Atypical manifestations and complications of hydatid disease: Spectrum of imaging findings.

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Learning objectives

The purpose of this pictorial essay is to illustrate the radiologic findings (US, CT, MR) of abdominal hydatid cyst disease with emphasis on pitfalls, diagnostic difficulties and differential diagnosis.

We have reviewed the sonographic, CT, and MRI features of extrahepatic hydatid disease; including visceral, intraperitoneal, retroperitoneal and hematogenous spread.

This review should allow us to get familiar with atypical manifestations and complications of hydatid disease that may be helpful in making an accurate and prompt diagnosis.
Background

Hydatid disease is the larval cystic stage of Echinococcus granulosus infection, that can cause illness in intermediate hosts; generally herbivorous animals and humans who are infected accidentally.

Clinical features of cystic echinococcosis are highly variable. The spectrum of symptoms depends on the involved organs, the size of cysts, the interaction between the expanding cysts and the adjacent organs and finally the disseminated spread of the disease.

The classical findings in hydatid disease are well known; however, findings related to disease complications and unusual anatomic locations are less frequently described in the literature.

Life Cycle of *E. granulosus* Fig. 1 on page 6

Hydatid disease is a common clinical pathology in many parts of the world. There are two clinical forms of this disease: cystic hydatidosis caused by *Echinococcus granulosus* and alveolar hydatidosis caused by *Echinococcus multilocularis*. The main species pathogenic for humans in Mediterranean and Southern European countries is *Echinococcus granulosus*.

In the normal life cycle of *Echinococcus* species, adult tapeworms (3-6 mm long) inhabit the small intestine of carnivorous definitive hosts, such as dogs or wolves, and echinococcal cyst stages occur in herbivorous intermediate hosts, such as sheep, cattle, and goats.

The adult worm of the parasite lives in the proximal small bowel of the definitive host, attached by hooklets to the mucosa. The tapeworm eggs are passed in the feces of an infected dog and may subsequently be ingested by grazing sheep; they hatch into embryos in the intestine, penetrate the intestinal lining, and are then picked up and carried by blood throughout the body to major filtering organs (mainly liver and/or lungs). After the developing embryos localize in a specific organ or site, they transform and develop into larval echinococcal cysts in which numerous tiny tapeworm heads (called protoscoleces) are produced via asexual reproduction.

These protoscoleces are infective to dogs that may ingest viscera containing echinococcal cysts (with protoscoleces inside), mainly because of the habit in endemic countries of feeding dogs viscera of home-slaughtered sheep or other livestock. Protoscoleces attach to the dog’s intestinal lining and, in approximately 40-50 days, grow
and develop into mature adult tapeworms, once again capable of producing infective eggs to be passed to the outside environment with the dog’s feces.

Because humans play the same role of intermediate hosts in the tapeworm life cycle as sheep, humans also become infected by ingesting tapeworm eggs passed from an infected carnivore. This occurs most frequently when individuals handle or contact infected dogs or other infected carnivores or inadvertently ingest food or drink contaminated with fecal material containing tapeworm eggs.

**Hydatid cyst structure** Fig. 2 on page 6

The larva of *E. granulosus* in intermediate host is known as hydatid cyst.

The cysts are more or less spherical in shape. It ranges from 1 to 20 cm, even reaching 40 cm in diameter, consisted of cyst wall and inclusion content (hydatid fluid, hydatid sand). Cyst fluid is clear or pale yellow and antigenic.

The cyst wall consists of three layers:

(a) the outer pericyst, composed of modified host cells that form a dense and fibrous protective zone;

(b) the middle laminated layer is a cream-white, 1-4mm thick, noncellular structure which allows the passage of nutrients;

(c) The inner germinal layer is a 22-25µm thick structure, possesses germinal cells from which masses of brood capsules and protoscoleces are differentiate and grow into the cyst cavity.

The middle laminated membrane and the germinal layer are known as the endocys.

The brood capsule wall is similar with germinal layer. Each brood capsule may contains 1 to 70 protoscoleces. Sometime, the brood capsule forms external laminated layer to form similar structure with that of maternal cyst, called as daughter cyst, that at gross examination resemble a bunch of grapes.

