REVIEW ARTICLE

Hepatic Infections: A Comprehensive Imaging Review

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Abstract

Hepatic infections are common and represent a heterogeneous group of disorders with varying severity. Patients with clinically suspected hepatic infections often undergo imaging evaluation. Imaging allows confirmation when the findings are typical. However, when the imaging appearances are atypical, imaging in conjunction with clinical and laboratory data allows narrowing the differential diagnoses. Further, image-guided sampling helps confirm diagnosis in certain situations. In this review, we describe the imaging appearance of various hepatic infections.

Keywords: Hepatic infections, Hydatid cyst, Imaging, Liver abscess.

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Introduction

Infections of the liver include bacterial, viral, fungal, mycobacterial, and parasitic involvement of the liver parenchyma. These infections may present with vague constitutional symptoms and pain abdomen and are usually identified incidentally during an abdominal ultrasound. Hepatotropic viruses often involve the liver; however, the clinical and radiological manifestations are, in most cases, a consequence of the body's immune response to the infected hepatocytes.

While imaging is an excellent tool for identification of liver lesions, characterization of these lesions is done only after careful consideration of the clinical details of the patient. Radiological appearances of most hepatic infections are nonspecific, thereby often requiring serological and histological confirmation. The most common differentials are neoplasms and cysts. Of late, there has been a paradigm shift in the clinical management of many common conditions. Liver abscesses and hydatid cysts are now being drained by minimally invasive procedures by interventional radiologists.

In this review, hepatic infections have been systematically discussed based on their etiology.

Liver Abscess

Liver abscess refers to a collection of suppurative material within the hepatic parenchyma. This may be a consequence of bacterial, parasitic, fungal, or granulomatous infection. There is a geographic and demographic divide in the etiological factors responsible for liver abscesses. In the developed countries, pyogenic abscesses are more common, while in the developing and underdeveloped regions, amebic abscesses form the majority.[1,2] Abscesses involve the right lobe in about two-third of cases, probably in accordance with the proportion of portal blood received by the right lobe. Up to a fifth of the patients may have multiple abscesses.[3]

Amebic and pyogenic abscesses may be indistinguishable on clinical and imaging features. However, they differ in terms of epidemiology as well as medical management. Amebic liver abscess is commonly seen in the 3rd and 4th decades, with a striking male preponderance (M:F ratio 4–10:1).[1,4,5] Most cases either reside in or have a history of recent travel to a tropical, endemic region. The imaging findings of amebic abscess have been discussed later.

Pyogenic abscesses, on the contrary, are usually seen in the 5th or 6th decade, with no gender predilection.[6,7] These are commonly polymicrobial, with *Escherichia coli* and *Klebsiella* being the most common causes. Intra-abdominal infections like appendicitis and diverticulitis used to be the most common etiological factors responsible for pyogenic liver abscesses in the pre-antibiotic days. However, early diagnosis of these conditions and timely institution of antimicrobials has led to a decline in these numbers. Biliary diseases and hepatic interventions are often the likely source in recent studies; however, no clear source is determined in most of the cases.[8]

Clinically, the patients usually present with right-sided abdominal pain, fever, and rigors. Biochemical alterations are nonspecific with mild to moderate elevation of alkaline phosphatase, modest hyperbilirubinemia, and raised total leukocyte count. Persistence of elevated bilirubin should alert the radiologist to look for an evidence of biliary obstruction.

Imaging Modalities

Ultrasound

An abdominal ultrasound is the first modality to be used in case of suspected liver abscess. Most cases are detected while workup for abdominal pain, fever, or constitutional symptoms. Abscesses are discrete hypoechoic lesions with acoustic through transmission. There may be presence of internal echoes (Figs 1A and B), or air within the cavity (Fig. 1C) seen as echogenic foci with reverberation

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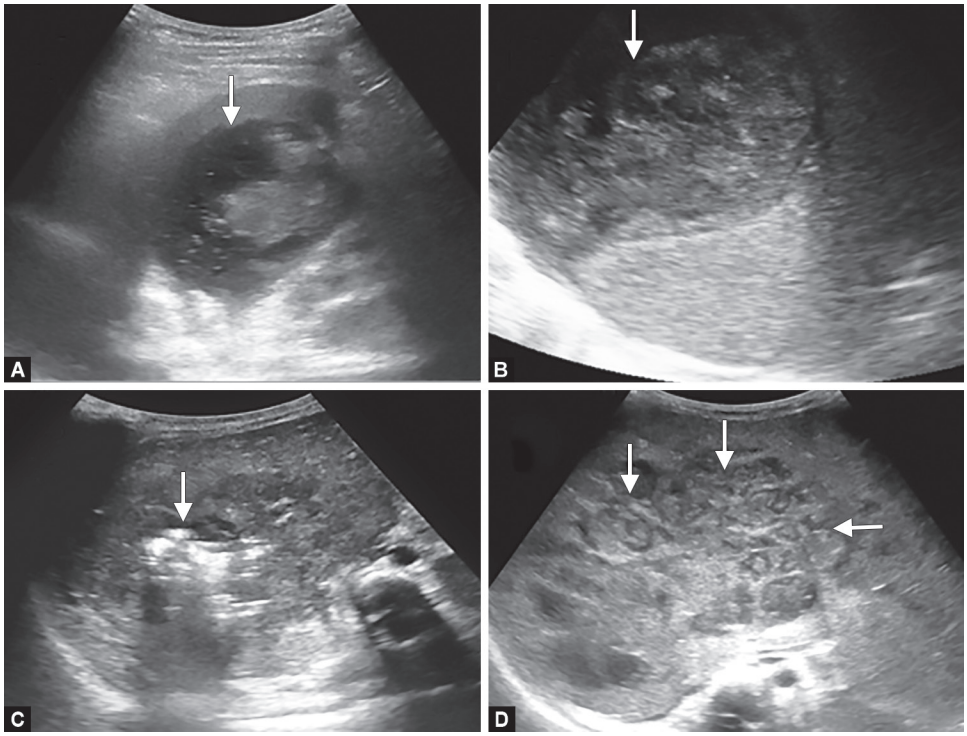


Figs 1A to D: Pyogenic liver abscess—USG: (A and B, arrows) Lesion with internal echoes; (C, arrow) Abscess with air-foci seen as echogenic foci with "dirty" acoustic shadowing; (D, arrow) Abscess seen as area of altered liver echotexture

artifacts.[9] Less commonly they present as ill-defined areas of distorted parenchymal echogenicity (Fig. 1D). Larger abscess may be hypoechoic or hyperechoic. Ultrasound has a sensitivity of 70–90% for detecting liver abscess.[10] Subdiaphragmatic lesions of the posterior segment may sometimes be difficult to visualize and may therefore be missed, especially in those with fatty liver. Therefore, in cases with high index of suspicion, and when the whole liver could not be adequately visualized on ultrasonography, a contrast-enhanced computed tomography (CECT) may be warranted.

