Cystic Hepatic Lesions: A Review and an Algorithmic Approach

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OBJECTIVE. The purpose of this article is to review the different cystic hepatic lesions, with an emphasis on the imaging features that help to differentiate them, and to propose a practical algorithm for approaching the diagnosis of these lesions.

CONCLUSION. The number and morphology of the lesions and determination of whether there is a solid component are key imaging features that are helpful for approaching the diagnosis of cystic hepatic lesions. Familiarity with these features and knowledge of the clinical associations will help the radiologist to establish a definitive diagnosis or provide a reasonable differential diagnosis.

Keywords: acute generalized exanthematous pustulosis, angioneurotic edema, cutaneous reactions, delayed adverse drug reactions, iodinated contrast media reactions

DOI:10.2214/AJR.13.12386

Received December 14, 2013; accepted after revision March 21, 2014.

Presented as a poster at the 2013 annual meeting of the Association of University Radiologists, Los Angeles, CA.

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AJR 2014; 203:1192–1204

0361–803X/14/2036–1192

Cystic hepatic lesions are commonly encountered in daily practice. The differential diagnoses range from benign lesions of no clinical significance to malignant and potentially lethal conditions. Many cystic hepatic lesions have classic imaging findings, and the diagnosis can be made with certainty on the basis of imaging alone. In other cases, recognizing key radiologic features in combination with reviewing the clinical data usually allows the correct diagnosis.

Cystic hepatic lesions can be divided into developmental, inflammatory, neoplastic, and trauma-related lesions (Table 1). An incidental simple hepatic cyst is the most commonly encountered pathologic finding. The number and morphology of the lesions and determination of whether there is a solid component are key imaging features that are helpful for approaching the diagnosis of cystic hepatic lesions. The pretest probability of a diagnosis is highly affected by the patient's comorbidities and the clinical and laboratory data; thus, imaging studies should be interpreted in the context of the other clinical information for that particular patient. Except simple hepatic cysts and polycystic liver disease, which can be confidently diagnosed on the basis of ultrasound only, contrast-enhanced CT or MRI is essential to establish a definitive diagnosis or provide a reasonable differential diagnosis.

Developmental Lesions

Hepatic Cysts

Cysts are the most commonly encountered hepatic lesion, occurring in 2.5% of the general population [1], and have a slight predominance in females (female-male ratio, 1.5:1) [2]. Hepatic cysts are thought to be of biliary origin as a result of deranged development of the biliary tree (i.e., a hamartoma of biliary origin or so-called "von Meyenburg complex") [2]. The wall of a hepatic cyst is lined by cuboidal biliary epithelium, and the cavity is filled with serous fluid similar to plasma; however, there is no communication with the biliary tree. Cysts are generally asymptomatic, and no treatment is needed unless they become large and symptomatic. In the latter cases, the treatment options include percutaneous drainage with sclerotherapy, surgical resection, or marsupialization [3, 4].

Hepatic cysts are typically round or ovoid structures that have an imperceptible wall. These cysts are usually multiple in number and vary in size. The ultrasound features of hepatic cysts are similar to those of simple cysts in other organs. Common features include a well-marginated, anechoic structure with enhancement of the posterior wall and increased through-transmission (Fig. 1). On CT and MRI, simple cysts have attenuation (0–15 HU) and signal intensity (T1 hypointensity, T2 hyperintensity) similar to water. Simple cysts do not show enhancement af-

TABLE 1: Summary and Key Imaging and Clinical Findings of Cystic Hepatic Lesions

Lesion	Key Imaging Findings	Key Clinical Data
Developmental		
Simple cyst	Solitary cyst or multiple cysts	
Biliary hamartoma	Multiple irregular lesions	
	May have enhancing component	
Caroli disease	Multiple lesions	
	Enhancing "central dot" sign	
	Communicating with biliary tree	
Polycystic liver disease	Multiple large cysts	History of polycystic renal disease
	Usually associated with renal cysts	
Ciliated foregut duplication cyst	Classic subcapsular location in medial segment	
Inflammatory		
Pyogenic abscess	Complex cyst with enhancing rim	Clinical and laboratory findings of infection
Amebic abscess	Complex cyst with "double-target" appearance	Patient is from endemic areas
Hydatid cyst	Complex cyst with peripheral daughter cysts	Patient is from endemic areas
Fungal microabscess	Innumerable small cysts	Patient is immunocompromised
	Splenic and renal lesions may be present	
Intrahepatic pseudocyst	Findings of pancreatitis	Clinical and laboratory findings of pancreatitis
	Pseudocysts may be present in lesser sac	
Neoplastic		
Biliary cystadenoma and cystadenocarcinoma	Large complex cystic lesions with enhancing septations	Absence of infection or known metastatic disease
Cystic HCC	Complex lesion	Liver cirrhosis and increased α-fetoprotein level
	Hypervascular component with washout on portal venous phase	
Cystic metastasis	Multiple complex cystic lesions with enhancing component	History of malignancy
Undifferentiated embryonal carcinoma	Large complex cystic lesion on CT and MRI	Usually seen in adolescents
	Solid appearance on ultrasound	
Trauma-related		
Biloma	Large simple cyst with or without an enhancing pseudocapsule	History of trauma, surgery, or intervention
Seroma and hematoma	Cyst with variable density and intensity	History of trauma, surgery, or intervention
	No enhancement	

Note—HCC = hepatocellular carcinoma.

ter the administration of IV contrast material. Hepatic cysts can rarely become complex as a result of hemorrhage or superinfection; sequelae include the development of internal septations, rim calcification, and increased attenuation or heterogeneous signal intensity.

Biliary Hamartoma (von Meyenburg Complex)

Biliary hamartomas, also known as von Meyenburg complexes, are benign congenital lesions consisting of dilated small bile ducts surrounded by fibrous stroma [5]. Although biliary hamartoma has a reported incidence of 5.6% in autopsies [6] and 0.6% in specimens from needle liver biopsies [7] in the pathology literature, it is rarely diagnosed radiologically presumably because of its small size and the fact that it does not cause symptoms [8]. No sex predilection has been reported. This condition is caused by a ductal plate malformation with deficient remodeling of the primitive ductal plate [5, 9]. Because biliary hamartomas are asymptomatic and are almost always discovered incidentally, they require no treatment.

On imaging, biliary hamartomas present as multiple, small (< 15 mm), round or irregular scattered cysts with a predilection for the subcapsular region (Fig. 2). They typically have a simple cystic appearance on CT (i.e., nonenhancing, hypoattenuation) and MRI (i.e., high T2 signal intensity). Occasionally rim enhancement is observed and is attributed to increased enhancement of the adjacent compressed liver parenchyma [10]. The ultrasound findings of biliary hamartomas are variable because of the small size of these lesions: Cysts might appear anechoic, hypoechoic, or hyperechoic. Additionally, some lesions may show reverberation artifact caused by the close proximity of their interfaces [8]. The lack of communication with

the biliary system helps to differentiate biliary hamartomas from Caroli disease.

