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Complications of Hepatic Hydatid Cysts: Ultrasound, Computed Tomography, and Magnetic Resonance Diagnosis

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Abstract: A total of 179 hepatic hydatid cysts (HHCs) were studied by ultrasound (US), computed tomography (CT), and magnetic resonance imaging (MRI). The diagnosis of HHC complications was established by US and CT, which permitted a distinction between intact cysts and those presenting with contained rupture, as demonstrated by a collapsed endocyst or a globally echogenic appearance. The diagnosis of HHC perforation into the main biliary tree was 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 was diagnosed by demonstrating cyst wall discontinuity and the presence of hydatid material within these spaces. Ruptured and infected cysts were difficult to distinguish from ruptured cysts with sterile content. The role of MRI is yet to be defined in the assessment of HHC complications.

Key words: Liver, hydatid cysts – Liver, imaging studies – Hydatid cyst rupture, diagnosis.

The evolution of an hepatic hydatid cyst (HHC) involves a number of stages [1–4]. Initially, the cyst progressively increases in size, while the host develops a pericyst layer in an attempt to limit expansion of the cyst. Subsequently, the cyst undergoes internal proliferation (involving hydatid sand, daughter vesicles, and membranes). The outer layer presents biliary radicles and neighboring structures involved in the development of posterior complications [5–7].

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With time, the cysts seem markedly prone to rupture [6, 8, 9]. The latter may occur in three different ways: contained rupture, rupture communicating with the biliary tract, and direct rupture [1, 5, 6]. We have attempted to characterize these three forms using ultrasound (US), computerized tomography (CT), and magnetic resonance imaging (MRI).

Materials and Methods

We studied 179 HHCs in 221 patients with abdominal hydatidosis seen over a period of $8^1/_2$ years (85.5% liver involvement). Clinical history and surgical as well as pathological findings were analyzed retrospectively in all cases.

All patients were subjected to real-time US studies with 3.5- and/or 5-MHz transducers. High-resolution CT was used in 34 patients before and after administration of intravenous iodine contrast. The MRI was in turn performed on 7 patients with SE T1-weighted (TR = 350 ms, TE = 20 ms) and T2-weighted (TR = 2100 ms, TE = 50 and 100 ms) sequences. The HHCs were classified into four groups: noncomplicated or simple (group I), and complicated or ruptured (infected or otherwise); the latter classification was in turn divided into contained rupture (endocyst rupture) (group II), communicating rupture (the broken endocyst allowing passage of hydatid material into the biliary tract) (group III), and direct rupture (both endo- and pericyst rupture, cyst contents passing into the peritoneal and/or pleural cavity) (group IV).

Results

Group I: Noncomplicated HHC (Figs. 1 and 2)

With 121 patients, this was the most numerous group (67.7%). Age varied from 17 to 79 (mean, 33 years). In general, the cysts were detected when exploring patients with expansive liver lesions, or were incidental observations. Surgery revealed crystalline, occasionally yellowish liquid, with daughter vesicles in some cases and few or no membranes.

