Imaging Spectrum of Hydatid Disease: Usual and Unusual Locations

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Summary

Hydatid disease is a parasitic infection caused by *Echinococcus granulosus* and *Echinococcus multilocularis*. It is common in endemic regions and can demonstrate a variety of imaging features that differ according to the affected organ and the stage of the disease. Liver and lungs are the most commonly affected organs. The classic features of hepatic hydatid disease are well known. However, diagnosing hydatid disease at unusual locations may be challenging because of myriad imaging features in each of these locations. Knowledge of the imaging spectrum in systemic hydatidoses in various organs is very valuable in improving the accuracy of radiological interpretation. The purpose of this article is to review the imaging features of hydatid disease at its varied locations.

MeSH Keywords: Echinococcosis, Hepatic • *Echinococcus multilocularis* • Magnetic Resonance Imaging • Multidetector Computed Tomography

Background

Epidemiology

Hydatid disease is a widely prevalent parasitic infection caused by cestode species, *Echinococcus granulosus* (also known as cystic hydatid disease/echinococcosis) and *Echinococcus multilocularis* (also known as alveolar hydatid disease/echinococcosis). Human infection is common in sheep-rearing countries such as Australia and New Zealand, throughout England and Europe, the Middle East, Russia, Northern China, and Japan. In the Americas, the disease is prevalent in Alaska, Canada, Argentina, Uruguay and Chile. Alveolar hydatid disease (*E. multilocularis*) is a less widespread disease. The disease is mainly prevalent in northern and central Europe and parts of Canada [1].

Life cycle of Echinococcus and cyst structure

Dogs are the definitive host and sheep are the intermediate hosts [2]. Humans become infected after ingestion of parasitic eggs, either directly from contact with dogs, or more frequently by indirect ingestion of contaminated food or water [2]. The adult form of the cestode resides in the small intestine of the definitive host, releasing eggs containing infective oncospheres to the environment. These eggs are consumed by the intermediate host, following which a larval stage – metacestode, develops in various internal organs [3].

The metacestode stage usually manifests as a cystic structure (hydatid cyst). The wall of a hydatid cyst (HC) has three layers, an outer pericyst, which is the dense fibrous capsule formed by the host’s inflammatory response, the middle laminated proteinaceous layer and the inner germinal layer, endocyst which is produced by the parasite itself. The layers tend to be thick in the liver, less developed in muscles, absent in bones, and sometimes visible in the brain [2].

Clinical presentation

Symptoms of cystic echinococcosis are site-specific with hepatic disease presenting with hepatomegaly and jaundice and pulmonary disease with chronic cough and chest pain. Complicated cysts may present with anaphylactic reaction.
On the other hand, alveolar hydatid disease is characterized by a long asymptomatic incubation period, presents later and has a chronic progressive course. The most common presenting symptoms of alveolar hydatid disease include cholestatic jaundice, epigastric pain, fatigue and weight loss.

**Radiological diagnosis**

Ultrasoundography (USG), computed tomography (CT), magnetic resonance (MR) imaging can help to image hydatid disease. The modality that is most helpful depends on the stage of the disease (growth of the cyst) and the affected organ [2]. USG can clearly demonstrate hydatid sand-fine internal debris, floating membranes and daughter cysts. CT is best for detecting calcification and osseous involvement whereas MR imaging is helpful in detecting hydatid disease with neural involvement [4].

Imaging findings in cystic echinococcosis range from cystic lesions to lesions that appear solid [2]. The cyst may appear as a well-defined fluid collection. Frequently, there may be detachment of endocyst from the pericyst causing the appearance of a floating membrane. Curvilinear or ring-like calcification of the cyst can be seen during the natural evolution and can lead to complete calcification [5].

**Serological diagnosis**

Confirmation of hydatid disease is done by detecting specific serum antibodies using immunodiagnostic tests. The tests include enzyme linked immunosorbent assay (ELISA) using hydatid fluid (less specific) or purified antigens (Antigen B – cystic echinococcosis, Em2 – alveolar echinococcosis), immunoblot analysis, immunoelectrophoresis, detection of immunoglobulin G4, etc. However, disease in the bone, brain, eye and muscle induces less antibody response and is more difficult to detect by serological methods [3].

**Types of hydatid disease and radiographic findings**

Hydatid cyst may be classified into the following 4 types according to a widely accepted imaging classification [5]. Appearance of hydatid disease at different sites (cystic echinococcosis) follows the general pattern presented below. Specific features of the disease at each site are mentioned in the subsequent sections. Alveolar hydatidosis shows an infiltrative growth pattern with multicystic honeycomb appearance and calcifications.

