FINAL YEAR MBBS · CLINICAL VIVA

Clinical Medicine Viva Voce

Common long and short cases for MBBS clinical exams
Based on *Davidson's Principles and Practice of Medicine*

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1000 viva questions with model answers · 20 cases

How to use this book

Each chapter is one common examination case. It opens with the typical case you may meet in the exam, followed by 50 questions that examiners commonly ask, from simple to hard: history and examination, diagnosis, investigations, management and complications.

The answers are short model answers, the way an examiner wants to hear them. Read the question, answer it aloud first, then check the model answer. Guidance follows UK and international guidelines (NICE, ESC, GINA, GOLD, WHO) as taught in Davidson; follow your local hospital protocols where they differ.

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CASE 1

Diabetes Mellitus

> **Typical exam case:** A 52-year-old man with type 2 diabetes for 10 years presents with burning feet, blurred vision and poor glucose control. He takes metformin and gliclazide. His HbA1c is 75 mmol/mol (9%).

HISTORY AND EXAMINATION

Q1. What symptoms of hyperglycaemia would you ask about?

Polyuria, polydipsia, weight loss, tiredness, blurred vision and recurrent infections (thrush, boils, UTIs).

Q2. What complications would you ask about?

Microvascular: eyes (vision), kidneys (frothy urine, swelling), nerves (numbness, burning feet, erectile dysfunction). Macrovascular: angina, stroke, claudication.

Q3. What symptoms suggest autonomic neuropathy?

Postural dizziness, gastroparesis (early fullness, vomiting), diarrhoea, bladder problems, erectile dysfunction and sweating changes.

Q4. What should you ask about hypoglycaemia?

Frequency, warning symptoms (sweating, tremor, hunger), night sweats, and episodes needing help.

Q5. What drug history is important?

Diabetes medicines and doses, adherence, steroids, thiazides and antipsychotics that raise glucose.

Q6. What would you examine in a diabetic patient?

BMI, BP (lying and standing), fundi, feet, pulses, sensation, injection sites, thyroid, and signs of hyperlipidaemia.

Q7. What do you look for in the feet?

Skin colour, ulcers, calluses, deformity (Charcot), nail infection, pulses, and sensation with a 10 g monofilament and tuning fork.

Q8. What is the pattern of diabetic peripheral neuropathy?

Symmetrical 'glove and stocking' sensory loss, starting in the feet, with loss of ankle reflexes and vibration sense.

Q9. What are signs of insulin resistance?

Central obesity and acanthosis nigricans.

Q10. What is lipohypertrophy?

Fatty lumps at repeated insulin injection sites that cause erratic insulin absorption; sites should be rotated.

CLASSIFICATION AND DIAGNOSIS

Q11. How is diabetes diagnosed?

Fasting plasma glucose 7.0 mmol/L or more, random or 2-hour OGTT glucose 11.1 mmol/L or more, or HbA1c 48 mmol/mol (6.5%) or more. Without symptoms, a second test is needed.

Q12. What is prediabetes?

Impaired fasting glucose (6.1–6.9 mmol/L), impaired glucose tolerance (2-hour 7.8–11.0 mmol/L) or HbA1c 42–47 mmol/mol.

Q13. How does type 1 differ from type 2 diabetes?

> Type 1 is autoimmune destruction of beta cells with absolute insulin deficiency, usually in young lean people prone to DKA. Type 2 is insulin resistance with relative deficiency, usually in older overweight people.

Q14. What is LADA?

> Latent autoimmune diabetes in adults: slowly progressive autoimmune diabetes that looks like type 2 at first but needs insulin within a few years.

Q15. What is MODY?

> Maturity-onset diabetes of the young: monogenic diabetes, autosomal dominant, often treated with sulphonylureas (HNF1A).

Q16. What are secondary causes of diabetes?

> Pancreatitis, pancreatic cancer, haemochromatosis, cystic fibrosis, Cushing's, acromegaly, phaeochromocytoma and drugs (steroids).

Q17. When is HbA1c unreliable?

> In haemoglobinopathies, anaemia, recent blood loss or transfusion, pregnancy and kidney failure.

Q18. Which antibodies support type 1 diabetes?

> GAD, IA-2 and ZnT8 antibodies; low C-peptide also supports it.

INVESTIGATIONS

Q19. What does HbA1c measure?

> Average blood glucose over the past 2–3 months.

Q20. What is the usual HbA1c target?

> Around 48–53 mmol/mol (6.5–7%), individualised; less strict in frail elderly patients.

Q21. How is diabetic kidney disease screened?

> Urine albumin:creatinine ratio and eGFR yearly.

Q22. What ACR shows microalbuminuria?

> 3–30 mg/mmol (moderately increased albuminuria).

Q23. How is retinopathy screened?

> Annual digital retinal photography.

Q24. What other yearly checks are done?

> BP, lipids, foot examination, weight, smoking status and HbA1c.

Q25. What lipid target is used?

> Statin therapy aiming for a 40% fall in non-HDL cholesterol.

Q26. Why check B12 in patients on metformin?

> Long-term metformin can cause B12 deficiency, which worsens neuropathy.

Q27. What tests are done if DKA is suspected?

> Capillary glucose, blood ketones, venous gas (pH, bicarbonate), U&E and potassium.

Q28. What test helps if the type of diabetes is unclear?

> C-peptide and islet autoantibodies.

MANAGEMENT

Q29. What is first-line drug therapy for type 2 diabetes?

> Metformin, with diet and exercise advice.

Q30. How does metformin work?

> It reduces liver glucose output and improves insulin sensitivity; it does not cause hypoglycaemia or weight gain.

Q31. When is metformin stopped?

> If eGFR is below 30 mL/min, in acute illness with dehydration, and before iodinated contrast in some cases (lactic acidosis risk).

Q32. Which drugs are added for patients with heart disease or heart failure?

> SGLT2 inhibitors (e.g. empagliflozin, dapagliflozin) because they reduce heart failure and cardiovascular events.

Q33. Which drug class protects the kidneys in diabetic kidney disease?

> SGLT2 inhibitors, together with ACE inhibitors or ARBs.

Q34. What are the side effects of SGLT2 inhibitors?

> Genital thrush, UTIs, dehydration and euglycaemic DKA.

Q35. What are GLP-1 receptor agonists?

> Injectable drugs (e.g. semaglutide, liraglutide) that raise insulin, lower glucagon, slow stomach emptying and cause weight loss.

Q36. What is the main side effect of sulphonylureas?

> Hypoglycaemia and weight gain.

Q37. When is insulin started in type 2 diabetes?

> When HbA1c remains high on oral drugs, in symptomatic hyperglycaemia, pregnancy or acute illness.

Q38. What is a basal-bolus insulin regimen?

> Long-acting insulin once daily plus rapid-acting insulin with meals; standard for type 1 diabetes.

Q39. How is hypoglycaemia treated in a conscious patient?

> 15–20 g of fast-acting glucose (glucose tablets, juice), recheck in 15 minutes, then a starchy snack.

Q40. How is hypoglycaemia treated in an unconscious patient?

> IV glucose (e.g. 100 mL of 20% glucose) or IM glucagon 1 mg.

Q41. What are the sick day rules?

> Never stop insulin, check glucose and ketones more often, keep drinking, and stop metformin and SGLT2 inhibitors if dehydrated.

Q42. How is painful diabetic neuropathy treated?

> Duloxetine, amitriptyline, pregabalin or gabapentin.

Q43. What BP target is used in diabetes?

> Below 140/90 mmHg, or below 130/80 mmHg with kidney disease, using an ACE inhibitor or ARB first line.

COMPLICATIONS AND RECENT ADVANCES

Q44. What is diabetic ketoacidosis?

> High glucose (over 11 mmol/L), ketones over 3 mmol/L and acidosis (pH below 7.3 or bicarbonate below 15), mainly in type 1 diabetes.

Q45. How is DKA treated?

> IV fluids, fixed-rate IV insulin (0.1 unit/kg/hour), potassium replacement, and treating the cause.

Q46. What is the hyperosmolar hyperglycaemic state?

> Very high glucose (often over 30 mmol/L) and high osmolality without significant ketosis, usually in older type 2 patients; mainly treated with fluids.

Q47. What are the stages of diabetic retinopathy?

> Background (microaneurysms, dot and blot haemorrhages, hard exudates), pre-proliferative (cotton wool spots), proliferative (new vessels) and maculopathy.

Q48. How is proliferative retinopathy treated?

> Pan-retinal laser photocoagulation or anti-VEGF injections.

Q49. What is a Charcot foot?

> Destruction of foot joints due to neuropathy, presenting as a warm, swollen foot; treated by offloading in a cast.

Q50. What is a recent advance in diabetes care?

> Continuous glucose monitoring and closed-loop insulin pumps for type 1, and SGLT2 inhibitors and GLP-1 agonists (including tirzepatide) with heart and kidney benefits in type 2.

CASE 2

Hypertension

> **Typical exam case:** A 48-year-old man is found to have a BP of 168/104 mmHg on two clinic visits. He smokes and is overweight. He has occasional headaches.

HISTORY AND EXAMINATION

Q1. What symptoms would you ask about?

Usually none. Ask about headache, visual problems, chest pain, breathlessness, palpitations and symptoms of stroke.

Q2. What history suggests a secondary cause?

Young age, sudden onset, resistant hypertension, episodes of sweating and palpitations (phaeochromocytoma), muscle weakness (Conn's), snoring (sleep apnoea) and kidney disease.

Q3. What drugs can raise BP?

NSAIDs, steroids, combined pill, decongestants, liquorice, cocaine and some antidepressants.

Q4. What lifestyle factors would you ask about?

Salt, alcohol, smoking, exercise, diet and stress.

Q5. What family history is relevant?

Hypertension, early heart disease, stroke and kidney disease.

Q6. How should BP be measured in clinic?

Seated, after 5 minutes rest, arm supported at heart level, correct cuff size, in both arms at the first visit.

Q7. Why measure BP in both arms?

A difference over 15 mmHg suggests subclavian stenosis or aortic coarctation; use the higher arm.

Q8. Why check for radiofemoral delay?

To detect coarctation of the aorta.

Q9. What signs of end-organ damage would you look for?

Left ventricular hypertrophy (heaving apex), fundal changes, signs of heart failure and reduced pulses.

Q10. What abdominal signs might suggest a secondary cause?

Enlarged kidneys (polycystic kidney disease) and a renal bruit (renal artery stenosis).

DIAGNOSIS AND CLASSIFICATION

Q11. How is hypertension diagnosed according to NICE?

Clinic BP 140/90 mmHg or higher, confirmed by ambulatory (ABPM) or home BP average of 135/85 mmHg or higher.

Q12. What are the NICE stages of hypertension?

Stage 1: ABPM 135/85–149/94. Stage 2: ABPM 150/95 or more. Severe: clinic systolic 180 or diastolic 120 or more.

Q13. What is white-coat hypertension?

High BP in the clinic but normal at home; it is confirmed by ABPM.

Q14. What is masked hypertension?

Normal clinic BP but high BP outside the clinic.

Q15. What proportion of hypertension is primary (essential)?

> About 90–95%.

Q16. What are the main secondary causes?

> Renal disease, renal artery stenosis, primary hyperaldosteronism, phaeochromocytoma, Cushing's, coarctation, sleep apnoea and drugs.

Q17. What is the commonest endocrine cause of secondary hypertension?

> Primary hyperaldosteronism (Conn's syndrome).

Q18. What is accelerated (malignant) hypertension?

> Severe hypertension (usually over 180/120) with retinal haemorrhages or papilloedema; a medical emergency.

INVESTIGATIONS

Q19. What baseline tests are done for all patients?

> Urine dipstick for blood and protein, urine ACR, U&E and eGFR, HbA1c, lipids and a 12-lead ECG.

Q20. Why is an ECG done?

> To look for left ventricular hypertrophy and ischaemia.

Q21. What does fundoscopy show in hypertensive retinopathy?

> Arteriolar narrowing, AV nipping, haemorrhages, cotton wool spots and papilloedema in severe cases.

Q22. How is cardiovascular risk assessed?

> Using QRISK3 to estimate 10-year risk.

Q23. What test screens for Conn's syndrome?

> Aldosterone:renin ratio (high aldosterone, low renin), often with low potassium.

Q24. What test screens for phaeochromocytoma?

> Plasma or 24-hour urine metanephrines.

Q25. What test screens for Cushing's syndrome?

> Overnight dexamethasone suppression test or 24-hour urinary free cortisol.

Q26. What test is used for renal artery stenosis?

> Renal artery Doppler, CT or MR angiography.

Q27. When is echocardiography done?

> To assess LVH and heart function, particularly with symptoms or ECG changes.

Q28. Who should be screened for secondary causes?

> Patients under 40, those with resistant hypertension, low potassium, sudden onset or other clinical clues.

MANAGEMENT

Q29. What lifestyle advice is given?

> Lose weight, reduce salt (under 6 g/day), limit alcohol, stop smoking, regular exercise and a diet rich in fruit and vegetables.

Q30. When is drug treatment started for stage 1 hypertension?

> If under 80 with target organ damage, cardiovascular disease, kidney disease, diabetes or a 10-year risk of 10% or more.

Q31. When is drug treatment started for stage 2?

> In all patients.

Q32. What is first-line therapy for patients under 55 (not Black African or Caribbean origin) or with diabetes?

> An ACE inhibitor or an ARB.

Q33. What is first-line therapy for patients 55 or older or of Black African or Caribbean origin?

> A calcium channel blocker (e.g. amlodipine).

Q34. What is step 2?

> Add a calcium channel blocker or thiazide-like diuretic to an ACE inhibitor/ARB, or vice versa.

Q35. What is step 3?

> ACE inhibitor/ARB plus calcium channel blocker plus thiazide-like diuretic (indapamide).

Q36. What is step 4 (resistant hypertension)?

> Check adherence, then add spironolactone if potassium is 4.5 mmol/L or less, or an alpha- or beta-blocker if higher.

Q37. What is the BP target for patients under 80?

> Clinic BP below 140/90 (home below 135/85).

Q38. What is the BP target for patients 80 and over?

> Clinic BP below 150/90 (home below 145/85).

Q39. What are side effects of ACE inhibitors?

> Dry cough, hyperkalaemia, rising creatinine and angioedema.

Q40. Why should ACE inhibitors and ARBs not be combined?

> Higher risk of hyperkalaemia and kidney injury without added benefit.

Q41. What is the main side effect of amlodipine?

> Ankle swelling, flushing and headache.

Q42. Which antihypertensives are avoided in pregnancy?

> ACE inhibitors, ARBs and thiazides; labetalol, nifedipine and methyldopa are used instead.

Q43. How is hypertensive emergency treated?

> Admission, IV labetalol or nitroprusside or GTN, lowering BP gradually (by no more than 25% in the first hour).

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of hypertension?

> Stroke, myocardial infarction, heart failure, LVH, atrial fibrillation, chronic kidney disease, retinopathy and aortic dissection.

Q45. Why is BP lowered slowly in severe hypertension?

> Rapid lowering can cause stroke or kidney injury because autoregulation is reset.

Q46. Why is a statin often added?

> To reduce overall cardiovascular risk if 10-year QRISK is 10% or more.

Q47. What is the role of home BP monitoring?

> Confirms diagnosis, monitors treatment and improves adherence.

Q48. What is the most common reason for 'resistant' hypertension?

> Poor adherence to medicines.

Q49. How does sleep apnoea affect BP?

> It causes resistant hypertension; CPAP treatment can lower BP.

Q50. What is a recent advance in hypertension?

> Renal denervation for resistant hypertension, and single-pill combination therapy to improve adherence.

CASE 3

Ischaemic Heart Disease and Acute Coronary Syndrome

> **Typical exam case:** A 58-year-old diabetic smoker presents with 40 minutes of crushing central chest pain radiating to the left arm, with sweating and nausea. His ECG shows ST elevation in leads II, III and aVF.

HISTORY AND EXAMINATION

Q1. What features of chest pain suggest cardiac ischaemia?

> Central, crushing or tight pain radiating to the arm, jaw or neck, with sweating, nausea and breathlessness.

Q2. What is stable angina?

> Chest pain brought on by exertion and relieved by rest or GTN within minutes.

Q3. What is unstable angina?

> Angina at rest, new-onset or worsening (crescendo) angina, without a rise in troponin.

Q4. What are the risk factors for coronary artery disease?

> Smoking, hypertension, diabetes, high cholesterol, obesity, family history, male sex, age and South Asian ethnicity.

Q5. Why can diabetics have silent myocardial infarction?

> Autonomic neuropathy can blunt pain; they may present with breathlessness or collapse.

Q6. What would you look for on examination?

> Pulse, BP, signs of shock, heart failure (crackles, raised JVP), new murmurs and risk factors (xanthelasma, corneal arcus).

Q7. What does a new pansystolic murmur after MI suggest?

> Mitral regurgitation from papillary muscle rupture, or a ventricular septal defect.

Q8. What are the differential diagnoses of acute chest pain?