Contrast-enhanced CT

With a sensitivity of over 95%, a CECT is an excellent investigation for liver abscess.[10] An additional advantage of CT is that it facilitates planning of percutaneous interventions in complex cases. A microabscess on CECT scan is typically a well-defined hypodense lesion with faint rim enhancement and perilesional edema (Fig. 2A). When multiple, small adjacent abscesses may coalesce to aggregate into a single larger cavity described as the cluster sign (Fig. 2B). Larger abscesses may range from smooth walled unilocular to complex multilocular septated lesions. They may also appear as phlegmonous masses. The classical double target rim enhancement is seen on dynamic contrast-enhanced imaging with the inner rim showing an early persistent enhancement and the outer edema showing delayed enhancement (Fig. 2C). Air within the abscess cavity (Fig. 2D) may be seen in the form of an air-fluid level or small bubbles but is uncommonly encountered.[11,12] Transient segmental enhancement of the hepatic parenchyma in arterial phase scans may be seen in patients with large vascular abscesses.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is not routinely required for the diagnosis of liver abscesses but may be invaluable in cases with suspected biliary involvement. Magnetic resonance cholangiopancreatography (MRCP) sequence can help define and characterize any biliary obstruction because of the liver abscess. Abscesses are usually T1 hypointense and T2 hyperintense lesions (Fig. 3A). However, their signal intensities are largely dependent upon the protein content. Mild T2 hyperintensity may be seen surrounding the abscess representing edema. The double target inflammatory rim showing dynamic enhancement characteristics may be seen with gadolinium enhanced MRI as seen on CT (Fig. 3B). Marked central diffusion restriction on diffusion weighted imaging (DWI) is a useful feature that can sometimes help differentiate it from neoplasms where marked restriction favors an abscess over neoplasm.

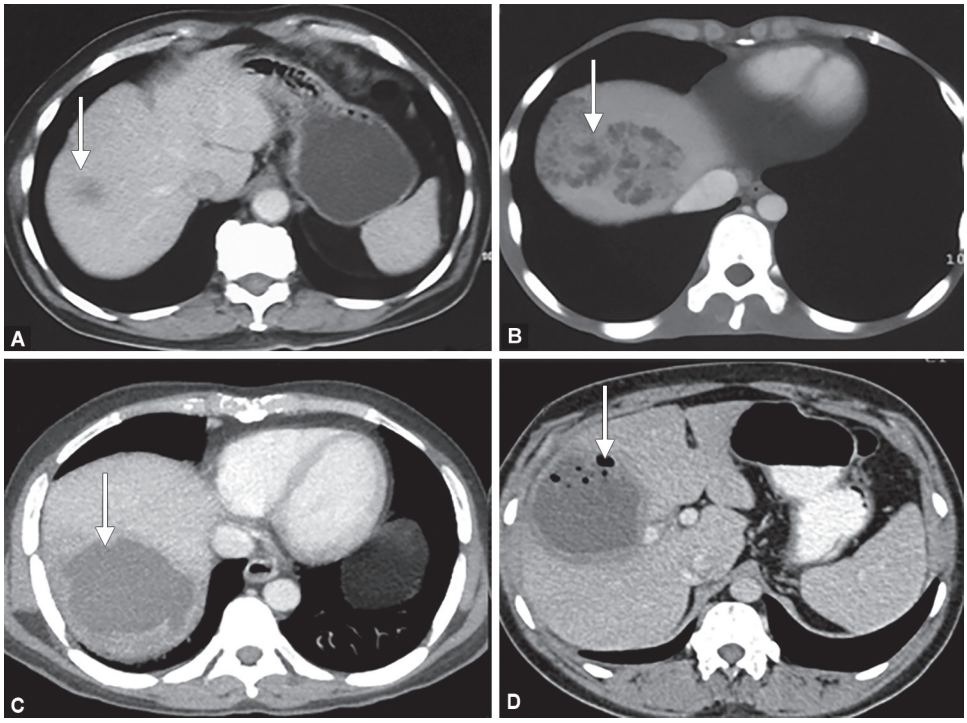
Fluorodeoxyglucose Positron Emission Tomography

A biomarker for glycolytic metabolism, any metabolically active lesion can demonstrate high uptake on fluorodeoxyglucose-positron emission tomography (FDG-PET). PET can be used as a problem-solving tool in patients with nondiagnostic imaging. For example, polycystic liver disease may be complicated by bacterial superinfection. However, it may be extremely difficult to differentiate from hemorrhage in the cyst based upon routine radiological investigations. In such cases, an FDG-PET can indicate presence of metabolic activity in an infected cyst.

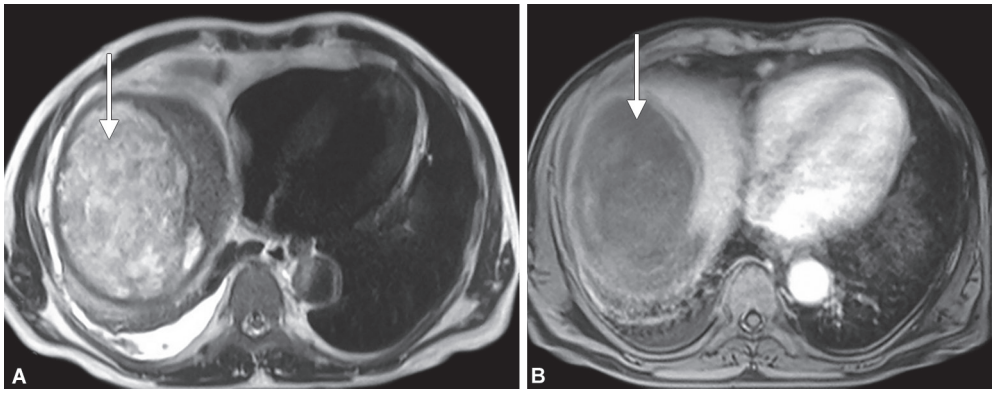
Differential Diagnosis and Imaging Pitfalls

Although the imaging findings in abscesses are nonspecific and various focal pathologies including neoplasms and complicated cysts may mimic them, the common differentials include the following:

- Neoplasms: A phlegmonous abscess may be difficult to differentiate from a necrotic neoplasm. Likewise, a necrotic mass may mimic a liquefied abscess. Unlike abscesses, neoplasms seldom show the layered appearance, segmental enhancement,



Figs 2A to D: Pyogenic liver abscess—CT: (A, arrows) Microabscess seen as a well-defined hypodense lesion with perilesional edema; (B, arrow) Cluster sign; (C, arrow) Larger abscess with inner liquefied component and wall showing two rims (double-target appearance); (D, arrow) Air-foci in pyogenic liver abscess



Figs 3A and B: Pyogenic liver abscess—MRI: (A, arrow) T2W MR image shows a large abscess showing heterogeneous hyperintensity; (B, arrow) T1W post-contrast image shows peripheral rim enhancement

perifocal edema, and central diffusion restriction. Other ancillary findings specific to tumor type can help differentiate them from abscesses (Fig. 4).

- Tubercular abscess: Discussed in the section hepatic tuberculosis.
- Complicated hydatid cyst: Discussed in the section hydatid disease.
- Fungal microabscesses: Discussed in the section fungal infection.
- Cysts: Lack of surrounding edema, diffusion restriction, and rim enhancement differentiate cysts from abscesses (Fig. 5).
- Biliary hamartomas: Although hepatic hamartomas may show enhancing rims to mimic abscesses, they do not show perilesional edema or diffusion restriction.

Key imaging features

Although differentiating an abscess from a neoplasm may be extremely challenging at times, the key imaging features include the typical enhancement kinetics of an inflammatory capsule with a layered appearance, transient segmental parenchymal enhancement, and restriction of diffusion within the lesion on DWI.

