FINAL MBBS · GENERAL MEDICINE · LONG CASE

A E T C M

Chronic Liver Disease Case Sheet

Ascites · Portal Hypertension — a complete abdominal case, from history to viva

A fully worked case sheet of decompensated alcohol-related cirrhosis with large-volume ascites, portal hypertension and hepatic encephalopathy, written the way a final-year MBBS student should record and present it in the university examination — with step-by-step case taking and the reasoning behind every question asked and every sign elicited.

Case-taking steps › | History of presenting illness › | Alcohol & viral history › | Stigmata of liver disease ›
Abdominal inspection › | Liver & spleen palpation › | Tests for ascites › | Signs of portal hypertension ›
Hepatic encephalopathy › | Final diagnosis & Child–Pugh › | Differential diagnosis › | LFT & viral markers ›
Ascitic fluid & SAAG › | USG abdomen › | Endoscopy & varices › | Ascites management › | Variceal bleed › | SBP · HE · HRS ›
Model presentation › | Viva questions ›

TAP A TOPIC TO JUMP TO IT

HISTORY › alcohol · viral · bleed · confusion

EXAMINATION › stigmata · ascites · spleen

INVESTIGATIONS › SAAG · USG · endoscopy

MANAGEMENT › LVP · β-blocker · lactulose

SEE THE SIGNS ONLINE — TAP TO OPEN IMAGE / SCAN / VIDEO

Ascites | Caput medusae | Spider naevi | Palmar erythema | Asterixis (video) | Shifting dullness (video)
Spleen palpation (video) | Cirrhosis USG | Gross ascites USG | CT cirrhosis | Varices endoscopy | All signs — visual atlas ›

AETCM
Academy for Emergency, Trauma & Critical care Medicine

By

Dr. Gireesh Kumar K.P.

WATCH THE VIDEOS · TAP TO PLAY ON YOUTUBE

► Abdominal examination 1 | ► Abdominal examination 2 | ► Ultrasound (USG) | ► USG abdomen | ► Portal hypertension

For final-year MBBS students, interns and teachers of clinical medicine

Chronic Liver Disease Case Sheet

Ascites and portal hypertension — a complete abdominal long case for final MBBS

Dr. Gireesh Kumar K.P.

Academy for Emergency, Trauma & Critical care Medicine (AETCM) · First edition · November 2026

How to use this case sheet

Chronic liver disease (CLD) with ascites and portal hypertension is the commonest **abdominal long case** in the final MBBS General Medicine examination. It tests whether a student can take a precise **alcohol, viral and drug history**, find and **explain** the stigmata of liver disease, **demonstrate ascites** in a methodical order, recognise the signs of **portal hypertension** and of **decompensation** (bleeding, encephalopathy, infection, kidney injury), interpret **ascitic fluid, SAAG and ultrasound**, and plan treatment. This booklet gives one fully worked case exactly as it should be written in the case record and presented at the bedside — together with **step-by-step instructions for taking the case**.

Each section has three layers: the **case record** itself (what you write), **green boxes** that summarise and interpret (what you conclude), and **blue boxes** with examiner's tips (what you are judged on). Tables turn the single case into a revision of the whole topic. The book ends with a model presentation to practise aloud and 42 viva questions with short answers.

New in this case sheet — tap to see the sign, the scan or the video

Wherever a sign, scan or technique is described, small buttons open free online pages: ► Photo clinical sign ► USG ultrasound / CT ► Video bedside technique ► Article free reading ► Tool calculator. A one-page **visual atlas** with QR codes (page 29) is provided for the printed copy. Only free sites are used.

The patient described is a composite teaching case; details have been chosen to illustrate the commonest examination scenario. Examine your own patient carefully and record only what you find. Doses follow AASLD 2021, EASL 2018 and Baveno VII and standard Indian practice — always check local protocols.

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CASE-TAKING STEPS

Case Taking in 12 Steps

Follow the same order every time — history → examination → summary → diagnosis → plan

Step	What you do	Details	Examiner's point
1	Particulars	Name (initials), age, sex, **occupation in full**, address (water source, hepatitis-endemic area), religion/education, informant and reliability.	Alcohol use is often under-reported — plan to re-check with a relative.
2	Chief complaints	List symptoms (not diagnoses) in **chronological order** with duration.	Separate the **chronic illness** (months) from the **decompensation** (days) that brought him to hospital.
3	History of presenting illness	Ascites → jaundice → GI bleed → confusion → fever/pain → urine; then the **precipitant** of the present episode.	Ask each of the five "decompensation" events one by one.
4	Aetiology screen	Alcohol · hepatitis B/C · metabolic (diabetes, obesity) · drugs & herbal medicines · autoimmune · Wilson/iron · cardiac & vascular (Budd–Chiari).	Quantify alcohol in **grams per day** and years.
5	Past history	Previous jaundice, transfusion, surgery, variceal bleed/banding, paracentesis, TB, diabetes, kidney disease.	Number of admissions for decompensation.
6	Treatment history	Diuretics, β-blocker, lactulose, antibiotics, NSAIDs, herbal "liver tonics"; adherence; salt restriction.	Ask: "Which tablets did you stop, and why?"
7	Personal history	Alcohol, tobacco, diet and **salt intake**, bowel (constipation), sleep pattern, sexual/injection/tattoo exposure.	Sleep reversal and constipation are early encephalopathy clues.
8	Family & socio-economic	Liver disease, hepatitis B, Wilson disease, diabetes; dependants, income loss, insurance.	Needed for transplant and drug-cost planning.
9	General examination	Consciousness, built, **icterus, pallor, spider naevi, palmar erythema, leuconychia, Dupuytren, gynaecomastia, parotids, oedema, JVP, flap**, fetor, vitals.	Raised JVP = think of a cardiac cause of ascites.
10	Abdominal examination	**Inspect → palpate → percuss → auscultate.** Ascites tests; liver; spleen; collaterals; hernia; genitalia.	Examine from the patient's right side, exposed from nipples to knees.
11	Other systems	Respiratory (hepatic hydrothorax), CVS, CNS (orientation, flap, reflexes), per-rectal (melaena, piles).	Never skip the nervous system in liver disease.
12	Summary → diagnosis → plan	Say a summary, give a provisional diagnosis, name investigations, then the **final diagnosis** with Child–Pugh class, and management.	Practise a 4–5 minute presentation.

The five questions every liver case must answer

(1) Is there **chronic liver disease / cirrhosis**? (2) What is the **cause** — alcohol, virus, metabolic, other? (3) Is there **portal hypertension** — ascites, varices, splenomegaly, collaterals? (4) What **complications** or decompensation are present — bleeding, encephalopathy, infection, kidney injury, hepatocellular carcinoma? (5) What is the **severity** — Child–Pugh, MELD — and what is the plan?

Examiner's tip — the three marks that are never missed

- **Quantify alcohol** in grams per day and years — "drinks a lot" scores nothing.
- **Demonstrate shifting dullness correctly** — percuss from the midline to the flank, mark, turn, repeat.
- **Look for the cause and the precipitant** — every decompensation has a trigger (bleed, infection, constipation, diuretics, NSAIDs, sedatives, alcohol).

Watch the bedside technique first

Two short videos show a full abdominal examination: ► Abdominal exam 1 ► Abdominal exam 2 Free OSCE guide: ► Geeky Medics Liver-focused exam and the 14 signs of chronic liver disease: ► Medmastery

SECTION A · HISTORY

Case Sheet — Chronic Liver Disease (Long Case, Abdomen)

Final MBBS General Medicine · Case-taking and presentation format

1. Particulars of the patient

Name	Mr. K. R. (initials only — always respect confidentiality)
Age / Sex	48 years / Male
Occupation	**Mason (construction worker) for 25 years** — always record the occupation in full; daily-wage work and alcohol use often go together
Address	Chalakudy, Thrissur district, Kerala (rural; house with a borewell)
Religion / Education	Hindu / Studied up to 10th standard
Informant	Patient and his wife — **reliable** (patient was drowsy on admission; history re-checked on day 2 when alert)
Date of admission / examination	Admitted through casualty; examined on day 3 of admission

2. Chief complaints (in chronological order, in the patient's own words)

- **Progressive swelling of the abdomen** — 3 months; increased with breathlessness on lying flat — 5 days
- **Swelling of both legs** — 2 months
- **Yellowish discolouration of eyes** and dark urine — 1 month
- **Vomiting of blood and passing black stools** — 1 episode, 6 weeks ago
- **Loss of appetite, weight loss and weakness** — 3 months
- **Day-time sleepiness, night-time wakefulness and irrelevant talk** — 4 days

> **Examiner's tip — two time-lines**
> In every liver case separate the **chronic illness** (months) from the **acute decompensation** (days). Write symptoms, never a diagnosis: 'abdominal distension × 3 months' is right; 'ascites × 3 months' or 'cirrhosis' is wrong.

3. History of presenting illness

The chronic illness. The patient was apparently well 3 months ago, when he noticed a **gradual, painless increase in abdominal girth** — his trousers became tight and he had to loosen his belt. The swelling was generalised, progressive and not related to meals; he felt full after a few mouthfuls. One month later both ankles began to swell, rising to the knees, worse in the evening and **pitting**; there was no swelling of the face. Over the same period he lost about **6 kg** although the abdomen enlarged — his arms, thighs and temples became thin. He noticed **yellow eyes with dark urine** for 1 month, without itching, pale stools, fever or abdominal pain (suggesting a hepatocellular, not obstructive, jaundice).

Variceal bleed 6 weeks ago. He suddenly vomited about two cupfuls of dark red blood without abdominal pain, followed by **black tarry stools for 3 days** and giddiness. He was admitted elsewhere, received 2 units of blood, and upper GI endoscopy showed large oesophageal varices, which were banded. He was started on propranolol but **stopped it after 3 weeks** because of weakness. He has had **no alcohol since the bleed**.

The present decompensation. For 5 days the abdominal distension has increased so that he cannot lie flat and feels full after a few mouthfuls. For 4 days his wife noticed that he sleeps by day and is awake at night, is forgetful and talked irrelevantly one night; she saw his outstretched hands "shaking" (asterixis). He has been **constipated for a week** and took **diclofenac tablets for leg pain** 3 weeks ago and an Ayurvedic "liver tonic" for 2 months. There is no fresh haematemesis or melaena, no fever, no abdominal pain, no diarrhoea or vomiting; urine output is slightly reduced. On admission he was drowsy with a flapping tremor; he was treated with lactulose, a diagnostic tap (negative for infection), diuretics and albumin, and is now alert, oriented and eating.

