

Systemic Echinococcosis- Radiological Presentation

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Abstract: Hydatid cyst (HC) is a zoonotic infection of the human caused by *Echinococcus granulosus*. The disease poses an important public health problem in many areas of the World. HC may develop in almost any part of the body. Most hydatid cysts occur in the liver (59-75%), followed in frequency by the lung (27%). Involvement of the kidney (3%), bone (1-4%) and brain (1-2%) is rare. In this article, the imaging findings of the hydatid cysts in different localization are reviewed. Findings in brain, lung, heart, liver, kidney, intra- and retro-peritoneum, spleen, pancreas, ovary, soft tissue and spine are discussed.

Keywords: Echinococcosis; Hydatid Disease; Computed Tomography; Magnetic Resonance Imaging; Ultrasonography

I. Introduction

Hydatid disease is one of the common diseases in tropical countries. It primarily affects the liver and shows typical imaging findings. However clinical presentations vary widely and are mostly nonspecific. Thus imaging plays an important role in diagnosis of Hydatid disease. The motive of this essay is to describe the imaging appearances of Hydatid disease and recognize the most frequent complications associated with hepatic hydatid disease [1].

Hydatid disease is a parasitic infection caused by *Echinococcus granulosus* characterized by cystic lesions in the liver, lungs, and rarely, in other parts of the body. The liver is reported to be the most commonly involved organ (in 52–77% of cases) (Babba et al. 1994). The non complicated hydatid cysts of the liver are symptomatic. The symptoms may be related to a toxic reaction due to the presence of the parasite and the local and mechanical effects depending on the location and nature of the cysts and the presence of complications (El-Tahir et al., 1992). Hepatic echinococcal cysts may be classified into five types according to the widely accepted imaging classification of Gharbiet al. (1981), based on sonographic patterns. Type I consists of a pure fluid collection, i.e. a non-complicated unilocular or monovesicular cyst. Type II is a fluid collection with a split wall (floating detached endocyst membrane). Type III is a cyst containing daughter cysts and septations with a predominantly fluid component on ultrasonography (US) (honeycomb image). Type IV is a cyst with a predominantly heterogeneous solid echopattern consisting of thick membranes with few daughter cysts. Type V is a calcified non-viable degenerated cyst with thick reflecting walls, representing an involute. Uncomplicated hepatic cysts are common lesions, lined by a single layer of epithelium. They could be solitary or multiple. The computed tomography (CT) appearance of a simple hepatic cyst consists of a well-circumscribed, homogeneous mass with no discernible wall. It has a near water attenuation value, and shows no enhancement after intravenous contrast material administration (Sayek & Onat, 2001; Suwan, 1995). On sonography it appears anechoic because of the clear fluid content with posterior acoustic enhancement, and has smooth regular wall. The aim of this study was to analyse the imaging findings on US and CT and to determine the effective diagnostic imaging of hepatic echinococcal cyst (HEC), in order to reduce diagnostic errors [2].

Life Cycle: Cystic echinococcosis is a zoonotic illness caused by infection with *Echinococcus granulosus*. In the life cycle of *E. granulosus*, humans sometimes become accidental intermediate hosts. Humans can orally take up the *E. granulosus* eggs from infected carnivore excretions by handling the animals or egg-containing feces, plants, eating vegetables, uncooked fruits, and drinking water with the eggs (3,4).

Epidemiology: The hydatid disease is endemic in some Mediterranean countries, the Middle East, the South America, the South Africa and Oceania. It is a disease of rural areas where farming is practiced traditionally (5). The hydatid cyst can occur also in non-endemic countries because of the upsurge of emigration and trade (6). Annual incidence rates of diagnosed human cases per 100,000 inhabitants vary widely, from less than 1 case per 100,000 to high levels.

Cystic echinococcosis causes not only illness but also productivity losses in human and agricultural animal population, and it can have large societal impacts on endemic areas (7). Cystic echinococcosis is rarely fatal. Occasionally, deaths occur because of anaphylactic shock or cardiac tamponade in heart echinococcosis (8).

Parasite Biology

Organ involvement: The liver is the most common site of infection followed by the lung in 10 to 30% of cases, and other sites (spleen, kidney, brain, bone) in about 10% cases. In one series of 386 patients with pulmonary hydatid cysts, 59 patients (15%) had extrapulmonary cysts, of which 54 were in the liver, 4 were in the kidney, and 1 was intraperitoneal. Approximately 60% were located in the lower lobes, and a predilection for the right lung was seen in 56% cases (9). Although hydatid cysts may be asymptomatic, but usually produce various symptoms due to compressive effect on surrounding vital structures such as dyspnea, retrosternal chest pain, cough, back pain, and superior vena cava syndrome (10,11).

