An Unusual Presentation of Gall Bladder Perforation with Hepatic Subcapsular Collection

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Abstract A 60 year old male presented with Gall Bladder perforation in a case of calculous cholecystitis with perforation at the tip of the fundus. The perforation lead to collection of bile under the liver capsule. This case is unusual as clinically suggestive of liver abscess and the perforation didn't lead to biliary peritonitis. Gall Bladder perforation is life threatening event associated with increased morbidity and mortality. Cholecystectomy with peritoneal lavage is the treatment of choice.

Keywords Gall bladder perforation · Hepatic sub-capsular collection · Complications of Cholelithiasis

Introduction

Gallbladder perforation is a life-threatening event, and it can be traumatic, iatrogenic, or idiopathic. Conditions such as cholelithiasis, infections, malignancy, steroid therapy, diabetes mellitus, and atherosclerotic heart disease are all predisposing factors. A study shows that gallbladder perforation accounts for 3–15 % in acute calculus

cholecystitis. The commonest site of perforation is fundus of the gallbladder [3].

Case Report

A 60-year-old man came with pain in the right upper abdomen for 6 days, nausea for 5 day, and retching for 5 days without any comorbidity.

On presentation, the patient had tachycardia and other vital parameters were within normal limits. Abdominal examination showed tenderness in the right hypochondrium and in the right lower intercostal area. Guarding was present. The liver was palpable two fingers below the costal margins. No rigidity or distension was noted. Bowel sounds were present.

Routine examination of blood showed hemoglobin 13.3 g/dl and TLC (total leucocyte count) $11,100/\text{mm}^3$; LFT (liver function tests) was within normal limits except alkaline phosphatase 121 IU/l (increased). BUN (blood urea nitrogen), serum creatinine, BT (bleeding time), CT (Clotting time), PT (prothrombin time), and urine routine microscopy were all normal. X-ray abdomen (erect) showed no signs of pneumoperitoneum.

USG abdomen and pelvis (Fig. 1a, b) showed the normal size liver with large multiloculated subcapsular collection, approximately $15.5\text{ cm} \times 13.5\text{ cm} \times 6.5\text{ cm}$ along the superolateral aspect of the right lobe of liver extending up to the left lobe on dorsal surface. USG-guided aspiration was done, and as the aspiration was not completed and seemed not to be ending, pig-tail catheterization (Fig. 2) was done. Aspirate was sent for cytology and culture sensitivity which showed no growth and was bile.

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Fig. 1 **a** Ultrasound showing hepatic subcapsular collection.
b Ultrasound showing hepatic subcapsular collection

After 3 days of pig-tail catheterization, bile was draining for 400–500 ml/day and not reducing. CT scan abdomen and pelvis (Fig. 3, 4) was done which showed a large collection in the subcapsular location of the right lobe of liver on lateral, superior, and anterior aspects up to the left lobe of liver with gallbladder sludge or calculi.

In spite of pig-tail catheter draining for 3 days, the size of collection did not reduce and it was decided to perform

exploratory laparotomy. Right subcostal incision was made. Intraoperative findings showed the following:

1. Huge biliary collection in the right subphrenic space. (Fig. 5)
2. Liver capsule torn and rolled up due to repeated aspiration.
3. Perforation seen to be covered by the liver capsule at the tip of the fundus of gallbladder of less than 5 mm. (Fig. 6)
4. Omentum had cordoned the area.

Open cholecystectomy was done. On cut section (Fig. 7, 8) of the gallbladder, multiple calculi were seen. Thorough peritoneal lavage was given and subhepatic drain was kept. The patient went home uneventfully on the eighth postoperative day.

