INTRODUCTION

Hydatid disease (HD) is a worldwide parasitic infection caused by the larval stage of *Echinococcus* tapeworm. The disease is endemic in many parts of the world, especially in cattle farming areas of South America, the Mediterranean region, the Middle East, Africa, Asia, and Australia.\[1\] HD is more commonly caused by *Echinococcus granulosus* and less commonly by *Echinococcus multilocularis* in its more aggressive form.

In its life cycle, the adult *E. granulosus* resides in the small bowel of its definite hosts, which are mostly dogs or other carnivorous animals, and then gravid proglottids release eggs that are passed in the feces. These eggs are ingested by intermediate host, which are commonly sheep or other ruminants. The eggs then release oncospheres in the small bowel and then migrate either through portal venous or lymphatic circulation to liver or less commonly lodged in other organs. At the organ site, the embryos either die or develop into hydatid cysts. Humans can be infected secondarily if they ingest substances such as water or vegetables contaminated with *Echinococcus* eggs.\[2\]

The liver acts as the first line of defense and, therefore, is the most common site of involvement in approximately 75% of cases by HD. Less commonly, hydatid cyst can involve lung, spleen, kidney, bones, brain, and other rare anatomic locations.\[3\]

ABSTRACT

Hydatid disease (HD) is endemic in many parts of the world. HD can affect virtually any organ system in body and should be kept as differential diagnosis of cystic lesion. HD is mostly asymptomatic; however, it demonstrates a variety of characteristic imaging findings depending on the site of involvement, stage of growth, mass effect, complications, or hematogenous spread, which helps in diagnosis. Radiography, ultrasonography (USG), computed tomography (CT), and magnetic resonance imaging (MRI) are commonly used imaging modalities. Radiography is helpful in chest and for demonstrating calcification. USG demonstrates characteristic findings such as cystic nature, daughter vesicles, membranes, septa, and hydatid sand. CT and MRI are modalities of choice for number, size, anatomic location, identification of local complications, and systemic spread. CT is, especially helpful for osseous involvement, and MRI is better for biliary and neurological involvement. Knowledge of these imaging findings helps in early diagnosis and timely initiation of appropriate therapy.

KEY WORDS

Computed tomography, hydatid disease, magnetic resonance imaging, ultrasonography

Access this article online

Quick Response Code:  
Website:  
www.tropicalparasitology.org

DOI:  
10.4103/2229-8070.190812

How to cite this article: Mehta P, Prakash M, Khandelwal N. Radiological manifestations of hydatid disease and its complications. Trop Parasitol 2016;6:103-12.

DOA: 30-05-2016, DOP: 19-09-2016

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

© 2016 Tropical Parasitology | Published by Wolters Kluwer - Medknow
Structurally, hydatid cyst is composed of three layers. The outer most layer is pericyst, which is formed by compressed host tissue and fibrous reaction; the middle laminated membrane, also referred to as ectocyst is an acellular structure; and the inner most germenerative layer referred as endocyst produces daughter vesicles containing protoscolices. The cystic fluid is antigenic in nature and can produce anaphylactic reaction if released into host circulation.

**IMAGING OF HYDATID DISEASE**

The imaging methods used for diagnosis and evaluation of the extent of HD are ultrasonography (USG), computed tomography (CT), magnetic resonance imaging (MRI), and less commonly radiography and urography. USG is screening modality of choice and is also used to monitor the efficacy of treatment. It clearly demonstrates the hydatid sand, floating membranes, daughter cysts, and vesicles inside the cyst. CT has high sensitivity and specificity for HD. CT is an important diagnostic modality in detecting cyst wall or septal calcification, demonstrating internal cystic structure posterior to calcification, assessing complications, depicting osseous lesions and in cases where USG has limitations (obesity, excessive bowel gases, abdominal wall deformities, and previous surgery). MRI is superior for demonstrating cyst wall defect, biliary communication, and neural involvement. Recently, MRI has been shown to be important in differentiating liver hydatid cysts from other simple cysts using diffusion-weighted sequence.