> Aortic dissection, pulmonary embolism, pericarditis, pneumothorax, oesophageal spasm or rupture, and musculoskeletal pain.

Q9. What features suggest aortic dissection?

> Tearing pain radiating to the back, unequal arm BPs, and a widened mediastinum.

Q10. What features suggest pericarditis?

> Sharp pain worse lying flat and better sitting forward, and a pericardial rub.

DIAGNOSIS

Q11. What are the types of acute coronary syndrome?

> STEMI, NSTEMI and unstable angina.

Q12. How is STEMI diagnosed on ECG?

> ST elevation of 1 mm or more in two adjacent limb leads, or 2 mm in two adjacent chest leads, or new left bundle branch block with symptoms.

Q13. Which ECG leads show an inferior MI and which artery is usually involved?

> II, III and aVF; usually the right coronary artery.

Q14. Which leads show an anterior MI?

> V1–V4; the left anterior descending artery.

Q15. Which leads show a lateral MI?

> I, aVL, V5–V6; the circumflex artery.

Q16. What is an NSTEMI?

> Myocardial infarction with a rise in troponin but without ST elevation; ECG may show ST depression or T wave inversion.

Q17. What is the universal definition of MI?

> A rise and/or fall of troponin above the 99th centile with clinical evidence of ischaemia (symptoms, ECG changes, imaging or thrombus).

Q18. Why should right-sided leads be checked in inferior MI?

> To detect right ventricular infarction (ST elevation in V4R), which needs fluids and avoidance of nitrates.

INVESTIGATIONS

Q19. What is the key blood test?

> High-sensitivity troponin, repeated at 0 and 1–3 hours according to local protocol.

Q20. Why is a single normal troponin not enough early on?

> Troponin rises a few hours after onset; a repeat test is needed.

Q21. What other bloods are taken?

> FBC, U&E, glucose, lipids and clotting.

Q22. Why is a chest X-ray done?

> To look for heart failure and other causes such as dissection or pneumothorax.

Q23. What is the role of echocardiography after MI?

> To assess left ventricular function and complications.

Q24. How is stable angina investigated?

> CT coronary angiography as first-line test, or functional tests (stress echo, perfusion scan).

Q25. What is the gold standard for coronary anatomy?

> Invasive coronary angiography.

Q26. Why are ECGs repeated?

> Changes can evolve over time.

Q27. What is the GRACE score?

> A score estimating death risk in ACS, used to guide timing of angiography in NSTEMI.

Q28. Which test monitors arrhythmias after MI?

> Continuous cardiac monitoring (telemetry).

MANAGEMENT

Q29. What is the immediate management of ACS?

> Aspirin 300 mg, pain relief (morphine with an antiemetic), GTN, oxygen only if saturation is below 94%, and a second antiplatelet.

Q30. What is the treatment of choice for STEMI?

> Primary PCI within 120 minutes of diagnosis (ideally 90).

Q31. When is thrombolysis used?

> If primary PCI cannot be given within 120 minutes and symptoms started within 12 hours.

Q32. Name a thrombolytic agent.

> Tenecteplase or alteplase.

Q33. What are absolute contraindications to thrombolysis?

> Previous haemorrhagic stroke, ischaemic stroke in the last 6 months, recent major trauma or surgery, active bleeding, aortic dissection and brain tumour.

Q34. How is NSTEMI managed?

> Aspirin, a second antiplatelet, anticoagulation (fondaparinux), and angiography within 72 hours (sooner if high risk).

Q35. What is dual antiplatelet therapy?

> Aspirin plus a P2Y12 inhibitor (ticagrelor, prasugrel or clopidogrel), usually for 12 months after ACS.

Q36. What secondary prevention drugs are given after MI?

> Aspirin, a second antiplatelet, high-intensity statin, beta-blocker, ACE inhibitor and, if LV dysfunction, an aldosterone antagonist.

Q37. What lifestyle advice is given after MI?

> Stop smoking, Mediterranean diet, exercise, weight loss, and cardiac rehabilitation.

Q38. How is stable angina treated?

> GTN for attacks, beta-blocker or calcium channel blocker, aspirin and statin; revascularisation if symptoms persist.

Q39. When is CABG preferred to PCI?

> Left main stem disease, three-vessel disease, especially with diabetes or reduced LV function.

Q40. When can a patient drive after MI?

> In the UK, usually after 1 week if treated with successful PCI and normal LV function, otherwise 4 weeks (car drivers).

Q41. When can sexual activity be resumed after MI?

> Usually after about 4 weeks if the patient is well; avoid sildenafil with nitrates.

Q42. What is the dose of high-intensity statin after ACS?

> Atorvastatin 80 mg daily.

Q43. Why are nitrates avoided in right ventricular infarction?

> They reduce preload and can cause severe hypotension.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the early complications of MI?

> Arrhythmias (VF, heart block), cardiogenic shock, heart failure, papillary muscle rupture, ventricular septal rupture and free wall rupture.

Q45. What are the late complications of MI?

> Dressler's syndrome (pericarditis), left ventricular aneurysm, heart failure and recurrent ischaemia.

Q46. What is Dressler's syndrome?

> Autoimmune pericarditis 2–10 weeks after MI, with fever and pleuritic pain.

Q47. What does persistent ST elevation weeks after MI suggest?

> Left ventricular aneurysm.

Q48. Which heart block often follows inferior MI?

> Complete heart block; it is often temporary.

Q49. What is the commonest cause of death in the first hours of MI?

> Ventricular fibrillation.

Q50. What is a recent advance in ACS care?

> High-sensitivity troponin 0/1-hour rapid rule-out pathways, and PCSK9 inhibitors and inclisiran for further lowering LDL cholesterol.

CASE 4

Heart Failure

Typical exam case: A 65-year-old man with previous MI presents with increasing breathlessness, orthopnoea, paroxysmal nocturnal dyspnoea and swollen ankles. He has a raised JVP, bibasal crackles and pitting oedema.

HISTORY AND EXAMINATION

Q1. What is heart failure?

A syndrome where the heart cannot pump enough blood to meet the body's needs, or does so only at high filling pressures.

Q2. What are the symptoms of left heart failure?

Breathlessness on exertion, orthopnoea, paroxysmal nocturnal dyspnoea, fatigue and cough with frothy sputum.

Q3. What are the symptoms of right heart failure?

Ankle swelling, abdominal swelling, liver discomfort, nausea and weight gain.

Q4. What is orthopnoea?

Breathlessness lying flat; ask how many pillows the patient uses.

Q5. What is paroxysmal nocturnal dyspnoea?

Waking at night gasping for breath, relieved by sitting up.

Q6. How is severity graded?

The NYHA class I–IV, from no limitation to symptoms at rest.

Q7. What signs would you look for?

Tachycardia, irregular pulse, raised JVP, displaced apex, third heart sound, murmurs, bibasal crackles, pleural effusion, hepatomegaly, ascites and pitting oedema.

Q8. What does a third heart sound suggest?

Rapid ventricular filling in a dilated ventricle; a sign of heart failure in adults.

Q9. What causes of heart failure would you ask about?

Ischaemic heart disease, hypertension, valve disease, alcohol, cardiomyopathy, arrhythmias, thyroid disease and chemotherapy.

Q10. What precipitates acute decompensation?

Infection, ischaemia, arrhythmia (AF), poor adherence, salt or fluid excess, NSAIDs and anaemia.

CLASSIFICATION AND DIAGNOSIS

Q11. What is HFrEF?

Heart failure with reduced ejection fraction (EF 40% or less).

Q12. What is HFpEF?

Heart failure with preserved ejection fraction (EF 50% or more), often in older, hypertensive, diabetic or obese patients.

Q13. What is HFmrEF?

Heart failure with mildly reduced ejection fraction (41–49%).

Q14. What is the commonest cause of heart failure?

Ischaemic heart disease.

Q15. What is cor pulmonale?

> Right heart failure due to lung disease, such as COPD.

Q16. What is high-output heart failure?

> Failure caused by high demand, e.g. severe anaemia, thyrotoxicosis, Paget's disease, AV fistula or beri-beri.

Q17. What are the Framingham criteria?

> Clinical criteria for heart failure combining major signs (e.g. PND, raised JVP, crackles) and minor signs (e.g. ankle oedema, night cough).

Q18. What is the first diagnostic test in suspected heart failure?

> NT-proBNP.

INVESTIGATIONS

Q19. What NT-proBNP level suggests heart failure in non-acute settings?

> Above 400 ng/L needs echocardiography; above 2000 ng/L needs urgent review within 2 weeks.

Q20. What conditions raise BNP apart from heart failure?

> Kidney disease, AF, sepsis, PE, older age and right heart strain.

Q21. What lowers BNP?

> Obesity and treatment with diuretics or ACE inhibitors.

Q22. What is the key imaging test?

> Transthoracic echocardiography to measure EF and find the cause.

Q23. What does the chest X-ray show in heart failure?

> ABCDE: alveolar oedema (bat wings), Kerley B lines, cardiomegaly, upper lobe diversion and pleural effusions.

Q24. What blood tests are done?

> FBC, U&E, LFTs, thyroid function, HbA1c, lipids and iron studies.

Q25. Why check iron studies?

> Iron deficiency is common in heart failure; IV iron improves symptoms.

Q26. What does the ECG help with?

> It may show previous MI, LVH, AF or conduction problems; a normal ECG makes HFrEF less likely.

Q27. When is coronary angiography considered?

> To find ischaemic heart disease that may be treatable by revascularisation.

Q28. What is cardiac MRI used for?

> To characterise cardiomyopathy, infiltration and scarring.

MANAGEMENT

Q29. How is acute pulmonary oedema managed?

> Sit up, oxygen if hypoxic, IV furosemide, IV nitrates if BP allows, CPAP if needed, and treat the cause.

Q30. What are the four 'pillars' of drug treatment for HFrEF?

> ACE inhibitor/ARB or sacubitril-valsartan, a beta-blocker, a mineralocorticoid receptor antagonist and an SGLT2 inhibitor.

Q31. Which beta-blockers are used in heart failure?

> Bisoprolol, carvedilol or nebivolol, started at low dose and increased slowly.

Q32. What is sacubitril-valsartan?

> A neprilysin inhibitor with an ARB; it reduces death and admission more than enalapril in HFrEF.

Q33. Why must ACE inhibitors be stopped 36 hours before starting sacubitril-valsartan?

> To avoid angioedema.

Q34. Which mineralocorticoid receptor antagonists are used?

> Spironolactone or eplerenone; monitor potassium and creatinine.

Q35. What is the role of diuretics?

> Loop diuretics relieve fluid overload and breathlessness but do not improve survival.

Q36. Which drugs should be avoided in heart failure?

> NSAIDs, non-dihydropyridine calcium channel blockers (verapamil, diltiazem) in HFrEF, pioglitazone and some antiarrhythmics.

Q37. What device treatments are used?

> ICD for patients at risk of sudden death, and cardiac resynchronisation therapy (CRT) for EF 35% or less with broad QRS.

Q38. What is the role of digoxin?

> Rate control in AF with heart failure and symptom relief in sinus rhythm.

Q39. How is HFpEF treated?

> Diuretics for fluid, SGLT2 inhibitors, and treatment of BP, AF, diabetes and obesity.

Q40. What lifestyle advice is given?

> Daily weights, salt restriction, fluid advice in severe cases, exercise rehabilitation, stop smoking and limit alcohol.

Q41. Which vaccines are recommended?

> Annual influenza and one-off pneumococcal vaccine.

Q42. What advice is given about daily weights?

> Report a gain of more than 1.5–2 kg in 2 days, which may mean fluid build-up.

Q43. What is the final option for end-stage heart failure?

> Heart transplant or a left ventricular assist device in selected patients, and palliative care.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of heart failure?

> Arrhythmias, sudden death, kidney impairment, thromboembolism, cachexia and depression.

Q45. What electrolyte problems occur with treatment?

> Hypokalaemia with diuretics, hyperkalaemia with ACE inhibitors and spironolactone, and hyponatraemia.

Q46. What is the prognosis of heart failure?

> Poor; about 50% die within 5 years of diagnosis, though modern treatment has improved outcomes.

Q47. Why is cardiac rehabilitation recommended?

> It improves exercise capacity and quality of life and reduces admissions.

Q48. What is cardiorenal syndrome?

> Heart failure and kidney dysfunction worsening each other.

Q49. Why is palliative care discussed early in advanced heart failure?

> Symptom burden is high and prognosis uncertain.

Q50. What is a recent advance in heart failure?

> SGLT2 inhibitors for all types of heart failure, and early rapid up-titration of all four drug classes (STRONG-HF trial).

CASE 5

Rheumatic Heart Disease and Mitral Stenosis

Typical exam case: A 30-year-old woman has breathlessness on exertion and palpitations. She had joint pains and fever in childhood. Her pulse is irregularly irregular. There is a loud first heart sound, an opening snap and a low-pitched mid-diastolic murmur at the apex.

HISTORY AND EXAMINATION

Q1. What is rheumatic fever?

An autoimmune inflammatory illness 2–4 weeks after group A streptococcal throat infection, affecting the heart, joints, skin and brain.

Q2. Why is rheumatic heart disease still common in Pakistan?

Untreated streptococcal sore throats, overcrowding and poor access to penicillin prophylaxis.

Q3. What are the symptoms of mitral stenosis?

Breathlessness, orthopnoea, fatigue, palpitations, haemoptysis, hoarseness (Ortner's syndrome) and embolic events.

Q4. What is the malar flush?

Purple-red cheeks due to low cardiac output and vasoconstriction in severe mitral stenosis.

Q5. What pulse is typical?

Small volume and often irregularly irregular due to atrial fibrillation.

Q6. What is the apex beat like in mitral stenosis?

Tapping (palpable first heart sound) and not displaced.

Q7. What are the auscultatory features?

Loud S1, opening snap, and low-pitched rumbling mid-diastolic murmur at the apex, best heard in left lateral position with the bell, with presystolic accentuation in sinus rhythm.

Q8. What signs suggest pulmonary hypertension?

Loud P2, left parasternal heave, raised JVP and features of right heart failure.

Q9. Why does the presystolic accentuation disappear?

It depends on atrial contraction, which is lost in atrial fibrillation.

Q10. What does a shorter gap between S2 and the opening snap mean?

More severe mitral stenosis.

DIAGNOSIS

Q11. What are the major Jones criteria?

Carditis, polyarthritis, chorea (Sydenham's), erythema marginatum and subcutaneous nodules.

Q12. What are the minor Jones criteria?

Fever, arthralgia, raised ESR or CRP, and prolonged PR interval.

Q13. How is rheumatic fever diagnosed?

Evidence of recent streptococcal infection plus two major, or one major and two minor criteria.

Q14. Which valve is most commonly affected in rheumatic heart disease?

The mitral valve, followed by the aortic valve.

Q15. What is the normal mitral valve area?

> 4–6 cm^2; severe stenosis is below 1.5 cm^2 (very severe below 1.0 cm^2).

Q16. What are causes of mitral stenosis other than rheumatic disease?

> Congenital, mitral annular calcification in older people, and rarely carcinoid or SLE.

Q17. What is the murmur of mitral regurgitation?

> A pansystolic murmur at the apex radiating to the axilla.

Q18. What is the Graham Steell murmur?

> An early diastolic murmur of pulmonary regurgitation due to pulmonary hypertension in severe mitral stenosis.

INVESTIGATIONS

Q19. What does the ECG show in mitral stenosis?

> P mitrale (bifid P waves) in sinus rhythm, atrial fibrillation, and right ventricular hypertrophy if pulmonary hypertension.

Q20. What does the chest X-ray show?

> Enlarged left atrium (straight left heart border, double right heart border, splayed carina), pulmonary congestion and Kerley B lines.

Q21. What is the key investigation?

> Echocardiography to measure valve area, gradient, pulmonary pressure and valve suitability for balloon valvotomy.

Q22. What is the Wilkins score?

> An echo score of leaflet mobility, thickening, calcification and subvalvular disease to judge suitability for balloon valvotomy.

Q23. Why is transoesophageal echo done before valvotomy?

> To exclude left atrial thrombus, especially in the appendage.

Q24. What tests support rheumatic fever?

> ASO titre or anti-DNase B, throat swab, ESR, CRP and ECG.

Q25. What blood test monitors warfarin?

> INR.

Q26. Why are blood cultures needed with fever in valve disease?

> To exclude infective endocarditis.

Q27. What is exercise echocardiography used for?

> To assess symptoms and pulmonary pressures when symptoms and resting findings do not match.

Q28. What test checks rhythm if palpitations are intermittent?

> Holter (24-hour ECG) monitoring.

MANAGEMENT

Q29. How is acute rheumatic fever treated?

> Penicillin to eradicate streptococcus, aspirin or NSAIDs for arthritis, rest, and steroids for severe carditis.

Q30. What is secondary prophylaxis?

> Long-term penicillin to prevent recurrences, usually benzathine penicillin IM every 3–4 weeks.

Q31. How long is secondary prophylaxis given?

> At least 10 years after the last attack or until age 40 if there is persistent valve disease (sometimes lifelong).

