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A CASE OF ACUTE CALCULOUS CHOLECYSTITIS WITH CONTAINED GALL BLADDER PERFORATION WITH PERICHOLECYSTIC COLLECTIONS EXTENDING TO LIVER PARENCHYMA

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ABSTRACT **CASE REPORT**

Gall bladder perforation is a serious complication of acute cholecystitis and represents an advanced stage of the disease. Its clinical presentation is varied and can range from non-specific symptoms to acute generalized peritonitis. In many cases, GB perforation in diagnosis during exploratory laparotomy for peritonitis and with no definite source identified. The presentation of gall bladder perforation can be indistinguishable from uncomplicated acute cholecystitis. A 60 yrs. old male with no known comorbidities presented to a tertiary care hospital with diffuse pain abdomen associated with vomiting and constipation of 04 days' duration. Clinical evaluation revealed icterus and tenderness in right hypochondrium and generalized peritonitis. Lab investigations revealed conjugated type hyperbilirubinemia and elevated serum Lipase levels and normal Amylase levels. Initial USG revealed Acute acalculous cholecystitis with normal IHBR and normal CBD. Later NCCT abdomen and MRCP were done which revealed calculous cholecystitis with sealed off GB perforation IHBR, CBD, and normal pancreas. The patient was managed non operatively and responded well. The inflammatory reaction of the peritoneum in bile peritonitis increases the absorption of the spilled conjugated bile from the peritoneal cavity. The conjugated type hyperbilirubinemia without dilatation of the bile duct on imaging examination was the sign of bile leakage into the peritoneal cavity.

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INTRODUCTION

Diagnosis of spontaneous gall bladder perforation is difficult to make even with

imaging examination, such as USG, CT scan, and or MRI [1–3]. The dilution of the previously hyperosmotic and irritative alkaline bile by the peritoneal fluid causes peritonitis and the increased absorption of the conjugated bilirubin causes direct hyperbilirubinemia [4, 5]. The combination of symptoms of peritonitis with no dilatation of the bile duct on imaging examination is specific signs in making the diagnosis of spontaneous gall bladder perforation [23].

Niemeier classified this complication into three types in 1934, and currently, these are described as type 1—free perforation, type 2—perforation with abscess, and type 3—chronic perforation with cholecysto-enteric fistula [23].

CASE REPORT

A 60-year-old man came to the emergency room suffering from pain in the whole abdominal region, fever, and jaundice since two days before. The vital signs were the temperature 99°F, HR 79, RR 18/min, and BP 112/68 mmHg. The patient had jaundice, and the abdomen was distended, guarding with rebound tenderness in the right hypochondrium and epigastric region and decreased bowel sound. The laboratory values were as follows: white blood cell count 13.100 mm3; total bilirubin 8.8 mg/dl; direct bilirubin 7.7 mg/dl; indirect bilirubin 1.1 mg/dl; AST 65 U/L; ALT 97 U/L; amylase 84 U/L; and lipase 735 U/L. On abdominal ultrasound, the gallbladder was normal with 6mm calculus inside obstructing the neck with collection 4.4 x 4.5 x 2.5 cm without dilatation of the bile duct system (Figure 1). NCCT Abdomen was done which revealed 8mm calculus near the neck of GB, a focal breach in the medial wall of gall bladder with well-circumscribed fluid collection measuring 45 x 15 x 40 cm (AP x TR x CC) suggestive of contained gall bladder perforation. No IHBRD, normal CBD, and pancreas. (Figure 2).

MRCP was performed due to jaundice and direct hyperbilirubinemia and elevated Lipase levels, and the result showed that there was gallstone of size 7mm noted near the neck of gall bladder with pericholecystic collections extending into segment IV b of liver parenchyma and normal pancreas. MRCP also showed the collection was shown in CT (Figure 2). As the perforation leak had become localized, the patient was managed conservatively with IV Fluids, antibiotics, analgesics, and supportive management and he responded well. The patient was gradually started on oral fluids and subsequently solids which he tolerated well. The patient has been planned up for laparoscopic/open cholecystectomy.

