FMGE Pathology Sample Paper – 3

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 35-year-old immigrant presents with low-grade fever, night sweats, and weight loss for 3 months. Chest X-ray shows a right upper lobe cavitary lesion. Sputum AFB smear is positive. Biopsy of a mediastinal lymph node is taken. Which type of necrosis is characteristically found in this condition, and what are its microscopic features?

Type A

Caseous Necrosis

- Amorphous, eosinophilic
- "Cheese-like" gross appearance
- No tissue architecture
- Surrounded by granuloma
- Classic: tuberculosis

Type B

Coagulative Necrosis

- Cell outlines preserved
- "Ghost" cells on H&E
- Ischaemic infarcts (most organs)
- Firm texture
- Classic: myocardial infarct

(A) Coagulative necrosis – ghost cell outlines preserved

(B) Caseous necrosis – amorphous eosinophilic material, no tissue architecture

(C) Liquefactive necrosis – pus formation with neutrophils

(D) Fat necrosis – saponification with calcium deposits

Q2. A pathologist is reviewing a tissue section from a tumour treated with chemotherapy. She identifies cells with condensed chromatin, nuclear fragmentation, and membrane-bound apoptotic bodies without surrounding inflammation. Which protein is the key "executioner" in the final common pathway of intrinsic apoptosis responsible for this morphology?

(A) Bcl-2 (anti-apoptotic regulator)

(B) Cytochrome c (released from mitochondria)

(C) Apaf-1 (apoptosome scaffold protein)

(D) Caspase-3 (executioner caspase)

Q3. A medical student compares necrosis and apoptosis in a pathology practical. Which of the following statements best distinguishes necrosis from apoptosis regarding their inflammatory consequences?

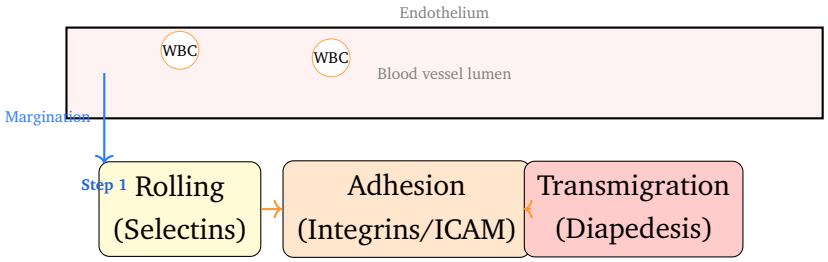
(A) Necrosis triggers inflammation; apoptosis does not

(B) Apoptosis triggers inflammation; necrosis does not

(C) Both necrosis and apoptosis trigger equal inflammation

(D) Neither necrosis nor apoptosis triggers inflammation

Q4. During acute inflammation following a bee sting, leukocytes must exit the blood vessel to reach the site of injury. The sequential steps of leukocyte extravasation are shown below. What is the FIRST step in this process?



(A) Transmigration (diapedesis) through the vessel wall

(B) Firm adhesion via integrin-ICAM-1 interaction

(C) Margination and rolling along the endothelium

(D) Chemotaxis toward the site of injury

Q5. A 68-year-old woman develops profuse watery diarrhoea 10 days after completing a course of clindamycin for a dental infection. Sigmoidoscopy reveals yellow-white plaques adherent to the colonic mucosa. Stool toxin assay is positive. Which organism is responsible?

(A) Staphylococcus aureus

(B) Clostridium difficile

(C) Campylobacter jejuni

(D) Escherichia coli O157:H7

Q6. A 32-year-old African-American woman presents with bilateral hilar lymphadenopathy on chest X-ray, erythema nodosum, and elevated serum ACE level. Transbronchial biopsy shows compact collections of epithelioid macrophages and multinucleated giant cells without central necrosis. What is the characteristic pathological finding in this disease?

(A) Caseating granuloma with central necrosis

(B) Suppurative granuloma with neutrophil infiltrate

(C) Foreign body granuloma with Touton giant cells

(D) Non-caseating epithelioid granuloma

Q7. A large infected wound on the lower limb is left open to heal by secondary intention. Weeks later, the wound contracts significantly in size before re-epithelialisation is complete. Which cell type is primarily responsible for wound contraction in this process?