Q32. How are symptoms of mitral stenosis treated?

> Diuretics for congestion and beta-blockers or rate-limiting drugs to slow heart rate and lengthen diastole.

Q33. How is AF managed in mitral stenosis?

> Rate control and anticoagulation with warfarin.

Q34. Why are DOACs not used in AF with moderate-severe mitral stenosis?

> They were not tested and are not recommended; warfarin is used.

Q35. What is the intervention of choice for suitable symptomatic mitral stenosis?

> Percutaneous balloon mitral valvotomy.

Q36. What are contraindications to balloon valvotomy?

> Left atrial thrombus, moderate or severe mitral regurgitation, heavy calcification and severe subvalvular disease.

Q37. What surgery is used if valvotomy is unsuitable?

> Mitral valve replacement (or open commissurotomy).

Q38. What anticoagulation is needed with a mechanical valve?

> Lifelong warfarin; INR target 2.5–3.5 for mitral mechanical valves.

Q39. Why are DOACs contraindicated with mechanical valves?

> They caused more valve thrombosis and bleeding than warfarin.

Q40. How is mitral stenosis managed in pregnancy?

> Beta-blockers and diuretics; balloon valvotomy in pregnancy if symptoms are severe.

Q41. What advice is given about dental care?

> Good oral hygiene and regular dental review to reduce endocarditis risk.

Q42. When is antibiotic prophylaxis for endocarditis considered?

> In high-risk patients (prosthetic valves, previous endocarditis) before high-risk dental procedures, per local guidelines.

Q43. What is the role of a heart team?

> Cardiologists and surgeons decide the best intervention for each patient.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of mitral stenosis?

> Atrial fibrillation, systemic embolism (stroke), pulmonary hypertension, right heart failure, haemoptysis and infective endocarditis.

Q45. Why is stroke a major risk in mitral stenosis with AF?

> Blood stasis in an enlarged left atrium forms thrombus.

Q46. What is Ortner's syndrome?

> Hoarseness due to compression of the left recurrent laryngeal nerve by an enlarged left atrium.

Q47. What is Sydenham's chorea?

> Involuntary jerky movements in rheumatic fever, often appearing weeks to months after infection.

Q48. How can rheumatic heart disease be prevented?

> Prompt penicillin treatment of streptococcal sore throat (primary prevention) and secondary prophylaxis.

Q49. What is the prognosis after successful balloon valvotomy?

> Good symptom relief for many years, though restenosis can occur.

Q50. What is a recent advance in rheumatic heart disease?

> Echocardiographic screening of school children to detect early disease and start penicillin prophylaxis (GOAL trial).

CASE 6

Chronic Obstructive Pulmonary Disease

Typical exam case: A 64-year-old man who has smoked 40 pack-years has a chronic productive cough and increasing breathlessness for 3 years. He has a barrel chest, pursed-lip breathing and prolonged expiration with wheeze.

HISTORY AND EXAMINATION

Q1. What is COPD?

A chronic lung disease with persistent airflow limitation that is not fully reversible, usually due to smoking.

Q2. What are the main symptoms?

Chronic cough, sputum, breathlessness on exertion, wheeze and frequent chest infections.

Q3. How do you calculate pack-years?

Number of packs (20 cigarettes) smoked per day multiplied by years smoked.

Q4. What risk factors other than smoking cause COPD?

Biomass fuel smoke, occupational dust and chemicals, air pollution and alpha-1 antitrypsin deficiency.

Q5. How is breathlessness graded?

Using the MRC dyspnoea scale (1–5).

Q6. What signs of hyperinflation would you look for?

Barrel chest, reduced cricosternal distance, reduced expansion, hyper-resonance and loss of cardiac dullness.

Q7. What is pursed-lip breathing?

Breathing out through pursed lips to keep airways open and reduce air trapping.

Q8. What signs suggest CO2 retention?

Bounding pulse, warm peripheries, flapping tremor, drowsiness and headache.

Q9. What signs suggest cor pulmonale?

Raised JVP, right ventricular heave, loud P2, hepatomegaly and ankle oedema.

Q10. What is the significance of finger clubbing in COPD?

COPD itself does not cause clubbing; look for lung cancer, bronchiectasis or fibrosis.

DIAGNOSIS

Q11. How is COPD confirmed?

Spirometry showing a post-bronchodilator FEV1/FVC ratio below 0.7.

Q12. How is severity of airflow obstruction graded (GOLD)?

By FEV1 % predicted: GOLD 1 (80% or more), 2 (50–79%), 3 (30–49%) and 4 (below 30%).

Q13. How does COPD differ from asthma?

COPD: older smoker, persistent symptoms, little reversibility. Asthma: younger, variable symptoms, atopy and significant reversibility.

Q14. What is chronic bronchitis?

Productive cough for at least 3 months in 2 consecutive years.

Q15. What is emphysema?

> Permanent enlargement of air spaces beyond the terminal bronchioles with destruction of their walls.

Q16. What is the difference between 'pink puffers' and 'blue bloaters'?

> Older descriptions: pink puffers (emphysema) are thin and breathless with near-normal gases; blue bloaters (bronchitis) are cyanosed, oedematous and retain CO2.

Q17. When should alpha-1 antitrypsin deficiency be suspected?

> Early-onset COPD (under 45), little smoking, basal emphysema or family history.

Q18. What is an exacerbation of COPD?

> An acute worsening of symptoms beyond normal day-to-day variation, needing a change in treatment.

INVESTIGATIONS

Q19. What baseline tests are done at diagnosis?

> Spirometry, chest X-ray, FBC (anaemia or polycythaemia), BMI, and alpha-1 antitrypsin level in selected patients.

Q20. What does the chest X-ray show?

> Hyperinflation: flat diaphragms, more than 6 anterior ribs visible, narrow heart and bullae.

Q21. Why is FBC checked?

> Polycythaemia from chronic hypoxia or anaemia may worsen breathlessness.

Q22. What does an ABG show in type 2 respiratory failure?

> Low PaO2 (below 8 kPa) and high PaCO2 (above 6 kPa).

Q23. When is pulse oximetry used to assess for oxygen?

> If saturation is 92% or below at rest, refer for ABG assessment for long-term oxygen.

Q24. What is the role of CT chest?

> To assess emphysema, bullae, bronchiectasis, and suitability for lung volume reduction.

Q25. What does an ECG show in cor pulmonale?

> Right axis deviation, P pulmonale and right ventricular hypertrophy.

Q26. What tests are done in an exacerbation?

> ABG, chest X-ray, FBC, CRP, U&E, sputum culture and ECG.

Q27. Why is sputum culture useful?

> To guide antibiotics in recurrent infections.

Q28. What is the BODE index?

> A prognostic score combining BMI, airflow obstruction (FEV1), dyspnoea and exercise capacity.

MANAGEMENT

Q29. What is the single most important intervention?

> Stopping smoking.

Q30. What non-drug treatments help?

> Pulmonary rehabilitation, vaccination (influenza, pneumococcal, COVID), nutrition and self-management plans.

Q31. What is the first-line inhaler?

> A short-acting bronchodilator (salbutamol or ipratropium) as needed.

Q32. What is the next step if symptoms continue without asthmatic features?

> A long-acting beta-agonist plus a long-acting muscarinic antagonist (LABA + LAMA).

Q33. When are inhaled steroids added?

> If there are asthmatic features, high blood eosinophils (300 or more) or frequent exacerbations.

Q34. What is the risk of inhaled steroids in COPD?

> Pneumonia.

Q35. Who should get long-term oxygen therapy?

> Non-smokers with PaO2 below 7.3 kPa, or 7.3–8 kPa with polycythaemia, pulmonary hypertension or oedema; used at least 15 hours a day.

Q36. Why must patients not smoke on oxygen?

> Risk of fire and burns.

Q37. How is an acute exacerbation managed?

> Nebulised bronchodilators, oral prednisolone 30 mg for 5 days, antibiotics if purulent sputum, and controlled oxygen.

Q38. What oxygen target is used in COPD with CO2 retention?

> Saturation 88–92% using a Venturi mask.

Q39. When is non-invasive ventilation (NIV) used?

> In acute hypercapnic respiratory failure with pH below 7.35 and PaCO2 above 6.5 kPa despite medical treatment.

Q40. When is invasive ventilation considered?

> If NIV fails or is not suitable, after considering the patient's wishes and prognosis.

Q41. What is the role of mucolytics?

> Carbocisteine may reduce exacerbations in patients with chronic productive cough.

Q42. What is the role of azithromycin?

> Long-term prophylactic azithromycin can reduce exacerbations in selected non-smoking patients.

Q43. What surgical options exist?

> Lung volume reduction surgery, endobronchial valves, bullectomy and lung transplant in selected patients.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of COPD?

> Exacerbations, respiratory failure, cor pulmonale, pneumothorax, polycythaemia, depression and lung cancer.

Q45. What is a secondary pneumothorax?

> Pneumothorax in someone with lung disease; it is more dangerous and often needs a chest drain.

Q46. Why is pulmonary rehabilitation important?

> It improves breathlessness, exercise capacity and quality of life and reduces admissions.

Q47. What factors suggest poor prognosis?

> Low FEV1, low BMI, hypercapnia, frequent exacerbations, cor pulmonale and need for NIV.

Q48. Why is a rescue pack sometimes given?

> So patients can start steroids and antibiotics early at the start of an exacerbation.

Q49. Why are end-of-life discussions important in severe COPD?

> To plan care, ceilings of treatment and symptom control.

Q50. What is a recent advance in COPD?

> Triple inhaler therapy (LABA/LAMA/ICS) in a single device, and dupilumab for COPD with type 2 inflammation (high eosinophils).

CASE 7

Bronchial Asthma

> **Typical exam case:** A 22-year-old woman has episodes of wheeze, chest tightness and night cough for 2 years, worse in spring and after exercise. She has eczema and hay fever.

HISTORY AND EXAMINATION

Q1. What is asthma?

> Chronic airway inflammation with variable, reversible airflow obstruction and airway hyper-responsiveness.

Q2. What are the typical symptoms?

> Wheeze, breathlessness, chest tightness and cough, often worse at night and early morning.

Q3. What triggers would you ask about?

> Allergens (dust mite, pollen, pets), cold air, exercise, infections, smoke, pollution, drugs (NSAIDs, beta-blockers) and occupational exposures.

Q4. What is atopy?

> A tendency to allergic diseases: asthma, eczema and hay fever.

Q5. Why ask about occupation?

> Occupational asthma improves on days away from work.

Q6. Why ask about aspirin use?

> Aspirin and NSAIDs can trigger asthma in some patients, especially with nasal polyps (Samter's triad).

Q7. How is control assessed?

> Daytime symptoms, night waking, reliever use, activity limitation and exacerbations (e.g. Asthma Control Test).

Q8. What signs might you find between attacks?

> Often none; maybe wheeze, signs of eczema or nasal polyps.

Q9. What would you look at in inhaler technique?

> Correct use, timing, breath-hold and use of a spacer.

Q10. What history suggests a near-fatal asthma risk?

> Previous ICU admission or ventilation, frequent hospital visits, heavy reliever use and poor adherence to preventers.

DIAGNOSIS

Q11. How is asthma diagnosed?

> A history of variable symptoms plus objective tests showing variable airflow obstruction or airway inflammation.

Q12. What spirometry finding supports asthma?

> Obstructive pattern (FEV1/FVC below 0.7) with a 12% and 200 mL rise in FEV1 after a bronchodilator.

Q13. What is FeNO?

> Fractional exhaled nitric oxide; a raised level (over 40–50 ppb) supports eosinophilic airway inflammation.

Q14. How is peak flow variability used?

> More than 20% variability over 2–4 weeks supports asthma.

Q15. What is a bronchial challenge test?

> Inhaling methacholine or histamine to show airway hyper-responsiveness.

Q16. What is the differential diagnosis?

> COPD, vocal cord dysfunction, heart failure, bronchiectasis, foreign body and hyperventilation.

Q17. What is cough-variant asthma?

> Asthma presenting mainly with chronic dry cough.

Q18. What is exercise-induced bronchoconstriction?

> Narrowing of airways during or after exercise, often treated with a reliever before exercise or better control.

INVESTIGATIONS

Q19. What blood test may show allergic asthma?

> Raised eosinophils and total IgE.

Q20. What is skin prick testing used for?

> To identify allergens.

Q21. Is a chest X-ray needed in all patients?

> No; only for atypical features or to exclude other conditions.

Q22. What tests are done in an acute attack?

> Peak flow, oxygen saturation, ABG if saturation is below 92% or life-threatening features, and chest X-ray if pneumothorax or infection suspected.

Q23. What ABG finding is worrying in acute asthma?

> A normal or raised PaCO2, which shows the patient is tiring.

Q24. What is aspergillus testing used for?

> To detect allergic bronchopulmonary aspergillosis in severe or poorly controlled asthma.

Q25. What does a peak flow chart show in occupational asthma?

> Worse values on work days and better values on days off.

Q26. Why measure blood eosinophils in severe asthma?

> To select patients for biologic therapy.

Q27. What is the role of FeNO in monitoring?

> It can guide steroid dose and check adherence.

Q28. How is severe acute asthma defined?

> Peak flow 33–50% of best or predicted, respiratory rate 25 or more, heart rate 110 or more, or unable to finish sentences.

MANAGEMENT

Q29. What are the general measures?

> Avoid triggers, stop smoking, correct inhaler technique, a written action plan and vaccination.

Q30. What is the modern first-line treatment for adults (BTS/NICE/SIGN 2024)?

> Anti-inflammatory reliever: as-needed low-dose ICS-formoterol (e.g. budesonide-formoterol).

Q31. What is MART?

> Maintenance and reliever therapy: a single ICS-formoterol inhaler used both regularly and as needed.

Q32. Why is SABA-only treatment no longer recommended?

> SABA alone does not treat inflammation and increases the risk of severe attacks and death.

Q33. What is the next step if not controlled on low-dose MART?

> Moderate-dose MART, then check FeNO and eosinophils and refer to a specialist.

Q34. What other add-on treatments are used?

> Leukotriene receptor antagonists (montelukast) and long-acting muscarinic antagonists (tiotropium).

Q35. What is the main side effect of montelukast?

> Mood changes, sleep problems and nightmares.

Q36. What are biologic therapies for severe asthma?

> Omalizumab (anti-IgE), mepolizumab and benralizumab (anti-IL-5), dupilumab (anti-IL-4R) and tezepelumab (anti-TSLP).

Q37. How is acute severe asthma treated?

> Oxygen to keep saturation 94–98%, nebulised salbutamol, ipratropium, oral prednisolone 40–50 mg (or IV hydrocortisone).

Q38. What is given if not improving?

> IV magnesium sulfate 2 g, and consider IV aminophylline after senior review; ICU referral if life-threatening.

Q39. What are life-threatening features?

> Peak flow below 33%, saturation below 92%, silent chest, cyanosis, poor effort, exhaustion, confusion, arrhythmia or hypotension.

Q40. When can a patient be discharged after an attack?

> When peak flow is above 75% of best, stable on discharge medication, inhaler technique checked and follow-up arranged.

Q41. How long is oral prednisolone given after an attack?

> At least 5 days.

Q42. What advice is given about inhaled steroid side effects?

> Rinse the mouth to prevent thrush and hoarseness; use a spacer.

Q43. How is asthma managed in pregnancy?

> Continue usual treatment; poorly controlled asthma is more dangerous than the medicines.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of asthma?

> Severe attacks, respiratory failure, pneumothorax, airway remodelling and death.

Q45. Why are beta-blockers avoided in asthma?

> They can cause severe bronchospasm.

Q46. What is steroid side-effect monitoring needed for?

> Frequent oral steroids cause osteoporosis, diabetes, cataracts and adrenal suppression.

Q47. Why is a written action plan important?

> It helps patients recognise worsening and act early, reducing admissions.

Q48. What is the main cause of asthma deaths?

> Under-use of preventer treatment, over-reliance on relievers, and delay in seeking help.

Q49. Why does adherence matter?

> Most poorly controlled asthma is due to poor adherence or technique.

Q50. What is a recent advance in asthma?

> The 2024 BTS/NICE/SIGN guideline moving to anti-inflammatory reliever and MART, and new biologics such as tezepelumab.

CASE 8

Community-Acquired Pneumonia

Typical exam case: A 70-year-old man presents with 3 days of fever, cough with rusty sputum and right-sided pleuritic chest pain. He is confused. RR 32/min, BP 95/55 mmHg, urea 9 mmol/L. There is bronchial breathing at the right base.

HISTORY AND EXAMINATION

Q1. What is pneumonia?

Infection of the lung tissue causing inflammation and consolidation of the alveoli.

Q2. What are the typical symptoms?

Fever, cough, sputum, breathlessness, pleuritic chest pain, and sometimes confusion in older people.

Q3. What risk factors would you ask about?

Older age, smoking, COPD, diabetes, alcohol, heart failure, immunosuppression and recent influenza.

Q4. What features suggest atypical pneumonia?

