Second Edition

Kundu's Practical Medicine

An overview of physical examination

As per the latest CBME Guidelines |
Competency Based Undergraduate Curriculum for the Indian Medical Graduate

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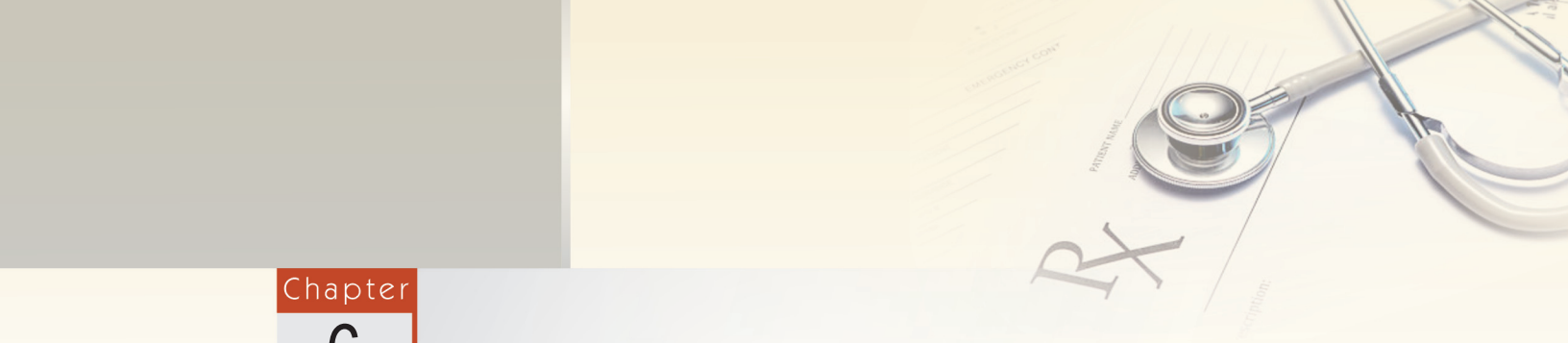

Chapter

6

Gastrointestinal (GI) and Hepatobiliary System

SCHEME OF SYSTEMIC EXAMINATION: AN OVERVIEW

I. Examination of upper GI tract: Lips, teeth, gum, cheek (buccal mucous membrane), tongue, tonsils, palate, breath, and oropharynx; oesophagus cannot be examined.

II. Examination of the abdomen

A. *Inspection:*

- Shape of abdomen and appearance of skin [striae, ulcer, sinus, pigmentation, puncture marks, bluish discolouration around umbilicus (Cullen's sign) and bluish-red discoloration of flanks in acute pancreatitis (Grey Turner's sign)]
- Flanks—full (in ascites) or not
- Any localised lump seen or not
- Venous prominence (examine in standing position of the patient after coughing)
- Movement of the abdomen (respiratory movements, peristalsis and pulsation)
- Umbilicus (discolouration, nodules, venous prominence, sinus, hernia, and granuloma in and around umbilicus)
- Hernial sites, groin and hairs (e.g., pubic hair).

B. *Palpation:*

- Superficial palpation: Surface temperature, tenderness, consistency, localised lump, pulsation, divarication of recti while rising from bed, fluid thrill, direction of venous flow, cough impulse at hernia orifices
- Deep palpation: Palpation of liver, spleen, kidneys, gall bladder, colon, testes (though remain outside of the abdomen), urinary bladder, aorta, any lump or mass, hernia sites, deep tender spots e.g., McBurney's point or epigastric point.

C. *Percussion:*

- General note of abdomen (in health, there is tympanitic note)
- Upper border of liver dullness (in health, in the right 5th ICS in right MCL)
- Shifting dullness (i.e., to diagnose ascites)
- Percussion over urinary bladder (if distended).

D. *Auscultation:*

- Bowel sounds (i.e., peristaltic sounds)

- Hepatic or splenic rub
- Venous hum
- Bruit
- Foetal heart sound (in pregnancy).

III. Per rectal examination

Q. Mention the common presentations (chief complaints and H/o present illness) in GI and hepatobiliary system.

The patients having complaints of GI and hepatobiliary system commonly present with:

1. Anorexia (loss of appetite), dyspepsia (medical term which signifies indigestion; it may be reflux-like, ulcer-like or dysmotility-like).
2. Dysphagia (difficulty in swallowing) or odynophagia (painful deglutition).
3. Nausea (feeling or desire to vomit), vomiting, hiccough.
4. Heartburn (a sense of hot, burning, retrosternal discomfort)—in gastro-oesophageal reflux disease (GERD), where the oesophageal mucosa is exposed to acidic gastroduodenal contents for a prolonged period.
5. Jaundice (e.g., yellow eyes) or yellow urine.
6. Pain abdomen.
7. Haematemesis, melaena, haematochezia, bleeding per rectum.
8. Flatulence and/or flatus (too much gaseous distension is due to aerophagia or air ingestion, malabsorption or lactase deficiency; aerophagia is a repetitive pattern of swallowing or ingesting air, and belching).
9. Swelling in the abdomen (collection of fluid or presence of a tumour).
10. Constipation, diarrhoea, dysentery, alteration of bowel habit (sometimes, point towards carcinoma of the rectum), or mucus in the stool.
11. *Others:*
    a. Fever (intestinal tuberculosis, amoebic liver abscess, abdominal lymphoma, subphrenic abscess, spontaneous bacterial peritonitis, cholangitis).
    b. Loss of weight (intra-abdominal malignancy, tuberculosis, or lymphoma).
    c. Oral ulcers or halitosis (i.e., bad breath).
    d. Confusion, drowsiness, or inversion of sleep rhythm (hepatic encephalopathy i.e., neuropsychiatric manifestations from cirrhosis of liver).
    e. Low urine output or oliguria from hepato-renal syndrome (i.e., developing from cirrhosis of liver).
    f. Pedal oedema (with features of malnutrition and vitamin deficiency from severe anorexia or malabsorption).

