TIN COLLOID
LIVER/SPLEEN
SCINTIGRAPHY

THIRD YEAR MEDICAL
STUDENTS
DIAGNOSTIC BLOCK
ANATOMY (liver)

Largest organ in body
Weighing 1500-1800g
In R hypochondrium and epigastrium
Four lobes: R and L, quadrate and caudate

Portal venous system:
Brings blood from stomach
Provide liver sinusoids that empties
In hepatic vein and IVC

Hepatic artery:
Brought arterial blood to liver

Porta hepatitis = division part between R and L lobes. Contains hepatic artery, portal vein and the common bile duct
Normal variability of liver contour.

Fig. 9-29  Normal variability of liver contour. Livers come in many shapes—triangular, round, even square. The inferior border may be convex or concave. The inferior lateral aspect of the right lobe may be indented due to rib compression, elongated (Riedel's), notched, or absent. The dome of the liver may be flattened due to depressed diaphragms from COPD or elevated due to phrenic nerve paralysis or congenital weakness (eventration).
ANATOMY (SPLEEN)

Beneath 9-11 rib left post
POST portion of left abdomen
10 x 6 cm and 4 cm thick
Vascular organ
RES and phagocytosis function
lymphocytes for immunity
Sequestration of blood elements
RB Cell production site

ENLARGED SPLEEN

POST ORGAN
LIVER LOBULE

Vascular sinusoids in the hepatic lobule, Are lined with reticulo-endothelial cells = Called the Kupffer cells = 10% of liver mass

OTHER RES CELLS:
Macrophages of the spleen and bone marrow

(Do not look at function of the Hepatic cells and bile ducts = part of DISIDA/HIDA scans)
RADIOPHARMACEUTICALS USED FOR LIVER/SPLEEN SCINTIGRAPHY

• Radio-colloids have been used since mid 1950’s
• We use $^{99m}$Tc labelled Tin Colloid
• Colloid particles is small particles that stay in suspension, and do not dissolve in water
• After IV administration, the colloid particles is removed from the blood by the RES cells. Blood clearance halftime is 2-3 min.
• Large particles are taken up by the liver, smaller particles distribute more to the bonemarrow. Very large particles may result in lung uptake
• Liver uptake is completed within 15 min. Phagocytosis fixes the colloids intra-cellular.
• Distribution of colloid particles in influenced by:
  a) blood flow
  b) particle size
  c) state of the kuppfer cells (normal/diseased)

Most diseases affect both hepatocytes and Kuppfer cells similarly
  a) focal disease: results in locally decreased due to destruction/displacement of normal liver tissue (filling defects due to tumors/abscess)
  b) diffuse liver disease produces a generalized reduction in extraction of colloid and increased distribution of activity in the spleen and bone marrow (this is called COLLOID SHIFT)

(seldom higher uptake of colloid in the liver = focal nodular hyperplasia = slightly higher uptake than in rest of liver)
Spleen on radio-colloid study

• **Normal study:** smooth contour, located left upper quadrant of abdomen, POST. The post liver and spleen images have about the same optical density (this change when the spleen is involved in a pathological condition)

• **Colloid shift:** the spleen: liver ratio of densities changes. The spleen takes up more radio-colloid in comparison to the liver
COLLOID SHIFT
(VERY IMPORTANT!!!)

• This may result from:

Main reasons

i) Congestive heart disease
ii) Policytemia
iii) Hodgkin lymphoma
iv) Extra medullary hemopoise
v) Hypersplenism
vi) Portal hypertention
vii) Liver cirrhosis
Colloid shift

• Other reasons:
1) Replacement or loss of liver function due to displacement of RES cells eg tumors
2) Hepatic damage from vessel obstruction
3) Portal/splenic vein obstruction
Some cases of hypersplenism
4) Following medications
5) Severe anemia
6) Hepatic damage due to alcoholism
7) Diabetic fatty infiltration of the liver
8) Artefactual due to GIT bleeding, liver tissue overlying spleen, hematoma or metal artefacts
LUNG RADIO-COLLOID UPTAKE

