

## Review Article

# Evaluation of Hepatic Cystic Lesion in Children

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### Abstract:

Hepatic cysts are very rare in children and most of them are simple and solitary, asymptomatic, benign, and do not require intervention. It is usually found as a mere coincidence on abdominal imaging techniques, such as ultrasonography (USG), computed tomography (CT), and magnetic resonance imaging (MRI). The incidental diagnosis of cystic lesions of the liver increased day by day due to increased use and sensitivity of abdominal imaging. Predominantly simple cysts are most common and diagnosed by characteristic Ultrasonogram findings. Serodiagnostic tests and contrast-enhanced ultrasound (CEUS) are useful when USG, CT, and MRI show ambiguous findings in differentiating other cystic lesions (complicated cysts, echinococcosis, and cyst adenoma/cyst adenocarcinoma) and reduce the need for invasive procedures. Although most cysts are benign, some are malignant or premalignant, so early detection of cystic lesions is important to manage them properly. Treatment is needed in symptomatic or complex cysts or those having malignant or premalignant features. This article outlines the major types of hepatic cysts, highlights clinical features that may aid in their appropriate diagnosis, and discusses therapeutic options.

**Key words:** Hepatic cyst, Cystic liver disease in children, Fibrocystic liver disease in children, Polycystic liver disease, Hydatid cysts.

### Introduction:

A cyst is a structure composed of a distinct wall with a liquid or solid content<sup>1</sup>. Cystic liver disease encompasses a heterogeneous group of fluid-filled lesions within the liver parenchyma<sup>2</sup>. Hepatic cysts include parasitic and nonparasitic cysts.

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Hepatic cysts may be solitary or multiple, such as in congenital polycystic disease of the kidney and liver, congenital dilatation of intrahepatic bile ducts (Caroli disease)<sup>3</sup>. According to the characteristics of lesions, hepatic cysts can be subdivided into simple and complex cysts. It is important to differentiate between them because it can implicate the need for further diagnostics and treatment<sup>2</sup>. These lesions predominantly remain asymptomatic, in most cases will follow a benign course and usually need no treatment<sup>4,5</sup>. However, it is essential to differentiate benign cysts from potentially harmful cysts, such as echinococcosis, cyst adenoma, and cyst adenocarcinoma, which require specific treatment<sup>6,7</sup>. The primary modality of differentiation is by abdominal imaging and in some cases, advanced imaging has made it possible to accurately diagnose hepatic cystic lesions bypass the need for invasive testing, such as biopsy or resection<sup>2</sup>. Incidental findings of asymptomatic hepatic cystic lesions are rising, due to the vast availability and increased use of abdominal imaging modalities over the last years<sup>8</sup>. This increased detection poses two main challenges: first, distinguishing a lesion as either benign or malignant, and second, choosing an imaging modality that will be diagnostically accurate, cost-effective, and safe<sup>2</sup>. This review will help in the evaluation of different types of hepatic cysts and also describe how various imaging modalities are used to evaluate and diagnose them along with management strategies for hepatic cysts.

**Differential diagnosis of cystic lesions in liver/ solitary liver cysts in children<sup>4,9</sup>.**

1. Monocystic disease	2. Polycystic disease
Simple cyst	Autosomal dominant
Complex cysts	polycystic kidney
Echinococcosis/Hydatid cysts	disease
Cystic echinococcosis	Autosomal dominant
Alveolar-echinococcosis	polycystic liver disease
Cystadenoma	
Cystadenocarcinoma	
a. Congenital	b. Acquired
Simple	Parasitic
Mesenchymal hamartoma	(hydatid) /Echinococcosis
Intrahepatic choledochal cyst	Posttraumatic
Caroli disease & Caroli syndrome	Neoplastic: cystadenoma, sarcoma, teratoma
Ciliated hepatic foregut cyst	Biliary cysts: biliary atresia post Kasai, traumatic
Epidermoid cyst	Abscess (eg, pyogenic, amoebic)
Lymphangioma	