History of presenting illness — relevant positives and negatives

Question asked	Answer in this patient	Why it is asked
Alcohol — type, quantity, duration, last drink, CAGE?	**Rum 180 mL a day for 22 years (≈ 57 g ethanol/day); CAGE 4/4; last drink 6 weeks ago**	Alcohol-related liver disease; risk of withdrawal
Jaundice, dark urine, pale stools, itching?	Jaundice 1 month, dark urine; no pale stools, no itching	Hepatocellular vs obstructive/cholestatic
Haematemesis, melaena, giddiness?	**Yes — 6 weeks ago**; large varices banded	Portal hypertension — varices; also ask about NSAIDs and ulcers
Confusion, sleep reversal, tremor, irritability?	**Yes — 4 days**; constipation, diclofenac, hyponatraemia as triggers	Hepatic encephalopathy — always find the precipitant
Fever, abdominal pain or tenderness?	No	Spontaneous bacterial peritonitis (SBP), TB peritonitis, alcoholic hepatitis
Urine volume, vomiting, diarrhoea, diuretics, NSAIDs?	Slightly reduced urine; diclofenac 3 weeks ago; no diarrhoea	Pre-renal injury, hepatorenal syndrome (HRS-AKI)
Breathless on lying flat, cough, chest pain?	Breathless supine (tense ascites); no chest pain	Massive ascites, hepatic hydrothorax; cardiac cause
Leg swelling first or abdomen first? Orthopnoea, PND, palpitations?	**Abdomen first**, legs a month later; no PND	Cardiac failure starts with legs; cirrhosis with abdomen
Frothy urine, puffy face?	No	Nephrotic syndrome
Weight loss, evening fever, night sweats, cough, TB contact?	Weight loss 6 kg; no fever or sweats; no contact	TB peritonitis, malignancy
Transfusion, injections, tattoo, surgery, IV drugs; family jaundice?	None before the recent transfusion	Hepatitis B and C
Diabetes, obesity, high triglycerides?	None; BMI normal	Metabolic dysfunction-associated steatotic liver disease (MASLD)
Herbal, Ayurvedic, ATT, methotrexate, amiodarone?	Ayurvedic “liver tonic” × 2 months	Drug / herb-induced liver injury
Tremor, psychiatric symptoms, young age, family history?	None	Wilson disease, haemochromatosis (if age <40)
Bruising, gum bleeding, loss of libido, breast enlargement, cramps?	Easy bruising, loss of libido, breast swelling	Coagulopathy; hyperoestrogenism; hypogonadism

4. Past history

- **Variceal haemorrhage** 6 weeks ago — 2 units of blood; endoscopic band ligation.
- No previous jaundice, hepatitis, blood transfusion (before this), surgery or hospital admission for liver disease.
- No diabetes, hypertension, tuberculosis, heart disease, kidney disease or thyroid disease.

5. Treatment history — and what he actually takes

Drug	How he actually takes it
Propranolol 20 mg BD	**Stopped after 3 weeks** — “felt weak”; was not told why it is needed
Spironolactone 50 mg OD	Irregular — takes only when legs swell; continues **high-salt diet** (pickles, dried fish)
Lactulose	Not taking — “makes the stomach loose”; became constipated
Diclofenac (self-medication)	3 tablets for leg pain 3 weeks ago — **NSAIDs precipitate kidney injury and bleeding**
Ayurvedic “liver tonic”	From a local shop for 2 months — contents unknown
Vaccines	Never vaccinated against hepatitis A or B

Examiner’s tip — ask “which tablets did you stop?”
Non-adherence to β-blocker, diuretics and lactulose and continued salt intake explain most readmissions. Record **adherence, salt intake, NSAID and herbal use**, and vaccination status — examiners love to hear them.

6. Personal history

Alcohol	**Rum (40 % alcohol) 180 mL — "a quarter" — daily for 22 years (from age 26); more at weekends. Stopped 6 weeks ago after the bleed.** CAGE 4/4
Ethanol per day	180 mL × 0.40 × 0.79 ≈ **57 g/day** (heavy drinking: more than 40 g/day in men)
Tobacco	Bidi 10/day for 20 years (smoking index 200); no chewing tobacco
Diet / appetite	Mixed diet; **high salt**; appetite reduced; early satiety
Bowel / bladder	Constipated for a week; urine slightly reduced; dark-coloured
Sleep	**Sleep reversal** (sleeps by day, awake at night) for 4 days
Risk exposures	No tattoo, injection-drug use, unsafe injections, multiple partners or foreign travel

How to quantify alcohol — the numbers examiners expect

Ethanol (g) = volume (mL) × % alcohol ÷ 100 × 0.79. A quarter (180 mL) of 40 % spirit ≈ 57 g; a 650-mL bottle of 5 % beer ≈ 26 g; 1 litre of toddy (4–5 %) ≈ 35 g. The risk of cirrhosis rises sharply above **40 g/day in men (20 g/day in women)** over 10 years or more. Use the **CAGE** questions — Cut down? Annoyed by criticism? Guilty? Eye-opener in the morning? — or AUDIT-C. Ask about the **last drink** (withdrawal, delirium tremens) and about illicit or home-brewed liquor (methanol).

7. Family and socio-economic history

Father, a heavy drinker, died at 58 of "jaundice and swelling of the belly". No family history of hepatitis B, Wilson disease, haemochromatosis or diabetes. He lives with his wife and two school-going children in a three-room house; lower-middle class (modified Kuppuswamy scale); daily-wage income is lost during each admission and the family is covered under a state health-insurance scheme — relevant when planning repeated paracentesis, endoscopy and transplant referral.

Causes of chronic liver disease — risk factors in this patient

Group	Examples	In this patient
Alcohol	Alcohol-related liver disease; alcoholic hepatitis	**Present — 57 g/day × 22 years**
Viral	Hepatitis B (± D), hepatitis C	Not suspected — to be excluded (HBsAg, anti-HCV)
Metabolic	MASLD/NASH, diabetes, obesity	Absent (normal BMI, no diabetes)
Drug / toxin	Herbal products, methotrexate, amiodarone, ATT	Ayurvedic tonic — to be stopped
Autoimmune / cholestatic	Autoimmune hepatitis, PBC, PSC	Not suspected
Genetic	Wilson, haemochromatosis, α_1-antitrypsin deficiency	Not suspected (age 48, no family history)
Vascular / cardiac	Budd–Chiari syndrome, right heart failure, constrictive pericarditis	Excluded on JVP, echo and Doppler

Indian context: alcohol, hepatitis B and MASLD are the leading causes of cirrhosis; a significant number remain cryptogenic. Non-cirrhotic portal hypertension (NCPF) and extrahepatic portal vein obstruction (EHPVO) are important causes of portal hypertension in young Indians with a normal liver function.

8. Summary of history

Summary (say this before you start the examination)

A **48-year-old man, a mason and heavy drinker (about 57 g alcohol a day for 22 years; abstinent for 6 weeks)**, presents with **progressive abdominal distension for 3 months, bilateral leg swelling for 2 months, jaundice for 1 month**, a **variceal bleed 6 weeks ago** treated with banding, and **4 days of sleep reversal and confusion** following **constipation, high salt intake, NSAID and herbal drug use** after stopping propranolol and lactulose. There is no fever, abdominal pain, diabetes, transfusion or injection-drug history, and no family history of liver disease other than an alcoholic father.

9. Provisional diagnosis from history

Decompensated chronic liver disease — probably alcohol-related cirrhosis — with portal hypertension (ascites, previous variceal bleed) and hepatic encephalopathy (precipitated by constipation, NSAIDs and non-adherence).

How the history leads to the diagnosis — the reasoning

Clue from history	Inference
22 years × 57 g/day alcohol, abstinent only 6 weeks	Alcohol-related liver disease; steatosis → hepatitis → **cirrhosis**
Painless, gradual abdominal distension before leg swelling	**Ascites** of hepatic origin (cardiac failure begins with leg oedema)
Jaundice, dark urine, no itching, no pale stools	Hepatocellular jaundice — **liver failure**
Haematemesis then melaena, large varices banded	**Portal hypertension** — oesophageal varices (a previous decompensating event)
Sleep reversal, irrelevant talk, flapping hands, constipation	**Hepatic encephalopathy** with a precipitant
No fever or abdominal pain	SBP less likely — but a diagnostic tap is mandatory in every admitted patient with ascites
Weight loss despite distension; thin limbs	Sarcopenia — protein–calorie malnutrition, poor prognosis
Easy bruising, breast swelling, loss of libido	Coagulopathy and hyperoestrogenism of chronic liver disease

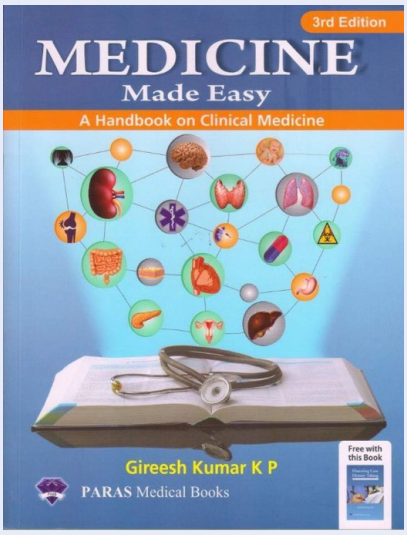
Examiner's tip — what the examiner looks for in the history

(1) **Alcohol in grams per day and years**; (2) the **five decompensation events** — ascites, jaundice, bleed, encephalopathy, infection; (3) **causes** — viral, metabolic, drugs; (4) **precipitants**; (5) **negatives** for cardiac, renal and tuberculous causes of ascites; (6) **adherence and salt intake**, vaccination.