- REASONS:
  I) Improper labeling of the colloid = excessive aluminium from the generator, causing large particle clumping
  II) Severe liver disease eg acute/chronic cirrhosis
  III) Bacterial endotoxins in septicemia
  IV) High serum levels of aluminium eg antacids
  V) Heparin therapy
  VI) High estrogen levels as in cirrhosis and with estrogen therapy
INDICATIONS

• Differential diagnosis of hepatomegali and abdominal masses (also splenomegali)
• Evaluation for liver mets in patients known with malignancies
• Diffuse liver diseases (cirrhosis and hepatitis)
• Liver position and shape evaluation
• Patients with ascites of unknown origin
• Patients and children with jaundice
• Suspected liver abscesses
• Follow up of patients with liver malignancies
• Localization of hepatic lesions for needle biopsy
• Patients with suspected liver hemangioma
NORMAL liver IMAGE

NORMAL SIZE:
VERTICAL MIDCLAVICULAR DIMENSIONS – 17cm
WIDTH = 15cm

ENLARGED LIVER:
cefalo caudal dimension more than 17cm

LEAD MARKERS are used to evaluate the size of the liver. Length of marker is 15 cm

SPLEEN SIZE > 14cm in longest axis is considered enlarged.
Elongation of the right lobe down in
The pelvis is a normal variation
called a Riedl’s lobe

Intensity of spleen uptake on the POST view is
usually equal or less than that of the liver

Normal sized spleen not seen on ant images
Artifacts seen on a liver scintigram

Artifacts:  
  i) Shadows from the female breast, male rib cage and the heart  
  ii) Barium in the colon  
  iii) Contrast medium in the gallbladder  
  iv) Foreign objects like coins/jewelry  
  v) Prominent porta hepatis due to extra hepatic compression due to enlarged lymph nodes  
  vi) Displaced liver due to pleural effusion or emphysema
ABNORMAL IMAGES

1) DECREASED UPTAKE IN LIVER DUE TO
   • i) displacement or destruction of Kupffer cells
   • ii) alterations in the bloodflow
   • iii) alterations in the disease process
2) COLD or PHOTOPHENIC defects = FILLING DEFECTS
   Mostly benign or malignant lesions.
   Can only be detected if lesions are larger than 2-3 cm in diameter
3) RECTANGULAR PORT SHAPED DEFECTS due to radiation therapy
4) DIFFUSELY INCREASED UPTAKE is UNCOMMON
5) FOCAL AREA’S OF INCREASED UPTAKE.
   can be due to – increased blood flow to the area
   - increased density of the Kuppfer cells
6) Focal nodular hyperplasia
7) NON VISUALIZATION DUE to severe phagocyte damage and diminished blood flow
8) PROMINENT PORTA HEPATIS: look for metastasis in lymphnodes originating from stomach and pankreas ca
LIVER DISEASES

- 1) HEPATITIS → ACUTE/CHRONIC
- 2) HEPATIC CIRRHOSIS → EARLY – LATE STAGES
- 3) ABSCESSES eg ENTAMOEBA HISTOLYTICA
- 4) PRIMARY LIVER CA
- 5) HEPATIC ADENOMAS
- 6) BENIGN TUMORS eg CYSTS/HEMANGIOMAS
- 7) MEATSTATIC CARCINOMAS from BREAST, LUNG, COLON, GIT AND GENITO-URINARY TRACTS
- 8) TRAUMA
- 9) SVC OBSTRUCTION = BUDD CHIARY SYNDROME
- 10) EXTRA HEPATIC DISEASES eg
  - RIGHT PLEURAL EFFUSION
  - UPWARD DISPLACEMENT OF R LIVER LOBE DUE TO DIAPHRAGM DISEASE
  - ASCITIS
HEPATO-MEGALY