**Type I: Simple Cyst with no Internal Architecture**

On ultrasound, Type I hydatid cysts appear as well-defined unilocular anechoic lesions. On CT, these appear as well-defined fluid attenuation lesions (Figure 1). MRI features are those of a fluid-attenuation cystic lesion with a T1 isointense and T2 hypointense peripheral rim (“rim sign”) surrounding the homogenous high signal cyst contents [6].

**Type II: Cyst with daughter cysts and matrix**

This type includes cysts with detached floating endocyst membranes or daughter cysts. The attenuation of the daughter cyst is hypodense/hypointense to maternal matrix on CT and MRI, respectively [4]. Multiple cysts when present are enclosed within a single capsule resulting in “wheel spoke” appearance [2].

At CT, type II HCs can be visualized in three stages depending on the age, number, and arrangement of the daughter cysts [7].

a. Type IIA: Contain round daughter cysts arranged at the periphery (Figure 2).

b. Type IIB: Contain larger, irregularly shaped daughter cysts that occupy almost the entire volume of the mother cyst (Figure 3).

c. Type IIC: Relatively high-attenuation round or oval masses with scattered calcifications and occasional daughter cysts (Figure 4A). This represents degeneration of old cyst.

d. Type III: Dead calcified cysts.

They manifest as round, hyper-attenuating areas on CT (Figure 4B) and hypointense areas on MR imaging.

**Type IV: Complicated hydatid cysts**

Hydatid cyst complications include rupture and superinfection. Cyst rupture is mainly due to degeneration of parasitic membranes. Cysts may rupture into pleural and peritoneal cavities or into the biliary radicals [4] (Figure 5). The incidence of hydatid disease at different sites with the various complications at each site is listed in Table 1 [3].

**Sites of Involvement**

The liver is the most frequently involved site in both isolated cases and in cases with cysts in other organs. The second most common site is the lung. Other sites of involvement include the spleen, kidney, adrenal glands, peritoneum, bladder, ovary, brain, spinal cord, lung, heart, bone and soft tissues [8].

**Liver**

Liver is the most commonly affected organ (68 to 75% of cases), with the right lobe being the most frequently involved portion. Echinococcal cysts appear as well-defined round fluid cystic lesions. Daughter cysts that indicate viability, give the lesion a multicellular appearance.

There may be detachment of endocyst from the pericyst, appearing as localized split with floating membranes inside the cavity [4]. During the natural course of the disease, complete calcification of the cyst may occur [4]. A classification was proposed by the World Health Organization Informal Working Group on Echinococcosis for the different types of E. Multilocularis cysts seen on ultrasound examination of the liver (Table 2) [3]. Complications such as rupture into the biliary tree or transdiaphragmatic migration to involve the thoracic cavity can occur [4] (Figure 5).

Alveolar echinococcosis is a rare parasitic disease caused by E. Multilocularis, liver being the most commonly involved site [9]. On USG, these lesions show a “hailstorm” pattern, characterized by multiple echogenic nodules with
Figure 1. Type I hydatid cyst. (A) Oblique sonogram in a 32-year-old male shows type I hydatid cyst in the right lobe of the liver. (B) Axial contrast-enhanced CT image in a 46-year-old male shows a well-defined fluid-attenuation simple cystic lesion in the right lobe of the liver (asterix) with no septations/wall calcification.

Figure 2. Type IIA hydatid cyst. (A) Oblique sonogram in a 38-year-old male shows a type IIA hydatid cyst, with multiple peripherally arranged (yellow arrow) daughter cysts. (B) Axial contrast-enhanced CT in a 44-year-old male shows a non-enhancing multiloculated cystic lesion with multiple peripherally arranged daughter cysts (black arrow) of lower attenuation than the mother cyst.

Figure 3. Type IIb. (A) Axial contrast-enhanced CT scan in a 38-year-old male patient demonstrates multiple cystic lesions in the right lobe, the larger lesion appears as an unenhanced hypoattenuating cyst with irregularly shaped daughter cysts (asterix) that occupies most of the left hepatic lobe (Rosette sign). (B) Axial contrast-enhanced CT scan in a 28-year-old female shows two well-defined multiloculated cystic lesions in the liver, with lower attenuation daughter cysts within.
Figure 4. (A) Type IIC: Partially calcified hydatid cyst - axial contrast-enhanced CT scan in a 55-year-old male shows a hypoattenuating lesion in the right hepatic lobe with partial wall calcification (yellow arrow) and few peripheral daughter cysts. (B) Type III: axial contrast-enhanced CT scan in a 36-year-old male shows a well-defined round hyperattenuating calcified lesion (white arrow) in the right lobe of the liver.

