FMGE Pathology Sample Paper – 2

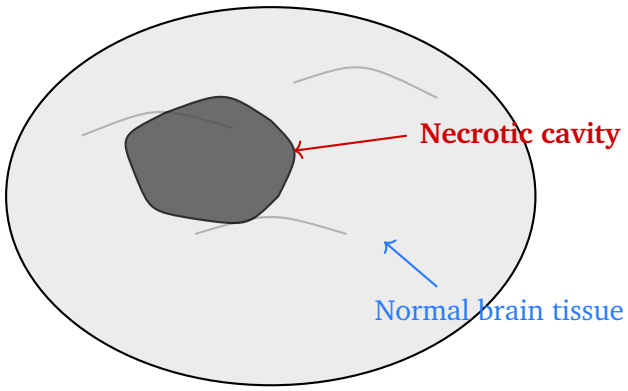
Duration: 60 Minutes **Maximum Marks: 40**

Instructions

- This paper contains **40 Multiple Choice Questions** modelled on the **Pathology** section of FMGE (General Pathology & Systemic Pathology).
- Each correct answer carries **+1 mark**. There is **no negative marking** – attempt all questions.
- Questions follow the clinically integrated pattern of the FMGE examination.
- Click a question number in the answer key to navigate to its solution.

FMGE Pathology – Q1 to Q40

Q1. A 55-year-old hypertensive man is brought to the emergency department with sudden-onset right-sided hemiplegia and aphasia. CT brain performed 48 hours later shows a low-density area in the left middle cerebral artery territory. At autopsy several weeks later, pathological examination of the infarcted area would most likely show which pattern of necrosis?



(A) Coagulative necrosis

(B) Caseous necrosis

(C) Liquefactive necrosis

(D) Fat necrosis

Q2. A pathologist examines renal tubular cells from a patient who suffered a brief ischaemic episode (15 minutes). Electron microscopy shows dilation of the endoplasmic reticulum, loss of microvilli from the apical surface, and swelling of mitochondria with normal cristae. The chromatin is uncondensed and the cell membrane is intact. These findings are most consistent with:

(A) Reversible cell injury (hydropic/cellular swelling)

(B) Irreversible cell injury leading to necrosis

(C) Apoptosis

(D) Normal ultrastructure

Q3. A 68-year-old man with primary hyperparathyroidism (serum calcium 13.2 mg/dL) undergoes autopsy after dying of a pulmonary embolism. Histological examination reveals basophilic calcium deposits in the walls of pulmonary blood vessels, renal tubular basement membranes, and gastric mucosa, all of which are histologically normal tissues. This pattern of calcification is best classified as:

(A) Dystrophic calcification

(B) Psammoma body formation

(C) Ossification

(D) Metastatic calcification

Q4. A 30-year-old woman presents with a dental abscess. Aspiration of the fluctuant swelling yields thick yellow-green pus. Microscopic examination of the pus would reveal which of the following as the predominant cellular component?

(A) Lymphocytes

(B) Neutrophils (polymorphonuclear leukocytes)

(C) Eosinophils

(D) Macrophages

Q5. A 25-year-old woman with systemic lupus erythematosus develops right-sided pleuritic chest pain and mild dyspnoea. Thoracentesis yields 400 mL of clear, straw-coloured fluid. Microscopy shows a few mesothelial cells and scattered lymphocytes with very few neutrophils. Protein content is 2.0 g/dL. This effusion is best characterised as which type of inflammatory exudate?

(A) Fibrinous inflammation

(B) Suppurative (purulent) inflammation

(C) Serous inflammation

(D) Haemorrhagic inflammation

Q6. A 45-year-old woman who underwent an abdominal operation 3 months ago now has a firm nodule at the surgical site. Excision biopsy shows granulomatous inflammation surrounding fragments of non-absorbable suture material. The giant cells seen in such a reaction are formed by fusion of which cells?

(A) Foreign body giant cells (fused macrophages/histiocytes)

(B) Langhans giant cells (epithelioid histiocytes with horseshoe nuclei, as in TB)

(C) Reed-Sternberg cells

(D) Touton giant cells (as in xanthomas)

Q7. A 40-year-old man sustains a large degloving wound to his thigh with extensive tissue loss. The wound is left open and allowed to heal without surgical closure. Compared with primary intention healing, this wound will heal by a process characterised by:

(A) Minimal granulation tissue formation

(B) Absence of wound contraction

(C) No scarring

(D) More granulation tissue, wound contraction by myofibroblasts, and a larger scar

Q8. A 28-year-old man receives a bee sting. At the site of injury, local vasodilatation and increased vascular permeability develop over 15–30 minutes. A researcher notes that one mediator generated from plasma kinin precursors via Hageman factor (Factor XII) activation causes both pain and sustained vascular permeability but is not responsible for the immediate vasodilatation. This mediator is:

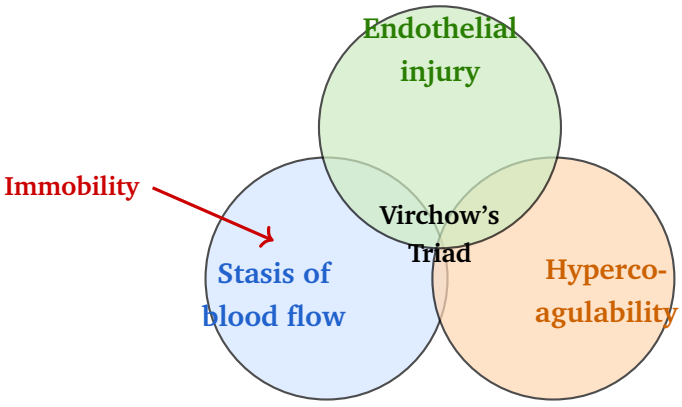
(A) Histamine

(B) Bradykinin

(C) Serotonin

(D) Prostaglandin E2

Q9. A 72-year-old woman undergoes elective hip replacement surgery under general anaesthesia lasting 4 hours. On the third post-operative day she develops calf pain and swelling. Doppler ultrasound confirms deep vein thrombosis. According to Virchow's triad, which of the three components was most directly promoted by prolonged immobility during surgery?



(A) Endothelial injury

(B) Hypercoagulability

(C) Stasis of blood flow

(D) Increased fibrinolysis

Q10. A 35-year-old woman presents with sudden-onset right-sided weakness and facial droop. MRI brain confirms acute ischaemic stroke. She has no atrial fibrillation, carotid stenosis, or cardiac thrombus. Transoesophageal echocardiography with bubble contrast study shows right-to-left shunting of microbubbles during a Valsalva manoeuvre. She was previously found to have a lower extremity deep vein thrombosis. What is the most likely mechanism of her stroke?

(A) Patent foramen ovale allowing venous thrombus to enter the arterial circulation (paradoxical embolism)

(B) Cardioembolism from atrial fibrillation

(C) Large-vessel atherosclerosis of the carotid artery

(D) Air embolism from a central venous catheter

Q11. A 65-year-old man is admitted with massive anterior myocardial infarction. He develops hypotension (BP 80/50 mmHg), tachycardia, cold clammy extremities, oliguria, and confusion. Central venous pressure is markedly elevated at 20 mmHg. Pulmonary artery catheterisation shows high pulmonary wedge pressure and low cardiac output. Which type of shock best explains this presentation?

(A) Hypovolaemic shock

(B) Distributive (septic) shock

(C) Obstructive shock

(D) Cardiogenic shock

Q12. A 58-year-old postmenopausal woman presents with bloating, early satiety, and a pelvic mass on CT scan. Serum tumour markers are ordered. Which marker is most specifically elevated in epithelial ovarian carcinoma (especially serous type) and is used for monitoring treatment response and detecting recurrence?

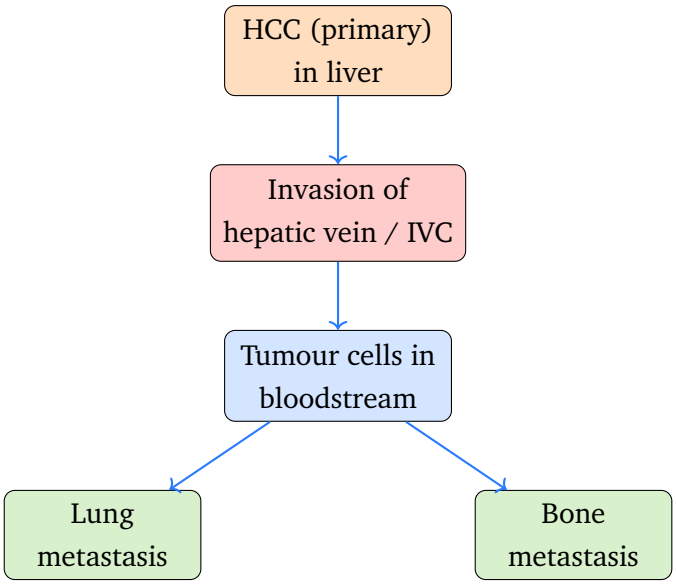
(A) AFP (alpha-fetoprotein)

(B) CA-125

(C) PSA (prostate-specific antigen)

(D) CEA (carcinoembryonic antigen)

Q13. A 55-year-old man with chronic hepatitis B-related cirrhosis is found to have a 6 cm hepatocellular carcinoma (HCC) on imaging. He subsequently develops pulmonary nodules and bone pain in the lumbar spine. The most common route by which HCC spreads to distant organs is illustrated below:



(A) Lymphatic spread

(B) Direct (contiguous) extension

(C) Hematogenous route

(D) Transcoelomic spread

Q14. A 38-year-old woman presents with episodic severe headaches, profuse sweating, and palpitations, each lasting 15–20 minutes. Between episodes her blood pressure is normal; during episodes it rises to 220/130 mmHg. 24-hour urine collection reveals markedly elevated vanillylmandelic acid (VMA) and metanephrines. CT abdomen shows a 4 cm right adrenal mass. The most likely diagnosis is:

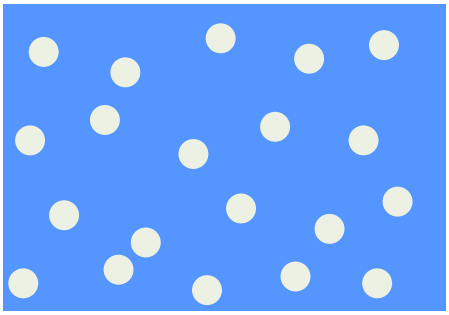
(A) Pheochromocytoma (catecholamine-secreting adrenal medullary tumour)

(B) Primary hyperaldosteronism (Conn syndrome)

(C) Cushing's syndrome (cortisol-secreting adrenal adenoma)

(D) Neuroblastoma

Q15. A 10-year-old African boy presents with a rapidly growing jaw mass. Biopsy shows sheets of uniform medium-sized lymphoma cells (B-cells) with a high mitotic rate and numerous tingible-body macrophages scattered among the tumour cells. Cytogenetics reveals t(8;14)(q24;q32). Which molecular event is the direct consequence of this translocation?



