

The Liver “Summary”

LIVER IMAGING TECHNIQUES

I- PLAIN RADIOGRAPHY : Limited - can see hepatomegaly & Ca.

II- ULTRASOUND

III- COMPUTED TOMOGRAPHY

- 1- **Single Phase CT** : “Precontrast + IV phase “as delayed phase in triphasic”
- 2- **Tri phasic** : Precontrast + 3 phases
- 3- **Angiographically Assisted CT**: CT with arterial Phase by Femoral catheter.
“obsolete after MDCT”

- The majority of solid liver lesions have a predominantly arterial blood supply
- Liver parenchyma receives 75–80 per cent of its blood supply via the portal v.

- **Phases of enhanced Triphasic CT imaging:**

- I- Early arterial phase
- II- Late arterial phase. (20–40 s post injection).
- III- Portal phase. (60–80 s post injection).
- IV- Late and delayed phase imaging.

Delayed CT imaging;

in selected cases for
characterization, e.g.
haemangiomas.

- CT arteriography and CT arteriportography (CTAP)

IV- MAGNETIC RESONANCE IMAGING : Wide range of protocols

V- LIVER SCINTIGRAPHY

- Contrast material: 99mTc-sulphur colloid or albumin colloid.
- Target the reticulo-endothelial system.
- Value: *It is infrequently used as a primary diagnostic investigation*

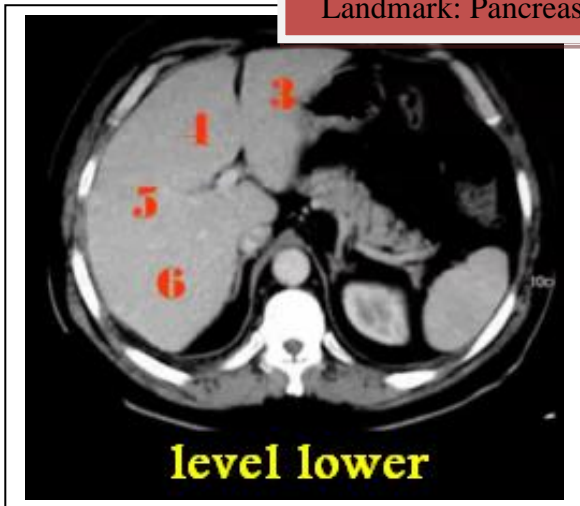
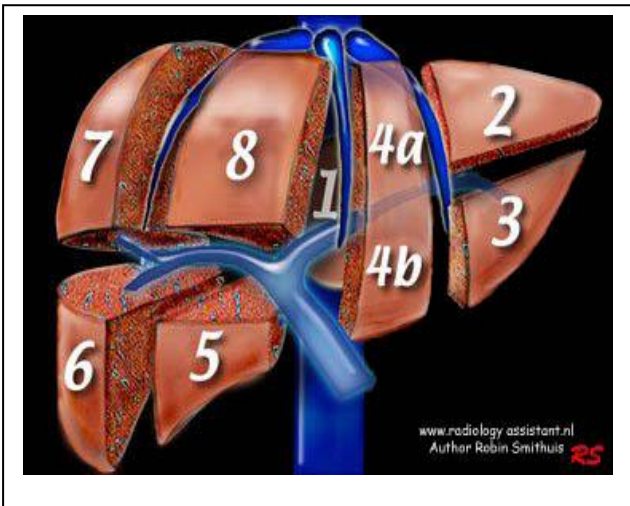
* Can help to further characterize known lesions when CT and MRI are not available.

* Liver scintigraphy lacks anatomical specificity.

VI- ANGIOGRAPHY : 1- Arteriography 2- Portal venography



Landmark: Pancreas



<i>Proximal</i>	7	8	4	2
<i>Distal</i>	6	5	4	3

Hepatic segmentation

caudate lobe	Segment I
Lateral segment left lobe superior	Segment II
Lateral segment left lobe inferior	Segment III
Medial segment left lobe	Segment IV
Anterior segment right lobe inferior	Segment V
Posterior segment right lobe inferior	Segment VI
Posterior segment right lobe superior	Segment VII
Anterior segment right lobe superior	Segment VIII

DIFFUSE HEPATIC DISEASE

→ BENIGN DIFFUSE DISEASE ←

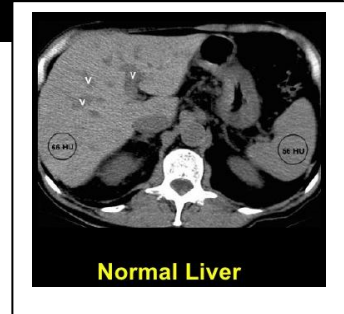
1 - Fat infiltration

- **Incidence:** relatively common finding
- **Etiology:** increased **triglyceride**

loading of hepatocytes.