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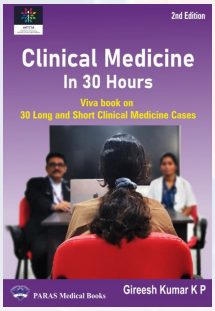
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SECTION B · EXAMINATION

General Physical Examination

Look for stigmata of chronic liver disease, hepatic encephalopathy, volume status and the clues to another cause

Consciousness / orientation	Conscious, alert and oriented today (drowsy on admission — West Haven grade 2)
Built / nourishment	**Thin limbs with a distended abdomen**; height 168 cm, weight 58 kg (**dry weight about 48 kg**); temporal and thenar wasting; mid-arm circumference 21 cm — sarcopenia
Decubitus	Prefers to sit propped up (tense ascites pushes up the diaphragm)
Breath	**Fetor hepaticus** — sweetish, musty odour (mercaptans)
Hands	Warm, **palmar erythema**, **leuconychia**, **Dupuytren's contracture** of the right ring finger; no clubbing; **flapping tremor absent today** (present on admission)

Sign	Finding	Relevance / mechanism
Jaundice (icterus)	**Present** — sclerae and under the tongue (bilirubin 3.6 mg/dL)	Hepatocellular failure; seen when bilirubin >2–2.5 mg/dL
Pallor	Mild	Blood loss, macrocytic anaemia of alcohol, hypersplenism
Spider naevi ► Photo	**Six** on the face, neck and upper chest; blanch on pressure with a central pulsating arteriole	Hyperoestrogenism; >3 are significant — present only in the SVC territory
Palmar erythema ► Photo	**Present** — thenar and hypothenar eminences	Hyperoestrogenism; also in pregnancy, thyrotoxicosis, RA
Leuconychia / Terry's nails	White nails with narrow distal pink band	Hypoalbuminaemia (albumin 2.4 g/dL)
Dupuytren's contracture ► Article	Right ring finger	Alcohol-related; palmar fascia fibrosis
Gynaecomastia & hair loss	**Bilateral gynaecomastia**; loss of axillary and chest hair; **testicular atrophy**	Failure to inactivate oestrogen; ↓ androgens; also spironolactone
Parotid enlargement	Bilateral, painless	Chronic alcohol use, malnutrition
Clubbing	**Absent**	Present in biliary cirrhosis, hepatopulmonary syndrome, lung disease
Bruising / purpura	Ecchymoses over forearms and shins	Low platelets, ↓ clotting factors (INR 1.8)
Oedema	**Bilateral pitting oedema to the knees**; sacral oedema	Hypoalbuminaemia, sodium retention, ascites compressing IVC
JVP	**Not raised** (≈ 2 cm above the sternal angle)	Raised JVP would suggest a cardiac cause of ascites
Lymph nodes	No supraclavicular (Virchow) node	Gastric/hepatobiliary malignancy
Xanthelasma, Kayser–Fleischer ring	Absent	PBC; Wilson disease (slit-lamp)
Flapping tremor (asterixis) ► Video	Absent today; **present on admission**	Hepatic encephalopathy grade ≥ 2

Vital signs

Vital	Finding	Interpretation
Pulse	98/min, regular, **bounding**; all pulses felt	Hyperdynamic circulation of cirrhosis (vasodilatation)
Blood pressure	**100/60 mmHg** (right arm, sitting)	Low-normal in cirrhosis; <90 systolic limits β-blocker and diuretic use
Respiratory rate	22/min, abdominal splinting	Tense ascites limits diaphragm movement
Temperature	37.2 °C	No fever — SBP less likely (but fever may be absent in SBP)
SpO_2	96 % on room air	Check for hepatopulmonary syndrome if low or ↓ on sitting up (orthodeoxia)
Weight / abdominal girth	58 kg; **girth 98 cm** at the umbilicus	Baseline for response to diuretics (target: loss of 0.5 kg/day)



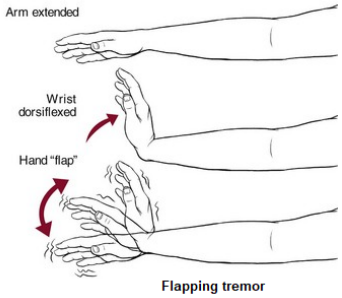


Fig. 1 Asterixis (flapping tremor): outstretched arms, wrists dorsiflexed — irregular lapses of posture. Negative myoclonus; test for 30 seconds.

Asterixis — how to elicit it

Ask the patient to stretch out both arms with wrists dorsiflexed and fingers spread, eyes closed, for **30 seconds**. A positive sign is sudden, irregular, **flexion "flaps" at the wrists** from loss of postural tone. It is a sign of metabolic encephalopathy — hepatic, uraemic or CO_2 — and appears from **West Haven grade 2**. Watch a real patient: ► Video ► West Haven

Hepatic encephalopathy — bedside grading (West Haven)

Grade	Mental state	Neuro-muscular signs
0 / minimal (covert)	Normal on bedside testing; abnormal psychometric tests only	None; may affect driving, work
1	Trivial lack of awareness, euphoria or anxiety, short attention span, difficulty with addition, **sleep reversal**	Mild incoordination; flap may be absent
2	Lethargy, disorientation to time, obvious personality change, inappropriate behaviour	**Asterixis present**, slurred speech
3	Somnolent but rousable, confused, grossly disoriented, bizarre behaviour	Hyperreflexia, clonus, extensor plantars
4	**Coma** — no response to pain	Decerebrate posture, absent reflexes

Examiner's tip — "Show me the signs of chronic liver disease"

Go from **head to toe** in a fixed order: hands (clubbing, leuconychia, palmar erythema, Dupuytren, flap) → arms (bruising, spider naevi) → face (icterus, parotids, xanthelasma, fetor, wasted temples) → chest (spider naevi, gynaecomastia, hair loss) → abdomen → legs (oedema, bruising). Then say: "I would like to examine the abdomen for ascites and portal hypertension."

SECTION B · ABDOMEN

Examination of the Abdomen

Position, expose, inspect → palpate → percuss → auscultate — and always examine the hernial orifices and genitalia

Position and preparation (do this every time)

Wash hands, introduce yourself, take consent and ask about pain. Patient **supine with one pillow, arms by the sides, knees slightly flexed**, exposed from **nipples to mid-thigh** (with privacy). Stand on the **right side**; warm hands; look at the patient's face while palpating. Watch the full sequence: ► Video 1 ► Video 2 ► OSCE guide ► Ascites tests

a) Inspection



Fig. 2 Tense, globular abdomen of large-volume ascites: flattened/everted umbilicus, bulging flanks, dilated veins and skin marks.



Fig. 3 Caput medusae — dilated veins radiating from the umbilicus (left: schematic; right: patient).

Feature	Finding	Interpretation
Shape and contour	**Distended, symmetrical, globular**; **bulging flanks**; abdomen is wider than it is deep when supine	Free fluid gravitates to the flanks (frog belly)
Umbilicus	**Everted/flat**; **small reducible umbilical hernia**	↑ intra-abdominal pressure from tense ascites
Skin	Shiny, tight, **stretch marks**; icterus; bruises; spider naevi over the chest	Rapid distension; clotting defect
Veins	**Dilated veins around the umbilicus (caput medusae)** ► Photo; flow **away from the umbilicus** (above → upward, below → downward)	Portosystemic shunt through paraumbilical veins
Movements	Moves poorly with respiration; **no visible peristalsis or pulsation**	Tense ascites
Scars, swellings	No scars; **no visible mass**; flanks full	Previous surgery, hepatoma
Hernial orifices	Umbilical hernia; inguinal orifices normal on coughing	Hernias are common with tense ascites
Genitalia	**Scrotal oedema**, small soft testes	Hypoalbuminaemia; hypogonadism

Direction of flow in abdominal wall veins — a favourite viva

Press a finger on a vein, strip it with another finger and release one — see which way it refills. **Portal hypertension / caput medusae:** veins radiate from the umbilicus; above it blood flows **upward**, below it **downward** (away from the navel). **IVC obstruction** (Budd–Chiari, thrombosis): veins in the flanks carry blood **upward** everywhere, the lower ones from groin to chest, and are also seen on the back. In **SVC obstruction** flow is downward.

b) Palpation

Feature	Finding	Interpretation
Tenderness, guarding, rigidity	**Tense but non-tender**; no guarding or rebound	SBP causes tenderness in only some patients; tenderness + fever + ascites → tap
Temperature, fluid thrill	**Fluid thrill positive**	Large-volume ascites
Liver ► Technique	**Not palpable** by superficial palpation; **dipping** (ballottement) method does not reveal an edge; liver dullness upper border in the 6th space; span **8 cm** (shrunken)	Shrunken, nodular, firm liver of cirrhosis (early cirrhosis may be enlarged and firm with a sharp edge)
Spleen	**Palpable 4 cm below the left costal margin** (Hackett grade 2), firm, notch present, moves with respiration, cannot get above it, dull on percussion	Congestive splenomegaly of portal hypertension (hypersplenism → ↓ platelets)
Kidneys	Not ballotable	Spleen vs left kidney: notch, no resonant band, cannot get above
Epigastrium / other masses	No mass; aorta pulsation not felt	Hepatocellular carcinoma may present as a hard nodular mass
Para-umbilical vein / thrill	**Venous hum** felt at the umbilicus	Cruveilhier–Baumgarten sign
Hernial sites	Reducible umbilical hernia	Risk of rupture and leak in tense ascites

Examiner's tip — palpating the liver in tense ascites
In tense ascites the liver floats away from the examining hand. Use the **dipping method**: with the fingers held stiff and together, press sharply downward and release — the liver edge "bounces" against the finger tips. Better still, **re-examine after paracentesis**. Always **measure the span** by percussion and record the **edge, surface, consistency, tenderness and pulsation**.

c) Percussion — the tests for ascites

Test	How to do it	Positive finding	Detects
Flank dullness / bulging flanks	Percuss from the umbilicus (tympanic, gas-filled bowel floats) outward to each flank	Dull in both flanks; tympany centrally	≈ 1–1.5 L — most sensitive sign; absence argues against ascites
Horse-shoe dullness (supine)	Percuss from the centre to the flanks and below; map the border of dullness	Dullness in the flanks and lower abdomen, **curved, concave upward** (Fig. 4, left)	Moderate–large ascites
Shifting dullness	Mark the dull–resonant boundary on one flank; roll the patient to the opposite side for **1 minute**; percuss again	Dullness **shifts to the new dependent flank**; the upper flank becomes resonant (Fig. 4, right)	Ascites ≥ 1 L
Fluid thrill (fluid wave)	Assistant presses the **edge of the hand along the midline**; examiner flicks one flank and feels the opposite flank	Impulse felt on the opposite hand	Large-volume (tense) ascites; most specific but insensitive
Puddle sign	Patient on knees and elbows for 3–5 minutes; percuss/auscultate from flank to the dependent midline	Change from dull to resonant note over the fluid pool (Fig. 5)	Small volumes (about 120 mL); poorly reliable, rarely used

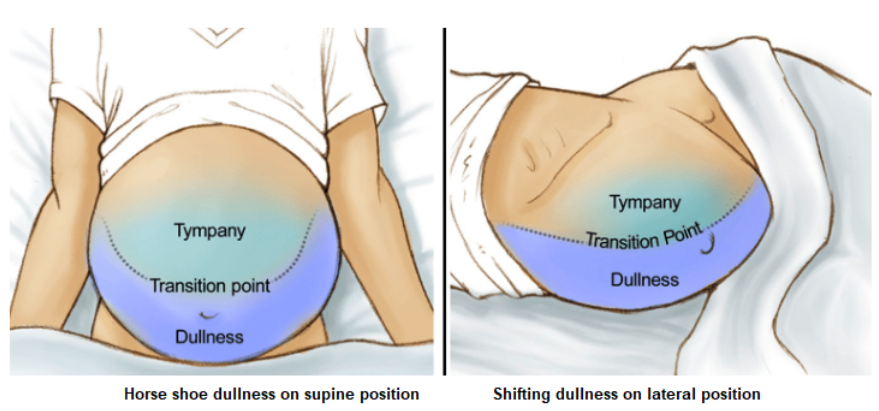


Fig. 4 Horse-shoe dullness (supine, left) and shifting dullness (lateral position, right): the transition point moves with posture.

