HYDATID CYST OF THE KIDNEY

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Clinical Presentation:
A 60 years old female suffered from dull pain and heaviness in her right flank since the last 10-15 years. In February 2007, she presented with colicky pain of several hours duration at the same site. Pain was not associated with nausea and vomiting. There was no fever. Abdominal examination demonstrated a pal-pable right kidney that was painful. Small bowel peristalsis was normal, and the liver was not en-larged. The remainder of the systemic examination was normal.

IMAGING FINDINGS

Ultrasonography (US) revealed a well-defined 14×12cm sized, hypoechoic multicystic mass with multiple hyperechoic septae arising from the upper pole of right kidney. No definite solid component was seen. No calcification or hydronephrosis seen in the kidney. No flow was picked up on colour doppler in the septae or walls. Rest of the viscera were unremarkable.

US was followed by Computed Tomography (CT) of the upper abdomen which showed hypoattenuating expansile, multilocular cystic mass arising anteriorly from upper, mid pole of right kidney. Post contrast CT showed faint enhancement of the wall and septae. Density varied from 3-25 HU.

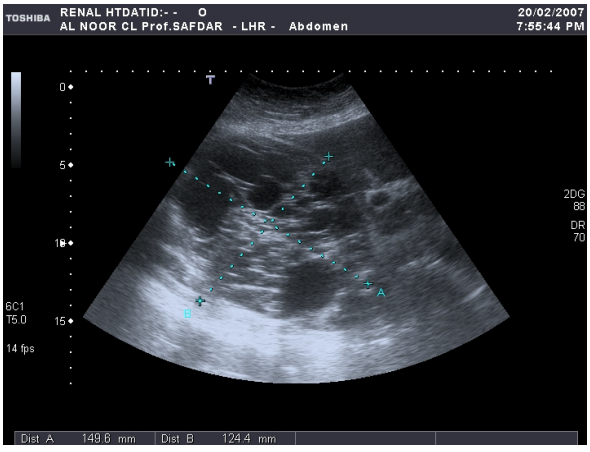

Fig. 1: Multi cystic mass with locules and septae.

LABORATORY FINDINGS

Immunological tests were carried out, they revealed elevated echinococcus antibody titers. US guided cyst aspiration was done which showed crystal clear fluid. The aspirate was negative for malignant cells. No other contents were found.

SURGERY

Later nephrectomy was performed and a large whitish cyst was removed with the kidney. No other similar cysts were found in the abdomen.

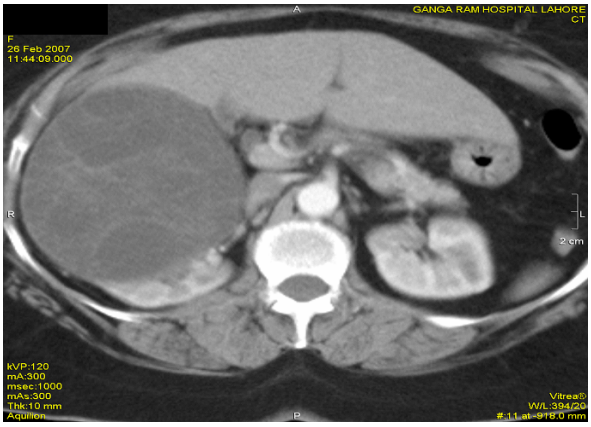

Fig. 2: Post contrast CT showing faint enhancement of wall and septae.

