

Laparoscopic Treatment of a Hepatic Subcapsular Abscess Secondary to Gallbladder Perforation: Case Report

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Rezumat

Tratamentul laparoscopic al unui abces hepatic subcapsular secundar perforaţiei vezicii biliare: raportarea unui caz

Prezentăm un caz rar de perforaţie a vezicii biliare, de tip Niemeier II, apărută ca o complicaţie a colecistitei acute litiazice. Un pacient în vârstă de 76 de ani s-a internat în clinica noastră cu diagnosticul prezumtiv de colecistită acută litiazică. Examenul computer tomograf a evidenţiat o colecţie subcapsulară dezvoltată pe faţa viscerală a lobului drept hepatic. Aceasta comunica printr-o bandă hipodensă subţire cu canalul cistic, distal de un calcul cistic impactat. Laparoscopia diagnostică a evidenţiat un abces hepatic subcapsular secundar perforaţiei veziculei biliare. Colecistectomia şi tratamentul abscesului s-au realizat integral pe cale laparoscopică. Articolul demonstrează că, în această situaţie, abordul laparoscopic poate fi sigur şi fezabil pentru tratamentul atât al cauzei cât şi al complicaţiei. Diagnosticul precoce şi abordul miniminvasiv reprezintă cheia rezolvării acestei rarităţi.

Cuvinte cheie: perforaţie tip II vezică biliară, abces hepatic subcapsular, colecistită acută litiazică

Abstract

We present a rare case of type II Niemeier gallbladder perforation (GBP) developed as a complication of acute calculous cholecystitis. A 76-year-old man was admitted to our surgical unit with the presumptive diagnosis of acute cholecystitis. CT scan revealed a subcapsular collection developed on the visceral surface of the right hepatic lobe. It was communicating through a thin hypodense band with the cystic duct, distal to an impacted stone. Through laparoscopy the collection was confirmed to be a subcapsular liver abscess secondary to GBP. The cholecystectomy and the abscess cavity treatment were completely handled via laparoscopic approach. The paper demonstrates that laparoscopic approach can be a safe and feasible method in order to treat both the cause and the complication in this situation. Early diagnosis and appropriate minimally invasive approach are the key to manage this rarity.

Key words: type II gallbladder perforation, subcapsular liver abscess, acute calculous cholecystitis

Introduction

In cohort retrospective studies acute cholecystitis may result in gallbladder perforation (GBP) in 2 to 12% of cases (1,2). The most important associated risk factor for this occurrence is cholelithiasis (2,3,4).

According to the inflammation progress and type of perforation, Niemeier subdivided GBP into 3 types (1,5,6,7). Type I (acute) is associated with free perforation into the peritoneal cavity. Type II (subacute) perforation consists in the

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localization of the fluid at the perforation site, pericholecystic abscess and localized peritonitis. If the perforation site is covered by the omentum, the intestines or the visceral surface of the liver, the infection remains limited in the supramesocolic space with formation of a plastron, pericholecystic fluid or an intrahepatic abscess (1,2,3). Therefore the GBP can cause a cholecystohepatic communication with consequent spreading of the infection into the liver. The type III (chronic) perforation consists of internal or external fistula formation (1,3,5).

An early cholecystectomy strategy and the improved antibiotic treatment have reduced the risk of GBP in acute cholecystitis to 0.8-3.2% today (6). Pericholecystic abscesses are rarely found in the liver. Sometimes the symptoms of type II perforation may not be differentiated from the other types or from uncomplicated acute cholecystitis causing the diagnosis to potentially be delayed, especially when acute symptoms including peritonism are missing (2,6,7).

Case report

A 76-year-old man was admitted to our service with a low grade fever, anorexia, nausea and right-sided abdominal pain. Hypertension and a repaired right sided inguinal hernia 6 months back were significant in the patient's history. Two weeks previously to this he was admitted in another institution with similar complaints. The laboratory tests done at that time showed elevated inflammatory markers. Initial ultrasonography (US) revealed gallbladder sludge with mildly oedematous gallbladder wall, but no calculus was apparently observed. Due to the patient's refusal to undertake more invasive diagnostic procedures and in accordance with his condition which improved under intravenous therapy he was subsequently discharged on oral antibiotics. His clinical condition worsened and acute cholecystitis was presumed, therefore the patient was referred to our hospital to undergo a further evaluation.

On admission he had a temperature of 38.2°C, pain and tenderness in the right upper quadrant, but there was no palpable mass. The pain was aggravated on respiratory movement and relieved by rest, with no other associated symptoms. Laboratory studies showed a white cell count of 11340/ μ L (ref. level 4000–9000/ μ L), C-reactive protein at a level of 7.3 mg/dL (ref. level <0.5 mg/dL) and a fibrinogen serum value of 780 mg/dl (ref. level 200–450 mg/dL). Apart from the above referenced results, the blood cell tests were normal.

