FMGE Pathology Sample Paper – 1

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 62-year-old man is brought to the emergency department 6 hours after the onset of crushing chest pain. He is found to have an acute myocardial infarction. A biopsy of the infarcted myocardium at 48 hours would most likely show cells in which of the following states?

(A) Liquefactive necrosis with neutrophilic infiltration

(B) Caseous necrosis with central acellular debris

(C) Fat necrosis with saponification

(D) Ghost cell outlines with preserved tissue architecture

Q2. A pathologist is reviewing histological slides and notes single cell death occurring without surrounding inflammatory infiltrate. The dying cells show chromatin condensation and membrane blebbing, and their fragments are rapidly phagocytosed by neighbouring cells. Which of the following features best distinguishes this process from necrosis?

(A) Release of lysosomal enzymes into the extracellular space

(B) Membrane blebbing without inflammatory response

(C) Cellular swelling with disruption of organelle membranes

(D) Mitochondrial swelling and ATP depletion

Q3. A 55-year-old woman with a history of recurrent pancreatitis undergoes a CT scan of the abdomen which reveals calcium deposits in a region of old fat necrosis. Laboratory investigations show a serum calcium of 9.2 mg/dL (normal) and serum phosphate of 3.8 mg/dL (normal). Which type of pathological calcification is demonstrated in this patient?

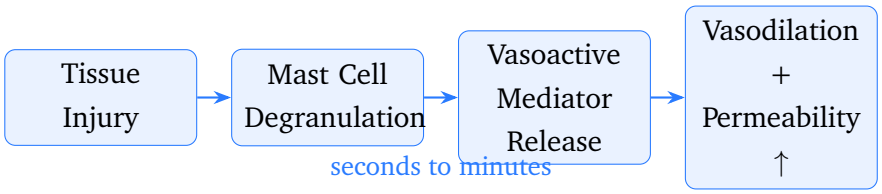
(A) Dystrophic calcification

(B) Metastatic calcification

(C) Calcinosis universalis

(D) Calcinosis circumscripta

Q4. A 28-year-old man sustains a bee sting on his forearm. Within seconds, he develops a wheal and flare reaction at the site. A pathologist studying the early vascular events of acute inflammation at this site identifies a vasoactive mediator responsible for the immediate transient increase in vascular permeability. The sequence of events is illustrated below:



Which mediator is primarily responsible for the immediate transient phase of increased vascular permeability?

(A) Bradykinin

(B) Leukotrienes C4, D4, E4

(C) Histamine

(D) Platelet-activating factor

Q5. A 58-year-old man dies 5 days after a large anterior myocardial infarction. At autopsy, the pericardial surfaces appear dull and rough, with a shaggy, yellow-white exudate that resembles two slices of buttered bread pulled apart. There is no significant effusion. Which of the following best describes this finding?

(A) Serofibrinous pericarditis

(B) Purulent (suppurative) pericarditis

(C) Haemorrhagic pericarditis

(D) Fibrinous pericarditis

Q6. A 35-year-old immigrant presents with a chronic cough, low-grade fever, and weight loss for 3 months. A lung biopsy is performed and reveals discrete granulomas composed of epithelioid macrophages, with large multinucleated giant cells at the periphery whose nuclei are arranged in a horseshoe or arc pattern. Which cell is being described?

(A) Foreign body giant cell

(B) Langhans giant cell

(C) Touton giant cell

(D) Reed-Sternberg cell

Q7. A 40-year-old woman undergoes an elective cholecystectomy. The clean surgical incision is closed primarily with sutures, with minimal tissue loss and well-apposed wound edges. The wound heals uneventfully over two weeks with a thin scar. This type of wound healing is best characterised as:

(A) Healing by primary intention

(B) Healing by secondary intention

(C) Healing by tertiary intention

(D) Healing by granulation tissue replacement

Q8. A microbiologist is studying the innate immune response against encapsulated bacteria. She notes that after activation of the complement cascade, a specific cleavage fragment coats the bacterial surface and dramatically enhances phagocytosis by binding to receptors on neutrophils and macrophages. Which complement component is primarily responsible for this function?

(A) C5a

(B) C1q

(C) C3b

(D) C4b2a

Q9. A 52-year-old man develops a deep vein thrombosis (DVT) in his left calf 3 days after undergoing an open femoral artery bypass graft surgery. Virchow's triad describes three factors predisposing to thrombosis. In the context of this surgical procedure, which component of Virchow's triad is most directly implicated by the vessel manipulation during surgery?

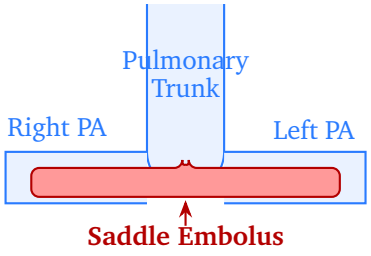
(A) Hypercoagulability

(B) Venous stasis

(C) Turbulent blood flow

(D) Endothelial injury

Q10. A 68-year-old woman with a known DVT suddenly develops severe dyspnoea and haemodynamic collapse. She dies before resuscitation can be completed. At autopsy, a large thrombus is found straddling the bifurcation of the pulmonary trunk, obstructing both main pulmonary arteries simultaneously, as shown in the diagram below:



What is the specific term for this type of pulmonary embolism?

(A) Paradoxical embolism

(B) Saddle embolus

(C) Venous air embolism

(D) Fat embolism

Q11. A 25-year-old man is brought to the emergency department following a stab wound to the abdomen with an estimated blood loss of 900 mL (approximately 18% of total blood volume). He is awake and anxious. Which of the following haemodynamic changes would be the *earliest* compensatory response expected in this patient?

(A) Tachycardia

(B) Hypotension

(C) Decreased urine output

(D) Metabolic acidosis

Q12. A 68-year-old man presents with difficulty in urination, nocturia, and lower back pain. Serum investigations reveal an elevated tumour marker that is organ-specific (though not cancer-specific) for the prostate gland. A subsequent transrectal biopsy confirms adenocarcinoma of the prostate. Which tumour marker was most likely elevated?

(A) Carcinoembryonic antigen (CEA)

(B) Alpha-fetoprotein (AFP)

(C) Prostate-Specific Antigen (PSA)

(D) Cancer antigen 125 (CA-125)

Q13. A 52-year-old woman is diagnosed with invasive ductal carcinoma of the left breast. She is found to have multiple ipsilateral axillary lymph node metastases at the time of surgery. Which route of spread is most commonly responsible for the nodal metastases seen in breast carcinoma?

(A) Haematogenous spread

(B) Lymphatic spread

(C) Transcoelomic spread

(D) Direct extension

Q14. A 45-year-old man presents with episodic facial flushing, watery diarrhoea, and wheezing for the past 6 months. Urinary 5-HIAA levels are markedly elevated. A CT scan reveals a 2 cm ileal mass with multiple liver metastases. Which substance secreted by the tumour is primarily responsible for the flushing and diarrhoea in this syndrome?

(A) Gastrin

(B) Histamine

(C) VIP (vasoactive intestinal peptide)

(D) Serotonin

Q15. A 22-year-old man presents with painless cervical lymphadenopathy and night sweats for 2 months. A lymph node biopsy reveals large binucleated cells with prominent eosinophilic "owl-eye" nucleoli set against a background of reactive lymphocytes, eosinophils, and plasma cells. These pathognomonic cells are diagnostic of which condition?

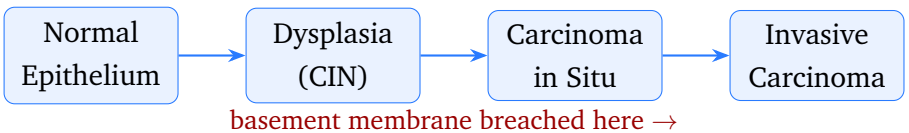
(A) Hodgkin lymphoma

(B) Diffuse large B-cell lymphoma

(C) Burkitt lymphoma

(D) Anaplastic large cell lymphoma

Q16. A colposcopy biopsy from the cervix of a 34-year-old woman shows disordered cell maturation, nuclear pleomorphism, increased nuclear:cytoplasmic ratio, and abnormal mitoses confined to the lower two-thirds of the epithelium, with an intact basement membrane. The progression from this lesion to invasive carcinoma is illustrated below:



What is the most accurate term for the histological finding described in this biopsy?

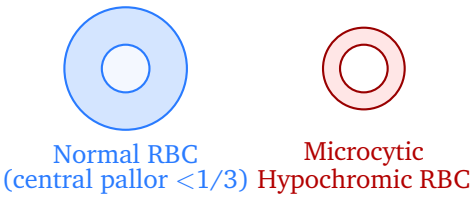
(A) Metaplasia

(B) Anaplasia

(C) Dysplasia

(D) Carcinoma in situ

Q17. A 28-year-old woman presents with fatigue and pallor. Her CBC shows: Hb 8.2 g/dL, MCV 68 fL, MCH 20 pg. Peripheral blood smear demonstrates the findings illustrated below:



Serum ferritin is 5 ng/mL (low) and TIBC is 480 μg/dL (high). Which of the following is the most likely diagnosis?

(A) Anaemia of chronic disease

(B) Iron deficiency anaemia

(C) Beta-thalassaemia trait

(D) Sideroblastic anaemia

Q18. A 45-year-old man presents with fatigue, weight loss, and a markedly enlarged spleen. CBC shows: WBC 145 $\times$ 10^9/L with a full myeloid spectrum including basophilia. Bone marrow biopsy is hypercellular with myeloid hyperplasia. Cytogenetic analysis reveals a specific chromosomal translocation resulting in a BCR-ABL fusion oncogene with constitutive tyrosine kinase activity. Which chromosomal abnormality is pathognomonic of this condition?

