2nd Edition

Part-2

An Insider's Guide to Cases in CLINICAL MEDICINE

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3 SECTION

Gastrointestinal System

Section Outline

- Case Sheet Format
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  - Cirrhosis
  - Hepatomegaly
  - Splenomegaly
  - Ascites

Case Sheet Format

HISTORY TAKING

Name:

Age:

Sex:

Residence:

Occupation:

Chief complaints

1. _____ × days
2. _____ × days
3. _____ × days

History of presenting illness

Abdominal distension

- Duration
- Onset
- Progression
- Aggravating factors
- Relieving factors
- Associated symptoms
- Is it preceded by pedal edema or followed by it?

Pedal edema:

- Duration
- Onset
- Progression
- Aggravating factors
- Relieving factors
- Is it preceded by facial puffiness or followed by it?

Abdominal pain:

- Onset
- Site
- Type of pain
- Radiation
- Aggravating factors
- Relieving factors
- Associated symptoms

Nausea and vomiting:

- Episodes
- Contents
- Blood tinged or not
- How many hours after consumption of food associated with pain abdomen?
- Conditions with nausea and vomiting but not associated with pain abdomen:
  - Metabolic
  - Neurologic
  - Drug, induced
  - Psychogenic

Other symptoms:

- Heart burn, flatulence, and waterbrash
- Hematemesis and melena
- Dysphagia
- Constipation and diarrhea

Altered bowel habit:

- Stool color
- Stool odor
- Stool frequency
- Blood tinged or melena

Jaundice—itching and high, colored urine

Other symptoms:

- Fever
- Weight loss
- Pain in oral cavity
- Halitosis
- Hiccups
- Other relevant history

Past history

- Asthma
- Chronic obstructive airway disease
- Tuberculosis
- History of contact with tuberculosis
- Diabetes mellitus (DM)
- Hypertension (HTN)
- Ischemic heart disease (IHD)
- Seizure disorder

Family history

Draw a three generations pedigree chart

Personal history

- Bowel habits
- Bladder habits
- Appetite
- Loss of weight
- Occupational exposure
- Sleep
- Dietary habits and taboo
- Food allergies
- Smoking index or pack years
- Alcohol history

Menstrual and obstetric history

- GPLA
- Age of menarche
- Menopause at
- Flow—ameno/oligo/menorrhagia

Summarize

Differential diagnosis:

1.
2.
3.

GENERAL EXAMINATION

Patient

- Conscious
- Coherent
- Cooperative
- Obeying commands

Body mass index (BMI)

- Weight (kg)/height^2 (meters)
- Grading according to WHO for Southeast Asian countries

Vitals

- **Pulse**
  - Rate:
  - Rhythm:
  - Volume:
  - Character:
  - Vessel wall thickening:
  - Radioradial delay and radiofemoral delay:
  - Peripheral pulses:
- **Blood pressure**
- **Respiratory rate**
  - Regular/irregular
  - Abdominothoracic/thoracoabdominal
  - Usage of accessory muscles:
- **Jugular venous pressure**
  - cm of blood above sternal angle (+ 5 cm water from right atrium)
- **Jugular venous pulse**
  - Waveform (describe waves)

On physical examination

- Pallor:
- Icterus:
- Cyanosis:
- Clubbing:
- Lymphadenopathy:
- Edema:

Other Head-to-Toe Signs of Liver Cell Failure

1. Alopecia
2. Fetor hepaticus
3. Jaundice
4. Parotid swelling
5. Gynecomastia
6. Testicular atrophy
7. Loss of secondary sexual characters
8. Spider nevi
9. Palmar erythema
10. Dupuytren's contracture
11. Asterixis
12. Xanthelasma
13. Signs of chronic cholestasis (scratch marks due to pruritus).

SYSTEMIC EXAMINATION

The order of examination of abdomen is preferably done—Inspection → Auscultation → Palpation → Percussion (as the auscultatory findings might change post palpation and percussion).

Inspection

- Shape/distension (localized/generalized) and flanks (free/full)
- Skin over the abdomen
- Symmetry
- Umbilicus
- Movement of corresponding quadrants with respiration
- Dilated veins
- Visible mass
- Visible pulsations
- Visible peristalsis
- Scars or sinuses
- Divarication of recti

Palpation

- Superficial palpation
  - Warmth
  - Tenderness
  - Guarding
  - Rigidity
- Deep palpation
  - Liver
    - Size
    - Shape
    - Border or edge
    - Surface
    - Tenderness
    - Consistency
    - Movement with respiration
    - Pulsation
  - Spleen
    - Location
    - Size
    - Shape
    - Consistency
    - Surface
    - Edge
    - Tenderness
    - Movement with respiration
  - Gallbladder
  - Other palpable mass

- Bimanual palpation
  - Kidneys
    - Location
    - Size
    - Shape
    - Consistency
    - Surface
    - Edge
    - Tenderness
    - Movement with respiration
- Dipping method (in case of large ascites)
- Hernia orifices
- Direction of flow in veins (if dilated veins present)
- Abdominal girth measurement
- Spinoumbilical distance
- Xiphisternum to umbilicus distance (x) in cm
- Umbilicus to pubic symphysis distance in cm (y)
  - Ratio of x/y

Percussion

- Liver
- Spleen
- Traube's space
- Fluid
  - Shifting dullness
  - Fluid thrill
  - Puddle sign

Auscultation

- Bowel sounds
- Succussion splash
- Bruit
- Venous hum
- Friction rub

Examination of

- Scrotum
- Spine
- Supraclavicular fossa

Per Rectal Examination

Per Vaginal Examination

Diagnosis Format

CIRRHOSIS/LIVER DISEASE

- Acute hepatitis <4 weeks
  or
  Subacute hepatitis
  or
  Chronic (cirrhosis/hepatitis >6 months)
  or
  Acute on chronic liver disease (ACLD)
- Compensated or decompensated
- Possible etiology—alcohol/post-viral/toxin/nonalcoholic steatohepatitis (NASH)
- With complications—portal hypertension with or without gastrointestinal (GI) bleed/hepatic encephalopathy (preferable to mention stage)/spontaneous bacterial peritonitis/hepatocellular carcinoma/hepatorenal syndrome/others.

Example: Decompensated chronic liver disease—cirrhosis secondary to alcohol, with portal hypertension, with upper gastrointestinal (UGI) bleed, patient in stage 2 hepatic encephalopathy with no evidence of spontaneous bacterial peritonitis or other complications.

Sample Case Sheet and Discussion

Case 1: Cirrhosis

Brief History

A 45-year-old male presented with following chief complaints:

- *Distension of abdomen for 6 months*
- *Abdominal discomfort and a sense of heaviness for 6 months*
- *Scanty micturition for 1½ months*
- *Weakness, malaise, loss of appetite for 1½ months.*

History of Present Illness

- *The patient states that he was reasonably well about 6 months back. Since then, he has been suffering from gradual swelling of his abdomen which has increased progressively over the last few days.*
- *It is not associated with abdominal pain, but there is discomfort and sense of heaviness. He also complains of scanty micturition, generalized weakness, malaise and loss of appetite for 1½ months.*
- *There is no history of hematemesis or loss of consciousness. There is history of passing dark colored stools whenever he is constipated.*
- *The patient does not give any history of fever, shortness of breath or cough, puffiness of the face, joint pain, skin rash, pigmentation, etc. His bowel habit and sleep pattern are normal.*

History of Past Illness

He suffered from jaundice 3 years back that lasted for about 5 months and then subsided. At that time, he took some herbal medications. There is no history of any injection, infusion, blood transfusion, IV drug abuse or sharing of needles.

Personal History

He is a government service holder. He smokes about 20–30 sticks of cigarettes per day for the last 25 years. There is no history of taking alcohol. Wife says he is irritable and excessively sleepy in the day for the last 2 weeks.

Family History

His parents are alive and in good health. His wife and two children are good health. There is no history of similar illness in his family.

Drug and Treatment History

Patient is on tablet lasix, aldactone, pantoprazole, ursocol and B complex.

General Examination

- *The patient is ill-looking, emaciated, conscious and oriented.*
- *Face—hollowed temporal fossa, pinched up nose, malar prominence, muddy complexion of the skin, shallow and dry face with icteric conjunctiva*
- *Pallor present, angular cheilitis present*
- *Generalized pigmentation is present. There are few ecchymoses in upper limbs.*
- *Pitting pedal edema—present.*
- *Multiple spider angiomas are present over the upper part of chest and back.*
- *There is leukonychia (fingers and toes) and palmar erythema.*
- *Gynecomastia present.*
- *No koilonychia or cyanosis.*
- *No lymphadenopathy or thyromegaly.*
- *Dupuytren's contracture and flapping tremor are absent.*

Vital signs: Pulse—88/min. BP—110/75 mm Hg. Temperature—99°F. Respiratory rate—18/min.

Systemic Examination Abdomen

Inspection

- *The abdomen is distended, flanks are full*
- *Umbilicus—everted*
- *Visible superficial veins with normal flow (away from umbilicus)*
- *There is no visible peristalsis, no scar mark.*

Palpation

- *No tenderness*
- *Liver—not palpable.*
- *Spleen is palpable, 4 cm from the left costal margin in anterior axillary line towards the right iliac fossa. The surface is smooth, firm in consistency, nontender.*
- *Testes—both testes are atrophied.*
- *Fluid thrill present.*

Percussion: Shifting dullness present. Auscultation: No abnormality detected.

Examination of Other Systems

Nervous system

Constructional apraxia present, no flaps, reflexes normal respiratory system (RS), cardiovascular system (CVS)—NAD.

Provisional diagnosis

Chronic decompensated parenchymal liver disease—cirrhosis with portal hypertension probably of etiology with ascites with features of hepatic encephalopathy and coagulopathy.

- *It is not associated with abdominal pain, but there is discomfort and sense of heaviness. He also complains of scanty micturition, generalized weakness, malaise and loss of appetite for 1½ months.*
- *There is no history of hematemesis or loss of consciousness. There is history of passing dark colored stools whenever he is constipated.*
- *The patient does not give any history of fever, shortness of breath or cough, puffiness of the face, joint pain, skin rash, pigmentation, etc. His bowel habit and sleep pattern are normal.*

DISCUSSION

Q. What are the gastrointestinal causes of clubbing?

Biliary cirrhosis, ulcerative colitis, Crohn's disease, gastrointestinal tract (GIT) malignancy.

Q. What is the evidence for portal hypertension clinically?

Splenomegaly is the clinical evidence for portal hypertension in a case of cirrhosis liver. Other evidences are paraxiphoid umbilical hum and caput medusae which indicate intrahepatic portal hypertension.

Q. What are the signs in favor of chronic liver disease?

- Spider angiomata, sparse axillary hair, ascites, Dupuytren's contracture, gynecomastia.
- Signs in favor of alcohol etiology in cirrhosis patients
- Dupuytren's contracture.
- Parotid swelling.
- Alcohol peripheral neuropathy.

Q. Consuming how much alcohol/day is associated with increased risk of developing alcoholic liver disease (ALD)?

Consumption of 60–80 g alcohol/day in males for 10 years and 20–40 g alcohol/day for 10 years in females increases the risk of ALD.

Q. What is spider angiomata and how many spider are significant?

Spider nevi (Table 3.1)

Table 3.1: Spider nevi (spider telangiectasia; vascular spiders; spiderangiomas; arterial spiders, and nevus araneus).

Description	✦ Consists of a central arteriole from which numerous small vessels radiate peripherally-resembling spider's legs. Whole spider disappears when central arteriole is compressed with a pinhead. When compression is released filling occurs from center to periphery ✦ Spider angioma has three features: A body, legs, and surrounding erythema. ✦ Spider nevi may also be associated with numerous small vessels scattered randomly through the skin on the upper arms **(paper money skin)**
Pathophysiology	Due to arteriolar changes induced by hyperestrogenism
Location	✦ Usually found only in the necklace area, i.e., above the nipples, territory drained by the superior vena cava, such as: head and neck, upper limbs, front and back of upper chest ✦ Rare below the diaphragm (possibly due to higher vasomotor gradient)
Size	Vary from pinhead to 0.5 mm in diameter
Clinical demonstration	Applying pressure over the body of spiders with a glass slide (diascopy) **(Fig. 3.1)**, or pin head **(Fig. 3.2)** leading to pallor with refilling following the release of pressure
Significance	They are a strong indicator of liver disease but can be found in other conditions
Causes	***Liver disorders*** ✦ Viral hepatitis ✦ Alcoholic hepatitis ✦ Hepatocellular carcinoma ✦ Treatment with sorafenib ***Others*** ✦ Third trimester of pregnancy ✦ Rheumatoid arthritis ✦ Thyrotoxicosis ✦ Also normally seen in 2% of healthy population ✦ Oral contraceptives also can cause
	Venous star, Campbell de Morgan spots, petechiae, insect/mosquito bites and hereditary hemorrhagic telangiectasias (Osler-Weber-Rendu syndrome)
Differential diagnosis	✦ Differentiating features of venous star – Blood flows from the periphery of the star centrally and thence into the collecting vein; the direction of flow is the exact opposite of that in the arterial spider – The pattern, shape and size are much more variable than in the arterial spider. – Color frequently is blue
	✦ Are common on the dorsum of the feet, around the ankle and the lower legs both front and back, and above the knee on the medial aspect of the thigh ✦ Histologically they are dilated veins
Clinical significance in liver disease	✦ Spider nevi correspond with a higher risk of mortality among patients with the alcoholic liver disease. They also suggest a high likelihood of esophageal varices and are indicative of the extent of hepatic fibrosis. ✦ No of spider naevi significant is if more than 5 ✦ Sudden disappearance of spider naevi may indicate an ongoing gastrointestinal bleed ✦ Size more 15 mm: 80% chances of variceal bleed ✦ *Florid spider telangiectasia, gynecomastia, and parotid enlargement are most common in **alcoholic hepatitis*** ✦ *Florid spiders and new onset clubbing in a patient with cirrhosis indicates **hepatopulmonary syndrome***

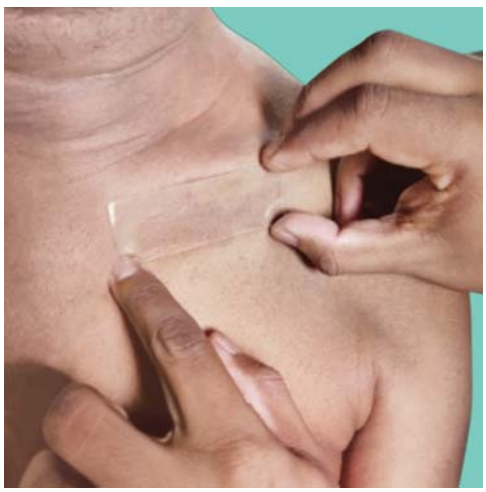


Fig. 3.1: Demonstration of spider nevi (glass slide method).

