

Case Report

Case series of spontaneous gall bladder perforation and review of literature

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ABSTRACT

Gall bladder perforation in acalculus cholecystitis is a rare condition and has a high mortality rate. A diagnosis before surgery is often difficult. We present two cases of gall bladder perforation encountered at an initial presentation without any clear radiological evidence. A 45 year old male patient, presented with chief complaints of diffuse abdominal pain since 1 day. No other complaints/symptoms were found on a detailed history. Exploratory Laparotomy of this patient, revealed a ruptured gall bladder with necrotic patches and approximately 3 litres of bilious fluid in the peritoneal cavity. Another 45 year old Female, known diabetic, hypertensive and asthmatic presented with diffuse abdominal pain. On laparotomy 2.5 litres of biloma with necrotic gall bladder was observed. Owing to rarity of gall bladder perforation, the condition is often misdiagnosed. Early diagnosis and immediate surgical intervention are the gold standard for decreasing the morbidity and mortality associated with perforation.

Keywords: Spontaneous perforation, Gallbladder, Acalculous cholecystitis

INTRODUCTION

Gall bladder rupture, usually in association with gall stones, is a well-known entity. However, a free perforation into the peritoneal cavity without associated gall stones is distinctly uncommon. Gall bladder perforation in acalculus cholecystitis is a rare condition; it has a high mortality rate of 10-30%.¹⁻³ We report a case of Biliary Peritonitis with ruptured gall bladder without cholelithiasis, the diagnosis being confirmed only in the operation room. Spontaneous gall bladder perforation is a rare condition and may be sequelae to acute cholecystitis.⁴⁻⁶ If left untreated, it is associated with a high mortality rate.^{7,8} The perforated gall bladder syndrome may occur in immunocompromised patients and others with numerous systemic diseases, namely generalised atherosclerosis, undernutrition and chronic

cardio pulmonary and renal disease. In addition, metabolic deficiency syndromes such as obesity, diabetes mellitus and collagen diseases result in numerous problems for these seriously ill patients.³

CASE REPORT

Case 1

A 45 year old male patient presented in the OPD with diffuse abdominal pain since 1 day. Patient was a known case of hypertension since 1 year, not on any treatment. He was non-diabetic and there was not any other chronic illness. Patient is a known case of hiatus hernia with prolapsed gastropathy. He also gives history of a perforated appendix for which an exploratory laparotomy was done 15 years prior to present admission. A

ureteroscopic stone retrieval was done a few months prior to present admission. There were no symptoms suggestive of any gall bladder disease.

General physical examination was unremarkable. On systemic examination, abdomen was found to be distended, diffuse tenderness being present over whole abdomen with guarding and rigidity. No audible bowel sounds. Rest of the systemic examination was unremarkable. Patient was advised admission with a working diagnosis of peritonitis with paralytic Ileus.

All routine investigations were sent, which revealed total leucocytic count of 7000/cumm, random blood sugar of 88 mg/dl, blood urea of 90 mg/dl, serum creatinine of 1.4 mg/dl, ALP of 744 IU/l, serum amylase of 213 IU/l, direct bilirubin 2.3mg/dl and total bilirubin 3.2 mg/dl. This gave a picture of obstructive jaundice. An urgent computerised tomography of abdomen revealed focal pancreatic lesion with mildly dilated main pancreatic duct and marked ascites (Figure 1a, 1b, 1c). To further evaluate the bilio pancreatic tree, an urgent MRCP was ordered which revealed moderate ascitis with 2 irregular cystic areas within the pancreas with calcification within one of them suggests a possibility of chronic pancreatitis (Figure 2). A diagnostic ascitic tap was done which aspirated bile stained fluid. On laparotomy, approximately 3 litres of bilious fluid (Figure 3) was drained from the abdominal cavity with a ruptured gall bladder with necrotic patches. A cholecystectomy was done following the drainage of the bilious peritoneal fluid. Thorough peritoneal lavage was given, and the incision was closed along with a suction drain in situ. The gall bladder specimen with perforation (Figure 4) was sent for histopathological examination which revealed no evidence of cholelithiasis with acute on chronic cholecystitis with perforation. During the post-operative period, condition of patient remained uneventful. The patient was discharged on the 7th post-operative day in a stable condition. Patient was followed up for 3 months post operatively, and found in a fair state.



Figure 1a: CT scan showing gross ascites.

