Clinical study, management and outcome of gall bladder perforation in a tertiary care hospital

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ABSTRACT

Background: The objective of the study was to present our clinical experience with gall bladder perforation cases. This may help in the management and decision making of such cases.

Methods: Records of 480 patients who received medical and/or surgical treatment with the diagnosis of acute cholecystitis in our hospital between 2007 and 2014 were reviewed retrospectively. Twenty nine (6%) of those patients had gall bladder perforation. Original Niemeier’s classification of gall bladder perforation used to describe the type of perforation. The parameters including age, gender, duration of symptoms, diagnostic procedures, medical or surgical or radiological interventional treatment used, morbidity and mortality were evaluated.

Results: Out of the 29 patients, 25 patients had subacute type of gall bladder perforation (Niemeier type II) and 4 patients had chronic (Niemeier type III) perforation. None of the patients encountered had generalized peritonitis (Niemeier type I). The diagnosis in all these patients was established on admission to the hospital by means of abdominal ultrasound and computed tomography. Twelve (43%) patients underwent early surgery. The rest (58.6%) either underwent conservative medical line of management or pigtail catheter insertion in the collection followed by interval cholecystectomy. Three (10.7%) patients died of sepsis and associated comorbid condition.

Conclusions: Early diagnosis of gall bladder perforation is of critical importance. Abdominal ultrasound coupled with computerized tomography is useful in diagnosis of gall bladder perforation. Management strategies include early surgery in patients with generalized peritonitis or suspicion of gall bladder necrosis, and initial conservative line of management and/or pigtail insertion in surgically high risk patients which can be followed up by interval cholecystectomy.

Keywords: Acute cholecystitis, Early diagnosis, Early surgery, Gall bladder perforation, Pig tailing

INTRODUCTION

Gall bladder perforation (GBP) is a potentially fatal disease, its presentation can vary and therefore is a dilemma for early diagnosis. It has been reported to occur in 2-15% of patients with acute cholecystitis.¹-³ Most perforation are subacute; causing pericholecystic abscess. Acute free perforation with bile peritonitis and chronic perforation with an internal biliary fistula are rare. The most common site of perforation is fundus of the gallbladder.⁴

Various prognostic factors have been proposed as risk factors that contribute to the development of complications, such as gangrene, empyema, emphysematous cholecystitis, and perforation, in patients with acute cholecystitis.⁵⁶ Advanced age, male sex, associated diseases, fever >38°C, and marked
leukocytosis should prompt an increased awareness for complications.\textsuperscript{3,9}

**Niemeier classification**

Niemeier in 1934 classified gall bladder perforation as: Acute or type I: free gallbladder perforation and generalized biliary peritonitis. Subacute or type II: pericholecystic abscess and localized peritonitis. Chronic or Type III: cholecystoenteric fistula.\textsuperscript{6}

It is important to realize that the three types of perforation have different presentations. Patients with type I perforation usually have risk factors leading to immunodeficiency that prevents localization of the inflammation, thus leading to free perforation and generalized peritonitis. Patients with type II perforations present with features not typical of acute cholecystitis, and type III patients present with features similar to those of chronic cholecystitis and so are difficult to identify preoperatively unless they have obstructive symptoms.\textsuperscript{3,5}

**METHODS**

**Sample size**- Records of 480 patients of acute cholecystitis admitted to our hospital between 2007 and 2014 were reviewed retrospectively. Twenty nine (6\%) of those patients had gall bladder perforation. Perforations due to trauma, iatrogenic causes and malignancy are excluded.

**Statistical method and tool**

Original Niemeier’s classification of gall bladder perforation used to identify and describe the type of perforation in patients. The parameters evaluated are age, gender, duration of symptoms, diagnostic procedures, medical or surgical or radiological interventional treatment used, morbidity and mortality. Chi square test used for analysis. Abdominal ultrasonography, abdominal contrast enhanced computed tomographic scan, complete blood count and blood chemistry tests were performed in all patients. Abdominal drains were placed in all operated patients.

As this was a retrospective observational study which included only data assessment of patients treated under single surgery unit at a tertiary care center where consent is always taken from patients on admission for using data and photographs for research purpose, so the need for ethics approval was waived by our IRB named Institutional Ethics Committee, Topiwala National Medical College and B. Y. L. Nair Ch. Hospital, Mumbai.

**RESULTS**

Out of the 29 patients, 25 patients had subacute type of gall bladder perforation (Niemeier type II) and 4 patient had chronic (Niemeier type III) perforation. None of the patients encountered generalized peritonitis (Niemeier type I) secondary to acute gall bladder perforation.

Of the 29 patients 15 patients were male and 14 were female; slight male preponderance was found in type II perforation while no difference in type III perforation cases. Type II perforation was seen mostly in 4\textsuperscript{th} decade and type III in 6\textsuperscript{th} decade of life. The mean age of presentation in type II was 55.5 years and 67.7 years in type III. Patients presented with complaints of abdominal pain, fever, nausea and vomiting. All the patients had pain localized to the right hypochondriac region, of which 3 patients presented with generalized pain over the abdomen.

![Figure 1: Type II Niemeier classification.](image)

Of the 29 patients, one patient experienced pain over a duration of 6 months which was diagnosed as type III perforation in a patient with Gilbert’s syndrome. Of the 4 patients with type III Niemeier’s perforation, all had diabetes mellitus as a co morbid condition and 1 patient had ischaemic heart disease and hypertension too. Among the patients with type II perforations, 4 patients had hypertension, 3 had diabetes mellitus and 3 had ischaemic heart disease as a co morbid condition. 1 patient was sero-positive, 1 was a case of typhoid and 1 patient was 21 weeks pregnant.