Fig. 3.2: Demonstration of spider nevi (pin head method).

Q. What is palmar erythema?

- Palmar erythema is reddening of the palms involving thenar and hypothenar eminences sparing the central portions of the palm.
- Involves thenar and hypothenar eminence, distal pads of fingers, circumungual areas on dorsum of fingers
- Central part of palm is clear
- Represents collection of A-V anastomosis
- Steroid estrogen precursors blamed
- Can occur in RA, pregnancy and OCP use

Q. What nail changes are seen in chronic liver disease?

Leukonychia (Fig. 3.3)

- White (Terry's) chalky and brittle nails, proximal 2/3—white, distal 1/3—red
- **Muehrcke's nails:** Characterized by transverse white lines that disappear on applying pressure and these lines do not move with growth of nail.

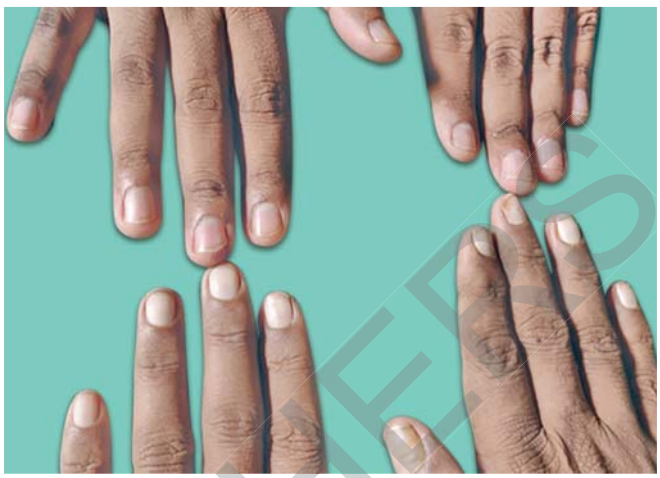


Fig. 3.3: Leukonychia—compare with nails of normal person (preferably hands to be placed side by side).

- Clubbing is present in primary biliary cirrhosis or hepatoma.

Q. Clubbing is common in which type of cirrhosis?

Clubbing is more common in biliary causes of cirrhosis.

Q. What is Dupuytren's contracture and name some conditions associated with it?

Dupuytren's contracture (Fig. 3.4)

- Fibrosis of palmar aponeurosis is probably caused by local microvascular ischemia.
- Platelet and fibroblast-derived growth factors promote fibrosis
- Flexion contracture of the finger especially ring and little fingers
- Sign of alcoholism
- **Other causes:** Diabetes mellitus, rheumatoid arthritis, and manual labor (workers exposed to repetitive handling tasks or vibration)

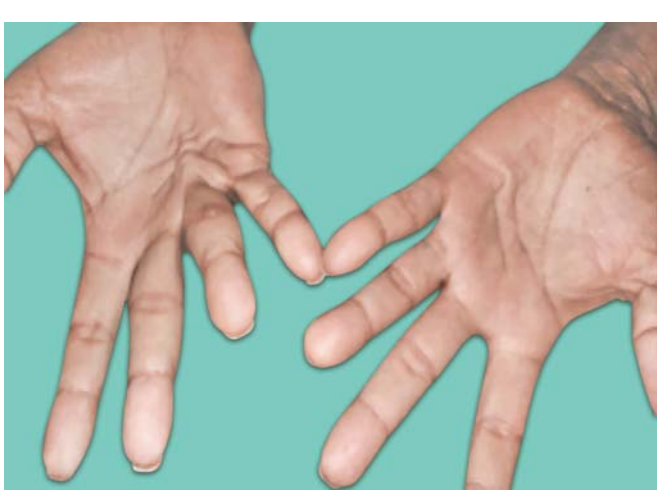


Fig. 3.4: Dupuytren's contracture.

Q. What is gynecomastia and briefly tell causes, pathophysiology behind it?

Gynecomastia: It occurs due to two mechanisms **(Fig. 3.5)**:

Mechanism 1: Increased conversion of weak androgenic steroids to estrogens in peripheral tissues especially adipose tissue causing local fat deposit. Alcohol induces androgenic steroids.

Mechanism 2: Steroidal estrogen precursors escape the enterohepatic circulation and then undergo peripheral conversion.

Causes of gynecomastia

- Cirrhosis of liver
- Drugs:
  - Spironolactone
  - Cimetidine
  - Digoxin
  - Ketoconazole
  - Estrogens
  - Isoniazid/antiandrogens
- Physiological (puberty/aging)
- Klinefelter's syndrome
- Hypogonadism
- Tumor:
  - Testes
  - Lung
  - **Examination (Fig. 3.6):** Appear as palpable nodule (2 cm or greater, subareolar).
  - **Microscopy**: Proliferation of glandular tissue of breast.

Q. What is fetor hepaticus?

- Established reason is mercaptans
- Mercaptans are thiols (sulfur-containing compounds) formed due to gut metabolism
- Newer evidence points to **dimethyl sulfide** as the reason for fetor hepaticus.

Fig. 3.6: Palpation breast bud in gynecomastia.

Q. What is the mechanism of testicular atrophy?

- High alcohol consumption causes damage to the Leydig cells and there by causes decreased testosterone levels **(Fig. 3.7)**.
- Direct effect of alcohol and not related to estrogen effect.
- Characteristic in alcoholic cirrhosis.
- Also occurs in hemochromatosis.
- Loss of testicular sensation
- Orchidometer
- The dimensions of the average adult testicle is 4.5 × 3.5 × 2.5 cm and the volume is 15–25 mL.

Q. What is pseudogynecomastia?

Pseudogynecomastia is accumulation of subareolar fat tissue without palpable nodule. Seen in obesity and Cushing's syndrome.

Q. When to suspect hepatoma in cirrhosis patients?

In patients with previously compensated cirrhosis who develop decompensation such as ascites, encephalopathy, jaundice, or variceal bleeding.

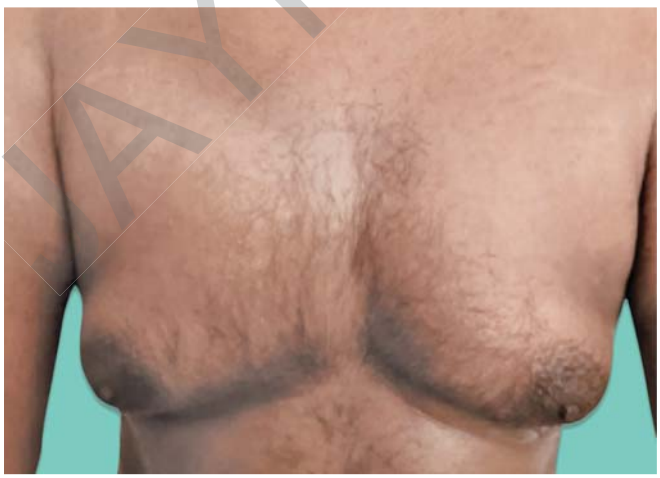


Fig. 3.5: Gynecomastia.

Fig. 3.7: White nails.

- Palpable mass in the upper abdomen.
- Mild to moderate upper abdominal pain
- Fever may develop in association with central tumor necrosis
- Intraperitoneal bleeding due to tumor rupture.
- Bone pain or dyspnea due to metastases
- A bruit heard over the liver.

Q. What is the typical presentation of a primary biliary cirrhosis?

The typical patient is a middle-aged woman with a complaint of fatigue or pruritus. Other symptoms include right upper quadrant abdominal pain, anorexia, and jaundice. Fatigue, although relatively nonspecific, is considered to be the most disabling symptom by many patients, and it worsens in some patients as the disease progresses. Pruritus may occur at any point, early or late, in the course of the disease, or intermittently throughout the course. Pruritus generally is intermittent during the day and is most troublesome in the evening and at night.

Symptoms and signs of primary biliary cirrhosis at presentation (Table 3.2)

Table 3.2: Signs and symptoms of PBC.

Symptoms or signs	*Frequency (%)*
Fatigue	21–85
Pruritus	19–55
Hyperpigmentation	25
Hepatomegaly	25
Splenomegaly	15
Xanthelasma	10
Jaundice	3–10
Right upper quadrant pain	8
None	25–61

Q. What is the morphological classification of cirrhosis?

Figure 3.8 shows the morphological classification of cirrhosis.

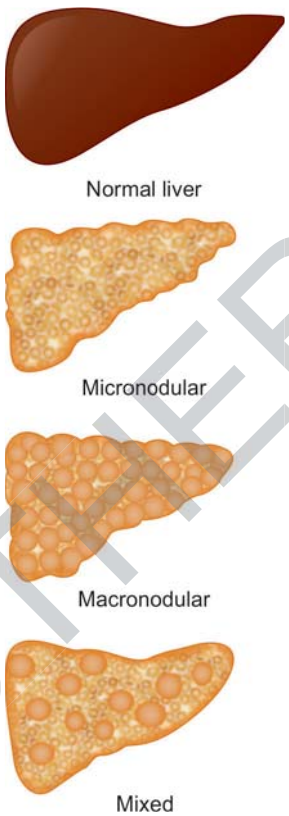


Fig. 3.8: Morphological classification of cirrhosis.

AUTOIMMUNE HEPATITIS

Q. What are the clinical features of autoimmune hepatitis?

Refer **Table 3.3**.

Table 3.3: Clinical features of autoimmune hepatitis.

Clinical features	*Occurrence (%)*
Symptoms	
Fatigue	86
Jaundice	77
Upper abdominal discomfort	48
Pruritus (mild)	36
None (at presentation)	25–34
Clinical features	*Occurrence (%)*
Anorexia	30
Myalgias	30
Diarrhea	28
Cushingoid features	19
Fever (≥40°C)	18
Physical findings	
Hepatomegaly	78
Jaundice	69
Spider angiomata	58
Concurrent immune disease	≤38
Splenomegaly	≥32
None	<25
Ascites	20
Encephalopathy	14

Q. What is constructional apraxia?

Refer **Figures 3.9 and 3.10**.

Inability to reproduce simple designs with blocks or matches. Number connection tests may be used serially to assess progress.

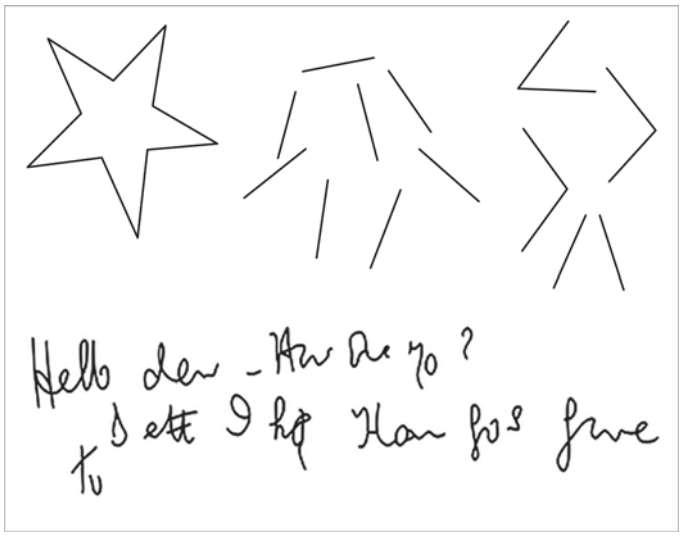


Fig. 3.9: Focal disorders in chronic portal-systemic encephalopathy elicited in patients with full consciousness and minimal intellectual defect, in the absence of gross tremor or visual disorder. Above: constructional apraxia. Below: writing difficulty. 'Hello dear. How are you? Better I hope. That goes for me too'.

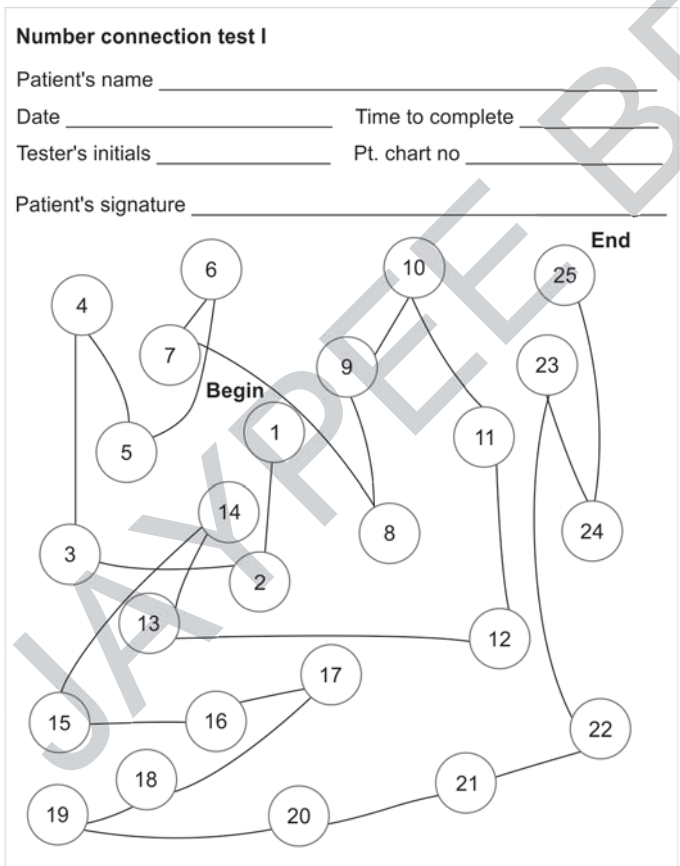


Fig. 3.10: The reitan number connection test.

Q. Define apraxia and classify?

Inability to carry out on request a high level, familiar and purposeful motor act in absence of any weakness, sensory loss, or other deficit involving the affected part. There are many varieties of apraxia. The ones seen most often are ideomotor, buccofacial, constructional, and dressing apraxia.

In ideomotor (motor) apraxia, the patient is unable to perform a complex command (e.g., salute, wave goodbye, snap the fingers, make a fist, show how to hitch-hike) with the involved extremity. In ideomotor apraxia, there may be a disconnection between the language or visual centers that understand the command and the motor areas tasked with carrying it out.

In ideational (conceptual) apraxia, the patient is able to carry out individual components of a complex motor act, but she cannot perform the entire sequence properly. Ideational apraxia may occur with damage to the left posterior temporoparietal junction or in patients with generalized cognitive impairment.