The daughter cysts are replicas in miniature of the complete hydatid cyst; these in turn produce brood capsules, which may contain protoscoleces. In this way, granddaughter cyst may be seen. The free protoscoleces, brood capsules, daughter cysts, and amorphous material found in the cyst are known as "hydatid sand".

Cyst fluid is clear or pale yellow and antigenic.

**Diagnosis**
In humans, most cases are infected during childhood period, but they do not present with symptoms until adulthood. Hydatid disease can involve almost every organ of the body; the liver (75%) and lungs (15%) being the predominant locations.

The diagnostic certainty is given us by the radiological and serological studies.

Analytical parameters can be altered while not specific; eosinophilia is only present in less than 15%of cases.

Plain radiography detects only calcified cysts in 20-30% of cases, so it is not technique of choice.

MRI, CT and ultrasound show the cyst and its features.

Ultrasound is commonly the first technique used as is the easiest and cheapest to perform, with a sensitivity of 90-95%.

Then it is recommended to perform a CT or MRI scan that with a sensitivity of 95-100%, will give you the number, size and location of the cysts, including extrahepatic cysts detected more accurately than ultrasound. More specific findings include the presence of "daughter vesicles" within the larger cysts and cyst wall calcification. They are also considered better than ultrasound for detecting cyst complications such as infections and intrabiliary spread.

Regarding the serological analysis, the test more sensitive, detecting IgG ELISA has a sensitivity near 90%; however the specificity of the test ranges from 98-100%.

Definitive diagnosis could be made by examining aspirated fluids looking scoleces hooks; however, the diagnosis is not recommended as suction conventional method because of the risk of liquid leakage by the spread of the infection or the possible occurrence of anaphylactic reactions.
Fig. 1: Life cycle of Echinococcus granulosus.

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Fig. 2: Internal structure of hydatid cyst

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Findings and procedure details

RADIOLOGICAL FINDINGS

Hydatid disease primarily affects the liver and typically demonstrates characteristic imaging findings depending on the stage of cyst growth (whether the cyst is unilocular, multilocular containing daughter vesicles, or if partially or completely calcified [dead]).

In radiologic studies the typical findings of hydatid disease are well known, but unusual imaging characteristics related with its rare complications are less frequently documented in the literature. It is expected that a complicated hydatid cyst is associated with higher morbidity and mortality. Therefore, familiarity with atypical radiological features is very important to prevent misdiagnosis and improper therapeutical management.

Although the liver (75%) and lung (15%) are the most commonly involved organs, the disease can be seen anywhere in the body (10%).

Unusual locations of hydatid cysts support the hypothesis that beside portal circulation, the echinococcus embryos can spread via other routes, such as the lymphatic system, the biliary tract and/or by dissemination of daughter cysts into peritoneal or other cavities with the rupture of the primary cyst.

Furthermore, secondary involvement due to hematogenous dissemination may be seen in almost any anatomic location (eg, lung, kidney, spleen, bone, brain).

However, there are many potential local or distant complications (eg, intrahepatic complications, exophytic growth, transdiaphragmatic thoracic involvement, perforation into hollow viscera, peritoneal seeding, biliary communication, portal vein involvement, abdominal wall invasion,...).

The diagnosis of hepatic hydatid cysts complications may be established by US, CT or MRI, that permit the distinction between intact cysts and those presenting with contained rupture, as demonstrated by a collapsed endocyst or a globally echogenic appearance.

CT and MR imaging may demonstrate cyst wall discontinuity as well as the passage of contents through the defect.

CT is the modality of choice in peritoneal seeding.

The diagnosis of HHC perforation into the main biliary tree is usually made by detection of a discontinuity in the cyst wall and/or the presence of hydatid material within the
biliary system. Similarly, direct HHC rupture into different thoracoabdominal spaces is diagnosed by demonstrating cyst wall discontinuity and the presence of hydatid material within these spaces.

Indirect signs of biliary communication include increased echogenicity at US and fluid levels and signal intensity changes at MR imaging.

CT allows precise assessment of osseous lesions, whereas MR imaging is superior in demonstrating neural involvement.