Tubercular Disease

Hepatobiliary tuberculosis is uncommon, accounting for <1% of all tubercular infections. Sources of hepatobiliary tuberculosis include hematogenous dissemination via hepatic artery from lungs or via the portal vein from the gastrointestinal tract, ascent along the common bile duct, or secondary infection form periportal adenitis. Spleen is more commonly involved in patients with HIV infection.[13]

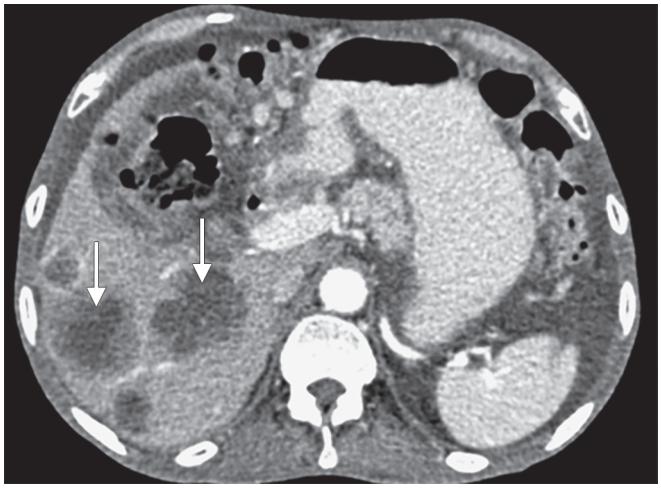


Fig. 4: Pyogenic abscesses: differential diagnosis (metastases). CT image shows multiple well-defined hypodense lesions in a patient with gastrointestinal stromal tumors (GIST) (arrows)

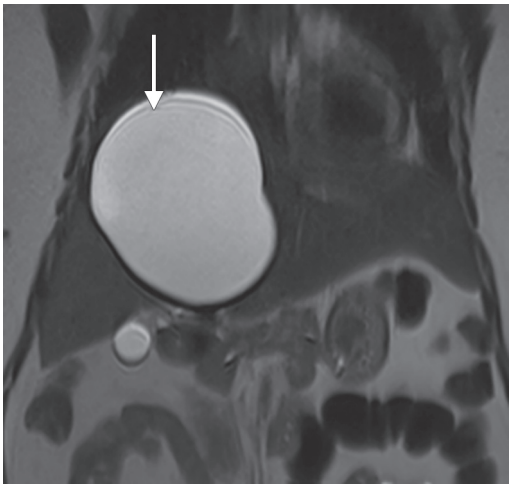


Fig. 5: Pyogenic abscesses: differential diagnosis (simple cyst). T2W MR image shows a large well-defined homogeneously hyperintense mass with no appreciable wall (arrow)

Hepatobiliary tuberculosis may present with hepatic, pancreatic, gallbladder, or biliary involvement.[14]

Hepatic tuberculosis may present in two forms. Hematogenous dissemination of tuberculosis to the liver presents as its micronodular form with tiny (<2 mm) nodules that calcify on healing. The macronodular hepatic tuberculosis presents as focal granulomas (usually 1–3 cm in size) or frank abscesses. It is less common than its miliary counterpart.[14] Tuberculomas often progress to abscesses resulting from liquefaction of caseous necrosis. By virtue of their mass effect, they may cause focal biliary dilatation.

Imaging

Ultrasonography

Miliary tuberculosis may present as hepatomegaly with coarsening of hepatic echotexture (Fig. 6A). Generally, a tuberculoma appears as a poorly defined hypoechoic mass (Figs 6B and C). At times, it may be entirely hyperechoic.[15,16] A tubercular abscess is a complex appearing lesion (Fig. 6D) that may not have a distinct wall at all, may have a circumferential hyperechoic wall, or may appear as a solid lesion (Fig. 6E).

CT

Hepatic tuberculosis may present as multiple hypodense nodules with or without peripheral enhancement (Figs 7A and B), a single hypodense mass with central hypoattenuation, a conglomerate mass with honeycomb appearance, or a fluid collection with enhancing thick walls (Fig. 7C). With multifocal disease, the nodules may have varying attenuation representing disease in different stages of progression.[17]

MRI

On MRI, miliary hepatic tuberculosis may be seen as diffusely T2 hyperintense parenchyma. The tuberculosis nodules are usually T1 hypointense. On T2WI, they are commonly iso to hyperintense with a less hyperintense rim.[18] They may also be T2 hypointense due to free radical production. Multifocal nodules may be seen on MRI having varying T2 signal intensity. On DWI, tuberculomas exhibit restricted diffusion. On contrast administration, peripheral enhancement is seen.[18] Abscesses are T1 hypointense and T2 hyperintense. An unusual presentation with a perihepatic collection associated with intrahepatic tuberculoma may also be seen. Tubercular cholangitis is an uncommon presentation. It presents as asymmetrical or segmental dilatation of bile ducts due to small and duct intrahepatic duct strictures, ductal thickening, ductal/periductal calcification, and periportal necrotic or calcified lymph nodes.

Differential Diagnosis and Imaging Pitfalls

Miliary hepatic tuberculosis closely mimics lesions of fungal infection, *Pneumocystic jiroveci* infection and sarcoidosis. The clinical profile of patients with the former two closely matches that of those with tuberculosis making diagnosis challenging and histology necessary. When differentiating tuberculosis from sarcoidosis, MRI plays an important role. On T1WI, the caseating granulomas in tuberculosis have a varying signal and show no enhancement, or sometimes peripheral enhancement in comparison to noncaseating granulomas in sarcoidosis which have intermediate signals on T1WI with enhancement that may persist on delayed images.

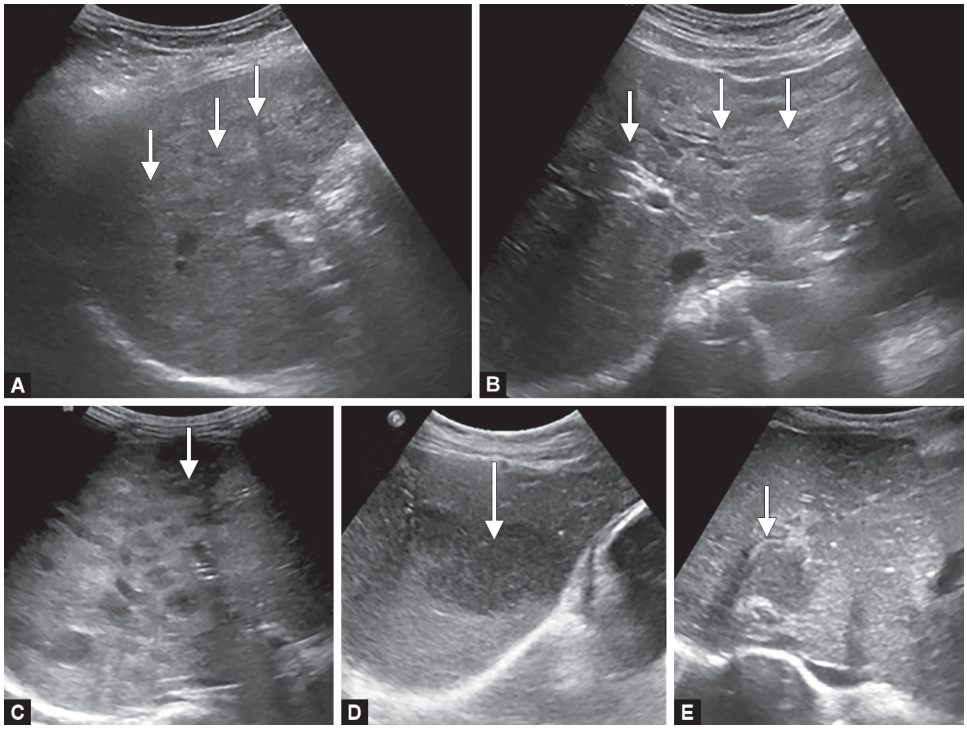
Other less common differentials include lymphoma, metastases, brucellosis, and coccidioidomycosis. The presence of necrotic lymph nodes and splenic involvement favors tuberculosis over all others.