Constructional or dressing apraxia may occur with parietal lobe lesions that interfere with the patient's ability to comprehend spatial relationships.

Q. How do you stage hepatic encephalopathy?

Refer **Table 3.4**.

Table 3.4: Clinical grades of hepatic encephalopathy.

Clinical grade	*Clinical sign*
Grade I	Mild confusion, euphoria, anxiety or depression
	Shortened attention span
	Slowing of ability to perform mental tasks (addition/subtraction)
	Reversal of sleep rhythm
Grade II	Drowsiness, lethargy, gross deficits in ability to perform mental tasks
	Obvious personality changes
	Inappropriate behavior
	Intermittent disorientation of time (and place)
	Lack of sphincter control
Grade III	Somnolent but rousable
	Persistent disorientation of time and place
	Pronounced confusion
	Unable to perform mental tasks
Grade IV	Coma with (IVa) or without (IVb) response to painful stimuli.

Q. Briefly explain how will you demonstrate hepatojugular reflux?

- Position the patient with his trunk initially around 45° from the horizontal, and observe the jugular pulsations during quiet breathing. Alter the position as needed to identify the

highest angle of elevation at which these pulsations can be seen. This is the baseline venous pressure. (You will be searching for a 3-cm rise in venous pressure. Accordingly, if the jugular vein is too short to demonstrate such a rise, you may have to crank up the head of the bed so that the vein rises 3 cm on the vertical).

- Apply your hand to the right upper quadrant or the middle of the abdomen. It is not necessary to press over the liver to produce the phenomenon. (In fact, if there is tenderness, you should not press in that area because you do not wish the patient to guard, perform a Valsalva maneuver, or interrupt his normal breathing pattern in any way.)
- Press down, maintaining a pressure of 35 mm Hg. (You can practice over a semi-inflated blood pressure cuff or place the blood pressure cuff over the abdomen to be sure that sufficient pressure is applied.)
- Instruct the patient to continue to breathe normally through his mouth. Do not attempt to measure the venous pressure for at least 10 seconds to allow both respiratory artifacts and tensing of the abdominal muscles to subside (each alters jugular venous pressure). The best time to take another venous pressure measurement is at 1 minute of pressure. This should be used as the gold standard in ambiguous cases for reasons given in the section "For the Attending."
- A venous pressure rise of more than 3 cm is abnormal and hepatojugular reflux (or "abdominojugular reflux" as some now prefer) is said to be present, assuming of course that pain or performance of a Valsalva maneuver has not produced a false positive.
- Watch for an abrupt drop in jugular venous pressure as the abdominal pressure is relieved. The sudden fall is generally easier to perceive than the gradual rise.

Q. What are the causes of drug-induced liver injury?

- **Acute hepatocellular injury:** Numerous drugs such as isoniazid, rifampicin, methyl dopa, telithromycin, ketoconazole, diclofenac
- **Mononucleosis:** Sulfonamides, phenytoin, dapsone
- **Fulminant hepatitis:** Paracetamol (acetaminophen)
- **Bland cholestasis:** Anabolic/androgenic steroids, ciclosporin
- **Cholestatic hepatitis:** Chlorpromazine, erythromycin, amoxicillin—clavulanate, clarithromycin
- **Chronic hepatitis:** Methotrexate, lisinopril, trazodone, uracil
- **Autoimmune hepatitis:** Nitrofurantoin, minocycline, methyldopa, oxyphenisatin
- **Macrovesicular hepatitis:** Corticosteroids, methotrexate, asparaginase, alcohol, halothane
- **Microvesicular hepatitis:** Valproic acid, tetracyclines, cocaine, amiodarone
- **Steatohepatitis:** Amiodarone, griseofulvin, perhexilline maleate
- **Cirrhosis:** Methotrexate, amiodarone
- **Granulomatous hepatitis:** Allopurinol, rosiglitazone, sulfonamide, phenylbutazone, quinidine
- **Primary biliary cirrhosis:** Chlorpromazine, erythromycin, amoxicillin—clavulanate, haloperidol
- **Peliosis hepatic:** Anabolic steroids, oral contraceptives
- **Portal vein thrombosis:** Oral contraceptives
- **Sinusoidal obstructive syndrome:** Pyrrolizidine alkaloids, adriamycin, floxuridine, oncotherapy
- **Nodular transformation:** Anabolic and contraceptive steroids
- **Adenoma:** Anabolic and contraceptive steroids
- **Hepatocellular carcinoma:** Thorotrast, anabolic and contraceptive steroids
- **Cholangiocarcinoma:** Thorotrast
- **Angiosarcoma:** Vinyl chloride, inorganic arsenicals.

Q. What histological findings are seen in alcoholic liver disease?

The histologic features of alcohol-induced hepatic injury include steatosis (fatty change), lobular hepatitis, periportal fibrosis, Mallory bodies, nuclear vacuolation, bile ductal proliferation, and fibrosis or cirrhosis. Development of large-droplet (macrovesicular) steatosis (fatty liver) is the earliest and most common manifestation of ALD.

Q. How to investigate a case of cirrhosis?

Occupation, age, sex, domicile.

Clinical history

- Fatigue and weight loss
- Anorexia and flatulent dyspepsia
- Abdominal pain
- Jaundice.
- Itching.
- Color of urine and feces
- Swelling of legs or abdomen
- **Hemorrhage:** Nose, gums, skin, alimentary tract
- **Loss of libido; menstrual history**
- **Past health:** Jaundice, hepatitis, drugs ingested, blood transfusion
- **Social:** Alcohol consumption
- **Family history:** Liver disease, autoimmune disease

Examination

- Nutrition, fever, fetor hepaticus, jaundice, pigmentation, purpura, finger clubbing, white nails, vascular spiders, palmar erythema, gynecomastia, testicular atrophy, distribution of body hair, parotid enlargement, Dupuytren's contracture, blood pressure
- **Abdomen:** Ascites, abdominal wall veins, liver, spleen, peripheral edema
- **Neurological changes:** Mental functions, stupor, tremor

Investigations

- **Hematology:** Hemoglobin, leukocyte and platelet count, prothrombin time (INR)
- **Serum biochemistry:**
  - Bilirubin
  - Transaminase
  - Alkaline phosphatase
  - γ-glutamyl transpeptidase
  - Albumin and globulin
  - Immunoglobulins
  - Transferrin saturation and serum ferritin
  - Serum ceruloplasmin and copper
  - α-1-antitrypsin phenotype
- **If ascites present:**
  - Serum sodium, potassium, bicarbonate, chloride, urea and creatinine levels
  - Weigh daily
  - 224 hours urine volume and sodium excretion
- **Serum immunological:**
  - Smooth muscle, mitochondrial, nuclear, LKM1 antibodies, and ANCA
  - Hepatitis B antigen (HBsAg), anti-HCV (other markers of hepatitis
  - α-fetoprotein
- **Endoscopy**
- **Hepatic ultrasound, CT or MRI scan**
- **Needle liver biopsy if blood coagulation permits**
- **EEG if neuropsychiatric changes**

Q. What are purpura, petechiae and ecchymoses?

Purpurae are discoloration of skin or mucus membrane due to extravasation of red blood cells. Petechiae are small purpuric lesions up to 2 mm in size whereas ecchymoses are larger (>2 mm) extravasations of blood. They do not blanch on pressure. Extravasated blood is broken down into various other pigments derived from hem in 2–3 weeks. This accounts for the characteristic color changes (purple, orange, brown, blue and green) which occur in purpuric lesions.

Q. Name the conditions where portal hypertension is present without dilated veins.

Dilated veins around the umbilicus (caput medusae) are a feature of **intrahepatic** portal hypertension. Here, some blood from the left branch of portal vein may be deviated via paraumbilical veins to the umbilicus where it reaches the veins of the caval system. These are absent in **extrahepatic** portal hypertension where dilated veins may appear in the left flank.

Q. What are the causes of extrahepatic portal hypertension?

Extrahepatic portal vein obstruction-usually due to thrombosis.

Important causes are:

- Infections—more common in children following spread of infection from umbilical vein. Other causes are acute appendicitis, peritonitis, biliary infections.
- Hypercoagulable states like myeloproliferative disorders, deficiency of protein C, S or antithrombin 3 or the presence of prothrombin gene mutation.
- Invasion and compression usually by hepatocellular carcinoma or carcinoma of pancreas.
- Post-splenectomy

Cirrhotic patients can develop portal vein thrombosis usually due to invasion by hepatocellular carcinoma.

Q. What are the causes of cirrhosis with enlarged liver?

- Hemochromatosis
- Primary biliary cirrhosis
- Primary sclerosing cholangitis
- Hepatocellular carcinoma in a cirrhotic liver
- Nonalcoholic steatohepatitis
- Cardiac cirrhosis
- Budd-Chiari syndrome
- Storage diseases of liver

Q. What is fulminant hepatic failure?

Fulminant hepatic failure is said to be present when the time interval between jaundice and hepatic encephalopathy is less than 2 weeks in patients without pre-existing liver disease. It is also characterized by coagulopathy. When the onset of hepatic encephalopathy is after 2 weeks it's called as subfulminant hepatic failure **(Table 3.5)**.

Table 3.5: Types of acute liver failure.

Types	*Interval: Jaundice to encephalopathy*
Hyperacute	<7 days
Acute	8–28 days
Subacute	29 days to 12 weeks

The following features help to differentiate acute liver failure from acute on chronic disease **(Table 3.6)**.

Table 3.6: Acute liver failure versus acute on chronic liver failure.

Clinical features	*Acute*	*Acute on chronic*
Nutrition	Good	Poor
Liver	±	+ Hard
Spleen	±	+ Hard
Spider naevi	Absent	++

Q. What is Gauchers disease and name the enzyme deficiency associated with Gauchers disease?

This is a rare autosomal recessive disease. It is the most common lysosomal storage disorder. It is due to the deficiency of lysosomal **acid beta glucosidase** leading to the accumulation of glucosylceramide, derived from membrane glycosphingolipids in the reticuloendothelial system

throughout the body, particularly in the liver, bone marrow and spleen.

There are three types depending on the type of mutations affecting the structural gene for acid beta glucosidase on chromosome 1:

1. **Type 1 (adult, chronic, non-neuronopathic):** Mildest and most common form. It occurs rarely in all ethnic groups but most common in Ashkenazi jews. The central nervous system is spared.
2. **Type 2 (infantile, acute, neuronopathic)—rare:** In addition to the visceral involvement there is massive, fatal neurological involvement with death in infancy.
3. **Type 3 (juvenile, subacute, neuronopathic):** Rare. There is gradual, heterogeneous neurological involvement.

The characteristic Gaucher cell (oval or polygonal in shape with pale cytoplasm with 2 or more peripherally placed hyperchromatic nuclei) accumulate in the perisinusoidal space and can form large aggregates associated with fibrosis which can be severe enough to resemble cirrhosis.

Treatment is by enzyme replacement therapy.

Q. What skin, nail, hand manifestations are seen in chronic liver disease?

- Spider nevi (telangiectatic superficial blood vessels with central feeding vessel)
- Clubbing of hands (especially biliary cirrhosis and hepatocellular carcinoma)
- Leukonychia
- Palmar erythema (blotchy appearance over the thenar and hypothenar eminence)
- Bruising
- Dupuytren's contracture (sign of alcoholism)
- Scratch marks (cholestatic jaundice)
- Pyoderma gangrenosa: Associated inflammatory bowel diseases (IBD) PBC or autoimmune cirrhosis.

Q. When does the abdominal bruit become significant?

Abdominal bruit becomes significant when it lateralizes to one side. Bruit will have both systolic and diastolic components and it suggests turbulent blood flow of partial arterial occlusion or arterial insufficiency.

Bruits confined to systole are relatively common and do not necessarily signify occlusive disease. Prolonged bruits are significant.

Q. Where does the paraumbilical vein drain into?

The paraumbilical vein drain into left portal vein.

Q. Briefly explain the anatomy of portal vein.

The portal vein begins at the level of the second lumbar vertebra and is formed from the convergence of the superior mesenteric and splenic veins. It lies anterior to the inferior vena cava and posterior to the neck of the pancreas. It lies obliquely to the right and ascends behind the first part of the duodenum, the common bile duct and gastroduodenal artery. At this point it is directly anterior to the inferior vena cava. It enters the right border of the lesser omentum, and ascends anterior to the epiploic foramen to reach the right end of the porta hepatis. It then divides into right and left main branches which accompany the corresponding branches of the hepatic artery into the liver. In the lesser omentum it lies posterior to both the common bile duct and hepatic artery. It is surrounded by the hepatic nerve plexus and accompanied by many lymph vessels and some lymph nodes. The right branch usually receives the cystic vein and then enters the right lobe. It usually forms an anterior division supplying segments V and VIII and a posterior division supplying segments VI and VII. The anterior division may give a branch to segment I. The left branch has a longer extra parenchymal course and tends to lie slightly more horizontal than the right branch but is often of smaller caliber. It gives off branches to segments I (caudate), II, III and IV (quadrate). As it enters the left lobe it is joined by paraumbilical veins and the ligamentum teres, which contains the functionless and partly obliterated left umbilical vein. It is connected to the inferior vena cava by the ligamentum venosum, a vestige of the obliterated ductus venosus. The small extrahepatic section of the left branch, from which the branches to segments II, III and IV arise, is a persistent part of the left umbilical vein.

The portal vein receives many branches including the splenic, superior mesenteric, left gastric, right gastric, paraumbilical and cystic veins. Portal venous blood is one route through which hepatic metastases from gastrointestinal primary malignancies may spread. Blood within the portal vein flows at such a rate that streaming may occur so that the blood from the splenic vein tends to remain on the left side of the portal bloodstream and drain preferentially to the left main branch. The clinical evidence to support this is very limited since colorectal cancer metastases commonly occur in the right lobe.

The following are common sites of portosystemic shunts:

- Between the left gastric and lower esophageal veins (portal) and the lower branches of the esophageal veins draining into the azygos and accessory hemiazygos veins (systemic). Enlargement of these anastomoses may result in the formation of varices, either esophageal or gastric. These may give rise to potentially fatal torrential bleeding.
- Between the superior rectal veins (portal) and the middle and inferior rectal veins draining into the internal iliac and pudendal veins (systemic). The dilated veins may be seen on the rectal wall, but rarely give rise to troublesome bleeding and are not a cause for internal hemorrhoids.
- Between persistent tributaries of the left branch of the portal vein running in the ligamentum teres and the periumbilical branches of the superior and inferior epigastric veins (systemic), forming the so-called 'caput medusae'.
- Between intraparenchymal branches of the right branch of the portal vein lying in liver tissue exposed in the 'bare

area' and retroperitoneal veins draining into the lumbar, azygos and hemiazygos veins.
- Between omental and colonic veins (portal) and retroperitoneal veins (systemic) in the region of the hepatic and splenic flexure.
- Rarely, between a patent ductus venosus connected to the left branch of the portal vein and the inferior vena cava.

