Case Report

Hydatid cyst of the gall bladder-A rare location

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Abstract. A 70 year old female presented with intermittent, dull aching pain in right upper abdomen lasting for four months . Examination revealed hepatomegaly and a hard globular swelling continuous with the right costal margin. Enzyme linked immunosorbant assay for hydatid was positive. Ultrasonography of abdomen revealed a large multicystic lesion in right lobe of the liver and hyperechoic shadows in gall bladder fossa. Contrast enhanced computerized tomography of abdomen documented huge hydatid cyst in right lobe of liver and a thick walled calcified cystic lesion in segment-VI of the liver which was confirmed as a gall bladder at laparotomy. Histopathological examination of the specimen confirmed hydatid cyst of the gall bladder. Larval stage of echinococcus causes hydatid disease. Four species can produce disease in humans. E. granulosus and E.multilocularis cause cystic and alveolar hydatid disease respectively while E. vogeli and E. oligarthus rarely infect humans. Hydatid cyst of the gall bladder is extremely rare and this case report highlights the rarest location of hydatid disease.

Key words: Gall bladder, hydatid cyst

1. Introduction

Hydatid disease has a worldwide distribution. It is a parasitic infection of the liver and other organs caused by the tapeworm *Echinococcus*, most commonly *E. granulosus*. It is a 5 mm long hermaphroditic tapeworm, that has dog , foxes or coyotes as a definitive host and sheep, swine or cattle as an intermediate host. Human are the accidental intermediate host and infection in them represents dead end of the parasite (1,2).

Once within the human or other intermediate host, the ingested eggs hatch in the duodenum to release the larvae that penetrate the mucosa of small intestines to enter the portal circulation. Liver is the most common site of involvement (65-75%) while lungs are involved in 10-25% of cases (1).

If larvae are not trapped in liver or lungs, it may lodge itself in any part of the body including peritoneal cavity (8-18%), spleen (2-3%), kidneys (1- 4%), uterus and adnexia (0.5-1%), retroperitoneum (0.5-1%), pancreas (0.5-0.8%), and subcutaneous sites (1-2%). Other rare sites for involvement by the hydatid disease include heart, brain, thyroid, vertebrae, breast and gall bladder (incidence of <1% each) (1-7). Concurrent occurrence of gall bladder hydatid cyst along with liver cysts, especially when biliary channels are clear of cysts, is very rare, as seen in our patient (5).

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2. Case report

A 70 year old female presented with the complaint of pain in right upper abdomen of four months duration. The pain was gradual in onset, mild in severity, intermittent, dull aching, not referred, radiated or shifted anywhere, with no aggravating or relieving factors. There was no other significant history like biliary colic, jaundice, fever, rigors or chills. General physical examination revealed only mild pallor. On abdominal examination, a bulge was seen in right upper abdomen. Liver was enlarged 4-5 cm below costal margin. A swelling was palpable which was continuous with the lower edge of the liver. It was about 8×6 cm in size, globular in shape, with well defined margins, smooth surface, non tender, hard in consistency, fixed to liver, and dull on percussion. Rest of the examination was normal as were the haemogram, kidney function, liver function, and chest X-ray. Enzyme linked immunosorbent assay (ELISA) for hydatid disease was positive. Abdominal ultrasonography (USG) revealed a large multicystic lesion, 12.3 ×11.8 cm in the right lobe of liver, with gall bladder fossa showing hyperechoic shadows (Figure 1).