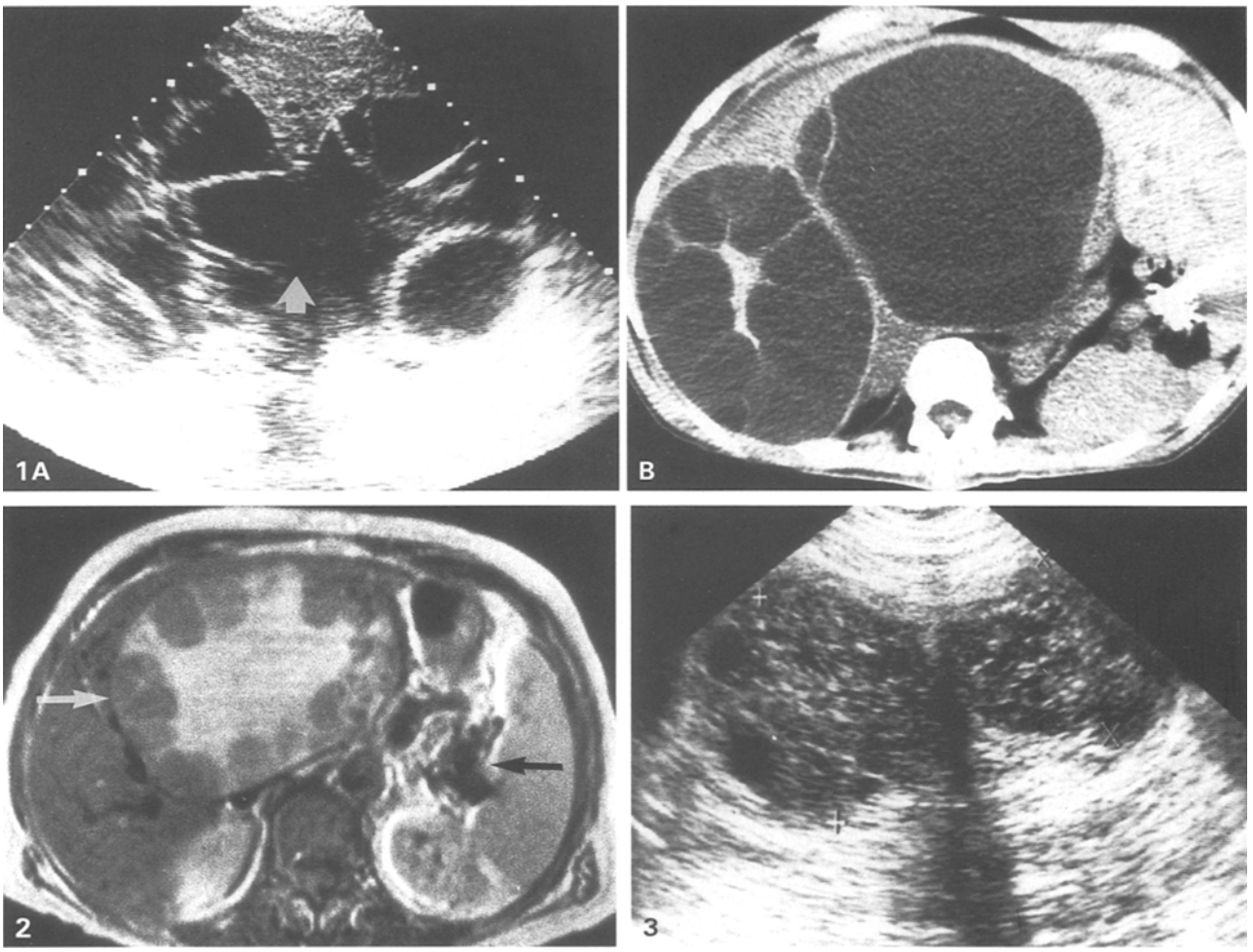

Fig. 1. Hydatid cysts of the liver in a 35-year-old man. **A** The US shows two multivesicular cysts with an apparent communication between them (*arrow*). **B** The CT demonstrated the integrity of both pericysts: note the internal calcification of the laterally situated cysts. At surgery, both pericysts were intact.

Fig. 2. Hydatid cyst in a 54-year-old woman with jaundice. Liver MRI in T2-weighted sequences (TR = 2100 ms, TE = 50 ms, FOV = 350, 0.5T). The pericyst (*white arrow*) appears hypointense, whereas the hydatid matrix is hyperintense in T2. Note how the peripheral daughter vesicles are less intense than the matrix. Attention is also drawn to the marked collateral splenic circulation (*black arrow*) produced by portal hypertension secondary to the compressive hepatic lesion. At surgery an intact HHC was observed, along with portal hypertension and concomitant choledocholithiasis.

Fig. 3. The HHC in a 32-year-old patient with right hypochondrial discomfort and sensation of mass. The US reveals two rounded, hypoechoic lesions with an internal snowstorm pattern, peripheral daughter vesicles, and posterior acoustic enhancement. This globally echogenic appearance of the HHC may be observed in both contained ruptures of the cyst (as in this case) and in communicating cyst ruptures due to fissures.

