

Chapter 5

Cirrhosis-Associated Lesions and Pseudolesions

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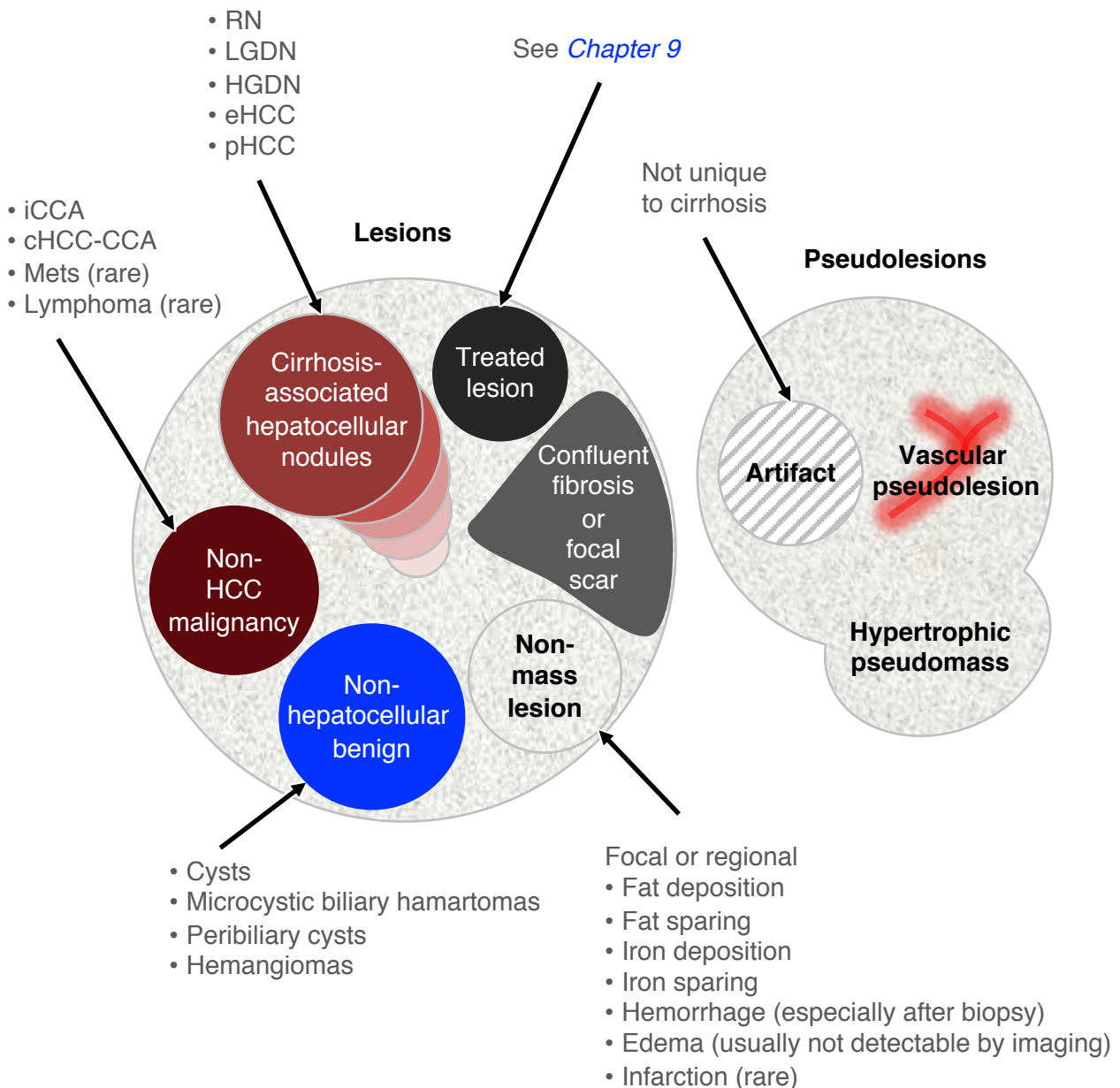
Cirrhosis-Associated Lesions and Pseudolesions

Introduction

This chapter reviews imaging manifestations of various lesions and pseudolesions that can be encountered in patients with cirrhosis.

What lesions are encountered at imaging in the cirrhotic liver?


As illustrated below and discussed on the pages that follow, the cirrhotic liver may contain a wide spectrum of lesions and pseudolesions at imaging.





Cirrhosis-Associated Hepatocellular Nodules

What are cirrhosis-associated hepatocellular nodules?

- These nodules are composed of hepatocytes (hence “hepatocellular”) and they are strongly associated with underlying cirrhosis (hence “cirrhosis-associated”).
- They represent a clinical and biological continuum ranging from benignity to overt malignancy.
- For simplicity and clinical utility, they are classified into five distinct categories based on their pathological features.
-  Note that classification is based on gross and microscopic pathology, not on imaging or molecular characterization.

Regenerative nodules (RNs)

- Also known as cirrhotic nodules (CNs) or cirrhotic regenerative nodules, RNs are histologically nonneoplastic, benign lesions representing a regenerative response to repetitive injury.
- Resembling other background nodules, they have no distinctive pathologic features. Thus, they are rarely if ever more steatotic or siderotic than the rest of the liver.

Low-grade dysplastic nodules (LGDNs)

- LGDNs are histologically benign clonal expansions of cells with no cellular atypia.
- Since they are clonal populations, they typically have distinctive phenotype compared to background nodules.
- Grossly, they differ in size, color, or degree of bulging from background nodules
- They are usually ≥ 8 mm
- LGDNs may be more steatotic or siderotic than the rest of the liver



Despite histological benignity and lack of cellular atypia, RNs and LGDNs may contain molecularly aberrant cells that may become neoplastic. Since these cells cannot be seen with noninvasive imaging, neither CT nor MRI can reliably categorize RNs or LGDNs as definitely benign even if they have no malignant imaging features. Thus, nodules thought to represent RNs or LGDNs cannot be categorized LR-1. They can be ignored or they can be categorized LR-2 or higher.




The presence of steatosis or siderosis virtually excludes RN.



Cirrhosis-Associated Hepatocellular Nodules


High-grade dysplastic nodules (HGDNs)

- Representing clonal expansions of neoplastic cells, HGDNs are premalignant lesions with high risk of transformation to HCC.
- Histologically, they are characterized by cellular and architectural atypia insufficient for diagnosis of HCC.
-  A key distinguishing histologic feature from eHCC is the absence of stromal invasion (see below).
- Like LGDNs, they differ in size, color, or degree of bulging from background nodules at gross pathology, and they usually measure ≥ 8 mm
- The nodules may be steatotic or siderotic.