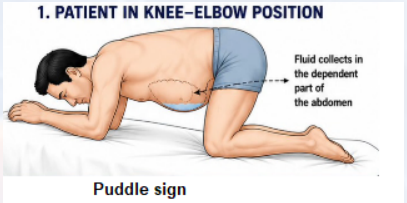


Fig. 5 Puddle sign (knee–elbow position): fluid collects in the dependent part.

See the examination performed: ► Video 1 ► Video 2 ► Ascites review ► Accuracy of signs ► Quick notes

Ascites vs ovarian cyst vs obesity — the percussion pattern

Ascites: central tympany (floating bowel) with **dull flanks**, shifting dullness, fluid thrill. **Large ovarian cyst:** central **dullness** with **resonant flanks**; no shifting dullness; pelvic mass; transmitted aortic pulsation. **Obesity / gas ("fat, flatus"):** tympany all over, no shifting dullness. Pregnancy, faeces and tumour complete the "F" list. **Ultrasound** settles the doubt.

Grading of ascites (EASL/AASLD): **grade 1 (mild)** — detectable only on ultrasound; **grade 2 (moderate)** — moderate symmetrical distension; **grade 3 (large/tense)** — marked distension. This patient has **grade 3 (large) ascites**. ► AASLD 2021

d) Auscultation

Feature	Finding	Interpretation
Bowel sounds	Present, normal; **reduced** if SBP/ileus	Silent abdomen suggests peritonitis or ileus
Venous hum over the umbilicus / epigastrium	**Present** — continuous, soft, louder on expiration (Cruveilhier–Baumgarten murmur)	Flow through a patent paraumbilical vein: portal hypertension
Hepatic bruit / friction rub	Absent	Bruit: hepatocellular carcinoma, alcoholic hepatitis; rub: peri-hepatitis, biopsy
Succussion splash	Absent	Gastric outlet obstruction

Signs of portal hypertension — look for all of them

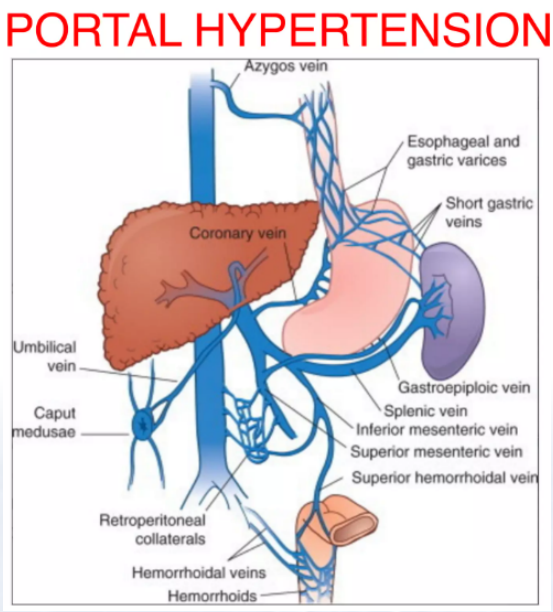


Fig. 6 Portosystemic collaterals in portal hypertension: oesophageal and gastric varices, caput medusae, rectal veins, retroperitoneal collaterals.

Site of collateral	Veins connected (portal → systemic)	Clinical result
Lower oesophagus / gastric fundus	Left gastric (coronary) and short gastric → azygos / oesophageal veins	**Oesophageal and gastric varices** — haematemesis, melaena
Umbilicus	Paraumbilical (recanalised umbilical) veins → epigastric veins	**Caput medusae**; Cruveilhier–Baumgarten hum
Anorectal junction	Superior rectal → middle and inferior rectal	**Haemorrhoids**, rectal varices
Retroperitoneum / bare area	Veins of Retzius, colonic, lumbar and phrenic veins	Portal colopathy; (usually silent)
Spleno-renal shunt	Splenic → left renal vein	Gastric varices; hepatic encephalopathy

Sign of portal hypertension	In this patient
Splenomegaly ± hypersplenism (↓ platelets)	**Present** — 4 cm; platelets 82,000/µL
Ascites	**Present — grade 3**
Caput medusae and venous hum	**Present**
Haemorrhoids / rectal varices	Small internal haemorrhoids (per rectal)
Oesophageal / gastric varices	**Large varices with previous bleed** (endoscopy)
Portal hypertensive gastropathy	Mild (endoscopy)

Examination of other systems

System	Findings	Relevance
Cardiovascular	JVP not raised; apex beat normal; heart sounds normal; no murmur, no pericardial knock	**Excludes cardiac ascites** (raised JVP, hepatomegaly, S_3) and constrictive pericarditis
Respiratory	RR 22; **decreased breath sounds and stony dullness at the right base** (small right hepatic hydrothorax); spider naevi on the chest	Hepatic hydrothorax (right-sided in 85 %); hepatopulmonary syndrome
Nervous system	Alert; **oriented to time, place, person**; serial 7s slowed; **no flap today**; plantars flexor; no focal deficit; no tremor of Wilson	Hepatic encephalopathy resolving; examine reflexes and plantars
Per rectal	Small internal haemorrhoids; **no melaena on the glove**	Portal haemorrhoids; ongoing GI bleed
Genitalia	Scrotal oedema; testicular atrophy	Hypogonadism, hypoalbuminaemia
Musculoskeletal	Wasting of quadriceps and small muscles; no bone tenderness	Sarcopenia; hepatic osteodystrophy
Skin	Excoriations absent; bruises; xanthomas absent	Cholestasis, coagulopathy

Conclusion — abdominal and systemic examination

There is **large-volume (grade 3) ascites** (bulging flanks, horse-shoe dullness, shifting dullness, fluid thrill) with **signs of portal hypertension** — caput medusae with venous hum, firm splenomegaly (4 cm), umbilical hernia, haemorrhoids — and **stigmata of chronic liver disease** (jaundice, spider naevi, palmar erythema, leuconychia, Dupuytren's contracture, gynaecomastia, testicular atrophy, parotid enlargement, muscle wasting, fetor). The liver is **small and not palpable** (span 8 cm). JVP is not raised. There is a small right hepatic hydrothorax; encephalopathy has resolved to grade 0–1.

Hepatomegaly, splenomegaly and ascites — what the combination suggests

Combination	Think of
Small liver + splenomegaly + ascites + stigmata	**Cirrhosis with portal hypertension** (this patient)
Large tender liver + ascites + no splenomegaly	Budd–Chiari syndrome, right heart failure, alcoholic hepatitis, hepatocellular carcinoma
Large smooth liver, raised JVP, ascites, oedema	Congestive cardiac failure, constrictive pericarditis
Massive splenomegaly + varices + normal liver function	Non-cirrhotic portal fibrosis / EHPVO (India), myeloproliferative disease
Hard nodular liver, cachexia, ascites, Virchow node	Metastases, hepatocellular carcinoma, peritoneal carcinomatosis
Doughy abdomen, fever, night sweats, weight loss	Tuberculous peritonitis (check ADA)

Examiner's tip — expose, sit up, compare and finish properly

Finish every abdomen with: "I would like to examine the **hernial orifices and external genitalia**, do a **per-rectal examination**, look at the **chest for effusion**, record the **weight and abdominal girth**, and see the **urine chart** and the **ascitic fluid report**."

SECTION C · DIAGNOSIS

Summary and Final Clinical Diagnosis

Disease → cause → portal hypertension → complications → severity → comorbidities

Summary of examination

Say this after your history summary

On examination he is conscious and oriented, thin with **muscle wasting**, **icteric**, with **spider naevi, palmar erythema, leuconychia, Dupuytren's contracture, gynaecomastia, parotid enlargement and fetor hepaticus**. There is **bilateral pitting oedema** and JVP is **not raised**. Pulse 98/min, bounding; BP 100/60 mmHg; no fever. The abdomen is **distended with bulging flanks and an everted umbilicus**, with **caput medusae and a venous hum**. **Shifting dullness and fluid thrill are positive** — large-volume ascites. The **liver is not palpable (span 8 cm)**; the **spleen is palpable 4 cm** below the costal margin. Flap is absent today but was present on admission. There is a small right pleural effusion.

Arriving at the diagnosis — step by step

Question	Answer	Conclusion
Is there chronic liver disease?	Spider naevi, palmar erythema, leuconychia, gynaecomastia, testicular atrophy, small liver	**Chronic liver disease — cirrhosis**
What is the cause?	Alcohol 57 g/day × 22 years; HBsAg and anti-HCV negative	**Alcohol-related cirrhosis**
Is there portal hypertension?	Splenomegaly, ascites, caput medusae, varices, low platelets	**Clinically significant portal hypertension**
Why is there ascites? (Cause)	Stigmata of CLD; JVP normal; SAAG ≥ 1.1 g/dL; ascitic protein low	**Portal-hypertensive (cirrhotic) ascites**
What decompensation has occurred?	Ascites, jaundice, variceal bleed, hepatic encephalopathy	**Decompensated cirrhosis**
Any infection or kidney injury?	Ascitic PMN < 250/mm³; culture negative; creatinine 1.0 mg/dL	No SBP; no HRS-AKI at present
What precipitated this episode?	Constipation, stopped β-blocker and lactulose, NSAIDs, salt, hyponatraemia	Non-adherence + NSAIDs + constipation
How severe is the disease?	Child–Pugh 13 (class C); MELD-Na 24	**Advanced; refer for transplantation**

Final clinical diagnosis

Write it in this format

Chronic liver disease — alcohol-related cirrhosis [disease and cause] — **decompensated, Child–Pugh class C (score 13), MELD-Na 24** [severity] — with **clinically significant portal hypertension** [large-volume (grade 3) ascites, splenomegaly with hypersplenism, large oesophageal varices with previous bleed, caput medusae] — complicated by **hepatic encephalopathy (grade 2, resolved)**, **hypo-albuminaemia, coagulopathy, dilutional hyponatraemia, anaemia** and a **small right hepatic hydrothorax** [complications] — precipitated by **constipation, non-adherence and NSAID use** — **no SBP, no HRS, no hepatocellular carcinoma** on evaluation [exclusions] — in a patient with **poor nutrition, unvaccinated and a heavy drinker, now abstinent** [management issues].

