mL (0-0.49); C-reactive protein (CRP) of 17.4 mg/dL (<0.16); lactate of 33 mg/dL (5-14); brain natriuretic peptide of 1206 ng/mL (0-18.4); α-Fetoprotein of 0.7 ng/mL (0-20). These results indicated that the patient had multiple comorbidities such as sepsis, anemia, liver and renal dysfunction, heart failure and coagulopathy.

On plain abdominal CT, swelling, and wall thickening of the gallbladder were not detected (Fig. 1a). Subcapsular high density area of the liver indicated subcapsular hemorrhage (Fig. 1b, c). A small amount of free fluid around the spleen was present (Fig. 1a). Initial diagnosis of sepsis due to cholan-

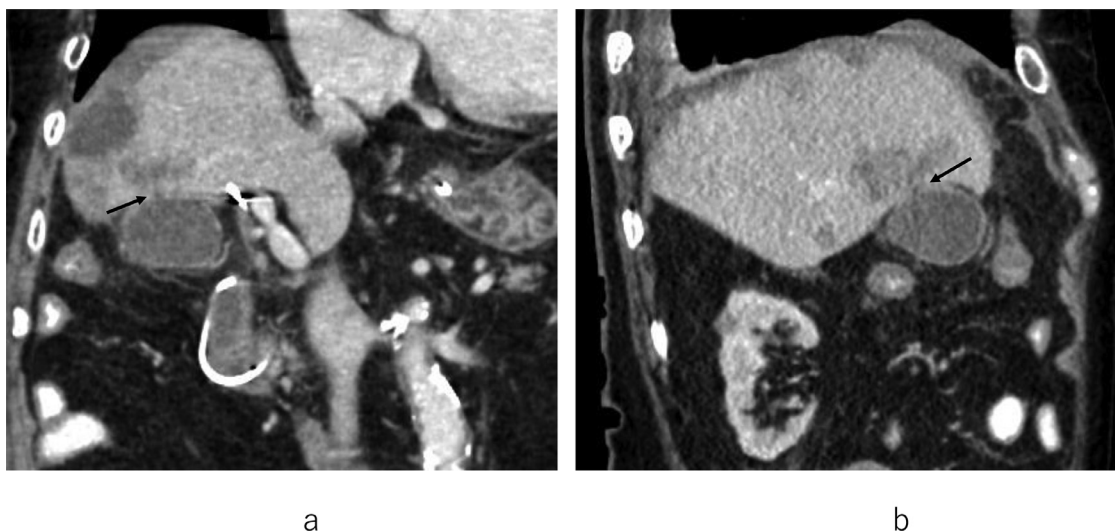
gitis or liver damage was made. He was admitted to intensive care unit. He was treated with intravenous antibiotics, meropenem hydrate. As cholangitis could not be excluded, endoscopic retrograde cholangiography was performed. There was no stone in the bile duct. Endoscopic nasobiliary drainage was performed to monitor quantity and property of bile. The patient was treated with noradrenaline and dobutamine hydrochloride for shock after above mentioned procedure.

Blood culture on the first day was positive for klebsiella oxytoca which was sensitive to meropenem hydrate. It was administered for the next 6 days. Laboratory results on the third day showed Hb of 7.5 g/dL, WBC of 19030/μL, CRP of 30.1 mg/dL, and PCT of 279.6 ng/mL, blood urea nitrogen of 50.1 mg/dL, creatinine of 2.57 mg/dL. Anemia progressed and required 2 units of blood transfusion. Renal damage slightly improved by fluid replacement, however, infection could not be controlled and the inflammatory markers remained high heralding surgical review. Contrast-enhanced abdominal CT showed intrahepatic and subcapsular low density areas (Fig. 2a-c). Although swelling and wall thickening of the gallbladder were not present, a wall defect was seen in coronal and sagittal - sections at the liver bed (Fig. 3a, b). A small amount of free fluid around the liver and spleen was present. Fluids obtained through the paracentesis were hemorrhagic in character. In the presence of these findings, a transhepatic perforation of acute cholecystitis was suspected for this patient. The patient was taken into emergency interventional radiology.