(A) Myofibroblast (alpha-smooth muscle actin positive)

(B) Type I collagen-secreting fibroblast

(C) Neutrophil (early debridement phase)

(D) Macrophage (orchestrating growth factor release)

Q8. During an acute bacterial infection, a patient develops fever and pain at the site of inflammation. Arachidonic acid is metabolised by cyclooxygenase (COX) enzymes to produce a mediator that acts on the hypothalamus to reset the temperature set-point and sensitises peripheral pain receptors. Which mediator is primarily responsible?

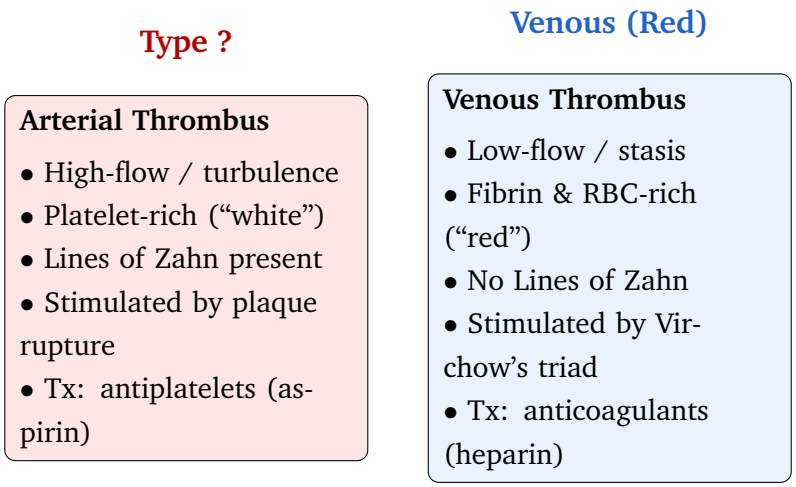
(A) Leukotriene B4 (LTB4)

(B) Thromboxane A2 (TXA2)

(C) Prostaglandin E2 (PGE2)

(D) Platelet-activating factor (PAF)

Q9. A 58-year-old man with hypertension and hypercholesterolaemia suffers an acute myocardial infarction. Coronary angiography reveals a thrombotic occlusion over a ruptured atherosclerotic plaque in the left anterior descending artery. The diagram below contrasts arterial and venous thrombi. What type of thrombus characteristically forms in arteries?



(A) Fibrin-rich (red) thrombus with many trapped erythrocytes

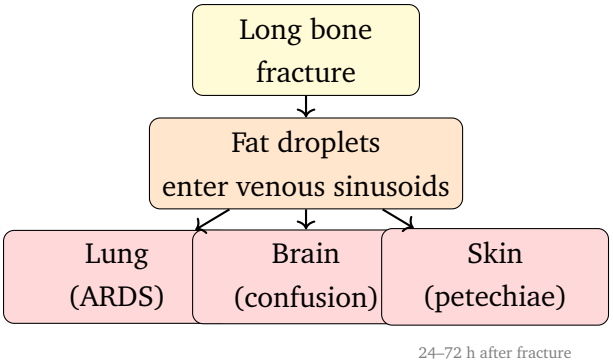
(B) Platelet-rich (white) thrombus with lines of Zahn

(C) Mixed thrombus with equal platelets and fibrin

(D) Hyaline thrombus composed of fibrin in microvasculature

Q10. A 22-year-old motorcyclist sustains fractures of the femur and tibia. Twenty-four hours later, he develops sudden-onset dyspnoea, confusion, and petechiae over the chest and axillae. Blood gas shows PaO2 of 55 mmHg.

Urinalysis shows fat globules. The diagram below illustrates the pathogenesis. What is the most likely diagnosis?



