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Cystic liver lesions: a pictorial review

Lésions kystiques hépatiques : revue iconographique

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Sous la direction de Madame le Docteur PAISANT Anita

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Liste des abréviations

HC	Hepatic cysts
HSC	Hepatic simple cysts
US	Ultrasonography
CT	Computed tomography
MRI	Magnetic resonance imaging
CEUS	Contrast enhanced ultrasonography
BH	Bile duct hamartomas
MRCP	Magnetic resonance cholangiopancreatography
CS	Caroli syndrome
CD	Caroli disease
PLD	Polycystic liver disease
ADPLD	Autosomal dominant polycystic liver disease
ADPKD	Autosomal dominant polycystic kidney disease
HLM	Hepatic lymphatic malformation
CHFC	Ciliated hepatic foregut duplication cysts
MCN-L	Mucinous cystic neoplasms of the liver
IPNB	Intraductal papillary neoplasms of the bile duct
GIST	Gastrointestinal stromal tumor
WHO	World Health Organization
CL	Cystic lesion
CE	Cyst echinococcosis
HU	Hounsfield units
CHF	Congenital hepatic fibrosis
18-F-FDG PET	18-Fluorine-fluorodeoxyglucose positron emission tomography
TAG-72	Tumor-associated glycoprotein 72
CA19-9	Carbohydrate antigen 19-9
CEA	Carcinoembryonic antigen
HEA	Hepatic alveolar echinococcosis

Cystic liver lesions: a pictorial review

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ABSTRACT

Hepatic cysts are focal lesions characterised by a cystic component, mostly found incidentally by imaging. This imaging pattern may be observed in a wide spectrum of common and uncommon non-neoplastic or neoplastic diseases.

Hepatic simple cysts are common and do not require treatment or follow-up, but untypical cysts should be carefully scrutinised in order to avoid misdiagnosis lesions and propose appropriate management when necessary. Therefore the knowledge of imaging patterns for each disease aids accurate diagnosis.

The aim of this review is to describe the imaging features of some of the most frequent hepatic cystic lesions.

INTRODUCTION

Hepatic cysts (HC) are mostly incidental and asymptomatic findings. They are well-defined fluid-filled lesions, either lined or not lined by epithelium. The cystic component is different according to the particular lesion: bilious, serous, mucinous, necrotic, haemorrhagic, proteinaceous or mixed fluid. As the cystic component is a common feature, it is important to be aware of other characteristic features in order to differentiate between types of cyst, especially distinguishing benign from malignant tumours. The aim of this article is to describe all different cystic hepatic lesions, including typical radiological features that enable non-invasive diagnosis.

1. Ductal abnormalities

Ductal plate malformations correspond to a group of congenital cystic liver lesions that can affect both the intra- and/or extrahepatic biliary ducts. They result from insufficient remodelling and resorption of cylindral ductal plates (1).

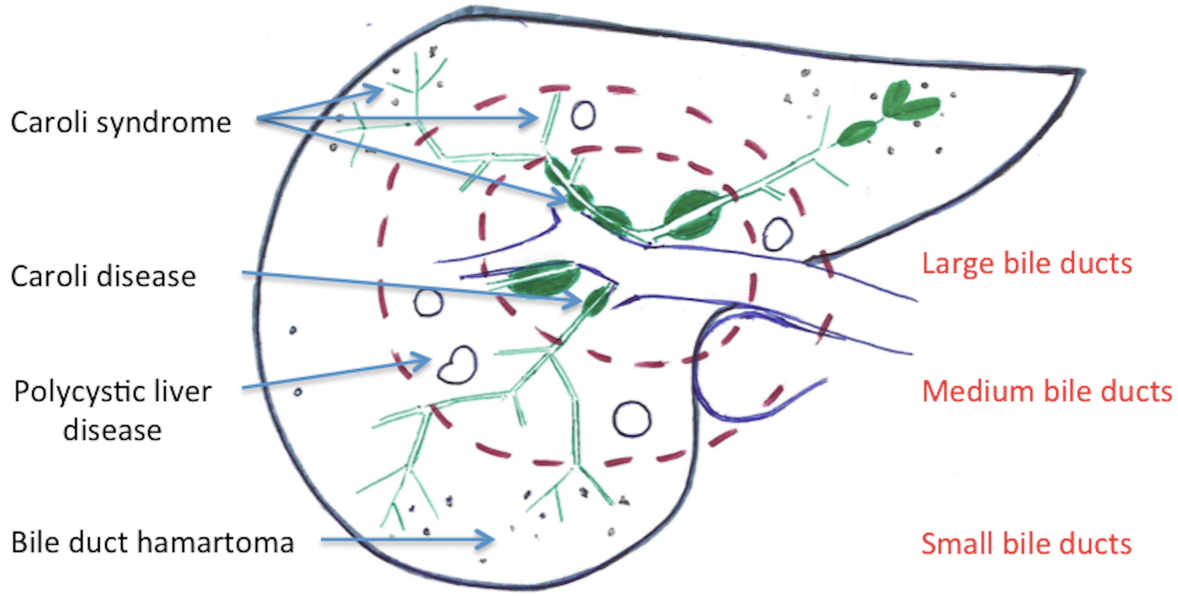


Figure 1: schematic figure (axial section of a liver) showing the different types of ductal plate malformation. The size of affected ducts is closely correlated to the ductal phase development.

1.1. Hepatic simple cyst

Aetiopathogenesis- Hepatic simple cysts (HSC) (also named bile duct cysts or biliary cysts) are congenital parts of the ductal tree detached from the main biliary system and dilated to become cystic lesion (2). Their origin is not consensual. They are thought to result, by some authors, from dilatation of bile duct hamartoma (cf. section) (3). They do not communicate with the biliary tract. In the literature, the prevalence is reported as ranging from 2,5% to 18%, increasing with age and more frequent in women (2-4).

HSC are benign lesions, usually asymptomatic. Cystic serous fluid is continually produces by a cuboidal biliary epithelium (5). A very large cyst can lead to abdominal pain or early satiety by compression. Complications (haemorrhage, infection, etc.) are rare, but when they do, they give rise to the "complex cyst".

Imaging- HSC are well-circumscribed unilocular lesions with a thin imperceptible wall, sometimes lobulated, anechoic on ultrasonography (US) with increased through-transmission. At computed tomography (CT), HSC are hypoattenuating (0–20HU); and hypointense on T1 / strongly hyperintense on T2 at magnetic resonance imaging (MRI). There is no internal nodule and no enhancement after intra-venous contrast (at US, CT or MRI) (Fig 2-3). Cysts can be bi- or multi-lobar (fused cysts) (Fig 5). Typical features on each imaging modality are sufficient for the diagnosis.

"Complex cyst"- Complications are rare but should be recognised, because they result in changes in the appearance of cysts and could therefore lead to misdiagnosis, especially with cystic tumours.

- In the case of **haemorrhage**, cysts can grow, and present with heterogeneous content and septations seen as echoic intra lesion material (Fig 4) (pain can occur during ultrasound examination). Attenuation is higher than simple fluid at CT and classical blood signal is seen at MRI (Fig 5-6). A fluid-fluid level can be seen (Fig 5). Inner septa are mobile but do not enhance. Pseudo-capsule in the form of a thin peripheral rim enhancement is depicted, and should not be mistaken for tumoral tissue.

- **Infection** is exceptional, except in cases of polycystic disease. Cysts present with a thick wall with heterogeneous enhancement, a fluid-fluid level and possibly some inner gas bubbles. Differentiating between cyst and collected abscess may be difficult without prior examination showing the HSC.

- **Rupture** is also very rare and, when it occurs, a "floating wall" inside the cyst is typically depicted. When cysts are subcapsular, rupture may be associated with peri-hepatic fluid (Fig 7).

- Large **symptomatic** HSC. Very large HSC remain asymptomatic in most patients, but they may also lead to pain, dyspnoea or duodenogastric compression. Treatment may be surgical (laparoscopic cyst fenestration) or image-guided by sclerotherapy. Due to the difficulty of attributing symptoms to the cyst with certainty, a diagnostic test may be carried out in a first instance by means of a simple aspiration of the cyst fluid.

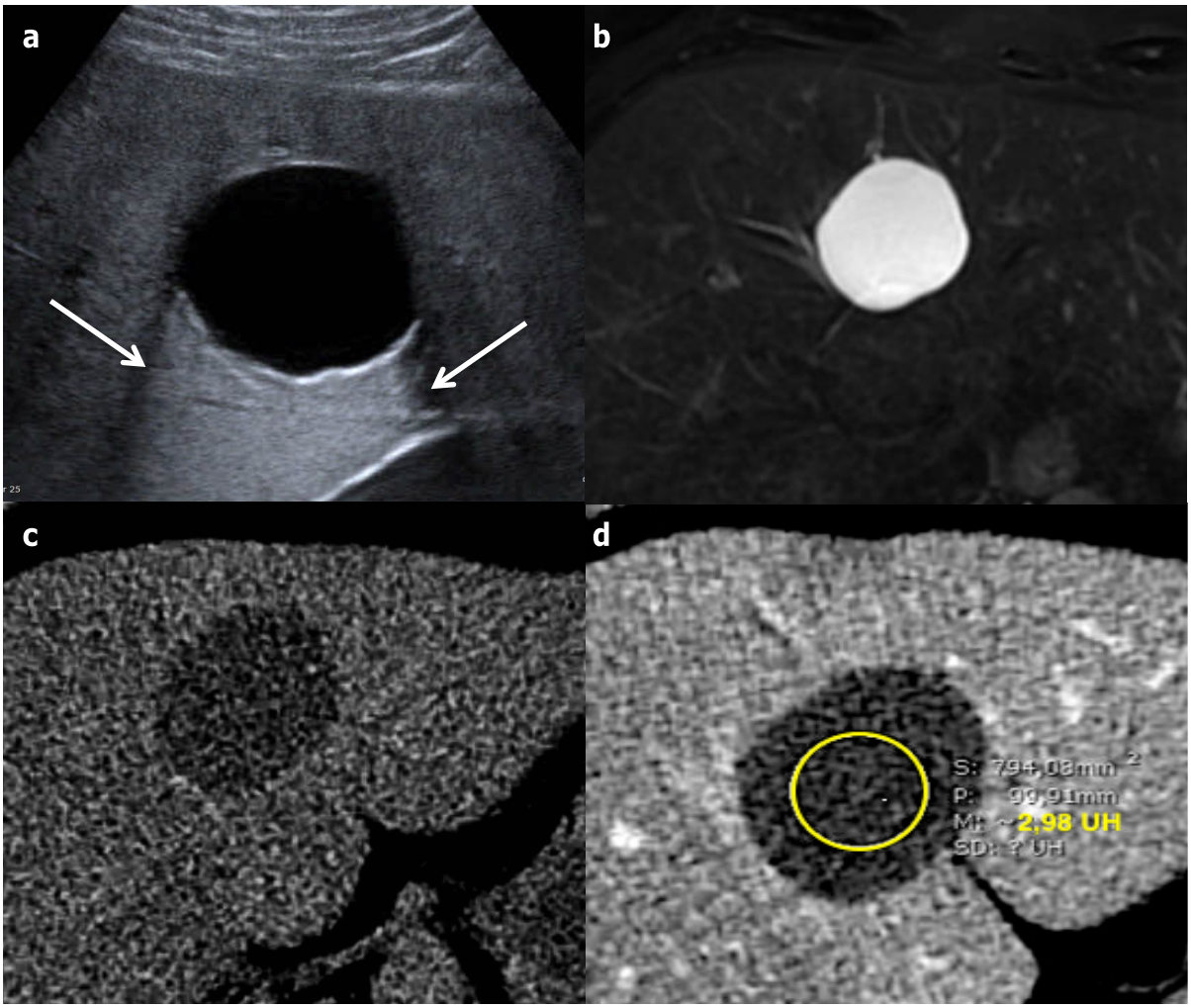


Figure 2: Incidentally hepatic simple cyst in an asymptomatic 64-year-old male patient. (a) Ultrasonography shows a round homogeneous anechoic well-circumscribed cystic hepatic lesion, without any mural nodule or vegetation. Arrows show an increased through transmission, confirming the cystic component of the lesion. (b) T2-weighted magnetic resonance imaging shows a heavily hyperintense round lesion. (c and d) Computed tomography without and with contrast at portal-venous-phase shows a round homogeneous non-enhancing hypoattenuating lesion (3 Hounsfield Units).

