

RARE CASE OF LIVER ABSCESS SECONDARY TO INTRAHEPATIC RUPTURE OF GALLBLADDER & CHOLECYSTOHEPATIC FISTULA COMPLICATING ACUTE CALCULOUS CHOLECYSTITIS IN AN UNCONTROLLED TYPE 2 DIABETIC: A CASE REPORT AND REVIEW OF LITERATURE

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ABSTRACT

Acute calculous cholecystitis rarely progresses to intrahepatic perforation with formation of a giant liver abscess. Such cases are associated with delayed presentation, uncontrolled diabetes mellitus, and significant morbidity. We report the case of a 62-year-old male with poorly controlled diabetes who presented with fever, right hypochondrial pain, vomiting, jaundice, and sepsis. Contrast-enhanced CT revealed acute calculous cholecystitis with intrahepatic rupture of the gallbladder resulting in a large multiloculated hepatic abscess. Initial management consisted of aggressive resuscitation, glycemic optimization, broad-spectrum antibiotics, and ultrasound-guided percutaneous drainage. Persistent postoperative biliary drainage suggested a cholecystohepatic fistula. After optimization of sepsis and metabolic status, laparoscopic cholecystectomy was successfully performed. The patient recovered uneventfully with complete resolution of symptoms. This case highlights the importance of staged management in complicated gallbladder perforation with intrahepatic abscess.

KEYWORDS: Acute calculous cholecystitis, Gallbladder perforation, Intrahepatic rupture, Liver abscess, Cholecystohepatic fistula, Diabetes mellitus.

INTRODUCTION

Gallbladder perforation is an uncommon but potentially life-threatening complication of acute cholecystitis, occurring in approximately 2–11% of patients. According to the classification described by Niemeier, perforations may present as acute free perforation, localized perforation with abscess formation, or chronic fistula formation.

Intrahepatic perforation of the gallbladder leading to a hepatic abscess is exceedingly rare because the gallbladder is usually separated from the liver by connective tissue. Chronic inflammation and pressure necrosis caused by impacted gallstones may result in erosion of the gallbladder wall into the adjacent liver

parenchyma. Delayed diagnosis is common due to nonspecific clinical findings.

Patients with uncontrolled diabetes mellitus are particularly susceptible because of impaired immunity and severe infective complications.

CASE PRESENTATION

A 62-year-old male presented to the emergency department with complaints of pain in the right hypochondrium for 15 days, associated with fever and multiple episodes of vomiting. He had initially been admitted to another hospital for one week before being referred to our tertiary care center because of persistent symptoms. He was a known case of type 2 diabetes mellitus on irregular treatment with poor glycemic

control. On admission, the patient appeared toxic and septic. His clinical examination revealed tachycardia, hypotension, icterus, abdominal distension, bilateral pedal edema, and marked tenderness over the right hypochondrium. He was immediately admitted and managed with aggressive intravenous fluid resuscitation, broad-spectrum intravenous antibiotics, analgesics, insulin therapy, and supportive care.

Laboratory investigations demonstrated severe leukocytosis with a total leukocyte count of 30,000/mm³, total bilirubin of 4.92 mg/dL, direct bilirubin of 4.52 mg/dL, alkaline phosphatase of 720 IU/L, random blood sugar of 621 mg/dL, and glycated hemoglobin (HbA1c) of more than 14%, indicating poorly controlled diabetes mellitus. The patient was co-managed with the medicine team, and intensive insulin therapy was initiated to optimize glycemic status before definitive intervention.

Contrast-enhanced computed tomography (CECT) of the abdomen revealed a two calculi measuring approximately 13 mm in the neck and 7 mm within the lumen. A defect measuring approximately 2.9 cm was identified along the hepatic surface of the gallbladder body, with direct communication between the gallbladder lumen and a large multiloculated intrahepatic collection involving hepatic segments IVA, IVB, V, and VIII. The collection demonstrated multiple internal air foci and measured approximately 13.2 × 11.6 × 13.5 cm, corresponding to an estimated volume of 1075 mL. Minimal perihepatic free fluid was also noted. These findings were consistent with acute calculous cholecystitis with intrahepatic gallbladder rupture and large intra hepatic collection formation.