(A) Pulmonary thromboembolism from deep vein thrombosis

(B) Air embolism following central line insertion

(C) Amniotic fluid embolism

(D) Fat embolism syndrome

Q11. A 70-year-old man with a urinary catheter develops high fever (39.8°C), warm flushed skin, bounding pulse, blood pressure of 80/50 mmHg, and elevated serum lactate. Blood cultures grow gram-negative rods. Lipopolysaccharide (LPS) from the bacterial cell wall triggers massive release of TNF-α and IL-1. What type of shock does this represent?

(A) Septic shock (distributive)

(B) Hypovolaemic shock (haemorrhagic)

(C) Cardiogenic shock (pump failure)

(D) Obstructive shock (tension pneumothorax)

Q12. A 55-year-old man with known hepatitis B cirrhosis is found on surveillance ultrasound to have a 4 cm hepatic mass. Serum tumour marker is significantly elevated. Which tumour marker is characteristically raised in hepatocellular carcinoma AND in yolk sac tumours (endodermal sinus tumour)?

(A) Carcinoembryonic antigen (CEA)

(B) CA 19-9 (pancreatic/biliary marker)

(C) Alpha-fetoprotein (AFP)

(D) CA-125 (ovarian epithelial marker)

Q13. A 45-year-old woman has a slowly growing mass in the parotid gland with facial pain radiating along the jaw. Imaging shows tumour extending along the facial nerve sheath. Biopsy reveals a tumour with cribriform (Swiss-cheese) pattern of basaloid cells. What is the characteristic route of spread of this tumour?

(A) Haematogenous spread to lungs

(B) Perineural spread (perineural invasion)

(C) Lymphatic spread to cervical nodes

(D) Direct invasion through the parotid capsule

Q14. A 50-year-old woman presents with profuse watery diarrhoea (up to 5 L/day), severe hypokalaemia (K^+ 2.1 mEq/L), and achlorhydria (no gastric acid). CT abdomen reveals a 3 cm tumour in the tail of the pancreas. Which tumour is responsible for this clinical syndrome?

(A) Insulinoma (secreting insulin)

(B) Glucagonoma (secreting glucagon)

(C) Gastrinoma (Zollinger-Ellison syndrome)

(D) VIPoma (WDHA – Watery Diarrhoea, Hypokalaemia, Achlorhydria)

Q15. A 19-year-old man presents with painless cervical lymphadenopathy and systemic B symptoms. Lymph node biopsy shows large pleomorphic lymphoid cells with horseshoe-shaped nuclei (hallmark cells) that are CD30-positive and ALK-positive. Which genetic abnormality is characteristically associated with this lymphoma?

(A) ALK gene rearrangement [t(2;5)(p23;q35)]

(B) BCL-2 rearrangement [t(14;18)]

(C) BCL-6 rearrangement [t(3;14)]

(D) c-MYC translocation [t(8;14)]

Q16. A 48-year-old woman has microcalcifications detected on routine mammography. Stereotactic core biopsy is performed. Histology shows malignant epithelial cells filling and distending ducts with central necrosis and granular calcification; the basement membrane is intact. No stromal invasion is identified. What is the diagnosis?

(A) Invasive ductal carcinoma (IDC)

(B) Lobular carcinoma in situ (LCIS)

(C) Ductal carcinoma in situ (DCIS), comedo subtype

(D) Phyllodes tumour of the breast

Q17. A 25-year-old man presents with progressive fatigue, frequent infections, and spontaneous bruising. CBC shows pancytopenia: Hb 6.2 g/dL, WBC 1.8×10^9/L, Platelets 22×10^9/L. Bone marrow biopsy shows markedly reduced cellularity with replacement by fat cells. Reticulocyte count is very low. What is the characteristic bone marrow finding in aplastic anaemia?

(A) Hypercellular marrow with blast cells $>$20%

(B) Hypocellular marrow with fat cell replacement

(C) Marrow packed with Reed-Sternberg cells

(D) Marrow with "wrinkled tissue paper" macrophages

Q18. A 72-year-old man is incidentally found to have a WBC of 85×10^9/L on a routine blood test. He has no symptoms. Peripheral blood smear shows a monotonous population of small mature-appearing lymphocytes with scant cytoplasm and clumped chromatin. Numerous disrupted cells are noted. Flow cytometry shows CD5+, CD19+, CD23+, surface Ig dim. What are the disrupted cells characteristically called?