Figure 5. (A) Axial contrast-enhanced CT scan in a 50-year-old male shows a subcapsular cystic lesion in the right lobe showing peripheral wall calcification, with rupture into the peritoneal cavity (yellow arrow). (B) Axial contrast-enhanced CT scan in a 40-year-old female demonstrates a type II hydatid cyst in the right lobe, showing rupture of the cyst into the biliary tree (white arrow). (C) Coronal CT image of a 35-year-old female with rupture of hepatic hydatid cyst into the right pleural cavity (black arrow) causing collapse of the underlying lung.

Table 1. Incidence and site specific complications of hydatid disease [3].

| Organ               | Percentage of cases | Complications                                                                 |
|---------------------|---------------------|-------------------------------------------------------------------------------|
| Liver               | 68.8–75             | Liver abscess, portal hypertension, inferior vena cava compression and thrombosis, Budd-Chiari syndrome, cyst rupture, peritoneal spread, biliary peritonitis, cholangitis, pancreatitis |
| Lung                | 17.2–22             | Biliiptysis, pneumothorax, lung abscess, eosionophilic pneumonia, parasitic lung embolism |
| Kidney              | 0.4–3.7             | Hematuria                                                                     |
| Spleen              | 1–3.3               | Rupture into peritoneal cavity                                                |
| Muscles and skin    | 0.2–2.2             | Pain, disturbances of movement                                                |
| Peritoneal and pelvic cavity | 2.0–5.2       | Mass effect                                                                   |
| Brain               | 0.03–1.1            | Headache, seizures, ‘tumor like symptoms’                                    |
| Bones               | 0.4–0.9             | Bone fragility, bone outgrowth                                                |

* Incidence of cystic echinococosis – 1–8 per 100,000 population, alveolar echinococcosis – 0.02–1.4 per 100,000 population.
Table 2. Types of cystic lesions (CL) and *E. granulosus* cysts (CE) which may be found on ultrasound (US) examination of the liver [3].

| Type of cyst | Description |
|--------------|-------------|
| Type CL      | Unilocular, cystic lesion(s) (CL) with uniform anechoic content, not clearly delimited by an hyperechoic rim (=cyst wall not visible) |
| Type CE1     | Unilocular, simple cyst with uniform anechoic content. Cyst may exhibit fine echoes due to shifting of brood capsules which is often called hydatid sand ('snowflake sign') |
| Type CE2     | Multivesicular, multiseptated cysts; cysts septations produce ‘wheel-like’ structures, and presence of daughter cysts is indicated by ‘rosette-like’ or ‘honeycomb-like’ structures. Daughter cysts may partly or completely fill the unilocular mother cyst |
| Type CE3     | Anechoic content with detachment of laminated membrane from the cyst wall visible as floating membrane or as ‘waterlily sign’ which is indicative of wavy membranes floating on top of remaining cyst fluid |
| Type CE4     | Heterogenous hypoechoic or hyperechoic degenerative contents. No daughter cysts. May show a ‘ball of wool’ sign which is indicative of degenerating membranes |
| Type CE5     | Cysts characterised by thick calcified wall which is arch shaped, producing a cone shaped shadow. Degree of calcification varies from partial to complete |

Figure 6. (A) Axial non-contrast and contrast-enhanced arterial (B) and venous phase (C) CT images of a 55-year-old male shows a heterogenous infiltrating non-enhancing mass with irregular margins (arrow) and scattered foci of calcification, with areas of hypoattenuation (asterix) corresponding to necrosis. Immunology and histology confirmed the diagnosis of *E. multilocularis*. MRI was not performed in this patient due to cardiac pacemaker.