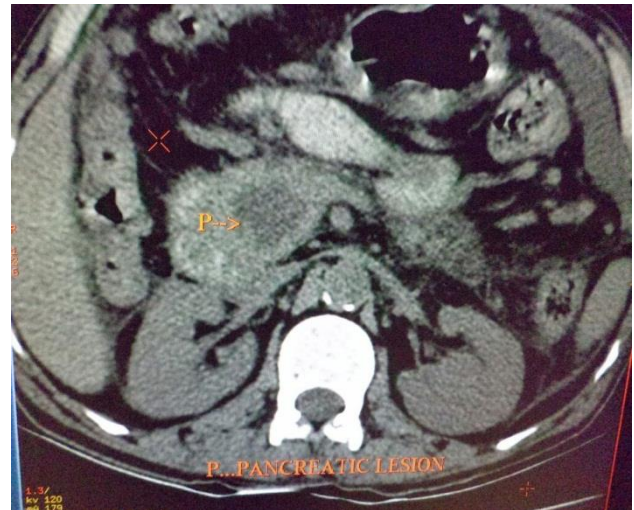


Figure 1b: CT scan suggestive of hypodense lesion of pancreas.

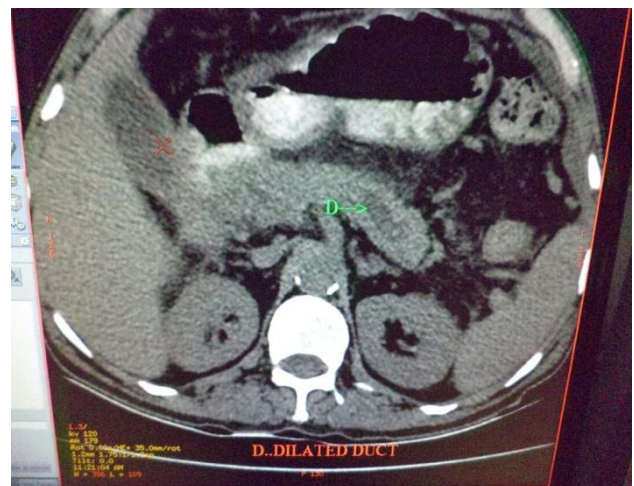


Figure 1c: CT scan suggestive of dilated pancreatic duct.



Figure 2: Magnetic resonance cholangiopancreatic image showing irregular cystic lesions with calcifications in pancreas.

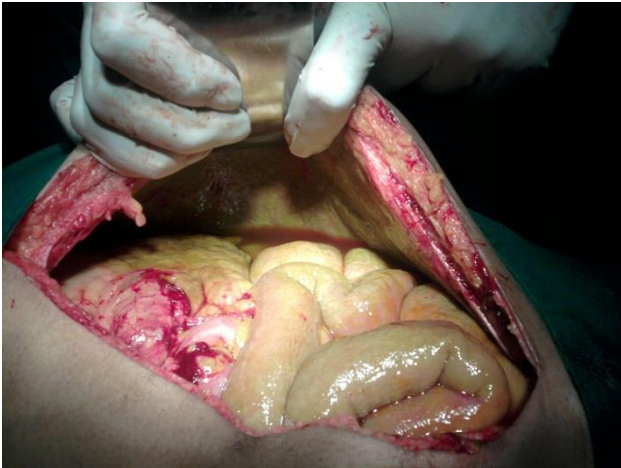


Figure 3: Laparotomy case one with intraperitoneal bilious fluid.

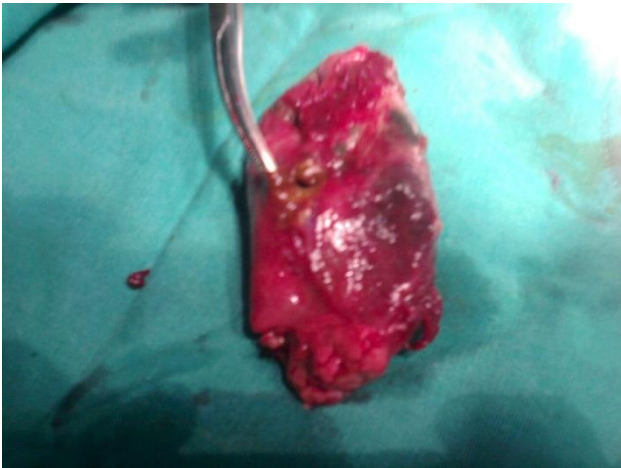


Figure 4: Cholecystectomy specimen case one with gall bladder fundus perforation.

Case 2

A 45 year old female patient known case of diabetes mellitus, hypertension, and asthma presented in emergency department with the complaints of diffuse abdominal pain and vomiting since 2-3 days. Pain in dull aching and is referring to back. Patient was also complaining of poor oral intake and not able to pass stools since 2 days. On examination, abdomen was tense, distended, generalised tenderness of abdomen with guarding, free fluid in abdominal cavity and absent bowel sounds. Patient was admitted with provisional diagnosis of acute pancreatitis with peritonitis. Her serum amylase was 12900 IU/L and lipase was 7630 IU/L, total leucocyte count was 17900/cumm. Ultrasound guided aspiration reveals clear bile in peritoneal cavity. And urgent MRCP was done which was suggestive of gross ascites with no evidence of pancreatitis (film not available). On laparotomy, around 2.5 litres of bilious peritoneal fluid (Figure 5), extensive fat saponification, gangrenous fundus of gall bladder, mildly oedematous pancreas, normal CBD and normal intestine were found

(Figure 6). Thorough peritoneal lavage with cholecystectomy was performed. Patient was managed in High Dependency Unit (HDU) for 2 days and then in surgical ward. Gall bladder specimen with perforation and gangrene (Figure 7) was sent for histopathological examination which was suggestive of chronic cholecystitis with perforation with transmural necrosis. Her post-operative period was uneventful. She was discharged on 7th post-operative day and was followed up for 3 months, and found in a fair state of health.



