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Presentation and Management of Spontaneous Extrahepatic Biliary Perforations in Adults in a Tertiary Care Centre

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Abstract:
Background: *Iatrogenic biliary injury is a known and quite common complication following hepatobiliopancreatic surgeries, but spontaneous non-traumatic perforation of biliary tree is not so common. Aim. To investigate and treat the patients presenting with spontaneous extrahepatic biliary perforations.*
Methods: *A total of 15 patients presented with spontaneous extrahepatic biliary perforation.*
Results: *9 patients were treated surgically and 6 patients responded with conservative management.*
Conclusion: *Spontaneous biliary perforations should be diagnosed and treated as early as possible to prevent diffuse biliary peritonitis and sepsis, thereby reducing morbidity and mortality.*
Keywords: *Gallbladder perforation, biliary peritonitis, abdominal paracentesis, T-tube drainage.*

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I. Introduction

Spontaneous non-traumatic biliary perforation is a rare entity. It was first described by Freeland in 1882[1]. Bile duct perforation in infants is caused by congenital biliary anomalies[2]. In adults the bile duct perforation is caused by CBD calculi, intramural infection, thrombosis leading to necrosis of wall of bile duct, obstruction, cirrhosis, after ERCP and abdominal trauma. The major cause of gallbladder perforation is acute cholecystitis which can be calculous or acalculous[3-4]. Incidence of acute cholecystitis in asymptomatic cholelithiasis is 2%, and the incidence of gallbladder perforation in acute cholecystitis is 2-18%[5]. Due to its rarity, gallbladder perforation represents a diagnostic and surgical challenge[6], as a result of which and due to associated medical comorbidity especially in elderly, the mortality can reach 30-50%[7].

II. Materials And Methods

The study was conducted in the post graduate department of surgery, Government medical college jammu. 15 patients presented in emergency with varied complaints like pain abdomen, vomiting, fever, jaundice, abdominal distension. The diagnosis of spontaneous extrahepatic biliary (gallbladder and CBD) perforations were made after detailed clinical examination, biochemical investigations and imaging modalities.

III. Results

15 patients were admitted with a diagnosis of spontaneous biliary perforation. 5 were males and 10 were females.

Table 1. Age and Sex distribution.

Age group (in years)	Male	Female
30-40	-	2
40-50	2	-
50-60	1	3
60-70	2	3
70-80	-	2
Total	5	10

Table 2. Presentations and various ways of management.

Parameters	No. of patients
Hemodynamic instability at presentation	5
Jaundice	2
Generalized peritonitis	3
Pleural effusion	5
Associated comorbidities	6
CECT abdomen	5
GB perforation	13
GB perforation with associated cholelithiasis	10
CBD perforation	2
CBD perforation with associated choledocholithiasis	2
Conservative management	6
Operative management	9

6 patients had different comorbidities which included hypertension, diabetes mellitus, ischemic heart disease, chronic kidney disease and chronic obstructive pulmonary disease. 3 patients presented with diffuse peritonitis. CECT abdomen was done in 5 patients. 6 patients were managed conservatively in view of improvement after initiating injectable antibiotics and CT documented sealed gallbladder perforation. 9 patients were treated surgically. 13 patients had gallbladder perforation (Figure 1) and 2 patients had CBD perforation.

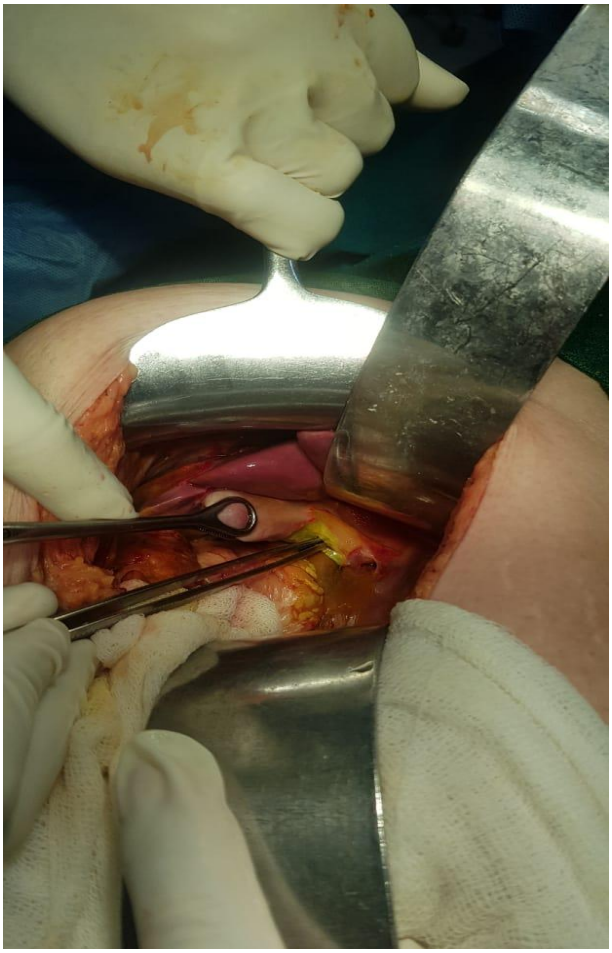

Figure 1. Perforation in distal body of gallblader with impacted large stone at Hartmann's pouch.