(A) t(15;17)

(B) t(8;14)

(C) t(14;18)

(D) t(9;22)

Q19. A 24-year-old woman presents with easy bruising and petechiae on her legs for 3 weeks following a mild upper respiratory illness. Investigations reveal: platelets 18 $\times$ 10^9/L, Hb 13.8 g/dL (normal), WBC 7.2 $\times$ 10^9/L (normal). PT and aPTT are both within normal limits. Bone marrow biopsy shows increased megakaryocytes. Anti-platelet IgG antibodies are detected in the serum. What is the most likely diagnosis?

(A) Immune thrombocytopenic purpura (ITP)

(B) Thrombotic thrombocytopenic purpura (TTP)

(C) Disseminated intravascular coagulation (DIC)

(D) Aplastic anaemia

Q20. A 16-year-old boy presents with recurrent episodes of spontaneous haemarthroses since early childhood. His mother's brother had a similar condition. Investigations show: aPTT 62 seconds (prolonged), PT 12 seconds (normal), platelet count normal, bleeding time normal. Which factor is deficient in this condition?

(A) Factor IX

(B) Factor XI

(C) Factor VIII

(D) von Willebrand factor

Q21. A 65-year-old man presents with bone pain, fatigue, and recurrent infections. Serum protein electrophoresis shows an M-spike in the gamma region. Urine protein electrophoresis reveals free immunoglobulin light chains that precipitate on heating to 60°C and redissolve at 100°C. X-ray of the spine shows multiple lytic "punched-out" lesions. What is the specific urinary protein described in this case?

(A) Tamm-Horsfall protein

(B) Bence Jones protein

(C) Microalbumin

(D) Beta-2 microglobulin

Q22. A 19-year-old Afro-Caribbean man presents with acute onset severe pain in his hands and feet (dactylitis) triggered by a cold weather exposure. His Hb electrophoresis shows predominantly HbS. A blood smear shows elongated, crescent-shaped red blood cells. Which of the following is the fundamental molecular mechanism underlying the pathological RBC shape change in this condition?

(A) Autoimmune haemolysis due to warm IgG antibodies

(B) Defective spectrin causing membrane fragility

(C) G6PD deficiency causing oxidative haemolysis

(D) HbS polymerisation in the deoxygenated state

Q23. A 9-year-old child is a contact of a sputum-positive tuberculosis patient. A chest X-ray shows a calcified nodule in the right lower lobe parenchyma accompanied by calcification of the right hilar lymph nodes, with calcified lymphangitic channels connecting the two. The child is asymptomatic. Which of the following terms best describes this radiological complex?

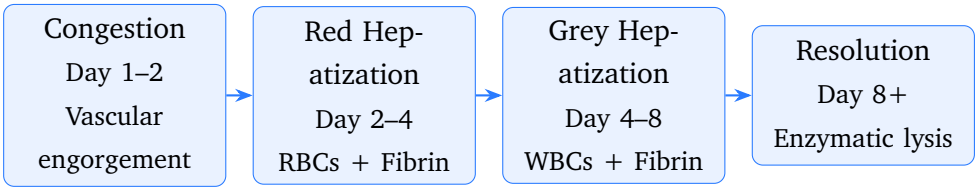
(A) Ghon complex

(B) Ranke complex

(C) Simon focus

(D) Rasmussen's aneurysm

Q24. A 55-year-old man presents with high fever, rigors, rust-coloured sputum, and right-sided pleuritic chest pain. CXR shows right lower lobe consolidation. He does not receive antibiotics and dies on day 3. At autopsy, the right lower lobe is firm and airless with a red, granular cut surface. Microscopically, alveoli are packed with red blood cells and fibrin. The four stages of lobar pneumonia are illustrated below:



Based on the gross and microscopic findings described (day 3, red, firm, RBCs + fibrin in alveoli), which stage of lobar pneumonia is present?

(A) Congestion

(B) Grey hepatization

(C) Red hepatization

(D) Resolution

Q25. A 62-year-old male smoker presents with confusion, hyponatraemia (serum Na^+ 118 mEq/L), and concentrated urine (urine osmolality 620 mOsm/kg). CXR reveals a hilar mass. Bronchoscopic biopsy shows small, round to oval cells with scant cytoplasm, nuclear moulding, and a high mitotic index, characteristic of a particular lung carcinoma subtype. Which paraneoplastic syndrome is most commonly associated with this tumour?

(A) Cushing syndrome (ectopic ACTH)

(B) SIADH (ectopic ADH secretion)

(C) Hypercalcaemia (ectopic PTHrP)

(D) Eaton-Lambert myasthenic syndrome

Q26. A 68-year-old retired shipyard worker presents with progressive dyspnoea and unilateral dull chest pain. CT thorax shows a large unilateral pleural effusion and pleural thickening encasing the lung. Lung tissue biopsy shows golden-brown beaded fibres coated with iron-containing protein (ferruginous bodies). A subsequent pleural biopsy confirms a malignant epithelioid neoplasm positive for calretinin and WT-1 on immunohistochemistry. What is the most likely diagnosis?

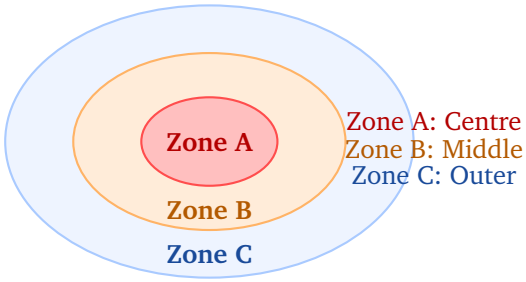
(A) Squamous cell carcinoma of the lung

(B) Lung adenocarcinoma

(C) Pleural fibroma (solitary)

(D) Malignant mesothelioma

Q27. Following an acute myocardial infarction, the pathological changes can be organised into three concentric zones as shown in the diagram below:



Which zone (Zone A – the central region) shows coagulative necrosis with ghost cell outlines?

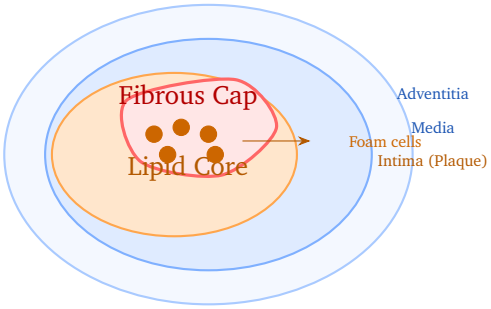
(A) Zone of coagulation (infarct centre)

(B) Zone of stasis

(C) Zone of hyperaemia

(D) Zone of haemorrhage

Q28. A 55-year-old man with hyperlipidaemia undergoes coronary angiography revealing significant stenosis. A segment of the coronary artery is examined histologically. The cross-sectional schematic of the atherosclerotic plaque is shown below:



The orange dots in the intima represent the key cells of the earliest atherosclerotic lesion (fatty streak). What are these cells?

(A) Smooth muscle cells migrating from media

(B) Lymphocytes infiltrating the intima

(C) Foam cells (lipid-laden macrophages)

(D) Platelet aggregates on endothelial injury

Q29. A 40-year-old man with a history of chronic alcohol use presents with progressive exertional dyspnoea, orthopnoea, and bilateral leg oedema. Echocardiography shows global four-chamber dilation with a severely reduced ejection fraction of 25%. The ventricular wall thickness is normal, but the chambers are markedly enlarged. Which type of cardiomyopathy does this patient have?

(A) Hypertrophic cardiomyopathy

(B) Dilated cardiomyopathy

(C) Restrictive cardiomyopathy

(D) Arrhythmogenic right ventricular cardiomyopathy

Q30. A 45-year-old man undergoes upper GI endoscopy for recurrent epigastric pain that worsens before meals and is relieved by eating. An antral biopsy shows chronic active gastritis and a rapid urease test (CLO test) is positive. Multiple duodenal ulcers are found at the duodenal bulb. Which site in the stomach is *H. pylori* most commonly found and most commonly associated with ulcer disease?

(A) Fundus

(B) Body (corpus)

(C) Cardia

(D) Antrum of stomach / duodenal bulb

Q31. A 28-year-old woman presents with right iliac fossa pain, diarrhoea, and weight loss for 6 months. Colonoscopy shows discontinuous areas of mucosal inflammation with deep longitudinal and transverse ulcers creating a cobblestone appearance, with normal intervening mucosa (skip lesions). A full-thickness biopsy of the terminal ileum shows inflamma-

tion extending through all layers of the bowel wall. Which histological feature is most characteristic of this disease?

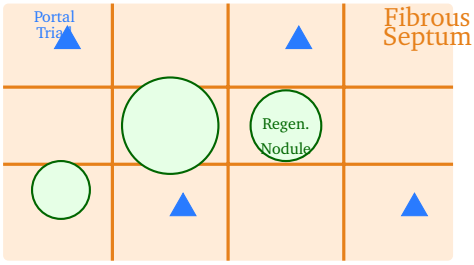
(A) Transmural inflammation

(B) Crypt abscesses with mucosal inflammation only

(C) Pseudomembrane formation

(D) Submucosal fibrosis only

Q32. A 58-year-old man with a 25-year history of alcohol use is found to have a small, nodular liver on imaging. He has jaundice, spider naevi, caput medusae, and ascites. A liver biopsy is performed and the histological findings are illustrated schematically below:



The green circles in the diagram represent which histological feature that is the hallmark of hepatic cirrhosis?

