Bile Ascites

Perforation of extrahepatic biliary ducts causing bile ascites is rare. It may follow perforations of congenital choledochal cysts or blunt trauma. Forty seven cases of the latter have been reported in children, including 16 of complete transection of the common bile duct (CBD). Two cases of ascites, one of each type are being reported.

Case Reports

**Case 1:** A 3-year-old boy was admitted with complaints of moderate, generalized, continuous pain in abdomen for 20 days, gradually developing pallor for 15 days, poor appetite and occasional vomiting for 10 days, yellow sclera and urine for 5 days and mild fever for 1 month. There was abdominal distension with free fluid but no lump. Investigations showed hemoglobin 7.8 g/dl; WBCs 16,500/mm³ with 77% polymorphs and 19% lymphocytes; serum bilirubin 2.21 (direct) and 3.77 (total) mg/dl; and serum amylase-135 U/l. The ascitic fluid was bilious having WBCs 700/mm³ with 90% lymphocytes, total proteins 3.2 g/dl, and bilirubin 22.77 (direct) and 30.71 (total) mg/dl. Stools were not acholic. Urine was deep yellow. Ultrasonography (USG) and CT scan reported free fluid in abdomen and right sided pleural effusion. X-ray chest was normal with clear costophrenic angles. Three hundred and fifty ml of bile was aspirated before an exploratory laparotomy was done. A large bile collection in the hepato-renal pouch was drained along with collections from other recesses. A loculated bile collection just above the upper border of duodenum was aspirated and a leak was seen from a choledochal cyst just beyond the union with cystic duct. Proximal part of extra-hepatic biliary ducts were patent, but dilators could not be passed into the distal part of CBD. The cyst with thick fibrous and inflamed walls was resected and a choledocho-duodenostomy was done. Post-operative recovery was uneventful and the child went home in 10 days.

**Case 2:** A 3-year-old boy became unconscious after a waterpot fell on his abdomen. He was initially treated conservatively by his local doctor. USG was normal. Seven days later, abdominal distension developed and repeatedappings of the ascitic fluid (nature of fluid
not known) were done by him, but the fluid soon filled up again. Parents of the child were told by the local doctor that he suspected an intra-abdominal injury, but the exploratory laparotomy showed no major visceral injury. The child was admitted to S.D.M. Hospital, Jaipur, 40 days after the initial injury. The abdomen was distended and tense but there was no local tenderness, muscle guarding or rebound rigidity. Hemoglobin was 11.1 g/dl and serum bilirubin 1.25 (direct) and 2.48 (total) mg/dl. Ascitic fluid was deep yellow with WBCs 500/mm³, and 90% lymphocytes, proteins 2.8 g/dl, and bilirubin 14.7 (direct) and 17.5 (total) mg/dl. Stool and urine were normal. USG and CT scan reported free fluid in the abdomen with no detectable cause. On exploratory laparotomy, the peritoneal cavity was full of bile in loculations which were drained. Fibrinous inflammatory adhesions were cleared. Dissection at the porta-hepatis revealed a transverse tear in the common hepatic duct with unclean sloughy edges. Proximal patency of the hepatic ducts was demonstrated, but the tear location prevented distal probing. The tear was repaired by a free omental graft. The CBD was opened at cystic duct level, proximal and distal patency demonstrated, and the CBD was drained by a T-tube. The peritoneal cavity was also drained. There was a continuous bile leak through the peritoneal drain for 2 weeks after removal of the T-tube on 15th day. The drainage tube was removed 15 days later and the tract closed soon thereafter.

Discussion

Idiopathic perforations of CBD, most often, occur about its junction with the cystic duct suggesting vulnerability of the site to developmental errors. In Case 1 of bile ascites, rupture occurred in a Type II choledochal cyst which is rare(5). What causes rupture of choledochal cysts is uncertain. A fibrous weak cyst wall, perhaps, gives way when intra-ductal pressure rises following partial or complete obstruction of CBD. Absence of acholic stools in the two cases reported here shows that some bile managed to reach the gut. The classical triad of pain, mass in abdomen and jaundice occurs in cases of choledochal cyst and the abdominals-mass in the triad is replaced by distension in bile ascites. Resection of ruptured choledochal cyst followed by choledochoduodenostomy is mandatory. Anastomotic drainage of the cyst may lead to stricture, ascending cholangitis or malignancy later on(6).

The blunt injury damage to the common hepatic duct (Case 2) may be explained by its relative fixity to the liver, as also occurs in CBD transection which occurs at the lower end, being fixed by pancreas(3). Local repair and drainage are adequate for isolated biliary duct damage.

Facilities for 99 mTc HIDA scan were not available and negative USG and CT scan reports kept the diagnosis in doubt till established at laparotomy.

REFERENCES


