



### A case study of chemical peritonitis due to biliary leakage in post pigtail catheterization.

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#### Abstract-

Generalized biliary peritonitis is a serious intra-abdominal emergency. In this situation, recognition of the pathology and its treatment is straightforward and is usually associated with a good outcome. There are a few unusual causes of biliary peritonitis, of which rupture of the biliary tree is one. Liver abscess with biliary communication poses management problem if percutaneous drainage is performed. We describe a rare case of biliary peritonitis in amoebic liver abscess due to rupture of an intra hepatic biliary radical after insertion of pigtail catheterisation. Prolonged high-output bile after percutaneous drainage of liver abscess resulted biliary peritonitis and after surgical drainage also suspicion of communication of abscess with intrahepatic bile ducts (biliary fistula). Unusual causes of peritonitis do interrupt our daily routine emergency surgical experience. Rapid recognition of the presence of peritonitis, adequate resuscitation, recognition of operative findings, establishment of biliary anatomy,

and performance of a meticulous surgical procedure resulted in a good outcome.

**Keywords:** chemical peritonitis, liver abscess, biliary radical rupture, pigtail catheterization, laparotomy, biliary leakage, biliary stent.

#### Introduction-

Liver abscess is the most common extra-intestinal manifestation of amoebiasis.<sup>[1]</sup> The formation of a communication between liver abscesses and intrahepatic bile ducts is an uncommon cause of bile leak. Biliary communication of amoebic liver abscess (ALA) has been reported in up to 27% of cases.<sup>[2]</sup> Amoebic liver abscess usually improves within 5–7 days following treatment with metronidazole. Some patients require percutaneous abscess drainage or surgery. Few series have reported the management of liver abscess with biliary communication.

Literatures on endoscopic intervention in the management of biliary communication in liver abscess are limited. We report our experience with the use of therapeutic

pigtail catheterization in the management of liver abscess causing biliary leakage and further complications.

Rupture of an intra-hepatic biliary duct leading to biliary peritonitis is a rare occurrence, with only few cases reported in the literature. This case report reinforces the complication due to rupture of biliary duct during insertion of pigtail catheterisation which used to drain liver abscess and necessity of complete and meticulous operative assessment of the biliary system in the case of bile peritonitis.

### Case report-

A 49 years-old male patient with severe abdominal pain, nausea and vomiting. (BP 90/60, pulse 120/min) and clinical examination revealed tenderness at right hypochondric and epigastric region. Patient was chronic alcoholic and tobacco chewer since 25years and known history of pulmonary Koch's twenty years ago. On examination, he had pallor, deep jaundice, and smooth mild tender hepatomegaly. His haemoglobin was 6.7 g/dl, total white blood cell count was  $22.9 \times 10^3$  /ul with 86% neutrophils, platelet count was  $435 \times 10^3$ /ul, and mean corpuscle volume was 69.9 fL. Liver function tests showed total bilirubin 3.92 mg/dl (normal up to 1.2), direct bilirubin 3.72 mg/dl, alkaline phosphatase 191.4 U/L (normal: 40–129), with hypoalbuminemia (2.72 g/dl). International normalized ratio was 1.08, and renal function tests were normal.

Also later on ultrasonography reports revealed the liver is just enlarged in size and show reduced echogenicity. Ill

defined iso to hypoechoic lesion of size 95 x 94 x 81mm and volume is 370cc is seen in caudate lobe of liver and extending into left lobe with thick septations in peritoneal cavity with significant ascites noted. And diagnosis confirmed as liver abscess with ascites. Due to the large volume of abscess and deterioration in clinical condition even after medical management with metronidazole and third-generation cephalosporin in adequate doses, decision for percutaneous drainage of abscess was made after due consent from patient.

Diagnostic paracentesis and conservative management done for ascites. For the liver abscess, pigtail catheterization done for drainage of liquefied lesion by interventional radiologist. Examination of pus from abscess revealed no organism on gram staining or culture. After pigtail catheterization, initially drainage was anchovy sauce and later on it was bile stained and that indicates the biliary leakage.