- 1- Alcohol abuse, 5- tetracyclines, steroids
- 2- Obesity, 6- malnourishment
- 3- Diabetes mellitus, 7- total parenteral nutrition
- 4- cystic fibrosis, 8- ileal bypass.

▪ **Diagnosis:**



Ⓡ **US:** increased reflectivity → obscures the portal vein margins. - Further imaging may be required for confirmation as fibrosis can also cause increased reflectivity.

Grades of Fatty Liver:

- **Grade I:** Visualized portal walls & Diaphragm
- **Grade II:** Obscure Portals – Visualized Diaphragm.
- **Grade III:** Obscure Both

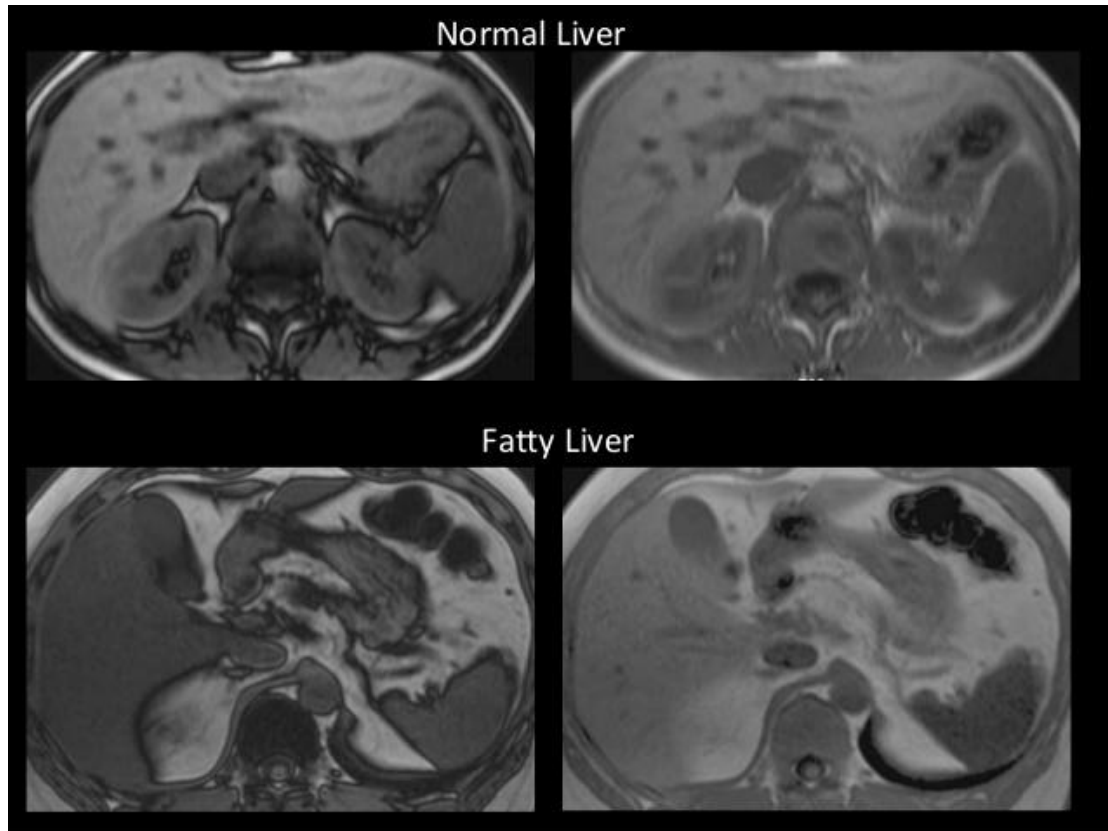
Ⓡ **CT:**

- Increase Each **mg of triglyceride** / per **gram of liver** → attenuation decreases by 1.6 HU per The architecture of the liver. i.e. CT can demonstrate and quantify diffuse fatty infiltration
- Increasing fat infiltration the liver → attenuation falls below that of the spleen → **reverse the normal liver–spleen difference.**

SUMMARY OF LIVER IMAGING

- In severe cases the liver attenuation $<$ blood, \rightarrow the hepatic vasculature appears '*enhanced*'.

Ⓜ MRI: (*most sensitive and specific technique for demonstrating hepatic fat infiltration.*)



2- Cirrhosis

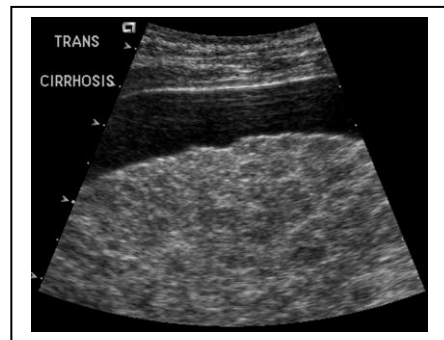
- **Def:** Cirrhosis is the endpoint of a wide variety of chronic disease processes → hepatocellular necrosis leading to hepatic fibrosis and nodular regeneration.
- **Pathology :**
 - The early changes ← Histological.
 - As cirrhosis progresses → widespread **fibrosis** and **nodular** regeneration develop → macroscopic changes of liver morphology.