The imaging findings depend on the organ involved, host reaction, stage of evolution, and maturity of disease. The findings can range from purely cystic lesions to solid-appearing masses. The cysts may be solitary or multiple, unilocular or multivesicular, and with or without calcification. Presence of daughter vesicles and membranes within the cyst, peripheral cyst wall, or internal matrix calcification are important findings for differential diagnosis of HD. Various classifications are being used to describe hydatid cysts. More commonly, depending on the imaging appearance the hydatid cysts are classified into four types.

**Type I: Simple cyst with no internal architecture**

During the initial stage of development, the hydatid cyst appears as a unilocular, well-defined cystic lesion with no internal architecture with or without hydatid sand or internal septa. On CT, there is a frequent postcontrast enhancement of cyst wall and septa [Figure 1]. On MRI, the hydatid cyst shows a characteristic hypointense rim on T2-weighted images representing the pericyst. MRI is useful in differentiating Type I hydatid cyst from simple cyst depending upon the apparent diffusion coefficient values, which are found to be lower in hydatid cysts (2.5 × 10³ ± 0.9) in comparison to that of simple cyst (3.5 × 10³ ± 0.5).[9]

**Type II: Cyst with daughter cyst(s) and/or matrix**

Multivesicular cysts appear as fluid collection with multiple internal septae representing walls of daughter cysts, which are usually arranged at the periphery, having attenuation lower than that of the mother cyst [Figure 2]. Depending upon the maturity and arrangement of daughter cysts within the maternal cyst, a Type II cyst can be seen as Type IIA cyst containing multiple-rounded daughter cysts arranged at periphery with central high-density matrix (wheel spoke appearance); Type II B cyst containing large irregular daughter cysts occupying almost the entire volume of maternal cyst (rosette appearance); or Type IIC cyst appearing as relatively high-attenuation lesion containing occasional calcification and daughter cyst suggesting degeneration of old cyst [Figure 3].[10]

On MRI, the daughter cysts or brood capsules may appear hypointense or isointense relative to the maternal matrix on T1- and T2-weighted images with T2 hypointense pericyst [Figure 4]. Detached endocyst appears as twisted-linear structure within the cyst as the serpent-sign or snake-sign.[10]

**Type III: Calcified cyst**

Type III hydatid cysts are calcified cysts. CT is the preferred imaging method to evaluate calcified hydatid cysts as in USG, they show strong posterior acoustic shadowing. In CT, they appear as well-defined hyperattenuating lesions and in MRI as hypointense areas [Figure 5].

**Type IV: Complicated hydatid cyst**

CT and MRI play an important role in imaging of complicated hydatid cysts, common complications include rupture and superinfection. Hydatid cyst rupture occurs in 50–90% cases, usually due to age, chemical reaction, or a host defense mechanism and can be of three types.[11,12] In contained rupture, the endocyst detaches from pericyst appearing as curvilinear structure.
or floating membranes, finding that is highly specific for HD [Figure 6]. In communicating rupture, the cyst ruptures into the structures incorporated in the pericyst usually in biliary and bronchial radicles. Direct rupture into peritoneal or pleural cavities or other structures is due to rupture of both the endocyst and the pericyst.\cite{10}

Up to 25% cases of ruptured cyst may become secondarily infected. The cyst may show internal gas, fluid-fluid, or air-fluid level or fat as indirect signs of infection and/or communication with hollow viscera or biliary tree [Figure 7].

**SITES OF INVOLVEMENT**

**Abdominal involvement**

**Liver**

Liver is the most common site of involvement by HD; most cysts are located in the right lobe. They can be single or multiple in number with or without involvement of other organs. Differential diagnosis of Type I cyst includes simple epithelial cyst. Type II multivesicular cyst appears as well-defined fluid attenuation lesion with multiple septae or daughter cysts in a honeycomb pattern [Figure 8]. In advanced stage, cysts become partially or completely calcified, hydatid cyst showing complete dense calcification is assumed to be inactive cyst [Figure 5].\cite{13}

**Complications of liver hydatid cyst**

Common complications of hydatid cyst include cyst rupture and superinfection. In contained rupture, detached endocyst appears as floating membrane inside the cyst at USG, CT, or MRI [Figure 6]. On imaging, defect in the cyst wall can be identified in both communicating