Pyogenic or amebic abscesses may not be differentiable from tubercular abscesses on imaging. Lack of response to antibiotics and percutaneous drainage should raise suspicion for tubercular etiology.

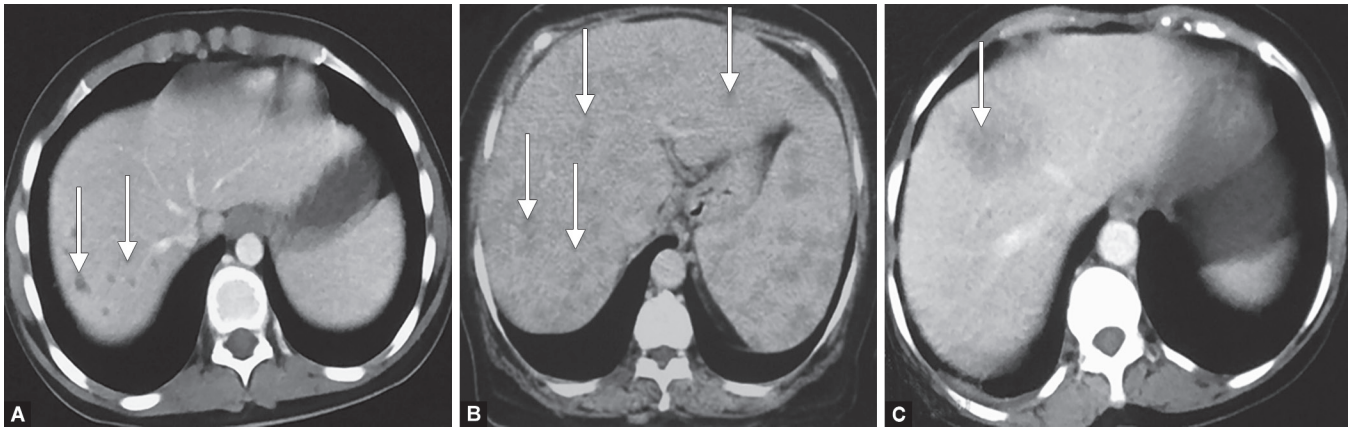
Neoplasms

Necrotic tumors including hepatocellular, cholangiocellular carcinoma, and metastases may mimic tuberculomas. No or minimal peripheral enhancement can differentiate tuberculomas from neoplasms.[19–21]

Key imaging features
Hepatic tuberculosis presents with a myriad of imaging features, often indistinguishable from fungal infections and neoplasia. Correlation with epidemiology and clinical presentation is of paramount importance. Tissue sampling and nucleic acid amplification test aid diagnosis.



Figs 6A to E: Hepatic tuberculosis—USG: (A) Coarsening of liver echotexture; (B and C, arrows) Poorly defined hypoechoic lesions; (D, arrow) Tubercular abscess seen as a well-defined hypoechoic lesion; (E, arrow) Solid-appearing lesion



Figs 7A to C: Hepatic tuberculosis—CT: (A and B, arrows) Multiple nodules; (C, arrow) Solitary mass-like lesion

Viral Hepatitis

The commonest viruses to infect the liver are hepatotropic viruses like hepatitis A, B, C, and E, herpes simplex virus, HIV, and coxsackie virus. The imaging findings of acute viral hepatitis are essentially nonspecific. The role of radiology is to rule out other causes of similar biochemical and clinical picture. HIV frequently affects liver and biliary tracts. Several opportunistic viruses, bacteria, and fungi can primarily affect the liver in cases of disseminated disease.

Imaging

Ultrasonography

Acute hepatitis appears as hepatomegaly and decreased parenchymal echogenicity with prominent portal tracts described classically as the starry sky appearance. The parenchymal echotexture may be completely normal. Associated gallbladder wall edema with luminal collapse is noted. Chronic hepatitis may have normal appearing liver, coarsened echotexture, or changes of frank cirrhosis. A cirrhotic liver demonstrates nodular parenchyma and surface contour. There may be redistribution of volume with enlargement of the caudate lobe and lateral segments of the left lobe of liver.

CT

The CT findings in viral hepatitis are nonspecific. Heterogeneous enhancement of liver with periportal edema is the usual findings on CT. There may sometimes be well-defined areas of hypoattenuation.[22] When complicated by portal hypertension, prominent portosystemic collateral channels may be seen in combination with ascites and splenomegaly. At times, the only finding on CT in acute or chronic hepatitis may be periportal adenopathy.[23]

MRI

MRI is not routinely used to investigate hepatitis. When imaged, an acutely inflamed liver may demonstrate diffuse periportal hyperintensity on T2WI representing periportal edema. Involved parenchyma may appear as ill-defined areas of T1 hypointensity and T2 hyperintensity.[24]

MRCP

HIV cholangiopathy may manifest in four patterns: papillary stenosis with upstream dilatation of the biliary tree, sclerosing cholangitis, combined papillary stenosis and sclerosing cholangitis, and long bile duct stricture.

Differential Diagnosis and Imaging Pitfalls

Hepatomegaly is a nonspecific finding and may be seen in several liver specific or systemic diseases. An edematous gallbladder may mimic acute cholecystitis. A collapsed gallbladder lumen favors reactionary edema of hepatitis over cholecystitis. Chronic hepatitis and cirrhosis will need differentiation from other causes of liver parenchymal disease.

Key imaging features

While ultrasound is the imaging modality of choice in acute viral hepatitis, the imaging features remain nonspecific and diagnosis is made in conjunction with liver function tests and serology. An enlarged liver with echogenic portal tracts against a hypoechoic parenchymal background gives the proverbial yet nonspecific starry-sky appearance. Concentric gallbladder edema is also seen.

Parasitic Infestation

Amebiasis

Etiopathogenesis

Caused by *Entamoeba histolytica*, hepatic amebiasis is more common in males. Amebic trophozoites migrate up the portal vein to invade hepatic parenchyma. Patients with amebic abscesses are sicker and younger than those with pyogenic abscesses.[25]

Imaging

The amebic abscess is classically unilocular and solitary. It is usually subcapsular in location and has a tendency for diaphragmatic disruption.

Ultrasonography: On ultrasonography, the abscess appears as a hypoechoic lesion with low-level internal echoes within the abscess cavity and no significant wall echoes (Fig. 8). Although amebic abscesses cannot be differentiated from pyogenic abscesses with certainty on imaging, a suggestive pattern of five sonographic signs has been described for amebic abscesses: (a) round or oval shape, (b) absence of wall echoes, (c) homogeneous low-level internal echoes, (d) acoustic through transmission, and (e) contiguity with hepatic capsule.[26]

CT: On CECT, characteristics of an amebic abscess are like those of a pyogenic abscess and appear as well-defined hypoattenuating rounded lesions (Fig. 9). They have thick enhancing walls with perilesional edema. The cavity may have septae, debris level, air, or hemorrhage. Often, there is associated evidence of colitis with circumferential mural thickening and edema involving the cecum and proximal ascending colon.