Considering the patient's sepsis, uncontrolled diabetes mellitus, and the large hepatic abscess, an initial non-operative approach was adopted. Ultrasound-guided percutaneous pigtail catheter drainage of the liver abscess was performed. Approximately 300 mL of frank pus was aspirated immediately following catheter placement. During the first postoperative day, nearly 500 mL of fluid was drained, which was predominantly bilious with only minimal residual pus. By the third day, purulent drainage had completely resolved and the output became entirely bilious. The patient's clinical condition improved progressively with resolution of fever and abdominal pain. Serial laboratory investigations demonstrated a declining leukocyte count, improving bilirubin levels, and decreasing alkaline phosphatase values. He was discharged with the pigtail catheter in situ along with oral antibiotics, insulin and was advised interval follow-up after one week.

During the second admission, the patient remained afebrile and clinically stable. Repeat laboratory investigations showed normalization of the leukocyte count and bilirubin levels with continued improvement in liver function tests. However, persistent hyperglycemia required further insulin optimization. The pigtail catheter

continued to drain approximately 150–200 mL/day of bile, suggesting persistent communication between the gallbladder and the hepatic cavity. Magnetic resonance cholangiopancreatography (MRCP) demonstrated findings similar to those observed on the initial CT scan without evidence of distal biliary obstruction. After optimization of the patient's metabolic status and resolution of sepsis, definitive surgical intervention was planned.

Laparoscopic cholecystectomy was subsequently performed. Intraoperatively, dense inflammatory adhesions were encountered around the gallbladder. A cholecystohepatic fistula involving the posterior wall of the gallbladder was identified, with the posterior wall densely adherent to the liver parenchyma. A large residual cavity corresponding to the previously drained hepatic abscess was also visualized. Laparoscopic cholecystectomy was performed. The posterior gallbladder wall was deliberately left attached to the liver, and the residual mucosa was ablated using electrocautery. A drain was placed in Morrison's pouch, while the previously inserted pigtail catheter was retained.

The postoperative recovery was uneventful. On postoperative day one, the pigtail catheter drained approximately 50 mL of bilious fluid, whereas the abdominal drain yielded approximately 30 mL of serosanguineous fluid. By postoperative day two, no further output was observed from either drain. The abdominal drain was removed on postoperative day three, followed by removal of the pigtail catheter on postoperative day four. The patient remained afebrile throughout the postoperative period, tolerated oral feeding well, and reported complete resolution of abdominal symptoms. He was discharged in stable condition, and at follow-up five days later, remained asymptomatic without any evidence of recurrent collection or bile leak.

