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Case Report

Liver abscess as a complication of intrahepatic gallbladder perforation in a septuagenarian: a case report and literature review

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ABSTRACT

Acute cholecystitis is a common complication of gallstone disease; however, gallbladder perforation with intrahepatic abscess formation is a rare and potentially life-threatening entity. We report the case of a 67-year-old man with long-standing type 2 diabetes mellitus, end-stage renal disease on peritoneal dialysis, and hypertension who presented with nonspecific constitutional symptoms and minimal abdominal findings. Laboratory evaluation revealed leukocytosis and abnormal liver function tests. Computed tomography demonstrated acute cholecystitis with intraluminal gas and adjacent hypodense lesions in hepatic segments VII and VIII, concerning for pyogenic liver abscesses secondary to gallbladder perforation. Exploratory laparotomy confirmed complicated acute cholecystitis with dual gallbladder perforation, including posterior extension into the hepatic parenchyma with intrahepatic abscess formation. Due to severe inflammation and unsafe biliary dissection, subtotal fenestrating cholecystectomy was performed as a bailout procedure with abscess drainage. Histopathology confirmed acute ulcerated cholecystitis with mucosal necrosis and intramural gallstones. The postoperative course was uneventful. This case highlights the diagnostic challenge of intrahepatic gallbladder perforation in high-risk patients and supports early cross-sectional imaging and operative source control when severe inflammation precludes safe definitive dissection.

Keywords: Liver abscess, Acute cholecystitis, Cholecystectomy, Gallstone disease, Case report

INTRODUCTION

Gallstone disease affects approximately 20 million people in the United States. It may remain asymptomatic in 60-80% of cases, while approximately 20% of patients will develop complications.[1,2] Acute cholecystitis represents the most common complication of cholelithiasis, occurring in 15-20% of cases, and is caused by obstruction of the cystic duct in 90-95% of cases. Only 20% of acute cholecystitis cases are associated with infection by enterobacteria, mainly *E. coli, Klebsiella*, and *Streptococcus faecalis.*[2]

Acute cholecystitis progresses through three phases: an initial inflammatory phase, characterized by wall congestion and edema, typically appearing 2-4 days after symptom onset. The second phase is characterized by hemorrhage and wall necrosis, which may predispose to perforation and biliary peritonitis, occurring between days 3 and 5. The third phase, chronic or suppurative, is characterized by leukocytic infiltration, necrotic tissue, and intraluminal purulent material.[2]

Gallbladder perforation is a known but uncommon complication, occurring in 1-4% of cases. Niemeier first classified gallbladder perforation in 1934 into three types: Type I (free perforation), type II (localized perforation with abscess formation), and type III (chronic perforation with fistula formation). This classification was later modified to include additional subtypes (Table 1).

Perforation with intrahepatic abscess formation represents a rare complication, with only a few cases reported in the literature.[4] Associated mortality is high, ranging from 12-

42%.[5] The most frequent age of presentation is 62.1±9 years. The most common type is Niemeier type II perforation (46%), followed by type I (40%) and type III (10%).[5]

Table 1: Niemeier´s modified classification.[1,5]

Types	Classification
Type 1	Acute perforation of the gallbladder with generalized biliary peritonitis
Type 2	Subacute perforation with pericholecystic abscess and localized peritonitis
Type 3	Chronic perforation with cholecystoenteric fistula formation
Type 4	Chronic perforation with cholecystobiliary fistula formation

The most frequent site of perforation is the gallbladder fundus, accounting for up to 57% of cases.[4,6] Predisposing factors for this complication include preexisting infections, malignancy, trauma, corticosteroid use, and diabetes mellitus. The most strongly associated predisposing factor is diabetes mellitus.[6]

Objectives were to report a rare case of intrahepatic gallbladder perforation complicated by hepatic abscess, to describe its clinical course and surgical management, and to contribute to the limited existing literature.

CASE REPORT

A 67-year-old male presented to our institution. On February 16, 2024, he developed nausea without vomiting, asthenia, adynamia, and subjective fever. He self-medicated with ceftazidime and paracetamol, with partial improvement. Treatment was discontinued on February 21, 2024. Due to persistent malaise and hyperthermia, he presented to the emergency department.