MRI: Homogeneous hypointensity on T1WI and hyperintensity on T2WI is noted within the abscess cavity. A peripheral zone of T2 hyperintensity denoting edema may be seen.[23] Central diffusion restriction is seen in abscesses.

Differential Diagnosis

- Pyogenic abscess
- Complicated hydatid cyst

Key imaging features

An amebic abscess simulates a pyogenic abscess on imaging. A complex fluid-attenuation lesion with "target" appearance of the enhancing capsule is typical on CT. In addition, these abscesses have a higher predisposition for extrahepatic extension.

Hydatid Disease

Echinococcus granulosus and *Echinococcus multilocularis* are the causative organisms for hepatic echinococcosis with cystic and alveolar echinococcosis being the commonest forms. Liver is the most commonly affected organ with a propensity for right lobar involvement. The cysts grow up to approximately 1 cm in size in 6 months and then progress at a rate of 2–3 cm per year.[27]

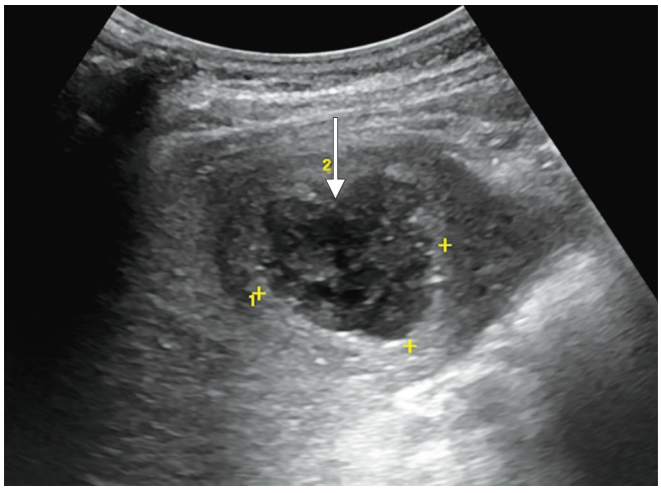


Fig. 8: Amebic liver abscess—USG. There is a well-defined lesion with thick wall and internal echoes (arrow)

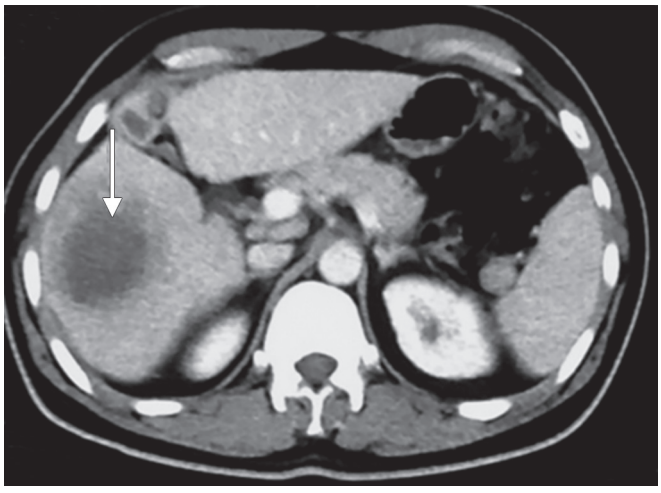
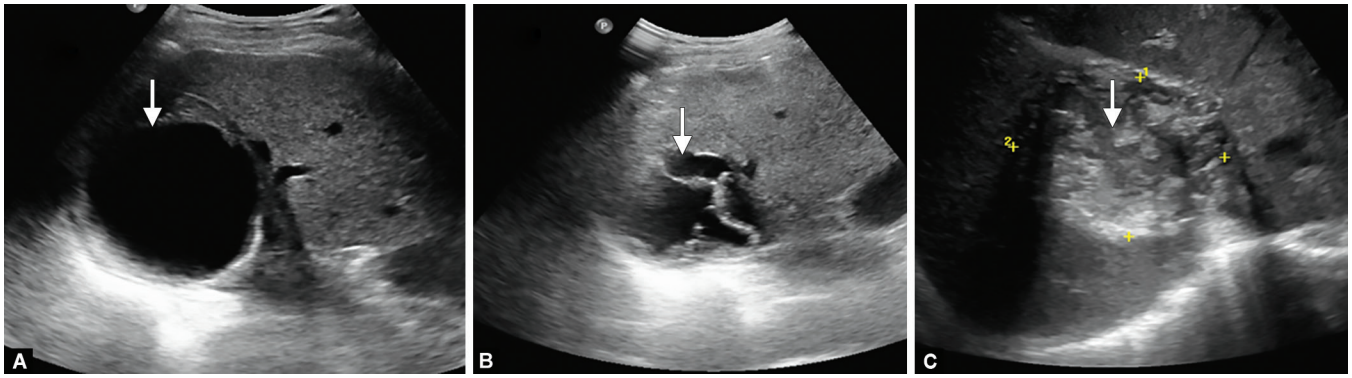
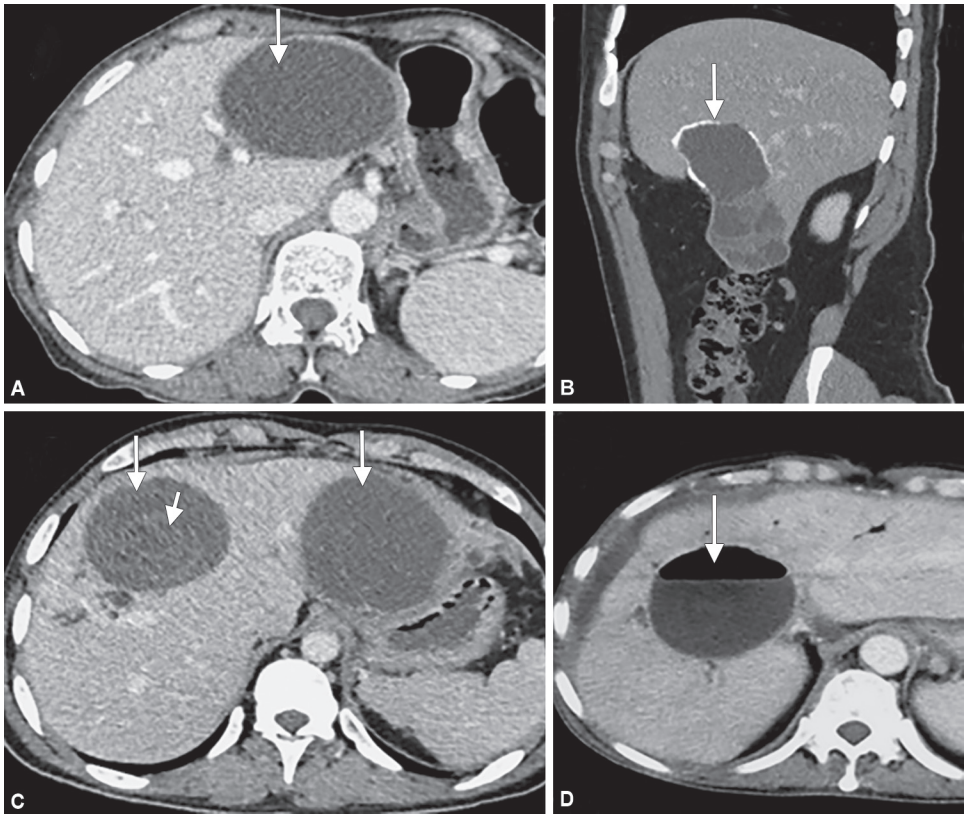


