An unusual case of obstructive jaundice

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Abstract: A case of hydatid cyst of liver rupturing into biliary tree causing obstructive jaundice is reported.

Key words: Obstructive jaundice, hydatid cyst ERSP

Introduction

Jaundice is a symptom complex, characterized by yellowish coloration of tissues and body fluids due to an increase in bile pigments. Jaundice is classified as hemolytic, hepatic and obstructive. Association of hepatic hydatid cyst with bile duct obstruction is not a common occurrence. It occurs after the rupture of hepatic hydatid cyst into the biliary tree. In a patient with rupture into the biliary system, daughter cysts and membranes pass into the common bile duct producing surgical jaundice. It was described by Dew for the first time. Most patients present with recurrent attacks of right hypochondriac pain, fever and intermittent or persistent obstructive jaundice and a palpable liver mass. Diagnosis is made by radiological investigation such as ultrasonography, CT scan of abdomen and ERCP.

Case report

38 years female presented to Tribhuvan University teaching Hospital with history of vomiting, pain in right upper quadrant and jaundice for 1 month. On examination she was icteric, liver function test showed features of obstructive jaundice. Ultrasound showed well defined smooth walled cystic lesion in right lobe of liver containing thin linear echogenic structures within it (fig 1). Bile duct was communicating with the cystic lesion. CBD was dilated and lumen contained linear echogenic structure. Intrahepatic bile ducts were dilated (fig 2). Contrast enhanced CT revealed a well demarcated cystic lesion in right lobe of liver with linear hyperdense septa within it. Intrahepatic bile ducts were dilated in both lobes of liver. Common bile duct and common hepatic duct were dilated with enhancing hyperdense structure within it (fig 3, fig 4 and fig 5). ERCP shows dilated common bile duct with multiple linear, irregular intraluminal filling defects (fig 6). Final diagnosis of Intrabiliary rupture of Hydatid cyst of liver was made subsequently endoscopic sphincterotomy was performed and hydatid sand was retrieved.

Fig. 1: USG shows a well defined cystic lesion in the right lobe of liver with multiple thin linear echogenic structures with in it.

Fig. 2: USG shows dilated common bile duct with linear echogenic structures within it.
Fig. 3: CT scan showing a well demarcated cystic lesion in right lobe of liver with linear hyperdense septa within it. Common bile duct and common hepatic duct is dilated with hyperdense structure within it.

Fig 4: CT scan showing dilated intrahepatic bile ducts in both lobes of liver.

Fig 5: Enhancing membrane with in the common bile duct

Fig 6: ERCP shows dilated common bile duct with multiple linear, irregular intraluminal filling defects.

Discussion

Hydatid disease is a major health problem worldwide since the time of Hippocrates. It is a human parasitic disease and Liver is the most common site (73%). The right lobe of the liver is affected in 80% of cases and the left lobe in 20%. Lewall and McCorkell have classified rupture of echinococcal cysts into three types: contained, communicating and direct.

Though uncommon, intrabiliary rupture is the most common complication of the hepatic hydatid cysts. The incidence of rupture into the biliary tree has been reported as 3 to 17%. When ruptured into the biliary tree, hydatid cysts commonly manifest with biliary obstruction and cholangitis and demand early surgical intervention.

USG is the most commonly employed initial investigation with an accuracy rate of 66–94%. Similar to our case, undulating membranes may be seen within common bile duct. Extrahepatic biliary dilatation is a constant feature.
Echogenic or non-echogenic material without posterior acoustic shadowing may be seen in biliary tree suggesting sludge and daughter cyst. Accuracy can be increased to near 100% with addition of CT. Other than the findings in this patient, CT can show Calcification of cyst wall. An interrupted area of the cyst wall proximal to a dilated duct may be identified as representing the site of communication. Cyst wall discontinuity, a direct sign of rupture, is seen in only 75% of cases. ERCP is the gold standard in confirming biliary tract involvement and may have therapeutic application in some cases. On ERCP, a swollen ampulla of Vater may be seen, with hydatid material protruding out, though this was not seen in our case. Dilated ducts with debris and daughter cysts may appear as radiolucent filling defects as in our case. However a small cystobiliary communication cannot always be excluded by ERCP.

MRI was not done in our case but may be of help when CT and USG findings are inconclusive. The wall of the hydatid cyst appears as low signal intensity. Daughter cysts have a lower signal intensity compared with the mother cyst. In addition, a breach in the low intensity rim of the cyst wall with extrusion of cyst contents, increased echogenicity, fluid levels and presence of air may be seen.

In conclusion patients with hepatic hydatid cyst with dilated common bile duct intrabiliary rupture should be suspected.

References