Q. What is venous hum and where will you look for venous hum?

It is a continuous, low-pitched, soft murmur that may become louder with inspiration and diminish when more pressure is applied to the stethoscope. Typically, it is heard between xiphisternum and the umbilicus in cases of portal hypertension. It may radiate to chest or over to the liver.

Large volumes of blood flowing in the umbilical or paraumbilical veins in the falciform ligament are responsible. These channel blood from the left portal vein to the epigastric or internal mammary veins in the abdominal wall. A venous hum may occasionally be heard over the large vessels such as inferior mesenteric vein or after portocaval shunting. Sometimes a thrill is detectable over the site of maximum intensity of the hum. The Cruveilhier-Baumgarten syndrome is the association of a venous hum at the umbilicus and dilated abdominal wall veins. It is almost always due to cirrhosis of liver. It occurs when patients have a patent umbilical vein, which allow portosystemic shunting at this site. The presence of a venous hum or of prominent central abdominal veins suggest that the site of portal obstruction is intrahepatic rather than in the portal vein itself.

Q. What are the cardinal features of IVC obstruction?

- Dilated veins on the flanks and back.
- Cyanosis and edema of the legs appear, dilated varicose veins involve the legs, abdominal wall and even the thorax.
- Veins of the lower abdominal wall, which normally fill from above downwards, fill from below upwards.

Q. What are the evidence of hepatic encephalopathy?

- Flapping tremor—asterixis
- Disturbed consciousness
- Inversion of sleep rhythm
- Personality changes like childishness and irritability
- Constructional apraxia—inability to reproduce simple designs with blocks
- Intellectual deterioration elicited easily by number connection test.
- Monotonous slow and slurred speech.
- Exaggerated deep tendon reflexes.
- Increased muscle tone
- Sustained ankle clonus often associated with rigidity.
- Flexor plantar response
- Extensor plantar in deep stupor or coma
- Hyperventilation and hyperpyrexia may be terminal.

Q. Name the staging criteria for hepatic encephalopathy and how will you grade hepatic encephalopathy?

West Haven criteria (Table 3.7)

Table 3.7: Grading of hepatic encephalopathy.

Clinical stage	*Impairment of intellectual function*	*Impairment of neuromuscular function*
Subclinical	Normal examination findings, but work or driving may be impaired	Subtle changes on psychometric or number connection tests
Stage 1	Impaired attention, irritability, depression, or personality change	Tremor, incoordination, apraxia
Stage 2	Drowsiness, behavioral changes, poor memory and computation, sleep disorders	Asterixis, slowed or slurred speech, ataxia
Stage 3	Confusion and disorientation, somnolence, amnesia	Hypoactive reflexes, nystagmus, clonus, and muscular rigidity
Stage 4	Stupor and coma	Dilated pupils and decerebrate posturing; oculocephalic ("doll's eye") reflex; absence of response to stimuli in advanced stages

Q. How will you test for constructional apraxia?

- Number connection test **(Fig. 3.11)**
- Digit symbol test **(Fig. 3.12)**
- Serial dotting **(Fig. 3.13)**
- Line tracing test **(Fig. 3.14)**.
- Subjects are asked to join the numbers in sequence as quickly as possible. The time taken to complete the task is recorded.
- To be more informative, time taken should be compared with normal values of same age group.

Digit symbol test

- Subjects are asked to insert symbols in the blank squares below the numbers using the key provided.
- The exercise is timed and the number correctly completed in 90 seconds recorded.

Serial dotting

Subjects are asked to place a dot in the center of each circle and to complete the page as quickly as possible. The time taken to complete the task is recorded.

Line tracing test

- Subjects are asked to trace a line between the two guidelines as quickly and accurately as possible without moving the paper.
- The time taken to complete the task and the number of errors made are recorded.

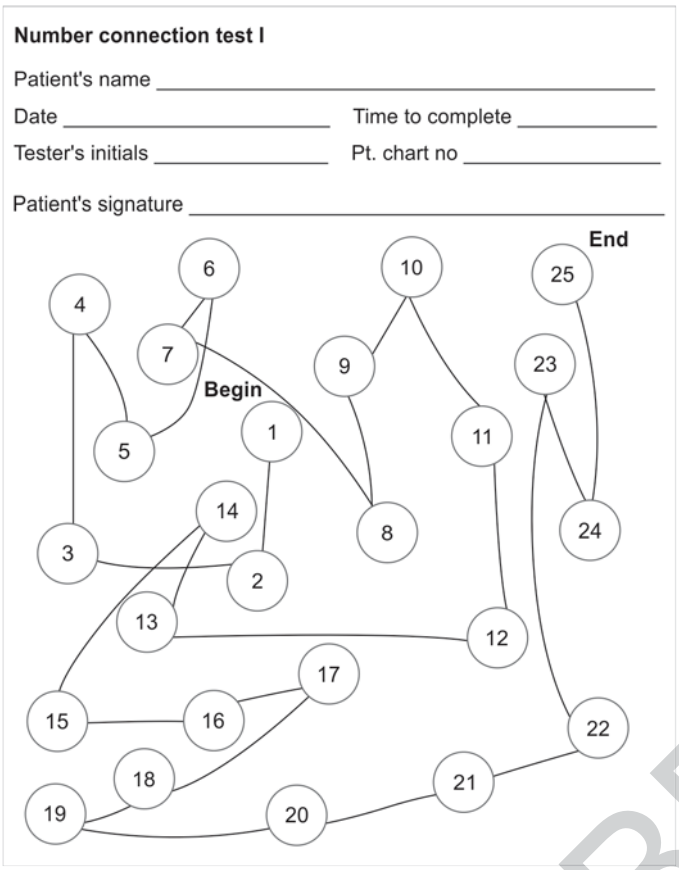


Fig. 3.11: Number connection test.

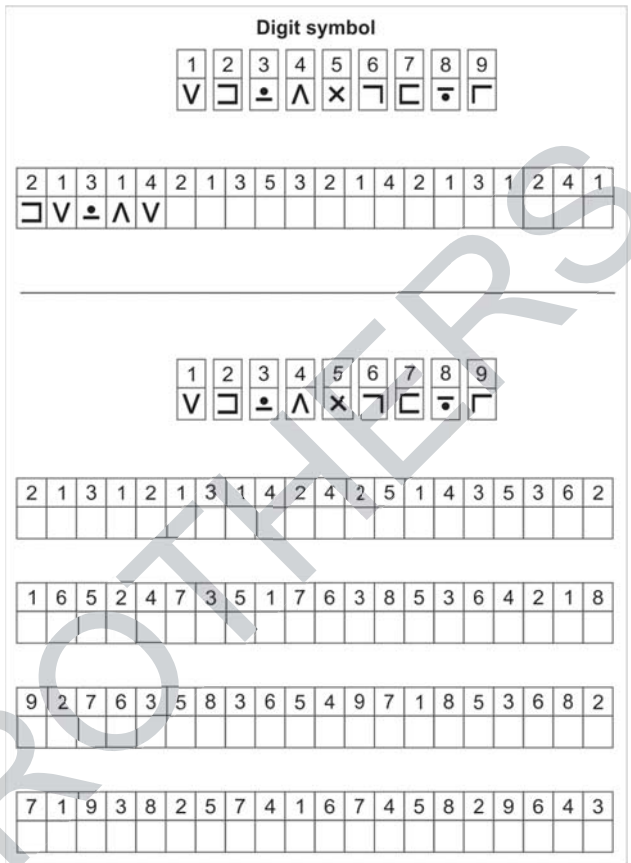


Fig. 3.12: Digit symbol test.

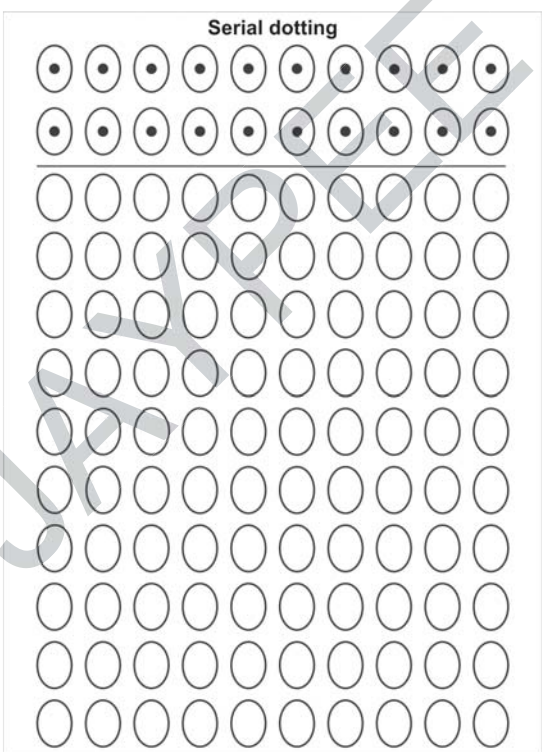


Fig. 3.13: Serial dotting.

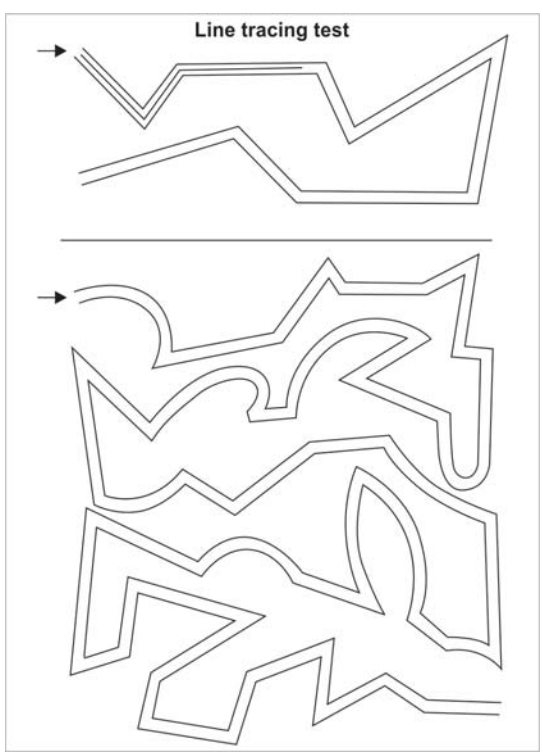


Fig. 3.14: Line tracing test.

This battery, which has been called the psychometric hepatic encephalopathy score (PHES), assesses the required domains of attention, visual perception and executive abilities; it is easily applied and has been shown to have a ***high specificity for the diagnosis of hepatic encephalopathy.***

Q. What is the significance of cervical venous hum?

Heard as a continuous humming sound over prominent veins seen in the root of neck in supraclavicular region.

- Venous hum indicates that there is hyperkinetic circulation most commonly chronic compensated anemia.

Characteristics of venous hum

- Soft and low-pitched
- Often continuous with diastolic accentuation
- Best heard in sitting or erect position with the bell of the stethoscope
- Best audible in inspiration
- Disappears in pressing the bell of the stethoscope and with Valsalva maneuver.

Q. What is a venous nevus?

Venous nevus is a vascular malformation that occurs due to a mutation during the postzygotic embryogenesis phase. These lesions never cross the midline.

These should be differentiated from other vascular malformations that are not categorized as nevi.

Examples: Varicose veins associated with Klippel-Trenaunay syndrome, deep vein anomalies of Parkes-Weber syndrome, multiple lesions of the blue rubber nevus syndrome.

Q. What is the triad of portal hypertension?

- Splenomegaly—definite evidence of portal hypertension
- Ascites
- Dilated abdominal wall veins/varices.

Q. How do you rule out chronic Budd-Chiari in this patient?

Patients with Budd-Chiari syndrome will have an absent hepatojugular reflux.

Q. What are the noncardiac causes of raised JVP?

- Innominate vein thrombosis
- Superior vena cava (SVC) obstruction
- Chronic obstructive pulmonary disease (COPD)
- Massive right sided pleural effusion
- Pulmonary embolism
- Ascites
- Pregnancy
- Renal disease
- Excess IV fluids

Q. How to know the site of obstruction in SVC by assessing JVP?

The rise in JVP is greater when the site of obstruction is near the junction with azygous and proximal to the opening of azygous **(Figs. 3.15 to 3.17)**.

Q. What is the significance of the flow of veins from bottom to top?

In intrahepatic portal hypertension, blood from the left branch of the portal vein is deviated via the paraumbilical veins to the umbilicus from where it reaches the caval veins. In extrahepatic portal obstruction dilated veins appear on the flanks.

In IVC obstruction the flow of blood through the collaterals is from top to bottom to reach the superior vena caval system.

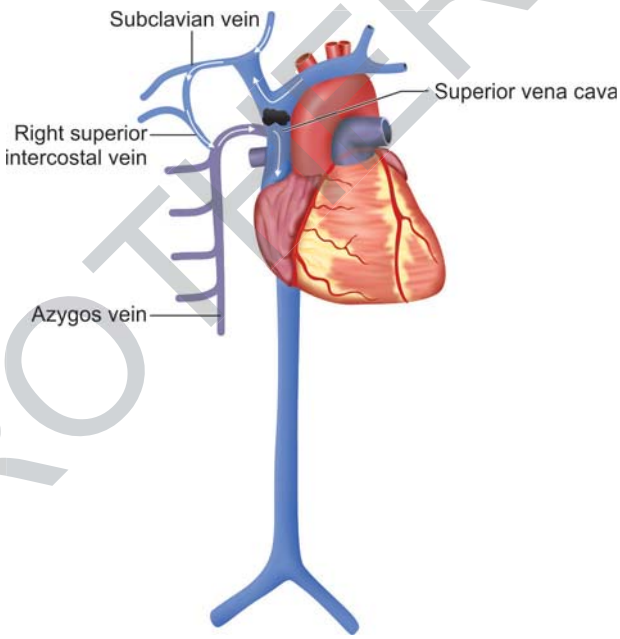


Fig. 3.15: Obstruction of SVC proximal to the opening of azygous vein.