---

**Figure 2:** Contrast-enhanced computed tomography of abdomen shows Type II liver hydatid cysts with multiple irregularly shaped daughter cysts that occupy almost the entire volume of the mother cyst - “rosette appearance”

**Figure 3:** Ultrasound (a), magnetic resonance imaging axial T2-weighted (b) and magnetic resonance cholangiopancreatography (c) images show heterogeneous soft tissue lesion with internal membranes (arrow), few calcifications and daughter cysts (arrow head) causing mild intrahepatic biliary radical prominence due to mass effect

**Figure 4:** Magnetic resonance imaging of abdomen T2-weighted axial image shows liver hydatid with multiple peripherally arranged brood capsules (arrow head) and hypointense pericyst

**Figure 5:** Contrast-enhanced computed tomography of abdomen shows calcified Type IIc (arrow head) and III (arrow) hydatid cysts showing calcification of wall, internal matrix, and membranes

**Figure 6:** Ultrasound (a), magnetic resonance imaging axial T2-weighted (b) and magnetic resonance cholangiopancreatography (c) images show heterogeneous soft tissue lesion with internal membranes (arrow), few calcifications and daughter cysts (arrow head) causing mild intrahepatic biliary radical prominence due to mass effect

**Figure 7:** Ultrasound (a), magnetic resonance imaging axial T2-weighted (b) and magnetic resonance cholangiopancreatography (c) images show heterogeneous soft tissue lesion with internal membranes (arrow), few calcifications and daughter cysts (arrow head) causing mild intrahepatic biliary radical prominence due to mass effect
Pooja, et al.: Radiological manifestations of hydatid

and direct rupture with or without partially collapsed cyst and passage of contents [Figure 7].

Communicating rupture can occur into adjacent biliary radicle, hepatic parenchyma, or sub-capsular hepatic space [Figure 9]. Biliary communication of HD has been demonstrated in up to 90% of hepatic cysts and can occur either through small fissures due to incorporation of biliary radicles (in 55% of cases) or through a wide direct perforation.\cite{14,15} Daughter vesicles or floating membranes can be visualized within the biliary radicles besides the visualization of defect in the cyst wall [Figures 10 and 11]. CT and MRI are superior to USG in demonstrating hydatid contents in distal common bile duct.

Direct rupture allows the passage of hydatid contents into the peritoneal cavity, pleural cavity, hollow viscera, or abdominal wall, and it is usually seen in peripherally located lesions [Figure 7]. Direct rupture can have important clinical consequences, including anaphylaxis, hydatid dissemination, or secondary bacterial infection.

Infection in hydatid cyst mimics hepatic abscess clinically and radiologically. Infected cyst demonstrates poorly defined margins with or without perilesional inflammation, presence of gas, or air-fluid level [Figure 12].\cite{16}

Compression of portal vein and thrombosis is a rare complication of hepatic hydatid and usually seen in hydatid cysts located in the caudate lobe and at hepatic bifurcation.\cite{17,18} Lack of flow within the portal venous system with or without visualization of cyst material may be demonstrated on imaging. Multiple venous collaterals at hepatic hilum, secondary cavernomatous transformation, or secondary Budd–Chiari syndrome can be seen [Figure 13].

**Spleen**

Involvement of spleen by HD has been reported from 0.9% to 8% of cases, and it is found to be the third most common location after liver and lungs in some

---

**Figure 6:** Contrast-enhanced computed tomography abdomen shows contained rupture of liver hydatid cyst with “floating membrane sign” (arrow) produced by detachment of the germinal membrane of the endocyst.

**Figure 8:** Contrast-enhanced computed tomography of abdomen shows Type II liver hydatids with multiple daughter cysts (arrows).

**Figure 7:** X-ray chest (a) and contrast-enhanced computed tomography of abdomen (b) show partially-calcified liver hydatid cyst with intracystic air and right pleural effusion, suggesting super-infection and rupture into the thoracic cavity.