II. Clinical Features:

The clinical features of cystic echinococcosis are highly variable. The spectrum of symptoms depends on the following; involved organs, size of cysts and their sites within the affected organ or organs, interaction between the expanding cysts and adjacent organ structures, particularly bile ducts and the vascular system of the liver, complications caused by rupture of cysts, bacterial infection of cysts and spread of protoscolices and larval material into bile ducts or blood vessels, immunologic reactions such as asthma, anaphylaxis, or membranous nephropathy secondary to release of antigenic material (7). The initial phase of primary infection is asymptomatic and may remain so for many years. Hydatid disease is seen in subjects of any age and sex, although it is more common in those aged 20–40 yrs (12,13). The rate of growth of cysts is variable depending on the strain differences and the organ involved. Typical measurements state that the average cyst growth is 1 cm to 1.5 cm/year (7). Patients come to the clinician's attention for different reasons, such as when a large cyst has some mechanical effect on organ function or rupture of a cyst causes acute hypersensitivity reactions. The cyst may also be discovered accidentally during radiographic examination, body scanning, surgery, or for other clinical reasons (14). Physical findings are hepatomegaly, a palpable mass if on the surface of the liver or other organs, and abdominal distention. If cysts in the lung rupture into the bronchi, intense cough may develop, followed by vomiting of hydatid material and cystic membranes (15).

III. Diagnosis:

The combination of imaging and serology usually enables diagnosis. The standard diagnostic approach for cystic echinococcosis involves imaging techniques, predominantly ultrasonography, computed tomography (CT), X-ray examinations, and confirmation by detection of specific serum antibodies by immunodiagnostic tests. Enzyme linked immunosorbent assay (ELISA) test using hydatid cyst fluid has a high sensitivity (>95%) but its specificity is often unsatisfactory. Finding a cyst using ultrasound, X-ray, or CT is typically expected in Echinococcus infection (16).

IV. Laboratory And Special Investigations:

Serological tests are commonly employed to supplement the radiological data in the diagnosis of hydatid cyst. The current gold standard serology test for echinococcosis detects IgG antibodies to hydatid cyst fluid-derive native or recombinant antigen B subunits. This is performed using ELISA or immunoblot formats (17). The lipoproteins antigen B (AgB) and antigen 5 (Ag5) are the major components of hydatid cyst fluid and are the most widely used antigens in current assays for immunodiagnosis of cystic echinococcosis (18). General consensus states that the ELISA test with crude hydatid cyst fluid has a high sensitivity of 95%; however, its specificity is low at 61.7% (19).

V. Types Of Hydatid Cysts

Type I Hydatid Cysts:

Type I cysts constitute the initial and active phase of hydatid disease. The three layers are intact. The external rupture of these cysts, due to either iatrogenic or traumatic causes, can cause disseminated disease. Type I cysts are important in disease spread to other anatomic sites in addition to the well-described hematogenous and lymphatic route. The walls of ruptured type I cysts are generally imperceptible since they become infected and rupture. Ultrasound (US) reveals an anechoic, well-defined cystic lesion with small echogenic foci or “falling snowflakes” consistent with hydatid sands changing with patient position. Computed tomography (CT) detects a water-attenuating lesion with well-defined borders. Pseudo contrast enhancement at the cyst wall can be seen due to compressed host tissue. True wall enhancement is seen in infected Type I cysts. Magnetic resonance imaging (MRI) reveals a homogeneous hypointense lesion on T1-weighted images and a homogeneous hyperintense lesion on T2-weighted images. The presence of a hypointense rim at the cyst periphery has been described as a characteristic of hydatid cysts (as opposed to non-parasitic cysts), but it is non-specific. This hypointense rim may be seen in long-standing Type I cysts due to a fibrotic response of neighbouring host tissue or to slight calcifications within the cyst wall.