(A) BCL-2 overexpression preventing apoptosis

(B) BCR-ABL fusion tyrosine kinase constitutive activation

(C) Cyclin D1 overexpression

(D) c-MYC oncogene activation (translocated to immunoglobulin heavy chain locus)

Q16. A 34-year-old woman with a history of multiple sexual partners and HPV 16 infection presents for a routine Pap smear. Colposcopy-directed biopsy shows full-thickness dysplastic changes throughout the entire thickness of the squamous epithelium, with numerous mitoses including abnormal mitoses. The basement membrane is intact. This lesion is best classified as:

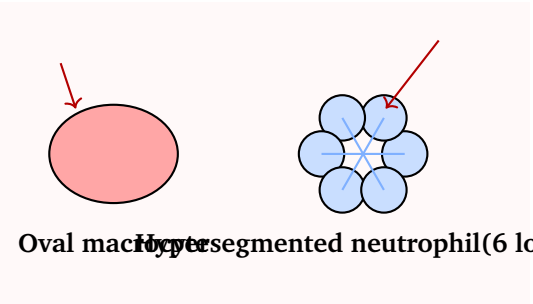
(A) CIN I (mild dysplasia)

(B) Carcinoma in situ of the cervix (CIN III / HSIL)

(C) Invasive squamous cell carcinoma

(D) Adenocarcinoma in situ

Q17. A 52-year-old woman with a history of total gastrectomy presents with progressive fatigue, glossitis, and mild jaundice. CBC shows haemoglobin 7.2 g/dL and MCV 118 fL. Peripheral blood smear findings are illustrated below:



(A) Microcytic hypochromic anaemia (iron deficiency)

(B) Normocytic normochromic anaemia

(C) Macrocytic anaemia with hypersegmented neutrophils (megaloblastic – vitamin B12 deficiency)

(D) Haemolytic anaemia with spherocytes

Q18. A 5-year-old boy presents with pallor, bone pain, and cervical lymphadenopathy. CBC shows WBC 65,000/μL with 80% blasts, haemoglobin 6.8 g/dL, and platelets 35,000/μL. Bone marrow biopsy shows replacement by lymphoblasts that are TdT-positive and express CD10, CD19, and CD20. Which diagnosis is most consistent?

(A) Acute lymphoblastic leukemia (ALL) – most common childhood malignancy

(B) Acute myeloid leukemia (AML)

(C) Chronic myeloid leukemia (CML)

(D) Burkitt lymphoma

Q19. A 28-year-old pregnant woman (38 weeks gestation) presents with abruptio placentae and is in haemorrhagic shock. Laboratory data: PT prolonged, PTT prolonged, platelets 28,000/μL, fibrinogen 60 mg/dL (low),

D-dimers markedly elevated, peripheral smear shows schistocytes. The most likely underlying haematological disorder is:

(A) Haemophilia A (Factor VIII deficiency)

(B) Immune thrombocytopenic purpura (ITP)

(C) Thrombotic thrombocytopenic purpura (TTP)

(D) Disseminated intravascular coagulation (DIC)

Q20. A 22-year-old woman presents with menorrhagia and prolonged bleeding after dental extraction. Her mother has a similar history. Laboratory findings: bleeding time prolonged, platelet count 240,000/μL (normal), PT normal, PTT mildly prolonged. Platelet aggregation with ristocetin is absent. Factor VIII activity is mildly reduced. The most likely diagnosis is:

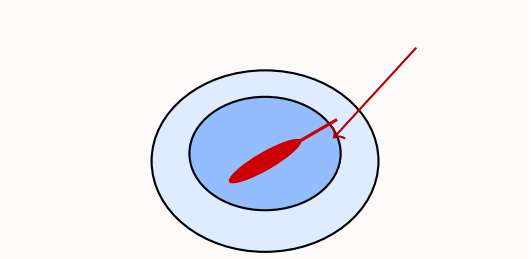
(A) Haemophilia A

(B) Von Willebrand disease (prolonged bleeding time with normal platelet count)

(C) Glanzmann's thrombasthenia

(D) Bernard-Soulier syndrome

Q21. A 55-year-old man presents with gum hypertrophy, fever, and petechiae. CBC shows WBC 80,000/μL with 75% blasts; platelets 20,000/μL. Myeloperoxidase stain is strongly positive on the blasts. Bone marrow aspiration findings are illustrated below:



Auer rod (elongated pink crystalline inclusion)
Myeloid blast, MPO+

(A) Acute lymphoblastic leukemia (ALL)

(B) Chronic myeloid leukemia (CML)

(C) Acute myeloid leukemia (AML) – Auer rods are pathognomonic

(D) Multiple myeloma

Q22. A 2-year-old child of Mediterranean descent presents with severe anaemia (Hb 4.5 g/dL), marked hepatosplenomegaly, frontal bossing, and maxillary overgrowth. Skull X-ray shows a "hair-on-end" pattern. Haemoglobin electrophoresis reveals HbF 90%, HbA2 5%, and absent HbA. Both parents have mild microcytic anaemia with elevated HbA2. The most likely diagnosis is:

(A) Beta-thalassemia major (Cooley anaemia) – HbF predominance, absent HbA, bone marrow expansion

(B) Sickle cell anaemia

(C) Alpha-thalassemia major (haemoglobin Bart's)

(D) Iron deficiency anaemia

Q23. A 6-year-old child develops primary pulmonary tuberculosis. Chest X-ray shows a small peripheral parenchymal opacity in the right lower lobe along with ipsilateral hilar lymphadenopathy. Together, these findings constitute which pathological entity?

(A) Miliary tuberculosis

(B) Simon foci (apical reactivation foci)

(C) Secondary (post-primary) tuberculosis

(D) Ghon complex (Ghon focus + regional hilar lymphadenopathy)

Q24. A 78-year-old bed-ridden man with stroke-related dysphagia develops fever, productive cough, and bilateral lower-lobe crackles. Chest X-ray shows patchy bilateral consolidation predominantly in the dependent zones. Post-mortem examination would most likely reveal:

(A) Lobar pneumonia with complete lobar consolidation

(B) Bronchopneumonia – patchy peribronchiolar consolidation in multiple lobules

(C) Interstitial pneumonia with diffuse alveolar damage

(D) Lung abscess with central liquefaction

Q25. A 62-year-old male heavy smoker (50 pack-years) presents with haemoptysis and a hilar mass on chest X-ray. CT shows a central cavitating mass in the right upper lobe. Bronchoscopy biopsy reveals malignant cells with keratin pearls and intercellular bridges. Serum calcium is elevated. The most likely diagnosis and its characteristic histological feature is:

(A) Small cell carcinoma – neuroendocrine granules, paraneoplastic SIADH

(B) Adenocarcinoma – mucin production, peripheral location, lepidic growth

(C) Squamous cell carcinoma – central hilar location, cavitation, keratin pearls

(D) Large cell carcinoma – undifferentiated peripheral mass, no keratin pearls

Q26. A 55-year-old sandstone quarry worker with 30 years of occupational exposure presents with progressive exertional dyspnoea and bilateral upper-zone nodular opacities on chest X-ray. Pulmonary function tests show a restrictive pattern. He is found to have latent tuberculosis on Mantoux testing. The inorganic dust responsible for this pneumoconiosis and its association with tuberculosis is:

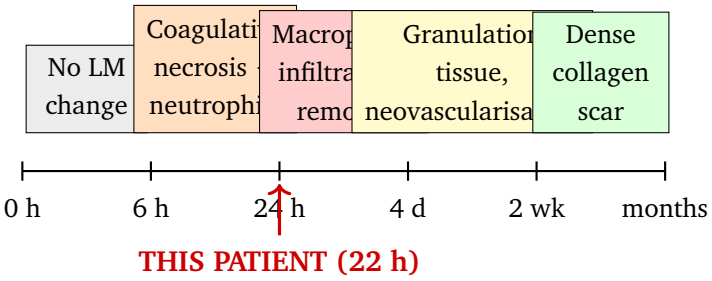
(A) Silicosis (silicon dioxide inhalation) – predisposes to tuberculosis (silicotuberculosis); birefringent particles surrounded by collagenous nodules

(B) Asbestosis (asbestos fibre inhalation) – associated with mesothelioma, not TB

(C) Anthracosis (coal dust) – simple coal worker's pneumoconiosis, Caplan syndrome

(D) Berylliosis (beryllium exposure) – non-caseating granulomas mimicking sarcoidosis

Q27. A 58-year-old man dies 22 hours after the onset of acute chest pain. At autopsy, the pathologist examines the myocardium of the left ventricle. Based on the timeline of myocardial infarction, the histological changes expected at this stage are illustrated below:



(A) No light microscopic changes (only EM-detectable changes)

(B) Macrophage infiltration and removal of necrotic debris

(C) Dense collagenous scar

(D) Coagulative necrosis of myocytes with early neutrophil infiltration

Q28. A pathologist examining a coronary artery plaque identifies lipid-laden foam cells within the intima. These cells play a central role in the early formation of atherosclerotic plaques (fatty streaks). The foam cells are derived from which circulating cell type?

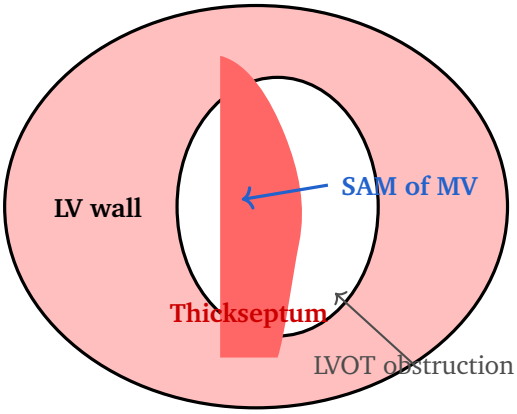
(A) Smooth muscle cells that have migrated to the intima

(B) Monocytes (circulating) that differentiate into macrophages and ingest oxidised LDL

(C) Lymphocytes that accumulate lipid

(D) Mast cells in the adventitia

Q29. A 22-year-old athlete collapses and dies during a basketball game. Autopsy reveals a heart weighing 620 g (normal: ∼350 g). Cross-section is illustrated below:



(A) Dilated cardiomyopathy

(B) Restrictive cardiomyopathy

(C) Hypertrophic cardiomyopathy (asymmetric septal hypertrophy, SAM, LVOT obstruction)

(D) Cardiac amyloidosis

Q30. A 55-year-old obese man with a 15-year history of heartburn and regurgitation undergoes upper endoscopy. Salmon-coloured mucosa is seen extending 4 cm above the gastro-oesophageal junction. Biopsy shows replacement of the normal stratified squamous epithelium by columnar epithelium with goblet cells (intestinal metaplasia). What is this condition and its most important clinical complication?