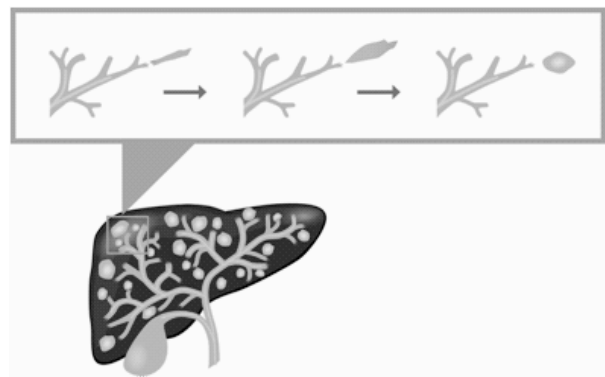
**Presenting features:**

Most of the hepatic cysts are asymptomatic. The presentation of symptoms is usually dependent on the size of hepatic cysts because, by an increase in size, they may become symptomatic<sup>10</sup>. Hepatic cysts with symptoms account for approximately 15-16%<sup>11</sup>. Symptoms are nonspecific, which include abdominal pain, early satiety, nausea, or vomiting loss of appetite, feeling of pressure, fullness & weight loss. Hepatomegaly or a palpable mass on physical examination may have found dependent on the size of the cysts<sup>2,4,12</sup>.

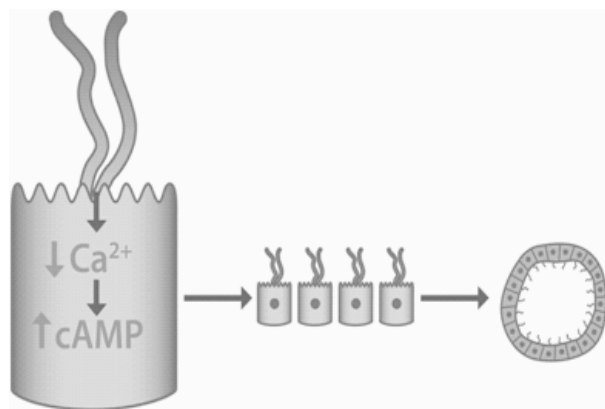
**Pathogenesis:**

Liver cysts are rare in children, they can be congenital or acquired. Alterations in the development of the intrahepatic bile ducts cause the formation of congenital cysts<sup>13</sup>. Acquired or complex cysts are the result of inflammation, post-traumatic, parasite infections, malignancy, or other miscellaneous etiologies<sup>2,13</sup>. Simple cysts include congenital cysts, biliary hamartomas, Caroli disease, and polycystic liver disease (PCLD). Most of them are congenital and form from biliary ducts that do not connect to the biliary system<sup>4,10,14</sup>. Biliary hamartomas are derived from

embryonic bile ducts<sup>14</sup>. Caroli disease is an autosomal recessive disorder which is a rare form of simple cyst characterized by cavernous ectasia of the bile ducts<sup>14,15</sup>. The formation of cyst in PCLD is not completely understood yet. During the process of the biliary tree formation, the ducts undergo cycles of apoptosis and regeneration. That time small portions of the ductal system may become detached from the main biliary system & gradually dilate into cystic lesions formation (Fig. 1)<sup>16</sup>. In a second proposed mechanism, the cilia on cholangiocytes transmit signals for decreased intracellular calcium and increased levels of cAMP in the response of changes in the bile flow or bile composition. This triggers a signaling cascade, stimulating DNA transcription and hyperproliferation of cholangiocytes. These proliferative cells aggregate to form cysts (Fig. 2)<sup>16</sup>.



**Fig. 1:** Retained abnormal bile ductules, which become detached from the biliary tree and progressively dilate, forming cysts



**Fig. 2:** Defect in biliary cilia, leading to hyperproliferation of cholangiocytes and generation of cysts

### Congenital hepatic cyst:

It is a rare and non-symptomatic condition in infants and children which maintain a silent natural history and diagnosis is usually made incidentally on radiologic imaging<sup>17,18</sup>. It is only found in the postnatal life about 2.5% & most common among girls<sup>18,19</sup>.

These cysts are formed due to congenital or secondary obstruction of the biliary glands followed by proliferation and continue to proliferate until adolescence. It usually does not contain bile<sup>17</sup>. These are superficial cysts, located just under the liver capsule and lined by a single layer of columnar or cuboidal epithelium & contain clear cystic fluid but rarely bile<sup>17</sup>.