- CAUSES:
  1) Primary and secondary Tumors
  2) Alcoholic liver disease and cirrhosis
  3) Congestive heart failure
  4) Hepatic vein thrombosis
  5) Infiltrative liver disease due to fatty infiltration due to alcohol and diabetic disease
  6) Amyloidosis
  7) Wilson disease
  8) lymphomas
SPLENOMEGALY with HIGH UPTAKE ("WARM SPLEEN")

CAUSES:

1) CIRRHOSIS
2) ACUTE INFECTIONS
3) HEMOLYTIC ANEMIA
4) HYPERSPLENISM
Splenomegaly with Low Uptake ("Cold Spleen")

- **CAUSES:**
  1) Myelofibrosis
  2) Lymphoma and Leucemia
  3) Metastatic disease
  4) Amyloied/sarcoid infiltration
  5) Cysts
  6) Bleeding/infarction
SPLENIC DEFECTS/FILLING DEFECTS

• DEFECTS WITHIN THE SPLEEN SUBSTANCE OR ALONG IT’S PERIPHERY

1) SUB CAPSULAR BLEEDING
2) CYSTS
3) SMALL MALIGNANCIES
4) INFILTRATING DISORDERS
5) MULTIPLE SMALL EMBOLI/VASCULAR OCCLUSIONS
6) DIFFUSE VASCULAR DISEASE
7) TUMORS THAT EXTEND INTO THE SPLEEN
8) CONGENITAL LOBULATIONS
9) INTRA SPLENIC GAS
HEPATITIS (Acute/Chronic)

- **ACUTE:** (TOXIC/VIRAL)
  - ENLARGEMENT OF BOTH LIVER LOBES
  - LOW UPTAKE IN LIVER
  - INCREASED VISUALIZATION OF THE SPLEEN
HEPATITIS

- **CHRONIC:** (VIRAL, NUTRITION, ALCOHOL)

--FATTY INFILTRATION CAUSES GLOBAL HEPATOMEGALY

--ENLARGEMENT OF THE LEFT LOBE OVER THE RIGHT LOBE

--INCREASED SPLEEN VISUALIZATION – COLLOID SHIFT TOWARDS THE SPLEEN
ALCOHOLIC LIVER DISEASE
VERY IMPORTANT!!!!

• PRESENT IN DIFFERENT STAGES

1) FATTY INFILTRATION
2) ACUTE ALCOHOLIC HEPATITIS
3) CIRRHOSIS RELATED TO THE DEGREE OF PATHOLOGY, AND THE PRESENCE OR ABSENCE OF PORTAL HYPERTENSION
FATTY INFILTRATION

LIVER SCAN:

1) CAN BE NORMAL
2) SHOW MILD NON-HOMOGENEITY
3) LATER ON SHOW → HEPATOMEGALY WITH COLLOIED SHIFT
ALCOHOLIC HEPATITIS

- LIVERSCAN
  1) RESULTS IN HEPATIC NECROSIS WITH INFLAMMATION AROUND THE CENTRAL VEINS
  2) SHOWS IRREGULAR DISTRIBUTION OF COLLOID
CIRRHOSIS

• **CAUSES:**
  - I) CHRON HEPATITIS B, NON A, NON B
  - 2) NUTRITIONAL/ALCOHOLIC
  - 3) CHRONIC BILIARY OBSTRUCTION
  - 4) TOXIC HEPATITIS
  - 5) HEMOCHROMATOSIS
  - 6) WILSON’S DISEASE