Caroli Disease

Caroli disease is a benign entity that manifests with saccular dilatation of large intrahepatic bile ducts [11]. It is a rare entity with no sex predilection. In most cases, transmission of the disease is autosomal recessive. Caroli disease is associated with other diseases along the spectrum of ductal plate malformations (e.g., biliary hamartomas, polycystic liver disease, or hepatic fibrosis), polycystic kidney disease, or renal tubular ectasia [12]. The combination of Caroli disease and hepatic fibrosis is designated as Caroli syndrome, which is the more common variant [13]. In the revised Todani classification of biliary cysts, Caroli disease is classified as type V [14]. The pathogenesis of this disease stems from the arrest or derangement in ductal plate remodeling of the large ducts. Patients usually become symptomatic by the age of 30 years, although symptoms may manifest earlier in those with Caroli syndrome. Complications include recurrent cholangitis and abscess, stone formation, cholangiocarcinoma, and the development of secondary biliary cirrhosis. Patients with recurrent bouts of cholangitis or those with biliary cirrhosis and portal hypertension will benefit from liver transplantation (level of evidence IIc). Hepatectomy can be curative in rare cases in patients with segmental or lobar disease (level of evidence IIc).

On imaging, Caroli disease manifests as multiple intrahepatic cysts of varying sizes that communicate with the biliary system [15] (Fig. 3). The extrahepatic ducts remain intact. The involvement can be diffuse or localized to one segment or one lobe, usually the left lobe. Thin-section CT images and multiplanar reformations are helpful in showing communication between the Caroli cysts and the biliary tree. Communication with the biliary system can be further confirmed on cholangiography (percutaneous transhepatic cholangiography or ERCP) or on MRI performed using a hepatobiliary contrast agent, such as gadoxetate disodium. On CT and MRI, the lesions are cystic and usually have a central enhancing component, the "central dot" sign, which is the portal radicle.

Polycystic Liver Disease

Polycystic liver disease is part of fibropolycystic liver disease manifesting with multiple simple hepatic cysts. It is an autosomal-dominant condition that can be associated with autosomal-dominant polycystic kidney disease, which is found in 50% of these patients [16]. Polycystic liver disease is a rare condition with a slight predominance in females [16]. Its cause is maldevelopment of the ductal plate that affects the small intrahepatic bile ducts [17]. Polycystic liver disease can be associated with other disorders along the spectrum of ductal plate malformations such as Caroli disease, biliary hamartoma, or hepatic fibrosis. Histologically, there are two types of cysts: intrahepatic and peribiliary cysts [18]. The intrahepatic cysts are similar to simple hepatic cysts: They are lined by cuboidal biliary epithelium and contain plasmalike serous fluid. The peribiliary cysts arise from dilated peribiliary glands. The cysts emerge after puberty and significantly increase in size and number during adulthood [16]. The majority of patients are asymptomatic, and polycystic liver disease progresses to become advanced liver disease or to cause symptoms as a result of massive hepatomegaly or a cyst complication in only a minority of cases. Common complications of polycystic liver disease include cyst hemorrhage, rupture, or superinfection. The treatment options include percutaneous aspiration, sclerosis, or resection of the dominant complicated cyst. The ultimate treatment of advanced cases is liver transplantation.

The intrahepatic cysts seen in patients with polycystic liver disease are usually peripherally located and vary in size, ranging from a few millimeters to 80 mm (Fig. 4). The peribiliary cysts are typically small (< 10 mm) and have a periportal distribution [19]. The cysts in patients with polycystic liver disease typically appear to be simple cysts on imaging. MRI is the best modality for identifying cysts complicated by hemorrhage or infection [19]. CT findings suggestive of cyst infection include the development of a fluid level, wall thickening, calcification, or internal gas [17].

Ciliated Hepatic Foregut Duplication Cyst

A ciliated hepatic foregut duplication cyst is a rare congenital cystic lesion that is thought to arise from the embryonic foregut. It is lined by ciliated pseudostratified columnar epithelium, which is similar to respiratory tract epithelium. A ciliated hepatic foregut duplication cyst has many similarities with a bronchogenic cyst except that a bronchogenic cyst lacks cartilage [20]. A ciliated hepatic foregut duplication cyst is a solitary lesion that typically measures less than 3 cm and is most commonly located in the subcapsular aspect of segment IV [21] (Fig. 5), but it may also occur in the anterior segment (segments V and VIII). The majority of patients are asymptomatic, and the ciliated hepatic foregut duplication cyst is discovered incidentally. However, one case of portal hypertension caused by mass effect [22] and a few cases of malignant transformation to squamous cell carcinoma [23, 24] have been reported. Given the reported risk of malignant transformation, ciliated hepatic foregut duplication cysts that are symptomatic, are enlarging, are larger than 4 cm, or contain atypical features (e.g., solid components, thick septations) should be resected (level of evidence of III and IV) [25].

These lesions are anechoic or hypoechoic on ultrasound, have high signal intensity on T2-weighted imaging, and do not show enhancement on MRI [26]. The cyst content ranges from clear serous fluid to mucous fluid of different viscosities. Accordingly, CT attenuation and T1 signal intensity vary [21].

Inflammatory Lesions

Pyogenic Liver Abscess

The number of liver abscesses due to bacterial infection has been increasing in the United States, purportedly because of increases in liver transplantations and biliary malignancies; however, the rate of liver abscess in the United States remains lower than that in Eastern Asia [27]. The disease has a slight preponderance in males [27], and risk factors include diabetes [28], gastrointestinal tract cancers [29], diverticulitis [30], cholangitis, cholecystitis, and recent hepatobiliary surgery or trauma [31]. Patients present with fever, chills, jaundice, and weight loss, and frank sepsis at presentation is rare [31]; the mortality rate is approximately 6% [27]. Cultures of aspirates are usually negative. When an organism is identified, *Klebsiella pneumonia*, *Escherichia coli*, and *Staphylococcus* species are most commonly isolated, and the infection may be polymicrobial [27, 32]. Management of pyogenic liver abscess always includes IV antibiotics, but percutaneous drainage catheter placement or aspiration will be required to treat half of patients [32]. For abscesses smaller than 5 cm, needle aspiration may be considered; large abscesses should be drained with a catheter [33]. Percutaneous treatment fails

in approximately 10% of abscesses; in those cases, surgical intervention is required [27]. Factors that increase the risk for failure of percutaneous therapy include multiloculation, connections with the biliary system, or infection with yeast organisms [34, 35].

Abscesses are more likely to form in the right lobe, although left lobar disease and bilobar disease also occur [31, 36, 37]. On ultrasound, a liver abscess may appear as an anechoic mass with well-defined or indistinct borders and with increased through-transmission and may possibly contain echogenic debris or gas. When the infectious agent is *Klebsiella* organisms, the lesion is more likely to be solid and to yield little pus at drainage [37]. On CT, a pyogenic liver abscess is typically iso- to hypoattenuating compared with background liver on the unenhanced phase and has a peripheral rim of enhancement on administration of IV contrast material (Fig. 6). A "double-target" sign—or a central hypoattenuating lesion surrounded by a ring of enhancing tissue and encircled by an outer rim of hypoattenuation—may also be evident; this sign indicates an abscess but is not specific for a pyogenic source [38]. Infrequently (i.e., < 10% of cases), there may be associated findings of thrombophlebitis, gas within the abscess cavity, or pneumobilia. Lesions may be as small as a few centimeters or as large as 14 cm [36]. On MRI, the central portion of the lesion will show low signal intensity on T1-weighted imaging and high signal intensity on T2-weighted imaging; in addition, a peripheral halo of hyperintensity indicating edema may be seen on T2-weighted imaging.