Percutaneous transhepatic gallbladder drainage (PTGBD) was attempted. Abdominal ultrasonography before PTGBD revealed high echogenic debris in the gallbladder, however, the findings of gallbladder swelling, wall thickening and defect were not detected (Fig. 4). A 7 Fr. drainage tube was inserted into the gallbladder. 90 mL of bad smelling red-yellow pus was aspirated. First cholangiography revealed an orifice of fistula (Fig. 5a). Further injection of contrast medium drained into the intrahepatic secondary abscess (Fig. 5b) and intraperitoneal cavity (Fig. 5c). As second procedure, percutaneous abscess drainage was attempted. 150 mL of hemorrhagic fluid was aspirated. As third procedure, abdominal angiography was attempted. There was no evidence of extravasation, hepatic



**Fig. 2 – Contrast-enhanced CT. (a) Contrast-enhanced CT showed intrahepatic low density area at the liver bed (arrow). A swelling and wall thickening of the gallbladder were not present. (b) Intrahepatic (arrow) and subcapsular low density areas were revealed. (c) Subcapsular 2 different low density areas were revealed. A small amount of free fluid around the liver and spleen (arrow head) was present.**



**Fig. 3 – Coronal and sagittal-sections of contrast-enhanced CT. A wall defect was seen in coronal (3a) and sagittal (3b)-sections at the liver bed (arrow).**

artery aneurysm or cystic artery aneurysm. The diagnosis of transhepatic gallbladder perforation with hemoperitoneum and sepsis due to acute cholecystitis was confirmed.

Following PTGBD and percutaneous abscess drainage, his general condition dramatically improved and emergency laparotomy was avoided. Culture of the red-yellow pus aspirated from gallbladder was positive for enterococcus avium, enterococcus gallinarum, and klebsiella oxytoca. Culture of intrahepatic abscess was positive for enterococcus gallinarum. Repeat blood culture on the fifth day was negative. On the seventh day, antibiotics was changed to sulbactam sodium/ampicillin sodium and it was administered for the next 7 days.

On the 17th day, contrast-enhanced CT showed reduction in size and localization of intrahepatic abscess and subcapsular hematoma. Cholangiography via PTGBD tube revealed recanalization of cystic duct. Previously noted orifice of fistula was not detected. Stone in the gallbladder and bile duct was not detected. He was discharged on the 33rd day.

## Discussion

Transhepatic gallbladder perforation is a very rare and life-threatening complication of acute cholecystitis [5]. It is difficult to diagnose transhepatic gallbladder perforation hence rate of emergency laparotomy is high [6]. We suspected transhepatic gallbladder perforation by contrast-enhanced CT, and it was confirmed by cholangiography via PTGBD tube. We could avoid emergency laparotomy.

Eide J et al [1] first presented a case of transhepatic gallbladder perforation causing massive hemoperitoneum. Transhepatic gallbladder perforation can cause massive hemoperitoneum with or without biliary peritonitis and vice versa [7]. Former develops hypovolemic shock, which needs emergency laparotomy, and is treated by hepatectomy or cholecystectomy according to intraoperative findings. Our case was without massive hemoperitoneum thus did not require surgical intervention.





**Fig. 4 – Abdominal ultrasonography before PTGBD.** Abdominal ultrasonography before PTGBD revealed high echogenic debris in the gallbladder, however, the findings of gallbladder swelling, wall thickening and defect were not detected.

Many risk factors causing perforation in acute cholecystitis have been reported [3–5]. Old age, male gender, previous cholecystitis attacks, complicated cases of other severe systemic diseases (malignant, coronary artery disease, cardiopulmonary, and renal diseases, diabetes mellitus, collagen tissue disease, obesity), arteriosclerosis, immunosuppression, long-term use of steroids are important risk factors. Our patient had many risk factors. Physical findings were fever, abdominal pain and tenderness. Blood laboratory examination revealed anemia, increased level of liver enzymes and inflammatory markers such as WBC, CRP and PCT. However, it took 3 days to come to a definitive diagnosis as explained above. This case had 2 problems, 1 was hemoperitoneum with subcapsular