Fundus of the gallbladder was the commonest site of perforation. One patient had anterior CBD perforation and one patient had perforation over posterior wall of CBD. 10 patients of gallbladder perforation had cholelithiasis and both patients of CBD perforation had choledocholithiasis. Exploratory laparotomy with peritoneal lavage with cholecystectomy was done in 6 patients. Exploratory laparotomy with peritoneal lavage with T-tube drainage was done in one patient. Exploratory laparotomy with peritoneal lavage with cholecystectomy and repair of posterior CBD perforation with T- tube drainage was done in one patient. Peritoneal drains under local anesthesia were placed in one patient of biliary peritonitis with multi organ dysfunction who was not fit for general anesthesia.

Table 3. Procedures performed in patients who were treated surgically.

Procedure	No. of patients
Exploratory laparotomy with cholecystectomy	6
Exploratory laparotomy with T- tube drainage	1
Exploratory laparotomy with cholecystectomy repair of posterior CBD perforation with anterior T-tube drainage	1
Placement of peritoneal drains under local anesthesia	1

One female patient with posterior CBD perforation was initially admitted as a case of acute biliary pancreatitis, who subsequently developed peripancreatic fluid collections and tense ascites (Figure 2).

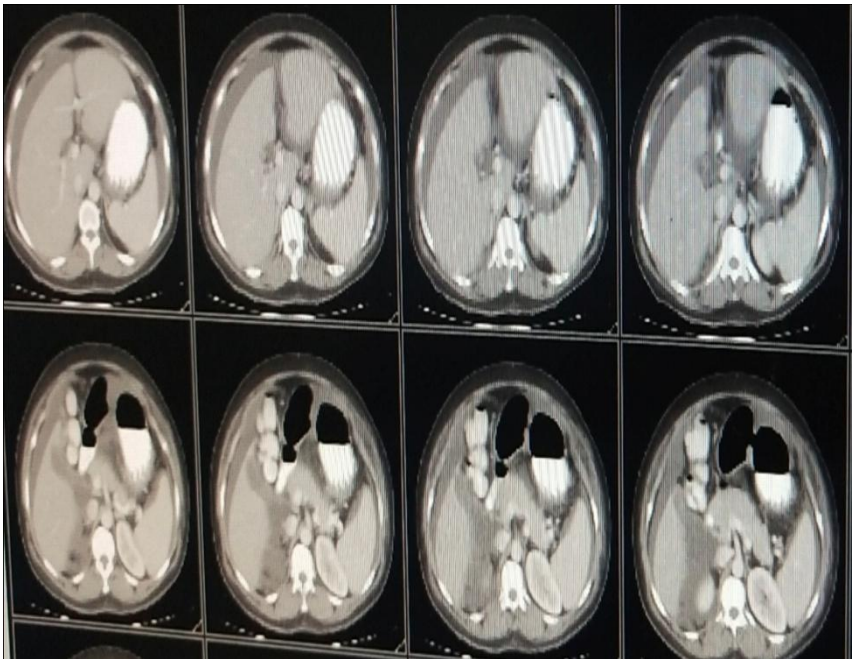

Figure 2. CECT Abdomen showing multiple small calculi in gallbllader with cholecystitis, bulky pancreas with peripancreatic fluid collections and gross ascites.

Peritoneal drain under local anaesthesia was placed in view of tense and gross ascites. Decision about exploratory laparotomy was made in view of high output bile drainage from peritoneal drain. Intraoperative finding were; a) one litre of bile in peritoneal cavity, b) omental saponification (Figure 3), c) about 0.5×0.5cm perforation in posterior wall of CBD in supraduodenal part with small rounded shiny calculi along with bile coming out of perforation (Figure 4, 5 and 6), d) gallbladder distended and contained multiple small calculi.