Fig. 9: Amebic liver abscess—CT. A well-defined hypodense lesion with thick wall is seen in segment V and VI



Figs 10A to C: Hydatid cyst—USG: (A) CE type I lesion; (B) CE type III lesion; (C) CE type IV lesion



Figs 11A to D: Hydatid cyst—CT: (A, arrow) Well-defined unilocular cyst; (B, arrow) Cyst with daughter cyst; (C, arrow) Cyst with detached membranes; (D, arrow) Cyst with air-fluid level

Structurally, a hydatid cyst has three layers. These include the innermost live germinal endocyst, the secreted multilamellated ectocyst, and the fibrous outer pericyst. Daughter cysts mark viability of the cyst whereas dense cyst wall calcification is a marker for nonviability. Alveolar echinococcosis has an infiltrative appearance and a propensity to involve the porta. When the portal vein itself is compressed, it leads to lobar atrophy. It does not form membranes or cysts. Hepatic alveolar echinococcosis (HAE) commonly causes compression of intrahepatic biliary radicals, portal veins, and hepatic veins with resultant portal hypertension.

Imaging

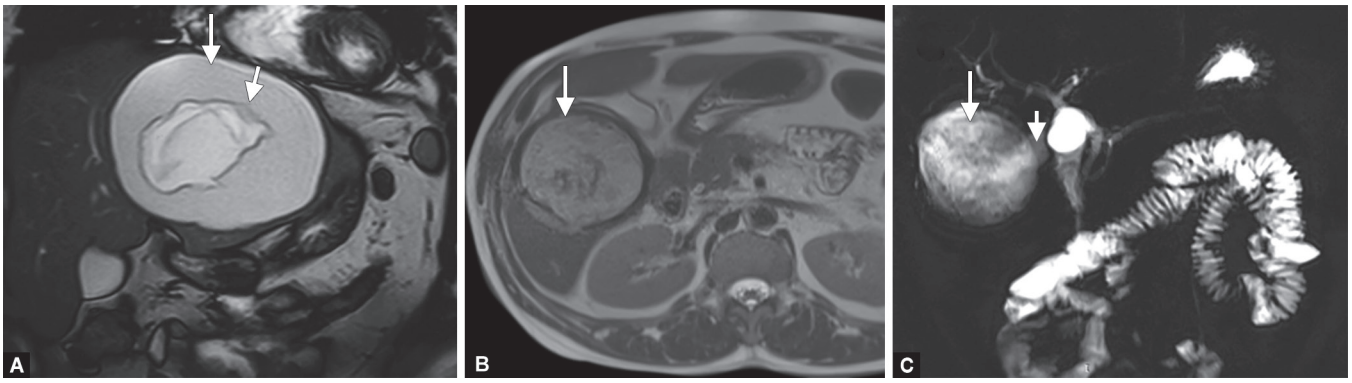
Ultrasonography: Based on growth, WHO has designated six stages of progression of hydatid cysts (Table 1 and Fig. 10).[28]

CT: Like ultrasound findings, based on stage, the imaging characteristics vary (Fig.11). The daughter cysts usually have a lower attenuation than the main cyst (Figs 11B and C). There may be air or fat within indicating superadded infection or communication (Fig. 11D). HAE presents as a heterogeneous infiltrative mass that does not enhance centrally. Clustered microcalcification or dense focal calcifications may be seen.

MRI: The pericyst is well delineated by MRI as a T1 and T2 hypointense rim. The daughter cysts are more T2 hyperintense than the mother cyst. When collapsed, the membranes appear hypointense on all sequences (Figs 12A and B) and this is called serpent sign.[29] Irregularity of cyst wall is better detected on MRI and represents loss of viability of cyst.[30] On administration of

Table 1: WHO classification of hydatid cyst

Stage	*Imaging*	*Sign*	*Viability*
CL	Clear unilocular cyst	–	Active
CE type I	Defined cyst wall with fine echoes that move with change of position (Fig. 11A)	Falling snowflakes sign	Active
CE type II	Multivesicular cyst with daughter cysts	Racemose or spoke wheel appearance	Active
CE type III	• 3A-Detached floating membrane within (Fig. 11B) • 3B -Cysts have a solid matrix	Water-lily sign	Transitional
CE type IV	Heterogeneous cyst with hypoechoic and hyperechoic contents (Fig. 11C)	Pseudotumor sign or ball of wool sign	Mostly inactive
CE type V	Partial or complete calcification of cyst wall and its contents	–	Nonviable



Figs 12A to C: Hydatid cyst—MRI: (A and B, arrows) Cyst with detached membrane; (C, arrow) Cyst with biliary communication (short arrow, C)

gadolinium, the cyst fluid and the septae do not enhance. The pericyst, being fibrous, may enhance on delayed scans.

Complications

- Rupture: A cyst may show communicating or contained rupture or may directly rupture into the surrounding anatomical structures including viscera, thorax, pancreatobiliary ducts (Fig. 12C), peritoneum, or skin.[31]
- Infection: An infected hydatid is always secondary to rupture. When infected, hydatid cysts enhance on contrast administration, making diagnosis difficult. They appear poorly defined and may have intracavitary air-fluid level.

Differential Diagnosis and Imaging Pitfalls

Simple hepatic cyst: Differentiating a simple cyst from a clear unilocular (CL) stage hydatid is difficult.

Cystadenoma or cystadenocarcinoma: Unlike hydatid cysts, the septae and nodules in cystadenomas and cystadenocarcinomas enhance on contrast administration (Fig. 13).

Abscess: An infected hydatid cyst mimics abscesses. Hydatid serology can help make the diagnosis.

Complicated hepatic cyst: A cyst complicated by hemorrhage appears hyperintense on T1WI. HAE can be mistaken for a neoplasm; however, the absence of enhancement differentiates it.[32]

Key imaging features

The imaging features correspond to the stage of cyst transition. While a CL cyst may be indistinguishable from a simple cyst, demonstration of daughter cysts on ultrasonography or MRI is diagnostic of hydatid disease. On contrast-enhanced imaging, the pericyst enhances avidly while the internal sepatations do not.

Visceral Larva Migrans and Eosinophilic Abscess

Caused by *Toxocara canis*, visceral larva migrans (VLM) can present as variable sized hypoechoic to isoechoic, round or oval lesions with or without a defined margin (Fig. 14).[33] On CECT, there may be multiple oval or elongated hypoattenuating lesions (Figs 15A and B) that may change locations on subsequent imaging indicating migration of larvae.[34] Metastasis is the most important differential diagnosis. Features including fuzzy margins, lack of peripheral venous phase enhancement, and subtle hypodensity favor larva migrans. MRI findings correspond to the changes seen on CT and include ill-defined lesions showing T1W hypo- and T2W heterogeneous hyperintense signal (Fig. 16). Patients usually have peripheral eosinophilia and may present with fever, abdominal discomfort, and nausea.