Table 3. PNM staging of alveolar hydatid disease [3].

| P  | Hepatic localization of disease                                      |
|----|---------------------------------------------------------------------|
| Px | Primary lesion cannot be assessed                                    |
| P0 | No detectable lesion in the liver                                    |
| P1 | Peripheral lesions without proximal vascular and/or biliary involvement |
| P2 | Central lesions with proximal vascular and/or biliary involvement of one lobe |
| P3 | Central lesions with hilar vascular and biliary involvement of both lobes and/or with involvement of two hepatic veins |
| P4 | Any liver lesion with extension along the vessels(b) and the biliary tree |

| N  | Extra-hepatic involvement of neighbouring organs                      |
|----|---------------------------------------------------------------------|
| Nx | Not evaluable                                                        |
| N0 | No regional involvement                                              |
| N1 | Regional involvement of contiguous organs or tissues                 |

| M  | Absence or presence of distant metastasis                             |
|----|---------------------------------------------------------------------|
| Mx | Not completely evaluated                                             |
| M0 | No metastasis                                                        |
| M1 | Metastasis                                                           |

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irregular, ill-defined margins. CT and MR features are those of multiple irregular, ill-defined lesions which are hypoattenuating on CT and hyperintense on T2-weighted MR imaging (Figure 6). In advanced stages, irregular calcifications are found within the areas of central necrosis [7]. Hilar infiltration, if present, causes intrahepatic biliary radical dilatation. There may be atrophy of the affected liver segments if there is invasion of the portal or hepatic veins due to hypoperfusion [7]. The European Network for Concerted Surveillance of Alveolar Echinococcosis has proposed a classification denominated as PNM for describing the anatomical extent of the disease (Table 3) [3].

The following sections describe the appearance of cystic hydatid disease at different sites. Extra-hepatic site of primary alveolar hydatid disease is extremely rare. Secondary involvement/infiltration from primary hepatic disease is more common. The imaging features of alveolar hydatidosis common to all sites (lung, spleen and brain) include an infiltrative growth pattern with multicystic honeycomb appearance and clustered microcalcifications or plaque-like calcifications [2].

**Lung**

Lungs are the most common site of involvement in children. Right lung is affected in 60%, both lungs in 20% of cases [10]. Pulmonary lesions can be primary or secondary to intrathoracic rupture of a hepatic hydatid cyst. It can also involve the pleural cavity, mediastinum and chest wall [11]. Pulmonary cysts may range between 1 and 20 cm in diameter [12]. Larger lesions cause mediastinal shift, can induce a pleural reaction or cause atelectasis of adjacent lung. Calcification of pulmonary cysts is rare [11].

Computed tomography (CT) scan may demonstrate a thin enhancing rim if the cyst is intact. The contents of closed simple cysts are homogeneous and show fluid attenuation (Figure 7). Daughter cysts may be seen attached to the endocyst or lying free within the main cyst [11].
The MRI characteristics of a hydatid cyst may differ depending on the developmental phase (infected/viable/dead). On magnetic resonance imaging (MRI), cysts show low signal intensity on T1-weighted images and high signal intensity on T2-weighted images [13].

**Kidney**

Involvement of the kidney is rare (approximately 3% of cases). When involved, the lesions are frequently solitary and located in the cortex. Imaging appearance is that of a well-defined uni- or multilocular cystic lesion which may show ring-like calcifications (Figure 8). A characteristic feature would be the presence of daughter vesicles. The differential diagnoses of unilocular HC include simple cyst while the differentials of multilocular HC include multilocular renal cyst, cystic nephroma and also cystic-type renal cell carcinoma [2,14].

**Bone hydatid cyst**

Hydatid disease of bone is rare (prevalence ranges from 1.1% to 4%) [15]. On radiography, there can be osteolytic and inflammatory changes which may mimic osteomyelitis. Bone erosion and destruction may often cause confusion with malignant tumour. The primary role of CT and magnetic resonance imaging is in delineating the area of destruction and in recognition of extraosseous spread of the hydatid disease into the soft tissues [16,17] (Figure 9). Extraosseous HCs may show calcification, whereas intraosseous HCs rarely show calcification [2].

Hydatid disease of muscle is rare (2.3% of cases), and requires a high index of suspicion. In soft tissues, a hydatid cyst typically contains a number of daughter vesicles due to proliferation of the endocyst (proliferous membrane), giving the appearance of cyst within cyst. Ultrasonography is capable of detecting most features of hydatid. However, MRI is the preferred diagnostic imaging modality as it provides a better evaluation of the locoregional extent of the lesion and relations with the nerve and vascular pedicles [18] (Figure 10).

**Spleen**

Primary splenic hydatidosis is rare (less than 2%) with E.granulosus being the causative organism in the majority of cases. Most commonly, it develops secondary to intraperitoneal spread from ruptured liver hydatid cyst or due to systemic dissemination. They are usually solitary and show the classic imaging features of well-defined cystic lesions with daughter cysts or internal septae (Figure 11). The differential diagnosis includes pseudocysts, abscess, epidermoid cysts and hematoma [19].

