

Rare Case of Gall Bladder Perforation following acute Calculus Cholecystitis in a Elderly Male: A Case Report

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How to cite this article:

Dipanjana Pradhan, Rohit Basu, Saurav Karmakar/Rare Case of Gall Bladder Perforation Following Acute Calculus Cholecystitis in a Elderly Male: A Case Report/New Indian J Surg. 2022;13(4): 167-170.

Abstract

Perforation of the gallbladder is a rare but dreaded complication following cholecystitis.¹ Spontaneous perforation is most commonly seen in the elderly, and in patients suffering from diabetes mellitus.² The most common site of perforation is the fundus of gall bladder.³ The gall bladder distension and edema associated with acute inflammation leads to venous and arterial obstruction. Subsequently, this leads to ischaemia and necrosis of the gall bladder wall. The fundus is most susceptible to this injury due to maximum distance from the cystic arterial supply. Aggravating factors include atherosclerosis, focal vasospasm and localized vasculitis. If diagnosed and treated late, perforation of gallbladder may be associated with high chances of morbidity and mortality.⁴⁻⁶ Here, we report a case of a 66-year-old male with perforation of gallbladder following acute calculous cholecystitis.

Key Words: Gall bladder perforation; Pericholecystic abscess; Perforation.

Introduction

The commonest cause of gallbladder perforation is acute cholecystitis.⁷ The incidence⁸ of gallbladder perforation in acute cholecystitis is 2-18%. Neimeier classified gall bladder perforation into 3 clinical subtypes⁹ acute free perforation, subacute pericholecystic abscess, and chronic cholecystoenteric fistula. Gangrenous cholecystitis¹⁰ or gall bladder perforation is suspected when the patient remains toxic and febrile even after conservative management for acute cholecystitis, and it advocates further investigations. CT scan is the modality of

choice⁷ to diagnose gall bladder perforation. Since CT scan is not routinely indicated in acute cholecystitis, diagnosis is often delayed, leading to higher chances of mortality and morbidity.

Case Report

A 66 year old male presented with complaints of right upper quadrant abdominal pain for 1 day, which was sudden in onset, gradually progressed to reach a steady level and persisted for more than 12 hours, with radiation to the back. It was not relieved by antispasmodics and proton pump inhibitors. It was associated with 3 episodes of vomiting. The patient gave history of yellowish discoloration of eyes and urine about 1 month back, which got relieved spontaneously. He was a known case of type-II diabetes mellitus, on oral medications.

His serum total bilirubin level was 2.7 mg/dl and alkaline phosphatase was 239 IU/L.

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Received on: 16.06.2022 **Accepted on:** 18.07.2022

Ultrasonography of the abdomen showed multiple calculi in the gall bladder lumen, with symmetrical wall thickening (maximum wall thickness of 6.0 mm), and pericholecystic fluid collection.

MRCP revealed a distended gall bladder with multiple 8-9 mm filling defects in its lumen, suggestive of calculi. GB wall thickness was around 17 mm. The neck of gall bladder compressed the common hepatic duct, causing mild upstream biliary tract dilatation. Overall the

features were suggestive of cholelithiasis with xanthogranulomatous cholecystitis.

Contrast enhanced CT scan of the abdomen revealed a distended gall bladder with diffuse symmetrical wall thickening (maximum wall thickness of 15.6 mm). Focal areas of perforation of gall bladder was noted in the body and fundus with pericholecystic collection and adhesions in the pericholecystic region with the gastric pylorus, first two parts of duodenum, and hepatic flexure of colon (Fig. 1).

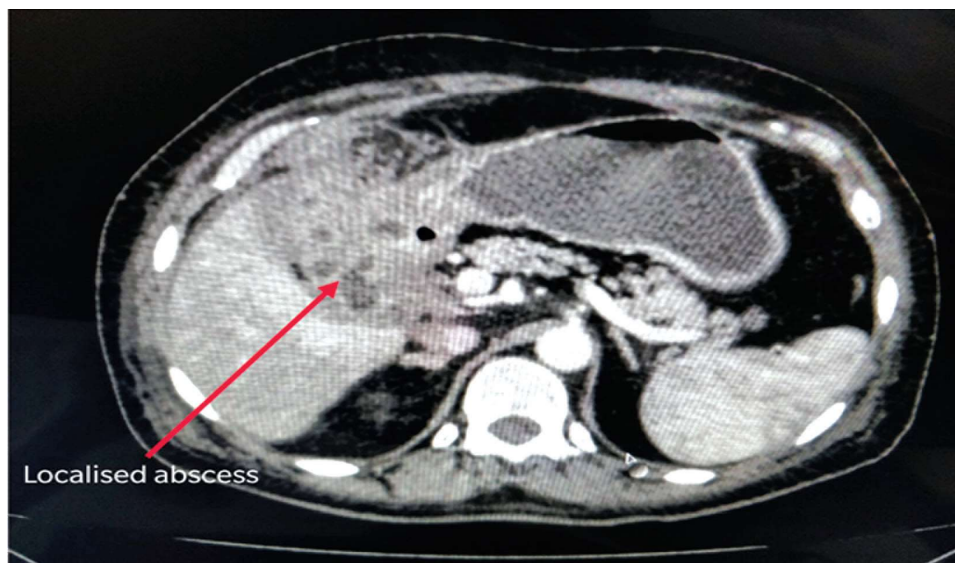


Fig. 1: Localized pericholecystic abscess (red arrow) following spontaneous rupture of gall bladder at the fundus

