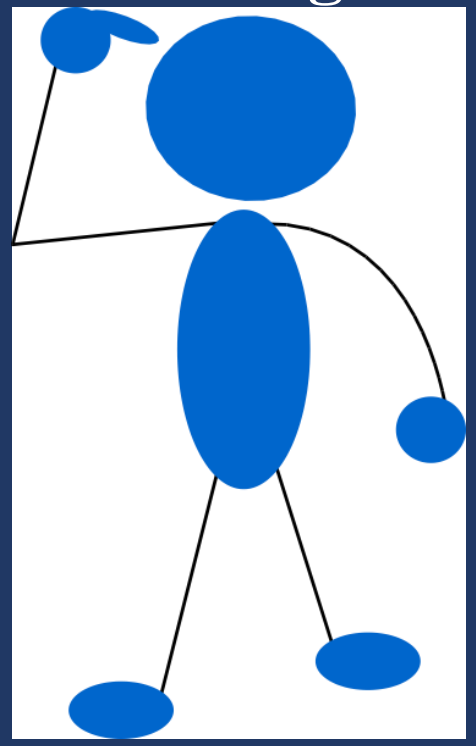
malignant hepatic lesion

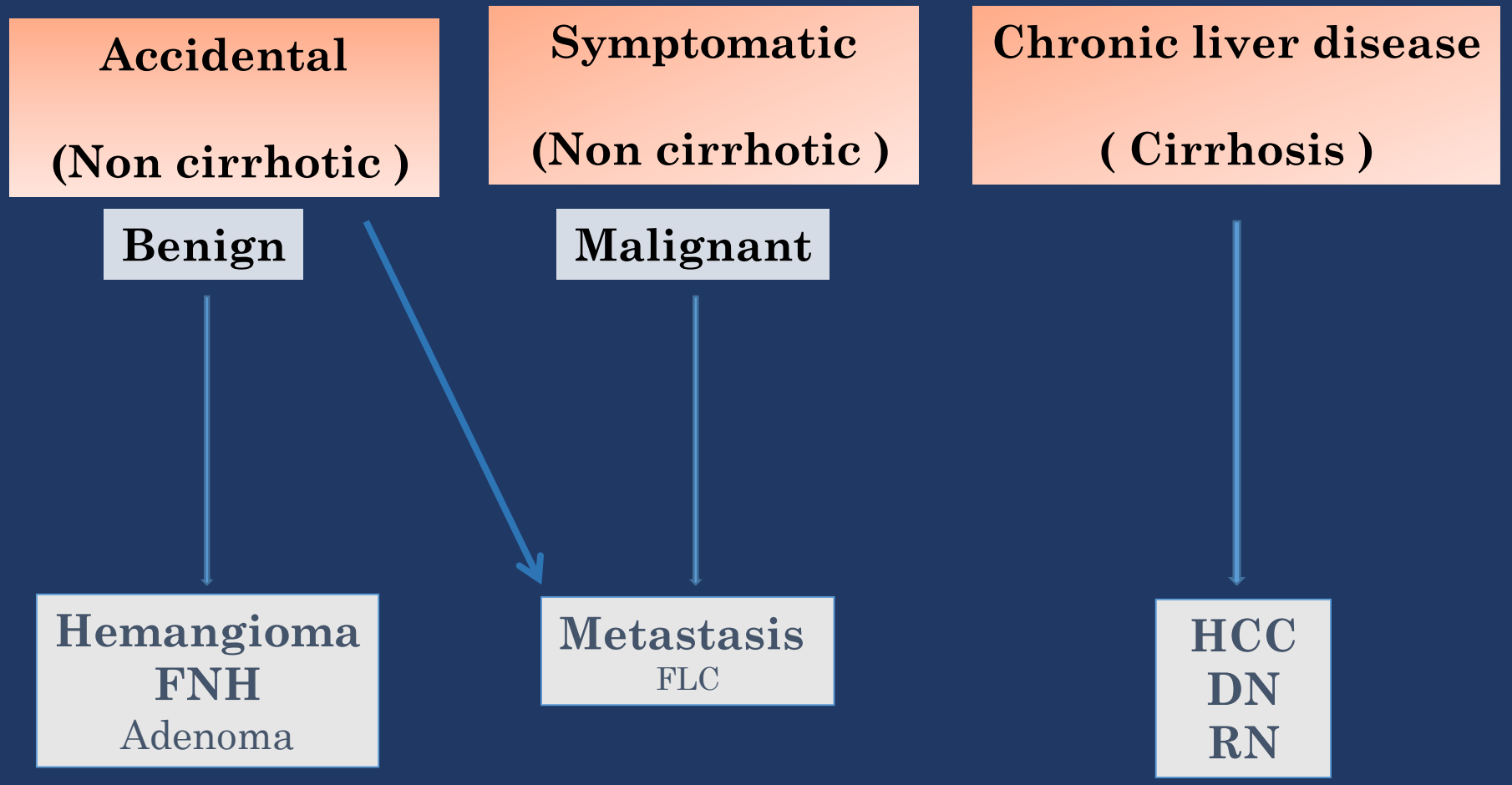
TARZAMNI, MD

Things to consider usually in liver mass

- **Symptomatic or Incidentally detected**
- **History of Hepatitis or extra hepatic malignant tumor**
- **Liver function tests**
- **Cirrhotic or Non cirrhotic**



A lesion is detected in the liver



Imaging methods:

- 1/ Ultrasound
- 2/ Ct scan
- 3/ MRI

- In most cases, liver masses are initially detected on ultrasound or single-phase CT scan.
- MRI is the best modality for characterizing liver masses, due to its improved sensitivity and temporal and contrast resolution.
- MRI is also preferred in cases when iodinated contrast is contraindicated due to allergy, or in young adults or pediatric patients. In cases of limited resource availability, however, multiphasic CT(arterial, portal venous, and delayed phases) can also adequately characterize liver masses.
- The case of MRI, either extracellular or hepatocyte-specific contrast agents (eg, gadoxetic acid) can be used. The underlying mechanism of the latter agent involves up-take and retention of the agent by functioning hepatocytes, which peaks at 20 minutes, with excretion into the biliary system.

1/ Benefits of hepatocyte-specific agents include functional assessment of liver and biliary excretion; improved sensitivity and accuracy for the detection of HCC and hypo-vascular metastases compared to CT and extracellular agents

2/ Ability to differentiate between lesions with hepatocytes that retain the agent (eg, FNH) from those that do not (ie, most adenomas) that have overlapping imaging features otherwise.

Hepatocyte-specific agents

- Hepatocyte-specific agents are not without its limitations.
- They are more expensive and require a longer imaging time.
- Arterial phase enhancement of lesions is less intense than with extracellular agents, and none hepatocyte-containing lesions will become hypointense on equilibrium phase (3-5minutes post injection) resulting in a pseudowashout appearance, which limits the assessment of lesions such as hemangiomas.
- The utility of hepatocyte-specific agents in cirrhosis is controversial; as uptake of the agent is reduced as liver function is compromised, lesion conspicuity decreases.
- Well-differentiated HCC may retain contrast on hepatocyte phase imaging and overlap in appearance with high-grade dysplasia

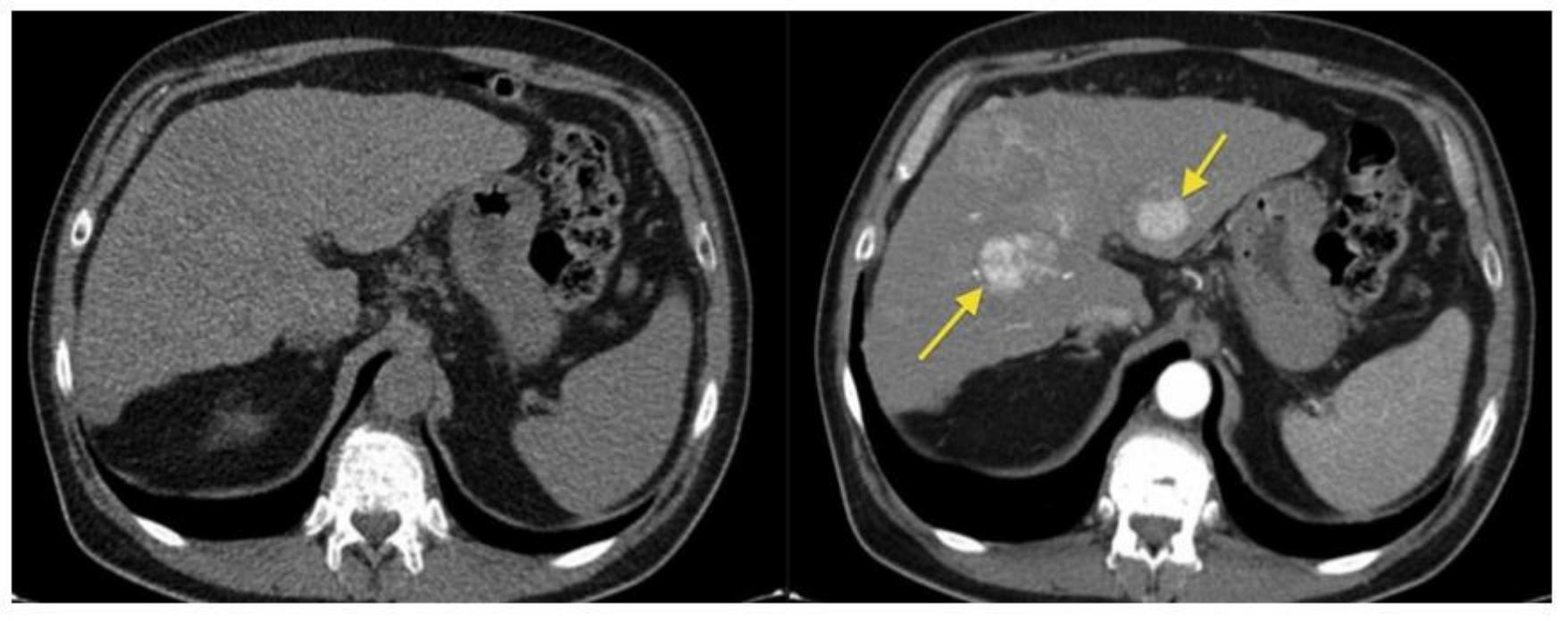
Positron emission tomography CT (PET-CT)

- PET-CT has limited utility in the diagnosis of hepatic lesions, with its primary role to look for sites of extrahepatic disease.
- A negative PET scan does not exclude malignancy (in particular, HCC with reported sensitivities of only 60%)
- A positive PET cannot differentiate among HCC, cholangiocarcinoma, or metastases.
- In addition, heterogenous background liver activity makes detection of small lesions challenging
- Benign lesions such as FNH and hemangiomas tend to uptake fluorodeoxyglucose similarly to normal liver, thus increased uptake in a hepatic lesion in a patient with known primary malignancy and no clinical features of infection is suggestive of metastases.

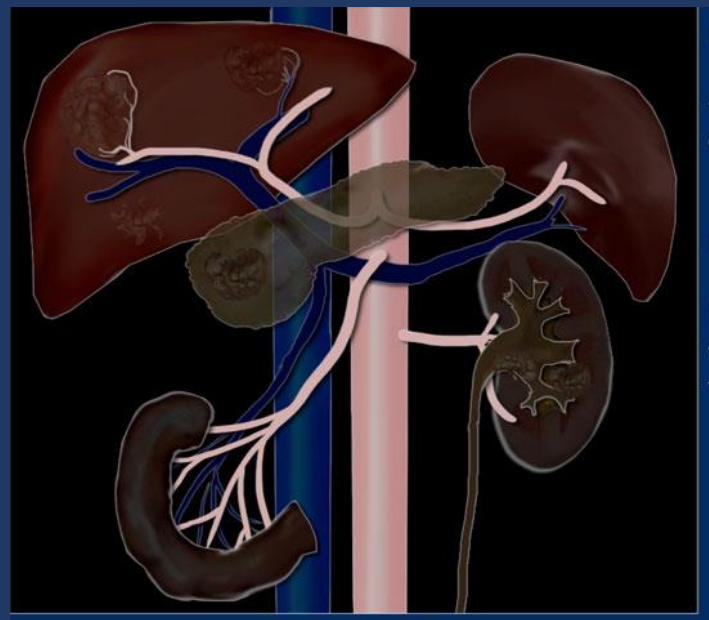
Detection of liver masses :

- The conspicuity of a liver lesion depends on the attenuation difference between the lesion and the normal liver.
  On a non enhanced CT-scan (NECT) liver tumors usually are not visible, because the inherent contrast between tumor tissue and the surrounding liver parenchyma is too low.
  Only a minority of tumors contain calcifications, cystic components, fat or hemorrhage and will be detected on a NECT.
- Normal parenchyma is supplied for 80% by the portal vein and only for 20% by the hepatic artery.
- All liver tumors however get 100% of their blood supply from the hepatic artery, so when they enhance it will be in the arterial phase.

Ct Scan:



On a non enhanced CT-scan (NECT) liver tumors usually are not visible, because the inherent contrast between tumor tissue and the surrounding liver parenchyma is too low.

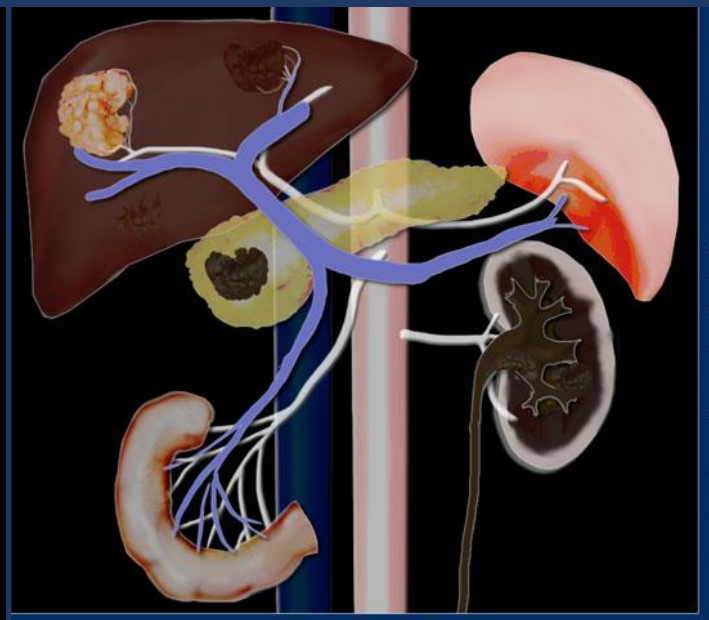


Early arterial phase

15-20 sec p.i.
or immediately
after bolustracking

Demarcation of vessels

Detection of :
- Dissection of aorta
- Arterial bleeding

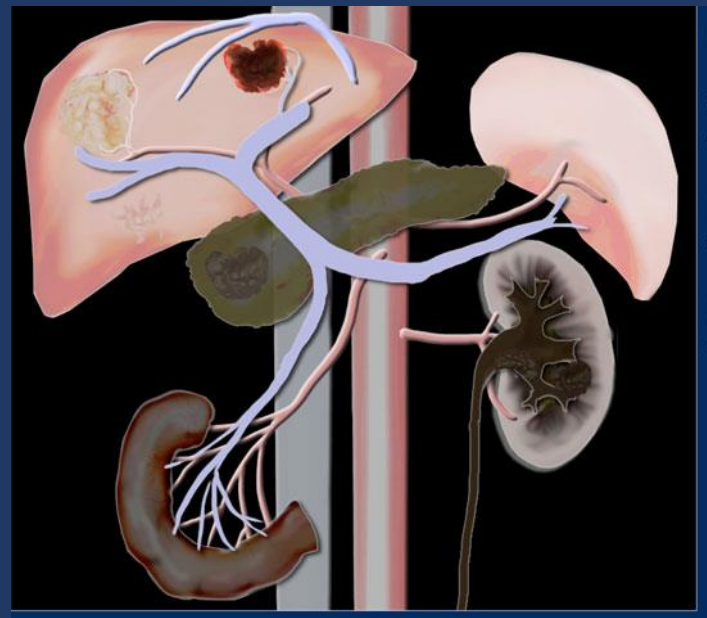


Late arterial phase

35-40 sec p.i.
or 20 sec after bolustracking

Enhancement of :
- hypervascular lesions
- stomach
- bowel
- pancreas parenchyma
- spleen
- kidney outer cortex

Detection of :
- Liver: HCC - FNH - Adenoma
- Pancreas: adenocarcinoma - Insulinoma
- Bowel ischemia

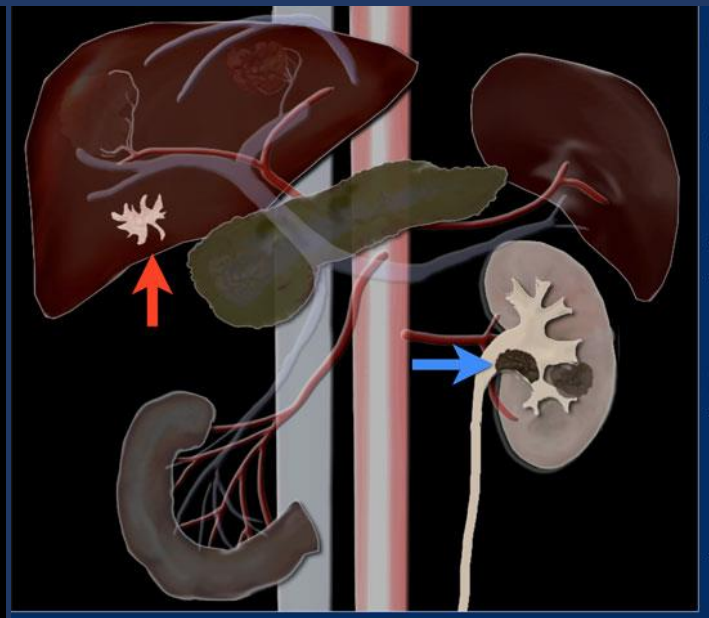


Hepatic phase

70-80 sec p.i.
or 50-60 sec after bolustracking

Enhancement of :
- Hepatic parenchyma

Detection of:
- Hypovascular liver lesions: cysts, abscess, most metastases



Delayed phase

6 minutes p.i.
or 6 minutes after bolustracking

Enhancement of :
- fibrotic lesions
- still enhancement of kidney and urinary collecting system

Detection of :
- Liver :
  - Cholangiocarcinoma (arrow)
  - Fibrotic metastases, most commonly breastcacer
- Kidney :
  - Transitional cell carcinoma (blue arrow)

CT and MRI
Dynamic Multi-phasic *Technique*

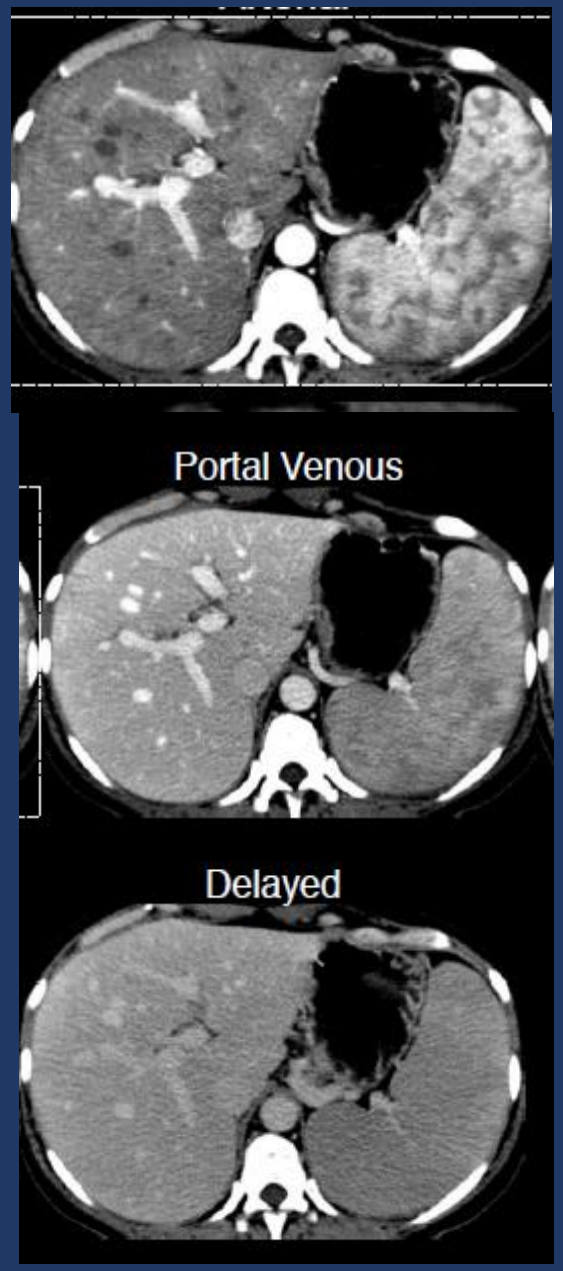
- **Non-contrast** imaging

- **arterial phase (20–40 sec post contrast injection**)

- Late hepatic arterial phase is **strongly preferred** for HCC diagnosis and staging, because the degree of enhancement in HCC usually is higher in the late than in the early hepatic arterial phase.

- **portal venous phase (60–80 sec post contrast injection)**

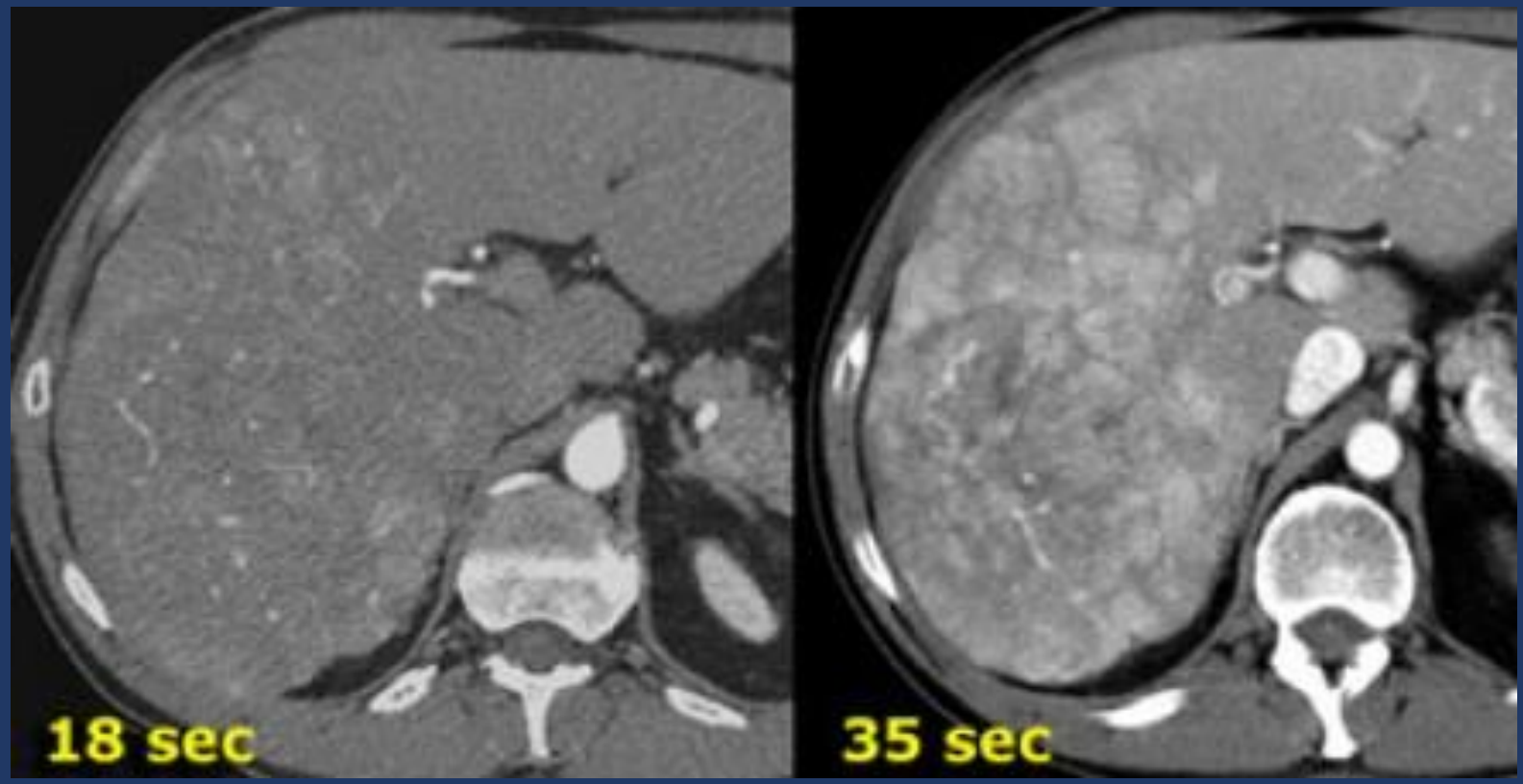
- **delayed phase(3-5 min) until 10 min**



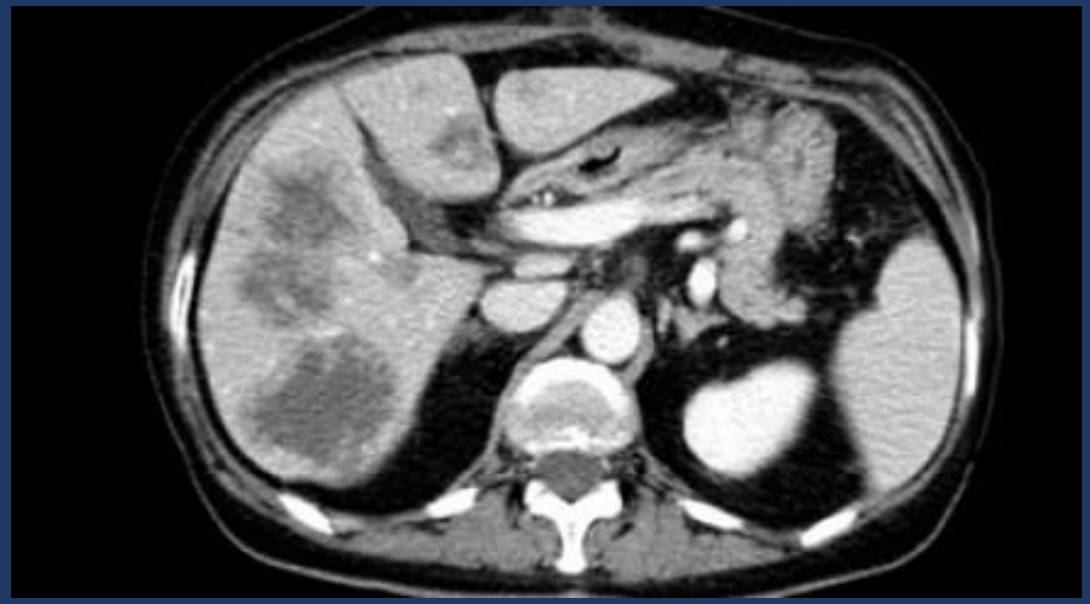
- **Late arterial phase** :strong enhancement of aorta, hepatic artery branches , and intrahepatic portal vein branches.
- Hepatic veins are not enhanced. characteristic heterogeneous enhancement of spleen.

- **Portal venous phase:** :
  - •Portal veins are fully and maximally enhanced.
  - •Hepatic veins are enhanced by antegrade flow.
  - •Liver parenchyma usually is at peak enhancement

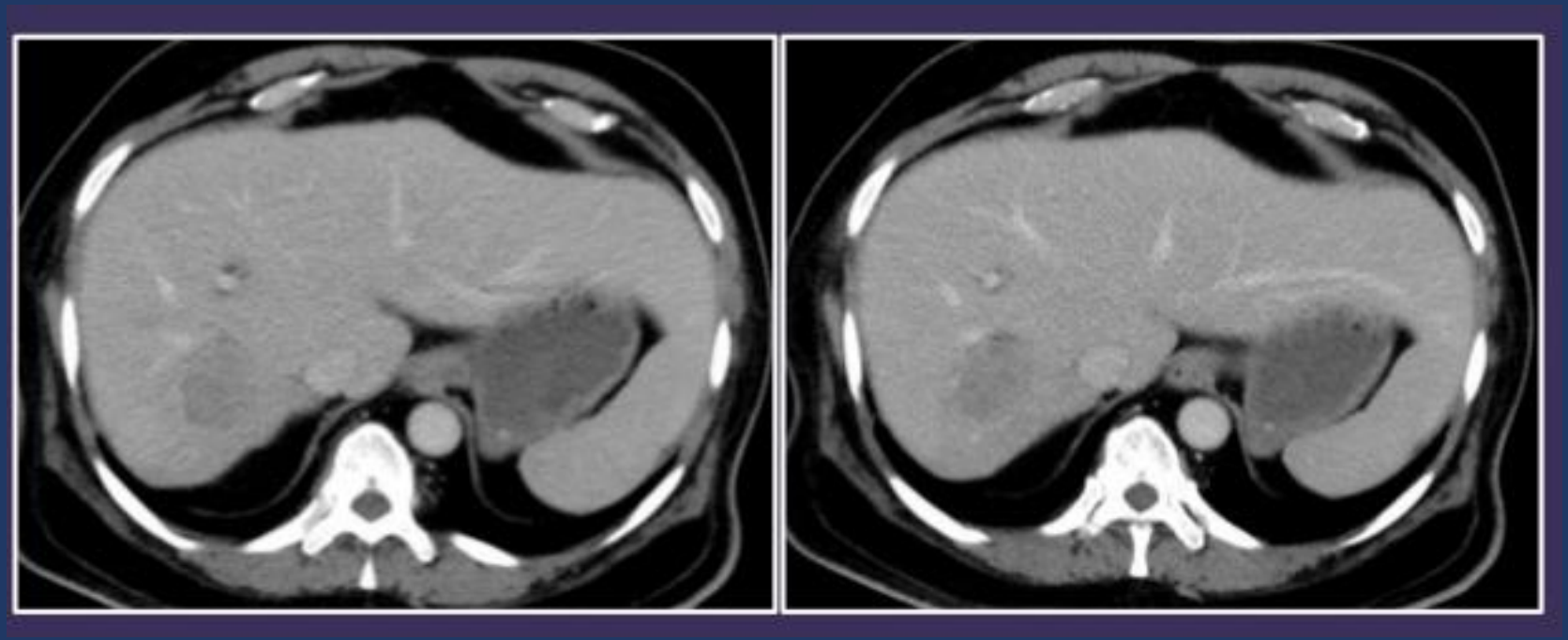
- **Delayed phase** :Portal and hepatic veins and liver parenchyma are enhanced but less than in portal venous phase.



- In the **arterial phase,** hypervascular tumors will enhance via the hepatic artery, when normal liver parenchyma does not yet enhances, because contrast is not yet in the portal venous system.
  These hypervascular tumors will be visible as hyperdense lesions in a relatively hypodense liver.
  However when the surrounding liver parenchyma starts to enhance in the portal venous phase, these hypervascular lesion may become obscured.
- Notice that in the late arterial phase there has to be some enhancement of the portal vein.
  The only time that an early arterial phase is needed is when you need an arteriogram, for instance as a roadmap for chemoembolization of a liver tumor.



- In In the **portal venous phase** hypovascular tumors are detected, when the normal liver parenchyma enhances maximally.
  These hypovascular tumors will be visible as hypodense lesions in a relatively hyperdense liver.
- The best moment to start scanning is at about 75 seconds, so this is a late portal venous phase.
- This late portal venous phase is also called the hepatic phase because there already must be enhancement of the hepatic veins.



- **equilibrium phase:** This phase begins after **3 to 4 minutes** of administering the contrast and the best imaging results are obtained at about 10 minutes of contrast injection.
  These lesions will become either relatively hyperdense or hypodense to the normal liver.
- This phase can be valuable if you're looking for: fast tumor washout in hypervascular tumors like HCC or retention of contrast in the blood pool as in hemangiomas or the retention of contrast in fibrous tissue in capsules (HCC) or scar tissue (FNH, Cholangiocarcinoma).

Liver mass enhancement

- Lesion vascularity: This assessment can only be made on hepatic arterial-dominant (HAD)-phase images and is a subjective comparison with background tissue
- The reference tissue of the lesion is the peripheral portion of the tumor, which is the most vascularized region of tumors.
- This is subdivided into hypovascular → less than background liver; isovascular → the same as background liver (may be seen in small HCCs or chemotherapy-treated metastases); and hypervascular → greater than background liver.
- A hypervascular tumor, which possesses only a thin peripheral rim of hypervascular tissue on HAD images, may be considered, perhaps incorrectly, as a hypovascular tumor.
- Generally, 1 cm thick peripheral tissue should be considered to reflect the vascularity, although at times we have used even 5 mm if it is especially hypervascular.

Perilesional enhancement.

- Enhancement observed beyond the confines of the tumor. Lesional versus perilesional can at times be difficult to distinguish, and correlation with precontrast images may be required: comparing the size of the area of enhancement with the size of the lesion on precontrast (we usually favor in-phase and out-of-phase images).
- Perilesional may be subdivided into thin rim (e.g., biliary hamartoma), circumferential (e.g., colon cancer metastases, bacterial abscess, lymphoma), or wedge shaped (e.g., pancreatic adenocarcinoma metastases).
- Most forms of perilesional enhancement fade (especially inflammatory, but also malignancy related), but some are persistent (biliary hamartoma). Most are also ill-defined and relatively large in size (inflammatory, malignancy related), while biliary hamartoma is very thin

Ring enhancement

- Enhancement of the outer, most vascularized portion of the tumor on HAD-phase images. This is the classic appearance for metastases and may be seen in any lesion that seeds the liver (travels from one site, even within the liver, to another site), such as abscesses, lymphoma, intrahepatic liver metastases from HCC. Uniform ring may be seen with metastases or abscesses. Irregular (scalloped) rings are usually seen with metastases.
- Nodular ring enhancement is seen in hemangiomas. Note that scalloped and nodular enhancement may at times be difficult to distinguish; see the sections “Hemangiomas” and “Liver metastases” for differentiating properties.

Diffuse homogeneous enhancement.

Uniform lesion enhancement on HAD-phase images. This is classic for adenoma, FNH, type 1 small hemangiomas, high-grade DNs, small HCC, and small hypervascular metastases.

Diffuse heterogeneous enhancement.

Enhancement throughout the tumor on HAD-phase images but with irregular distribution of enhancement. This is classic for large HCC; may be seen in any large malignant tumor of primary liver origin.

Diffuse mosaic (or patterned) enhancement.

Enhancement throughout the tumor on HAD-phase images but with a regular appearance or pattern of enhancement. This is a rare appearance. This may imply the tumor is benign, because of the regular organized pattern of the vessels (e.g., angiomyolipoma); however, this can be simulated by some malignant tumors, such as leiomyosarcoma.

Progressive enhancement.

Progressive enhancement often is used interchangeably for retention of contrast. This is the classic appearance for hemangioma, cholangiocarcinoma and for chemotherapy-treated metastases. Subdivision of types of progressive enhancement are therefore important, as described in the following.

Progressively intense capsule or septal enhancement without enhancement of previously unenhanced stroma is classic for inflammatory conditions, in particular bacterial abscesses. The abscess wall, and internal septations if present, becomes progressively more intense on venous-phase images (up to 3 min), but there is no enhancement of additional stroma that was not enhanced on arterial-phase images. This is a critical distinguishing feature from hypovascular, noncystic metastases, which will show enhancement of stroma over time that was not enhanced on arterial-phase images.

Centripetal enhancement refers to progressive enhancement of the more central portion of a tumor over time. This classically has been used to describe hemangiomas. The problem with this term is that metastases will often also show centripetal enhancement. As a result, we prefer the terms enlargement and coalescence of nodules when describing hemangiomas, and progressive central enhancement for metastases

Washout.

Decrease in signal on serial images to below the signal of liver.

