**INTRODUCTION**

Biliary ascites in children due to spontaneous perforation of the bile duct is a rare entity but well documented in the literature. The first case was described by Caulfield in 1936, since that time more than 150 cases have been reported. The perforation usually occurs at the junction of the common bile duct and the cystic duct. The cause of these perforations is mainly unknown. Biliary ascites can be secondary to perforation of a choledochal cyst. Biliary ascites in older children may occur as a result of trauma or after cholecystectomy. Biliary ascites presents with progressive abdominal distension and jaundice or septic shock, fluctuating course and mild derangement in liver functions with an evidence of cholestasis. Evidence of bilirubin in ascitic fluid, ultrasound, CT and MRCP are mainstay of investigation tools. Surgical intervention has a definitive role including from percutaneous tube placement to complicated biliary procedures.

We report a case of biliary ascites in a 7 months old infant who presented with jaundice and abdominal distension.

**CASE REPORT**

A 7 months old male infant who was a known case of hypoxic ischaemic encephalopathy admitted with history of jaundice and progressive abdominal distension for one month with intermittent non-bilious vomiting and pale stools. He was a preterm (30 weeks gestation) baby, born by C-section due to fetal distress.

On examination, he was mildly jaundiced and pale with obvious abdominal distension but without any respiratory compromise. His weight was < 3rd and length was between 10 - 25th centiles. Abdomen was markedly distended with shiny skin and umbilical hernia. Liver was palpable 3 cm below costal margin on the right side which was soft in consistency without splenomegaly but with evidence of ascites.

The baseline investigations showed normal liver enzymes except for high Gamma Glutamyl Transferase (GGT) and alkaline phosphatase which were 242 U/L and 654 U/L respectively with evidence of cholestasis revealing total bilirubin of 54 mmol/L with 48 direct fractions. CBC showed low hemoglobin which was 9.2 gm/dl. Ultrasound abdomen showed fluid collection with septations and rest of the study was unremarkable for liver, spleen and biliary system.

The next day, his abdominal distension worsened with respiratory compromise and therapeutic ascitic tap was done to relieve his tense abdomen and respiratory compromise. The fluid was greenish in color with positive bilirubin on dipstick and later on confirmed presence of bilirubin (178 mmol/L) from analysis of ascitic fluid with gram positive cocci. He developed septic shock with recollection of the intraperitoneal fluid and respiratory compromise. An intraperitoneal drain was placed for the collection and respiratory compromise by the surgeon. He was further investigated with CT abdomen which showed extensive ascites extending to the right lateral wall as well as medial aspect of the liver with possible communication with the biliary system. MRCP (Figure 1 and 2) report was also consistent with CT, however, the exact location of the biliary leak was not shown by both.

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**CASE REPORT: An Infant with Biliary Ascites**

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**ABSTRACT**

Biliary ascites in children due to perforation of bile duct is a rare entity. The exact pathogenesis is not known but there are proposed mechanisms including congenital weakness of ductal wall, pancreaticobiliary malunion, tuberculosis, necrotizing enterocolitis and rupture of choledochal cyst. Presentation may be acute or sub-acute. Progressive insidious course is the common presentation in children with jaundice, clay colored stool, abdominal distension with slightly elevated liver enzymes but well documented cholestasis. Clinical suspicion with ultrasound, CT, MRCP and ascitic tap provides clue to the diagnosis. Both conservative and surgical interventions are in practice for managing these children. We report a 7 months old infant with biliary ascites due to perforation of bile duct.

**Key Words:** Infant. Spontaneous biliary perforation. Biliary ascites. Bile duct perforation.
He underwent laparotomy and per-operative findings confirmed the biliary leak at posterior and medial aspect of common hepatic duct 2 x 1 cm, almost sealed with dilatation. There was massive biliary ascites and adhesions around the common bile duct with normal gall-bladder and cystic duct. A 10 Fr T-tube was placed in the common hepatic duct for flow of bile. In next 3 weeks, his T-tube was removed as bile flow in the tube started decreasing with good flow of contrast to small intestine evident by cholangiogram. Three months after surgery, he has been jaundice-free and doing well with normal colored stool and liver functions.

**DISCUSSION**

Spontaneous rupture or perforation of bile duct leading to biliary ascites in children is a rare disorder. More often, it is seen in less than 4 years of age with peak incidence during the first year though one case has been reported in the literature at 25 weeks gestation *in-utero.*

There is no sex predilection. The etiology is mainly idiopathic but there are proposed theories which precipitate the rupture such as congenital weakness of the bile duct, pancreaticobiliary anomalies, pancreatitis, viral infection of the bile duct, tuberculosis and necrotizing enterocolitis. Rupture of the bile duct is often associated with pancreaticobiliary malunion with or without a choledochal cyst.

Presentation may be acute or sub-acute. Acute presentation is with signs of peritonitis or shock. This case had sub-acute picture initially but complicated with septic shock. Majority of the children presents with progressive abdominal distension, jaundice and vomiting with pale or normal stool as seen in this case. The clinical findings are mainly of localized or generalized peritonitis and ascites. The diagnosis should be suspected in any infant with an insidious onset of jaundice and abdominal distension with acholic stools. A clinical suspicion of bile duct perforation should be thought of when a previously normal child develops obstructive jaundice in the absence of liver derangement and presence of bile in the peritoneal cavity.

The main laboratory and radiological investigations to diagnose these children are normal or mildly elevated liver enzymes with cholestasis, ascitic tap, ultrasound, radionuclide scan including CT and MRCP. Abdominal sonography will identify generalized ascites or localized collection of fluid. Scintigraphy can localize the perforation site but most of biliary leaks are identified per-operatively as in this case where the site of perforation was sealed off in the common hepatic duct with lot of adhesions around. The evidence of adhesions around the bile duct made the suspect site of leak when there is no clear identifiable leakage seen.

Surgical intervention has good outcome in the majority of these children. The interventions range from simple percutaneous placement of tube in the peritoneum to complicated biliary tract procedures. This case had initial placement of intraperitoneal drain but end-up with laparotomy with insertion of T-tube in the hepatic duct. Percutaneous tube drainage is the initial preferred step in view of that these perforation spontaneously heals as there is usually no distal obstruction but it never helped in this case and it continued to leak. If T-tube is placed with external drainage it should be left *in situ* as long as there is bilious drainage, in this case it lasted around 21 days.

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