Amebic Liver Abscess

Infection with *Entamoeba histolytica* is endemic in Mexico, Central and South America, India, Southeast Asia, and Africa, and the number of symptomatic infections worldwide is estimated at 50 million [39]. In the United States, amebic liver abscesses are rare (1.38 infected persons per 1 million people) and are distributed mostly among young Hispanic males in the Southwest region of the country [40]. Transmission of *Entamoeba histolytica* infection is through the fecal-oral route, with trophozoites invading the colonic epithelium and spreading to other sites hematogenously [41]. Liver abscess formation is the most common extraintestinal manifestation of *Entamoeba* infection, although fewer than 1% of those infected will have infection outside the gastrointestinal tract, such as within the liver, peritoneum, pleural space, lung, pericardium, skin, or brain [41]. Hepatic involvement is up to 10 times more common in males than females, possibly because of hormonal factors and sex differences in background liver disease [42]. Mortality from amebic liver abscess is approximately 30% [43]. Treatment of amebic liver abscess is an antiparasitic agent, such as metronidazole, and possibly includes image-guided drainage. For abscesses smaller than 5 cm in diameter, drug therapy is sufficient. There is no firm evidence in the literature about using percutaneous therapy for the management of lesions between 5 and 10 cm. Large (> 10 cm) abscesses are at high risk for treatment failure with drugs alone [44], and lesions on or in the left lobe are more likely to have complications such as rupture [41]: These abscesses should be drained percutaneously, and the drain should be left in place [45, 46].

The imaging characteristics of amebic and pyogenic liver abscesses are virtually indistinguishable, and the diagnosis is typically made on the basis of clinical and serologic findings [47–49]. Extrahepatic disease, such as a right pleural effusion, a pericardial effusion, or intraperitoneal rupture, when present, may suggest an amebic abscess [50]. Amebic abscesses are typically solitary round-to-oval lesions that occur most often in the posterior segment [50, 51]. A miliary pattern mimicking a fungal or pyogenic infection may be seen rarely [52]. On ultrasound, findings suggestive of an amebic abscess are hypoechoic round or oval lesions located close to the liver capsule that show low-level internal echoes and posterior acoustic enhancement [51]. On CT, these lesions have slightly higher attenuation than water, may have smooth or nodular borders, and have a thick (3–15 mm) wall that typically enhances (Fig. 7). A ring of edema, forming a double-target sign, may be present [50]. On MRI, the central portion of the lesion appears cystic, and the "rind" exhibits variable intensities on T1- and T2-weighted imaging [53].

Hydatid Cyst

Hydatid cysts are caused by infestation with *Echinococcus granulosus.* This entity is mostly seen in developing and underdeveloped regions of the world and in patients from these endemic areas who had close contact with sheep [54]. The human is an intermediate host that becomes infested by accidental handling of material contaminated with larva. The infestation classically occurs during childhood, but the disease usually remains undiagnosed until the 3rd and 4th decades. The majority of patients are asymptomatic. The symptoms include pain; biliary obstruction; superinfection; and, rarely, cyst rupture, which can lead to anaphylactic reaction. The diagnosis is confirmed with serologic tests. Anthelminthic drugs have been the mainstay of treatment but with disappointing results. Open surgery, when not contraindicated, is the treatment of choice for patients with hepatic hydatid cysts, whereas treatment using laparoscopic surgery, percutaneous cyst drainage, or injection of scolicidal agents is reserved for selected patients [54].

On imaging, the lesions present as unilocular or multilocular cysts. Four different radiographic appearances have been described: a simple cyst with no internal architecture, a cyst with daughter cysts and a matrix, a calcified cyst, and a complicated cyst [55, 56]. The classic type is a cyst containing multiple peripheral daughter cysts (Fig. 8). The content of the daughter cysts is different from that of the mother cyst; hence, the daughter cysts are usually hypodense on CT and have a slightly different signal intensity than the mother cyst.

Fungal Microabscesses

Organ involvement with fungal microorganisms may manifest in the liver as numerous disseminated small fluid collections. Invasive fungal infections are typically seen in the immunocompromised population, including diabetic patients, organ transplant recipients, postsplenectomy patients [57], premature neonates [58], and patients in ICUs [59]. The causative organism is most commonly *Candida* species, although other infective fungi include *Cryptococcus* [60] and *Aspergillus* [61] species, among others. Treatment is with IV antifungal agents.

On imaging, the lesions are usually small (< 2 cm) and disseminated throughout the liver and the spleen (Fig. 9A). The ultrasound appearance of fungal microabscesses is classically described as a "bull's eye": a round hyperechoic lesion with an outer hypoechoic ring [62]. Adding a central hypoechoic dot to the bull's eye has been described as a "wheel within a wheel"; uniform hyper- or hypoechoic small rounded masses have been described as well [63]. On CT, triphasic liver imaging is most sensitive for detecting hepatic microabscesses, with most lesions being detectable on the arterial phase. The appearance of fungal microabscesses is variable on the portal venous phase: Fungal microabscesses may be uniformly hypoatten-

uating, may have a ring-enhancing appearance akin to the appearance on ultrasound, or may be uniformly hyperenhancing [64]. On MRI, the lesions are most conspicuous on the T2-weighted sequence. The timing and host factors may alter the signal intensity characteristics and enhancement patterns of the lesions on MRI. For example, early disease in a neutropenic patient may be occult on MRI, whereas subacute treated lesions may have a ring of hemosiderin on MRI [65]. Mimics of hepatosplenic fungal infection include granulomatous diseases (e.g., sarcoidosis) (Fig. 9B) and rarely aseptic abscesses in the setting of autoimmune diseases such as Behçet syndrome and Crohn disease [66, 67].

Neoplastic Lesions

Biliary Cystadenoma and Cystadenocarcinoma

Biliary cystadenoma (BCA) and biliary cystadenocarcinoma (BCAC) are rare slow-growing neoplasms arising from the bile ducts. Both lesions are more common in women, although the female predominance is much more pronounced in BCA (female-male ratio, 9:1) [68]. The mean age at presentation is 45 years for BCA and almost 55 years for BCAC. The proposed pathogenesis is that these lesions arise from ectopic rests of embryonic bile ducts or aberrant ducts [69]. BCAC is usually a result of malignant transformation of BCA but can also arise de novo [69, 70]. The risk of malignant transformation of BCA to BCAC can be as high as 20% [71]. The majority of BCAs and BCACs are intrahepatic, although a few extrahepatic cases occurring in extrahepatic ducts or in the gallbladder have been reported. Classically, BCAs have ovarian-type stroma [72]. Patients are usually asymptomatic or present with nonspecific symptoms [69]. Treatment of both BCAs and BCACs is surgical excision.