Ⓡ **US:** It demonstrates the following:

- **In relatively early cirrhosis**

“Changes detected by high-frequency transducers.”

- **In advanced cirrhosis:**



- **Nodular liver margin**, *“particularly when ascites is present”*.
- **Increases reflectivity** → loss of the margins of the portal vein branches,
- Both fatty infiltration & fibrosis are coexist in alcoholic cirrhosis.
- The overall texture becomes **coarser** or **more heterogeneous** as cirrhosis progresses, *but this is difficult to quantify.*
- **In end-stage cirrhosis** → the liver atrophies.

Ⓡ **Doppler US:** → other nonspecific features of cirrhosis:

- **Reduced** main portal vein blood flow (**<10 cm/s mean peak**) or hepatofugal flow.
- **Increased Hepatic arterial flow** in advanced cirrhosis *“ may be mistaken for enlarged bile ducts on US”*.

Ⓡ CT: Is *relatively insensitive* to the changes of *early* cirrhosis.

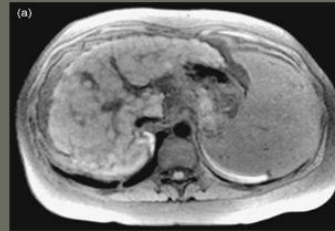
Ⓡ

- Nodularity
 - Best seen affecting the liver margin (especially left lateral)
- Cobblestone appearance
- Diffuse heterogeneity of liver parenchyma
- Atrophy of the right lobe and hypertrophy of the left and caudate lobes



CT Normal

Van Beers, et al. *AJR*, 2001



MRI Chronic Cirrhosis

Murakami, *Seminars*, 2001.

Ⓡ MRI:

- Is also relatively insensitive to the changes of early cirrhosis.
- There are *no specific changes* of parenchymal signal intensity on T1w or T2w imaging.
- **In advanced cirrhosis** MRI delineates the morphological changes
- non-invasive assessment of portal vein patency along .

3- VIRAL HEPATITIS

- ***Incidence:*** significant disease worldwide;
- ***Diagnosis:***
 - Based on serological tests. - imaging is relatively nonspecific.
 - ***Acute hepatitis*** → Imaging exclude other causes of jaundice such as bile duct obstruction.
 - ***In chronic hepatitis*** with cirrhosis, → help monitor the progression of disease, development of portal venous hypertension and complications such as hepatocellular carcinoma.

Ⓡ US:

- in acute viral hepatitis → nonspecific *decreased reflectivity*
- In majority of cases → normal parenchyma.
- GB wall thickening: common nonspecific finding in acute hepatitis.

Ⓡ *CT, MRI and angiography* are of limited value until cirrhotic changes develop.

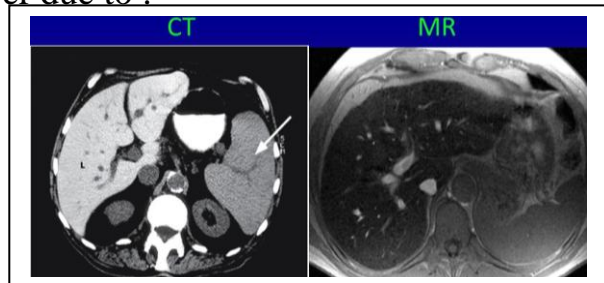
4-Haemochromatosis and iron overload

● **Etiology:** Iron deposition in the liver due to :

- Multiple transfusions.
- Genetic (autosomal recessive)

● **Pathology:**

- Increase risk of malignancy esp. “hepatocellular carcinoma “.
- Iron deposited in hepatocytes → cirrhosis.
- In other organ tissues including the myocardium, skin and endocrine glands.
- **iron deposition** Initially → generalized.
- development of cirrhosis → uneven distribution.



Ⓡ MRI: *most specific imaging technique*

- Reducing the parenchymal T2.
- When severe it will affect T1w images.
- **In other organs** , spleen and pancreas → Abnormally reduced signal on T2w imaging “ main feature”.

The liver signal is abnormally reduced (to less than that of adjacent muscle).

Ⓡ Unenhanced CT:

- Hepatic iron deposition → **increase in HU value** (>75 HU).
- *Nonspecific finding*, → several other causes.

SUMMARY OF LIVER IMAGING

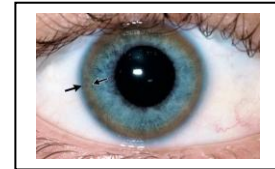
- Increased attenuation / correlated with the amount of iron deposition *but* this is neutralized by the influence of any **fat deposition**.