HCC

- Malignant neoplasm composed of cells with hepatocellular differentiation
Subdivided into early and progressed

Early HCC (eHCC)

- Analogous to carcinoma-in-situ, eHCC is the earliest histologically definable stage of HCC development (hence “early” HCC).
- With little if any capacity to invade vessels or metastasize, the tumor is not overtly malignant but it has high risk of progressing to overt malignancy.
- Histologically, the lesion is characterized by well-differentiated cells resembling HGDN
-  A key distinguishing histologic feature from HGDN is the presence of stromal invasion, defined as the presence of tumor cells in the stroma of the intranodular portal triads and/or in the fibrous scars surrounding the nodules
- Unlike progressed HCC (below), the nodule exhibits “replacing growth”: the tumor cells gradually encroach into the surrounding parenchyma without destroying underlying architecture
- Due to the replacing growth, the lesion appears vaguely nodular at gross pathology, with indistinct margins that blend into surrounding tissue
- A tumor capsule is not present,
- eHCC is often steatotic; some studies suggest that of all cirrhosis-associated HC nodules, eHCC has the highest frequency of intralesional fat
- eHCC is rarely siderotic as the cells are felt to be iron “resistant”
- eHCCs are often undetectable at imaging. When visible, they tend to be well defined despite their vaguely nodular pathologic appearance.
- Usually ≤ 1.5 cm in size



Cirrhosis-Associated Hepatocellular Nodules

Progressed HCC (pHCC)

- pHCC is an overtly malignant cancer with capacity for vascular invasion and metastasis (hence, “progressed”).
- It is usually composed of moderately or poorly differentiated cells.
- Unlike the replacing growth of eHCC, pHCC exhibits “expansile growth”: the cells destroy normal hepatic architecture and compress adjacent parenchyma.
- The parenchymal compression may induce the formation of a tumor capsule. Hence, pHCC may be encapsulated.
- Even if unencapsulated, it has a distinctly nodular morphology, with sharply defined margins.
- pHCC is rarely steatotic or siderotic
- It is sub-divided into small (<2 cm) and large (≥ 2 cm).
- Large tumors may be aggressive and they may penetrate the tumor capsule if present.
- Very aggressive tumors may exhibit permeative growth.
 - This is sometimes described as “infiltrative” HCC.
 - Radiologists should be aware, however, that the term infiltrative HCC has not been used consistently in the radiology literature and is sometimes used as a synonym for diffuse HCC.

► Table on next page summarizes the distinguishing features of the various nodules

Cirrhosis-Associated Hepatocellular Nodules

Table. Cirrhosis-Associated Hepatocellular Nodules

	Size	Biology	Histology	Gross Pathology
RN	Usually < 1 cm	“Benign”*	No cytologic atypia or architectural changes	Identical to background liver
LGDN	Usually < 1 cm	“Benign”*	No cytologic atypia Mild architectural changes ± clonal features ± steatosis ± siderosis	Distinctive in color, texture, or degree of bulging
HGDN	Usually < 1.5 cm	Premalignant	Cellular atypia Mod architectural changes Clonal features ± steatosis ± siderosis	Distinctive in color, texture, or degree of bulging
eHCC	Usually < 1.5 cm	Microinvasive cancer, analogous to “carcinoma-in-situ” No vascular invasion Rare metastases	Well-differentiated malignant cells “Stromal invasion” Often steatotic Iron resistant	Vaguely nodular Indistinct margins No capsule
Small pHCC	< 2 cm	Malignant ± vascular invasion ± metastases	Moderately differentiated malignant cells Rarely steatotic Iron resistant	Distinctly nodular Well-defined margins Capsule frequent
Large pHCC	≥ 2 cm	More aggressive Frequent vascular invasion Frequent metastases	Moderately to poorly differentiated Rarely steatotic Iron resistant	Distinctly nodular, Well defined margins Capsule frequent May become diffuse (“infiltrative”)

* Although they are considered “benign”, RNs and LGDNs may contain molecularly aberrant, neoplastic cells. Despite the molecular aberrations, the cells may be normal phenotypically, undetectable by noninvasive imaging or routine histology.



Morphological Patterns of HCC: Pathology

What are the morphological patterns of HCC?

The gross morphological patterns of HCC have been classified as follows:

- Egel H (1901): nodular; massive; diffuse
- Trevisani F (1993): nodular solitary; multinodular; massive; diffuse
- Liver cancer study group of Japan (2000): simple nodular; simple nodular with extranodular growth; confluent multinodular growth

Below we elaborate on the classification system developed by the Liver cancer study group of Japan.

Morphological patterns on pathology (classification system of Liver cancer study group of Japan)

Nodular

- Simple nodular
 - Indistinctly (vaguely) nodular
 - Nodular growth with a vaguely nodular appearance, lack of capsule and vascular invasion and it is usually well-differentiated and smaller than the distinctly nodular type. Early HCC is a small (usually < 1.5 cm) lesion with a indistinctly nodular pattern.
 - Distinctly nodular
 - Nodular growth with a clear demarcation with or without a capsule or fibrous septa and is the gross morphological manifestation of a progressed HCC.
- Nodular with extranodular growth
 - Nodular growth with extension of tumor beyond the expected boundary of the dominant nodule
- Confluent multinodular
 - Confluence of variably sized tumor nodules into a single conglomerate mass

Multifocal

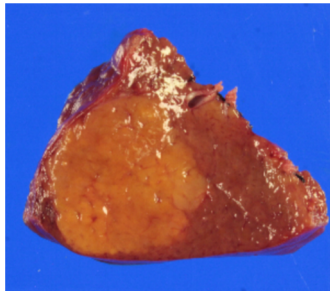
- Two or more nodules with any combination of the above described growth patterns

Diffuse

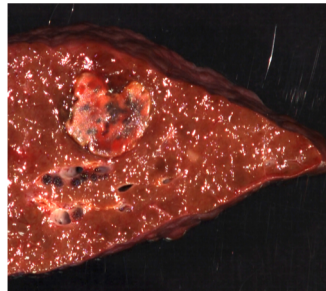
- Spread of multiple small tumor nodules throughout large contiguous portion of liver or whole liver.