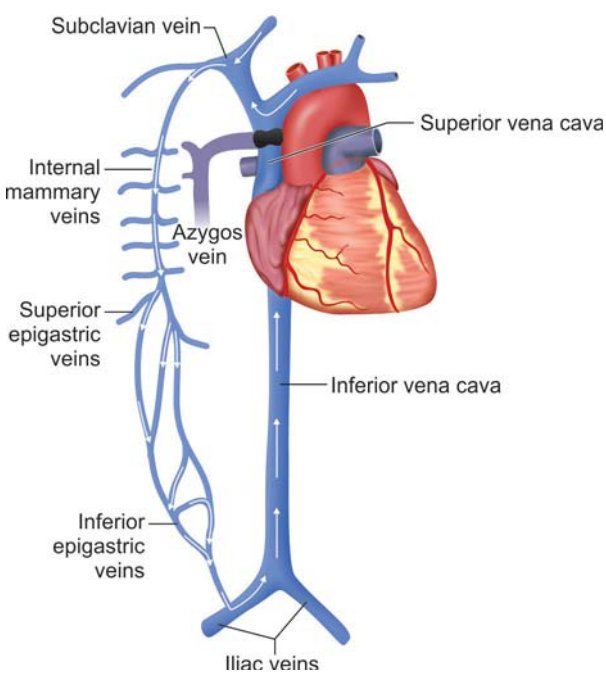


Fig. 3.16: Obstruction of SVC at junction with azygous.

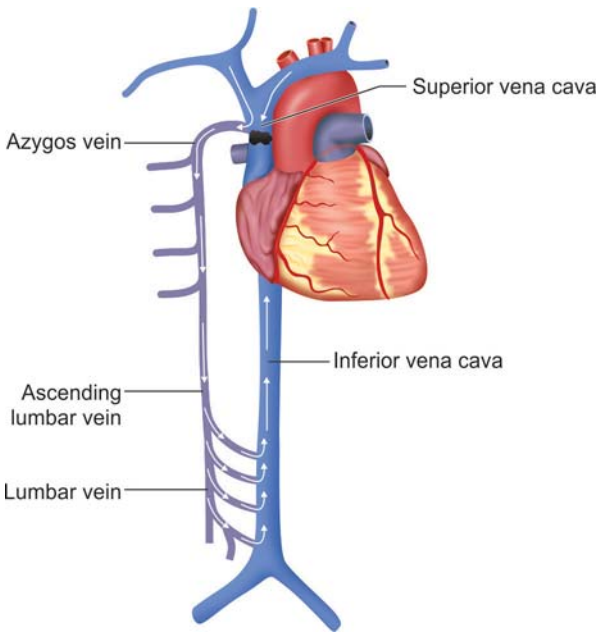


Fig. 3.17: Obstruction of SVC distal to opening of azygous vein.

Hence if collateral veins are present and the flow of veins is from bottom to top, it is highly suggestive of IVC obstruction **(Figs. 3.18A to C)**.

Sometime tense ascites may lead to functional obstruction of the IVC and cause difficulty in interpretation.

Q. How do you classify portal hypertension?

Portal hypertension can be classified into two types **(Fig. 3.19)**:

1. **Presinusoidal:**
   - Extrahepatic: Blocked portal vein and increased splenic flow. Examples: splenic vein and portal vein thrombosis, idiopathic tropical splenomegaly, IVC thrombosis, hepatic vein thrombosis, constrictive pericarditis, Budd-Chiari
   - Intrahepatic: Portal zone infiltrates, toxic and hepatoportal sclerosis. Examples: Schistosomiasis, early primary biliary cirrhosis, sarcoidosis
2. **Hepatic:**
   - Sinusoidal: Cirrhosis, cytotoxic drugs, acute alcoholic hepatitis
   - Postsinusoidal: Veno-occlusive disease, alcoholic central hyaline sclerosis.

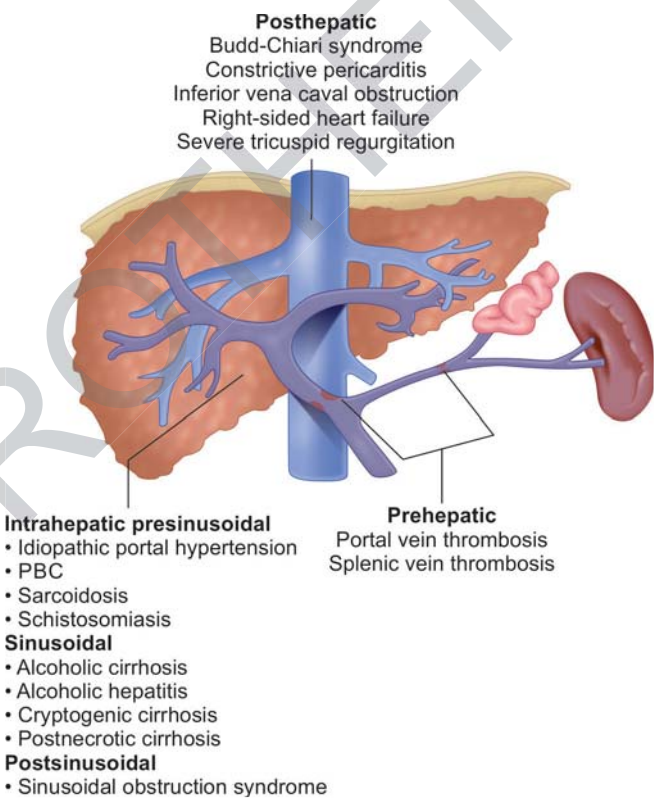
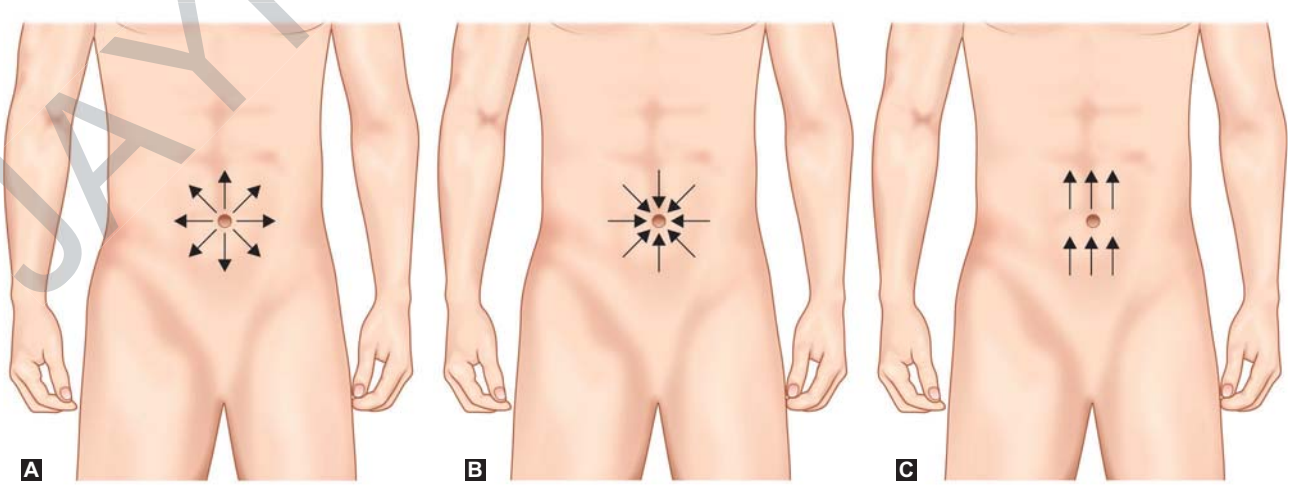


Fig. 3.19: Classification of portal hypertension.



Figs. 3.18A to C: Direction: (A) Portal hypertension; (B) Portal vein thrombosis; (C) IVC obstruction.

Q. What is the sine qua non of portal hypertension?

Splenomegaly.

Q. What are the clinical signs of IVC obstruction? Cyanosis and edema of the legs.

Varicosities of the lower limbs and varicocele of the testes. Large dilated tortuous veins over the abdomen and chest.

Filling of the veins of the lower abdominal wall from bottom to top hematuria, albuminuria and casts if the renal vein is involved. Venous ulcer in the lower limbs due to stasis.

Q. What are the differences between hepatic and prehepatic portal hypertension?

Refer **Table 3.8**.

Table 3.8: Hepatic versus prehepatic portal hypertension.

Clinical features	*Hepatic*	*Prehepatic*
Ascites	Persistent and late	Transient and early
Sings of liver cell failure	Present	Absent
Dilated veins	Periumbilical dilated veins	Dilated veins over the flank
Venous hum	Present	Absent
Size of liver	Usually shrunken	Liver is normal
Causes	Cirrhosis, tumors of the liver, alcoholic hepatitis	Thrombosis of the splenic vein, portal vein thrombosis, IVC obstruction

Q. What are the causes and clinical features of Budd-Chiari syndrome?

It is a syndrome which consists of hepatomegaly, abdominal pain, ascites and hepatic histology showing zone 3 sinusoidal distension and pooling. It is caused due obstruction of the hepatic vein at any site from the efferent vein of the acinus to the entry of the IVC. Other causes of obstruction like pericardial disease, cardiac disease and veno-occlusive disease have to be ruled out before a diagnosis of Budd- Chiari can be made **(Figs. 3.20 and 3.21)**.

It can present as acute, chronic and asymptomatic forms.

- **Acute:** Characterized by acute liver injury with elevated transaminases, jaundice, hepatic encephalopathy, and an elevated prothrombin time/INR. Hepatic encephalopathy develops within 8 weeks after the development of jaundice.
- **Chronic:** Patients present with complications of cirrhosis
- **Asymptomatic:** Diagnosed fortuitously by imaging or by abnormal LFT values.

The common causes include:

- Myeloproliferative disorders like polycythemia rubra vera
- Connective tissue disorders like SLE, APLA, idiopathic granulomatous vasculitis, Behcet's
- Paroxysmal nocturnal hemoglobinuria
- Protein C and protein S deficiency

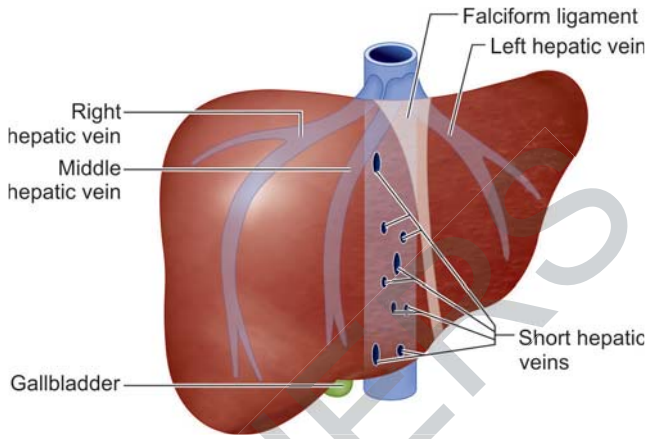


Fig. 3.20: Figure showing the anatomy of the hepatic veins.

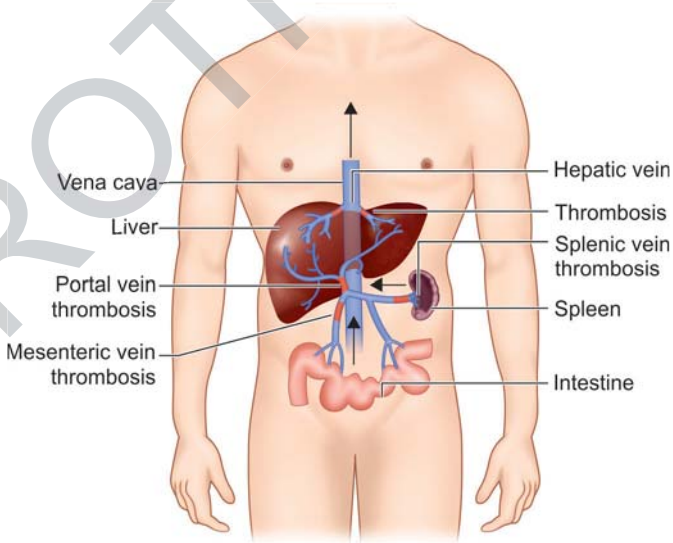


Fig. 3.21: Budd-Chiari syndrome.

- Antithrombin III deficiency
- Drugs—oral contraceptives
- Secondary to obstruction of the IVC due to thrombus produced by tumors like renal cell carcinoma, hepatocellular carcinoma

Q. What is Jamaican Bush Tea syndrome?

It is a syndrome characterized by the development of veno-occlusive disease resulting in cirrhosis of the liver. It was seen in Jamaica where the bush tea leaves were used as food and medicinal purposes. It was seen particularly in children and poorer sections of the community. Pharmacologic analysis of the tea revealed the presence of a toxic substance in a common plant in Jamaica, *Crotalaria fulva* known locally as 'white back'.

The disease was common in the early 1900s. By 1960 due to successful public health awareness and campaigning, there was a dramatic fall in the number of cases.

Internationally, it was recognized as the first cause of veno-occlusive disease leading to cirrhosis of the liver. It is rare to see the syndrome nowadays.

Q. Define acute liver failure.

Acute liver failure is defined as the rapid progressive deterioration in liver function, specifically coagulopathy and mental status changes (encephalopathy) in a patient without known prior liver disease.

Q. What are the clinical features of acute liver failure?

- Hepatomegaly
- Fatigue/malaise
- Lethargy
- Anorexia
- Nausea and/or vomiting
- Right upper quadrant pain
- Pruritus
- Jaundice
- Abdominal distension from ascites

Q. What are the features of cholestatic jaundice?

- Deep jaundice with greenish hue
- Scratch marks (pruritis—as bile salts saponify the fat surrounding the free nerve endings)
- Bradycardia
- Xanthelasmas on the eyelids **(Fig. 3.22)** and xanthomas over tendons due to lipid deposit
- Palpable gallbladder in carcinoma head of pancreas
- Large hard irregular liver in malignancy
- Late features: Secondary biliary cirrhosis and signs of liver cell failure.

Q. What is Courvoiser's law?

In the presence of jaundice, a palpable nontender gallbladder is unlikely due to gallstones.

In obstruction of common bile duct due to a stone, the gallbladder as a rule is impalpable (no distension). This is because the gallbladder is already shriveled, fibrotic and nondistensible and hence will not be palpable.

In obstruction from other causes (carcinoma head of pancreas) distension of gallbladder is common and hence gallbladder may be palpable.

Fig. 3.22: Xanthelasmas around the eyes.