**COMPLICATIONS**

In order to classify the complications and atypical manifestations observed in our center, we will consider on one hand exclusive liver involvement and on the other hand the extension from the liver to other anatomical locations. Complications of hydatid disease confined to the liver itself (intrahepatic), will contemplate; superinfection of the cyst, contained rupture of the cyst, communication with the biliary tree and direct vascular invasion of the portal vein. For extrahepatic extension, we consider direct communication to adjacent visceral structures (gallbladder, colon, stomach, right kidney, lung), retroperitoneal and peritoneal dissemination (omentum, pouch of Douglas, ...), and finally hematogenous spread (spleen, pancreas, pulmonary artery, bone,...).

**1- INTRAHEPATIC COMPLICATIONS** (LOCAL COMPLICATIONS)

**1a) Superinfection of hydatid cyst** (Fig. 3 on page 15)

Previous reports have shown a rate of superinfection in cystic hydatidosis of 1-8%. The superinfection probably occurs from sites next to the hydatid cyst (e.g., biliary tree) or as a complication of bacteriemia of any cause. Thus, a small rupture on the cyst's wall could induce infection.

The presence of a thickening of the wall, significant peripheral enhancement, air bubbles or fluid levels within the cyst can support the diagnosis of infection, although these findings are not specific. In selected cases fine needle aspiration can confirm the diagnosis.

**1b) Contained rupture of the cyst** (Fig. 4 on page 15 , Fig. 5 on page 16 , Fig. 6 on page 17 ).

It occurs when only the endocyst is torn and cyst contents are confined within the surrounding layer of host reactive tissue, the pencyst. Endocyst detachment is seen at cross-sectional imaging as floating membranes within the cyst. Contained rupture may be related to degeneration, trauma, infection or response to therapy.
1c) **Communication between cyst and biliary tree** (Fig. 7 on page 18, Fig. 8 on page 18, Fig. 9 on page 19)

Communication of hydatid disease with the biliary tree can be explained by the fact that during cyst growth, small biliary radicles are incorporated into the pericyst. Frank rupture into the biliary tree occurs in only 5%-15% of cases. Jaundice, fever, and chills are the most frequent symptoms related to biliary obstruction and cholangitis.

In imaging studies these alterations are usually manifested as hepatic cysts with irregular wall and presence of slightly echogenic or dense material, associated with bile duct dilatation.

The presence of material with increased viscosity inside the bile ducts may lead to complications such as biliary obstructive syndrome, acute suppurative cholangitis and even secondary acute pancreatitis (Fig. 8 on page 18).

1d) **Direct vascular invasion of the portal vein** (Fig. 10 on page 20 and Fig. 11 on page 21)

Direct portal invasion by hydatid cyst contents is an extremely rare complication. The mechanism usually involves compression of the portal vein and thrombosis with secondary cavernomatosis caused by cysts located in the caudate lobe and hepatic bifurcation. The latter phenomenon could be explained by an inflammatory response of the vessel wall caused by external compression of the cyst, facilitating the creation of a fistula, allowing passage of vesicles through the vascular wall to the portal lumen.

2. **EXTRAHEPATIC COMPLICATIONS** (EXTRAHEPATIC EXTENSION OF THE DISEASE)

Direct rupture occurs when both pericyst and endocyst are torn, allowing free spillage of hydatid material into the surrounding adjacent organs, or free body cavities.

Hydatid cyst rupture occurs due to degeneration of parasitic membranes caused by aging, chemical reactions, or a host defense mechanism.

Hydatid lesions can appear at any abdominal visceral location. When cystic lesions appear in these locations with the typical characteristics described (multivesicular, internal membranes, calcifications,...) hydatid disease should be suspected.

2a) **Direct communication to adjacent visceral structures**

2a1) **Extension to gallbladder** (Fig. 12 on page 22)

Because of the small number of cases reported, opinions about the pathogenesis of the primary gallbladder hydatid cysts are divided depending on the location of the cyst: in the lumen of the gallbladder or on the external surface. Cysts inside the gallbladder are described as a result of brood capsules dissemination through the biliary tract. When it
grows extramucosally is more likely to have occurred by lymphatic circulation as a result of transport of oncospheres from the intestine to the gallbladder. However, that should be confirmed in a larger number of patients.