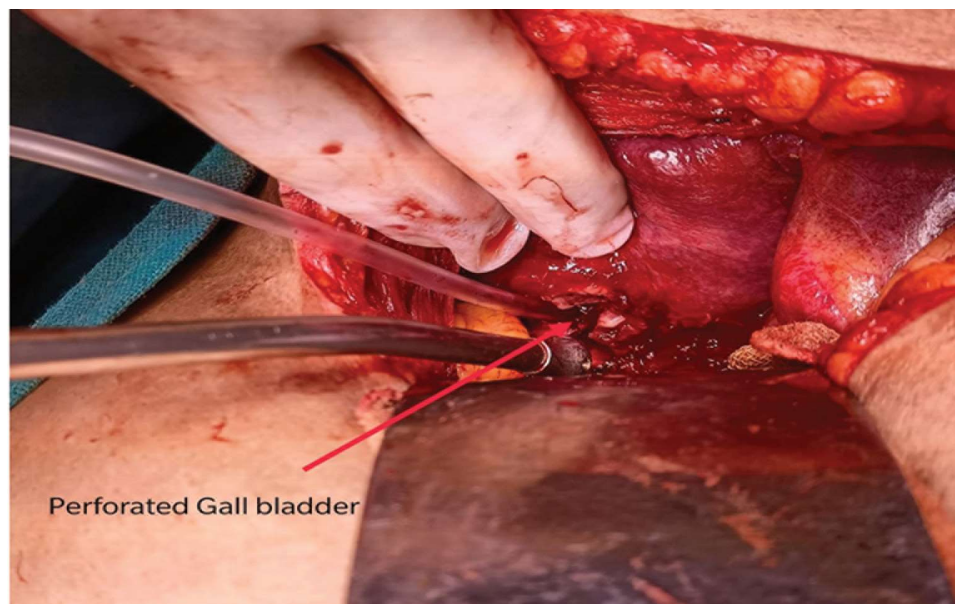


Fig. 2: Spontaneously perforated fundus of gall bladder (marked by the red arrow). The biopsy of the specimen identified it as a specimen of acute calculous cholecystitis with focal area of perforation at the fundus.

A provisional diagnosis of gall bladder perforation, as a sequelae of acute calculous cholecystitis, was made, and the patient was posted for open cholecystectomy after optimization with conservative treatment using iv fluid and broad spectrum antibiotics. There were extensive adhesions in the pericholecystic region, involving the gastric pylorus, duodenum, and hepatic flexure of colon. After adhesiolysis, a perforation was found in the fundus of the gall bladder (Fig. 2). No gallstones were found in the peritoneal cavity. After cholecystectomy, the bowel was examined for any iatrogenic injury. Post-operative period was uneventful and patient was discharged on 7th post-operative day.

Discussions

Gallbladder perforation is a rare but life-threatening event.³ Based on Neimeier classification, Roslyn et al⁴ reported in his study that type 1 and type 2 gallbladder perforations are mostly seen in young patients (<50 years), and type 3 is seen in elderly with long history of stone disease. Our patient had a type 2 gallbladder perforation. The most possible mechanism of gallbladder perforation in acute cholecystitis is cystic duct obstruction (mostly by a stone at the neck) that raises the intraluminal pressure, thereby compromising the venous and lymphatic drainage of gallbladder resulting in necrosis and finally perforation.¹³ As gallbladder perforation occurs most commonly at fundus due to least blood supply (60%)³, this proves the role of ischemia. Conditions such as cholelithiasis, malignancy, diabetes, atherosclerosis, steroid therapy, etc. are all risk factors for gallbladder perforation.¹⁴

Clinical diagnosis of spontaneous gallbladder perforation is very difficult and often delayed or missed because there are no classical symptoms and signs of gall bladder perforation. A sudden decrease in pain intensity caused by the relief of high intracholecystic pressure might herald the perforation.¹⁵ Perforation and abscess formation should be suspected in those patients of acute cholecystitis who suddenly become toxic and deteriorates clinically.¹⁶ Ultrasonography is the initial radiological investigation¹² done, but findings are very non-specific for gallbladder perforation and mimic those seen in acute uncomplicated cholecystitis such as gallbladder

distention, gallbladder wall thickening (> 3 mm), pericholecystic fluid collection, gallstones, and bile duct dilatation. Distention of the gallbladder and edema of its wall may be the earliest detectable signs of imminent perforation.¹² Computed tomography (CT) scan is the most sensitive tool to diagnose gallbladder perforation¹¹. CT scan finding can be divided into primary gallbladder changes, and pericholecystic changes. Primary gallbladder changes include wall thickening, wall enhancement, wall defect, intramural abscess, intramural gas, mural hemorrhage, presence of gallstones, bile duct stones or cystic duct stones, and intraluminal gas. Pericholecystic changes include pericholecystic fat stranding, pericholecystic fluid collection, abscess or biloma formation and presence of extra-luminal stones. The direct signs of gallbladder perforation include demonstration of either calculi outside the gallbladder or a ruptured segment of the gallbladder wall, whereas indirect indicators include the presence of an abscess outside the gallbladder and the presence of gallstones together with thickening of gallbladder wall.¹⁷

Magnetic resonance imaging (MRI) may demonstrate the wall of the gallbladder and defects also. The most specific is the hole sign on MRI.⁸ The biliary tree is better demonstrated by MRCP than other modalities in presence of gallbladder perforation in suspected cases of acute cholecystitis.⁸ If USG and CT scans are inconclusive, the MRI is the modality of choice.

Conclusion

In conclusion, to decrease the morbidity and mortality associated with gallbladder perforation, early diagnosis and surgical intervention are of prime importance. Presence of risk factors certainly warrants a vigilant approach. Gallbladder perforation should be considered in differential diagnosis in elderly patients presenting with peritonitis with an unknown etiology as was in this patient.

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