Q. Queries in past, family, personal and treatment history in GI system.

Past history	*Family history*	*Personal history*	*Treatment history*
1. Jaundice 2. Blood transfusion (e.g., viral hepatitis B and C), recent tattooing	1. Any member of family suffering from similar type of illness (e.g., Wilson's disease)? 2. Family H/o tuber-	1. Dose of alcohol consumption (type, quantity with duration) 2. 'Substance abuse',	1. Drug history (rifampicin, INH, oral contraceptive pills, NSAID, or any herbal remedies)—NSAID

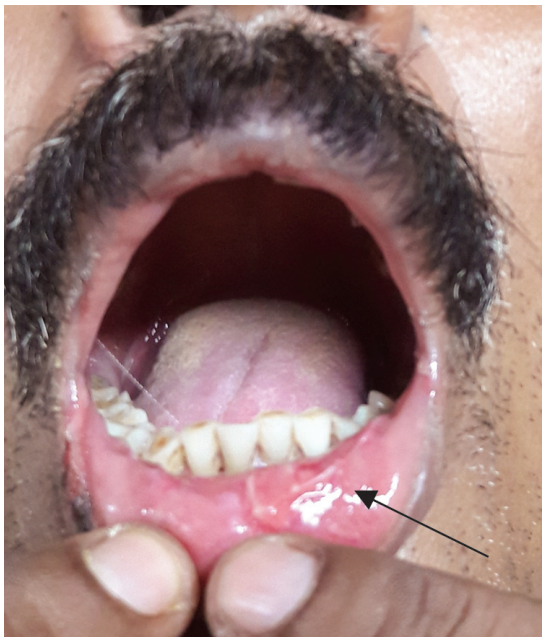

Fig. 6.1: Ulcers inside mouth and over lower lip as a result of cytotoxic drug therapy

Q. How tongue helps in diagnosing diseases in clinical medicine?

Previously, tongue was thought to be 'mirror of GI tract' only but it is now regarded as mirror of dysfunction of all systems in human body. Now examine tongue in the following ways:

1. Ask the patient to protrude the tongue: The patient cannot protrude it in 'tongue tie' (or ankyloglossia), malignancy of the tongue and bilateral XIIth cranial nerve paralysis.
2. Tremor (e.g., in anxiety neurosis, thyrotoxicosis) in tongue can be seen on protrusion, while fasciculation (e.g., in motor neuron disease or MND) is seen while tongue rests on floor of the mouth.
3. Large tongue is common in acromegaly, myxoedema, cretinism and Down's syndrome (macroglossia), while small tongue (microglossia) is seen in cerebral diplegia, MND, bulbar and pseudobulbar palsy.
4. Dry (dehydration, xerostomia) and moist (sialorrhoea, heavy metal poisoning) tongue.
5. Yellow (jaundice), pale (severe anaemia), blue (cyanosis), bluish-red (polycythemia), magenta-coloured (riboflavin deficiency, Fig. 6.2), black (ingestion of bismuth, charcoal, or Addison's disease), 'furred' (heavy smoking, sore throat), 'bald' (total loss of papillae is common in pernicious anaemia, tropical sprue) tongue, and candidiasis of tongue (common in immunodeficiency, Fig. 6.3).
6. Spastic (pseudobulbar palsy) and flaccid (bulbar palsy) tongue.
7. Raw-beefy (red, swollen and painful tongue in nicotinic acid and vitamin B_{12} deficiency), angry-looking (central coating with pointed tip in typhoid fever), geographical (denuded and migrating red patches due to loss of papillae, and of no

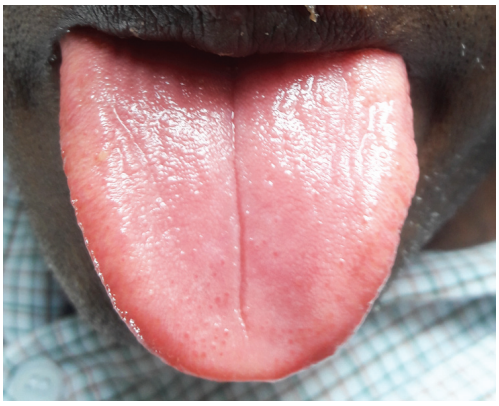

Fig. 6.2: Magenta-coloured tongue in riboflavin deficiency

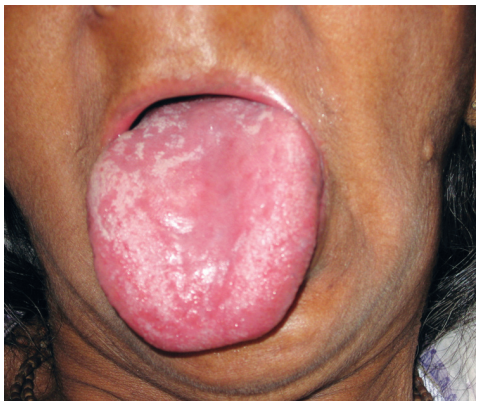

Fig. 6.3: Candidiasis of tongue in a patient of diabetes mellitus

clinical significance, Fig. 6.4), and strawberry and raspberry (scarlet fever) tongue; mushroom-like growth (malignancy of the tongue, Fig. 6.5), and ulcers (aphthous ulcer, Behçet's disease) in the tongue. Haematoma in the tongue may be seen in accidental tongue bite or coagulation disorders (Fig. 6.6).

8. Deviation of tongue while protruding and loss of power of tongue are seen in XIIth cranial nerve paralysis. Loss of taste sensation is found in VIIth (anterior two-thirds) and IXth (posterior one-third) cranial nerve paralysis.