Dry cough, headache, myalgia, diarrhoea and a less dramatic chest examination.

Q5. What history suggests aspiration pneumonia?

Stroke, alcohol, seizures, swallowing problems or reduced consciousness.

Q6. What signs of consolidation would you find?

Reduced expansion, dull percussion, increased vocal resonance, bronchial breathing and coarse crackles.

Q7. What does pleural rub suggest?

Inflamed pleura (pleurisy).

Q8. What is the significance of confusion in pneumonia?

It is a severity marker and is part of the CURB-65 score.

Q9. What signs suggest sepsis?

Fever or hypothermia, tachycardia, low BP, fast breathing, confusion and low urine output.

Q10. Why is the travel and contact history important?

Legionella (hotel showers), psittacosis (birds), Q fever (farm animals) and TB.

DIAGNOSIS AND SEVERITY

Q11. What is the CURB-65 score?

Confusion, Urea over 7 mmol/L, Respiratory rate 30 or more, BP systolic below 90 or diastolic 60 or less, and age 65 or more. One point each.

Q12. What is this patient's CURB-65 score?

5 (confusion, urea 9, RR 32, low BP, age 70): severe pneumonia.

Q13. How is CURB-65 used?

0–1: low risk, consider home treatment; 2: moderate, consider admission; 3–5: severe, admit and consider ICU.

Q14. What is the commonest cause of community-acquired pneumonia?

Streptococcus pneumoniae.

Q15. What are atypical organisms?

> Mycoplasma pneumoniae, Legionella, Chlamydophila pneumoniae and Chlamydophila psittaci.

Q16. Which organism causes pneumonia after influenza?

> Staphylococcus aureus.

Q17. Which organism is linked to alcoholics and red-currant jelly sputum?

> Klebsiella pneumoniae.

Q18. What is hospital-acquired pneumonia?

> Pneumonia starting 48 hours or more after admission, often due to Gram-negative organisms.

INVESTIGATIONS

Q19. What is the key imaging test?

> Chest X-ray, showing consolidation.

Q20. Which blood tests are done?

> FBC, CRP, U&E, LFTs, blood cultures, and ABG if hypoxic.

Q21. What microbiology tests are done in moderate or severe pneumonia?

> Blood cultures, sputum culture, pneumococcal and Legionella urinary antigens.

Q22. What does hyponatraemia with deranged LFTs suggest?

> Legionella pneumonia.

Q23. What does cold agglutinin haemolysis suggest?

> Mycoplasma pneumonia.

Q24. Why is CRP useful?

> To support diagnosis and monitor response.

Q25. Why should HIV testing be considered?

> Recurrent or unusual pneumonia may indicate HIV infection.

Q26. When is a repeat chest X-ray done?

> At about 6 weeks for patients with persistent symptoms or at higher risk of cancer (e.g. smokers over 50).

Q27. What tests are done if TB is suspected?

> Sputum smear for AFB, GeneXpert and culture.

Q28. What test helps decide on antibiotics in lower respiratory infection?

> CRP in primary care; procalcitonin in some hospitals.

MANAGEMENT

Q29. What antibiotic is used for low-severity CAP?

> Amoxicillin for 5 days (doxycycline or clarithromycin if allergic).

Q30. What antibiotic is used for moderate severity?

> Amoxicillin plus clarithromycin.

Q31. What antibiotic is used for high severity?

> IV co-amoxiclav plus clarithromycin (or per local policy).

Q32. How soon should antibiotics be given in severe pneumonia with sepsis?

> Within 1 hour.

Q33. What supportive care is needed?

> Oxygen to keep saturation 94–98% (88–92% in CO2 retainers), IV fluids, analgesia and VTE prophylaxis.

Q34. When is ICU referral considered?

> CURB-65 of 4–5, respiratory failure, shock or failure to respond.

Q35. How is Legionella treated?

> Clarithromycin or a fluoroquinolone (levofloxacin).

Q36. How is aspiration pneumonia treated?

> Antibiotics covering anaerobes (e.g. co-amoxiclav) and a swallowing assessment.

Q37. How long should treatment usually last?

> 5 days for most patients, longer if slow to respond or with complications.

Q38. When should IV antibiotics be switched to oral?

> When the patient is improving, afebrile for 48 hours and able to take oral medicines.

Q39. How is pleuritic pain managed?

> Paracetamol or NSAIDs to allow deep breathing and coughing.

Q40. What advice is given about recovery time?

> Fever settles in a week, cough and sputum within 6 weeks, and tiredness may last up to 6 months.

Q41. What vaccines prevent pneumonia?

> Pneumococcal and annual influenza vaccines, and COVID-19 vaccines.

Q42. What should you do if a patient does not improve after 72 hours?

> Review diagnosis, check for complications (empyema, abscess), repeat imaging and cultures, and reconsider antibiotics.

Q43. What is the role of steroids in pneumonia?

> Hydrocortisone may be used in severe CAP needing ICU; not routine.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of pneumonia?

> Parapneumonic effusion, empyema, lung abscess, sepsis, respiratory failure, ARDS and death.

Q45. How is empyema diagnosed?

> Pleural fluid that is pus, or pH below 7.2, low glucose or positive culture.

Q46. How is empyema treated?

> Chest drain, prolonged antibiotics and sometimes intrapleural fibrinolytics or surgery.

Q47. Which organisms cause lung abscess?

> Staph aureus, Klebsiella and anaerobes (after aspiration).

Q48. What is the mortality of severe pneumonia?

> High, up to 30% or more with CURB-65 of 4–5.

Q49. Why is follow-up chest X-ray important in smokers?

> Pneumonia may be caused by an underlying lung cancer.

Q50. What is a recent advance in pneumonia?

> Hydrocortisone in severe CAP reducing mortality (CAPE COD trial) and multiplex PCR panels for faster organism identification.

CASE 9

Pulmonary Tuberculosis

> **Typical exam case:** A 28-year-old man presents with cough for 6 weeks, evening fever, night sweats, weight loss and one episode of blood in sputum. Chest examination shows crackles at the right apex.

HISTORY AND EXAMINATION

Q1. What are the classic symptoms of pulmonary TB?

> Cough over 2–3 weeks, haemoptysis, fever (often evening), night sweats, weight loss and loss of appetite.

Q2. Which organism causes TB?

> Mycobacterium tuberculosis, an acid-fast bacillus spread by airborne droplets.

Q3. What risk factors would you ask about?

> Contact with TB, overcrowding, poverty, malnutrition, HIV, diabetes, steroids or other immunosuppression, smoking, alcohol and previous TB.

Q4. Why ask about previous TB treatment?

> Previous incomplete treatment raises the risk of drug-resistant TB.

Q5. Why is TB common in Pakistan?

> Pakistan is a high-burden country with overcrowding, poverty and incomplete treatment.

Q6. What signs may be found?

> Weight loss, fever, lymph nodes, apical crackles, signs of consolidation, cavity or pleural effusion; sometimes clubbing.

Q7. What is the typical site of reactivation TB?

> The apical and posterior segments of the upper lobes.

Q8. What extrapulmonary sites should you ask about?

> Lymph nodes, pleura, spine (Pott's disease), abdomen, meninges, kidneys and joints.

Q9. What is miliary TB?

> Widespread blood-borne TB with tiny nodules throughout the lungs and other organs.

Q10. What is primary TB?

> First infection, usually in children, forming a Ghon focus and hilar lymph nodes (Ghon complex).

DIAGNOSIS

Q11. What is latent TB?

> Infection with TB bacteria that are dormant, without symptoms and not infectious.

Q12. What is the most important test for pulmonary TB?

> Sputum examination: smear for acid-fast bacilli, GeneXpert MTB/RIF, and culture.

Q13. How many sputum samples are sent?

> At least two to three, ideally including an early morning sample.

Q14. What stain is used for AFB?

> Ziehl-Neelsen (or auramine fluorescent) stain.

Q15. What is GeneXpert MTB/RIF?

> A rapid PCR test that detects TB DNA and rifampicin resistance within about 2 hours.

Q16. How long does culture take?

> 2–6 weeks in liquid culture (MGIT), up to 8 weeks on solid media.

Q17. What is the gold standard for diagnosis?

> Culture, which also allows full drug sensitivity testing.

Q18. How is latent TB diagnosed?

> Tuberculin skin test (Mantoux) or interferon-gamma release assay (IGRA).

INVESTIGATIONS

Q19. What does the chest X-ray show in reactivation TB?

> Upper lobe consolidation, cavities, fibrosis, nodules and sometimes calcification or effusion.

Q20. What does a miliary pattern look like?

> Many tiny (1–3 mm) nodules evenly throughout both lungs.

Q21. What blood tests are done before treatment?

> FBC, LFTs, U&E, HbA1c, HIV test, and hepatitis B and C serology.

Q22. Why test for HIV in every TB patient?

> HIV is a major risk factor and changes treatment.

Q23. Why check liver function before treatment?

> Isoniazid, rifampicin and pyrazinamide can cause hepatitis.

Q24. Why check visual acuity and colour vision before ethambutol?

> Ethambutol can cause optic neuritis.

Q25. How is TB diagnosed when sputum is negative?

> Induced sputum, bronchoscopy with lavage, or biopsy of affected tissue.

Q26. What does a pleural fluid analysis show in TB effusion?

> Exudate, lymphocytes, raised adenosine deaminase (ADA) and often negative culture; pleural biopsy helps.

Q27. What is the role of CT chest?

> To show cavities, tree-in-bud nodules and complications when X-ray is unclear.

Q28. How is drug resistance detected?

> GeneXpert for rifampicin resistance, line probe assays and culture sensitivity testing.

MANAGEMENT

Q29. What is the standard treatment for drug-sensitive TB?

> 2 months of isoniazid, rifampicin, pyrazinamide and ethambutol (intensive phase), then 4 months of isoniazid and rifampicin (continuation phase).

Q30. How long is treatment for TB meningitis?

> 12 months, with steroids.

Q31. What is DOTS?

> Directly observed treatment, short-course: a health worker watches the patient take medicines to ensure adherence.

Q32. Why is pyridoxine given?

> To prevent isoniazid-induced peripheral neuropathy.

Q33. What are the side effects of rifampicin?

> Orange-red urine and tears, hepatitis, flu-like symptoms and liver enzyme induction (reduces effect of the pill, warfarin and other drugs).

Q34. What are the side effects of isoniazid?

> Peripheral neuropathy, hepatitis and drug-induced lupus.

Q35. What are the side effects of pyrazinamide?

> Hepatitis, gout (raised uric acid) and arthralgia.

Q36. What is the main side effect of ethambutol?

> Optic neuritis (reduced visual acuity and red-green colour blindness).

Q37. What contraceptive advice is given to a woman on rifampicin?

> Use a non-hormonal method or depot injection, as rifampicin reduces the effect of the pill and implant.

Q38. How is drug-induced hepatitis managed?

> Stop the drugs if ALT is over 5 times normal (or 3 times with symptoms or jaundice), then reintroduce one at a time once liver tests settle.

Q39. What is MDR-TB?

> TB resistant to at least isoniazid and rifampicin.

Q40. How is MDR-TB treated?

> Specialist treatment with all-oral regimens such as BPaLM (bedaquiline, pretomanid, linezolid, moxifloxacin) for 6 months.

Q41. When are steroids added to TB treatment?

> TB meningitis and TB pericarditis.

Q42. How is latent TB treated?

> 3 months of isoniazid and rifampicin, or 6 months of isoniazid.

Q43. What infection control measures are needed?

> Isolation for the first 2 weeks of treatment for smear-positive patients, masks, ventilation, and contact tracing.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of pulmonary TB?

> Massive haemoptysis, pneumothorax, bronchiectasis, aspergilloma in old cavities, empyema, respiratory failure and spread to other organs.

Q45. What is an aspergilloma?

> A fungal ball growing in an old TB cavity; it can cause haemoptysis.

Q46. What is IRIS in TB-HIV coinfection?

> Immune reconstitution inflammatory syndrome: worsening of TB after starting antiretrovirals as immunity recovers.

Q47. When should antiretroviral therapy be started in TB-HIV?

> Within 2 weeks of starting TB treatment (later in TB meningitis), depending on CD4 count.

Q48. Why is contact tracing important?

> To find and treat household contacts with active or latent TB.

Q49. What is BCG vaccine?

> A live attenuated vaccine given at birth in high-burden countries; it protects mainly against severe childhood TB.

Q50. What is a recent advance in TB?

> Shorter 6-month all-oral MDR-TB regimens (BPaLM) and the 4-month rifapentine-moxifloxacin regimen for drug-sensitive TB.

CASE 10

Pleural Effusion

Typical exam case: A 55-year-old man has breathlessness for 3 weeks. On examination the trachea is shifted to the left, the right side has reduced expansion, stony dull percussion and absent breath sounds at the base.

HISTORY AND EXAMINATION

Q1. What is a pleural effusion?

An abnormal collection of fluid in the pleural space.

Q2. What symptoms occur?

Breathlessness, pleuritic chest pain, dry cough, and symptoms of the cause (fever, weight loss, heart failure).

Q3. What are the signs of a large effusion?

Reduced expansion, stony dull percussion, reduced or absent breath sounds and vocal resonance, and tracheal shift away from the effusion.

Q4. What may be heard just above an effusion?

Bronchial breathing from compressed lung.

Q5. What is the difference between dullness of effusion and consolidation?

Effusion: stony dull with absent breath sounds. Consolidation: dull with bronchial breathing and increased vocal resonance.

Q6. Why does the trachea shift away from a large effusion?

The fluid pushes the mediastinum to the opposite side.

Q7. What history suggests malignancy?

Smoking, weight loss, haemoptysis and asbestos exposure.

Q8. What history suggests TB?

Fever, night sweats, weight loss and contact with TB.

Q9. What signs suggest heart failure as the cause?

Raised JVP, bilateral effusions, ankle oedema and a third heart sound.

Q10. What other general signs might help?

Clubbing (lung cancer, empyema), lymph nodes, breast lumps, ascites and signs of liver disease.

DIAGNOSIS

Q11. How are effusions classified?

Transudates and exudates.

Q12. What are causes of transudate?

Heart failure, cirrhosis, nephrotic syndrome, hypoalbuminaemia, hypothyroidism and peritoneal dialysis.

Q13. What are causes of exudate?

Infection (parapneumonic, TB), cancer, pulmonary embolism, rheumatoid arthritis, SLE and pancreatitis.

Q14. What are Light's criteria?

An effusion is an exudate if: fluid/serum protein ratio over 0.5, fluid/serum LDH ratio over 0.6, or fluid LDH over two-thirds the upper limit of normal serum LDH.

Q15. When are Light's criteria used?

> When pleural fluid protein is between 25 and 35 g/L.

Q16. What is the commonest cause of a transudate?

> Heart failure.

Q17. What is the commonest cause of an exudate in Pakistan?

> Tuberculosis, with malignancy and pneumonia also common.

Q18. What is a haemothorax?

> Blood in the pleural space, usually after trauma.

INVESTIGATIONS

Q19. What is the first imaging test?

> Chest X-ray; at least 200 mL is needed to blunt the costophrenic angle on a PA film.

Q20. What is the role of ultrasound?

> To confirm fluid, guide aspiration and show septations.

Q21. What tests are done on pleural fluid?

> Appearance, protein, LDH, glucose, pH, cell count, Gram stain and culture, AFB, ADA, and cytology.

Q22. What does a low pleural glucose suggest?

> Empyema, TB, rheumatoid arthritis or malignancy.

Q23. What pH suggests a complicated parapneumonic effusion needing a drain?

> pH below 7.2.

Q24. What does a high ADA suggest?

> Tuberculous pleural effusion, especially in high-prevalence areas.

Q25. What does milky fluid suggest?

> Chylothorax (high triglycerides), from thoracic duct injury or lymphoma.

Q26. What does a lymphocyte-rich exudate suggest?

> TB or malignancy.

Q27. What is the next step if cytology is negative but cancer is suspected?

> CT chest with contrast and pleural biopsy (image-guided or thoracoscopic).

Q28. Why should a bilateral transudate with heart failure not always be tapped?

> If typical heart failure is present and responds to diuretics, diagnostic aspiration may not be needed.

MANAGEMENT

Q29. What is the main principle of management?

> Treat the underlying cause and relieve symptoms.

Q30. How is a large symptomatic effusion relieved?

> Therapeutic aspiration or chest drain.

Q31. How much fluid should be removed at one time?

> Usually no more than 1.5 L, to avoid re-expansion pulmonary oedema.

Q32. What is re-expansion pulmonary oedema?

> Oedema in the lung after rapid drainage of a large effusion or pneumothorax.

Q33. How is a heart failure effusion treated?

> Diuretics and heart failure treatment.

Q34. How is a parapneumonic effusion treated?

> Antibiotics; a chest drain if pH below 7.2, pus, or positive culture.

Q35. How is empyema treated?

> Chest drain, antibiotics, intrapleural tPA and DNase in some cases, and surgery (VATS decortication) if it fails.

Q36. How is a TB effusion treated?

> Standard anti-TB treatment; therapeutic aspiration for symptom relief.