**Peritoneum and Retroperitoneum**

Peritoneal hydatid cysts are usually secondary to rupture of hepatic hydatid cysts into the peritoneal cavity. These are generally multiple and can arise anywhere within the peritoneal cavity, with imaging features similar to those located elsewhere (Figure 12). The differential diagnoses include mesenteric cysts and intestinal duplication cysts [10].
Figure 10. Axial non-contrast CT images in a 35-year-old female with painless swelling of the forearm shows a fluid attenuation cystic lesion in the muscles of the forearm (asterix) with laminated membranes within. Hydatid serology and excision confirmed hydatid disease.

Figure 11. (A, B) Coronal T2 fat saturated images in a 45-year-old male patient show a well-defined heterogeneously hyperintense lesion in the lower pole of the spleen with daughter cyst and detached membranes (asterix) within. Note also the heterogeneous lesion involving T8-T10 vertebrae and left paravertebral region with daughter cyst within (yellow arrow). (C, D) Axial T2-weighted MRI images show heterogeneous lesion in the spleen that contains detached membranes (white arrow) with a serpentine appearance.
Figure 12. (A) Sagittal and (B, C) axial contrast-enhanced CT images of a 50-year-old male showing multiple well-defined intraperitoneal and retroperitoneal fluid attenuation cystic lesions with multiple septations and daughter cysts within. Also seen is a hydatid cyst in the left lobe of the liver (asterix).

Figure 13. (A) Axial contrast-enhanced CT image in a 32-year-old female shows a heterogeneous fluid-attenuation cystic lesion with laminated membrane in the head of the pancreas (thin arrow). Also seen in this section is an intraperitoneal multiloculated cystic lesion (solid arrow). (B) More cephalad section in the same patient demonstrated multiloculated cystic lesion in the right hepatic lobe (asterix).
Figure 14. (A) Axial unenhanced CT scan shows a cystic lesion within the pelvis, posterior to the urinary bladder (UB) and showing multiple peripherally arranged low-attenuation daughter cysts within. (B) Cephalad scans showed calcification of the cyst wall (yellow arrow).

Figure 15. (A) Axial T1-weighted image in the same patient showing cystic lesion within the pelvis posterior to the bladder (UB) showing peripheral low signal intensity daughter cysts within. (B) Coronal T2-weighted image shows the cystic lesion causing compression of the prostate and seminal vesicles. The right seminal vesicle is separately seen from the lesion; however the left seminal vesicle could not be separately delineated. (C) Sagittal T2-weighted image shows the large cyst located posterior to the bladder (UB) with curvilinear T2 hypointensity along the superior aspect of the lesion – suggestive of cyst wall calcification (white arrow).
Figure 16. (A) Sagittal contrast-enhanced CT image scan in an 80-year-old female shows a multilocular ovarian cystic lesion with scattered calcifications. (B) Axial contrast-enhanced CT image shows the multilocular ovarian cystic lesion. (C) Caudal axial sections show scattered calcification within the multilocular cystic lesion. The excision was done and hydatid disease of ovaries was confirmed.

Figure 17. Anterior mediastinal type IIB hydatid cyst. (A) Coronal and (B) axial contrast-enhanced CT scan shows a hydatid cyst with daughter cysts in the pericardial sac (asterixes) causing compression on the left ventricle.
Figure 18. (A, B) Axial contrast-enhanced computed tomography of a 48-year-old male shows a well-defined fluid-attenuation cystic lesion with internal septations within the left ventricle. Also seen is the fluid-attenuation lesion in the transverse sinus of the pericardial cavity. (C, D) Axial T2-weighted MR image of the same patient shows a well-defined hyperintense cystic lesion with internal septations within the left ventricle and hyperintense lesions in the transverse sinus of the pericardial cavity (white arrows).

Figure 19. Type II HC of the brain in a 13-year-old boy. Axial CECT (A, B) scan shows two well-defined multiloculated CSF attenuation lesions in the left fronto-temporal region causing mass effect in the form of effacement of the frontal horn of the left lateral ventricle and displacement of the interhemispheric fissure (white arrow) to the right. There is no perilesional edema.
Primary pancreatic involvement is very rare (0.25% of the cases), and is often associated with hepatic hydatid disease. The most common location is in the pancreatic head. The cyst may appear radiologically similar to pancreatic pseudocyst. However, a thickened laminated wall with a thin calcified layer and concomitant hepatic lesions suggest a hydatid cyst. The differential diagnosis includes pseudocyst and cystic neoplasms such as mucinous and serous cystadenoma [20]. We had a case of multiple hepatic and intra-peritoneal hydatid cysts with incidental finding of a cystic lesion in the head of the pancreas with laminated appearance of the cyst wall (Figure 13).