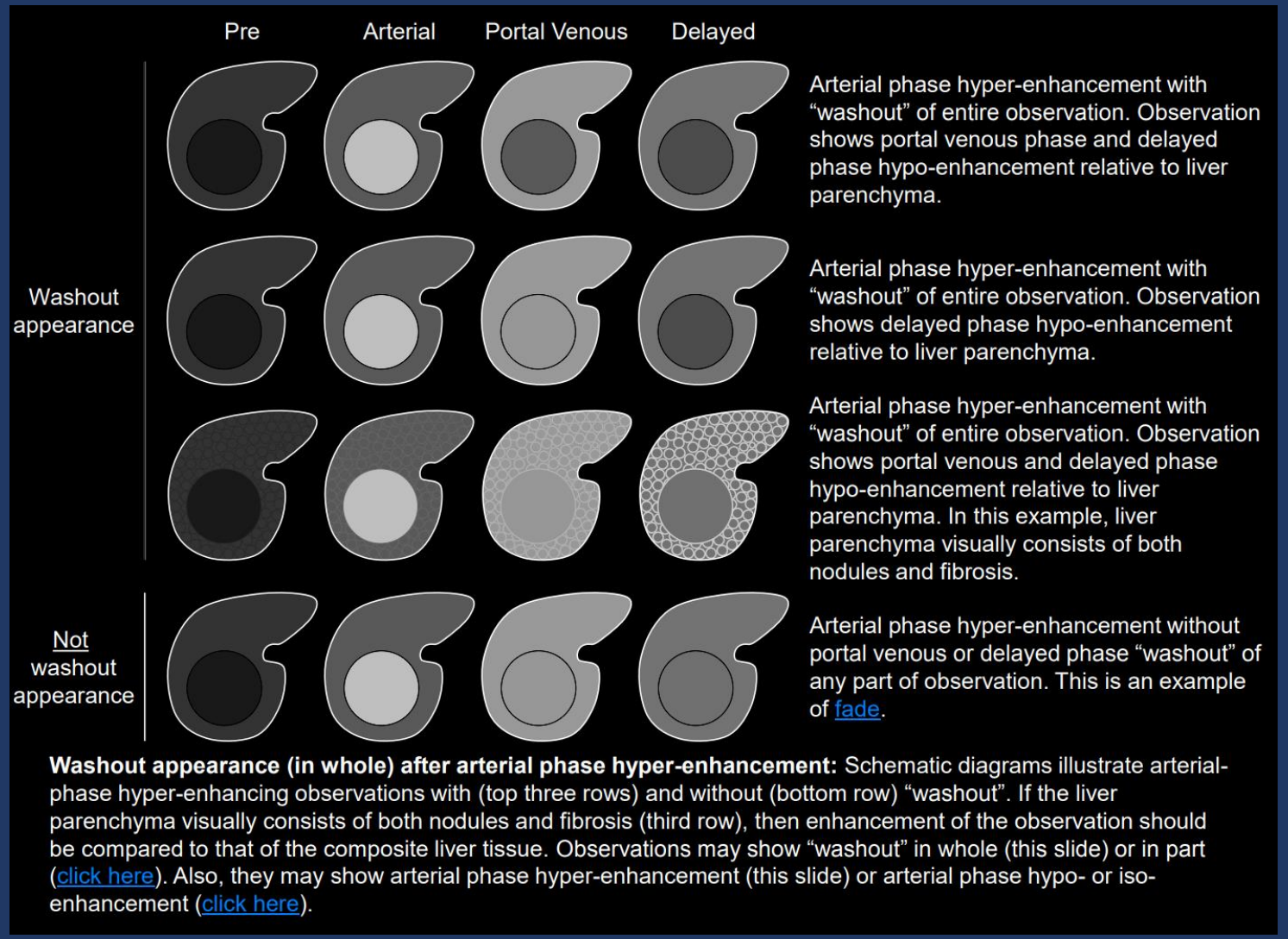
• Uniform washout is classic for HCC; also seen in carcinoid and other neuroendocrine tumor metastases.

• Peripheral washout may be seen in hypervascular metastases. The classic is gastrinoma.

• Delayed capsule is the appearance of a rim around the lesion on venous-phase images. This is classic for HCC, but also seen in carcinoid metastases.

• Persistent capsule is the appearance of a capsule on immediate postcontrast images that remains largely unchanged on later venous phases. For benign lesions this is classic for biliary hamartoma. For malignant lesions this may be seen in cystic metastases (e.g., ovarian cancer) and may also be seen in colon cancer metastases and hypovascular cholangiocarcinoma. In the case of colon cancer metastases, this rim is in addition to the peripheral rim of enhancement with centripetal enhancement

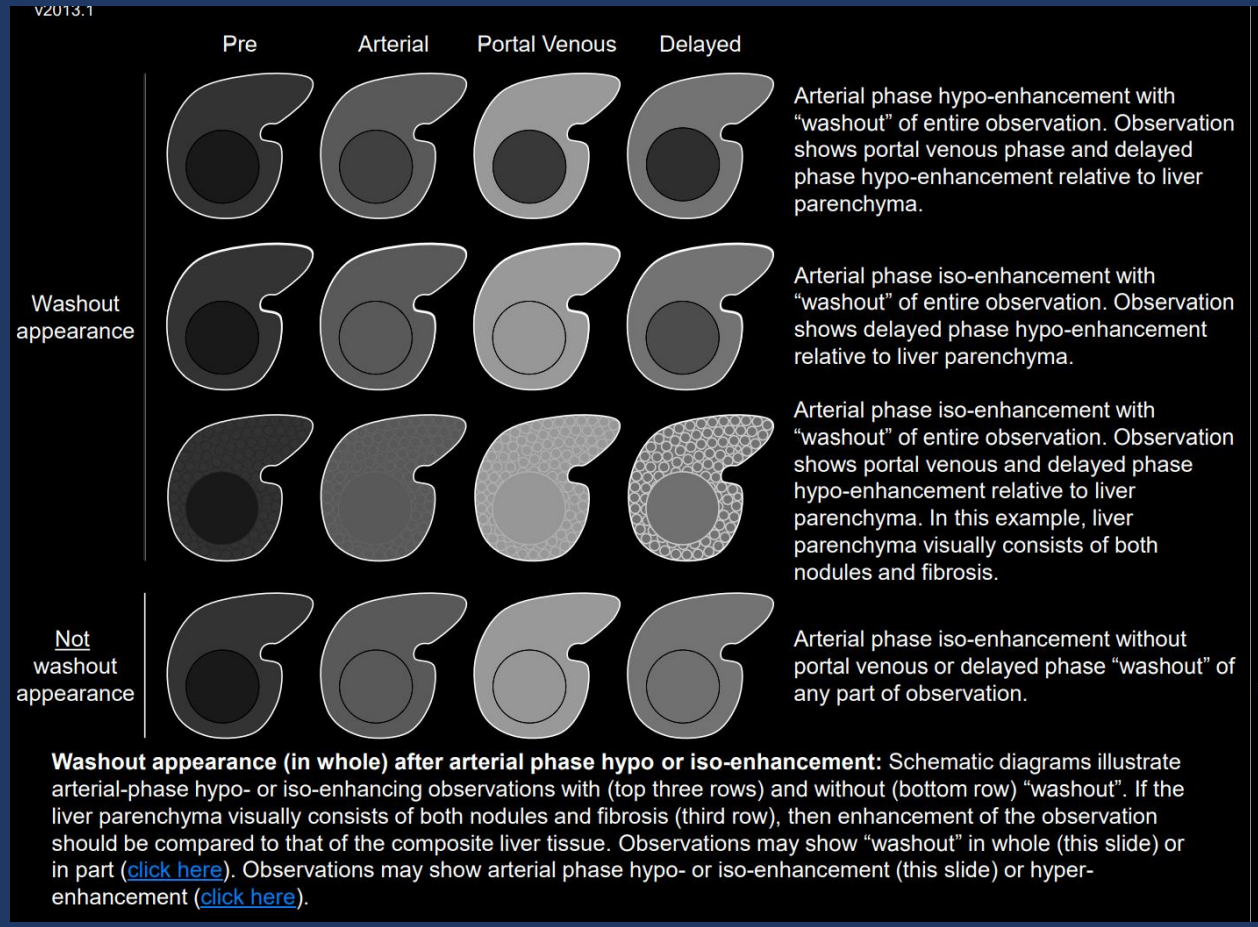
Wash out



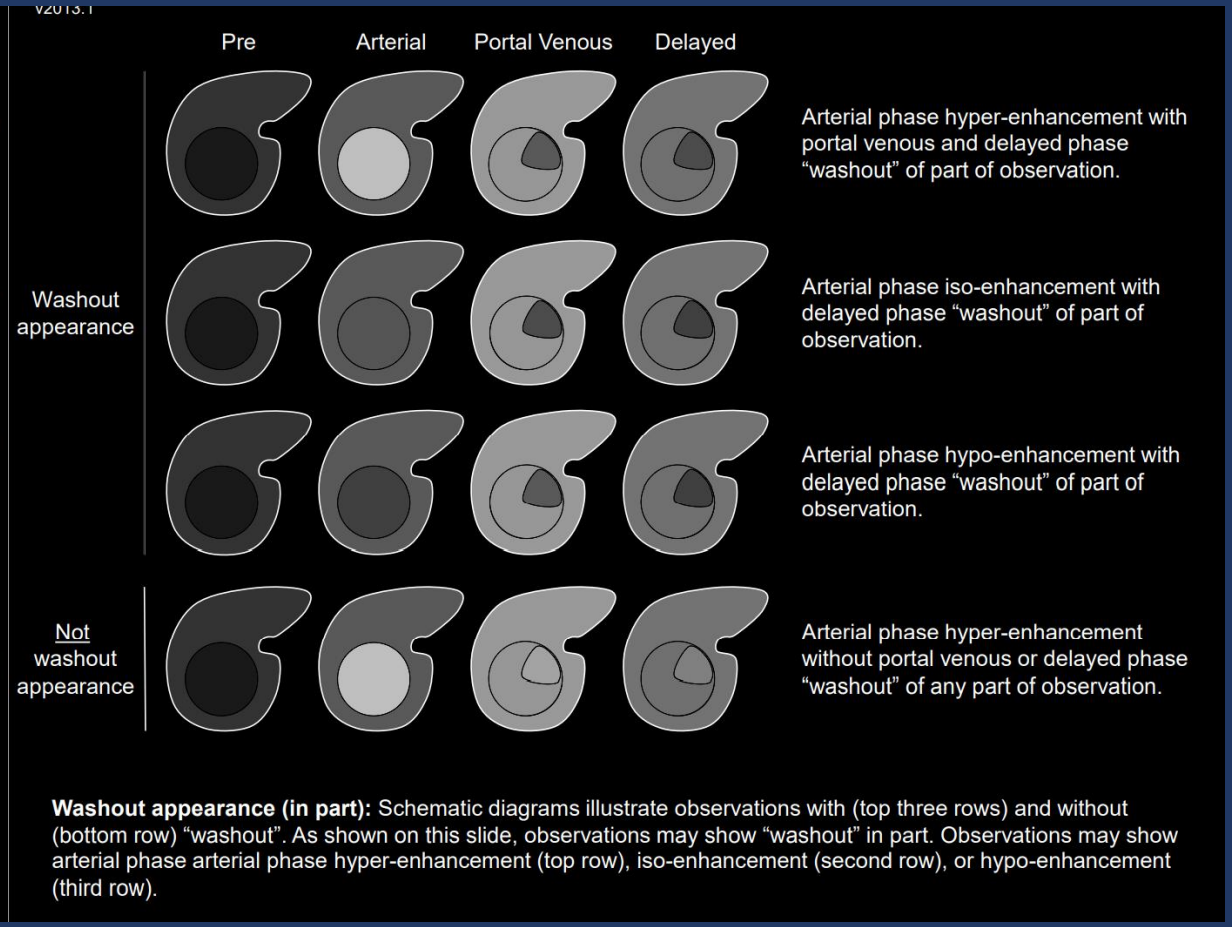
Washout appearance (in whole) after arterial phase hyper-enhancement: Schematic diagrams illustrate arterial-phase hyper-enhancing observations with (top three rows) and without (bottom row) "washout". If the liver parenchyma visually consists of both nodules and fibrosis (third row), then enhancement of the observation should be compared to that of the composite liver tissue. Observations may show "washout" in whole (this slide) or in part (click here). Also, they may show arterial phase hyper-enhancement (this slide) or arterial phase hypo- or iso-enhancement (click here).

Visually assessed temporal reduction in enhancement relative to liver from an earlier to a later phase resulting in portal venous phase hypoenhancement or delayed phase hypoenhancement.

Wash out

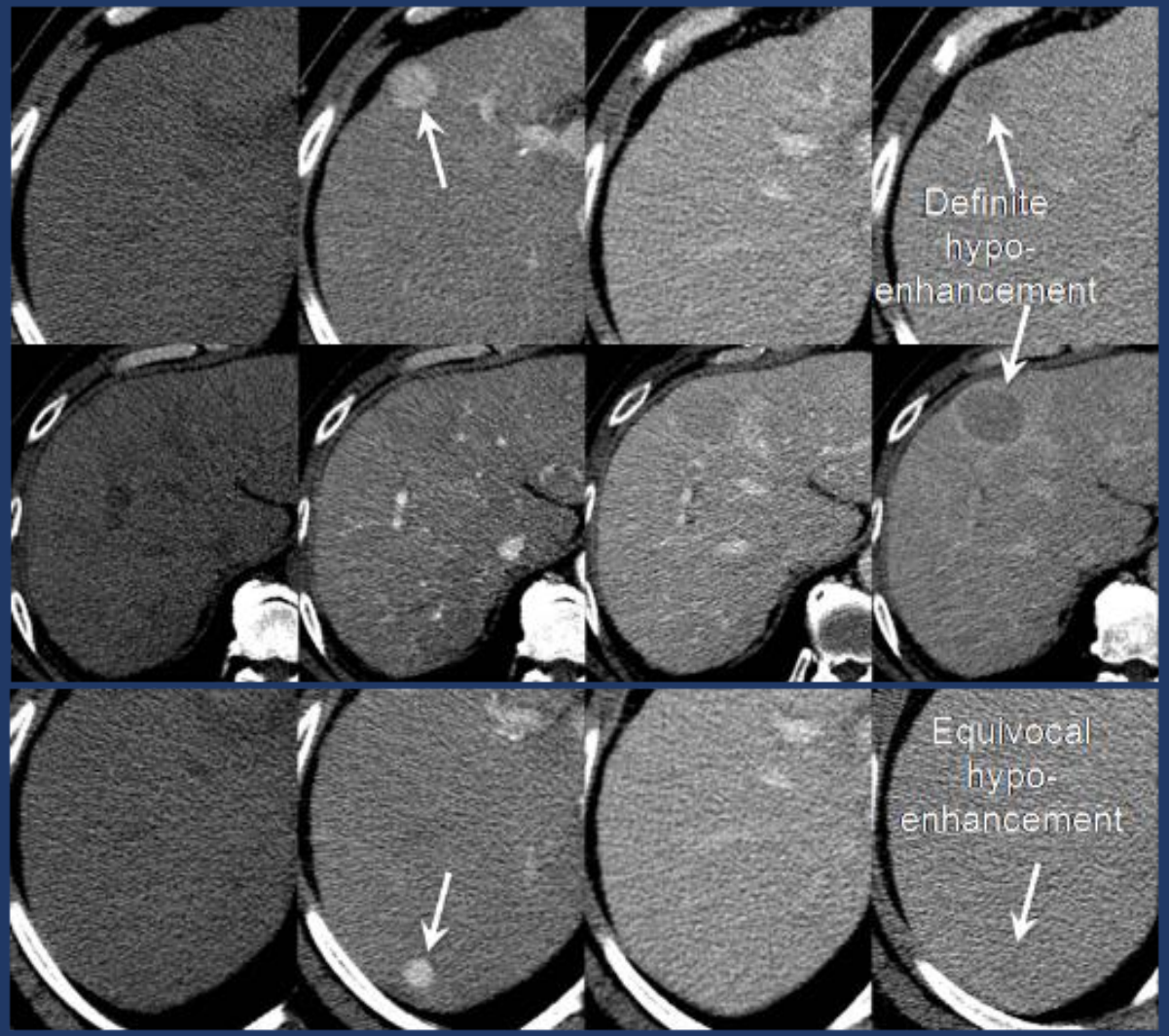


Washout appearance (in whole) after arterial phase hypo or iso-enhancement: Schematic diagrams illustrate arterial-phase hypo- or iso-enhancing observations with (top three rows) and without (bottom row) "washout". If the liver parenchyma visually consists of both nodules and fibrosis (third row), then enhancement of the observation should be compared to that of the composite liver tissue. Observations may show "washout" in whole (this slide) or in part (click here). Observations may show arterial phase hypo- or iso-enhancement (this slide) or hyper-enhancement (click here).

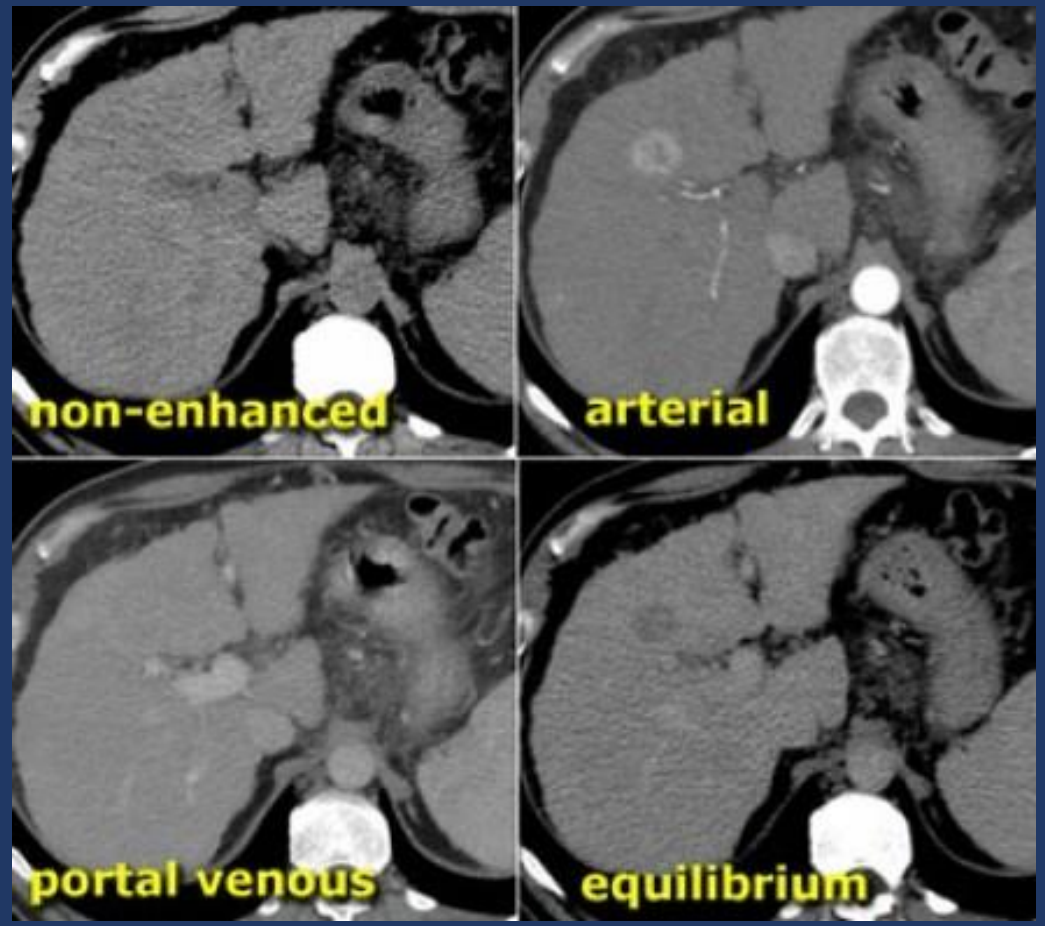


Washout appearance (in part): Schematic diagrams illustrate observations with (top three rows) and without (bottom row) "washout". As shown on this slide, observations may show "washout" in part. Observations may show arterial phase arterial phase hyper-enhancement (top row), iso-enhancement (second row), or hypo-enhancement (third row).

Washout appearance:



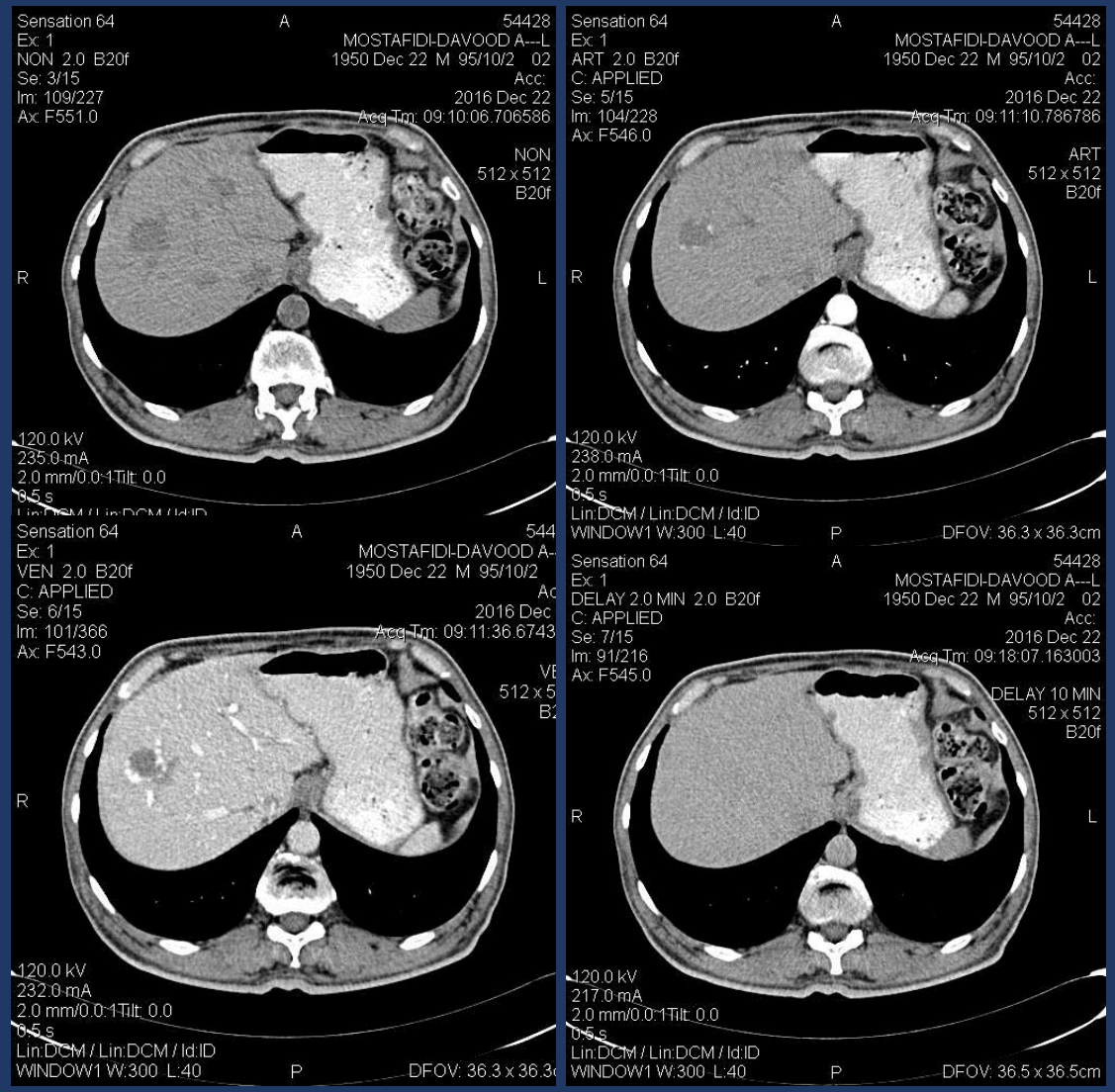
Relative hypodense lesions in the delayed phase (washed out)

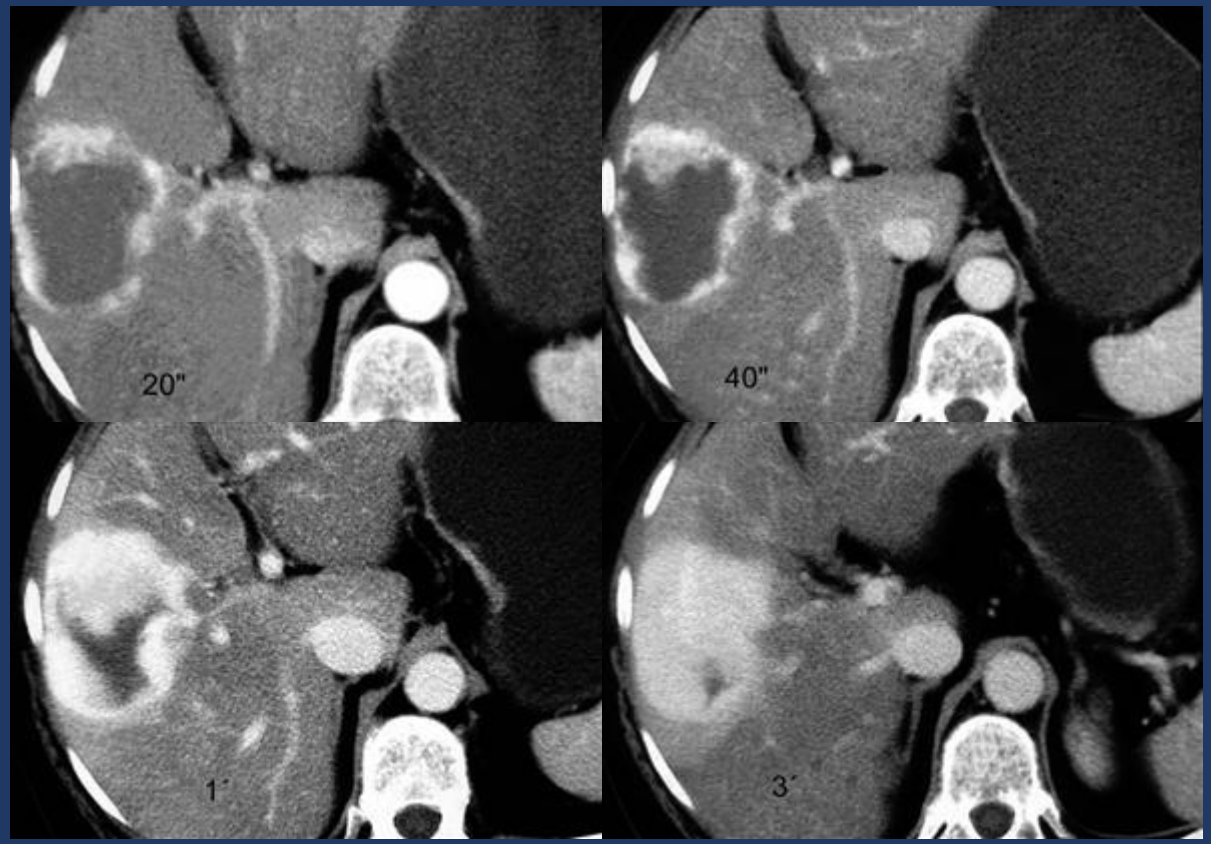


Now the issue at hand is in small enhancing lesions in a cirrhotic liver whether it is a benign lesion like a regenerating nodule or a HCC.
In the delayed phase we see that the tumor is washed out more than the surrounding liver parenchyma.

Blood pool

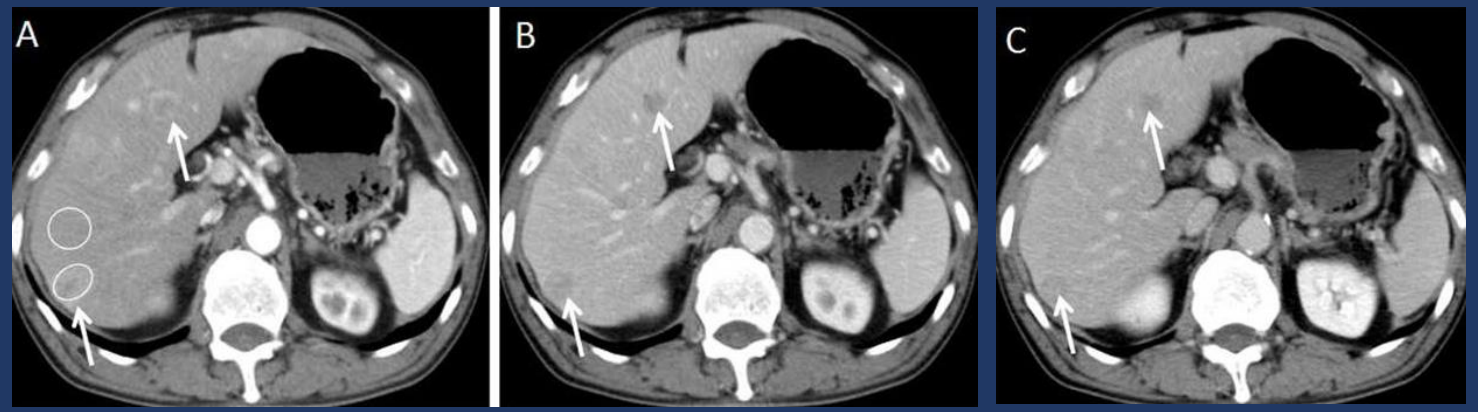
- Normally when we look at lesions filling with contrast, the density of these lesions is always compared to the density of the liver parenchyma.
- In hemangiomas however should not compare the density of the lesion to the liver, but to the blood pool.
- This means that the areas of enhancement in a hemangioma should match the attenuation of the appropriate vessels (bloodpool) at all times.
- in the arterial phase the enhancing parts of the lesion must have almost the same attenuation value as the enhancing aorta.
- in the portal venous phase it must match the enhancement of the portal vein.
- If it does not match the bloodpool in every single phase of contrast enhancement forget the diagnosis of a hemangioma.



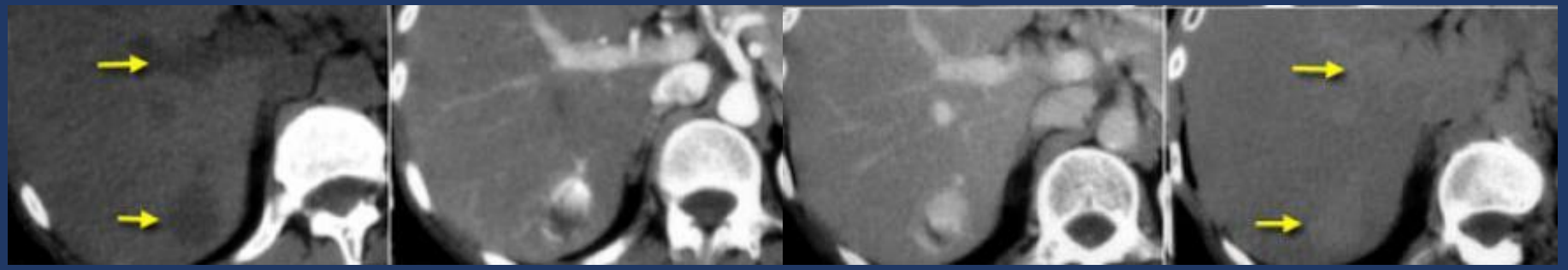


- Hemangioma on NECT, late arterial, late portal venous and equilibrium phase. Notice that the attenuation of the hemangioma matches the bloodpool in every single phase.

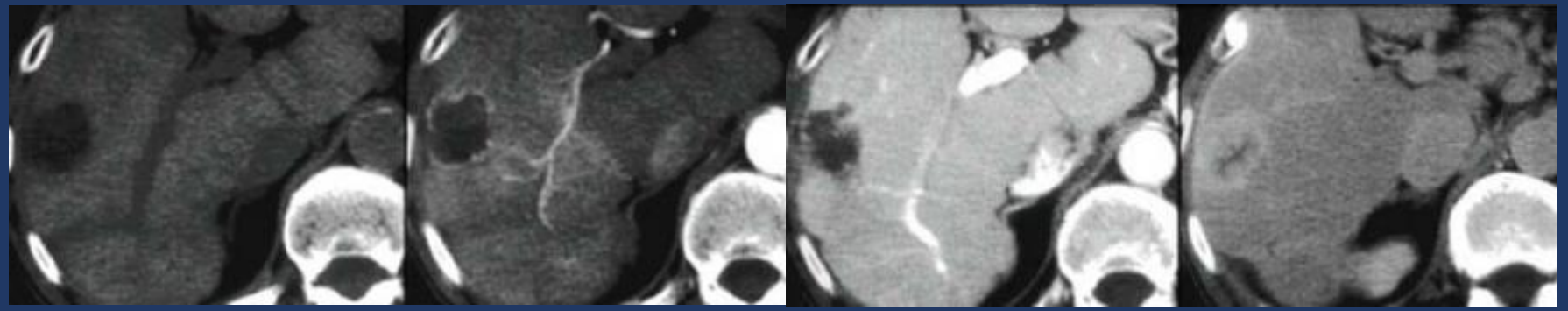
Progressive fill in:



The pattern of a peripheral, discontinuous, intense nodular enhancement during the arterial-dominant phase with progressive centripetal fill-in is considered pathognomic for hemangiomas



Metastasis
lesion does not match bloodpool in all phases, so it cannot be a hemangioma.



Progressive fill in is a non-specific feature, that can be seen in many other **lesions** like metastases or primary **liver tumors** like cholangiocarcinoma.
The delayed enhancement in this **lesion** is due to fibrotic tissue in a cholangiocarcinoma and is a specific feature of these **tumors**.