(A) Central vein dilation

(B) Fatty change (steatosis)

(C) Regenerative nodules

(D) Bridging fibrosis without nodule formation

Q33. A 55-year-old man with alcoholic cirrhosis presents to the emergency department vomiting large amounts of bright red blood. Upper GI endoscopy reveals dilated, tortuous submucosal veins in the lower oesophagus. These represent portosystemic collaterals that have developed due to portal hypertension. Which complication of portal hypertension is responsible for this presentation?

(A) Caput medusae

(B) Oesophageal varices

(C) Haemorrhoids

(D) Splenomegaly

Q34. An 8-year-old boy presents with massive proteinuria (urine protein 8 g/day), hypoalbuminaemia, and generalised oedema. Serum complement levels are normal. A renal biopsy is performed: light microscopy shows no abnormality, immunofluorescence is negative, but electron microscopy reveals diffuse effacement of podocyte foot processes. Which ultrastructural finding is characteristic of this condition?

(A) Sub-endothelial electron-dense deposits

(B) Subepithelial "spike and dome" pattern

(C) Mesangial IgA deposits

(D) Effacement of podocyte foot processes on EM

Q35. A 3-year-old girl is brought to the clinic with a painless abdominal mass that was noticed by her mother during bathing. The mass does not cross the midline. CT abdomen reveals a large intrarenal mass. Histology of the resected specimen shows triphasic components: blastemal cells, stromal cells, and epithelial elements forming primitive tubules and glomeruloid structures. Which of the following is the most likely diagnosis?

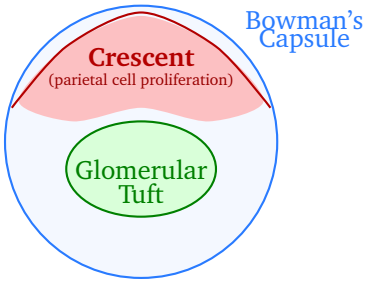
(A) Wilms tumour (nephroblastoma)

(B) Neuroblastoma

(C) Renal cell carcinoma

(D) Clear cell sarcoma of kidney

Q36. A 30-year-old woman presents with rapidly worsening oliguria, haematuria, and hypertension over 3 weeks. Serum creatinine has risen from 1.2 to 6.8 mg/dL. Urinalysis shows RBC casts. Renal biopsy findings are illustrated below:



The crescent seen in Bowman's space is formed by proliferation of which cells?

(A) Mesangial cells

(B) Endothelial cells of glomerular capillaries

(C) Parietal epithelial cells of Bowman's capsule

(D) Visceral epithelial cells (podocytes)

Q37. A 42-year-old woman presents with fatigue, weight gain, constipation, and a painless goitre. TFTs show TSH 12 mIU/L and free T4 0.6 ng/dL. A thyroid biopsy shows marked lymphocytic infiltration with germinal centre formation, destruction of follicles, and Hurthle cell (oxyphilic) change of the follicular epithelium. Which antibody is most characteristically elevated in this condition?

(A) Anti-TSH receptor (stimulating) antibodies

(B) Anti-thyroid peroxidase (anti-TPO) antibodies

(C) Anti-double-stranded DNA antibodies

(D) Anti-microsomal antibodies only

Q38. A 35-year-old woman presents with rapid weight gain, central obesity, a "buffalo hump" on the back of her neck, purple striae on her abdomen, facial plethora, and hypertension. Random cortisol is 68 μg/dL and 24-hour urinary free cortisol is markedly elevated. Overnight 1 mg dexamethasone suppression test is non-suppressive. Which is the primary pathophysiological mechanism causing this syndrome?

(A) Excessive mineralocorticoid production

(B) Excessive catecholamine secretion

(C) Excess growth hormone and IGF-1

(D) Excess cortisol production

Q39. A 58-year-old man presents with a changing pigmented lesion on his back. Excision biopsy confirms malignant melanoma. The pathologist uses Clark's level of invasion to assess depth. Clark level V is defined as tumour invasion into the subcutaneous fat. A patient whose melanoma is classified at Clark level V has which prognostic implication?

(A) Clark level V (deepest) – worst prognosis

(B) Clark level I – confined to epidermis, best prognosis

(C) Clark level III – fills the papillary dermis

(D) Clark level II – invades papillary dermis

Q40. A 24-year-old man of African descent notices a large, raised, firm, rubbery scar on his chest 6 months after a minor laceration. The scar has grown beyond the original wound margins and has not regressed spontaneously. Histologically, it shows thick, haphazardly arranged collagen bundles (type I and III collagen) extending into adjacent normal tissue. Which of the following best characterises this lesion?

(A) Hypertrophic scar (remains within wound margins)

(B) Desmoid tumour

(C) Extends beyond original wound margin

(D) Dermatofibroma

Detailed Solutions

Q1.

Solution

Concept – Coagulative Necrosis: Coagulative necrosis is the most common form of necrosis caused by ischaemia (except in the brain). The cell architecture is preserved because denatured proteins resist enzymatic digestion, creating *ghost cell outlines* – cells retain their shape but lose their nuclear staining (karyolysis/karyorrhexis).

Key Point: In myocardial infarction, the affected cardiomyocytes show preserved cell outlines with eosinophilic cytoplasm but absent nuclei (ghost cells) – the hallmark of coagulative necrosis. This appearance is maintained for 24–72 hours before neutrophilic infiltration and subsequent liquefaction begin.

Why other options are wrong:

- Option A: Liquefactive necrosis occurs in brain infarcts and bacterial abscesses; myocardial infarction does not produce liquefaction at 48 hours.
- Option B: Caseous necrosis with acellular debris is characteristic of tuberculosis granulomas, not MI.
- Option C: Fat necrosis with saponification is seen in pancreatic or breast injury, not myocardial infarction.

Final Answer: MI at 48 hours $\Rightarrow$ ghost cells with preserved outlines $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q1

Q2.

Solution

Concept – Apoptosis vs. Necrosis: Apoptosis is programmed cell death – an energy-dependent, genetically controlled process. It is characterised by cell shrinkage, chromatin condensation, membrane blebbing, and formation of apoptotic bodies that are rapidly phagocytosed. **Crucially, there is no inflammatory response.**

Key Point: Membrane blebbing without triggering an inflammatory response is the defining feature that distinguishes apoptosis from necrosis. In necrosis, membrane integrity is lost, releasing cellular contents that trigger acute inflammation.

Why other options are wrong:

- Option A: Release of lysosomal enzymes into the extracellular space is characteristic of *necrosis*, not apoptosis.
- Option C: Cellular swelling (oncosis) occurs in necrosis due to failure of the Na^+/K^+-ATPase pump, not in apoptosis (which shows cell shrinkage).
- Option D: Mitochondrial swelling and ATP depletion are features of necrosis; apoptosis uses the mitochondrial (intrinsic) pathway but requires ATP.

Final Answer: Apoptosis hallmark distinguishing from necrosis $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q2

Q3.

Solution

Concept – Dystrophic Calcification: Pathological calcification is of two types. *Dystrophic calcification* occurs in dead or dying tissue with **normal serum calcium and phosphate**. *Metastatic calcification* occurs in normal tissues due to **hypercalcaemia or hyperphosphataemia**.

Key Point: This patient has calcium deposits in a region of *necrotic* fat tissue (consequence of pancreatitis), with *normal* serum calcium and phosphate. This is classic dystrophic calcification – the necrotic tissue acts as a nidus for calcium deposition despite normal systemic levels.

Why other options are wrong:

- Option B: Metastatic calcification requires hypercalcaemia (e.g., hyperparathyroidism, vitamin D toxicity, multiple myeloma); serum calcium here is normal.
- Option C: Calcinosis universalis is diffuse calcium deposition in skin/subcutaneous tissue in connective tissue diseases (e.g., dermatomyositis), not focal.
- Option D: Calcinosis circumscripta is localised calcium deposition in soft tissues in scleroderma (CREST syndrome), not in post-pancreatitic fat necrosis.

Final Answer: Calcium in necrotic tissue + normal serum $Ca^{2+} \Rightarrow$ Dystrophic calcification $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – Acute Inflammation Mediators: Increased vascular permeability in acute inflammation occurs in two phases: (1) immediate transient (seconds to 30 minutes) mediated primarily by **histamine** and serotonin, and (2) delayed sustained (4–24 hours) mediated by leukotrienes, bradykinin, and complement.

Key Point: Histamine, preformed in mast cell granules, is released immediately upon tissue injury and causes rapid venular endothelial contraction, increasing vascular permeability. The wheal and flare reaction within seconds of a bee sting is a prototypical histamine-mediated response.

Why other options are wrong:

- Option A: Bradykinin causes a prolonged, delayed increase in permeability (generated by the kinin system, not preformed); it also causes pain and bronchoconstriction.
- Option B: Leukotrienes C4/D4/E4 (slow-reacting substances of anaphylaxis, SRS-A) cause a sustained, delayed increase in permeability and are synthesised de novo.
- Option D: Platelet-activating factor (PAF) causes a later phase of permeability increase and is involved in platelet aggregation; it is not the primary mediator of the immediate transient phase.

Final Answer: Immediate transient phase mediator $\Rightarrow$ Histamine $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q4

Q5.

Solution

Concept – Fibrinous Pericarditis: Post-MI pericarditis (Dressler syndrome or early reactive pericarditis) results in fibrin deposition on the pericardial surfaces. The classic gross description is the **"bread and butter" pericarditis** – when the pericardial surfaces are pulled apart, they resemble buttered bread being separated.

