

Original Research Article

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Study of clinical profile and outcome of gall bladder perforations at a tertiary care centre from central India

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ABSTRACT

Background: Amongst the diseases of gall bladder calculous cholecystitis is the most common. Acute cholecystitis can worsen and result in various complications. Gall bladder perforation leading to generalised peritonitis is a lethal complication.

Methods: In this retrospective observational study we have reviewed 550 cases of acute cholecystitis who were admitted to our institution in 8 years i.e. from 2008 to 2015. Perforations due to other causes (trauma, iatrogenic causes, and carcinoma) were excluded. Niemeier classification was used to identify the patients. Direct-abdominal X-ray series, abdominal ultrasound scanning (US), abdominal contrast-enhanced computerized tomography (CECT), routine blood cell count, and blood chemistry tests were performed.

Results: Fourteen cases (2.5%) were found to have gall bladder perforations during the course of investigations and intraoperatively. Ten (71.42%) were females out of the total fourteen patients. Mean age was 65 years and presenting features on admission were nausea, vomiting, abdominal pain, fever and variable general condition. Aetiopathology of gall bladder perforation remains obscure till date. This clinical condition still remains a diagnostic as well as therapeutic challenge to surgeons. On detailed review of literature, we could not get few references, as the condition is uncommon.

Conclusions: We have done this study to analyse the variable clinical presentation, correlation of non-invasive investigations and intraoperative findings and outcome in cases of gall bladder perforations so as to improve our further management of such cases.

Keywords: Cholecystitis, Diabetes mellitus, Gallbladder perforation

INTRODUCTION

Amongst the diseases of the gall bladder calculous cholecystitis is the most common. Acute cholecystitis can get complicated and result in complications like empyema, gall stone ileus, cholecystoenteric fistula, emphysematous cholecystitis, gall bladder perforation and biliary peritonitis.¹ Of these, gall bladder perforation (GBP) is an uncommon but life-threatening complication of acute cholecystitis, with a reported mortality rate of 12-42%.²⁻⁴

Niemeier in 1934 has classified gall bladder perforations in three types as follows;⁵

- Type I: Free gall bladder perforation with generalised peritonitis
- Type II: Pericholecystic abscess and localised peritonitis
- Type III: Chronic cholecystoenteric fistula.

Neimeir classification is still in use with minor modifications after so many years. The aetiology of the

condition is poorly understood and it still remains a diagnostic and therapeutic challenge to the surgeons.

METHODS

This is a retrospective observational study which was carried out by retrieving the clinical data of 550 patients with acute cholecystitis who were admitted in our hospital from 2008 to 2015 i.e. for a period of 8 years, of which 14 (2.5%) patients had a gall bladder perforation. Niemeier classification was used to identify the patients. This study was carried out on a retrospective basis by collection of data of patients with gall bladder perforation from the case files of those patients who were admitted with a diagnosis of acute cholecystitis. These patients were studied for the demographic profile, clinical features, co-morbidities, laboratory investigations, ultrasonographic findings, CT scan findings and operative findings with the outcome of the treatment. All the collected data was entered on a master chart. At no stage the identity of an individual patient was disclosed.

RESULTS

In this study of 550 patients of acute cholecystitis fourteen (2.54%) had gall bladder perforation. None was

due to trauma, malignancy or any iatrogenic cause. All of these patients were admitted in hospital and were under the management of consultant surgeons after thorough investigations.

Table 1: Distribution of patients according to Neimeier classification (n = 14).

Type	Features	Number of patients	Percentage
Type I	Free perforation with generalised peritonitis	6	42.85%
Type II	Pericholecystic abscess with localised peritonitis	8	57.14%
Type III	Chronic perforation or cholecystoenteric fistula	nil	nil

Gall bladder perforations were diagnosed by ultrasonography and/or CT scan in 8 preoperatively whereas in 4 perforations were diagnosed at operation.

Table 2: Patient data, comorbidities and management.