**Figure 9:** Contrast-enhanced computed tomography of abdomen shows communicating rupture of liver hydatid into surrounding hepatic parenchyma and sub-capsular hepatic space (arrow).
Splenic involvement is commonly secondary to systemic dissemination or intraperitoneal spread from ruptured liver hydatid cysts, isolated primary splenic HD is rare and constitutes for <2% of cases. Imaging appearances are similar to those of hydatid cysts in liver [Figure 14a and b]. Complications are rare and include infection; rupture into pleural or peritoneal cavity; fistula formation into hollow organs, such as colon or stomach; rupture into the bronchial tree; splenothoracic fistula; pleural effusion; calcification or hypersplenism [Figures 15 and 16].

Kidneys
Renal involvement of HD is rare and has been reported in 2–3% of cases. Renal hydatid cysts are usually solitary and located in cortex, at upper or lower pole [Figure 17] and can reach up to 10 cm before they become symptomatic. Renal hydatid may appear as uni- or multi-locular well-defined cystic lesion with or without calcification or curvilinear structure. Hydatid cyst may infect secondarily or rupture into collecting system or perinephric tissues. Ruptures into the collecting system have been reported in up to 18% of cases. Differential diagnoses are renal cyst, cystic nephroma, and necrotic renal cell carcinoma. HD of adrenal gland is rare (0.06–0.18%).

Pancreas
Primary pancreatic involvement of HD is very rare (0.25%), and it is often secondary to hepatic disease. The diagnosis of pancreatic hydatid cyst is possible with high index of suspicion as it is commonly confused with pancreatic pseudocyst or cystic neoplasm. They appear as single or multiple, well-defined cystic lesion with thickened wall with or without calcification.

Peritoneum and retroperitoneum
Primary intra- or retro-peritoneal hydatid cysts are very rare. Peritoneal hydatid cysts are prevalent in approximately 13% of abdominal hydatid cysts and are usually secondary to spontaneous, traumatic, or surgical
Pooja, et al.: Radiological manifestations of hydatid rupture of hepatic, liver, or mesenteric hydatid cysts [Figure 18]. Retroperitoneal cysts are also secondary to involvement of other organs, most common being liver. On imaging, they look similar to hydatid cyst at any other location. Peritoneal hydatid cysts are usually multiple and can be seen anywhere [Figure 19]. Presence of daughter cysts helps in differentiating hydatid cyst from mesenteric or intestinal duplication cyst.

An isolated hydatid cyst in the pelvic cavity in retrovesical location has also been reported causing urinary symptoms due to mass effects [Figure 20]. In such cases, the hydatid embryo gains access to pelvis by hematogenous or lymphatic route.\[26,27\]

**Extraabdominal involvement**

**Brain**

Involvement of central nervous system by HD is rare and has been reported in 1–2% of cases.\[28\] Cerebral HD is more commonly seen in children and young adults. Brain hydatid cysts are usually single and unilocular, most common location being cerebral hemispheres, particularly in the territory of the middle cerebral artery. The less common sites of involvement are cerebellum, pons, ventricles, cavernous sinus, extradural, skull, and eye ball.\[29\] Cystic lesions such as porencephalic cyst, arachnoid cyst, cystic tumor of the brain, and pyogenic abscess are included as differential diagnosis of intracerebral hydatid cyst.

The cerebral hydatid cysts grow slowly and present late generally due to the compression of adjacent structures. In CT and MRI, cerebral hydatid cyst appears as well-defined, cerebrospinal fluid (CSF) signal density, or intensity lesion with or without brood capsules or daughter cysts [Figure 21]. Thin rim of peripheral enhancement can be visualized due to fibrous capsule or secondary to superadded infection. Calcification of brain hydatid cyst is rare (<1%). Perilesional edema is usually not seen as compare to brain abscesses or cystic neoplasms.

Hydrocephalus due to ventricular compression can be seen. Rarely, hydatid cyst may rupture into the ventricular system or subarachnoid spaces with demonstration of floating membranes or fluid-CSF level [Figure 22]. Peripherally located hydatid cyst can protrude into the meninges and calvaria or can
cause bone erosions. Sutural separation or unilateral enlargement of vault can be seen in childhood.

Spinal cord hydatid cysts are rare (<1%), thoracic spine being most common location.\[29\] They are usually multiple and can be intramedullary, intradural extramedullary, or extradural intraspinal in location. Spinal cord hydatid cyst usually appears as cystic lesion of CSF signal intensity without postcontrast enhancement.