2a2) **Extension to stomach** (Fig. 13 on page 23)

That is a very uncommon complication of hydatid disease. Ruptured cysts into the stomach derive most commonly from the liver. Long standing cysts seem to erode adjacent hollow viscera due to compression and resulting ischaemia. The site of communication may rarely be demonstrated at imaging studies such as computed tomography. An indirect sign may also be the presence of air-fluid levels, with or without hydatid membranes within the cyst.

2a3) **Extension to colon** (Fig. 14 on page 24)

Perforation of an hydatid cyst into the right colon may be either secondary to infection of the cyst or to primary pathology of the perforated organ. The content of the cyst can drain into the large bowel through the fistula, and also can allow gas from the large bowel to penetrate into the cyst. Spontaneous rupture of the cyst into hollow viscera is an extremely rare complication with an estimated frequency of 0.5%. This complication may be accompanied by clinical findings of hydatidorrhea. Typically, the communication is not discovered until surgery, although in some cases it is found at radiology. CT may demonstrate a cyst with an air-fluid level or orally administered contrast material inside the cavity.

2a4) **Extension to kidney** (Fig. 15 on page 25)

Involvement of the kidney is rare (3% of cases). Renal HCs are usually located in the upper or lower pole. HCs are frequently solitary and located in the cortex, and they may reach 10 cm in diameter before any clinical symptoms are noted. Complications of renal HCs include infection and rupture, in either the renal sinus or the perinephritic tissues.

An unilocular HC can mimic necrotic renal cell carcinoma, and the presence of calcification does not help distinguish HC from renal cell carcinoma due to the concomitant presence of calcification in the latter. Multilocular HCs can be misdiagnosed as simple renal cysts, cystic nephroma, and cystic variants of renal cell carcinoma.

2a5) **Extension to lung** (transdiaphragmatic migration) (Fig. 16 on page 26)

Involvement of the diaphragm and thoracic cavity occurs in 0.6%-15% of cases of hepatic hydatid disease. Transdiaphragmatic migration of hydatid disease from the posterior segments of the right hepatic lobe has been reported to be a common complication and is probably related to their proximity to the diaphragm, specially the bare area of the liver.
This may be due to the lack of peritoneal covering in this particular area, resulting in decreased resistance to cyst growth. Transdiaphragmatic migration varies from simple adherence to the diaphragm to rupture into the pleural cavity, seeding in the pulmonary parenchyma, and chronic bronchial fistula.

2b) **Peritoneal seeding:**

Omental (Fig. 17 on page 27), Douglas pouch (Fig. 18 on page 28, Fig. 18 on page 28 Fig. 19 on page 29).

Peritoneal hydatid dissemination has a prevalence of about 13% of cases and is almost always secondary to hepatic hydatid disease. Multiple cystic lesions can be observed anywhere in the peritoneal cavity. Peritoneal echinococcosis usually goes undetected until cysts are large enough to produce symptoms.

The differential diagnosis can be made easily owing to the characteristic appearance of daughter cysts but sometimes it can be difficult to differentiate from mesenteric cysts or intestinal duplication cysts.

2c) **Retroperitoneal spread** (Fig. 20 on page 30).

Hidatid cysts at this location are extremely rare with an estimated frequency of 0.8%. One of the postulated hypothesis is hematogenous spread, where the embryos once in circulation would not pass the liver and/or lungs and would be implemented elsewhere. Another possible route could be lymphatic spread from the intestine into the thoracic channels and then to other body sites. It has also been suggested that the embryo could be maintained in the rectal ampulla and migrates through the hemorrhoidal vessels eventually reaching a pre-rectal or retro-bladder location.

2d) **Hematogenous dissemination**

2d1) **Spleen hydatidosis** (Fig. 21 on page 31)

Splenic hydatid disease has been reported to constitute up to 4% of cases of abdominal hydatid disease. The imaging characteristics of splenic hydatid cysts are similar to those of hydatid cysts: calcification of the cyst wall, the presence of daughter cysts and membrane detachment. The differential diagnosis includes other splenic cystic lesions such as epidermoid cysts, pseudocysts, splenic abscesses, hematomas and cystic neoplasms of the spleen. Owing to the risk of spontaneous or traumatic rupture, splenic hydatid cysts are usually treated surgically.