(A) Barrett's oesophagus – intestinal metaplasia predisposing to oesophageal adenocarcinoma

(B) Squamous cell carcinoma arising from normal squamous mucosa

(C) Achalasia – failure of lower oesophageal sphincter relaxation

(D) Mallory-Weiss tear – submucosal laceration from vomiting

Q31. A 28-year-old woman presents with bloody diarrhoea, urgency, and cramping for 4 months. Colonoscopy shows continuous mucosal involvement from the rectum to the splenic flexure with friable, erythematous mucosa and absence of skip lesions. Rectal biopsy shows crypt abscesses. The histological and anatomical features that most distinguish ulcerative colitis from Crohn's disease are:

(A) Transmural inflammation with granulomas and skip lesions

(B) Cobblestone appearance with fissuring ulcers penetrating deeply

(C) Perianal disease with fistulae

(D) Inflammation limited to the mucosa and submucosa; continuous; no granulomas; crypt abscesses

Q32. A 48-year-old man with 20-year history of heavy alcohol consumption is admitted with right upper quadrant pain, jaundice, and fever. AST/ALT ratio is $>$ 2:1. Liver biopsy shows hepatocyte ballooning degeneration, neutrophilic infiltration, and eosinophilic intracytoplasmic inclusions within ballooned hepatocytes. These inclusions are composed of aggregated intermediate filament proteins (cytokeratin 8 and 18). They are called:

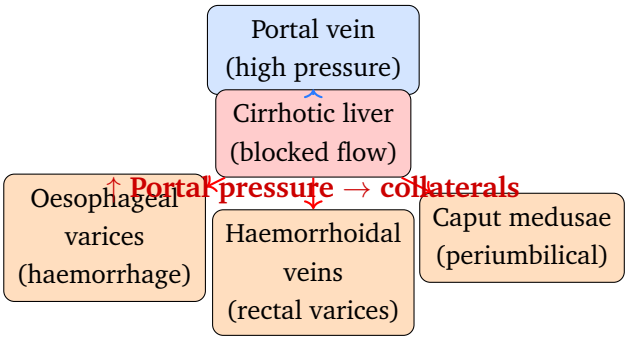
(A) Councilman bodies (acidophilic bodies of apoptotic hepatocytes)

(B) Mallory-Denk bodies (cytokeratin clumps – pathognomonic of alcoholic hepatitis)

(C) Dutcher bodies (intranuclear immunoglobulin inclusions)

(D) Psammoma bodies (concentric calcification)

Q33. A 52-year-old man with liver cirrhosis (Child-Pugh class C) presents with massive haematemesis. Endoscopy reveals large tortuous varices at the lower oesophagus that are actively bleeding. The development of these oesophageal varices is a direct consequence of which pathophysiological process? The portosystemic collateral pathways are illustrated below:



(A) Hypoalbuminaemia causing decreased oncotic pressure

(B) Hepatic artery thrombosis

(C) Portal hypertension causing portosystemic shunting to oesophageal venous plexus

(D) Superior vena cava obstruction

Q34. An 18-year-old man develops painless macroscopic haematuria 24 hours after an upper respiratory tract infection. He had a similar episode 6 months ago. Urinalysis shows 3+ blood, no casts. Serum complement (C3, C4) is normal. Renal biopsy immunofluorescence shows mesangial IgA deposits. The most likely diagnosis is:

(A) IgA nephropathy (Berger disease) – mesangial IgA deposits; haematuria 1–3 days after URTI

(B) Post-streptococcal glomerulonephritis – subepithelial humps, decreased C3, latent period 2–3 weeks

(C) Thin basement membrane disease

(D) Alport syndrome (hereditary nephritis with sensorineural deafness)

Q35. A 58-year-old man presents with the classic triad of painless haematuria, left flank pain, and a palpable left flank mass. CT abdomen shows a heterogeneous 9 cm mass in the left kidney with areas of necrosis. He is also noted to have polycythaemia and hypercalcaemia. Histopathology shows tumour cells with abundant clear cytoplasm (high glycogen/lipid content) arranged in nests. The most common genetic alteration in this tumour is:

(A) BRCA1 mutation

(B) MET proto-oncogene mutation

(C) TP53 loss of heterozygosity

(D) Loss-of-function mutation/hypermethylation of the VHL tumour suppressor gene (clear cell renal cell carcinoma)

Q36. A 40-year-old man presents with nephrotic syndrome (4+ proteinuria, oedema, hypoalbuminaemia). Renal biopsy light microscopy shows thick-

ened glomerular capillary walls with a “spike and dome” pattern on silver staining. Immunofluorescence shows granular IgG and C3 along the capillary loops. Electron microscopy reveals subepithelial electron-dense deposits with intervening basement membrane projections (spikes). The characteristic finding on EM is:

(A) Subendothelial deposits (lupus nephritis class III/IV)

(B) Subepithelial immune complex deposits with spike-and-dome pattern (membranous nephropathy)

(C) Mesangial deposits only (IgA nephropathy)

(D) No immune deposits (minimal change disease)

Q37. A 32-year-old woman presents with heat intolerance, weight loss despite increased appetite, tremor, and palpitations. On examination she has a diffuse smooth goitre with a bruit, bilateral exophthalmos, and pretibial myxoedema. TSH is undetectable; free T4 is markedly elevated. The pathogenesis of this condition is mediated by:

(A) Anti-thyroglobulin antibodies causing thyroid destruction

(B) Anti-TPO antibodies causing hypothyroidism

(C) TSH-receptor stimulating autoantibodies (thyroid-stimulating immunoglobulin – TSI) causing continuous thyroid stimulation

(D) Activating mutation in the TSH receptor gene

Q38. A 35-year-old woman presents with progressive fatigue, weight loss, postural hypotension, and hyponatraemia. She has generalised hyperpigmentation including buccal mucosa, skin creases, and recent scars. Laboratory findings: serum sodium 128 mEq/L, potassium 5.8 mEq/L, fasting glucose 62 mg/dL. Morning cortisol is very low and fails to rise after ACTH stimulation test (Synacthen test). The diagnosis is:

(A) Primary adrenocortical insufficiency (Addison disease) – destruction of adrenal cortex, elevated ACTH causes hyperpigmentation

(B) Secondary adrenocortical insufficiency (pituitary ACTH deficiency) – no hyperpigmentation

(C) Phaeochromocytoma – episodic hypertension, not hypotension

(D) Cushing's syndrome – hypertension, hyperglycaemia, not hypoglycaemia

Q39. A 70-year-old farmer with extensive sun exposure presents with a pearly, translucent nodule with a rolled border and central telangiectasia on the nose. Biopsy shows islands of basaloid cells with peripheral palisading of nuclei and retraction artefact between the tumour nests and stroma. The tumour rarely metastasises but is locally invasive. The characteristic histological feature of this tumour is:

(A) Keratin pearls and intercellular bridges

(B) Pagetoid spread through the epidermis

(C) Reed-Sternberg cells

(D) Peripheral nuclear palisading of basal cells with stromal retraction artefact (basal cell carcinoma)

Q40. A 28-year-old woman presents with a ring-shaped, flesh-coloured papular rash on the dorsum of her hand. She has no joint pain, and her fasting blood glucose is 85 mg/dL (normal). Skin biopsy shows palisading histiocytes surrounding a central zone of degenerated collagen (necrobiosis) with mucin deposition. There is no evidence of caseating necrosis. The most likely diagnosis is:

(A) Necrobiosis lipoidica diabeticorum (associated with diabetes mellitus)

(B) Granuloma annulare (central necrobiosis + palisading histiocytes; normal blood glucose)

(C) Rheumatoid nodule (subcutaneous, associated with rheumatoid arthritis)

(D) Sarcoidosis (non-caseating granulomas, elevated ACE)

Detailed Solutions

Q1.

Solution

Concept – Liquefactive necrosis: Liquefactive necrosis occurs when enzymatic digestion of dead cells completely dissolves the tissue, producing a liquid-filled cavity. The brain is uniquely susceptible because of its high lipid content and relatively poor structural proteins – ischaemic infarcts of the brain result in liquefactive (not coagulative) necrosis. Bacterial infections (abscesses) also cause liquefactive necrosis via proteolytic enzymes released by neutrophils.

Key Point: All organs except the brain show coagulative necrosis in ischaemia. Brain ischaemia always produces liquefactive necrosis – the infarcted region liquefies into a cystic cavity over weeks.

Why other options are wrong:

- Option A (Coagulative necrosis): Seen in ischaemia of heart, kidney, spleen – NOT brain.
- Option B (Caseous necrosis): Characteristic of tuberculosis – cheese-like amorphous necrosis with granulomas.
- Option D (Fat necrosis): Occurs in pancreatic enzymatic injury or saponification of breast fat.

Final Answer: Brain infarction $\Rightarrow$ liquefactive necrosis (cystic cavity). $\Rightarrow \boxed{C}$

Answer: (C) **Go Back to Q1**

Q2.

Solution

Concept – Reversible cell injury (hydropic/cellular swelling): Brief ischaemia (15 minutes) causes reversible cell injury. The ATP depletion leads to failure of the Na^+/K^+-ATPase pump, causing intracellular Na^+ and water influx – **cellular swelling (hydropic change)**. On electron microscopy, reversible changes include: ER dilation ("vacuolation"), loss of microvilli, myelin figures (phospholipid whorls), swollen mitochondria with normal cristae, and clumping of chromatin. The cell membrane remains intact and nuclei are not pyknotic.

Key Point: The key distinction between reversible and irreversible injury is: (1) **membrane integrity** (intact in reversible) and (2) **mitochondrial changes** (swelling reversible; amorphous densities / flocculent densities in matrix = irre-

versible).

Why other options are wrong:

- Option B (Irreversible necrosis): Would show membrane rupture, karyolysis/pyknosis/karyorrhexis.
- Option C (Apoptosis): Cell shrinkage, chromatin condensation, apoptotic bodies – not swelling.
- Option D (Normal): The described EM changes (ER dilation, loss of microvilli) are abnormal.

Final Answer: Cellular swelling with intact membrane = reversible cell injury. $\Rightarrow$ $\boxed{A}$

Answer: (A) **Go Back to Q2**

Q3.

Solution

Concept – Metastatic calcification: Metastatic calcification occurs in the setting of **hypercalcaemia** (elevated serum calcium) and deposits in **normal, undamaged tissues**. Causes include hyperparathyroidism, hypervitaminosis D, malignancy with bone destruction, sarcoidosis, and milk-alkali syndrome. Preferential sites: **lungs, kidneys, gastric mucosa, and blood vessel walls** (all sites that lose acid rapidly and become alkaline, favouring calcium salt precipitation).