The most common presentation is an incidental finding of an asymptomatic lesion on antenatal imaging but in infants, it may presents with abdominal distension, feeding difficulties, respiratory distress, duodenal obstruction and in some cases cholestasis due to the compression of the hepatic parenchyma and the biliary system by a large cyst<sup>20</sup>. In the older group, symptoms may include abdominal pain, nausea, vomiting, and rarely obstructive jaundice or portal hypertension<sup>21</sup>.

The diagnosis is primarily made via Ultrasonography shows an anechoic unilocular fluid-filled space with posterior acoustic enhancement. Septations rarely found & it represents the bridging of bile ducts and vessels. Magnetic resonance imaging (MRI) typically reveals a well-demarcated water-attenuated lesion without enhancement but it is not routinely needed<sup>22</sup>. Chance of malignant transformation is extremely rare, and the only recognized risk factor for this transformation is a cyst size greater than 12 cm<sup>19,21</sup>.

Management is conservative with periodic ultrasound monitoring to ensure their stability, especially for large cysts ( $\geq 4$  cm in diameter)<sup>20,22</sup>. Most simple hepatic cysts are benign and have a spontaneous resolution. Surgical intervention with aspiration, sclerotherapy, or excision is indicated only for complicated cases, like hydrops, progressive enlargement, hemorrhage, torsion, or if doubtful imaging characteristics<sup>19,20</sup>.

### Simple hepatic cyst:

Simple cysts are thin, smooth walls lesions lined by cuboidal epithelium; same as bile duct that's why secrete bile-like fluid<sup>2</sup>. Simple hepatic cysts are usually  $< 1$  cm and can grow up to 30cm<sup>10</sup>. The prevalence ranges from 2.5% to 18% and increases with age<sup>4,23</sup>. It is usually asymptomatic but in a small fraction of patients present with abdominal pain, early satiety, nausea, and vomiting due to mass effect<sup>4,6</sup>. Complications such as hemorrhage, rupture, and biliary obstruction are uncommon but may occur in larger cysts<sup>4</sup>.

Laboratory investigation may reveal, raised serum  $\gamma$ -glutamyl transferase ( $\gamma$ -GT) and cancer antigen 19-9 (CA 19-9) may be elevated. CA 19-9 is expressed in the simple cyst inner epithelial & is not elevated due to malignancy rather than cyst infection<sup>24</sup>.

Anechoic, homogenous, fluid-filled lesions with smooth margins are the characteristics findings in ultrasound. CT shows a well-demarcated, water-attenuated, smooth lesion without an internal structure and contrast enhancement. Similarly, MRI shows a well-defined, homogeneous lesion with low signal intensity on T1 weighting, and high intensity on T2, without contrast enhancement<sup>10</sup>.

The microbubble contrast-enhanced ultrasound (CEUS) enables us to visualize vascular flow within septa or solid components of cysts, which is absent in simple cysts with intra-cystic hemorrhage, that's why it is helpful in case of hemorrhagic cysts<sup>28</sup>.

Most of the case management is "wait and- see" policy, and no further treatment is required. If there are symptoms and a complication arises, aspiration-sclerotherapy is the preferred treatment<sup>4,26</sup>. According to availability & local expertise, laparoscopic deroofing rather than aspiration and sclerotherapy may be an option in a case of symptomatic simple hepatic cysts<sup>10</sup>.

### Complex cyst:

Complex cyst is usually large ( $> 4$ -5 cm), multiple ( $> 20$ ), and defined by the presence of complex features within a lesion, like septations, mural thickening or nodularity, calcifications, fenestrations, loculations, hemorrhagic or proteinaceous contents, debris-containing fluid, radiographic enhancement. Heterogeneity, daughter cysts, or symptoms on presentation are present in a complex cyst<sup>2,10</sup>. If on USG these findings are present then CT or MRI should prompt for further evaluation<sup>10</sup>.