Type II Hydatid Cysts:

Type II HCs represent the active phase of hydatid diseases in the parasite life cycle and in the dissemination of the hydatid disease. Although Type I HCs cannot spread to host parenchyma other than by external rupture, type II HCs can be spread to nearby tissue by outpouching a new cyst from main cyst cavity. An hourglass appearance and additional type II HCs can be seen. This type of HCs can be classified into three stages according to the arrangement of daughter vesicles within the cyst cavity. In type IIA HCs, daughter cysts arrange at the cyst periphery. The CT density of the mother cyst is higher than the daughter cysts. MR imaging shows the daughter cysts as hypointense or isointense relative to the maternal matrix on T1- and T2-weighted images. Type IIB HCs contain larger, irregularly shaped daughter cysts that occupy almost the entire volume of mother cyst, creating a “rosette” appearance. Type IIC HCs are type IIA and type IIB cysts that contain scattered calcifications within the cyst wall and daughter cysts within the cavity. Scattered calcification at the cyst wall does not imply a dead cyst in the presence of daughter cysts, but simply degeneration at the cyst wall.

Type III: Calcified Hcs:

Type III HCs constitute the inactive or dead phase of HD. In this phase, HD cannot spread, and in the absence of mass effect or other complications, there is no need for surgery. These HCs are seen in three types: (1) Total and thick continuous calcification (ring-like) of the cyst wall, (2) total calcification within the cyst matrix and a decrease in cyst size, and (3) curvilinear calcification within the ruptured internal membranes. CT is the preferred imaging method to evaluate these types of HCs due to calcification.

Type IV: Complicated Hcs:

The complications of HCs can be seen in all types of HCs except completely calcified Type III cysts. The complications, explained below, can be due to profound cyst size and subsequent mass effect on neighbouring organs. Other complications include internal and external rupture of HCs, secondary site involvement due to invasion of various anatomic barriers (e.g., diaphragm) and superinfection. Ruptures can be seen in 50-90% of cases. Internal cyst rupture is detected by the detachment of the endocyst from the pericyst and is probably related to decreasing intracystic pressure, degeneration, host response, trauma or response to medical therapy and percutaneous drainage [20-22]. Internal rupture causes death of the parasite. In the acute phase of rupture, internal membranes can be seen as floating structures inside the cavity; this is called the “water lily sign”. With time, cystic fluid decreases, and the HCs mimics a solid mass. Collapsed membranes within the cavity are detected as serpentine structures.

Complications Of Hydatid Cyst⁽²⁰⁻²²⁾:

Mass Effect

One of the most important complications occurs when HCs (type I and II) reach large sizes due to an active growth phase. Large type I and II HCs can compress neighbouring tissue and cause mass effect. If there is compression of vascular structures, especially portal vein, the involved lobe becomes atrophic due to decreased portal venous supply, and compensatory hypertrophy of the other lobes results. This is especially true for *E. alveolaris* due to microinvasion of portal veins and small biliary ducts. However, we also detected compensatory hypertrophy of uninvolved lobes and atrophy in the involved lobe without complete biliary obstruction. Large cysts in the hepatic parenchyma can cause biliary duct dilatation by either compression of a nearby duct by mass effect or by perforation into biliary ducts. The compression of nearby biliary ducts can cause micro erosion within the bile duct wall and fistula formation. There can be wide perforation that allows cystic contents to spill within the biliary ducts. Hydatid sands in Type I HCs, daughter cysts in type II HCs and ruptured germinal membranes in type IV HCs may pass through the fistula or perforation, obstruct bileflow, dilate biliary ducts and cause jaundice.

In the presence of a small fistula, it is difficult to show direct communication between cyst and biliary tracts by imaging methods. MR cholangiography may show small biliary ducts entering in the HCs. The detection of dilated biliary ducts and echogenic material within the lumen may be suspicious in the presence of HCs by ultrasound. A ruptured internal membrane floating freely within the cystic cavity may pass through the communication between HCs and biliary tract. This may be seen as tubular structures within the biliary lumen. When daughter cysts pass through a direct communication, a cystic lesion may also be seen within the biliary lumen.

VI. Rupture Of Hcs

The rupture of HCs can be contained (internal), communicating, and direct. Contained or internal rupture occurs when the detachment of the endocyst from the pericyst takes place, but the pericyst remains intact. Internal rupture may be related to degeneration, trauma or response to therapy, and it represents the

inactive phase of the HCs. Communicating rupture implies passage of cystic contents into biliary tracts. Communicating rupture may be seen in type I, II and IV HCs that internally rupture. When both the pericyst and endocyst rupture, direct rupture occurs. Internal rupture of cyst implies the death of the parasite, but external rupture causes dissemination to other organs. Type II HCs can only rupture externally. Trauma, degeneration and iatrogenic rupture due to surgical and percutaneous treatment cause external rupture of type I and II HCs. Sudden death, anaphylactic shock and dissemination of disease can be seen with cystic content spillage into the peritoneal cavity. When type I and II HCs are found near the hepatic capsule, extension to other neighbouring organs is easier. Rupture can also be seen in these locations due to a lack of protective tissue surrounding the cyst and deficient pericyst. Spillage of cystic content within peritoneal cavity causes disseminated disease.

