Diagnosis of Gallbladder Perforation—a Puzzle!

Sanjeev R. Chowksey · Hiteshwari Baghel ·
Priyank Sharma · Brijesh Singh

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Abstract Gallstone disease is common and complications frequently encountered include acute cholecystitis, acute pancreatitis, and cholangitis, but gallbladder perforation (GBP) is rare. A definitive diagnosis is uncommon before surgery and morbidity and mortality associated with this condition are high. Reported incidence ranges between 2 and 10.6 % in patients with acute cholecystitis but is higher in patients managed conservatively. We report here five patients with GBP to show the difficulty in making an early diagnosis.

Keywords Gallbladder perforation (GBP) · Gangrenous gallbladder · Acute cholecystitis · Emphysematous cholecystitis

Introduction

Gallbladder perforation (GBP) is a rare complication of cholecystitis and cholelithiasis. High morbidity and mortality rates associated with this condition are due to delay in diagnosis and treatment, old age, and sepsis. Because symptoms and signs of perforation do not differ significantly from those of uncomplicated cholecystitis, it is commonly misdiagnosed [1].

Perforation is the end result of acute cholecystitis. Factors that cause acute cholecystitis include obstruction of cystic

duct, chemically induced inflammation by concentrated bile, and bacterial infection. Obstruction is due to calculi impacted in the bile duct (92–95 %), congenital anomalies, adhesions, scars, edema, and some unknown causes. Complete obstruction of bile duct results in concentration of bile that acts as an irritant, leading to inflammation. Secondary bacterial infection may then occur. The organisms, most commonly *Escherichia coli* and *Streptococcus faecalis*, are thought to reach gallbladder via lymphatics causing edema, ulceration, suppuration, local or diffuse gangrene, and eventually perforation. A special mention here is about acute emphysematous cholecystitis [2] associated with a higher incidence of GBP caused by gas-forming organism *Clostridium* characterized by the presence of gas in lumen or wall of gallbladder.

Niemeier in 1934 proposed a classification on his observations:

- Type I (acute): Free GBP and generalized peritonitis (68 %)
- Type II (subacute): Localized peritonitis and pericholecystic abscess (16 %)
- Type IIa: Abscess located adjacent to gallbladder
- Type IIb: Abscess located intramurally
- Type IIc: Abscess located intraperitoneally
- Type III: Cholecystoenteric fistula (16 %)
- Type IIIa: Cholecystoduodenal, most common type (70 %)
- Type IIIb: Cholecystocolic (14 %)
- Type IIIc: Cholecystogastric (6 %)
- Type IIId: Choledochoduodenal (4 %)

Derici et al. [1] suggested that when gallbladder is perforated at fundus, it is least commonly covered by omentum and bile drains into peritoneal cavity, resulting in type I perforation. If perforation site is not at the fundus, it is easily sealed by omentum or intestine and the condition remains

S. R. Chowksey · H. Baghel · P. Sharma · B. Singh
Department of Surgery, SS Medical College and SG Memorial
Hospital, Rewa, Madhya Pradesh, India

S. R. Chowksey (✉)
Nirmal Bhavan, Dr. Ambedkar Marg, Indora Chowk,
Nagpur, Maharashtra, India 440017
e-mail: crystal_sanjeev@yahoo.com

localized to the right upper quadrant with the formation of a plastron and pericholecystic abscess.

Risk Factors

According to Stefanidis et al. [3], risk factors for GBP are as follows:

1. Age: >60 years
2. Systemic diseases: Diabetes mellitus (DM), atherosclerotic heart diseases

Roslyn and Bussutil suggested that spontaneous GBP is caused by hypoperfusion of viscera secondary to systemic diseases.

Immunocompression prolongs steroid use for any systemic disease. Inability to combat infection plays a major role in spontaneous perforation of gallbladder in immunocompromised patients.

Material and Methods

Five patients with acute GBP form the basis of this study. Perforations due to trauma, iatrogenic causes, and gallbladder carcinoma were excluded. The aim was to study the incidence of spontaneous GBP and various ways in which acute GBP can present and to find any pointers that may help in correct preoperative diagnosis.