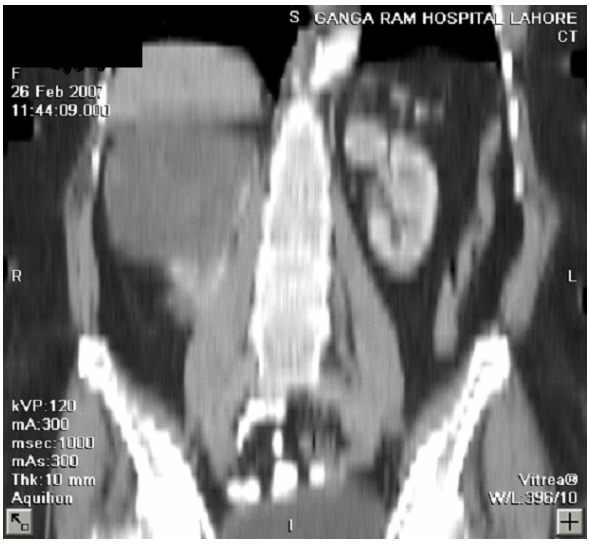

Fig. 3: Coronal reconstruction showing the right renal mass

DISCUSSION

Echinococcosis is a worldwide zoonosis produced by the larval stage of the *Echinococcus* granulosus. The adult worm lives in the proximal small bowel

of the definitive host (dog), attached by hooklets to the mucosa. Eggs are released into the host's intestine and excreted in the faeces. Humans may become intermediate hosts through contact with a definitive host (usually a domesticated dog) or ingestion of contaminated water or vegetables. The ovum loses its protective layer as it is digested in the duodenum. Once the parasitic embryo passes through the intestinal wall to reach the portal venous system or lymphatic system, the liver acts as the first line of defense and is therefore the most frequently involved organ. In humans, hydatid disease involves the liver in approximately 75% of cases and the lung in 15%. Secondary involvement due to haematogenous dissemination may be seen in almost any anatomical location[1]. Kidney involvement in echinococcosis is extremely rare (2%–3% of cases), even in areas where hydatid disease is endemic[2]. Renal hydatid cysts usually remain asymptomatic for many years. There are no pathognomonic clinical signs except cystic rupture into the collecting sys-tem, which leads to acute renal colic and hydatid-uria.[3,4]

Imaging findings in hydatid disease depend on the stage of cyst growth (ie, whether the cyst is unilocular, contains daughter cysts, or is partially or completely calcified [dead])[1]. A difference in attenuation and signal intensity between the fluid in the central portion of the cyst and that in the peripheral cysts is a typical finding in echinococcosis due to a difference in content. About.5-7 daughter vesicles (brood capsules) are small spheres that are formed from rests of the germinal layer and appear as cysts within a cyst. They contain the scolices and hooklets, along with sodium chloride, proteins, glucose, ions, lipids, and polysaccharides[1]. When daughter cysts are separated by the hydatid matrix, they demonstrate a "wheel spoke" pattern as was seen in this case[8]. The matrix represents hydatid sand containing membranes of broken daughter vesicles and scolices[9]. Regressive changes occur in the center, whereas peripheral proliferation continues indefinitely. The cytotoxic effects of the vesicular fluid may result in an exuberant granulomatous response by the host's immune system. This reaction has two main consequences: fibrosis and necrosis. Complete calcification of the wall of a hydatid cyst can be considered an indication of quiescence or perhaps death of the parasite.[10]

Renal hydatid disease may mimic other diseases. Detection of a cystic lesion with internal septations and sand, wall calcifications, or the rosette sign in the proper clinical setting allows the correct diagnosis in a majority of cases.[10,11] Lesions with a solid appearance may be encountered. Lack of internal contrast enhancement allows one to classify them as hyperattenuating or hyperintense cystic lesions and to avoid misinterpreting them as tumours. Intact cysts may produce a low level of antigenic stimulation. Advanced radiological techniques such as CT and MR imaging remain the mainstays of diagnosis.[12,13]

In general, surgery is the treatment of choice in renal hydatid cyst**.** Kidney-sparing surgery (cystectomy with pericystectomy) is possible in most cases (75%). Nephrectomy (25% of cases) must be reserved for destroyed kidneys.[14] A major concern during surgery for cyst removal is that, if a cyst ruptures, the brood capsules can spread throughout the body, and secondary cysts can grow wherever their contents come to rest. Although puncture of hydatid cyst has been considered as a possible source of anaphylactic reactions and spread of the parasite, studies have shown good long-term results with percutaneous hydatid cyst treatment under US guidance.[15,16]

The patient in this case made an uneventful recovery.

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