Q37. How is a recurrent malignant effusion managed?

> Talc pleurodesis or an indwelling pleural catheter.

Q38. What is pleurodesis?

> Sealing the pleural space by causing inflammation, usually with talc, to prevent fluid re-collecting.

Q39. What is an indwelling pleural catheter?

> A tunnelled catheter allowing repeated drainage at home in malignant effusion.

Q40. Why is ultrasound guidance recommended for pleural procedures?

> It reduces complications such as pneumothorax and organ injury.

Q41. Where is a chest drain inserted?

> In the 'safe triangle': mid-axillary line, anterior border of latissimus dorsi, lateral border of pectoralis major, above the 5th intercostal space.

Q42. Why is the needle inserted just above a rib?

> To avoid the neurovascular bundle that runs below each rib.

Q43. What is the complication of chest drain insertion?

> Pain, bleeding, infection, organ injury, drain blockage and surgical emphysema.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of pleural effusion?

> Respiratory compromise, empyema, trapped lung and fibrothorax.

Q45. What is a trapped lung?

> Lung that cannot re-expand because of a thick visceral pleural peel.

Q46. What is the prognosis of malignant pleural effusion?

> Usually poor, with median survival of a few months, depending on the cancer.

Q47. Which cancers commonly cause malignant effusion?

> Lung, breast, lymphoma, ovary and mesothelioma.

Q48. What is mesothelioma?

> A cancer of the pleura linked to asbestos exposure, often decades earlier.

Q49. What is the LENT score?

> A score (LDH, ECOG performance, neutrophil:lymphocyte ratio, tumour type) to predict survival in malignant effusion.

Q50. What is a recent advance in pleural disease?

> Indwelling pleural catheters with talc (IPC-plus) for faster pleurodesis, and intrapleural tPA/DNase for pleural infection (MIST2 trial).

CASE 11

Chronic Liver Disease and Cirrhosis

Typical exam case: A 50-year-old man with chronic hepatitis C presents with abdominal swelling and ankle oedema. He has jaundice, spider naevi, palmar erythema, gynaecomastia, splenomegaly and shifting dullness.

HISTORY AND EXAMINATION

Q1. What is cirrhosis?

Diffuse liver fibrosis with regenerative nodules, the end result of chronic liver injury.

Q2. What are the common causes of cirrhosis?

Hepatitis B and C (common in Pakistan), alcohol, non-alcoholic fatty liver disease (MASLD), autoimmune hepatitis, primary biliary cholangitis, haemochromatosis and Wilson's disease.

Q3. What risk factors for hepatitis would you ask about?

Blood transfusions, reused needles, dental or surgical procedures, injections by unqualified practitioners, tattoos, IV drug use and sexual contact.

Q4. What symptoms suggest decompensation?

Jaundice, abdominal swelling (ascites), confusion (encephalopathy) and vomiting blood or black stools (variceal bleeding).

Q5. What signs of chronic liver disease are seen in the hands?

Clubbing, leukonychia, palmar erythema, Dupuytren's contracture and flapping tremor.

Q6. What signs are seen on the face and chest?

Jaundice, spider naevi, gynaecomastia and loss of body hair.

Q7. What abdominal signs are seen?

Caput medusae, hepatomegaly or a small liver, splenomegaly and ascites.

Q8. How do you demonstrate ascites?

Shifting dullness and a fluid thrill in large ascites.

Q9. What is asterixis?

A flapping tremor of the outstretched hands, a sign of hepatic encephalopathy.

Q10. What does splenomegaly show in cirrhosis?

Portal hypertension.

DIAGNOSIS AND CLASSIFICATION

Q11. What is portal hypertension?

Raised pressure in the portal venous system (gradient over 5 mmHg; clinically significant over 10 mmHg).

Q12. What are the consequences of portal hypertension?

Varices, splenomegaly, ascites and portosystemic encephalopathy.

Q13. What is the Child-Pugh score?

A score of liver disease severity using bilirubin, albumin, INR, ascites and encephalopathy (classes A, B, C).

Q14. What is the MELD score?

A score using bilirubin, INR, creatinine (and sodium) to predict mortality and prioritise transplantation.

Q15. What is compensated versus decompensated cirrhosis?

> Compensated: no major complications. Decompensated: ascites, variceal bleeding, encephalopathy or jaundice.

Q16. What is MASLD?

> Metabolic dysfunction-associated steatotic liver disease (previously NAFLD), linked to obesity and diabetes.

Q17. What is hepatorenal syndrome?

> Kidney failure in advanced cirrhosis due to intense renal vasoconstriction, without structural kidney disease.

Q18. What is spontaneous bacterial peritonitis?

> Infection of ascitic fluid without an obvious surgical source; ascitic neutrophils over 250/mm³.

INVESTIGATIONS

Q19. What blood tests are done?

> LFTs, albumin, INR, FBC (low platelets from hypersplenism), U&E, glucose and alpha-fetoprotein.

Q20. Why are platelets low in cirrhosis?

> Hypersplenism and reduced thrombopoietin.

Q21. Which tests find the cause?

> Hepatitis B and C serology, autoantibodies, immunoglobulins, ferritin and transferrin saturation, caeruloplasmin, and alpha-1 antitrypsin.

Q22. How is hepatitis C confirmed?

> Anti-HCV antibody, then HCV RNA PCR to confirm active infection.

Q23. What does ultrasound show?

> A small nodular liver, splenomegaly, ascites, portal vein flow and any liver lesion.

Q24. What is transient elastography (FibroScan)?

> A non-invasive measure of liver stiffness to assess fibrosis.

Q25. What is the role of liver biopsy?

> When the cause or stage is unclear; less used now.

Q26. What is the serum-ascites albumin gradient (SAAG)?

> Serum albumin minus ascitic albumin; 11 g/L or more indicates portal hypertension.

Q27. What ascitic tests are done?

> Cell count, albumin, protein, Gram stain and culture (in blood culture bottles), and cytology if needed.

Q28. Why is endoscopy done?

> To screen for oesophageal varices.

MANAGEMENT

Q29. How is ascites managed?

> Salt restriction (about 5 g/day) and diuretics: spironolactone first, adding furosemide.

Q30. How is tense ascites managed?

> Large-volume paracentesis with IV albumin (8 g per litre removed if over 5 L).

Q31. What is refractory ascites treated with?

> Repeated paracentesis, TIPS, or liver transplant.

Q32. How is spontaneous bacterial peritonitis treated?

> IV antibiotics (e.g. cefotaxime) and IV albumin to protect the kidneys.

Q33. Who needs SBP prophylaxis?

> Patients with previous SBP, and those with low ascitic protein and advanced disease; ciprofloxacin or co-trimoxazole.

Q34. How is hepatic encephalopathy treated?

> Find and treat the trigger, lactulose to achieve 2–3 soft stools a day, and rifaximin to prevent recurrence.

Q35. What triggers encephalopathy?

> GI bleeding, infection, constipation, sedatives, electrolyte disturbance, dehydration and alcohol.

Q36. How is acute variceal bleeding managed?

> Resuscitation, terlipressin, IV antibiotics, urgent endoscopy with band ligation, and TIPS if bleeding continues.

Q37. How are varices prevented from bleeding?

> Non-selective beta-blockers (carvedilol or propranolol) or band ligation.

Q38. How is hepatitis C treated?

> Direct-acting antivirals such as sofosbuvir with velpatasvir (or daclatasvir) for 12 weeks; cure rates over 95%.

Q39. How is hepatitis B cirrhosis treated?

> Long-term tenofovir or entecavir.

Q40. What nutritional advice is given?

> Adequate protein (1.2–1.5 g/kg/day), late evening snack, and avoid long fasting.

Q41. Which drugs should be avoided in cirrhosis?

> NSAIDs, sedatives, aminoglycosides and high-dose paracetamol.

Q42. What screening is needed for liver cancer?

> Ultrasound every 6 months (with or without AFP).

Q43. What is the definitive treatment of end-stage liver disease?

> Liver transplantation.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the major complications of cirrhosis?

> Variceal bleeding, ascites, SBP, hepatic encephalopathy, hepatorenal syndrome, hepatocellular carcinoma and liver failure.

Q45. How is hepatorenal syndrome treated?

> Stop diuretics and nephrotoxins, IV albumin and terlipressin, and consider transplant.

Q46. What is the risk of liver cancer in cirrhosis?

> About 1–4% per year.

Q47. What is the prognosis of decompensated cirrhosis?

> Poor; median survival about 2 years without transplant.

Q48. Why is hepatitis B vaccination important?

> It prevents infection and cirrhosis; it is part of the EPI schedule in Pakistan.

Q49. Why is safe injection practice important in Pakistan?

> Reused needles are a major cause of hepatitis C spread.

Q50. What is a recent advance in liver disease?

> Pan-genotypic direct-acting antivirals for hepatitis C and the WHO 2030 hepatitis elimination goal, and carvedilol to prevent decompensation.

CASE 12

Chronic Kidney Disease

> **Typical exam case:** A 60-year-old man with long-standing diabetes and hypertension has tiredness, itching, poor appetite and ankle swelling. His eGFR is 18 mL/min/1.73 m² and urine ACR is 120 mg/mmol.

HISTORY AND EXAMINATION

Q1. What is chronic kidney disease?

Abnormal kidney structure or function lasting more than 3 months, such as eGFR below 60 or albuminuria.

Q2. What are the common causes of CKD?

Diabetes, hypertension, glomerulonephritis, polycystic kidney disease, obstruction, recurrent infections and drugs (NSAIDs).

Q3. What symptoms occur in advanced CKD?

Tiredness, itching, poor appetite, nausea, weight loss, breathlessness, swelling, cramps, restless legs and reduced urine.

Q4. What drug history is important?

NSAIDs, ACE inhibitors, lithium, aminoglycosides and herbal remedies.

Q5. What family history is relevant?

Polycystic kidney disease and Alport syndrome.

Q6. What signs would you look for?

Pallor, scratch marks, BP, fluid overload, pericardial rub, peripheral neuropathy and fistula or catheter sites.

Q7. What is the colour change in uraemia?

A sallow, yellow-brown skin colour.

Q8. Why examine the abdomen?

For enlarged polycystic kidneys, bladder enlargement and transplant kidneys.

Q9. What signs suggest uraemic emergency?

Pericarditis, encephalopathy, flapping tremor and severe acidosis breathing.

Q10. Why examine the fundi in CKD?

To look for diabetic or hypertensive retinopathy.

DIAGNOSIS AND STAGING

Q11. How is CKD staged?

By eGFR (G1–G5) and albuminuria (A1–A3).

Q12. What are the eGFR stages?

G1 90 or more, G2 60–89, G3a 45–59, G3b 30–44, G4 15–29, G5 below 15.

Q13. What are the albuminuria stages?

A1 ACR below 3, A2 3–30, A3 over 30 mg/mmol.

Q14. What is this patient's stage?

G4 A3.

Q15. Why are both eGFR and ACR used?

> Albuminuria adds risk of progression and cardiovascular events at any eGFR.

Q16. How does CKD differ from acute kidney injury?

> CKD is long-standing (over 3 months), often with small kidneys and anaemia; AKI is a sudden rise in creatinine.

Q17. What is end-stage kidney disease?

> eGFR below 15 or need for dialysis or transplant.

Q18. What is the commonest cause of CKD worldwide?

> Diabetic kidney disease.

INVESTIGATIONS

Q19. What blood tests are done?

> U&E, eGFR, bicarbonate, calcium, phosphate, PTH, FBC, ferritin, lipids and HbA1c.

Q20. What urine tests are done?

> Urine dipstick for blood and protein, ACR, and microscopy.

Q21. What does ultrasound show in CKD?

> Usually small, echogenic kidneys; large kidneys in diabetes, polycystic disease or amyloidosis.

Q22. When is a renal biopsy done?

> When the cause is unclear, for example suspected glomerulonephritis.

Q23. Why is haematuria with proteinuria important?

> It suggests glomerulonephritis.

Q24. Which tests screen for glomerulonephritis?

> ANCA, ANA, complement, anti-GBM, immunoglobulins and serum protein electrophoresis.

Q25. What anaemia is seen in CKD?

> Normocytic normochromic anaemia due to low erythropoietin.

Q26. What bone and mineral changes occur?

> Low calcium, high phosphate, high PTH and low active vitamin D (CKD-mineral bone disorder).

Q27. How often should eGFR and ACR be monitored?

> At least yearly, more often in advanced stages.

Q28. What is the kidney failure risk equation?

> A score predicting 2- and 5-year risk of kidney failure; used to decide referral.

MANAGEMENT

Q29. What is the main aim of CKD management?

> Slow progression, reduce cardiovascular risk and treat complications.

Q30. What BP target is used in CKD with albuminuria?

> Below 130/80 mmHg.

Q31. Which antihypertensives are preferred?

> ACE inhibitor or ARB, especially with albuminuria.

Q32. Which drug class slows CKD progression in diabetes and non-diabetic CKD?

> SGLT2 inhibitors (dapagliflozin, empagliflozin).

Q33. What is finerenone?

> A non-steroidal mineralocorticoid antagonist that slows diabetic kidney disease.

Q34. Why are statins given?

> To reduce cardiovascular risk.

Q35. How is anaemia of CKD treated?

> Correct iron deficiency (often IV iron), then erythropoiesis-stimulating agents, aiming for Hb 100–120 g/L.

Q36. How is high phosphate treated?

> Low phosphate diet and phosphate binders (calcium acetate, sevelamer).

Q37. How is secondary hyperparathyroidism treated?

> Control phosphate, give active vitamin D (alfacalcidol) and cinacalcet if needed.

Q38. How is metabolic acidosis treated?

> Oral sodium bicarbonate if bicarbonate is below 20 mmol/L.

Q39. What are the indications for urgent dialysis?

> Refractory hyperkalaemia, severe acidosis, fluid overload not responding to diuretics, uraemic pericarditis or encephalopathy.

Q40. What are the types of renal replacement therapy?

> Haemodialysis, peritoneal dialysis and kidney transplantation.

Q41. Why is an AV fistula preferred for haemodialysis?

> It has lower infection and clotting risk than catheters.

Q42. What is the best treatment for end-stage kidney disease?

> Kidney transplant, which gives better survival and quality of life than dialysis.

Q43. Why must drug doses be adjusted?

> Many drugs are cleared by the kidneys and can build up, e.g. metformin, gabapentin, digoxin and some antibiotics.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of CKD?

> Anaemia, bone disease, hyperkalaemia, acidosis, fluid overload, hypertension, cardiovascular disease and uraemia.

Q45. What is the main cause of death in CKD?

> Cardiovascular disease.

Q46. What is renal osteodystrophy?

> Bone disease from mineral and hormone disturbance in CKD.

Q47. How is hyperkalaemia treated acutely?

> IV calcium gluconate, insulin with dextrose, nebulised salbutamol, and dialysis if refractory.

Q48. What dietary advice is given?

> Limit salt, potassium and phosphate as needed, adequate protein and fluid guidance.

Q49. What is conservative kidney management?

> Supportive care without dialysis, often for frail elderly patients.

Q50. What is a recent advance in CKD?

> SGLT2 inhibitors and finerenone slowing progression, and GLP-1 agonists (semaglutide) protecting the kidneys in diabetes (FLOW trial).

CASE 13

Nephrotic Syndrome

Typical exam case: A 35-year-old man presents with swelling of the face in the morning and of the legs in the evening for 3 weeks. His urine is frothy. Urine dipstick shows 4+ protein. Serum albumin is 18 g/L and cholesterol is high.

HISTORY AND EXAMINATION

Q1. What is nephrotic syndrome?

Heavy proteinuria (over 3.5 g/day in adults), low serum albumin and oedema, usually with high cholesterol.

Q2. What are the typical symptoms?

Facial and periorbital swelling, leg oedema, frothy urine, weight gain and tiredness.

Q3. Why is the swelling facial in the morning?

Fluid shifts to the face lying down overnight.

Q4. What drug history is relevant?

NSAIDs, gold, penicillamine and lithium.

Q5. What systemic diseases cause nephrotic syndrome?

Diabetes, SLE, amyloidosis, hepatitis B and C, HIV, myeloma and cancers.

Q6. What signs would you look for?

Pitting oedema, facial puffiness, ascites, pleural effusions, BP and xanthelasma.

Q7. What do Muehrcke's lines show?

Paired white bands on the nails due to low albumin.

Q8. Why examine for lymph nodes and organs?

To look for lymphoma or other cancers linked to secondary nephrotic syndrome.

Q9. What symptoms suggest thrombosis?

Leg swelling (DVT), chest pain (PE), and flank pain with haematuria (renal vein thrombosis).

Q10. How does nephritic syndrome differ?

Nephritic: haematuria, red cell casts, hypertension, oedema and reduced kidney function, with less proteinuria.

CAUSES AND PATHOLOGY

Q11. What is the commonest cause in children?

Minimal change disease.

Q12. What are common primary causes in adults?

Membranous nephropathy, focal segmental glomerulosclerosis (FSGS) and minimal change disease.