Child–Pugh score — learn the five parameters

Parameter	1 point	2 points	3 points	This patient
Bilirubin (mg/dL)	< 2	2–3	> 3	3.6 → **3**
Albumin (g/dL)	> 3.5	2.8–3.5	< 2.8	2.4 → **3**
INR	< 1.7	1.7–2.3	> 2.3	1.8 → **2**
Ascites	None	Mild / controlled	Moderate–severe / refractory	Large → **3**
Encephalopathy	None	Grade 1–2 (or controlled)	Grade 3–4	Grade 2 (resolved) → **2**
Total	A = 5–6	B = 7–9	C = 10–15	**13 → Class C**

Approximate one-year survival: **class A ≈ 100 %, class B ≈ 80 %, class C ≈ 45 %**. **MELD** = 10 × [0.957 ln(creatinine) + 0.378 ln(bilirubin) + 1.12 ln(INR) + 0.643]; here ≈ **18**, and with sodium 129 mmol/L the **MELD-Na** ≈ **24** (≥ 15 → refer for transplantation). Calculator: ► Child–Pugh calculator

Differential diagnosis of ascites — by SAAG and protein

SAAG	Ascitic protein	Causes	Pointers at the bedside
≥ 1.1 g/dL (portal hypertension)	< 2.5 g/dL	**Cirrhosis**, alcoholic hepatitis, massive liver metastases, fulminant hepatic failure, portal vein thrombosis	Stigmata of CLD, splenomegaly, caput, normal JVP
≥ 1.1 g/dL	≥ 2.5 g/dL	**Cardiac** ascites (RHF, constrictive pericarditis), **Budd–Chiari** syndrome, sinusoidal obstruction	Raised JVP, S_3, hepatomegaly, tender liver, dilated flank veins
< 1.1 g/dL	≥ 2.5 g/dL	**Tuberculous** peritonitis, peritoneal carcinomatosis, pancreatic ascites, nephrotic syndrome (low protein), serositis, bowel obstruction	Fever, weight loss, doughy abdomen; palpable omental cake; periorbital oedema, frothy urine
Any	Chylous / bloody / bile-stained	Lymphoma, trauma, chylous ascites; bile peritonitis	Milky fluid, triglycerides > 200 mg/dL

SAAG = serum albumin − ascitic albumin (same-day samples). **This patient:** 2.4 − 0.6 = **1.8 g/dL** (≥ 1.1 → portal hypertension); ascitic protein 1.2 g/dL. SAAG is ≈ 97 % accurate for portal-hypertensive ascites. Reading: ► Ascites review ► StatPearls

Differential diagnosis of the clinical picture — for and against

Condition	Points in favour	Points against
Alcohol-related cirrhosis with portal hypertension (most likely)	Heavy alcohol, stigmata, small liver, splenomegaly, SAAG 1.8, varices	— (confirm: USG, endoscopy, viral markers)
Chronic hepatitis B / C cirrhosis	Common in India	HBsAg, anti-HCV negative; no risk factors
MASLD / NASH cirrhosis	Common	No obesity or diabetes; AST:ALT 2:1; heavy alcohol
Cardiac cirrhosis / constrictive pericarditis	Ascites, oedema	JVP normal; echo normal
Budd–Chiari syndrome	Ascites, hepatomegaly	Liver small; hepatic veins patent on Doppler
Tuberculous peritonitis	Weight loss, India	No fever, SAAG ≥ 1.1, ADA 10 U/L, lymphocytes few
Peritoneal carcinomatosis / hepatocellular carcinoma	Weight loss, ascites	SAAG high, cytology negative, AFP 6, no mass on USG
Non-cirrhotic portal hypertension (NCPF/EHPVO)	Splenomegaly, varices	Stigmata, jaundice, low albumin, small nodular liver

Causes of portal hypertension — classification

Site	Examples	Liver function
Pre-hepatic	Portal or splenic vein thrombosis (EHPVO), massive splenomegaly (tropical), arteriovenous fistula	Normal
Intrahepatic — pre-sinusoidal	Non-cirrhotic portal fibrosis (NCPF, idiopathic), schistosomiasis, congenital hepatic fibrosis, PBC (early), sarcoid	Normal / mildly ↑
Intrahepatic — sinusoidal	**Cirrhosis** (alcohol, HBV, HCV, MASLD, autoimmune), alcoholic hepatitis	Impaired
Intrahepatic — post-sinusoidal	Sinusoidal obstruction syndrome (veno-occlusive disease)	Variable
Post-hepatic	Budd–Chiari syndrome, right heart failure, constrictive pericarditis, IVC web	Congestive

Portal hypertension — definitions you must know

Portal pressure gradient (HVPG) normal 1–5 mmHg; **portal hypertension > 5 mmHg**; **clinically significant (CSPH) ≥ 10 mmHg** — varices and ascites appear; **≥ 12 mmHg** — variceal bleeding risk. In compensated advanced chronic liver disease, **liver stiffness > 25 kPa** by elastography rules in CSPH (Baveno VII). ► Baveno VII ► Endoscopy video

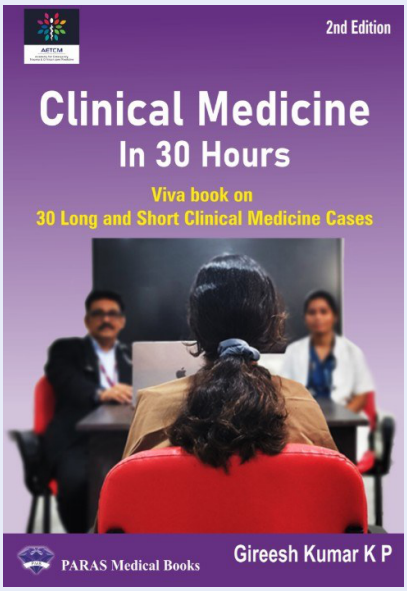
Cirrhosis vs non-cirrhotic portal fibrosis (NCPF) — an Indian favourite

Feature	Cirrhosis	NCPF / EHPVO
Age, cause	Middle age; alcohol, HBV, HCV, MASLD	Young adults; low socio-economic, arsenic, infections
Stigmata of CLD	Present	Absent
Jaundice, ascites	Common (decompensation)	Rare; ascites only after bleed or hypoalbuminaemia
Liver size	Small and nodular	Normal or small, smooth
Spleen	Moderate	**Massive**
Variceal bleed	Poorly tolerated, Child class matters	**Well tolerated**; liver function normal
LFT	Deranged; INR ↑; albumin ↓	Normal or near-normal
Liver biopsy	Nodules with fibrous septa	Obliterative portal venopathy, no cirrhosis

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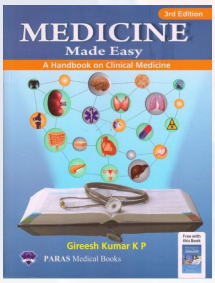
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SECTION D · INVESTIGATIONS

Investigations — Plan, Rationale and Results

Confirm cirrhosis and portal hypertension, find the cause, exclude infection, grade severity, screen for complications

Investigation	Why	Result in this patient
Complete blood count	Anaemia, MCV (alcohol), platelets (hypersplenism), TLC (infection)	Hb **9.4 g/dL**, MCV 104 fL, TLC 5,800/µL, **platelets 82,000/µL**
PT / INR	Synthetic function; bleeding risk	**INR 1.8** — does not correct with vitamin K (hepatocellular)
Liver function tests	Pattern of injury; synthetic function	Bilirubin **3.6** (direct 1.9) mg/dL; AST 96, ALT 48 (AST:ALT ≈ 2:1); ALP 142; GGT 210 U/L; **albumin 2.4**, globulin 3.8 g/dL (A:G reversed)
Urea, creatinine, sodium, potassium	Hyponatraemia, HRS, hypokalaemia	Urea 34, creatinine **1.0** mg/dL, **Na 129**, K 3.4 mmol/L
Viral markers	HBsAg, anti-HCV (± HIV, HBV DNA)	HBsAg **negative**; anti-HCV negative; HIV negative
Ascitic fluid analysis (diagnostic tap)	SAAG, PMN count, culture, protein, ADA, cytology	See next page — **SAAG 1.8; PMN 40/mm³**; culture negative
Ultrasound abdomen with Doppler	Liver, spleen, portal vein, ascites, focal lesion	See below
Upper GI endoscopy	Varices, gastropathy, source of bleeding	**Large (grade III) oesophageal varices** with scars of banding; mild portal hypertensive gastropathy; no gastric varices
Alpha-fetoprotein (AFP)	Hepatocellular carcinoma surveillance	AFP 6 ng/mL (normal)
Echocardiography, chest X-ray, ECG	Exclude cardiac cause; hydrothorax; QT before drugs	Normal LV/RV, PASP 24 mmHg; small right pleural effusion; ECG normal
Urine routine, urinary sodium	Bile pigments, protein; HRS vs ATN	Bile pigments +; protein nil; urine Na 12 mmol/L
Optional: ANA/AMA, ceruloplasmin, ferritin/TSAT, α_1-antitrypsin	Only if the cause is unclear or the patient is young	Not required — cause evident

Examiner's tip — a diagnostic paracentesis for every patient

Perform a **diagnostic tap in every patient with new ascites and every hospitalised patient with ascites**, and whenever there is GI bleeding, fever, abdominal pain, encephalopathy or worsening renal function. **Do not wait for the INR** — a tap is safe even with coagulopathy. Watch the technique: ► Ascitic tap guide ► AASLD 2021

Ascitic fluid analysis — what to send and how to read it

Test	This patient	Interpretation
Appearance	Straw-coloured, clear	Cloudy = infection; bloody = HCC/trauma; milky = chylous
Total protein	**1.2 g/dL**	< 1.5 g/dL with renal or liver failure = high risk of SBP → **primary prophylaxis**
SAAG	**2.4 − 0.6 = 1.8 g/dL**	≥ 1.1 g/dL = portal hypertension
Cell count and differential	WBC 120/mm³; **neutrophils (PMN) 40/mm³**	**PMN ≥ 250/mm³ = spontaneous bacterial peritonitis**; lymphocytes > 70 % → TB
Culture (blood-culture bottles at the bedside)	No growth	Inoculate 10 mL in each bottle at the bedside — positive in about 80 %
ADA	10 U/L	> 30–40 U/L suggests TB peritonitis (use only if SAAG < 1.1 or suspicion)
Cytology	Negative	Only if cirrhosis is not the likely cause
Amylase, triglyceride, glucose, LDH	Not required	Pancreatic, chylous or secondary (surgical) peritonitis

Ultrasound of the abdomen — what you should look for

Watch an ultrasound examination of the abdomen: ► USG video ► USG abdomen video Real cases with images: ► Cirrhosis + portal HTN ► Full spectrum ► Gross ascites ► CT cirrhosis

Finding	In this patient	Meaning
Liver size, surface, echotexture	**Small**, **nodular surface**, **coarse echotexture**; **caudate lobe hypertrophy**	Cirrhosis (right lobe shrinks, caudate/left lateral hypertrophy)
Focal lesion	None	Hepatocellular carcinoma — screen 6-monthly with USG ± AFP
Portal vein	**14 mm** (normal ≤ 13 mm); hepatopetal flow, reduced velocity (11 cm/s); no thrombus	Portal hypertension; hepatofugal flow or thrombus are important complications
Hepatic veins	Patent, narrow, monophasic	Cirrhosis; exclude Budd–Chiari
Spleen	**15 cm**; splenic vein 10 mm	Congestive splenomegaly
Collaterals	**Recanalised paraumbilical vein (4 mm)**; splenorenal collaterals	Portosystemic shunting
Ascites	**Gross, free, anechoic** fluid in all quadrants and pelvis	USG detects very small volumes (≈ 100 mL), best in Morison's pouch
Gallbladder and bile ducts	Thick-walled gallbladder (hypoalbuminaemia); CBD normal	No biliary obstruction
Pleura	Small right pleural effusion	Hepatic hydrothorax