Key Point: The shaggy yellow-white exudate without significant effusion, with the "bread and butter" appearance, is pathognomonic of *fibrinous* pericarditis. The exudate is composed predominantly of fibrin protein strands.

Why other options are wrong:

- Option A: Serofibrinous pericarditis has both serous (straw-coloured fluid) and fibrinous components, producing a larger effusion – not described here.
- Option B: Purulent pericarditis has turbid/creamy purulent exudate, usually from bacterial infection; the exudate is frankly suppurative.
- Option C: Haemorrhagic pericarditis shows blood-stained fluid, typically in TB, malignancy, or uraemia.

Final Answer: "Bread and butter" pericarditis post-MI $\Rightarrow$ Fibrinous pericarditis $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q5

Q6.

Solution

Concept – Langhans Giant Cell: In granulomatous inflammation (e.g., tuberculosis), epithelioid macrophages fuse to form multinucleated giant cells. The **Langhans giant cell** has nuclei arranged in a horseshoe/arc or peripheral ring pattern at the periphery of the cell, characteristic of TB and other mycobacterial granulomas.

Key Point: The key distinguishing feature of the Langhans giant cell is the arrangement of nuclei in a *horseshoe or arc pattern around the periphery* of a large cell formed by fusion of epithelioid macrophages. This is distinct from the foreign body giant cell where nuclei are scattered haphazardly throughout the cytoplasm.

Why other options are wrong:

- Option A: Foreign body giant cells have nuclei scattered randomly/centrally throughout the cytoplasm; they surround non-degradable material.
- Option C: Touton giant cells have a wreath of nuclei around a central zone of foamy cytoplasm; seen in xanthomas and juvenile xanthogranuloma.
- Option D: Reed-Sternberg cells are large binucleated cells with owl-eye nucleoli, characteristic of Hodgkin lymphoma – not a giant cell of granulomatous inflammation.

Final Answer: Horseshoe-arranged nuclei in TB granuloma $\Rightarrow$ Langhans giant cell $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q6

Q7.

Solution

Concept – Wound Healing by Primary Intention: Healing by primary (first) intention occurs when a clean wound has well-apposed edges with *minimal tissue loss*. It involves: haematoma formation → epithelial regeneration → fibrous union → scar maturation. It results in a thin, neat scar with minimal granulation tissue.

Key Point: A clean, sutured surgical incision with no infection, minimal tissue loss, and well-opposed edges heals by primary intention. This is the most efficient form of healing, with rapid re-epithelialisation (24–48 hours) and minimal scarring.

Why other options are wrong:

- Option B: Healing by secondary intention occurs in large wounds with tissue loss (e.g., pressure sores, burns); involves extensive granulation tissue, wound contraction, and larger scars.
- Option C: Tertiary intention (delayed primary closure) involves deliberate delay before wound closure (e.g., contaminated wounds); not applicable to clean surgical wounds.
- Option D: Granulation tissue replacement describes secondary intention healing, not the clean sutured wound described.

Final Answer: Clean sutured surgical wound with minimal tissue loss ⇒ Primary intention ⇒ $\boxed{A}$

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – Complement C3b and Opsonisation: The complement system generates several biologically active fragments. **C3b** (opsonin) coats bacteria and foreign particles, enhancing phagocytosis by binding to CR1 (complement receptor 1) on neutrophils and macrophages. This process is called opsonisation.

Key Point: C3b is the principal opsonin of the complement system. When deposited on bacterial surfaces, it acts as a "tag" recognised by phagocyte CR1 receptors, dramatically increasing the efficiency of phagocytosis. This is especially important for encapsulated bacteria (e.g., *Streptococcus pneumoniae, H. influenzae*).

Why other options are wrong:

- Option A: C5a is a potent chemoattractant and anaphylatoxin – it recruits neutrophils to the site of infection and activates mast cells/basophils, but is not primarily an opsonin.
- Option B: C1q initiates the classical pathway by binding to antibody-antigen complexes; it does not directly opsonise bacteria for phagocytosis.
- Option D: C4b2a is the classical pathway C3 convertase enzyme complex – it cleaves C3 to generate C3b, but is not itself the opsonin.

Final Answer: Complement fragment coating bacteria to enhance phagocytosis $\Rightarrow$ C3b $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q8

Q9.

Solution

Concept – Virchow's Triad: Virchow's triad describes three factors predisposing to thrombus formation: (1) **Endothelial injury** (vessel wall damage), (2) **Stasis or turbulent flow**, and (3) **Hypercoagulability**. All three may be present simultaneously in surgical patients.

Key Point: During femoral artery bypass graft surgery, direct mechanical manipulation and surgical dissection of the vessel wall causes *endothelial injury*. Exposed subendothelial collagen activates platelets and the coagulation cascade, initiating thrombosis. This is the component of Virchow's triad most directly related to vessel manipulation.

Why other options are wrong:

- Option A: Hypercoagulability is present postoperatively (surgical stress, immobility increasing coagulation factors), but is not the most *direct* consequence of vessel manipulation.
- Option B: Venous stasis results from immobility and reduced muscle pump activity postoperatively, but is not directly caused by vessel manipulation.
- Option C: Turbulent blood flow results from plaque/stenosis or vessel geometry changes, not from the surgical manipulation itself.

Final Answer: Vessel manipulation during surgery $\Rightarrow$ Endothelial injury $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q9

Q10.

Solution

Concept – Saddle Embolus: A **saddle embolus** is a large thrombus that lodges at the bifurcation of a major artery, straddling ("saddling") both branches simultaneously. In pulmonary embolism, a saddle embolus sits at the bifurcation of the pulmonary trunk, obstructing both main pulmonary arteries and causing sudden haemodynamic collapse or death.

Key Point: The thrombus straddling the pulmonary trunk bifurcation (blocking both left and right pulmonary arteries) is the defining anatomical feature of a saddle embolus. This produces massive, bilateral obstruction of pulmonary blood flow leading to acute cor pulmonale and rapid death.

Why other options are wrong:

- Option A: Paradoxical embolism is a venous thrombus that crosses to the systemic circulation via a patent foramen ovale or ASD; it does not straddle the pulmonary bifurcation.
- Option C: Venous air embolism occurs when air enters the venous circulation (e.g., during central line insertion or surgery); it is gaseous, not a thrombus.
- Option D: Fat embolism results from release of fat globules after fracture of long bones or liposuction; it does not produce a discrete thrombus at the bifurcation.

Final Answer: Thrombus straddling the pulmonary trunk bifurcation $\Rightarrow$ Saddle embolus $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q10

Q11.

Solution

Concept – Haemorrhagic Shock (Compensatory Phase): With blood loss of 15–30% (Class II haemorrhagic shock), the body activates compensatory sympathoadrenal mechanisms. The **earliest** response is stimulation of the sinoatrial node by catecholamines, resulting in **tachycardia**. Blood pressure may be maintained initially by increased heart rate and peripheral vasoconstriction.

Key Point: Tachycardia is the *earliest* and most sensitive clinical sign of haemorrhagic shock. At 18% blood loss (Class I–II), baroreceptor unloading triggers a catecholamine surge causing increased heart rate before blood pressure falls or urine output decreases significantly.

Why other options are wrong:

- Option B: Hypotension is a *late* sign of haemorrhagic shock, occurring with >30% blood loss (Class III); compensatory vasoconstriction maintains BP initially.
- Option C: Decreased urine output (<30 mL/hr) occurs with >30% blood loss due to renal vasoconstriction; it follows tachycardia.
- Option D: Metabolic acidosis (lactic acidosis) from tissue hypoperfusion is a late manifestation, occurring after sustained hypoperfusion in Classes III–IV shock.

Final Answer: Earliest compensatory response to haemorrhage $\Rightarrow$ Tachycardia $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q11

Q12.

Solution

Concept – Prostate-Specific Antigen (PSA): PSA is a serine protease produced by prostatic epithelium. It is an **organ-specific** tumour marker (specific to prostate tissue) but **not cancer-specific** – PSA can be elevated in BPH, prostatitis, and prostate cancer. It is the most clinically useful screening and monitoring marker for prostate carcinoma.

Key Point: PSA elevation in the context of lower urinary tract symptoms and back pain should raise suspicion for prostate carcinoma. PSA >10 ng/mL is highly suggestive of prostate cancer, though biopsy confirmation is required. It is organ-specific (prostate) but not cancer-specific.

Why other options are wrong:

- Option A: CEA is a marker for colorectal, gastric, lung, and breast cancers – not specific to prostate. It is also non-specific and non-organ-specific.
- Option B: AFP is elevated in hepatocellular carcinoma, germ cell tumours (especially yolk sac tumour), and normal pregnancy; not prostate.
- Option D: CA-125 is a marker for epithelial ovarian carcinoma; not used for prostate cancer.

Final Answer: Organ-specific prostate marker elevated in prostate carcinoma $\Rightarrow$ PSA $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q12

Q13.

Solution

Concept – Routes of Tumour Spread: Carcinomas (epithelial malignancies) most commonly spread via the **lymphatic route** initially, whereas sarcomas tend to spread haematogenously. Breast carcinoma is a classic example of lymphatic spread – it first metastasises to regional lymph nodes (axillary nodes for the lateral breast), before distant haematogenous spread.

Key Point: Invasive breast carcinoma characteristically spreads first to the *ipsilateral axillary lymph nodes* via lymphatic channels. Axillary nodal status is the single most important prognostic factor in breast cancer. The sentinel node concept (first node in lymphatic drainage) is based on this predictable lymphatic spread pattern.