Morphological Patterns of HCC: : Pathology

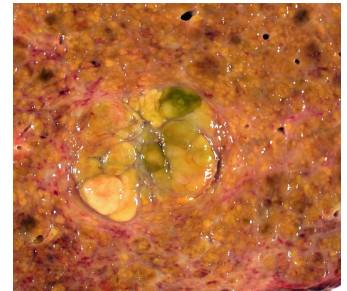
Indistinctly nodular



Distinctly nodular



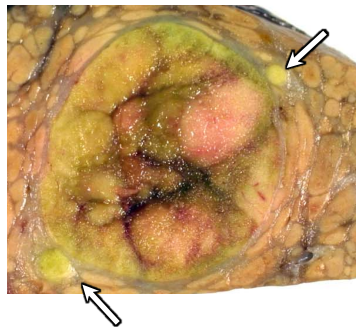
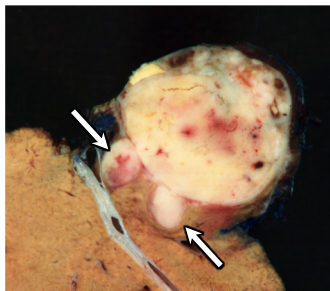
Distinctly nodular



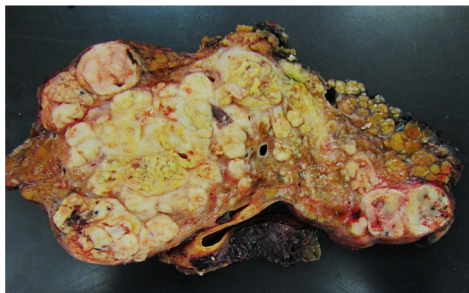
Simple nodular

Nodular with extranodular growth

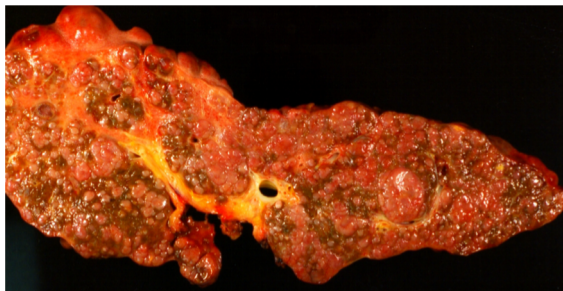
Simple nodular with extranodular growth



Confluent multinodular



Diffuse



Morphological Patterns of HCC: Imaging

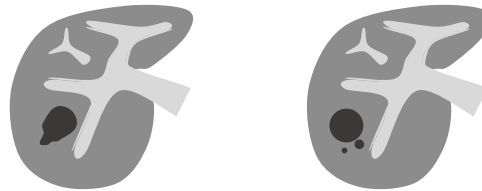
Nodular

Nodules manifest as distinct, typically circumscribed lesions. The pathology classification of indistinctly vs. distinctly nodular does not apply to imaging, where most detectable nodules appear circumscribed, including those that would be poorly margined at gross pathology.



Nodular with extranodular growth

Extranodular growth manifests as focal extension of tumor beyond the expected margin of a nodule, irregular margins of a nodule, or satellite nodules. Satellite nodules are defined as small nodules with the same imaging pattern as a dominant nodule located no more than 2 cm from the margin of the dominant nodule.



Confluent multinodular

Synchronous or metachronous nodules, which due to proximity and growth, have become confluent, and resemble a single mass.



Morphological Patterns of HCC: Imaging

Multifocal

Synchronous or metachronous nodules, which are not necessarily contiguous, and may manifest in variety of the nodular growth patterns listed above.



The individual nodules may represent multicentric hepatocarcinogenesis, intrahepatic metastases from a primary HCC, or a combination of both pathways.

A clue to multicentric hepatocarcinogenesis is the presence of multiple nodules with variable appearances whose imaging features suggest different points along the hepatocarcinogenesis spectrum (see [Chapter 6](#)).

A clue to metastatic spread is the presence of a dominant mass in conjunction with satellite nodules and/or more remote intrahepatic nodules with similar imaging features (see [Chapter 6](#)).

However, imaging cannot distinguish multicentric hepatocarcinogenesis from intrahepatic metastatic spread reliably, and the clinical relevance of distinguishing these pathways has not been established. For these reasons, LI-RADS does not recommend that radiologists attempt to make the distinction in their reports.



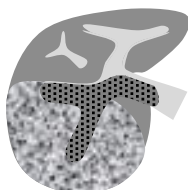
Morphological Patterns of HCC: Imaging

Diffuse

Segmental or lobar distribution of micronodules, which often have a “infiltrative” or “permeative” appearance on imaging. Commonly associated with tumor in vein.

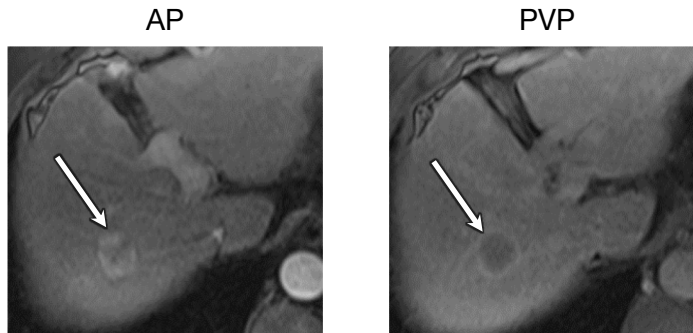
Features:

- AP:
 - Mild heterogeneous arterial-phase hyperenhancement, or
 - Wedge-shaped arterial-phase hyperenhancement mimicking transient hepatic intensity difference
- PVP/DP
 - Subtle, usually miliary, “washout”
- T2 and DWI
 - Typically hyperintense on T2- and diffusion-weighted sequences. Often more conspicuous on T2 and diffusion weighted sequences than on multiphase contrast enhanced T1-weighted sequences.
- Other: Tumor in vein is often present and can be a key diagnostic clue.



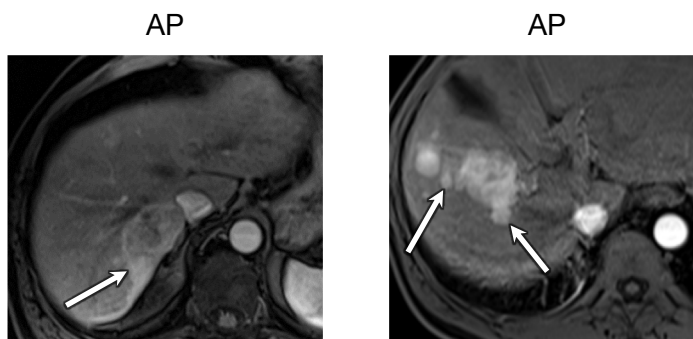
Morphological Patterns of HCC: Imaging

Example: Nodular



Well-defined, round nodule or mass with APHE, WO and “capsule”

Example: Nodular with extranodular growth (two different patients)

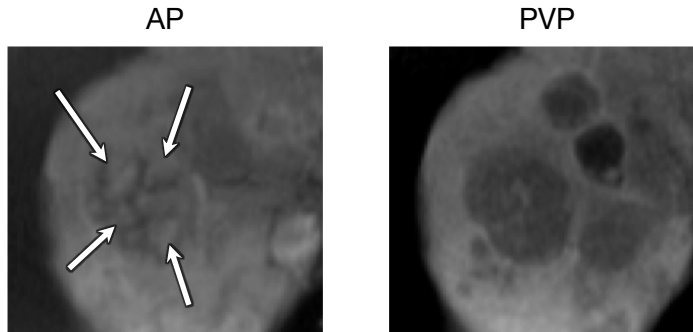


Non-circular or irregularly shaped mass, indicating growth beyond/outside of the initial mass.