DISCUSSION

The etiopathogenesis of spontaneous gall bladder perforation is still obscure; however, the most plausible mechanism is the complication from the stones and acute cholecystitis [6, 7]. Early diagnosis of gall bladder perforation is very crucial as delayed diagnosis and management increase morbidity and mortality [23]. Gall bladder perforation has been classified into three types: -Type 1 free perforation, Type 2 perforation with abscess, and Type 3 chronic perforation with cholecysto-enteric fistula [23]. This case presented with features of jaundice, fever, and abdominal pain with the total bilirubin 8.8 mg/dl and direct bilirubin 7.7 mg/dl. The sign of infection WBC 13,100/uL. However, after we found that there was no dilatation of the bile duct on MRCP, the diagnosis of acute cholangitis was excluded. In general, the clinical features of perforation of the gallbladder pose a diagnostic difficulty in distinguishing them from cholangitis because of similar symptoms[11].

Ultrasonography was not conclusive in the presumptive diagnosis of gall bladder perforation. CT Abdomen and MRCP confirmed the sealed-off GB perforation in this case. HIDA scan is a more sensitive tool in diagnosing gall bladder perforation in

complicated cases. However, the practicality of this scan is an issue [12].

The inflammatory reaction of the peritoneum in bile peritonitis induces neoangiogenesis which increases the effective endothelial surface layer leading to relatively faster absorption of the spilled conjugated bile from the peritoneal cavity [4]. Moreover, the dilution of the previously hyperosmotic and irritative bile by the peritoneal fluid increases the osmotic conductance of the bile through the peritoneal membrane[5]. This causes the patient to have jaundice and direct hyperbilirubinemia on blood examination. In the case of biliary peritonitis, indirect bilirubin is less absorbed by the peritoneum because it predominantly exists in its acid form which has low aqueous solubility and tends to aggregate on the lipid membrane [14]. On the contrary, the direct bilirubin is water-soluble [15]. The acidity (pKa) also affects the bioavailability of bilirubin. The direct bilirubin pKa is approximately 1.5, whereas the indirect bilirubin pKa varies widely from 4 to 9 [16]. At the pH of the peritoneal fluid, direct bilirubin will be ionized and more water-soluble than indirect bilirubin is, and therefore, the former is potentially absorbable [17]. Spontaneous common bile duct perforation also shows similar features, that is, obstructed type jaundice. However, jaundice would have begun long before the signs of generalized peritonitis because it would be preceded by the prior common bile duct obstruction [18]. Hyperbilirubinemia is also found in generalized peritonitis caused by perforated or gangrenous appendicitis; however, in this case, not only the direct hyperbilirubinemia but also concomitant with indirect hyperbilirubinemia may occur. The pathogenesis is thought to be because of bacteremia or endotoxemia brought through the portal vein causing an imbalance between the production and excretion of bilirubin in the liver, and its impaired excretion from the bile canaliculi [19, 20]. It should be emphasized that the symptom of peritonitis concomitant with obstructive-type jaundice and no dilatation of the bile duct is fairly specific in making the diagnosis of gall bladder perforation [23]. However, when there is a dilatation of the bile duct, then the diagnosis of acute choledocholithiasis is suspected. The patients with sealed-off GB perforation can be effectively managed conservatively without active surgical intervention and can be followed up for elective cholecystectomy after resolution of active disease.