Past medical history

The patient had a history of: Type 2 diabetes mellitus (30 years), treated with linagliptin and insulin glargine, systemic arterial hypertension (30 years), treated with nifedipine, end-stage renal disease (stage V) on peritoneal dialysis for 4 years, anuric, psoriasis (35 years), untreated glaucoma complicated by retinal detachment, surgical history included right hip prosthesis placement and Tenckhoff catheter insertion. He reported allergy to penicillin. Other history was unremarkable.

Physical examination

On admission, vital signs were stable. The patient was alert (Glasgow Coma Scale 15). Uremic frost and signs of dehydration were noted. Cardiopulmonary examination was unremarkable. The abdomen was flat with a functional Tenckhoff catheter, without signs of infection. Murphy's sign was negative, and no peritoneal irritation was observed.

Laboratory findings

Initial laboratory results showed leukocytosis (14,150/µl) with neutrophilia (91.3%), anemia (hemoglobin 9.1 g/dl), and elevated renal function markers (creatinine 13.64 mg/dl). Liver enzymes were elevated (AST 146 U/l, ALT 69 U/l, ALP 258 U/l, GGT 121 U/l). Hyperglycemia was noted (347 mg/dl).

Follow-up labs demonstrated worsening leukocytosis (20,900/µl).

Peritoneal dialysis fluid analysis showed no evidence of peritonitis.

Imaging studies

Liver and biliary tract ultrasound

Findings suggestive of acute cholecystitis and emphysematous pyogenic abscess in the right hepatic lobe; changes suggestive of hepatic steatosis. CT scan recommended.

Non-contrast abdominopelvic CT

Hepatic abscesses, likely pyogenic, in segments VII and VIII.

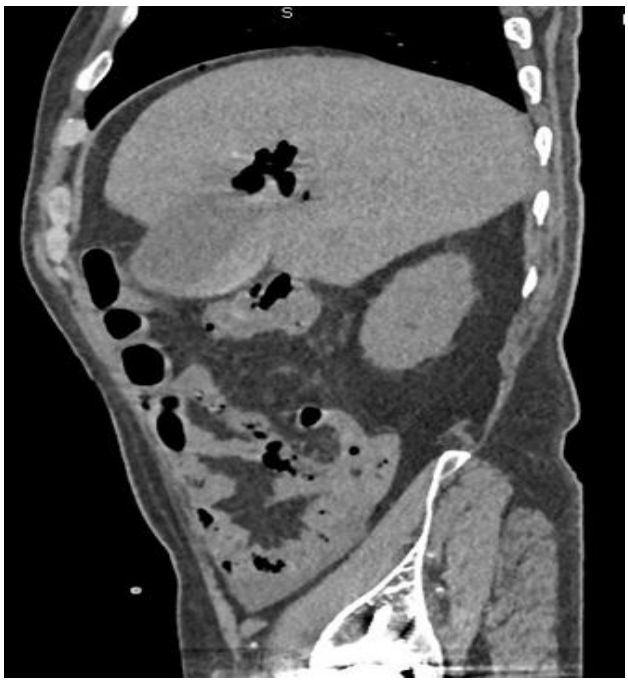

Figure 1: Non-contrast abdominopelvic CT scan (sagittal view).
*Distended gallbladder with intraluminal gas and surrounding inflammatory changes, associated with a hypodense lesion with has in the adjacent hepatic parenchyma suggestive of abscess.

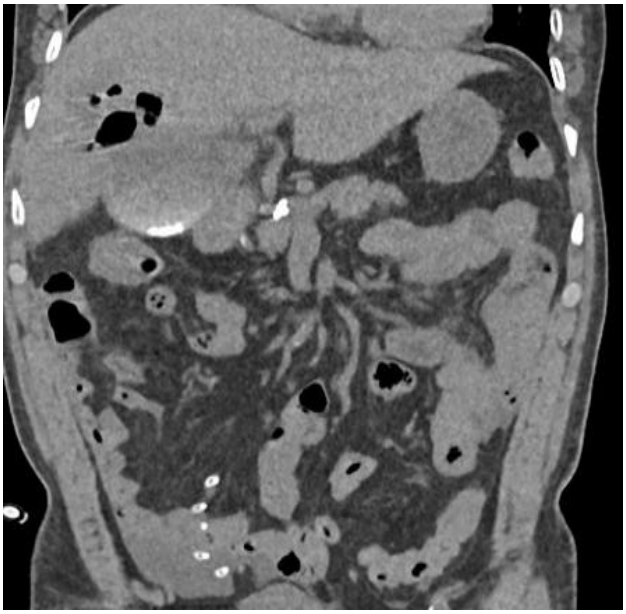

Figure 2: Non-contrast abdominopelvic CT scan (coronal view).
*Demonstrating hypodense lesions in hepatic segments VII and VIII consistent with pyogenic abscesses.