The US (Fig. 1 A) revealed an oval or rounded image, with posterior acoustic enhancement in all cases. The cysts were uni- or multivesicular in appearance, with an anechoic matrix. In six univesicular cases a germination membrane was either intact or slightly detached from the internal layer. Sixteen HHCs were globally calcified. Computed tomography (Fig. 1 B) was performed in 3 cases belonging to this group, all homogeneously hypodense with good pericyst visualization and lack of signs suggesting rupture. Magnetic resonance imaging, performed in 2 cases, showed the cysts with an hypointense pericyst and daughter vesicles less intense than the matrix in all sequences (Fig. 2).

Group II: HHC Presenting with Contained Rupture (Fig. 3)

This group involved 21 patients (11.7%), who were from 21 to 75 years old (mean: 48 years). Right hypochondrial pain or discomfort was the main cause of consultation; expansive hepatic lesions

were less common and a minority were asymptomatic. Surgery revealed hydatid content with many daughter vesicles of varying size and numerous internal membranes. Intraoperative cholangiograms were normal. No cases were infected.

The US showed an oval or rounded morphology with posterior acoustic enhancement in all cases. No cyst was simple univesicular in appearance; 5 patients presented uni- or multivesicular cysts with internal linear or spiral structures representing the detached collapsed endocyst. The predominant internal echogenic pattern (12 cases) was in the hypoechoic or "snowstorm" pattern. Computed tomography was performed in 9 cases; peripheral calcium was observed in 6, daughter vesicles in 4, and membranes in 5 cases. The overall HHC attenuation coefficient was similar to water (0–15 HU) in 8 cases; only in 1 case was the cyst isodense with the liver prior to contrast injection and hypodense following the latter (the US appearance was typically in the snowstorm pattern). In 2 cases, MRI showed a cyst with internal hypointense linear septations, and no discernible daughter vesicles (being globally echogenic on US).

Group III:
HHC with Communicating Rupture (Fig. 4)

This group involved 27 patients (15%), varying in age from 19 to 76 years (mean: 46 years). They were divided into two subgroups according to the type of communication with the biliary tree: (a) fissures (15 cases, 55.5%), i.e., small fistulas opening into peripheral biliary radicles; and (b) perforations (12 cases, 44.5%), i.e., cyst communication with larger biliary radicles or the main biliary tract [6]. Among the first 15 patients biliary colic predominated in 7, along with fever in 6 infected cases. One patient presented with jaundice, whereas another was asymptomatic. In the perforation subgroup the predominant clinical feature was jaundice (8 cases, 2 with cholangitis); biliary colic was observed in 2 patients, whereas only 1 case was asymptomatic. There had been previous episodes of jaundice and/or cholangitis in 8 cases. Fever was detected in 1 infected case.

In the group of fissured HHC, almost all cases were globally echogenic on US, with a predominance of the hypoechoic or snowstorm pattern. Only one cyst was multivesicular, with scarce echogenic matrix. The CT (Fig. 4C and D) was performed on 6 patients with cyst fissures opening into the biliary tree: cyst wall calcification was encountered in 4 cases, with germinative membrane detachment and curved linear membranes within the cyst in 3 patients. All HHCs were hypodense, and air was detected in 1 of them, which proved to be infected. The MRI was performed in 2 cases, their appearance being similar to that of multivesicular cysts.

In the perforated cyst subgroup, all HHCs were globally echogenic, except for 1 case, which was multivesicular and presented an echogenic matrix. The extrahepatic biliary tract was visualized in 10 of the 12 perforated cases; the tract was in turn distended in 9 patients (mean 10 mm, with a range of 8–15 mm). In 8 cases hydatid material was detected within the main biliary tract in the form of rounded (echogenic or cystic) or linear structures without posterior acoustic shadowing; these structures represented daughter vesicles and/or membranes. In 1 case the biliary tract was not distended, and surgery failed to reveal hydatid material within it. In 2 patients the main biliary tract was not visualized, but surgery revealed hydatid material within it. Hydatid material was detected in the gallbladder in 4 cases, with cholelithiasis in 4 (both findings coincided in 2 patients). Nine patients presented HHC communication with a biliary radicle or the extrahepatic ducts in the form of a projection emerging from the cyst; in 7 of these cases hydatid material was identified crossing the communication.