May also be seen as adjacent satellite nodule(s) around a mass

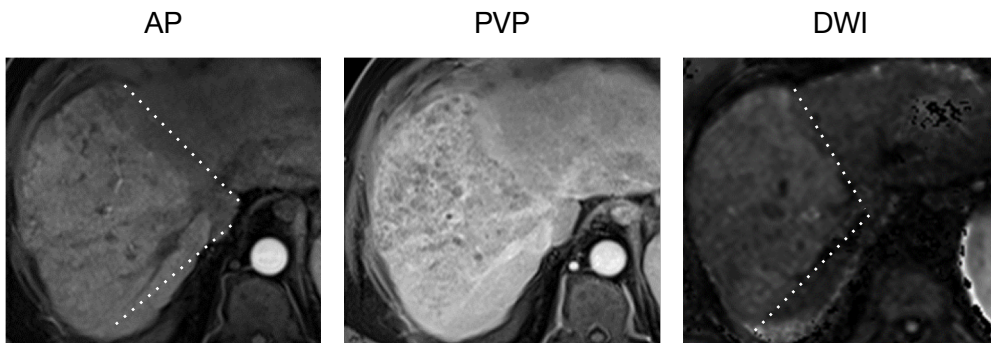
Morphological Patterns of HCC: Imaging

Example: Confluent multinodular



Multiple confluent nodules

Example: Diffuse



Typically, a wedge-shaped region of APHE, multifocal “washout”, diffusion restriction, and T2 hyperintensity. Tumor in vein is usually present and occurs in the vascular distribution of the tumor



Morphological Patterns of HCC

Diffuse HCC: Pitfalls

Benign conditions and non-HCC malignancies may be mistaken for diffuse HCC

- Confluent hepatic fibrosis:
 - Clues to the correct diagnosis of confluent fibrosis: often in the anterior and medial hepatic segments, typically associated with capsular retraction, enhances progressively on delayed phase images (using ECA). See [Chapter 15, page 18](#).
 - Geographic fat deposition:
 - Clues to the correct diagnosis of fat deposition: signal loss on opposed-phase T1w GRE, especially if uniform or homogeneous, is key for proper diagnosis. See [Chapter 15, page 14](#).
 - Severe micronodular cirrhosis:
 - Often has heterogeneous AP enhancement as well as progressive enhancement of lace-like fibrosis in the delayed phase. The intervening liver parenchyma is hypoenhanced relative to the fibrosis in the delayed phase, which mimics the appearance of “washout”.
 - Clues to the correct diagnosis of severe micronodular cirrhosis: involves entire liver.
 - Other malignancies:
 - iCCA: targetoid appearance (see [Chapter 16, page 205](#)). TIV possible but rare
 - Diffuse metastatic disease (“pseudo-cirrhosis”): diffuse hepatic metastases with marked alteration of the hepatic morphology mimicking cirrhosis. Usually in patients receiving chemotherapy for metastatic breast cancer (not a LI-RADS population).
-

Diffuse HCC may be difficult to detect despite its large size

- APHE may be subtle or absent
 - WO may be subtle or absent
 - Hepatobiliary uptake may be similar to that of background cirrhotic liver
 - The mass may blend imperceptibly with background cirrhotic liver
 - The heterogeneity of diffuse HCC may resemble the heterogeneity of cirrhosis
-



Morphological Patterns of HCC

Diffuse HCC: Practical considerations

Diffuse HCC: clues to diagnosis



Tumor in vein is often the first clue to the presence of diffuse HCC in the liver parenchyma.

- If tumor in vein is detected or suspected, scrutinize the adjacent liver parenchyma for diffuse HCC.

Alpha-fetoprotein is often markedly elevated.

Diffuse HCC: MRI vs. CT



MRI offers potential advantages over CT for detection of diffuse HCC due to multiple contrast mechanisms.

- If diffuse HCC is suspected on CT, consider multiphase MRI, including DWI, if possible.
-

Diffuse HCC: biopsy



- Tissue sampling may be required in ambiguous cases.
-

Diffuse HCC: Terminology

The LI-RADS RAD-PATH committee prefers the use of the term “diffuse HCC” and discourages the use of the following terms:

- Massive HCC
- Extensive HCC
- Infiltrative HCC
- Cirrhotico-mimetic HCC
- Cirrhosis-like HCC

Rationale

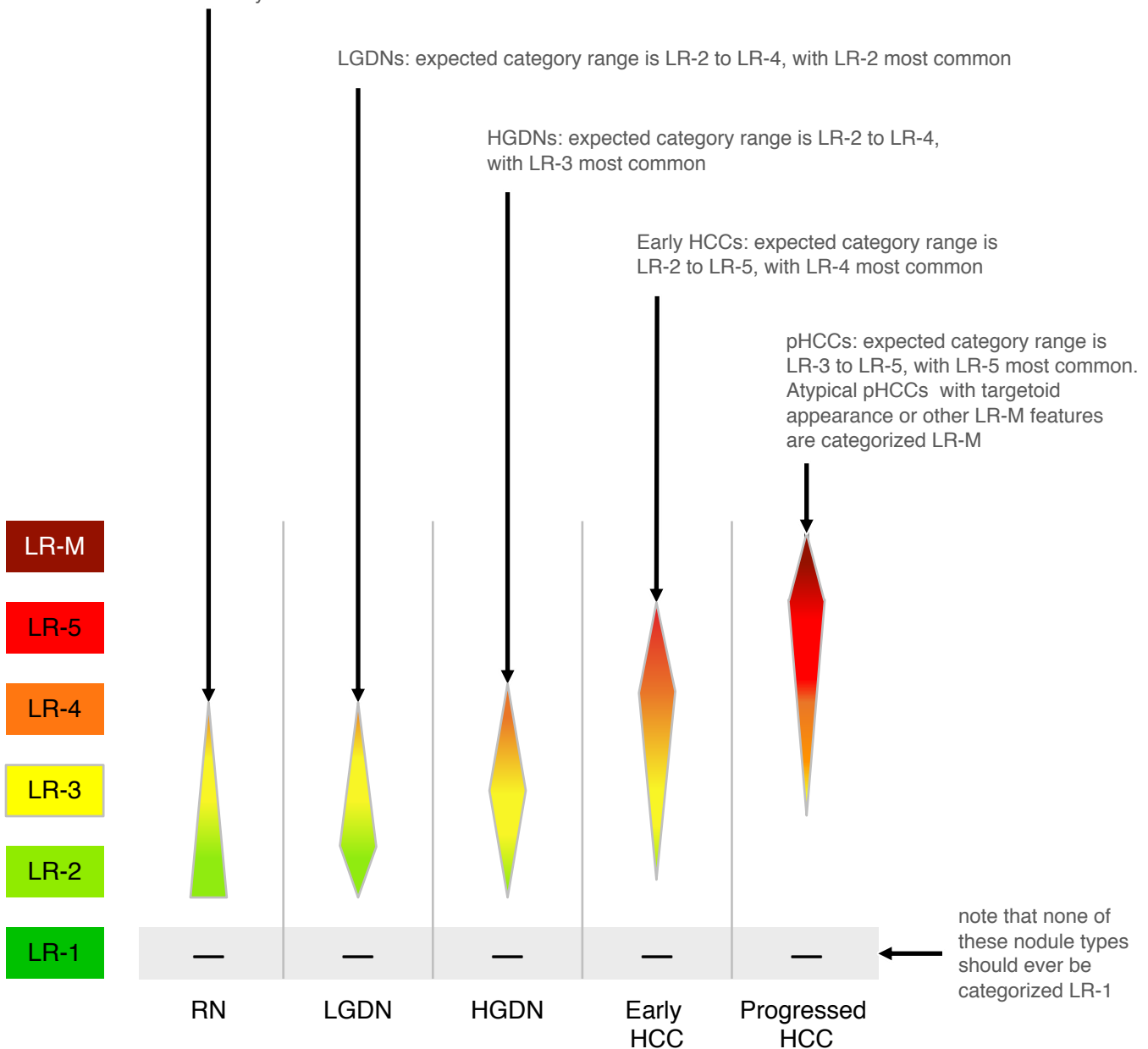
- The term diffuse HCC is preferred to convey the idea of the spread of tumor nodules in a large portion of liver parenchyma and to avoid confusion with the “infiltrative” growth of a progressed, nodular HCC.
-