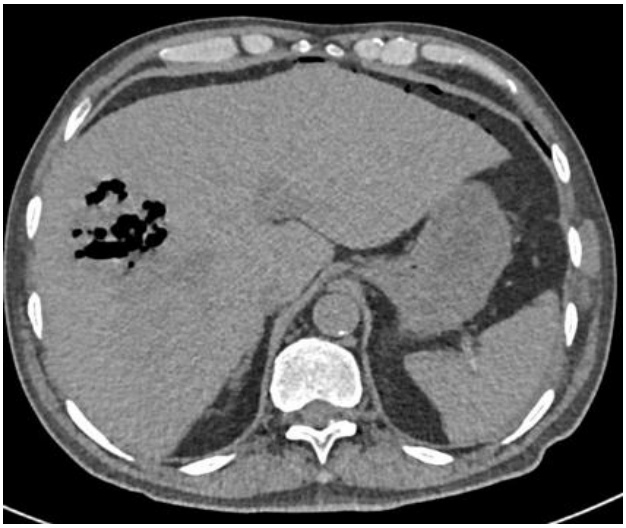

Figure 3: Non-contrast abdominopelvic CT scan (axial view).
*Demonstrating hypodense lesions in hepatic segments VII and VIII consistent with pyogenic abscesses.

Surgical findings

An exploratory supraumbilical midline laparotomy was performed on February 23, 2024. Intraoperative findings included approximately 200 ml of purulent material consistent with complicated acute cholecystitis and microlithiasis. A 1 cm perforation was identified on the anterior gallbladder wall.

Due to inability to achieve the critical view of safety, an antegrade (fundus-first) approach was performed. A subtotal fenestrated cholecystectomy was completed.

A second 1 cm perforation was identified on the posterior wall extending into the hepatic parenchyma, with drainage of approximately 40 mL of purulent material consistent with an intrahepatic abscess. A drain was placed directed toward the abscess cavity. The patient was transitioned to hemodialysis postoperatively.

The patient had a favorable postoperative course and was discharged on postoperative day three with outpatient follow-up. At follow-up, he remained asymptomatic, with no evidence of recurrent infection or biliary complications. The surgical drain was removed one month later.

Pathology report

Acute ulcerated cholecystitis with mucosal necrosis and intramural gallstones.

DISCUSSION

Gallbladder perforation remains an uncommon but severe complication of acute cholecystitis, occurring in approximately 1-4% of cases and associated with substantial morbidity and mortality when diagnosis is delayed.[5,6.] Elderly patients with diabetes and chronic kidney disease are at increased risk of atypical clinical presentations and delayed diagnosis, which may contribute to disease progression and the development of complications such as perforation and abscess formation.[5,6] Date et al reported that gallbladder perforation occurs most frequently in older patients and remains associated with significant mortality, particularly when diagnosis and treatment are delayed.[5] Likewise, Wani et al confirmed that advanced age and diabetes are among the most frequent risk factors in contemporary series of gallbladder perforation.[6]

Intrahepatic gallbladder perforation is an especially rare form of localized perforation in which inflammation and mural necrosis extend directly into the adjacent hepatic parenchyma, resulting in hepatic abscess formation.[4] Hussain et al described this entity as an unusual manifestation of Niemeier type II perforation and emphasized its diagnostic difficulty due to its low incidence and nonspecific presentation.[4] While gallbladder perforation itself is uncommon, hepatic abscess secondary to cholecystohepatic fistulization is even rarer, with only isolated case reports described in the literature.[4,7] The presence of emphysematous features in our patient, demonstrated by intraluminal gas on computed tomography, makes this presentation even more unusual and clinically significant, as emphysematous cholecystitis is associated with more aggressive disease and a greater risk of perforation.[1,2]

Our patient shared several recognized risk factors for complicated gallbladder disease, including advanced age, long-standing diabetes mellitus, and chronic kidney disease. These factors are consistently associated with delayed diagnosis and more advanced disease at

presentation, likely due to impaired inflammatory response, autonomic dysfunction, and atypical symptomatology.[2,5,6] Unlike the classic presentation of acute cholecystitis, our patient had no Murphy's sign, no overt peritoneal irritation, and only subtle abdominal findings despite severe necrotizing gallbladder disease with dual perforation and intrahepatic abscess formation. This atypical presentation is clinically important because it illustrates how gallbladder perforation may progress insidiously in elderly diabetic and immunocompromised patients despite minimal physical findings.