**Thoracic involvement**

**Pulmonary**

Lungs are the second most frequent site of hematogenous spread in adults and have been reported as the most common site in children (15–25% of cases).\[2,11\] Concurrent involvement of the liver and lungs is seen in approximately 6% of all patients.\[30\] The size of the cyst may vary from 1 to 20 cm due to the compressibility of lungs.\[11\] Cysts are more commonly located in lower lobes (60% cases) and can be multiple (30% cases) and bilateral (20% cases).\[10,31\] Complications of pulmonary hydatid cyst include rupture (50–90% cases), superinfection, and rarely hepato bronchial fistula or acute pulmonary embolism secondary to rupture.

Uncomplicated cysts appear as round, oval, or polycyclic well-defined mass lesion. Calcification of the pericyst is very rare (0.7%) in pulmonary hydatid cysts. Daughter cysts are rarely seen in lung HD. As the cyst grows, it may cause erosions in the incorporated bronchioles allowing introduction of air within the cyst, appearing either as thin crescent of air between the pericyst and the laminated membrane (the “crescent sign” or “meniscus sign”) or air-fluid level within the endocyst. Sometimes, collapsed membranes may be seen floating on dependent portion of the cyst or on the surface of the cyst also known as the “water-lily sign” [Figure 23].

The “air bubble sign” seen as the presence of air bubbles in regions surrounding the cyst may develop as a consequence of cyst rupture, secondary to bacterial spread.
infections with presence of consolidation in adjacent lung parenchyma [Figure 24]. The “air bubble sign” is reported to be very sensitive and specific (85.7% sensitivity and 96.6% specificity) in establishing the diagnosis of complicated hydatid cyst.[32] Cysts may rupture directly into the pleural or peritoneal cavity. Rarely, during the medical treatment, pulmonary hydatid would be seen as thin-walled air-filled cavitary lesion [Figure 25].

**Intrathoracic extrapulmonary**

Intrathoracic but extrapulmonary hydatid cysts are rare and can be located in the fissures, pleural cavity, chest wall, mediastinum, myocardium, and diaphragm. They can be solitary or multiple and appearance can vary from Type I to Type III cysts.[33] CT and MRI are the main diagnostic tools in such cases. Pleural and mediastinal involvement are generally secondary to lung, hepatic, or splenic involvement [Figures 26 and 27].

Cardiac HD is rare (0.02–2% cases) and may be due to hematogenous spread or rupture of a lung hydatid cyst.[32] Most common location being left ventricle followed by interventricular septum, right ventricle, pericardium, and right or left atrium.[34,35] CT and MRI demonstrate unilocular or multivesicular cystic lesion in relation to cardiac chambers [Figure 28]. Pericardial hydatid cysts are usually seen in costophrenic recess, at

![Figure 22: Magnetic resonance imaging axial T2-weighted (a and b), T1-weighted (c) and diffusion-weighted (d) images of brain show a well-circumscribed cystic lesion in right cerebral hemisphere with detached germinal membrane (arrow heads) and intraventricular rupture (black arrow) causing mass effect and multiple brain infarcts (white arrow)](image)

![Figure 23: Non-contrast computed tomography of thorax shows a cystic lesion with air-fluid level (arrow) and collapsed endocyst (arrow head) lying in the dependent part of the cyst (“Water-lily sign”)](image)

![Figure 24: Contrast-enhanced computed tomography of chest (mediastinal window) shows hypodense lesion in left lower lobe with thick-enhancing wall with air bubbles and surrounding infection- The “air bubble” sign](image)

![Figure 25: Contrast-enhanced computed tomography of chest shows thin walled air-filled cavitary lesion in left lower lobe during the course of pulmonary hydatid disease](image)
Bones and soft tissues

Osseous involvement has been reported in 0.5–2% of cases, most common location being spine followed by pelvis, long bones, skull, and ribs.\(^{[36]}\) On imaging, bone hydatid appears as a well-defined, multiloculated expansile lytic lesion with irregular branching. Pathological fracture can also be seen. As the lesion grows, it causes thinning and breach of cortex and extension into the adjacent soft tissue. Intraosseous cyst rarely demonstrates calcification; however, extrasosseous component can calcify.