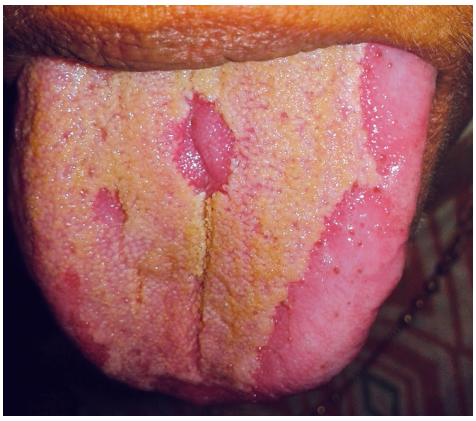

Fig. 6.4: Geographical tongue

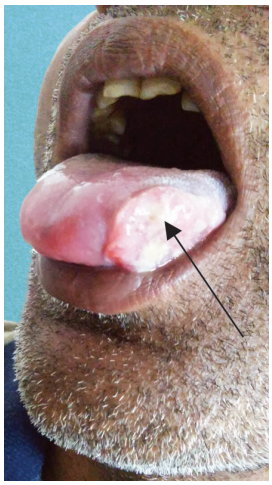

Fig. 6.5: Squamous cell carcinoma of the tongue

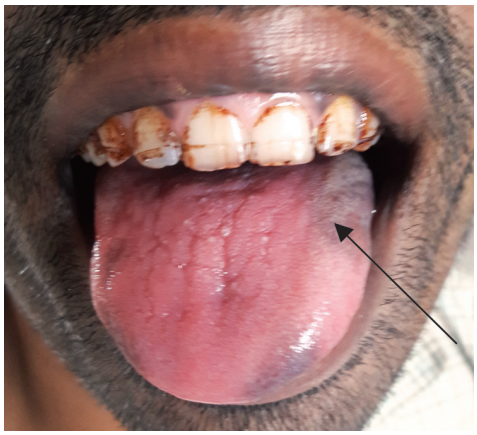

Fig. 6.6: Haematoma in the tongue in coagulation disorders; blood-stained teeth are evident

Q. Do you know the different types of dysphagia?

Difficulty in swallowing is of two types:

- Oropharyngeal (due to neuromuscular disorder): Bulbar and pseudobulbar palsy, myasthenia gravis.
- Oesophageal: Malignancy of oesophagus, peptic stricture, achalasia cardia, diffuse oesophageal spasm, mediastinal growth mechanically causing obstruction in oesophagus.

Q. Causes of persistent vomiting.

Nausea and vomiting (protracted) are commonly encountered in:

- Acute viral hepatitis, increased intracranial pressure (ICP), acute labyrinthitis, Ménière's disease, migraine, meningitis.
- CRF or uraemia, diabetic ketoacidosis, AMI, chronic alcoholism, hyperemesis gravidarum, drug-induced (e.g., digoxin), functional vomiting (psychogenic).
- Surgical: Acute pancreatitis, acute cholecystitis, intestinal obstruction, colics.

NB:

1. 'Pyloric stenosis' (or gastric outlet obstruction) is an intestinal obstruction at pylorus of stomach where the patients complain of projectile vomiting which is copious in amount, offensive and contains food taken 1–2 days back. It is diagnosed by a diffuse swelling in upper abdomen, visible peristalsis from left to right hypochondrium and succussion splash (splashing sound audible after shaking the patient's abdomen).
2. Vomiting of raised ICP occurs in the morning. It occurs suddenly without preceding nausea (i.e., projectile vomiting).

Q. 'Red flag' or 'alarming signs' in gastroenterology are:

Patients present with these symptoms/signs should immediately be investigated, especially if ≥ 55 years:

- Loss of weight (progressive)
- Anorexia (profound)
- Persistent dysphagia
- Protracted jaundice
- Haemaemesis/melaena
- Chronic diarrhoea.

Q. What is 'peptic ulcer disease (PUD)'?

PUD is a general term for ulcers that occur in the lower part of oesophagus, stomach (gastric ulcer) or duodenum (duodenal ulcer).

The aetiology of PUD is *Helicobacter pylori*, NSAID (non-steroidal anti-inflammatory drugs, e.g., aspirin), smoking, stress ulcer (e.g., Curling's ulcer from severe burn or Cushing's ulcer from raised ICP), gastrinomas (Zollinger-Ellison syndrome), or reflux of bile and intestinal contents into stomach.

Helicobacter pylori is a spiral, Gram-negative, flagellated, urease-producing bacillus which is transmitted through oral-oral or faecal-oral (enters through mouth by ingestion

hepatitis is caused by hepatitis A, B ± D, and E virus, whereas hepatitis B, C and D are notorious for chronicity. Hepatitis C is the commonest cause of post-transfusion hepatitis. Hepatitis C virus very rarely produces acute hepatitis.

Route of entry: By faecal-oral route: Hepatitis A and E, parenteral route: Hepatitis B, C, and D.

Q. How a patient of acute viral hepatitis presents (enumerate symptoms and signs)?

The patient may present in any of the three stages like prodromal stage (fever, anorexia, nausea, vomiting, distaste for smoking, urticaria and arthralgia (especially, hepatitis B virus), myalgia, headache, malaise, discomfort in right hypochondrium, dark urine), icteric stage (jaundice*, mild/soft/tender hepatomegaly, splenomegaly in 10% patients, dark urine with pale stool—Fig. 6.7) or recovery stage (urine and eyes become less yellow, stool becomes yellow and there is return of appetite). The course of the disease is usually 2–4 weeks.

*Read 'Jaundice' from section of 'General examination'

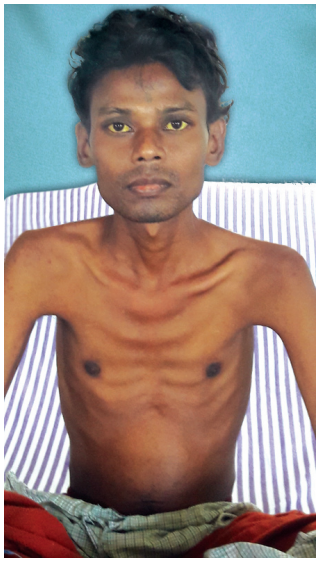

Fig. 6.7: Patient suffering from acute viral hepatitis with jaundice

Q. Common liver function tests done in clinical practice.

- Serum bilirubin (conjugated and unconjugated fractions)
- Aminotransferases (transaminases)—SGPT (serum glutamic pyruvic transaminase) is known as ALT (alanine aminotransferase), and SGOT (serum glutamic oxaloacetic transaminase) is known as AST (aspartate aminotransferase)
- Alkaline phosphatase
- Serum albumin and globulin
- Prothrombin time
- Gamma-glutamyl transpeptidase (GGT).