Computed tomography was performed on 9 perforated cases, with daughter vesicles and membranes observed in 8, and cyst wall discontinuity in 7 patients. This discontinuity was always in close proximity to the hepatic hilus, and associated with intra- or extrahepatic biliary tract distension. In 2 of these cases lateral cyst wall discontinuity was also observed, without evidence of biliary tract communication. Hydatid material was observed within the gallbladder in 4 of the 9 cases, along with high-density linear structures in the biliary tract in 7 patients.

Group IV:
HHC with Direct Rupture (Figs. 5 and 6)

This group included 10 patients (5.6%), varying in age from 23 to 68 years (mean: 59 years). The predominant clinical finding was right hypochondrial pain in 9. Fever was detected in 3 cases (all infected). Leukocytosis (over 10,000 cells per field) was observed in 8 cases. Surgery revealed hydatid material within the pleural or peritoneal cavity, or within other spaces.

The US (Fig. 5A) showed all cases to be globally echogenic, with predominance of an isoechoic pattern. All cases of diaphragmatic rupture

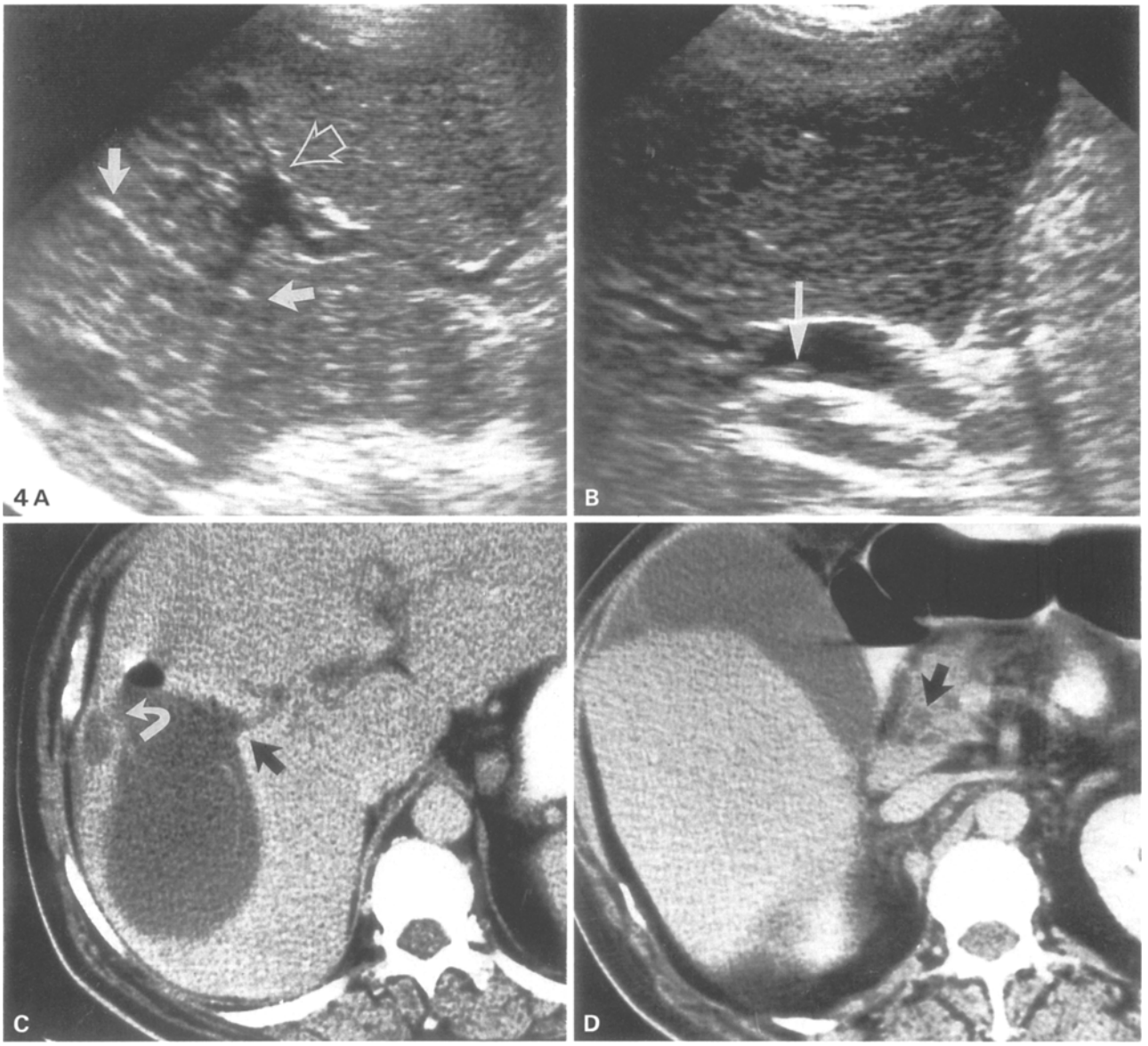

Fig. 4. Ruptured HHC in a patient with episodes of cholangitis, jaundice, and leukocytosis. The US (**A and B**) shows a collapsed echogenic HHC (*short arrows* in A) with a perforation (*open arrow*) into an intrahepatic biliary radicle. Note the presence of an echogenic daughter vesicle