Ⓡ US:

- No specific features In US except increased parenchymal reflectivity.

Wilson's disease

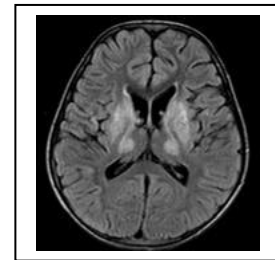
- **Etiology:** autosomal recessive condition.
- **Pathology:** **copper** is deposited in : cornea / lenticular nucleus of the brain. / liver → inflammation → cirrhosis



Ⓡ CT: rarely increase in hepatic attenuation.

Ⓡ MRI : may an *increase* in signal on *T1w* & a *decrease* on *T2w* images.

Ⓡ US : There are no specific features



→ MALIGNANT DIFFUSE DISEASE ←

- **Incidence:** Occasionally occur.
- **Etiology:**
- **Metastases** is commonest ex. from breast carcinoma.
- **Some primary** hepatic tumours, as HCC.
- **Lymphoma and leukaemia**
- **Diagnosis:** difficult. **Biopsy** is required .

FOCAL DISEASE

1- Calcification

→ **Benign**: - TB, - Pneumocystis infection, - Sarcoidosis, - pyogenic Abscess

- parenchymal haematoma. – Or in relation to a Giant haemangioma.

→ **In Malignant** lesions:

* **mucinous adenocarcinoma** of the colon.

* Primary : such as **H**epatoblastoma and **F**ibrolamellar hepatoma .

Ⓡ **Plain films**: may demonstrate gross calcification,

Ⓡ **unenhanced CT**: is more sensitive and will detect the subtle calcification occasionally found in metastases.

Ⓡ **US**: calcification foci have increased reflectivity with a posterior acoustic shadow.

- Focal pockets of gas → similar picture.

Ⓡ Scintigraphy and MRI are *insensitive* to calcification.

2- Aerobilia

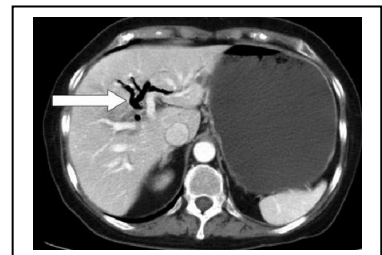
● **Etiology**:

1- Incompetence sphincter of Oddi:

-Sphincterotomy, - Large stone passage - Elderly.

2- Postoperative: *Roux loop procedure (GB – intestinal anastomosis).

3- Biliary – intestinal **fistula**: by *Stone eroding GB wall, *Duodenal ulcer, *Malignancy



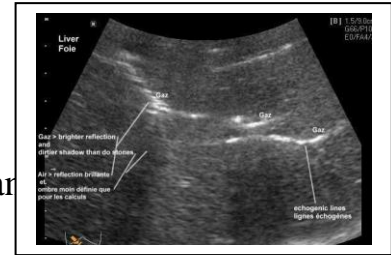
SUMMARY OF LIVER IMAGING

- **Pathology:** gas linear distribution radiating from the hilum → More centrally & Not reaching capsule (Due to direction of bile drainage toward the hilum)

Ⓡ **US:** - Ducts appears as echogenic linear structures.

Ⓡ Differentiated from calcification by: the distribution and

Ⓡ **CT:** - is extremely sensitive to the presence of gas.



3- Portal vein gas

- **Etiology:** increases intestinal permeability *and/or* **Increase in intestinal luminal pressure due to:

-Infections -Neonatal necrotizing enterocolitis -Crohn's disease.

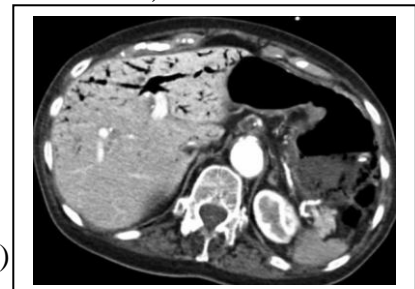
-Invasive abdominal malignancies (colon carcinoma, ovarian carcinoma),

-Gastric emphysema, -intestinal volvulus, -Blunt abdominal trauma,

- **Pathology:** *outcome relates to etiology*

- The gas radiates out from the hilum

(*Reaching the capsule* as blood pass from the hilum & Out ward)



Ⓡ **US:** is the *most sensitive* method of detection as moving gas bubbles.

Ⓡ **CT:** it may become visible if a large amount of gas accumulates.

4- Parenchymal gas

- **Etiology:** - Abscess Gas-forming organism / or infarct.

- Trauma
- Hepatic arterial thrombosis ← liver transplantation.
- Embolization or thermal ablation of liver tumours.

