

Disseminated Intra-Abdominal Hydatidosis: A Case Report

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Abstract—Hydatid cyst disease is a zoonotic disease caused by the larval stage of *Echinococcus granulosus* and *Echinococcus Multilocularis*. In human beings, apart from involving the liver and lungs commonly, it also affects other organs like brain, kidney and spleen. Rupture of Hydatid cyst into abdominal cavity causes disseminated abdominal hydatidosis which is a rare complication. Here this rare case was presenting as a 48 years old female patient of disseminated intra-abdominal hydatidosis. Disseminated abdominal hydatidosis occurs secondary to traumatic or surgical rupture of a hepatic cyst. Ultrasonography or Computed Tomography findings are helpful in making a definitive diagnosis. For localized hydatid cysts in liver or lungs, the management of choice is preferably surgical while the treatment for disseminated intra-abdominal hydatidosis remains medical. Albendazole is the treatment of choice for disseminated abdominal hydatidosis.

Keywords: Zoonotic, Disseminated, Abdominal, Hydatid Cyst

I. INTRODUCTION

Hydatid disease (HD) is a zoonotic infection caused by the parasitic cestode tapeworm *Echinococcus Granulosus* and *Echinococcus Multilocularis*¹. In humans, Hydatid disease involves the liver (75%) and the lungs (15%). Some cases include the other regions of the body like brain, kidney and spleen². Disseminated abdominal hydatidosis is a rare presentation. Non-symptomatic hydatid disease may present with complications, but unusual locations as well as multiple primary or secondary hydatid disease pose special therapeutic hurdles.

Here a very rare case of disseminated intra-abdominal hydatidosis involving liver, spleen, and ovary and at adnexal spaces with calcification and septations was reported.

II. METHODOLOGY

A typical case of Intra-abdominal Disseminated abdominal hydatidosis attended in Medicine OPD of SMS Hospital, Jaipur. So case study was done thoroughly and case report was prepared to publish this rare case.

III. CASE REPORT

A 48 years old female presented to medicine outpatient department with the complaint of abdominal pain for one year. The pain was insidious in onset, moderate in intensity, intermittent, not radiating, aggravated by movements and relieved by lying down. It was associated with low grade fever, fatigability, decreased appetite, undocumented weight loss, constipation, dyspepsia and nausea. Patient had past history of similar complaints 5 years back for which she was operated and intra-abdominal cystic lesion had been evacuated.

On examination she was stable and afebrile, vitals were also normal; pulse: 78 beats/minute, blood pressure: 110/70mm of Hg (right arm supine position), respiratory rate: 17/minute. She had mild pallor

but no lymphadenopathy, cyanosis, or jaundice. Abdominal examination revealed tender hepatomegaly with tenderness in epigastric and left lumbar regions. Respiratory systemic examination revealed decreased air entry at right infrascapular area with dull note on percussion. Cardiovascular, nervous system and musculoskeletal examination were within normal limits.

Hematological and biochemical profiles were as follows; TLC: $26.3 \times 10^9/L$ ($4-10 \times 10^9/L$), haemoglobin: 10 gm%, absolute eosinophil count: $0.12 \times 10^9/L$ ($0.04-0.45 \times 10^9/L$), hs C-reactive protein: 446 mg/L ($<1\text{mg/L}$), Erythrocytes sedimentation rate of 44 mm/ 1stHour (<20 mm/Hr) and urea: 58 mg/dL (17-43 mg/dL), creatinine: 1.07mg/dL(0.5-1.0 mg/dL), SGOT: 43U/L ($<35\text{U/L}$), SGPT: 37 U/L (<35 U/L), alkaline phosphatase: 254 U/L (30–120 U/L), total bilirubin: 1.42mg/dL (0.2–1.2mg/dL), and direct bilirubin : 0.56 mg/dL and albumin/globulin ratio was 0.74.

Abdominal contrast enhanced computed tomography (CT) revealed a large multiloculated well defined cystic intrahepatic mass of size 16x12x11 cm involving right lobe of liver with foci of calcification. There was a cystic lesion of 8x9x10 cm seen in spleen without any septae and calcifications, and large multilocular cystic structure of size 22x9x5 cm was also seen in the left Para umbilical region of peritoneal cavity; bilateral adnexa also showed multilocular cystic structures.(Figures 1 & 2).

Figure: 1
CECT Abdomen suggestive of hepatic and splenic Hydatid cyst



Figure: 2
CECT Abdomen suggestive of intraperitoneal Hydatid cystic mass



IV. DISCUSSION

Hydatid disease is a unique parasitic disease which can occur almost anywhere in the body and demonstrates a spectrum of imaging features that vary according to growth stage, associated complications, and affected tissue³.

Peritoneal hydatidosis is a rare entity of hydatid disease. Secondary peritoneal disease occurs as a result of traumatic or surgical rupture of a hepatic cyst.

Mostly patients remain asymptomatic for long time. Symptoms arises due to enlarging abdominal cysts or rupture which may present as acute abdominal pain or vague abdominal symptoms such as nonspecific dull aching pain, abdominal fullness, dyspepsia and vomiting. Compression of a bile duct or leakage of cyst fluid into the biliary tree may mimic recurrent cholelithiasis and biliary obstruction can result in jaundice¹. Acute allergic reaction may occur due to spillage of antigenic fluid into the peritoneal cavity.

The diagnosis is based on the ultrasonography and Computed Tomography findings which may be supplemented by specific IgG antibodies and ELISA (Enzyme Linked Immuno-Sorbent Assay) tests.

The sensitivity of various serological tests used for hydatid disease varies from 64 to 87 %⁴. In recent times, many PCR (Polymerase Chain Reaction) protocols have been published on the identification of DNA of *Echinococcus granulosus* from eggs or from adult parasites and many new ways of diagnosing this cestode are under development⁵.

Management for Hydatid disease is relied on considerations of the size, location and manifestations of cysts and the overall well being of the patient. For localized hydatid cysts in liver or lungs, the management of choice is preferably surgical while the treatment for disseminated intra-abdominal hydatidosis remains medical³. Surgery can be performed with removal of the cyst intact if possible after first sterilising the cyst with formalin or alcohol.

However, perioperative oral course of albendazole should be considered to sterilise the cyst, as it decreases the risk of anaphylaxis and reduces the post-operative recurrence rate⁶. Albendazole's active sulfoxide metabolite achieves high cystic concentrations and is also active against both the germinal membranes and protoscolices⁷.

The development of disseminated peritoneal hydatidosis in our patient was attributed either to the rupture of her primary hepatic lesion or spillage from her previous operation. She was started on Albendazole 400mg twice daily and surgical opinion was taken. After 3 months of medical management, patient improved satisfactorily.

V. CONCLUSION

Disseminated abdominal hydatidosis is a rare presentation of Hydatid disease. This is a slowly progressive disease with symptoms that may last for long time as in this patient. For localized hydatid cysts in liver or lungs, the management of choice is preferably surgical while the treatment for disseminated intra-abdominal hydatidosis remains medical. Medical therapy with albendazole has shown better results than surgical treatment and is now considered as drug of choice for disseminated peritoneal hydatidosis

CONFLICT OF INTEREST

None declared till now.

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