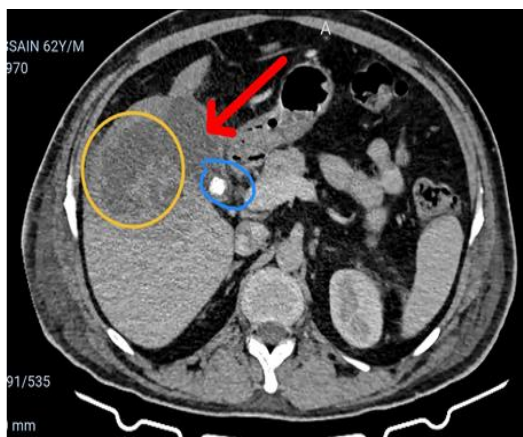


Fig1- CT SCAN SHOWING

RED – GB WITH CHOLECYSTOHEPATIC FISTULA
 BLUE- GB STONE
 YELLOW- INTRAHEPATIC COLLECTION



Fig2- Pigtail Draining Bile



Fig3- MRCP

Showing intrahepatic collection with no distal obstruction

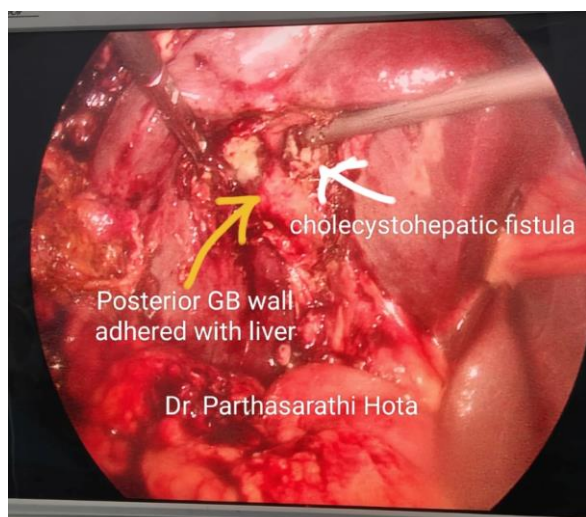


Fig. 4: Intraoperative photograph showing cholecystohepatic fistula [white arrow], densely adhered posterior wall of gall bladder [yellow arrow] & the cystic duct stump in the bottom left corner.

DISCUSSION

Gallbladder perforation (GBP) is an uncommon but potentially life-threatening complication of acute calculous cholecystitis, occurring in approximately 2–11% of patients and carrying a mortality rate of 10–16%, particularly in patients with delayed diagnosis, advanced age, diabetes mellitus, or established sepsis.^[1,2] The pathophysiological mechanism begins with persistent cystic duct obstruction leading to progressive gallbladder distension, impaired venous and lymphatic drainage, mural ischemia, gangrene, and eventual transmural perforation.^[2] Because the fundus has the poorest vascular supply, it represents the commonest site of perforation; however, perforation involving the body or hepatic surface may remain localized by the adjacent liver, resulting in hepatic abscess formation rather than generalized biliary peritonitis.^[2]

The present case represents an exceptionally rare manifestation of acute calculous cholecystitis

complicated by intrahepatic rupture with formation of a giant multiloculated pyogenic liver abscess and cholecystohepatic fistula. Direct extension of gallbladder inflammation into the liver parenchyma is uncommon because the gallbladder is normally separated from the liver by connective tissue and Glisson's capsule. Chronic inflammation, pressure necrosis caused by impacted gallstones, and progressive erosion of the gallbladder wall eventually establish communication with the hepatic parenchyma, producing an intrahepatic abscess.^[3,4] Only isolated case reports and small case series have been published, emphasizing the rarity of this condition and the absence of universally accepted management guidelines.^[3,4]

According to Niemeier's classic classification, gallbladder perforation is categorized as Type I (acute free perforation with generalized biliary peritonitis), Type II (localized perforation with abscess formation), and Type III (chronic perforation resulting in internal or external fistula formation).^[1] However, intrahepatic perforation with cholecystohepatic fistula demonstrates characteristics of both Type II and Type III disease and therefore represents an overlap between localized perforation and chronic fistulous communication.^[3,4] Our patient exhibited both a giant localized hepatic abscess and persistent biliary communication, making this presentation particularly unusual.