Key Point: Dystrophic calcification occurs in previously damaged/necrotic tissue despite *normal* serum calcium (e.g., in atherosclerotic plaques, caseous TB, dead parasites). **Metastatic calcification** occurs in normal tissue due to *elevated* serum calcium.

Why other options are wrong:

- Option A (Dystrophic): Serum calcium is normal in dystrophic calcification; occurs in damaged tissue.
- Option B (Psammoma bodies): Laminated concentric calcification in meningioma, papillary thyroid Ca, serous ovarian Ca – not related to hypercalcaemia.
- Option C (Ossification): True bone formation; not seen in this context.

Final Answer: Hypercalcaemia + normal tissue calcification = metastatic calcification. $\Rightarrow$ $\boxed{D}$

Answer: (D) **Go Back to Q3**

Q4.

Solution

Concept – Composition of pus (suppurative inflammation): Pus consists of **neutrophils** (polymorphonuclear leukocytes – alive and dead), necrotic tissue debris, dead bacteria, and fluid exudate. Neutrophils are the principal effector cells of acute inflammation and are the dominant cells in any purulent exudate. They arrive first at the site of bacterial infection via chemotaxis (C5a, IL-8/CXCL8, LTB4, bacterial formyl peptides).

Key Point: **Neutrophils** are the hallmark of **acute inflammation**. Pus = neutrophils + dead bacteria + necrotic tissue. Abscess = loculated collection of pus within dead tissue.

Why other options are wrong:

- Option A (Lymphocytes): Predominant in chronic inflammation, viral infections, and granulomatous disease.
- Option C (Eosinophils): Predominant in parasitic infections and allergic reactions (IgE-mediated).
- Option D (Macrophages): Arrive later than neutrophils; principal cells of chronic inflammation.

Final Answer: Pus is predominantly neutrophils. $\Rightarrow \boxed{B}$

Answer: (B) **Go Back to Q4**

Q5.

Solution

Concept – Serous inflammation: Serous inflammation is characterised by the production of a watery, protein-poor (transudate-like) exudate derived from plasma or mesothelial cell secretions. It contains **few cells** (sparse lymphocytes and mesothelial cells) and minimal fibrin. It is the mildest form of inflammation. Examples: viral pleuritis, early rheumatoid pleuritis, skin blisters (2nd degree burns, herpes simplex). The straw-coloured pleural fluid with protein < 3 g/dL and few cells is classic for serous inflammation.

Key Point: Distinguish from **fibrinous pleuritis** (fibrin strands visible; "bread and butter" pericarditis); **suppurative pleuritis** (empyema – pus, neutrophils); **haemorrhagic** (red blood cells, seen in TB, malignancy, pulmonary infarction).

Why other options are wrong:

- Option A (Fibrinous): Thick fluid, fibrin threads, rub heard on auscultation.
- Option B (Suppurative/purulent): Pus – neutrophils dominant, thick green/yellow fluid.
- Option D (Haemorrhagic): Bloody fluid, seen in anthrax, rickettsial infections, malignancy.

Final Answer: Clear straw-coloured fluid with few cells = serous inflammation. $\Rightarrow \boxed{C}$

Answer: (C) **Go Back to Q5**

Q6.

Solution

Concept – Foreign body granuloma: When macrophages cannot digest a persistent indigestible material (suture, silica, talc, breast implant material), they fuse to form **foreign body giant cells** – large cells with many nuclei haphazardly arranged (not in horseshoe pattern). This is a **non-immune granuloma** (no sensitised T-lymphocytes required). The granuloma surrounds but cannot destroy the material.

Key Point: Foreign body giant cells: nuclei scattered randomly throughout cytoplasm; no caseation. **Langhans giant cells** (TB, sarcoidosis): nuclei arranged in a horseshoe at the periphery of the cell; require T-cell sensitisation. **Touton giant cells**: wreath of nuclei around central lipid-laden zone (xanthomas).

Why other options are wrong:

- Option B (Langhans): Seen in tuberculosis and sarcoidosis (immune granulomas) – nuclei at periphery in horseshoe arrangement.
- Option C (Reed-Sternberg): Owl-eye binucleate giant cells in Hodgkin lymphoma – not in granulomas.
- Option D (Touton): Found in xanthomas (lipid disorders) with central lipid and nuclear wreath.

Final Answer: Suture material $\rightarrow$ foreign body giant cell (fused macrophages, random nuclei). $\Rightarrow \boxed{A}$

Answer: (A) **Go Back to Q6**

Q7.

Solution

Concept – Healing by secondary intention: Secondary intention healing applies when there is **extensive tissue loss** and the wound edges cannot be approximated. It involves: (1) larger amounts of granulation tissue formation, (2) **wound contraction** mediated by **myofibroblasts** (express α-smooth muscle actin), and (3) re-epithelialisation from wound margins over a large surface. The result is a **larger scar** compared with primary intention (where clean incised wound edges are sutured together).

Key Point: Myofibroblasts are the key cells of wound contraction in secondary intention healing. Excessive myofibroblast activity leads to **contractures**. Keloid/hypertrophic scarring is excess collagen deposition beyond wound margins.

Why other options are wrong:

- Option A: Secondary intention requires *more* granulation tissue, not minimal.
- Option B: Wound contraction by myofibroblasts is a *defining* feature of secondary intention.
- Option C: Scarring always occurs in secondary intention (in fact larger scar than primary intention).

Final Answer: Large tissue-loss wound $\rightarrow$ secondary intention: more granulation tissue, contraction, larger scar. $\Rightarrow \boxed{D}$

Answer: (D) **Go Back to Q7**

Q8.

Solution

Concept – Bradykinin as a mediator of vascular permeability and pain: Bradykinin is generated from kininogen by **kallikrein** which is activated by Hageman factor (Factor XII, contact activation system). Bradykinin causes: (1) vasodilation (via NO and PGI2), (2) **increased vascular permeability**, (3) **pain** (acts on B1/B2 receptors on nociceptive fibres), and (4) smooth muscle contraction. Unlike histamine (which acts immediately – within seconds to minutes), bradykinin acts over 15–30 minutes. It is inactivated by ACE (angiotensin-converting enzyme) – hence ACE inhibitors cause bradykinin accumulation and a dry cough.

Key Point: Histamine = immediate (preformed, released from mast cells);

Bradykinin = delayed (generated by contact system). Both increase vascular permeability. Only bradykinin causes pain directly.

Why other options are wrong:

- Option A (Histamine): Preformed mediator – acts within seconds to minutes; not generated by Hageman factor.
- Option C (Serotonin): Released from platelets and enterochromaffin cells; minor role in humans.
- Option D (PGE2): Sensitises nociceptors (hyperalgesia) but is derived from arachidonic acid, not Hageman factor pathway.

Final Answer: Hageman factor $\rightarrow$ kallikrein $\rightarrow$ bradykinin (permeability + pain). $\Rightarrow \boxed{B}$

Answer: (B) **Go Back to Q8**

Q9.

Solution

Concept – Virchow's triad – stasis: Virchow's triad describes the three factors predisposing to thrombosis: (1) **Endothelial injury** (e.g., atherosclerosis, trauma), (2) **Stasis of blood flow** (e.g., immobility, atrial fibrillation, varicosities), (3) **Hypercoagulability** (e.g., factor V Leiden, antiphospholipid syndrome, malignancy). Prolonged immobility during surgery dramatically reduces venous return from the lower limbs, causing **venous stasis** – blood pools in the deep veins of the calf, promoting thrombus formation.

Key Point: Venous thrombi (red thrombi) form under **low-flow** conditions (stasis) and are fibrin and RBC-rich. Arterial thrombi (white thrombi) form at sites of **endothelial injury** (high shear stress) and are platelet-rich.

Why other options are wrong:

- Option A (Endothelial injury): Not directly caused by immobility alone; relevant in arterial disease.
- Option B (Hypercoagulability): May coexist but is not the primary consequence of immobility.
- Option D (Increased fibrinolysis): Would protect against thrombosis, not promote it.

Final Answer: Immobility $\rightarrow$ venous stasis $\rightarrow$ deep vein thrombosis. $\Rightarrow \boxed{C}$

Answer: (C) **Go Back to Q9**

Q10.

Solution

Concept – Paradoxical embolism via patent foramen ovale (PFO): Normally, the foramen ovale closes after birth due to higher left atrial pressure. In ∼25% of adults, a **patent foramen ovale (PFO)** remains. During straining (Valsalva manoeuvre), right atrial pressure transiently exceeds left, allowing a venous thrombus originating in the deep veins to pass directly into the left atrium and then to the systemic arterial circulation – **paradoxical embolism**. This is an important and underdiagnosed cause of cryptogenic stroke in young adults.

Key Point: Bubble echocardiography (agitated saline) with Valsalva is the investigation of choice for PFO. Bubbles normally disappear in the right heart; if they appear in the left heart, a right-to-left shunt is confirmed.

Why other options are wrong:

- Option B (AF embolism): No atrial fibrillation documented in this young patient.
- Option C (Carotid atherosclerosis): Rare at age 35 without major risk factors; carotid imaging was normal.
- Option D (Air embolism): No central line or iatrogenic procedure described.

Final Answer: DVT $\rightarrow$ PFO $\rightarrow$ arterial embolism $\rightarrow$ stroke (paradoxical embolism). $\Rightarrow \boxed{A}$

Answer: (A) **Go Back to Q10**

Q11.

Solution

Concept – Cardiogenic shock: Cardiogenic shock results from **pump failure** (most commonly massive MI, large myocardial loss $>$ 40% of LV). Haemodynamic profile: **low cardiac output (CO)**, **elevated central venous pressure (CVP)**, elevated pulmonary capillary wedge pressure (PCWP), and high systemic vascular resistance (SVR) as the body compensates. Clinical: cold/clammy peripheries, oliguria, altered consciousness, pulmonary oedema.

Key Point: The key distinguishing feature of cardiogenic shock is **elevated CVP and PCWP** (distinguishes from hypovolaemic shock where CVP is low). Mortality remains very high ($>$ 50%) despite modern intervention.

Why other options are wrong:

- Option A (Hypovolaemic): Low CVP/PCWP; responds to fluids; no pump failure.
- Option B (Distributive/septic): *Low* SVR, warm peripheries ("warm shock"), normal or high CO early.
- Option C (Obstructive): Caused by PE or cardiac tamponade – different pathophysiology though CVP may also be elevated.

Final Answer: Massive MI $\rightarrow$ pump failure $\rightarrow$ cardiogenic shock (high CVP, low CO). $\Rightarrow \boxed{D}$

Answer: (D) **Go Back to Q11**

Q12.

