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Giant abdominal hydatid cyst masquerading as ovarian malignancy

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ABSTRACT

Hydatid disease, caused by *Echinococcus granulosus*, is a common parasitic infection of the liver. Disseminated intra-abdominal hydatid disease may occur with the rupture of the hydatid cyst into the peritoneal cavity, producing secondary echinococcosis. Occasionally, the cyst may not rupture, and instead, enlarge, thus mimicking gross ascites or huge ovarian tumours. We present a 30-year-old woman with a giant intra-abdominal hydatid cyst communicating with the liver, and discuss the management of her case.

Keywords: ascites, *Echinococcus*, hydatid disease, ovarian tumour

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INTRODUCTION

Echinococcosis (hydatid disease), a zoonosis, is characterised by worldwide distribution and frequent hepatic involvement. It is caused by the larval stage of *Echinococcus granulosus*, a parasite of the order Cestoda and family Taeniidae. Humans are the accidental dead-end intermediate hosts. Animals (e.g., dog, wolf, fox and jackal) are definitive hosts, while pigs, cattle, horses and goats are intermediate hosts. Hydatid cysts are characterised by indolent yet unremittent growth in the majority of patients, with the potential for cyst metastasis to the peritoneal surface and the lungs. We present a case of intraperitoneal hydatid disease with a continuous communicating cavity, and review the literature.

CASE REPORT

A 30-year-old woman presented to the Department of Obstetrics and Gynaecology with a history of abdominal distension for three years. The abdominal swelling had been gradually increasing in size. The patient had persistent abdominal pain, which was dull aching in nature and localised to the lower abdomen. There was a loss of weight and appetite associated with nausea, with no history of jaundice and no alteration in bladder habits. The swelling was so enormous that it made the patient breathless even at rest. The patient was P4L4, married for 14 years, with her last menstrual period four months ago. The pregnancy test was negative. The patient had sought treatment from a local practitioner but did not get any relief. No definite diagnosis was made at that time.

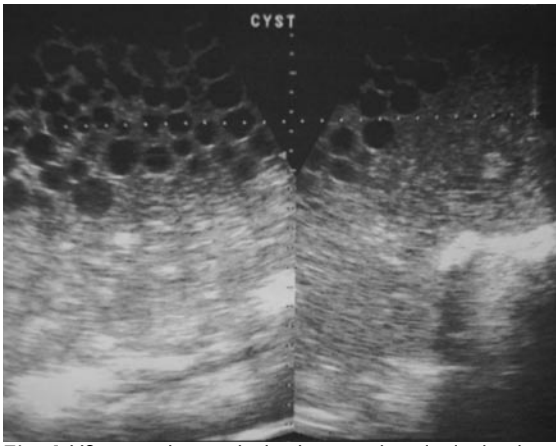

Fig. 1 US image shows a hydatid cyst with multiple daughter cysts and hydatid sand.

Physical examination revealed moderate general cachexia. There was a large mass arising from the pelvis, measuring 20 cm × 20 cm × 15 cm, rising above the umbilicus to the diaphragm. The abdominal girth measured 118 cm. Hepatosplenomegaly could not be assessed. The mass was dull on percussion with a fluid thrill. Shifting dullness was not present, indicating either a tense collection of, or a singular huge mass. Further investigations were conducted on the patient to address clinical suspicious of ovarian malignancy. Laboratory investigations revealed a haemoglobin level of 8.2 g/dL, neutrophilic leucocytosis (16,000/mm^3), alkaline phosphatase was 2.35 nkat/L, serum bilirubin total 26.65 μmol/L, direct 8.55 μmol/L , SGOT 0.367 μkat/L and SGPT 0.683 μkat/L.

Ultrasonography of the abdomen and pelvis revealed a huge cystic mass lesion with a honeycomb appearance. It had a mobile matrix with a few calcific foci within, and occupying the abdomen and pelvis (Fig. 1). The mass was reported to be a hydatid cyst with multiple daughter cysts and hydatid sand within. The cysts were multiloculated with multiple internal septations, suggestive of hydatid disease. The

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patient was immediately transferred to the surgical ward. Emergency exploratory laparotomy with enucleation of the hydatid cyst was performed. Intraoperatively, the pericyst was found adherent to the peritoneum and was not separated. The bowel loops were felt through the pericyst. 15 L of fluid with multiple daughter cysts and hydatid sand were removed. The pericyst cavity was thoroughly washed with betadine solution. A small communication was seen with the liver in the form of an indentation, which was also covered with pericyst. The drain was kept in the corresponding subhepatic and pelvic area of the pericyst, and the pericyst wall was closed. The patient made an uneventful recovery with return to a normal appetite. The patient was kept under regular follow-up and to date, there is no recurrence of the hydatid cyst.

DISCUSSION

A typical hydatid cyst is formed from its embryo. It consists of three layers.[1] The outer layer (pericyst or adventitia) consists of fibrous tissue, is grey in colour, and blends immediately with the liver. It is formed from the host tissue as a result of chronic inflammatory reaction to the parasite. The pericyst usually increases in thickness as the cyst expands. Liver and spleen hydatid cysts have a thick pericyst, as compared to peritoneal hydatid cysts, in which the pericyst is extremely thin. The hydatid cysts in the lung and brain have no pericyst at all. The present case was an exception as a thick pericyst was encountered. As the cyst grows in the liver, bile ducts and blood vessels stretch and finally become incorporated within this structure, thus accounting for the propensity for biliary and haemorrhagic complications of cyst growth and resection. With time, portions of the pericyst may calcify. Complete calcification of the pericyst may interrupt the nutrient and oxygen supply to the parasites and thus marks the death of the hydatid cyst.