Q. What are the exceptions to Courvoisier's law?

- **Double impaction:** Stones, simultaneously occluding the cystic duct and distal common bile duct
- Pancreatic calculus obstructing the ampulla of Vater
- Oriental cholangiohepatitis
- Periampullary carcinoma in the patients with cholecystectomy
- Mirizzi syndrome—common hepatic duct obstruction caused by an extrinsic compression from an impacted stone in the cystic duct or Hartmann's pouch of gallbladder

Q. What are the ultrasound features of portal hypertension?

- **Liver size:** <10 cm
- **Hepatic vein:** 11 mm
- **Portal vein:** 12 mm
- **Spleen:** >13 cm

Q. What are the ultrasound features of chronic portal hypertension?

Intrahepatic portal cavernoma **(Fig. 3.23)**—occurs when the native portal vein is thrombosed, and myriads of collateral channels develop in the porta hepatis to bypass the occlusion. Cavernous transformation results from recanalization of the portal venous thrombus as well as dilatation of paracholedochal veins in an effort to bypass the portal venous obstruction.

In cirrhosis, cavernous transformation of the portal vein is rare because stasis of portal venous flow prevents the formation of collateral channels in and around the portal venous thrombus.

Q. What is Murphy sign?

It is seen in acute cholecystitis, ask the patient to breath in deeply, and now try to palpate the gallbladder in sitting position. There is tenderness and catch in the breath at the height of inspiration with a mass felt there.

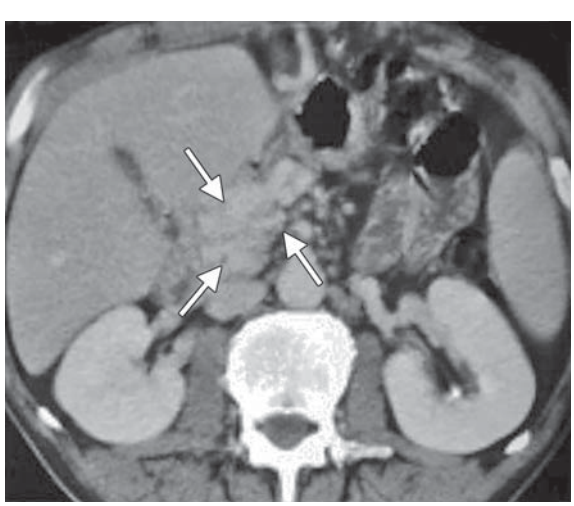


Fig. 3.23: CECT axial section reveals cavernoma formation in the extrahepatic portal vein marked by arrow heads.

Q. What differentials will you consider when a CLD patient with ascites develops fever?

- Spontaneous bacterial peritonitis
- Pneumonia, UTI, endocarditis, lymphangitis
- Hepatocellular carcinoma

Q. What to look for in the hands in liver disease?

- Leukonychia—hypoalbuminemia
- Knuckle pigmentation—hemochromatosis, Addison's disease
- Dupuytren's contracture and palmar erythema—alcoholic liver disease
- Clubbing—primary biliary cirrhosis.

Q. If it is stomach carcinoma, what else to be looked for?

- Virchows node (Troisier's sign)
- Jaundice—due to metastasis to liver
  - Or involvement of porta hepatis

Q. Why are there dilated veins on the anterior wall of abdomen?

This is suggestive of either portal hypertension or IVC obstruction due to massive ascites. Look for veins below the umbilicus and check the direction of flow. If the flow is away from the umbilicus suggestive of portal hypertension. If it is towards the umbilicus, due to IVC obstruction.

Q. Define cirrhosis.

Cirrhosis is the end stage of any chronic liver disease.

It is a diffuse process (entire liver is involved) characterized by fibrosis and conversion of normal architecture to structurally abnormal regenerating nodules of liver cells. **Table 3.9** summarizes types of cirrhosis.

Table 3.9: Types of cirrhosis.

Micronodular cirrhosis	*Macronodular cirrhosis*	*Mixed cirrhosis*
✦ Also called Laennec's cirrhosis ✦ Regular and small regenerating nodules of <3 mm diameter ✦ Uniform thin regular fibrous connective tissue septa ✦ Involvement of every lobule of whole liver ✦ Alcoholic cirrhosis, biliary cirrhosis, venous occlusion	✦ Regenerating, irregular, coarse nodules of variable size, usually of >3 mm diameter ✦ Fibrous connective tissue is broad and variable in thickness ✦ Liver surface grossly distorted ✦ Increased risk of developing hepatocellular carcinoma ✦ Most common cause is postnecrotic cirrhosis (chronic viral hepatitis)	✦ Features of both micro and macronodular cirrhosis

The three main morphologic characteristics of cirrhosis are:

1. Fibrosis
2. Regenerating nodules
3. Loss of architecture of the entire liver

Factor affecting liver cirrhosis is summarized in **Table 3.10** and main causes of cirrhosis are discussed in **Box 3.1**.

Q. Why is it a decompensated cirrhosis?

The symptom triad of decompensated cirrhosis:

- Abdominal distension (ascites)
- **Internal/external bleeding:** Varices/portal hypertension

Table 3.10: Factors affecting liver cirrhosis.

Quantity of alcohol and gender	✦ **Males:** 40–80 g/day—fatty liver, >160 g/day—hepatitis, cirrhosis; >14 drinks/week—causes damage ✦ **Females:** >20 g/day or >7 drinks/week—causes damage
Coinfections	Moderate alcohol consumption but chronic infection with hepatitis B, C accelerates development of cirrhosis
Genetics	Patatin-like phospholipase domain-containing protein 3 (PNPLA3) is associated with alcoholic cirrhosis
Fatty liver	Co-existence of obesity and NASH are risk factors for development of cirrhosis in alcoholics

Box 3.1: Main causes of cirrhosis.

- ✦ Alcohol (most common causes)
- ✦ Chronic viral hepatitis (most common cause)
  - Hepatitis B
  - Hepatitis C
  - Delta hepatitis (hepatitis D) + hepatitis B
- ✦ Nonalcoholic steatohepatitis (NASH) or nonalcoholic fatty liver disease (NAFLD) (earlier was considered as cryptogenic cirrhosis)
- ✦ Biliary cirrhosis
  - Primary biliary cholangitis
  - Secondary biliary cirrhosis
  - Primary sclerosing cholangitis
  - Autoimmune cholangiopathy, IgG4 cholangiopathy
- ✦ Autoimmune hepatitis
- ✦ Budd-Chiari syndrome
- ✦ Intrahepatic or extrahepatic biliary obstruction: Recurrent biliary obstruction (e.g., gallstones)
- ✦ Inherited metabolic liver disease
  - Hemochromatosis
  - Wilson's disease
  - α_1 antitrypsin deficiency
  - Cystic fibrosis
  - Glycogen storage disease
- ✦ **Drug-induced cirrhosis:** For example, methotrexate, methyldopa, isoniazid, phenylbutazone, sulfonamides
- ✦ **Others:** Indian childhood cirrhosis, cardiac cirrhosis, chronic venous outflow obstruction, celiac disease. Hereditary hemotelangiectasia, infection [e.g., brucellosis, syphilis, echinococcosis, porphyria, idiopathic adulthood ductopenia (Carolia disease)]

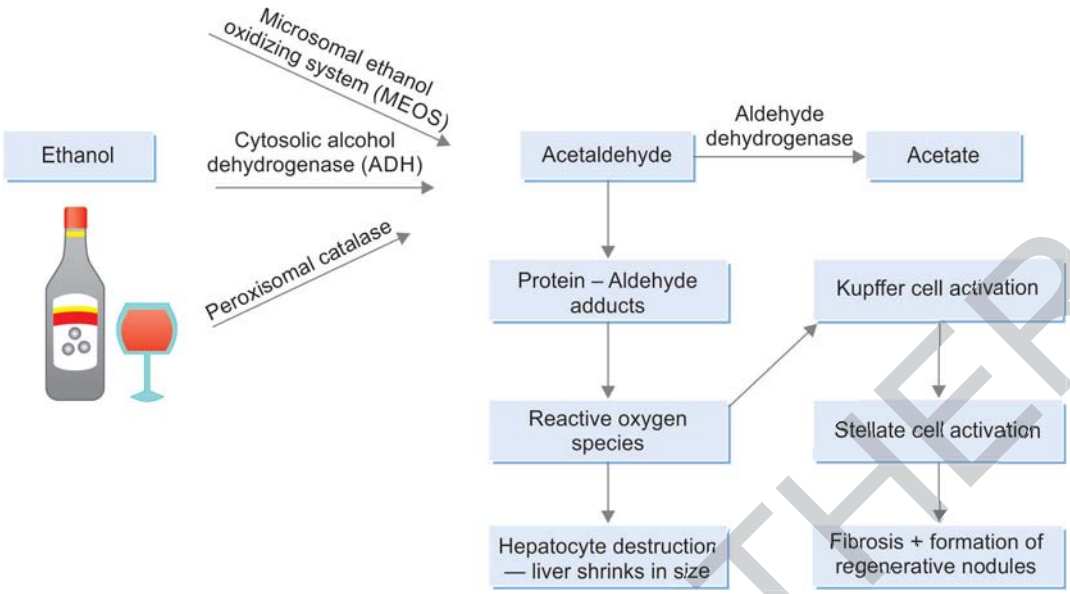


Fig. 3.24: Pathogenesis of alcoholic cirrhosis.

- Behavioral/mental changes—Encephalopathy

Other decompensations:

- Jaundice
- Hepatocellular carcinoma (HCC)

Pathogenesis of alcoholic cirrhosis is given in **Figure 3.24**.

Q. What is the ophthalmic sign of liver cell failure?

Bitot's spots **(Fig. 3.25)**

- Caused due to vitamin A deficiency as a consequence of malabsorption due to decreased fat content in bile.
- Rare in cirrhosis among adults irrespective of etiology.
- It is a sign of liver cell failure.

Q. What is Kayser-Fleischer (KF) ring?

- Named after Bernhard Kayser and Bruno Fleischer
- Copper deposited in Descemet's membrane.
- First appears at 12 o'clock position then at 6 o'clock position, then encircles completely.
- About 85–100% of patients with neurological and/or psychiatric manifestations of Wilson's disease but only 33–86% of patients with hepatic disease and 0–59% of asymptomatic patients.

Fig. 3.25: Bitot's spot.

- Slit lamp examination is mandatory to make a diagnosis of KF rings particularly in the early stages unless the rings are visible to the naked eye in conditions of severe copper overload.
- Also seen in other liver diseases such as primary biliary cirrhosis, neonatal hepatitis, and cryptogenic cirrhosis.
- Or elevated copper for other reasons such as in multiple myeloma, pulmonary carcinoma, benign monoclonal gammopathies, chronic lymphocytic leukemia, or even oral contraceptive use.
- After the initiation of treatment, the Kayser-Fleischer ring disappears in 85–90% of cases.

Differences between KF ring and Arcus is discussed in **Table 3.11.**

Table 3.11: KF rings versus arcus.

Features	*KF ring*	*Arcus*
Naked eye examination	May be seen	Not seen
Site of the ring	Superior/interior/ circumferential	Always circumferential
Color	Golden brown	Yellowish green
Texture	Granular	Homogenous
Layer of cornea involved	Descemet's membrane	Peripheral stroma
Relation to bilirubin level	Absent	Present
Response to chelation therapy	Improved	Not applicable

Q. Why cirrhosis patients are prone for increased risk of bleeding?

- Often thought due to decreased coagulation factors
- May occur due to thrombocytopenia
- Increase in plasma fibrinolysins in cirrhosis
- Dysfibrinogenemia due to increased sialic acid in cirrhosis

Q. Why does parotid enlargement seen in alcoholic cirrhosis?

- Occurs in 50% of alcoholic cirrhosis
- Painless and soft enlargement
- Earlier thought due to hypersecretory parotid
- Edema and fatty infiltration
- Now appears to be due to presence of autonomic neuropathy
- Size can fluctuate during heavy alcohol intake

Q. Why is there a loss of facial/chest hair in cirrhosis?

- Loss of male pattern of hair **(Fig. 3.26)**
- Density of hair over face and chest is not different in cirrhosis compared to controls
- Asians by nature have sparse chest hair
- Clinical significance is questionable
- Scalp hair usually spared

Q. What are the causes of jaundice in a cirrhosis patient?

- Mostly due to progressive hepatocellular injury
- Can be due to hypersplenism related to hemolysis
- Can be due to obstruction by gallstones (increase on account of hemolysis) or pancreatitis
- Hepatoma
- Superadded acute injury

Q. What are the causes of acute decompensation in a chronic liver disease patient?

- Superadded hepatitis
- Sepsis including SBP
- Malignant transformation
- GI bleed

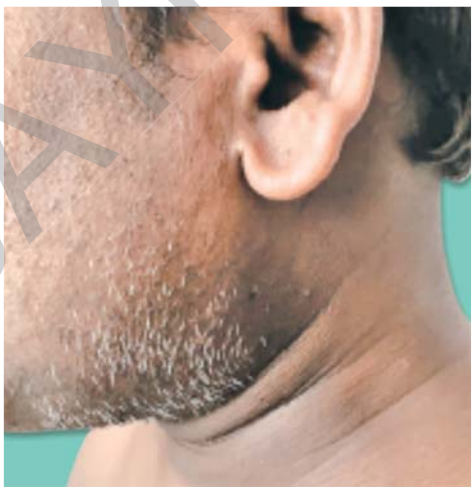


Fig. 3.26: Diminished facial hair with parotid enlargement.

- Renal failure
- Cardiac failure
- Noncompliance

Q. Enumerate the complications of cirrhosis.

Figure 3.27 shows the complication of cirrhosis.

- Portal hypertension
- Esophageal varices **(Fig. 3.28)** and gastropathy.
- Splenomegaly
- Edema and ascites.
- Spontaneous bacterial peritonitis.
- Bruising and bleeding. Due to coagulopathy and thrombocytopenia
- **Jaundice:** It is rare and can be seen with obstructive causes, hepatoma, or superadded hepatitis.
- Hepatic encephalopathy.
- Hepatocellular carcinoma
- Insulin resistance and type 2 diabetes. Diabetes mellitus is seen in 15–30% of patients with cirrhosis
- Immune system dysfunction
- Hepatorenal and hepatopulmonary syndromes
- Hepatic hydrothorax
- Portopulmonary hypertension
- Cirrhotic cardiomyopathy
- Hepatic osteodystrophy
- Hepatic neuropathy
- Hepatic myelopathy.

Q. What are the causes of anemia in cirrhosis?

It can be due to various causes:

- Acute and chronic blood loss from varices
- Nutritional deficiency of vitamin B12 and folate
- Hypersplenism
- Bone marrow suppression by alcohol
- Hemolysis
- **Zieve's syndrome:** Alcohol-induced hemolytic anemia with hypercholesterolemia.

Q. What is asterixis?

Refer **Table 3.12**.

Table 3.12: Causes of asterixis.