Solution

Concept – CA-125 and epithelial ovarian carcinoma: CA-125 (cancer antigen 125) is a mucin-like glycoprotein expressed on the surface of epithelial ovarian tumours, particularly the **serous type**. It is elevated in $\sim$80% of advanced epithelial ovarian cancers. CA-125 is used for: (1) monitoring treatment response after chemotherapy, (2) detecting recurrence, and (3) as part of the risk malignancy index. It is not specific enough for screening (elevated in endometriosis, uterine fibroids, pregnancy, peritonitis).

Key Point: AFP = yolk sac tumour and HCC. **PSA** = prostate carcinoma. **CEA** = colorectal, gastric, lung carcinoma. **CA-125** = epithelial ovarian carcinoma. β**-hCG** = choriocarcinoma, gestational trophoblastic disease.

Why other options are wrong:

- Option A (AFP): Elevated in yolk sac (endodermal sinus) tumours and HCC, not epithelial ovarian Ca.
- Option C (PSA): Prostate-specific; not relevant to ovarian carcinoma.
- Option D (CEA): Elevated in colon, gastric, pancreatic, and lung carcinomas; less specific for ovary.

Final Answer: CA-125 is the tumour marker for epithelial ovarian carcinoma. $\Rightarrow \boxed{B}$

Answer: (B) **Go Back to Q12**

Q13.

Solution

Concept – Hematogenous metastasis of HCC: Hepatocellular carcinoma (HCC) has a propensity for **vascular invasion** – it commonly grows into the hepatic veins and inferior vena cava, giving it direct access to the pulmonary circulation. The most common route of distant metastasis is **haematogenous**, leading to pulmonary metastases (most common), bone metastases, and adrenal metastases. HCC rarely metastasises via lymphatics, and transcoelomic spread is uncommon.

Key Point: Tumours that commonly spread haematogenously: HCC, renal cell carcinoma, follicular thyroid carcinoma, choriocarcinoma, osteosarcoma. Sarcomas also spread haematogenously ("via the blood") rather than lymphatically.

Why other options are wrong:

- Option A (Lymphatic): Common route for carcinomas of the breast, stomach, colon – not the primary route for HCC.
- Option B (Direct extension): HCC invades adjacent liver and diaphragm, but distant metastases require vascular access.
- Option D (Transcoelomic): Classic for ovarian carcinoma, GI carcinomas seeding the peritoneum.

Final Answer: HCC spreads primarily via haematogenous route (hepatic vein invasion). $\Rightarrow \boxed{C}$

Answer: (C) **Go Back to Q13**

Q14.

Solution

Concept – Pheochromocytoma: Pheochromocytoma is a **catecholamine-secreting tumour** arising from the chromaffin cells of the **adrenal medulla** (or sympathetic ganglia – paraganglioma). It causes episodic (paroxysmal) hypertension with the "5 Ps": **Pressure** (hypertension), **Pain** (headache), **Perspiration**, **Palpitations**, **Pallor**. Diagnosis: elevated **urine/plasma metanephrines and VMA**. The tumour follows the "rule of 10s": 10% bilateral, 10% malignant, 10% extra-adrenal, 10% familial (MEN 2A/2B, VHL, NF1).

Key Point: VMA (vanillylmandelic acid) and **metanephrines** are the metabolic breakdown products of adrenaline and noradrenaline – elevated in pheochromocytoma. Surgical resection is curative; pre-operative alpha-blockade (phenoxybenzamine) is mandatory.

Why other options are wrong:

- Option B (Conn syndrome): Hyperaldosteronism causes *sustained* hypertension, hypokalaemia, no episodic features, no elevated catecholamines.
- Option C (Cushing's): Cortisol excess causes central obesity, striae, moon face, buffalo hump – not episodic hypertensive crises.
- Option D (Neuroblastoma): Occurs in young children ($<$ 5 years), arises in adrenal medulla/sympathetic chain, rarely episodic hypertension.

Final Answer: Episodic hypertension + elevated VMA/metanephrines + adrenal mass = pheochromocytoma. $\Rightarrow \boxed{A}$

Answer: (A) **Go Back to Q14**

Q15.

Solution

Concept – Burkitt lymphoma and c-MYC: The translocation **t(8;14)(q24;q32)** juxtaposes the **c-MYC proto-oncogene** (chromosome 8q24) next to the **immunoglobulin heavy chain (IgH) gene** (chromosome 14q32). IgH is constitutively active in B-cells, so c-MYC is overexpressed, driving uncontrolled cell proliferation. Burkitt lymphoma is the fastest growing human tumour (tumour doubling time $\sim$24 hours). The "starry sky" pattern = tingible-body macrophages (pale stars) engulfing apoptotic tumour cells among sheets of dark lymphoma cells (high proliferation AND apoptosis).

Key Point: Variant translocations: t(2;8) and t(8;22) – c-MYC with kappa/lambda light chain loci. **EBV** is associated with the endemic (African) form (jaw involvement). The sporadic form more commonly involves the ileo-caecal region.

Why other options are wrong:

- Option A (BCL-2): Overexpressed in follicular lymphoma via t(14;18) – prevents apoptosis.
- Option B (BCR-ABL): Philadelphia chromosome t(9;22) – CML and some ALL.
- Option C (Cyclin D1): Overexpressed in mantle cell lymphoma via t(11;14).

Final Answer: t(8;14) $\rightarrow$ c-MYC activation $\rightarrow$ Burkitt lymphoma. $\Rightarrow \boxed{D}$

Answer: (D) **Go Back to Q15**

Q16.

Solution

Concept – Carcinoma in situ of cervix (CIN III / HSIL): Carcinoma in situ (CIS) of the cervix is characterised by **full-thickness dysplastic changes** throughout the entire squamous epithelium. Mitoses (including abnormal/tripolar forms) are present throughout all layers. The **basement membrane is intact** – no stromal invasion. This is classified as **CIN III** or **high-grade squamous intraepithelial lesion (HSIL)** in the updated nomenclature. **HPV 16 and 18** (high-risk types) are responsible. The progression from normal $\rightarrow$ CIN I $\rightarrow$ CIN II $\rightarrow$ CIN III $\rightarrow$ invasive carcinoma takes years to decades.

Key Point: CIN I = lower third of epithelium involved. CIN II = lower 2/3 involved. CIN III = full thickness (including carcinoma in situ). **Basement membrane intact** = in situ (no invasion); basement membrane breached = invasive carcinoma.

Why other options are wrong:

- Option A (CIN I): Only lower 1/3 dysplasia; reflects transient HPV infection.
- Option C (Invasive carcinoma): Requires stromal invasion through the basement membrane.
- Option D (Adenocarcinoma in situ): Glandular epithelial dysplasia – different cell type from squamous.

Final Answer: Full-thickness atypia + intact BM + HPV 16 = carcinoma in situ (CIN III). $\Rightarrow \boxed{B}$

Answer: (B) **Go Back to Q16**

Q17.

Solution

Concept – Megaloblastic anaemia (B12 deficiency): Vitamin B12 is absorbed from the terminal ileum bound to **intrinsic factor (IF)** secreted by gastric parietal cells. Total gastrectomy removes all parietal cells, abolishing IF production, causing B12 deficiency over years (large hepatic stores). B12 is required for DNA synthesis (via folate metabolism); its deficiency causes **megaloblastic anaemia**: (1) **large oval macrocytes** (MCV $>$ 100 fL), (2) **hypersegmented neutrophils** (5+ lobes or $\geq$1 cell with 6+ lobes), and (3) pancytopenia in severe cases. Neurological features (subacute combined degeneration) distinguish B12 from folate deficiency.

Key Point: The single most characteristic peripheral smear finding of megaloblastic anaemia is the **hypersegmented neutrophil** (6 or more lobes in a single cell). Schilling test (historical) diagnoses pernicious anaemia vs dietary B12 deficiency.

Why other options are wrong:

- Option A (Microcytic hypochromic): Iron deficiency or thalassaemia – low MCV, not high.
- Option B (Normocytic): Aplastic anaemia, acute blood loss, early renal disease.
- Option D (Haemolytic with spherocytes): Hereditary spherocytosis, AIHA – spherocytes, raised reticulocytes.

Final Answer: Post-gastrectomy $\rightarrow$ B12 deficiency $\rightarrow$ macrocytic anaemia + hypersegmented neutrophils. $\Rightarrow \boxed{C}$

Answer: (C) **Go Back to Q17**

Q18.

Solution

Concept – Acute lymphoblastic leukemia (ALL): ALL is the **most common malignancy in children** (peak age 2–5 years) and is the most common acute leukaemia in children. It arises from lymphoid progenitor cells. **TdT (terminal deoxynucleotidyl transferase)** is the most important marker – positive in virtually all ALL cases. B-cell ALL markers: CD10 (CALLA – common ALL antigen), CD19, CD20, CD22. T-cell ALL: CD2, CD3, CD5, CD7. The favourable prognosis cytogenetic feature is hyperdiploidy or t(12;21)/ETV6-RUNX1 in B-ALL.

Key Point: ALL vs AML: **TdT+** = ALL; **TdT-** = AML. **Auer rods** = AML (pathognomonic). **Myeloperoxidase (MPO)+** = AML; **MPO-** = ALL. ALL has better prognosis in children (cure rate ~80–90%).

Why other options are wrong:

- Option B (AML): TdT-negative, MPO-positive, Auer rods may be present; more common in adults.
- Option C (CML): Chronic course, Philadelphia chromosome t(9;22); predominantly granulocytic differentiation.
- Option D (Burkitt lymphoma): B-cell lymphoma with t(8;14); presents as mass lesion, not primarily as leukaemia.

Final Answer: Child + lymphoblasts + TdT+ + CD10/19/20 = ALL. $\Rightarrow \boxed{A}$

Answer: (A) **Go Back to Q18**

Q19.

Solution

Concept – Disseminated intravascular coagulation (DIC): DIC is a **consumptive coagulopathy** characterised by simultaneous widespread microvascular fibrin thrombi (causing organ ischaemia) and consumption of clotting factors and platelets (causing haemorrhage). Triggers include obstetric complications (abruptio placentae, amniotic fluid embolism), sepsis, malignancy, and trauma. Lab: **prolonged PT and PTT** (factor consumption), **low fibrinogen**, **low platelets**, **elevated D-dimers** (fibrin degradation products), and **schistocytes** (microangiopathic haemolytic anaemia from RBCs shearing through fibrin strands).

Key Point: DIC is the only condition that simultaneously prolongs *both* PT and PTT with *low* fibrinogen and *elevated* D-dimers. **Abruptio placentae** releases thromboplastin (tissue factor) into the circulation, triggering DIC.