(A) Auer rods in blast cells

(B) Reed-Sternberg cells (owl-eye nuclei)

(C) Flame cells (IgA myeloma)

(D) Smudge cells (basket cells)

Q19. A 62-year-old man with metastatic pancreatic cancer develops migratory superficial thrombophlebitis (Trousseau sign), then DIC. Laboratory shows prolonged PT/PTT, low fibrinogen, elevated D-dimer, and microangiopathic haemolytic anaemia. Which property of the tumour triggers this hypercoagulable state?

(A) Mucin secretion activating coagulation (Trousseau syndrome)

(B) Tumour neovascularisation releasing tissue plasminogen activator

(C) Bone marrow infiltration reducing platelet production

(D) Liver metastases reducing coagulation factor synthesis

Q20. A 32-year-old woman develops a deep vein thrombosis during her second trimester of pregnancy. She has a family history of thrombosis. Laboratory testing shows that her plasma does not prolong the APTT when activated protein C (APC) is added (APC resistance). What is the most common inherited thrombophilia responsible?

(A) Protein C deficiency

(B) Antithrombin III deficiency

(C) Factor V Leiden mutation (APC resistance)

(D) Prothrombin G20210A mutation

Q21. A 55-year-old man presents with massive splenomegaly, pancytopenia, and recurrent infections. Bone marrow trephine shows a hypercellular marrow with cells having abundant pale cytoplasm and “fried egg” appearance with prominent hairy cytoplasmic projections. A specific cytochemical stain is strongly positive even after tartrate treatment. Which finding is diagnostic?

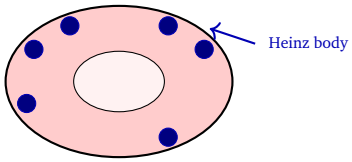
(A) PAS (periodic acid-Schiff) positivity in blasts

(B) TRAP (tartrate-resistant acid phosphatase) positivity

(C) Sudan black B positivity

(D) Myeloperoxidase positivity

Q22. A 28-year-old man of African descent is started on primaquine for malaria prophylaxis. Three days later, he develops jaundice, dark urine, and pallor. Blood film with supravital stain (brilliant cresyl blue) shows dark inclusions within red blood cells near the periphery of the cell membrane, as shown below. What are these inclusions and what causes them?



RBC with peripheral dark inclusions
(supravital stain – methylene blue)

(A) Howell-Jolly bodies – DNA remnants from hyposplenism

(B) Basophilic stippling – lead poisoning/thalassaemia

(C) Pappenheimer bodies – iron-containing granules (sideroblastic)

(D) Heinz bodies – denatured haemoglobin from oxidative stress (G6PD deficiency)

Q23. A 45-year-old immunocompromised patient develops high fever, progressive dyspnoea, and a chest X-ray showing numerous 1–2 mm nodular opacities distributed uniformly throughout both lung fields. Sputum culture is negative but bronchoalveolar lavage grows acid-fast bacilli after 6 weeks. What is the pathogenesis of this form of tuberculosis?

(A) Haematogenous dissemination producing millet-seed (miliary) lesions throughout the lungs and other organs

(B) Direct lymphatic spread from a primary Ghon focus to hilar nodes

(C) Reactivation of latent TB confined to the apical lung segment

(D) Aspiration of infected secretions causing lobar consolidation

Q24. A 60-year-old woman presents with a 6-week history of dry cough and breathlessness. HRCT shows bilateral patchy consolidation in a peribronchovascular distribution. Lung biopsy shows plugs of loose fibroblastic tissue filling alveolar ducts and alveoli, with preservation of overall lung architecture and no honeycombing. What is this histological pattern?