NB: Severity assessment of hepatic injury is best assessed by prothrombin time and partly by serum albumin.

Q. What is cirrhosis of liver?

Cirrhosis, by itself a pathological term, is the chronic and diffuse involvement of hepatic parenchyma of varied aetiology, and is clinically manifested by features of portal hypertension and hepatocellular failure in varying combination, and pathologically by irreversible necrosis, extensive fibrosis, and regenerative nodule formation in such a way that the normal hepatic architecture is totally lost. Cirrhosis is a classical example of chronic liver disease (CLD) and the other common CLD is chronic hepatitis (Fig. 6.8).

Aetiology of cirrhosis is alcoholic, biliary, cryptogenic (i.e., no cause found), cardiac, Wilson's disease, haemochromatosis, galactosaemia, Indian childhood cirrhosis, Budd-Chiari syndrome, drug-induced (e.g., methotrexate) and non-alcoholic steatohepatitis (NASH).

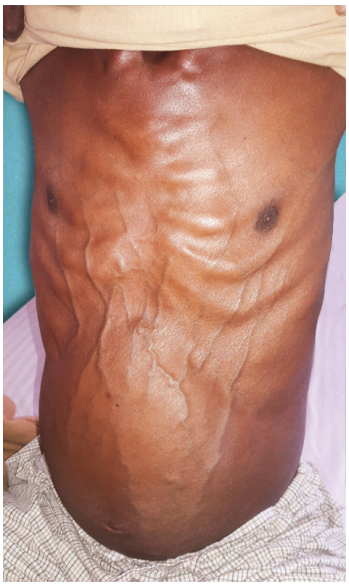

Fig. 6.8: Classical cirrhosis of liver—prominent and tortuous veins with ascites point towards chronic liver disease

Q. How to diagnose cirrhosis at the bedside?

A patient of cirrhosis will have features of both hepatocellular failure (i.e., liver failure) and portal hypertension. Both the subsets have some specific features which help in bedside diagnosis. The features of:

- Hepatocellular failure (i.e., hepatic failure)
  - Jaundice
  - Ascites and bipedal oedema
  - Fever, loss of muscle mass
  - Skin changes: Spider naevi* (Fig. 6.9), palmar erythema* (Fig. 6.10), white nails, diffuse pigmentation
  - Endocrine changes*: Gynaecomastia, testicular atrophy, loss of axillary and pubic hair, breast atrophy and menstrual irregularities, loss of libido (i.e., low sexual desire)
  - Fetor hepaticus: Sweetish-faecal smell in breath

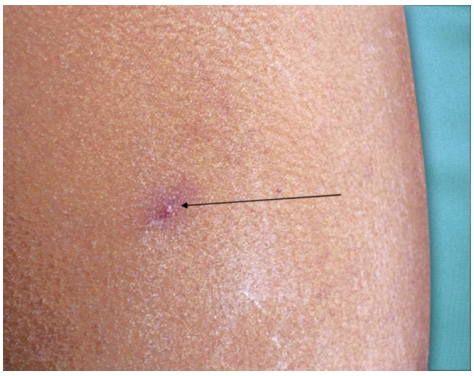

Fig. 6.9: Spider naevi over the upper part of back of chest—a hallmark sign of hepatocellular failure

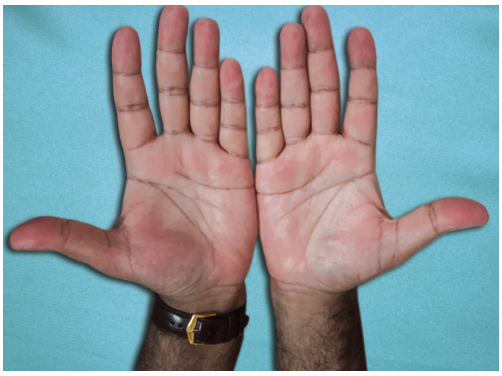

Fig. 6.10: Palmar erythema in a patient of cirrhosis of liver

- Hepatic encephalopathy (i.e., the neuropsychiatric syndrome): Inversion of sleep rhythm, confusion, flapping tremor (the toxins like NH_3, methionine, octopamine, etc. go directly to brain via collateral veins by-passing liver)
- Bleeding manifestations: Petechiae, purpura, ecchymoses (due to low prothrombin level)
- Circulatory changes: Capillary pulsation, clubbing, cyanosis.

*All endocrine changes along with spider naevi and palmar erythema are due to 'hyperoestrogenaemia'.

❍ Portal hypertension
- Evidences of porto-systemic anastomosis (i.e., collateral circulation), that means engorged and tortuous veins seen at:
  - Lower end of oesophagus: Oesophageal varix (their rupture leads to haematemesis and melaena)
  - Around the umbilicus or over epigastrium (Fig. 6.11): Known as 'caput medusae' (looks like Greek Goddess Medusae's hair) when present around the umbilicus
  - Rectum and anal canal: Formation of haemorrhoids
- Splenomegaly (cardinal feature at bedside)
- Ascites.

NB: Feature common to hepatocellular failure and portal hypertension is ascites. Liver failure → hypoproteinaemia → low oncotic pressure → formation of ascites; portal hypertension only localises the fluid within the peritoneal cavity.

