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Acute gall bladder perforation: case series over three years

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ABSTRACT

Background: Acute perforation of gall bladder is a life threatening condition. It is a complication of acute cholecystitis. This is not an uncommon condition and resembles acute cholecystitis in its presentation. If there is delay in diagnosis and management there is high morbidity and mortality. It should be diagnosed as early as possible for better prognosis.

Methods: This is a retrospective study wherein data of cholelithiasis, acute and chronic cholecystitis and perforated gall bladder from our hospital for the last three years 2014-2016 was collected. The clinical presentation, investigations routine and radiological, surgical and medical management was analysed.

Results: Total numbers of patients with chronic cholelithiasis were 3534, patients with acute cholecystitis were 133, and numbers of patients with gall bladder perforations were 22, making it 0.6% of gall bladder disease patients and 16.5% among patients with acute cholecystitis. TLC was invariably raised in all the cases. 12 out of 22 cases were managed conservatively while others underwent surgery. There was no mortality in the series.

Conclusions: Gall bladder perforation occurs in about two weeks or several weeks after episode of acute cholecystitis, the incidence of occurrence of perforation increases to four folds if there is delay in proper management of acute cholecystitis more than two days after the onset of symptoms. Clinical presentation varies from an acute generalized peritonitis to nonspecific abdominal symptoms and thus requires prompt diagnosis for better prognosis.

Keywords: Acute cholecystitis, Cholelithiasis, Gall bladder perforation

INTRODUCTION

Acute perforation of gall bladder is a life threatening condition. It is a complication of acute cholecystitis. This is not an uncommon condition and resembles acute cholecystitis in its presentation. If there is delay in diagnosis and management there is high morbidity and mortality. It should be diagnosed as early as possible for better prognosis. In acutely ill patient with severe signs and symptoms of pain in right hypochondrium, tenderness, guarding, rigidity, fever and other manifestation of toxemia, the possibility of gall bladder perforation should always be kept in mind. It is the high degree of suspicion which will be very helpful for

accurate diagnosis and investigating the patient accordingly.

In 1934 Neimier presented his classic description of the gall bladder perforation and proposed a classification .He concluded that this condition demands eternal vigilance for prompt recognition and treatment to lower the mortality. This classification still persists and seems to be complete and explanatory. Roslyn et al reviewed the risk factors for gall bladder perforation. He found that in older patients of gall bladder perforation there are other disease. associated co-morbidities like heart atherosclerosis and diabetes. In these patients there may be compromised blood supply of all the organs of the body including gastrointestinal tract. This hampered blood supply is an important factor for gall bladder perforation. In younger patients immunosuppression and other debilitating conditions were major factors because there was inability to combat infection which led to perforation of gall bladder.²

Relevant anatomy of gall bladder

The gall bladder is divided in four anatomic areas: the fundus, the body, the infundibulum and the neck .After the neck there is cystic duct. The fundus contains most of the smooth muscle of the organ in contrast to body. The body contains most of the elastic tissue and this is the main storage area of gall bladder. Sometimes gall bladder has complete covering of peritoneum and then it is suspended to the liver surface with a mesentery. But most of the time, the peritoneum covers the fundus and the inferior surface of gall bladder continuous with the liver. Sometimes gall bladder is embedded in the liver parenchyma. The gall bladder is lined by a single, highly folded, tall columner epithelium containing cholesterol and fat globules. These tubuloalveolar glands secreting the mucus are present in the infundibulum and neck but absent in the body of the gall bladder. The inner epithelial lining is supported by lamina propria. The muscle layer of gallbladder has longitudinal and oblique fibres but it lacks well developed muscle layers. The perimuscular subserosa contains connective tissue, nerves, vessels, lymphatics and adipocytes. The gall bladder lacks a muscularis mucosa and submucosa so it differs from the rest of the gastrointestinal tract in microscopic anatomy.³

The cystic artery is a branch of right hepatic artery in 90% of cases. It is an end artery with essentially no collateral circulation. Upon reaching the neck of gall bladder it divides into anterior and posterior branches. Venous return is carried either directly to the liver or rarely to a large cystic vein that carries blood to portal vein. Gall blabber lymphatics drain into nodes at the neck of gall bladder. Most of the time a visible lymph node overlies the insertion of cystic artery to the gall bladder. It is named as lymph node of Lund. Nerves of gall bladder arise from vagus and sympathetic branches from celiac plexus.