(A) Usual interstitial pneumonia (UIP) with honeycombing

(B) Diffuse alveolar damage (DAD) – hyaline membranes

(C) Organizing pneumonia (OP) with Masson bodies (fibroblastic plugs)

(D) Respiratory bronchiolitis-ILD with pigmented macrophages

Q25. A 52-year-old non-smoking woman is found to have a 2.5 cm peripheral right lower lobe mass on CT chest. PET scan shows mild FDG avidity. Biopsy reveals malignant cells growing along pre-existing alveolar walls (lepidic pattern) with focal acinar formation. Molecular testing shows an EGFR exon 19 deletion. What is the most likely diagnosis?

(A) Small cell lung carcinoma (oat cell)

(B) Adenocarcinoma of the lung

(C) Squamous cell carcinoma of the lung

(D) Large cell neuroendocrine carcinoma

Q26. A 58-year-old retired coal miner presents with slowly progressive exertional dyspnoea. HRCT shows small nodular opacities in the upper and mid zones with areas of progressive massive fibrosis. There is no increased susceptibility to tuberculosis. Which pneumoconiosis does this represent?

(A) Silicosis (crystalline silicon dioxide – quartz)

(B) Asbestosis (with pleural plaques)

(C) Berylliosis (non-caseating granulomata)

(D) Coal workers' pneumoconiosis (CWP – coal dust macules)

Q27. A 62-year-old man dies 3 days after the onset of severe chest pain. Autopsy of the heart shows an area of yellow softening in the anterior wall of the left ventricle. Histological examination of the necrotic area shows the most prominent cellular infiltrate at this time point. What is the dominant inflammatory cell infiltrate at this stage of myocardial infarction?

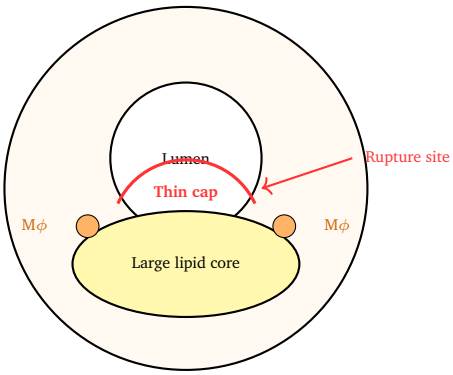
(A) Neutrophils (predominant at days 1–3)

(B) Macrophages (predominant at days 3–7, removing debris)

(C) Lymphocytes (predominant in healing phase, weeks 2–3)

(D) Granulation tissue with new capillaries (weeks 1–2)

Q28. A cardiovascular pathologist studies two types of atherosclerotic plaques. The diagram below represents a cross-section of a coronary artery. Which structural feature makes a plaque most susceptible to rupture and acute coronary syndrome?



(A) Thick fibrous cap with small lipid core and few macrophages

(B) Dense calcification of the fibrous cap

(C) Thin fibrous cap over a large lipid core with macrophage-rich shoulders

(D) Smooth muscle cell proliferation within the plaque

Q29. A 70-year-old man with multiple myeloma develops progressive exertional dyspnoea and lower limb oedema. Echocardiography shows a "sparkling" or granular appearance of the myocardium, normal LV cavity size, increased wall thickness, and markedly impaired diastolic filling (diastolic dysfunction) with preserved systolic function. What is the most likely type of cardiomyopathy and its cause in this context?

(A) Dilated cardiomyopathy from doxorubicin toxicity

(B) Restrictive cardiomyopathy from amyloid deposition

(C) Hypertrophic cardiomyopathy (HOCM) – asymmetric septal hypertrophy

(D) Arrhythmogenic right ventricular cardiomyopathy (ARVC)

Q30. A 48-year-old woman presents with early satiety and progressive weight loss. Upper GI endoscopy shows a rigid, non-distensible stomach ("leather bottle stomach"). Biopsy reveals individual malignant cells with abundant intracytoplasmic mucin displacing the nucleus to the periphery, infiltrating diffusely through the gastric wall without forming a discrete mass. What cell type is diagnostic?