The imaging findings of cystadenoma and cystadenocarcinoma overlap (Fig. 10). These lesions are usually multilocular with enhancing walls, fine septations, and variable calcification and can be as large as 30 cm [73–75]. Ultrasound can better show internal septations than other imaging modalities. Enhancing mural nodules are more common in BCAC than BCA. Associated biliary ductal dilatation and localization in the left lobe are also common features of BCA and BCAC [76]. The morphology can mimic that of pyogenic abscess, amebic abscess, or cystic metastasis, and knowledge of clinical information is paramount for providing the correct diagnosis. After exclusion of mimics, the diagnosis of BCA or BCAC should be considered, and the patient should be referred for surgical consultation.

Cystic Hepatocellular Carcinoma

Classically, hepatocellular carcinoma (HCC) appears on dynamic cross-sectional imaging as a hypervascular mass with rapid washout on the portal venous phase and an enhancing peripheral capsule [77, 78]. Very rarely, HCC may manifest as a predominantly cystic mass with enhancing septa [79, 80]. Cystic HCC has been reported as having an unusual clinical presentation of acute fever and leukocytosis, with imaging findings on CT suggesting an abscess: an irregular multilocular hypoattenuating lesion with a peripheral rim of enhancement [81] (Fig. 11). Pathologic evaluation of this entity has shown that the hypoattenuating central portion is necrosis and the peripheral enhancing septa contain malignant cells [82]. Liquefactive necrosis after locoregional treatment, such as chemoembolization, cryoablation, or radiofrequency ablation, is a more common cause for the cystic morphology that results in the cystic appearance of HCC.

Cystic Liver Metastases

Approximately 10% of focal liver lesions in patients with a known primary carcinoma are found to be metastatic disease [83]. Typical metastatic lesions may be hypoattenuating to background liver on CT but will usually have an irregular peripheral rim of enhancement [84] (Fig. 12). Some tumors have been reported to produce truly cystic lesions in the liver, which typically, but not necessarily, appear with a rim of enhancement. Malignancies that have been described to appear cystic include neuroendocrine tumors, gastrointestinal stromal tumor (GIST), lung adenocarcinoma, colorectal carcinoma, transitional cell carcinoma, adenoid cystic carcinoma, ovarian carcinoma, choriocarcinoma, sarcoma, and lesions treated with chemotherapy [85]. The cystic appearance of some metastatic lesions could be because of the high mucinous content of the lesion, such as in mucinous colorectal or ovarian carcinomas, or the rapid growth of the tumor with hemorrhage, necrosis, or cystic degeneration, such as in neuroendocrine tumors, sarcoma, melanoma, GIST, or certain lung and breast carcinomas. Additionally, metastatic lesions can undergo necrosis or cystic degeneration after chemotherapy.

Miscellaneous Lesions

Peribiliary Cyst

Peribiliary cysts are believed to be caused by obstruction of the neck of the periductal glands (extramural-type glands) due to inflammation or deranged portal circulation [86]. This condition has a high association with cirrhosis, portal hypertension, and autosomal-dominant polycystic disease [86]. Although found more commonly on microscopic examination (seen in up to 50% of patients with liver disease), peribiliary cysts are encountered less frequently on imaging. On CT examination, the reported prevalence of peribiliary cysts in patients with cirrhosis is 9% [87]. The cysts have an epithelial lining and contain mucin or serum. The lesions usually increase in size and number as cirrhosis and portal hypertension progress. Patients are usually asymptomatic, although obstruction of the bile ducts may rarely occur [88].

Peribiliary cysts are multiple and can be discrete, clustered, or confluent. They are typically located along the portal tracts in the hilum and adjacent to the large intrahepatic ducts (Fig. 13) and are rarely found in the periphery. The confluent type can mimic biliary ductal dilatation, but distribution on both sides of the portal vein is helpful to differentiate the two entities. A "string-of-beads" pattern, which can mimic primary sclerosing cholangitis, has been described on CT. Ultrasound can depict the thin septa between the cysts, and this finding can be used to differentiate them from abnormal bile ducts that are seen in the setting of primary sclerosing cholangitis [89].

Intrahepatic Pseudocyst

An intrahepatic pseudocyst is an extremely rare condition that may occur in the setting of pancreatitis, usually as a complication of acute alcoholic pancreatitis [90]. The demography parallels that of acute pancreatitis: Intrahepatic pseudocysts most commonly affect young and middle-age men. The suggested pathophysiology is spread of pancreatic enzymes and lesser sac fluid along the hepatogastric and hepatoduodenal ligaments or along the portal triad into the liver parenchyma that results in intrahepatic tissue damage and necrosis [90]. This fluid collection, similar to other fluid collections in the setting of pancreatitis, can spontaneously resolve or can progress to become a pseudocyst with a fibrous capsule. An intrahepatic pseudocyst may require percutaneous or endoscopic drainage or surgical resection if it

is large or symptomatic. On imaging, the lesions manifest as a simple fluid collection with an enhancing thin peripheral capsule; these lesions have a high propensity for the right lobe [91]. The cysts will have a complex appearance if superinfected or if complicated by hemorrhage. Knowing the clinical history and the ancillary findings of pancreatitis is key to establishing the diagnosis.

Trauma-Related Lesions

The collection of bile, lymph, or blood products after injury to the liver parenchyma will result in the formation of a biloma, seroma, or hematoma, respectively [92]. These lesions may occur after blunt or penetrating trauma or iatrogenic injury to the liver, such as after cholecystectomy, liver surgery, liver transplantation, or percutaneous liver procedures. Inflammatory response can lead to pseudocapsule formation. Depending on the type of collection, its size, the time frame, and the patient's symptoms, the treatment options range from conservative management to percutaneous drainage, biliary diversion and stent placement in cases of biloma, or open surgery. On imaging, seromas and bilomas appear as a simple fluid collection that may or may not show a thin rim of enhancement (i.e., a pseudocapsule) (Fig. 14). Superinfection results in a complex cystic appearance. Hematomas, on the other hand, have different density and intensity based on the age of the blood products. MRI—specifically, the gradient-echo T2-weighted sequence—is the most sensitive method for detecting blood products. Cholescintigraphy, MRI with a hepatobiliary contrast agent, or cholangiography is extremely useful for showing a bile leak and differentiating biloma from other fluid collections.

Mimics

Undifferentiated Embryonal Sarcoma

Undifferentiated embryonal sarcoma (UES) is a highly malignant hepatic neoplasm that is usually seen in the pediatric age group (typical age at presentation, 6–10 years; equal sex distribution [93]), but UES can rarely be seen in late childhood and early adulthood [94]. UES is the third most common malignant liver tumor in the pediatric population, accounting for approximately 10% of pediatric liver cancers [95]. It is of mesenchymal origin with sarcomatous features. Histologic similarities with mesenchymal hamartoma exist, and some authors suggest that UES arises from mesenchymal hamartoma [72]. Patients usually present with nonspecific symptoms. Treatment includes surgical resection and multiagent chemotherapy [96]. Historically, UES was known to have a very poor prognosis, but new reports show promising prognosis with a 20-year survival reported in up to 70% of patients [97].