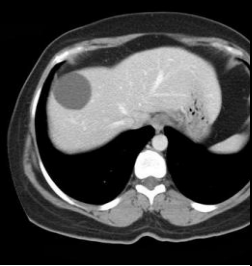

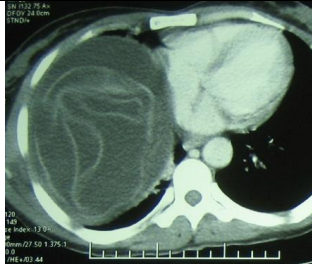
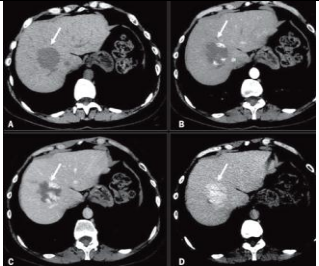
Ⓡ **CT:** best delineates parenchymal gas and any related pathological changes.

Ⓡ **US:** when large or peripheral or may be confused with adjacent bowel.

SUMMARY OF LIVER IMAGING

CYSTIC LESIONS

4 Common	2 Rare
1- Simple 2- Abscess 3- Hydatid 4- Hemangioma <i>“Commonest benign Hepatic lesion”</i>	1-Cystic Mets 2- Cystadenocarcinoma

Simple Cyst	Abscess	Hydatid	Hemangioma
-Thin wall -Clear contents -No enhance -No Ca	- C.P. -Ring enhance +/- Air	-Ca very Common - Daughter cysts *** - Floating shadows	*** Iris sign “Marginal nodular enhancement with delay fill in --Giant = 6:10 cm
			

Cystic Metastases	Cystadenoma / Cystadenocarcinoma
Known primary + Cystic lesion	-3 : 40 cm -Multilocular +Mural nodule -Both Not differentiated should excised

SUMMARY OF LIVER IMAGING

SOLID & MIXED LESIONS

⇒ Common	⇒ Rare
1-Metastases	1-Hepatic Adenoma
2-HCC	2-Focal Nodular Hyperplasia
3-Hepatoblastoma	3-Angiolipoma
4-Cholangiocarcinoma	
5-Lymphoma	

Metastases	- Commonest Hepatic M. Lesion -+Known Primary - Multiple -Arterial enh - Ca from Mucinos tumor
HCC	- Commonest 1ry Hepatic M. Lesion - more e cirrhosis DO NOT BIOPSY IF EARLY STAGE & < 5 CM -8M:1F -US & AFP for screening -Arterial enh/Venous washout -3Types: Focal/MultiFocal/Diffuse - Lo T1 / Hi T2
Hepatoblastoma	-- Commonest 1ry Hepatic M. < 5 y -Ca common -Diffuse of multifocal
Cholangiocarcinoma	-2 nd common after HCC “1:10” - - 3 Types: Small ducts/Large / Bifurcation = Klatskin - IHBCD + Hypdense/ Heter. Enh
Lymphoma	-1ry rare -Common as 2ry - Non specific finding - e Aids & organ transplant -Diffuse→ hypodense as fatty
Hepatic Adenoma	
Focal Nodular Hyperplasia “FNH”	- Very Vascular - use small Needle Highly Enhancing - Central scar - Not DD From Fibrolanular HCC - Confirm By : *US , AFP if small *Biopsy if large
Angiolipoma	Fat containing Lesion

LIVER MASS DIFFERENTIATION

- *Items to evaluate to differentiate Liver mass:*

- | | |
|--|------------------------|
| • Fat | Hypervascular lesions |
| • Hemorrhage | • Hypovascular lesions |
| • Cystic components | • Scar |
| • Retraction of liver capsule | • Capsule |
| • Peripheral enhancement & progressive fill in | • Calcification |

⇒ **FINDING IN DIFFERENT PHASES**

In the arterial phase:

- *Hypervascular tumors* ← enhance via the hepatic artery ,
- *Normal liver* parenchyma does not yet enhances , because contrast is not yet in the portal venous system.

In the portal venous phase:

Hypovascular tumors are detected when the normal liver parenchyma enhances maximally → hypodense lesions in a relatively hyperdense liver.