VII. Infection Of The Hcs

The presence of air within the cystic cavity is also an important clue. Cyst infection is generally seen in HCs that rupture. Rupture may be internal (communicating) or direct. Rupture of HCs permits bacteria to pass easily into cyst. The wall becomes thicker and exhibits contrast enhancement on CT and MR imaging. Patchy contrast enhancement of neighbouring liver parenchyma represents inflammatory changes. CT is superior in detecting gas or air-fluid levels within the cyst.

VIII. Exophytic Growth

The two most common routes of exophytic growth are via the bare area of the liver and gastrohepatic ligament. HCs extend to lung and mediastinum when located at the bare area of liver. The cyst located near the gastrohepatic ligament can extend into peritoneal cavity. Trans-diaphragmatic lung, mediastinum and cardiac involvement. Trans diaphragmatic involvement of lungs and mediastinum occurs in 0.6- 16% of hepatic hydatid cysts. Trans -diaphragmatic migration is most common in HCs located in the 7th and 8th segments of right lobe due to their close proximity to the diaphragm. All HCs types can be spread in this way including completely calcified cysts that migrate into the thoracic cavity by adherence to the diaphragmatic surface. When a HC is located in close proximity to the diaphragm and grows large, initial indirect or reactionary findings may be seen on imaging methods, including pleural effusion, atelectasis or consolidation due to decreased diaphragmatic motion and incomplete lung expansion. Elevation of the diaphragm due to mass effect may also be seen with large HCs.

A direct sign of transdiaphragmatic migration is the presence of cystic lesion growing through diaphragm into lung, mediastinum and heart (Figure 16). The cyst typically has a characteristic hourglass shape. Sagittal and coronal MR images are helpful. Migration to the lung is frequent in HCs located in the 7th to 8th segments of the liver while mediastinal and cardiac involvement is associated with the 4th and 2nd liver segments.

Migration of HCs into lung from liver is seen as a continuation of the main cyst in the liver. Erosion or adhesion to the diaphragm is the cause of cyst migration. If cyst erodes the bronchial wall, as seen in primary lung HCs, internal perforation is considered. Expectoration of cystic membranes can occur with a bronchial fistula. Direct perforation of HCs into lung parenchyma causes prominent parenchymal consolidation and widespread hydatid disease. Perforation of HCs into the pleural cavity causes pleural empyema or multiple pleural cysts.

IX. Peritoneal Seeding

Peritoneal HD is almost always secondary to hepatic disease. Peritoneal echinococcosis in our series was generally due to previous hepatic surgery or spontaneous or traumatic rupture. Spontaneous rupture is a possibility with HCs located near the hepatic capsule or gastrohepatic ligament that grow exophytic. Peritoneal hydatid disease is generally multiple and remains undetected until cysts are large enough to produce symptoms. The overall incidence of peritoneal disease in cases of abdominal HD is approximately 13%. CT and MR imaging is valuable by imaging the entire peritoneal cavity. All kinds of HCs can be seen in the abdominal cavity.

X. Radiologic Findings

A pathology-based classification for this disease has been described by Lewall (23). The early lesions (type I hydatid cysts [HCs]) have a non-specific anechoic cystic appearance on ultrasonography (US) (23-26). On CT scans, Type I HC appears as a well-defined, round or oval cystic mass with an attenuation density near that of water (3-30 HU). On magnetic resonance imaging (MRI), HC is seen as hypointense on T1-weighted images and as marked hyperintense lesions on T2-weighted images. MRI can display a low-signal-intensity rim that surrounds the cyst (the "rim sign"), which is more evident on the T2-weighted sequences (24-30). For type II HCs, many daughter cysts and/or matrix develop within the parent cyst with or without cyst wall calcification

(23). Daughter cysts, indicating viability, have a lower attenuation value than that of mother cysts on CT scans. On MRI, daughter cysts may appear slightly hypointense or isointense relative to the maternal matrix on the T1-weighted images and hyperintense on the T2-weighted images. When present, floating membranes are seen as low signal intensity linear structures within the cyst on both the T1- and T2-weighted images (24, 25, 30).