2d2) **Extension to pancreas** (Fig. 22 on page 32)
Primary HC of the pancreas is very rare, with a reported prevalence of 0.25%. The cyst may easily be confused with a pseudocyst of the pancreas. However, the presence of a thickened and more laminated wall than a simple cyst and a thin layer of calcification within the lesion associated with liver HC may suggest a hydatid cyst. The differential diagnosis includes pseudocyst, serous cystadenoma, and mucinous cystic neoplasm.

2d3) **Pulmonary artery involvement** (Fig. 23 on page 33).
Hydatid disease is a rare cause of pulmonary embolism.

This uncommon complication is sometimes seen when there is embolisation of infectious particles (intravascular thrombus containing microorganisms) through the pulmonary arterial system or when direct invasion of vena cava occurs.

2d2) **Lung hydatid disease** (Fig. 24 on page 34).

The lung is the second most common site of involvement of *E. granulosus* in adults (10-30% of cases). The coexistence of liver and lung disease is present in only 6% of patients.

Most cysts are acquired in childhood, remain asymptomatic for a long period of time, and are later diagnosed incidentally at chest radiography.

Uncomplicated cysts appear as well-defined masses that may vary from 1 to 20 cm. Cysts are multiple in 30% of cases, bilateral in 20%, and located in the lower lobes in 60%.

Cyst growth produces erosions in the bronchioles that are included in the pericyst, and as a result, air is introduced between the pericyst and the laminated membrane. This air collection appears as a thin, radiolucent crescent in the upper part of the cyst and is known as the crescent sign or meniscus sign.

If the content is only partially evacuated, a waterline image appears, commonly referred to as the “water-lily or camalote sign”.

Sudden coughing attacks, hemoptysis, and chest pain are the most common clinical symptoms. After cyst rupture, expectoration of cyst fluid, membranes, and scolices may occur. Rupture into the pleural cavity may also occur. Although allergic episodes may develop, fatal anaphylaxis is uncommon. Bacterial infection of the cyst is the most serious complication after rupture.

2d4) **Bone involvement** (Fig. 25 on page 35).

The frequency of osseous involvement in hydatid disease is 0.5%-4%.
In bone involvement, pericyst formation does not occur, thereby allowing aggressive proliferation along trabecular bone.

The parasite replaces the osseous tissue between trabeculae due to slow growth of multiple vesicles. With time, the parasite destroys the cortex, with subsequent spread of the disease through surrounding tissues. Extraosseous cysts may calcify, whereas intraosseous disease rarely demonstrates calcification. The most common form of bone involvement is spinal hydatid disease, accounting for more than 50% of cases. It is usually difficult to distinguish from tuberculous spondylitis or chronic osteomyelitis.

Although CT allows precise assessment of osseous lesions and clearly depicts hydatid cyst calcification, MR is superior in demonstrating neural involvement.

**TREATMENT**
The definitive treatment of complicated hydatid disease is surgery with removal of the cyst and instillation of escolicides solutions such as hypertonic saline or ethanol, but in recent years due to the development of techniques for liver resection, pericysto-resection or even hepatectomy have been performed, and thereby obtained a low rate of morbidity and shortening of the postoperative period.

Also adjuvant treatment is recommended with albendazole, starting at least 4 days prior to removal and staying for more than 4 weeks. Sometimes as recurrences after surgery, in inoperable patients or those who refuse to surgery, a percutaneous aspiration can be done, with infusion of escolicide agents and re-aspiration (PAIR technique) by ultrasound control.

Recent studies have shown that this technique achieves cure rates equivalent to those achieved with surgery recurrence, but with lower perioperative morbidity and a shorter hospital stay. However more studies to specify the indications for this technique are needed.
Fig. 3: 64 years male presenting with abdominal pain and fever. Antecedents of hydatid disease (HC). a) Abdominal contrast enhanced CT axial image shows intrahepatic complex focal lesion in RHL, with hypodense center, wall thickening and peripheral enhancement. It is accompanied by inflammatory changes in the liver capsule affecting muscles of the abdominal wall. Puncture drainage was performed under ultrasound guidance obtaining purulent material confirming the diagnosis of superinfection of HC. b)&c) Abdominal CT coronal and sagittal sections showing contiguous extension of the inflammatory process (blue arrow) with loss of cleavage plane with the hepatic angle of colon.