Histologic vs. LI-RADS Categories

Is there a correspondence between histologic and LI-RADS categories?

As illustrated in the diagram below, histologic and LI-RADS categories do not have a 1:1 correspondence. The histologically defined nodules overlap in imaging features relevant to LI-RADS categorization. Hence, each histologic category may correspond to a range of LI-RADS categories.

The vast majority of RNs are NOT assigned LI-RADS categories since they closely resemble other background nodules at imaging (i.e., they are not distinctive and so not LI-RADS observations). Atypically an RN may be distinctive: the expected category range for such RNs is LR-2 to LR-4, with LR-2 by far the most common.





FNH and HCA

What about FNH and HCA? Why are these lesions not mentioned in the prior slides as examples of hepatocellular lesions encountered in cirrhosis?

Although relatively frequent hepatocellular nodules in patients without cirrhosis, focal nodular hyperplasia (FNH) and hepatocellular adenoma (HCA) are uncommon in patients with cirrhosis.

Moreover, FNH and HCA may overlap in appearance with HCC. For these reasons, FNH and HCA are diagnoses of exclusion in high-risk patients.

To discourage radiologists from inappropriately suggesting the diagnosis of FNH or HCA in cirrhosis, these lesions are purposely omitted from the preceding pages.

So how should nodules with features highly suggestive of FNH or HCA be categorized?

They usually should be categorized LR-3.

With caution, they may be categorized LR-2.

They should not be categorized LR-1 or LR-5.

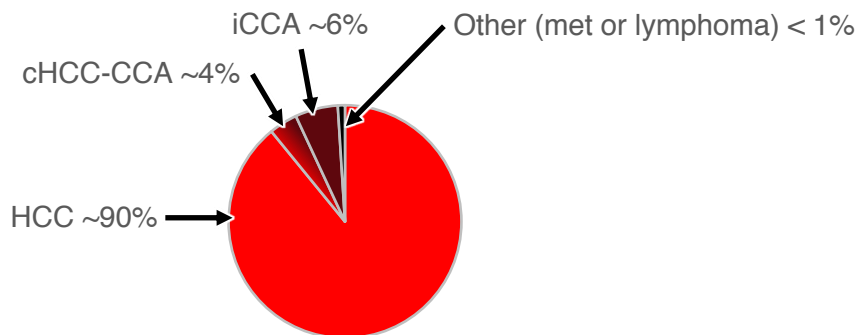
Non-HCC Malignancies

What are non-HCC malignancies?

Non-HCC malignancies include intrahepatic cholangiocarcinoma (iCCA), combined hepatocellular-cholangiocarcinoma (HCC-CCA), metastasis to liver from extrahepatic site, and lymphoma

What is the relative proportion of the various types of malignancy in cirrhosis?

This is not yet well understood. As an order of magnitude, it is about 90% : 6% : 4% : <1%



What is iCCA?

iCCAs are cancers composed of cells with cholangiocyte differentiation. They are the second most common primary liver cancer in cirrhosis.

iCCAs have a vascularized cellular rim and a fibrotic core. This architecture accounts for their characteristic targetoid imaging appearance: AP rim enhancement, peripheral washout appearance, progressive central enhancement, and target appearance at DWI and in the HBP. Small iCCAs may be diffusely hypercellular without a prominent central stroma and overlap in appearance with HCC, however. Additionally, some HCCs may have a targetoid appearance due to central fibrosis, fat, or hemorrhage.

What is cHCC-CCA?

cHCC-CCAs are malignancies composed of cells with both cholangiocellular and hepatocellular differentiation. The two cell types may be mixed together or may be in separate components of the tumor. They are the third most common primary liver cancer in cirrhosis.

Their imaging appearance is not yet well understood. Emerging reports suggest they resemble iCCAs in appearance although they may exhibit hepatocellular features such as intralesional fat. Depending on their degree of hepatocellular differentiation, it may be impossible to differentiate some cHCC-CCAs from HCC.

Non-HCC Malignancies

Why are iCCA and cHCC-CCA important?

Although neither is as common in cirrhosis as HCC, these non-HCC malignancies are more common in patients with cirrhosis than in the general population.

iCCAs tend to metastasize to extrahepatic sites early in their development. Liver transplantation is not done for these lesions except under investigational protocols, because of high post-transplant recurrence risk.

The prognosis and biology of cHCC-CCA is not yet well understood. Conservatively, these tumors are assumed to have poor prognosis and biology, similar to iCCA.

Non-HCC malignancies are relatively common in cirrhosis and require different treatment than HCC. Hence, a diagnostic test such multiphase CT or MRI imaging study—should differentiate HCC from iCCA and from cHCC-CCA.

Unfortunately, perfect differentiation between HCC and these other malignancies is not always possible due to overlap in imaging features.



The LI-RADS categorization algorithm is designed to maximize specificity for HCC, with an unavoidable trade-off in sensitivity.

When observations have classic features for HCC, they should be categorized LR-5, with the understanding that this category virtually excludes the possibility of iCCA or other non-HCC malignancy.

Unfortunately, it is impossible to completely exclude all non-HCC malignancies, especially cHCC-CCAs. Depending on their degree of hepatocellular differentiation, cHCC-CCAs may overlap substantially in appearance with HCC.

When observations have features suggestive of iCCA or other non-HCC malignancy, they should be categorized LR-M, with the understanding that this category does not exclude HCC, since some HCCs may have a targetoid appearance of other atypical features.