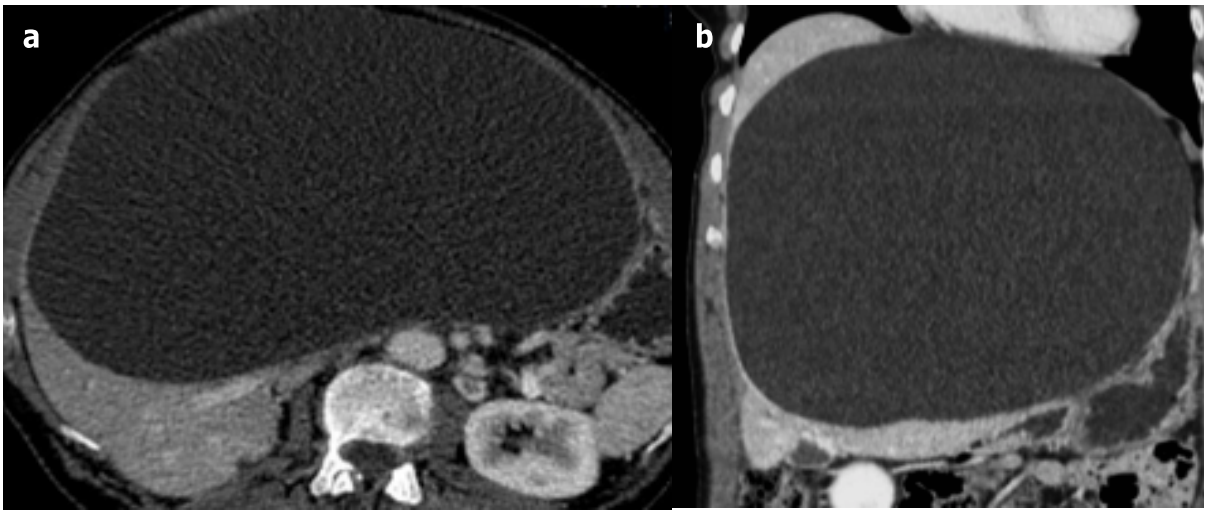


Figure 3: Very large hepatic simple cyst in a symptomatic 64-year-old female patient with abdominal pain. (a) Axial and (b) coronal computed tomography at portal-venous-phase shows a very large hepatic simple cyst. The cyst had been progressively growing for a number of years and has been finally treated by laparoscopic fenestration with an improvement of pain.

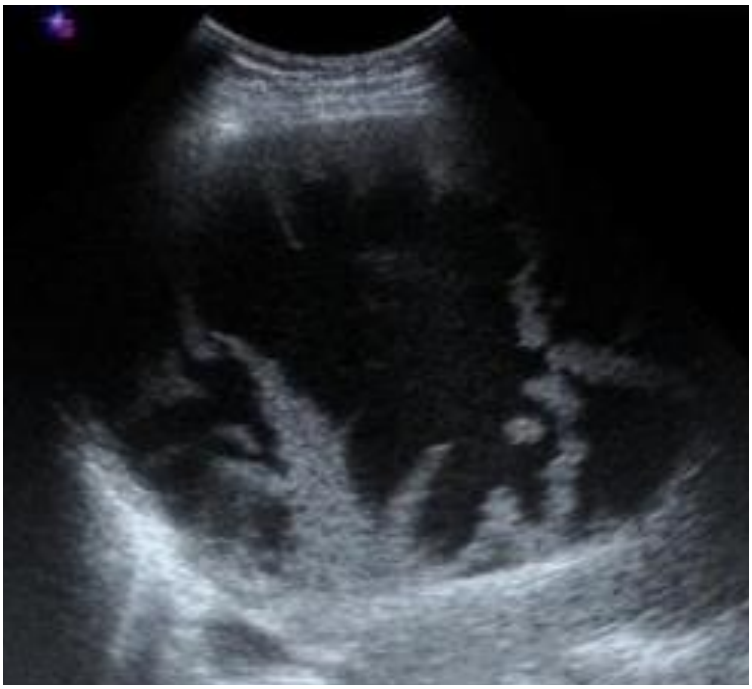


Figure 4: Semi-recent haemorrhage in a hepatic simple cyst in a 70-year-old male patient. Ultrasonography shows a spontaneous mobile hyperechogenicity inside the cyst, described as a "fern leaf".

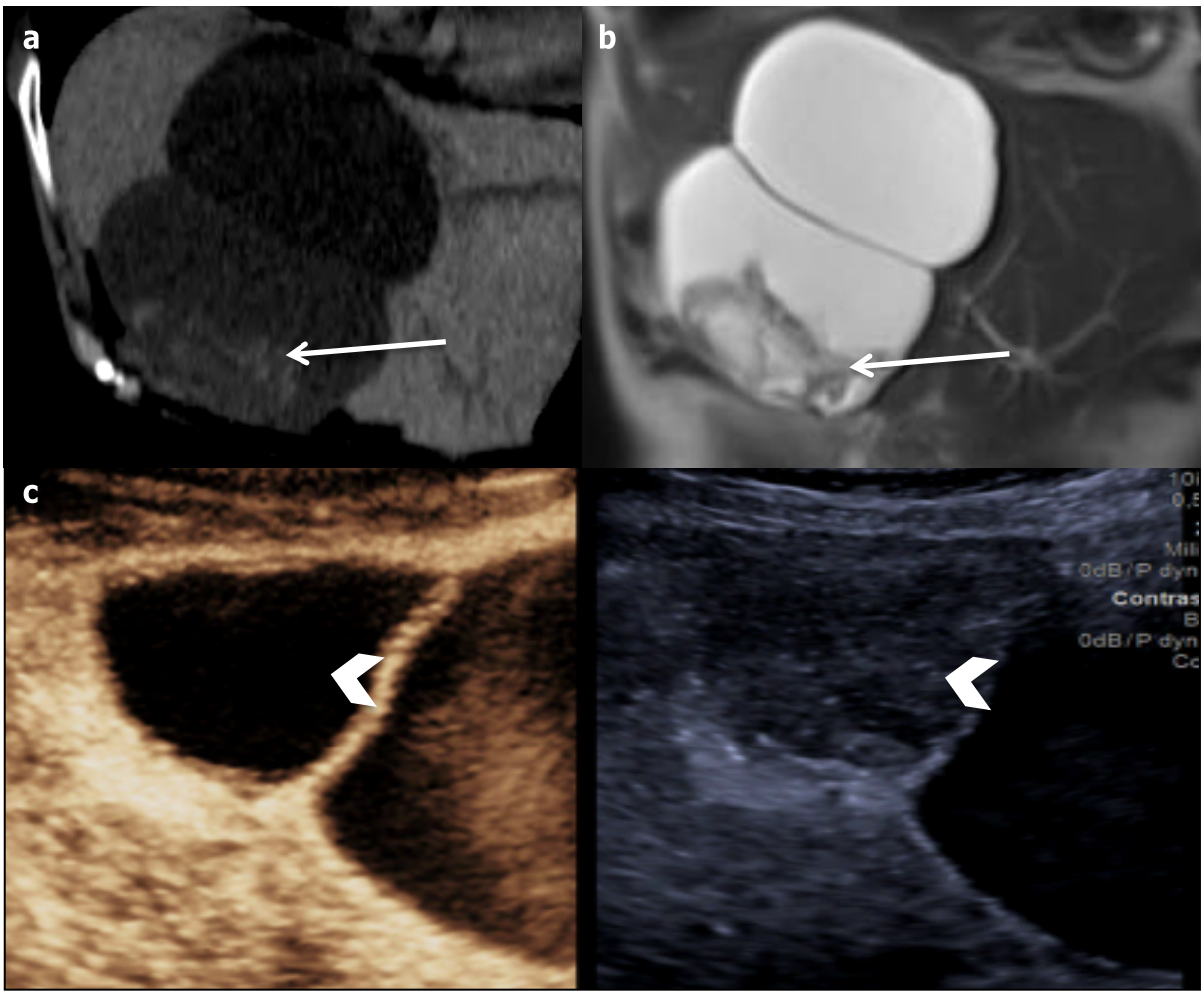


Figure 5: Bilobulated haemorrhagic hepatic cyst in a 73-year-old female patient. (a) Coronal non-enhanced computed tomography shows a bilobulated cyst with spontaneous declive hyperattenuation (arrow); (b) Coronal T2-weighted magnetic resonance imaging shows a declive heterogeneous hypointensity inside the inferior part of the cyst (arrow). (c) Contrast enhanced ultrasonography confirms the non-enhancement of the cystic part of the lesion (arrowhead).

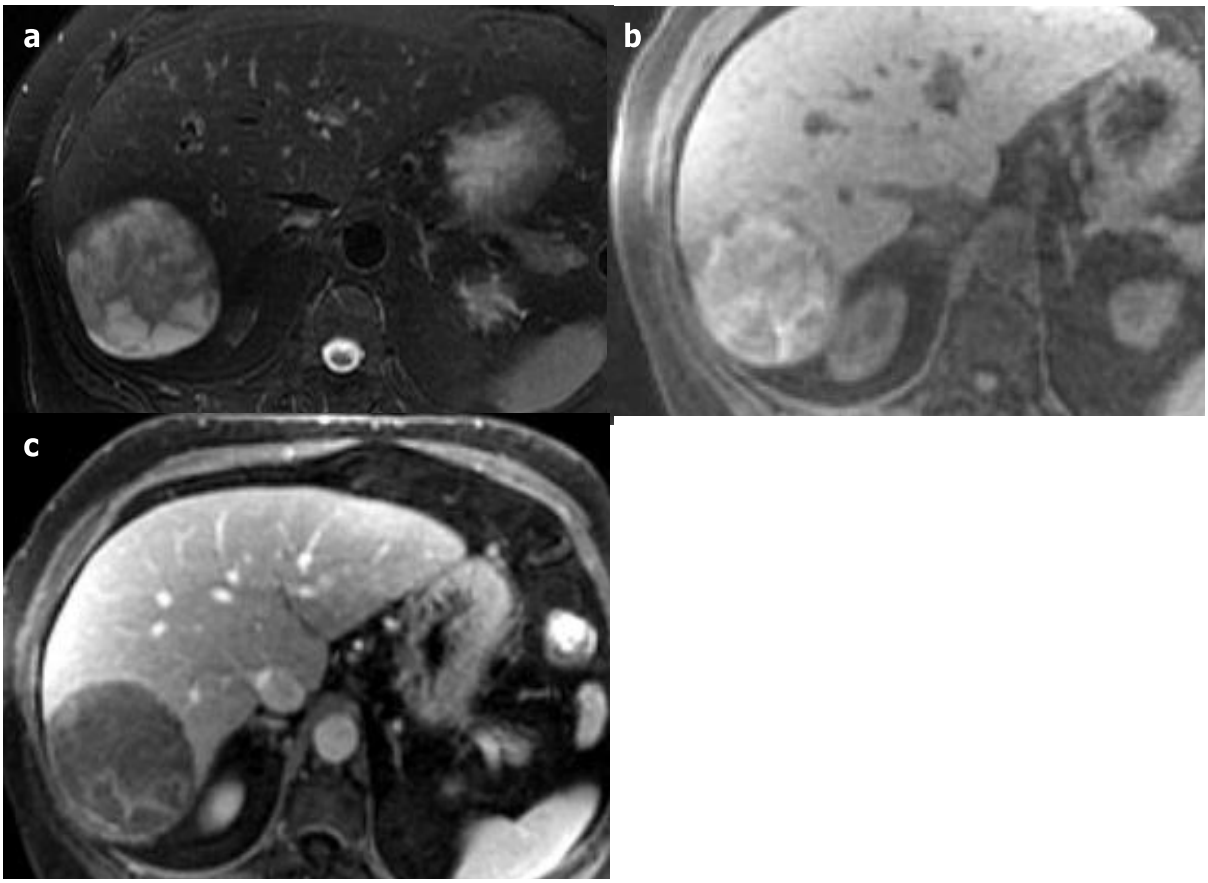


Figure 6: Haemorrhagic cyst at recent stage in a 66-year-old female patient. (a) Axial T2-weighted magnetic resonance imaging shows heterogeneous hyperintensity and (b and c) Axial T1 fat-sat-weighted magnetic resonance imaging shows a lesion spontaneously hyperintense with no enhancement after gadolinium-chelate injection.

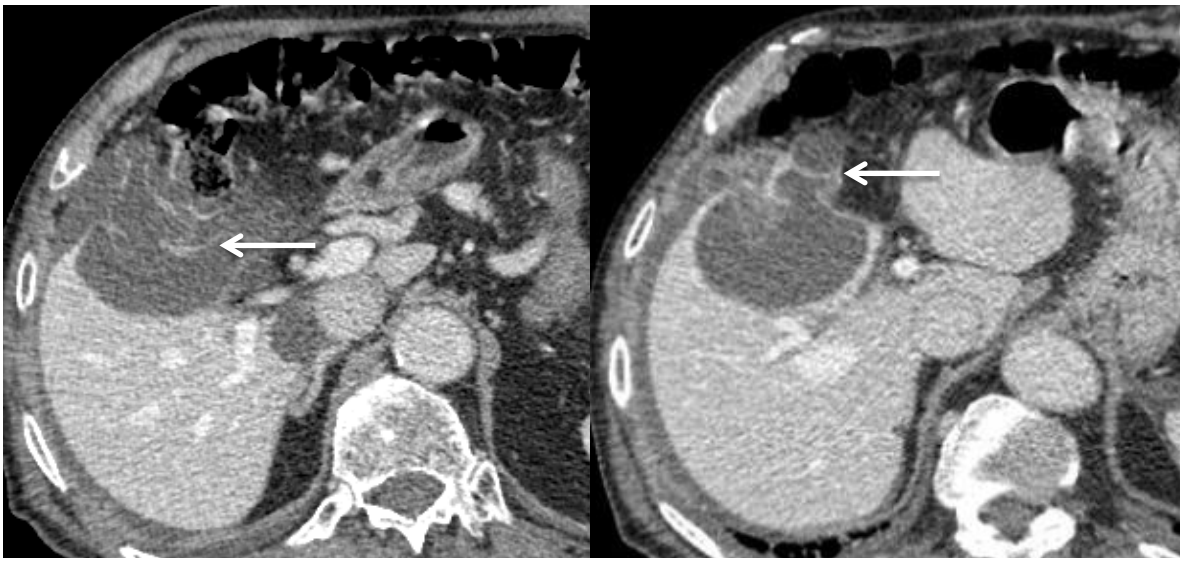


Figure 7: Rupture of a hepatic cyst in an 89-year-old male patient with violent abdominal pain. Axial computed tomography at portal-venous-phase shows a floating wall (arrow) associated with peri-cystic and peri-hepatic fluid collection.