Fig. 2 Pigtail catheter in right hypochondrium

Discussion

Gallbladder perforation accounts for 1–4 % in cases of acute calculus cholecystitis [1]. Perforation can occur as early as

Fig. 3 CT scan showing tip of pigtail catheter in subphrenic space and peri-hepatic collection

2 days after the onset of acute cholecystitis, or after several weeks [3, 4] the sequence of events that leads to acute cholecystitis and subsequently to perforations is thought to result from occlusion of the cystic duct (most often by a calculus), resulting in retention of intraluminal secretions. Distension of the organ with a consequent rise in intraluminal pressure impedes venous and lymphatic drainage, leading to vascular compromise and ultimately to necrosis and perforation of the wall of the gallbladder [5, 6]. Because of its poor blood supply, the fundus of the gallbladder is the most common site of perforation [3]. Despite a propensity

for perforation in acalculous cholecystitis, most cases of perforation are associated with a calculus because of the higher incidence of calculus cholecystitis [7]. In our case, the perforation was at the tip of the fundus which was covered by the liver capsule.

Apart from cholelithiasis and infection, factors that predispose to perforation are malignancy, trauma, and drugs (e.g., corticosteroids). Systemic diseases such as diabetes mellitus and atherosclerotic heart disease are also thought to be contributory [3], and elderly patients are especially susceptible to gallbladder perforation [8]. Glenn and Moore

Fig. 4 CT scan showing peri-hepatic collection

Fig. 5 Liver surface laden with bile

in an early study reported an incidence of perforation of the gallbladder five times higher among those patients treated conservatively than among those who underwent cholecystectomy [5]. The incidence of perforation is known to increase fourfold with a delay in surgery of more than 2 days from the onset of abdominal symptoms [9]. At surgery, the site of perforations is most commonly found to be sealed off by omentum with adhesion to the liver capsule [10, 11], as seen in our study. Williams and Scobie reported that in cases of gallbladder perforation in patients with acute cholecystitis, the mean age was 69 years with a female-to-male ratio of 3:2 [13].

In the early decades of the last century, Niemeier categorized perforation into three types [12]: type 1 (acute 33–37 %), which manifests with generalized peritonitis; type 2 (subacute 43–53 %), which denotes localization of fluid at the site of perforation with the formation of a pericholecystic abscess; and type 3 (chronic 10 % to 19 %), in which internal (bilio-biliary or bilio-enteric) or external fistulae occur [1]. Recent studies cite a higher incidence of subacute perforation/type II perforations as compared to other types [2]. Our study could not be classified as there was no sign of biliary peritonitis, liver abscess, or fistula.

Fig. 6 Perforation of gall bladder fundus

Fig. 7 Multiple calculi from gall bladder

As calculi are often missed on CT, sonography followed by CT is preferred in suspected biliary pathology [15,16]. Ultrasound and CT in combination are used to diagnose suspected gallbladder perforation; recently MRI scan has also been suggested as more accurate than these two conventional imaging techniques [12]. Ultrasonography is usually the initial mode of investigation in cases of suspected gallbladder perforation. As the sensitivity of CT in the detection of gallbladder perforation and biliary calculi was found to be 88 and 89 %, respectively, it is recommended to be performed in all cases of gallbladder perforation [14]. In our case, ultrasound was suggestive of liver abscess or collection and did not diagnose gallbladder perforation. CT scan only suggested of gallbladder sludge or calculi.

Conclusion

Our case of gallbladder perforation is unusual because it does not fit in any of the three groups of Niemeier's classification. Our patient had subcapsular bile collection over the dorsal and anterolateral surface of the liver.

The traditional diagnostic modalities such as USG and CT scan could not diagnose gallbladder perforation although subcapsular collection under liver capsule and gallbladder sludge or calculi were diagnosed.

In our case, repeated aspirations and pig-tail drainage of collection did not cure the patient. The patient required

Fig. 8 Gall bladder with multiple calculi

exploratory laparotomy where subcapsular gallbladder perforation at the tip of the fundus was seen. Cholecystectomy with peritoneal lavage and abdominal drainage was performed as definitive procedure.

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