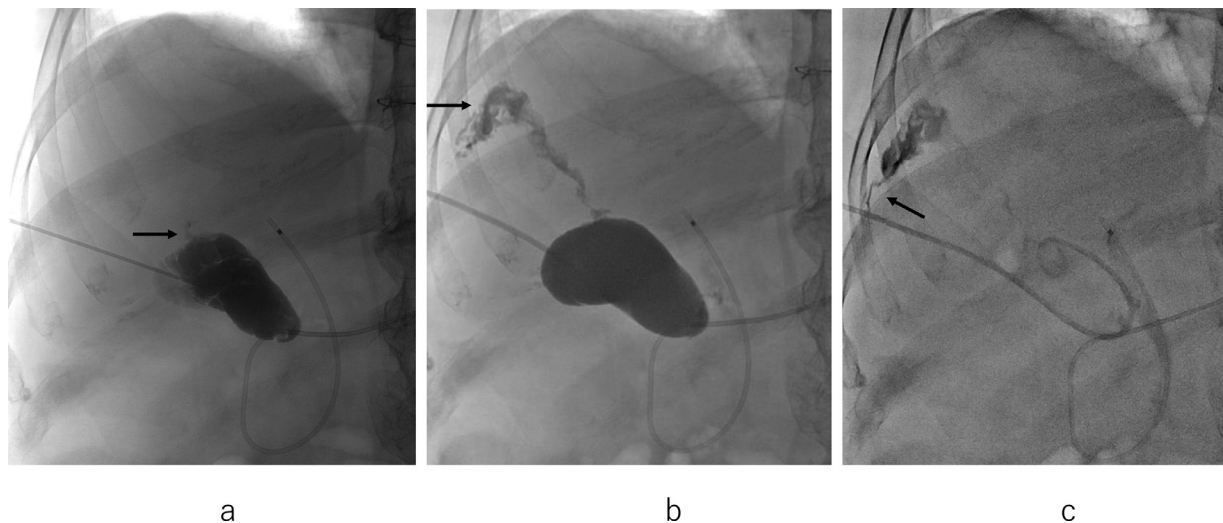
hemorrhage and the other was sepsis. Initially, we could not explain coexistence of hemoperitoneum and sepsis. Plain CT has its own limitation as was shown in this case. By contrast-enhanced CT with coronal and sagittal - sections on the third day, we could suspect transhepatic gallbladder perforation (Fig. 3a, b). Nural MS et al [3] emphasized that the determination of the defect on the lining of the gallbladder wall is very important for diagnosis of perforation. Use of anticoagulants can cause hemoperitoneum. Hemoperitoneum was caused by ruptured hepatic parenchyma, fortunately however, hemoperitoneum was not massive in our case.

Development of gallbladder perforation is triggered by obstruction of the cystic duct, which leads to biliary stasis and gallbladder distension, an increase in intraluminal pressure, compromise of venous and lymphatic drainage, and vascular damage that cause gallbladder necrosis and, finally, a perforation [8]. Niemeier OW [9] classified gallbladder perforations in acute cholecystitis into 3 groups:

- (1) Chronic perforations with the presence of a fistulous communication between the gallbladder and some other viscus.
- (2) Subacute perforations where the perforated gallbladder is surrounded by an abscess walled off by adhesions from the general peritoneal cavity.
- (3) Acute perforation of the gallbladder into the free peritoneal cavity without protective adhesions.

Intrahepatic abscess [10,11] with an intrahepatic perforation may be considered as a special condition of groups 1 and 2. Furthermore, it may be said that transhepatic gallbladder perforation is a very special condition of groups 1, 2, and 3. Once the intraperitoneal leakage from the intrahepatic abscess by cholangiography via PTGBD tube is seen (Fig. 5a-c), diagnosis is accepted as hemoperitoneum caused by transhepatic gallbladder perforation.

Gallbladder perforation should always be considered in the differential diagnosis of peritonitis, especially in elderly



**Fig. 5 – Cholangiography.** (a) First cholangiography showed an orifice of fistula (arrow). (b) Further injection of contrast medium drained into the intrahepatic secondary abscess (arrow). (c) Finally, contrast medium drained into the intraperitoneal cavity (arrow).

patients with findings of cholelithiasis. When hemoperitoneum associates with cholelithiasis, transhepatic gallbladder perforation should be considered. Contrast-enhanced CT with coronal and sagittal - sections is first recommended to detect intrahepatic low density area, gallbladder wall line defect, and intraperitoneal fluid collection. Cholangiography via PTGBD tube becomes a gold standard examination to confirm diagnosis of transhepatic gallbladder perforation. If hemoperitoneum is not massive, emergency laparotomy can be avoided.

We conclude that contrast-enhanced CT with coronal and sagittal - sections and cholangiography via PTGBD tube are useful to confirm diagnosis of transhepatic gallbladder perforation.

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### Patient consent statement

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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