

A rare cause of jaundice in a case: Hydatid cyst infestation

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ABSTRACT

Although the most common cause of obstructive jaundice is viral infections, there are some rare disorders which can cause this picture. We present a 15-year-old girl, admitted with an obstructive jaundice. Her laboratory tests including viral infections were normal. The diagnosis of hydatid cyst was performed with both ultrasonography and indirect hemagglutination assay. The patient was operated on and hydatid cyst was seen to be ruptured into the choledochus. In conclusion, nonviral causes of hepatitis are less common, but must be considered in the differential diagnosis. An important complication of hydatid disease is rupture to biliary tree. This situation may cause obstructive jaundice.

Key Words: Jaundice, hydatid cyst, choledochus, hepatitis

Introduction

Acute hepatitis is characterized by inflammation and necrosis of the liver. The underlying triggering factor for the inflammatory process may be toxic, autoimmune, infective or metabolic. Although the most common cause of obstructive jaundice is viral infections, there are some rare disorders which can cause this picture. One of them is a hydatid cyst infestation (1). We reported a rare case presented with obstructive jaundice and diagnosed as hydatid cyst infestation.

Case report

A 15-year-old girl was admitted to our clinic with complaints of jaundice and abdominal pain. Physical examination revealed hepatomegaly (4 cm palpable at the costal margin), and icter. Splenomegaly was not present. The results of laboratory tests were as follows: Hemoglobin 10.3 g/dL; leukocyte 11,300/mL; platelet 463,000/mL; alanine aminotransferase (ALT) 103 IU/L; aspartate aminotransferase (AST) 77 U/L; total bilirubin 22 mg/dL; direct bilirubin 18.8 mg/dL; total protein 5.8 g/dL; albumin 2.4 g/dL; alkaline phosphatase 1625 IU/L. Prothrombin time, partial thromboplastin time, and INR levels were normal. Anti-hepatitis A virus (HAV) immunoglobulin (Ig) G was positive, and anti-HAV IgM was negative. Other viral tests for hepatitis B, hepatitis C, cytomegalovirus, Epstein-Barr virus, parvovirus

B19 did not indicate an acute infection. C-reactive protein was 117 mg/L (range: 0-5 mg/L).

Abdominal ultrasonography revealed a cyst, in the segment 4 of liver with 7x5 cm in diameter, and a dilated choledochal diameter (15 mm). Then, abdominal computerized tomography was performed and it was commented that a hydatid cyst was ruptured into choledochus (Figure 1). Indirect hemagglutination test for hydatid cyst was positive.



Fig. 1. Computerized tomography shows a hydatid cyst and dilated choledochus.

Therefore, the patient was operated on. In operation, hydatid cyst was seen to be ruptured into choledochus. Cholecystectomy, choledocotomy, removal of cyst membranes

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which obstructed choledochus and drainage procedures were administered. Any complication did not develop. Albendazole treatment was begun at a dose of 400 mg/day. Seven days after the operation, the laboratory tests improved (ALT 32 IU/L; total bilirubin 3 mg/dL; direct bilirubin 2 mg/dL) and the patient was discharged.

Discussion

Hydatid disease, a zoonotic infection, is caused by adult or larval stages of cestodes belonging to the genus *Echinococcus* and the family *Taeniidae* (2). Human infection with *Echinococcus granulosus* may occur after ingestion of ova excreted by infected dogs. Dogs generally acquire infection via consumption of sheep liver or intestine containing hydatid cysts (3). Ingestion of eggs typically occurs after the handling of an infected dog or the drinking of contaminated water. The ingested embryo, after release from the egg in the duodenum, penetrates the intestinal mucosa and enters the portal circulation (3).

Hydatid disease is still an important public health problem in many endemic countries. While it can develop anywhere in the human body, the liver and the lungs are the most frequent locations (1-3). Other sites of infection in approximately 10% of children include the brain, bones, genitourinary tract, eyes, spleen, and heart (3). Our case had a cyst solely in the liver.

Infestation is common in childhood, although symptoms may not occur for many years (3). Hepatic cysts are usually slow growing and lead to asymptomatic hepatomegaly, but may manifest due to secondary infection or greatening in size (1). Hepatic involvement is marked by the development of cysts within the hepatic parenchyma, most often within the right lobe (3,4). Hydatid cyst of our case within the left lobe (Segment 4). Typically, the cyst is surrounded by a fibrous capsule. An acellular, hyalinized layer forms the exocyst, underlaid by a germinal layer. Extrusions of the germinal layer form brood capsules which contain protoscolices. Hydatid sand, composed of separated brood cysts and protoscolices, floats within the main cyst cavity. Septation may occur, as may formation of daughter cysts (3).

A common complication of hepatic hydatid disease is the rupture of the cyst caused by the increased pressure within it. Cysts may rupture into the biliary tree, the peritoneal or pleural cavity, the pericardium, the gastrointestinal tract,

or even into blood vessels (5). Hydatid disease was reported as noncomplicated in 46.3% of the patients. The most common complications were reported intrabiliary rupture (34.4%), cyst infection (32.7%), and their combination (24.5%) (6).

Hydatid cyst rupture into the biliary tree may involve the common hepatic duct, lobar biliary branches, or the small intrahepatic bile ducts. In such cases, the suggestion of the diagnosis may be obtained by ultrasonography (7). Intrabiliary rupture can occur with two different clinical picture: Occult communication (10%-64%) and frank intrabiliary rupture (3%-35%). The occult rupture is usually silent and may be accompanied by suppuration or it can progress to a frank rupture. In the frank rupture, daughter vesicles and fragmented membranes escape into the biliary tree causing obstruction, cholangitis or septicemia (5,8,9). Communications with the biliary system were reported to be encounter in 21% of the patients with hepatic hydatid cyst. A history of jaundice had been taken in 25% of them (8).

In the diagnosis of liver hydatid cysts, abdominal ultrasonography was reported to give correct diagnosis in 94% of the patients (9). Indirect hemagglutination assay confirms the diagnosis in 80% to 94% of hepatic hydatidosis cases (10). In a study (11), hepatic cystic echinococcosis was detected in 0.3% by ultrasonography, while 8.9% and 10.1% of the cases were found to be positive for cystic echinococcosis by ELISA and indirect hemagglutination, respectively. In our case, the diagnosis of hydatid cyst had been confirmed with both ultrasonography and indirect hemagglutination assay. Albendazole has been known to be a useful choice in the management of cystic echinococcosis both when used as sole treatment or as an adjunct to surgery (12).

In conclusion, nonviral causes of hepatitis are less common, but must be considered in the differential diagnosis. An important complication of hydatid disease is rupture to biliary tree. This situation may cause obstructive jaundice. Therefore, abdominal ultrasonography must be performed in differential diagnosis for a case presenting with obstructive jaundice.

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