1.2. Bile duct hamartoma

Aetiopathogenesis- Bile duct hamartomas (BH) (or von Meyenburg complexes) are benign lesions resulting from the lack of involution of small interlobular bile ducts at late phase of embryogenesis (6-7). They are asymptomatic and disconnected bile duct lesions with only limited dilatation, lined by biliary epithelium. The prevalence based on autopsy series is up to 5.6% in adults (7). They can be observed in a healthy liver or in association with other ductal plate anomalies (7). 35 cases of cholangiocarcinoma associated with BH have been reported in the literature up to 2016, without any other ductal plate malformation associated (7).

Imaging- They are commonly subcapsular or located at the periphery of portal tracts (6). US shows multiple small (<1.5cm) intra-hepatic cysts, mostly appearing hyperechoic due to their small size and to a fibrous stroma (Fig 8 a), with comet-tail artefacts (3,7). These are described as a "snowstorm". At CT, they are poorly visible, and appear round or irregular in shape with strict fluid density and no enhancement (Fig 8 c). MRI allows for better detection with a strongly T2-weighted sequence (7) showing a "starry sky" appearance (8) (Fig 8 d), and confirms the lack of communication with the biliary tract on magnetic resonance cholangiopancreatography (MRCP) sequence (Fig 8 b). In some cases, a thin regular persistent enhanced rim can be seen due to the compressed surrounding liver parenchyma (3). Typical features on each imaging modality are sufficient for a definitive diagnosis.

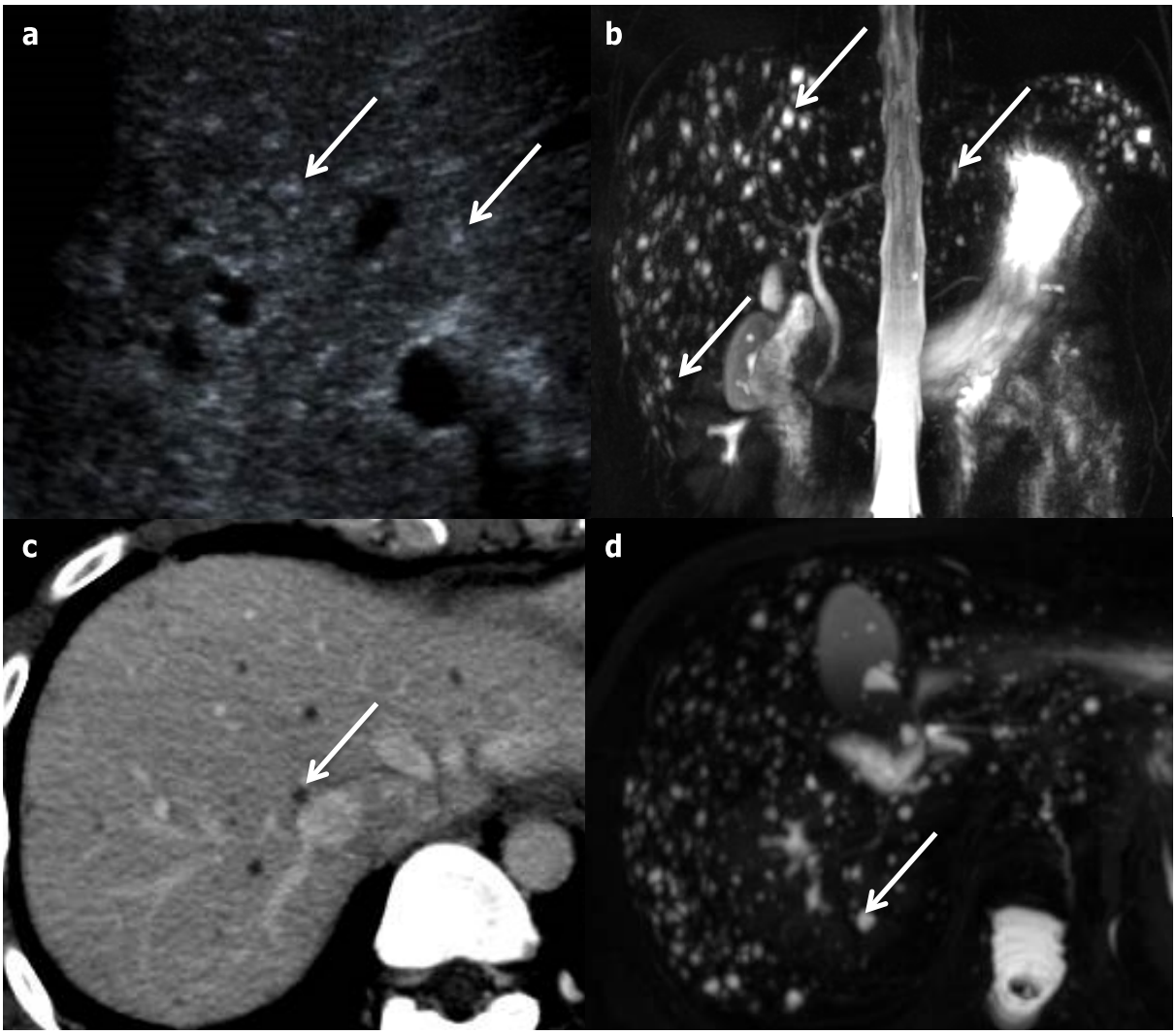


Figure 8: Bile duct hamartomas in a 52-year-old female patient. (a) Multiple hyperechoic round nodules (arrows) at ultrasonography, seen as "snowstorm". (b) Coronal magnetic resonance cholangiopancreatography shows several small hyperintense nodules not communicating with the biliary tract (arrows). (c) Axial computed tomography at portal-venous phase shows bilobar, small hypoattenuating round nodules, better detected on an axial magnetic resonance cholangiopancreatography sequence (d).

1.3. Caroli syndrome and disease

They are both extremely rare congenital anomalies in the ductal plate development (prevalence of less than one in 1,000,000 inhabitants) (5,9). Caroli disease (CD) is less frequent than Caroli syndrome (CS). CS must be differentiated from CD. The origin of CD is unclear. It is mostly thought to be non-hereditary and rarely transmitted in an autosomal dominant mode. CS is transmitted in an autosomal recessive mode. CD is thought to result from a partial or complete interruption of ductal plate remodelling at an early phase. In CS the anomalies occur both at an early and late phases involving peripheral bile ducts (1,8). Follow-up is mandatory for both, due to the risk of cholangiocarcinoma (10). In addition, the cysts and biliary tract can contain stones, resulting from the stagnation of bile.

1.3.1. Caroli disease

Aetiopathogenis- The lack of peri-portal biliary epithelium regression during embyogenesis creates an aneurysmal dilatation of large intra-hepatic bile ducts (3,8). The liver parenchyma is normal. Symptoms are possible: abdominal pain, jaundice, and/or cholangitis.

Imaging- Biliary tract is abnormal with focal dilated bile ducts without stenosis. The cystic dilatations are connected to the biliary tract and have thin walls with a "central dot sign", corresponding to a residual portal vein and arterial branch (8) (Fig 9). The Doppler or contrast-enhanced CT/MRI attest to its vascular origin (5).

MRCP is the modality of choice for assessing lesions. Cystic hepatic lesions communicate with the biliary tract. The use of hepatobiliary MRI contrast agents can be

useful for confirming communication between the biliary tracts and cysts (5,8). There is no liver atrophy.

1.3.2. Caroli syndrome

Aetiopathogenesis- This syndrome combines bile duct dilatations and congenital hepatic fibrosis. CS may be associated with autosomal recessive polycystic kidney disease (PKHD1 mutation) (11). Symptoms are a consequence of liver fibrosis. Follow-up is required to prevent the complications induced by hepatic fibrosis but also due to the high risk of cholangiocarcinoma (about 7%) (10).

Imaging- Features are closely similar to those of CD, showing cysts communicating with dilated bile ducts. They are usually smaller in CS than in CD (<3cm), with diffuse fusiform dilatation of the biliary tract (5).

The associated hepatic fibrosis may lead to specific dysmorphia (hypertrophy of the left and caudate lobes, *normal sized or hypertrophy* of segment IV and hypotrophy of the right lobe (12)) and features of portal hypertension (porto-systemic collateral vessels, splenomegaly and ascites) (Fig 10).

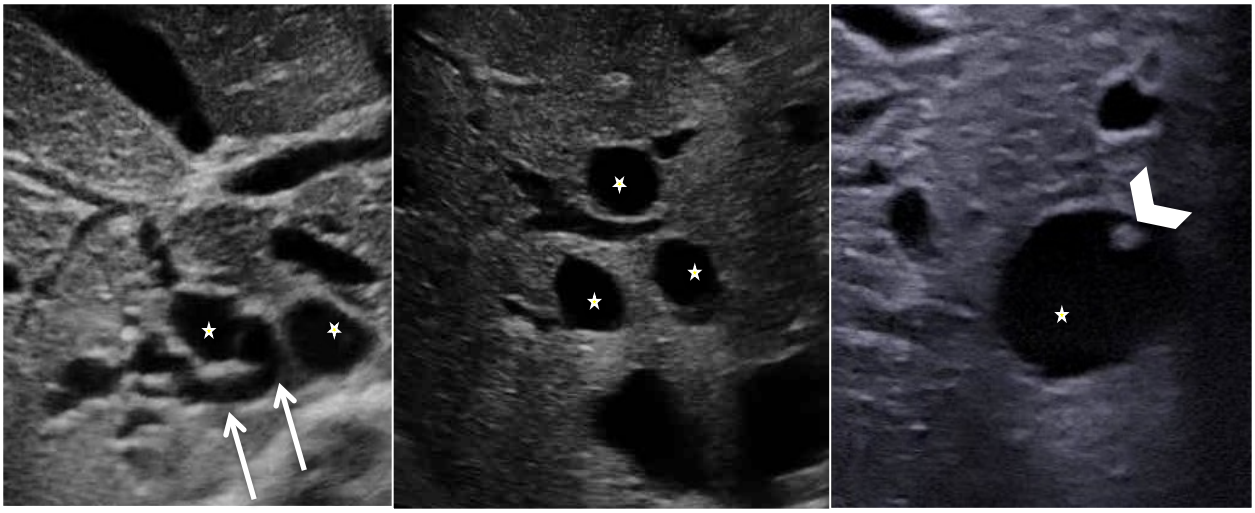


Figure 9: Caroli syndrome in a 7-year-old female patient, at ultrasonography. Several round anechoic cysts (stars) with thin wall, increased through-transmission, connected to the dilated fusiform biliary tract (arrows), with some of them showing the highly specific "central dot-sign" (arrowhead).

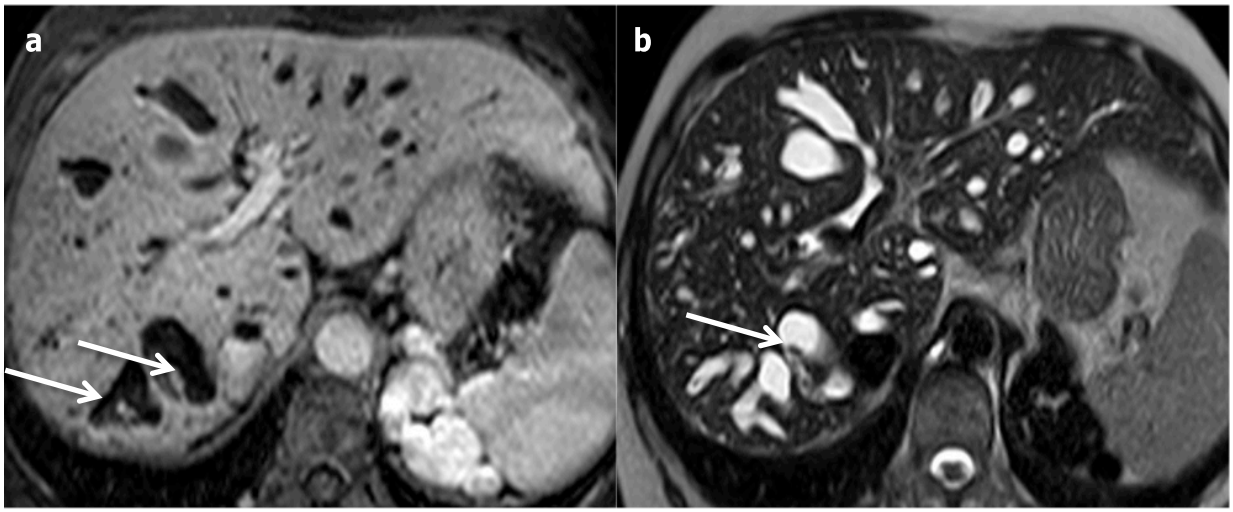


Figure 10: Caroli syndrome in a 53-year-old female patient. (a) Axial magnetic resonance imaging at portal-venous-phase shows peripheric fusiform dilated bile duct, hyperintense at axial T2-weighted magnetic resonance imaging (b), with the specific "central dot-sign" (arrows). Note the presence of spleno-renal shunts due to portal hypertension.