(A) Intestinal-type adenocarcinoma forming glands

(B) MALT lymphoma – small B-cell infiltrate

(C) GIST (gastrointestinal stromal tumour) – spindle cells

(D) Signet ring cell carcinoma (diffuse-type – linitis plastica)

Q31. An 80-year-old man presents with left lower quadrant pain, fever, and elevated WBC. CT abdomen shows multiple outpouchings in the sigmoid colon with surrounding fat stranding. On resection, histology shows that the wall of the outpouchings consists only of mucosa and submucosa herniating through gaps in the muscularis propria at sites of penetrating vessels. These are best classified as:

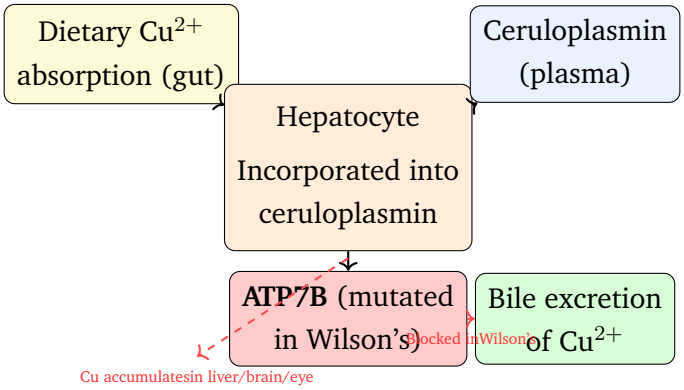
(A) False diverticula (mucosa and submucosa only – no muscularis propria)

(B) True diverticula (all layers of the bowel wall)

(C) Meckel's diverticulum (true, congenital – all layers)

(D) Duplication cysts (adjacent to the bowel wall)

Q32. A 20-year-old man presents with liver cirrhosis, dysarthria, tremor, and psychiatric disturbance. Slit-lamp examination reveals golden-brown rings at the periphery of the cornea. Serum ceruloplasmin is low and 24-hour urinary copper is elevated. The diagram below shows the normal copper metabolism pathway. Which gene mutation is responsible for this disease?



(A) ATP7A gene mutation (Menkes disease – X-linked, copper deficiency)

(B) HFE gene mutation (hereditary haemochromatosis)

(C) ATP7B gene mutation (Wilson's disease – AR, copper excess)

(D) ABCB4 gene mutation (progressive familial intrahepatic cholestasis)

Q33. A 58-year-old man with a long history of hepatitis C-related cirrhosis undergoes surveillance imaging. A 5 cm arterially enhancing hepatic nodule with washout on portal phase is detected. Serum alpha-fetoprotein (AFP) is 3200 ng/mL (normal $<$10). Which primary liver tumour is this, and why is AFP elevated?

(A) Cholangiocarcinoma – AFP not characteristically elevated

(B) Hepatocellular carcinoma (HCC) – recapitulates fetal liver AFP production

(C) Hepatic haemangioma – benign, AFP normal

(D) Metastatic colorectal carcinoma – CEA elevated, not AFP

Q34. A 35-year-old HIV-positive man presents with nephrotic syndrome: massive proteinuria (7 g/day), hypoalbuminaemia, and oedema. Renal biopsy on light microscopy shows segmental areas of sclerosis involving portions of some glomeruli. Electron microscopy shows effacement of podocyte foot processes. Immunofluorescence is negative. What is the diagnosis?

(A) Minimal change disease (MCD) – normal LM, effaced foot processes

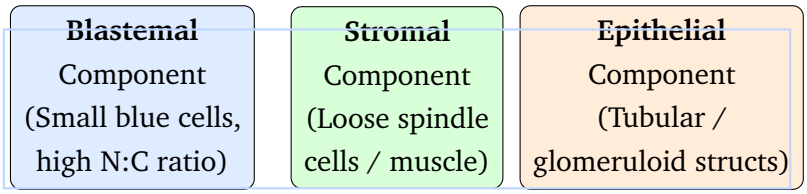
(B) Membranous nephropathy – diffuse capillary wall thickening, subepithelial deposits

(C) Membranoproliferative glomerulonephritis – tram-track appearance

(D) Focal segmental glomerulosclerosis (FSGS)

Q35. A 3-year-old girl presents with a large abdominal mass. Ultrasound reveals a well-defined 8 cm intrarenal mass. CT shows the mass is confined to the kidney with no vascular invasion. Biopsy shows a triphasic tumour with blastemal, stromal, and epithelial components (tubules/glomeruloid structures), as depicted below. Which tumour suppressor gene is mutated in sporadic cases of this tumour?