Patient responded well to the treatment and became hemodynamically stable. His haematological blood profile as well as his urea and electrolytes were unremarkable. Plain radiology (chest x-ray and erect abdominal films) did not demonstrate any pneumoperitoneum.

Patient was asymptomatic with good bowel sounds and regular bowel emptying.

After two weeks USG repeated and the findings were abscess is reduced in size and inflammatory exudate with pigtail catheter in situ. Also there is large multiloculated collection with organised solid collection in intrahepatic, right flank, right lumbar peritoneal cavity. And

interventional radiologist advised surgical drainage and cannot drain percutaneously.

A diagnosis of peritonitis was made and suspicious of formation of biliary fistula. When the patient was optimized, he underwent surgery. Midline incision taken and during laparotomy, there was no evidence of gastrointestinal perforation; however, free intra-peritoneal bile with biliary leakage found at pigtail catheterization site. And due to this chemical peritonitis was developed and bile collection with plaques formation (approximately 7 litres), Resuscitation done. Thick pseudoperitonium was formed. Multiple small adhesions of small intestine seen with inflamed appendix. Adenolysis done. Biliary leakage seen at the right and at the caudate lobe of liver and that was repaired with the help of prolene 2-0. Haemostasis achieved, And tube drain kept at right hepatorenal pouch and left paracolic gutter. sample of fluid culture reported as growth of E-coli organism. According to sensitivity higher antibiotics started (Inj polymyxin and clindamycin).

After surgical intervention, patient was haemodynamically stable. Patient was hydrated properly and parenteral nutrition given with albumin and PCV transfusion given according to the need under physician's guidance. After proper bowel movements, NBM broke and soft diet started and kept under observation. By drain, the remaining collection drained out in catheter bag and measured daily. Gradually, the patient improved clinically and biochemically, he was afebrile with decreased jaundice, but drain was persistently bilious and thick purulent from tube drain on an average of 500-600 ml/day. The patient had lost weight,

developed pedal edema, and had low albumin level at 1.6 g/dl. Due to persistent bilious drain of 500 ml/day up to 8 days after surgery, suspected biliary fistula and resulting this, recollection occurred in the peritoneum.

Later on hepatobiliary surgeon advised MRCP. On MRCP impression there was abscess in segment IV of the liver of size 5.3 x 4.48 x 6.37cm heterogeneously hyperintense lesion. Suspicious communication of dilated segment II and III duct with abscess. Narrowing with irregularity of CHD by extrinsic impression (considered more likely) or involvement by the abscess. Resultant upstream prominence of left hepatic duct and right posterior hepatic duct and right posterior hepatic duct. Abscess in segment VI of the liver of size 2.91 x 2.13 x 2.99 cm communicating with a subcapsular abscess. Anomalous insertion of right anterior hepatic duct on the CHD. Ascites with bilateral pleural effusions. Small in size right kidney with cortical scarring.

The collection was still increasing. for 5-10 days again conservative management performed. During this bowel habits and bowel sounds of the patient was regular. And collection was used to drain by tube drain with biliary and also thick purulent in nature. But on 15<sup>th</sup> day tube drain released out itself and after 2-3 days' patient came with the complaint of severe abdominal pain, vomiting and distension. investigations done and erect abdomen x-ray suggests multiple fluid levels with dilated bowel loops with obstruction. Haematological investigations suggesting septicemia due to increased WBC count. Severe hyponatremia with hyperkalemia

and low urinary output. Patient was hospitalised for approximately 30 days.

According to patient's condition, the patient was transferred to the high dependency unit for immediate care. Emergency laparotomy and stent application performed with slow but steady post-operative recovery.

## Discussion-

Peritonitis requiring surgical intervention is caused by perforated peptic ulcer in about 40% cases (duodenum:gastric: 3:1), appendicitis in 20%, gangrene of the small bowel or gall bladder in 15%, post-operative complications in 10% and miscellaneous causes in 15% cases.<sup>[3]</sup> Most commonly, peritonitis in the clinical setting is due to microorganisms, though the initial insult is usually chemical as in peptic ulcer perforation where bile, pancreatic enzymes, blood, etc., gain access into the peritoneal cavity.