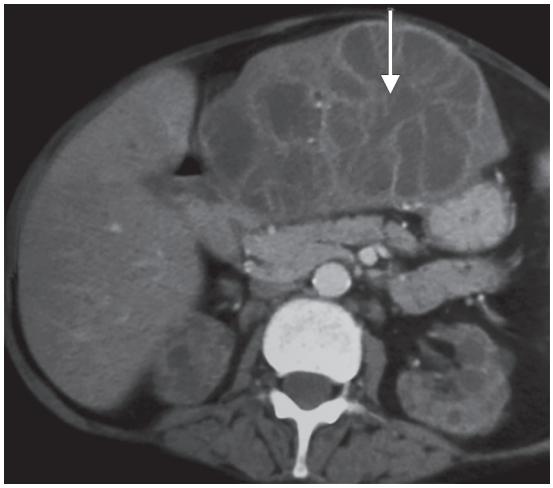


Fig. 13: Hydatid cyst—differential diagnosis (biliary cystadenoma). CT image shows a cystic lesion with thick enhancing septations (arrow)

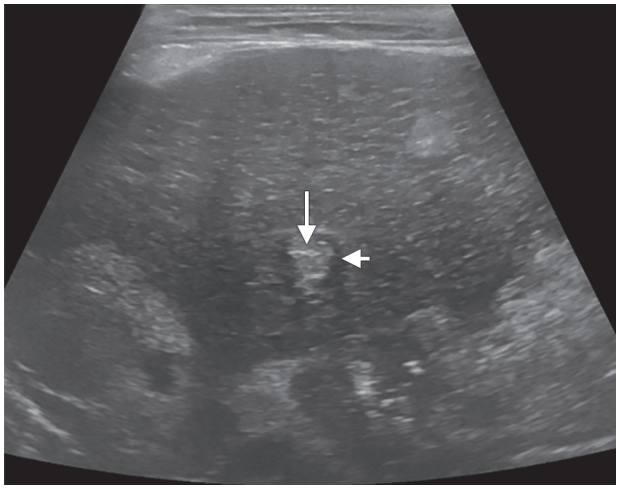
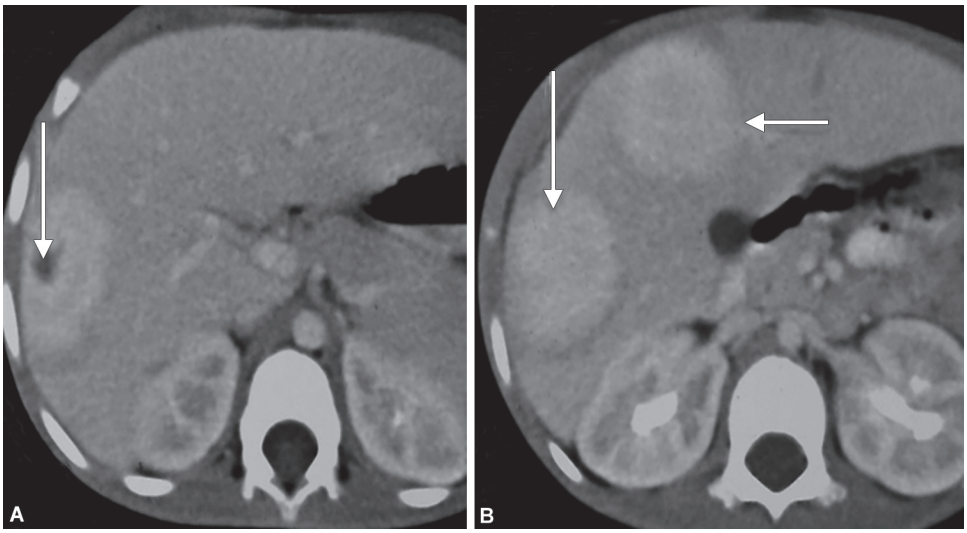


Fig. 14: Fungal infection—USG: Multiple well-defined echogenic lesions (arrow) with hypoechoic rim (short arrow) in a child with candidiasis



Figs 15A and B: Fungal infection—CT: (A, arrow) Well-defined enhancing lesion with hypodense center; (B, arrow) Multiple lesions with homogeneous enhancement in a patient with histoplasmosis

Other Parasitic Infections

Ascariasis

This results from migration of the worm through the ampulla of Vater into the biliary tree and the gallbladder. This incited inflammation resulting in cholangitis, besides causing mechanical obstruction of the common bile duct and ampulla causing pancreatitis. Ultrasonography reveals the worm as a nonshadowing tubular echogenic structure within the bile duct. Anechoic central line representing gastrointestinal tract may also be seen.[35] Filling defect is seen at MRCP and endoscopic retrograde cholangiopancreatography.[36]

Schistosomiasis

A granulomatous inflammation of liver, schistosomiasis is characterized by capsular calcifications. The liver forms gross septations with a turtle back appearance on CT scans. On MRI, these septations are hypointense on T1WI, hyperintense on T2WI, and enhance on post contrast scans. The calcified parasite

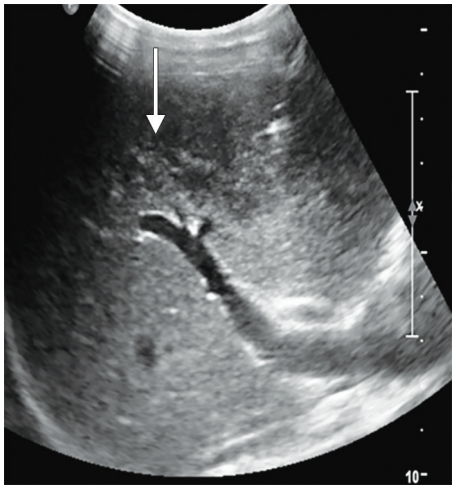


Fig. 16: Eosinophilic abscess—USG. An ill-defined heteroechoic lesion is seen (arrow)

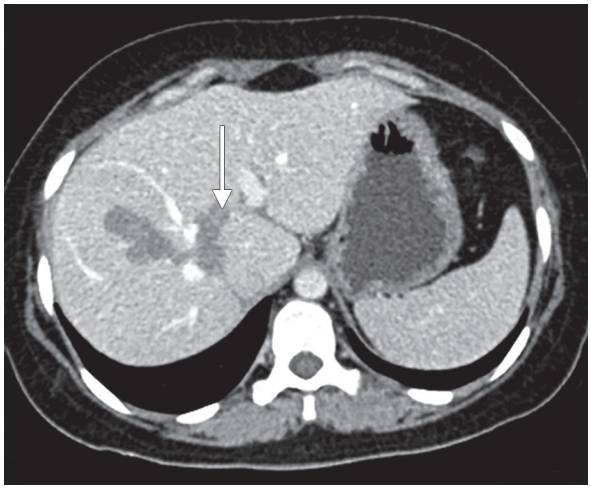


Fig. 17: Visceral larva migrans—CT. Well-defined tubular lesions are seen in right lobe (arrows)

appears hypointense on all sequences, best seen on gradient sequence.

Fascioliasis

Imaging features in fascioliasis are stage dependent. Initially, the patients have subcapsular nodules or abscesses. These may restrict on DWI. The disease progresses to the flukes presenting as fillings defects within the biliary tree with or without imaging features of cholangitis. These include biliary thickening and edema. These patients have a high eosinophil count, which often points toward the diagnosis.[37]

Clonorchiasis

Due to ascent of the flukes up the biliary tracts, on imaging, clonorchiasis presents with calcifications within the peripheral intrahepatic biliary radicals. There may also be associated biliary dilatation making differentiation from sclerosing cholangitis challenging.

Fungal Infection

The typical hosts for fungal infections include bone marrow transplant and AIDS patients or those with prolonged neutropenia. The commonest fungi to infect the liver include Candida, Aspergillus, and Cryptococcus. Their imaging appearances are similar and cannot be distinguished without tissue sampling and complementary studies like polymerase chain reaction (PCR) or β-D-glucan or galactomannan assay.