**Fig.3:** Congenital hepatic cyst by USG

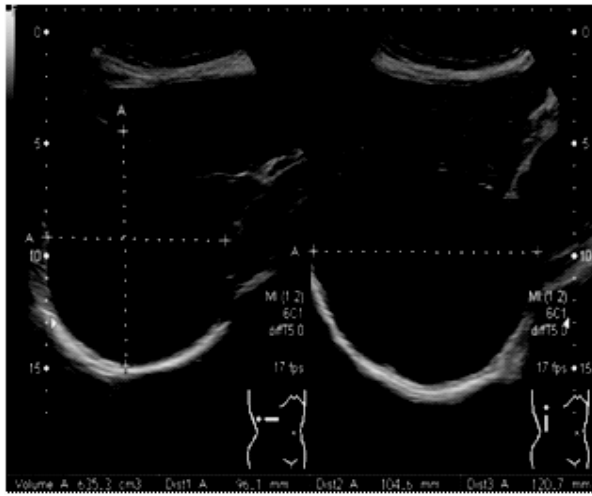


Fig.4: Simple hepatic cyst on USG



Fig.5: Complex hepatic cyst on USG



Fig 6: Hydatid cyst in liver

**Table-I:** Distinguishing characteristics of simple versus complex hepatic cysts<sup>2</sup>.

Simple cyst	Complex cyst
Thin, smooth wall	Septated
May contain up to two septae	Mural irregularity or nodularity
	Debris
	Calcification
	Fluid levels

**Parasitic (Hydatid disease):**

Hydatid disease is a parasitic infection caused by larval stages of cestodes (tapeworms) belonging to the Echinococcus, characterized by a cystic lesion in the liver, lungs, and rarely in other parts of the body<sup>27</sup>. Hydatidosis can also affect the brain, heart, kidney, ureter, spleen, uterus, fallopian tube, pancreas, diaphragm, bone, and muscles<sup>28</sup>. Only two species cause solitary cystic lesions in humans: (i) *E. granulosus*, responsible for cystic echinococcosis, and (ii) *E. multilocularis*, responsible for alveolar echinococcosis<sup>7</sup>.

It is prevalent where livestock is raised in association with dogs. After ingestion of contaminated food with Echinococcus eggs, which are excreted by infected carnivores (dogs and other canids) people become infected by acting as intermediate hosts of *E. granulosus*<sup>7,28</sup>. Hydatid cysts are most common in the Mediterranean, South America, Australia, East Africa, and Central Asia<sup>10,29</sup>.

**Cystic echinococcosis/ Hydatid cysts in liver:**

Echinococcus granulosus involvement in children has a different pattern because it is more common in the lungs while in the case of adults it is the liver<sup>27,28</sup>. It is more predominant in boys over girls most probably due to more exposure with boys and behavioral differences between sexes<sup>27</sup>. The cysts become visible in the liver at 3 to 4 weeks and grow into a mature cyst that has a germinal layer surrounding a fluid-filled central hydatid cavity<sup>4</sup>. The Hydatid cyst in the liver is usually a slow grower than in lung due to less elasticity & can remain asymptomatic for a long time in most of the cases<sup>30</sup>.

Most small cysts, < 5 cm, are asymptomatic & larger cysts can present with abdominal pain due to inflammatory reaction. Other symptoms may be loss of appetite, nausea, fever, hepatomegaly & abdominal mass<sup>10,30</sup>.

An acute presentation with pain may be due to rupture or secondary infection of the cyst. Ascites, peritonitis, and shock due to a severe allergic reaction of antigenic

content of cyst followed by incidental rupture or iatrogenic puncture may occur<sup>10</sup>. Rarely, the cysts can extrude into the biliary tree, leading to jaundice and cholangitis (rupture into the biliary tree), biliary obstruction, and an intraperitoneal rupture followed by anaphylaxis are common complications requiring immediate hospitalization<sup>7,10</sup>.

Diagnosis is confirmed usually by following criteria: endemic region history, clinical findings (e.g., abdominal pain, fever, chest pain, and dyspnea), serological test for anti echinococcal antibody and characteristic sonographic features like anechoic, round or oval-shaped, or atypical (i.e., snowflake-like inclusions) echo pattern with multiple septa confined by a laminated border (Table II)<sup>4</sup>. Usually, a CT scan is not needed for diagnosis but it is required for surgical management.

Common therapeutic options are medical treatment with antihelminthic (Albendazole, praziquantel), PAIR (puncture, aspiration, injection, and respiration) and surgery (radical pericystectomy or conservative deroofting)<sup>31</sup>. PAIR with adjunct antihelminthic chemotherapy is as effective as open surgical drainage with fewer complications and lower cost. PAIR is recommended if hydatid cysts > 5 cm & patients not fit for or decline surgery, or who relapse after surgery<sup>10</sup>. It is contraindicated in patients with biliary fistulas (risk of biliary sclerosis), inaccessible cysts, or complicated, multivesicular cysts<sup>10</sup>. Surgical treatment has been reserved for complicated cysts that have fistulas, multiple daughter vesicles, rupture, hemorrhage, or secondary infection<sup>10</sup>.