The parasite constitutes the laminated membrane (ectocyst) and the germinal layer (endocyst). The ectocyst has the appearance of the white of a hard-boiled egg. It is elastic, made up of gelatinuous, chitinous material and when incised or ruptured, curls in on itself, exposing the inner layer. The innermost germinal layer is cellular and consists of a number of nuclei embedded in a protoplasmic mass. It is a very thin, vital layer of the cyst, and produces brood capsules with scolices, secretes hydatid fluid and forms the outer layer. The cyst fluid is crystal clear and colourless with a specific gravity of 1,005 to 1,010, is slightly alkaline, and is highly antigenic and toxic. Contact with the fluid can give rise to anaphylactic shock. Hydatid cysts expand slowly and asymptomatically, and thus, tend to be quite large at presentation. The high secretion pressure is responsible for the progressive enlargement of the cyst. Fluid pressure within a hydatid cyst can reach up to 70 cm of water. Bile-stained cyst fluid is an indicator of communication of the cyst with the biliary tract.[1]

Complications are seen in one-third of patients with hydatid cysts. The most common complication encountered is rupture of the cyst, which can occur internally, or less frequently, externally. Internal rupture occurs due to trauma or pressure necrosis from the growing cyst. External rupture may occur into the biliary duct, thoracic cavity or peritoneal cavity. The cyst can rupture into the stomach, duodenum, small intestine or into the general peritoneal cavity as well. This may result in anaphylactic shock and formation of localised or generalised secondary echinococcosis.[2] In the present case, the hydatid cyst had not ruptured but had started expanding from the liver, and invaded the abdomen. Approximately 80% of hydatid cysts are single and located in the right lobe of the liver. In one quarter of cases, there are multiple cysts. The incidence of intra-abdominal hydatid disease is still small. Hydatid disease remains asymptomatic for a long period of time. When symptoms appear, pain is the commonest symptom of hydatid disease. It may occur as an acute onset if the cyst ruptures or is secondarily infected, or as an insidious onset which presents as a continuous dull ache. Fever with chills and rigors can occur if the cyst is secondarily infected. Jaundice, biliary colic and urticaria will develop if there is an intrabiliary rupture of the cyst.[3] Intrathoracic rupture may lead to shoulder pain and phlegmatic cough containing blood and bile.[4]

Ultrasonography and computed tomography are both excellent imaging modalities for the detection of hydatid cysts. Ultrasonography is cost-effective in endemic areas and when the diagnosis of hydatid cyst is certain. However, ultrasonography is less accurate in localising and delineating the extent of the cyst. The sensitivity of computed tomography is 90%–100%.[5] It provides a three-dimensional view and delineates the cyst, which is useful when diagnosis is uncertain, or when rupture or infection has occurred.[6] If a complication in the biliary tree is suspected, endoscopic retrograde cholangiopancreatography should be carried out to delineate the biliary connections within the hydatid cyst wall. The presence of biliary complications may alter the operative management.

The World Health Organisation has recently outlined the treatment guidelines for hydatid cysts. Surgery is the treatment of choice for all patients with symptomatic disease and who are fit for

surgery.[7] The goals of surgery are removal of cyst, prevention of spillage, and preservation of liver function. Albendazole is given both preoperatively and postoperatively. Not only does it soften the cysts and facilitates removal during the operation, it also prevents recurrence after the operation. The dose duration is five days before, to one month after the operation. Further dose can lead to hepatotoxicity. The other alternatives are chemotherapy with albendazole or mebendazole, and PAIR therapy (puncture, aspiration, injection, re-aspiration) with concomitant chemotherapy. The efficacy of sole medical therapy is limited. Antihelminthics work best when prescribed for small, unilocular, hydatid cysts. Successful treatment for such cases has been reported in up to 40% of cases. In PAIR therapy, ultrasound-guided percutaneous aspiration of cysts is done, followed by injection of protoscolicidal substances (such as, 20% sodium chloride solution, 95% ethanol or betadine solution). The solution is left on for a contact period of a minimum 15 minutes and then re-aspiration of the fluid cyst content is done. The indications of PAIR therapy are: (1) large, multiple cysts of the liver, spleen, kidney and bones; (2) inoperable cases; and (3) relapses after surgery. The contraindications are lung cysts and communicating cysts.

The hydatid cyst is managed surgically by partial or total cyst resection. Once the cyst is exposed and isolated from the remainder of the field, partial resection is done by unroofing the hydatid cyst, removing the laminated and germinal membranes, and leaving a portion of pericyst. In case of unilocular cyst, omentoplasty is done; in multilocular cyst, simple drainage is done with the placement of drain. If the cystic cavity is reapproximated, there are fewer chances of biliary fistulas. In extrahepatic hydatids, the pericyst is very thin, and therefore, a plane of dissection can always be found. The recurrence rate of hydatid disease after surgical treatment is approximately 2%. The survival rates in patient undergoing operative intervention is 95%. A postoperative long term follow-up regimen is essential. Repeated imaging every six weeks is also essential.[1]

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