Q13. What is the commonest secondary cause in adults?

Diabetic nephropathy.

Q14. What antibody is linked to primary membranous nephropathy?

Anti-PLA2R antibody.

Q15. What does electron microscopy show in minimal change disease?

> Effacement (fusion) of podocyte foot processes.

Q16. Which cancers are linked with membranous nephropathy?

> Solid tumours such as lung, colon and breast.

Q17. Which cancer is linked with minimal change disease?

> Hodgkin lymphoma.

Q18. Why does oedema occur?

> Low oncotic pressure from low albumin and sodium retention by the kidney.

INVESTIGATIONS

Q19. How is proteinuria quantified?

> Urine protein:creatinine ratio (over 300 mg/mmol is nephrotic range).

Q20. What blood tests are done?

> Albumin, U&E, lipids, FBC, glucose and clotting.

Q21. What tests look for a cause?

> HbA1c, ANA, dsDNA, complement, hepatitis B and C, HIV, anti-PLA2R, serum protein electrophoresis and free light chains.

Q22. What does urine microscopy show?

> Fatty casts and oval fat bodies ('Maltese crosses').

Q23. What is the key test in adults?

> Renal biopsy to find the histological type.

Q24. Why are children not usually biopsied?

> Most have steroid-sensitive minimal change disease; biopsy is reserved for steroid resistance or atypical features.

Q25. What does ultrasound show?

> Usually normal sized kidneys; it excludes other causes and renal vein thrombosis with Doppler.

Q26. Why check thyroid function?

> Thyroid-binding proteins are lost in urine, which can affect results.

Q27. What is checked if amyloidosis is suspected?

> Serum free light chains and biopsy with Congo red stain.

Q28. Why is age-appropriate cancer screening considered in membranous nephropathy?

> Because of its link to solid tumours.

MANAGEMENT

Q29. What are the general measures?

> Salt restriction, diuretics for oedema, ACE inhibitor or ARB to reduce proteinuria, and a statin.

Q30. Which diuretic is used?

> Loop diuretics such as furosemide, often in higher doses because of low albumin.

Q31. Why is an ACE inhibitor used?

> It reduces proteinuria and protects the kidney.

Q32. Why is anticoagulation considered?

> Nephrotic syndrome is pro-thrombotic, especially with albumin below 20–25 g/L in membranous nephropathy.

Q33. How is minimal change disease treated?

> High-dose oral prednisolone; most respond within weeks.

Q34. What is the treatment for frequently relapsing or steroid-dependent disease?

> Calcineurin inhibitors (tacrolimus, ciclosporin), cyclophosphamide or rituximab.

Q35. How is membranous nephropathy treated?

> Supportive care at first; rituximab or cyclophosphamide with steroids if high risk or not improving.

Q36. How is FSGS treated?

> Steroids, then calcineurin inhibitors; secondary FSGS is treated by addressing the cause.

Q37. How is diabetic nephrotic syndrome treated?

> Glucose and BP control, ACE inhibitor/ARB and SGLT2 inhibitors.

Q38. Why is fluid removal done slowly?

> To avoid hypovolaemia and acute kidney injury.

Q39. Why are vaccines given?

> Patients are at risk of infection, especially pneumococcal.

Q40. What protein intake is advised?

> Normal protein intake; high protein diets are not recommended.

Q41. How is the response to treatment judged?

> By falling urine protein:creatinine ratio and rising albumin.

Q42. When should steroid side effects be prevented?

> Give bone protection and stomach protection and monitor glucose and BP.

Q43. What does steroid resistance mean in children?

> Failure to remit after 4–6 weeks of steroids; biopsy is needed.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of nephrotic syndrome?

> Thrombosis (DVT, PE, renal vein thrombosis), infections, hyperlipidaemia, AKI and progressive CKD.

Q45. Why are infections common?

> Loss of immunoglobulins and complement in urine, plus immunosuppressive treatment.

Q46. Which infection is classically linked with nephrotic syndrome in children?

> Spontaneous pneumococcal peritonitis.

Q47. Why does thrombosis occur?

> Loss of antithrombin in urine, more liver clotting factors and haemoconcentration.

Q48. What is the prognosis of minimal change disease?

> Good kidney prognosis, though relapses are common.

Q49. What is the prognosis of FSGS?

> Many progress to CKD; it can recur after transplant.

Q50. What is a recent advance in nephrotic syndrome?

> Anti-PLA2R antibody testing to diagnose and monitor membranous nephropathy, and rituximab as first-line therapy (MENTOR trial).

CASE 14

Stroke (Cerebrovascular Accident)

> **Typical exam case:** A 68-year-old hypertensive man woke up with weakness of the right arm and leg and difficulty speaking. On examination he has right hemiplegia with upper motor neuron signs, right facial weakness sparing the forehead, and expressive aphasia.

HISTORY AND EXAMINATION

Q1. What is a stroke?

> A sudden focal neurological deficit of vascular origin lasting more than 24 hours or leading to death.

Q2. What is a TIA?

> A transient episode of neurological dysfunction due to ischaemia without infarction, usually lasting minutes.

Q3. What history is key in suspected stroke?

> Exact time of onset or last seen well, symptoms, risk factors, anticoagulant use and functional baseline.

Q4. What are the risk factors for stroke?

> Hypertension (most important), AF, diabetes, smoking, high cholesterol, previous TIA, carotid disease, heart disease and age.

Q5. What is the FAST test?

> Face drooping, Arm weakness, Speech difficulty, Time to call emergency services.

Q6. What are upper motor neuron signs?

> Weakness, increased tone (spasticity), brisk reflexes, clonus and an up-going plantar.

Q7. Why is the forehead spared in a UMN facial palsy?

> The upper face has bilateral cortical supply.

Q8. What is expressive (Broca's) aphasia?

> Slow, effortful speech with good understanding; lesion in the dominant frontal lobe.

Q9. What is receptive (Wernicke's) aphasia?

> Fluent but meaningless speech with poor understanding; lesion in the dominant temporal lobe.

Q10. What else would you examine?

> Pulse (AF), BP, carotid bruits, heart murmurs, visual fields, swallowing and conscious level (GCS).

CLASSIFICATION AND LOCALISATION

Q11. What are the two main types of stroke?

> Ischaemic (about 85%) and haemorrhagic (about 15%).

Q12. What are the causes of ischaemic stroke?

> Large artery atherosclerosis, cardioembolism (AF), small vessel (lacunar) disease and rarer causes such as dissection.

Q13. What is the Oxford (Bamford) classification?

> Total anterior circulation (TACS), partial anterior circulation (PACS), lacunar (LACS) and posterior circulation (POCS) strokes.

Q14. What defines a TACS?

> All three of: hemiparesis or sensory loss, homonymous hemianopia, and higher cortical dysfunction (aphasia or neglect).

Q15. What is a lacunar stroke?

> A small deep infarct causing pure motor, pure sensory, sensorimotor, or ataxic hemiparesis, without cortical signs.

Q16. What are signs of a posterior circulation stroke?

> Cerebellar signs, cranial nerve palsies, crossed signs, isolated hemianopia and reduced consciousness.

Q17. What does a middle cerebral artery stroke cause?

> Contralateral weakness and sensory loss affecting face and arm more than the leg, hemianopia, and aphasia (dominant) or neglect (non-dominant).

Q18. What does an anterior cerebral artery stroke cause?

> Contralateral leg weakness more than arm.

INVESTIGATIONS

Q19. What is the first investigation?

> Urgent non-contrast CT head to exclude haemorrhage.

Q20. What does early CT show in ischaemic stroke?

> Often normal in the first hours; later a low-density area.

Q21. What additional imaging is used for thrombectomy?

> CT angiography (and CT perfusion if late presentation).

Q22. Why check blood glucose immediately?

> Hypoglycaemia can mimic stroke.

Q23. What blood tests are done?

> FBC, U&E, glucose, lipids, clotting, ESR and HbA1c.

Q24. Why is an ECG and cardiac monitoring done?

> To find atrial fibrillation.

Q25. Why is carotid Doppler done?

> To find significant carotid stenosis in anterior circulation stroke or TIA.

Q26. When is MRI preferred?

> For posterior circulation symptoms, small strokes, uncertain diagnosis or wake-up strokes (DWI-FLAIR mismatch).

Q27. When is echocardiography done?

> If a cardioembolic source is suspected, such as endocarditis or a heart thrombus.

Q28. What tests are done in a young stroke patient?

> Thrombophilia screen, vasculitis screen, bubble echo for PFO and imaging for arterial dissection.

MANAGEMENT

Q29. What is the time window for IV thrombolysis?

> Within 4.5 hours of onset, after CT excludes haemorrhage.

Q30. Which drugs are used for thrombolysis?

> Alteplase or tenecteplase.

Q31. What is the BP threshold before thrombolysis?

> BP must be below 185/110 mmHg.

Q32. What is mechanical thrombectomy?

> Removing a clot from a large artery with a catheter; offered up to 6 hours, and up to 24 hours in selected patients with imaging showing salvageable brain.

Q33. What antiplatelet is given after ischaemic stroke?

> Aspirin 300 mg for 2 weeks (after excluding haemorrhage), then clopidogrel 75 mg long term.

Q34. How is TIA managed?

> Aspirin 300 mg at once, and specialist assessment within 24 hours; dual antiplatelets in some cases.

Q35. How is stroke with AF managed long term?

> Anticoagulation (a DOAC), usually started around 2 weeks after a large stroke (earlier for small ones).

Q36. When is carotid endarterectomy offered?

> For symptomatic carotid stenosis of 50–99%, ideally within 2 weeks.

Q37. What secondary prevention is given?

> Antiplatelet or anticoagulant, high-intensity statin, BP control, diabetes control, stop smoking and lifestyle change.

Q38. Why is swallowing assessed before oral intake?

> To prevent aspiration pneumonia.

Q39. How is intracerebral haemorrhage managed?

> Reverse anticoagulants, lower BP (systolic target 140 mmHg) and neurosurgical review if needed.

Q40. When is neurosurgery considered for stroke?

> Large cerebellar haemorrhage or infarct, hydrocephalus, or malignant MCA infarct needing decompressive hemicraniectomy.

Q41. Why are patients managed in a stroke unit?

> It reduces death and disability.

Q42. What rehabilitation is needed?

> Physiotherapy, occupational therapy, speech and language therapy and psychological support.

Q43. How is VTE prevented in immobile stroke patients?

> Intermittent pneumatic compression devices.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of stroke?

> Aspiration pneumonia, DVT, pressure sores, depression, spasticity, shoulder pain, seizures and recurrence.

Q45. What is the risk of stroke after TIA?

> Highest in the first days; the ABCD2 score was used but all suspected TIAs now need urgent review.

Q46. Why is depression common after stroke?

> Brain injury and loss of function; screening and treatment improve recovery.

Q47. What are driving rules after stroke or TIA in the UK?

> Car drivers must not drive for at least 1 month.

Q48. What is the prognosis of stroke?

> About a third recover well, a third have disability and a third die within a year, depending on severity.

Q49. Why is hypertension control vital in Pakistan?

> Hypertension is the main risk factor and is often undiagnosed or untreated.

Q50. What is a recent advance in stroke care?

> Tenecteplase as a single-bolus thrombolytic and thrombectomy for large core infarcts and late presentations up to 24 hours.

CASE 15

Paraplegia and Spinal Cord Compression

Typical exam case: A 40-year-old man has progressive weakness of both legs for 2 months with back pain, numbness below the umbilicus and difficulty passing urine. He has spastic paraparesis, brisk reflexes, up-going plantars and a sensory level at T10.

HISTORY AND EXAMINATION

Q1. What is paraplegia?

Weakness or paralysis of both lower limbs; paraparesis is partial weakness.

Q2. What history is important?

Speed of onset, back pain, sensory symptoms, bladder and bowel problems, fever, weight loss, trauma, TB contact and cancer history.

Q3. What does sudden onset suggest?

Trauma, spinal cord infarction, haemorrhage, or acute disc prolapse.

Q4. What does gradual onset with back pain and fever suggest?

Spinal TB (Pott's disease) or epidural abscess.

Q5. What does a cancer history suggest?

Metastatic spinal cord compression.

Q6. What are the signs of an upper motor neuron lesion in the legs?

Spasticity, weakness in a pyramidal pattern, brisk reflexes, clonus and up-going plantars.

Q7. What is a sensory level?

The level below which sensation is lost; it localises a spinal cord lesion.

Q8. What is the T10 dermatome?

The level of the umbilicus.

Q9. Why examine the spine?

For tenderness, deformity (gibbus in TB), swelling and scars.

Q10. What is spinal shock?

Early flaccid paralysis with absent reflexes after acute cord injury; spasticity develops later.

CAUSES AND LOCALISATION

Q11. How are causes of paraplegia grouped?

Compressive (TB, tumours, disc, trauma, abscess) and non-compressive (transverse myelitis, multiple sclerosis, B12 deficiency, MND, hereditary spastic paraparesis).

Q12. What is the commonest cause of spinal cord compression in Pakistan?

Spinal tuberculosis (Pott's disease).

Q13. Which cancers commonly spread to the spine?

Lung, breast, prostate, kidney, thyroid and myeloma.

Q14. What is subacute combined degeneration of the cord?

Vitamin B12 deficiency affecting dorsal columns and corticospinal tracts: loss of vibration and position sense, ataxia, absent ankle jerks and up-going plantars.

Q15. What is cauda equina syndrome?

> Compression of nerve roots below L1, causing saddle anaesthesia, bladder and bowel dysfunction, and lower motor neuron leg weakness.

Q16. What is Brown-Séquard syndrome?

> Cord hemisection: weakness and loss of position sense on the same side, loss of pain and temperature on the opposite side.

Q17. What is anterior spinal artery syndrome?

> Infarction of the front of the cord: paralysis and loss of pain and temperature, with preserved vibration and position sense.

Q18. What is transverse myelitis?

> Inflammation across the spinal cord causing sudden weakness, sensory level and bladder problems, often after infection.

INVESTIGATIONS

Q19. What is the investigation of choice?

> Urgent MRI of the whole spine.

Q20. What does MRI show in spinal TB?

> Destruction of adjacent vertebral bodies and the disc, with paravertebral or epidural abscess.

Q21. What does MRI show in metastatic disease?

> Vertebral body lesions with sparing of the disc and cord compression.

Q22. What blood tests are done?

> FBC, ESR, CRP, B12, calcium, protein electrophoresis, PSA (men) and HIV.

Q23. How is spinal TB confirmed?

> CT-guided biopsy of the lesion for histology, AFB, GeneXpert and culture.

Q24. What is the role of CSF examination?

> In non-compressive myelopathy (e.g. transverse myelitis, MS) for cells, protein and oligoclonal bands.

Q25. What does a plain X-ray show in spinal TB?

> Disc space narrowing, vertebral collapse and kyphosis (gibbus).

Q26. What is checked for MS?

> MRI brain and spine for demyelinating lesions and CSF oligoclonal bands.

Q27. What bladder test is done?

> Post-void residual volume by bladder scan.

Q28. What is the role of chest X-ray?

> To look for pulmonary TB or a lung primary tumour.

MANAGEMENT

Q29. What is the urgency of suspected cord compression?

> It is a medical emergency; MRI within 24 hours (sooner with neurological deficit).

Q30. How is metastatic spinal cord compression treated?

> Dexamethasone 16 mg daily, flat bed rest if spinal instability is suspected, and urgent radiotherapy or surgical decompression.

Q31. How is spinal TB treated?

> Anti-TB treatment for 9–12 months, rest and bracing; surgery for cord compression, instability or deformity.

Q32. How is cauda equina syndrome treated?

> Urgent surgical decompression.

Q33. How is B12 deficiency myelopathy treated?

> Intramuscular hydroxocobalamin; avoid giving folate alone.

Q34. How is transverse myelitis treated?

> High-dose IV methylprednisolone, and plasma exchange if severe.

Q35. How is the bladder managed?

> Catheterisation, then intermittent self-catheterisation if needed.

Q36. How is the bowel managed?

> Regular laxatives and a bowel program.

Q37. How are pressure sores prevented?

> 2-hourly turning, pressure-relieving mattress, good nutrition and skin care.

Q38. How is spasticity treated?

> Physiotherapy, baclofen, tizanidine and botulinum toxin.

Q39. How is DVT prevented?

> LMWH and compression devices.

Q40. What is the role of rehabilitation?

> Physiotherapy, occupational therapy, wheelchair training and psychological support.

Q41. What sexual health support is needed?

> Counselling and treatment of erectile dysfunction, and fertility advice.

Q42. What advice prevents autonomic dysreflexia triggers?

> Keep the bladder and bowel empty, avoid tight clothing and check skin.

Q43. What is the prognosis for recovery?

> Best when compression is relieved early and before complete paralysis.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of paraplegia?

> Pressure sores, UTIs, kidney stones, DVT, contractures, osteoporosis, constipation, depression and autonomic dysreflexia.

Q45. What is autonomic dysreflexia?