#### **Alveolar Echinococcosis (AE):**

Approximately 99% of cases AE similar to simple cysts which are usually confined to a solitary alveolar lesion in the lung. Symptoms are related to mass effects such as chest pain, dyspnea, or are nonspecific, such as weight loss or fatigue<sup>4,32</sup>. Diagnosis is usually made by conventional X-ray and computed tomography. The cyst is identified on X-ray as a round or oval homogenous opacity that can be differentiated from pulmonary parenchyma<sup>27</sup>. The choice of treatment for AE includes radical surgery followed by 2 years of chemotherapy<sup>27</sup>.

#### **Mucinous cystic neoplasm:**

Biliary cystadenoma (BCA) also is known as mucinous cystadenoma and biliary cystadenocarcinomas are biliary cyst tumors and usually, they are the most common complex cysts. Above the two cystic lesions along with intraductal papillary neoplasms (IDPNs) constitute a collection of cysts known as mucinous cystic neoplasms<sup>2</sup>.

Biliary cystadenomas are congenital, benign, and rare cystic lesions in children but in adults, it comprises up to 1-5 % of total hepatic cysts, and up to 10% of cysts > 4 cm<sup>4,10,33</sup>. BCAs are multilocular lesions filled with mucinous (95 %) or serous (5%) material and derived from aberrant bile duct remnant<sup>10,34</sup>. Histologically, they are composed of three layers: outer collagenous layer; a middle layer of mesenchymal smooth muscle cells and fibroblasts (stromal layer); and, an inner cuboidal/columnar epithelial layer that secretes Mucin. They are slow-growing lesions, most commonly found in the right lobe of the liver and ranging in size from 1.5 cm to 35 cm.<sup>2,10</sup> Biliary cystadenomas are thought to be the precursor of adenocarcinoma and usually, differentiation is done histologically because imaging is not very much useful to distinguish them<sup>35</sup>.

The presentation of both this cystic lesions is usually asymptomatic or may present with symptoms like a simple cysts or echinococcosis. A large tumor may present with abdominal pain, palpable abdominal mass, nausea, vomiting, and clinical signs & symptoms of biliary obstruction<sup>36</sup>.

The cystic lesion may be unilocular or multilocular and resemble abscesses, echinococcal cysts, teratomas, hematomas, necrotic tumors, and polycystic disease on imaging<sup>35</sup>. Abdominal ultrasound typically shows anechoic, irregular wall lesions with multiloculated areas and septations like "a cyst within a cyst"<sup>37</sup>.

Because of difficulty in diagnosing by imaging and chance of recurrence and transformation into malignancy, the gold standard for treating these lesions is complete surgical resection with confirmed clear margins<sup>36</sup>.

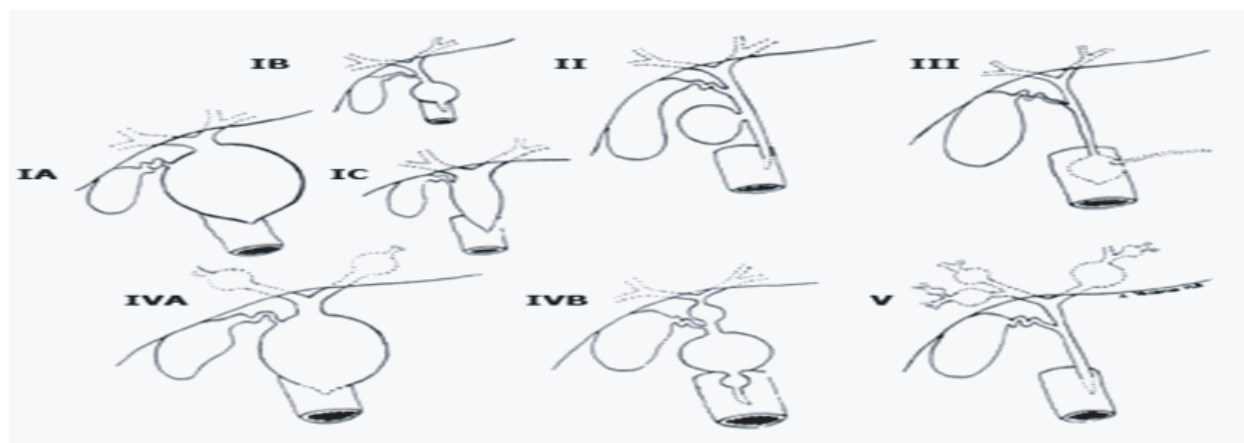
#### **Choledochal cyst (intrahepatic), Caroli disease & Caroli syndrome:**