UES presents as a large (> 10 cm) solitary lesion commonly in the right lobe [98]. On ultrasound, it appears as a solid lesion that is usually iso- to hyperechoic to liver parenchyma and that contains small anechoic areas corresponding to areas of necrosis or cystic degeneration (Fig. 15). On unenhanced CT, however, UES appears cystic with near-water attenuation, reflecting the high water content of its myxoid stroma, and contains septations and peripheral nodules [99, 100]. Contrast-enhanced CT can show different degrees of enhancement, which helps to confirm its solid nature [99, 100]. UES appears cystic on unenhanced T1- and T2-weighted sequences (i.e., low signal intensity on T1-weighted imaging and high signal intensity on T2-weighted imaging) because of its myxoid stroma but will show heterogeneous enhancement after contrast administration, which is better seen in the late portal venous phase [99, 100].

Pseudoaneurysm

Intrahepatic pseudoaneurysm is a rare entity that is usually a delayed complication of trauma or can be caused by iatrogenic injuries from prior surgery or percutaneous procedures [92]. Pseudoaneurysms appear cystic on ultrasound and on unenhanced CT. The vascular nature of these lesions can be easily established on color and spectral Doppler imaging. Contrast-enhanced CT and MRI will show enhancement similar to the blood pool. All cases should be treated because of the high risk of perforation [101].

Focal Steatosis

Steatosis can result in near-water attenuation of the affected areas. Hence, nodular steatosis rarely can mimic a cystic lesion on unenhanced CT. These areas, however, appear solid on ultrasound and contrast-enhanced CT. MRI with the use of chemical-shift gradient-echo imaging is considered the best imaging modality to establish the diagnosis [102].

Conclusion

Familiarity with the key imaging features of cystic hepatic lesions and knowledge of the clinical associations will help the radiologist to establish a definitive diagnosis or provide a reasonable differential diagnosis. The radiologist can also play an active role in the management of patients by performing image-guided percutaneous biopsy, aspiration, or drainage of many of these lesions, as we discussed earlier. We propose a practical algorithm that can simplify the approach for the diagnosis of these lesions (Fig. 16). By applying these workup strategies, one can prevent unnecessary tests, avoid a delay in initiating the appropriate management, and improve the cost-effectiveness of diagnostic tests. A potential area for future research is establishing new anatomic biomarkers for more accurate diagnosis of cystic hepatic lesions.

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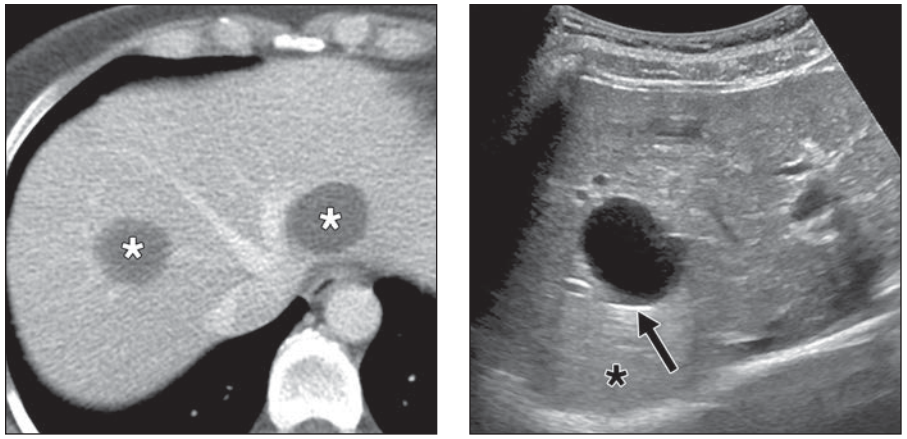

Fig. 1—Simple hepatic cyst in 49-year-old woman who presented with abdominal pain.
A, Axial contrast-enhanced CT image shows incidental cystic lesion (*asterisks*) in lateral segment.
B, Ultrasound image obtained for further evaluation confirms simple nature of cyst. Note classic sonographic features of simple hepatic cyst including well-marginated borders, posterior acoustic enhancement (*asterisk*), and enhancement of posterior wall (*arrow*).

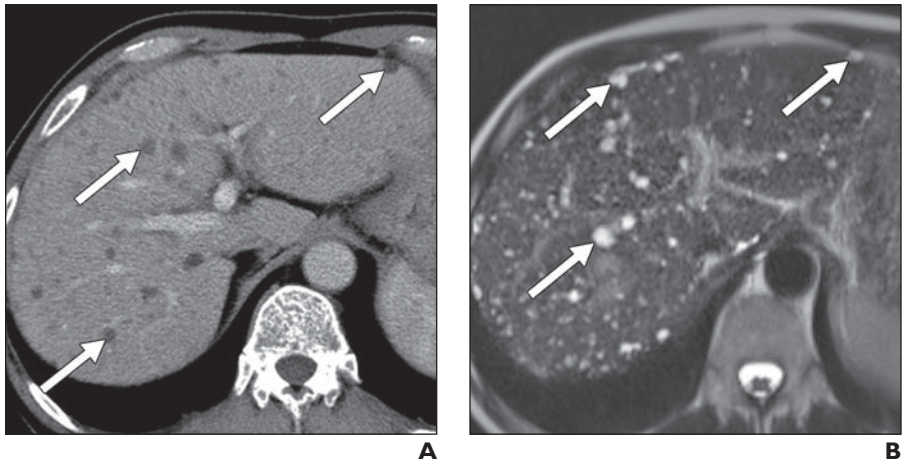

Fig. 2—Biliary hamartomas in 73-year-old man with history of chronic hepatitis C.
A and **B,** Axial contrast-enhanced CT (**A**) and axial T2-weighted MR (**B**) images show innumerable small irregular cystic lesions (*arrows*) that have been stable since prior examinations.

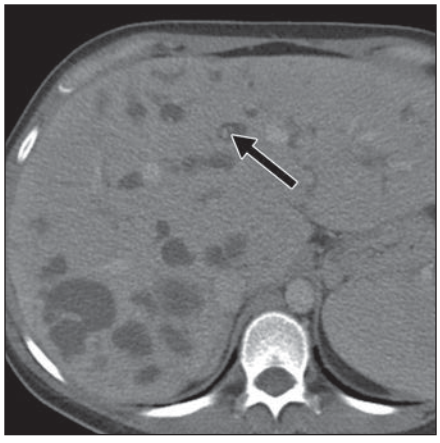

Fig. 3—Caroli disease in 37-year-old woman who presented with fever. Contrast-enhanced CT image shows multiple cystic lesions of variable sizes throughout liver. These lesions were shown to be communicating with biliary system on ERCP (not shown). Note classic "central dot" sign (*arrow*).