Case 1

A 70-year-old man presented with a 4-day history of generalized abdominal pain with distension, vomiting, and constipation. He had history of generalized, dull abdominal pain for 2 years. Abdominal X-ray showed no significant findings. Liver enzymes were normal. Patient underwent laparotomy with cholecystectomy 2 days later. A perforated gallbladder at the fundus with multiple, black-colored, sand-like tiny gallstones with generalized biliary peritonitis was found. The patient recovered well in postoperative period and was discharged on 9th day without any events.

Case 2

A 50-year-old woman came with a 4-day history of abdominal pain with distension, constipation, and vomiting. X-ray of the abdomen was inconclusive. Liver function test (LFT) was normal. Widal test was positive. On laparotomy, gangrenous gallbladder perforated at fundus with pericholecystic abscess with a large gallstone of around 3×3 cm was found in the body of gallbladder. Cholecystectomy was performed. The patient was discharged on 13th postoperative day.

Case 3

A 72-year-old man with type 2 DM on oral antidiabetic drugs presented in emergency care with 7-day history of abdominal pain and vomiting. Abdominal X-ray was inconclusive. Ultrasonography showed mild hepatomegaly with an impacted gallstone at the neck of distended gallbladder with pericholecystic edema. On peritoneal tapping, hazy, thick, non-foul-smelling, yellowish fluid was aspirated. Cytology suggested exudate. LFTs were normal. The patient was treated for peritonitis and he underwent laparotomy 4 days later. Thickened gallbladder perforated at body with a large gallstone impacted at neck with pericholecystic abscess and pyoperitoneum were found. Cholecystectomy with drainage of pyoperitoneum was done. The patient deteriorated postoperatively and succumbed on 6th day.

Case 4

A 40-year-old-man presented in emergency care with 1-day history of abdominal pain and distension and vomiting. X-ray of the abdomen was inconclusive. Liver enzymes [serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), alkaline phosphatase] were normal with raised serum bilirubin levels (2.1 mg%). The patient underwent laparotomy 4 days later. Gallbladder with a perforation of about 5×3 mm near the fundus with a solitary calculus impacted at perforation site with generalized peritonitis was found. Cholecystectomy was performed. The patient was discharged on 9th postoperative day.

Case 5

An 85-year-old nondiabetic man came with 2-day history of abdominal pain and distension. X-ray of abdomen showed distended small bowel loops, but no free gas under the diaphragm. The patient had very high levels of blood sugar on routine biochemical investigations. Liver enzymes were raised more than five to seven times of the normal values (SGOT 280 U/L, SGPT 110 U/L) with serum bilirubin level of 4.5 mg% and serum alkaline phosphatase value of 240 IU. The patient underwent laparotomy next day. Gangrenous gallbladder with a perforation of 10×5 mm in the body of gallbladder with generalized peritonitis was found for which cholecystectomy was done. Postoperatively, the patient suffered diabetic coma with septicemia and burst abdomen and expired on 17th postoperative day.

Discussion

GBP is a very rare complication of chronic cholecystitis found in 2 % of patients operated for cholecystectomy and

is associated with high mortality. In patients with acute cholecystitis, inflammation may progress and cause ischemia and necrosis, resulting in GBP. GB fundus, being the most distal part for blood supply, is the most common site of perforation [4]. In our study, fundus was the most common site of GBP (three of five).

Although uncomplicated acute cholecystitis is more common among females (2:1) [5], GBP is more frequent in males [3, 5]. In our study, 80 % (4/5) were males.

Studies by Glenn and Moore have reported mortality rates of around 42 %, whereas other recent studies by Lennon and Green and Roslyn and Bussutil have reported that mortality rates decreased to 12–16 % owing to developments in anesthesiology and intensive care conditions. A mortality of 40 % was reported in our study. This could be attributed to the fact that both patients who died were elderly diabetics, which supports evidence that diabetics and elderly patients have a higher mortality rates and associated with a complicated disease [3].