Why other options are wrong:

- Option A: Haematogenous spread is the primary route for breast cancer *distant* metastases (bone, lung, liver, brain), but regional nodal spread is lymphatic.
- Option C: Transcoelomic spread is characteristic of ovarian carcinoma and mesothelioma (seeding peritoneal/pleural surfaces); not the primary route for breast cancer.
- Option D: Direct extension may occur locally into chest wall or skin, but is not the primary explanation for axillary lymph node involvement.

Final Answer: Axillary nodal metastases in breast carcinoma $\Rightarrow$ Lymphatic spread $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – Carcinoid Syndrome: Carcinoid tumours arise from enterochromaffin cells (APUDoma) most commonly in the ileum. When they metastasise to the liver, hepatic degradation of secreted amines is bypassed, leading to **carcinoid syndrome**: flushing, diarrhoea, bronchospasm, and right-sided cardiac lesions. The elevated urinary 5-HIAA (5-hydroxyindoleacetic acid) is the diagnostic hallmark.

Key Point: Serotonin (5-hydroxytryptamine) is the primary mediator responsible for the flushing and watery diarrhoea of carcinoid syndrome. Serotonin stimulates intestinal motility (diarrhoea) and causes vasoactive effects (flushing). Urinary 5-

HIAA is a serotonin metabolite and the key diagnostic marker.

Why other options are wrong:

- Option A: Gastrin excess causes the Zollinger-Ellison syndrome (peptic ulcers, hypersecretion of gastric acid), not flushing and diarrhoea with elevated 5-HIAA.
- Option B: Histamine causes flushing in some atypical carcinoid tumours (gastric carcinoids), but is *not* the primary mediator in midgut carcinoids with elevated 5-HIAA.
- Option C: VIP causes the Verner-Morrison syndrome (watery diarrhoea, hypokalaemia, achlorhydria – WDHA); VIP-omas do not cause flushing and 5-HIAA is not elevated.

Final Answer: Flushing + diarrhoea + elevated 5-HIAA in carcinoid syndrome $\Rightarrow$ Serotonin $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q14

Q15.

Solution

Concept – Reed-Sternberg Cell: The **Reed-Sternberg (RS) cell** is the pathognomonic neoplastic cell of **Hodgkin lymphoma**. It is a large binucleated or bilobed cell with prominent eosinophilic "owl-eye" nucleoli. RS cells are CD15+ and CD30+ (in classical Hodgkin lymphoma) and arise from germinal centre B cells. Their presence in an appropriate reactive background is required for diagnosis.

Key Point: The owl-eye nucleoli in a binucleated large cell, surrounded by a reactive mixed infiltrate of lymphocytes, eosinophils, and plasma cells, is pathognomonic of Hodgkin lymphoma. No other lymphoma has this specific cell type and background composition.

Why other options are wrong:

- Option B: DLBCL shows sheets of large B cells with round/irregular nuclei; no RS cells or reactive background.
- Option C: Burkitt lymphoma shows a "starry sky" pattern (tingible body macrophages among monomorphous intermediate-sized cells) with t(8;14) MYC translocation.
- Option D: Anaplastic large cell lymphoma has large cells with horseshoe-shaped nuclei (hallmark cells), CD30+, ALK+; not the classic binucleated

owl-eye cells.

Final Answer: Owl-eye binucleated cells with reactive background $\Rightarrow$ Hodgkin lymphoma $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q15

Q16.

Solution

Concept – Dysplasia: Dysplasia is disordered cellular growth characterised by cellular atypia (nuclear pleomorphism, increased N:C ratio, abnormal mitoses, loss of polarity) that is **reversible** and **confined within the basement membrane**. It is considered a preneoplastic (premalignant) condition. When the entire epithelial thickness is involved, it is termed carcinoma in situ (CIS).

Key Point: The biopsy shows atypia in the lower two-thirds of the epithelium with an *intact basement membrane* – this defines moderate dysplasia (CIN 2 in the cervical context). It is reversible and has not yet become invasive carcinoma. The distinction from carcinoma in situ is that CIS involves the full thickness.

Why other options are wrong:

- Option A: Metaplasia is the replacement of one mature cell type by another mature cell type (e.g., Barrett's oesophagus); it does not show cellular atypia or abnormal mitoses.
- Option B: Anaplasia refers to extreme pleomorphism and loss of differentiation, typical of high-grade malignancies; it is not a specific premalignant lesion confined to epithelium.
- Option D: Carcinoma in situ involves the *full thickness* of the epithelium with severe atypia but intact basement membrane; the biopsy here shows only the lower two-thirds.

Final Answer: Atypia in lower 2/3 of epithelium, intact BM, reversible $\Rightarrow$ Dysplasia $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q16

Q17.

Solution

Concept – Iron Deficiency Anaemia: Iron deficiency is the most common cause of anaemia worldwide. It produces a **microcytic (low MCV), hypochromic (low MCH)** anaemia. Laboratory findings: low serum ferritin (most sensitive marker of depletion), high TIBC (transferrin unsaturated due to low iron), low serum iron, low transferrin saturation.

Key Point: The triad of microcytic hypochromic anaemia + low ferritin + high TIBC is diagnostic of iron deficiency anaemia. Ferritin is an acute phase protein, so it may be falsely normal in concurrent inflammation; TIBC rising reflects increased transferrin synthesis as the body tries to capture more iron.

Why other options are wrong:

- Option A: Anaemia of chronic disease shows microcytic or normocytic anaemia but with *high* ferritin (iron is sequestered) and *low* TIBC – the opposite pattern.
- Option C: Beta-thalassaemia trait shows microcytic hypochromic anaemia but with *normal or elevated* ferritin, normal TIBC, and increased HbA2 on electrophoresis.
- Option D: Sideroblastic anaemia shows hypochromic cells with normal or elevated serum iron and ferritin, and ring sideroblasts on bone marrow biopsy.

Final Answer: Microcytic hypochromic + low ferritin + high TIBC $\Rightarrow$ Iron deficiency anaemia $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q17

Q18.

Solution

Concept – CML and Philadelphia Chromosome: Chronic Myeloid Leukaemia (CML) is caused by the **Philadelphia chromosome** – a reciprocal translocation between chromosomes 9 and 22, written as **t(9;22)(q34;q11)**. This creates the BCR-ABL fusion gene on chromosome 22 (the small "Philadelphia" chromosome), encoding a constitutively active tyrosine kinase that drives uncontrolled myeloid proliferation.

Key Point: t(9;22) is the most important chromosomal abnormality in haematological malignancy. BCR-ABL fusion protein is the target of imatinib (Gleevec), a breakthrough in targeted cancer therapy. CML presents with massive

splenomegaly, markedly elevated WBC with the full myeloid spectrum, and basophilia.

Why other options are wrong:

- Option A: t(15;17) – PML-RARA fusion – is the hallmark of Acute Promyelocytic Leukaemia (APL/AML M3), sensitive to all-trans retinoic acid (ATRA).
- Option B: t(8;14) – MYC-IGH fusion – is the hallmark of Burkitt lymphoma (and some aggressive B-cell lymphomas).
- Option C: t(14;18) – BCL2-IGH fusion – is the hallmark of follicular lymphoma; BCL2 overexpression prevents apoptosis.

Final Answer: BCR-ABL, CML, constitutive tyrosine kinase $\Rightarrow$ t(9;22) $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q18

Q19.

Solution

Concept – Immune Thrombocytopenic Purpura (ITP): ITP is an acquired autoimmune condition where anti-platelet IgG antibodies (targeting GPIIb/IIIa or GPIb/IX on platelet surface) cause splenic destruction of antibody-coated platelets. It presents with isolated thrombocytopenia (low platelets, normal PT/aPTT, normal Hb/WBC). Bone marrow shows increased megakaryocytes (compensatory response).

Key Point: The classic ITP pattern: isolated low platelets + normal coagulation studies (PT/aPTT) + increased bone marrow megakaryocytes + anti-platelet antibodies. The normal PT and aPTT rule out DIC and other coagulation factor deficiencies. Megakaryocyte increase indicates adequate platelet production being destroyed peripherally.

Why other options are wrong:

- Option B: TTP shows a pentad: thrombocytopenia + microangiopathic haemolytic anaemia (schistocytes) + neurological symptoms + fever + renal failure; ADAMTS13 deficiency.
- Option C: DIC shows thrombocytopenia *with* prolonged PT, aPTT, low fibrinogen, and elevated D-dimers – coagulation studies are abnormal.
- Option D: Aplastic anaemia shows pancytopenia (low RBC, WBC, AND platelets) with a hypocellular bone marrow; not isolated thrombocytopenia with increased megakaryocytes.

Final Answer: Anti-platelet antibodies + isolated thrombocytopenia + increased megakaryocytes $\Rightarrow$ ITP $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q19

Q20.

Solution

Concept – Haemophilia A (Factor VIII Deficiency): Haemophilia A is an X-linked recessive disorder caused by deficiency of coagulation **Factor VIII**. It presents with spontaneous haemarthroses, muscle haematomas, and prolonged bleeding after minor trauma. Laboratory: *prolonged aPTT* (intrinsic pathway) with *normal PT* (extrinsic pathway intact).

Key Point: Factor VIII is a component of the intrinsic pathway (Factor VIIIa is a cofactor for Factor IXa in the tenase complex). Its deficiency prolongs aPTT while PT remains normal. Haemophilia A is X-linked (maternal uncle affected – classic pedigree), accounting for 80% of haemophilia cases.