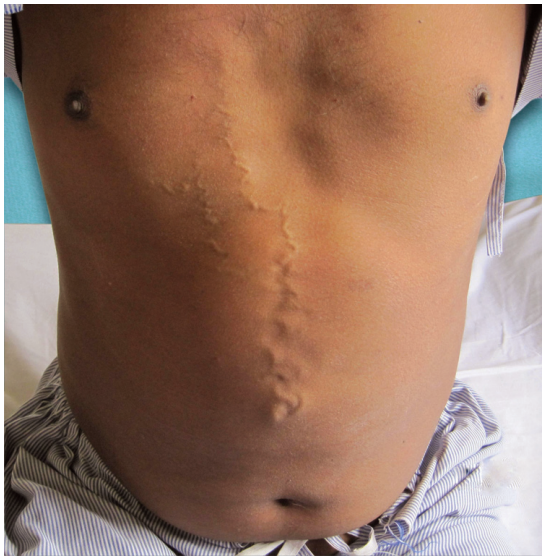

Fig. 6.11: Portal hypertension—venous collaterals are seen

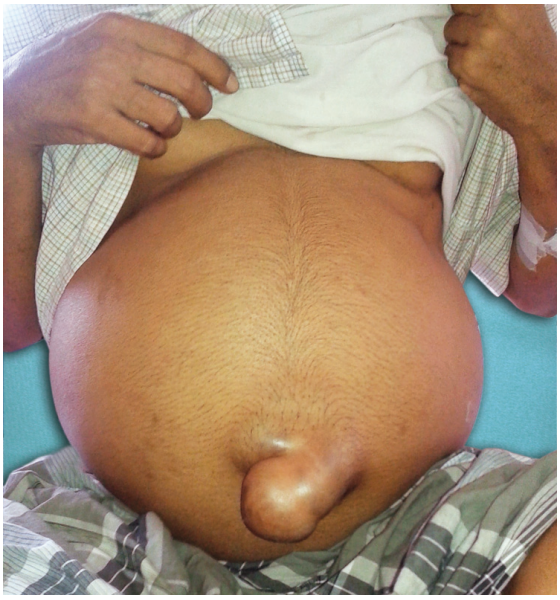

Fig. 6.12: Huge ascites with umbilical hernia

A huge ascites may have umbilical hernia associated with (Fig. 6.12).

Decompensated cirrhosis: Cirrhosis with one or more features of ascites, jaundice, GI bleeding and hepatic encephalopathy.

Compensated cirrhosis: Asymptomatic cirrhosis without those features.

Q. What is portal hypertension and its common causes?

As described in the previous question, portal hypertension is clinically diagnosed by venous collaterals in the abdomen, ascites and splenomegaly. To diagnose cirrhosis of liver in a patient, features of portal hypertension should be present along with features of hepatocellular failure. Portal hypertension is defined as:

- *Definition:* Normal pressure in the portal vein is 5–8 mm of Hg or 10–15 cm of saline. Portal pressure >30 cm of saline or >12 mm of Hg is known as portal hypertension.
- *Causes:* Cirrhosis of liver (commonest), Budd-Chiari syndrome (hepatic venous obstruction), portal vein thrombosis (e.g., umbilical sepsis in neonates, hyperviscosity syndrome), non-cirrhotic portal fibrosis (arsenic toxicity, schistosomiasis), veno-occlusive disease (by Jamaica bush tea).

Q. What are the causes of acute (fulminant) hepatic failure?

Acute hepatic failure is a fatal condition. It is caused by:

- Acute viral hepatitis (most common cause)
- Drug-induced (rifampicin, INH, paracetamol overdose, halothane), copper sulphate poisoning
- Shock and multi-organ failure
- Sepsis
- Acute fatty liver of pregnancy

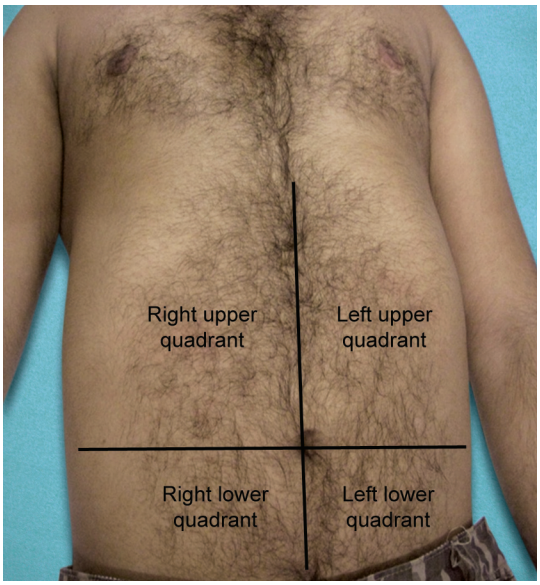

Fig. 6.16: Different quadrants of abdomen for the purpose of clinical examination