Choledochal cysts (CC) are rare congenital disorders & it may be intra-hepatic and/or extrahepatic biliary dilatation<sup>38</sup>. It is more commonly found in children than adults and predominantly among girls (4:1)<sup>39</sup>. According to Todani and colleagues there are five types of choledochal cyst. Among them only type IVa and type V has intrahepatic cystic dilatation and type V (Caroli disease) has intrahepatic cystic dilatation without evidence of extra-hepatic dilatation<sup>40</sup>.

Clinical presentation is variable among infants and children. Jaundice, clay-colored stools are the usual presentation in infants whereas the pediatric group is more likely to present classic triad of abdominal pain, jaundice, and right upper quadrant mass. Jaundice, cholangitis, pancreatitis, portal hypertension, liver function abnormalities, and coagulopathy are the complication as well as the presentation of choledochal cyst.

**Table 2:** Ultrasonography features for the diagnosis of monocystic diseases of the liver<sup>4</sup>

	Simple cyst	Cystic echinococcosis	Alveolar echinococcosis	Cystadenoma and cystadenocarcinoma
<b>Border</b>	Sharp & smooth	Laminated	Irregular	Irregular
<b>Shape</b>	Spherical or oval	Round or oval	Irregular	Round or oval
<b>Echo pattern</b>	Anechoic	Anechoic or atypical (snowflake-like inclusions or floating laminated membrane)	Hyperechogenic outer ring & hypoechogenic centre	Hypoechogenic with hyperechogenic septations
<b>Appearance</b>	No septa	Multiseptated	Multivesicular	Septated and/or solid structures (papillary projections)
<b>Wall</b>				Wall enhancement
<b>Posterior acoustic features</b>	Strong posterior wall echoes & relative accentuation of echoes		Dorsal shadowing (calcified area)	Dorsal shadowing (calcified areas)



**Fig 7:** Classification of choledochal cyst according to Todani and colleagues. (IA) Common type, (IB) Segmental dilatation, (IC) Diffuse dilatation, (II) Diverticulum, (III) Choledochoceles, (IVA) Multiple cysts (intra and extrahepatic), (IVB) Multiple cysts (extrahepatic), (V) Single or multiple dilatations of the intrahepatic ducts (Caroli disease)<sup>39</sup>

Ultrasonogram alone may be sufficient if there is no intrahepatic biliary dilatation present. For diagnosis of type and management purpose noninvasive and highly sensitive (70-100%) and specific (90-100%), MRCP is the investigation of choice<sup>38</sup>. Cyst excision and restore biliary enteric drainage, either primarily into the duodenum or via Roux-en-Y hepaticojejunostomy (RYHJ) is the definitive treatment for Choledochal cyst<sup>38</sup>.

**Caroli Syndrome:**

Caroli disease and Caroli syndrome are congenital dilatation of the intrahepatic biliary tree. If there is only ductal ectasia or segmental dilatation of the larger intrahepatic ducts present, then it is called Caroli disease. Caroli syndrome is characterized by

malformations of small bile ducts and the presence of congenital hepatic fibrosis. This form is less common than Caroli syndrome, which is also present. This process can be either diffuse or segmental and may be limited to one lobe of the liver, more commonly the left lobe<sup>41</sup>.

Both conditions can be inherited as an autosomal recessive manner and associated with autosomal recessive polycystic kidney disease (ARPKD) or very rarely with autosomal dominant polycystic kidney disease (ADPKD). Clinical presentation & complications are like a choledochal cyst<sup>41</sup>.

Surgical resection is the best curative option if unilateral disease or disease confined in a single lobe present, but if there is progressive liver decompensation, complications of portal hypertension,

recurrent cholangitis or suspicion of cholangiocarcinoma present then liver transplantation is the treatment of choice<sup>42</sup>.