Why other options are wrong:

- Option A (Haemophilia A): Isolated PTT prolongation; normal PT, normal platelets, no D-dimers.
- Option B (ITP): Isolated thrombocytopenia; PT and PTT normal; no fibrinogen consumption.
- Option C (TTP): Thrombocytopenia + MAHA but normal PT/PTT; caused by ADAMTS13 deficiency.

Final Answer: Abruptio placentae $\rightarrow$ DIC (consumption of all clotting factors, thrombocytopenia, elevated D-dimers). $\Rightarrow \boxed{D}$

Answer: (D) **Go Back to Q19**

Q20.

Solution

Concept – Von Willebrand disease: Von Willebrand disease (VWD) is the **most common inherited bleeding disorder**. VWF is a multimeric protein that (1) mediates platelet adhesion to subendothelial collagen (via GPIb receptor) and (2) carries and protects Factor VIII in plasma. Deficiency or dysfunction of VWF causes: **prolonged bleeding time** (platelet plug defective – primary haemostasis failure), **normal platelet count**, and **mildly prolonged PTT** (Factor VIII mildly reduced). Ristocetin agglutination is absent/reduced. Mucocutaneous bleeding

(menorrhagia, epistaxis, easy bruising) is typical.

Key Point: Prolonged BT + normal platelet count = VWD (or platelet function defect). **Low platelet count** = ITP, TTP, DIC. **Prolonged PTT only** = Haemophilia A or B.

Why other options are wrong:

- Option A (Haemophilia A): Factor VIII deficiency – prolonged PTT, normal BT and platelet count; X-linked; males affected; no ristocetin defect.
- Option C (Glanzmann's): Deficiency of GPIIb/IIIa – prolonged BT, normal platelet count, absent aggregation with ALL agonists except ristocetin.
- Option D (Bernard-Soulier): Deficiency of GPIb – prolonged BT, *low* platelet count (thrombocytopenia); giant platelets.

Final Answer: Prolonged BT + normal platelet count + absent ristocetin aggregation = Von Willebrand disease. $\Rightarrow \boxed{B}$

Answer: (B) **Go Back to Q20**

Q21.

Solution

Concept – Acute myeloid leukaemia (AML) and Auer rods: Auer rods are elongated, red-pink crystalline inclusions in the cytoplasm of myeloid blasts, composed of fused azurophilic (primary) granules containing **myeloperoxidase**. They are **pathognomonic of AML** and are most prominent in **AML-M3 (acute promyelocytic leukaemia / APML)** due to t(15;17)/PML-RARA. Gum hypertrophy (gingival infiltration) is classic for M4 (myelomonocytic) and M5 (monocytic) AML. MPO positivity confirms myeloid lineage.

Key Point: APML (M3) is the most dangerous due to DIC (from procoagulant granules) but is also the most curable with **ATRA (all-trans retinoic acid)** which induces differentiation. **Auer rods are NEVER seen in ALL.**

Why other options are wrong:

- Option A (ALL): TdT+, MPO-, no Auer rods – lymphoid blasts.
- Option B (CML): Chronic; mature granulocytes, not blasts; Philadelphia chromosome t(9;22).
- Option D (Multiple myeloma): Plasma cell malignancy; Bence-Jones proteins; lytic bone lesions; not leukaemic blasts with Auer rods.

Final Answer: Auer rods + MPO+ blasts = AML (pathognomonic). $\Rightarrow \boxed{C}$

Answer: (C) **Go Back to Q21**

Q22.

Solution

Concept – Beta-thalassemia major (Cooley's anaemia): Beta-thalassemia major results from **mutations in both β-globin genes** (homozygous), completely abolishing HbA ($\alpha_2\beta_2$) synthesis. Excess free α-chains precipitate and damage erythroid precursors (ineffective erythropoiesis) and red cells (haemolysis). Compensatory: **HbF** ($\alpha_2\gamma_2$) predominates (90%) and **HbA is absent**. Massive compensatory marrow expansion leads to **frontal bossing, maxillary overgrowth, "hair-on-end" skull X-ray** ("Cooley's facies"). Splenomegaly from extramedullary haematopoiesis.

Key Point: Beta-thalassemia **major** (homozygous) $\rightarrow$ transfusion-dependent anaemia, iron overload. Beta-thalassemia **minor** (heterozygous carrier) $\rightarrow$ mild microcytic anaemia, **elevated HbA2** ($>$3.5%).

Why other options are wrong:

- Option B (Sickle cell): HbS on electrophoresis; sickle-shaped cells; vaso-occlusive crises; no bony changes.
- Option C (Alpha-thalassemia major / Hb Bart's): Incompatible with life – hydrops fetalis at birth; HbH (β_4) inclusions.
- Option D (Iron deficiency): No hepatosplenomegaly, no bony changes; low ferritin, not absent HbA.

Final Answer: Absent HbA + HbF 90% + bone marrow expansion = beta-thalassemia major. $\Rightarrow \boxed{A}$

Answer: (A) **Go Back to Q22**

Q23.

Solution

Concept – Ghon complex in primary tuberculosis: The **Ghon focus** is the initial small (1–1.5 cm) subpleural parenchymal granulomatous focus in primary pulmonary tuberculosis, most often in the lower lobe or mid-lung (where ventilation is greatest). Together with the **enlarged hilar or mediastinal lymph nodes** (which represent lymphatic spread from the Ghon focus), they form the **Ghon complex** (= Ranke complex). This is the hallmark of **primary TB** in children and immunologically naive individuals. The lesion typically undergoes caseous

necrosis and calcification.

Key Point: **Ghon focus** alone = parenchymal lesion only. **Ghon complex** = Ghon focus + draining hilar lymphadenopathy. **Simon foci** = apical lesions from haematogenous spread during primary TB, which reactivate later as secondary TB.

Why other options are wrong:

- Option A (Miliary TB): Haematogenous dissemination – 1–2 mm millet-seed lesions throughout lungs.
- Option B (Simon foci): Apical reactivation lesions of secondary TB in adults.
- Option C (Secondary TB): Apical fibrocavitary disease in previously sensitised adults; NOT the primary complex.

Final Answer: Primary TB peripheral focus + hilar lymphadenopathy = Ghon complex. $\Rightarrow \boxed{D}$

Answer: (D) **Go Back to Q23**

Q24.

Solution

Concept – Bronchopneumonia: Bronchopneumonia (lobular pneumonia) is characterised by **patchy consolidation** beginning in the bronchioles (bronchiolitis) and spreading to the surrounding alveoli in **multiple lobules**, usually affecting both lower lobes. It is the typical pneumonia of the elderly, debilitated, and bedridden. Common causative organisms: Staphylococcus aureus, Klebsiella, Pseudomonas (in debilitated/hospital patients), and aspiration organisms. Contrast with **lobar pneumonia**: complete consolidation of an entire lobe (typically Streptococcus pneumoniae).

Key Point: **Bronchopneumonia** = patchy, multi-lobular, lower zones, begins in bronchioles. **Lobar pneumonia** = entire lobe consolidated, 4 stages (congestion $\rightarrow$ red hepatisation $\rightarrow$ grey hepatisation $\rightarrow$ resolution). **Aspiration** (stroke patients) favours the right lower lobe.

Why other options are wrong:

- Option A (Lobar pneumonia): Complete lobar consolidation – not patchy bilateral; less common in elderly.
- Option C (Interstitial/DAD): Viral, atypical pathogens; diffuse bilateral "ground glass" – not consolidation.
- Option D (Lung abscess): Localised cavity with pus; specific pathogen

(Staphylococcus, anaerobes); not the immediate presentation described.

Final Answer: Aspiration-prone stroke patient + bilateral patchy lower-lobe consolidation = bronchopneumonia. $\Rightarrow \boxed{B}$

Answer: (B) **Go Back to Q24**

Q25.

Solution

Concept – Squamous cell carcinoma (SCC) of the lung: SCC of the lung is strongly associated with **heavy cigarette smoking**. It arises centrally from the bronchial mucosa (squamous metaplasia → dysplasia → carcinoma in situ → invasive). Key features: (1) **central/hilar location**, (2) **cavitation** (central necrosis – "coin lesion with cavity"), (3) **keratin pearls and intercellular bridges** on histology, (4) **paraneoplastic hypercalcaemia** (secretion of PTHrP – parathyroid hormone-related protein). SCC spreads locally and to hilar nodes before distant metastases.

Key Point: Paraneoplastic syndromes: **SCC** = hypercalcaemia (PTHrP). **Small cell carcinoma** = SIADH (ADH) or Cushing's (ACTH). **Adenocarcinoma** = hypertrophic pulmonary osteoarthropathy.

Why other options are wrong:

- Option A (Small cell): Central, rapidly growing, neuroendocrine; but NO keratin pearls; "oat cell" appearance; SIADH or Cushing's.
- Option B (Adenocarcinoma): Peripheral location, lepidic growth (formerly BAC); mucin production; not keratin pearls; does not typically cavitate.
- Option D (Large cell): Peripheral, undifferentiated, no keratin pearls or gland formation – diagnosis of exclusion.

Final Answer: Central cavitating mass + keratin pearls + hypercalcaemia = squamous cell carcinoma of lung. $\Rightarrow \boxed{C}$

Answer: (C) **Go Back to Q25**

Q26.

Solution

Concept – Silicosis: Silicosis is caused by inhalation of **crystalline silicon dioxide (silica)** particles by miners, quarry workers, sandblasters, and pottery work-

ers. Macrophages ingest silica particles but cannot digest them – the particles are cytotoxic and kill macrophages, releasing inflammatory mediators that activate fibroblasts. The hallmark lesion is the **silicotic nodule**: concentric laminated collagen surrounding birefringent silica particles (apple-green birefringence under polarised light). Silicosis predisposes to **tuberculosis (silicotuberculosis)** because silica impairs macrophage killing of mycobacteria. Progressive massive fibrosis (PMF) occurs with prolonged exposure.

Key Point: Silicosis + TB risk is so high that patients with silicosis should be screened for TB. **Asbestosis** $\rightarrow$ mesothelioma, lung adenocarcinoma – asbestos bodies (ferruginous bodies). **Caplan syndrome** = pneumoconiosis + rheumatoid arthritis.

Why other options are wrong:

- Option B (Asbestosis): Associated with mesothelioma and lung carcinoma; not specifically linked to TB; asbestos bodies (golden-brown dumbbell-shaped).
- Option C (Anthracosis): Coal dust – "black lung"; Caplan syndrome (with RA); not strongly linked to TB.
- Option D (Berylliosis): Granulomatous lung disease mimicking sarcoidosis; associated with aerospace/nuclear industries.