Bilateral asterixis	*Unilateral asterixis*
Metabolic: Liver failure, azotemia, respiratory failure **Sedatives:** Benzodiazepines, barbiturates **Anticonvulsants:** Phenytoin (phenytoin flap), carbamazepine, valproic acid, gabapentin **Antipsychotics:** Ceftazidime **Others:** Metoclopramide **Dyselectrolytemia:** Hypomagnesemia, hypokalemia **Bilateral structural brain lesions**	**Focal brain lesions at:** ✦ Thalamus ✦ Corona radiata ✦ Anterior cerebral artery territory ✦ Primary motor cortex ✦ Parietal lobe ✦ Cerebellum ✦ Midbrain ✦ Pons

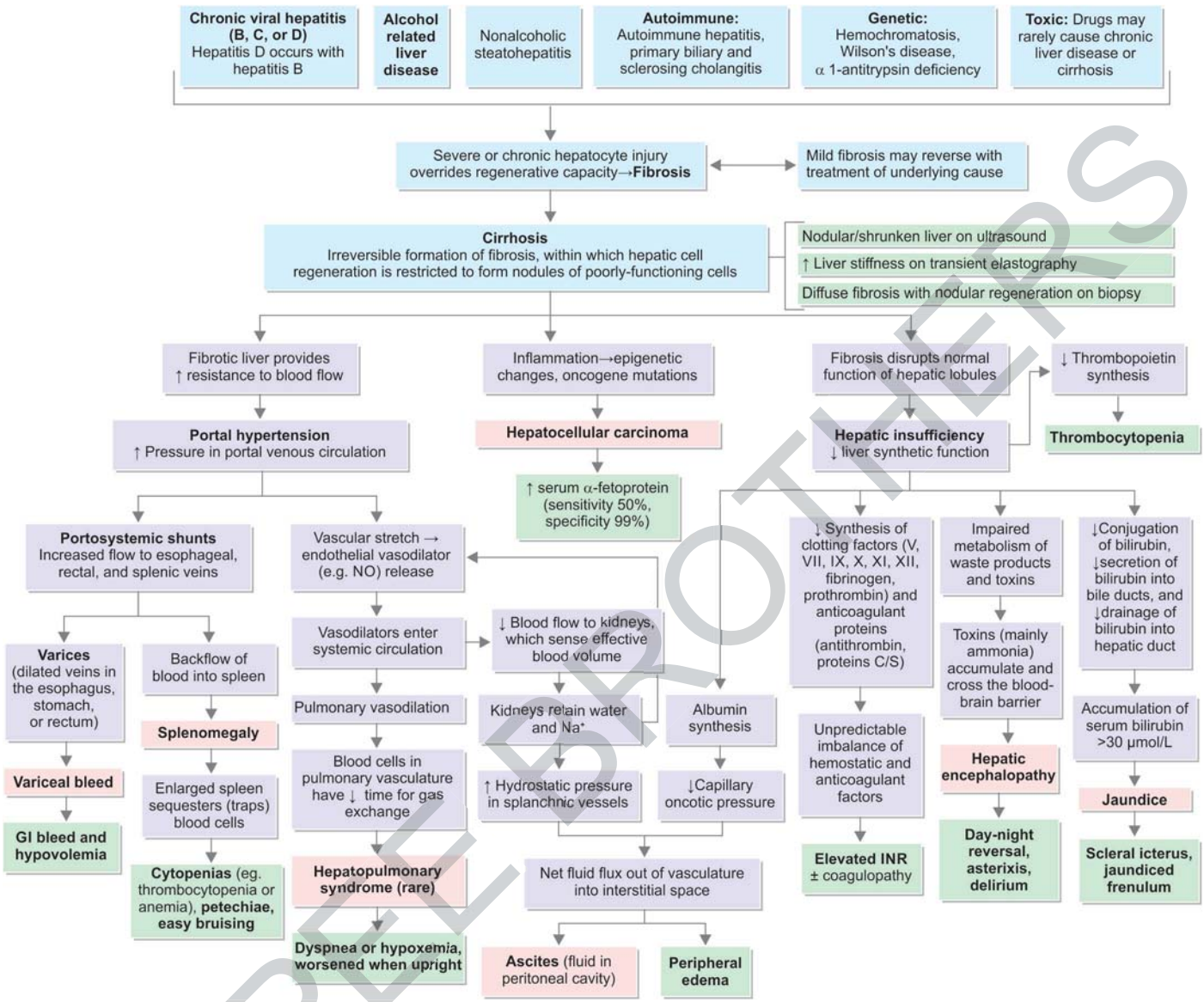


Fig. 3.27: Complications of cirrhosis.

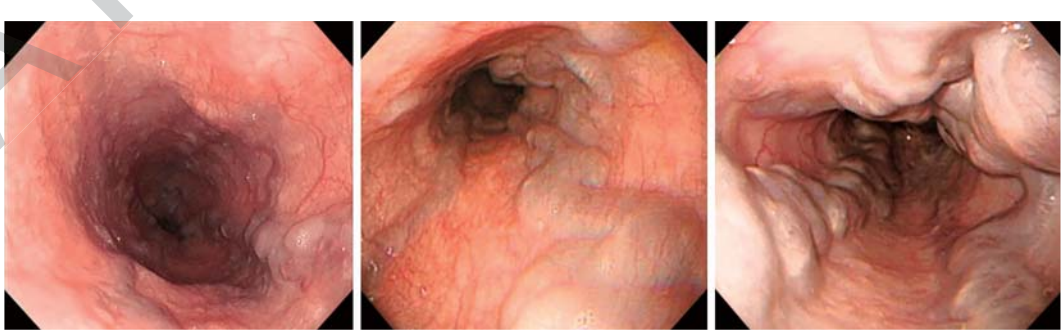


Fig. 3.28: Endoscopy view of esophageal varices (grade I, II, and III).

- Asterixis is a disorder of motor control characterized by an inability to actively maintain a position and consequent irregular myoclonic lapses of posture affecting various parts of the body independently.
- It is a type of negative myoclonus characterized by a brief loss of muscle tone in agonist muscles followed by a compensatory jerk of the antagonistic muscles.

Q. What is the mechanism of asterixis?

Projections from the medial frontal cortex to the brainstem reticular formation have a role in regulating muscle tone or posture.

There is dysregulation of the diencephalic motor centers in the brain that regulate innervation of muscles responsible for maintaining position.

Fluid shifts cause swelling of Alzheimer type II astrocytes and metabolic derangements, leading to compromise in the blood–brain barrier, upregulation of peripheral benzodiazepine receptor, and the production of neurosteroids.

They are brief, arrhythmic interruptions of sustained voluntary muscle contraction causing brief lapses of posture, with a frequency of 3–5 Hz. It is bilateral, but may be asymmetric.

The exact mechanism underlying asterixis remains elusive and many explanations have been forthcoming. The following pathogenic mechanisms have been suggested:

- "Receptive inattentiveness to incoming information", which could thus result from a dysfunction of the sensorimotor integration occurring in the contralateral parietal lobe and midbrain.
- Episodic dysfunction within neural circuits concerned with maintenance of sustained or tonic muscle contraction, due to focal, specific brain lesions or by a generalized neurochemical imbalance. The existence of a possible neural subsystem whose dysfunction could result in asterixis rather than "nonspecific" CNS lesions was hypothesized. Drowsiness in normal people and diffuse CNS lesions can also produce asterixis, perhaps by their effects on alerting or arousal mechanisms rather than by nonspecific CNS actions.
- Electrophysiological evaluation of asterixis using silent period locked averaging method revealed negative sharp waves in the contralateral central area. It was suggested that asterixis is due to abnormal activity in the motor field in the cerebral cortex.
- Recently, mini-asterixis which is a part of the spectrum of the gross flapping tremor seen in hepatic encephalopathy, was proposed as being due to the involvement of motor cortex causing a pathologically slowed and synchronized motor cortical wave.

Q. How will you demonstrate asterixis in upper limb?

Asterixis is tested by extending the arms, dorsiflexing the wrists, and spreading the fingers to observe for the "flap" at the wrist **(Fig. 3.29)**. The flap is due to irregular myoclonic lapses of posture caused by involuntary 50–200 ms silent periods appearing in tonically active muscles.

Fig. 3.29: Demonstration of asterixis in hands.

There may be a latent period between adopting the posture and the beginning of asterixis, so it is important to wait at least 30 seconds before concluding the test.

Q. What is mini-asterixis?

"Mini-asterixis" has been coined as a term to describe very fine asterixis affecting the fingers, which may be mistaken for tremor.

On electromyography (EMG) of the involved muscles, each loss of tone is associated with a silent period of between 50 and 200 ms.

Q. How will you demonstrate asterixis in lower limbs?

Testing asterixis at the hip joint involves keeping the patient in a supine position with knees bent and feet flat on the table, leaving the legs to fall to the sides. Negative myoclonus of the lower limbs at the hip joints repetitively occurs and is appreciated by looking at the knees **(Fig. 3.30)**.

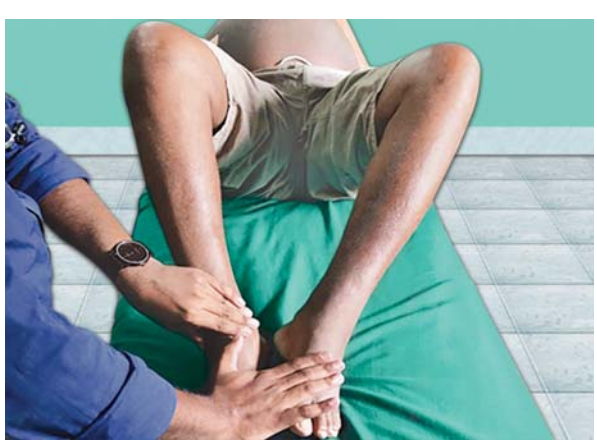


Fig. 3.30: Demonstration of flapping tremors in legs—on leaving the legs to fall apart a negative myoclonus can be noticed by observing the knee.

Q. Name the other methods to elicit asterixis.

- Request the patient to squeeze the doctor's hand or the doctor's extended fingers. Patients who are unable to maintain a posture usually are unable to maintain a steady squeeze.
- Have the patient squeeze a semi-inflated blood pressure cuff with instructions to maintain the reading. The readings bounce dramatically in patients with asterixis.

Q. What are the signs pointing to the different etiologies of cirrhosis?

Table 3.13 summarizes signs pointing the etiology of cirrhosis.

Table 3.13: Signs pointing the etiology of cirrhosis.

Signs	*Etiology of cirrhosis*
Parotid enlargement, Dupuytren's contracture	**Alcohol**
Tattoo marks, jaundice	**Hepatitis B/C**
Metabolic syndrome	**NASH**
Xanthoma, xanthelasma, obstructive jaundice	**Primary biliary cirrhosis**
Skin hyperpigmentation, organomegaly, diabetes	**Hemochromatosis**
Emphysema and cirrhosis	**Alpha-1 antitrypsin deficiency**
Long-standing heart failure	**Cardiac cirrhosis**
Tender liver with absent abdominojugular reflux	**Budd–Chiari syndrome**
Arthritis, skin changes, nephritis	**Autoimmune**
Deforming arthritis on treatment	**Methotrexate induced**
Kayser–Fleischer (KF) ring on cornea	**Wilson's disease**

Q. What is hepatorenal syndrome?

Hepatorenal syndrome is a severe life-threatening complication occurring in cirrhotic patients with ascites, and it is characterized by the development of renal failure in the absence of any identifiable renal pathology. It is a functional disturbance in the renal function rather than a structural defect.

Q. What are the diagnostic criteria for hepatorenal syndrome?

Refer **Box 3.2**.

Box 3.2: Diagnostic criteria for hepatorenal syndrome (HRS).

All of the following must be present for the diagnosis of HRS:

- Cirrhosis with ascites
- Serum creatinine >1.5 mg/dL
- No improvement of serum creatinine (decrease to a level of 1.5 mg/dL or less) after at least 2 days of diuretic withdrawal and volume expansion with albumin
- Absence of shock
- No current or recent treatment with nephrotoxic drugs
- Absence of parenchymal kidney disease as indicated by proteinuria >500 mg/day, microhematuria (>50 red blood cells per high power field), and/or abnormal renal ultrasonography

Q. What are the types of HRS?

Refer **Table 3.14**.

Table 3.14: Types of hepatorenal syndromes (HRS).

Acute kidney injury (AKI) type of HRS (HRS-AKI) Type 1 hepatorenal syndrome	*Non-AKI type of HRS (HRS-NAKI) Type 2 hepatorenal syndrome*
• It is characterized by progressive oliguria, a rapid rise of the serum creatinine to above 2.5 mg/dL and has a very poor prognosis • Usually precipitated by spontaneous bacterial peritonitis • Without treatment, median survival is less than 1 month and almost all patients die within 10 weeks after the onset of renal failure	• It is characterized by a reduction in glomerular filtration, moderate and stable increase in serum creatinine (>1.5 mg/dL), but it is fairly stable and has a better prognosis than type 1 HRS • Usually occurs in patients with refractory ascites (resistant to diuretics) • Median survival is 3–6 months

Q. What are the precipitating factors of hepatorenal syndrome?

Refer **Box 3.3**.

Box 3.3: Precipitating factors for hepatorenal syndrome.

- Gastrointestinal bleeding
- Aggressive paracentesis
- Diuretic therapy
- Sepsis including spontaneous bacterial peritonitis
- Diarrhea

Q. What are the types of hepatic encephalopathy?

Refer **Figure 3.31**.

Q. What are the sites of portosystemic anastomosis?

Figure 3.32 shows sites of portosystemic anastomosis in cirrhosis.

Q. How do you classify portal hypertension?

Figure 3.33 shows the classification of portal hypertension according to site of vascular obstruction.