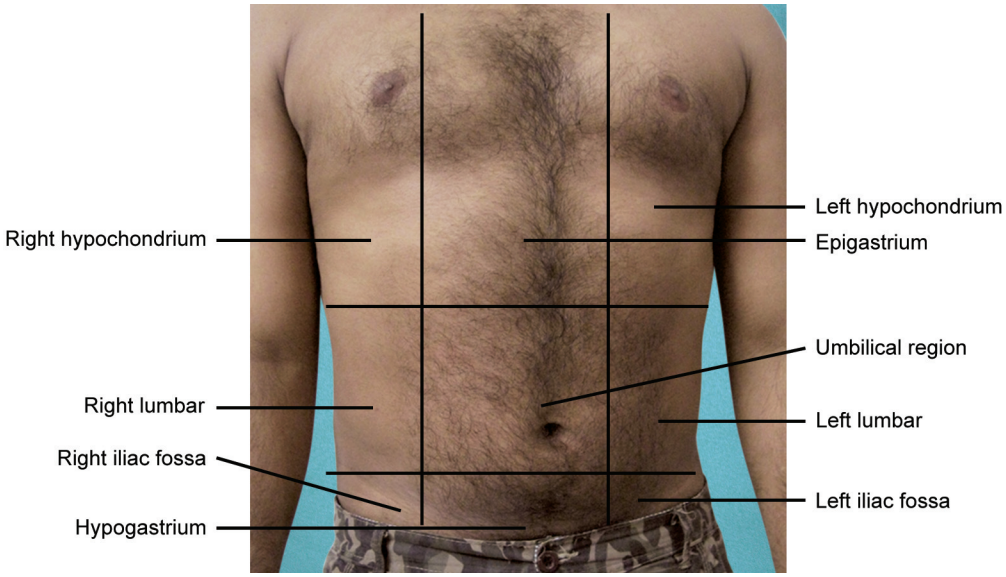

Fig. 6.17: Various (nine) regions of abdomen with anatomical correlations

Q. What are the significance/importance of dilated veins over abdomen/chest (Fig. 6.18)?

Dilated veins over abdomen is basically found in portal hypertension, SVC and IVC obstruction. Their presence in specific region of abdomen along with the direction of blood flow clinch the clinical diagnosis very promptly.

A. *Around umbilicus (caput medusae) or over epigastrium:* Portal hypertension where the direction of flow of blood is 'away from the umbilicus'; a venous hum (similar

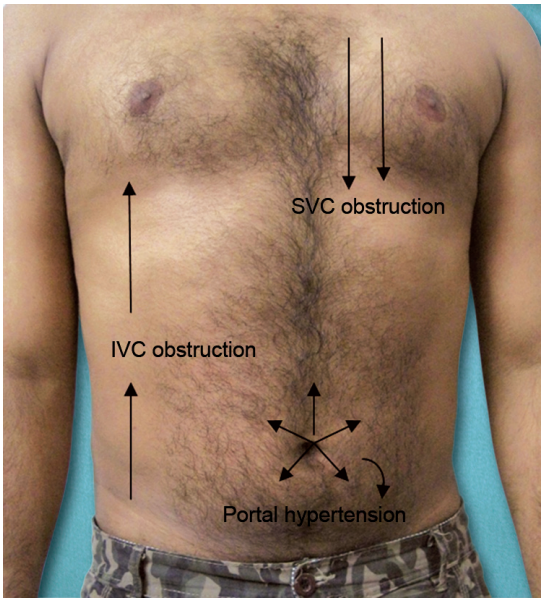

Fig. 6.18: Dilated abdominal wall veins with direction of blood flow (SVC—superior vena cava, IVC—inferior vena cava)

sound like bruit in arteries) may be audible over dilated abdominal wall veins around umbilicus.

B. *Over abdomen, lateral chest wall and back:* Inferior vena caval (IVC) obstruction where the direction of flow of blood is 'below upwards' i.e., from hypogastrium towards epigastrium.

C. *Over chest and upper arm:* Superior vena caval (SVC) obstruction where the direction of flow of blood is 'above downwards' i.e., from upper chest towards epigastrium.

NB: In health, the flow of blood is away from the umbilicus but one cannot see the venous prominences.

Q. How to palpate liver, spleen and gall bladder?

The patient should lie flat comfortably in bed. Proper light should be there for examination. Explain briefly to the patient what you are going to do. To gain confidence of the patient, a gentle palpation with proper care of the painful part is necessary. The prerequisites before palpation of abdominal viscera are (Fig. 6.19):

1. Stand on the right side of the patient as commonly we are right-handed (a left-handed clinician may stand on the left side of the patient for better palpation).
2. Hands of the patient should lie by his/her side (to prevent undue obliquity).
3. Flex the thighs (to relax the muscles of abdomen).
4. Turn the patient's face to opposite side and ask him/her to breathe deeply but regularly with open mouth.
5. Expose the abdomen from xiphisternum to just above the inguinal ligament.

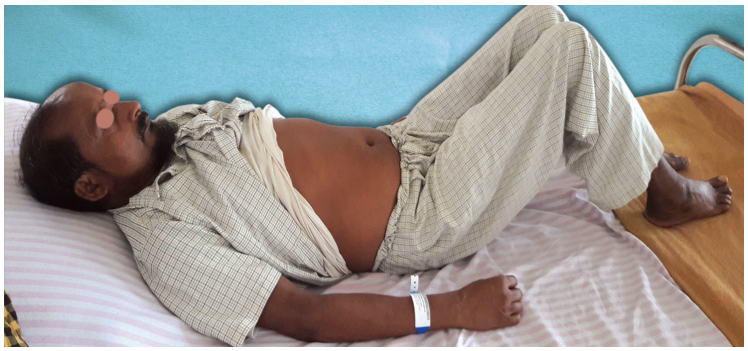

Fig. 6.19: Proper position of the patient before abdominal examination while the clinician will stand on the right side of the patient

Palpation of:

A. *Liver:* Stand on the right side of the supine patient. Place your right palm over the right iliac fossa (parallel to arbitrary lower border of liver, and lateral to right rectus abdominis muscle) and press the hand inwards and upwards. The liver can be felt at the height of inspiration, if enlarged, when the palpating hand gradually goes upwards towards right hypochondrium (conventional method). Now, try to palpate the left lobe of liver by moving the palpating hand from right hypochondrium to midline.

There are three methods of palpation of liver:

- *Conventional method* (described above; mostly practised in India) (Fig. 6.20).
- *Preferred method:* Place both hands side by side flat over right hypochondrium lateral to rectus abdominis muscle with fingers pointing upwards. If any resistance is felt (i.e., due to hepatomegaly), move the hands downwards till the resistance disappears. Now confirm the lower border of liver by asking the patient to inspire and moving the hands upwards and inwards. This method is preferred by rest of the world (Fig. 6.21).
- *Alternative method:* It is just like conventional method but the fingers of right hand are placed in the right iliac fossa directing upwards. This method is less sensitive than other two methods and thus not practised widely (Fig. 6.22).