1.4. Polycystic liver disease

Aetiopathogenesis- Polycystic liver disease (PLD) involves either liver disease (autosomal dominant polycystic liver disease (ADPLD): ≈ 1:100,000) or liver and kidney disease (autosomal dominant polycystic kidney disease (ADPKD): ≈ 1:600-1,000, autosomal recessive polycystic kidney disease (ARPKD) ≈ 1:20,000) (13). The number of cysts required for diagnosis has not been clearly defined. The presence of at least 10 hepatic simple cysts suggests the diagnosis according to the international PLD registry steering committee (14). As for HSC, complications may include organ compression (including biliary and hepatic vessels), haemorrhage, infection (Fig 11), or rupture. Detection of an infected cyst among the multiple cysts might be difficult. 18-Fluorine-fluorodeoxyglucose positron emission tomography (18-F-FDG PET) CT may help in identification (Fig 12).

Imaging- Cysts are similar to HSC and are diffuse in the liver (Fig 13). They may coalesce and linear calcification can be seen between cysts. They frequently lead to hepatomegaly and to vascular compression with venous collaterals (15).

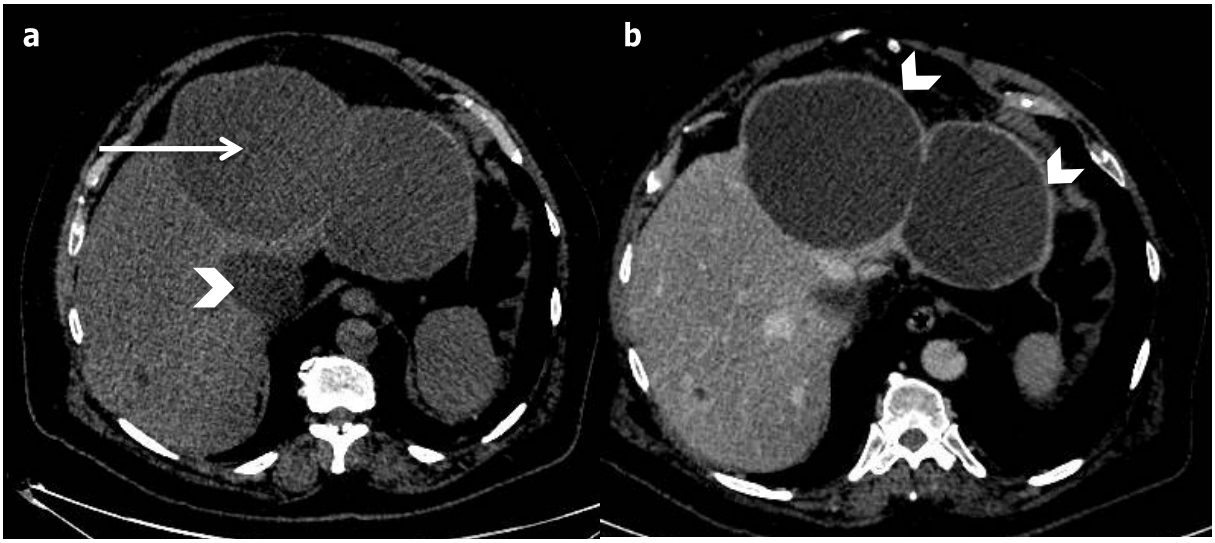


Figure 11: Infected simple hepatic cysts in a 77-year-old male patient with autosomal dominant polycystic liver disease. (a) Axial non-enhanced computed tomography shows two tensioned cysts (arrow) slightly more attenuating than non-infected cysts (arrowhead); (b) with thick enhanced wall at portal venous-phase (arrowhead).

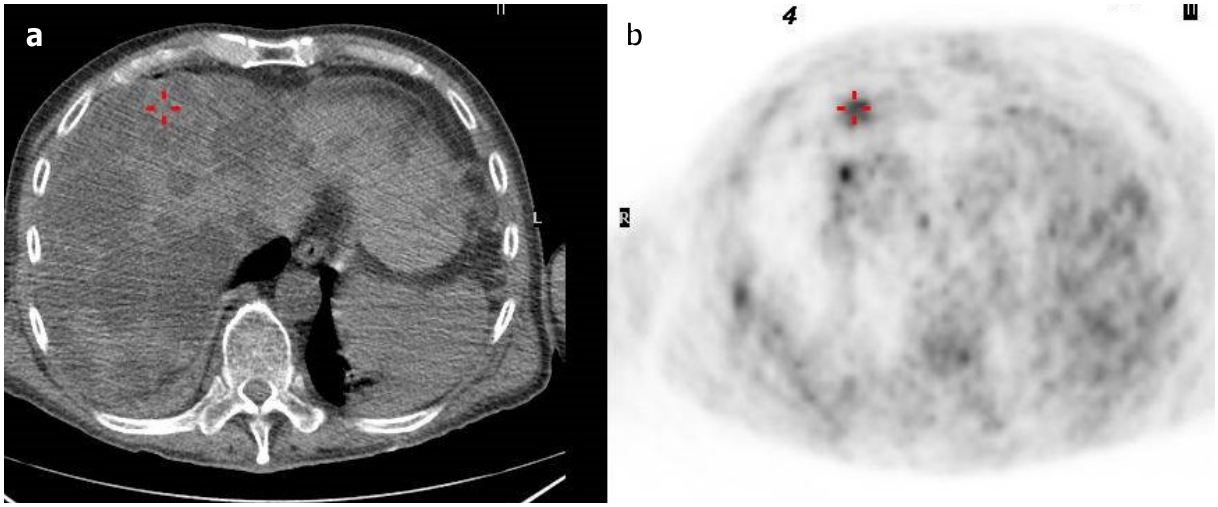


Figure 12: Infected cyst in a 65-year-old male patient with autosomal dominant polycystic liver disease. (a) Axial non-enhanced computed tomography shows one more attenuating cyst (cross) among multiple simple cysts; (b) hypermetabolic at 18-F-FDG PET computed tomography.

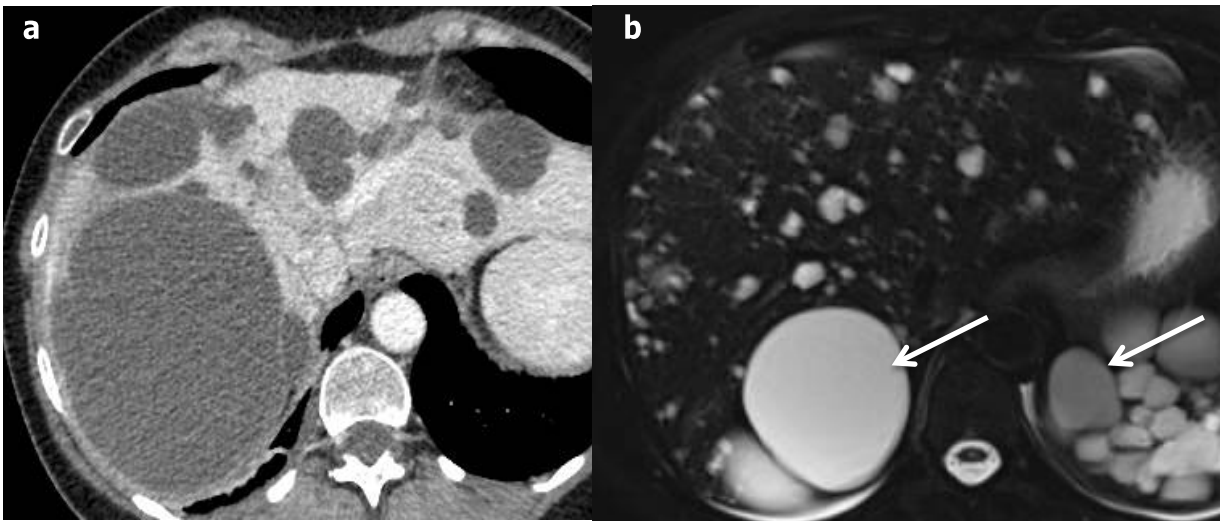


Figure 13: Autosomal polycystic kidney disease in a 38-year-old male patient. (a) Axial computed tomography at portal-venous-phase and (b) axial T2-weighted magnetic resonance imaging show several hepatic simple cysts and also numerous kidney cysts (arrows), with fluid signal (hypodense/hyperintense on T2).

2. Other cystic lesions

2.1. Peribiliary cyst

Aetiopathogenesis- Peribiliary cysts are small serous cysts resulting from obstructed extramural hilum peribiliary glands. They must not be confused with biliary dilatations. Aetiopathogenesis is not known. Their exact prevalence is unknown but they are mostly found in cirrhotic liver (50% in autopsy studies (16)), in ADPKD or ADPLD (17). On rare occasions, they have been reported to cause compression and therefore a dilatation of bile ducts.

Imaging- They are multiple, usually small (from 1 to 55mm (17)), simple cystic lesions, seen as a "string of pearls", around hilar portal veins (Fig 14). They are predominant in the left lobe due to the preponderance of the peribiliary gland (5). They can communicate with each other mimicking biliary dilatations but do not communicate with the biliary tract. MRCP helps differentiate them from biliary dilatation (Fig 14). Hepatobiliary MRI contrast agent can be used to prove the absence of biliary communication (5). On rare occasions, they may be confused with dilated lymphatic vessels (18).

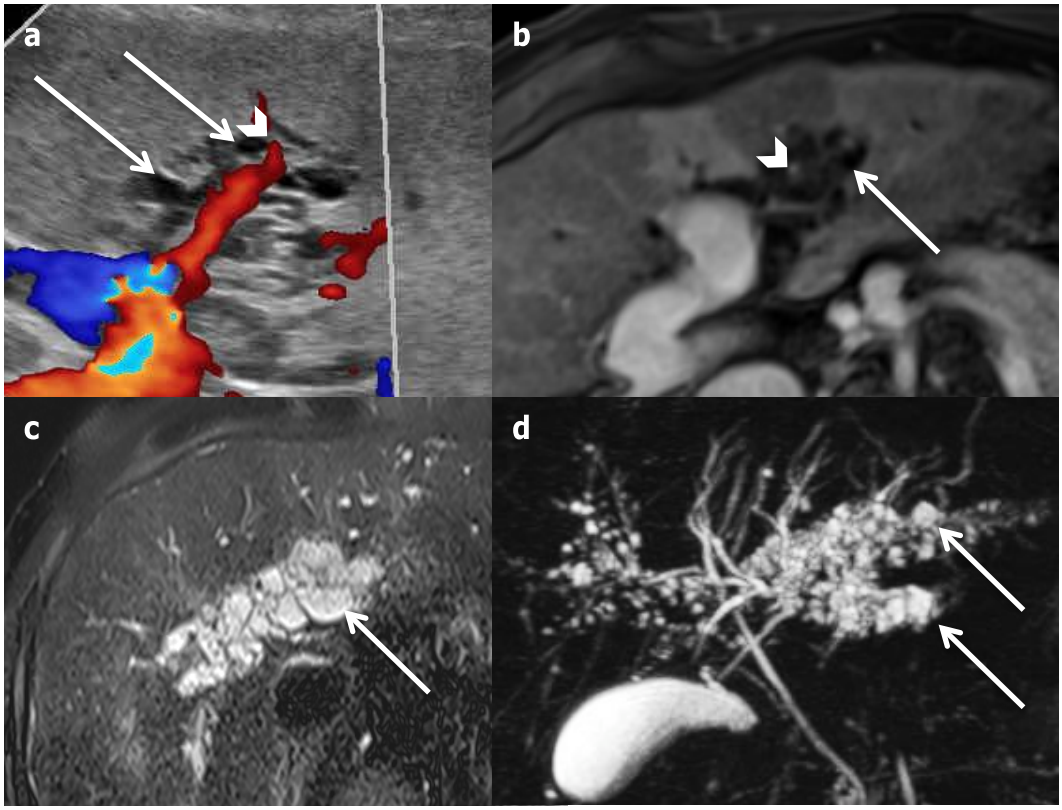


Figure 14: Peribiliary cyst in a 69-year-old cirrhotic male patient. (a) Ultrasonography shows multiple periportal (arrowhead) cysts (arrows). (b) The portal vessels are clearly visible (arrowhead) at axial portal-venous phase magnetic resonance imaging, assessing the periportal location of the cysts. (c and d) Axial T2-weighted magnetic resonance imaging and magnetic resonance cholangiopancreatography show multiples small cysts along thin, non-dilated bile ducts.

2.2. Hepatic lymphatic malformation

Aetiopathogenesis- Hepatic lymphatic malformation (HLM) is a extremely rare (8 cases reported in the literature up to 2010 (19)) benign cystic dilatation of the liver lymphatic spaces, commonly developing in systemic lymphangiomatosis (20-21). Cysts are lined with endothelial cells, filled with lymph (20). Three subtypes are described: capillary (super-microcystic), cavernous (microcystic), or cystic (macrocystic) (20-21).