within the distended common bile duct (*arrow* in B). The CT (**C**) reveals an HHC with air in its interior and two ruptures or discontinuities of the pericyst – one communicating with a distended intrahepatic biliary radicle (*black arrow*) and the other reflecting a direct intrahepatic rupture (*white curved arrow*). (**D**) A distended distal intrapancreatic choledochus with intraluminal hydatid material (*arrow*).

with hepatopulmonary communication (4 cases) had pleural effusion, and the diaphragmatic rupture and communication was demonstrated in all. In 3 cases air was detected within the cyst. The CT (Fig. 5B) in these 4 cases revealed both the air and pleural effusion, along with secondary lung involvement in all cases. In 2 patients, daughter vesicles were visualized within the thoracic cavity (not detected by US).

In ruptures opening to the subcapsular hepatic space (2 cases) or abdominal wall (1 case), and in intrahepatic ruptures (1 case, with hydatid material outside the mother cyst and delimited by liver parenchyma), US was effective in demonstrating the communication. Only in the case of rupture opening of the peritoneal cavity was US unable to detect direct communication, although the echogenic appearance of the HHC – including the snowstorm pattern – with internal membranes and free intraperitoneal liquid, was suggestive of complicated HHC. In this latter case CT revealed a thick, irregular, and partially discontinuous peri-