> Sudden severe hypertension, headache and sweating in patients with lesions above T6, triggered by a full bladder or bowel.

Q46. How is autonomic dysreflexia treated?

> Sit the patient up, remove the trigger (catheterise, empty bowel) and give fast-acting antihypertensives.

Q47. Why are UTIs common?

> Incomplete bladder emptying and catheter use.

Q48. What is the most important factor in outcome for MSCC?

> The patient's ability to walk at the time of diagnosis.

Q49. Why should back pain in a cancer patient be taken seriously?

> It may be the first sign of spinal metastases before cord compression.

Q50. What is a recent advance?

> Stereotactic body radiotherapy for spinal metastases and short-course TB regimens with better spinal surgery outcomes.

CASE 16

Meningitis

> **Typical exam case:** A 22-year-old student presents with fever, severe headache, vomiting and photophobia for 1 day. He has neck stiffness and a positive Kernig's sign. A non-blanching purpuric rash is seen on his legs.

HISTORY AND EXAMINATION

Q1. What is meningitis?

> Inflammation of the meninges, usually due to infection.

Q2. What are the classic symptoms?

> Fever, headache, neck stiffness, photophobia, vomiting and confusion.

Q3. What does a non-blanching rash suggest?

> Meningococcal septicaemia, a medical emergency.

Q4. What is the glass test?

> Pressing a glass on the rash; if it does not fade, it is non-blanching.

Q5. What is Kernig's sign?

> Pain or resistance when straightening the knee with the hip flexed.

Q6. What is Brudzinski's sign?

> Flexing the neck causes the hips and knees to flex.

Q7. What history suggests TB meningitis?

> Gradual onset over weeks, low-grade fever, weight loss, behaviour change, cranial nerve palsies and TB contact.

Q8. What features suggest encephalitis?

> Confusion, behavioural change, seizures and focal signs, more than neck stiffness.

Q9. What should you assess urgently?

> Airway, breathing, circulation, GCS, signs of shock and raised intracranial pressure.

Q10. What signs suggest raised intracranial pressure?

> Reduced consciousness, papilloedema, focal signs, bradycardia with hypertension, and seizures.

CAUSES

Q11. What are the common bacterial causes in adults?

> Streptococcus pneumoniae and Neisseria meningitidis.

Q12. Which bacterium is important in the elderly, pregnant and immunosuppressed?

> Listeria monocytogenes.

Q13. What are the causes in newborns?

> Group B streptococcus, E. coli and Listeria.

Q14. What is the commonest cause of meningitis overall?

> Viral (enteroviruses), usually mild.

Q15. Which virus causes encephalitis that needs urgent aciclovir?

> Herpes simplex virus.

Q16. What causes chronic meningitis in Pakistan?

> Tuberculosis, and less often fungal (cryptococcal in HIV) and brucellosis.

Q17. What is aseptic meningitis?

> Meningitis with negative bacterial cultures, usually viral.

Q18. Which vaccines prevent bacterial meningitis?

> Hib, pneumococcal and meningococcal (MenB, MenACWY) vaccines.

INVESTIGATIONS

Q19. What is the key investigation?

> Lumbar puncture with CSF analysis, if there is no contraindication.

Q20. When should CT head be done before LP?

> With reduced GCS, focal neurological signs, papilloedema, new seizures or immunosuppression.

Q21. What are contraindications to LP?

> Signs of raised ICP, coagulopathy, low platelets, infection at the site and shock.

Q22. What are CSF findings in bacterial meningitis?

> Cloudy, high neutrophils, high protein and low glucose (CSF:blood ratio below 0.4).

Q23. What are CSF findings in viral meningitis?

> Clear, lymphocytes, normal or slightly high protein and normal glucose.

Q24. What are CSF findings in TB meningitis?

> Lymphocytes, very high protein, low glucose, and sometimes a fibrin web.

Q25. What blood tests are done?

> Blood cultures, FBC, CRP, U&E, glucose (for CSF ratio), clotting and meningococcal PCR.

Q26. What CSF tests are sent?

> Cell count, protein, glucose, Gram stain, culture, PCR for bacteria and viruses, AFB and GeneXpert.

Q27. Which test is used for cryptococcal meningitis?

> CSF India ink stain and cryptococcal antigen.

Q28. Why measure opening pressure?

> High pressure is common in cryptococcal and TB meningitis and guides management.

MANAGEMENT

Q29. What is the first action in suspected meningococcal sepsis in the community?

> Give IM or IV benzylpenicillin and transfer urgently to hospital.

Q30. What empirical antibiotic is used in hospital for adults?

> IV ceftriaxone 2 g every 12 hours.

Q31. When is amoxicillin added?

> In patients over 60, pregnant or immunosuppressed, to cover Listeria.

Q32. When is vancomycin added?

> If the patient has travelled to areas with penicillin-resistant pneumococcus.

Q33. Why is dexamethasone given?

> It reduces hearing loss and neurological complications, especially in pneumococcal meningitis; given with or just before the first antibiotic dose.

Q34. Should antibiotics be delayed for LP or CT?

> No. Give antibiotics within 1 hour if LP cannot be done quickly.

Q35. What is given if HSV encephalitis is suspected?

> IV aciclovir.

Q36. How is TB meningitis treated?

> Anti-TB treatment for 12 months with dexamethasone.

Q37. How is shock managed?

> IV fluids, vasopressors and ICU support.

Q38. How long are antibiotics given?

> Usually 7 days for meningococcal, 10–14 days for pneumococcal, and 21 days for Listeria.

Q39. What is given to close contacts of meningococcal meningitis?

> Prophylaxis with ciprofloxacin (single dose) or rifampicin.

Q40. Which public health step is needed?

> Notify public health authorities.

Q41. How is raised ICP managed?

> Head elevation, airway protection, controlled ventilation and treatment of seizures.

Q42. How are seizures treated?

> Benzodiazepines then levetiracetam or phenytoin.

Q43. How is cryptococcal meningitis treated?

> Amphotericin B with flucytosine, then fluconazole, and repeated LPs for high pressure.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of bacterial meningitis?

> Hearing loss, seizures, hydrocephalus, brain abscess, cranial nerve palsies, cognitive problems and death.

Q45. What is Waterhouse-Friderichsen syndrome?

> Adrenal haemorrhage in meningococcal septicaemia, causing shock.

Q46. What complication is typical of TB meningitis?

> Hydrocephalus, strokes from vasculitis and cranial nerve palsies.

Q47. Why is a hearing test done after meningitis?

> Sensorineural hearing loss is common, especially in children.

Q48. What is the mortality of bacterial meningitis?

> About 10–20%, higher with pneumococcus and delayed treatment.

Q49. Why is early treatment vital?

> Every hour of delay increases death and disability.

Q50. What is a recent advance in meningitis?

> Rapid multiplex CSF PCR panels, and the WHO roadmap to defeat meningitis by 2030 through vaccination.

CASE 17

Anaemia

Typical exam case: A 35-year-old woman has tiredness, breathlessness on exertion and palpitations. She has heavy periods. She looks pale and has koilonychia and angular stomatitis. Hb is 78 g/L, MCV 66 fL.

HISTORY AND EXAMINATION

Q1. What is anaemia?

A haemoglobin below the normal range: below 130 g/L in men and below 120 g/L in women (WHO).

Q2. What are the symptoms of anaemia?

Tiredness, breathlessness, palpitations, dizziness, headache, angina and reduced exercise tolerance.

Q3. What history would you take for iron deficiency?

Blood loss (heavy periods, GI symptoms, melaena), diet, pregnancy, coeliac symptoms, NSAIDs and worms.

Q4. Why is GI blood loss important in men and postmenopausal women?

Iron deficiency in them must be investigated for GI cancer.

Q5. What signs of anaemia are seen?

Pallor of conjunctiva and palms, tachycardia, flow murmur and signs of heart failure if severe.

Q6. What signs suggest iron deficiency?

Koilonychia (spoon nails), angular stomatitis, glossitis and brittle hair.

Q7. What signs suggest B12 deficiency?

Lemon-yellow skin, glossitis, peripheral neuropathy, loss of vibration and position sense, and dementia.

Q8. What signs suggest haemolysis?

Jaundice, splenomegaly and dark urine.

Q9. What signs suggest bone marrow failure or leukaemia?

Bruising, bleeding, infections, lymph nodes and splenomegaly.

Q10. Why do you check for lymph nodes and spleen?

To look for haematological cancers, chronic infection and haemolysis.

CLASSIFICATION AND CAUSES

Q11. How is anaemia classified by MCV?

Microcytic (below 80 fL), normocytic (80–100 fL) and macrocytic (over 100 fL).

Q12. What are causes of microcytic anaemia?

Iron deficiency, thalassaemia, anaemia of chronic disease (sometimes) and sideroblastic anaemia.

Q13. What are causes of normocytic anaemia?

Acute blood loss, chronic disease, CKD, haemolysis, bone marrow failure and mixed deficiency.

Q14. What are causes of macrocytic anaemia?

B12 or folate deficiency, alcohol, liver disease, hypothyroidism, drugs (methotrexate, hydroxyurea), myelodysplasia and reticulocytosis.

Q15. What is pernicious anaemia?

Autoimmune destruction of gastric parietal cells, causing lack of intrinsic factor and B12 malabsorption.

Q16. What is anaemia of chronic disease?

> Anaemia in chronic inflammation, infection or cancer, with low iron but normal or high ferritin.

Q17. What is the commonest cause of anaemia worldwide?

> Iron deficiency.

Q18. What are the causes of haemolytic anaemia?

> Inherited (thalassaemia, sickle cell, G6PD deficiency, spherocytosis) and acquired (autoimmune, drugs, malaria, mechanical valves).

INVESTIGATIONS

Q19. What is the first test?

> Full blood count with red cell indices and reticulocyte count.

Q20. What is the best test for iron deficiency?

> Serum ferritin; a low level confirms iron deficiency.

Q21. What does a blood film show in iron deficiency?

> Microcytic hypochromic cells, pencil cells and target cells.

Q22. How do you distinguish iron deficiency from thalassaemia trait?

> Thalassaemia trait has a normal ferritin, high red cell count and raised HbA2 on electrophoresis (Mentzer index below 13).

Q23. How is iron deficiency investigated in a man or postmenopausal woman?

> Coeliac serology, and upper GI endoscopy and colonoscopy.

Q24. What tests are done in macrocytic anaemia?

> B12, folate, LFTs, TFTs, and blood film (hypersegmented neutrophils in megaloblastic anaemia).

Q25. How is pernicious anaemia confirmed?

> Intrinsic factor antibodies (specific) and parietal cell antibodies.

Q26. What tests suggest haemolysis?

> High reticulocytes, high bilirubin, high LDH, low haptoglobin and a direct Coombs test.

Q27. When is a bone marrow biopsy done?

> For unexplained anaemia, pancytopenia, suspected leukaemia, myeloma or myelodysplasia.

Q28. What does the reticulocyte count tell you?

> High in haemolysis and bleeding (marrow working); low in deficiency or marrow failure.

MANAGEMENT

Q29. How is iron deficiency anaemia treated?

> Treat the cause and give oral iron, e.g. ferrous sulfate 200 mg once daily or on alternate days.

Q30. How fast should Hb rise on iron?

> About 20 g/L in 3–4 weeks.

Q31. How long is iron continued?

> For about 3 months after Hb normalises to replenish stores.

Q32. When is IV iron used?

> Intolerance or failure of oral iron, malabsorption, CKD, heart failure, or a need for rapid correction.

Q33. How is B12 deficiency treated?

> IM hydroxocobalamin 1 mg on alternate days for 2 weeks (longer if neurological), then every 2–3 months for life in pernicious anaemia.

Q34. Why must B12 be given before folate?

> Giving folate alone in B12 deficiency can worsen subacute combined degeneration of the cord.

Q35. How is folate deficiency treated?

> Oral folic acid 5 mg daily for 4 months, after excluding B12 deficiency.

Q36. When is blood transfusion indicated?

> Acute bleeding, severe symptomatic anaemia, heart failure or Hb below 70 g/L in most patients (80 g/L with heart disease).

Q37. What is a risk of transfusing a patient with chronic severe anaemia?

> Fluid overload and heart failure; transfuse slowly with diuretics.

Q38. How is autoimmune haemolytic anaemia treated?

> Steroids, then rituximab or splenectomy if needed.

Q39. How is beta-thalassaemia major treated?

> Regular transfusions with iron chelation (desferrioxamine, deferasirox), folic acid, and bone marrow transplant.

Q40. How is G6PD deficiency managed?

> Avoid triggers such as fava beans, primaquine, dapsone and sulphonamides; transfuse if severe.

Q41. How is anaemia of CKD treated?

> Iron and erythropoiesis-stimulating agents.

Q42. What dietary advice is given for iron?

> Red meat, liver, green leafy vegetables, lentils and fortified cereals; take with vitamin C and avoid tea with meals.

Q43. What prevention steps help in Pakistan?

> Iron and folic acid supplements in pregnancy, deworming, food fortification and thalassaemia carrier screening.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of severe anaemia?

> High-output heart failure, angina, falls, poor pregnancy outcomes and reduced cognition in children.

Q45. What are the complications of B12 deficiency?

> Neuropathy, subacute combined degeneration of the cord, dementia and optic atrophy.

Q46. What is iron overload from repeated transfusion?

> Haemosiderosis affecting heart, liver and endocrine glands; treated with chelation.

Q47. Why is thalassaemia screening important in Pakistan?

> Carrier rates are about 5–7%; premarital and antenatal screening can prevent thalassaemia major.

Q48. What is the risk with a ferritin result in inflammation?

> Ferritin rises in inflammation and may hide iron deficiency.

Q49. What is the danger of missing GI cancer?

> Iron deficiency anaemia may be its only sign.

Q50. What is a recent advance in anaemia?

> Single-dose IV iron (ferric carboxymaltose, ferric derisomaltose), luspatercept for thalassaemia, and gene therapy for beta-thalassaemia and sickle cell disease.

CASE 18

Thyroid Disease: Hyperthyroidism and Hypothyroidism

> **Typical exam case:** A 30-year-old woman has weight loss despite a good appetite, palpitations, heat intolerance, tremor and anxiety for 3 months. She has a diffuse goitre with a bruit, lid lag and proptosis.

HISTORY AND EXAMINATION

Q1. What are the symptoms of hyperthyroidism?

> Weight loss, increased appetite, heat intolerance, sweating, palpitations, tremor, anxiety, diarrhoea and light or absent periods.

Q2. What are the symptoms of hypothyroidism?

> Weight gain, cold intolerance, tiredness, constipation, dry skin, hair loss, slow thinking, depression and heavy periods.

Q3. What signs of hyperthyroidism would you look for?

> Tachycardia or AF, fine tremor, warm sweaty palms, lid lag, lid retraction, goitre and proximal muscle weakness.

Q4. What signs are specific to Graves' disease?

> Exophthalmos, ophthalmoplegia, pretibial myxoedema, thyroid acropachy and a goitre with bruit.

Q5. What signs of hypothyroidism would you look for?

> Bradycardia, dry coarse skin, puffy face, loss of outer third of eyebrows, slow-relaxing reflexes and carpal tunnel syndrome.

Q6. How do you examine the thyroid?

> From behind, with the patient swallowing water; check size, nodules, tenderness, lymph nodes and retrosternal extension.

Q7. Why does a thyroid swelling move on swallowing?

> It is attached to the larynx by the pretracheal fascia.

Q8. What does a bruit over the thyroid suggest?

> Increased blood flow in Graves' disease.

Q9. What drug history is important?

> Amiodarone, lithium, iodine, interferon and immune checkpoint inhibitors.

Q10. Why ask about recent neck pain or viral illness?

> Subacute (de Quervain's) thyroiditis causes a painful goitre and temporary hyperthyroidism.

CAUSES AND DIAGNOSIS

Q11. What is the commonest cause of hyperthyroidism?

> Graves' disease.

Q12. What causes Graves' disease?

> Autoantibodies that stimulate the TSH receptor (TRAb).

Q13. What are other causes of hyperthyroidism?

> Toxic multinodular goitre, toxic adenoma, thyroiditis (subacute, postpartum), and drugs (amiodarone, excess thyroxine).

Q14. What is the commonest cause of hypothyroidism in iodine-sufficient areas?

> Hashimoto's (autoimmune) thyroiditis.

Q15. What is the commonest cause worldwide?

> Iodine deficiency.

Q16. What are other causes of hypothyroidism?

> After radioiodine or thyroidectomy, drugs (carbimazole, lithium, amiodarone) and pituitary failure.

Q17. What is subclinical hypothyroidism?

> High TSH with normal free T4.

Q18. What is secondary (central) hypothyroidism?

> Low or normal TSH with low T4, due to pituitary or hypothalamic disease.

INVESTIGATIONS

Q19. What is the first test?

> TSH, with free T4 (and free T3 if TSH is low).

Q20. What are the results in primary hyperthyroidism?

> Low TSH, high free T4 and/or T3.

Q21. What are the results in primary hypothyroidism?

> High TSH, low free T4.

Q22. What is T3 toxicosis?