Primary soft-tissue involvement is rare and represents 0.5–4.7% of cases.\(^{[37]}\) Muscular HD preferentially involves muscles of the neck and trunk and at the root of the limbs. At imaging, they may appear as unilocular or multilocular cystic lesion or complex solid lesion depending upon growth of the cyst [Figure 30]. Differential diagnoses include abscess, synovial cyst, chronic hemato, and necrotic soft tissue tumor.

**CONCLUSION**

HD can affect virtually any organ system in body. Commonly, it demonstrates characteristic imaging findings that help to differentiate HD from other entities. However, at times, it can present itself with atypical manifestations due to secondary complications or at atypical sites. Familiarity with these imaging findings helps in early diagnosis and timely initiation of appropriate therapy, thereby reducing patient morbidity.
Acknowledgment
Nathani Sanjay MD, Consultant Radiologist, SKG Scan (Unit of Goyal Hospital and Research Centre PVT., Ltd.), Jodhpur, Rajasthan, India.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES
1. Marsden PD. The cestodes. In: Beeson PD, McDermott W, editors. Textbook of Medicine. Philadelphia, PA: WB Saunders; 1975. p. 510-1.
2. Beggs I. The radiology of hydatid disease. AJR Am J Roentgenol 1985;145:639-48.
3. Engin G, Acunas B, Rozanes I, Acunas G. Hydatid disease with unusual localization. Eur Radiol 2000;10:1904-12.
4. Dahniya MH, Hanna RM, Ashebu S, Muhtaseb SA, Hanna RM. Imaging features in the nutrition of the liver and cysts of the liver. AJR Radiol 2001;74:283-9.
5. Gharbi HA, Hassine W, Brauner MW, Mamone G, Carollo V. Imaging of cystic liver diseases. J Radiol 2007;88:1061‑72.
6. Marrone G, Crino F, Caruso S, Mamone G, Carollo V, et al. Multidisciplinary imaging of liver hydatidosis. World J Gastroenterol 2012;18:1438-47.
7. Oruç E, Yıldırım N, Topal NB, Kılıçturgay S, et al. The role of diffusion-weighted MRI in imaging of hydatid disease at some unusual sites. Br J Radiol 2000;74:283-9.
8. Precetti S, Gandon Y, Vilgrain V. Imaging of cystic liver diseases. J Radiol 2007;88:1061‑72.
9. Inan N, Arslan A, Akansel G, Anik Y, Sarisoy HT, et al. Radiological manifestations of hydatid disease of the adrenal gland: Review of nine patients. World J Surg 2004;28:97-9.
10. Polat P, Kantarci M, Alper F, Suma S, Okur A. Hydatid disease from head to toe. Radiographics 2001;21:227-9.
11. Pedrosa I, Saiz A, Arrozal J, Ferreiros J, Pedrosa CS. Hydatid disease: Radiologic and pathologic features and complications. Radiographics 2000;20:795-817.
12. Shah DS, Parikh H, Shah B, Banuprakash S, Shah J. Imaging appearances of hydatid cyst. Indian J Radiol Imaging 2006;16:533-5.
13. Mendez Montero JV, Arrazola Garcia J, Lopez Lafuente J, Antela Lopez J, Mendez Fernandez R, Saiz Ayala A. Fat-fluid level in hepatic hydatid cyst: A new sign of rupture into the biliary tree? AJR Am J Roentgenol 1996;167:91-4.
14. Moguillasnski SJ, Gimenez CR, Villavicencio RC. Radiologia de la hidatidosis abdominal. Radiologias imagen diagnostica terapeutica: Abdomen. Vol 2. Philadelphia, Pa: Lippincott Williams and Wilkins; 1999. p. 47-72.