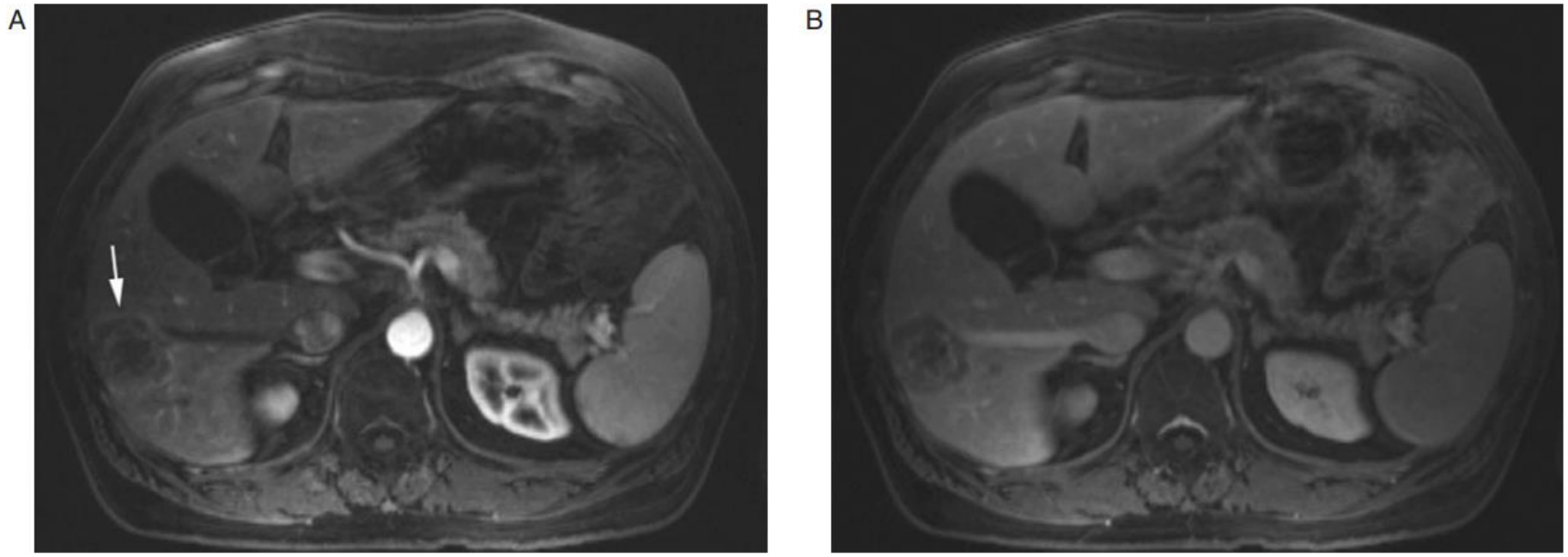
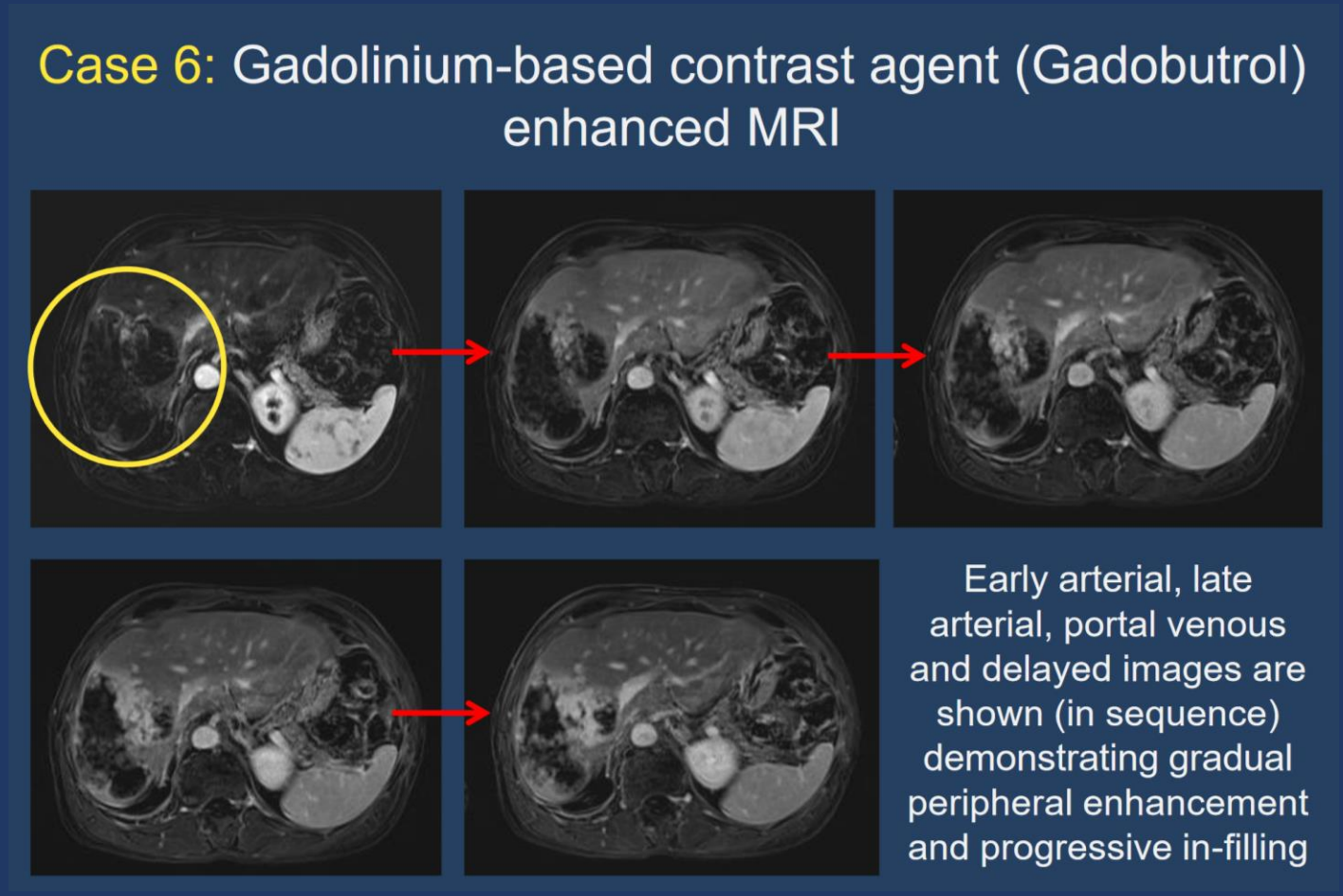


Figure 8 **Metastatic colon carcinoma. T1-weighted arterial phase gadolinium-enhanced MR image (A) shows a heterogeneously enhancing mass (arrow) in the right lobe of the liver. On an equilibrium phase image (B) the periphery of the lesion, which demonstrated enhancement during the arterial phase, is now less intense than the center of the lesion. This phenomenon is termed 'peripheral washout'.**

Peripheral washout on delayed images is a finding that is characteristic of malignancy and can be seen in intrahepatic cholangiocarcinoma and some hepatic metastases. This finding refers to a peripheral rim that is hypointense or hypoattenuating to the center of the lesion on delayed contrast enhanced MR or CT images

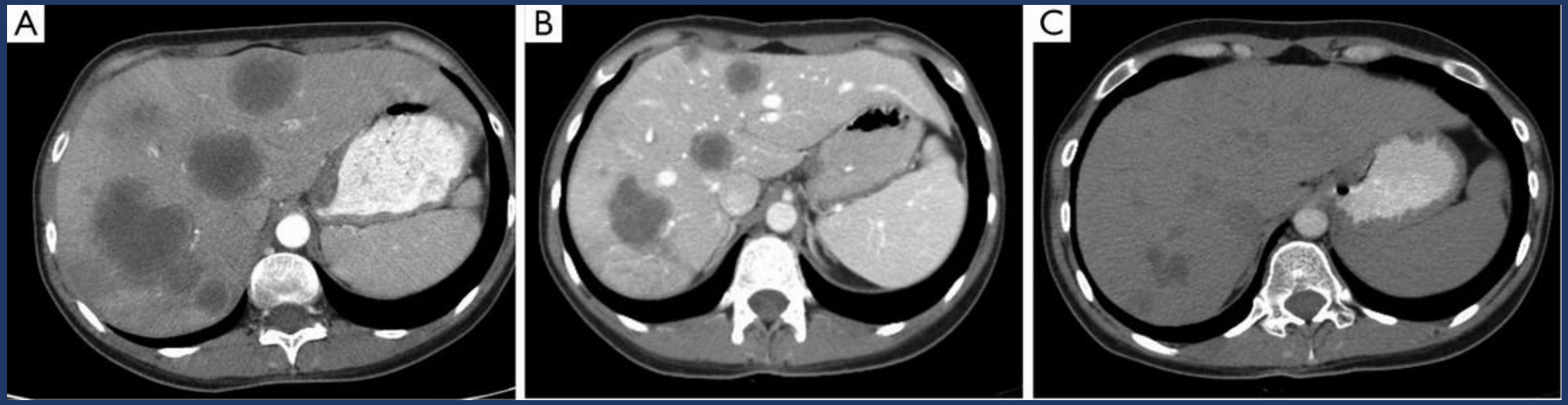
progressive in-filling



Hepatic Angiosarcoma

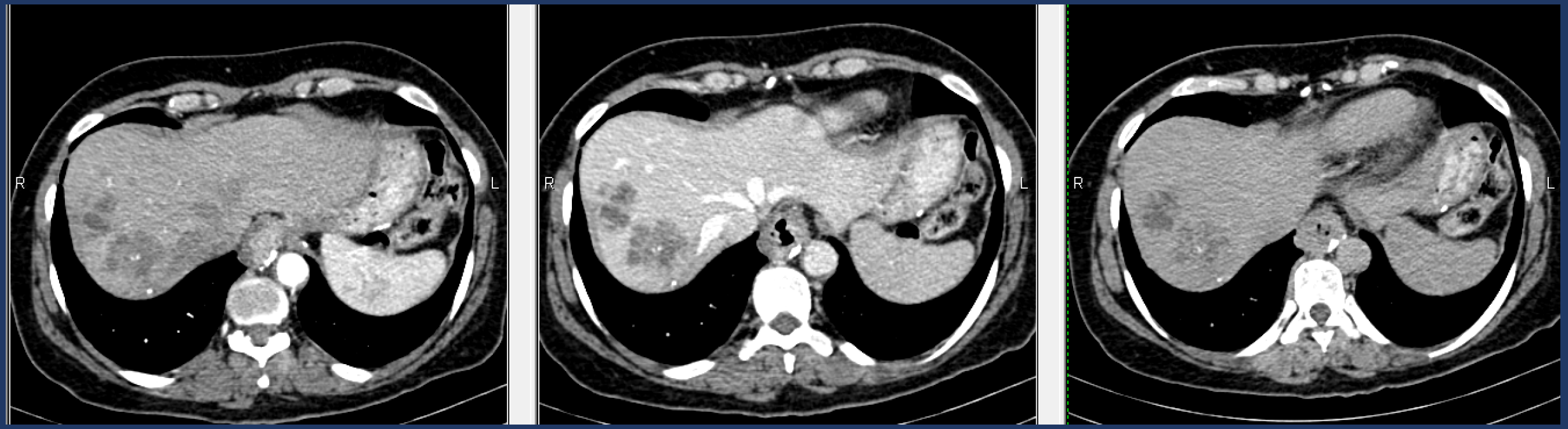
Imaging features: nodular enhancement is common with progressive in-filling, fluid-fluid levels may be present (haemorrhage), lesions are typically FDG PET avid•
Differential: hemangioma, hypervascular metastases, atypical HCC

Progressive fill in (colorectal cancer)



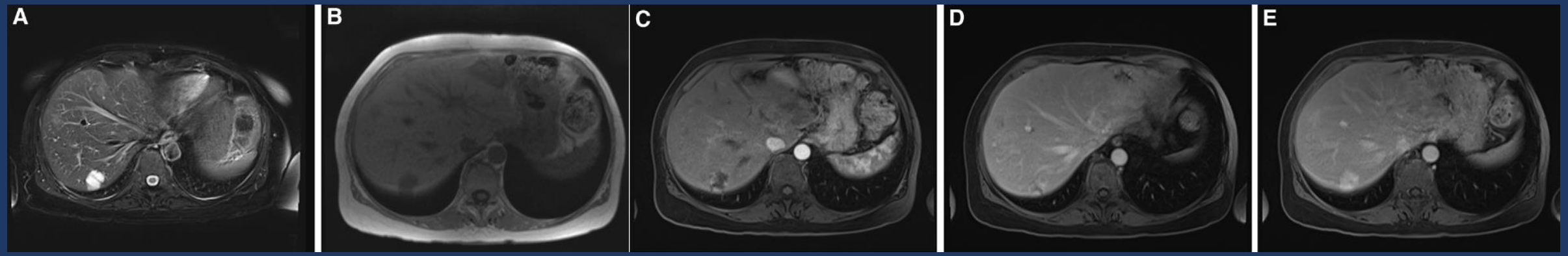
Enhancement is typically peripheral, and although there may be central filling in, on portal venous phase, the delayed phase will show washout; helpful in distinguishing a metastasis from a hemangioma.

Progressive fill in (colorectal cancer)

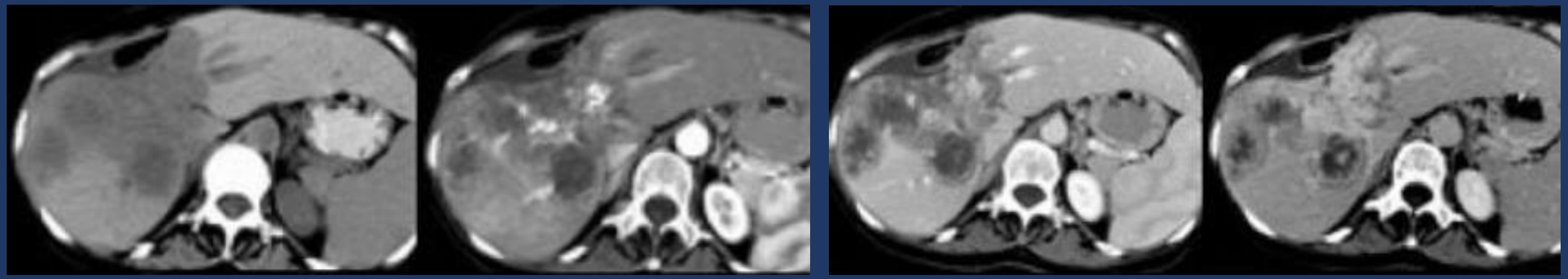


Enhancement is typically peripheral, and although there may be central filling in, on portal venous phase, the delayed phase will show washout; helpful in distinguishing a metastasis from a hemangioma.

Persistence enhancement: Relative hyperdense lesions in the delayed phase

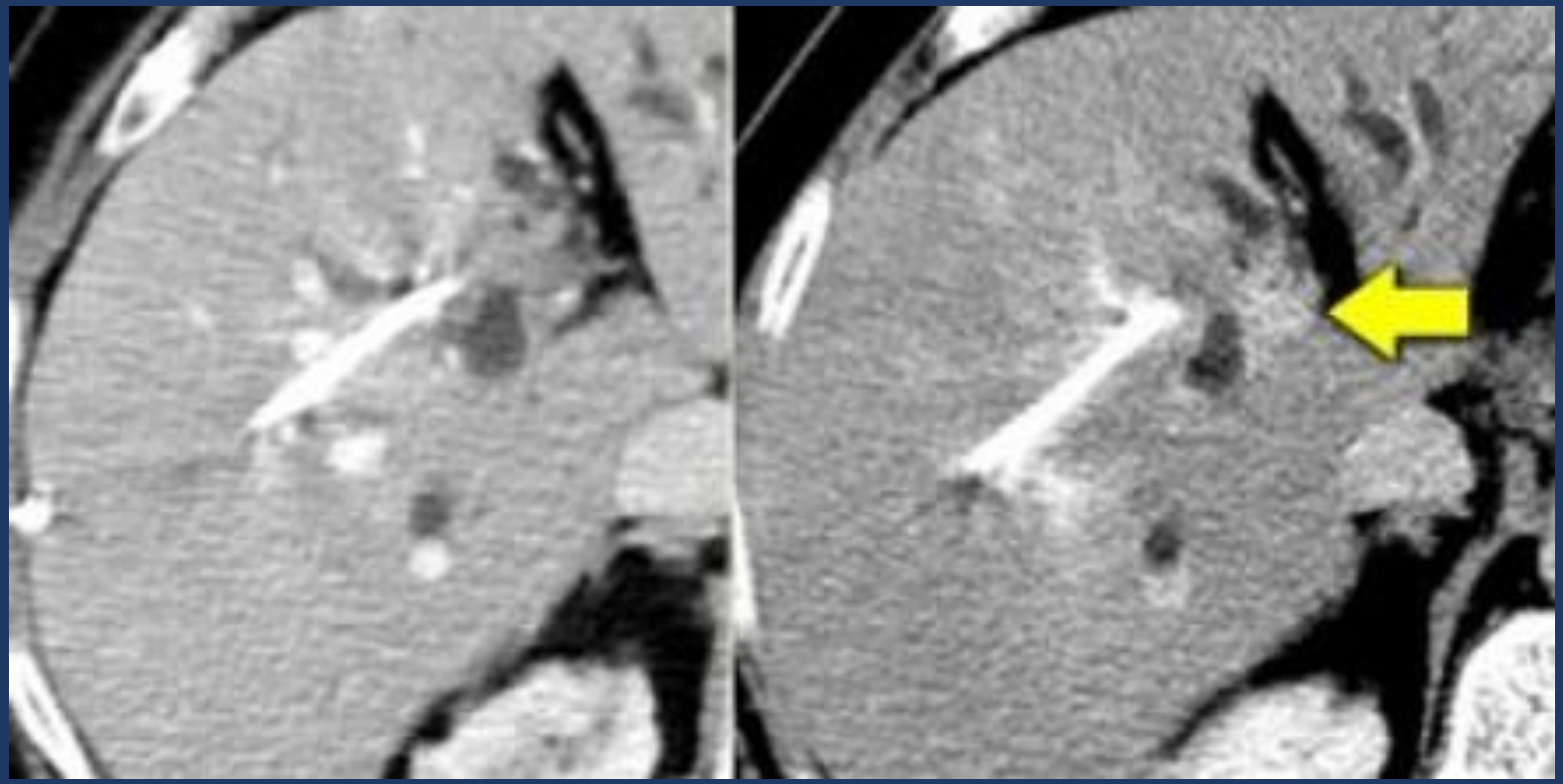


Hemangioma



delayed persistent enhancement (fibrous tissue, scar)
Cholangiocarcinoma

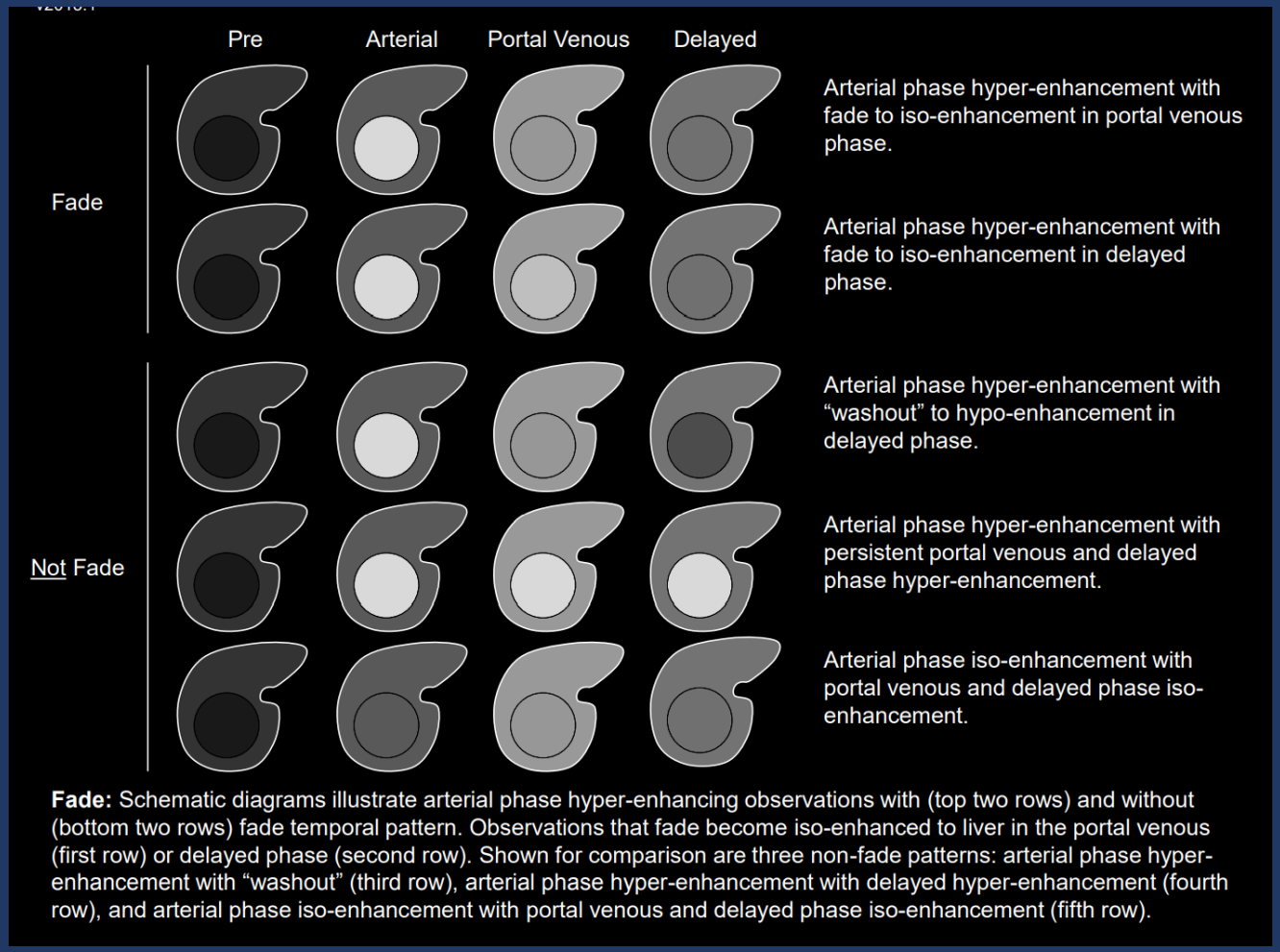
Relative hyperdense lesions in the delayed phase



CHOLLANGIOCARCINOMA

Faded:

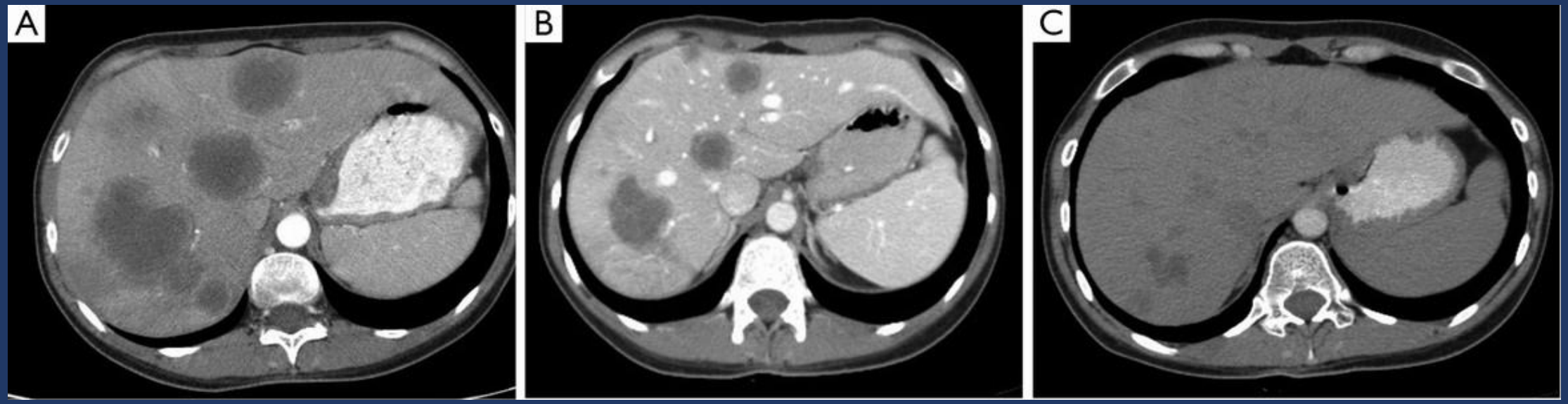
This refers to the lesion decreasing in signal to background (normal) liver on serial postgadolinium images. This is classic for FNH.



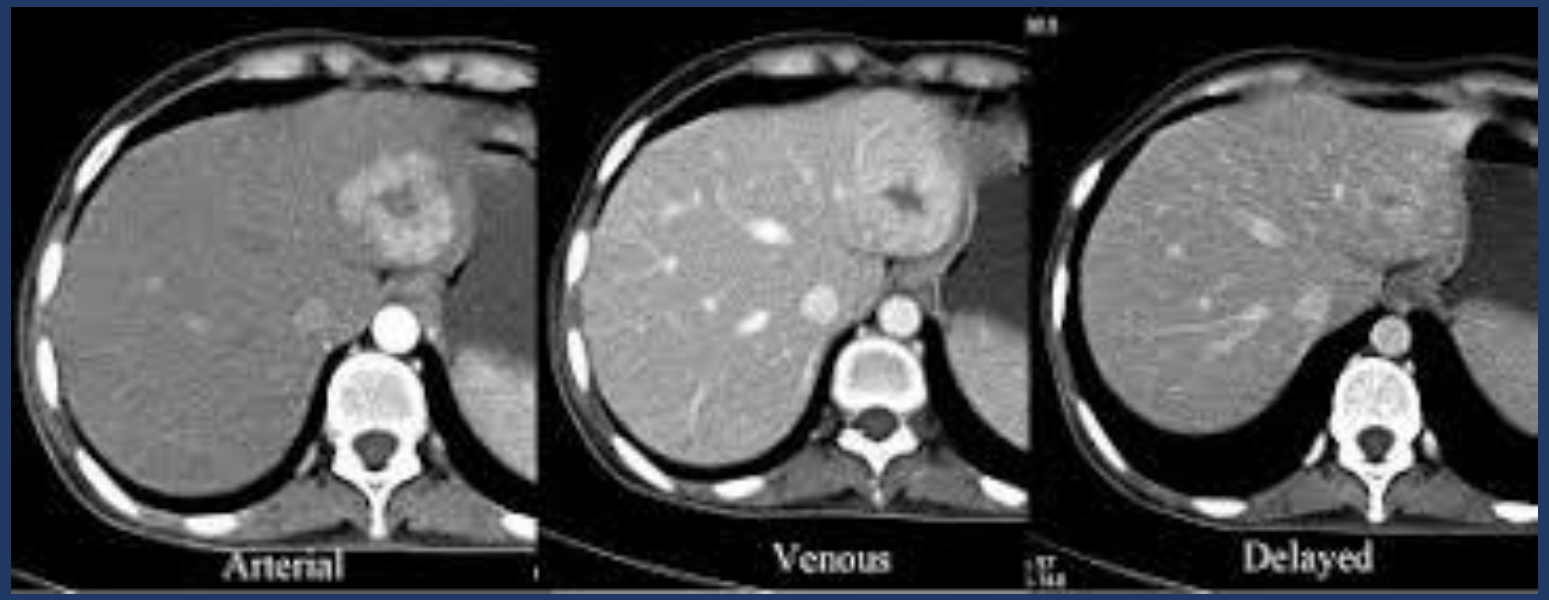
Fade: Schematic diagrams illustrate arterial phase hyper-enhancing observations with (top two rows) and without (bottom two rows) fade temporal pattern. Observations that fade become iso-enhanced to liver in the portal venous (first row) or delayed phase (second row). Shown for comparison are three non-fade patterns: arterial phase hyper-enhancement with "washout" (third row), arterial phase hyper-enhancement with delayed hyper-enhancement (fourth row), and arterial phase iso-enhancement with portal venous and delayed phase iso-enhancement (fifth row).

Temporal reduction in enhancement from hyperenhancement in the arterial phase to iso enhancement or faint residual hyper-enhancement in later phases.
This pattern may be observed with perfusion alterations (e.g., arterio-portal shunts), some small hemangiomas (more frequently at CT than MRI), FNH-like lesions,some dysplastic nodules, and some small HCCs.

Faded:

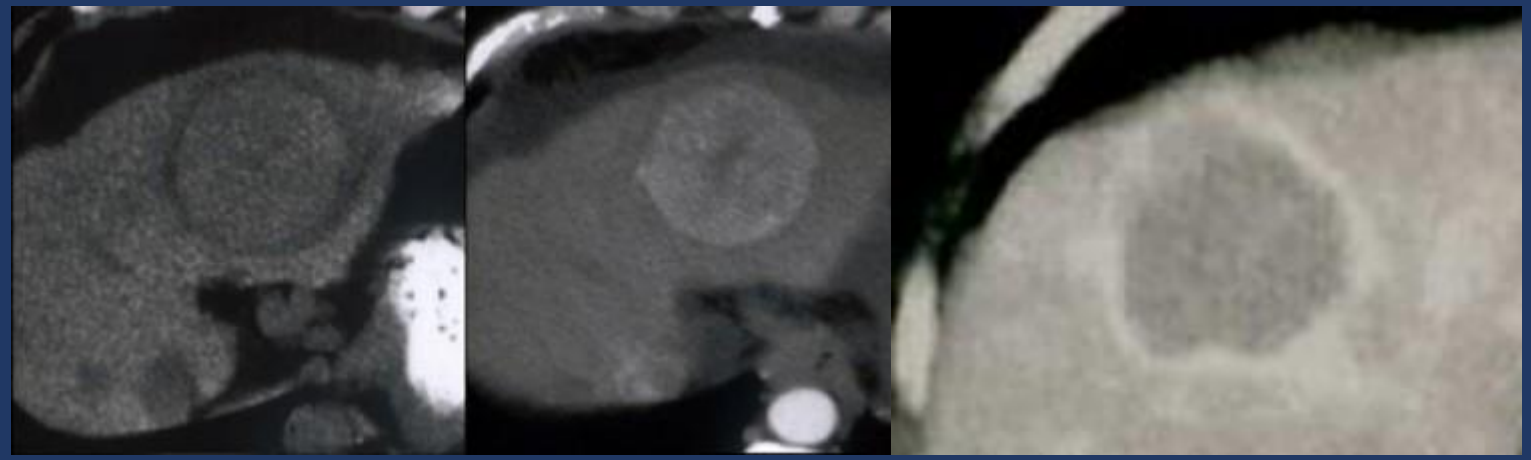


Scar:

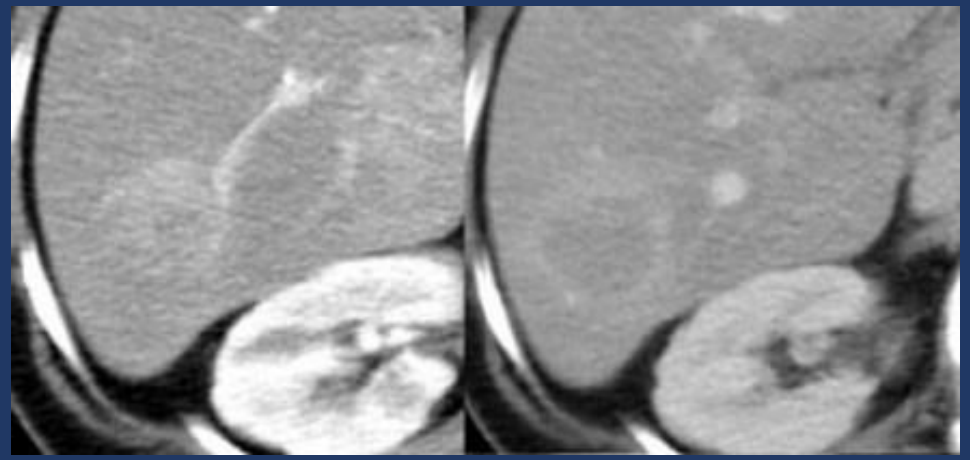


- Central scar :FNH, fibrolamellar carcinoma, cholangiocarcinoma, hemangioma and hepatocellular carcinoma.
- On CT a scar is sometimes visible as a hypodense structure.
- On MR scar tissue is hypointense on both T1WI and T2WI due to intense fibrotic changes.
- An exception: the central scar in FNH is hyperintense on T2WI due to edema.
- T2WI can be very helpful if there is a problem in differentiating FNH from FLC.
- Both on CT and MRI scar tissue will enhance in the delayed phase.

Capsule:



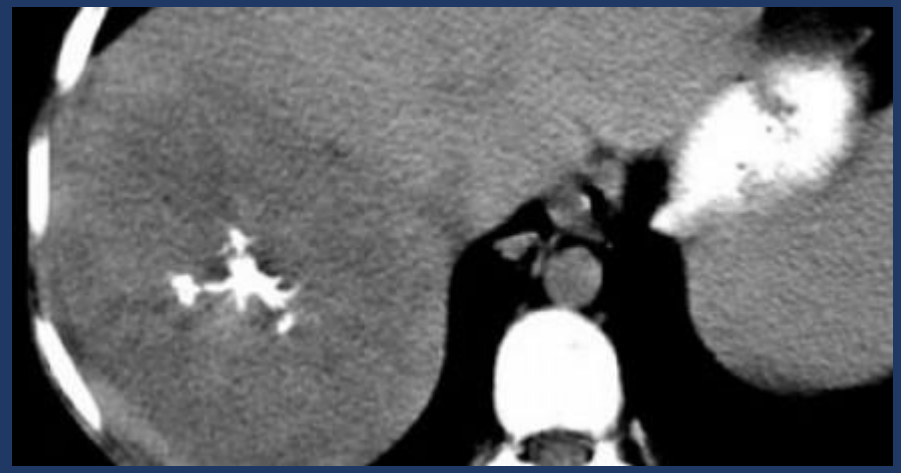
HCC



Adenoma (thin rim)

- Liver lesions which may have a capsule are Adenoma, HCC and cystadenoma or cystadenocarcinoma.
- The most common tumor with a capsule is HCC.
- The capsule will not enhance in the arterial phase and even in the portal venous phase it will be hypodense, because the fibrous tissue enhances very slowly.
- A capsule is usually best seen in the delayed phase as a relative hyperdense structure.

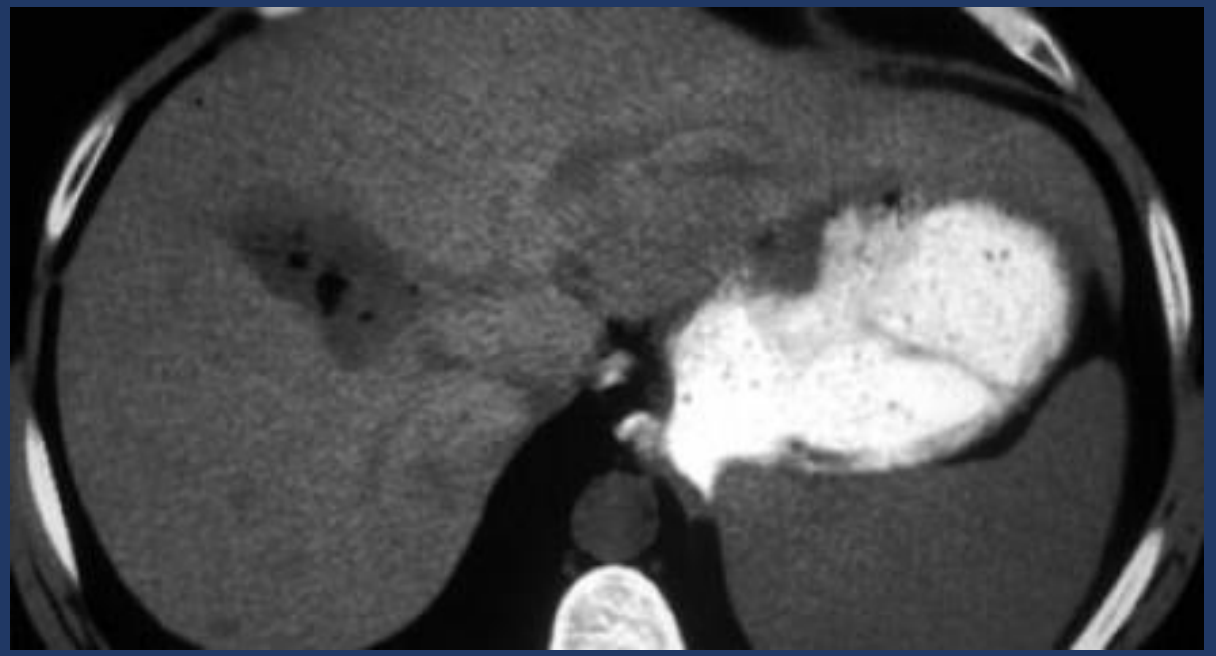
Calcifications



- Central calcifications are seen in:
  - Metastases (especially in colorectal tumors)
  - Fibrolamellar carcinoma (FLC)
  - Cholangiocarcinoma
  - Hemangiomas
- These calcifications are hyperdense on CT and hypointense on T1 and T2 MR images.

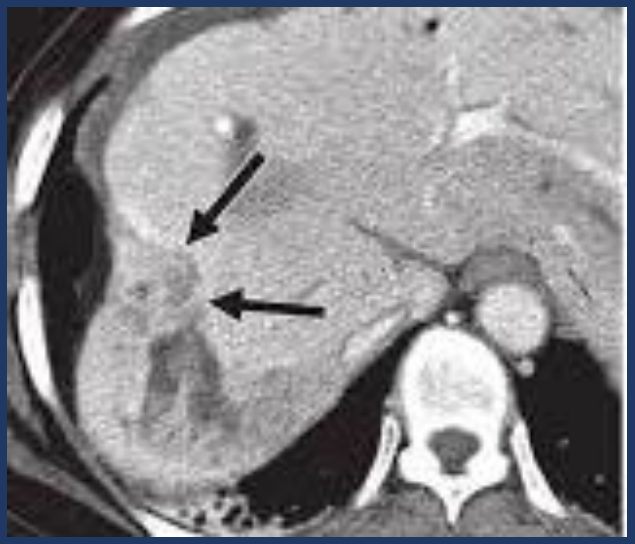
Fat:

- Adenoma
- HCC
- Metastatic liposarcoma
- Angiomyolipoma



Hemorrhage

- Adenoma (Hemorrhage is most commonly seen in adenomas)
- HCC



Retraction of liver capsule

- Some tumors however have an infiltrative growth pattern with a lot of fibrous tissue and do not cause mass effect.
- The most common tumor however to cause retraction is cholangiocarcinoma.
- Breast cancer metastases can be infiltrative. When they shrink they can cause multiple retractions.
  This will give a pseudo-cirrhosis appearance.