Q. What are the causes of hepatosplenomegaly/splenomegaly with ascites?

- Cirrhosis of liver with portal hypertension
- Lymphomas
- Systemic lupus erythematosus
- Disseminated tuberculosis
- Acute leukemias

Q. What is metabolic dysfunction-associated fatty liver disease?

Metabolic dysfunction-associated fatty liver disease (MAFLD) is a novel concept proposed in 2020 aiming to replace the term NAFLD (nonalcoholic fatty liver disease). Unlike NAFLD,

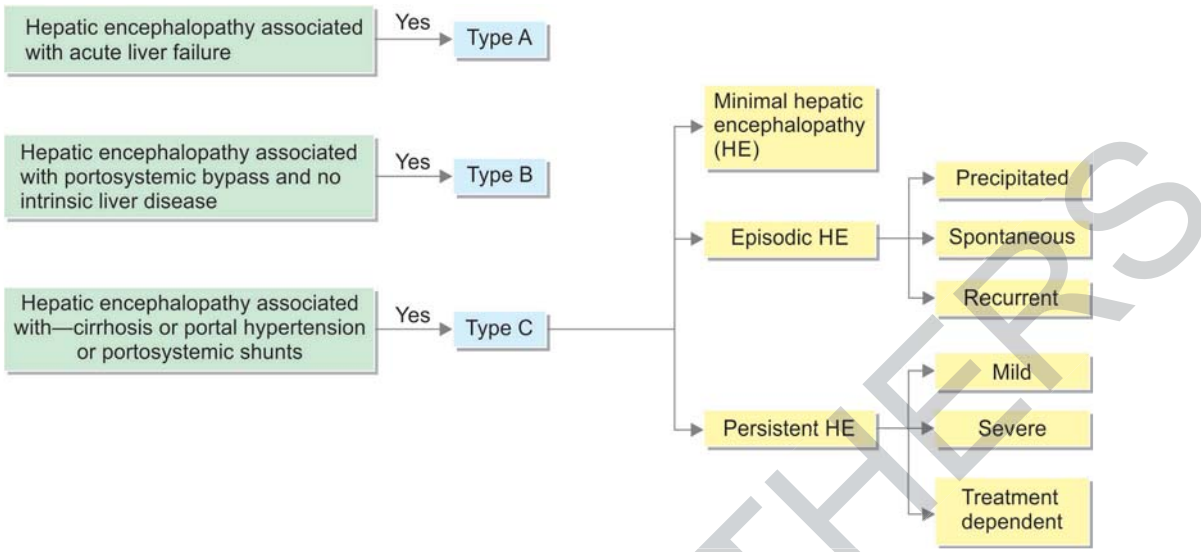


Fig. 3.31: Types of hepatic encephalopathy.

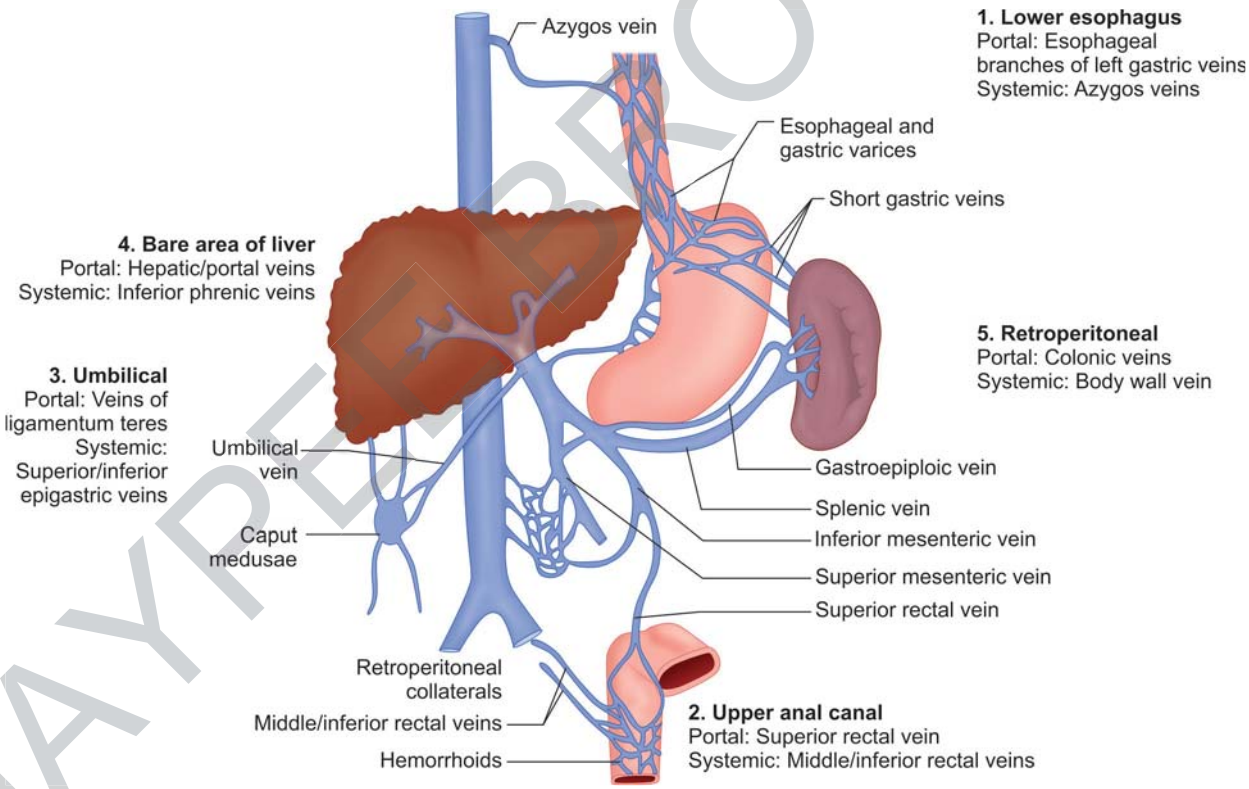


Fig. 3.32: Sites of portosystemic anastomosis in cirrhosis.

MAFLD does not require the exclusion of other etiologies of liver disease, such as excessive alcohol consumption or viral hepatitis **(Fig. 3.34)**.

MAFLD is diagnosed in patients when they have both hepatic steatosis and any of the following three metabolic conditions: overweight/obesity, diabetes mellitus, or evidence of metabolic dysregulation (MD) in lean individuals.

According to MAFLD definition, MD in this study was defined as the presence of at least two of the following criteria:

- Waist circumference ≥102 cm in men and 88 cm in women.
- Prediabetes [glycated hemoglobin (HbA1c) of 5.7–6.4%, or fasting plasma glucose (FPG) of 5.6–6.9 mmol/L, or 2-hour post-load glucose levels of 7.8–11.0 mmol/L].

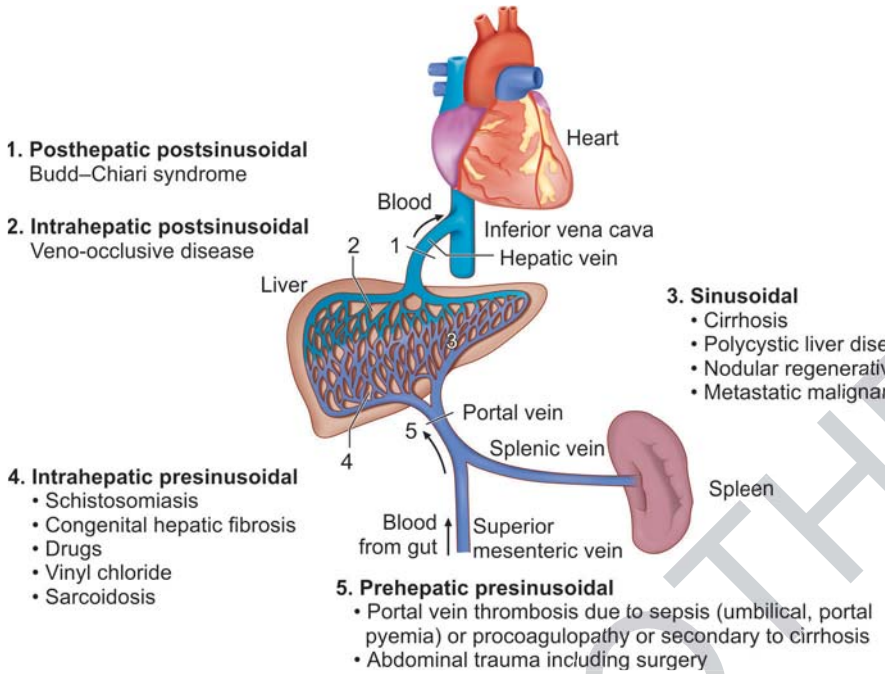


Fig. 3.33: Classification of portal hypertension according to site of vascular obstruction.

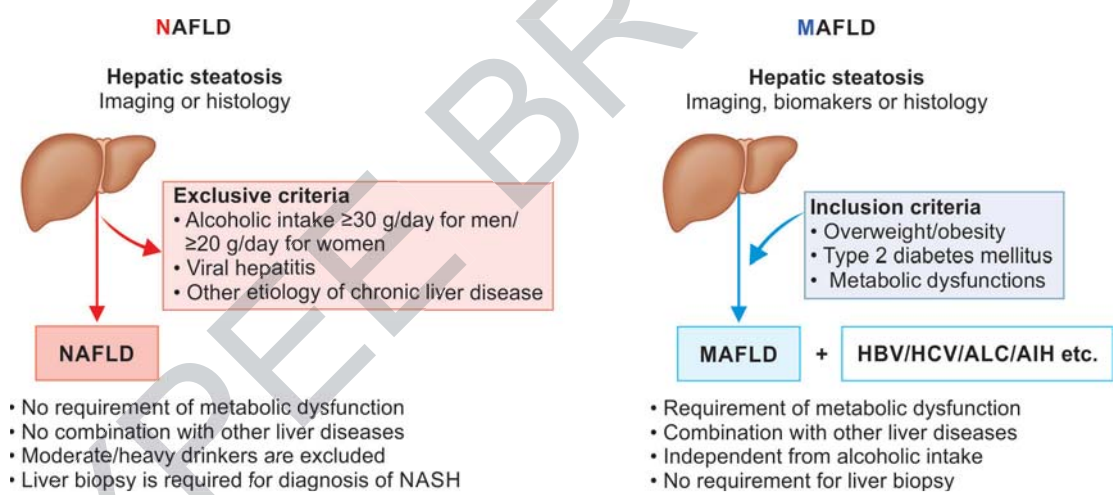


Fig. 3.34: Nonalcoholic fatty liver disease (NAFLD) versus metabolic (dysfunction)-associated fatty liver disease (MAFLD).

- Blood pressure ≥130/85 mm Hg or under antihypertension therapy.
- High-density lipoprotein cholesterol (HDLC) <1.0 mmol/L for males and <1.3 mmol/L for females.
- Triglyceride (TG) ≥1.70 mmol/L or specific drug treatment.
- Homeostasis model assessment-insulin resistance (HOMA-IR) score ≥2.5
- Hypersensitive C-reactive protein (hs-CRP) level >2 mg/L.

MAFLD must be evaluated as a multisystemic disease affecting many extrahepatic organs.

The disease burden extends beyond liver-related complications, underlining the importance of multidisciplinary screening and disease management.

Moreover, patients with MAFLD should also be examined for CVD and cardiovascular risk. Further, treatment of dyslipidemia, T2DM, and hypertension is recommended to decrease the risk of cardiovascular and kidney diseases.

Importantly, the high rate of co-existing CVD, CKD, OSA, hypothyroidism, osteoporosis, and PCOS indicates that MAFLD patients should be evaluated for these extrahepatic diseases.

Q. What is sinistral, or left-sided, portal hypertension?

Sinistral portal hypertension (SPH) is also known as splenoportal, left-sided, segmental, regional, localized, compartmental or lineal portal hypertension.

It is a rare entity, accounting for less than 5% of all patients with portal hypertension, and results from splenic vein thrombosis or occlusion, with patent extrahepatic portal vein.

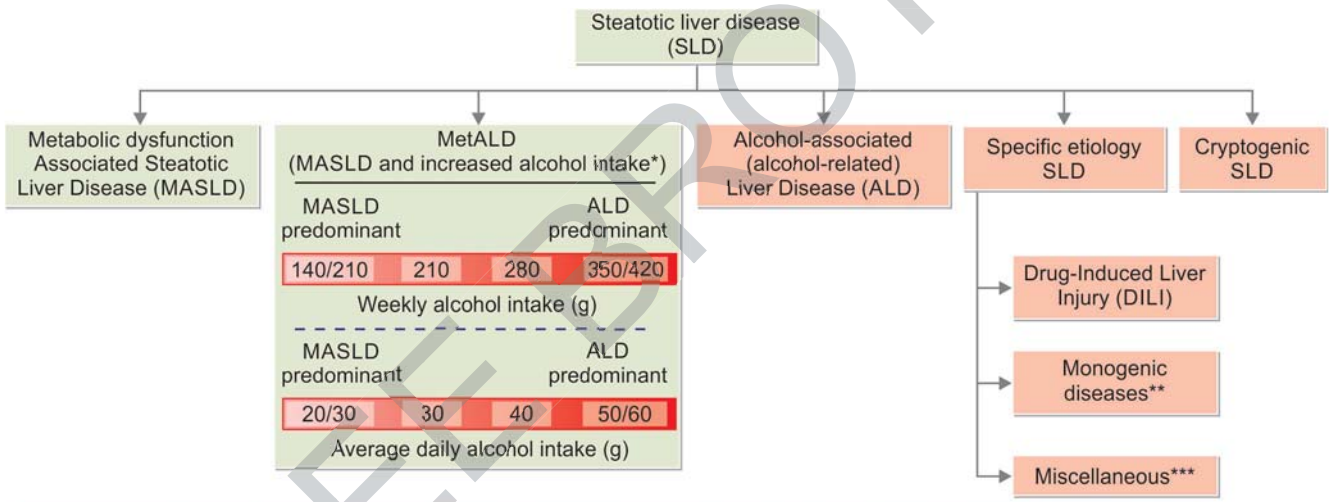
In fact, the name sinistral portal hypertension is a misnomer since portal pressure is usually within the normal range in these cases.

It is characterized by localized portal hypertension, most commonly due to obstruction of the splenic vein (SV) by a pancreatic pathology (acute or chronic pancreatitis, pancreatic pseudocysts, and malignancies) and/or subsequent surgery resulting in venous hypertension.

Q. What is steatotic liver disease (SLD)?

- Steatotic liver disease (SLD) is overarching term to encompass the various etiologies of steatosis.
- Non-alcoholic fatty liver disease (NAFLD) is metabolic dysfunction-associated steatotic liver disease (MASLD). MASLD encompasses patients who have hepatic steatosis and have at least one of five cardiometabolic risk factors.
- A new category, outside pure MASLD, termed MetALD (pronunciation: Met A-L-D) is used to describe those with MASLD who consume greater amounts of alcohol per week (140 g/week and 210 g/week for females and males respectively).
- Metabolic dysfunction-associated steatohepatitis (MASH) is the replacement term for NASH.

Steatotic Liver Disease Sub-classification



*Weekly intake 140–350 g female, 210–420 g male (average daily 20–50 g female, 30–60 g male)
**e.g., Lysosomal Acid Lipase Deficiency (LALD), Wilson disease, hypobetalipoproteinemia, inborn errors of metabolism
***e.g., Hepatitis C virus (HCV), malnutrition, celiac disease

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