In the equilibrium phase “ 3:4 min..... best 10 minutes after contrast injection” ,

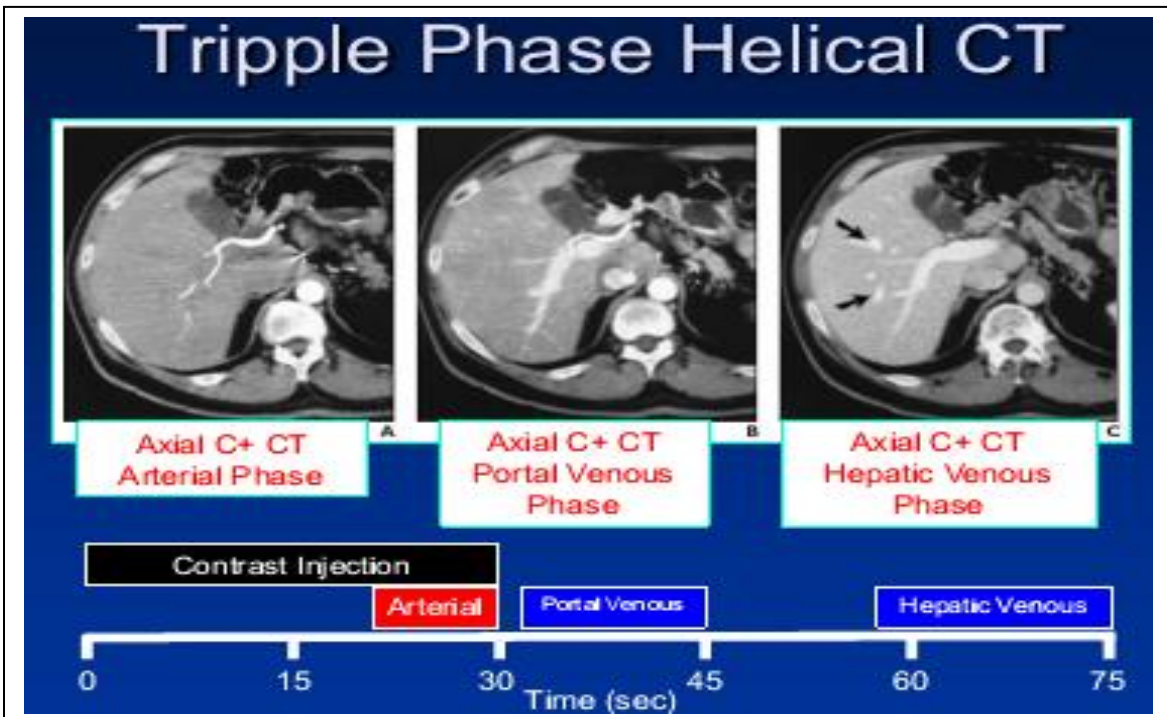
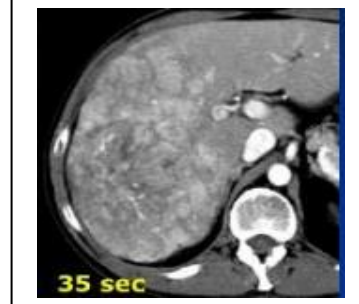
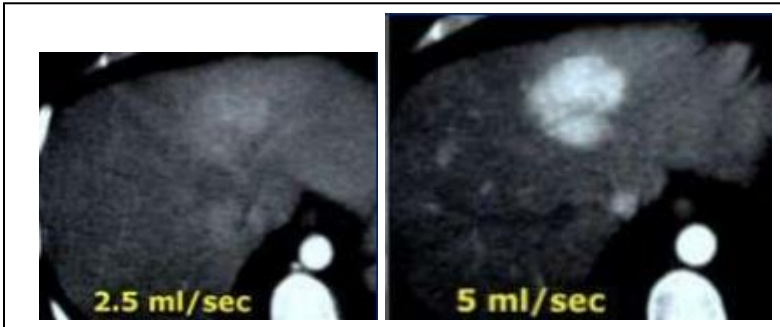
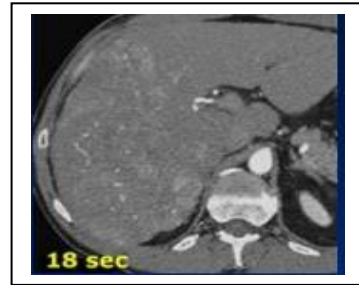
tumors become visible, that either :

- loose their contrast slower than the normal liver ,
- or wash out their contrast faster than normal liver parenchyma.
- Lesions will become either relatively hyperdense or hypodense to the normal liver

ARTERIAL PHASE IMAGING

For detection of hypervascular lesions :

- **Optimal Timing** : 35 mm “Late arterial phase”
- **Speed** : 5 ml/ sec



Value of Equilibrium Phase	
Tumoral wash out	Vascular tumors
Retention of contrast in blood pool	Hemangioma
Retention of contrast in fibrous tissue	Capsule around HCC Cholangiocarcinoma when fibrous Central fibrous scar (FNH)

SUMMARY OF LIVER IMAGING

	Scar	Capsul	Ca ⁺⁺	Fat	Blood	Cystic
Hemangioma	+		+		+	
FNH	+			+		
Adenoma		+		+	+	
HepatoCellCa	+	+		+	+	+
FibrolamellCa	+		+			
CholangioCa	+		+			
Metastases			+		+	+
Abces						+
Angiosarcoma					+	
Cystadenoma		+				+
Angiomyolipoma				+		