The length of cystic duct is quite variable. It may be short or absent and there may be a high union of it with the common hepatic duct or it may run parallel, behind or spirally to the common hepatic duct before joining it; or it may run parallel to join it near the duodenum. The neck part of the cystic duct has mucosal folds called the valves of Heister. They do not have any valvular function as described in the literature. The diameter of common bile duct may be 5 to 10 mm. The union of common bile duct and main pancreatic duct follows three patterns. In about 70% of people these ducts unite outside the duodenal wall and traverse the duodenal canal as a single duct. In about 20%, they join within the duodenal wall and have a short or no common duct, but open through the same opening in the duodenum. In about 10% subjects they

open through the separate openings. The sphincter of Oddi surrounds the common bile duct at the ampulla of Vater in the wall of duodenum.⁶

In fasting state approximately 80% of bile secreted by liver is stored in gall bladder. The gall bladder mucosa has the greatest absorptive power per unit area of any other organ in the body. It rapidly absorb sodium chloride and water against significant concentration gradient, thus concentrating the bile as much 10 folds and so there is marked change in bile composition. This rapid absorption of water and salts prevents the rise in pressure in the biliary system. The mucosal glands in the infundibulum and neck of the gall bladder secrets mucus glycoprotein and this mucus protect the mucosa from the lytic action of bile and facilitates smooth passage of bile through the cystic duct.⁶

Pathogenesis of acute cholecystitis and gall bladder perforation

In 90% to 95% cases the acute cholecystitis is due to gall stone blocking the cystic duct. In 1% of cases of acute cholecystitis the cause is a tumour at the neck of gall bladder causing obstruction. Acute cholecystitis initially is an inflammatory process probably mediated by the mucosal toxin lysolethicin, a product of lecithin. Bile salts and platelets activating factor contribute to this process. There is increase in prostaglandin synthesis which amplifies the inflammatory process. There also occurs bacterial contamination caused by stasis of bile in the gall bladder due to obstruction of cystic duct by a stone or a tumour.

The gall bladder wall becomes grossly thickened and reddish with subserosl heaemorrage. Pericholecystic fluids also get accumulated around the gall bladder. Mucosa becomes hyperaemic with patchy necrosis. In about 5% to 10% cases, the inflammatory process leads to ischemia and necrosis of the gall bladder wall. This may lead to perforation of gall bladder wall. But most of the time the obstruction is relieved by dislodging of the gall stone and this resolves the inflammatory process.⁸

But when gall bladder remains blocked the secondary bacterial infection supervenes leading to acute gangrenous cholecystitis, empyma or perforation. The perforation usually remains in the subhepatic space confined by omentum or adjacent organs. But free perforation in the peritoneal cavity with peritonitis, intrahepatic abscesses due to perforation in liver, in adjacent organ like duodenum or colon can occur. With secondary bacterial infection gas may be seen in gall bladder lumen or wall of the gall bladder. This gas can be seen by X- ray abdomen or in computerized tomography8 scan.

Acute acalculous cholecystis also occurs without a stone. It develops in critically ill patients, patients with severe sepsis, extensive burns, after major operations, multiple

traumas or in patients with multi organ failure. Gall bladder distension with bile stasis causing ischemia has been implicated in these cases. There is again obstruction due to biliary sludge, viscous bile and gall bladder mucus leading to cystic duct obstruction in the absence of frank stone formation.¹⁰

There is no morphologic difference between acute acalculous or calculous cholecystitis 10 except for the absence of macroscopic stones in the acalculous cholecystitis.

Diagnosis of perforation of gall bladder

USG findings of gall bladder perforation are very specific; the `hole sign' showing the defect in perforated gall bladder, wall thickening, distension, pericholecystic fluid and positive Murphy' sign. ¹⁵

Soiva et al showed that edematous and distended gall bladder may be the early sign of impending perforation Kim et al showed that both the modalities USG and CT demonstrated pericholecystic fluid collection, gall bladder wall thickening and cholelithiasis. ^{16,17}

Sood et al showed that CT is little better than USG in the detection of gall bladder defect.¹⁸ The choice of management is only early exploration as delay increases morbidity and mortality. Early diagnosis and immediate surgical intervention is the mainstay treatment for survival. High index of suspicion is required for early management to improve the morbidity and mortality.

METHODS

Study collected the data of cholelithiasis, acute and chronic cholecystitis and perforated gall bladder from our hospital for the last three years 2014-2016. The clinical presentation, investigations routine and radiological, surgical and medical management was analysed. The data is as follows:

Table 1: Total no. of cholelithiasis patients during 2014-2016.