**Autosomal dominant polycystic liver disease & polycystic kidney disease:**

PCLD is a distinct clinical & genetic entity characterized by multiple (usually > 20) bile duct derived cysts & usually not associated with cystic kidney disease<sup>4,41</sup>. It is very rare in children & incidence is less than 0.01%<sup>41</sup>. Cysts are microscopically similar to simple hepatic cysts, vary in diameter (rarely >10cm), contain clear, bile-like or blood-tinged fluid, the inner lining of cholangiocytes and usually remain asymptomatic even with large cysts<sup>4,41</sup>.

It may be isolated PLD, associated with ADPKD, or associated with ARPKD. Larger cysts and a greater number of cysts found in patients with isolated PLD and less morbidity than associated with ADPKD<sup>41</sup>.

There are female predilection and PCLD tends to be asymptomatic until the size and number of cysts increase to a critical level. Clinical manifestation includes abdominal pain, abdominal mass, hepatomegaly, and symptoms from mass effects, such as abdominal bloating, pain, fullness, and shortness of breath, cyst infection with abscess formation, cyst rupture and bleeding, extrinsic compression and obstruction of the biliary tree. Patients may develop

portal hypertension but the hepatic synthetic function is maintained during all stages of the disease<sup>10,41</sup>.

ADPKD occurs in 1 in 400-1000 individuals and 10% of patients and accounts for 10% of all cases of end-stage renal disease<sup>41</sup>. It is characterized by the renal and hepatic cyst and often associated with other visceral anomalies like an aortic aneurism, mitral valve prolapse, inguinal hernias, and colonic diverticula<sup>43</sup>. Renal function remains unaffected in PCLD-associated renal cysts but in ADPKD renal cysts ultimately lead to renal failure<sup>10</sup>. Renal cysts' findings are irregularly sized, distributed throughout the organ with normal intervening parenchyma. Systemic hypertension, proteinuria, hematuria, and pyelonephritis are common complications in ADPKD<sup>41</sup>.

There are no specific laboratory tests for PCLD but may cause increased Y-GT and Alkaline phosphatase levels in both PCLD and ADPKD patients, the renal function remains intact in PCLD, but impaired in ADPKD<sup>41,44</sup>. Diagnosis requires a positive family history and the presence of > 4 liver cysts in PCLD and enlarged bilateral cystic kidneys in combination with a positive family history for ADPKD<sup>45</sup>.