Hemangioma

Sharply demarcated lesion
Large Lesions usually heterogeneous
Peripheral Nodular enhancement
Enhancement - attenuation of appropriate vessels
 - look at Bloodpool
 - at all times (2 - 3 phases)

HCC

Tumoral Capsule
Internal Mosaic Pattern
Vascular Invasion Biliary Tract Invasion
Uncommon Ca⁺⁺
Predisposing conditions: Hepatitis B/C
 Hemochromatosis
 Cirrhosis

Cholangiocarcinoma

Gross pathologic structure	Annular, constricting Infiltrative and expanding Intraluminal, polypoid
Underlying histologic stroma	Fibrous versus glandular stroma
Locations	Intrahepatic, Proximal or Distal CBD,
Associations	PSC, Choledochal cysts; infections, chemical toxins

Focal Nodular Hyperplasia (FNH)

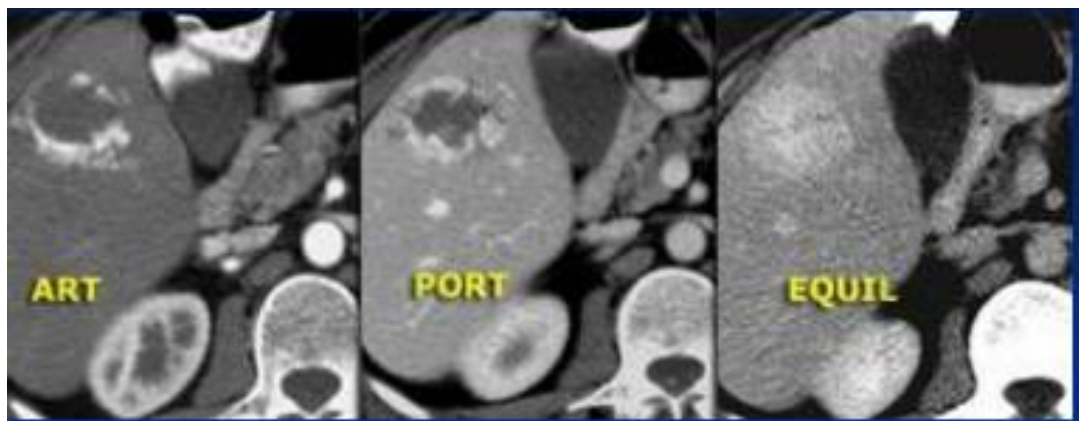
Etiology	Congenital vascular malformation or vascular injury
Morphology	Usually lobulated/well circumscribed No capsule Central fibrous scar with large vessels feeding the lesion
Enhancement	Hyperenhancing Arterial Phase in 100% Enhances Homogeneously in 95% Central scar enhances in equilibrium phase - Large Lesions 60 - 70% - Small lesions 30 - 35%

Fibrolamellar carcinoma (FLC)

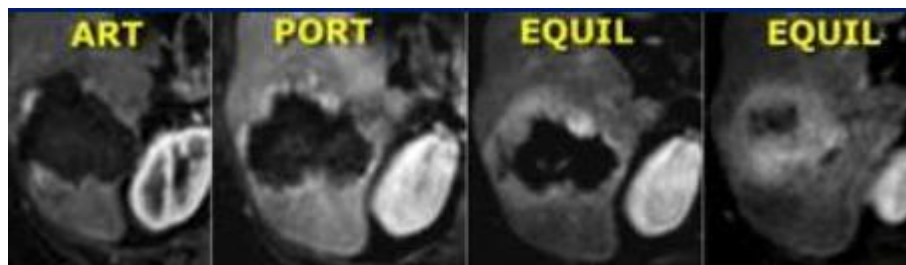
Noncirrhotic liver in younger patients
Large, often solitary
Fibrous bands through mass, coalescing in scar
Central scar ~ 40% (CT: hypo, MR: hypo,
 Delayed enhancement)
Ca⁺⁺ in ~50%
Arterial enhancement
Heterogeneous