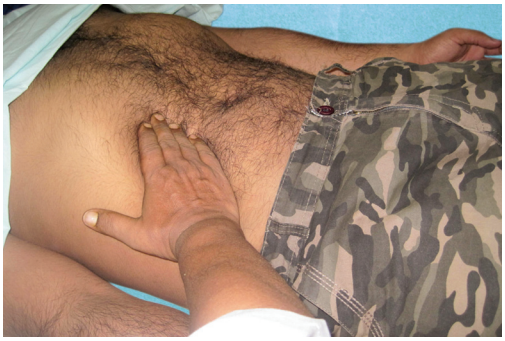

Fig. 6.20: Palpation of liver (conventional method)

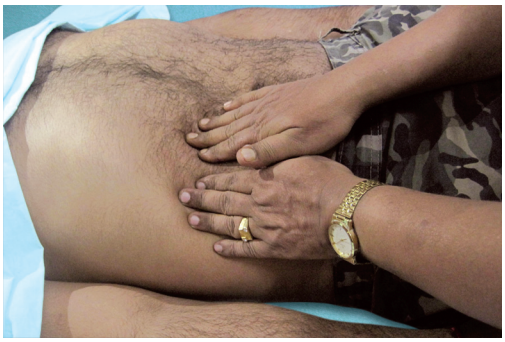

Fig. 6.21: Palpation of liver (preferred method)

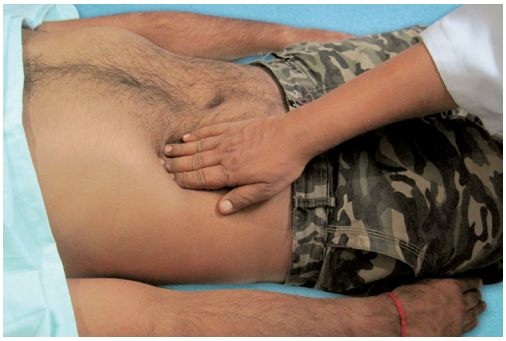

Fig. 6.22: Palpation of liver (alternative method)

In hepatomegaly, the points noted are:

- Degree of enlargement: The anterior liver span is 12–15 cm, measured from upper (determined by percussion) to lower border of liver at right MCL. It can also be expressed by distance (centimetres/inches) between right costal margin and lower border of liver at right MCL.
- Consistency: Soft (CCF) , firm (cirrhosis), hard (malignancy).
- Tenderness: Tender (viral hepatitis) or non-tender (fatty liver, cirrhosis).
- Surface: Smooth (CCF) or irregular (cirrhosis or malignancy).
- Margin or border: Sharp (cirrhosis) or rounded (fatty liver, CCF).
- Whether the left lobe is enlarged (e.g., amoebic liver abscess) or not.
- Any pulsation present (CCF) or not. Pulsatile liver (by placing right palm over enlarged liver and left palm over the back) may be felt in functional tricuspid incomptence (i.e., from CCF).
- Upper border of liver dullness: Percuss the right chest from above downwards. The upper border of liver dullness lies in the right 5th ICS in right MCL.
- Any hepatic bruit audible (hepatoma, haemangioma of liver), or hepatic rub palpable (after liver biopsy, metastatic carcinoma of liver)?

NB: It is very important to learn the art of palpation of liver and spleen (see below) from the initial days of clinical training.

B. ***Spleen:*** Stand on the right side of the supine patient and ask the patient to breathe deeply. Place your left palm firmly over left costal margin posterolaterally, and press it forward and medially. Start palpation with your right hand from right iliac fossa and proceed towards the left hypochondrium in search of palpable spleen (Fig. 6.23). Spleen becomes palpable when it is 2–3 times more than its normal size.

If spleen is not palpable in supine position, turn the patient in right lateral decubitus and palpate the spleen (Short's manoeuvre) (Fig. 6.24). If spleen is still not palpable, you can palpate it by standing on the left side of the patient with hooked fingers of left hand placed below left costal margin (Fig. 6.25).

In splenomegaly, the points noted are:

- Degree of enlargement: Spleen usually enlarges from left hypochondrium towards right iliac fossa (Fig. 6.26). The measurement is taken in centimetres/inches along

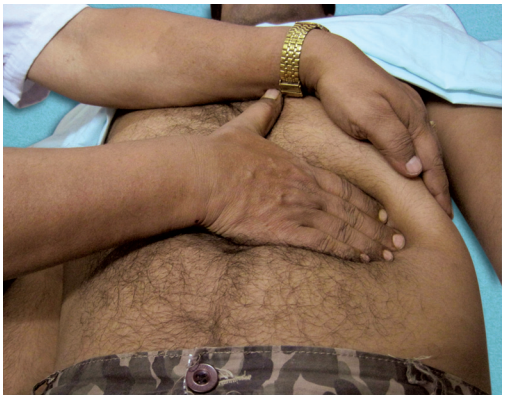

Fig. 6.23: Palpation of spleen

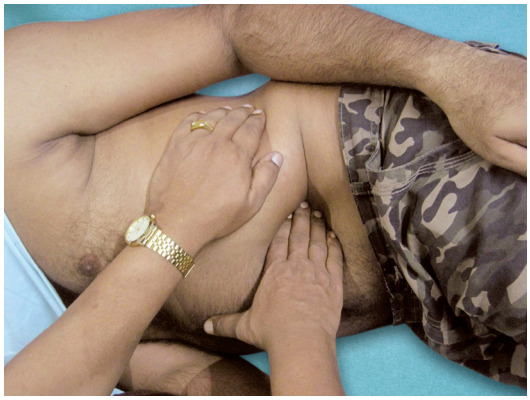

Fig. 6.24: Palpation of spleen in right lateral position

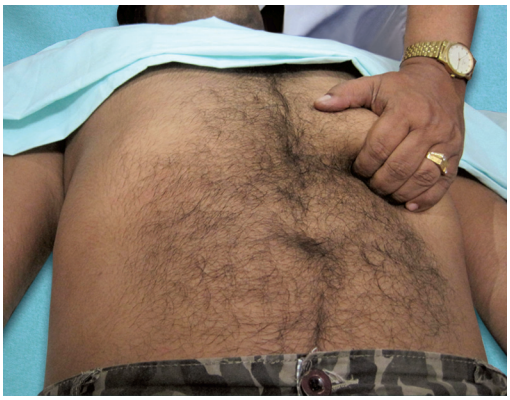

Fig. 6.25: Palpation of spleen by hooking method done from left side of the patient

