Renal Hydatid Disease

By Harpreet Singh, MD [2]

Case History: A 30-year-old female patient with history of dull pain in her left flank.
Figures 1-4: NCCT section show left renal cystic mass with multiple small cysts within.
Figure 2
Figure 3
Figure 4
Figures 5-9: CECT sections show no enhancement of cyst wall.
Figure 6
Figure 7
Figure 8
Figure 9

CT showed large well defined soft tissue attenuating mass lesion showing multiple cystic areas within it arising from cortex of lower pole of left kidney. The lesion is measuring about 31 (CC) x 85 (AP) x 77 (TR) mm in size. No obvious contrast enhancement is noted on arterial/venous phase. No e/o any calcification is seen.

**Diagnosis:** Renal hydatid disease

**Discussion:** Hydatid disease is a zoonosis with worldwide distribution and is caused by the larvae of Echinococcus tapeworms, primarily E. granulosus. It is endemic to many sheep- and cattle-raising regions of the world, including the Mediterranean, Africa, South America, the Middle East, Australia, and New Zealand.

The life cycle of E. granulosus involves two hosts: a definitive host, usually a dog or other carnivore, and an intermediate host, most commonly a sheep. The adult worm resides in the proximal small intestine of the definitive host, and the parasitic ova are excreted in the host’s feces. Sheep and other ruminants that graze on contaminated ground may become infected, and humans may
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become intermediate hosts by ingesting food or water contaminated with the ova. After it is ingested, the ovum loses its protective outer layer, which is digested in the duodenum of the intermediate host. The freed embryo (oncosphere) then penetrates the duodenal wall and enters the portal venous or lymphatic system. The embryo usually becomes lodged in hepatic capillaries and develops into a cyst within the liver, where it enlarges at a rate of approximately 2–3 cm per year. Rarely, the embryo is deposited within the lungs or other organs. The cycle is completed when the definitive host ingests infected viscera from the intermediate host.

The wall of a hydatid cyst comprises three layers. The outermost layer, or pericyst, is a protective covering that forms in response to the presence of the parasite and consists of modified host cells. The middle, laminated membrane is a thin, easily ruptured, acellular layer that allows the passage of nutrients. The inner, germinal layer is where the laminated membrane and scolices (infectious larval tapeworms) are produced. The latter two layers compose the true wall of the cyst, or the endocyst. Daughter vesicles, which contain protoscolices, are formed from the germinal layer and are initially attached to the mother cyst by a pedicle. The cyst fluid is a clear transudate that contains sodium chloride, proteins, glucose, ions, lipids, and polysaccharides. It is antigenic, and if released into the host’s circulation as a result of cyst rupture, it may cause eosinophilia or anaphylaxis. When daughter vesicles rupture into the parent cyst, scolices may pass into the cyst fluid and form a layering sediment known as hydatid sand.

The liver (75 percent of cases) and lung (15 percent of cases) are most commonly affected by hydatid disease, although any organ may be involved. Renal involvement is rare, occurring in 2%–3% of cases. Patients with renal involvement often are asymptomatic for many years, and cysts can become quite large (>10 cm) before clinical signs or symptoms appear. The most common manifestations of renal hydatid disease are a flank mass, pain, and dysuria. In 18 percent of cases, renal hydatid disease is complicated by cyst rupture into the collecting system, with resultant acute renal colic and hydatiduria.

Renal hydatid cysts typically are unilateral, solitary, and found in the cortex of the upper or lower pole of the kidney. Imaging appearances vary with the development of the parasite, and three types of cysts may be distinguished: Type 1 cysts correspond to the initial developmental stage of the parasite and appear unilocular, without internal architecture; type 2 cysts are seen at an intermediate stage of parasitic development and contain multiple daughter cysts; and type 3 cysts are completely calcified and represent the death of the parasite.

Abdominal radiography may depict a soft-tissue mass that corresponds to the hydatid cyst. Ring-shaped or curvilinear calcifications may be seen in 20 percent to 30 percent of cases because of calcification of the pericyst. Complete calcification also may occur during the healing phase. Infundibular and caliceal distortion is the most common finding at excretory urography, but obstruction and renal dysfunction also may be seen.

At ultrasonography (US), the appearance of renal hydatid disease varies. A unilocular (type 1) cyst may mimic a simple renal cyst. Multiseptated daughter cysts (type 2) may be mistaken for polycystic kidney disease. However, the presence of a thick, bilayered wall is suggestive of hydatid disease. The “falling snowflake” or “snowstorm” sign—multiple echogenic foci produced by hydatid sand that is dispersed when the patient rolls—is pathognomonic of hydatid disease. Detachment of the endocyst from the pericyst with a “floating membranes” appearance also is characteristic. Multiple daughter cysts separated by a fluid matrix that contains a mixture of membranes of broken daughter vesicles, scolices, and hydatid sand with mixed echogenicity may give rise to a “wheel-spoke” pattern. Type 3 cysts appear as a bright echogenic focus with strong posterior acoustic shadowing.

Typical CT findings of renal hydatid disease include a unilocular cyst (type 1), a multilocular cyst (type 2) with mixed internal attenuation and daughter cysts with lower attenuation than that of the maternal matrix, and a completely calcified cyst (type 3). In type 1 and type 2 cysts, the cyst wall may be thick or calcified, and both the wall and internal septa often enhance after contrast material is administered.

On T2-weighted MR images, hydatid cysts characteristically have a low-signal-intensity rim, a finding that probably corresponds to the dense, fibrous pericyst. Type 1 lesions are hypointense on T1-weighted images and markedly hyperintense on T2-weighted images, findings similar to those of simple cysts. The maternal matrix of type 2 cysts also typically is hypointense on T1-weighted images and hyperintense on T2-weighted images. The contents of daughter cysts demonstrate the signal intensity of fluid on both T1- and T2-weighted images; however, the signal intensity may differ, depending on the contents of the maternal matrix. Collapsed membranes of the daughter cysts may appear as linear intracystic structures that have low signal intensity on all MR images, regardless of the pulse sequence used. These collapsed membranes and the parent cyst wall may
enhance after the administration of contrast material. Type 3 cysts also have low signal intensity on all MR images, regardless of the pulse sequence used.

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