Through immunohistochemistry, D2-40 has been reported to be a highly specific antibody of the lymphatic endothelium assessing the diagnosis at histology (22).

Imaging- Hepatic lymphatic malformations can be unilocular but are more often multilocular, with internal septations sometimes enhanced as the wall. They can be confused with cystadenoma, cystadenocarcinoma or scleroting haemangioma (19,23). MRI has been shown to be very effective in distinguishing them from the latter. They demonstrate varying signal on T1- and T2-weighted sequences depending on the quantity of fat and fluid components (24). Lympho-MR sequences can show dilated lymph duct surrounding the lesion, supporting the diagnosis (22). They can spontaneously disappear (Fig 15).

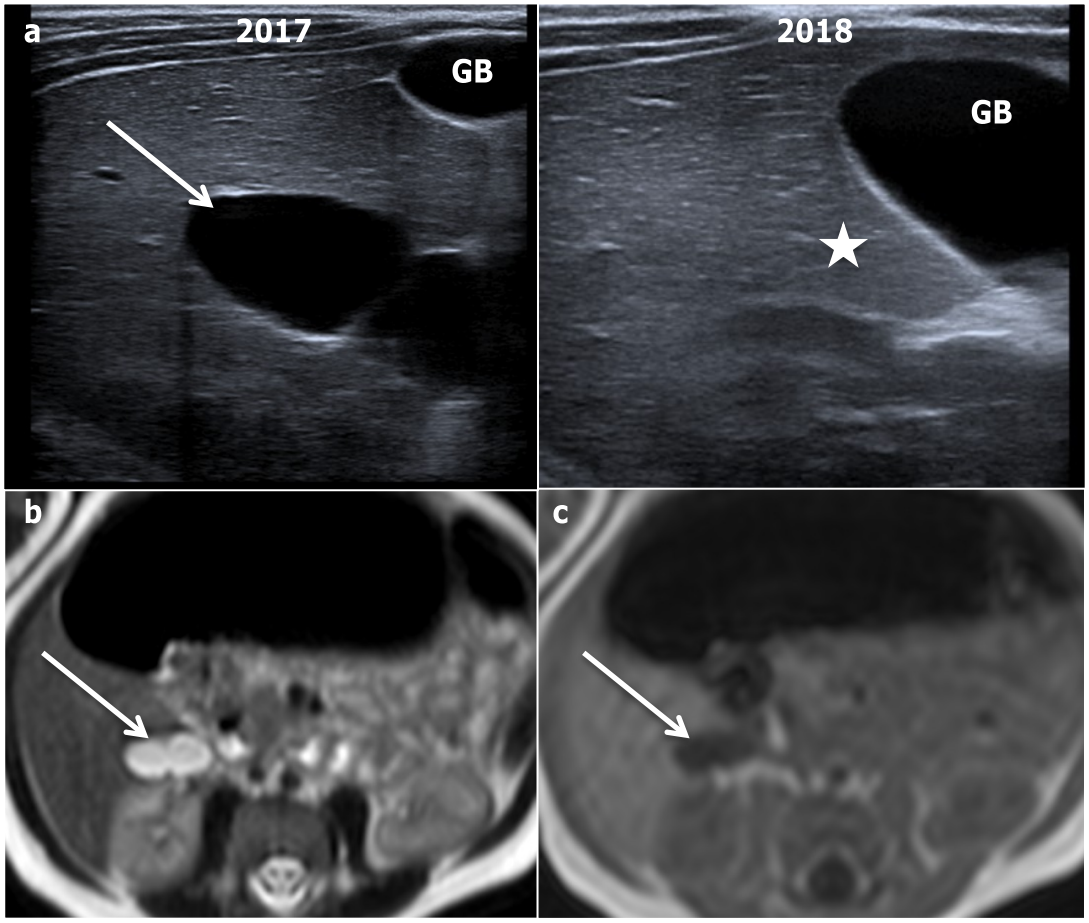


Figure 15: Spontaneous disappearance of a hepatic lymphatic malformation in 5-year-old male patient. (a) Ultrasonography shows biloculated homogeneous cyst (arrow) that has vanished one year later (star). It displays fluid signal (arrows) at axial T2-weighted (b) and T1-weighted magnetic resonance imaging (c).

2.3. Ciliated hepatic foregut duplication cyst

Aetiopathogenesis- Ciliated hepatic foregut duplication cyst (CHFC) is a very rare (about 100 cases reported in the literature up to 2012 (25)) and usually incidental finding. It seems to result from a congenital malformation from the embryonic foregut (25). It comprises four layers: inner pseudo stratified columnar epithelial lining; a subepithelial loose connective tissue; smooth muscle; and fibrous capsule (26). Abdominal discomfort is possibly associated due to its subcapsular localization (27).

The diagnosis can be assess only by fine needle aspiration showing ciliated pseudostratified tall columnar epithelial cells suspended in a mucoid background with immunohistochemical stains, or by histology (28). Complete surgical excision is recommended (26) due to the risk of malignancy, especially when larger than 5 cm (29-30).

Imaging- A solitary small (<4cm) subcapsular cystic lesion in the IV segment is very suggestive of CHFC. However, it has also been described in segment V or VIII (26,27,30). Cysts are non-enhancing, without septa, partition, nor internal nodule.

Content is usually fatty or protein-rich. In this case, it can lead to a fluid-fluid layer (30). CHFC are hypoechoic at US but usually not entirely transonic, and they can be spontaneously hyperattenuating at CT. At MRI, cysts are hyperintense on T2-weighted,

but the signal is lower than a hepatic simple cyst. On T1-weighted, signal is usually spontaneously hyperintense. They do not demonstrate any enhancement (Fig 16) (31). However, classical features can be missing due the different component of the cyst: mucinous component, cholesterol component, or calcium deposits (5).

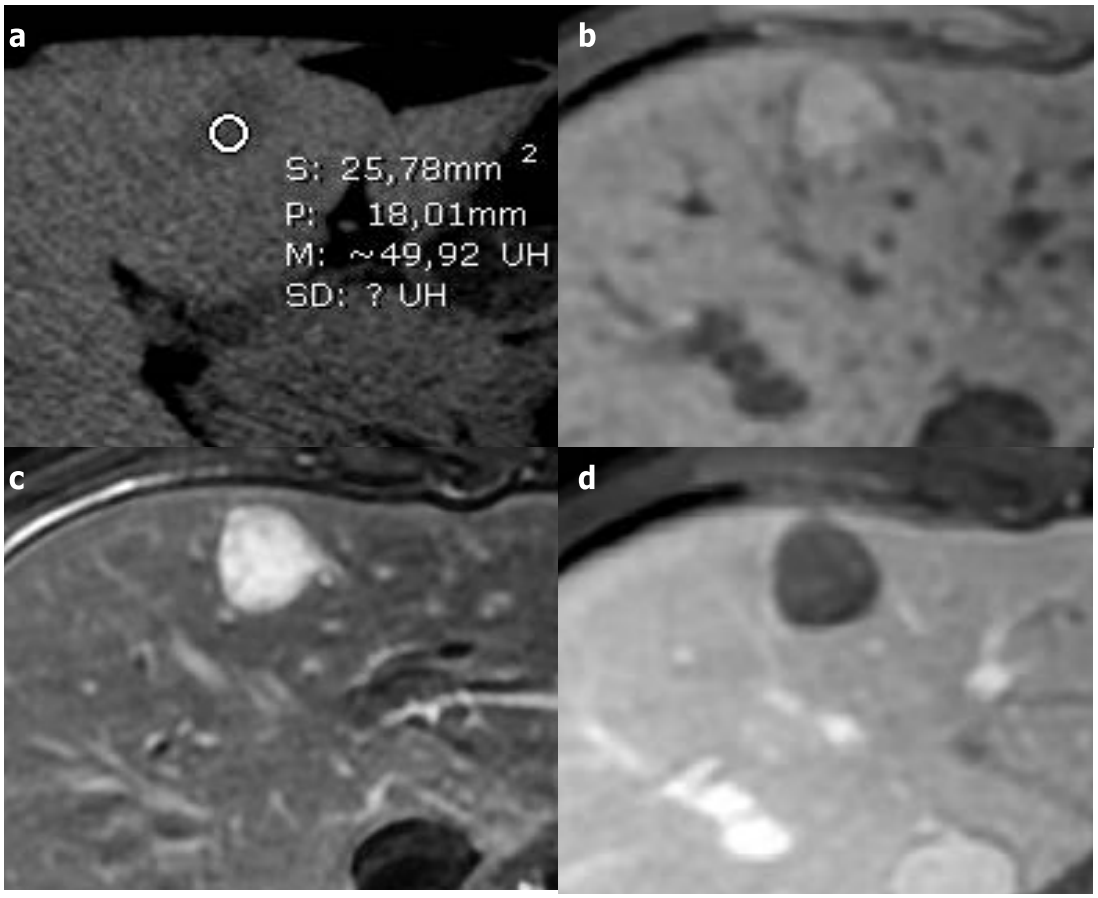


Figure 16: Ciliated hepatic foregut duplication cyst in a 15-year-old male patient. (a) Axial non-enhanced computed tomography shows a subcapsular nodule of the IVth segment with spontaneous attenuation around 50 Hounsfield Units. (b) At axial T1-weighted magnetic resonance imaging, it is spontaneous hyperintense. (c) It displays a high level of hyperintensity at axial T2-weighted magnetic resonance imaging. (d) Axial portal-venous phase T1-weighted magnetic resonance imaging shows neither enhancement nor wall thickening.

3. Cystic tumours

3.1. Mucinous cystic neoplasm of the liver

Aetiopathogenesis- Mucinous cystic neoplasms of the liver (MCN-L), previously known as biliary cystadenoma and cystadenocarcinoma, are rare cystic neoplasms (3-5% of hepatic cysts (2)) without biliary communication. They are composed of cuboidal to columnar, variably mucin-producing epithelium, associated with ovarian-type subepithelial stroma and are subdivided into non-invasive and invasive types (32). They frequently occur in women. The diagnosis can be hinted at by the intralesional assay of specific tumour markers (TAG-72) (table I) (33).

Treatment requires surgery, because radiology cannot differentiate non-invasive from invasive types with certainty (2).

Imaging- They are frequently large (1.5 - 35cm) solitary multilocular cystic lesion, mostly located close to the hepatic hilum in the segment IV, with well-circumscribed irregular margins and septations. Internal septa can be calcified (34). Septations arising from the internal cyst wall without external indentation seem to be a specific sign for differentiate MCN-L from HSC (35).

At US, they appear as large unilocular anechoic lesion, with thickened and irregular walls and internal septations (36). CEUS can confirm the enhancement of inner septas.

At CT, they appear as a solitary hypoattenuating lesion with septations, a thick wall, rarely with calcification (36). Cysts show different levels of attenuation according to their component.

At MRI, the signal intensity of the lesion may vary according the cystic fluid in every stall (e.g. protein rich), which is very specific (36). A fluid-fluid level can be seen in cases of haemorrhagic content. The capsule and the septas are enhanced (Fig 17). Haemorrhagic fluid, solid mural nodule or calcification are most commonly associated to the invasive type, with high specificity of mural nodules (5,34) (Fig 18).

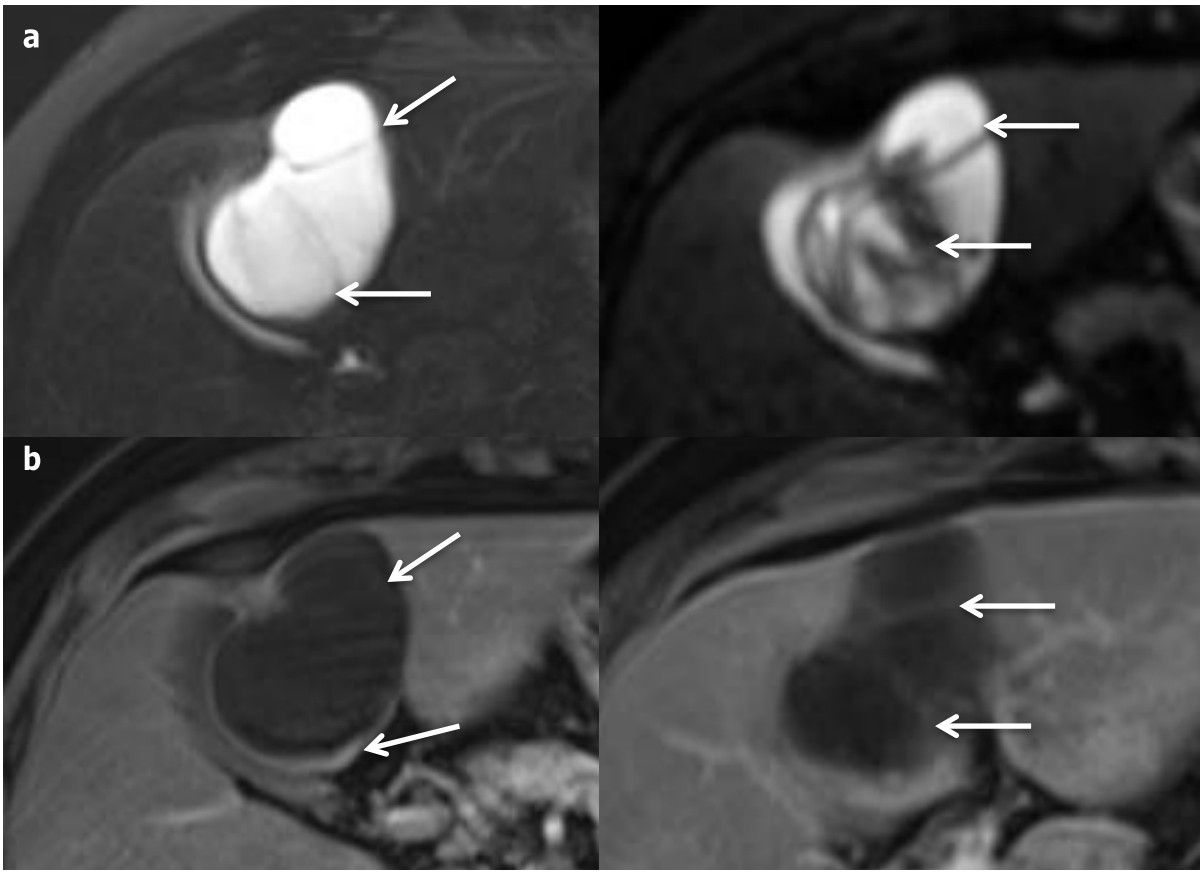


Figure 17: Typical non-invasive mucinous cystic neoplasm of the liver in a 59-year-old female patient. (a) Axial T2-weighted magnetic resonance imaging shows a solitary hyperintense cystic lesion, with hypointense septas (arrows). (b) Axial portal-venous-phase T1-weighted magnetic resonance imaging shows enhancement of the capsule and septas (arrows).