Enhancement

- Heterogen
- Peripheral regular
- Peripheral irregular
- Peripheral nodular

Focal liver mass:

- First step : lesion is cyst or hemangioma (two common liver masses)
- Hemangioma ruled out with peripheral nodular enhancement with centripetal extension and the same density as the bloodpool in all phases.
- Second step:

Hypervascular mass with arterial enhancing

- Small hemangioma
- FNH
- Adenoma
- HCC
- FLC
- Hypervascular metastasis

Hypovascular mass most apparent in portal phase

- Metastasis
- Peripheral cholangiocarcinoma
- Lymphoma

Malignant hepatic lesions

- Primary
- secondary

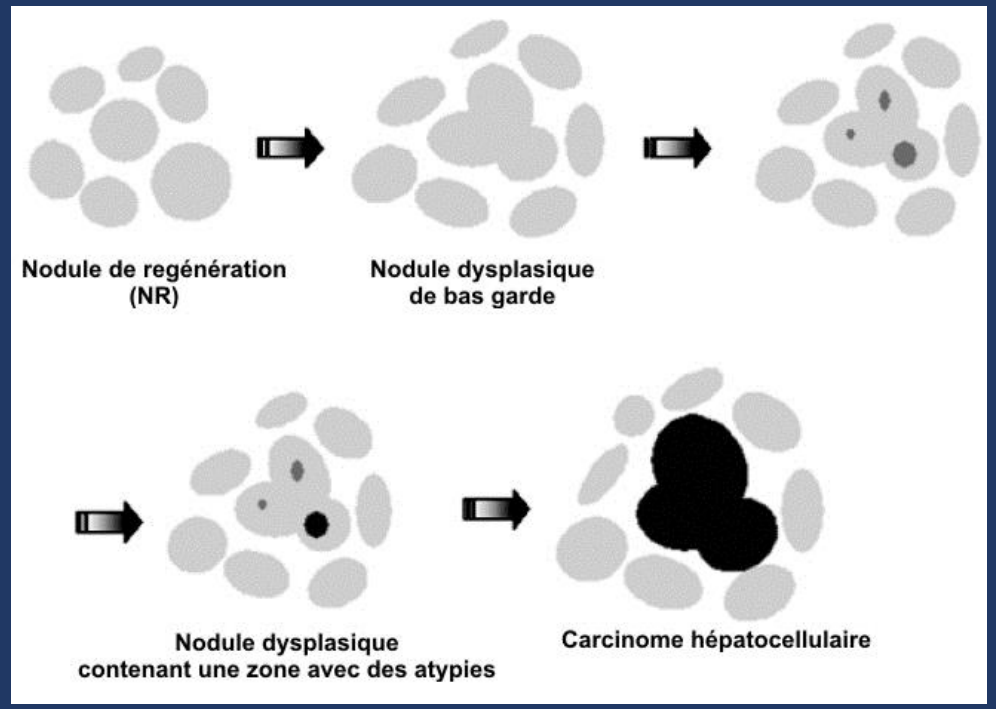
- Hypervascular
- Hypovascular

Hepatocellular Carcinoma (HCC)

- The fifth most common tumor
- Rarely occurs before age of 40 and peaks at 70 years
- Male to female: 4/1
- Cirrhosis is the strongest predisposing factor for HCC
- 80% of cases of HCC developing in a cirrhotic liver
- Causes of cirrhosis: hepatitis (B and C virus infection), alcohol, Hemochromatosis, biliary cirrhosis and aflatoxin, and possibly obesity and diabetes.

Two types of human hepatocarcinogenesis are now recognized:
de novo carcinogenesis and multistep carcinogenesis in a liver with cirrhosis or chronic hepatitis.
Most HCCs develop by means of a multistep progression:dysplastic nodule (DN), early HCC, well-differentiated HCC, moderately or poorly differentiated HCC
(from a low-grade dysplastic nodule to a high-grade dysplastic nodule, to a dysplastic nodule with a focus of HCC, and finally to overt carcinoma.)

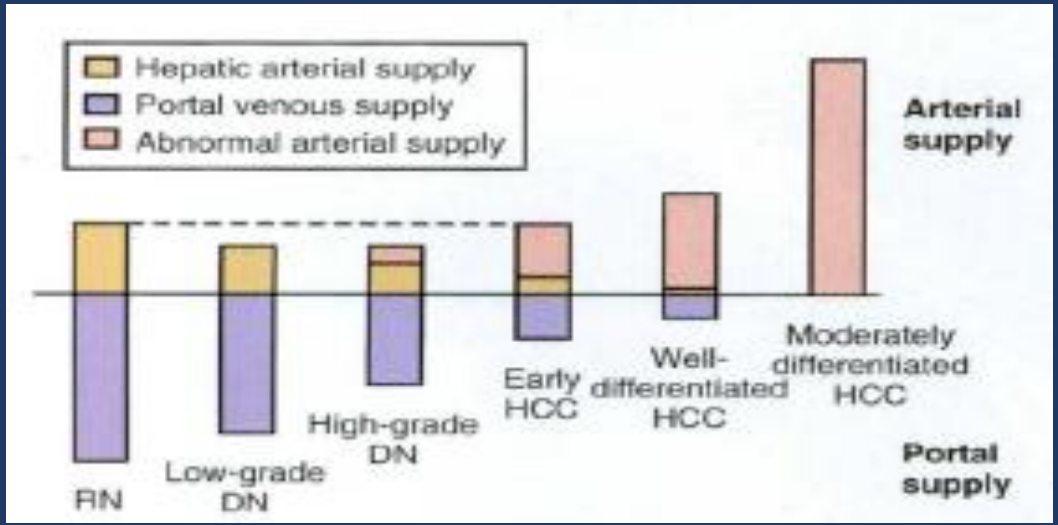
Hepatocellular Carcinoma (HCC)



Gross features of HCC are classified into five major types:

(1) small nodular with indistinct margin
(2) simple nodular
(3) simple nodular with extranodular growth
(4) confluent multinodular,
(5) infiltrative.

In advanced lesions, cancer cells often invade the portal veins through the blood drainage route from the tumor and then into the hepatic veins, forming tumor thrombi.



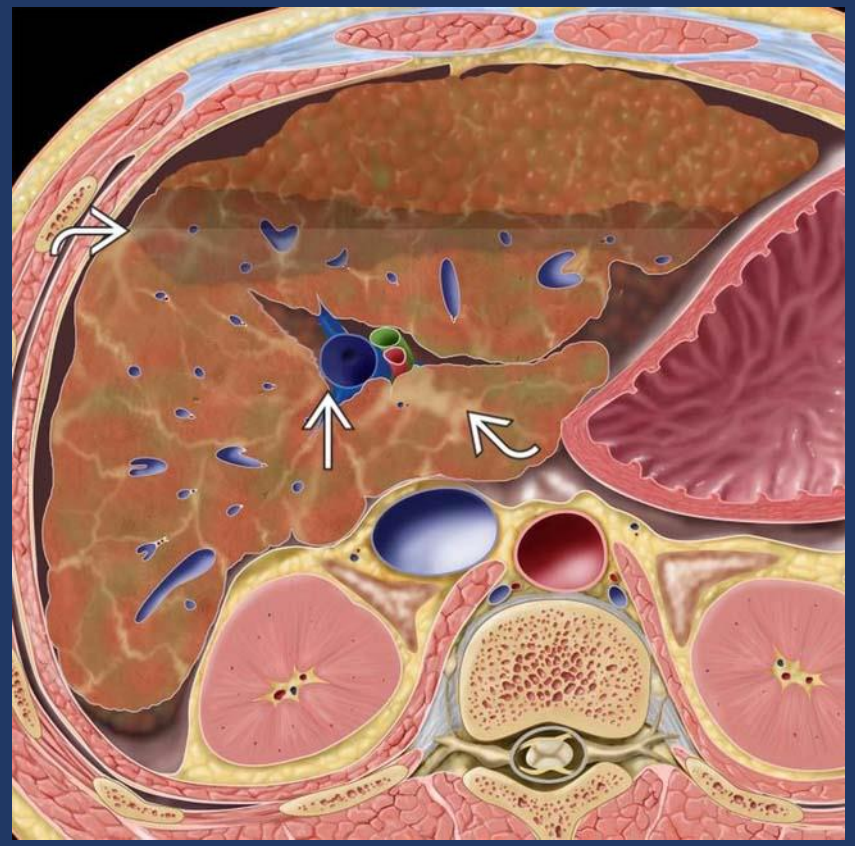
- Stepwise changes of intra nodular blood supply
- During multistep hepatocarcinogenesis.

  DN (dysplastic nodule)

  HCC (hepatocellular carcinoma)

  RN (regenerative nodule)

Cirrhosis



Regenerating Nodules

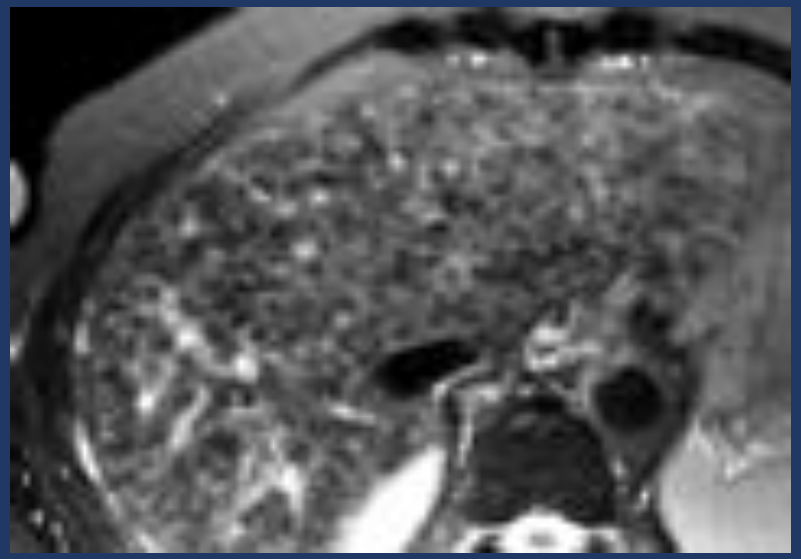
Usually too small to detect by imaging

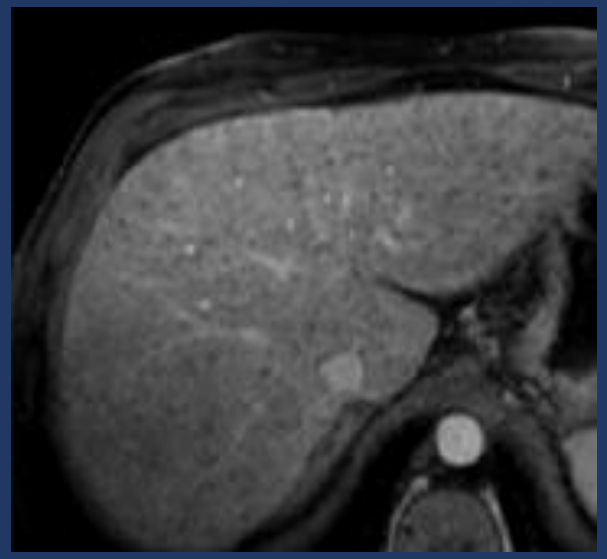
–May be surrounded by fibrotic septa

–May contain iron, copper

Siderotic or non siderotic

Siderotic regenerating nodules (When there is an accumulation of iron in the nodules, they are called siderotic nodules.)



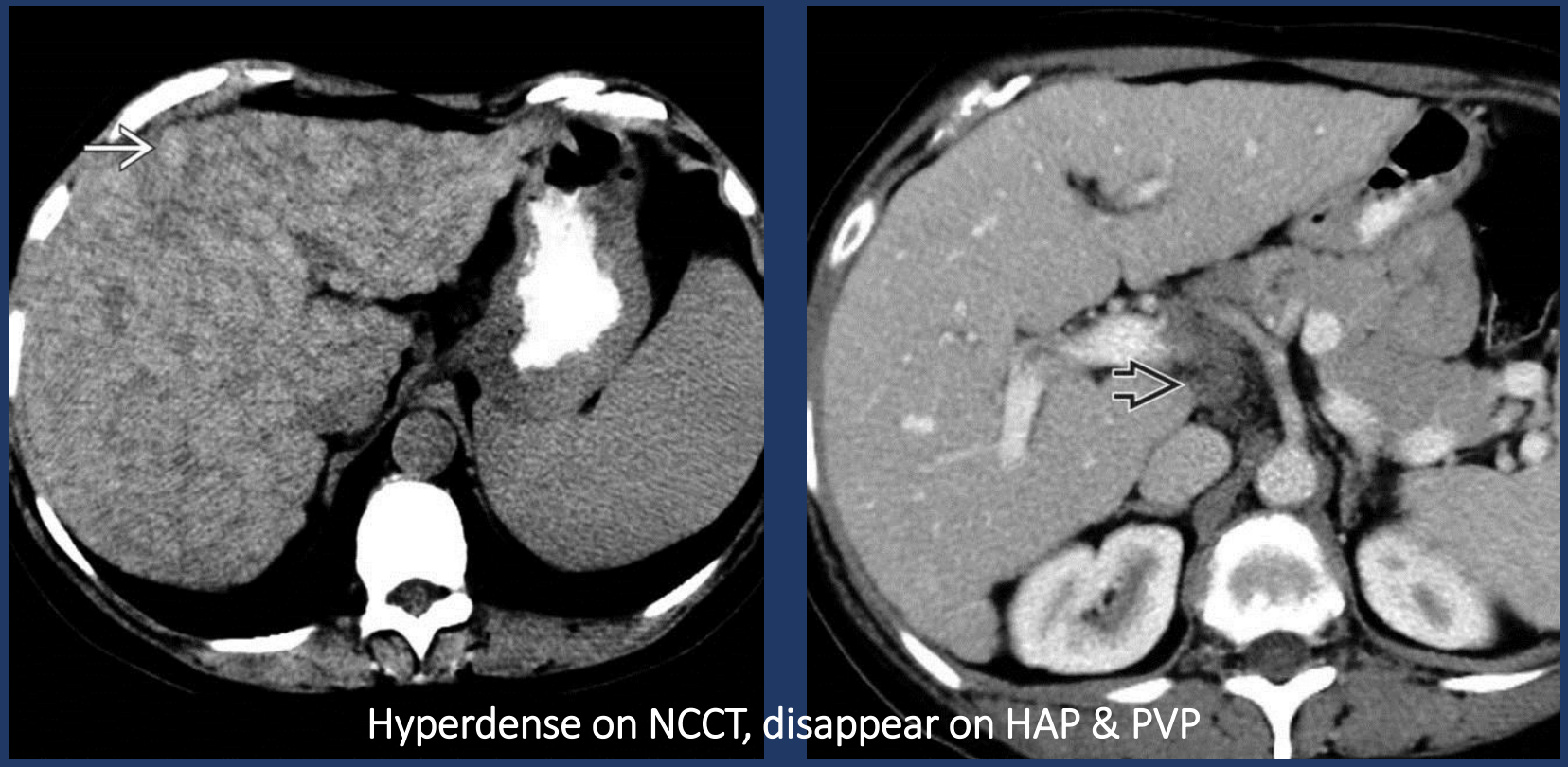


Regenerating Nodules

CT

Regenerative nodules are rarely visible on non-contrast CT unless they are siderotic.
Siderotic regenerative nodules (containing iron) are hyperdense to liver on precontrast imaging
Non siderotic regenerative nodule is isodence on non contrast imaging

On post-contrast CT and MRI, regenerative nodules enhance similar to the normal liver parenchyma in both portal venous and delayed phases, and may not be distinguishable in a cirrhotic liver. There is no arterial phase enhancement.



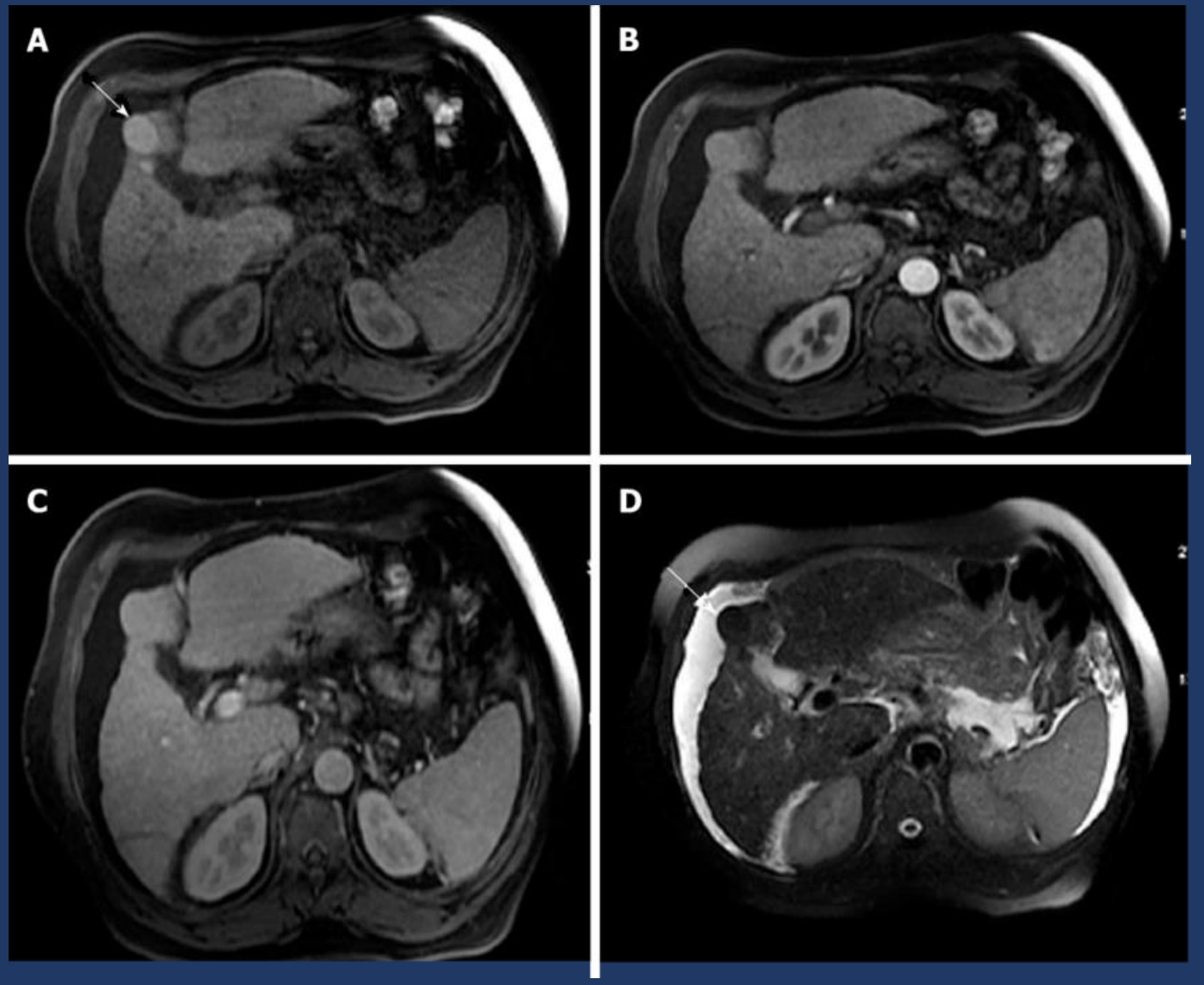
Hyperdense on NCCT, disappear on HAP & PVP

Importance of NC imaging

Regenerating Nodules

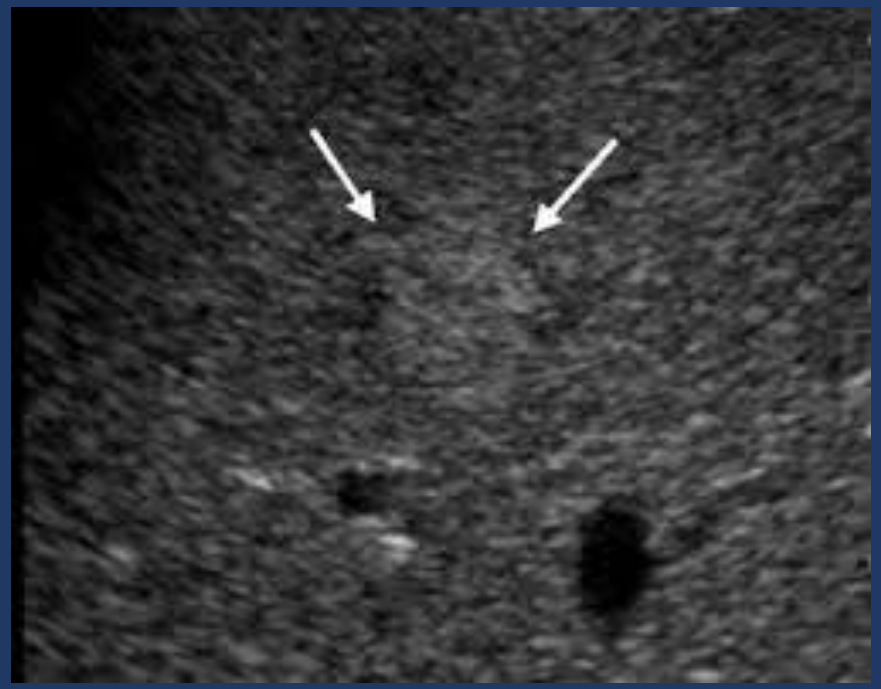
MRI finding:

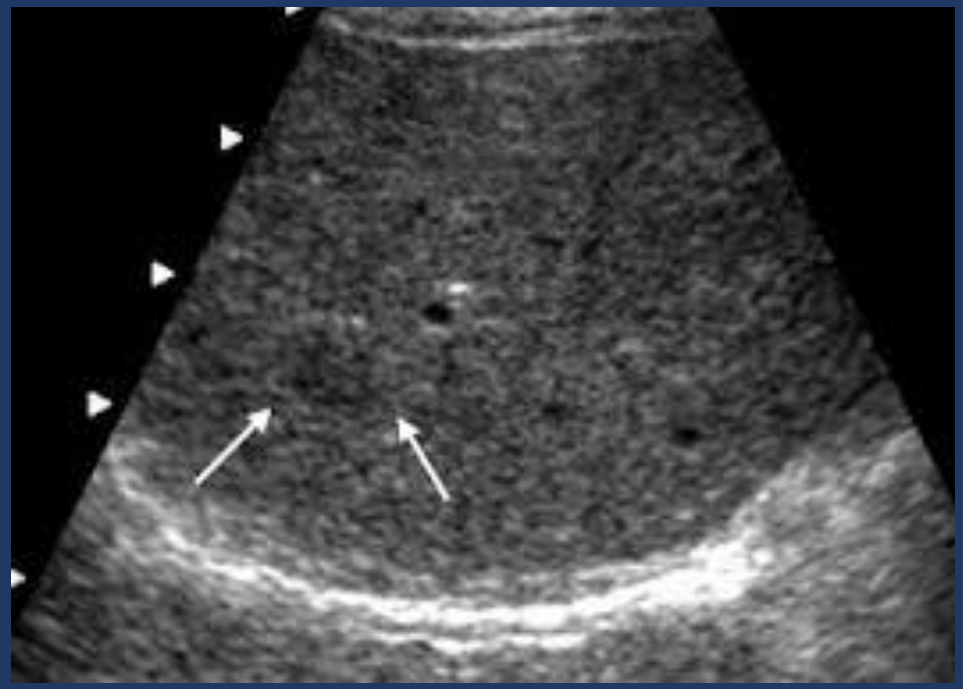
T1WI: Hypointense (Variable on T1)
T2WI: low signal intensity (Hypointense)
T2 gradient-echo and fast low-angle shot (FLASH) images: Markedly hypointense (best sequence for detection)
T1C+: usually do not enhance or enhance less than the liver parenchyma



Dysplastic Nodules

Ultrasound
Cirrhotic changes are present but the nodules may not be visualized on ultrasound. A few cases have shown hypo- and hyperechoic nodules and the echogenicity relates to the fat content in the nodule





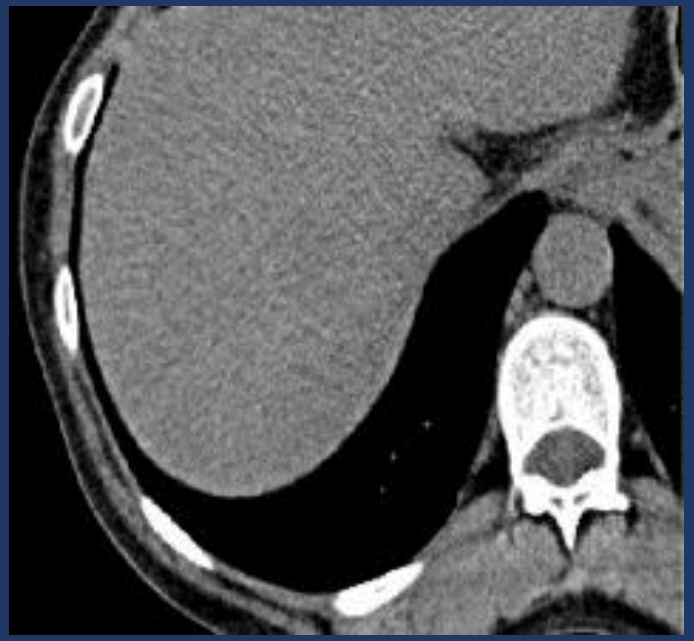
Dysplastic Nodules

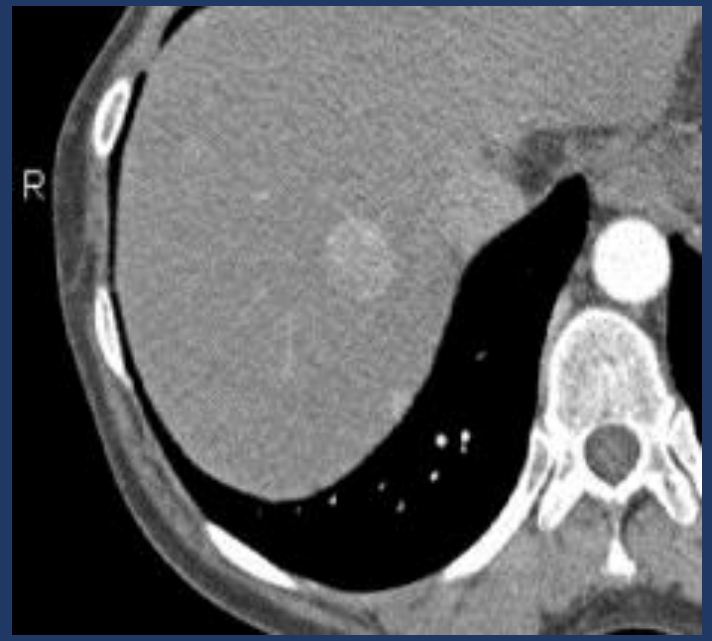
Rarely diagnosed by US or CT

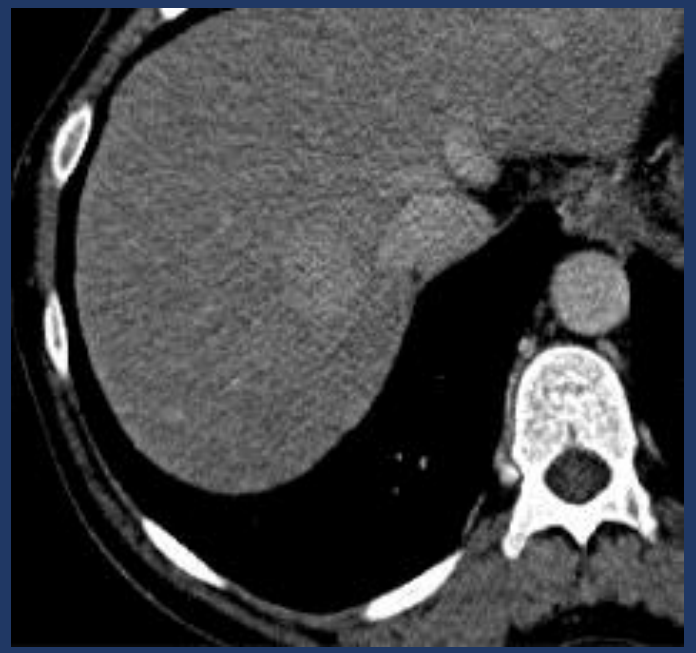
CT

Usually hypoattenuating, however, they may be iso- or hyperattenuating to the hepatic parenchyma.

multiphase contrasted images: they may show early arterial uptake but the contrast does not wash out on delayed phase (unlike HCC)



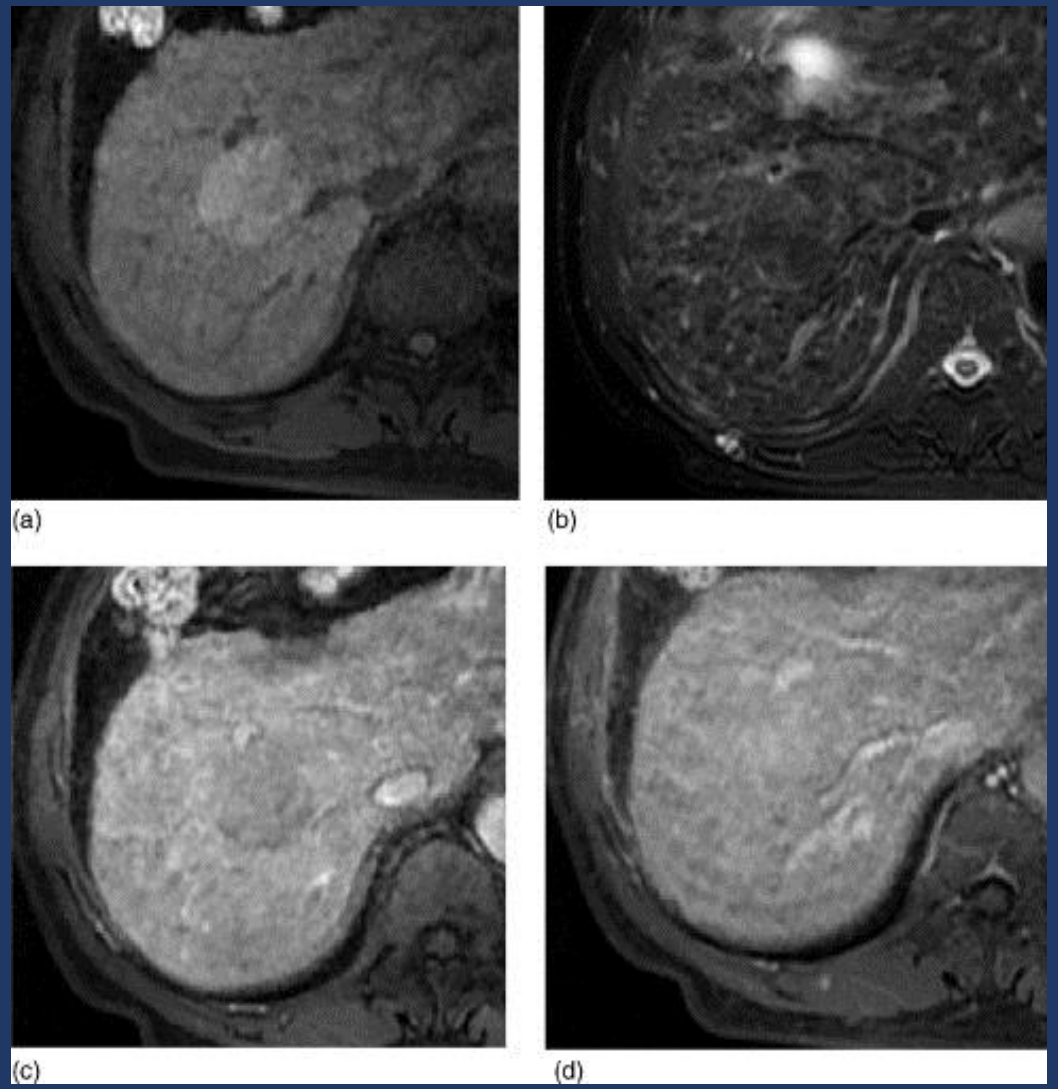




Dysplastic Nodules

MRI:

- **T1:** although the signal intensity may vary broadly, most of them have high T1 signal (copper)
- **IP-OOP:** shows fat accumulation characterized by signal drop on the out-of-phase sequence
- **T2:** iso- to hypointense (opposite of HCC)
- **DWI:** no restricted diffusion
- **T1 C+ (Gd):** high-grade nodules show early contrast enhancement without washout on delayed phase



Hepatocellular Carcinoma (HCC)

Several morphological forms

Massive(>3cms)

Nodular (<3cms)

Diffuse

AFP (Alfa feto protein)

Is an HCC tumor marker

Values more than 100ng/ml are highly suggestive of HCC

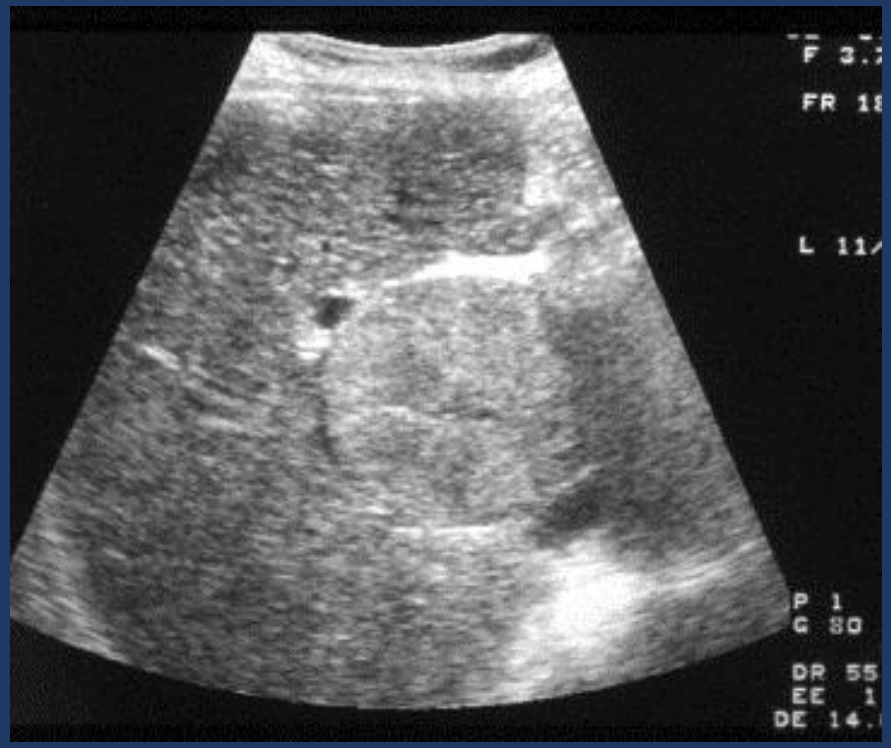
Elevation seen in more than 70%

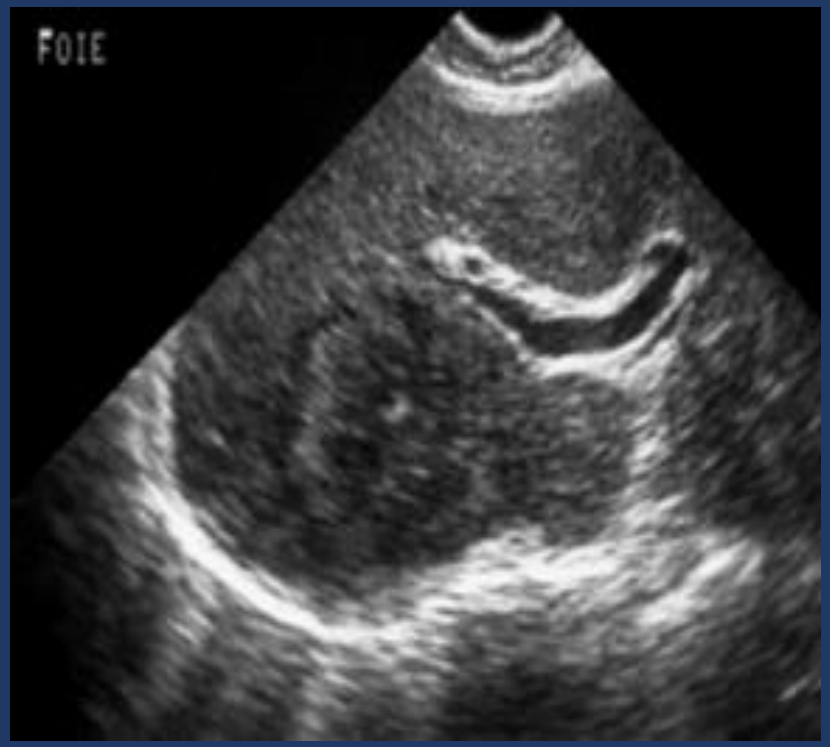
Hepatocellular Carcinoma (HCC)

US : hyperechoic, smaller tumors are hypoechoic.