Type III HCs represent a calcified, non-viable degenerated cyst (23). US demonstrates cyst calcification as hyperechoic areas with a strong posterior shadow. Cyst calcification can be seen as round, hyperattenuating areas on CT and as hypointense areas on MRI (24, 25). Complications of HC include rupture and superinfection of type I and II cysts (23). Rupture occurs in 50% 90% of cases. Three types of rupture can occur: contained, communicating and direct (23). In contained rupture, the endocyst ruptures and becomes detached from the pericyst. In communicating rupture, the cyst ruptures into an anatomical diversion structure like the biliary or bronchial tree (23). Direct rupture occurs when both the endocyst and the pericyst are ruptured with the cystic contents spilling into the pleural and peritoneal cavities or a hollow viscus (23). The cyst may become considerably smaller and less spherical both in communicating and direct ruptures (23). MRI may demonstrate disruption in the low-signal intensity rim of the cyst wall and extrusion of the contents through the defect (25, 30).

XI. Liver

The liver is the most common site of HC. The cysts may cause pain, discomfort, abdominal swelling or a palpable mass or thrill (27, 28). In the early stages of the disease, the appearance of HCs may be uncharacteristic and mimic that of simple cysts. However, the double-line sign can often be seen on sonography in unruptured HCs (25, 26).

Simple liver cysts do not demonstrate internal structures (24, 25), although multiple echogenic foci due to hydatid sand may be seen within the lesion by repositioning the patient on sonography (27). On MRI, a low signal intensity rim can be helpful to differentiate a unilocular HC from a simple liver cyst (24). Multiple unilocular cysts are indistinguishable from polycystic disease (24, 27). When echinococcal cysts become enlarged, they can produce pericystic biliary tract dilatation due to the mass effect (24, 27, 31). Decreasing intracystic pressure, endocyst degeneration, host response, trauma and medical treatment can cause separation of the endocyst from the pericyst (24, 25). Complete collapse of the endocyst results in a sonographic water-lily sign when the parasite lies in the most dependent part of the cyst or this produces an irregular, solid echo pattern (27). The wall of HC, even without calcification, is typically seen as a high-attenuated structure on unenhanced CT (25). The calcification may occur in the cyst wall or internally in the cyst, and this is detected on radiography in 20% to 30% of liver HCs (24, 25, 27). Following the formation of HC, many potential local complications may develop such as rupture, infection, perforation to the biliary tree (up to 90% of HCs), and involvement of the portal venous system, diaphragm and thoracic cavity (0.6% 16% of the cases with hepatic HCs) (24, 25).

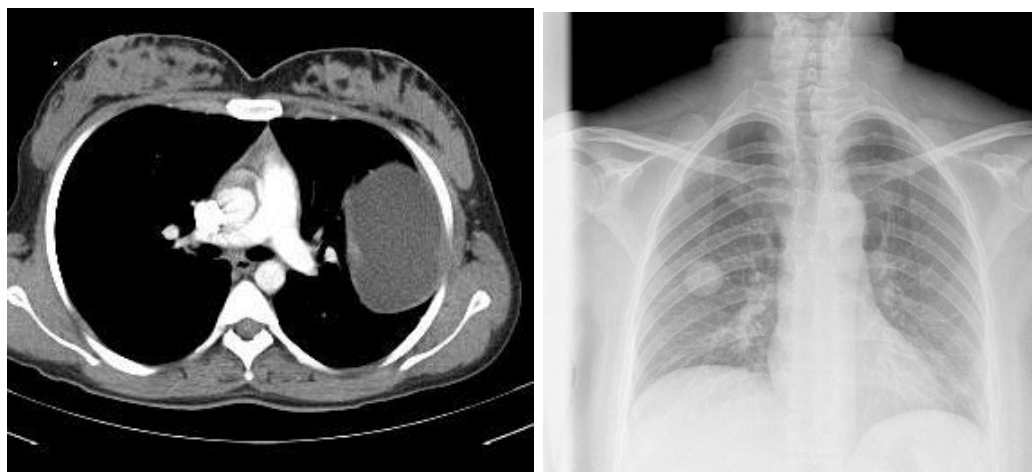


Partially calcified HC. CT scan shows a hypopattenuating lesion with peripheral wall calcification in the right hepatic lobe.