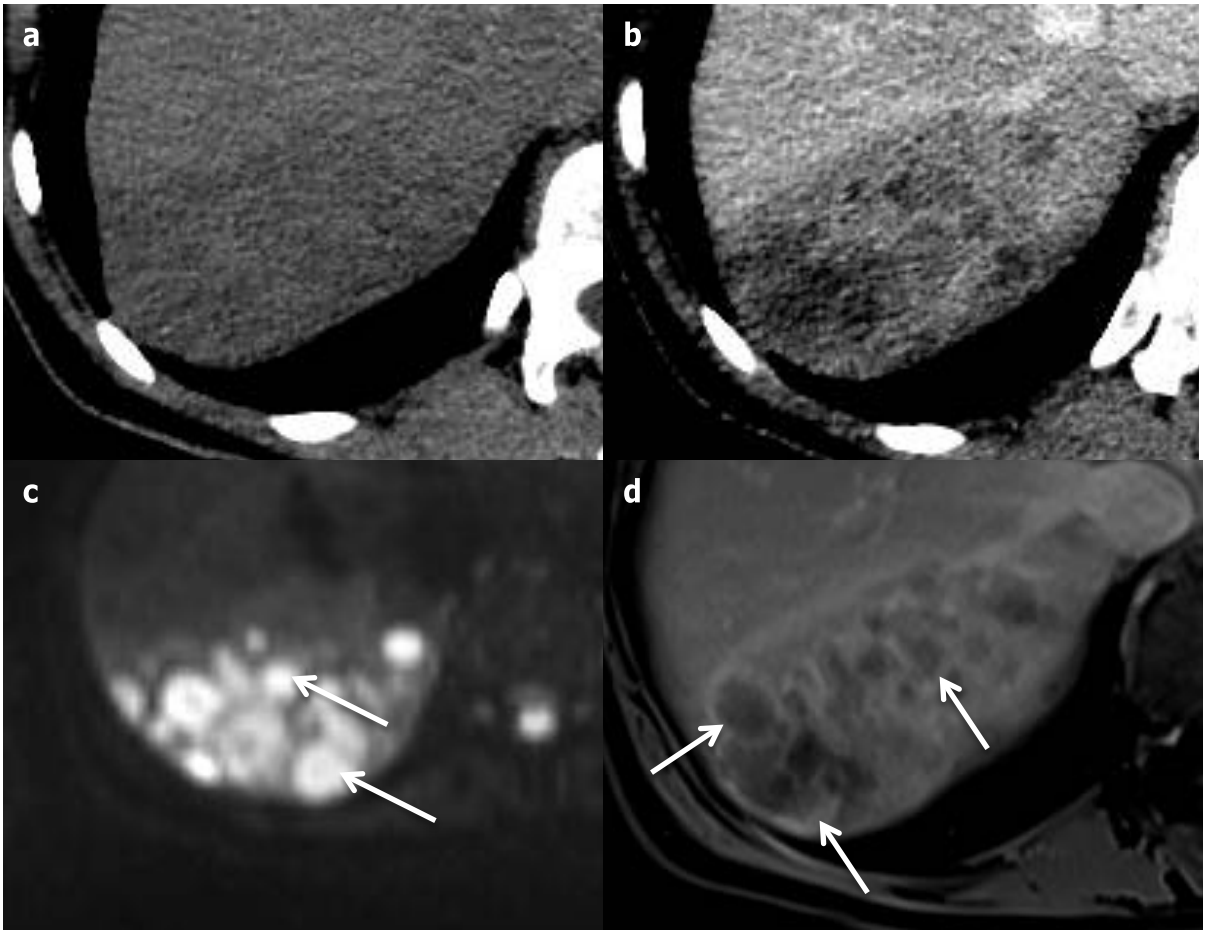


Figure 18: Invasive mucinous cystic neoplasm of the liver in a 50-year-old female patient. (a) Axial non-enhanced computed tomography shows a hypoattenuating lesion with thick heterogeneous enhancing wall at portal-venous-phase (b) better seen at axial portal-venous-phase T1-weighted magnetic resonance imaging (d) (arrows). Note the presence of cystic portions on axial T2-weighted sequence (c), in very intense hypersignal T2 (arrows).

3.2. Intraductal papillary neoplasm of the bile duct

Aetiopathogenesis- Intraductal papillary neoplasm of the bile duct (IPNB) is a rare premalignant neoplasm of the bile duct (10% of all bile ducts tumours (37)), however 40% can contain malignant component (37). The risk of malignant transformation is high and lesions must be treated by surgical resection when possible (38-39).

Imaging- The appearance of the lesions depends on the degree of papillary and mucin production of the tumours (40). They mostly appear as an intraluminal cystic mass within the bile duct, with upstream duct dilatation (Fig 19). If there is an important mucin production, lesions can appear only as focal or diffuse bile duct dilatations, without any tissular part visible. Aneurysmal dilatation of a branch of the biliary tree is considered a characteristic sign of IPNB by some authors (41). Intracystic tissular obvious mass points to the likelihood of a malignant transformation (40). The intra-luminal mass may appear hypo- or hyperechoic at US, highly enhanced at CEUS, CT and MRI at arterial phase (Fig 19 d). CEUS is very sensitive for detecting cystic wall or nodule enhancement (38). In opposite to intraductal cholangiocarcinoma, enhancement is not increasing on portal and delayed phases (40). Diffusion may be restricted but it is not indistinguishable from that of cholangiocarcinoma.

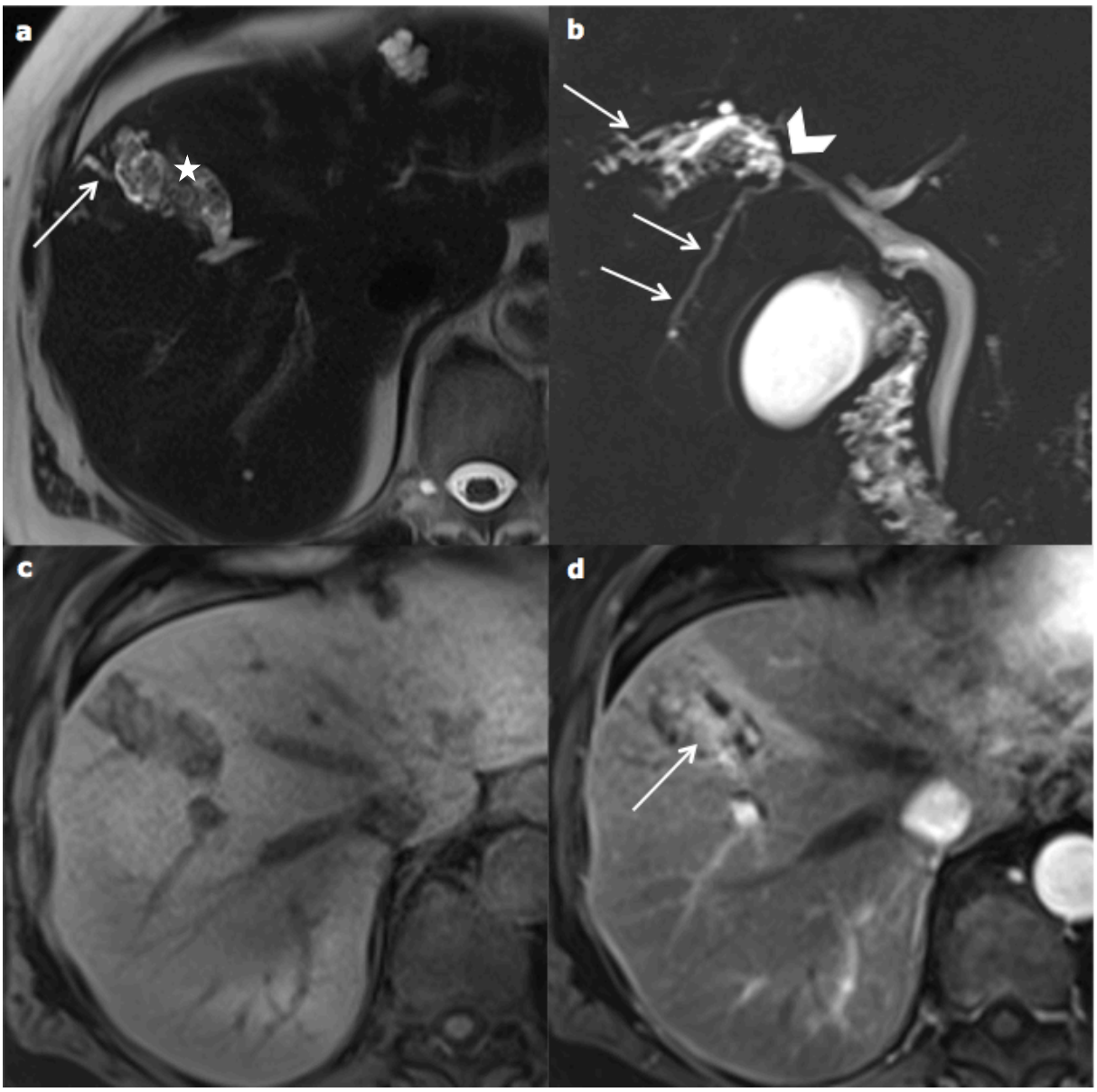
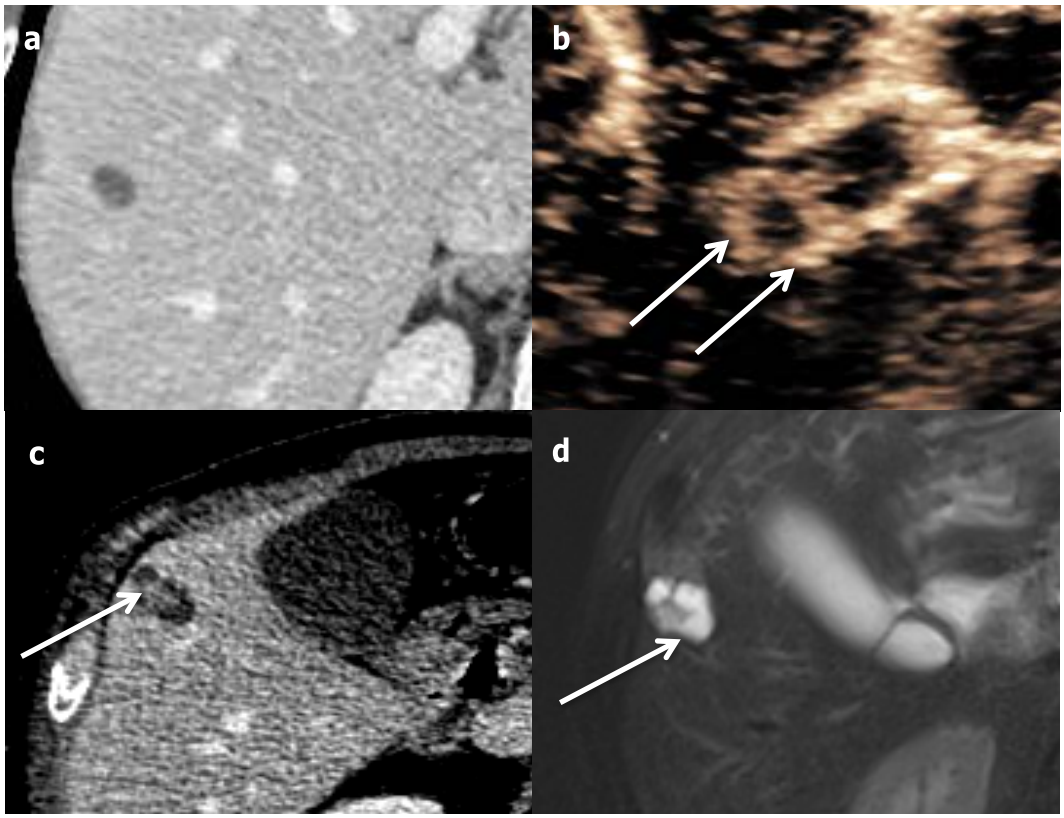


Figure 19: Intraductal papillary neoplasm of the bile duct in a 67-year-old female patient. (a) Axial T2-weighted magnetic resonance imaging shows intraductal heterogeneous cystic and tissular mass (star) with dilated bile duct in contact (arrows). The cystic part of the mass is better seen at magnetic resonance cholangiopancreatography (b) as well as upstream biliary dilatation (arrows) and the connection to the biliary tree (arrow-head). (c and d) Axial T1-weighted non-enhanced and at arterial-phase magnetic resonance imaging shows a highly enhanced tissular part.