Cross-sectional imaging played a central role in diagnosis. Although ultrasonography remains the initial imaging modality for suspected acute cholecystitis, computed tomography is more sensitive for identifying emphysematous changes, mural discontinuity, perforation, and associated hepatic collections.[2,4] In the present case, computed tomography was critical in demonstrating intraluminal gas and adjacent hepatic hypodense lesions, findings that raised suspicion for gallbladder perforation with secondary pyogenic liver abscess. This radiologic pattern has been described in previous reports and should prompt early operative consideration, particularly in high-risk patients in whom ultrasonographic findings may underestimate disease severity.[1,4]

Management of intrahepatic gallbladder perforation remains poorly standardized because of its rarity. Both staged and definitive approaches have been described, including initial antibiotic therapy with percutaneous drainage followed by interval cholecystectomy, as well as early operative intervention for definitive source control.[8,10] Paramythiotis et al and Alshammari et al demonstrated that drainage-first or minimally invasive strategies may be appropriate in selected hemodynamically stable patients; however, these approaches may be limited in the setting of advanced necrosis, purulent contamination, or uncertain biliary anatomy.[8,10] In our patient, surgery was appropriate because definitive source control was required in the setting of perforated necrotizing cholecystitis with intrahepatic abscess and gross purulence.

Given the severe inflammatory process and inability to safely obtain critical view of safety, subtotal fenestrating cholecystectomy was performed as a bailout procedure. This approach allowed definitive source control while minimizing risk of bile duct injury in a hostile operative field and was safer than attempting total cholecystectomy in the setting of distorted anatomy.[3,8] Prior studies have shown subtotal cholecystectomy to be a safe and effective alternative in difficult gallbladder surgery, particularly when severe inflammation precludes safe dissection, although postoperative bile leakage remains a recognized risk.[3,8] For this reason, drain placement was appropriate and allowed controlled postoperative monitoring of the gallbladder fossa and abscess cavity. In present case, this strategy was associated with an uneventful recovery and no postoperative biliary complications.

An additional distinguishing feature of this case was the patient's history of end-stage renal disease on peritoneal dialysis. Few reports have examined gallbladder perforation in this population, but delayed diagnosis may be more likely because abdominal symptoms can be subtle and alternative intra-abdominal sources of infection are often considered first.[9] Wu et al highlighted the diagnostic complexity of gallbladder perforation in peritoneal dialysis patients, emphasizing the need for heightened suspicion in this subgroup.[9] In this context, our case expands the limited literature by demonstrating that severe gallbladder pathology may occur in peritoneal dialysis patients without overt peritoneal signs/dialysis-associated peritonitis.

The educational value of this case lies not only in its rarity, but in its diagnostic and operative implications. The absence of peritonitis does not exclude severe gallbladder disease, and minimal abdominal findings should not reassure clinician in elderly diabetic/ immunocompromised patients with systemic inflammatory features. This case reinforces the need for a high index of suspicion, early cross-sectional imaging, and prompt source control in medically complex patients with suspected complicated cholecystitis.

CONCLUSION

Intrahepatic gallbladder perforation with hepatic abscess is a rare and potentially life-threatening complication of acute cholecystitis that often presents with subtle or atypical clinical findings, particularly in patients with diabetes and significant comorbidities. The absence of peritoneal signs does not exclude advanced gallbladder disease, and delayed recognition may allow progression to perforation and intrahepatic extension. Cross-sectional imaging is essential when this diagnosis is suspected, particularly in the presence of adjacent hepatic collections or emphysematous changes. Early operative source control remains the cornerstone of treatment, and subtotal cholecystectomy represents a safe and effective bailout strategy when severe inflammation precludes safe biliary dissection. This case expands the limited literature on intrahepatic gallbladder perforation and highlights the importance of maintaining a high index of suspicion in high-risk patients with subtle abdominal findings.

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