Abstract
Introduction
Gall bladder perforation is a rare but life threatening complication of acute cholecystitis with or without stones and is associated with increased morbidity and mortality due to late diagnosis. The late diagnosis is attributed to the fact that most of them are present with the same symptoms as in an uncomplicated acute cholecystitis. Most of them are identified and confirmed by laparotomy as preoperative diagnosis is very rare, that is, on computed tomography scan and ultrasound. We are reporting two cases of gall bladder perforation.
Case report
The first case was a 70-year-old man who was presented to the emergency department with complaints of pain in the abdomen for 7 days and obstipation for 3 days. The second case was a 58-year-old man who was presented to the emergency department with complaints of pain in the abdomen for 1 week and obstipation for 2 days.
Conclusion
Gall bladder perforation is a fatal and life threatening complication of acute cholecystitis so early diagnosis is the key, as delay will result in the increase of mortality and morbidity.

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Case Report 1
A 70-year-old man was presented to the emergency department with complaints of pain in the abdomen for 7 days and obstipation for 3 days. There were no history of nausea, vomiting, diarrhoea or burning micturition, gall bladder disease, NSAIDs or other medication and no other comorbidity and no family history of gall stones and malignancy. He was a hookah smoker for the past 30 years. Patient was vitally stable and was afebrile to touch. Abdomen was distended with generalised guarding and tenderness. In chest bilateral air entry was reduced in the basal zone. Laboratory investigations revealed haemoglobin, 10.8%; total leucocyte count, 14,000 cells/mm³; serum glucose, 96 mg%; serum amylase, 36 U/dl; lipase, 40 U/dl and serum creatinine, 1.6. APACHE-II score of patient in preoperative period was 11. Patient was urgently shifted to the emergency operating room.
Operative diagnosis was gall bladder perforation (TYPE-I), and emergency cholecystectomy was done. The operative findings were 1 l of biliary contamination, perforation noted at Hartmann’s pouch and multiple stones in lumen with the largest at Hartmann’s pouch (Figures 1, 2 and 3).
Due to sepsis and low saturation, patient was shifted to the ICU. Patient deteriorated in postoperative days and expired on POD-13.

Figure 1: Perforation in gall bladder at Hartman’s pouch.

Figure 2: Perforation in gall bladder at Hartman’s pouch.

Figure 2: Ligated cystic duct stump after cholecystectomy.
Case Report 2
A 58-year-old man was presented to the emergency department with complaints of pain in the abdomen for 1 week and obstipation for 2 days. History of weight loss (not documented) was present although he denied vomiting, burning micturition, jaundice, loose stools, constipation, bleeding per rectum and intake of any medication. No history of any comorbidity, no similar episodes in the past and no major surgery earlier were reported. On abdominal examination, generalised tenderness and guarding were present; liver dullness was obliterated, and bowel sounds were absent. On chest examination, bilateral air entry was absent in basal zones. Patient was in septicemic shock but was conscious, oriented resuscitated and shifted to the OR. His laboratory parameters were normal except the kidney function test. Ultrasound abdomen showed well-defined iso- to hyper-echoic lesion of size 53 × 51 mm with central cystic part (necrosis) in the right lobe of the liver with thickening of bowel loops in the right hypochondriac region, inflamed mesentery and septate collection. Gall bladder was not visualised, and CBD was dilated (11 mm).

Operative diagnosis was gall bladder and caecal perforation peritonitis with gall bladder fossa growth. Exploratory laparotomy with quadrictectomy with end ileostomy and ascending colon mucus fistula with tube cholecystostomy was done. Later biopsy from the gall bladder perforation showed muciin secretting adenocarcinoma. Postoperative period was uneventful, and patient was discharged after 1 week.

Discussion
Acute uncomplicated cholecystitis is more commonly seen in females, but gall bladder perforation is more common in males. Out of all the patients with cholelithiasis, around 10% have asymptomatic cholelithiasis and 2%–11% of asymptomatic group present gall bladder perforation. The mortality rate ranges from 12% to 16% in the perforation group. They usually present in 1 or 2 days and in some cases a few weeks after the onset of symptoms. Gall bladder perforation is mostly seen in stone disease and very rarely involving the acalculus gall bladder although the patients with acute acalculus cholecystitis are more susceptible for perforation as they were mostly seen in patients with the above-described risk factors.