**LIVER SCAN:**
1) THE RIGHT LOBE SHRINKS EN LEFT LOBE COMPENSATES WITH HYPERTROPHY
2) COLLOID REDISTRIBUTION TO THE SPLEEN TAKES PLACE AS WELL AS TO THE BONE MARROW (COLLOID SHIT)
3) THERE IS INTERFERENCE WITH THE PORTAL BLOOD FLOW, AND IN THE END STAGE THERE WILL BE PORTAL HYPERTETION WITH SECONDARY ENLARGEMENT OF THE SPLEEN
4) THE NON HOMOGENEOUS APPEARANCE RESEMBLES FOCAL FILLING DEFECTS, CAUSING A DIAGNOSTIC DILEMMA DUE TO HIGH INCIDENCE OF HEPATOMA IN CIRRHISIS PATIENTS
CIRRHOSIS IMAGES

??NORMAL
SLIGHTLY NON-HOMOGENEOUS

HEPATO-MEGALI, COLLOID SHIFT AND BONE MARROW UPTAKE
CIRRHOSIS

LOW IRREGULAR UPTAKE IN LIVER

HEPATITIS IMAGE
CIRRHOSIS

ASCITIS

IMPAIRMENT OF LIVER FUNCTION

SPLENOMEGAL COLLOID SHIFTY

BONE MARROW UPTAKE
CIRRHOSIS

NO LIVER SUBSTANS LEFT
PRIMARY LIVER CARCINOMA

HUGH FILLING DEFECTS
LIVER METASTASES

1) IS ONE OF THE MOST IMPORTANT INDICATIONS OF THIS INVESTIGATION

2) LIVER METS RESULTS FROM PRIMARY MALIGNANCIES OF THE COLON, LUNG, PANKREAS, BREAST AND GENITO-URINARY TRACT

3) OP PLANAR IMAGES THE LESIONS MUST BE AT LEAST > 2CM IN DIAMETER TO BE SEEN (3-4cm)

4) SPECT IMAGES INCREASES THE ACCURACY AND LESIONS OF 1cm CAN BE DETECTED WITH SPECT
FOCAL LIVER DEFECTS
FILLING DEFECTS

• CAUSES:
  1) CYSTS
  2) TUMORS = BENIGN or MALIGNANT
  3) ABSCESSES
  4) HEMATOMAS \(\rightarrow\) FOLLOW UP WITH RBS LABELLED STUDY
  1) LOCALIZED HEPATITIS
  2) RADIATION THERAPY
  3) INFARCTIONS
  4) CIRRHOSIS
  5) FATTY INFILTRATION
LIVER CYSTS OR ABCESESSES

CAN BE SINGLE OR MULTIPLE COLD SPACE OCCUPYING LESIONS
BUDD CHIARI SYNDROME
HEPATIC VEIN THROMBOSIS

• CAUDATE LOBE HAS MORE UPTAKE THAN THE REMAINDER OF THE LIVER

Normally the caudate lobe receives blood from IVC, while Rest of liver receives blood from hepatic vein

With hepatic vein thrombosis, there is impaired venous drainage of the majority of the liver, with poor hepatic function

While the caudate lobe retains good function as result of it’s direct venous drainage into the IVC
Sup Vena Cava Obstruction

• The injected RA is routed through collateral thoracic veins, to the subdiaphragmatic collaterals (this include the umbilical vein)

• If the umbilical vein has remained opened, it emptied into the liver, above the porta, where it ends abruptly, in the region of the caudate lobe

• With SVC obstruction the liver shows a “hot” spot in this caudate area.
Non visualization of spleen

• Causes:
  1) Congenital a-splenism
  2) Splenectomy
  3) Acquired a-splenia due to vascular occlusion/tumor replacement
  4) Functional asplenia
  5) Reversible hyposplenia due to anoxia
FOCAL INCREASED UPTAKE IN THE LIVER

• CAUSES:

1) SVC SYNDROME
2) IVC SYNDROME
3) FOCAL NODULAR HYPERPLASIA
4) BUDD CHIARI SYNDROME
5) CIRRHOsis
FOCAL NODULAR HYPERPLASIA
LIVER HEMANGIOMA

LABELLED REDCELL STUDY