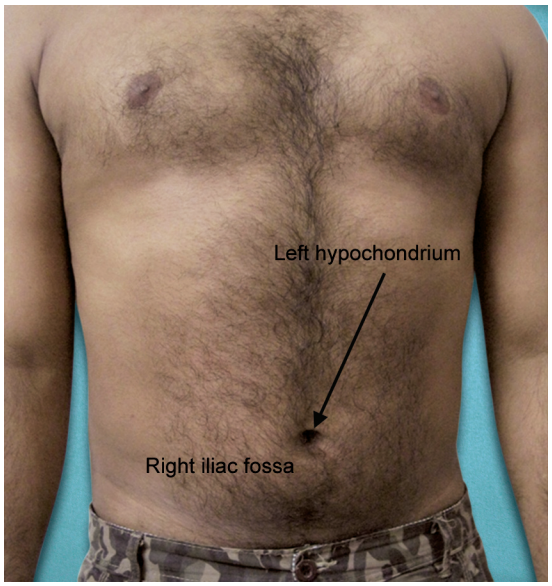

Fig. 6.26: Direction of splenic enlargement from left hypochondrium towards right iliac fossa

splenic axis by joining two points i.e., above where left costal margin is crossed by left MCL, and below up to the furthest apart point of spleen (Note: Left kidney enlarges towards left iliac fossa).

In health, spleen lies behind and below the left 9th, 10th and 11th rib, with its long axis along the line of 10th rib.

- Splenic notch: On its anterior border and very characteristic of splenic enlargement (helpful to differentiate kidney lump).
- Margin: Usually sharp.
- Consistency: Soft (acute malaria or acute kala-azar), firm (cirrhosis of liver), hard (chronic myeloid leukaemia/CML).
- Tenderness: Tender (SBE, infectious mononucleosis) or non-tender.
- Surface: Smooth (CML) or irregular (splenic cysts).

Q. Different methods of palpation of abdomen.

Usually there are four methods of palpation of abdominal viscera. They are:

- *Conventional method (classical method):* Palpation of liver, gall bladder.
- *Hooking method (by hooked fingers):* Classically used in splenic palpation.
- *Bimanual method (using both hands):* Classically used for palpation of kidneys, or abdominal palpation in an obese or poorly relaxed patient*.
- *Dipping method (a sharp tap is given over abdomen by flexing the metacarpophalangeal joints of right hand suddenly):* Palpation of abdominal viscera in the presence of ascites.

*Palpation by using two hands, placing the left hand over the right hand, is necessary in patients who are obese, muscular or with poorly relaxed abdomen (Fig. 6.27).

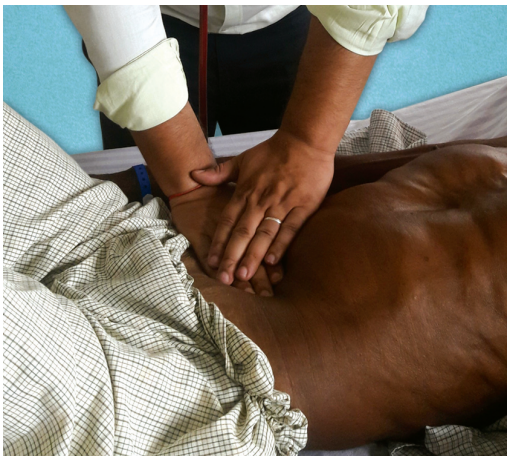

Fig. 6.27: Bimanual palpation of abdomen in an obese, muscular or poorly relaxed patient when the left hand is placed over the right hand

Q. Distension of the abdomen due to accumulation of fluid—causes behind?

Accumulation of fluid in abdomen may be due to collection of free fluid (ascites) or cystic fluid. Ascites is commonly due to cirrhosis of liver or tuberculous/malignant peritonitis. Cystic intra-abdominal swellings are due to distended urinary bladder, ovarian cyst, mesenteric cyst, pseudopancreatic cyst, hydatid cyst and polycystic kidney. Diagnosis of an abdominal lump (whether solid or cystic) is done by its site, size, margin, surface, consistency, movement with respiration, mobility, pulsation, or by the findings like whether it is bimanually palpable and ballottable.

Q. How to differentiate ascites from a cystic swelling?

There are two very sensitive methods for their diagnosis in clinical medicine. They are:

- *Fluid thrill: Fluid thrill is present both in ascites and encysted fluid.* For its presence, in case of ascites, fluid must remain under tension and should be large in volume (say, 2 litres). Tap or flick either flank of abdomen in a supine patient, while feel for a shock-like wave in other flank in case of presence of fluid thrill. Place ulnar border of one hand of the patient in the abdomen in the midline to prevent transmission of impulse through abdominal parietes (Fig. 6.28).

- Flicking, stroking or gentle massage of anterior abdominal wall
- Drinking 1–2 glasses of water at a time
- Pouring a few drops of ether over the abdomen.

The site of obstruction in intestine can be obtained by analysing peristaltic waves like:

1. *Stomach:* Waves are golf-ball like, and moves from left to right hypochondrium; 2–3 waves are seen at a time. Commonly it is due to pyloric stenosis (or gastric outlet obstruction).
2. *Small intestine:* Seen as 'ladder pattern' (zigzag fashion) in periumbilical region; 10–12 waves formation/minute.
3. *Large intestine:* In obstruction of transverse colon, waves move from right to left hypochondrium. Waves are seen in right or left flank in obstruction of ascending and descending colon respectively. Waves are a bit larger i.e., cricket-ball like.

Decoding Abbreviations

IBD	Inflammatory bowel disease
IBS	Irritable bowel syndrome
SVC	Superior vena cava
IVC	Inferior vena cava.