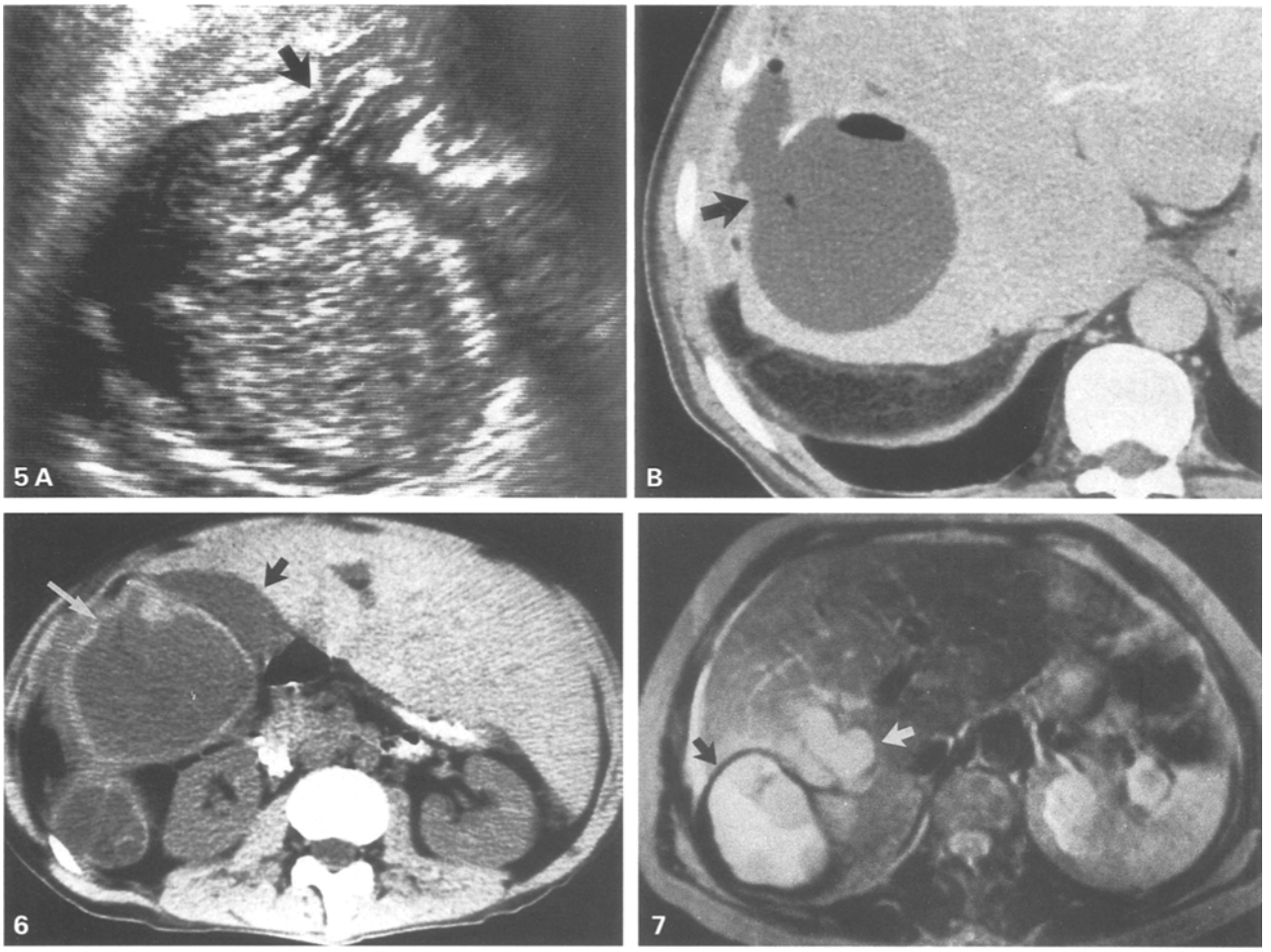

Fig. 5. Perforating HHC in a 58-year-old patient with sudden painful crises in the right hypochondrium. The US (**A**) shows a globally echogenic HHC with perforation of the wall and release of its contents through the gap (*arrow*); diaphragmatic integrity is difficult to evaluate. The CT (**B**) shows the cyst with an air–liquid level within its interior and a perforation to the subhepatic space (*arrow*) that extends to the pleuropulmonary cavity. The presence of cholangiographic contrast within the biliary tree facilitates the exclusion of any concomitant communication with the biliary tract.

Fig. 6. The HHC in a 46-year-old patient with peritoneal signs. Liver CT shows an HHC in the right lobe close to the gallbladder (*black arrow*). Note the irregular cyst contour and the discontinuity in its wall (*white arrow*), including pericyst liquid. This set of data suggested HHC with direct rupture into the abdominal cavity (as later confirmed surgically). Attention is drawn to a second cyst in its proximity, together with a displacement of the left hepatic lobe due to the cysts.