Standard X-rays revealed no signs of intestinal obstruction or pneumoperitoneum in the abdomen. The upper gastro-intestinal endoscopy showed an extrinsic compression of the antropyloric region by a posterior tumor-like mass which did not infiltrate the mucosa.

Abdominal ultrasound and the CT scan revealed an impacted stone (6 mm in diameter) in the distal segment of the cystic duct, a slightly distended gallbladder with diffuse and light wall thickening. The common bile duct diameter was normal. The CT exam also presented a subcapsular collection with clear margins on the visceral surface of the right hepatic lobe which was communicating through a thin



Figure 1. CT scan shows a 4/4 cm subcapsular hypodense lesion which is connected with the cystic duct through a hypodense 0.8 mm band. A 6 mm stone (arrow) impacted in the cystic duct

hypodense band with the cystic duct, distally to the stone (Figs. 1, 2). No enhancement was seen following contrast agent administration. There were also inflammatory changes of the subhepatic fat and Morison's pouch observed.

Laparoscopy was performed and it revealed a subhepatic plastron with dense inflammatory adhesions of the gall bladder to the surrounding structures. As the Calot triangle could not be well identified initially, the cholecystectomy was performed



Figure 2. CT-sagittal view: The collection (arrow) is located on the visceral surface of the right hepatic lobe. Inflammatory changes in the hepatorenal fossa

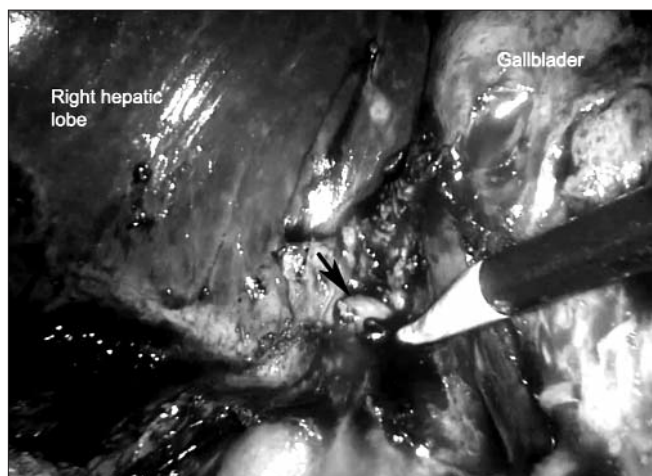


Figure 3. Laparoscopic view: The abscess is opened. Arrow indicates pus coming out from the abscess cavity

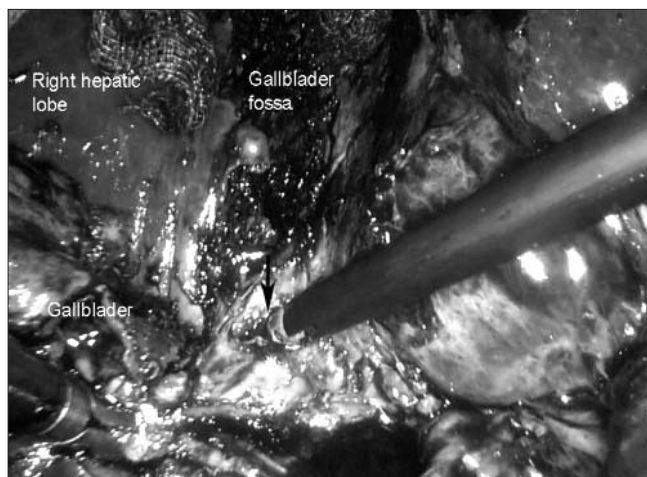


Figure 4. Laparoscopic view: After antegrade dissection from its fossa, the gallbladder is tractioned to the right. The abscess cavity is washed-out (arrow)

by antegrade blunt and gentle dissection. Posteriorly at the gall bladder's neck level we identified a fistulous communication between the bladder and an abscess cavity developed in the subcapsular area of the right hepatic lobe, on its visceral surface, which contained a mix of bile and pus (Figs. 2, 3). The pus was aspirated and sent for culture and antibiogram. The abscess cavity was washed out, the stone was removed from the cystic duct and the cholecystectomy was finalized. The abscess cavity and the subhepatic space were drained. At the time of general anaesthesia induction and during hospital stay the patient received 3rd generation cephalosporins.

The postoperative course was uneventful and his drains were removed on the 2nd (the intracavitary one) and the 3rd (subhepatic one) postoperative day. The follow-up CT showed that the abscess cavity disappeared. He was discharged from the hospital 5 days after the surgery. A histopathological diagnosis of acute on chronic cholecystitis was noted. A localized necrosis was seen at the site of perforation and several thrombi within the intramural vessels were detected (Fig. 5).