Several factors contributed to the severity of disease in the present patient. Most importantly, he had poorly controlled type 2 diabetes mellitus, evidenced by a random blood glucose of 621 mg/dL and HbA1c >14%. Diabetes impairs neutrophil chemotaxis, phagocytosis, intracellular bacterial killing, and cellular immunity while simultaneously producing microvascular ischemia, thereby predisposing patients to severe biliary infection, gangrenous cholecystitis, emphysematous cholecystitis, hepatic abscess formation, and septic complications.^[2,5] Furthermore, the patient's delayed presentation of nearly two weeks, impacted gallstones, and extensive biliary infection likely accelerated gallbladder wall necrosis and intrahepatic extension of infection. Similar associations between uncontrolled diabetes and complicated gallbladder perforation have been consistently reported in the literature.^[2,5]

Contrast-enhanced computed tomography (CECT) played a pivotal role in diagnosis. Although ultrasonography remains the first-line imaging modality for suspected acute cholecystitis, CT is considerably more sensitive for identifying gallbladder wall defects, intrahepatic extension, gas within collections, and associated complications.^[4] In the present case, CT demonstrated a 2.9-cm defect along the hepatic surface of the gallbladder with direct communication to a 13.2 × 11.6 × 13.5 cm multiloculated abscess involving hepatic segments IVA, IVB, V, and VIII. Subsequent MRCP excluded distal biliary obstruction while supporting persistent communication between the gallbladder and

hepatic cavity, thereby facilitating appropriate operative planning.^[3,4]

Current international guidelines recommend that management of severe acute cholecystitis should be individualized according to disease severity and physiological status. The Tokyo Guidelines 2018 (TG18) recommend urgent gallbladder drainage in patients with Grade III acute cholecystitis who are unsuitable for immediate surgery, followed by delayed cholecystectomy after stabilization.^[6] Similarly, the World Society of Emergency Surgery (WSES) guidelines advocate aggressive resuscitation, broad-spectrum antibiotics, early source control, and delayed definitive surgery in patients presenting with severe sepsis or major comorbidities.^[7] These recommendations closely matched the management strategy adopted in our patient.

Initial ultrasound-guided percutaneous pigtail drainage achieved effective source control by evacuating approximately 300 mL of frank pus, followed by progressive reduction in inflammatory markers, improvement in liver function tests, and complete resolution of fever. However, persistence of 150–200 mL/day of bilious drainage after disappearance of purulent material strongly suggested communication between the abscess cavity and the gallbladder. Persistent biliary drainage following hepatic abscess drainage has been recognized as an important indicator of cholecystohepatic or biliary fistula and should prompt definitive surgical treatment after adequate optimization.^[3,4]

Delayed laparoscopic cholecystectomy offered several important advantages. Performing emergency surgery in the presence of severe sepsis, uncontrolled diabetes, and dense inflammatory adhesions would have significantly increased operative difficulty, postoperative morbidity, and the risk of major bile duct injury.^[6,7] Following adequate control of sepsis and metabolic optimization, laparoscopic exploration became feasible. Dense adhesions of posterior wall of gallbladder was difficult to separate and was left in situ and mucosal of left out wall was cauterized.

Compared with previously reported cases, our patient possessed several unique features, including extremely poor glycemic control (HbA1c >14%), a giant (>1 L) multiloculated pyogenic liver abscess, persistent postoperative biliary drainage confirming a cholecystohepatic fistula, and successful staged management using percutaneous drainage followed by laparoscopic cholecystectomy. Most previously published cases required open surgery or were diagnosed intraoperatively.^[3,4] The favorable outcome achieved in our patient demonstrates that a staged minimally invasive approach is both feasible and safe when meticulous source control, metabolic optimization, and careful operative planning are employed.

CONCLUSION

Intrahepatic rupture of acute calculous cholecystitis with giant pyogenic liver abscess is an exceptionally rare but potentially fatal complication. Early recognition with cross-sectional imaging, prompt control of sepsis through image-guided drainage, aggressive optimization of uncontrolled diabetes, and delayed definitive surgery are fundamental to successful management. Persistent bilious drainage following abscess drainage should alert the surgeon to the possibility of a cholecystohepatic fistula. Percutaneous drainage followed by laparoscopic cholecystectomy represents a safe bailout strategy. This case adds to the limited published literature and supports a staged multidisciplinary approach for achieving favorable outcomes in this rare clinical entity.

Conflict of interests: None.

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