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Fig. 4: 81 y.o woman presenting with abdominal pain in May 2013. a) Abdominal ultrasound shows a complex liver injury in left hepatic lobe suggestive of hydatid cyst (HC). b) In August 2013 the patient was visiting again the emergency department with pain, fever and leukocytosis. Abdominal ultrasound study shows variation in the sonographic appearance of the liver injury, which now has a predominantly hyperechoic content.

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**Fig. 5:** Same patient as Figure 4. Axial and coronal contrast-enhanced abdominal CT

a) There is a large cystic lesion in LHL with significant parietal enhancement and another lobulated lesion adjacent to its upper lateral side and perihepatic fluid. These findings suggest complicated/superinfected hydatid cyst.  
b) A focal parietal discontinuity in the upper surface of the HC is seen (yellow arrow) with adjacent subdiaphragmatic fluid, suggesting a contained rupture of the cyst.

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Fig. 6: Same patient as Figures 4 and 5. a) An 8F pigtail catheter was placed under ultrasound guidance for drainage in the collection (red arrow), obtaining purulent material. b) After introduction of contrast with fluoroscopic guidance, communication between the collection and the hydatid cyst is demonstrated. c) CT MIP reconstruction image shows proper location of the catheter.

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Fig. 7: 39 y.o. patient presenting with abdominal pain. a) Abdominal Doppler-ultrasound. Hydatid cyst (white arrows) with slight dilatation of adjacent biliary ducts (green arrow) b) Hepatic ultrasound. Marked dilatation of common bile duct (calipers) at porta hepatis with echogenic material inside corresponding to multiple daughter vesicles filling the duct.

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**Fig. 8:** Same patient as Figure 7. Contrast enhanced abdominal CT scan, coronal plane. Hydatid cyst (HC) in VIII liver segment communicates with biliary duct (green arrow). There is significant fatty stranding (yellow arrow) around the head of the pancreas (P), in relation to acute pancreatitis.

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**Fig. 9:** a) Axial MRI T2 fat sat image. A multilocular cystic lesion corresponding to the hidatid cyst is seen in the RHL with mild adjacent intra-hepatic biliary radicles dilatation (yellow arrow). b) Coronal MRI 3D MRCP ASSET. Communicating with the hydatid cyst there is a dilated bile duct (yellow arrow) that is extending across the right hepatic duct.

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Fig. 10: 73 y.o. woman. Abdominal discomfort and weight loss. a) Abdominal Doppler ultrasound. Anechoic lesion in RHL suggesting HC. Colour doppler at the level of the porta hepatis reveals the absence of flow in the main porta and portal vein branches. b) Contrast enhanced abdominal CT scan. Portal vein (orange arrow) is increased in caliber and has hypodense content corresponding to cystic filling of the portal lumen, secondary to HC invasion. c) Coronal abdominal CT MIP projection. Multilocular cystic lesion in segment VII (star) conditions portal venous invasion, appreciating hydatid intraluminal content in the portal vein (orange arrow) and portal cavernomatosis development (green arrow). d) CT coronal MIP projection at other level shows vascular recanalization of superior mesenteric vein (yellow arrow)

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Fig. 11: Same patient as Figure 10. a) Axial T2 MRI. Dilated portal vein with intraluminal hypointense linear tracts (orange arrow). b) Coronal 2D FIESTA MRI. Dilated portal vein and intrahepatic portal branches, showing hyperintense intraluminal content with multilocular appearance (orange arrow), secondary to vascular invasion of HC and portal cavernomatosis (green arrow). c) Coronal 3D MIP ASSET image showing multiple daughter vesicles replacing the lumen of the dilated main portal vein (orange arrow). A normal common bile duct is seen (blue arrow).

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Fig. 12: 80 y.o. woman with anemia and right iliac fossa pain. a&b) Abdominal ultrasound. Hypoechoic lesion suggestive of hepatic multiseptated HC. Gallbladder is not well delimited, presenting heterogeneous echogenic content and some acoustic shadow, which suggest biliary sludge/stones. c&d) Sagittal and coronal enhanced contrast abdominal CT. Multiseptated focal hypodense lesion suggesting HC (orange arrow). The gallbladder presented heterogeneous hypodense content, very similar to HC (green arrow).