Patient no.	Age in years	Gender	Diagnosis	Type of perforation	Co-morbidities	Management
1	55	F	AC	I	HTN,DM	Laparoscopic cholecystectomy
2	67	F	AC	II	HTN,DM,COPD on steroids	Drainage
3	65	F	AC	I	HTN,DM,AHD	Open cholecystectomy
4	64	F	Acalculous cholecystitis	II	DM	Open cholecystectomy
5	70	F	AC	II	HTN,DM,AHD	CT guided pigtail drainage
6	50	M	AC	I	HTN,DM	Open cholecystectomy
7	65	M	AC	I	Diffuse atherosclerosis	Laparoscopic cholecystectomy
8	68	F	AC	II	-	Open cholecystectomy
9	69	F	AC	II	IHD with poor ejection fraction	Unfit-drainage
10	71	M	AC	II	HTN,DM	Open cholecystectomy
11	66	F	AC	I	Severe COPD	Unfit-drainage
12	68	F	AC	II	-	Open cholecystectomy
13	65	M	AC	I	HTN,DM unstable	Drainage
14	69	F	AC	I	DM	Subtotal cholecystectomy

AC-Acute cholecystitis, HTN-Hypertension, DM-Diabetes mellitus, COPD-Chronic obstructive pulmonary disease, IHD-Ischemic heart disease, AHD-atherosclerotic heart disease.

Out of the 14 patients with gall bladder perforation, ten (71.42%) were females and 4 were males. Their ages

ranged between 50-75 years with a mean age of 65 years. All of them had several comorbidities out of which

diabetes mellitus was the most common as it was seen in 9 out of 14 (64.28%). One patient had advanced atherosclerosis leading to severe peripheral vascular disease and on exploration his gall bladder showed gangrenous changes leading to perforation. Neimeir's classification was used to categorise the 14 patients, and out of 14 patients six patients were of Type I and eight patients were of Type II. None belonged to Type III.

Clinical presentation of these patients was ambiguous and could not correlate to Neimeir's classification. Some patients (2 out of 14) features of localised peritonitis with ill-defined mass in the right hypochondrium who had type II perforation. Features of overwhelming sepsis were seen in all the patients. All patients had polymorphonuclear leucocytosis. Ten out had significantly high fever. One patient on admission showed advanced sepsis and developed MODS and could not be revived inspite all the resuscitative efforts. Three patients in Type I presented with SIRS.

Abdominal ultrasound was done in all the patients and revealed gall stones in ten (71.42%) out of the fourteen. One patient had acalculous cholecystitis and the other with advanced atherosclerosis had gangrenous gall bladder on exploration but no gall stones on ultrasonography. Ultrasound examination showed pericholecystic fluid in 5 could locate the defect in wall due to perforation in 4 and generalised peritoneal collection in six patients. CT scan was done in 12 out of 14 patients. In addition to the findings of presence of gall stones, peritoneal collections and site of perforation CT scan showed the correct thickness of gall bladder wall which helped in making the diagnosis.

In one patient CT scan and USG also diagnosed an associated liver abscess. Perforations of gall bladder were seen at the fundus, corpus and infundibulum. The most common site of perforation was fundus of the gall bladder which was seen in 60% of patients in this study.

Table 2 shows the details of patients with diagnosis, comorbidities and type of management with outcome in each of fourteen patients in this study.

Open cholecystectomy was done in seven out of fourteen patients and laparoscopic cholecystectomy in two out of 14 patients in this study. In one patient a subtotal cholecystectomy was done with T-tube drainage. One patient with advanced atherosclerosis had gangrene of the gall bladder with perforation which is a rare clinical entity. In the remaining five patients with significant comorbidities, ultrasound or CT guided continuous drainage was done by insertion of a pig-tail catheter. These patients were further managed conservatively.

Hospital stay of the patients in this study ranged between 7 to 30 days with a mean stay of sixteen days. One patient (7.2%) out of the fourteen died of sepsis related complication leading to MODS. Four patients (28%) had

significant complications like acute renal failure, acute pancreatitis, sub-hepatic or pelvic abscesses which required drainage by percutaneous method under ultrasound guidance.

DISCUSSION

Ischemia and necrosis occurring as a result of infection and inflammation results in GBP in 2% to 11% of acute cholecystitis patients.⁶⁻⁸ GBP also develops following acalculous cholecystitis, although rare.^{9,10} GB fundus, the most distal part with regard to blood supply, is the most common site of perforation.^{7,11}

The incidences of type I and type II perforation are 42.85% and 57.15% in our case series. The type I perforation varies from type II in clinical diagnosis (e.g. in the form of peritonitis) assisted by radiology, and its treatment in the form of urgent laparotomy (or laparoscopy) and cholecystectomy, or cholecystostomy. Whereas the decision to treat type II perforations is far more complex due to late presentation and chronic nature of perforation and the lack of consensus within the published literature about the most appropriate investigative or treatment modality. The incidence of GBP was 2.5% and the most frequent site of perforation was the fundus in 9 (64.2%) in our study.