HCCs with targetoid appearance include those with a central fibrous stroma (e.g., scirrhous HCC), progressed HCCs with intralesional fat (e.g., steatohepatic HCC), and HCCs with intralesional hemorrhage or necrosis.

- ▶ These concepts are illustrated in the next two slides
- ▶ See also [Chapter 8, page 5](#).

Summary

Summary

Although the most common liver cancer in cirrhosis is HCC, patients with cirrhosis are also at increased risk for developing iCCA and cHCC-CCA. Differentiation of HCC from these other cancers is important due to differences in prognosis and treatment.

Patients with cirrhosis also may have a plethora of benign and premalignant lesions. These may obscure true HCCs (lower sensitivity) or be mistaken for HCCs (lower specificity).

FAQs

What are the LI-RADS categories associated with the most common malignancies in cirrhosis?

The diagram below illustrates the range of LI-RADS categories that should be assigned to the most common malignancies in cirrhosis.

Most early HCCs, if discernible, are categorized LR-4, although they may be categorized LR-2 to LR-5, depending on features

About 1/2 of pHCCs can be categorized LR-5

Atypical pHCCs with targetoid appearance or other LR-M features are categorized LR-M

Some pHCCs are categorized LR-3 or LR-4 due to absence of one or more major HCC features

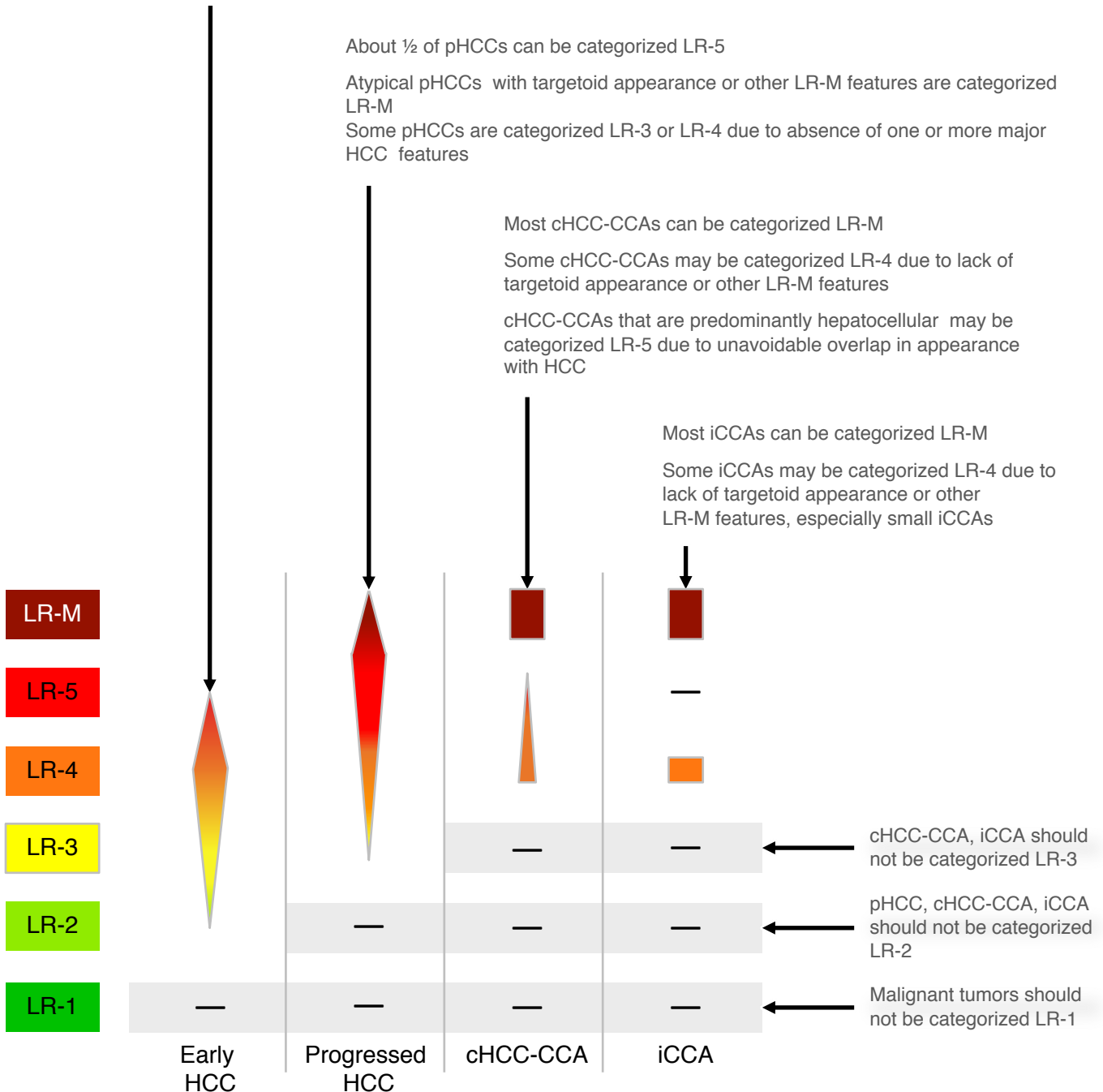
Most cHCC-CCAs can be categorized LR-M

Some cHCC-CCAs may be categorized LR-4 due to lack of targetoid appearance or other LR-M features

cHCC-CCAs that are predominantly hepatocellular may be categorized LR-5 due to unavoidable overlap in appearance with HCC

Most iCCAs can be categorized LR-M

Some iCCAs may be categorized LR-4 due to lack of targetoid appearance or other LR-M features, especially small iCCAs