Fig. 7. The MRI study to detect or exclude liver metastases in a 31-year-old female cancer patient. Sequences obtained in T2 (TR = 2100 ms, TE = 50 ms, FOV = 350, 0.5T); an HHC is observed within the right hepatic lobe, with a hypointense pericyst (*black arrow*). This layer is irregular and presents a medial rupture through which daughter vesicles are released (*arrow*). These are easily distinguished from the hydatid matrix in a direct intrahepatic rupture.

cyst with liquid in its vicinity; diagnosis was given as rupture opening to the peritoneal cavity (Fig. 6). One of the 2 cases of rupture into the subhepatic space evolved toward hepatopulmonary communication, with a decrease in cyst size and air in its interior. Magnetic resonance imaging was performed in 1 case of direct intrahepatic rupture. Daughter vesicles were clearly demonstrated outside the HHC (Fig. 7).

Discussion

The complications observed with HHCs are rupture and infection; it is accepted that the latter does not occur without the former [5]. The natural evolution of HHC implies rupture as a complication in 50–90% of cases [8, 9]. In our series, rupture incidence was approximately 35%.

Cyst rupture is mainly due to degeneration of the parasite's membranes as a result of age, chemical reactions, or host defense mechanisms [1, 5]. In contained ruptures the endocyst breaks, with collapse and wrinkling of the germinative mem-

brane within the cyst: the pericyst remains intact. This type of rupture may with time evolve toward a communicating rupture if hydatid material passes into the biliary radicles incorporated to the pericyst. This phenomenon is favored by an intracyst pressure (60–80 mm water) greater than the intrabiliary pressure (20–25 cm water) [6, 10]. Cyst ruptures in which hydatid material passes into small biliary radicles are considered fissures, whereas communications with larger biliary radicles or the main biliary duct are considered perforations [1, 6, 9]. Direct rupture represents the third variety; in this case both layers of endo- and pericyst rupture, whereby hydatid material passes into the pleural or peritoneal cavity, or into other spaces. Only communicated and direct ruptures are susceptible to infection, as they alone disrupt cyst isolation from the exterior [11, 12].

Ruptured HHCs are rarely asymptomatic [1, 11, 12]. A clinical warning sign is often encountered; this takes the form of right hypochondrial pain or discomfort in contained ruptures, biliary colic in communicating ruptures with fissures, and obstructive jaundice in direct perforation [6]. Fever indicates infection of the ruptured HHC, which in turn is more common in the case of fissured cysts. Leukocytosis may be observed in both communicating and direct ruptures – whether infected or not. Hence, it cannot be used to differentiate between infected and uninfected ruptured cysts.

The different evolutive stages of the cyst offer different radiological images [1, 2, 4]. With US, contained ruptured cysts contain floating, wavy, or collapsed membranes (the water lily sign); this occurs when the endocyst breaks and detaches from the pericyst. A globally solid appearance may also be observed [1, 2, 11, 12], and was in fact the most common image found in our series. This echogenic appearance is a result of the proliferative capacity of the germinative membrane in the case of contained rupture, favored by the mature condition of the cyst [1]. A differential diagnosis should be made between contained cyst rupture, and both intact HHC and fissured communicating rupture. Differentiation of the former is usually straighforward with imaging techniques; in this sense, US is sufficiently specific [1–3, 11, 12].

Cyst rupture and communication with the biliary system is common [8, 9]. In our experience the diagnosis of communicating rupture opening to the biliary system cannot be established preoperatively in most cases involving small fissures. Differentiation between contained ruptures and small communicating ruptures was usually based on the findings of preoperative cholangiography. Neither US nor CT were able to make this distinction, as the fissures were too small to be detected as cyst wall discontinuities. Even in the case of a patient with jaundice, globally echogenic HHC, and a distended biliary tract without intraluminal material, a differential diagnosis should include choledocholithiasis – the most commonly associated pathology in these patients [9]. In patients with fissure complications (biliary obstruction due to hydatid sand or odditis secondary to the passage of hydatid liquid), the correct diagnosis is better established by retrograde endoscopic cholangiography [13].