Discussion

GBP represents a rare complication of acute cholecystitis which is preoperatively diagnosed with a frequency of 8.3 to 23.8% (3,8). Although acute uncomplicated cholecystitis is more common among females, GBP is more frequent in males over 60 years of age, especially for the type II and type III perforation (2,7). Perforation is usually associated with the presence of stones but it can also be encountered in acute acalculous cholecystitis, however with a lower rate (3). Cholelithiasis can be unknown prior to perforation as we faced in our case as well (8). The duration of symptoms in our case was 14 days and the mean reported in previous studies was 8.5-9 days for the type II perforation (2,8). The clinical presentation of GBP is often non-specific and the

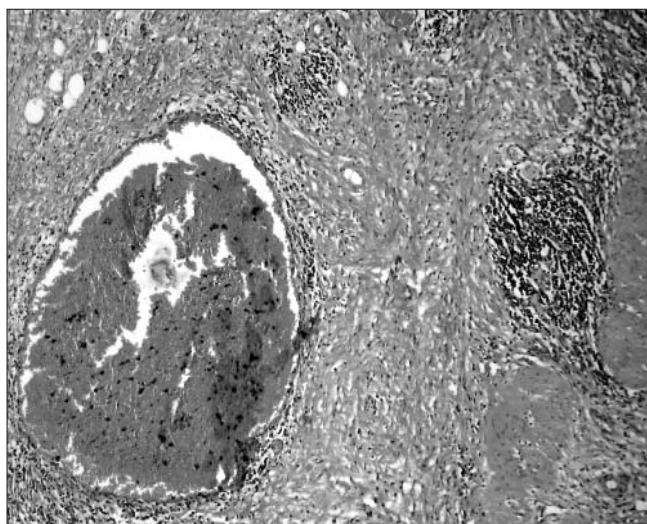


Figure 5. Histology (hematoxylin-eosin, magnification 10x): A thrombus within the intramural vessel. Adjacent inflammatory changes

laboratory tests are usually similar to other common diseases. Therefore the differential diagnosis with uncomplicated acute cholecystitis or other benign gastroenterological diseases is unclear and may not be established preoperatively. This could result in a delayed positive diagnosis which increases morbidity and mortality (2,4). For the cases diagnosed intraoperatively as a type II GBP, the most common preoperative diagnosis is acute uncomplicated cholecystitis (4,8).

Being a noninvasive procedure, ultrasound is the first choice when it comes to imaging procedures in patients clinically suspected of having acute cholecystitis and also for evaluating the suspicions of gallbladder perforation. The detection of the defect in the gallbladder wall ("the hole sign") is the only reliable sign of gallbladder perforation (4,7,9).

Although some authors cited higher rate of findings of this sign (9), the event is rare, the ultrasound being more effective in demonstrating pericholecystic or intrahepatic fluid collections, gallbladder wall thickening and cholelithiasis (2,8). CT scan is particularly useful in cases of discrepancies between clinical symptoms and an inconclusive ultrasound, in the assessment of suspected complications of acute cholecystitis and for better pre-operative planning if the ultrasound proved the perforation (6). However it can more accurately show the pericholecystic changes, the abscess and the defect of the wall due to the perforation, as it happened in our case as well. MRI exam, by its superior soft tissue resolution and multiplanar capability, can be a possible diagnostic option in order to demonstrate the defects of the gallbladder wall (1,7). In some isolated cases the color Doppler ultrasonography was used to show a flow signal passing through the perforated site, being a promising alternative in the diagnosis of GB perforation (4,8,9). Percutaneous radiological - guided aspiration with bilirubin testing can be relevant for a bile duct perforation. The fluid obtained must be also microbiologically analysed in order to diagnose an infection.

In the case presented the perforation site was at the junction between the gall bladder's neck level and the cystic duct. For a type II perforation it represents a less common intraoperative finding than the case of a fundus defect, as it was revealed by different studies (14.3 - 23.8% as compared to 33.3 - 42.9%) (2,8).

Early surgery may be a safe option in acute cholecystitis (10). A data meta-analysis proved that as compared to delayed cholecystectomy, early laparoscopic approach in acute cholecystitis allows significantly shorter total hospital stay. This could also increase the cost-effective benefits. The operation time is significantly longer and no important differences in conversion or overall complications rates were described (10,11,12,13).

In the case of a liver abscess preoperatively confirmed by the imaging exams the infection can be initially controlled by US guided percutaneous drainage of the collection $\pm$ cholecystotomy, followed by elective cholecystectomy. This strategy is recommended especially in high risk surgical patients (8,11,12,15).

We preferred an early laparoscopic approach due to his minimally invasive character and the possibility to treat both the disease and his complication over the same surgical time, in accordance with the general patient state. However, laparoscopic complete management is technically possible in less than 25% of the type II perforation cases (2,8,10). In type II GBP the anatomy is often unclear and thus conversion is required. Hereby laparoscopy is advised for its diagnostic value which is crucial, particularly if the preoperative radiological findings are equivocal (1,8,9,10).

Conclusions

In summary, GBP is difficult to diagnose preoperatively based only on the clinical features and US findings. In acute cholecystitis the surgeon should be aware of the possibility to identify a surrounding collection, even in the liver. Early diagnosis and appropriate minimally invasive approach are the key to managing this rarity (12,13,14,15).

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