Heterogeneous, hypervascular

US sensitivity about 75%.





Hepatocellular Carcinoma (HCC)

Arterial Phase: CT or MR

liver(30-35 sec)

HCC as supplied by arterial branch/neovascularization

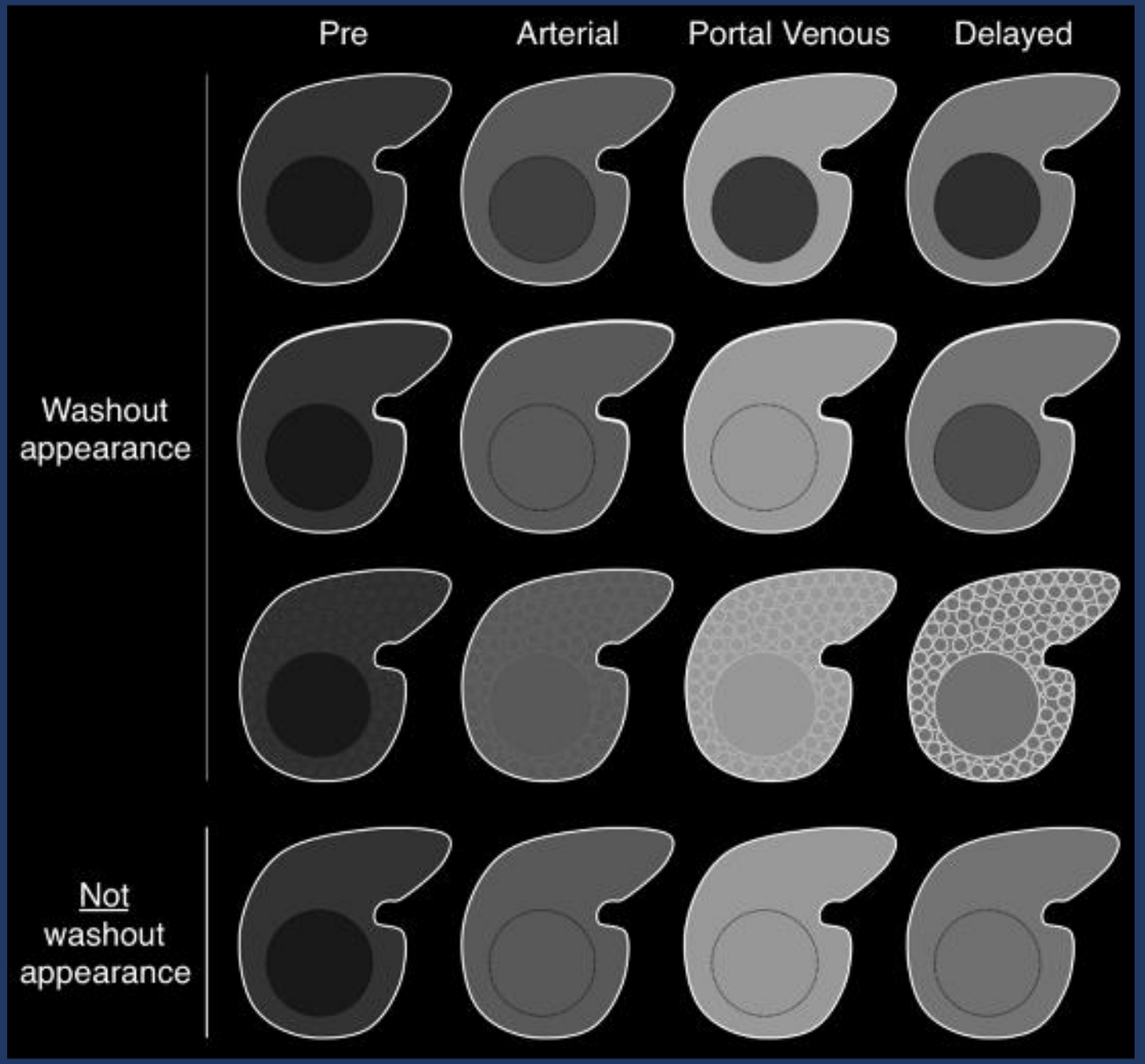
→ Enhancement

Venous Phase:

HCC which is enhanced during arterial phase has lost its contrast, hence no enhancement of the tumor but rest of the liver enhances.

Contrast in brightness of the lesion with respect to surrounding liver.

→ Wash out phenomenan

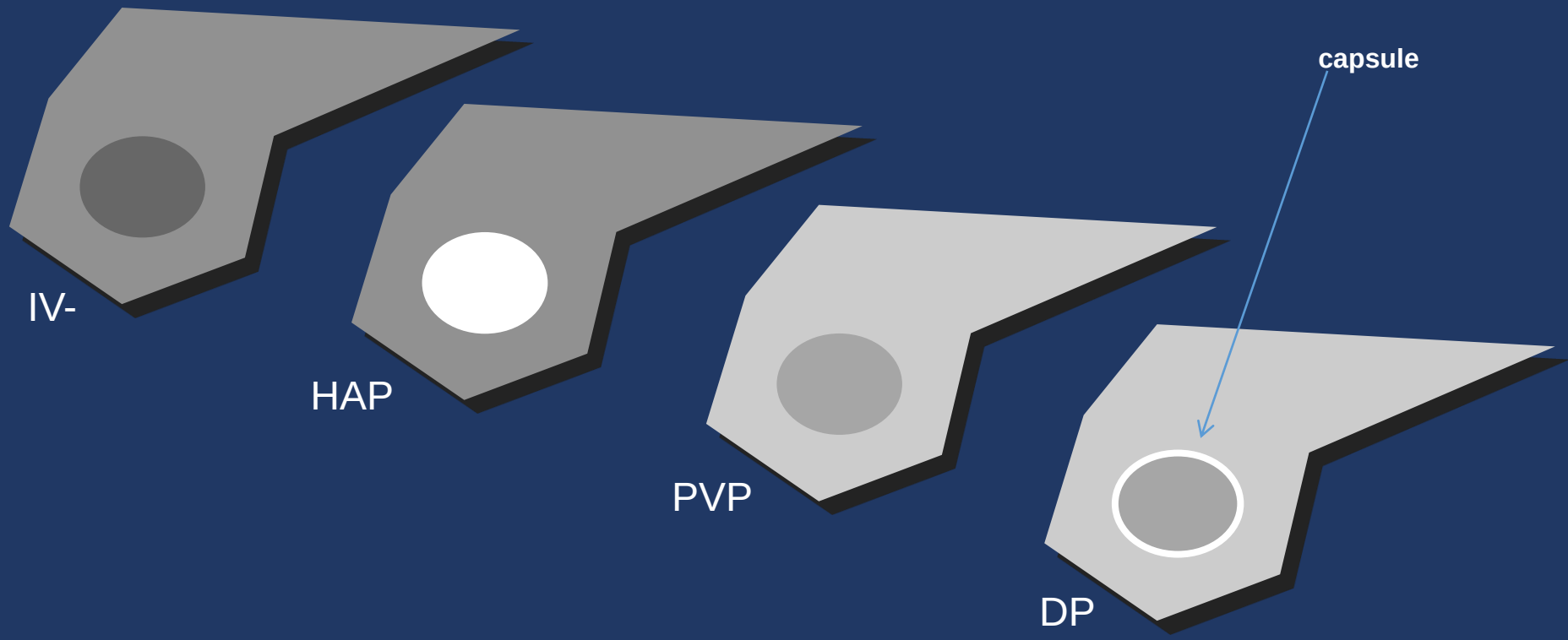

Pre
Arterial
Portal Venous
Delayed
Washout appearance
Not washout appearance

Hepatocellular Carcinoma (HCC)

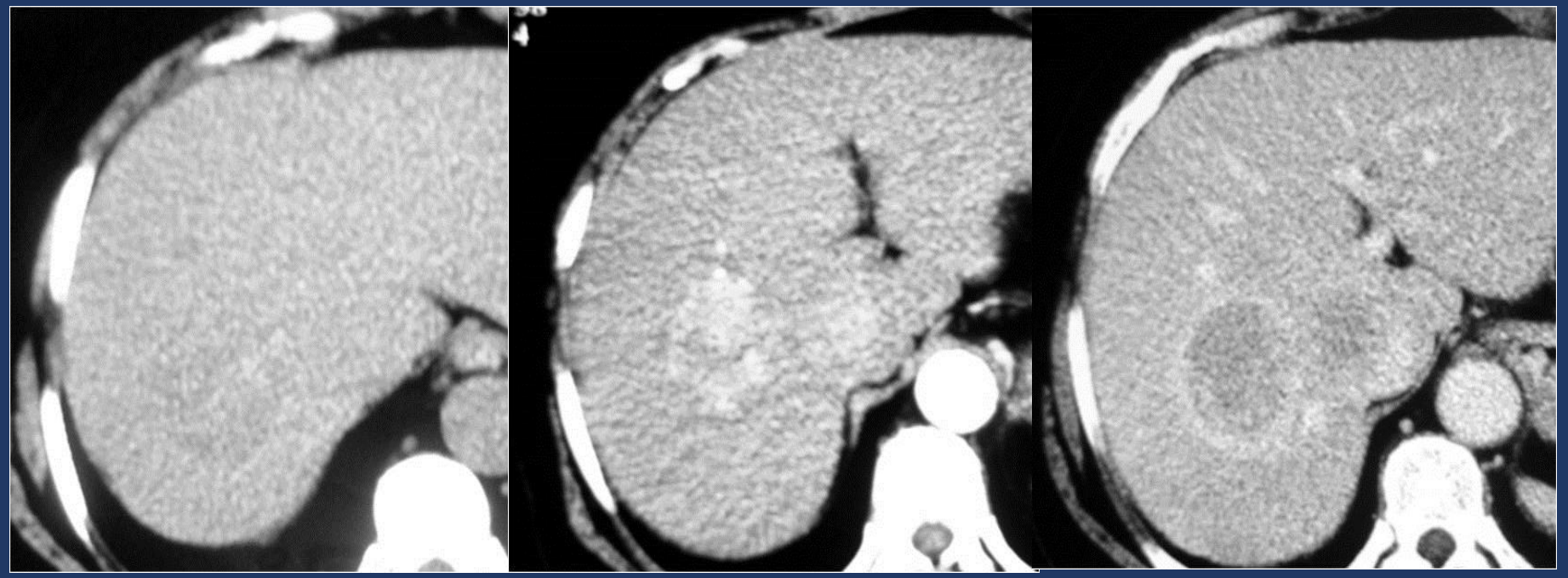
Delayed Phase :

Wash -out phenomenan persists and often exaggerated in smaller lesions.

The tumor capsule

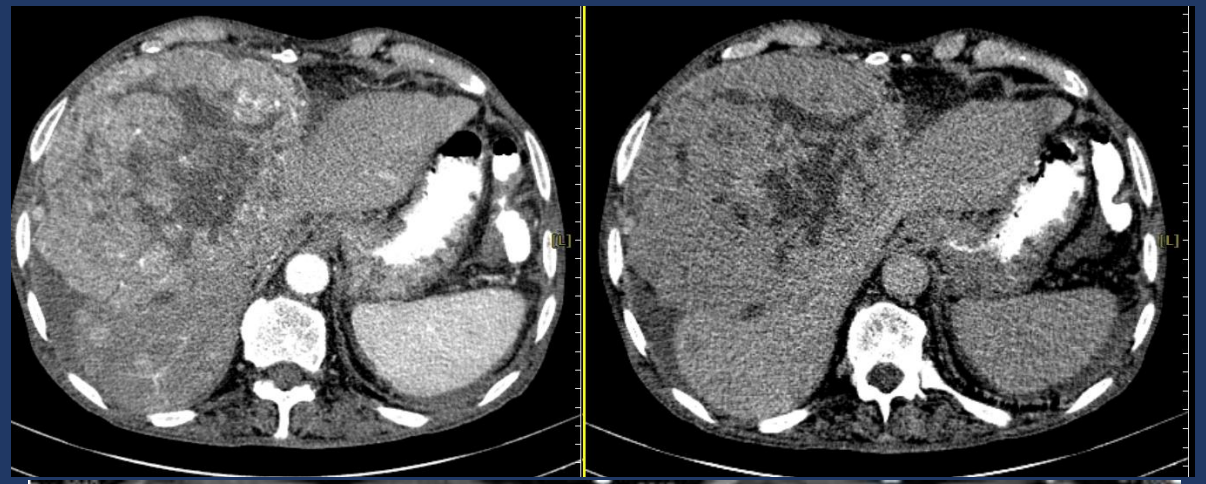


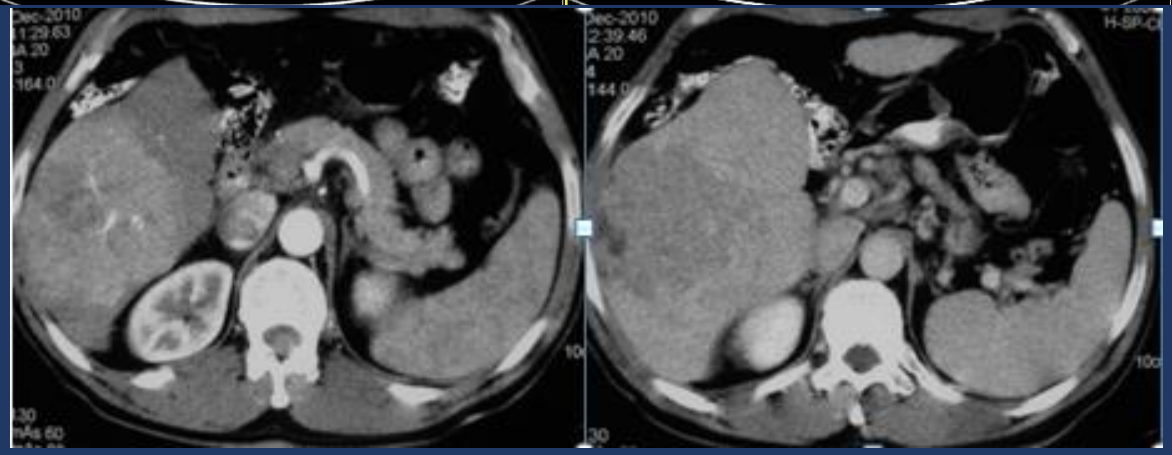
Hepatocellular Carcinoma (HCC)

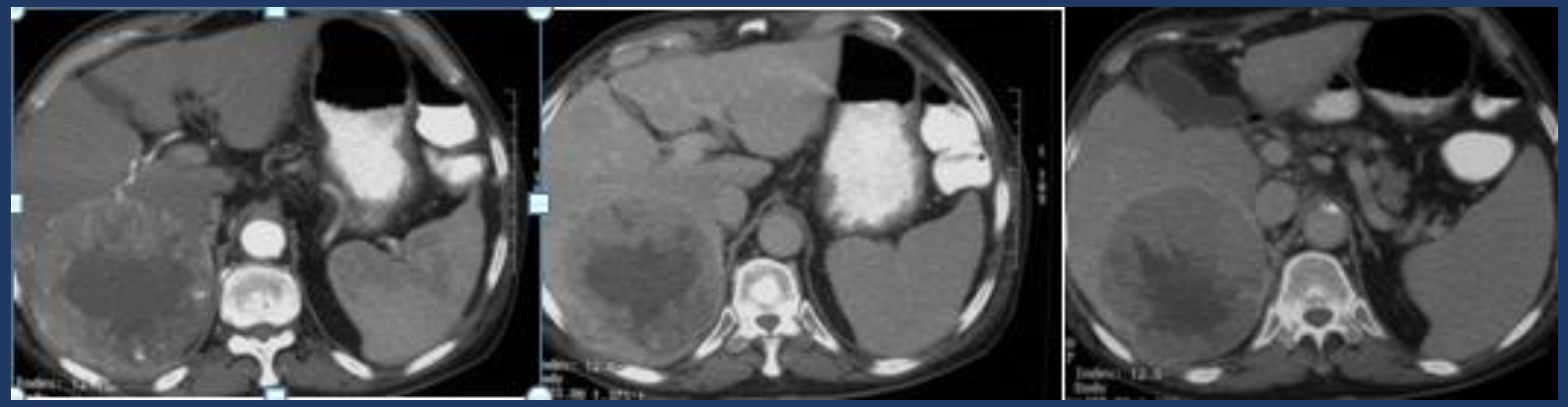


Abdominal CT level= w: 400 l: 50

Liver CT level= w: 150 l: 50







Hepatocellular Carcinoma (HCC)

MRI

. Variable intensity of HCC on T1

. 35% hyper, 25% iso-, 40 % hypo

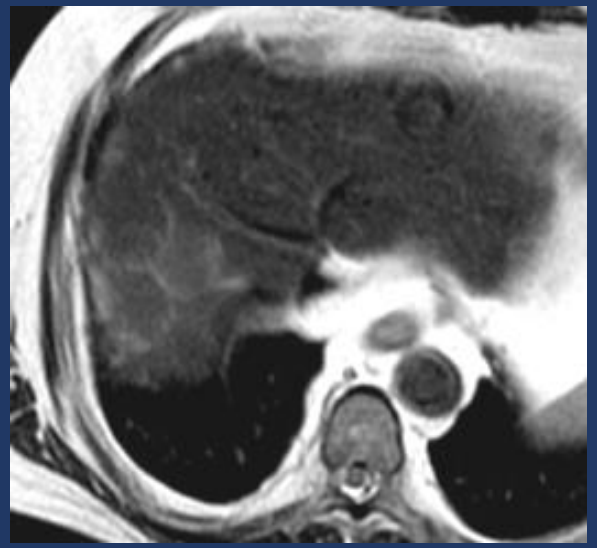
. Hyperintense (T1) often well-differentiated, contain fat, copper, glycogene

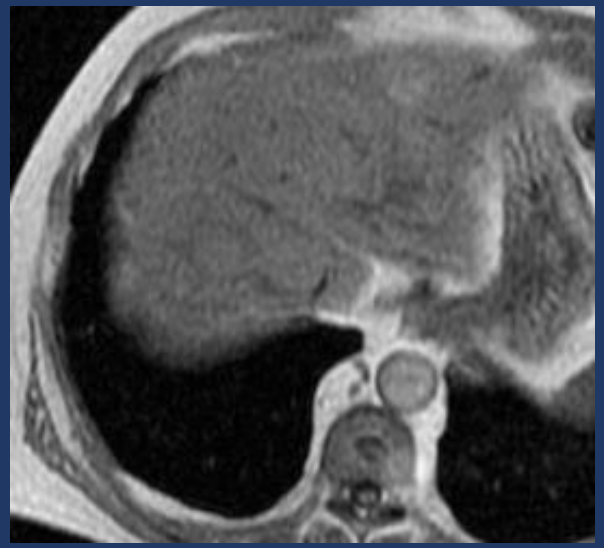
. Almost always hyperintense on T2 MR

. The tumor capsule is hypointense on both T1- and T2-weighted images in most cases

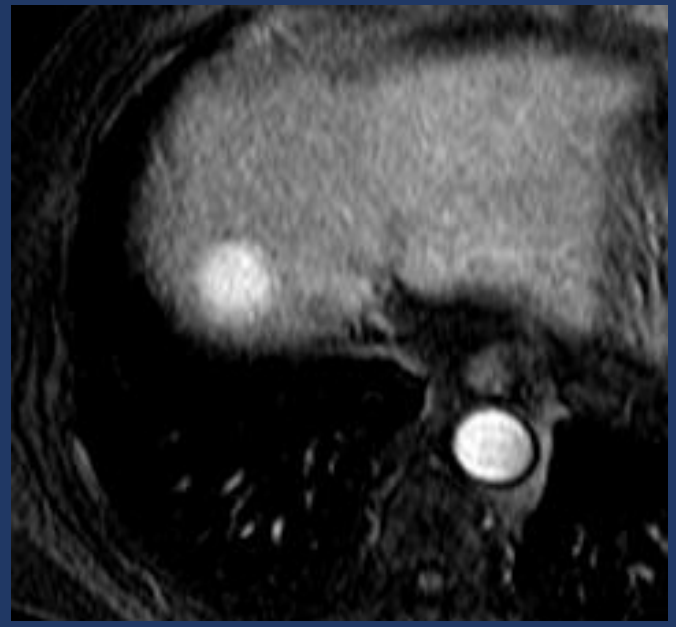
. Other Features: Focal fat

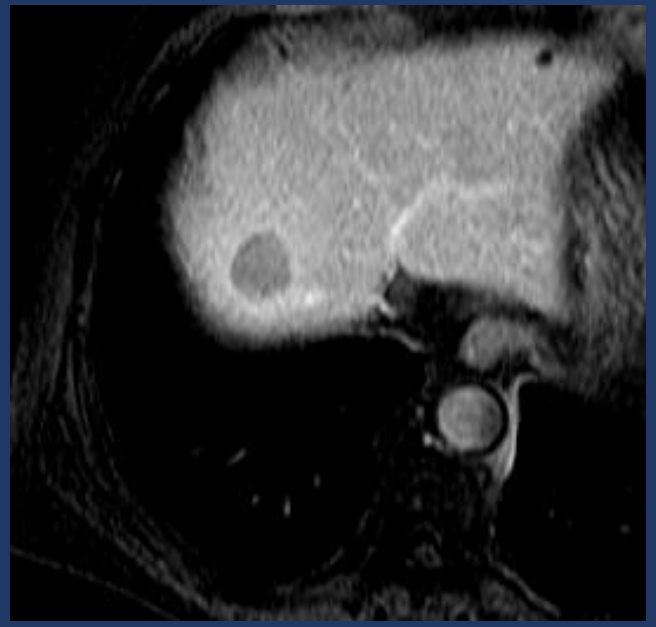
Hepatocellular Carcinoma (HCC)

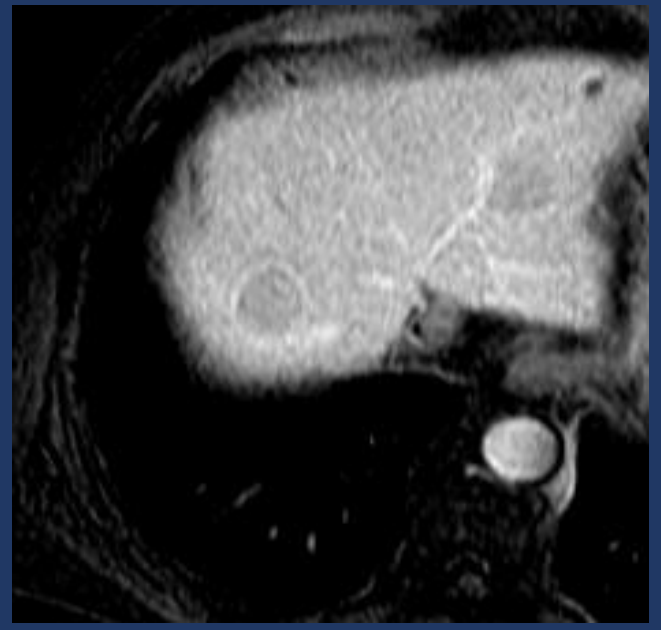


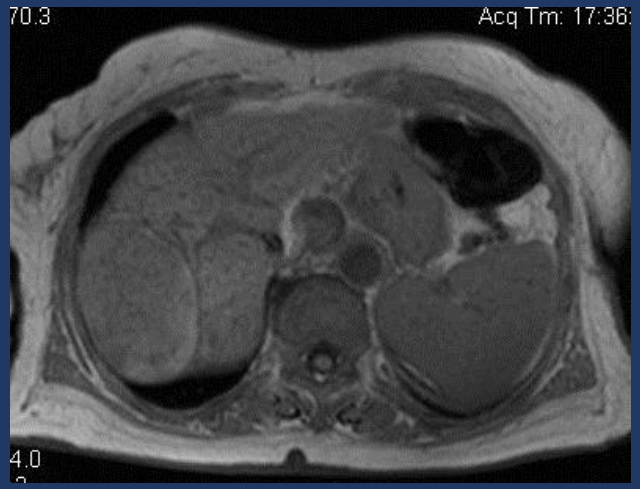


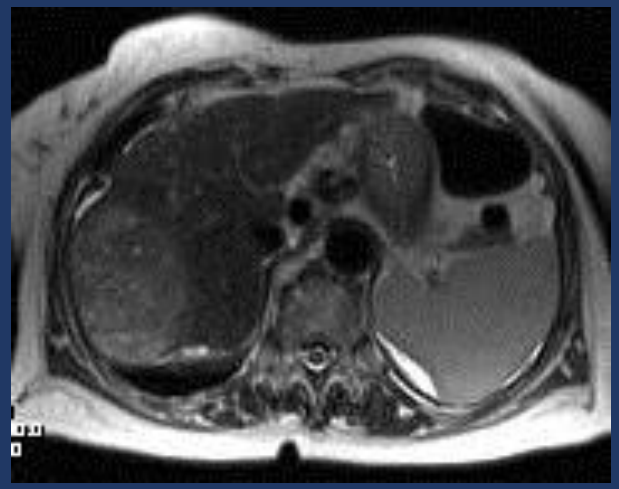
MRI

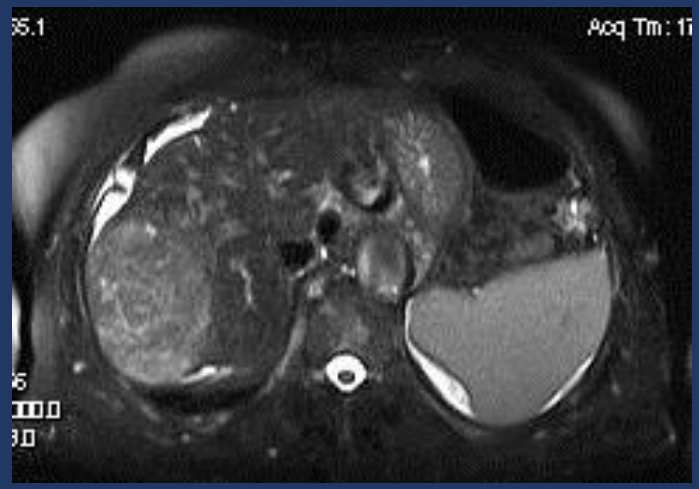


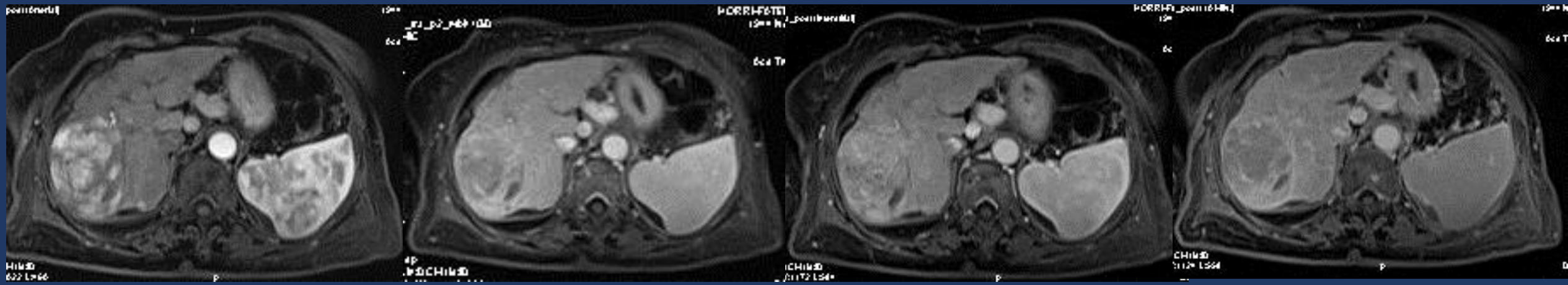


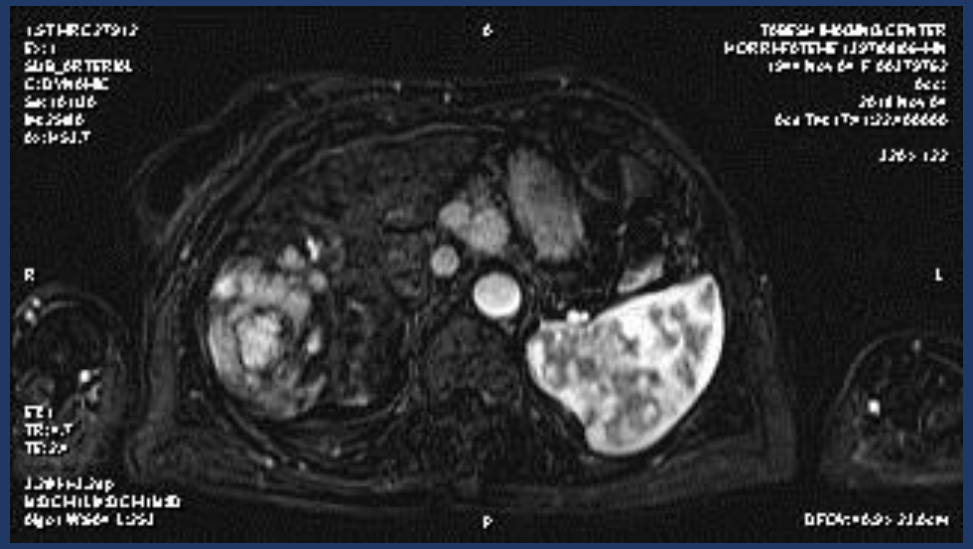


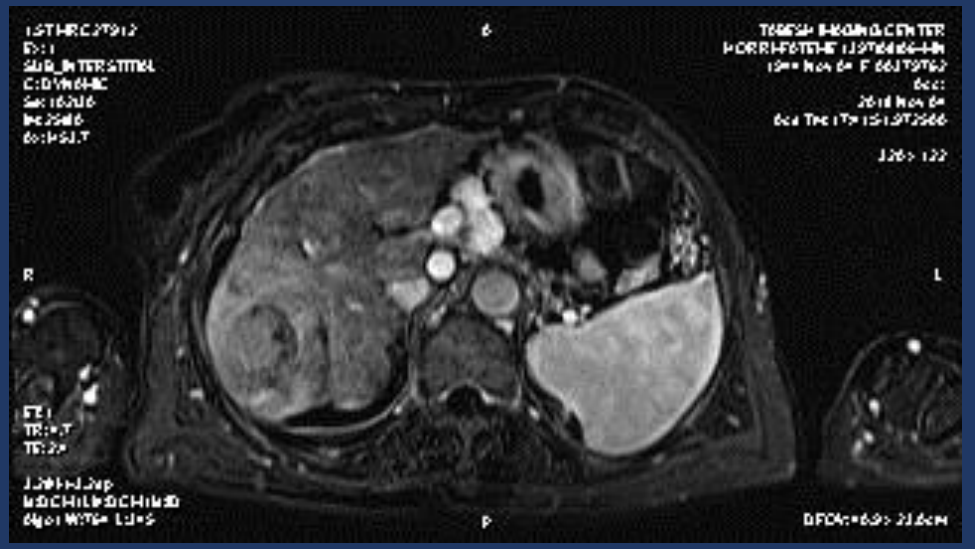

70.3
Acq Tm: 17:36:
4.0

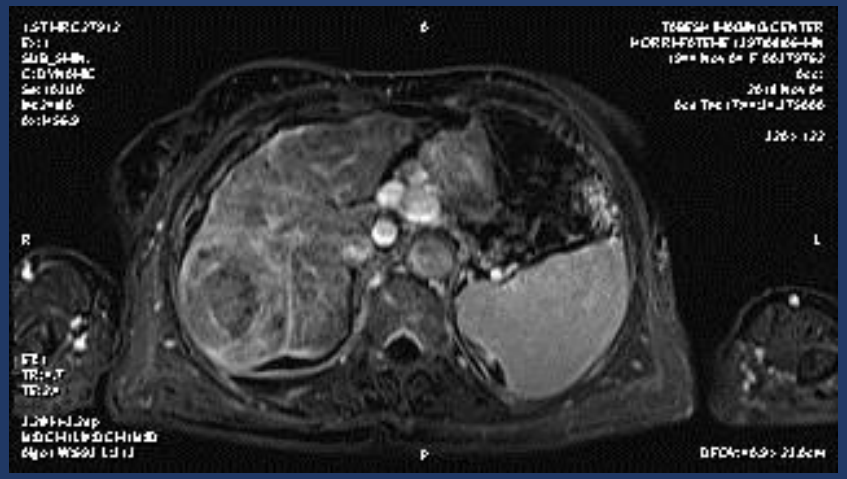


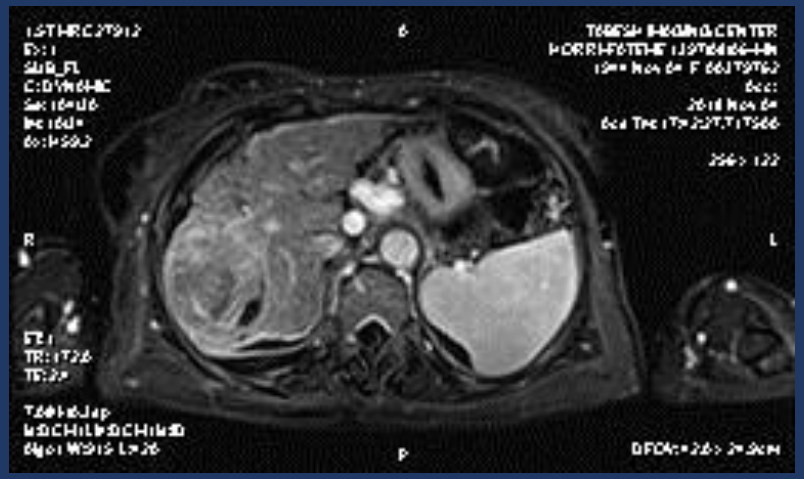

35.1
Acq Tm: 1

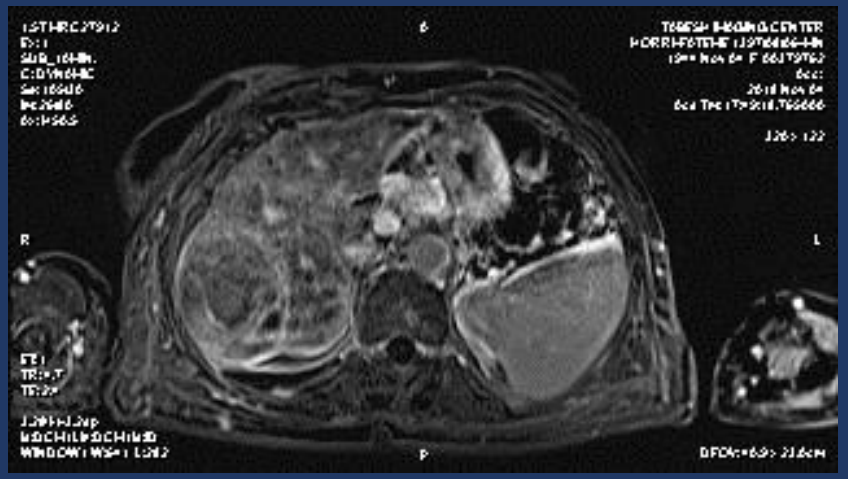












Nodular Lesions in Cirrhosis

	CT				MR			
	NC	HAP	PVP	Delay	T1	HAP	PVP	T2
Regenerative Nodule	— or ↑	—	—	—	— or ↑	—	—	— or ↓
Dysplastic Nodule	— or ↑	— or ↑	—	—	— or ↑	— or ↑	—	— or ↓
Well-diff HCC	— or ↓	— or ↓	↓	↓	— or ↑	— or ↑	— or ↑	↑
Mod-diff HCC	— or ↓	— or ↑	— or ↓	↓	— or ↓	↑	— or ↑	↑