The diagnosis of cyst perforation opening to the main biliary tree is made by demonstrating the cyst characteristics and presence of hydatid material within the biliary duct system. The observation of a cyst wall discontinuity provides definitive diagnosis. Ruptured cysts opening to the biliary system tend to be globally echogenic on US, although their CT appearance is generally similar to that of other HHCs. Occasionally, the observation of air within the cyst in the presence of aerobilia produced by the passage of hydatid material through the duodenal papilla makes it possible to establish a diagnosis of communication rupture (provided infection is clinically excluded). A liquid–liquid level has also been described in communicated cysts, although similar levels have been reported in intact HHCs caused by hydatid sand [12] and in infected cysts [14]; in our experience such levels are rare. Within the biliary system the daughter vesicles present sonographically as rounded anechoic or echogenic structures; the latter represent younger daughter vesicles. In turn, the fragmented membranes present as highly echogenic linear structures. Occasionally, the gallbladder may be seen containing daughter vesicles and fragmented membranes without hydatid material within the biliary ducts.

The demonstration of a communication between the cyst and biliary system establishes the diagnosis of perforation. This was detected sonographically as a projection of the cyst toward the hepatic hilus, with a distended biliary tree (segmentary, lobar, or main). The observation of hydatid material prolapsing from the cyst into the biliary tract and crossing the communication is pathognomonic. On CT scan the communication is seen as a hypodense line in the wall (pericyst discontinuity), more accentuated in the area of the HHC close to the hepatic hilus after administering intravenous iodine contrast.

When the pericyst ruptures and the cyst interior communicates with an anatomical cavity or space, the rupture is termed direct. The released hydatid

material may remain confined to the vicinity of the mother cyst, or discharge into the subcapsular space, abdominal wall, peritoneum, or pleural cavity (through the diaphragm). These complications reflect an evolutive stage, as prior to rupture into the pleural or peritoneal cavity the cyst must have undergone both intrahepatic and subcapsular ruptures. In our experience, CT is more sensitive and specific than US in establishing diagnosis. This is made by demonstrating the HHC and the communication in the form of a pericyst discontinuity. The observation of hydatid material emerging from the cyst or within the above-mentioned cavities confirms diagnosis.

Bacterial infection of the HHC is always secondary to cyst rupture opening to the exterior [12]. A moderate percentage of ruptured cysts (approximately 25% in our series) may become infected and present clinically as abcesses. The literature describes numerous signs of cyst infection: poorly defined delimitation [2, 12, 14], both hypoechoic [2] and hyperechoic internal echoes [4], and the presence of air with an air–liquid level [14, 15] or liquid–liquid level [11, 14]. However, both in our experience and in that of others [11], these characteristics may be observed in intact HHCs as well as in ruptured uninfected cysts. Computed tomography shares this lack of sensitivity [11, 14, 15], as the observation of irregular walls with nonhomogeneous content and levels may also be seen in aseptic cysts. The presence of air within the cyst establishes the diagnosis of rupture (communicating or direct), although it does not necessarily imply infection. This may be due to three main mechanisms: penetration of air through the diaphragm via a bronchial fistula, communication with the biliary tract in the presence of aerobilia, and cyst infection by anaerobes. Consequently, current radiological methods are unable to differentiate an infected ruptured HHC from a sterile cyst.

Magnetic resonance imaging may likewise be applied to the evaluation of liver echinococcosis. This technique clearly visualizes the pericyst, daughter vesicles, and matrix (Fig. 2). In our experience and in that of others [16], the pericyst is hypointense in both T1 and T2 sequences (Figs. 2 and 7). This observation, together with the lesser spatial resolution of MRI compared with CT, suggests that the demonstration of pericyst discontinuities is less feasible with MRI, thus making rupture demonstration more difficult. The hydatid matrix appears hypointense in T1 and markedly hyperintense in T2, whereas the daughter vesicles are more hypointense in T2 than the matrix of the mother cyst (Figs. 2 and 7). This relationship is apparently maintained in the presence of different types of rupture [17] (Fig. 7). Possibly, cyst infection may alter the HHC signal in both T1 and T2 weighted sequences. This would make it possible to establish a diagnosis of abscess.

The ruptured hepatic hydatid cyst is associated with a high mortality rate [8]. Consequently, cyst rupture should be diagnosed preoperatively to avoid any unnecessary delay in prompt surgical management of such complications.

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