Candidiasis

Candida is the most common fungal infections to affect liver and spleen. The presence of multiple hepatic lesions in a patient with febrile neutropenia on or after chemotherapy must raise suspicion for fungal infection. It is essential to note that detection rate and imaging characteristics of fungal abscesses vary with neutrophil counts of immunocompromised patients. Lesions may not appear, and preexisting lesions may disappear during neutropenia. Hence, the host's immune response plays a defining role in characterizing the lesions on imaging.

Ultrasonography

On ultrasonography, hepatic candidiasis may present with varying imaging patterns that represent progressive stages of maturity of candida abscesses.

This has been subdivided into several patterns:[38]

- Wheel-within-wheel pattern composed of the hypoechoic center (necrosis), an inner hyperechoic ring (inflammation), and an outer hypoechoic ring (fibrosis)
- Wagon wheel appearance where the echogenic spokes represent inflammatory process, the hypoechoic areas between them represents fibrous tissue, and the axis of the wheel is the necrotic nidus.
- Bull's eye appearance which lacks the necrotic nidus and is composed of only the inner and outer rings (Fig. 17).
- Hypoechoic nodule representing fibrosis that has completely replaced inflammation
- Echogenic focus representing scar or calcification indicating resolution of infection

CT

Arterial phase CT imaging is more sensitive in detecting hepatic lesions than noncontrast and portal phase imaging.[39] Four types of lesions have been described on CT of which type IV lesion is most often missed on a portal venous scan.

- Type I: Hypoattenuating
- Type II: Hypoattenuating center with a hyperattenuating rim
- Type III: Any lesion with an associated transient hepatic enhancement
- Type IV lesion: Hyperattenuating lesion

The usual appearance is that of multiple small hypodense nodules in the liver and spleen. Rarely, central hyperdense foci may be seen within these nodules representing pseudohyphae.

MRI

Dynamic contrast-enhanced MRI is more sensitive to lesion detection than a portal phase CT scan.[40] Depending on their stage, fungal abscesses have varying presentations. Acute fungal microabscesses appear T1 hypointense and T2 hyperintense. No perilesional edema is seen in fungal abscesses differentiating them from other infections. In subacute treated phase, the abscesses appear hyperintense on both T1 and T2WI. A T2 hypointense rim may be seen representing peripheral macrophage aggregation. Chronic treated abscesses are minimally T1 hypointense, T2 iso to mildly hyperintense, and show no post gadolinium enhancement. On contrast-enhanced scans, ring enhancement may be seen in patients with relatively higher neutrophil counts. On DWI, fungal abscesses may show restriction of diffusion.[41]

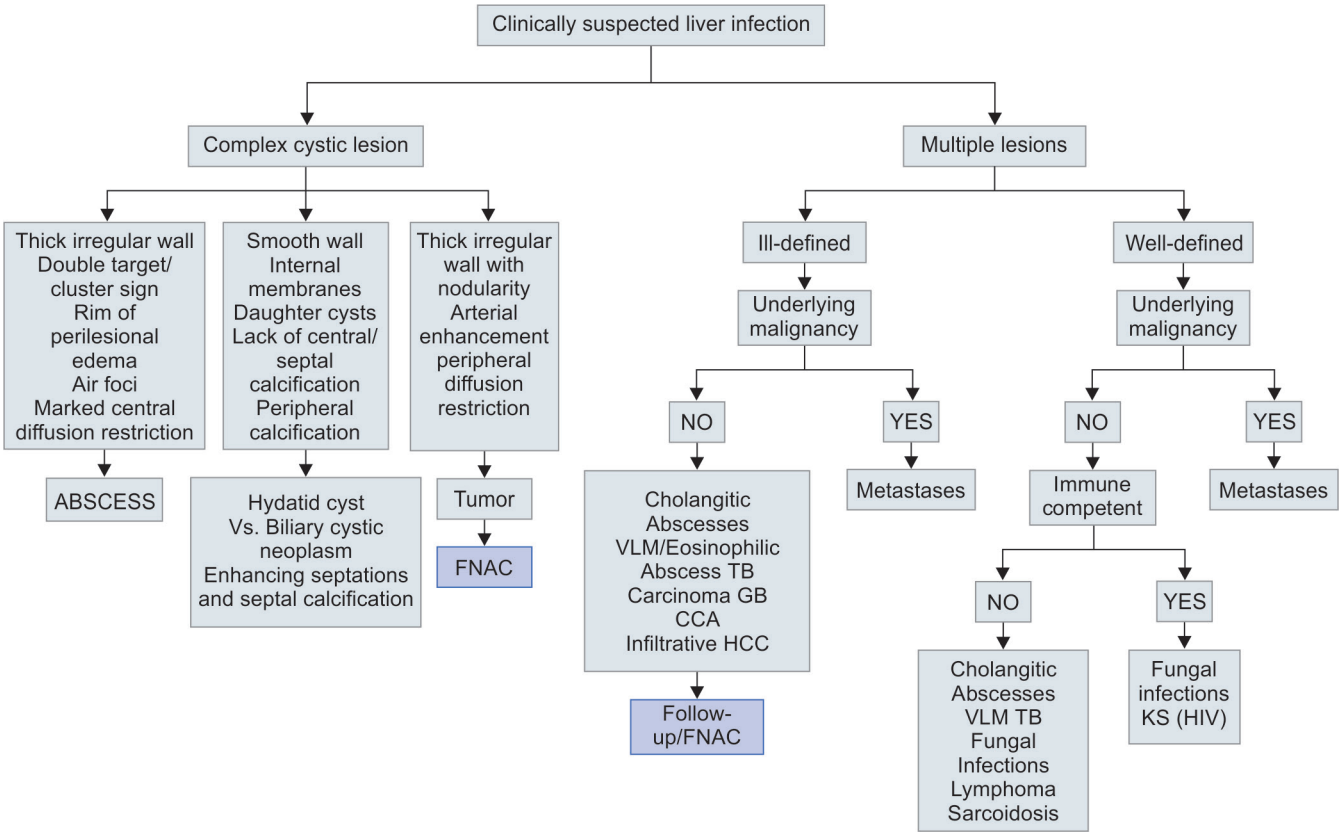
Histoplasmosis

Imaging appearance of hepatic histoplasmosis is similar to that of candidiasis with involvement of spleen (Flowchart 1).

Differential Diagnosis and Imaging Pitfalls

Tuberculosis, lymphoma, sarcoidosis, and metastases may present as multifocal hepatic nodules mimicking fungal infections. Presence of neutropenia is the primary pointer toward fungal etiology.

Flowchart 1: Approach to suspected liver infections. Modified from Bachler et al.[42]



Key imaging features
Imaging in disseminated fungal disease remains nonspecific. However, a resistance to antibiotics and a compromised immune status help narrow the differential.

Conclusion

Hepatic infections are common. Imaging findings are mostly nonspecific. However, if correctly interpreted, any peripheral solid component and irregular peripheral or central enhancement as well as septal calcification should favor a diagnosis of neoplasm rather than infection. Ultrasonography allows accurate characterization of cystic lesions in general and for hydatid cysts allow accurate detection of daughter cysts and membranes. The presence of fever, jaundice and immunosuppression favour the clinical diagnosis. Laboratory investigations favoring infective etiology include elevated total leukocyte count, absolute eosinophil count, hydatid serology, and other parasite serology in selected cases. Needle aspiration may sometimes be used, when despite all measures diagnosis remains unclear.

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