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Fig. 13: 70 y.o male. with severe epigastric pain. a) Axial contrast-enhanced abdominal CT (February 2012). Calcified cystic lesion in segment VII and large partially calcified hypodense lesion that occupies great part of LHL, with air-fluid level, contacting with the stomach, viewing several air bubbles on the surface of the gastric wall (yellow arrow). At surgery a fistulous orifice was demonstrated between the stomach and the HC. b&c) Axial and sagittal abdominal contrast-enhanced CT (June 2012). Postsurgical changes of partial gastrectomy (green arrow). Multiple nodular mesenteric lesions are seen according to hydatid peritoneal dissemination, some of them with air-fluid level (red arrow). Implants are also seen in the muscular plane (blue arrow)

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Fig. 14: 82 y.o woman. Fever and abdominal pain. a) AP Abdominal X-ray showing a calcified lesions in right upper quadrant and a large air-fluid level (orange arrow). b&c) Coronal and sagittal contrast-enhanced abdominal CT. A large HC is communicating with the hepatic flexure of colon (green arrow) with secondary formation of significant air-fluid level (orange arrow) by fistulation mechanism. d) Barium enema 4 weeks later, showing postinflammatory parietal irregularity in the colonic wall, with minimal residual fistulous tract (red arrow).

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**Fig. 15:** 35 y.o female. Fever maintained for weeks and right flank pain. a)&b) Contrast-enhanced abdominal CT with sagittal and coronal reconstruction. Extensive hypodense tumor in the right hepatic lobe (RHL) invading the upper pole of right kidney c)&d) MRI Coronal 2D FIESTA fat suppressed image and 3D reconstruction. Great multi-lobulated mass is invading the right kidney. There is also segmental dilatation of intrahepatic bile ducts in the RHL.

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**Fig. 16:** 68 y.o. patient with right subcostal pain. History of previous hydatid disease.

a). Axial contrast-enhanced thoracic CT. Increased soft tissue in the extrapleural space of right hemithorax associated with subsegmental atelectasis bundles. b). Thoracic CT sagittal image shows focal diaphragmatic discontinuity (green arrow), with hydatid contents extending into the lung, developing a cavitated lesion at the posterior segment of LID. A calcified hydatid cyst in the right liver parenchyma (yellow arrow) is also evident.

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**Fig. 17:** 34 y.o. man arriving from Morocco presenting with fever, abdominal pain and vomiting for one week. a)&b). Axial and sagittal contrast-enhanced abdominal CT. Hypodense multilocular lesion in the caudate lobe (yellow arrow) in continuity with a cystic lesion in the lesser sac (blue arrow). A larger hypodense lesion in the mesenteric root (green arrow) is also seen. c) and d). MRI T2-weighted fat sat axial section and coronal 2D FIIESTA demonstrate a predominantly hyperintense extrahepatic lesion (blue arrows) with internal hypointense linear images, that may correspond to collapsed and thickened walls of the cyst. These findings suggest hydatid peritoneal dissemination, probably secondary to previous asymptomatic intra-peritoneal rupture of hydatid cyst.

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Fig. 18: 78 y.o. Extension study of vulvar cancer. a)Coronal T2-weighted MRI of the pelvis reveals a large tumor in Douglas sac, with smooth edges, predominantly hyperintense with several internal septa (yellow arrow). b)Axial T1-weighted MRI T1. Well defined large heterogeneous tumor in the presacral space with predominantly hypointense signal. c)3D LAVA dynamic contrast-enhanced MRI in sagittal projection. The large pelvic mass presents linear enhancement at the peripheral part of the capsule. Intense contrast enhancement is also seen corresponding to the vulvar carcinoma (green arrow)

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**Fig. 19:** Same patient as Figure 18. a). Axial contrast-enhanced pelvic CT. Large heterogeneous tumor in pelvis, with smooth edges and hypodense intralesional nodes appearing as a "wheel spoke" pattern, characteristic of hydatid disease. b). Sagittal abdomino-pelvic CT demonstrating the large pelvic cystic mass (star) and a small peritoneal calcified nodule adjacent to the anterior surface of the liver (arrow), indicative mechanism of peritoneal seeding of the disease.