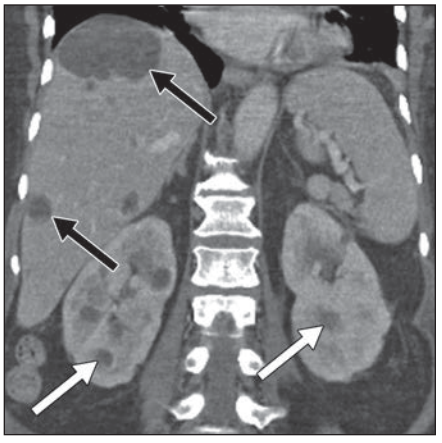

Fig. 4—Polycystic liver disease in 56-year-old woman. Coronal contrast-enhanced CT image shows multiple cysts of varying sizes in liver (*black arrows*) and kidneys (*white arrows*).

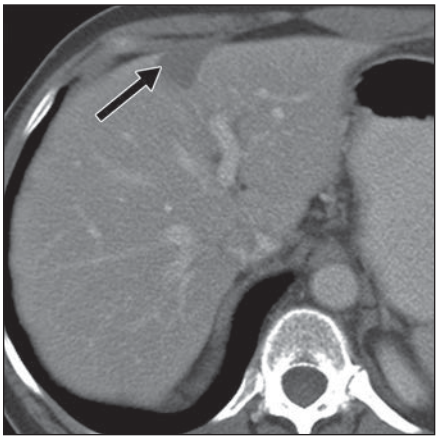

Fig. 5—Ciliated foregut cyst in 58-year-old man who presented with abdominal pain and nausea. Axial contrast-enhanced CT image shows incidental wedge-shaped peripheral hypodense lesion in segment IVA (*arrow*). Although lesion showed higher attenuation (50 HU) than water on CT, follow-up ultrasound (not shown) confirmed its cystic nature. Higher density on CT is presumably caused by proteinaceous content of cyst.

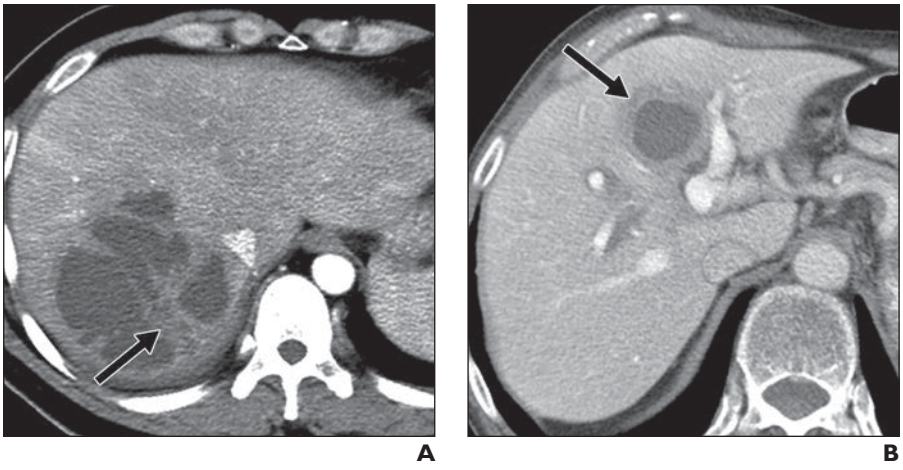

Fig. 6—Pyogenic abscess.
A, 33-year-old man with history of *Escherichia coli* and *Klebsiella* pneumonia who presented with abdominal pain and fever. Axial contrast-enhanced CT image shows clustered, large, complex cystic lesions with enhancing walls (*arrow*).
B, 66-year-old man with history of diverticulitis. Axial contrast-enhanced CT image shows complex cystic lesion with thick enhancing rim and perilesional edema (*arrow*).

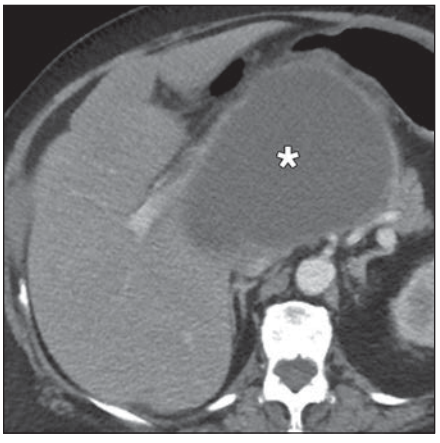

Fig. 7—Amebic abscess in 64-year-old woman who presented with fever and abdominal pain. Axial contrast-enhanced CT image shows large unilocular cystic lesion (*asterisk*) in caudate lobe. Patient had history of recent trip to central Africa.

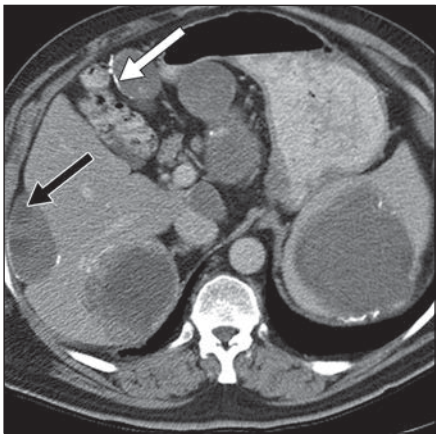

Fig. 8—Hydatid cyst in 65-year-old man who presented with abdominal pain. Axial contrast-enhanced CT image shows multiple cystic lesions in liver and spleen. Note wall calcification (*white arrow*) and daughter cysts (*black arrow*). Cysts medial to lesser gastric curvature are exophytic hepatic cysts protruding into lesser sac.

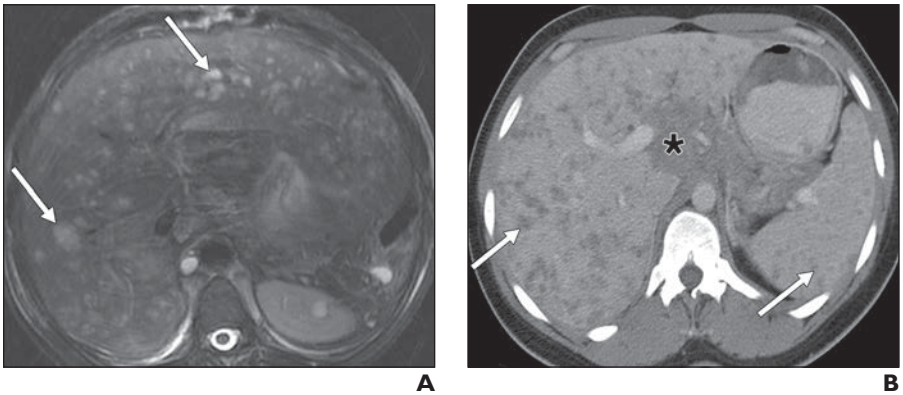

Fig. 9—Fungal microabscesses and mimic of hepatosplenic fungal infection.
A, Fungal microabscesses. Axial T2-weighted MR image of 35-year-old man with history of AIDS who presented with fever shows innumerable cystic lesions (*arrows*) throughout liver and spleen. Patient was found to have candidiasis.
B, Hepatic sarcoidosis. Axial contrast-enhanced CT image of 42-year-old man with history of pulmonary sarcoidosis shows innumerable hypodense hepatic and splenic lesions (*arrows*) and mesenteric lymphadenopathy (*asterisk*). Although these lesions are not truly cystic, they can mimic cysts on CT.