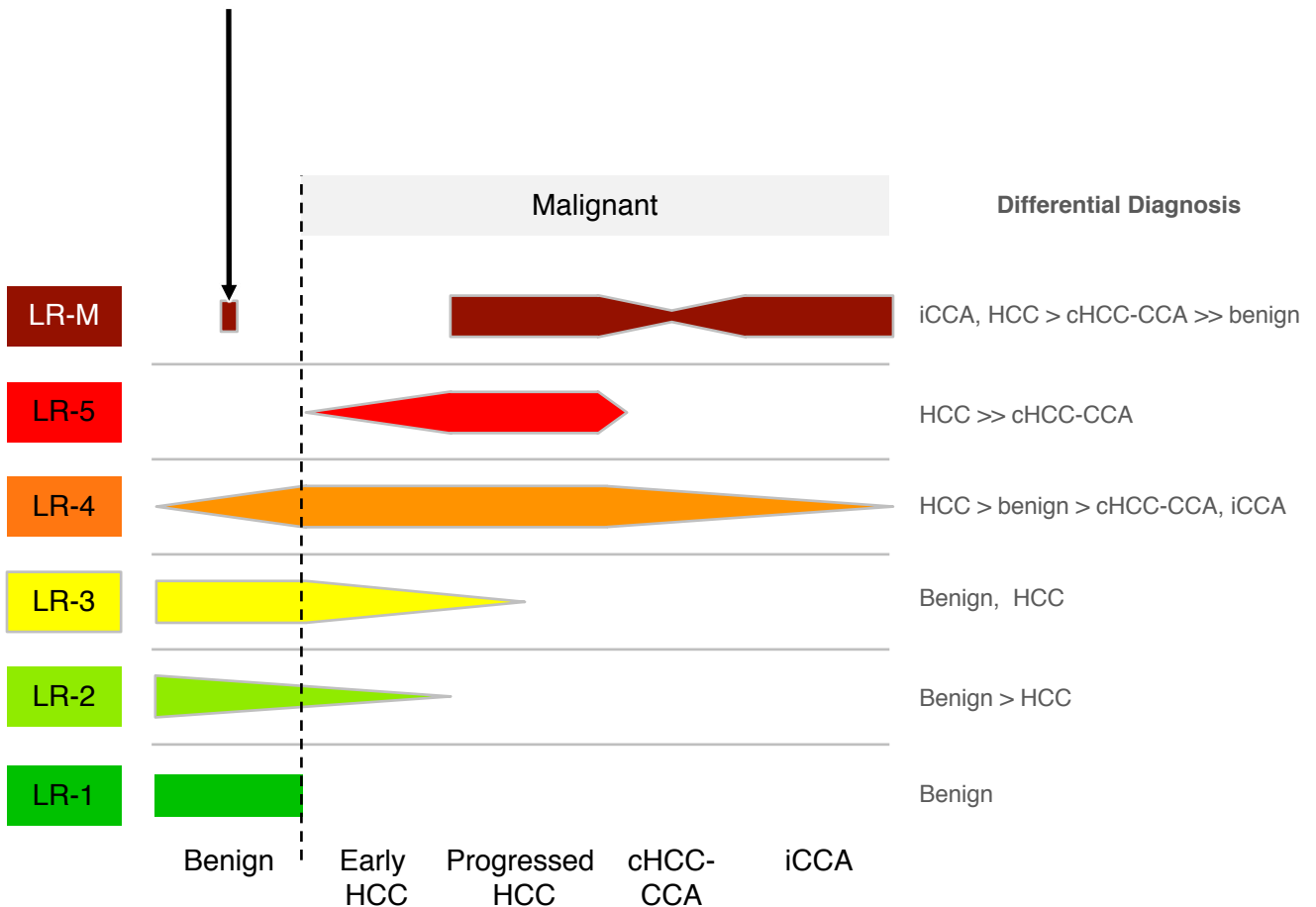


FAQs

What are the the most common malignancies in cirrhosis associated with each LI-RADS category?

The diagram below illustrates the expected differential diagnosis for LI-RADS categories. Only cancers that are relatively common in cirrhosis are shown.

Some observations categorized as LR-M ultimately prove to be benign. Examples include sclerosing hemangiomas which may have rim APHE, infarcted regenerative nodules, and rare perfusion alterations with rim pattern



FAQs

What are non-hepatocellular benign lesions?

Non-hepatocellular benign lesions include cysts, microcystic biliary hamartomas, peribiliary cysts, and hemangiomas

Some of these are more common in patients with cirrhosis than in the general population. Some are less common but may have unusual imaging appearances, challenging the interpretation.

What are cysts?

Cysts are benign cystic lesions lined by biliary epithelium but without connection to the biliary system.

Cysts are the most common non-hepatocellular focal lesions in cirrhosis. They tend to involute and become small as cirrhosis develops. At CT, tiny cysts may be difficult to characterize but at MRI the diagnosis is straightforward.

What are microcystic biliary hamartomas?

Microcystic biliary hamartomas are focally dilated, disorganized bile ducts surrounded by fibrocollagenous stroma. They usually do not communicate with the biliary tree.

Microcystic biliary hamartomas are seen rarely in cirrhosis. The imaging diagnosis is the same as in the normal liver.

What are peribiliary cysts?

Peribiliary cysts are cystic dilations of the peribiliary glands. They are unique but rare manifestations of cirrhosis.

They develop when fibrotic bands obstruct the peribiliary glands, causing them to dilate into cysts. Since they course along the portal tracts, the cysts may be mistaken at imaging for dilated bile ducts. One clue is that they tend to be more bulbous than bile ducts. In challenging cases, a hepatobiliary agent may be administered as the obstructed glands do not communicate with the biliary system.

What are hemangiomas?

Hemangiomas are benign vascular malformations composed of disorganized blood vessels and spaces.

Hemangiomas are relatively frequent in patients with early cirrhosis but infrequent in patients with advanced cirrhosis, because they involute as cirrhosis progresses and eventually disappear or become so small that they escape detection. As they involute, they become fibrotic (“sclerosing hemangioma”) and develop unusual imaging features, which challenges diagnosis. See [Chapter 1, page 4](#).

FAQs

What non-mass benign lesions are commonly encountered in cirrhosis?

Non-mass benign lesions include focal or regional

- Fat deposition excess lipid in hepatic parenchyma relative to surrounding liver
- Fat sparing paucity of lipid in hepatic parenchyma relative to surrounding liver
- Iron deposition excess iron in hepatic parenchyma relative to surrounding liver
- Iron sparing paucity of iron in hepatic parenchyma relative to surrounding liver
- Edema edema in hepatic parenchyma (often not discernible at imaging)
- Hemorrhage hemorrhage in hepatic parenchyma (most commonly seen after biopsy)

These lesions are important mainly because they may cause diagnostic errors, either by obscuring true HCC nodules or by being falsely misinterpreted as HCC nodules.

Infarction is another non-mass benign lesion. It is not listed above due to its rarity.

What is confluent fibrosis?

Confluent fibrosis is a macroscopically evident benign process of scarring in the liver parenchyma caused by conglomeration of dense fibrotic tissue into a confluent mass.

Confluent fibrosis is important because it may cause diagnostic errors, either by obscuring true tumor nodules or by being falsely misinterpreted as a fibrotic tumor such as iCCA. A key distinguishing feature is that confluent fibrosis does not exhibit the targetoid appearance characteristic of iCCA and other fibrotic tumors.

Confluent fibrosis also may be a clue to the etiology of liver disease, as it is more common in alcoholic liver disease and in PSC than in NASH or viral liver.

See [Chapter 15, page 18](#) for imaging appearance of confluent fibrosis.

What are focal scars?

A focal scar is a macroscopically visible scar limited to a small area of hepatic parenchyma. Pathologically it is the same as confluent fibrosis, only smaller, and so it has similar enhancement characteristics of confluent fibrosis. It often manifests as a thin band and rarely causes diagnostic confusion with a neoplastic mass.



FAQs

What is a pseudolesion?