> Hyperthyroidism with high T3 but normal T4.

Q23. What antibody confirms Graves' disease?

> TSH receptor antibody (TRAb).

Q24. What antibody suggests Hashimoto's?

> Anti-TPO antibodies.

Q25. When is a thyroid uptake scan used?

> To distinguish causes of hyperthyroidism: diffuse high uptake (Graves), patchy (multinodular), single hot nodule (adenoma), low uptake (thyroiditis).

Q26. What other tests may be abnormal?

> Raised ALP, high calcium and low WBC in hyperthyroidism; high cholesterol, low sodium and macrocytosis in hypothyroidism.

Q27. Why is ultrasound used?

> To assess nodules and goitre structure, and guide FNA.

Q28. Why check FBC and LFTs before starting carbimazole?

> As a baseline because of risks of agranulocytosis and liver injury.

MANAGEMENT

Q29. How are hyperthyroid symptoms controlled quickly?

> Beta-blockers such as propranolol.

Q30. What antithyroid drugs are used?

> Carbimazole first line; propylthiouracil in the first trimester of pregnancy.

Q31. What is the most serious side effect of carbimazole?

> Agranulocytosis; patients must stop it and have an urgent FBC if they get a sore throat or fever.

Q32. What is the block-and-replace regimen?

> High-dose carbimazole with levothyroxine added.

Q33. How long is antithyroid drug treatment given in Graves' disease?

> 12–18 months, after which about half relapse.

Q34. What is radioactive iodine?

> Oral I-131 that destroys thyroid tissue; it often causes hypothyroidism later.

Q35. What are contraindications to radioiodine?

> Pregnancy, breastfeeding and active moderate-severe thyroid eye disease.

Q36. When is thyroidectomy preferred?

> Large goitre with pressure symptoms, suspected cancer, eye disease, or patient preference.

Q37. How is thyroid eye disease treated?

> Stop smoking, lubricants, selenium for mild disease, steroids for active moderate-severe disease, and orbital decompression if needed.

Q38. What is thyroid storm and how is it treated?

> Life-threatening hyperthyroidism with fever, tachycardia, confusion; treat with propranolol, PTU, hydrocortisone, iodine (after PTU) and supportive care.

Q39. How is hypothyroidism treated?

> Levothyroxine, adjusted by TSH every 6–8 weeks until stable.

Q40. How should levothyroxine be started in elderly or heart disease patients?

> At a low dose (25 micrograms) and increased slowly to avoid angina.

Q41. How does pregnancy change levothyroxine needs?

> Dose increases by about 25–50%; check TSH early in pregnancy.

Q42. What is myxoedema coma and how is it treated?

> Severe hypothyroidism with hypothermia, confusion and bradycardia; IV thyroid hormone, IV hydrocortisone and supportive care.

Q43. Why give hydrocortisone before thyroxine in suspected hypopituitarism?

> To avoid precipitating an adrenal crisis.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of hyperthyroidism?

> Atrial fibrillation, heart failure, osteoporosis, thyroid storm and eye disease.

Q45. What are the complications of hypothyroidism?

> Hyperlipidaemia, heart disease, infertility, depression and myxoedema coma.

Q46. What are the complications of thyroidectomy?

> Bleeding with airway compression, recurrent laryngeal nerve injury, hypocalcaemia (parathyroid damage) and hypothyroidism.

Q47. What is congenital hypothyroidism?

> Hypothyroidism from birth; screened by the heel-prick test and treated early to prevent learning disability.

Q48. Why is iodised salt important in Pakistan?

> Iodine deficiency causes goitre and hypothyroidism; salt iodisation prevents it.

Q49. What is sick euthyroid syndrome?

> Abnormal thyroid tests during severe illness without true thyroid disease.

Q50. What is a recent advance in thyroid disease?

> Teprotumumab (IGF-1 receptor antibody) for active thyroid eye disease, and radiofrequency ablation of benign nodules.

CASE 19

Rheumatoid Arthritis

Typical exam case: A 42-year-old woman has painful swollen small joints of both hands and wrists for 6 months, with morning stiffness lasting 2 hours. There is symmetrical swelling of the MCP and PIP joints; the DIP joints are spared.

HISTORY AND EXAMINATION

Q1. What is rheumatoid arthritis?

A chronic autoimmune inflammatory arthritis mainly affecting the synovium of small joints, symmetrically.

Q2. What are the typical symptoms?

Symmetrical pain and swelling of small joints of the hands and feet, morning stiffness over 30 minutes, fatigue and low-grade fever.

Q3. Which joints are typically involved?

MCP, PIP, wrists and MTP joints; DIP joints are usually spared.

Q4. What is the significance of prolonged morning stiffness?

It suggests inflammatory rather than mechanical arthritis.

Q5. What are the late hand deformities?

Ulnar deviation, swan neck, boutonnière, Z-thumb and volar subluxation of MCPs.

Q6. What are rheumatoid nodules?

Firm, non-tender subcutaneous nodules over pressure areas such as the elbows; linked to seropositive disease.

Q7. What extra-articular features would you look for?

Nodules, anaemia, dry eyes (Sjögren's), scleritis, lung fibrosis, pleural effusion, pericarditis, vasculitis and neuropathy.

Q8. Why examine the cervical spine?

Atlantoaxial subluxation can cause cord compression, important before anaesthesia.

Q9. What risk factors are linked with RA?

Female sex, smoking, family history and HLA-DR4.

Q10. How is functional impact assessed?

By asking about daily activities, work and self-care (HAQ questionnaire).

DIAGNOSIS

Q11. What are the 2010 ACR/EULAR classification criteria based on?

Number and size of joints involved, serology (RF, anti-CCP), acute phase reactants (ESR, CRP) and duration of 6 weeks or more.

Q12. Which antibody is most specific for RA?

Anti-cyclic citrullinated peptide (anti-CCP) antibody.

Q13. What is rheumatoid factor?

An antibody against IgG; positive in about 70% of RA but also in other diseases and healthy elderly people.

Q14. How does RA differ from osteoarthritis?

> RA: symmetrical small joints, long morning stiffness, soft swelling, raised ESR. OA: large weight-bearing joints and DIPs, short stiffness, bony swelling (Heberden's nodes).

Q15. How does psoriatic arthritis differ?

> It often involves DIP joints, is asymmetrical, with psoriasis, nail pitting and dactylitis.

Q16. What is Felty's syndrome?

> RA with splenomegaly and neutropenia.

Q17. What is palindromic rheumatism?

> Recurrent short attacks of joint inflammation that may progress to RA.

Q18. What other conditions can mimic RA?

> SLE, viral arthritis, gout, hypothyroidism and haemochromatosis.

INVESTIGATIONS

Q19. What blood tests are done?

> FBC, ESR, CRP, rheumatoid factor, anti-CCP, U&E, LFTs and ANA.

Q20. What does the FBC often show?

> Normocytic anaemia of chronic disease and raised platelets.

Q21. What are the early X-ray features of RA?

> Soft tissue swelling and periarticular osteopenia.

Q22. What are the later X-ray features?

> Loss of joint space, marginal erosions, subluxation and ankylosis.

Q23. What is the role of ultrasound and MRI?

> To detect synovitis and early erosions before X-ray changes.

Q24. What is the DAS28 score?

> A disease activity score using tender and swollen joint counts in 28 joints, ESR or CRP and patient global assessment.

Q25. What DAS28 score indicates remission?

> Below 2.6.

Q26. Why are hepatitis B, C and TB screened before biologics?

> Biologics can reactivate these infections.

Q27. What tests are needed before methotrexate?

> FBC, LFTs, U&E, chest X-ray and hepatitis serology.

Q28. What does synovial fluid show in RA?

> Inflammatory fluid with high white cells, mainly neutrophils, and no crystals.

MANAGEMENT

Q29. What is the principle of treating RA?

> Early treatment with DMARDs to control inflammation and prevent joint damage, aiming for remission (treat to target).

Q30. What is the first-line DMARD?

> Methotrexate, often with short-term steroids as bridging.

Q31. How is methotrexate given?

> Once weekly, with folic acid on another day.

Q32. What are the side effects of methotrexate?

> Bone marrow suppression, liver toxicity, pneumonitis, mouth ulcers and teratogenicity.

Q33. What are other conventional DMARDs?

> Sulfasalazine, hydroxychloroquine and leflunomide.

Q34. What eye check is needed with hydroxychloroquine?

> Retinal screening because of retinal toxicity after long-term use.

Q35. What are biologic DMARDs?

> Anti-TNF (adalimumab, etanercept, infliximab), rituximab, abatacept and tocilizumab.

Q36. What are JAK inhibitors?

> Oral targeted DMARDs such as tofacitinib and baricitinib.

Q37. When are biologics used?

> When disease remains active despite at least two conventional DMARDs.

Q38. What is the role of NSAIDs?

> For symptom relief only; they do not change the disease course.

Q39. How are flares treated?

> Short courses of oral steroids or intra-articular or IM steroid injections.

Q40. What non-drug treatments help?

> Physiotherapy, occupational therapy, splints, exercise and stopping smoking.

Q41. When is surgery needed?

> For joint destruction, tendon rupture or nerve compression, e.g. joint replacement or synovectomy.

Q42. How is RA managed in pregnancy?

> Stop methotrexate and leflunomide before conception; hydroxychloroquine, sulfasalazine and some biologics are safer.

Q43. Why is cardiovascular risk managed in RA?

> RA doubles the risk of heart disease; control inflammation, BP, lipids and smoking.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of RA?

> Joint destruction, disability, anaemia, cardiovascular disease, osteoporosis, infections, lung disease and lymphoma.

Q45. What lung problems occur in RA?

> Interstitial lung disease, nodules, pleural effusion and methotrexate pneumonitis.

Q46. What is Caplan's syndrome?

> Lung nodules in RA patients exposed to coal dust.

Q47. Why are vaccines important?

> Immunosuppression increases infection risk; give influenza and pneumococcal vaccines (avoid live vaccines on biologics).

Q48. What is the prognosis of RA?

> Variable; early aggressive treatment greatly improves outcomes.

Q49. What predicts severe disease?

> Positive anti-CCP and RF, early erosions, high CRP and many joints involved.

Q50. What is a recent advance in RA?

> JAK inhibitors and biosimilar biologics making treatment more affordable, and the treat-to-target strategy.

CASE 20

Fever: Enteric Fever, Malaria and Dengue

Typical exam case: A 25-year-old man from Karachi has had high fever for 7 days with headache, abdominal pain and constipation. His pulse is 84/min despite a temperature of 39.5 °C. He has a coated tongue and a soft, palpable spleen.

HISTORY AND EXAMINATION

Q1. What history is important in a patient with fever?

Duration and pattern, travel and residence, contacts, water and food sources, mosquito exposure, rash, bleeding and localising symptoms.

Q2. What are the features of typhoid fever?

Step-ladder rising fever, headache, abdominal pain, constipation or diarrhoea, relative bradycardia, coated tongue, rose spots and splenomegaly.

Q3. What is relative bradycardia?

A pulse slower than expected for the degree of fever; seen in typhoid and some other infections.

Q4. What are the features of malaria?

Fever with chills and rigors, sweating, headache, myalgia, and in severe cases confusion, jaundice and dark urine.

Q5. What are the features of dengue fever?

Sudden high fever, severe headache, pain behind the eyes, muscle and joint pain ('break-bone fever') and a rash.

Q6. What are the warning signs in dengue?

Abdominal pain, persistent vomiting, bleeding, fluid accumulation, lethargy, enlarged liver and a rising haematocrit with falling platelets.

Q7. What signs would you look for on examination?

Pallor, jaundice, rash, lymph nodes, liver and spleen size, signs of bleeding, hydration and BP.

Q8. What is the tourniquet test?

A BP cuff inflated between systolic and diastolic for 5 minutes; 10 or more petechiae per square inch suggests dengue.

Q9. Why is the season important in Pakistan?

Dengue and malaria peak after the monsoon (August–November).

Q10. What features suggest other diagnoses?

Neck stiffness (meningitis), cough (pneumonia, TB), urinary symptoms (UTI), and night sweats with weight loss (TB, lymphoma).

CAUSES AND DIAGNOSIS

Q11. Which organism causes typhoid?

Salmonella Typhi (and Paratyphi for paratyphoid), spread by contaminated food and water.

Q12. What is XDR typhoid?

Extensively drug-resistant Salmonella Typhi, first seen in Sindh in 2016, resistant to first-line drugs, fluoroquinolones and third-generation cephalosporins.

Q13. Which malaria species occur in Pakistan?

> Mainly Plasmodium vivax, with P. falciparum causing most severe cases.

Q14. Which malaria species causes severe disease?

> Plasmodium falciparum.

Q15. What is severe malaria?

> Malaria with features such as impaired consciousness, seizures, severe anaemia, kidney injury, jaundice, hypoglycaemia, acidosis, shock or high parasitaemia.

Q16. What spreads dengue?

> The Aedes aegypti mosquito, which bites in the daytime.

Q17. What are the phases of dengue?

> Febrile phase, critical phase (days 3–7, plasma leakage), and recovery phase.

Q18. What is severe dengue?

> Dengue with severe plasma leakage (shock), severe bleeding or severe organ involvement.

INVESTIGATIONS

Q19. What is the best test for typhoid in the first week?

> Blood culture.

Q20. Why is the Widal test unreliable?

> It has many false positives and negatives, especially in endemic areas.

Q21. When is bone marrow culture useful?

> It is the most sensitive test, especially after antibiotics have been taken.

Q22. How is malaria diagnosed?

> Thick and thin blood films, or rapid diagnostic tests (antigen tests).

Q23. Why is the thin film useful?

> To identify the species and count parasitaemia.

Q24. How is dengue diagnosed in the first 5 days?

> NS1 antigen or PCR.

Q25. How is dengue diagnosed after day 5?

> Dengue IgM antibodies.

Q26. What blood count changes occur in dengue?

> Low white cells, falling platelets and a rising haematocrit (plasma leakage).

Q27. What blood count changes occur in typhoid?

> Normal or low white cells, sometimes with anaemia and low platelets.

Q28. What other tests are done in severe malaria?

> Glucose, U&E, LFTs, lactate, ABG and blood cultures.

MANAGEMENT

Q29. What is the treatment of uncomplicated typhoid in Pakistan?

> Azithromycin orally, as XDR strains are common.

Q30. What is the treatment of severe or XDR typhoid?

> Meropenem IV, often with azithromycin.

Q31. How long is typhoid treated?

> Usually 7–14 days depending on the drug and severity.

Q32. How is uncomplicated vivax malaria treated?

> Chloroquine (or an ACT) followed by primaquine to clear liver hypnozoites.

Q33. What test is needed before primaquine?

> G6PD level, because primaquine can cause haemolysis in G6PD deficiency.

Q34. How is uncomplicated falciparum malaria treated?

> Artemisinin combination therapy (e.g. artemether-lumefantrine).

Q35. How is severe malaria treated?

> IV artesunate, then a full course of ACT, with supportive care.

Q36. How is dengue without warning signs managed?

> Rest, oral fluids, paracetamol for fever, and daily monitoring of platelets and haematocrit.

Q37. Why are NSAIDs and aspirin avoided in dengue?

> They increase bleeding risk.

Q38. How is dengue with warning signs managed?

> Admission and careful IV crystalloid fluids guided by haematocrit and urine output.

Q39. How is dengue shock managed?

> Rapid IV crystalloid boluses, then colloids if needed, with close monitoring and blood transfusion for significant bleeding.

Q40. When are platelet transfusions given in dengue?

> Only for significant bleeding, not routinely for low platelet counts.

Q41. Why must fluids be reduced in the recovery phase of dengue?

> Fluid returns to the circulation and can cause overload and pulmonary oedema.

Q42. What prevents typhoid?

> Safe water, sanitation, hand washing and the typhoid conjugate vaccine (introduced in Pakistan's routine schedule).

Q43. What prevents malaria and dengue?

> Mosquito control, bed nets, repellents and covering skin; removing standing water for Aedes breeding.

COMPLICATIONS AND RECENT ADVANCES

Q44. What are the complications of typhoid?

> Intestinal perforation, GI bleeding, encephalopathy, myocarditis, hepatitis and relapse or chronic carriage.

Q45. When does typhoid perforation usually occur?

> In the third week, in the terminal ileum (Peyer's patches).

Q46. What is a chronic typhoid carrier?

> A person who excretes the bacteria for more than a year, often from the gallbladder.

Q47. What are complications of falciparum malaria?

> Cerebral malaria, severe anaemia, AKI, ARDS, hypoglycaemia, shock and blackwater fever.

Q48. Why can vivax malaria relapse?

> Dormant liver forms (hypnozoites) reactivate unless primaquine or tafenoquine is given.

Q49. What is the danger period in dengue?

> The critical phase, when fever falls (days 3–7) and plasma leakage peaks.

Q50. What is a recent advance?

> The typhoid conjugate vaccine for children in Pakistan, the RTS,S and R21 malaria vaccines, and tafenoquine single-dose radical cure for vivax malaria.