### Adrenal Gland

Hydatid cysts of the adrenal gland are also rare, and are usually secondary to disseminated hydatidoses. Hydatid cysts account for 6–7% of all adrenal cysts. Early lesions appear as simple cysts with daughter cysts with floating membranes and wall calcifications seen in later stages. The differential diagnosis of adrenal hydatid lesions includes endothelial cysts, pseudocysts, lymphangiomatous and...
angiomatous cysts, cystic degeneration of adrenal neoplasms [21].

**Pelvis**

Primary hydatid cyst in the pelvis is extremely rare and occurs in approximately 0.7% of the patients with this disease. We had a case of primary pelvic hydatid disease involving the left seminal vesicle showing multiple daughter cysts within and calcification of the cyst wall (Figures 14, 15).

Ovarian involvement is rare and is generally secondary to peritoneal spread from ruptured hepatic hydatid. Unilocular hydatid cysts can mimic an ovarian cystadenoma. Presence of daughter cysts and internal septations results in a radiological appearance similar to complex ovarian cysts (Figure 16) and even ovarian malignancy [22].

**Bladder**

Primary hydatid cyst of the bladder is rare. Urinary tract and bladder involvement can occur secondary to renal hydatid cysts, due to rupture of cysts into the collecting system [5].

**Cardiac and Pericardial Hydatid Cyst**

Hydatid cyst of the heart is very rare (0.02–2% of cases) and is usually secondary to hematogenous spread or rupture of a lung HC [23]. The most commonly affected cardiac chamber is the left ventricle (60% of cases). Other sites include interventricular septum (10–20%), right ventricle (10%), pericardium and less commonly atria [24].

On chest radiography, localised bulge of cardiac borders can be seen [8]. The cyst may be unilocular or multilocular. Transthoracic echocardiography, CT, and MR imaging are helpful in the evaluation of cardiac hydatid cysts [5] (Figures 17, 18).

**Central Nervous System**

Cerebral Hydatid occurs in about 3% of cases of hydatid infestation [25]. They are usually solitary lesions, commonly in the middle cerebral artery territory [26]. We had
a case of multiple giant cerebral Hydatid cysts (Figure 19). Other sites include pons, ventricle, meninges, aqueduct of Sylvius, cerebellum (Figure 20).

CT and MRI show a well-defined, smooth, thin-walled, homogeneous cystic lesion with cyst fluid showing CSF isointensity on CT and MRI, respectively. On unenhanced CT, the cyst wall is iso-hyperdense to brain tissue and on MRI it shows a T1&T2-hypointense rim. Wall calcification is rare [26]. Usually no rim enhancement or perilesional edema is evident unless superinfection is present. The differential diagnoses include cerebral abscess, cystic astrocytoma, arachnoid cyst and porencephalic cyst [27].

**Spinal Column**

Spinal hydatid cysts are extremely rare (<1%) [28]. Order of distribution according to incidence is thoracic (50%) > lumbosacral > cervical spine [26]. These can be classified into five groups: intramedullary, intradural extramedullary, extradural, vertebral and paravertebral hydatid disease (Figures 21, 22).

Most common spinal hydatid cysts are those that primarily affect the vertebral bodies as a result of portovertebral venous shunts [28]. Cysts show growth in the direction of least resistance; with time the parasite may destroy the cortex causing spread into surrounding soft tissues. CT and MRI show cystic fluid of CSF attenuation and signal intensity. Usually these lesions show no rim enhancement or wall calcification [26].

**Treatment**

There are several treatment methods for a patient with hydatid disease, including surgery, puncture aspiration injection reaspiration (PAIR), and chemotherapy. For asymptomatic patients, a “wait and observe” approach is likely more prudent. PAIR is a minimally invasive procedure, recommended for patients with unilocular hepatic cysts ≥5 cm in diameter, for cysts with daughter cysts, for
cysts with detached membranes, and for multiple cysts if accessible to puncture. Chemotherapy with benzimidazoles is recommended for patients with inoperable cysts [29].

Conclusions

Hydatid disease is common in endemic regions and can demonstrate a variety of imaging features that vary according to the affected organ and the stage of the disease. Knowledge of the imaging spectrum of hydatid disease in the various organs is very valuable in improving the accuracy of radiological interpretation.

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