3.3. Secondary cystic lesion: cystic metastasis

Aetiopathogenesis- Cystic metastases are less frequent than solid metastases and are rarely completely cystic. The cystic part of the metastases can be due to spontaneous necrosis especially in case of hyperenhancing metastases (neuroendocrine tumour, malignant melanoma, sarcoma or gastrointestinal stromal tumour (GIST)), or secondary to a systemic or locoregional treatment (34). There are exceptional cases of cystic liver metastases from squamous cell carcinoma (42). Mucin production (mucinous colorectal or ovarian adenocarcinoma) can also lead to cystic feature.

Imaging- They can be multilocular or unilocular, and present thick walls, irregular contours or mural nodules (Fig 20 c-d). Irregular thick septas are possible. The fluid component is a result of mucin, necrosis or haemorrhage (34), therefore the attenuation at CT and the signal at MRI are different from pure fluid. US may be useful to distinguish truly pure fluid cystic mass from cystic metastases, especially when there are only few. CEUS can show fine peripheral arterial enhancement with early washout (Fig 20 b). The clinical setting has to be considered.



Figures 20: (a-b) Cystic metastasis from malignant melanoma in a 72-year-old female patient. (a) Axial portal-venous-phase computed tomography shows unique hypoattenuating (20 Hounsfield Units) nodule of the right liver lobe, too small to be characterised. (b) Contrast-enhanced ultrasonography demonstrates an enhanced wall as a "rim" (arrows). (c-d) Metastatic pancreatic neuroendocrine tumour in a 60-year-old male patient. (c) Axial portal-venous phase computed tomography shows a lobulated hypodense cystic nodule with enhanced mural nodule and septas (arrow), very hyperintense on T2-weighted magnetic resonance imaging (d).

4. Infectious cystic lesions

4.1. Pyogenic abscess

Aetiopathogenesis- Frequent organisms of pyogenic abscesses are *Escherichia coli*, *Klebsiella pneumoniae*, *Enterococcus*, and *Streptococcus* (43). Hyper-virulent strains of *Klebsiella pneumoniae* have been responsible for severe emerging disease for the past two decades, particularly in Southeast Asia. Some of which are responsible for a

combining bacteremia, with multiple abscesses (monomicrobial liver abscesses, endophthalmitis, brain abscesses) (44).

The differential diagnosis of pyogenic abscess is the amoebaean abscess.

Imaging- Abscesses are rarely purely cystic and their appearance varies according to the pathologic stage. Prior to becoming cystic they display a pre-suppurative phase. Then they appear as multiple cystic nodules (honeycomb pattern), which coalesce in a unique large cystic cavity (cluster sign) with irregular shape surrounded by hypoattenuating inflammatory parenchyma (Fig 21). The cluster sign is specific to pyogenic abscess (43). The cystic central part appears at US as a large heterogenous cystic mass, with a mixture of anechoic, hypoechoic and hyperechoic parts (Fig 22). At CT and MRI, the rim and septa enhancement are described and the "double target" characteristic sign could be present. This sign is a hypoattenuating central pus area surrounded by an inner hyperattenuating ring (granulation tissue) and an outer hypoattenuating zone (inflammatory oedema) (34). Gas may be present (Fig 22). Central restriction of diffusion due to pus may be seen in larger abscess.

Appearances of abscesses can vary due to their pathogenesis: cholangitic abscesses are mostly small and multiples cystic nodules, unlike abscesses by hematogenous dissemination that are mostly larger and less numerous or abscesses by contiguous spread, commonly solitary (45).

Digestive venous thrombosis, particularly in the liver, is an additional feature to support the diagnosis of pyogenic abscess.

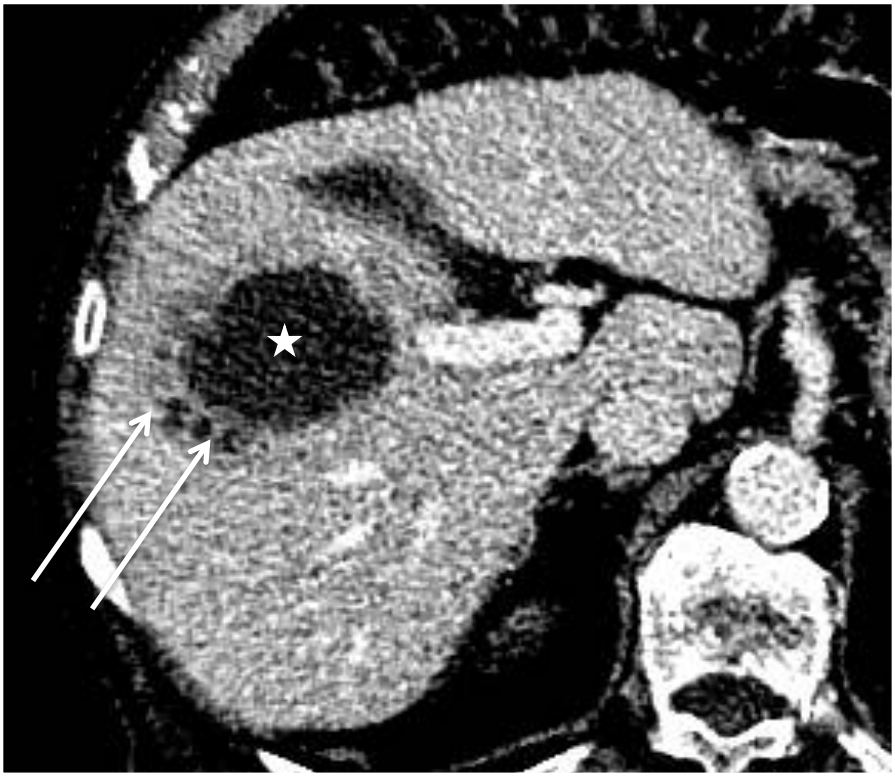


Figure 21: *Escherichia Coli* abscess in an 89-year-old male patient. Axial portal-venous-phase computed tomography shows three small hypoattenuating areas (arrows) corresponding to the initial lesion, which coalesce to form the large cavity (star).

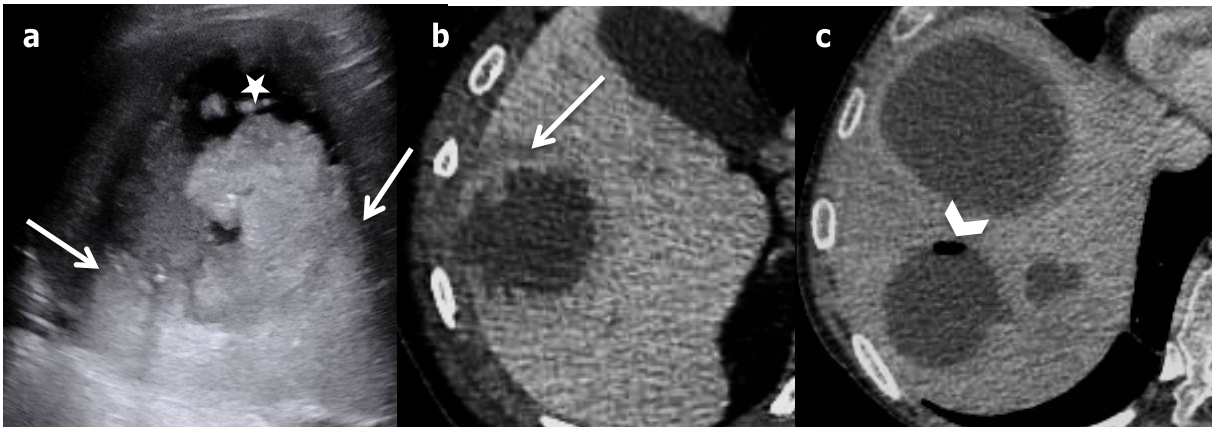


Figure 22: Hepatic pyogenic abscesses in a 49-year-old male patient. (a) Ultrasonography shows a large hypoechoic lesion, heterogeneous, with anechoic parts (star) and increased through-transmission (arrows) confirming its cystic nature**.** (b and c) Axial venous portal-phase computed tomography shows multiple abscesses appearing as cystic masses, with a specific sign: the double target (arrows). Note the bubble of gas in one of them (arrow-head).

4.2. Hydatid cyst

Aetiopathogenesis- Hydatid cysts result from *Echinococcus granulosis* infection (34). They have three layers: the outer pericyst, which corresponds with compressed and fibrosed liver tissue; the endocyst, an inner germinal layer; and the ectocyst, a thin, translucent interleaved membrane (46). The cysts are mainly asymptomatic but can become complicated (biliary fistula, compression, rupture) (47).

Imaging- The characteristic appearance varies, depending on the stage. For US, both the World Heath Organization (WHO) and the Gharbi classifications are used.

According to the WHO classification, there are seven categories, each depending on the stage of the disease. The first is called cystic lesion (CL). It is a unilocular anechoic lesion that appears as a hepatic simple cyst. Cyst echinococcosis (CE) 1 (Fig 23) corresponds to an active stage: it appears as a uniform anechoic cyst with internal echos ("hydatid sand") at US that may only be seen after patient repositioning. A peripheral hypointense enhanced capsule at MRI is visible (Fig 23). CE2 (active stage) (Fig 24) is as a well-defined multicystic mass with septa, with among half of the cases calcification of the cyst wall (47) better seen at CT. It appears multivesicular as a "honeycomb" due to daughter cysts surrounding a bigger cyst corresponding to the mother cyst. At CT, the attenuation of mother cyst is usually higher than those of daughter cysts because of debris (hydatid sand, and detached cysts wall); and the signal higher on T1-weighted and lower on T2-weighted at MRI (Fig 24). T2-weighted MRI sequences are more sensitive than CT to detect and characterise the cystic mass as typical wheel-spoke pattern (Fig 24). It exists septa in hyposignal on T2 due to the presence of calcifications and to the fibrotic component. They have a different signal on

T1-weighed images due to the cyst fluid that varies according to the amount of proteinaceous debris (34). The diffusion is not restricted (47). CE3 is a transitional stage. CE3A is a unique cyst with detached laminated membranes described as "water lily sign"(48). CE3B shows daughter cysts within a solid matrix. CE4 corresponds to an inactive stage. There are no more daughter cysts. It is a mixed hypoechoic and hyperchoic, resembling to a ball of wool (48). CE5 (inactive/degenerative stage) is partially or completely calcified.

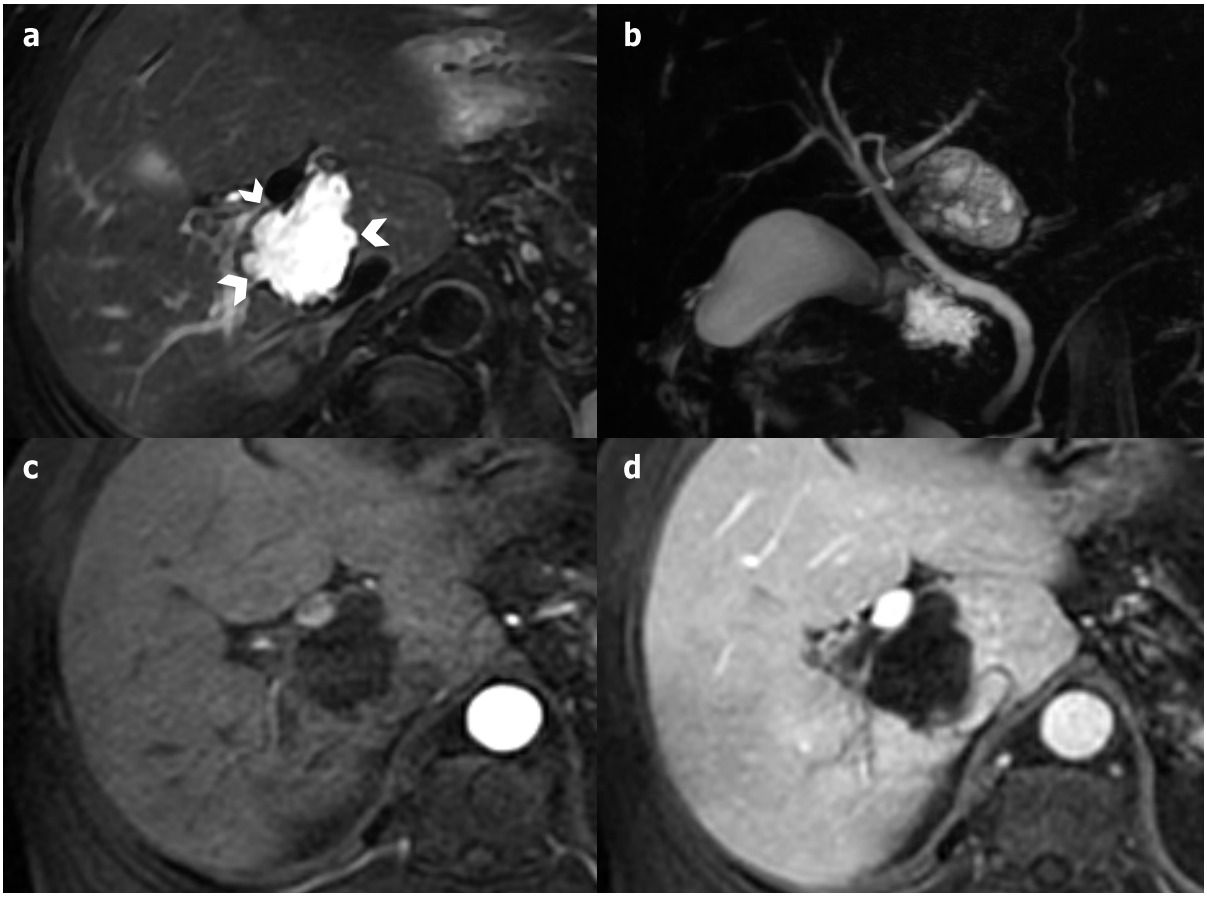


Figure 23: Cyst echinococcosis 1 in a 50-year-old female patient. (a) Axial T2-weighted magnetic resonance imaging shows a highly hyperintense lesion with lobulated margins that are typically hypointense. (b) Coronal 3D magnetic resonance cholangiopancreatography allows eliminating a biliary fistula. (c and d) Axial T1 fat-sat-weighted without and with contrast at portal-venous phase does not show any enhancement.