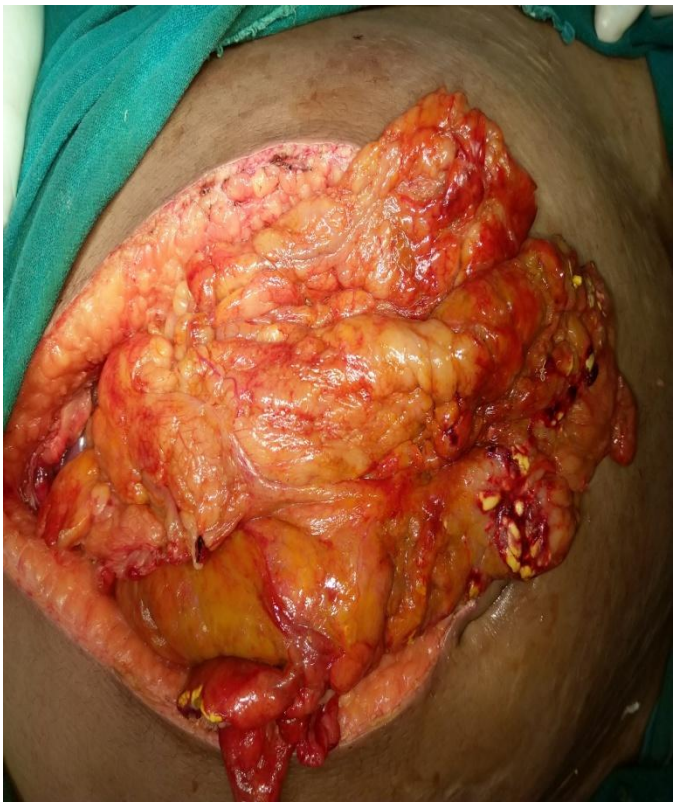

Figure 3. saponification over greater omentum.

Figure 4. Perforation over posterior wall of CBD with small glistening stone coming out.

Figure 5. Small stone which came out of perforation.

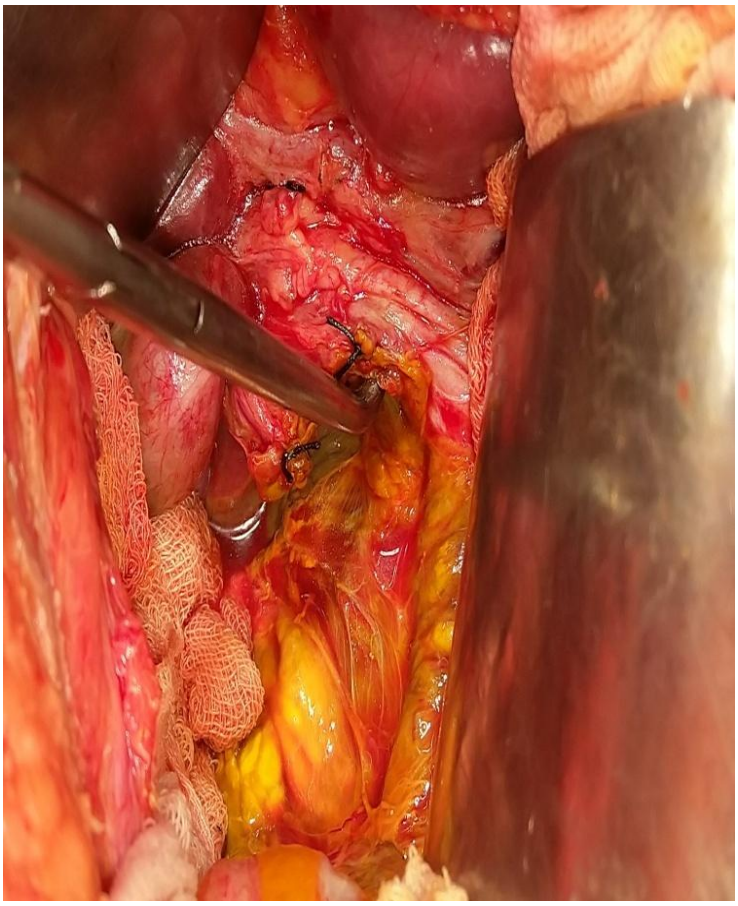

Figure 6. Tip of lahey's right angled clamp introduced inside CBD through perforation posteriorly.

After thorough peritoneal lavage, cholecystectomy with primary repair of posterior CBD perforation with anterior T- tube drainage was done (Figure 7). One tube drain kept in subhepatic space and another in pelvic cavity.

Figure 7. T tube inserted after making anterior choledochotomy.

IV. Discussion

Spontaneous non-traumatic extrahepatic biliary perforation is a rare entity in children and even rare in adults, which can lead to increased mortality in spite of adequate surgical treatment. The gallbladder is common site of biliary tract perforation. Spontaneous perforation of CBD in infants is mainly due to congenital anomalies and in adults due to acquired disorders.

The causes of CBD perforation include congenital weakness of the wall of CBD, choledochal cysts, pancreaticobiliary duct junction anomalies, pancreatitis, trauma, choledocholithiasis, ampullary stenosis, spasm of sphincter of oddi, diverticulum of CBD, inspissated bile leading to distal obstruction, ascariasis, viral infections of CBD, intramural thrombosis and previous biliary tract surgeries[8–10]. The proposed mechanism of CBD perforation is thought to be abrupt increase in intraluminal pressure and decreased blood flow in the vessels which run along the lateral border of bile ducts, resulting in ischemia on anterior surface of the bile duct[11]. The most common site of perforation is the anterior wall of CBD, at the junction of cystic duct and CHD[12].