Why other options are wrong:

- Option A: Factor IX deficiency causes Haemophilia B (Christmas disease) – clinically identical but less common (X-linked recessive); also prolongs aPTT only.
- Option B: Factor XI deficiency causes Haemophilia C – autosomal recessive, milder bleeding tendency; the question's X-linked pedigree (maternal uncle) points to X-linked Factor VIII or IX deficiency.
- Option D: von Willebrand factor (vWF) deficiency (von Willebrand disease) is the most common inherited bleeding disorder; it prolongs bleeding time and often aPTT (because vWF stabilises Factor VIII), but the clinical picture here of haemarthroses is more consistent with haemophilia A than vWD.

Final Answer: X-linked, haemarthroses, prolonged aPTT, normal PT $\Rightarrow$ Factor VIII $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q20

Q21.

Solution

Concept – Bence Jones Protein (Multiple Myeloma): Multiple myeloma is a plasma cell malignancy producing excess monoclonal immunoglobulin. When ex-

cess free immunoglobulin **light chains** (kappa or lambda) are produced, they appear in urine as **Bence Jones protein**. These free light chains have the unique property of precipitating at 56–60°C and redissolving at 100°C (heat test).

Key Point: Bence Jones protein = free immunoglobulin light chains in urine. They are nephrotoxic (cause cast nephropathy / "myeloma kidney") and are detected by urine protein electrophoresis. Note: Bence Jones protein is *not* detected by standard urine dipstick (which detects albumin only), requiring specific urine electrophoresis.

Why other options are wrong:

- Option A: Tamm-Horsfall protein is a normal urinary glycoprotein secreted by the thick ascending limb; it forms the matrix of urinary casts but is not related to myeloma.
- Option C: Microalbumin is a marker of glomerular damage (diabetic nephropathy, early renal disease); it is albumin in small quantities, not immunoglobulin light chains.
- Option D: Beta-2 microglobulin is a prognostic marker in myeloma (higher levels = worse prognosis) and is found in serum/urine, but it is the light chain of MHC class I, not the Bence Jones protein.

Final Answer: Free light chains in urine, precipitate at 60°C, redissolve at 100°C $\Rightarrow$ Bence Jones protein $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q21

Q22.

Solution

Concept – Sickle Cell Disease (HbS Polymerisation): Sickle cell disease is caused by a point mutation in the beta-globin gene (Glu→Val at position 6), producing haemoglobin S (HbS). In the **deoxygenated state**, HbS polymerises into long rigid fibres that distort the RBC into the characteristic sickle (crescent) shape, causing vaso-occlusion, haemolysis, and organ damage.

Key Point: HbS polymerisation is triggered by *deoxygenation* (low O_2 tension, cold, acidosis, or dehydration). The polymerised HbS forms tactoids (elongated fibres) that stiffen and deform the RBC membrane into the sickle shape. Re-oxygenation can reverse sickling initially, but repeated episodes lead to irreversibly sickled cells.

Why other options are wrong:

- Option A: Warm autoimmune haemolytic anaemia involves IgG antibody-mediated extravascular haemolysis (Coombs positive); RBCs are spherocytic, not sickled.
- Option B: Hereditary spherocytosis involves defective spectrin causing spherocytes, not sickle cells; the defect is in the RBC membrane skeleton, not haemoglobin.
- Option C: G6PD deficiency causes episodic oxidative haemolysis (triggered by drugs, infections, fava beans) with bite cells and Heinz bodies; not sickle cells.

Final Answer: HbS crescent-shaped RBCs in low O_2 $\Rightarrow$ HbS polymerisation in deoxygenated state $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q22

Q23.

Solution

Concept – Primary Tuberculosis Complex: In primary TB, the organism implants at a peripheral subpleural site (usually right lower lobe), forming a **Ghon focus** (small caseous granuloma). Lymphangitic spread to hilar/mediastinal lymph nodes produces the draining lymphadenitis. Together, the **Ghon focus + lymphangitic channel + draining lymph node** constitute the **Ghon complex** (primary complex).

Key Point: The Ghon complex represents the primary TB infection. In the majority of immunocompetent individuals, the lesion heals with *calcification* of both the parenchymal focus and the hilar lymph node, leaving a calcified scar visible on CXR. An asymptomatic calcified Ghon complex is evidence of past primary TB infection.

Why other options are wrong:

- Option B: Ranke complex is the healed/calcified Ghon complex (some use this term specifically for the calcified stage); strictly, the Ghon complex is the primary complex before or after calcification. In standard pathology, the term Ghon complex is preferred.
- Option C: Simon focus refers to apical secondary TB foci (haematogenous reactivation in the lung apex); not the primary lower lobe complex.
- Option D: Rasmussen's aneurysm is a pseudoaneurysm of a pulmonary artery wall eroded by a TB cavity, causing massive haemoptysis; not related to the primary complex.

Final Answer: Calcified lower lobe focus + calcified hilar node in asymptomatic child $\Rightarrow$ Ghon complex $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q23

Q24.

Solution

Concept – Stages of Lobar Pneumonia: Lobar pneumonia progresses through four classical morphological stages: (1) **Congestion** (day 1–2): vascular engorgement, bacteria-filled oedema fluid; (2) **Red hepatization** (day 2–4): firm, airless, red lobe with alveoli filled with RBCs and fibrin; (3) **Grey hepatization** (day 4–8): WBCs replace RBCs, grey/brown firm lobe; (4) **Resolution**: enzymatic dissolution and restoration.

Key Point: Day 3 with a *red, firm, airless* lobe and alveoli filled with *RBCs and fibrin* on microscopy is the classic description of **red hepatization**. The lobe is red because of the haemorrhagic exudate (extravasated RBCs) and firm ("liver-like" consistency = hepatization) due to fibrin consolidation.

Why other options are wrong:

- Option A: Congestion (day 1–2): the lobe is hyperaemic but not firm; alveoli contain serous fluid and few cells, not predominantly RBCs and fibrin.
- Option B: Grey hepatization (day 4–8): the lobe is grey-brown; alveoli contain predominantly neutrophils replacing the RBCs; not the picture on day 3.
- Option D: Resolution (day 8+): enzymatic digestion of exudate; lung architecture restored; no fibrin/RBC-filled alveoli.

Final Answer: Day 3, red firm lobe, alveoli full of RBCs + fibrin $\Rightarrow$ Red hepatization $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q24

Q25.

Solution

Concept – Small Cell Lung Carcinoma and SIADH: Small cell lung carcinoma (SCLC) is a high-grade neuroendocrine carcinoma strongly associated with smoking. It is the most common cause of paraneoplastic syndromes. SCLC can secrete **ectopic ADH** (antidiuretic hormone), causing the Syndrome of Inappropriate ADH

Secretion (SIADH): hyponatraemia + concentrated urine + euvolaemia/mild hypervolaemia.

Key Point: SIADH is the most common paraneoplastic syndrome of SCLC. The oat cell/small cell morphology (round-oval cells with nuclear moulding, scant cytoplasm, salt-and-pepper chromatin, high mitotic index) is pathognomonic of SCLC. Hyponatraemia with inappropriately concentrated urine in a smoker with a hilar mass = SCLC + SIADH.

Why other options are wrong:

- Option A: Cushing syndrome (ectopic ACTH) is also caused by SCLC, but is less common than SIADH in SCLC; the clinical presentation here shows hyponatraemia, not hypokalaemic alkalosis/hyperglycaemia.
- Option C: Hypercalcaemia (ectopic PTHrP) is classically caused by squamous cell carcinoma of the lung, not SCLC.
- Option D: Eaton-Lambert myasthenic syndrome (proximal muscle weakness, absent reflexes improving with repeated stimulation) is also a SCLC paraneoplastic syndrome, but the clinical features here (hyponatraemia, concentrated urine) point specifically to SIADH.

Final Answer: SCLC + hyponatraemia + concentrated urine $\Rightarrow$ SIADH $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q25

Q26.

Solution

Concept – Malignant Mesothelioma: Malignant mesothelioma is a primary tumour of the mesothelial cells lining the pleura (most common), peritoneum, or pericardium. It is **strongly associated with asbestos exposure** (especially crocidolite/blue asbestos), with a latency of 20–40 years. The pathological hallmarks are **ferruginous bodies** (asbestos fibres coated with iron-protein) in lung tissue.

Key Point: IHC positivity for *calretinin* and *WT-1* (Wilms tumour antigen) and negativity for TTF-1 and CEA distinguishes mesothelioma from pulmonary adenocarcinoma metastatic to pleura. The golden-brown beaded ferruginous bodies in lung tissue confirm asbestos exposure.

Why other options are wrong:

- Option A: Squamous cell carcinoma of the lung is associated with smoking and centrally located masses; it would be CK5/6 and p63 positive, not

calretinin/WT-1.

- Option B: Lung adenocarcinoma is TTF-1 positive and may cause pleural effusion but does not produce ferruginous bodies; it originates from lung parenchyma, not the pleural surface.
- Option C: Solitary fibrous tumour (pleural fibroma) is a benign localised pleural tumour; it is not associated with asbestos and is CD34 positive.

Final Answer: Asbestos exposure + pleural encasement + calretinin/WT-1+ + ferruginous bodies $\Rightarrow$ Malignant mesothelioma $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q26

Q27.

Solution

Concept – MI Zones (Jackson's Burn Model Applied to MI): After MI, ischaemic injury can be divided into three concentric zones: (1) **Zone of coagulation (infarct centre)**: irreversible cell death, coagulative necrosis with ghost cell outlines; (2) **Zone of injury (middle)**: cells at risk, potentially salvageable; (3) **Zone of ischaemia (outer)**: reversible ischaemia, viable cells.