Perforation results from the cystic duct occlusion (mostly by a stone at neck) that causes retention of intraluminal secretions ultimately resulting in the rise of intraluminal pressure. This raised intraluminal pressure leading to compromise venous and lymphatic drainage resulting in necrosis and finally gall bladder perforation. Perforation is most commonly seen at the fundus after the onset of acute cholecystitis, as it is the most distal part of the bladder and has the least blood supply. Fundus is not usually covered by omentum after perforation resulting in free biliary peritonitis and stones in the peritoneal cavity (TYPE-I) and rarely results in cholecystoenteric fistula (TYPE-III) most commonly involving the transverse colon. Perforation occurring in the gall bladder other than fundus is usually sealed by omentum or intestine, and the condition remains confined to the right hypochondrium. Roslyn et al. reported in their study that TYPE-I and -II gall bladder perforations are mostly seen in younger patients (<50 years), and TYPE-III is seen in elderly with a long history of stone disease.

As there are no specific symptoms and signs of gall bladder perforation, it is hard to differentiate clinically between gall bladder perforation and uncomplicated acute cholecystitis. According to Chen et al., a sudden relief in the abdomen pain was observed after the release of intra-cholecystic pressure due to perforation.

Gore et al. suggested that gall bladder perforation should be suspected in patients of acute cholecystitis who suddenly deteriorated and become toxic.

Tsai et al. proposed that gall bladder perforation should be considered in patients >70 years of age and high neutrophil count (80%).

Ultrasoundographic findings are very non-specific for gall bladder perforation and are also seen in acute uncomplicated cholecystitis such as gall bladder distension (largest diameter >3.5–4 cm), gall bladder wall thickening (>3 mm), pericholecystic fluid collection, coarse intracholecystic echogenic debris, bile duct dilatation, and positive sonographic Murphy sign. The ‘hole sign’, which is a defect seen in the gall bladder, is the only reliable and specific sign of gall bladder perforation. Intrahepatic perforation can present as liver abscess and confirmed on ultrasound.

Soiva et al. showed in their study that distension of gall bladder and oedema of its wall may be the earliest signs of impending perforation.

Ultrasoundography is the initial radiological investigation done in most of the cases, but it has some limitations in suspected cases of gall bladder perforation due to gaseous distension of bowel and pain; the sonography is compromised and unable to locate the perforation.

Computed tomography (CT) scan is the most sensitive tool to diagnose gall bladder perforation, but in the cases of suspected biliary pathology like missed stones, sonography can be done after the scan. CT scan findings can be divided into primary gall bladder changes, pericholecystic changes and findings of extra-gall bladder organs. Primary gall bladder changes include wall thickening, wall enhancement, wall defect, intramural abscess, intramural gas, mural haemorrhage, presence of gallstones, common bile duct stones or cystic...
duct stones, intraluminal membrane and intraluminal gas. Pericholecystic changes include pericholecystic fat stranding, pericholecystic fluid collection, pericholecystic abscess or biloma formation and presence of extraluminal stones. Findings in organs other than the gall bladder consist of pericholecystic liver enhancement, liver abscesses, portal vein thrombosis, reactive mural thickening of adjacent hollow organs (hepatic flexure of colon and duodenum), presence of lymph nodes, intraperitoneal free air, ascites, ileus and Mirizzi syndrome. The gall bladder perforation signs can be divided into direct and indirect signs: the demonstrations of either calculi outside the gall bladder or a ruptured segment of the gall bladder wall are direct indicators according to Pedrosa et al. Indirect indicators include the presence of an abscess outside the gall bladder and the presence of gall-stones together with thickening of the gall bladder wall.

Kim et al. in their study by comparing the sensitivity of CT and ultrasound in detecting the perforation found out that in 50% of patients, site of perforation was seen on CT but not a single perforation was identified on sonography.

Magnetic resonance imaging (MRI) demonstrates the wall of the gall bladder and defects, also MR colangiopancreatography images demonstrate the biliary tree better than other modalities. Showed if gall bladder perforation is suspected in acute cholecystitis and USG and CT scan are not conclusive, the MRI is the modality of choice. However, cost is the limiting factor.

Cholecystectomy (open or laparoscopic) with drainage of abscess if present and abdominal lavage are more than sufficient in almost all the cases of gall bladder perforation except in TYPE-III perforation where an additional repair of the fistula is also required.

**Conclusion**

Gall bladder perforation is a fatal and life-threatening complication of acute cholecystitis so early diagnosis is the key as delay will result in the increase of mortality and morbidity. Early surgical intervention as soon as the diagnosis is made either clinically or by using diagnostic tools such as ultrasound and CT scan.

**Consent**

Written informed consent was obtained from the patients for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

**References**