Gall bladder perforation in acalculous cholecystitis

Abstract

Acute acalculous cholecystitis is relatively rare as compared to the calculous cholecystitis. But complications like gangrenous changes and gall bladder (GB) perforation is more common with acalculous variety than calculous cholecystitis. GB perforation following acalculous cholecystitis has a mortality of 10-50% as compared to that of 1% following calculous cholecystitis. The literature pertaining to this is very few and does not conclude a standard management in such cases. Hence we try to review the literature pertaining to gall bladder perforation in acalculous cholecystitis and try to predict when a surgical intervention is crucial in reducing the morbidity and mortality associated with it.

Key words: Acalculous cholecystitis, gall bladder perforation, cholecystectomy

INTRODUCTION

Acute acalculous cholecystitis is relatively rare as compared to the calculous cholecystitis. But complications like gangrenous changes and gall bladder (GB) perforation is more common with acalculous variety than calculous cholecystitis. Acute acalculous cholecystitis was first described by Duncan in 1844 A.D in a case of incarcerated hernia. Traditionally, acute episode of acalculous cholecystitis is managed conservatively. Complication like GB perforation following acalculous cholecystitis is one indication for surgical intervention. The incidence of GB perforation happens to be 5% to 10% of patients with acute cholecystitis but usually is associated with the presence of gall stones. GB perforation following acalculous cholecystitis are usually seen in elderly patients > 60 years.

Various reasons are attributed to GB perforation like trauma, iatrogenic or idiopathic in case of spontaneous perforation. In case of acalculous cholecystitis the majority of spontaneous GB perforation do have some predisposing factors like critical illness, infections like enteric fever, malignancy, systemic disease like diabetes mellitus and atherosclerotic heart disease on drugs like steroids.

Neimeier’s classification

In 1934, Neimeier proposed a classification for GB perforation. According to which

Type I - Acute free perforation into peritoneal cavity.

Type II - Subacute perforation with pericholecystic collection/abscess.

Type III - Chronic perforation with cholecystoenteric fistula formation.

A number of modifications have been proposed but basic classification still stands good.

Epidemiology

According to US studies GB perforation accounts for 5-10% of all acute cholecystitis. GB perforation following acalculous cholecystitis has a mortality of 10-50% as compared to that of 1% following calculous cholecystitis. There is a slight male preponderance in GB perforation following acalculous cholecystitis when compared to calculous variety where female preponderance is noted. Elderly age groups are more affected in age group of 4th to 7th decade. Regarding type of perforation recent studies have cited higher rates of type II GB perforation followed closely
by type I. The timing of GBP can be as early as 2 days from the onset of acute cholecystitis, or after a few weeks.

**Pathophysiology**

To understand the pathophysiology of GB perforation following acalculous cholecystitis a brief idea of its vascularity is necessary. GB is supplied by cystic artery which is a branch of right hepatic artery in 60% of cases. This cystic artery divides into branches and supplies the GB from infundibulum to fundus. Thus, fundus happens to be the site of perforation in case of any ischemia.

Due to any illness like infection, systemic disease there is bile stasis and increased lithogenicity of bile. In critically ill patients there is increased bile viscosity due to fever, dehydration and absence of CCK-induced contraction of GB. Hence, low flow state due to fever or dehydration leads to GB wall ischemia resulting in perforation.

Based on site of perforation the peritoneal contamination is noted, when perforated at fundus it is less possibly covered by omentum and there is high chances of peritoneal contamination. When perforation is proximal to fundus it is easily covered by omentum and intestine and forms a contained collection restricted to right upper quadrant. Roslyn et al reported that type I and II GB perforation tend to occur in younger patients <50 years where type III noted in elderly.

In support of this hypothesis of ischemia, GB specimen arteriography revealed marked difference between calculous and acalculous variety. Gall stone-related disease showed arterial dilatation and extensive venous filling whereas acalculous variety was associated with multiple arterial occlusion and minimal to absent venous filling.

**Clinical presentations**

It is difficult to differentiate GB perforation from cholecystitis in initial period as both have similar symptoms. This delay in diagnosis is the main cause for morbidity and mortality. Patient present with fever, pain in right upper abdomen with secondary generalization on gross contamination. When there is biliary peritonitis patient may have guarding and rigidity but in type II GB perforation patient will have only tenderness in right hypochondrium (Murphy’s sign positive). Parker et al reported that high fever and high WBC count could be observed in 56% and 59% of patients with acute cholecystitis, respectively.

Complete blood count may show raised total count with neutrophils elevated. Liver function test is also normal in majority of cases. Abdominal radiography rarely helps in diagnosis of GB perforation. High resolution ultrasonogram (USG) can pick up GB perforation in the absence of gall stones in 70% of cases where demonstration of ‘hole sign’ [Figure 1] is a reliable sign of GB perforation. Sood et al noted that the sonographic hole sign, in which the defect in GB wall is visualized, is the only reliable sign of GB perforation.

Computed tomography (CT) is better in demonstrating this ‘hole sign’ along with quantifying the collection and wall thickness of GB wall. It is more specific in detecting the GB perforation in acalculous cholecystitis. The sensitivity of CT in detecting GBP and biliary calculi reported to be 88% and 89%, respectively is higher than those reported for the ultrasound studies.

Diagnosis of acute acalculous cholecystitis based on CT scan requires either two major criteria or one major and two minor criteria. They are as follows:

- **Major criteria**
  - GB wall thickness > 3 mm
  - GB distention > 5 cm

- **Transverse**
  - Minor criteria
  - subserosalhalo
  - high attenuation bile
  - pericholecystic fatty inflammation
  - pericholecystic fluid
  - mucosal sloughing
  - intramural gas

Doppler ultrasound, magnetic resonance imaging and radionuclide imaging are also being tried with no advantage over CT scan in diagnosing GB perforation. Cholescintigraphy is a useful tool for early diagnosis in acalculous cholecystitis and can provide a clue for early surgical intervention.

Diagnostic laparoscopy can be a suggested option in clinically suspicious patients with predisposing risk factors with increased GB wall thickness when other investigations are not fruitful.

**Management**

Management of patients with GB perforation mainly depends on general condition of patient as most of them are critically ill. In critically ill patients minimally invasive procedures like percutaneous drainage of collection with pigtail catheterization can be tried along with good antibiotic coverage along with supportive treatment and plan for definitive surgery once patient is stable. In clinically stable patients laparoscopic cholecystectomy can be performed in GB perforation as in uncomplicated cholecystitis but a conversion may be required in case of difficulty due to unclear anatomy. Type II GB perforation can be managed by
Srinivasan, et al.: Gall bladder perforation in acalculous cholecystitis

CONCLUSION

Although CT scan is an important role in identifying the GB perforation, good amount of clinical suspicion is required in to diagnose GB perforation. Laparoscopic cholecystectomy can be an option in managing GB perforation but should never hesitate to convert to open when operating surgeon finds anatomy unclear. To conclude in a clinically suspicious patient with risk factors of GB perforation should be managed with surgical intervention in early phase of illness, thereby drastically reducing the morbidity and mortality associated with spontaneous GB perforation following acalculous cholecystitis [Figure 2].

REFERENCES


Figure 2: CECT demonstrating Hole in GB wall

laparoscopic cholecystectomy with less chances of conversion into open.

Type III GB perforation requires additional surgical procedure for repair of fistula along with cholecystectomy.[20,21]