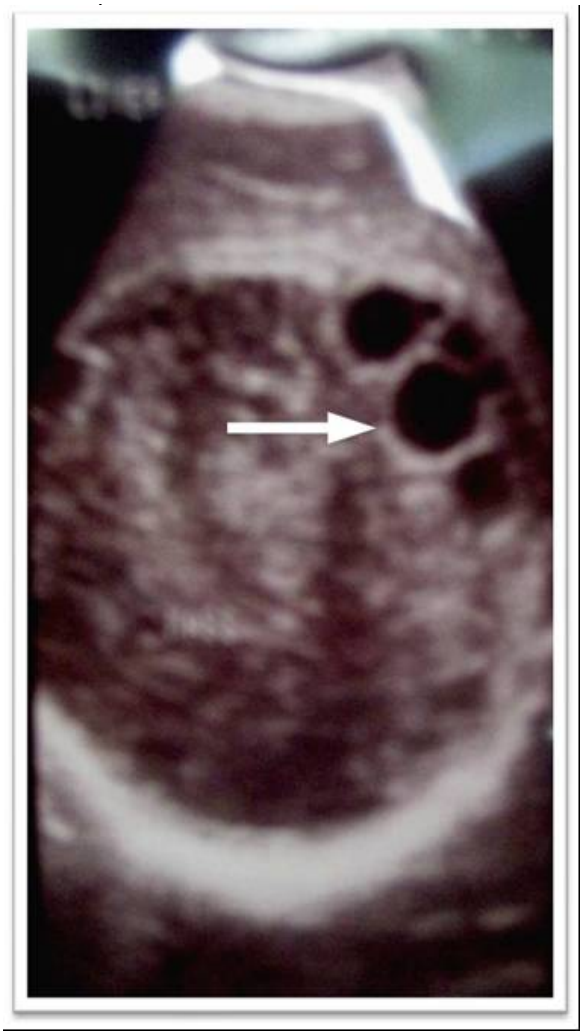

Fig. 1. Abdominal ultrasonography revealed a large multicystic lesion, 12.3×11.8 cm in the right lobe of liver (arrow), with gall bladder fossa showing hyperechoic shadows.

Contrast enhanced abdominal computerized tomography (CECT) documented a cyst at water density, 11×12 cm in size, with well defined wall and internal daughter cysts seen in right lobe of the liver. Another cystic lesion with thick calcified wall was seen in segment-VI of liver measuring 5×5 cm. A calcified area 2 cm in diameter was also seen medial to the above calcified cystic lesion. The gall bladder was not seen separately from the calcified cystic lesion. The intrahepatic biliary radicles (IHBR) and common bile duct (CBD) were normal (Figure 2). Intra-operative exploration revealed that almost whole right lobe was converted into a cyst, with adhesions of its surface with the parietal wall. Multiple daughter cysts and 2-3 cysto-biliary communications about 2-3 mm in diameter were seen within the liver hydatid cyst cavity.

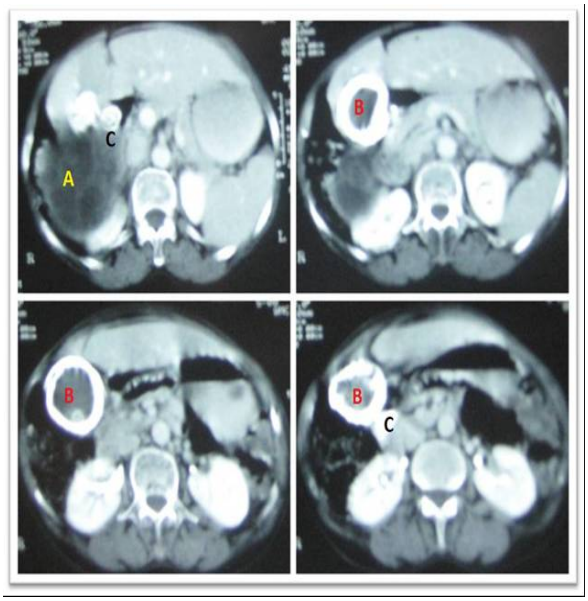

Fig. 2. Contrast enhanced abdominal computerised tomography documented hydatid cyst in right lobe of the liver (A). Another cystic lesion with thick calcified wall was seen in segment-VI of liver measuring 5×5 cm (B). A calcified area 2 cm in size was also seen medial to the above calcified cystic lesion (C). The gall bladder was not seen separately from the calcified cystic lesion. IHBR and CBD were normal.