CASES

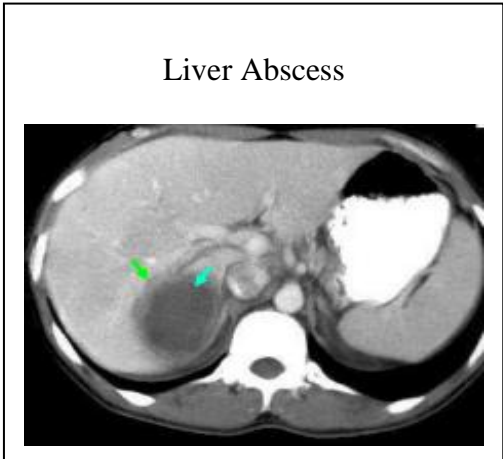
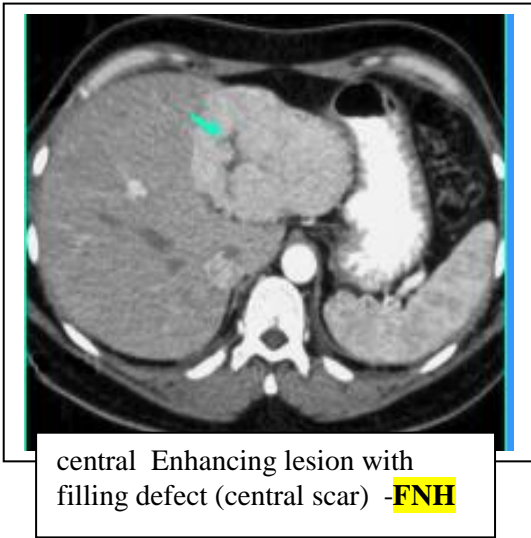
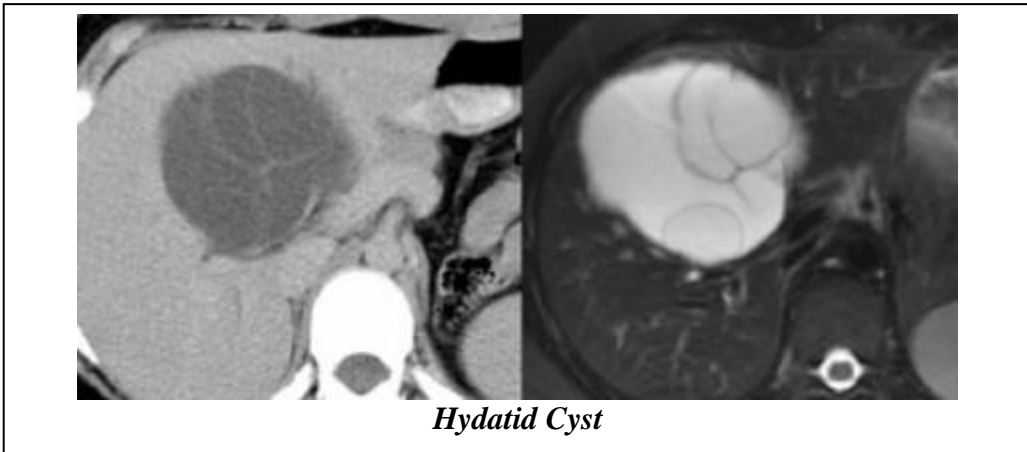
HCC
in a cirrhotic
liver.
Notice fast
wash out in
equilibrium
phase
compared to
surrounding
liver
parenchyma



Hemangioma



SUMMARY OF LIVER IMAGING





















<u>Lesions</u>	<u>Classical CT Findings</u>
Hepatic Cyst	Sharply demarcated wall, water density, non-enhancing
Hemangioma	Peripheral filling in of contrast over time "Light Bulb Sign" on T2 MRI
Focal Nodular Hyperplasia (FNH)	Early filling in arterial phase with central filling defect (scar)
Hepatocellular Adenoma	Variable, central changes due to hemorrhage often seen
Metastasis	Mostly multiple low attenuation lesions, rim enhancement without "filling in"
Abscess	Well demarcated hypodense areas with peripheral enhancement, may see gas
Hepatocellular Carcinoma (HCC)	Early arterial enhancement, fast washout, delayed fibrous capsule enhancement

SUMMARY OF LIVER IMAGING

Sources :

- Grainger & Allison's 5th ed.
- Lecture of Prof. Mamdouh Mahfouz
- <http://www.radiologyassistant.nl/>
- Lecture of Dr. Ahmad Elrefa'ey

	Arterial Phase	Portal-Venous Phase	Late Venous Phase
Hemangioma	 Peripheral Nodular Enhancement/ Complete Enhancement	 Partial or Complete Centripetal Enhancement	 Partial or Complete Enhancement
Focal Nodular Hyperplasia	 Centrifugal Hyper-enhancement/ Spoke-wheel Pattern	 Complete Hyper-enhancement	 Iso-/Hyper-enhancing, with or without Non-enhancing Central Scar
Hepatic Adenoma	 Rapid Enhancement/Centripetal Enhancement	 Iso-/Hyper-enhancing	 Iso-/Hyper-enhancing
Focal Fat	 Iso-enhancing	 Iso-enhancing	 Iso-enhancing
Regenerative Nodule	 Iso-/Hyper-enhancing	 Iso-enhancing	 Iso-enhancing
Malignant	 Hyper-enhancement, Rim hyper-enhancement, hyper-enhancement with Non-enhancing Areas	 Hypo-enhancement(Early Washout)	 Hypo-enhancement