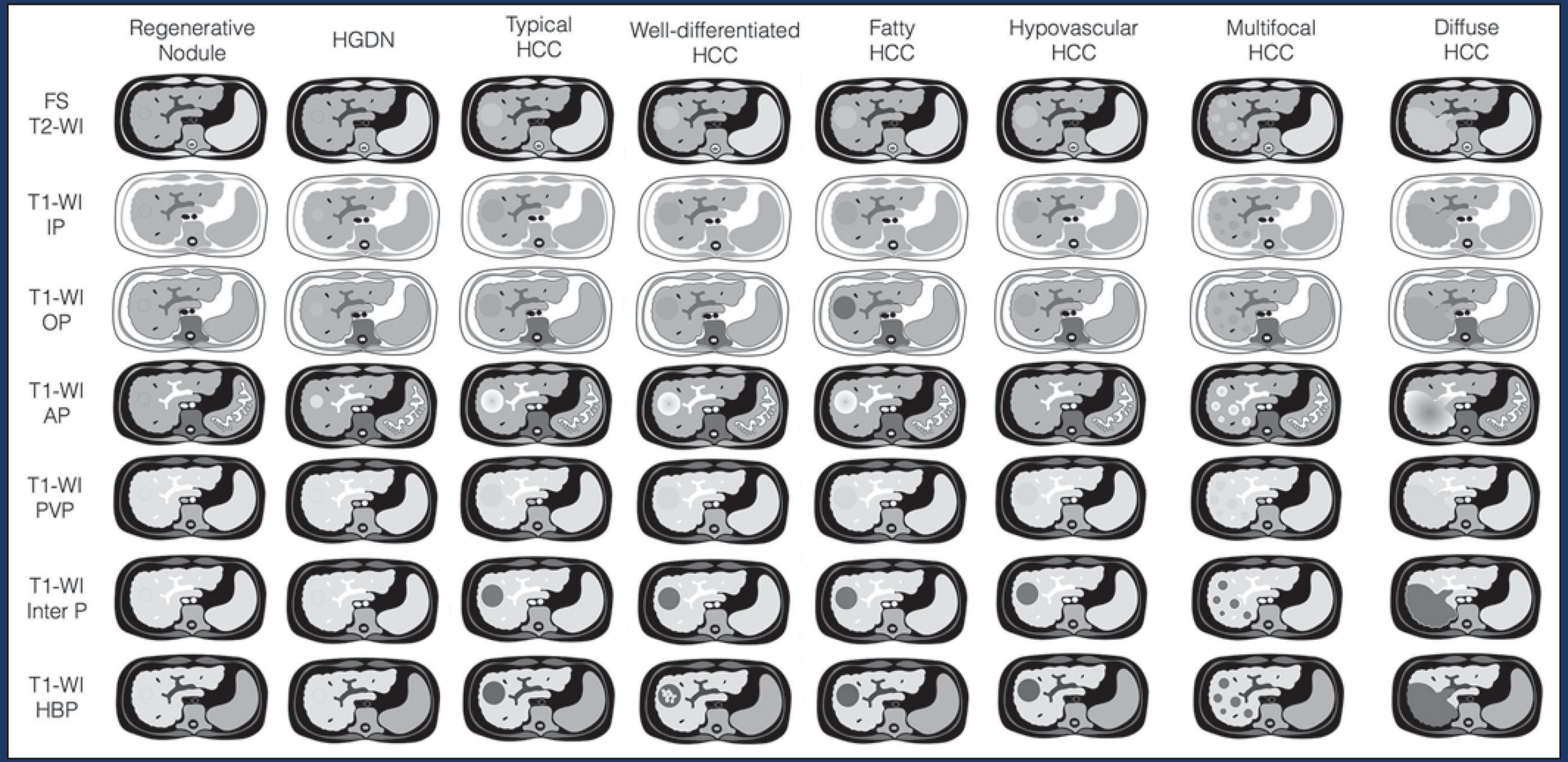
— = not seen (isodense, isointense)

↑ = hyperdense (-intense) to liver

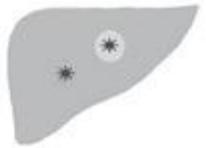
↓ = hypointense (-intense) to liver

Lesion	Sequences						
	Unenhanced T1	Arterial	Portal venous	Delayed	Hepatobiliary	DWI	T2
RN							
HGDN							
Early HCC							
HCC classic							
HCC green							
HCC ipo-vascular							

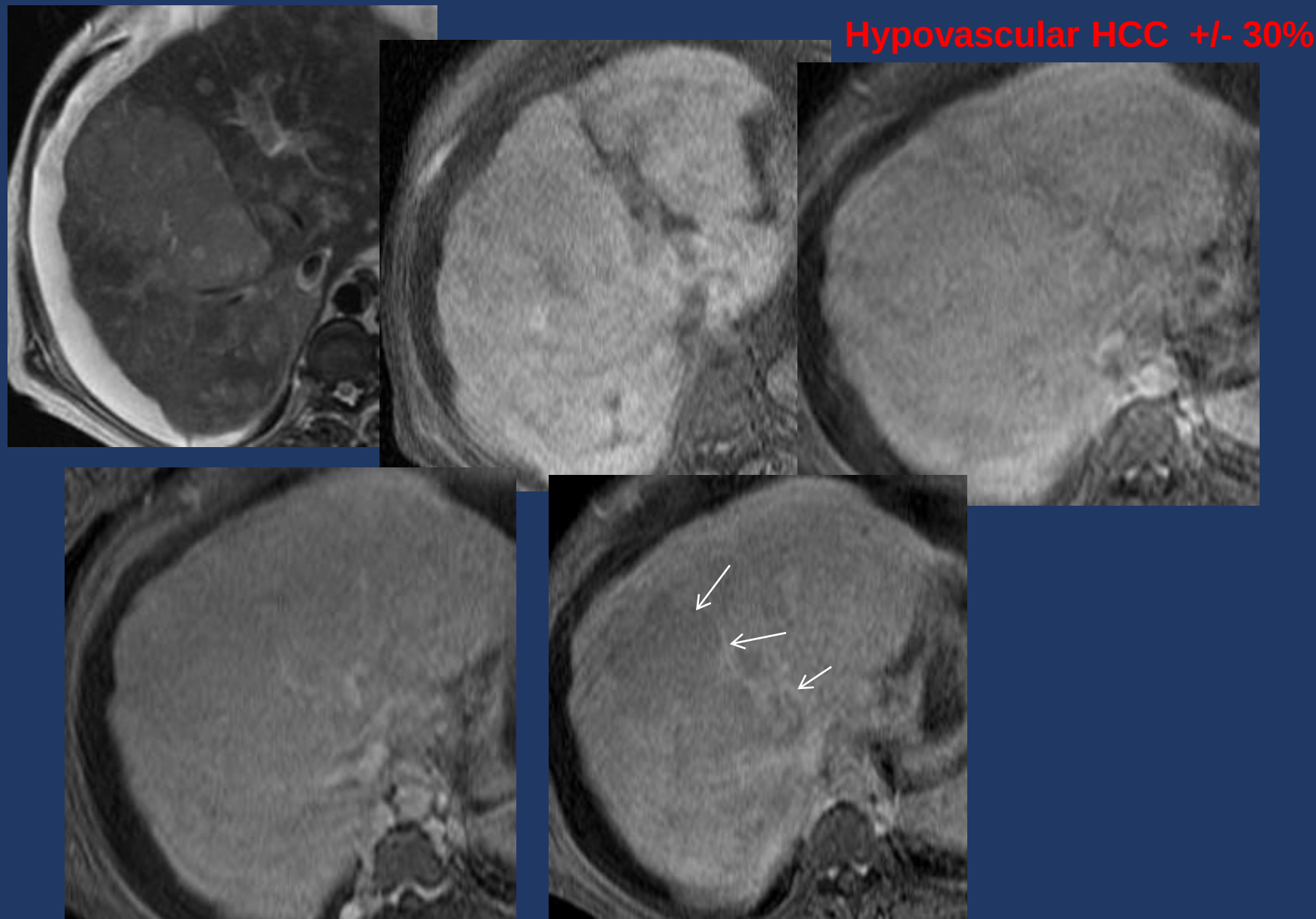
 Isointense lesion  Hyperintense lesion  Hypointense lesion  Slightly hyperintense lesion


Regenerative Nodule
HGDN
Typical HCC
Well-differentiated HCC
Fatty HCC
Hypovascular HCC
Multifocal HCC
Diffuse HCC
FS T2-WI
T1-WI IP
T1-WI OP
T1-WI AP
T1-WI PVP
T1-WI Inter P
T1-WI HBP

HCC TYPES

Liver Lesion	T2-Weighted	T1-Weighted Before Injection	Arterial Phase	Hepatocyte Phase
Simple cyst				
Hemangioma				
Flash-filling hemangioma				
Adenoma				
Focal nodular hyperplasia				
Cholangiocarcinoma				
Fibrolamellar hepatocellular carcinoma				
Metastasis				

Hepatocellular Carcinoma (HCC)



Hypovascular HCC +/- 30%

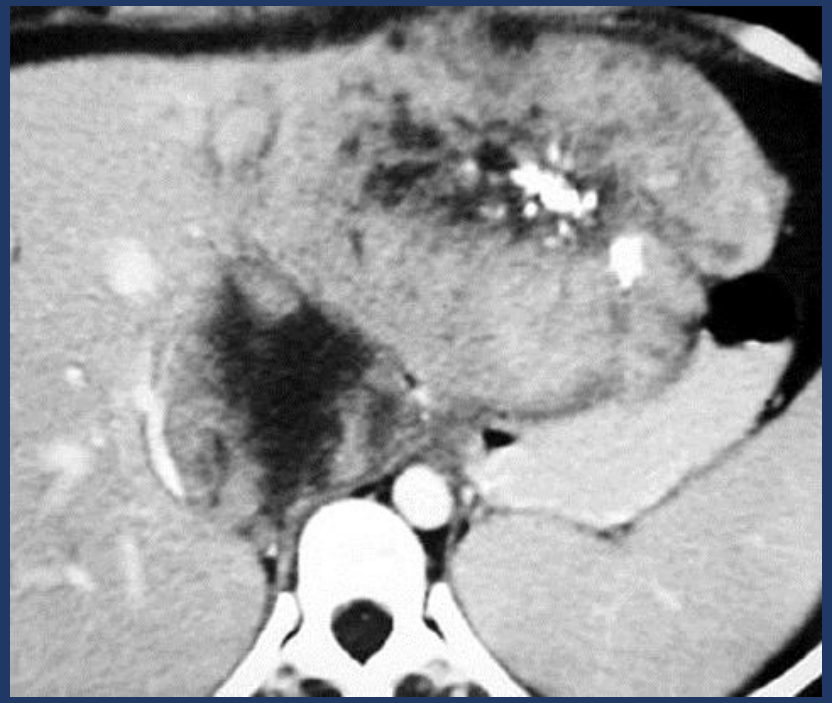
Fibro-Lamellar Carcinoma

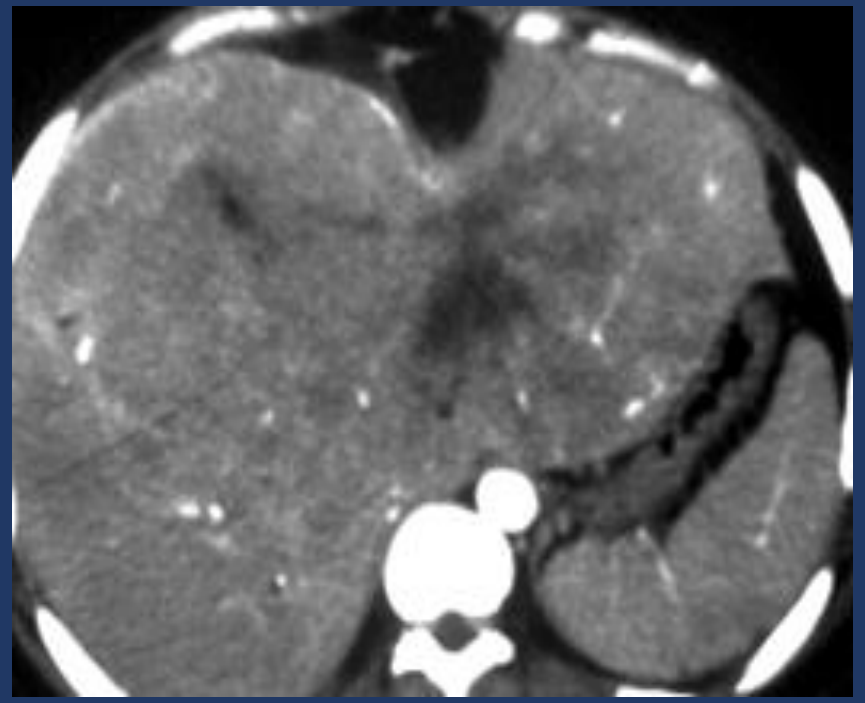
. Presents in young pt (5-35)

. Not related to cirrhosis, AFP is normal

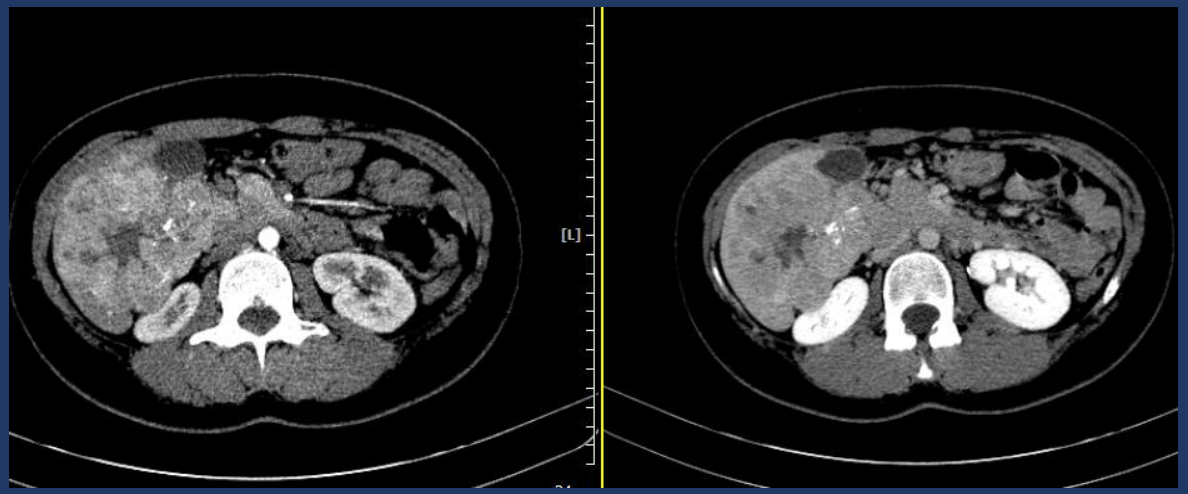
. CT/MRI shows large mass with peripheral enhancement and typical stellate scar with radial septa showing persistant enhancement

. Calcifications





fibrolamellar Carcinoma (FLC)

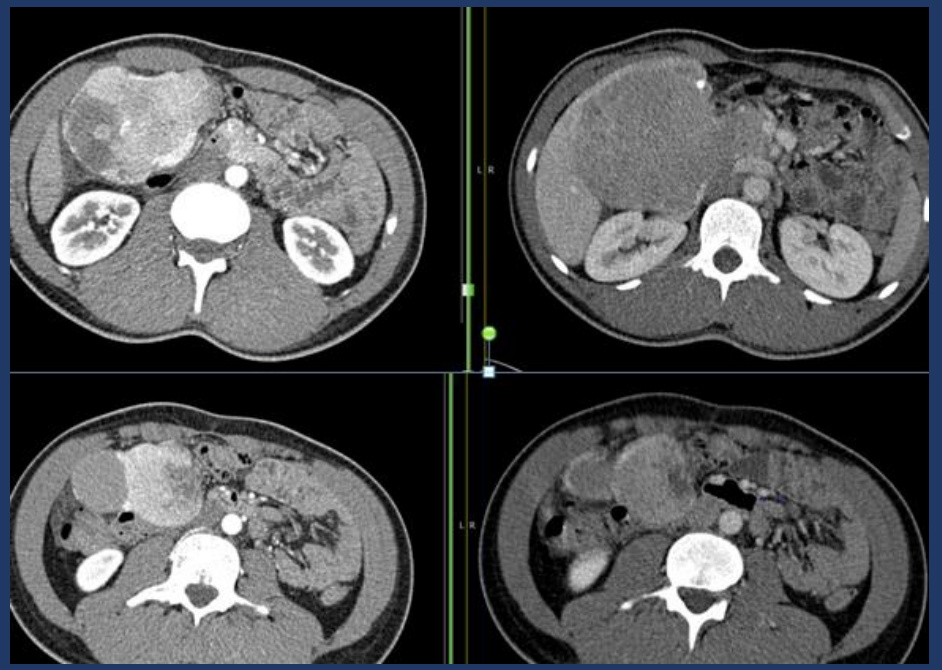


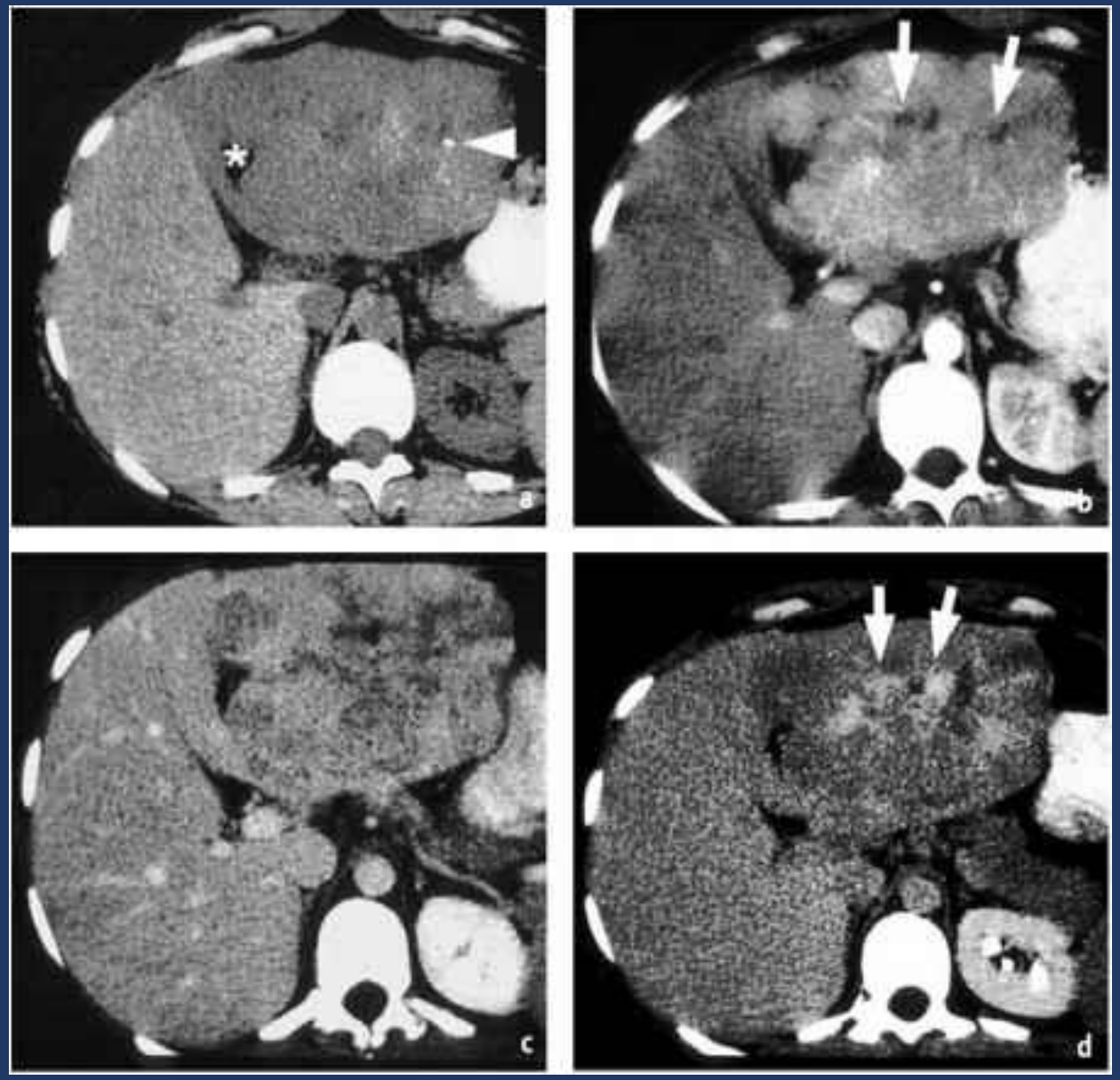
Fibrolamellar carcinoma develops in normal livers in young adults, with no clear sex predilection

Tumor markers are usually not elevated, although α-fetoprotein (AFP) may be slightly increased;

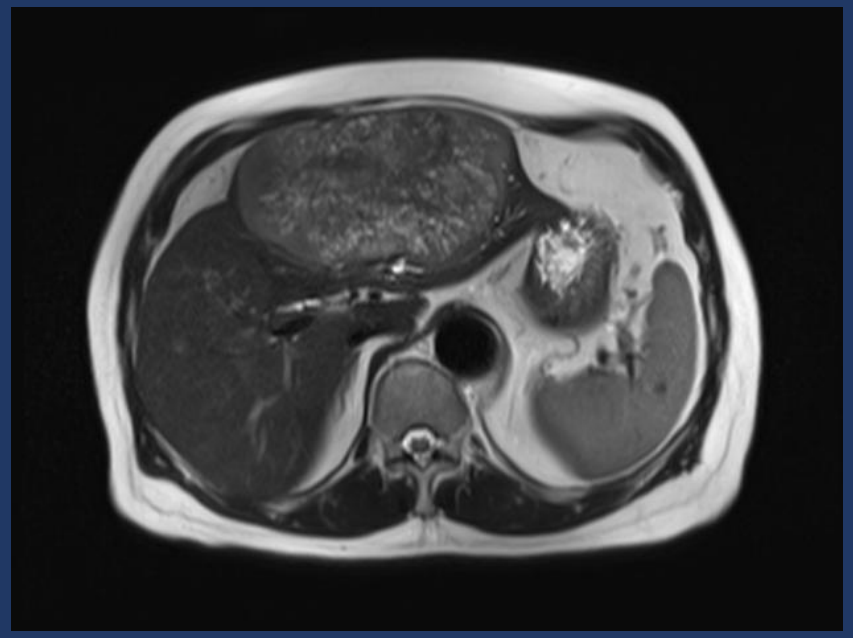
it is solitary, well demarcated, and lobulated. Necrosis and hemorrhage are seen in the center. A central fibrous scar like that seen in FNH is sometimes present.

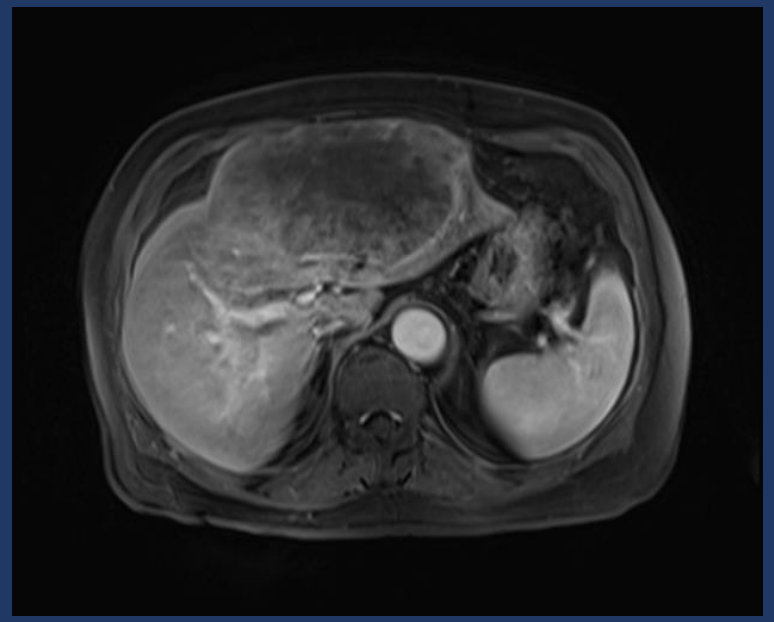
On dynamic CT inhomogeneous enhancement is observed during the arterial phase. A poorly enhanced hypodense area generally reflects necrosis. In the equilibrium (or delayed) phase, delayed enhancement of the central scar is seen in about 25% of cases

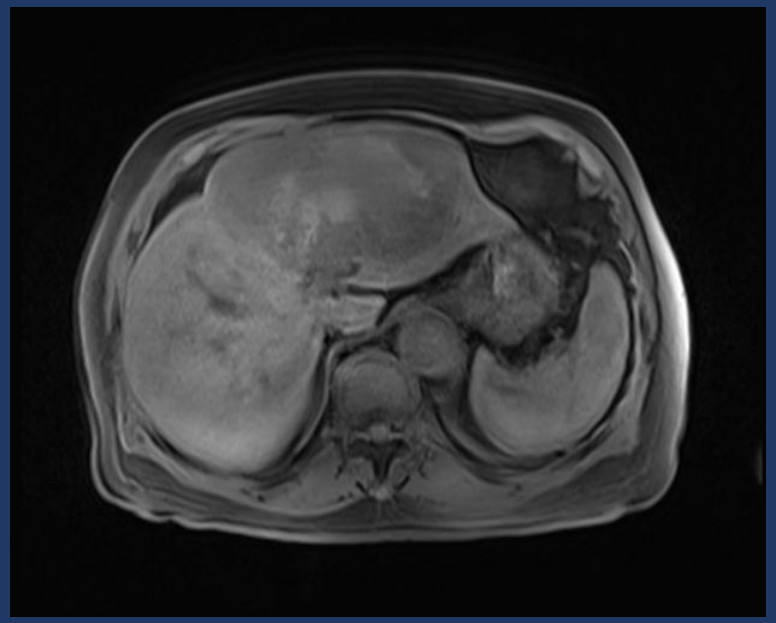



a
b
c
d

fibrolamellar Carcinoma (FLC)



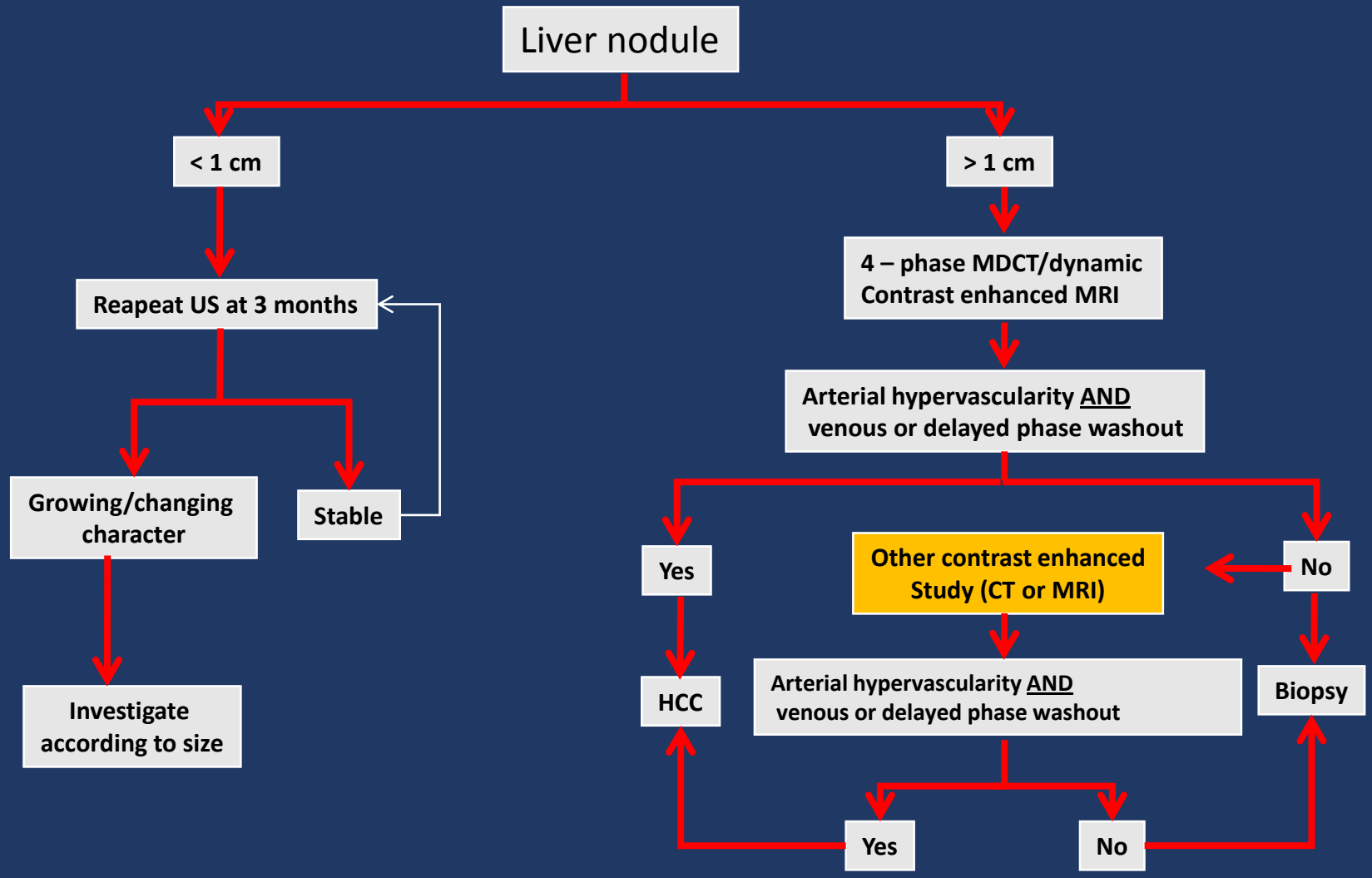




In the all sequences central scar is low signal
But shows delay enhancement

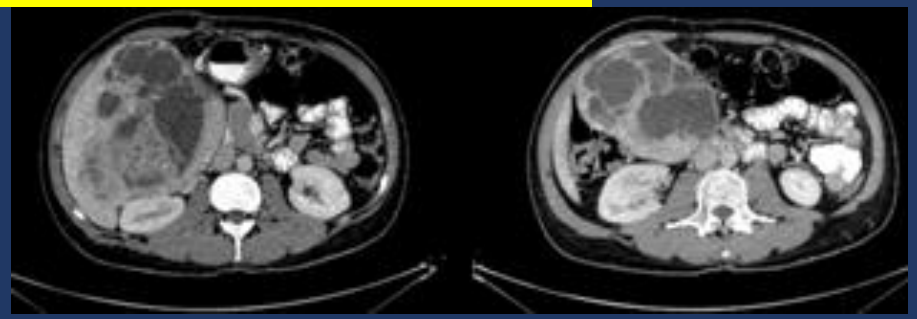
2010 AASLD Algorithm for Investigation of Small Nodules Found On Screening in Patients with Cirrhosis

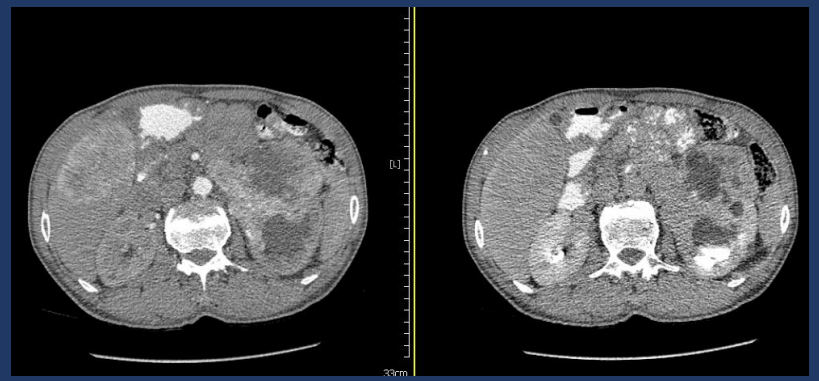
DIAGNOSIS : patients with cirrhosis or chronic hepatitis (even without cirrhosis)

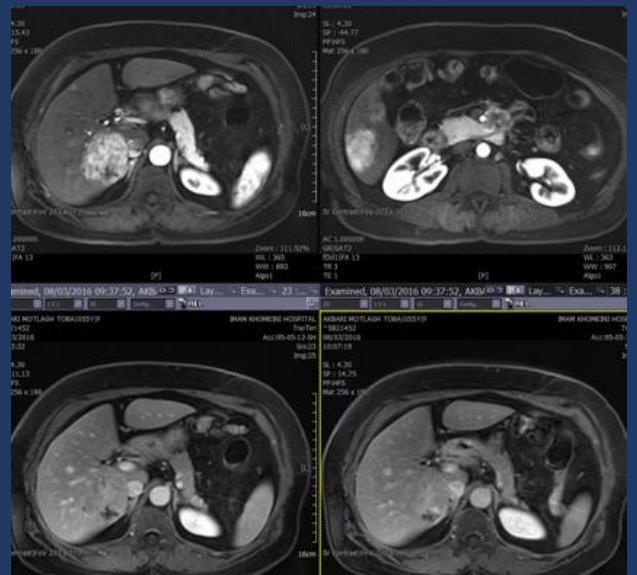


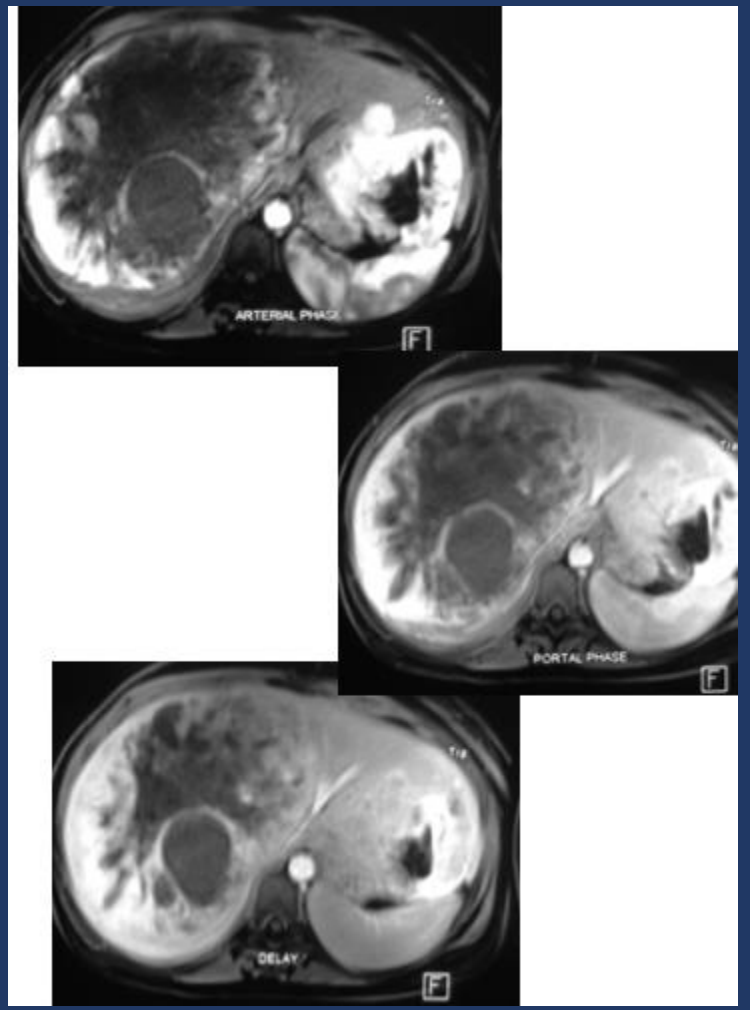
Bruix J and Sherman M. AASLD Practice Guidelines , Management of Hepatocellular Carcinoma ***Hepatology November 2011***