15. von Sinner WN, New diagnostic signs in hydatid disease; radiography, ultrasound, CT and MRI correlated to pathology. Eur J Radiol 1991;12:150-9.
16. Khaldi F, Braham N, Ben Chehida F, Ben Jaballah N, Bennaure B. Hepatic hydatidosis and portal hypertension in children. Is it the Budd-Chiari syndrome? Ann Pediatr (Paris) 1993;40:831-4.
17. Gil-Egea MJ, Alameda F, Girvent M, Riera R, Sitges-Serra A. Hydatid cyst in the hepatic hilum causing a cavernous transformation in the portal vein. Gastroenterol Hepatol 1998;21:227-9.
18. Franquet T, Montes M, Lecumberri FJ, Esparza J, Bescos JM. Hydatid disease of the spleen: Imaging findings in nine patients. AJR Am J Roentgenol 1990;154:525-8.
19. Durgun V, Kapan M, Kapan M, Karabiyik C, Aydoğan F, Goksoy E. Primary splenic hydatidosis. Dig Surg 2003;20:38-41.
20. Odev K, Kilinc M, Arslan A, Aygün E, Günsör S, Durak AC, et al. Renal hydatid cysts and the evaluation of their radiologic images. Eur Urol 1996;30:40-9.
21. Von Sinner WN, Hellström M, Kagevi I, Norlen BJ. Hydatid disease of the urinary tract. J Urol 1993;149:577-80.
22. Akça MN, Akça G, Balık AA, Böyük A. Hydatid cysts of the adenral gland: Review of nine patients. World J Surg 2004;28:97-9.
23. Khiari A, Mzali R, Ouali M, Kharraat M, Kechoua MS, Beyrouti ML. Hydatid cyst of the pancreas. Apropos of 7 cases. Ann Gastroenterol Hepatol (Paris) 1994;30:87-91.
24. Karavias DD, Vigianos CE, Kakkos SK, Panagopoulos CM, Androulakis JA. Peritoneal echinococcosis. World J Surg 1996;20:337-40.
25. Ouafdjel J, Assem A, Errougani A, Jalil A, Belkacem R, Balařeʃ S. Isolated retroperitoneal hydatid cyst. Apropos of a case. Ann Chir 1990;44:243-5.
26. Fernández Larrañaga A, Silmi‑Moyano A, Rodríguez‑Vallejo JM, Úsón‑Calvo A. Retrovesical hydatidosis. Actas Urol Esp 1983;7:165-74.
27. Ersahin Y, Mutluer S, Güzellab E. Intracranial hydatid cysts in children. Neurosurgery 1993;33:219-24.
28. Tüzün M, Hekimoglu B. Hydatid disease of the CNS: Imaging features. AJR Am J Roentgenol 1998;171:1497-500.
29. Pomelov VS, Karimov SH, Nishanov KH. Surgical tactics in associated echinococcosis of the liver and lung. Khirurgiia (Mosk) 1991;11:69-74.
30. Aytaç A, Yurdakul Y, Ikizler C, Olga R, Saylam A. Pulmonary hydatid disease: Report of 100 patients. Ann Thorac Surg 1977;23:145-51.
31. Yuncu G, Ors KS, Sevinc S, Karabulut N, Alper H. The diagnostic value of the ‘air bubble sign’ in complicated pulmonary hydatid cysts. J Thorac Cardiovasc Surg 2007;133:1524-74.
32. Mathur RK, Doda SS, Buxi TB, Talwar JR. Primary mediastinal echinococcosis. J Comput Tomogr 1985;9:195-7.
33. Yekeler I, Koçak H, Aydın NE, Basoğlu A, Okur A, Senocak H, et al. A case of cardiac hydatid cyst localized in the lungs bilaterally and an anterior wall of right ventricle. Thorac Cardiovasc Surg 1993;41:261-3.
34. Kaplan M, Demirtas M, Cimen S, Ozler A. Cardiac hydatid cysts with intracavitary expansion. Ann Thorac Surg 2001;71:1587-90.
35. Kizilkaya E, Silit E, Basekim C, Karsli AF. Hepatic, extrahepatic soft tissue and bone involvement in hydatid disease. Turk J Diagn Interv Radiol 2002;8:101-4.
36. Gossios KJ, Kontoyiannis DS, Dascalogianaki M, Gourtsoyiannis NC. Uncommon locations of hydatid disease: CT appearances. Eur Radiol 1997;7:1303-8.