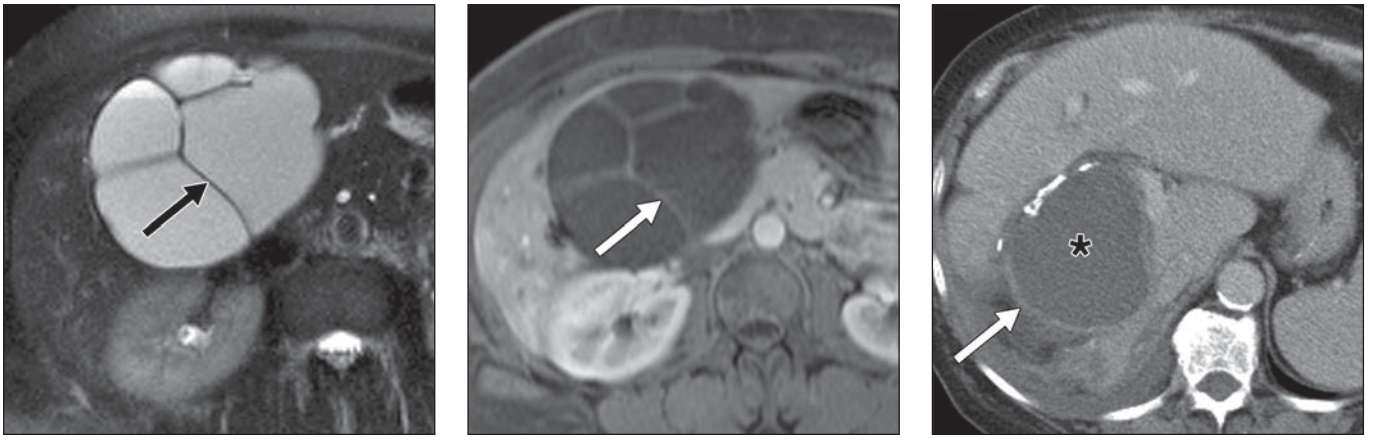

Fig. 10—Biliary cystadenoma (BCA) and biliary cystadenocarcinoma (BCAC).
A and **B,** BCA in 43-year-old woman who presented with abdominal pain. Axial T2-weighted (**A**) and contrast-enhanced T1-weighted (**B**) MR images show large multilocular cystic lesion with fine enhancing septations (*arrows*). Patient underwent surgical resection of lesion.
C, BCAC in 73-year-old woman who had prior resection of BCAC. Contrast-enhanced CT image shows large cystic lesion (*asterisk*) adjacent to surgical sutures with thick enhancing rim (*arrow*) compatible with recurrent BCAC.

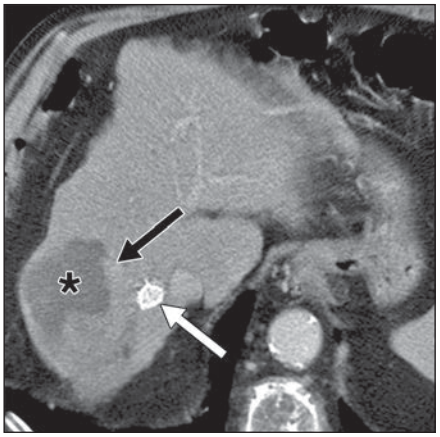

Fig. 11—Cystic hepatocellular carcinoma in 56-year-old man with history of hepatitis C cirrhosis. Axial arterial phase contrast-enhanced CT image shows complex cystic lesion (*asterisk*) in posterior segment with peripheral hypervascular component (*black arrow*). Note morphologic features of cirrhosis and transjugular intrahepatic portosystemic shunt stent (*white arrow*).

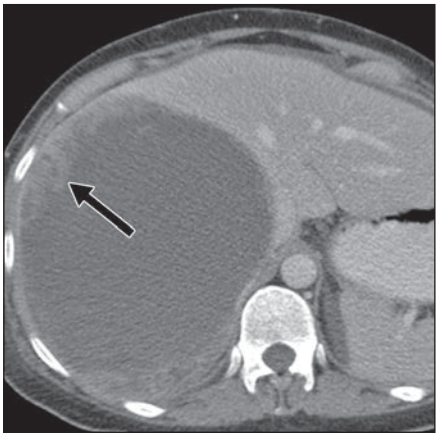

Fig. 12—Cystic metastasis in 40-year-old woman with history of retroperitoneal sarcoma. Axial contrast-enhanced CT image shows new complex cystic mass with peripheral solid components (*arrow*) compatible with metastasis.

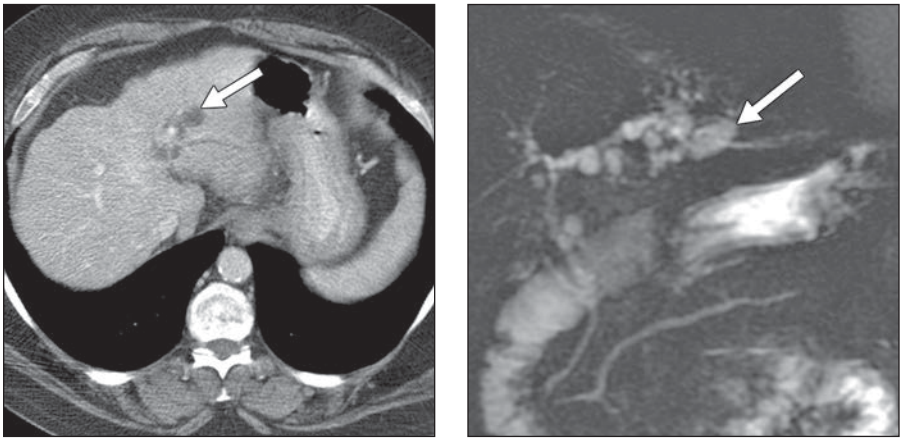

Fig. 13—Peribiliary cysts in 61-year-old man with history of cryptogenic cirrhosis.
A and **B,** Axial contrast-enhanced CT (**A**) and coronal MRCP (**B**) images show multiple cystic lesions (*arrows*) with periportal distribution. Note cirrhotic morphology of liver.

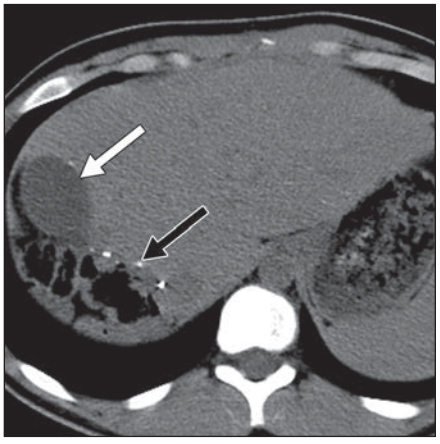

Fig. 14—Seroma in 20-year-old female living liver donor. Axial unenhanced CT image shows simple fluid collection (*white arrow*) close to lobar resection site (*black arrow*). Follow-up imaging revealed complete resolution of collection.