Figure 5: Laparotomy case two with intraperitoneal bilious fluid.



Figure 6: Intraoperative picture showing fat saponification and gall bladder perforation.



Figure 7: Cholecystectomy specimen case two showing gall bladder fundus gangrene and perforation.

DISCUSSION

Gall bladder perforation was 1st reported by Duncan in 1844. Several authors have reported mortality of 15-30% and significant morbidity.^{2,3} Most commonly the site of ruptured gall bladder is the fundus as it was in this case, presumably owing to its poor supply, as it is the most distal part of the gall bladder.⁹⁻¹⁵ Perforation at the fundus is less likely to be covered by the omentum, thus bile spills into the entire peritoneal cavity, instead of localised peritonitis (which would more likely be seen with a neck/body perforation), thus resulting in type i perforation. Inflammation after the cholecystitis may progress. Inflammation after the cholecystitis may progress and cause ischaemia and necrosis, resulting in a ruptured gall bladder.¹⁰⁻¹² Gall bladder perforation also develops following acalculous cholecystitis, as seen in this case, although rare.^{13,14} Lein HH & Huang CS¹⁶ suggested that gall bladder rupture is more common among male, although incidence of cholecystitis is more prevalent among females. Niemeir in 1934 reported 1 case and 1st classified this entity in 3 types, type I being chronic perforation with fistula formation between gall bladder and another viscus, type II being subacute perforation of gall bladder with percholecystic abscess and type III being acute perforation with generalised peritonitis.¹⁷ Fletcher & Ravdin¹⁸ in 1951 and Roslyn & Busutti¹⁹ in 1979 described type i perforation as acute with generalised peritonitis, type ii as pericystic abscess, type ii as chronic perforation with cholecysto enteric or cutaneous fistula. The modification in this classification includes type iv for chronic perforation with

cholecystobiliary fistula formation. Acute gall bladder perforation is infrequent, although not an uncommon complication of cholecystitis with cholelithiasis. It is rarely diagnosed pre operatively, although with high index of suspicion, leading to certain diagnostic procedure pre operatively. The preoperative diagnosis can be confirmed intra operatively. Delay in making the definitive diagnosis usually accounts for increased incidence of morbidity and mortality associated with this complication.^{4,5} Unlike most studies, in which the patients are generally aged 60 or more, this case was a 40 year old male.^{19,20} Gall bladder rupture may occur early in the course of cholecystitis or may it may occur late.^{21,22} Roslyn & Busutte suggested that spontaneous gall bladder perforation is caused by hypoperfusion of viscera secondary to systemic disease. Other hypothesis includes trauma, congenital abnormality, infection, pancreatic secretion, obstruction by calculi, malignancy, steroid therapy, abnormal bile or diabetes mellitus.^{1,21} In chronic diseases, Bile stasis triggers release of inflammatory enzymes (e.g., phospholipase A, which converts lecithin to lysolecithin, which then may mediate inflammation). The damaged mucosa secretes more fluid into the gallbladder lumen than it absorbs. The resulting distension further releases inflammatory mediators (e.g., prostaglandins), worsening mucosal damage and causing ischemia, all of which precipitates the gall bladder perforation. According to the causes of the gall bladder perforation Estevao-Costa J²³ proposes a classification as follows:

I. Spontaneous:

- a. Idiopathic
- b. Secondary:
 - i. Lithiasis
 - ii. Inflammation/infection (predisposing factor - diabetes, atherosclerosis, malignant, pregnancy)
 - iii. Other (congenital obstruction, salmonella typhi, anticoagulants)

II. Traumatic:

- c. Penetrating
- d. blunt

III. Iatrogenic:

This case is unusual, as the age does not fit into a usual clinical picture. In addition, there was no radiological feature suggestive of cholelithiasis and histopathological examination revealed an acute on chronic cholecystitis without cholelithiasis. This proved unusual, as the patient had no prior symptom of any gall bladder disease, such as pain upper abdomen, vomiting or dyspepsia. This suggests that the previous cholecystitis was silent. Another

fact to note in 1st case is that, the initial presentation of the disease was gall bladder rupture. In today's modern day and age, with extremely accurate diagnosis and radio imaging studies available, a presentation of a gall bladder rupture is extremely rare.

CONCLUSION

Owing to rarity of gall bladder perforation, the condition is often misdiagnosed. Early diagnosis and immediate surgical intervention are the gold standard for decreasing the morbidity and mortality associated with perforation.

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