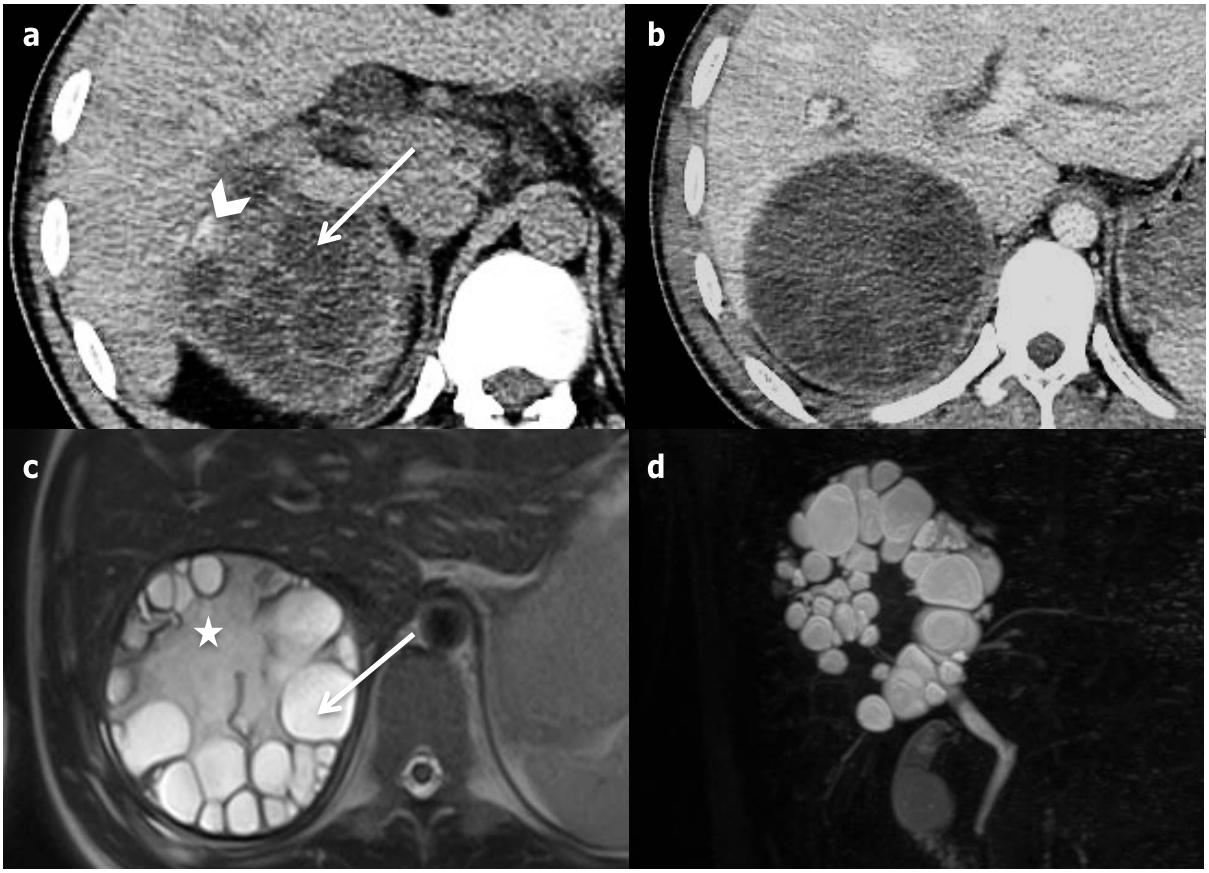


Figure 24: Cyst echinococcosis 2 in a 50-year-old male patient. (a) Axial non-enhanced computed tomography shows heterogeneous mass with cystic part (arrow) and calcification of the wall (arrowhead). (b) At axial portal-venous phase computed tomography there is no enhancement of the cystic component. (c) The cystic part and the differentiation between the daughter (arrow) and the mother (star) cysts are better evaluable at axial T2-weighted magnetic resonance imaging. (d) Coronal 3D magnetic resonance cholangiopancreatography eliminates biliary fistula.

4.3. Hepatic alveolar echinococcosis

Aetiopathogenesis- This is a rare parasitic infection resulting from *Echinococcus multilocularis*. In contrast to the hydatid cyst, a fibrous capsule does not usually demarcate the outer margin. It is a chronic and latent disease mostly found incidentally

(47). When symptomatic, the symptoms are related to compression. The treatment is medical and surgical. The prognosis is usually poor, sometimes leading to liver transplantation.

Imaging- Hepatic alveolar echinococcosis (HEA) lesions present irregular margins, and a mixed component of solid and cystic parts. There are frequently calcifications. The lack of enhancement is very helpful in forming a diagnosis.

At US, HEA lesions appear as a large heterogeneous mass, with areas of hyper- and hypoechogenicity (Fig 25), hyperechoic spots (corresponding to calcifications), and a central cystic anechoic cavity (due to necrosis) (47). More rarely they appear as multiple hyperechoic nodules with calcification and a cystic part. The CT helps to see calcification. Lesions are hypoattenuating, heterogeneous, with irregular margins, and calcification. Lesions may attract the surrounding parenchyma and deform the hepatic capsule. Usually, no enhancement is observed. At late phase, the fibrotic component may display a slender enhancement. MRI is essential prior to surgery. It helps to assess the diagnosis showing the multivesicular structure of the lesion, and allows the depiction of vascular or biliary tree involvement (47). The cystic part is in low to intermediate signal on T1-weighted images and high signal intensity on T2-weighted images; the fibrous part in low signal on T1 and T2-weighted images (Fig 25).

Therapeutic efficacy in HAE is essentially evaluated on the basis of repeated 18-F-FDG PET CT (49).

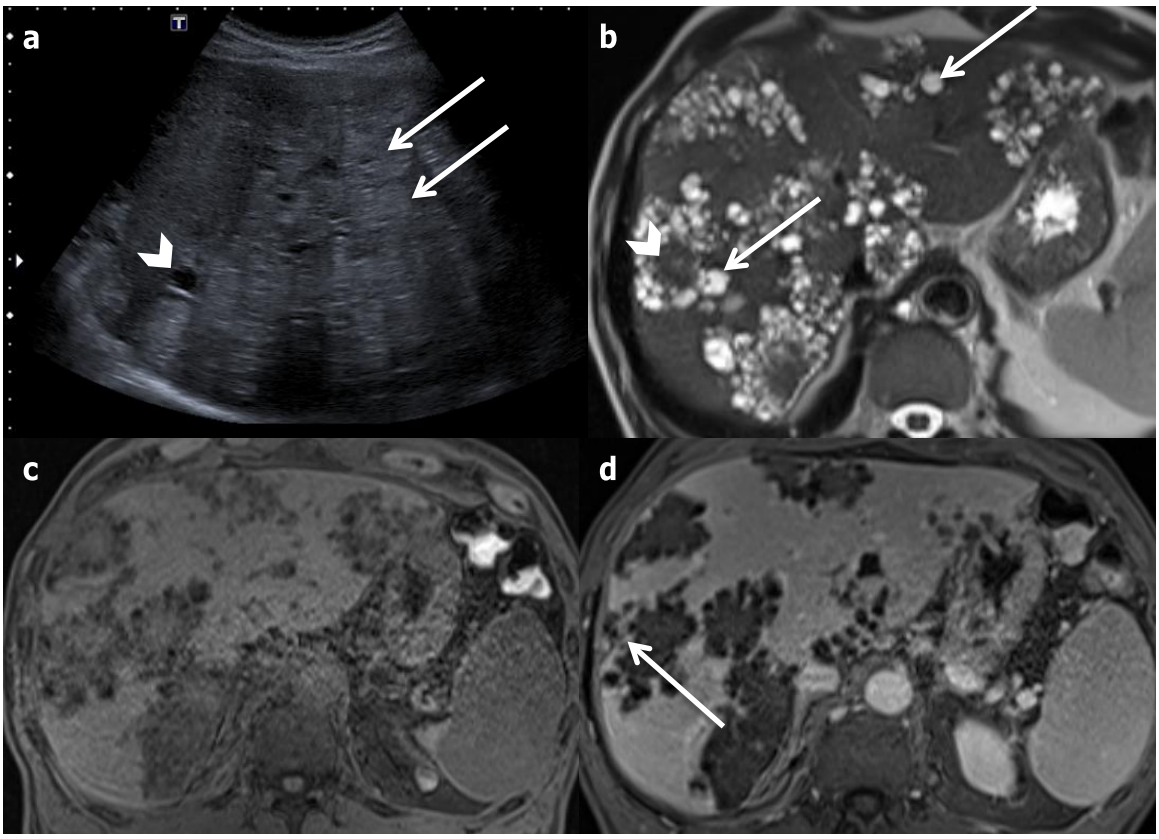


Figure 25: Hepatic alveolar echinococcosis in a 40-year-old male patient. (a) Ultrasonography shows multiple round hyperechoic nodules (arrows) with cystic anechoic parts (arrowhead). (b) At axial T2-weighted magnetic resonance imaging, the lesions appear as multivesicular cystic masses (arrows) with fibrous component (arrowhead). (c and d) Axial non-enhanced and at late-phase T1-fat-sat-weighted magnetic resonance imaging: the fibrous part is enhancing at late phase (arrow).

CONCLUSION

There is a wide spectrum of cystic liver lesions. To characterize cystic liver lesions, a particular attention should be paid to the wall thickness, the presence of enhanced septa or tissular components, the fluid signal on MRI and the clinical context. Cystic liver lesions may be malignant. Therefore, all non-HSC should be investigated to provide definitive diagnosis.

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CHENIN Mégane

Lésions kystiques hépatiques : revue iconographique

RÉSUMÉ

Les kystes hépatiques sont des lésions focales caractérisées par une composante liquidienne. Ils sont le plus souvent découverts fortuitement à l'imagerie. Les étiologies des kystes hépatiques sont nombreuses et variées, comprenant des maladies fréquentes et rares, tumorales et non tumorales.

Les kystes hépatiques simples sont fréquents et ne nécessitent aucun traitement ni suivi. Ils ne présentent aucune caractéristique atypique à l'imagerie. En revanche, les kystes hépatiques atypiques, ne présentant pas tous les critères de bénignité, nécessitent d'être repérés afin de ne pas méconnaître une lésion à risque d'évolution défavorable. Par conséquent, la connaissance des caractéristiques spécifiques de chaque lésion kystique hépatique aide le radiologue à tendre vers un diagnostic précis et non invasif.

L'objectif de cette revue est de décrire les caractéristiques en imagerie des différentes lésions kystiques hépatiques.

Mots-clés : kyste, hépatique, imagerie, échographie, tomodensitométrie, imagerie par résonance magnétique

Cystic liver lesions: a pictorial review

ABSTRACT

Hepatic cysts are focal lesions characterised by a cystic component, mostly found incidentally by imaging. This imaging pattern may be observed in a wide spectrum of common and uncommon non-neoplastic or neoplastic diseases.

Hepatic simple cyst is frequent and do not require any treatment nor follow up, but non-typical cysts should be carefully analysed to do not misdiagnosis lesion needing an appropriate management. Therefore the knowledge of each disease imaging patterns helps in accurate diagnosis.

The aim of this review is to describe imaging feature of the different hepatic cystic lesions.

Keywords: cyst, hepatic, liver, imaging, ultrasonography, computed tomography, magnetic resonance imaging

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