XII. Lung

The lung is the second most common location of hematogenous HC spread in adults and it is probably the most common site in children (24, 27). Pulmonary hydatid cysts are often asymptomatic, and they are usually found as incidental findings on routine chest radiography, but occasionally symptoms occur due to the

pressure effects on adjacent structures (27). The most prominent radiological finding is a dense, round, well-demarcated opacity that can resemble a neoplasm. Calcification (0.7% of cases) and daughter cysts are rarely seen in lung HD (24, 32). When the growth of the cyst produces erosion in the bronchioles, air between the endocyst and pericyst can produce a “crescent or inverse crescent sign” between the cyst wall and the pericyst. If air continues to enter the cyst cavity, then the “water lily” sign can be seen (an endocyst membrane floating in the most dependent part of the pericyst cavity) (24, 33). The radiologic appearances of infected cysts are similar to those of a lung abscess: a thick walled cavity with an air-fluid level and surrounding pneumonia (27). When HCs are infected, they can cause a solid appearance and give rise to a diagnostic error such as malignant tumor. Other complications of lung HC include rupture and recurrent acute pulmonary embolism (24).



A. Cystic lesion in left lower lobe. B. Cystic lesion with visible in right middle zone.

XIII. Kidney

Renal HD is rare (3% of case), usually solitary and located in the cortex (24). These patients may present with a flank mass, dysuria, pyuria, hematuria, persistent fever, renal stones, hypertension or renal colic (32). Hydatiduria can occur after rupture of the cyst into the collecting system (27). Any form of HC can be seen in renal HD (24, 25, 32). Mural calcification and daughter cysts often coexist. These findings are helpful in the differential diagnosis of HCs from a simple renal cyst, necrotic renal cell carcinoma, renal abscess and infected cysts, but it sometimes can be difficult to differentiate HC from necrotic renal cell carcinoma since calcifications may be encountered in both lesions (24, 32).



Axial unenhanced CT scan through the middle pole of the kidneys shows a hypoattenuating mass with peripheral daughter cysts and calcification (type II HC).

XIV. Peritoneal Cavity

Peritoneal HC, either primary or secondary, represents an uncommon but significant manifestation of this disease (approximately 13%). It is always secondary to traumatic or surgical rupture of a hepatic, splenic or mesenteric cyst (24, 25, 32). CT is the modality of choice for these patients because it permits imaging of the entire abdomen and pelvis (25). The lesions are generally multiple and any type of HC can arise anywhere in the

peritoneal cavity. Unilocular cysts (type I) should be distinguished from mesenteric cysts or intestinal duplication cysts (24, 28, 32).



Axial contrast-enhanced CT scan through the middle pole of the kidneys shows multiple low-attenuation daughter cysts in the peritoneal cavity.

XV. Spleen

Primary splenic involvement is very rare (less than 2%). The symptoms are mainly abdominal pain, splenomegaly and fever. Splenic HCs are usually solitary, and their imaging characteristics are similar to those of hepatic HCs (24, 32). Other splenic cystic lesions such as epidermoid cyst, pseudocyst, splenic abscess, hematoma and cystic neoplasm of the spleen should be considered in the differential diagnosis (32).



Large type II hydatid cyst with primary splenic involvement in a 50-year-old woman. Contrast-enhanced axial CT scan of the abdomen demonstrates unenhanced, well-defined hypodense lesion with a hydatid matrix and peripheral daughter cysts (arrows) in the upper part of the spleen.