Key Point: The central zone (Zone A in the diagram) undergoes **coagulative necrosis** – the defining feature of myocardial infarction. Ghost cell outlines persist because the denatured structural proteins maintain tissue architecture despite cell death, which is the hallmark of coagulative necrosis.

Why other options are wrong:

- Option B: Zone of stasis is not a standard pathological term for MI zones; stasis refers to blood flow stagnation in infarct tissue.
- Option C: Zone of hyperaemia describes the reactive peripheral zone in early inflammation around the infarct; it is reversible ischaemia, not coagulative necrosis.
- Option D: Zone of haemorrhage is seen in haemorrhagic infarcts (e.g., in reperfused infarcts or venous infarcts), not the central zone of ischaemic MI.

Final Answer: Central zone of MI with ghost cell outlines $\Rightarrow$ Zone of coagulation (infarct centre) $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q27

Q28.

Solution

Concept – Atherosclerosis and Foam Cells: The **fatty streak** is the earliest grossly visible lesion of atherosclerosis. It consists of **foam cells** – lipid-laden macrophages that have ingested oxidised LDL via scavenger receptors. Foam cells accumulate in the intima beneath the endothelium, giving the streak a yellow, flat appearance on the arterial intima.

Key Point: Foam cells are derived from circulating monocytes that migrate into the intima, differentiate into macrophages, and engulf oxidised LDL via scavenger receptors (CD36, SR-A). Oxidised LDL is the key driver: unlike native LDL, oxidised LDL is taken up unregulated (no feedback inhibition), causing foam cell formation. This is the critical initiating event in atherogenesis.

Why other options are wrong:

- Option A: Smooth muscle cells (SMCs) migrate from media to intima in later stages of atherosclerosis (fibrous plaque formation), contributing to the fibrous cap; they are not the primary cells of the earliest fatty streak.
- Option B: Lymphocytes are present in atherosclerotic plaques but are not the primary foam cell-forming cells; T cells play a modulatory role in plaque progression.
- Option D: Platelet aggregates occur on disrupted/ulcerated plaques (complicated plaques) and contribute to thrombosis; they are not the foam cells of the early fatty streak.

Final Answer: Earliest atherosclerotic lesion (fatty streak) – foam cells = lipid-laden macrophages $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q28

Q29.

Solution

Concept – Dilated Cardiomyopathy (DCM): DCM is characterised by **global four-chamber dilation** with *systolic dysfunction* (reduced EF). The ventricular walls are *thinned* (eccentric hypertrophy – increased chamber volume, normal or thin wall). Common causes include: chronic alcohol use, viral myocarditis (Coxsackie B), genetic (25% familial), peripartum, and idiopathic.

Key Point: Four-chamber dilation + severely reduced EF (25%) + normal wall thickness = dilated cardiomyopathy. Alcohol is the most common *identifiable*

cause of DCM. The normal wall thickness excludes hypertrophic cardiomyopathy; the global dilation excludes restrictive (which has small chambers with thick walls).

Why other options are wrong:

- Option A: Hypertrophic cardiomyopathy (HCM) shows *thickened* ventricular walls (especially asymmetric septal hypertrophy) with normal or *increased* EF (diastolic dysfunction); chambers are small, not dilated.
- Option C: Restrictive cardiomyopathy shows *normal or reduced* chamber size with markedly impaired diastolic filling; wall thickness may be increased (e.g., amyloidosis, sarcoidosis).
- Option D: ARVC (Arrhythmogenic Right Ventricular Cardiomyopathy) shows fibrofatty replacement of the RV myocardium with right ventricular dilation; EF may be preserved initially; not four-chamber dilation.

Final Answer: Four-chamber dilation + low EF + alcohol history $\Rightarrow$ Dilated cardiomyopathy $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q29

Q30.

Solution

Concept – *H. pylori* and Peptic Ulcer Disease: *Helicobacter pylori* is a gram-negative, urease-positive, spiral bacterium that colonises the gastric mucosa. It is the most common cause of chronic gastritis and peptic ulcer disease. *H. pylori* preferentially colonises the **antrum** of the stomach, causing antral gastritis and duodenal ulcers (most common), and body gastritis and gastric ulcers.

Key Point: *H. pylori* is found predominantly in the gastric **antrum**, where it causes antral gastritis, increases gastrin secretion (G cells in antrum), and leads to increased gastric acid output – resulting in ulcers in the duodenal bulb (most common site of peptic ulcers, accounting for 95% of peptic ulcers in *H. pylori* infection).

Why other options are wrong:

- Option A: The fundus is the dome of the stomach; *H. pylori* does not preferentially colonise the fundus (which is the site of acid-producing parietal cells and chief cells).
- Option B: The body (corpus) may be involved in pangastritis in some cases, but the primary site of *H. pylori* colonisation and antrum-type gastritis is the

antrum.

- Option C: The cardia (gastro-oesophageal junction) is less commonly colonised; cardia involvement is associated with GERD rather than peptic ulcer disease.

Final Answer: *H. pylori* colonisation site most associated with ulcer disease ⇒ Antrum / duodenal bulb ⇒ $\boxed{D}$

Answer: (D) Go Back to Q30

Q31.

Solution

Concept – Crohn's Disease (Transmural Inflammation): Crohn's disease is an idiopathic chronic granulomatous inflammatory bowel disease that can affect any part of the GI tract from mouth to anus. Its hallmark pathological features are: **transmural inflammation** (through all layers), skip lesions, cobblestone mucosa, linear fissuring ulcers, non-caseating granulomas, and fistula formation.

Key Point: Transmural inflammation is the most important distinguishing histological feature of Crohn's disease. It involves all layers (mucosa, submucosa, muscularis propria, and serosa), predisposing to fistulae, strictures, and perforations. This contrasts with ulcerative colitis which is limited to the mucosa and submucosa.

Why other options are wrong:

- Option B: Crypt abscesses with mucosal/submucosal inflammation only is the hallmark of *ulcerative colitis*, not Crohn's disease.
- Option C: Pseudomembrane formation is characteristic of *Clostridioides difficile* colitis (pseudomembranous colitis); not of Crohn's disease.
- Option D: Submucosal fibrosis alone is not the hallmark of Crohn's; fibrosis occurs in Crohn's (causing strictures) but as a consequence of transmural inflammation, not as an isolated finding.

Final Answer: Skip lesions + cobblestone + full-thickness involvement ⇒ Transmural inflammation ⇒ $\boxed{A}$

Answer: (A) Go Back to Q31

Q32.

Solution

Concept – Hepatic Cirrhosis: Cirrhosis is defined as **diffuse fibrosis with regenerative nodule formation**. The two essential components are: (1) **Regenerative nodules** – islands of hepatocytes surrounded by fibrous septa, formed by hepatocyte regeneration in response to chronic injury; and (2) **Bridging fibrous septa** – dense collagen bands connecting portal tracts to central veins.

Key Point: The green circles in the diagram represent **regenerative nodules** – the most essential diagnostic feature of cirrhosis on histology. These nodules lack normal lobular architecture and are surrounded by fibrous septa. Without regenerative nodules, the diagnosis of cirrhosis cannot be made (fibrosis alone = hepatic fibrosis, not cirrhosis).

Why other options are wrong:

- Option A: Central vein dilation is seen in right heart failure (congestive hepatopathy); it is not a feature of cirrhosis per se.
- Option B: Fatty change (steatosis) is an early feature of alcoholic liver disease and NAFLD; it is not the hallmark of cirrhosis and may regress with abstinence.
- Option D: Bridging fibrosis without nodule formation is *pre-cirrhotic* hepatic fibrosis (e.g., METAVIR stage F3); cirrhosis requires BOTH fibrosis AND nodule formation.

Final Answer: Fibrous septa + hepatocyte islands = regenerative nodules = hallmark of cirrhosis $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q32

Q33.

Solution

Concept – Portal Hypertension and Oesophageal Varices: Portal hypertension (portal pressure > 12 mmHg) causes dilation of portosystemic collaterals at sites where portal and systemic venous systems communicate. The most **clinically dangerous** collateral is the **oesophageal varices** (gastro-oesophageal junction), because their rupture causes life-threatening upper GI haemorrhage.

Key Point: Oesophageal varices arise at the gastro-oesophageal junction where the left gastric vein (portal) anastomoses with the oesophageal veins (systemic via azygous). Their rupture causes massive haematemesis (vomiting bright red

blood), which is the most common cause of death in cirrhotic patients. Urgent endoscopic banding or sclerotherapy is the treatment.

Why other options are wrong:

- Option A: Caput medusae refers to dilated periumbilical veins (portal $\leftrightarrow$ systemic via re-canalised umbilical vein); visible on the abdominal wall but does not cause haematemesis.
- Option C: Haemorrhoids (anorectal varices) form at the ano-rectal junction (superior rectal [portal] $\leftrightarrow$ middle/inferior rectal [systemic]); they cause rectal bleeding, not haematemesis.
- Option D: Splenomegaly is a consequence of portal hypertension (congestive splenomegaly), not a specific collateral channel; it does not directly cause upper GI bleeding.

Final Answer: Haematemesis in cirrhosis due to portosystemic collaterals $\Rightarrow$ Oesophageal varices $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q33

Q34.

Solution

Concept – Minimal Change Disease (Nephrotic Syndrome): Minimal Change Disease (MCD) is the most common cause of nephrotic syndrome in children. The name reflects the **normal appearance on light microscopy and immunofluorescence**. The only abnormality is seen on **electron microscopy**: **diffuse effacement (fusion) of podocyte foot processes**. This causes loss of the glomerular filtration barrier's charge selectivity, resulting in massive proteinuria.