On analysis of haematological parameters, leucocytosis was seen in 20 patients of which 3 were type III and rest were type II gall bladder perforation. 4 patients had increased total bilirubin of which 1 patient was type III and rest were cases of type II perforation.

Abdominal ultrasonography (USG) was able to detect gall bladder perforation in 18 cases of type II perforation. Not a single case of type III perforation was diagnosed on USG. CT scan was diagnostic for gall bladder perforation in all 29 cases. The most common site of perforation was fundus, seen in 23 cases followed by site of perforation in the body seen in 5 cases and 1 case had perforation at Hartmann pouch. 15 patients had associated cholelithiasis along with gall bladder perforation; among them 1 patient
was of type III gall bladder perforation and the rest were type II. Another important finding noted was the irregularity of gall bladder wall. Presence of pericholecystic collection was seen mainly in type II perforation cases.

Twelve (43%) patients underwent early surgery. The rest 17 (58.6%) either underwent conservative medical line of management or pigtail catheter insertion in the localized collection followed by interval cholecystectomy.

In our study there was mortality in 3 (10.7%) patients; out of which 2 patients had Niemeier type II and one patient had Niemeier type III perforation. All the 3 patients were diabetic and died due to sepsis.

**DISCUSSION**

Gallbladder perforation is a rare but life threatening event.\(^5\) Gall bladder perforation can be traumatic, iatrogenic or idiopathic. It is not possible to predict reliably in which patients this complication will develop.\(^3,5,7,10\) Strohl et al reported the results of a series involving 31 patients with perforation whose symptoms were similar to those in patients with uncomplicated Gallbladder perforation with acute cholecystitis.\(^11\) Conditions such as cholelithiasis, infections, malignancy, steroid therapy, diabetes mellitus and atherosclerotic heart disease are all predisposing factors for gallbladder perforation.\(^11\) Inflammation of the gall bladder wall due to cholecystitis or any bladder wall pathology can lead to ischemia and necrosis and eventually gall bladder wall perforation. Perforation of the gallbladder (GBP) is an important complication of acute cholecystitis. Acute uncomplicated cholecystitis is more common among females, with a female to male ratio of two to one; however, GBP is more frequent in the male gender.\(^1,2,5,7,12\)

In our study also the male preponderance was seen.

Fundus of gall bladder is the most common site of perforation as it is the most distal part with regards to blood supply.\(^4\) In our study too fundus was the most common site accounting for about 79.31%. 20 cases of the type 2 and 3 cases of the type 3 perforation was at fundus. This denotes the importance of ischaemic mechanism of gall bladder perforation.\(^17\) Parker et al reported that high fever, right upper quadrant pain, and elevated WBC count are not diagnostic features for GBP.\(^13\) In our study we found high fever in 58.62% and high WBC count in 72.41% of the cases. As has been suggested by other investigators, our study revealed that high fever and leukocytosis were associated with a higher incidence of perforation.\(^7,9,14\) Some systemic diseases, such as atherosclerotic heart disease and diabetes, may induce ischemia of the gallbladder wall, leading to necrosis and perforation.\(^1,5\) Stefanidis et al reported that cardiovascular comorbidity appears to be a risk factor for perforation, with half of the patients with perforation affected by it.\(^2\) In our study, cardiovascular comorbidity was more commonly detected in the patients with GBP. Gallbladder perforation may develop as early as 2 days after the onset of the symptoms of acute cholecystitis or as late as several weeks. The duration of presentation in our series was short of 2 to 10 days except in 1 patient who experienced pain for more than 6 months duration.
which was diagnosed as type III perforation in a case of Gilbert’s syndrome.

Use of USG and CT scan helped in the diagnosis. In one review, a correct diagnosis was established preoperatively in 18 (62%) patients. Sood et al noted that the sonographic hole sign, in the defect in the gallbladder wall is visualized, is the only reliable sign of GBP. However, in Kim et al’s study, the site of the defect was not visualized by USG in any of the 13 patients. USG is the primary investigation for GBP while CT scan appears to improve the diagnostic accuracy. CT scan can also show gallbladder wall thickness, and the defect on the wall due to perforation. In our series also CT scan was the gold standard for diagnosing gall bladder perforation and diagnosed all the cases.

Cholecystectomy, drainage of the abscess, if present, and abdominal lavage are usually sufficient to treat GBP. In our series, 12 patients underwent early surgery and 17 were managed conservatively including ERCP and biliary stenting in 3 patients, while 6 patients were managed with pigtailing. In this study mortality was encountered in 3 (10.7%) patients in which 2 patients were of type II and 1 patient was of type III perforation, all the patients were above 60 years of age and having diabetes mellitus as a co morbid condition. In the presented series 4 patients of type II perforation who were managed conservatively are awaiting surgery. Unlike gall bladder perforation as a complication of cholecystitis, cases of gall bladder perforation without an apparent cause are rare and are reported vaguely as being idiopathic or spontaneous. A literature search found only 17 cases worldwide reported as idiopathic or as spontaneous gall bladder perforation. Jose EC et al insisted that almost all cases of gall bladder perforation were in fact secondary to a co existent disease such as inflammation, trauma or obstruction.

Limitation of study was that this was a single institution study.

CONCLUSION

Early diagnosis of gall bladder perforation is of critical importance. Abdominal computerized tomography is the gold standard in diagnosis of gall bladder perforation.

Management strategies include early surgery in patients with generalized peritonitis or suspicion of gall bladder necrosis. In patients with significant comorbidities, initial therapy in the form of pigtail insertion for collections, if patients had CBD stones ERCP with stone retrieval and CBD stenting in acute phase which can be followed up by interval cholecystectomy.

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