Other investigations in portal hypertension and cirrhosis

Test	Role	Comment
Upper GI endoscopy ► Video	Varices (small < 5 mm / large ≥ 5 mm), red signs, gastric varices, gastropathy	Screen at diagnosis of cirrhosis; repeat band ligation every 2–4 weeks until eradication
Elastography (FibroScan)	Liver stiffness: < 10 kPa excludes advanced fibrosis; > 15 kPa = cirrhosis; > 25 kPa = CSPH	Not reliable in tense ascites, acute hepatitis or obesity; Baveno VII uses it with platelets
CT / MRI (triple-phase)	Hepatocellular carcinoma, portal vein thrombosis, collaterals	Arterial enhancement with washout = HCC; Radiopaedia ► CT
Liver biopsy (transjugular)	When the cause is unclear or the diagnosis is doubtful	Needed rarely; coagulopathy and ascites favour the transjugular route
Blood culture, urine culture, chest X-ray	Look for infection in every decompensation	Infection is the commonest precipitant

Liver function tests — pattern recognition

Pattern	Findings	Think of
Hepatocellular	ALT/AST ↑↑ (> 3×), ALP minimally ↑	Viral hepatitis, drug injury, ischaemia; **AST:ALT ≥ 2:1 with GGT ↑ and AST < 300 = alcohol**
Cholestatic	ALP/GGT ↑↑, bilirubin ↑, AST/ALT mildly ↑	Obstruction, PBC, PSC, drugs
Synthetic failure	Albumin ↓, INR ↑ (not corrected by vitamin K), bilirubin ↑	Advanced cirrhosis / acute liver failure

Interpretation of this patient's investigations

Cirrhosis (nodular small liver, thrombocytopenia, low albumin, prolonged INR) with **portal hypertension** (splenomegaly, portal vein dilatation, collaterals, large varices, SAAG 1.8), of **alcoholic aetiology** (AST:ALT 2:1, high GGT, macrocytosis, negative viral markers). **No SBP** (PMN 40/mm³), **no HCC**, **no HRS** (creatinine 1.0). Dilutional **hyponatraemia** and **hypokalaemia** need correction. Child–Pugh class C.

SECTION E · MANAGEMENT

Management of Decompensated Cirrhosis

Ascites · variceal bleeding · SBP · encephalopathy · kidney injury · cause · prevention · transplantation

Definition — decompensated cirrhosis

Cirrhosis with one or more of: **ascites, variceal haemorrhage, hepatic encephalopathy or jaundice**. Median survival falls from > 12 years (compensated) to about 2 years. Every decompensation needs three things: **treat the event, find and remove the precipitant, prevent the next one**. Guidelines (free): ► AASLD 2021 ► Baveno VII

A. Ascites — step by step

Step	Action
Diet	**Sodium restriction: 2 g (88 mmol) a day ≈ 5 g salt** — no added salt, no pickles, dried fish, papads, processed food. **No fluid restriction** unless sodium < 125 mmol/L. **Protein 1.2–1.5 g/kg** and 35 kcal/kg a day; small frequent meals and a **late-evening snack**; never restrict protein.
Diuretics	**Spironolactone 100 mg + furosemide 40 mg** once every morning (ratio 100 : 40). Increase every 3–7 days to a maximum of **400 mg + 160 mg**. Target weight loss **0.5 kg/day** (1 kg/day if leg oedema). Monitor weight, sodium, potassium and creatinine every 2–3 days at first.
Stop diuretics if	Sodium < 125 mmol/L (EASL) · creatinine rising (AKI) · hepatic encephalopathy · severe cramps · K^+ > 6 mmol/L (stop spironolactone) or < 3 mmol/L (stop furosemide).
Large-volume paracentesis (LVP)	For **grade 3 (tense) ascites**: drain the whole volume in one session, with **albumin 6–8 g per litre removed** if more than 5 L. Then start salt restriction and diuretics. **This patient:** 6 L drained with 48 g of albumin (8 g × 6 L).
Avoid	**NSAIDs**, ACE inhibitors / ARBs, aminoglycosides, non-selective α-blockers, sedatives and opioids, herbal medicines.
Refractory ascites	No response to maximal diuretics (400/160 mg) for 1 week with sodium restriction, or intolerable side-effects: **serial LVP + albumin**, **TIPS** (selected), stop β-blocker if SBP < 90 mmHg or AKI, and **refer for liver transplantation**.
Hepatic hydrothorax	Sodium restriction + diuretics; therapeutic thoracentesis; **do not insert a chest drain**; TIPS or transplant if refractory.
Hyponatraemia	Na 126–135: no action; < 125: stop diuretics, restrict fluid to 1–1.5 L, give albumin; hypertonic saline only for severe symptoms; vaptans are not recommended.

B. Variceal bleeding — acute and long-term

Step	Action
Resuscitate	ABC; two large-bore lines; **restrictive transfusion — haemoglobin target 7–8 g/dL** (over-transfusion raises portal pressure and rebleeding); correct hypovolaemia gently; airway protection if encephalopathy or massive bleed; **no sedatives**.
Vasoactive drug — start at once, before endoscopy	**Terlipressin** 2 mg IV 4-hourly (then 1 mg 4-hourly) or **octreotide** 50 µg bolus then 50 µg/h; continue 2–5 days.
Antibiotic prophylaxis	**Ceftriaxone 1 g IV daily** (up to 7 days) — reduces infection, rebleeding and mortality.
Endoscopy within 12 hours	**Endoscopic variceal ligation (EVL)** for oesophageal varices; cyanoacrylate glue for gastric varices. Balloon tamponade (Sengstaken–Blakemore) or covered metal stent as a bridge if uncontrolled.
High-risk patients	**Pre-emptive TIPS** within 72 hours for Child–Pugh C (score 10–13) or B (> 7) with active bleeding.
Secondary prophylaxis (this patient)	**Non-selective β-blocker** (propranolol 20–40 mg BD or carvedilol 6.25–12.5 mg/day, titrate to heart rate 55–60, SBP > 90) **+ EVL every 2–4 weeks until eradication**; TIPS if rebleeding.
Primary prophylaxis	Large varices without bleed, or clinically significant portal hypertension: **carvedilol** (preferred) or propranolol, or EVL if β-blocker is not tolerated.

C. Spontaneous bacterial peritonitis (SBP) · encephalopathy · hepatorenal syndrome

Complication	Diagnosis	Treatment and prevention
SBP	Ascitic **PMN ≥ 250/mm³** (no surgical source); organisms: *E. coli*, *Klebsiella*, *S. pneumoniae*	**Ceftriaxone 2 g/day IV** (or cefotaxime 2 g 8-hourly) for 5–7 days + **albumin 1.5 g/kg on day 1 and 1 g/kg on day 3**; repeat tap at 48 h. **Prophylaxis:** ciprofloxacin 500 mg OD or norfloxacin 400 mg OD after an episode; primary prophylaxis if ascitic protein < 1.5 g/dL with Child–Pugh ≥ 9 and bilirubin ≥ 3, or renal dysfunction or Na ≤ 130
Hepatic encephalopathy	Clinical (West Haven); ammonia not needed; exclude other causes of confusion	**Treat the precipitant** (bleed, infection, constipation, electrolytes, sedatives); **lactulose 30–45 mL 2–3 times daily** to 2–3 soft stools a day (enemas if comatose); **add rifaximin 550 mg BD** after a second episode; **no protein restriction**; avoid benzodiazepines
HRS-AKI	Cirrhosis + ascites; creatinine rise ≥ 0.3 mg/dL in 48 h or ≥ 50 %; **no response to 2 days of diuretic withdrawal and albumin 1 g/kg/day**; no shock, nephrotoxin or structural disease	**Stop diuretics, β-blocker and nephrotoxins; albumin 1 g/kg/day (max 100 g) + terlipressin** (bolus 0.5–1 mg every 4–6 h or infusion) or norepinephrine in ICU; treat infection; renal replacement as a bridge; **liver transplantation** is the definitive therapy

D. Treat the cause and prevent decompensation

Measure	Details
Alcohol abstinence	The most effective treatment of alcohol-related cirrhosis; counselling and de-addiction referral; **thiamine 100 mg daily**, folate; **baclofen** is the best-studied anti-craving drug in cirrhosis; avoid disulfiram. Watch for withdrawal.
Hepatitis B / C	Hepatitis B: tenofovir or entecavir lifelong; hepatitis C: sofosbuvir-based direct-acting antivirals (can be used in decompensated disease with expert care).
MASLD, others	Weight loss, diabetes and lipid control; immunosuppression for autoimmune hepatitis; chelation for Wilson disease; venesection for haemochromatosis.
Nutrition	Energy 35 kcal/kg, protein 1.2–1.5 g/kg, late-evening snack, branched-chain amino acids if intolerant of protein; vitamin D, zinc; treat sarcopenia.
Vaccines	Hepatitis A and B, pneumococcal, influenza, COVID-19, tetanus.
HCC surveillance	**USG abdomen ± AFP every 6 months** in all patients with cirrhosis.
Drugs to avoid	NSAIDs, ACE inhibitors/ARBs, aminoglycosides, sedatives/opioids, herbal and Ayurvedic tonics of unknown content; paracetamol ≤ 2 g/day is safe; no alcohol.
Liver transplantation	Refer at the **first major decompensation or MELD ≥ 15**. Indications: refractory ascites, recurrent bleeding or encephalopathy, HRS, HCC within criteria. Living-donor transplantation is widely available in India. Abstinence and a supportive family are required.