GBP is usually seen over the age of 60 years. Stefanidis et al. [3] reported that types I and II GBP tend to occur in younger patients. Patients with type I GBP were relatively younger than those with type II perforation in our study. No type III GBP was seen.

No gallbladder mass was found in any of the cases. However, four of the five patients had gallstones intraoperatively, which supports that patients with chronic cholelithiatic disease are more prone for GBP.

Biliary calculi are often missed on computed tomography (CT); hence, ultrasonography is usually the initial mode of investigation in cases of suspected GBP. Following perforation, ultrasonography and CT both show complex fluid collections surrounding the gallbladder. The wall of the gallbladder can appear focally disrupted and the gallbladder lumen may be seen within, or peripheral to, the pericholecystic abscess [6].

CT is commonly used in the evaluation of abdominal pain when other diagnoses in addition to acute cholecystitis such as emphysematous cholecystitis, hemorrhagic cholecystitis, and GBP are considered, which are very difficult to establish at sonography [6]. Sensitivity of CT in the detection of GBP and biliary calculi is 88 and 89 %, respectively [7]. Ancillary findings such as pneumobilia are more likely to be detected on CT. Specific findings that suggest gangrenous cholecystitis include foci of gas within the gallbladder wall, lack of gallbladder wall enhancement (focal or diffuse), mural striation, intraluminal membranes, pericholecystic abscess formation, and adjacent hepatic parenchyma hyperenhancement [8]. Bennet et al. [6] in their comparative study of CT and ultrasonography on 13 patients with GBP detected the site of perforation in 50 % of patients on CT, but in no patient on ultrasonography.

Magnetic resonance (MR) imaging, including MR cholangiopancreatography, can be a valuable complement to ultrasonography and CT when additional information is needed. MR images have excellent tissue contrast and can provide

more specific information. Abnormally hyperintense areas of fat-suppressed T1- and T2-weighted signal within and adjacent to the gallbladder wall on MR cholangiopancreatography suggest possible perforation in the setting of acute cholecystitis. Causes for such areas of signal abnormality include gallbladder wall ulceration, intramural hemorrhage, mural necrosis, and abscess formation [8]. Contrast-enhanced MR imaging more clearly delineates the wall disruption.

Endoscopic retrograde cholangiopancreatography (ERCP) has an important role in diagnosis and therapy of biliary tract injuries. It demonstrates the exact site of bile duct disruption and may allow stent placement in selected bile duct injuries. ERCP images show disruption of the bile duct wall and extravasation of contrast material [9].

Cholescintigraphy (technetium 99m isotope scan, hepatobiliary iminodiacetic acid (HIDA) scan) is most commonly used to diagnose problems with the gallbladder when other more commonly performed tests, particularly ultrasonography, are unequivocal. A radioactive dye (technetium 99m-labeled hepatic 2,6-dimethyl imino diacetic acid) is injected intravenously and the radioactivity is measured through the gamma scintillation camera. HIDA scans have sensitivity (94 %) and specificity (65–85 %) for acute cholecystitis, and they are sensitive (65 %) and specific (6 %) for chronic cholecystitis. In patients with GBP, HIDA scan shows a defect in the wall of gallbladder and extravasation of radioactive dye in peritoneal cavity [10, 11].

Lau et al. [12] and Gurusamy et al. with Cochrane Collaboration Library [13] suggested early laparoscopic (keyhole) cholecystectomy. This reduces the morbidity during the quiescent period, need for open cholecystectomy and development of complications, and decreases operating time and hospital stay. Early diagnosis of GBP and immediate surgical intervention are key factors to decrease mortality and morbidity in the management of acute cholecystitis with GBP.

All our patients were diagnosed as GBP intraoperatively pointing to the difficulty in making a diagnosis. Although statistical analysis was not possible because of insufficient number of patients in this series, this observation may be supported by larger series.

Case 1: Intraoperative view of a gallbladder perforated at the fundus

Case 2: Intraoperative view of a gangrenous gallbladder perforated at the fundus

Case 5: Intraoperative view showing perforated gallbladder at the fundus with generalized biliary peritonitis

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