Final Answer: Silicon dioxide inhalation $\rightarrow$ silicosis $\rightarrow$ silicotuberculosis risk. $\Rightarrow$ $\boxed{A}$

Answer: (A) **Go Back to Q26**

Q27.

Solution

Concept – Histological changes in MI at 24 hours: The sequential histological changes in myocardial infarction are:

- **0–6 hours**: No light microscopic changes (only EM changes: mitochondrial swelling, glycogen loss).
- **6–24 hours**: **Coagulative necrosis** becomes evident – "ghost outlines" of myocytes with hypereosinophilic cytoplasm, pyknotic nuclei; **neutrophil infiltration begins**.
- **1–4 days**: Dense neutrophil infiltration, necrotic debris removal begins.
- **5–14 days**: Macrophage infiltration, granulation tissue (neovascularisation, fibroblasts).

- **Weeks–months**: Collagen scar (dense white scar).

Key Point: At 22 hours, this patient is in the **6–24 hour window**: coagulative necrosis + early neutrophil infiltration. Wavy fibres are the earliest LM change (within first few hours). The "mummified" appearance of coagulative necrosis preserves cell outlines.

Why other options are wrong:

- Option A (No LM changes): Applies to 0–6 hours only.
- Option B (Macrophage infiltration): Occurs after 5 days.
- Option C (Dense collagenous scar): Occurs weeks to months later.

Final Answer: 22-hour MI $\rightarrow$ coagulative necrosis + early neutrophil infiltration. $\Rightarrow \boxed{D}$

Answer: (D) **Go Back to Q27**

Q28.

Solution

Concept – Foam cells in atherosclerosis (monocyte-derived macrophages): Atherosclerosis begins when **LDL** accumulates in the subintimal space and undergoes oxidative modification. **Circulating monocytes** are recruited to the intima by endothelial-expressed adhesion molecules (VCAM-1, ICAM-1) and MCP-1. In the intima, monocytes differentiate into **macrophages**, which ingest oxidised LDL via unregulated **scavenger receptors** (SR-A, CD36 – unlike the regulated LDL receptor). The lipid-laden macrophages are called **foam cells** and form the **fatty streak** – the earliest atherosclerotic lesion.

Key Point: Scavenger receptors are NOT downregulated by intracellular cholesterol accumulation (unlike LDL receptors), so macrophages continue to ingest oxidised LDL until they become foam cells. Foam cells are the hallmark of the fatty streak.

Why other options are wrong:

- Option A (Smooth muscle cells): Smooth muscle can also become foam cells but they migrate from media – the primary foam cell precursor is the blood monocyte.
- Option C (Lymphocytes): Do not phagocytose lipid to become foam cells.
- Option D (Mast cells): Present in adventitia; not foam cell precursors.

Final Answer: Monocytes (blood-derived) $\rightarrow$ macrophages $\rightarrow$ ingest oxidised LDL via scavenger receptors $\rightarrow$ foam cells. $\Rightarrow \boxed{B}$

Answer: (B) **Go Back to Q28**

Q29.

Solution

Concept – Hypertrophic cardiomyopathy (HCM): HCM is characterised by **asymmetric septal hypertrophy** (septum disproportionately thicker than the posterior LV wall), **systolic anterior motion (SAM) of the mitral valve** (abnormal anterior displacement of the anterior mitral leaflet during systole), and **left ventricular outflow tract (LVOT) obstruction**. It is the most common cause of **sudden cardiac death in young athletes**. Mutations in sarcomeric proteins (most commonly β-myosin heavy chain, cardiac myosin-binding protein C) are responsible. Autosomal dominant inheritance. Histology: myocyte disarray ("whorling").

Key Point: HCM = hypertrophied but non-dilated ventricle with *diastolic* dysfunction (impaired filling). LVOT obstruction worsens with decreased preload (dehydration, Valsalva) – explaining syncope during exercise. Treatment: beta-blockers, septal myectomy, ICD.

Why other options are wrong:

- Option A (Dilated cardiomyopathy): *Dilated*, thin-walled ventricle; systolic dysfunction; most common form of cardiomyopathy.
- Option B (Restrictive): Non-dilated, non-hypertrophied ventricle; impaired diastolic filling; caused by amyloid, sarcoid, haemochromatosis.
- Option D (Cardiac amyloidosis): Restrictive physiology; "sparkling" appearance on echo; symmetric LV wall thickening without LVOT obstruction.

Final Answer: Young athlete + asymmetric septal hypertrophy + LVOT obstruction + SAM = HCM. $\Rightarrow \boxed{C}$

Answer: (C) **Go Back to Q29**

Q30.

Solution

Concept – Barrett's oesophagus: Barrett's oesophagus is defined as **intestinal metaplasia of the oesophageal squamous epithelium to columnar epithelium with goblet cells**, resulting from chronic gastro-oesophageal reflux disease

(GORD). It requires at least 1 cm of endoscopically visible salmon-coloured mucosa proximal to the gastro-oesophageal junction with biopsy-confirmed goblet cells. **Barrett's is a premalignant condition**: it predisposes to **oesophageal adenocarcinoma** (risk 30–50× higher than general population). Surveillance endoscopy is recommended.

Key Point: The sequence is: GORD → oesophagitis → squamous metaplasia → Barrett's (intestinal metaplasia with goblet cells) → dysplasia (low grade → high grade) → **adenocarcinoma**. Goblet cells are the essential diagnostic feature of Barrett's.

Why other options are wrong:

- Option B (Squamous cell carcinoma): Arises from normal squamous mucosa; different risk factors (alcohol, smoking, achalasia, nitrosamines); upper/middle third of oesophagus.
- Option C (Achalasia): Failure of LES relaxation; megaoesophagus on barium swallow; not related to GORD.
- Option D (Mallory-Weiss): Mucosal laceration from forceful vomiting; acute presentation; no metaplasia.

Final Answer: Chronic GORD → intestinal metaplasia with goblet cells → Barrett's oesophagus → risk of adenocarcinoma. ⇒ $\boxed{A}$

Answer: (A) **Go Back to Q30**

Q31.

Solution

Concept – Ulcerative colitis (UC) vs Crohn's disease: The fundamental pathological distinction between UC and Crohn's disease:

- **UC**: inflammation limited to **mucosa and submucosa**; **continuous** from rectum proximally; **crypt abscesses**; no granulomas; pseudopolyps; increased risk of colorectal carcinoma.
- **Crohn's**: **transmural** inflammation (all layers); **skip lesions**; **non-caseating granulomas**; fissuring ulcers; cobblestone mucosa; perianal disease; affects any part of GI tract (mouth to anus).

Key Point: Continuous rectal involvement + mucosal/submucosal only + crypt abscesses = **UC**. Transmural + skip lesions + granulomas + fistulae = **Crohn's**.

Why other options are wrong:

- Option A (Transmural + granulomas + skip): These are Crohn's features, not UC.
- Option B (Cobblestone + fissuring ulcers): Crohn's disease.
- Option C (Perianal disease + fistulae): Crohn's disease – perianal fistulae, skin tags, abscesses.

Final Answer: UC = mucosal and submucosal involvement, continuous, crypt abscesses, no granulomas. $\Rightarrow \boxed{D}$

Answer: (D) **Go Back to Q31**

Q32.

Solution

Concept – Mallory-Denk bodies in alcoholic hepatitis: Mallory-Denk bodies (formerly "Mallory's hyaline" or "alcoholic hyaline") are **irregular eosinophilic intracytoplasmic inclusions** within ballooned hepatocytes in alcoholic hepatitis. They are composed of **aggregated intermediate filament proteins**, specifically cytokeratins 8 and 18, which form misfolded, ubiquitinated protein aggregates. While most characteristic of alcoholic hepatitis, they can also be found in non-alcoholic steatohepatitis (NASH), primary biliary cirrhosis, and Wilson disease (but are most pathognomonic of alcohol).

Key Point: The AST/ALT ratio $>$ 2:1 is a classic clue for **alcoholic liver disease** (alcohol preferentially depletes ALT due to pyridoxine deficiency). Neutrophilic infiltration (not lymphocytic) is seen in alcoholic hepatitis.

Why other options are wrong:

- Option A (Councilman bodies): Acidophilic apoptotic hepatocytes – seen in viral hepatitis (yellow fever, hepatitis A/B).
- Option C (Dutcher bodies): Intranuclear pseudoinclusions of immunoglobulin – seen in lymphoplasmacytic lymphoma/Waldenström macroglobulinaemia.
- Option D (Psammoma bodies): Concentric calcified lamellae – seen in meningioma, papillary thyroid Ca, serous ovarian Ca.

Final Answer: Alcoholic hepatitis + eosinophilic intracytoplasmic cytokeratin aggregates = Mallory-Denk bodies. $\Rightarrow \boxed{B}$

Answer: (B) **Go Back to Q32**

Q33.

Solution

Concept – Portal hypertension and portosystemic shunting: In cirrhosis, increased intrahepatic resistance raises **portal venous pressure** ($>$ 12 mmHg = clinically significant). High pressure forces blood through **portosystemic collaterals**:

- **Oesophageal varices**: portal $\rightarrow$ left gastric $\rightarrow$ oesophageal veins (most dangerous – haemorrhage).
- **Haemorrhoidal veins**: superior rectal $\rightarrow$ inferior rectal (anorectal varices).
- **Caput medusae**: portal $\rightarrow$ paraumbilical $\rightarrow$ epigastric veins (periumbilical dilatation).

Oesophageal varices represent the most life-threatening complication; bleeding carries 20–30% mortality per episode.

Key Point: Portal hypertension causes (Triad): (1) Splenomegaly, (2) Oesophageal varices + haemorrhoids + caput medusae, (3) Ascites. Treatment of varices: endoscopic band ligation, propranolol (non-selective β-blocker), TIPS (transjugular intrahepatic portosystemic shunt).

Why other options are wrong:

- Option A (Hypoalbuminaemia): Causes ascites and oedema – but not specifically varices.
- Option B (Hepatic artery thrombosis): Causes hepatic ischaemia – not variceal formation.
- Option D (SVC obstruction): Causes facial/upper body oedema (SVC syndrome) – not oesophageal varices.

Final Answer: Cirrhosis $\rightarrow$ portal hypertension $\rightarrow$ portosystemic shunting $\rightarrow$ oesophageal varices. $\Rightarrow \boxed{C}$

Answer: (C) **Go Back to Q33**

Q34.

Solution

Concept – IgA nephropathy (Berger disease): IgA nephropathy is the **most common primary glomerulonephritis worldwide**. It is characterised by **mesangial IgA deposits** detected by immunofluorescence. The classic presentation is **episodic gross haematuria** occurring **1–3 days after an upper respiratory in-**

fection ("synpharyngitic haematuria") – much earlier than post-streptococcal GN (2–3 weeks). Serum complement (C3, C4) is **normal** (unlike lupus or MPGN). Pathogenesis involves galactose-deficient IgA1 that forms immune complexes depositing in the mesangium.