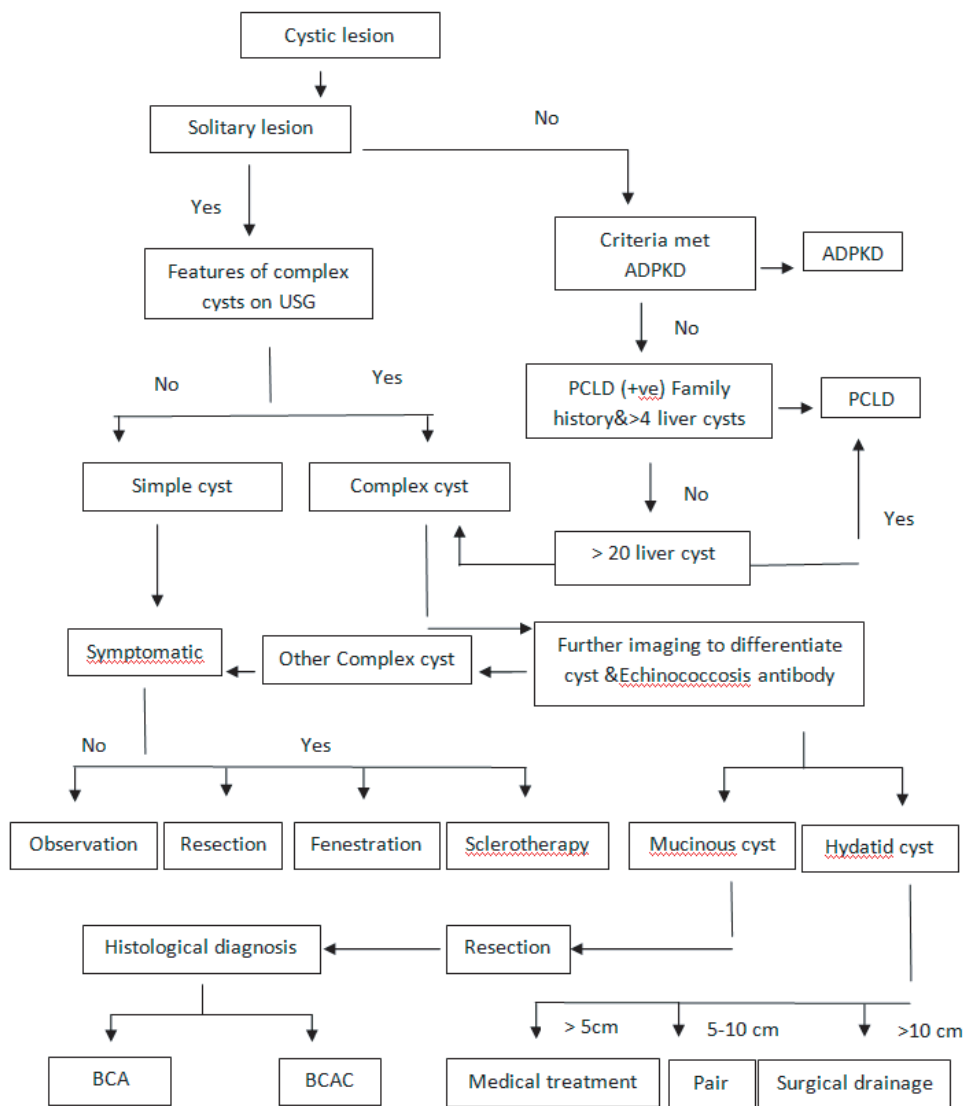
In PCLD usually, no treatment is required but in symptomatic cases, treatment should be focused on decompressing the liver or reducing the cyst volume. It can be done by transhepatic fenestration (Aspiration, deroofting of the cyst) or a combination of fenestration and resection procedure<sup>10,41</sup>. In severe form of PCLD liver transplantation and in ADPKD liver and kidney transplantation is the only curable treatment option<sup>10,41</sup>.

**Distinguishing characteristics of different hepatic cysts with various imaging modalities:**

**Table III: Imaging characteristics of hepatic cystic lesions**<sup>2,10,46</sup>

Lesion	Ultrasonogram	Computer tomography (CT)	MRI	Contrast enhanced Ultrasonogram (CEUS)
<b>Simple hepatic cyst (SHC)</b>	Anechoic, homogeneous, thin & smooth margins, fluid-filled.	Well-demarcated, water-attenuated, smooth margins without an internal structure, Non enhancing, hypodense	Well-defined, homogeneous, no enhancing T1: hypointense signal intensity T2: hyperintense signal intensity	No enhancing
<b>Complex hepatic cysts</b>	Irregular border, hyperechogenic septations, loculations, shadowing beyond calcifications	Multilocular, mural and septal enhancement, mural thickening and/or nodules, calcifications, debris containing fluid	T1: hypointense cyst contents T2: hyperintense with low signal border	Mural and septal enhancement
<b>Hydatid cysts</b>	May appear similar to SHC. Progress to develop thick, calcified walls, hyperechoic / hypoechoic contents. Daughter	Hypodense lesion with hypervascular pericyst wall, distinct endocyst wall. Calcified walls and septa easily detected. Daughter cysts seen peripherally within mother cyst	T1: Hypointense T2: Hyperintense Hypointense rim on T2. Daughter cysts seen peripherally within mother cyst. Collapse parasitic membranes seen as floating linear	

**Evaluation and management strategy of a cystic lesion in the liver in a child <sup>2,4</sup>**



**Fig 8.** Diagnosis & management of different hepatic cysts.

**Conclusion:**

Different types of the cystic lesion may be found in the liver in a child. Frequent use of various imaging modalities increased the incidental findings of hepatic cysts. Simple cysts are the most common and usually follow a benign course, need only follow up by Ultrasonogram. Symptomatic and complicated cysts like Hydatid cysts, cystic neoplasm (BCA & BCAC) need further evaluation by different imaging modalities because they may cause diagnostic enigma. Identification of any malignant or premalignant lesions, to treat any symptoms associated with hepatic cysts, and to prevent complications is the primary goal in the evaluation and treatment of hepatic cysts. Furthermore, the presence of multiple hepatic cystic lesions requires further screening to exclude PCLD or ADPKD.

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