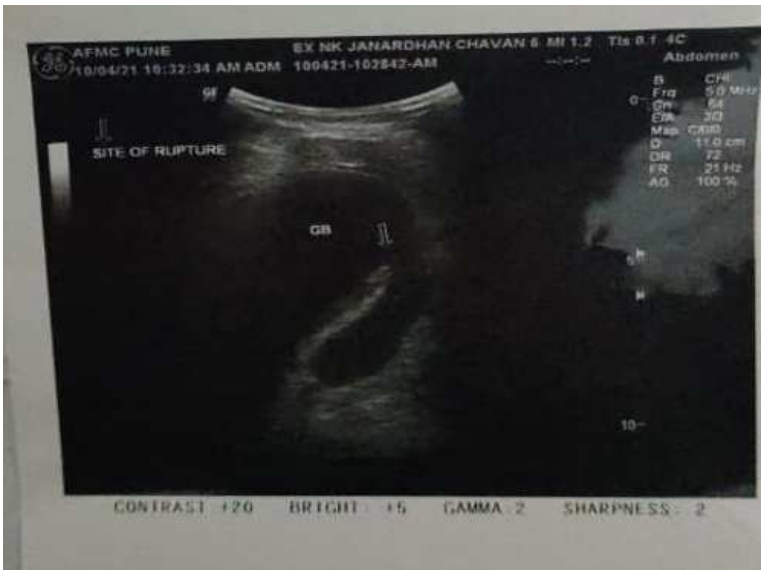
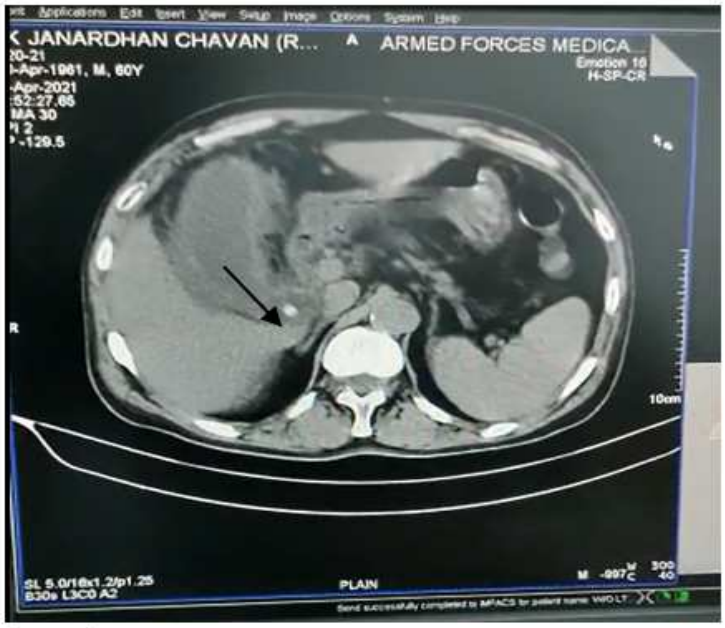
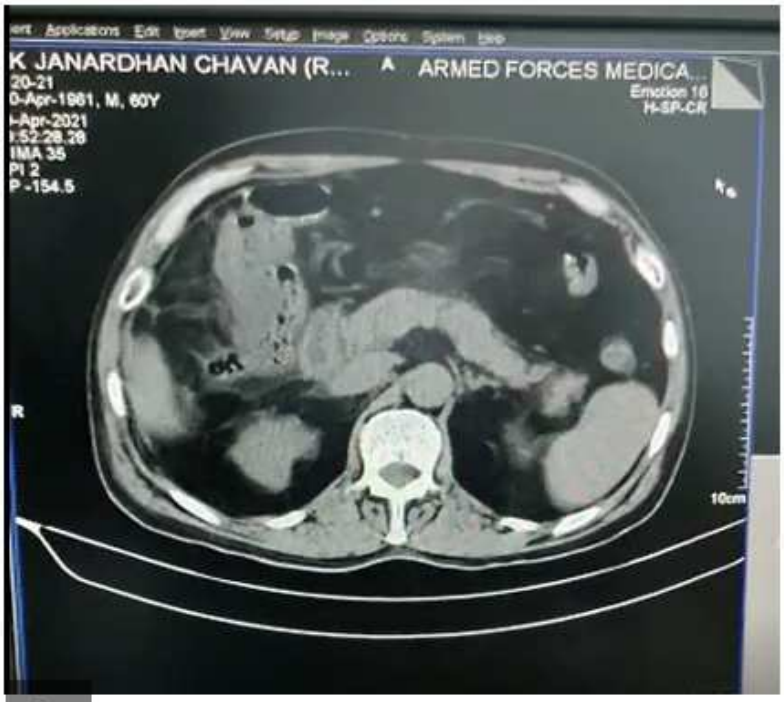


Figure 1: Abdominal ultrasound: normal gall bladder with stones without dilatation of the bile duct.



a



b

Figure 2: Abdominal CT: (a) gall bladder with the stone inside (arrow) and free peritoneal fluid.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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