HYPERVASCULAR METASTASIS



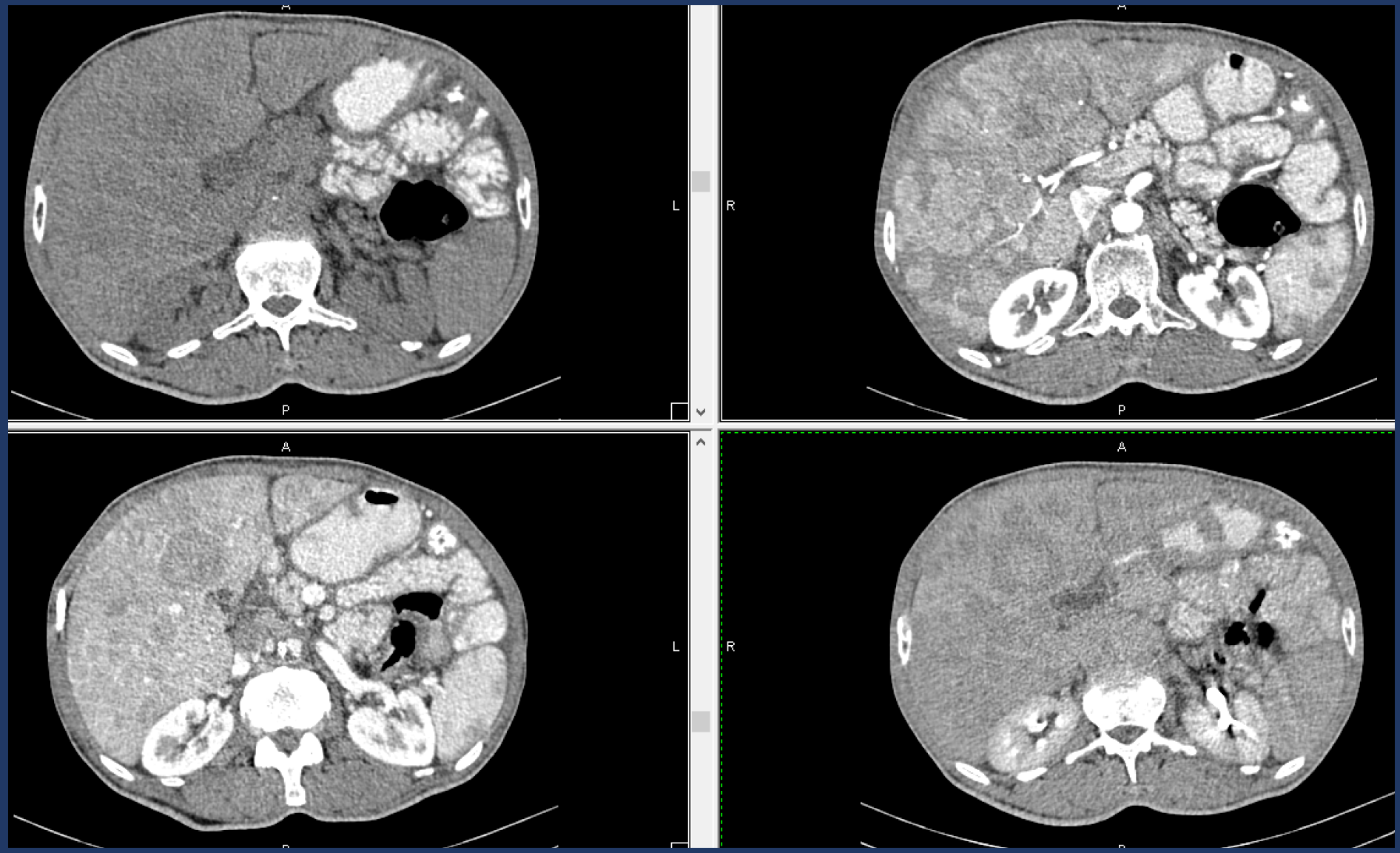






including renal cell carcinoma
thyroid carcinoma
neuroendocrine tumor
Choriocarcinoma
Breast
melanoma

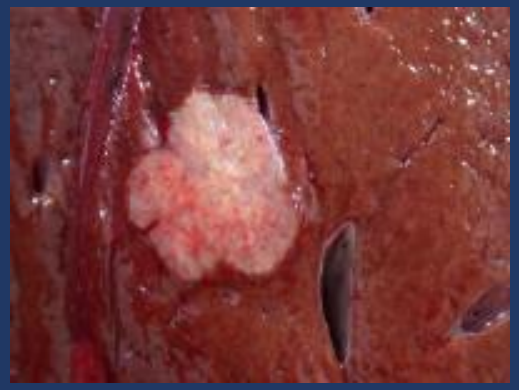
Hypervascular metastasis



Hypo vascular mass

- **Hypo vascular mass most conspicuous in portal phase**
  - Metastasis
  - Peripheral cholangiocarcinoma
  - Lymphoma

Metastatic disease



. Most common malignant hepatic tumor

. Presence of extrahepatic malignancy should be sought in patients with characteristic liver lesions per imaging studies. Physical exam and history is very helpful.

. Common primaries : colon, breast, lung, stomach, pancreases, and melanoma

. Mild cholestatic picture (ALP, LDH) with preserved liver function

. CT or US guided biopsy provides definitive diagnosis but not always required.

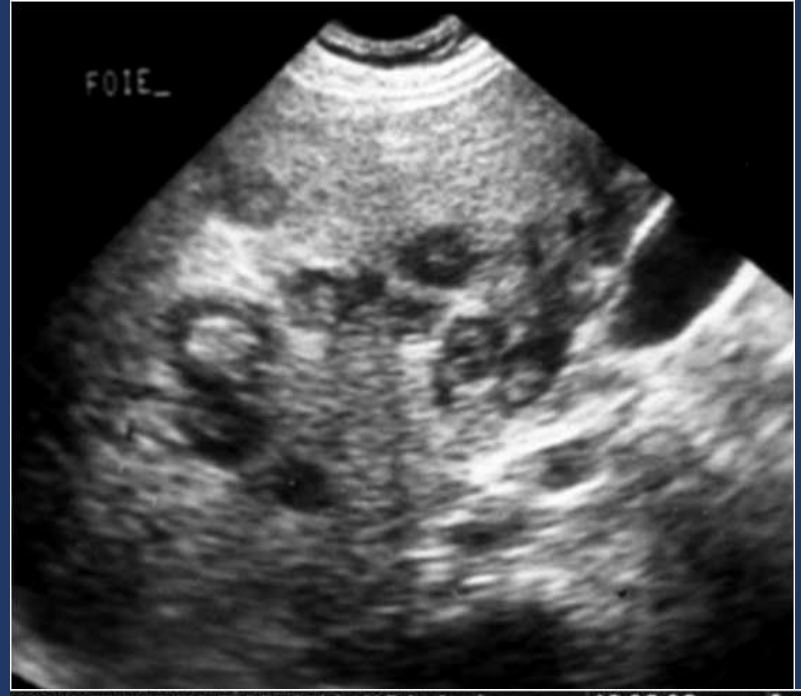
Metastatic disease

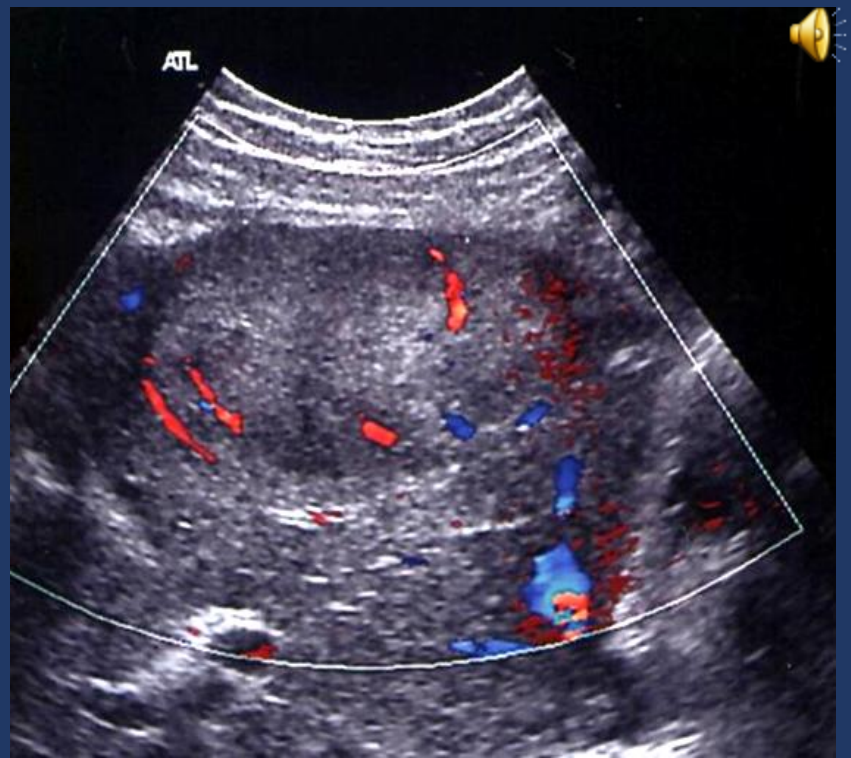
Variable US features

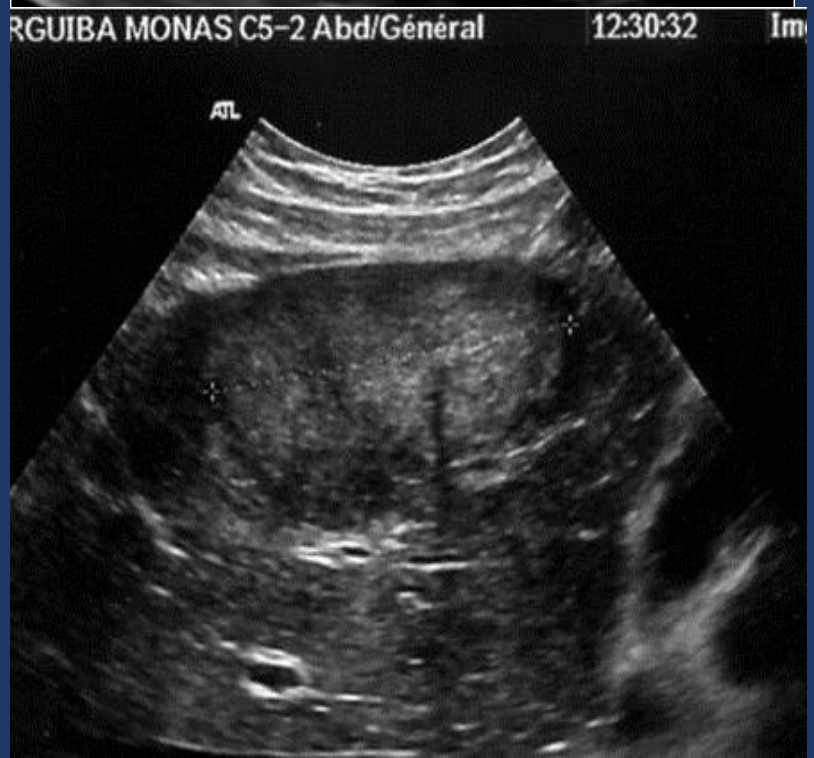
Iso, hyper or hypo echoic

Contrast-enhanced US (CEUS) (84% accuracy)

Intraoperative US (IOUS) (96% accuracy)







Metastatic disease

. MDCT are the most commonly used imaging modalities for detection and characterization of hepatic metastases

. Most liver metastases are hypovascular and are best imaged during the portal venous phase (colon, stomach and pancreas)

. Hypervascular metastases enhancing on the arterial phase (neuroendocrine tumors, renal cell, breast, melanoma, thyroid)

. Calcification may be present with metastases from mucinous gastrointestinal tract tumors and from primary ovarian, breast, lung, renal, and thyroid cancer

. Other features : Hemorrhagic or cystic metastases

Metastatic disease

. On MRI, metastases are variable but are usually hypo- to isointense on T WI and iso- to hyperintense on T2 WI

. Metastatic tumors with liquefactive necrosis or cystic neoplasms show higher signal intensity on T2 WI

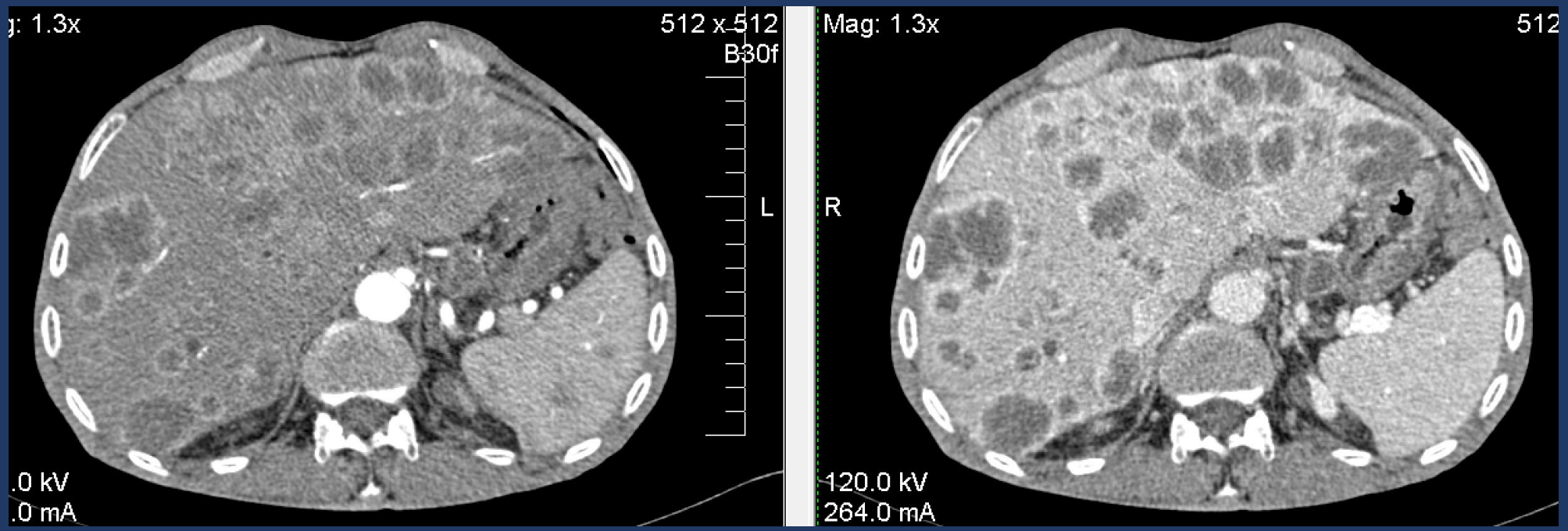
. Metastases may show central hypointensity on T2WI (coagulative necrosis, fibrin, and mucin)

. High T1 signal intensity can be seen with metastases from melanoma, colonic adenocarcinoma, ovarian adenocarcinoma, multiple myeloma and pancreatic mucinous cystic tumor

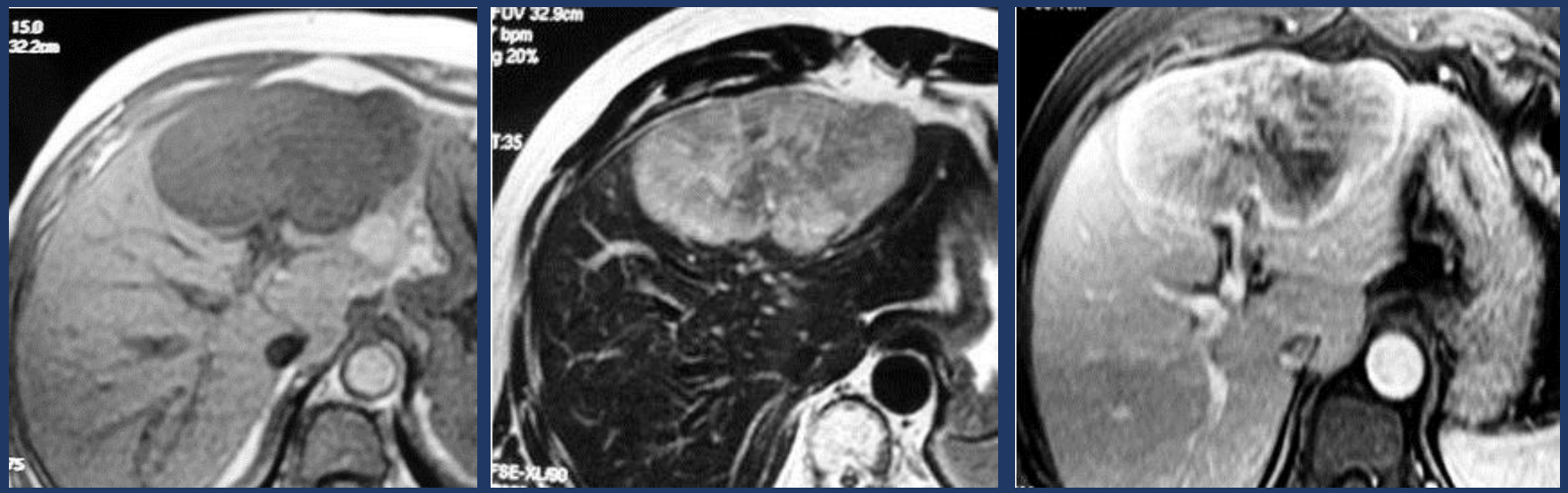
. Comparing T2-weighted (TE 90) and T2-weighted (TE 160) sequences, metastases become less intense→

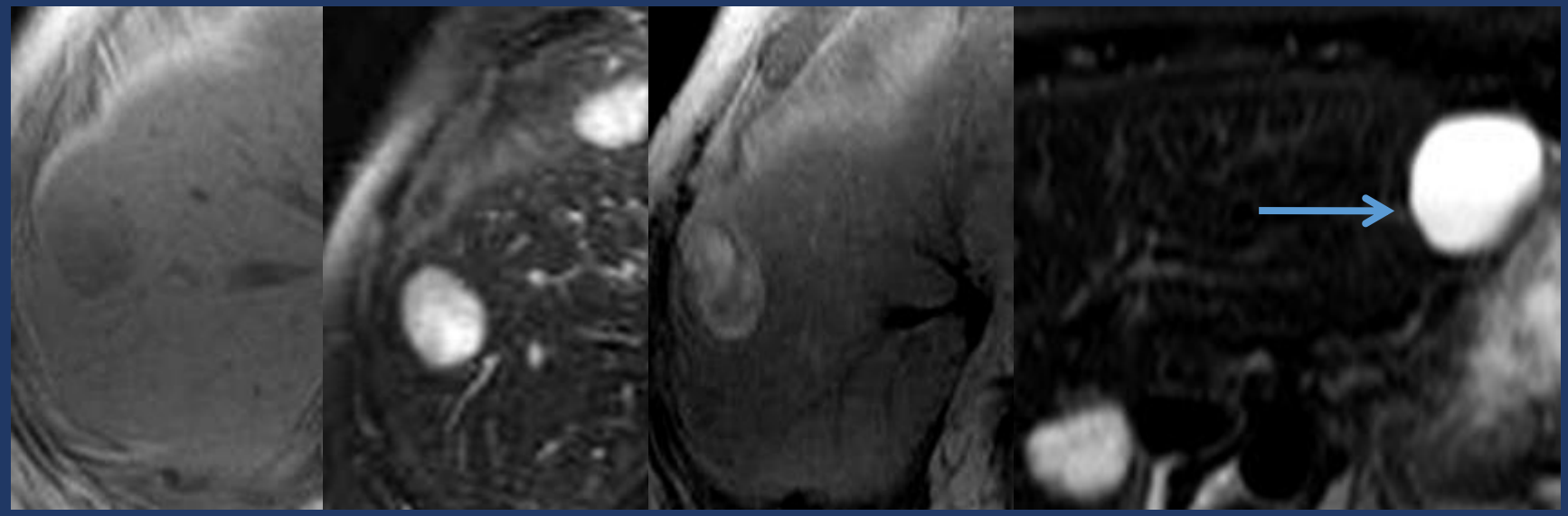
Characterization

. T1-weighted 3D dynamic contrast-enhanced MRI→ Detection


g: 1.3x
512 x 512
B30f
L
.0 kV
.0 mA
Mag: 1.3x
512
R
120.0 kV
264.0 mA

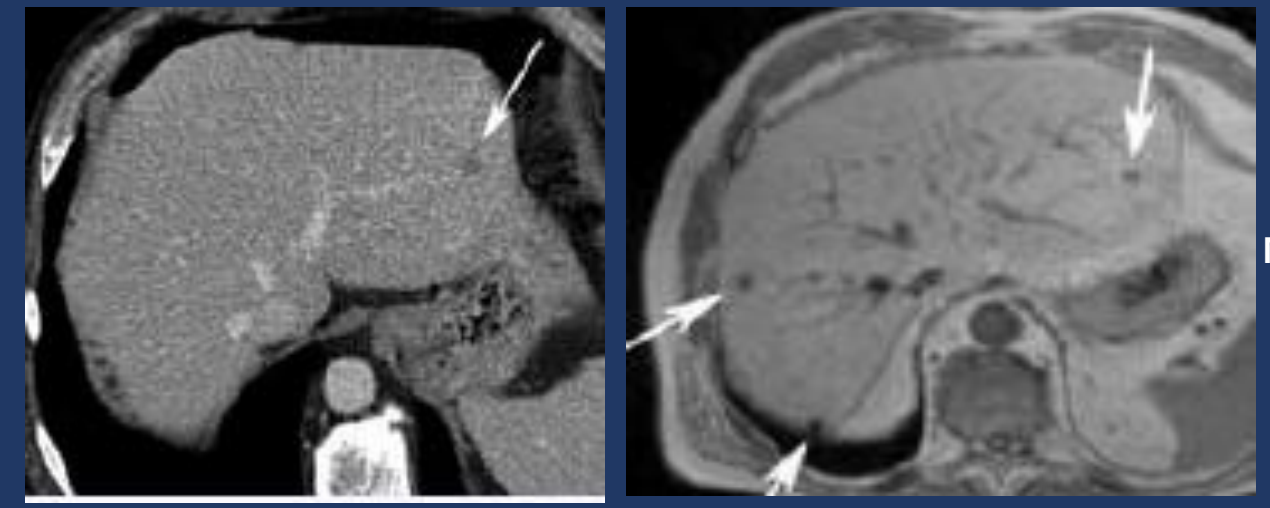
Metastatic disease





Metastatic disease

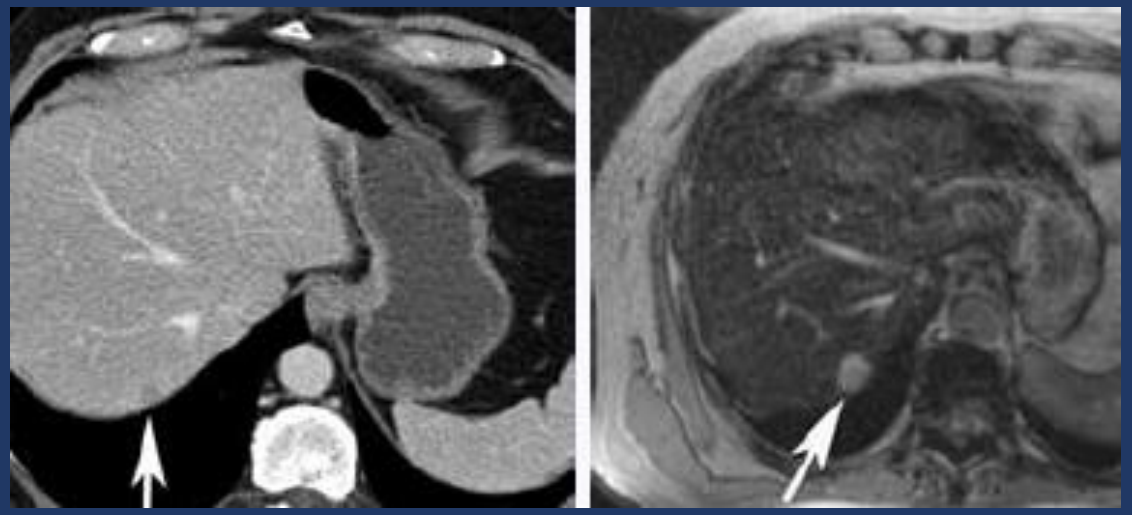
. Liver-specifc contrast agent: hepatobiliary agent(T1) or reticuloendothelial agent (superparamagnetic agent; T2)



T1

Multihance*
Primovist*

HB Agents



T2

Endorem*

USPIO Agents

Cholangiocarcinoma

- malignant tumors arising from cholangiocytes in the biliary tree and are the second most common primary hepatic malignancy after hepatocellular carcinoma (HCC) They tend to have a poor prognosis and high morbidity.
- much higher rates seen in south-east Asia and the Middle East
- with a mean age of 65 years
- Based on growth characteristics, cholangiocarcinomas are classified as mass-forming, periductal-infiltrating, and intraductal-growing types.
- Intrahepatic cholangiocarcinomas include peripheral and hilar cholangiocarcinomas. The vast majority of peripheral cholangiocarcinomas are the mass-forming type, whereas the majority of hilar cholangiocarcinomas are the periductal-infiltrating type.

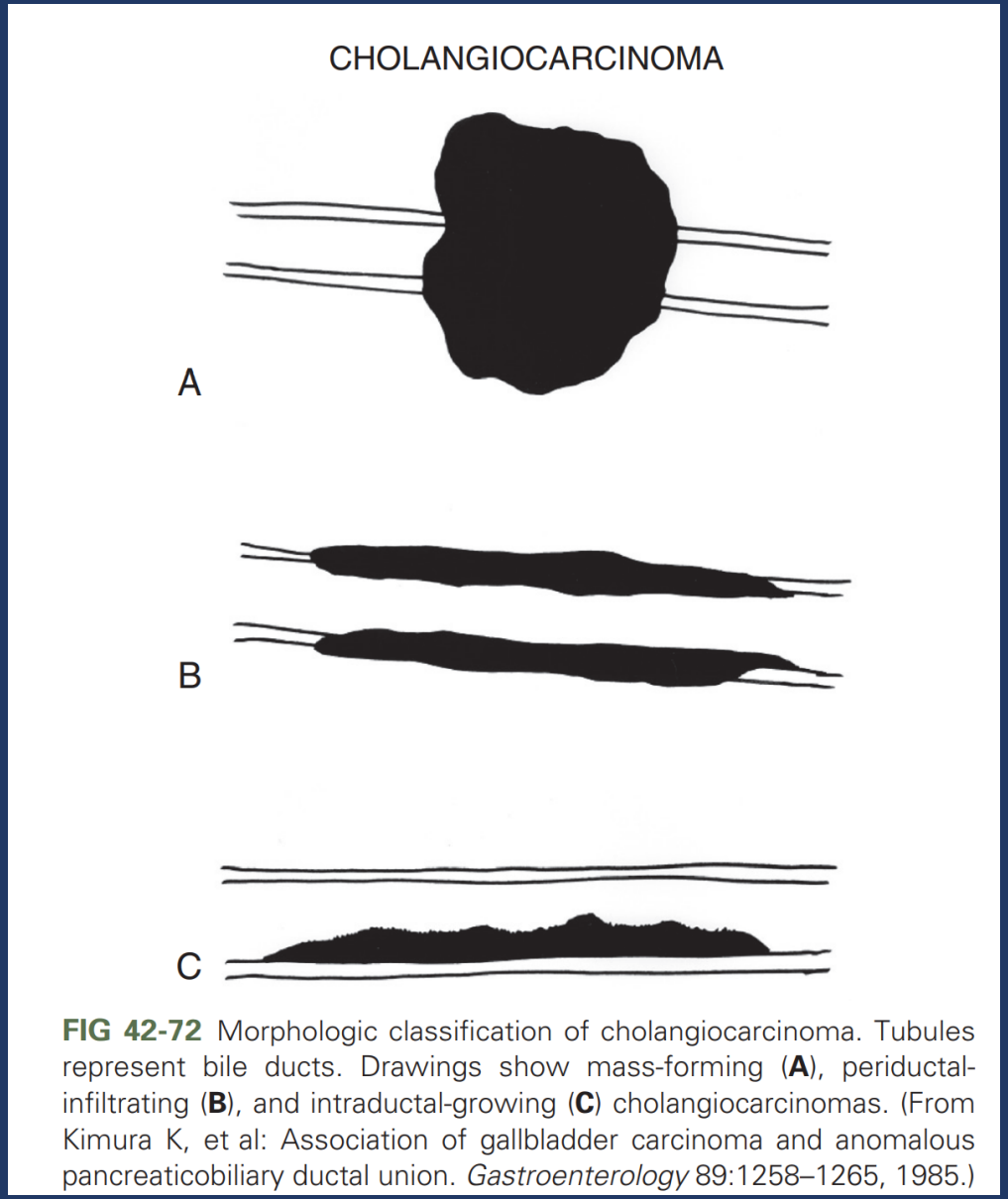
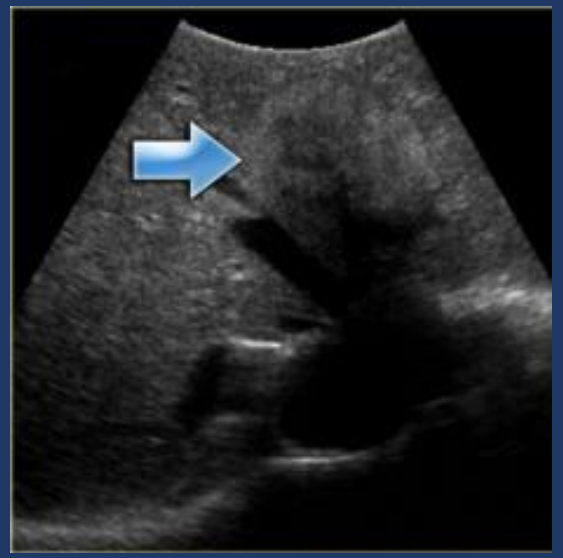
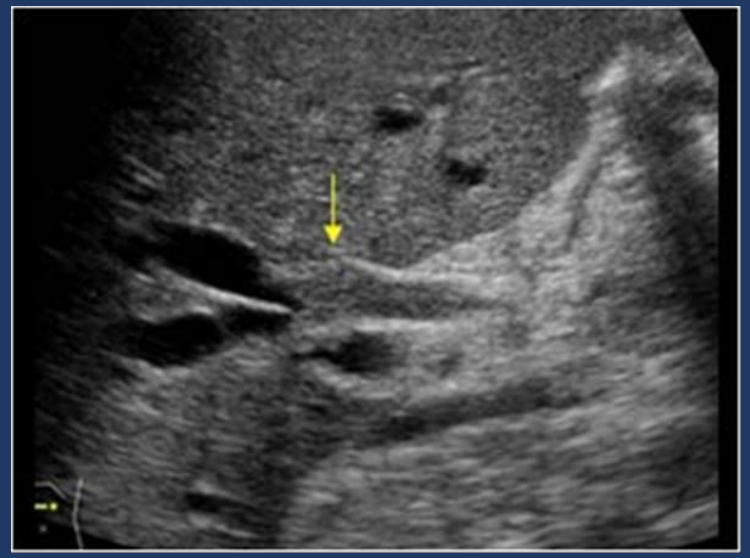


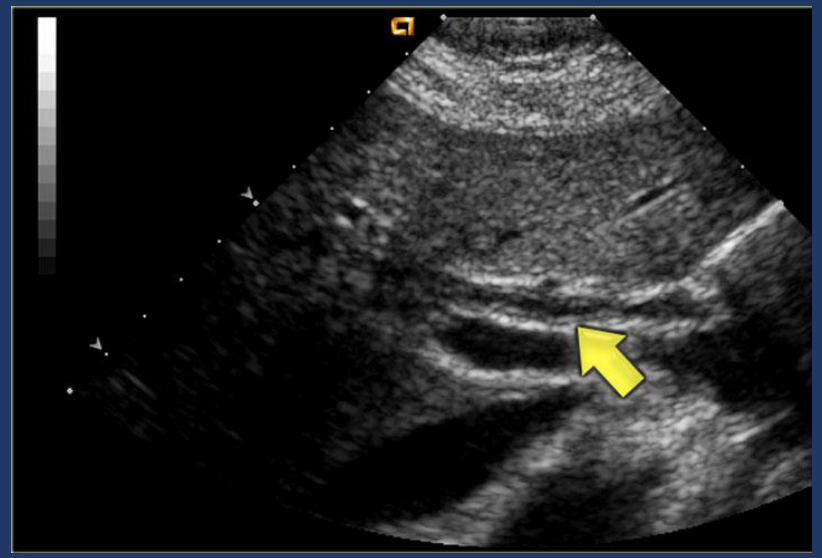
FIG 42-72 Morphologic classification of cholangiocarcinoma. Tubules represent bile ducts. Drawings show mass-forming (**A**), periductal-infiltrating (**B**), and intraductal-growing (**C**) cholangiocarcinomas. (From Kimura K, et al: Association of gallbladder carcinoma and anomalous pancreaticobiliary ductal union. *Gastroenterology* 89:1258–1265, 1985.)

Ultrasound

- **Mass-forming intrahepatic:** tumors will be a homogeneous mass of intermediate echogenicity with a peripheral hypoechoic halo of compressed liver parenchyma.
- They tend to be well delineated but irregular in outline and are often associated with capsular retraction which, if present, is helpful in distinguishing cholangiocarcinomas from other hepatic tumors.







CT scan

- **Mass-forming cholangiocarcinomas:**

typically homogeneously low in attenuation on noncontrast scans, and demonstrate heterogeneous minor peripheral enhancement with gradual centripetal enhancement.

The rate and extent of enhancement depend on the degree of central fibrosis.

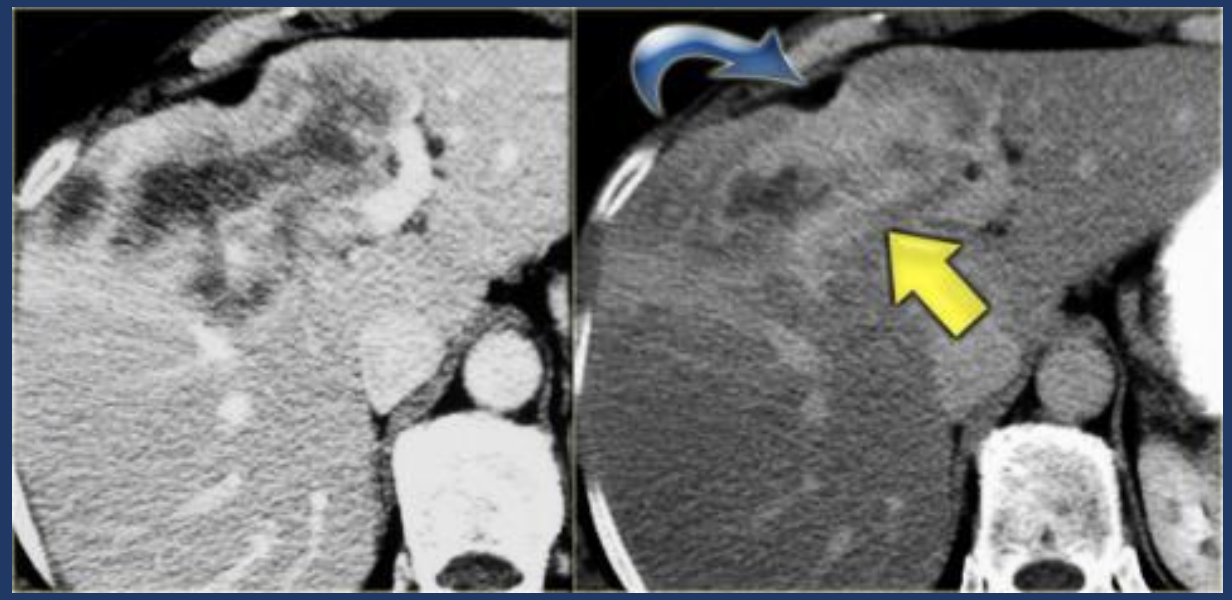
Slow diffusion of contrast media from the vascular space results in delayed and prolonged enhancement of the tumor, best seen 10–20 min after contrast media administration.