There was compensatory hypertrophy of the left lobe of the liver. Gall bladder was completely calcified (6×7cm), and converted into hard structure like a shell (Figure 3) with a stone 2.5 cm in diameter impacted at its neck. No communication was found between calcified gall bladder hydatid and the liver cyst. Cystic duct and CBD were normal. The patient was subjected to partial cystectomy, closure of the cystobiliary communications using 2/0 vicryl, tube drainage of the residual cavity and cholecystectomy using fundus first method, as the anatomy at calots triangle was distorted. Surgery was completed in 2 hours, and the patient was discharged on 12th day after cavitogram revealed no residual cystobiliary communication. Patient did not have any complaints on follow-up. Histopathological examination of the calcified gall bladder revealed the gall bladder mucosa with underlying laminated membranes of hydatid cyst (Figure 4).

3. Discussion

Hydatid disease is seen endemically in Middle East as well as in other parts of the world, including Africa, India, South America, Turkey and Southern Europe (7). Involvement of the gall bladder by hydatid disease is extremely rare and is usually due to intrabiliary rupture of the liver hydatid or by direct cyst rupture into the gall bladder (4,8).

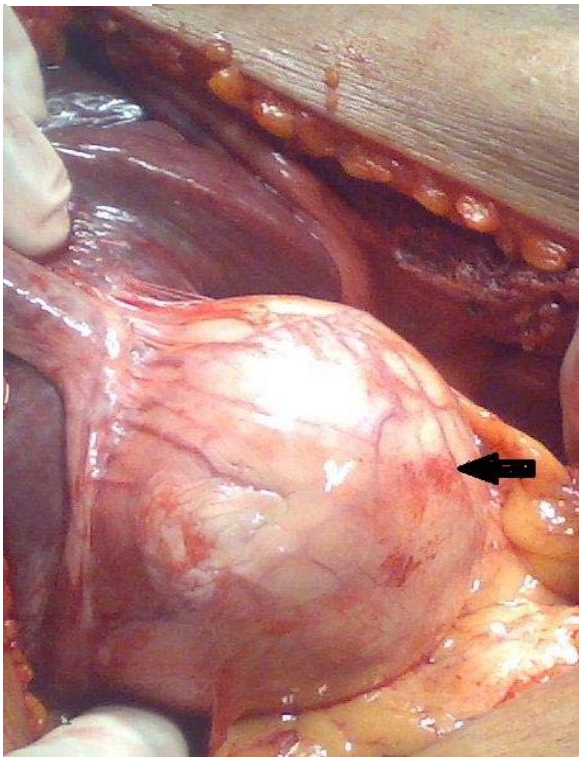

Fig. 3. Intraoperatively, calcified gall bladder is shown. Arrow shows the hard shell like structure.

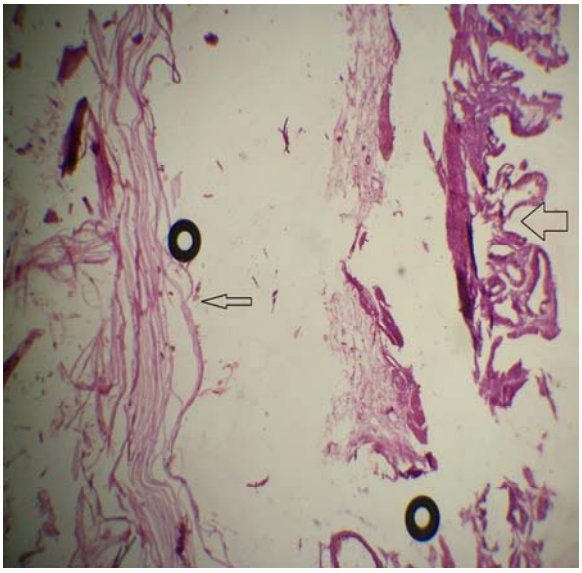

Fig. 4. Histopathology shows gall bladder mucosa (thick arrow) with underlying laminated membrane of hydatid cyst (thin arrow).