Roslyn et al reported that GBP is more frequent in male gender, but in our study there is a female preponderance (2.5:1).¹² Roslyn et al reported that type I and II GBP tend to occur in younger patients, especially more or less at the age of 50 years, whereas type III gallbladder perforations are more common in the elderly. The patients with type I gallbladder perforation were relatively younger than those with type II in our study and type III GBP are not reported in our study.¹²

Severe AHD has been reported in 21% of patients with type I and II gallbladder perforations, and diabetes, in 25% of patients with type I gallbladder perforation.^{7,13} Such high rates are related to vascular disorders caused by these systemic diseases. Diabetes has been observed in (9)64.2% of our patients. It has been reported that type I GBP occurs more frequently.^{4,8,11,12} The type I and type II GBP's in our study are in the ratio of 1:1.3 and the most frequent association is with the diabetes mellitus and cholelithiasis.

Since the symptoms of type I and II GBP and uncomplicated cholecystitis are similar, differential diagnosis may be difficult based on physical examination, laboratory tests, and radiological methods and the diagnosis may not be established preoperatively.^{6,11}

Surgical procedures and morbidities of the patients

Delayed diagnosis is the major cause of its high morbidity and mortality.^{8,12,14,15} Tanaka et al reported that only one patient had a concrete diagnosis preoperatively

in their series of 9 patients.¹⁶ The majority of GBP patients include those who undergo early surgery with the diagnosis of acute cholecystitis. The main complaint of the patients is abdominal pain accompanied with nausea and vomiting during the last 7 d.^{11,17} The duration of symptoms was shorter in patients with type I GBP than in patients with type II GBP in our study.

Sonography findings in acute cholecystitis, such as the GB wall thickening, GB distension, pericholecystic free fluid, and positive sonographic Murphy sign, may also be present in gallbladder perforation cases.^{8,18,20} Sood et al noted that the sonographic hole sign, in which the defect in GB wall is visualized, is the only reliable sign of gallbladder perforation.¹⁸ They were able to diagnose in 70% of patients with a high resolution ultrasound scanner device. However, Kim et al reported that the site of defect could not be visualized on US in any patient.²⁰ In our study, using US we are able to detect the GBP in 4 out of 14 patients. On the other hand, CT can show more accurate signs of free intraperitoneal fluid, pericholecystic fluid, and abscess.^{12,18,21} CT scan also show GB wall thickness and the defect on the wall due to perforation.^{18,19} In our study, twelve patients were diagnosed by CECT. Kim et al reported that the defect could not be visualized on CT in 54% of patients. Doppler ultrasound, magnetic resonance imaging and radionuclide methods have been used in the diagnosis of gallbladder perforation.^{20,22-24}

The majority of our patients with type II GBP were initially treated conservatively and then underwent surgery as no improvement was observed during 24-48 hour period. Cholecystectomy can be performed after the infection is relieved by US guided percutaneous drainage in typeII GBP.²¹

Laparoscopic cholecystectomy can be performed for acute, gangrenous, and/or perforated cholecystitis as well as uncomplicated cholecystitis, but a conversion may be necessary in case of difficulties like an unclear anatomy.^{8,14} In our study, laparoscopic procedure was initiated in 6 patients but conversion was required for four.

Higher morbidity and mortality rates are reported because of difficulty in diagnosis and delay in treatment.^{8,12,18} Glenn and Moore in their study, reported a mortality rate of gallbladder perforation patients of 42%, while other studies reported that the mortality rates are decreased to 12%-16% owing to the developments in anesthesiology and intensive care conditions.^{11,15,25} While in our study 14% mortality was observed.

CONCLUSION

Gall bladder perforation is an uncommon complication of acute cholecystitis which can lead to increased morbidity and mortality. Predisposing aetiopathological factors leading to perforation are still not understood. Further

studies on gall bladder perforations will be helpful in improving the chances of early diagnosis and appropriate treatment although it will be difficult to have large controlled trials on gall bladder perforations as the condition is uncommon.

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