The peritoneum can be contaminated with bile through a number of routes. The commonest is post-cholecystectomy. This is usually due to the division of small bile channels between the gall bladder and liver, imperfect clipping of the cystic duct, residual CBD stones causing raised intra-biliary pressure and inadvertent division of an accessory hepatic duct. The latter is potentially serious usually requiring biliary reconstruction. Other causes include post liver transplant biliary peritonitis, spontaneous hepatic rupture in pregnancy and trauma to the extra-hepatic biliary system such as that following minimal access renal surgery.<sup>[4]</sup>

Perforation of the biliary tract secondary to rupture of the gall bladder (empyema/gangrene) is well documented. However, spontaneous rupture of the CBD is exceedingly rare and here the aetiologies are increased intra-ductal pressure, calculus erosion and necrosis of the duct wall secondary to thrombosis.<sup>[5]</sup> Spontaneous perforation of extra hepatic ducts is also a very rare cause of jaundice in infancy.<sup>[6]</sup> The commonest site is the confluence of the cystic and common hepatic ducts.<sup>[7]</sup> Biliary peritonitis secondary to intra-hepatic duct rupture is rarely reported in the literature; the causes are pigtail catheterisation in liver abscess (as in this case).

The clinical picture associated with biliary peritonitis varies and the correct pre-operative diagnosis is difficult. Though the initial insult by bile is chemical, secondary bacterial infection is the usual sequelae. Furthermore, it has been clearly shown that, in the presence of bacteria, bile further impairs local host defence mechanism through its detergent lytic effects.<sup>[8]</sup> Paralytic ileus is also a frequent complication. Laboratory findings are usually non-contributory but biliary peritonitis should always be suspected in any patient with unexplained abdominal symptoms.

Communication of Amoebic liver abscess(ALA) with intrahepatic bile duct is an uncommon complication of ALA. The rarity of this complication is probably due to resistance offered by vasculobiliary sheath, a tough fibrous tissue level that surrounds the main and segmental portal structures: bile duct, hepatic artery, and portal vein.

Liver abscess in the present case was diagnosed by ultrasound. The majority of liver abscess responds to conservative management. Complications or poor response to medical management requires intervention such as percutaneous abscess drainage and rarely surgery. Ultrasonography-guided percutaneous drainage of abscess in our case was done due to jaundice, large volume of abscess, and poor response to medical treatment even after surgical intervention. Prolonged high-output bilious with purulent drainage (500-600 ml/day) from the drain for a week prompted us for other form of therapy to deal with the bile leak.

Literatures on endoscopic intervention in the management of biliary communication with amoebic liver abscess cavity are limited. There are no definitive guidelines for the management of patients with liver abscess communicating with the biliary tree. The patient was taken for laparotomy and biliary stenting for communication of abscess with bile ducts documented on cholangiogram.

The aim of treatment is to prevent sepsis in the abdominal cavity and thus prompt recognition of the condition and control of source of the contamination with appropriate drainage/reconstruction of the biliary system is of paramount importance. The type of surgery is dependent upon the general condition of the patient as well as on biliary anatomy. Regardless, biliary peritonitis requires some form of drainage, either externally via the percutaneous route or internally via the endoscopic/open surgery route. In our patient, the intra-hepatic duct rupture was presumably due to insertion of pigtail catheterisation and then leakage forming the biliary peritonitis.

Along with surgical and conservative management slow and steadily recovers the patient. 'T' tube drainage resulted in an uneventful resolution. Prompt recognition of this condition before biliary/systemic sepsis supervened played a major role in the positive outcome for this patient.

**In conclusion**, due to the insertion of pigtail catheter may rupture such biliary duct and develops further complications, prompt laparotomy in a well-resuscitated patient and an adequately tailored operation depending on the operative findings are the mainstay to avoid local and systemic sepsis and long-term morbidity in these cases of peritonitis.

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*Conflict of Interest:* Non

*Source of funding:* Nil

**Cite this article:**

*"A case study of chemical peritonitis due to biliary leakage in post pigtail catheterization."*

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**Ayurlog: National Journal of Research in Ayurved Science- 2020; (8) (4):01- 06**