Data	Total number
Chronic cholecystitis (cholelithiasis)	3534
Acute cholecystitis	133
Gall bladder perforation	22
% of gall bladder perforation	0.6
% of gall bladder perforation among acute cholecystitis	16.5

This is a significant finding. It may be due to being a referral hospital. All the serious cases from periphery are sent to this hospital.

Management

All the patients were treated with IV fluids, antibiotics, nasogastric aspiration at the outset. Out of 22 patients, 12 patients were treated conservatively as these patients presented with a gap of 4-7days after developing pain in abdomen. The thought behind this approach was that there would be very much adhesion and the visceral contents of abdomen would be a phlegmon mass and nothing could be recognizable. The operative attempt might have created increased morbidity and mortality as these patients were compromised haemodynamically and nutritionally.

The remaining 10 patients were treated surgically as per standard protocol. All the 22 patients fared well in our hospital and were discharged in satisfactory condition.

No follow up of the non-operated patients was available in the records.

RESULTS

The following is the observation in our study in our hospital.

Table 2: Total no. of chronic cholelithiasis in last three years.

Years	Total no. chronic cholelithiasis
2014	1413
2015	1529
2016- till date (18.11.2116)	592
Total in three years	3534

Table 3: No. of acute cholecystitis.

Years	Total no. chronic cholelithiasis
2014	64
2015	37
2016	32
Total in three years	133

Table 3: No. of gall bladder perforation.

Years	Total no. chronic cholelithiasis
2014	64
2015	37
2016	32
Total gall bladder perforatio	133

All the patient of gall bladder disease related to stone were 3689 in the last three years (2014- 2016).

Presentation of acute cholecystitis

Among all gall stone disease acute cholecystitis was 3.60%. The presentation of acute cholecystitis was very small and most patients presented with chronic gall stone disease. Most of the cases of gall bladder stone were operated prophylactically in a stable state.

Incidence of gall bladder perforation

There were 133 patients presenting with acute symptoms. Among these patients the gall bladder perforation was in 22 patients, a percentage of 16.5. This is a fair number.

Table 4: Management of gall bladder perforation in this hospital.

Total no. of perforation	Conservatively managed	Operatively managed
22	12	9 (1 pt. left LAMA)
%	52.7	47.3

During three years period (2014-2016) the following was the mode of management.

Laboratory findings

In all the cases there was increase in TLC count invariably. This is a sign of sepsis and acute disease process.

Operation and operative findings gall bladder perforation. In eight cases exploratory laparatomy was done. In one case pigtail drainage in the sub-hepatic space was performed. This case showed collection in sub-capsular space and in pelvis in USG findings. This also showed necrotized perforated gall bladder on USG. In exploratory laparotomy different findings were noted as has been described in surgical texts.

Mortality

In this series no mortality was noted. It seems very odd because as per other literature finding it is in range of 12% to 16%. ¹² In this hospital during the course of treatment, the reason may be that when the patient was critically ill the attendants of the patient might have taken the patient to other place. Moreover it is a very small sample size so may not give the exact results as per the other literature.

DISCUSSION

Gall bladder perforation presents as a complication in approximately 3% of acute cholecystitis, mostly occurs in the presence of gall stones. The mortality is in the range of 12% to 16%.¹² Neimeier classified the gall bladder perforation as generalized peritonitis as acute or type

1,pericholecystic abscess and localized peritonitis as subacute or type 2 and cholecystoenteric fistulas chronic or type 3.13 The pathology of perforation has already been discussed previously. The most distal part of gall bladder is the fundus, in terms of blood supply making it the most vulnerable part for gangrene formation and so perforation. The perforation occurs early in two weeks or several weeks after episode of acute cholecystitis. 14 Old patients are more vulnerable for perforation of gall bladder because of other associated co- morbidities specially atherosclerosis. The incidence of occurrence of perforation increases to four folds if there is delay in proper management of acute cholecystitis more than two days after the onset of symptoms. Clinical presentation varies from an acute generalized peritonitis to nonspecific abdominal symptoms when fundus is involved in perforation. Gall bladder perforation with uncomplicated cholecystitis presents difficulty in diagnosis because the bile leaked from the gall bladder gets accumulated in gall bladder fossa and may not produce sign and symptoms of peritonitis initially.14

CONCLUSION

Gall bladder perforation occurs in about two weeks or several weeks after episode of acute cholecystitis, the incidence of occurrence of perforation increases to four folds if there is delay in proper management of acute cholecystitis more than two days after the onset of symptoms. Clinical presentation varies from an acute generalized peritonitis to nonspecific abdominal symptoms and thus requires prompt diagnosis for better prognosis.

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