The causes of gallbladder perforation include acute cholecystitis (calculous or acalculous), typhoid fever, perinatal asphuxia and sepsis[13]. The possible mechanisms for gallbladder perforation involves: 1) bile stasis due to cystic duct obstruction, dehydration, total parenteral nutrition, fasting, which leads to change in bile concentration; 2) vascular impairment of gallbladder due to distension of viscus, sepsis, shock, atherosclerosis; and 3) ischemia, necrosis and perforation of gallbladder wall[14]. Gallbladder perforation in the absence of gallbladder distension is likely related to infection of the Rokitansky-Aschoff sinuses with subsequent necrosis and rupture[15]. Gallbladder perforation occurs most commonly at fundus, which has the least blood supply.

Niemeier[16] in 1934, proposed a classification of gallbladder perforation: type 1- acute free perforation into peritoneal cavity; type 2- subacute perforation with pericholecystic abscess; and type 3- chronic perforation with cholecystoenteric fistula.

There are no classical symptoms and signs of spontaneous extrahepatic biliary perforation. So the diagnosis is difficult and often delayed or even missed, the majority being made only at surgery by observing biliary ascites[17]. Perforation and abscess formation should be suspected in patients of acute cholecystitis who suddenly become toxic and whose clinical condition deteriorates rapidly[18].

Abdominal paracentesis may reveal presence of bilious ascites with increased bilirubin levels in ascitic fluid. Presence of bile in peritoneal cavity, associated with obstructive jaundice in the absence of derangements in liver functions, may be considered as pathognomic for the biliary perforation.

Imaging studies have limited use in the diagnosis of biliary perforations. Ultrasound is the initial radiologic investigation done in most cases, but initial findings are non-specific. Distension of gallbladder and edema of the walls may be the earliest signs of imminent perforation. The typical signs of perforation include the 'hole sign' i.e., a defect in the gallbladder wall, which bears a stiking resembelance to a defect in a perforated balloon or a pericholecystic fluid collection and the formation of an abscess[19]. In cases of spontaneous CBD perforation, ultrasound will show a free or loculated intraperitoneal collection, with normal intrahepatic and extrahepatic ducts[20].

CT findings include gallbladder wall thickening, wall enhancement, wall defect, intramural abscess, intramural gas, mural hemorrhage, presence of gallstones, bile duct stones, intraluminal membrane, intraluminal gas, pericholecystic fat stranding, pericholecystic fluid collection, abscess or biloma formation, extraluminal stones, intraperitoneal free air, ascites, Mirizzi syndrome and mural thickening of hepatic flexure and duodenum. Sensitivity of CT scan in the detection of gallbladder perforation and biliary calculi has been reported to be between 88% and 89%[21].

MRI is better imaging tool in evaluating biliary perforation[22]. A loculated fluid collection or a pseudocyst formation can be more easily visualized on MRCP than on ultrasonography. It is useful in demonstrating wall of gallbladder, the defect and biliary tree. MRCP is also useful in evaluating pancreaticobiliary junction anomalies. Gd-BOPTA and Gd-EOB-DTPA help in demonstrating bile leakage[23].

Hepatobiliary scintigraphy can provide information about the patency of the biliary tree, the site of perforation and any free leak of bile into the peritoneum[24].

After initial pre operative optimization, surgical intervention is recommended to prevent development of sepsis and resultant mortality. No single procedure can be recommended for all the patients with biliary perforations. The surgical procedure has to be tailored according to the age of the patient, the general condition of the patient, the severity of the peritonitis, the site of perforation and the associated intraoperative finding. At laparotomy the biliary ascites is usually obvious. However, the site of site of perforation is not always easily identifiable. There may be inflammatory mass/pseudocyst formation/collection. After careful dissection, the site of leak is then identified. Thorough peritoneal lavage is performed. For gallbladder perforations, a cholecystectomy is curative. For CBD perforations various options include: a simple drainage with or without cholecystectomy, a primary repair of the perforation with or without an external biliary drainage(T-tube drainage or cholecystostomy) , excision of choledochal cyst followed by biliary reconstruction as a primary or a secondary procedure, ERCP and biliary stenting[25], diagnostic laparoscopy and a simple percutaneous drainage.

V. Conclusion

Spontaneous non-traumatic extrahepatic biliary perforation is a rare entity which is associated with high mortality if not diagnosed and treated promptly. Clinical features are non-specific. High index of suspision should be there especially in elderly patients. Treatment should be instituted as early as possible to prevent mortality.

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