Again, capsular retraction may be evident.

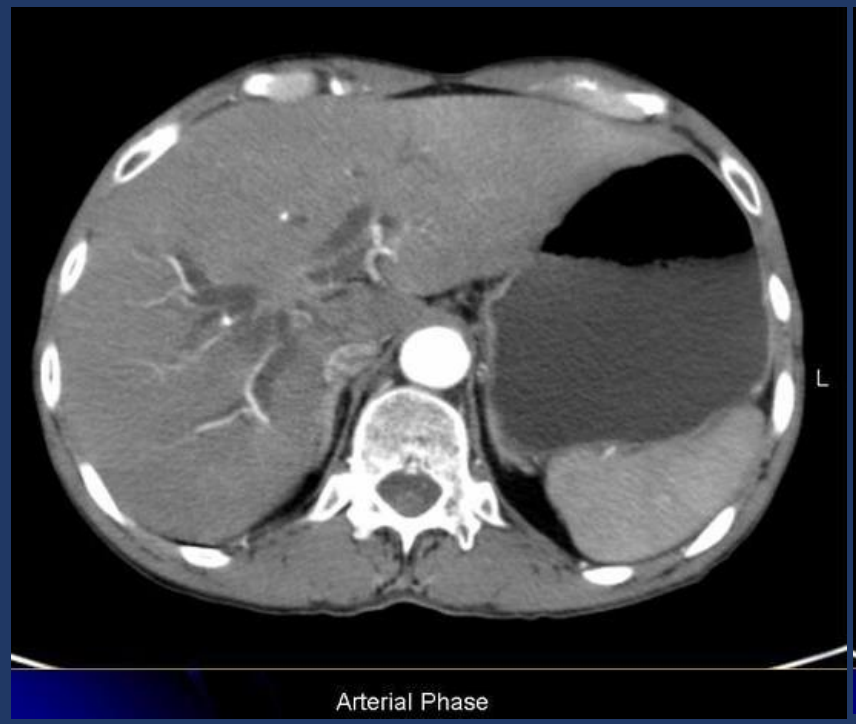
The bile ducts distal to the mass are typically dilated.

Although narrowing of the portal veins - or less frequently, hepatic veins - is seen, unlike HCC, cholangiocarcinoma only rarely forms a tumor thrombus .

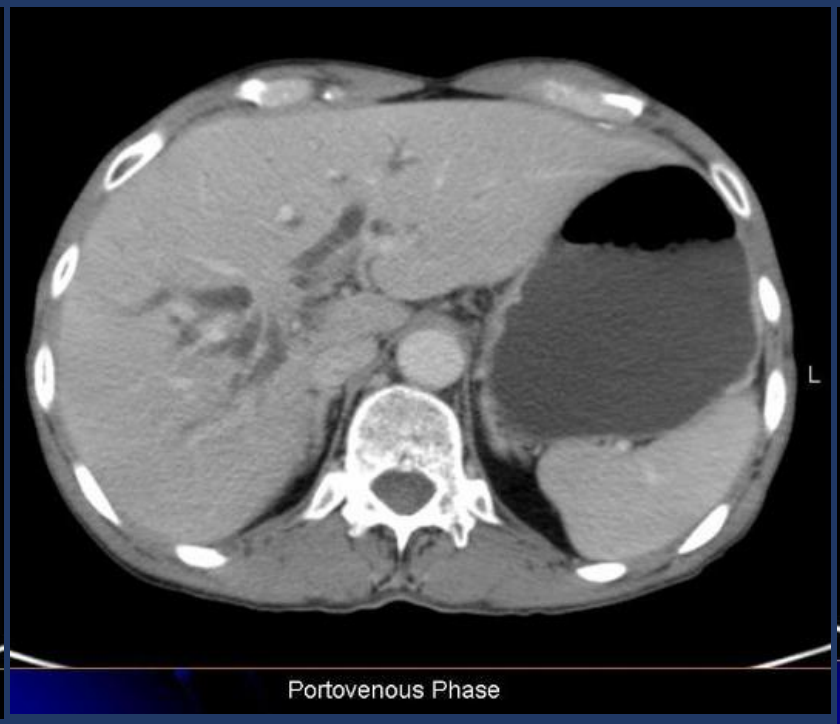
Lobar or segmental hepatic atrophy is usually associated with vascular invasion.



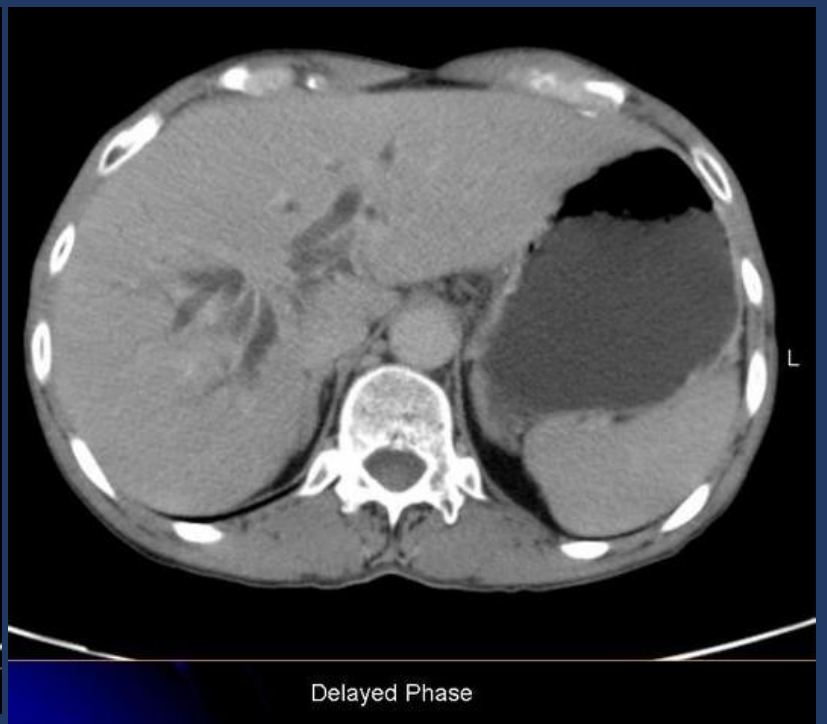
The key findings to look for are:
•Delayed enhancement
•Peripheral biliary dilatation
•Capsular contraction



Arterial Phase

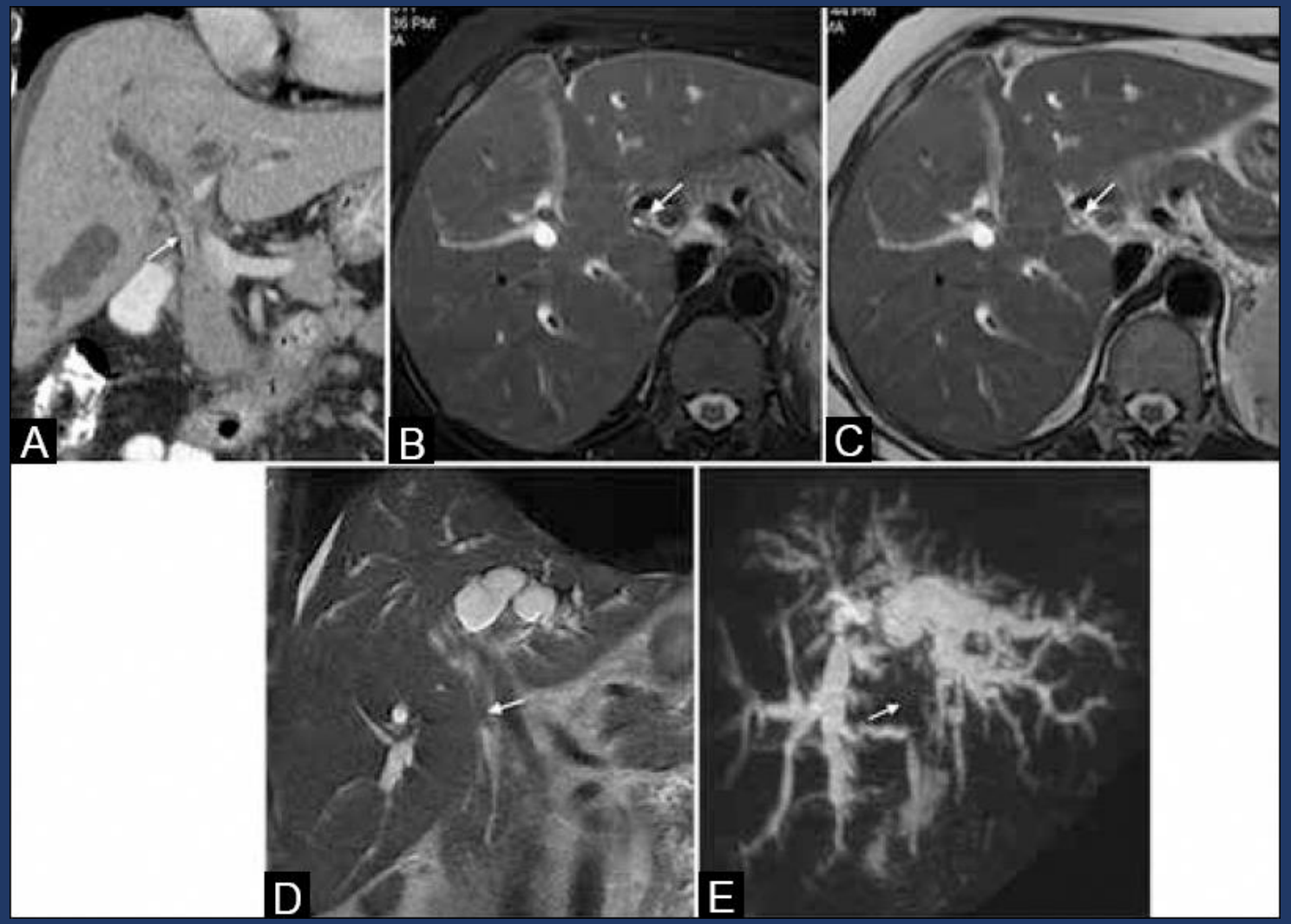


Portovenous Phase

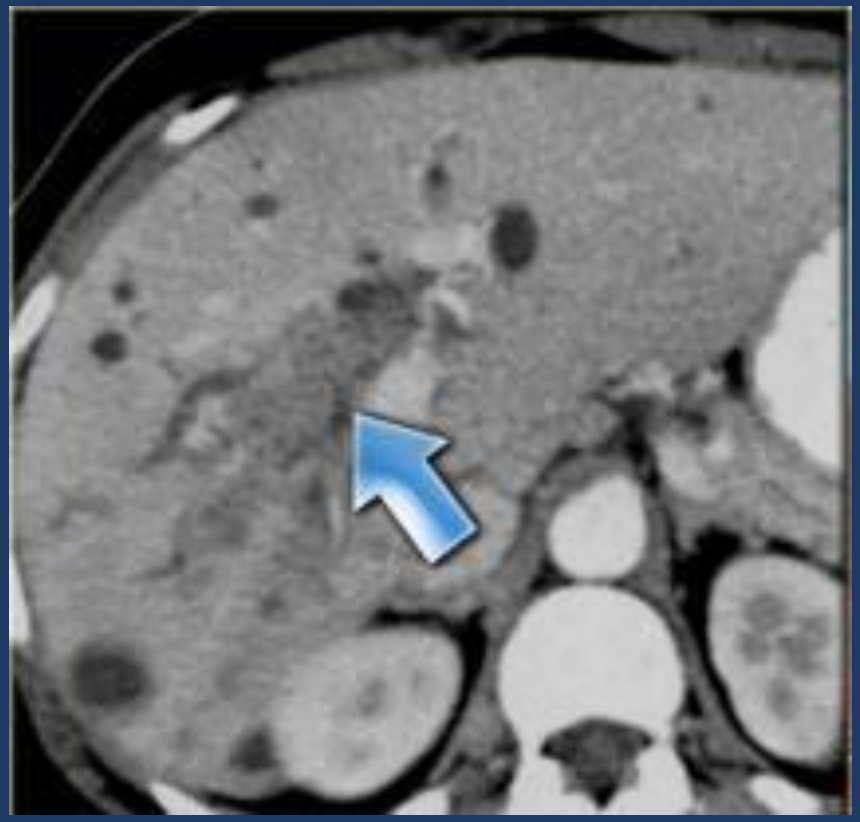


Delayed Phase

Periductal-infiltrating type



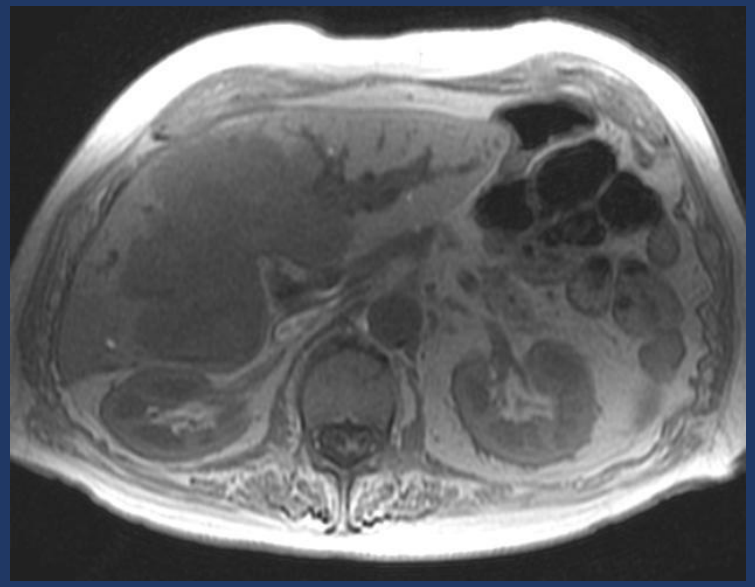
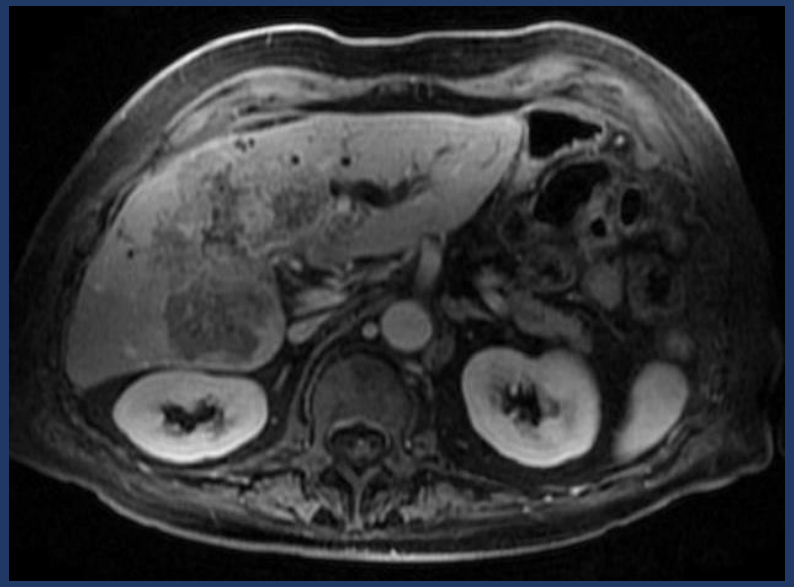
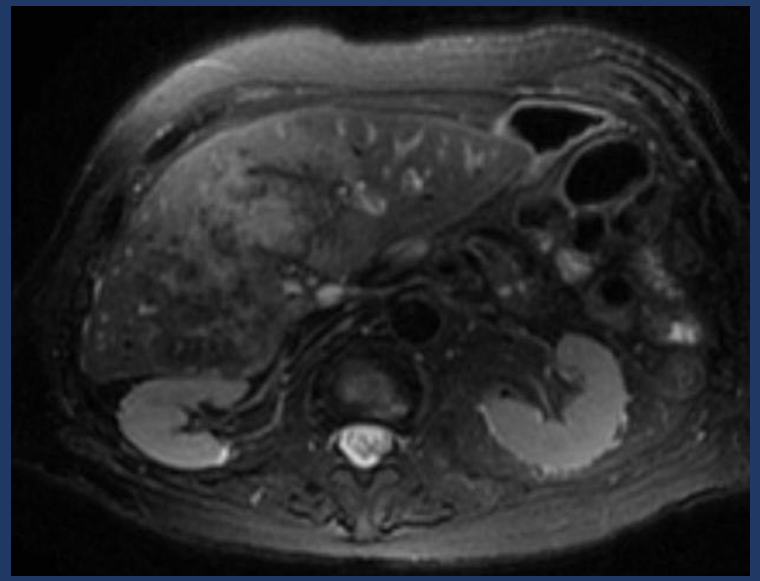
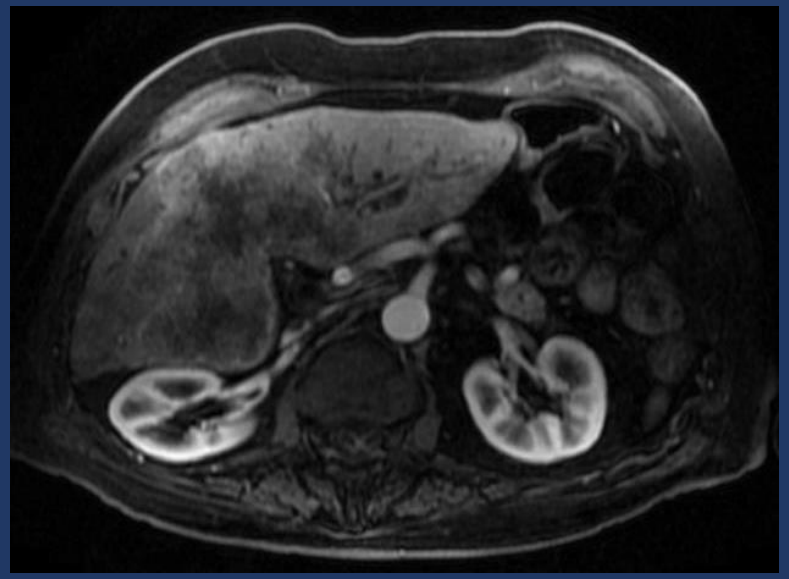
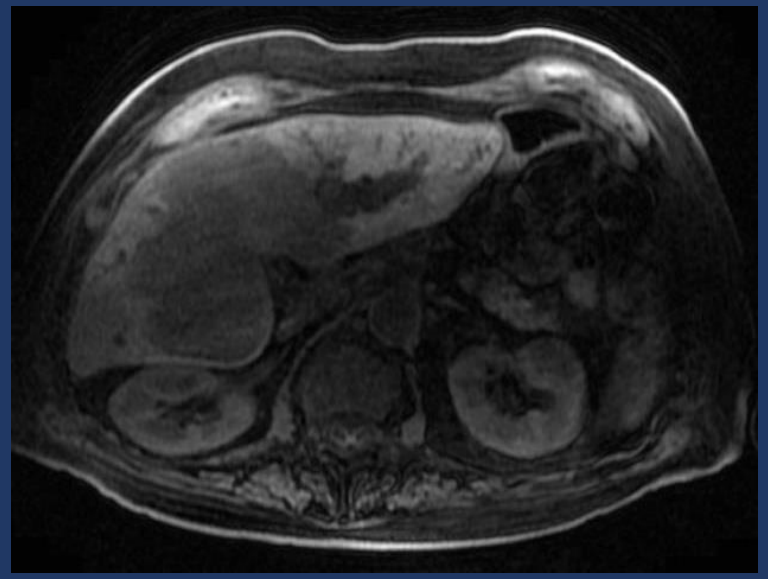
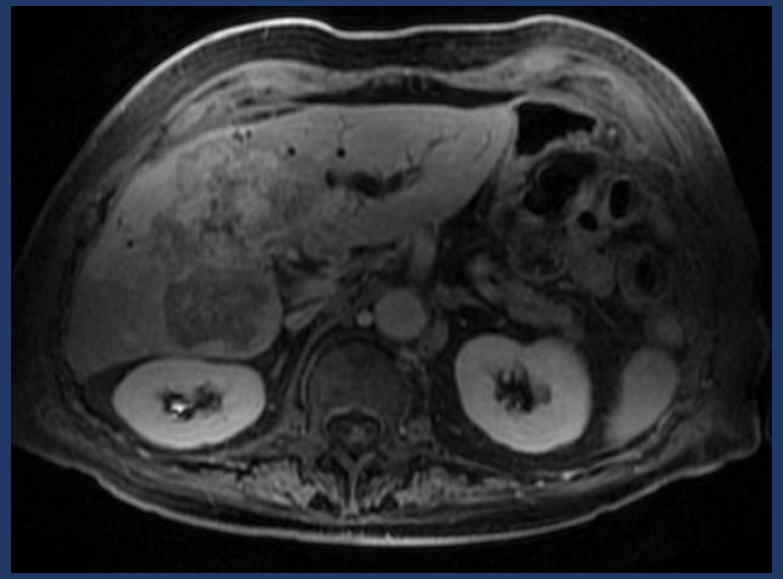
Intraductal Cholangiocarcinoma



These are very rare tumors.
They present as a intrabiliary mass with biliary dilatation peripheral to the mass.

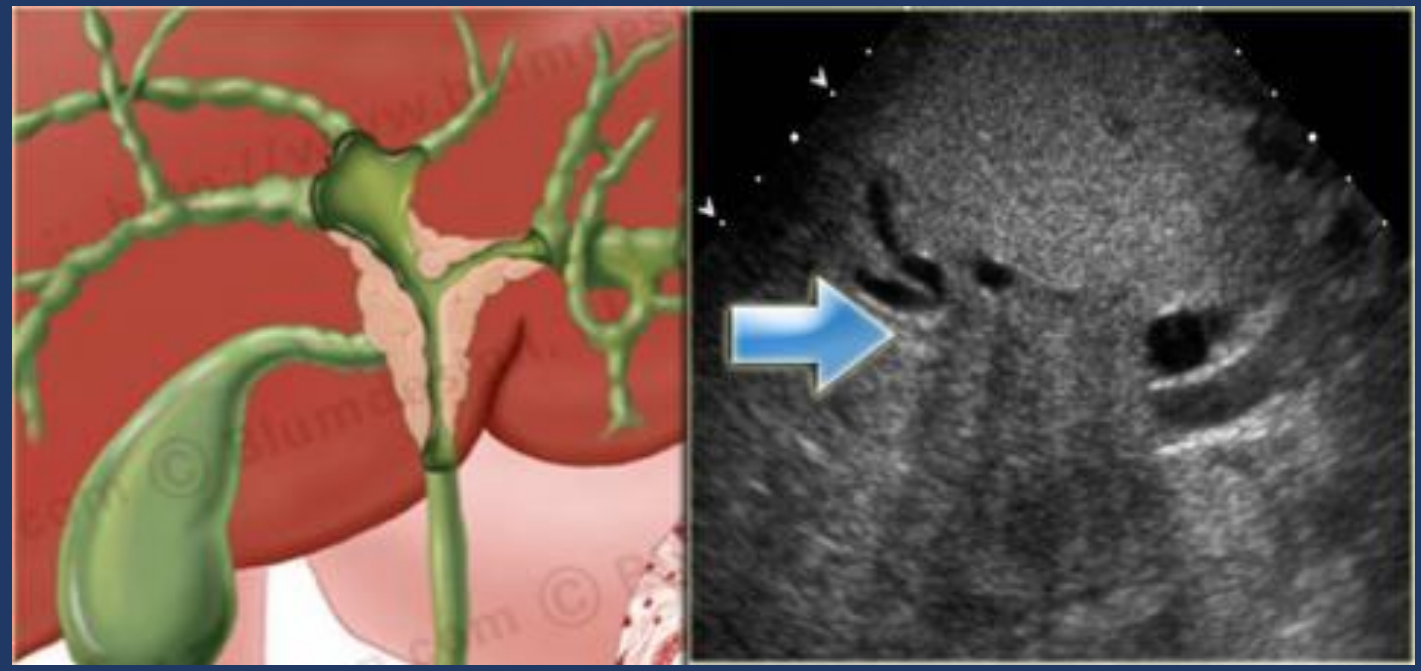
MRI/MRCP

- MRI is the imaging modality of choice, as it can best visualize all three the tumor itself, the biliary ducts and the blood vessels, all of which are essential for determining resectability. Appearances on MR are similar to those described above for CT, except that MR is more sensitive to contrast enhancement and bile duct visualization.
- Using MRI ICC has a nonspecific appearance:
- on T2 w the signal intensity ranges from markedly increased to mildly increased relative to liver; tumors with high fibrous content tend to be hypointense on T2 w.
- Precontrast T1 w is iso- to hypo-intense
- The enhancement pattern is similar to that seen on CT: minimal or moderately incomplete rim enhancement at the tumor periphery on the early images with progressive central contrast enhancement in later phases
- DWI/ADC: a peripherally hyperintense "target" appearance on DWI favors cholangiocarcinoma over hepatocellular carcinoma

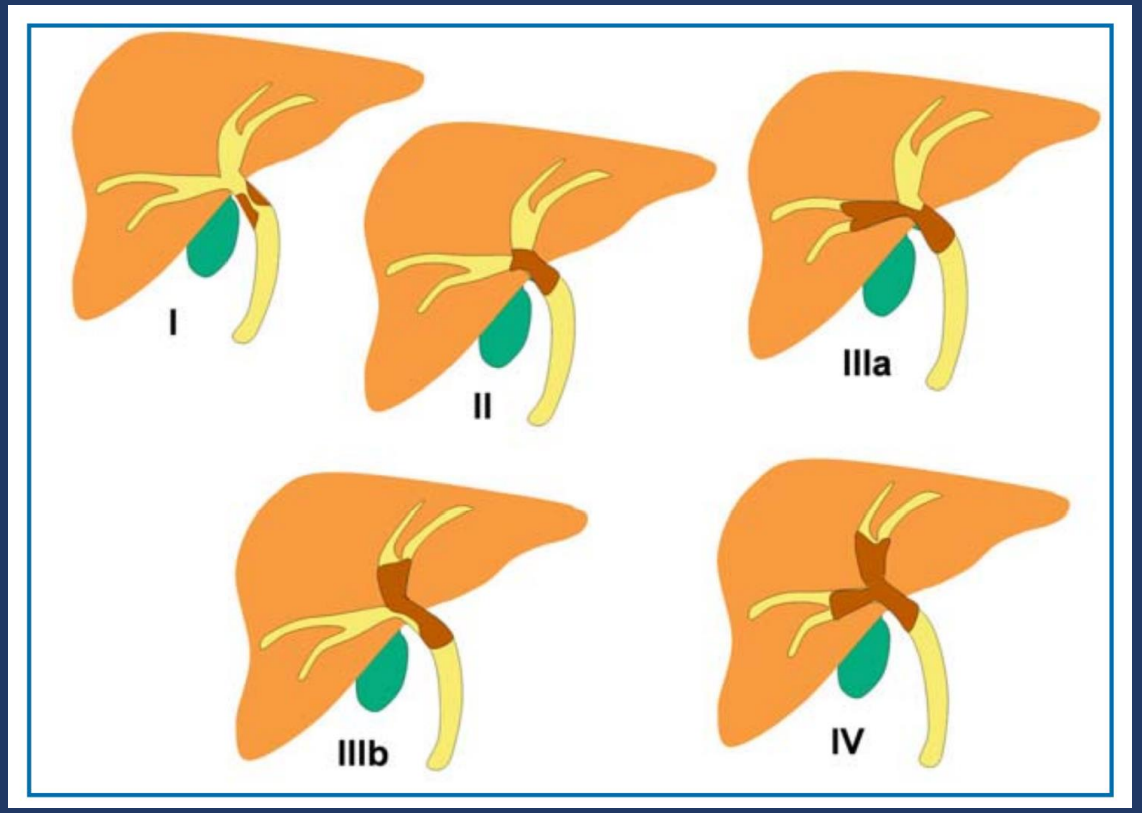
Klatskin Tumor - Hilar Cholangiocarcinoma

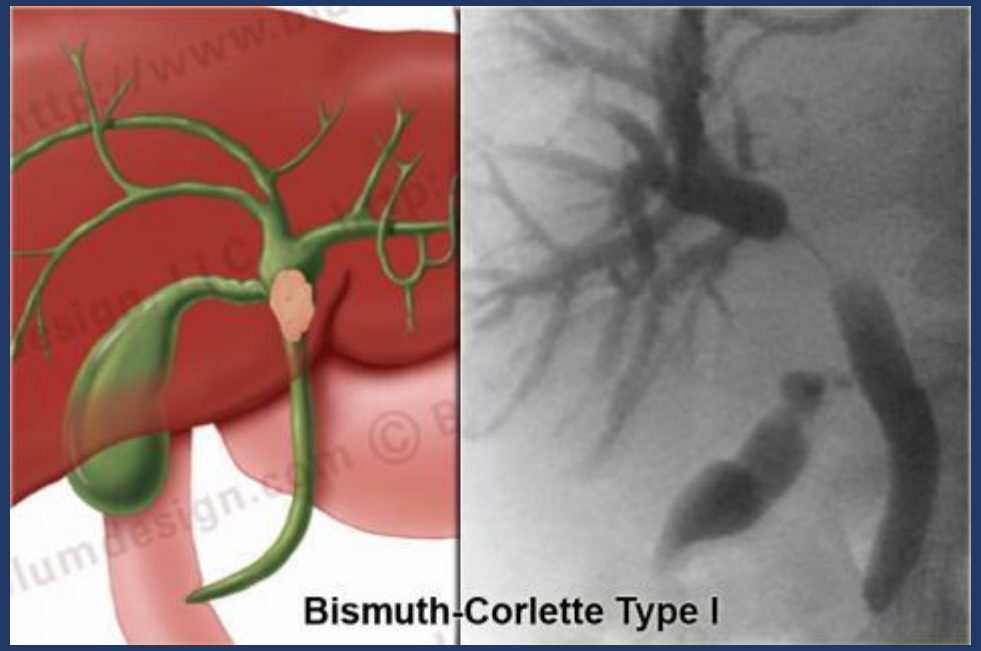
- **Klatskin Tumor - Hilar Cholangiocarcinoma**
- The most common site of biliary adenocarcinoma is at or near the confluence of the right and left hepatic ducts.
  These tumors are also known as Klatskin tumors.
  Klatskin tumors have an aggressive biologic behavior.
- Imaging features:
- Duct dilatation
- Ill-defined mass
- Lobar atrophy
- Vascular invasion

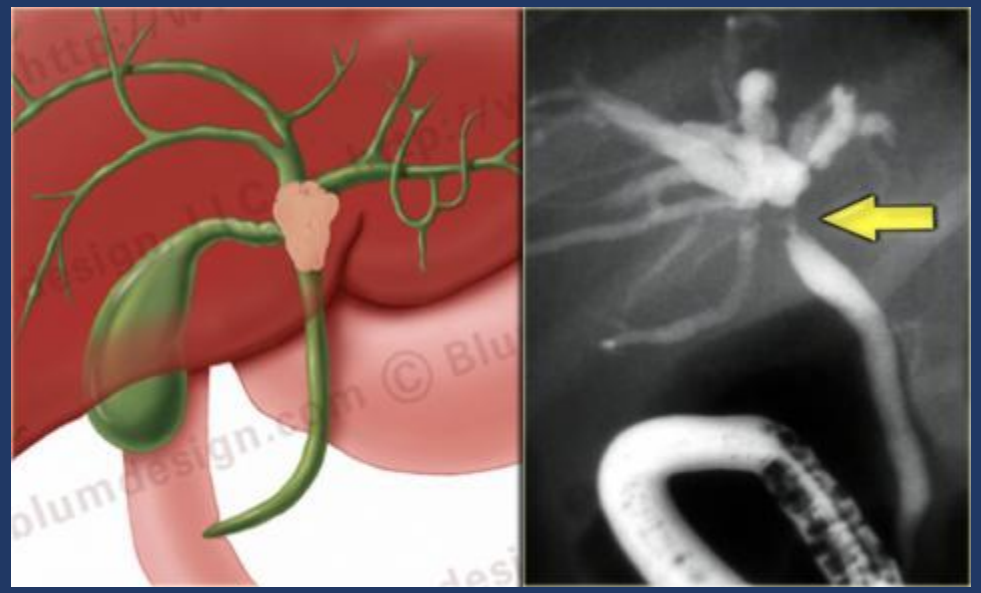


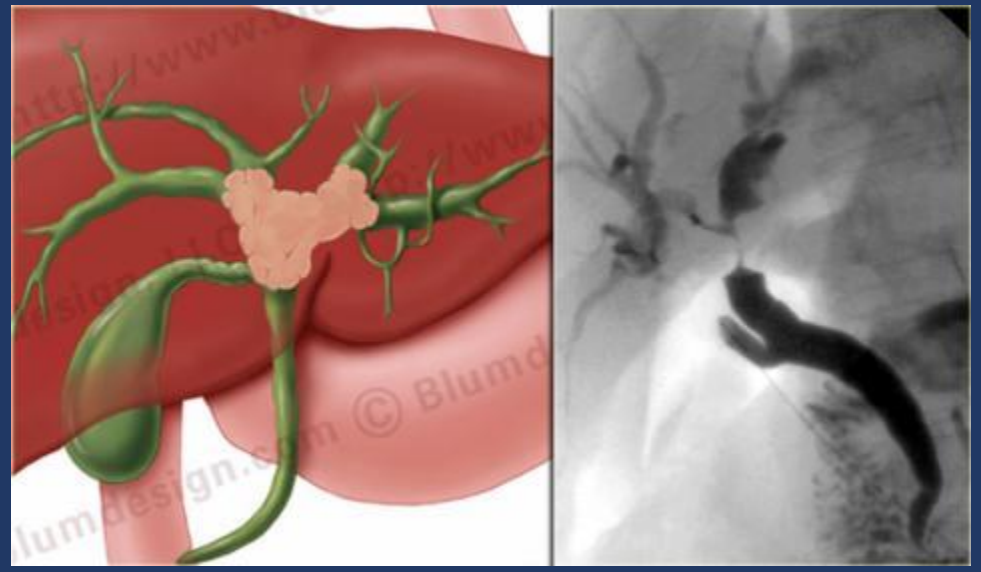
Bismuth-Corlette classification

- **type I**
  - limited to the common hepatic duct, below the level of the confluence of the right and left hepatic ducts
- **type II**
  - involves the confluence of the right and left hepatic ducts
- **type IIIa**
  - type II and extends to the bifurcation of the right hepatic duct
- **type IIIb**
  - type II and extends to the bifurcation of the left hepatic duct
- **type IV**
  - extending to the bifurcations of both right and left hepatic ducts
    or
  - multifocal involvement
- **type V**
  - stricture at the junction of common bile duct and cystic duct




Bismuth-Corlette Type I





Radiology report

- bile ducts (Bismuth-Corlette classification)
  - tumor confined to the common or hepatic bile duct?
  - extension to right or left hepatic duct or both?
  - does tumor involve second-order radicles and on which side?
- portal vein: does tumor abut/encase main/right/left portal vein and to what extent?
- hepatic artery
  - common hepatic artery/hepatic artery proper involved and to what extent?
  - right/left hepatic artery involved and to what extent?
  - variant arterial anatomy, if any
- lymph nodes: enlarged regional (N1) or distant (N2) lymph nodes?
- assess for distal metastases

Klatskin Tumor: Resectability

These tumors are unresectable when:

- Bilateral tumor extension
  - Into secondary ducts
  - Into hepatic parenchyma
  - Hepatic artery or portal vein
- Occlusion main portal vein
- N2 nodes (nodes around the pancreas)
- Distant metastases