A pseudolesion is an area of parenchyma that appears abnormal at imaging without a corresponding pathologic abnormality. Examples include:

- Vascular pseudolesion
- Hypertrophic pseudomass
- Imaging artifact or anatomic structure (e.g., intrahepatic varix) mistaken for a lesion

What is a vascular pseudolesion?

Vascular pseudolesions are areas of the liver that appear abnormal at imaging due to alterations in perfusion but without a causative parenchymal abnormality.

Most commonly, these manifest as ill-defined, wedge-shaped, or geographic areas of arterial phase hyperenhancement. Typically they are small, located peripherally, and occult on all non-arterial phase images, although they may be faintly hyperenhanced in the extracellular phases, faintly hyperintense on T2w and DW images, and faintly hypointense on HBP T1w images.

As they are not true lesions, they exert no mass effect, although their small size may complicate reliable assessment of this feature.

The most common cause is arterioportal shunting caused by cirrhosis-associated alterations in the cirrhotic microcirculation. High-pressure contrast-enhanced arterial blood enters the sinusoid rapidly through the microscopic shunt, leaks rapidly into the interstitium, and causes transient arterial-phase hyperenhancement of the affected parenchyma.

These hyperenhancing pseudolesions are especially common at MRI, in part due to the high sensitivity of MRI to contrast enhancement, although they may occur at CT.

They may cause diagnostic errors, either by obscuring true HCC nodules or by being falsely misinterpreted as HCC nodules.

Lack of visibility on non-arterial phase images (e.g., T2w images, DW images, and, if obtained HBP images) are clues to the correct diagnosis.

They are not visible at and so do not cause diagnostic confusion at CEUS, perhaps because CEUS microbubbles are blood pool contrast agents that do not leak into the liver interstitium.

Sometimes, vascular pseudolesions manifest as areas of nonmasslike portal or delayed phase hypoenhancement. Absence of mass effect suggests the correct diagnosis.

What is a hypertrophic pseudomass?

It is a hypertrophic area of liver surrounded by atrophic, fibrotic liver parenchyma. It may have a bulging appearance at imaging and resemble a mass.

FAQs

What are the LI-RADS categories associated with the most common benign entities in cirrhosis?

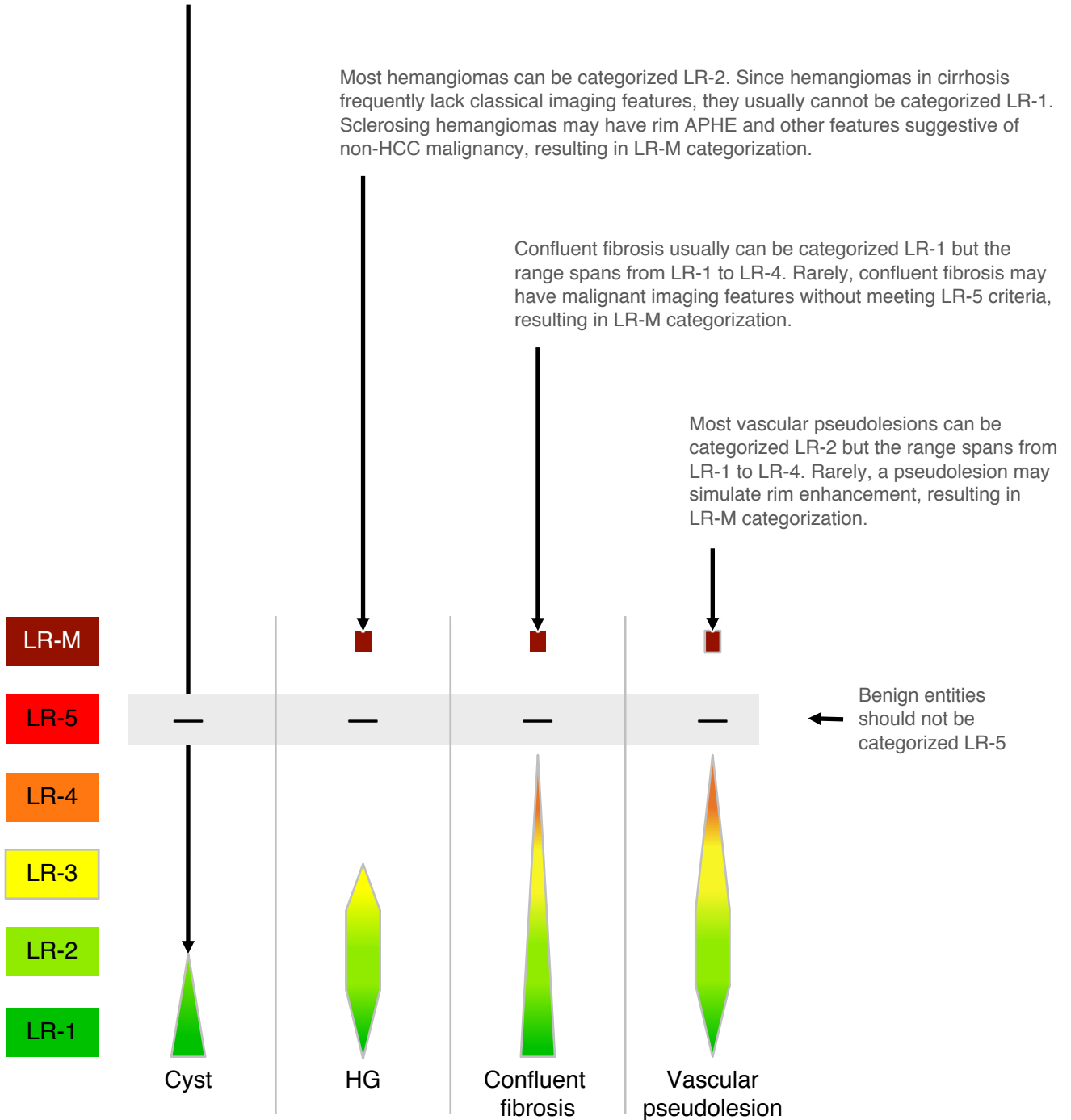
The diagram below illustrates the range of LI-RADS categories that should be assigned to the most common benign entities in cirrhosis.

Virtually all cysts should be categorized LR-1. Atypical cysts can be categorized LR-2.

Most hemangiomas can be categorized LR-2. Since hemangiomas in cirrhosis frequently lack classical imaging features, they usually cannot be categorized LR-1. Sclerosing hemangiomas may have rim APHE and other features suggestive of non-HCC malignancy, resulting in LR-M categorization.

Confluent fibrosis usually can be categorized LR-1 but the range spans from LR-1 to LR-4. Rarely, confluent fibrosis may have malignant imaging features without meeting LR-5 criteria, resulting in LR-M categorization.

Most vascular pseudolesions can be categorized LR-2 but the range spans from LR-1 to LR-4. Rarely, a pseudolesion may simulate rim enhancement, resulting in LR-M categorization.



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