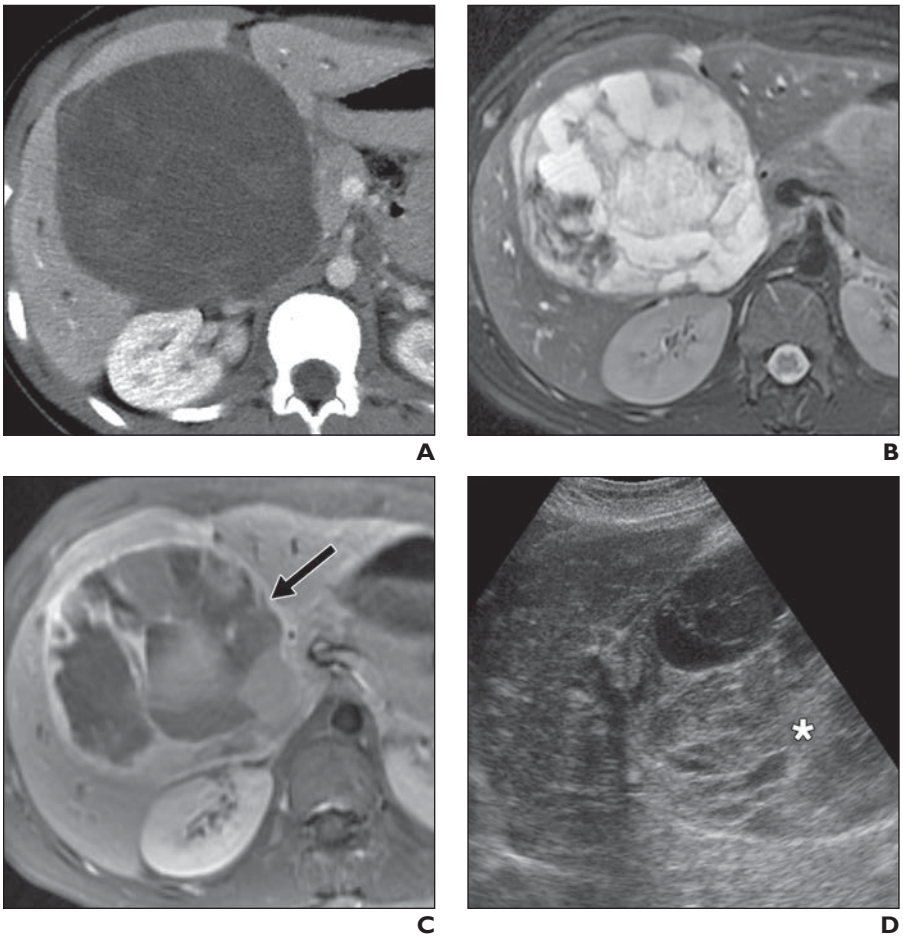

Fig. 15—Undifferentiated embryonal sarcoma in 14-year-old girl with palpable abdominal mass on physical examination.
A–C, Axial contrast-enhanced CT (**A**), axial T2-weighted MR (**B**), and axial contrast-enhanced T1-weighted MR (**C**) images show large multilocular cystic hepatic mass with enhancing rim and internal septa (*arrow*, **C**).
D, Ultrasound image. Although mass appears predominantly cystic on CT and MRI (**A–C**) because of its myxoid stroma, it has predominantly solid features (*asterisk*) on ultrasound.

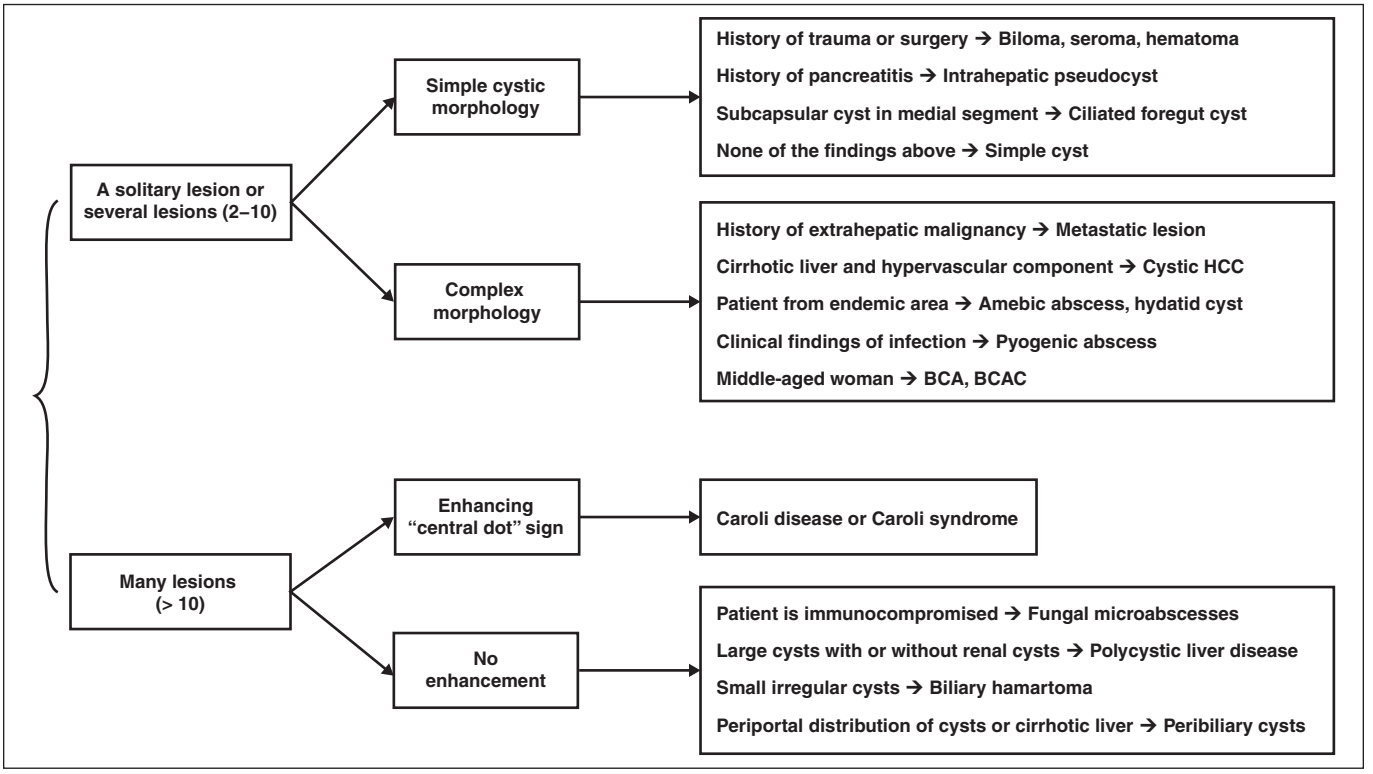

Fig. 16—Simplified algorithm for identifying and differentiating cystic hepatic lesions. HCC = hepatocellular carcinoma, BCA = biliary cystadenoma, BCAC = biliary cystadenocarcinoma.

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