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**Fig. 20:** 68 y.o. male presents with palpable mass in the left flank. 

a) Abdominal ultrasound at left iliac fossa demonstrates a large oval mass, predominantly hypoechoic, with well-defined edges.

b) Axial contrast-enhanced pelvic CT. The large hypodense tumor (star) located in left iliac fossa contacts with psoas muscle and the ipsilateral iliac bone originating cortical thinning (orange arrow).

c) Sagittal contrast-enhanced abdominopelvic CT. Besides the known tumor in the left flank (star), postoperative sequelae of prior thoracic surgery secondary to hydatid disease are evident, with multiple rib fracture callus formations (green arrows).

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**Fig. 21:** a) Ultrasound of upper abdomen demonstrating a predominantly hypoechoic heterogenous lesion suggestive of degenerated splenic hydatid cyst. b) Abdominal CT shows multiple splenic hydatid cysts. Detached calcified membranes produce a characteristic whirl pattern. Curvilinear calcifications can be seen in the pericyst. c) Coronal T2wi SSFSE MRI. Multiple splenic hydatid cysts are depicted as multilocular lesions with moderate hyperintensity on T2wi and a fibrotic thickened low signal intensity rim surrounding the cysts.

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Fig. 22: 65 y.o woman. Previous history of right liver resection and cholecystectomy due to hydatid disease. In a routine ultrasound a pancreatic tumor is detected. a). Axial contrast-enhanced abdominal CT demonstrates a well defined hypodense lesion in the neck of the pancreas (orange arrow). b). Coronal CT image. Hypodense nodular pancreatic lesion (orange arrow) and secondary changes to prior lobectomy (blue arrow) are evident. c). Axial T2 MRI reveals a lesion that is predominantly hyperintense, suggesting pancreatic cystic nature. d). 2D FIESTA coronal MRI. A predominantly hyperintense pancreatic lesion (orange arrow) is seen, not associated with dilatation of the main pancreatic duct.

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**Fig. 23:** 59 y.o. male presenting with dyspnea and chest pain. a). Axial contrast-enhanced thoracic CT reveals a hypodense excrescent lesion arising from LHL (star). b) Sagittal, axial and coronal contrast-enhanced thoracic CT. Hypodense heterogeneous solid tumor growing out of the left pulmonary artery wall and showing significant endoluminal growth (orange arrows). These finding correspond to vascular dissemination (hematogenous) of hydatid disease.

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**Fig. 24:** a) PA Chest radiograph showing a cavitated lesion in the right upper lobe (green arrow). b) Thoracic CT scan axial image showing a pulmonary cavitated lesion (green arrow) in the right upper lobe representing collapsed and crumpled endocyst floating freely in the cyst fluid ("water lily or camalote sign"). Other pulmonary nodular lesions are seen. c) Thoracoabdominal CT coronal image showing the cavitated lung lesion (green arrow) associated with multiple hydatid cysts in the liver (blue arrows).

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Fig. 25: 61 y.o. patient presented lumbar pain and sacral ulcer with history of vertebral hydatidosis surgically intervened. a) Abdominal radiography showing postchirurgical changes in thoracolumbar transition and bony destruction of T11, T12 and L1 vertebral bodies. b) Sagittal T2 and T1 weighted MRI scan of the thoracic spine showing multi-level involvement by numerous hydatid cysts with spinal intrusion and penetration of surrounding soft tissue. The cyst fluid appears iso-intense with the CSF in both T2 and T1-weighted images. There is mechanical failure with significant spondylolysthesia. Note also an hydatid cyst in paravertebral soft tissue (green arrow). c) Axial T2-weighted MRI scan showing multiple echinococcal cysts in the body and appendages of the vertebra, with intrusion of the hydatid cysts into the spinal canal

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Conclusion

Hydatid disease primarily affects the liver and typically demonstrates well-known characteristic imaging and serological findings.

However, it should be kept in mind that hydatid cysts can cause many potential local complications and secondary extrahepatic involvement that may be seen in different anatomic locations presenting as unusual US, CT and/or MRI findings.

The major teaching point of this exhibit is to learn the different appearances of abdominal hydatid disease and to understand the main complications and atypical manifestations in order to be able to recognize them and make a prompt accurate diagnosis and treatment.
References


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