XVI. Pancreas

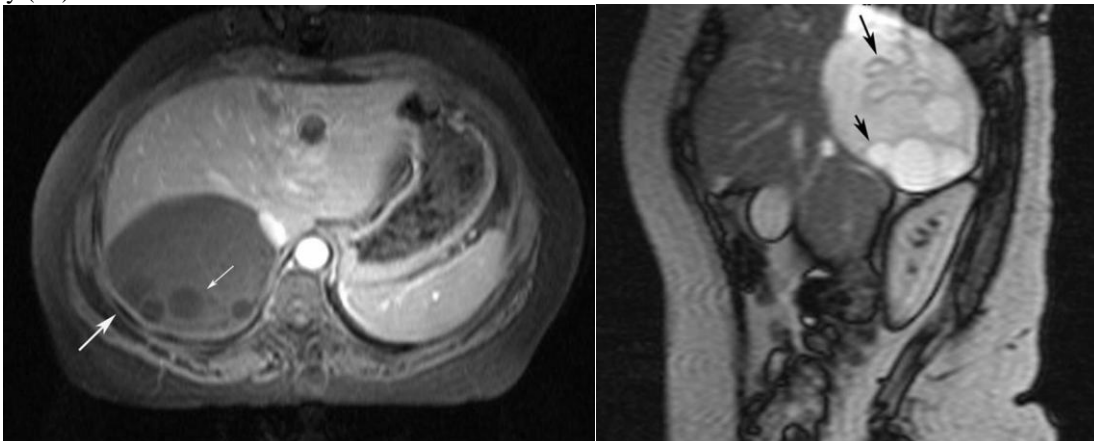
Primary HC of the pancreas is rare, representing 0.2-2% of all human infestation. It is usually single and located in the head of the pancreas (24, 32). The clinical symptoms depend on the size and location of the cyst within the pancreas. The lesion in the head of pancreas frequently presents with jaundice due to obstruction of the common bile duct. However, the body or tail lesions rarely cause symptoms. The differential diagnosis includes abscess or cystic neoplasms of the pancreas (32). The typical appearance and location of HCs within the pancreas is not established due to their relatively rare occurrence (24). In this paper we present the CT findings of a partially calcified HC located in the tail of the pancreas.



Partially calcified hydatid cyst of the pancreas in a 27- year-old woman. Contrast material-enhanced CT scan shows a hypodense lesion with dense peripheral calcification (arrow) in the tail of the pancreas.

XVII. Adrenal Gland:

The adrenal gland is an extremely rare location for HC and it is usually involved as a part of systemic echinococcosis. It is usually asymptomatic and these patients usually present with the symptoms that are caused by space occupying lesions (34). The imaging features depend on the stage of evolution of the disease. Unilocular HCs should be differentiated from exophytic renal cysts that originate from the upper pole of the kidney (24).



Type II hydatid cyst of the right adrenal gland in a 47-year-old woman. The axial (A) spin echo T1-weighted MR image demonstrates low-signal-intensity hydatid cyst with peripheral daughter cysts (arrows) in the right adrenal gland. Note that the daughter cysts have lower signal intensity compared to the mother cyst. The sagittal (B) plane spin echo T2-weighted MR image shows linear structures within the mother cyst, which represent detached membranes (long black arrow), and round, nodular lesions within the mother cyst, which represent daughter cysts (short black arrows).

XVIII. Ovary:

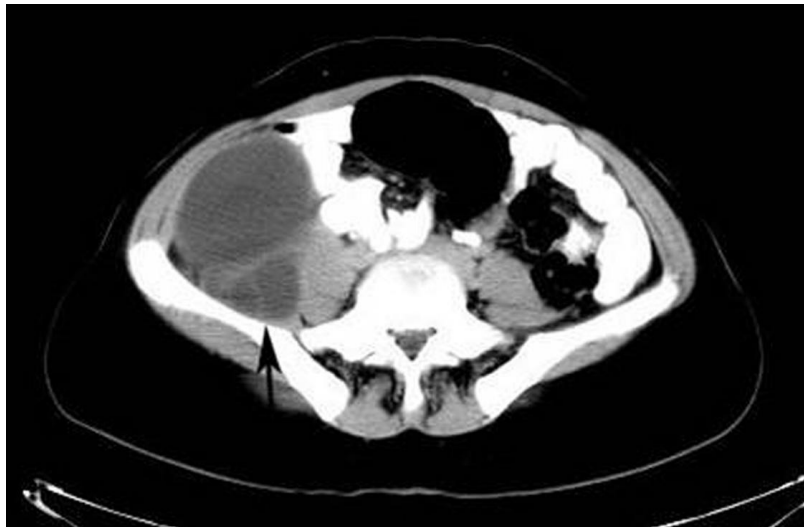
Ovarian involvement is also very rare and generally secondary to peritoneal spread of daughter cysts due to rupture of a liver HC. Ovarian HCs are usually asymptomatic and they can be discovered incidentally or they may cause irritation or compression symptoms. The ovarian lesion may be unilocular or contain daughter cysts that can give rise to a multiloculated appearance, and ovarian lesion should be considered in the differential diagnosis of cystic pelvic masses such as cystadenoma or cystadenocarcinoma (24).