Key Point: IgA nephropathy: haematuria 24–72 hours after URTI, mesangial IgA, normal complement. **Post-streptococcal GN**: haematuria 2–3 weeks after pharyngitis, subepithelial "humps", low C3.

Why other options are wrong:

- Option B (Post-streptococcal GN): Latent period 2–3 weeks after strep throat; low C3; subepithelial humps on EM.
- Option C (Thin BM disease): Persistent microscopic haematuria, no IgA; benign prognosis.
- Option D (Alport syndrome): X-linked; sensorineural deafness; basket-weave thinning/thickening of GBM on EM; no IgA.

Final Answer: Haematuria 24 h after URTI + mesangial IgA deposits + normal complement = IgA nephropathy (Berger disease). $\Rightarrow \boxed{A}$

Answer: (A) **Go Back to Q34**

Q35.

Solution

Concept – Clear cell renal cell carcinoma and VHL gene: Clear cell RCC (ccRCC) is the most common type of renal cell carcinoma (75%). Tumour cells have abundant **clear cytoplasm** due to high glycogen and lipid content. The **VHL tumour suppressor gene** (chromosome 3p25) is mutated or hypermethylated in the vast majority of sporadic ccRCCs, as well as in all cases of von Hippel-Lindau disease. VHL protein normally targets HIF-1α for degradation; loss of VHL $\rightarrow$ HIF-1α accumulation $\rightarrow$ VEGF and erythropoietin upregulation $\rightarrow$ angiogenesis and polycythaemia. Paraneoplastic: **polycythaemia** (EPO), **hypercalcaemia** (PTHrP), hypertension (renin).

Key Point: Classic triad of RCC: haematuria + flank pain + palpable mass ("too late" triad – seen in $< 10\%$ but indicates advanced disease). VHL mutation $\rightarrow$ clear cell RCC; **MET** mutation $\rightarrow$ papillary RCC type 1; **FH** mutation $\rightarrow$ hereditary leiomyomatosis and RCC.

Why other options are wrong:

- Option A (BRCA1): Associated with hereditary breast/ovarian cancer, not RCC.
- Option B (MET): Papillary RCC type 1 (hereditary) – not clear cell type.
- Option C (TP53): Loss of TP53 is common in many cancers but is not the defining mutation of clear cell RCC.

Final Answer: Clear cytoplasm + VHL mutation $\rightarrow$ clear cell RCC (most common subtype). $\Rightarrow \boxed{D}$

Answer: (D) **Go Back to Q35**

Q36.

Solution

Concept – Membranous nephropathy – subepithelial deposits: Membranous nephropathy (MN) is the most common cause of **nephrotic syndrome in adults** (age 40–60 years). The pathognomonic finding on electron microscopy is **subepithelial electron-dense deposits** between the glomerular basement membrane (GBM) and the visceral epithelial cell (podocyte) foot processes, with **intervening GBM projections (spikes)**. The "spike-and-dome" pattern on **silver (Jones) stain** reflects the BM spikes growing between deposits. Immunofluorescence: granular IgG and C3 along capillary loops. Most cases are primary (anti-PLA2R antibodies) or secondary (hepatitis B, malignancy, penicillamine, gold).

Key Point: Subepithelial deposits = membranous nephropathy. **Subendothelial** = lupus nephritis class III/IV, MPGN type 1. **Mesangial** = IgA nephropathy, early lupus. **No deposits** = minimal change disease (fusion of foot processes only).

Why other options are wrong:

- Option A (Subendothelial): Lupus nephritis III/IV; complement consumption.
- Option C (Mesangial only): IgA nephropathy – mesangial, not subepithelial.
- Option D (No immune deposits): Minimal change disease – negative IF; podocyte foot process fusion only.

Final Answer: Spike-and-dome subepithelial deposits = membranous nephropathy. $\Rightarrow \boxed{B}$

Answer: (B) **Go Back to Q36**

Q37.

Solution

Concept – Graves disease: Graves disease is an **autoimmune hyperthyroidism** caused by **thyroid-stimulating immunoglobulin (TSI)** – IgG autoantibodies that bind and *stimulate* (not block) **TSH receptors** on thyroid follicular cells. This mimics TSH action, causing continuous thyroid hormone synthesis and secretion, and follicular hypertrophy (diffuse goitre). TSI crosses the placenta (neonatal Graves). The unique extrathyroidal features – **exophthalmos (proptosis)** (from GAG deposition and lymphocytic infiltration of orbital tissue) and **pretibial myxoedema** (localised dermopathy) – are caused by the autoimmune process, not thyroid hormones.

Key Point: TSI (thyroid-stimulating immunoglobulin = thyroid-stimulating antibody = TSHR-Ab-stimulating) causes Graves. Anti-TPO antibodies cause Hashimoto's thyroiditis (hypothyroidism). The low/undetectable TSH with high free T4 confirms primary hyperthyroidism.

Why other options are wrong:

- Option A (Anti-thyroglobulin): Causes Hashimoto's – hypothyroidism, not hyperthyroidism.
- Option B (Anti-TPO + hypothyroidism): Hashimoto's thyroiditis – eventual hypothyroid.
- Option D (Activating TSH receptor mutation): Causes toxic adenoma (uninodular) or toxic multinodular goitre – autosomal dominant; no exophthalmos.

Final Answer: Diffuse toxic goitre + exophthalmos + pretibial myxoedema = Graves disease (TSH-receptor stimulating antibody). $\Rightarrow \boxed{C}$

Answer: (C) **Go Back to Q37**

Q38.

Solution

Concept – Addison disease (primary adrenocortical insufficiency): Addison disease is caused by **destruction of all three zones of the adrenal cortex** (most commonly autoimmune in developed countries; also tuberculosis, metastatic carcinoma, bilateral adrenalectomy). Loss of cortisol (zona fasciculata), aldosterone (zona glomerulosa), and DHEA causes the characteristic features: (1) **Hyperpigmentation** – due to elevated ACTH and MSH (from POMC cleavage) stimulating melanocytes; (2) **Hypotension, hyponatraemia, hyperkalaemia** (aldosterone

deficiency); (3) **Hypoglycaemia** (cortisol deficiency). The **Synacthen (ACTH stimulation) test** confirms primary disease (cortisol fails to rise).

Key Point: Hyperpigmentation occurs ONLY in *primary* adrenocortical insufficiency (elevated ACTH) – NOT in secondary (pituitary failure, where ACTH is low). Addisonian crisis: life-threatening acute presentation with shock, hypoglycaemia, and electrolyte crisis – treat with IV hydrocortisone.

Why other options are wrong:

- Option B (Secondary insufficiency): Low ACTH (pituitary failure – e.g., exogenous steroids) – NO hyperpigmentation; aldosterone usually preserved (regulated by renin-angiotensin).
- Option C (Phaeochromocytoma): Hypertension (not hypotension); episodic; catecholamine excess.
- Option D (Cushing's): Cortisol *excess* – hypertension, hyperglycaemia, not hypoglycaemia.

Final Answer: Buccal hyperpigmentation + hypotension + failed Synacthen test = Addison disease (primary adrenocortical insufficiency). $\Rightarrow \boxed{A}$

Answer: (A) **Go Back to Q38**

Q39.

Solution

Concept – Basal cell carcinoma (BCC): BCC is the **most common skin malignancy** (and most common malignancy overall in humans), arising from the basal layer of the epidermis or hair follicle. Risk factors: **UV radiation** (sun exposure), fair skin, immunosuppression, arsenic. The classic presentation is a **pearly, translucent nodule** with **rolled/"pearly" border** and telangiectases. On histology: **nests of basaloid cells with peripheral nuclear palisading** (nuclei line up in a picket-fence arrangement at the periphery of tumour nests) and a characteristic **stromal retraction artefact** (cleft between tumour nests and surrounding stroma). BCC rarely metastasises but is locally destructive.

Key Point: Peripheral palisading is the histological hallmark of BCC. **Keratin pearls** = squamous cell carcinoma. **Pagetoid spread** = Paget's disease, melanoma in situ. **Reed-Sternberg cells** = Hodgkin lymphoma.

Why other options are wrong:

- Option A (Keratin pearls + intercellular bridges): Squamous cell carcinoma

– central keratin with surrounding squamous cells.
- Option B (Pagetoid spread): Paget's disease of nipple or extramammary Paget's; melanoma in situ.
- Option C (Reed-Sternberg cells): Hodgkin lymphoma – owl-eye giant cells; not a skin tumour finding.

Final Answer: Pearly nodule + peripheral palisading + stromal retraction = basal cell carcinoma. $\Rightarrow \boxed{D}$

Answer: (D) **Go Back to Q39**

Q40.

Solution

Concept – Granuloma annulare: Granuloma annulare is a **benign idiopathic dermatosis** characterised by **palisading granulomas**: histiocytes arranged in a ring around a central zone of **degenerated collagen (necrobiosis) with mucin deposition**. It presents as ring-shaped flesh-coloured or erythematous papules on the dorsum of hands and feet, elbows, and knees. Blood glucose is **normal** (distinguishing from necrobiosis lipoidica diabeticorum, which is associated with diabetes). The aetiology is unclear; the generalised form has an association with diabetes mellitus but the localised form does not.

Key Point: Both **granuloma annulare** and **necrobiosis lipoidica diabeticorum (NLD)** show palisading granulomas with necrobiosis. **Key difference**: NLD occurs on the *shins* in diabetic patients (thick yellowish plaques); granuloma annulare occurs on *dorsal hands/feet* with normal blood glucose in the localised form.

Why other options are wrong:

- Option A (Necrobiosis lipoidica): Also shows necrobiosis + palisading histiocytes but is associated with diabetes; located on shins; yellow waxy plaques.
- Option C (Rheumatoid nodule): Subcutaneous, firm; over bony prominences; associated with RA and elevated RF; also shows palisading histiocytes but deeper in skin.
- Option D (Sarcoidosis): Non-caseating granulomas without necrobiosis or mucin; elevated serum ACE; hilar adenopathy.

Final Answer: Ring-shaped rash + palisading histiocytes + necrobiosis + normal glucose = granuloma annulare. $\Rightarrow \boxed{B}$

Answer: (B) **Go Back to Q40**

Answer Key – FMGE Pathology Sample Paper 2

1	C	2	A	3	D	4	B	5	C
6	A	7	D	8	B	9	C	10	A
11	D	12	B	13	C	14	A	15	D
16	B	17	C	18	A	19	D	20	B
21	C	22	A	23	D	24	B	25	C
26	A	27	D	28	B	29	C	30	A
31	D	32	B	33	C	34	A	35	D
36	B	37	C	38	A	39	D	40	B