E. Discharge prescription (sample)

Drug	Dose	Remarks
Tab. Spironolactone	100 mg OD (morning)	Titrate; K^+ and creatinine at 1 week
Tab. Furosemide	40 mg OD (morning)	Weight daily — target loss 0.5 kg/day
Tab. Propranolol (or carvedilol)	20 mg BD (carvedilol 3.125 mg BD → 6.25 mg BD)	Hold if SBP < 90, HR < 55, AKI or SBP; start low — BP is 100/60
Syp. Lactulose	30 mL TDS	Adjust to 2–3 soft stools a day
Tab. Rifaximin	550 mg BD	For recurrent encephalopathy
Tab. Ciprofloxacin (or norfloxacin)	500 mg OD (norfloxacin 400 mg OD)	Primary SBP prophylaxis: ascitic protein 1.2, Child C
Tab. Thiamine, folic acid, multivitamin; vitamin D	Thiamine 100 mg OD; folic acid 5 mg OD	Nutritional support
Diet and advice	2 g sodium; protein 1.2–1.5 g/kg; no alcohol; no NSAIDs or herbal tonics	Written plan; family counselling
Procedures	Repeat EVL in 2–4 weeks; LVP with albumin as needed; vaccines	Review in 1 week with sodium, potassium, creatinine
Referral	Liver transplant evaluation; de-addiction clinic; dietician	MELD-Na ≈ 24

F. Complications of cirrhosis and prognosis

System	Complication
Portal hypertension	Ascites, variceal bleeding, portal hypertensive gastropathy, hypersplenism, hepatic hydrothorax, hepatopulmonary syndrome, portopulmonary hypertension
Liver failure	Hepatic encephalopathy, coagulopathy, jaundice, hypoalbuminaemia, hypoglycaemia, sarcopenia
Infection	SBP, UTI, pneumonia, cellulitis, bacteraemia — often the first sign of decompensation
Kidney	Pre-renal AKI, hepatorenal syndrome, hyponatraemia
Neoplastic	**Hepatocellular carcinoma** (1–4 % a year)
Others	Hepatic osteodystrophy, type 2 diabetes, hypogonadism, portal vein thrombosis

Prognosis: cirrhosis is progressive but the course can be changed. Poor prognostic factors in this patient: **ascites with hyponatraemia, jaundice, prolonged INR, low albumin, a previous variceal bleed, encephalopathy, malnutrition and Child–Pugh class C (score 13)**; MELD ≈ 18 (MELD-Na ≈ 24). Favourable factors: he has stopped alcohol, has family support, and can benefit from β-blocker + banding, albumin and diuretics, nutrition and **transplant referral**.

Examiner's tip — answer "How will you manage this patient?" in four parts
(1) **The acute event** — tap, albumin + LVP, treat the precipitant, lactulose; vasoactive + antibiotic + EVL if bleeding. (2) **The complications** — SBP prophylaxis, hyponatraemia, HRS prevention (avoid NSAIDs). (3) **The cause** — alcohol abstinence, antivirals. (4) **Prevention** — β-blocker + banding, nutrition, vaccines, HCC surveillance, transplant referral.

SECTION F · PRESENTATION

How to Present This Case in the Examination

A 5-minute presentation that impresses the examiner

Structure of the presentation

- **Introduce** — name (initials), age, sex, occupation, place.
- **Chief complaints** with duration — chronic first, then the decompensation.
- **HOPI** — ascites, jaundice, bleed, confusion, fever/pain, urine; precipitant; relevant negatives (cardiac, renal, TB).
- **Aetiology** — alcohol in g/day and years, viral risks, drugs and herbs, metabolic, family; treatment and adherence.
- **Summary of history** and your provisional diagnosis.
- **General examination** — stigmata of CLD, icterus, oedema, JVP, flap, vitals.
- **Abdomen** — inspection, palpation (liver, spleen), percussion (ascites tests), auscultation; then respiratory and nervous systems.
- **Diagnosis** in the complete format with Child–Pugh; differential only if asked; investigations you would request.

Model presentation (read aloud and practise until you can say it in 4–5 minutes)

Sir / Madam, I would like to present the case of…
"…Mr K. R., a **48-year-old mason from Chalakudy**, who presented with **progressive abdominal distension for 3 months, bilateral leg swelling for 2 months, yellowish discolouration of the eyes for 1 month**, **one episode of haematemesis and melaena 6 weeks ago**, and **sleep reversal with irrelevant talk for 4 days**. The distension was gradual and painless and preceded the leg swelling; there is no fever, abdominal pain, orthopnoea, frothy urine, haemoptysis or TB contact. The bleed was treated with transfusion and band ligation; he stopped propranolol after three weeks. He took diclofenac and an Ayurvedic tonic, was constipated and eating a high-salt diet.

He drank 180 mL of rum a day for 22 years — about 57 g of alcohol daily — and has been abstinent for 6 weeks. There is no history of transfusion before this admission, tattoos, injection drugs, diabetes or family liver disease other than an alcoholic father.

On examination he is thin with **muscle wasting, icterus, six spider naevi, palmar erythema, leuconychia, Dupuytren's contracture, gynaecomastia, parotid enlargement and fetor hepaticus**; bilateral pitting oedema; **JVP not raised**; pulse 98, bounding; BP 100/60; no fever; flap absent today. The abdomen is **distended with bulging flanks, everted umbilicus, caput medusae and a venous hum**. **Shifting dullness and fluid thrill are positive — large-volume (grade 3) ascites**. The **liver is small, not palpable, span 8 cm; the spleen is palpable 4 cm**. There is a small right pleural effusion.

So my diagnosis is **alcohol-related cirrhosis of the liver, decompensated, Child–Pugh class C (13), with portal hypertension — grade 3 ascites, splenomegaly and large oesophageal varices with a previous bleed — complicated by hepatic encephalopathy (grade 2, resolved) and hyponatraemia, precipitated by constipation, NSAIDs and non-adherence**. I would like to confirm with an **ascitic fluid analysis (SAAG and cell count), ultrasound with Doppler, endoscopy, liver function tests and INR**."

Do's and don'ts during presentation

Do	Don't
Quantify alcohol in g/day and years	Say "alcoholic" or "drinker" without numbers
State "JVP not raised" and why it matters	Call every ascites "cirrhotic" without excluding cardiac, TB, malignancy
Demonstrate shifting dullness with a marked boundary	Skip the percussion note from midline to flank
Mention the precipitant of decompensation	Present the case as a list of findings without a cause
Give the Child–Pugh class and what it means	Diagnose cirrhosis without mentioning the cause and the portal hypertension
Say you would do a diagnostic paracentesis	Quote INR as a reason not to tap

SECTION G · VIVA VOCE

Frequently Asked Viva Questions with Answers

Short, exam-ready answers

Q1 Define cirrhosis.

A diffuse, **irreversible (late stage) transformation of the liver** into regenerative nodules surrounded by fibrous septa, with distortion of the vascular architecture. Causes **portal hypertension** and **liver failure**. The diagnosis is histological, but clinically from stigmata, imaging and elastography.

Q2 Name the stigmata of chronic liver disease.

Jaundice, spider naevi, palmar erythema, leuconychia, Dupuytren's contracture, clubbing, gynaecomastia, testicular atrophy, loss of axillary/pubic hair, parotid enlargement, muscle wasting, bruising, fetor, flapping tremor, ascites, caput medusae, splenomegaly.

Q3 Why does ascites form in cirrhosis?

Portal hypertension → **splanchnic arterial vasodilatation (nitric oxide)** → effective arterial under-filling → activation of renin–angiotensin–aldosterone, sympathetic system and ADH → **sodium and water retention**; with ↑ sinusoidal hydrostatic pressure, ↓ albumin (oncotic pressure) and ↑ lymph formation.

Q4 What is SAAG and why is it useful?

Serum albumin − ascitic albumin. It reflects the **hydrostatic–oncotic pressure difference** across the sinusoids: ≥ 1.1 g/dL means portal hypertension (cirrhosis, cardiac, Budd–Chiari); < 1.1 g/dL means non-portal ascites (TB, malignancy, nephrotic, pancreatic). Accuracy ≈ 97 %.

Q5 How do you detect ascites clinically?

Bulging flanks, everted umbilicus, **flank dullness** (≈ 1–1.5 L), **shifting dullness** (≥ 1 L), **fluid thrill** (large volume), horse-shoe dullness, puddle sign. USG detects very small amounts.

Q6 How do you elicit shifting dullness? Why does it shift?

Percuss from the umbilicus to the flank until the note turns dull; keep the finger there, roll the patient to the opposite side for a minute and percuss again. Free fluid gravitates to the dependent flank, so the **upper flank becomes resonant** as gas-filled bowel floats up.

Q7 Why does the fluid thrill need an assistant?

The assistant's hand edge pressed along the midline **blocks the transmission of the impulse through the abdominal wall fat**, so only a wave through the fluid reaches the other hand.

Q8 How do you differentiate ascites from an ovarian cyst?

Ascites: **central tympany, dull flanks, shifting dullness**. Large ovarian cyst: **central dullness, resonant flanks**, no shifting dullness, pelvic mass, transmitted aortic pulsation. USG confirms.

Q9 Define portal hypertension. What is HVPG?

Sustained rise in portal pressure with HVPG > 5 mmHg; clinically significant ≥ 10 mmHg (varices, ascites); ≥ 12 mmHg bleeding risk. HVPG = wedged − free hepatic venous pressure, measured by catheter.

Q10 Name the sites of portosystemic anastomoses.

Lower oesophagus (left gastric–azygos), **umbilicus** (paraumbilical–epigastric: caput), **anorectal junction** (superior–middle/inferior rectal), **retroperitoneum** and the bare area of the liver; splenorenal and gastro-renal shunts.

Q11 What is caput medusae? What is the Cruveilhier–Baumgarten syndrome?

Dilated, tortuous veins radiating from the umbilicus due to recanalised paraumbilical veins. **Cruveilhier–Baumgarten syndrome** — a venous hum heard over the epigastrium/umbilicus with caput medusae, due to a patent paraumbilical vein. Flow is **away from the navel**.

Q12 Why is the liver small and not palpable in advanced cirrhosis?

Fibrosis and loss of hepatocytes shrink the liver (especially the right lobe); it lies high under the costal margin; ascites floats it away. A palpable firm liver with sharp edge in cirrhosis suggests early cirrhosis, alcoholic hepatitis, fatty liver or HCC.

Q13 Why is the spleen enlarged? What is hypersplenism?

Congestion from portal hypertension. **Hypersplenism** = splenomegaly with **cytopenias** (thrombocytopenia first), a cellular marrow, and correction after splenectomy.

Q14 Explain spider naevi, palmar erythema and gynaecomastia.

The failing liver does not inactivate **oestrogens** (and ↑ conversion of androgens to oestrogen): spider naevi (central arteriole), palmar erythema (vasodilatation) and gynaecomastia. Spironolactone also causes gynaecomastia.

Q15 What is leuconychia? What is clubbing's relation to liver disease?

White nails with ↓ albumin (Terry's nails: distal pink band). Clubbing occurs in biliary cirrhosis, hepatopulmonary syndrome and cirrhosis of other causes in a minority of patients.

Q16 What is asterixis? Mechanism? Causes?

Negative myoclonus — sudden lapse of sustained posture, from impaired integration in the brain stem and thalamus. Causes: hepatic, uraemic, CO_2 narcosis, drugs (phenytoin), hypomagnesaemia.

Q17 Pathogenesis of hepatic encephalopathy and precipitants?

Gut-derived ammonia bypasses the liver (portosystemic shunt), enters astrocytes as **glutamine** → swelling, neuroinflammation, ↑ GABAergic tone. **Precipitants:** GI bleed, infection, constipation, dehydration/diuretics, hypokalaemia, alkalosis, sedatives, TIPS, renal failure, alcohol, high protein load.