Incidentally found type I hydatid cyst of the ovary in a 39-year-old woman. The axial (A, B) contrast-enhanced CT scan shows a unilocular low-attenuation lesion (arrows) in the right ovary.

XIX. Retroperitoneum:

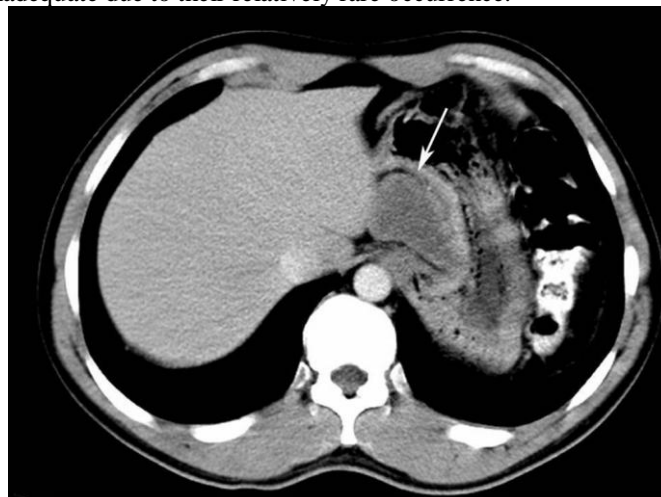
Isolated retroperitoneal HCs are also uncommon and they are usually secondary to the involvement of other organs or to previous surgery. Any type of HC can be seen in the retroperitoneum (24, 35).



Type II retroperitoneal hydatid cyst in a 25-year-old woman. The axial contrast-enhanced CT scan through the pelvis demonstrates a hydatid cyst with daughter cysts (arrow) adjacent to the right psoas muscle.

XX. Omentum:

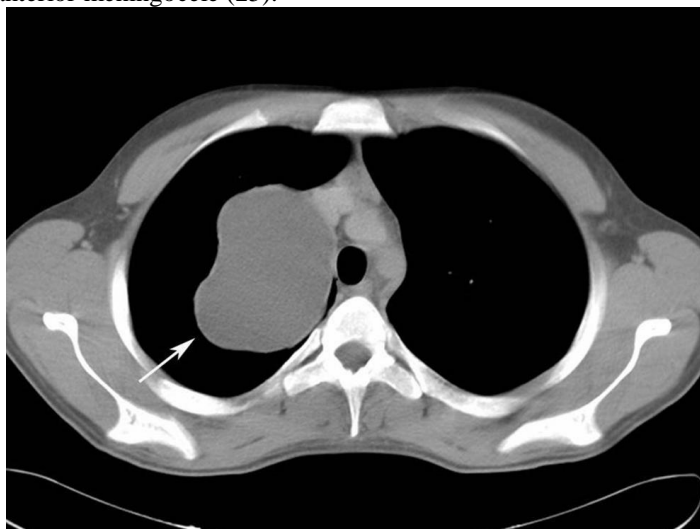
Isolated omental HC is one of the most unreported sites(36). Information about the appearances of HCs within the omentum is inadequate due to their relatively rare occurrence.



An omental hydatid disease in a 36-year-old man. The axial contrast-enhanced CT scan obtained through the stomach shows unilocular low-attenuation lesion (arrow) in the lesser omentum, which represents type I hydatid cyst.

XXI. Mediastinum:

Mediastinal HCs are also rare and they can be solitary or multiple lesions. The symptoms and complications of cyst depend on the size, location and involvement of adjacent structures. The imaging appearance can vary from type I to type III (24). The HCs in the mediastinum should be differentiated from cysts of a bronchogenic, pleuropericardial, thymic or enteric origin, and from intramural esophageal cysts such as lymphangioma and anterior meningocele (25).



A mediastinal hydatid disease in a 20-year-old man. The axial contrast-enhanced CT scan shows a well-defined, low attenuation lesion (arrow) that is consistent with type I unilocular hydatid cyst. Compression of the superior vena cava by the hydatid cyst is also seen.

XXII. Conclusion

HD is a dynamic entity with varying imaging appearances. It can arise in any part of the body the bloodstream reaches. Familiarity with imaging findings, especially in patients living in countries where this disease is endemic, provides important advantages in making the diagnosis. Despite the characteristic imaging findings, HD in unusual anatomic locations may make differential diagnosis difficult, even in patients from endemic regions. However, HC should be kept in mind when a cystic lesion is encountered anywhere in the body.

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