The diagnosis of uncomplicated hydatid cyst of liver depends on clinical suspicion. The symptomatic cysts are more than 5 cm in diameter and can present with abdominal pain, hepatomegaly, or a palpable abdominal lump. Rupture of the cyst into biliary tree causes biliary colic and jaundice (9), while intraperitoneal rupture may cause urticaria, anaphylaxis or implantation of cysts into other viscera (10). Our patient presented only with dull aching pain in right upper abdomen and abdominal examination revealed hepatomegaly and a hard right subcostal lump.

Routine laboratory tests can only reveal eosinophilia. Diagnosis is established by imaging and serological tests. Highly sensitive tests include indirect haemagglutination test (IHA), and latex agglutination test (LA). Specific tests include double diffusion test (DD), immunoelectrophoresis (IEP), ELISA, and radioallergosorbant test (RAST) (1). In our patient, IHA and ELISA suggested hydatid disease. Abdominal USG, CECT, and MRI are highly sensitive and specific and can reveal the morphological characteristics of a cyst, its exact site, size, number, its relation with surrounding structures, and can distinguish it from other lesions (7,11). In our patient, abdominal USG documented a large multicystic lesion in the right lobe of liver with hyperechoic shadows in the gall bladder fossa. Abdominal CECT also revealed hydatid cyst in right lobe of the liver along with another thick calcified cystic lesion in segment - VI of the liver.

Medical treatment alone may be effective in 30-40% of the cases. It is most effective in alveolar hydatid, less so for liver infection, and essentially ineffective for the diseases of bone, brain, eye, gall bladder and other sites (1). Many authors recommend preoperative use of antihelmenthics to sterilize the cyst, and reduce the chances of spillage associated anaphylaxis and dissemination at surgery (10). Postoperative medical treatment reduces the chances of recurrence (12). Our patient received albendazole (10mg/kg/day) for 4 weeks preoperatively. Surgery still remains the mainstay of treatment for hydatid disease. The principles of surgery include removal of the cyst contents without contaminating the patient, and appropriate management of the residual cavity (13). In suspected cases of hydatid disease of the gall bladder, open cholecystectomy is the treatment of choice and minimal access approach should be avoided because of the risk of perforation and dissemination of disease (peritoneal hydatidosis) (1). Our patient was subjected to exploratory laparotomy through a right subcostal incision. There was compensatory hypertrophy of the left lobe of the liver as almost whole of the right lobe was converted into a cyst which probably was not contributing adequately to liver function. All the principles of surgery were applied to the patient. Liver hydatid cyst was dealt first. Scolicidal

(Citrimide 0.5%) soaked packs were used to isolate the operative field. Cystobiliary communications were closed using 2/0 vicryl and cholecystectomy was performed. The gall bladder fossa was inspected for any bile leak or any communication with the above lying hydatid cyst, which was not found in our case. The drain in the cavity was removed on 12th postoperative day after a cavitogram which did not reveal any communication with the biliary tree. Postoperatively, patient was put on 3 cycles of albendazole for 21 days each with a gap of 1 week in-between.

The sections of calcified gall bladder on histopathological examination confirmed hydatid cyst of the organ.

Communication with biliary tree has been described in up to 90% cases of liver hydatid cyst (14). This is due to incorporation of biliary radicals into the pericyst (15). However frank rupture into biliary tree occurs in only 5 -15% of cases (14). Direct cyst rupture or communication between hepatic hydatid cyst and gall bladder is rare (8). In our patient, the gall bladder involvement was probably due to small cystobiliary communications which may have allowed small leak of the cyst contents into the biliary tree and involvement of the gall bladder via cystic duct. Other rare routes of spread to gall bladder (bloodborne / lymphatics) could not be ruled out as the patient did not have any symptom or sign suggestive of frank rupture of liver cyst into the biliary tree.

Cystic lesions of gall bladder should not be overlooked especially in gall stone disease (16) and in regions where hydatid disease is endemic. Hydatid disease of gall bladder is rare and should be kept in differential diagnosis of a cystic lesion of the organ in endemic areas (5).

To conclude, hydatid disease can affect any organ in the body and a high suspicion of disease is justified in a cystic lesion of any organ in endemic region, with a preoperative diagnosis being possible radiologically and serologically (7).

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