Key Point: Podocyte foot process effacement on EM is the *ultrastructural hallmark* of MCD. It results from cytoskeletal disruption of podocytes (possibly due to a circulating T-cell factor or overexpression of CD80). The effaced foot processes lose the slit diaphragm, allowing albumin to pass freely into the filtrate.

Why other options are wrong:

- Option A: Sub-endothelial electron-dense deposits are seen in Class III/IV lupus nephritis and membranoproliferative GN (type I); not MCD.
- Option B: Subepithelial "spike and dome" pattern (epimembranous deposits) is characteristic of membranous nephropathy (most common cause of nephrotic syndrome in adults); not MCD.
- Option C: Mesangial IgA deposits are characteristic of IgA nephropathy

(Berger's disease), which presents with haematuria, not pure nephrotic syndrome.

Final Answer: Normal LM + negative IF + podocyte foot process effacement on EM $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q34

Q35.

Solution

Concept – Wilms Tumour (Nephroblastoma): Wilms tumour is the **most common primary renal tumour in children** (peak age 3–4 years). It presents as a large flank mass, usually unilateral and intrarenal. Histology is **triphasic**: blastema (small blue primitive cells), stroma (mesenchymal), and epithelial (primitive tubules/glomeruli). It is associated with WT1 gene mutation (chromosome 11p13) and WAGR syndrome.

Key Point: The triphasic histology (blastemal + stromal + epithelial) in a 3-year-old with an intrarenal mass is pathognomonic of Wilms tumour. Unlike neuroblastoma, Wilms tumour is *intrarenal* (distorts the kidney) and does not cross the midline. Prognosis is excellent with surgery + chemotherapy (80–90% cure rate).

Why other options are wrong:

- Option B: Neuroblastoma originates from the adrenal medulla or sympathetic chain; it is *extrarenal*, may cross the midline (wraps around vessels), and shows rosette formation (Homer-Wright) on histology; often presents with urinary catecholamines.
- Option C: Renal cell carcinoma (RCC) is rare in children; it arises from tubular epithelium and does not show the triphasic pattern; it is predominantly a disease of adults (50–60 years).
- Option D: Clear cell sarcoma of the kidney is a rare, aggressive childhood renal tumour known for bone metastases; it does not show the triphasic pattern of Wilms tumour.

Final Answer: Triphasic intrarenal mass in a 3-year-old $\Rightarrow$ Wilms tumour (nephroblastoma) $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q35

Q36.

Solution

Concept – Rapidly Progressive GN (RPGN) and Crescent Formation: RPGN presents as rapid deterioration of renal function (days to weeks) with haematuria and RBC casts. The hallmark histological finding is **crescent formation** in Bowman's space, caused by **proliferation of parietal epithelial cells** (PECs) lining Bowman's capsule, along with infiltrating macrophages, in response to fibrin leakage through the damaged GBM.

Key Point: Crescents are formed by *parietal epithelial cells* of Bowman's capsule (not visceral/podocytes or endothelial cells). Fibrin leaking from damaged glomerular capillaries through the GBM acts as the scaffold for PEC proliferation, creating a crescent-shaped mass that compresses the glomerular tuft and rapidly obliterates filtration capacity.

Why other options are wrong:

- Option A: Mesangial cells proliferate in IgA nephropathy, lupus nephritis class II, and MPGN, causing mesangial hypercellularity – not crescent formation.
- Option B: Endothelial cell proliferation (along with mesangial cells) occurs in diffuse proliferative GN (lupus class IV, MPGN); endothelial proliferation does not form the Bowman's space crescent.
- Option D: Visceral epithelial cells (podocytes) undergo foot process effacement in minimal change disease; podocyte injury is the initiating event in RPGN (GBM breach), but the crescent itself is formed by *parietal* cells.

Final Answer: Crescent in Bowman's space formed by proliferating parietal epithelial cells $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q36

Q37.

Solution

Concept – Hashimoto's Thyroiditis: Hashimoto's thyroiditis is the most common cause of hypothyroidism in iodine-sufficient regions. It is an **autoimmune** condition characterised by lymphocytic infiltration of the thyroid with germinal centre formation, Hurthle (oxyphilic) cell metaplasia of follicular epithelium, and progressive gland destruction. It is associated with **anti-TPO antibodies** (most sensitive) and **anti-thyroglobulin antibodies**.

Key Point: Anti-thyroid peroxidase (anti-TPO) antibodies are the most sensitive and specific marker for Hashimoto's thyroiditis, present in >95% of cases. They target thyroid peroxidase, an enzyme essential for thyroid hormone synthesis. They are also found in Graves' disease but at lower titres; the key distinction is that Hashimoto's causes hypothyroidism, Graves' causes hyperthyroidism.

Why other options are wrong:

- Option A: Anti-TSH receptor *stimulating* antibodies (TSAb/TRAb) are the pathological antibodies in **Graves' disease** (hyperthyroidism), not Hashimoto's; they mimic TSH and constitutively activate the TSH receptor.
- Option C: Anti-double-stranded DNA (anti-dsDNA) antibodies are specific to **systemic lupus erythematosus (SLE)**; they are not found in thyroid autoimmune diseases.
- Option D: Anti-microsomal antibodies are essentially the same as anti-TPO antibodies (the microsomal antigen is thyroid peroxidase); this option is misleading. The correct and specific answer is anti-TPO antibodies.

Final Answer: Hashimoto's thyroiditis autoantibody marker $\Rightarrow$ Anti-TPO antibodies $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q37

Q38.

Solution

Concept – Cushing Syndrome: Cushing syndrome is the clinical syndrome resulting from **chronic exposure to excess glucocorticoids (cortisol)**. Causes include: (1) Exogenous corticosteroid administration (most common), (2) ACTH-secreting pituitary adenoma (Cushing's *disease*), (3) Ectopic ACTH secretion (SCLC), and (4) Adrenal cortical adenoma/carcinoma (ACTH-independent). All share the common endpoint of excess cortisol.

Key Point: The pathophysiological mechanism in ALL causes of Cushing syndrome is **excess cortisol production** (or exogenous administration). Cortisol excess causes fat redistribution (central obesity, buffalo hump, moon face), protein catabolism (striae, muscle wasting), hyperglycaemia, hypertension, immunosuppression, and osteoporosis.

Why other options are wrong:

- Option A: Excess mineralocorticoid (aldosterone) causes Conn syndrome (primary hyperaldosteronism): hypertension + hypokalaemia + metabolic

alkalosis; no fat redistribution or striae.

- Option B: Excess catecholamines (adrenaline/noradrenaline) from a phaeochromocytoma cause episodic hypertension, palpitations, sweating, and headache – not central obesity or striae.
- Option C: Excess GH and IGF-1 cause acromegaly (adults) or gigantism (children): coarse features, large hands/feet, prognathism, organomegaly – not the cushingoid phenotype.

Final Answer: Buffalo hump + central obesity + striae + hypertension + elevated cortisol $\Rightarrow$ Excess cortisol production $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q38

Q39.

Solution

Concept – Clark's Levels of Melanoma Invasion: Clark's levels describe the anatomical depth of melanoma invasion:

- Level I: confined to epidermis (in situ)
- Level II: invades papillary dermis
- Level III: fills papillary dermis to reticular interface
- Level IV: invades reticular dermis
- **Level V: invades subcutaneous fat** (worst prognosis)

Key Point: Clark level V, with invasion into the subcutaneous fat, carries the **worst prognosis** among Clark's levels. It represents the deepest invasion, correlating with high risk of metastasis. Note: In current practice, Breslow thickness (mm) has largely replaced Clark's level as the primary prognostic indicator, but Clark's levels remain exam-relevant.

Why other options are wrong:

- Option B: Clark level I (in situ) is confined to the epidermis with no dermal invasion; it is the earliest and carries the *best* prognosis.
- Option C: Clark level III fills the papillary dermis (expanding to but not into the reticular dermis); intermediate prognosis.
- Option D: Clark level II invades the papillary dermis superficially; better prognosis than levels III–V.

Final Answer: Invasion into subcutaneous fat = Clark level V = deepest, worst prognosis $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q39

Q40.

Solution

Concept – Keloid: A **keloid** is an exuberant, tumour-like fibrous proliferation at a wound site that **extends beyond the original wound margins** and does not regress spontaneously. It is more common in individuals with darker skin pigmentation and has a genetic predisposition. Histologically, it contains thick, haphazardly arranged "glassy" collagen bundles (type I and III) extending into the surrounding dermis.

Key Point: The defining feature of a keloid (differentiating it from a hypertrophic scar) is that it **extends beyond the original wound margin** and does not regress. Hypertrophic scars remain within the wound boundary and may regress over time. Both involve excess collagen deposition, but keloids show more extensive, uncontrolled fibroblast activity.

Why other options are wrong:

- Option A: A hypertrophic scar also shows raised, firm scar tissue with excess collagen, but it *remains within* the original wound margins and tends to regress over months to years – distinguishing it from a keloid.
- Option B: A desmoid tumour is a deep fibrous tissue proliferation (fibromatosis) arising from musculoaponeurotic structures; it is locally aggressive and not related to superficial wound healing.
- Option D: A dermatofibroma is a benign dermal fibrohistiocytic lesion; it is typically small, firm, and dimples when pinched – not an exuberant post-wound scar beyond wound margins.

Final Answer: Exuberant fibrous scar extending beyond wound margins, no regression $\Rightarrow$ Keloid $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q40

Answer Key – FMGE Pathology Sample Paper 1

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