Q18 How do you grade hepatic encephalopathy?

West Haven: grade 1 — sleep reversal, short attention; 2 — lethargy, disorientation, asterixis; 3 — somnolent, confused, rousable; 4 — coma. Covert (minimal/grade 1) vs overt (≥ 2).

Q19 How do you treat hepatic encephalopathy?

Find and treat the precipitant; **lactulose** to 2–3 soft stools a day; **rifaximin** 550 mg BD for recurrence; no protein restriction; avoid sedatives; consider ICU/airway in grade 3–4.

Q20 Describe the Child–Pugh score.

Bilirubin, albumin, INR, ascites, encephalopathy, each 1–3 points; A 5–6, B 7–9, C 10–15. One-year survival ≈ 100 %, 80 %, 45 %. Used for prognosis and drug dosing.

Q21 What is MELD? How is it used?

Based on **creatinine, bilirubin and INR** (± sodium: MELD-Na). Predicts 3-month mortality and **ranks patients for liver transplantation**; ≥ 15 is the usual threshold for listing.

Q22 Define SBP. Organisms, treatment and prophylaxis?

Ascitic fluid infection with **PMN ≥ 250/mm³** without a surgical source. Gram-negative enteric bacteria (*E. coli*, *Klebsiella*) and *S. pneumoniae*. **Ceftriaxone** + **albumin** (1.5 g/kg day 1, 1 g/kg day 3); prophylaxis with ciprofloxacin or norfloxacin.

Q23 What is hepatorenal syndrome?

A **functional kidney injury** due to extreme renal vasoconstriction in advanced cirrhosis with ascites; diagnosis of exclusion. **HRS-AKI** (rapid, formerly type 1) and HRS-CKD (slow, type 2). Treat with **albumin + terlipressin**; transplantation is definitive.

Q24 Outline the management of grade 3 ascites.

Diagnostic tap, then **large-volume paracentesis with albumin** (6–8 g/L), **2 g sodium diet**, spironolactone + furosemide (100:40), avoid NSAIDs, treat the cause; refractory → serial LVP, TIPS, transplant.

Q25 Why spironolactone first? What is the dose ratio?

Ascites is driven by **hyperaldosteronism**; spironolactone acts on the distal tubule and spares potassium; furosemide adds natriuresis. Ratio **100 : 40** keeps serum potassium normal.

Q26 Why albumin after large-volume paracentesis?

To prevent **post-paracentesis circulatory dysfunction** (a fall in effective volume with renin rise, hyponatraemia and renal impairment) — 6–8 g per litre removed when > 5 L.

Q27 What is refractory ascites?

Ascites that cannot be mobilised or recurs early despite **sodium restriction and maximal diuretics** (400/160 mg for 1 week), or that cannot be treated because of side-effects of diuretics. Median survival ≈ 6 months without transplant.

Q28 Management of acute variceal bleeding?

ABCs, restrictive transfusion (Hb 7–8), terlipressin/octreotide, ceftriaxone, EVL within 12 h; balloon tamponade or stent if uncontrolled; pre-emptive TIPS in Child C ≤ 13; treat encephalopathy risk (lactulose).

Q29 Primary and secondary prophylaxis of variceal bleeding?

Primary: carvedilol/propranolol or EVL. Secondary: **NSBB + EVL**. Avoid β-blockers in refractory ascites with SBP < 90, AKI or HRS.

Q30 Why antibiotics and vasoactive drugs in variceal bleeding?

Vasoactive drugs cause **splanchnic vasoconstriction** → ↓ portal inflow and pressure. Antibiotics prevent **bacterial infection and rebleeding** and reduce mortality.

Q31 How do you differentiate cirrhosis from NCPF/EHPVO?

NCPF: young, **massive splenomegaly**, bleeding well tolerated, **normal liver function**, no stigmata or ascites, patent hepatic veins. Cirrhosis: small nodular liver, stigmata, ascites, low albumin and ↑ INR.

Q32 What are the commonest causes of cirrhosis in India?

Alcohol, **hepatitis B**, hepatitis C, **MASLD**; others — autoimmune, Wilson, haemochromatosis, cryptogenic; vascular (Budd–Chiari) and NCPF cause portal hypertension without cirrhosis.

Q33 What is the significance of AST:ALT ratio?

AST:ALT ≥ 2 with GGT elevated and AST < 300 U/L suggests alcohol-related injury (pyridoxine deficiency lowers ALT; mitochondrial AST). In advanced cirrhosis of any cause AST may exceed ALT.

Q34 Hepatopulmonary syndrome vs portopulmonary hypertension?

HPS: **intrapulmonary vasodilatation** → hypoxaemia, platypnoea–orthodeoxia, clubbing, spider naevi; contrast echo positive. POPH: **pulmonary arterial hypertension** with cirrhosis — dyspnoea, RV failure. Both are transplant considerations.

Q35 What is hepatic hydrothorax?

Pleural effusion (usually **right-sided**, 85 %) in cirrhosis from movement of ascitic fluid through diaphragmatic defects. Treat as ascites; avoid chest drains; TIPS/transplant if refractory.

Q36 Which drugs are dangerous in cirrhosis?

NSAIDs (renal failure, bleeding), ACE inhibitors/ARBs, aminoglycosides, **benzodiazepines and opioids** (encephalopathy), herbal remedies, high-dose paracetamol (> 2 g/day).

Q37 How do you follow up a patient with cirrhosis?

USG ± AFP every 6 months (HCC), endoscopy for varices, nutrition, vaccines, treat cause, track Child–Pugh and MELD, refer for transplant.

Q38 When do you refer for liver transplantation?

At the **first major decompensation** (ascites, bleed, encephalopathy) or **MELD ≥ 15**, refractory ascites, HRS, HCC within Milan criteria. Alcohol abstinence and social support are required.

Q39 How is a diagnostic paracentesis done? Complications?

Supine or lateral, **left iliac fossa** 2–3 cm above and medial to the ASIS (avoid epigastric vessels), ultrasound guidance, Z-track, 20–22G needle; send cells, albumin, protein, culture in bottles. Complications: leak, haematoma, infection, bowel injury. Safe despite raised INR.

Q40 What is the role of elastography and platelets in portal hypertension?

Liver stiffness > 25 kPa rules in clinically significant portal hypertension; < 15 kPa with platelets > 150,000 safely avoids endoscopy (Baveno VII).

Q41 How do you grade oesophageal varices?

Small (< 5 mm) and large (≥ 5 mm) (Baveno); older scale I–IV by size, red wale marks, cherry-red spots and gastric varices (GOV1, GOV2, IGV1, IGV2) indicate bleeding risk.

Q42 What is the prognosis of decompensated cirrhosis?

Median survival ≈ 2 years after the first decompensation; Child C one-year survival ≈ 45 %; transplantation improves survival to > 80 % at 5 years.

Last-minute checklist before you go to the examiner

✔ Alcohol in g/day and years · ✔ viral, drug and herbal history · ✔ five decompensation events · ✔ precipitant · ✔ adherence and salt · ✔ icterus, spider naevi, palmar erythema, leuconychia, gynaecomastia · ✔ JVP and oedema · ✔ flap and orientation · ✔ inspection of veins and direction of flow · ✔ liver span and spleen · ✔ shifting dullness and fluid thrill · ✔ hernial orifices, genitalia, PR · ✔ SAAG and PMN · ✔ complete diagnosis with Child–Pugh · ✔ management in four parts.

VISUAL ATLAS

See the Signs — Tap or Scan

Free photographs, videos, ultrasound and CT cases, guidelines and calculators — from Radiopaedia, NCBI, YouTube and other open sites

Abdominal examination — video 1
► Video
Full abdominal examination

Abdominal examination — video 2
► Video
Ascites, liver and spleen technique

Ultrasound — how it is done
► Video
USG technique and views

USG abdomen — liver, spleen, portal vein
► Video
Normal and abnormal findings

Portal hypertension — endoscopy
► Video
Gastropathy, enteropathy, colopathy

Asterixis (liver flap)
► Video
Flapping tremor of outstretched hands

Caput medusae
► Photo
Veins radiating from the umbilicus

Spider naevi & palmar erythema
► Photo
Vascular signs of liver disease

Skin signs of liver disease
► Article
Cutaneous markers of cirrhosis

14 signs of chronic liver disease
► Article
Liver-focused physical examination

Abdominal examination OSCE guide
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Step-by-step free guide

Ascitic tap (paracentesis)
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Diagnostic paracentesis guide

USG — cirrhosis + portal hypertension
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Radiopaedia case with images

USG — full spectrum of cirrhosis
► USG
Small nodular liver, ascites, splenomegaly

USG — gross ascites in cirrhosis
► USG
Radiopaedia case

CT — cirrhosis with ascites
► CT
Nodular liver, caudate hypertrophy

Ascites — StatPearls
► Article
Free NCBI Bookshelf review

Ascites — diagnostic review
► Article
Neth J Med full text

Bedside diagnosis of ascites
► Article
Accuracy of clinical signs (PubMed)

Ascites — quick notes
► Article
Exam, SAAG, fluid analysis

AASLD 2021 ascites, SBP, HRS
► Guideline
Practice guidance

Baveno VII portal hypertension
► Guideline
Consensus statement (free full text)

West Haven criteria
► Article
Grading hepatic encephalopathy

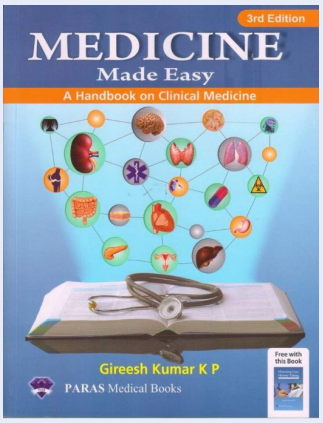
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Links open third-party sites; page content and availability can change. Only free resources are listed. The case-report and image pages show photographs that illustrate the sign, not a particular patient of this case.

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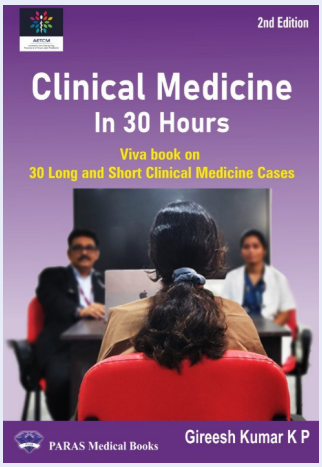
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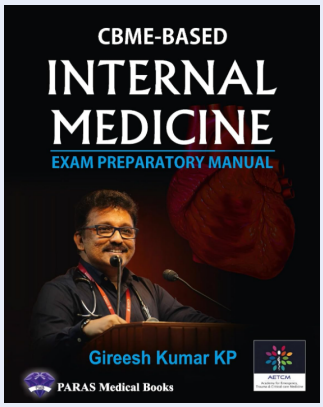
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