Wilms Tumour (Nephroblastoma) – Triphasic Histology



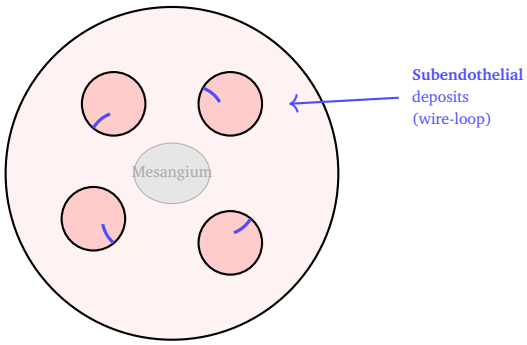
(A) WT1 tumour suppressor gene (chromosome 11p13)

(B) RB1 tumour suppressor gene (chromosome 13q14)

(C) p53 tumour suppressor gene (chromosome 17p13)

(D) VHL tumour suppressor gene (chromosome 3p25)

Q36. A 28-year-old woman with known systemic lupus erythematosus develops haematuria, proteinuria, and rising serum creatinine. Renal biopsy on light microscopy shows diffuse proliferative glomerulonephritis with prominent thickening of capillary walls creating a characteristic appearance. Immunofluorescence shows a "full house" pattern (IgG, IgA, IgM, C3, C1q all positive). The diagram below shows a glomerular capillary cross-section. Which light microscopy finding is characteristic of Class IV lupus nephritis?



Glomerular capillary cross-section
(Class IV Lupus Nephritis – LM)

(A) Spike and dome pattern on silver stain (Jones methenamine silver)

(B) Crescent formation with fibrin deposition

(C) Wire-loop lesion (subendothelial immune deposits on light microscopy)

(D) Nodular mesangial deposits (Kimmelstiel-Wilson nodules)

Q37. A 35-year-old woman presents with a painless anterior neck mass and palpable cervical lymph nodes. Fine needle aspiration cytology shows cells with ground-glass ("Orphan Annie eye") nuclei, nuclear grooves, and intranuclear cytoplasmic inclusions. Psammoma bodies are seen in the background. What is the diagnosis?

(A) Follicular thyroid carcinoma (FTC) – capsular and vascular invasion

(B) Papillary thyroid carcinoma (PTC) – Orphan Annie nuclei, psammoma bodies

(C) Medullary thyroid carcinoma (MTC) – parafollicular C-cells, calcitonin

(D) Anaplastic thyroid carcinoma – undifferentiated, rapidly fatal

Q38. A 40-year-old woman presents with episodic hypertension, palpitations, headache, and diaphoresis lasting 20 minutes. Blood pressure during an episode is 210/130 mmHg. CT adrenal shows a 4 cm right adrenal mass. Which laboratory investigation is the most sensitive biochemical marker for confirming the diagnosis of phaeochromocytoma?

(A) Serum aldosterone-to-renin ratio

(B) Serum ACTH and 24-hour urinary free cortisol

(C) Serum calcitonin and CEA levels

(D) 24-hour urinary vanillylmandelic acid (VMA) and/or plasma metanephrines

Q39. A 65-year-old fair-skinned farmer has multiple rough, scaly erythematous patches on the dorsa of his hands and face (actinic keratoses). One lesion has become indurated, ulcerated, and 1.5 cm in diameter. Biopsy reveals a malignant tumour of keratinocytes showing intercellular bridges (desmosomes) and keratin pearl formation. What is the diagnosis?

(A) Squamous cell carcinoma of the skin

(B) Basal cell carcinoma (BCC) – palisading basal cells

(C) Melanoma – malignant melanocytes, S100/Melan-A positive

(D) Merkel cell carcinoma – neuroendocrine, CK20 paranuclear dots

Q40. A 30-year-old woman notices a 1 cm firm, slightly pigmented papule on her lower leg that has been present for 2 years. On pinching the overlying skin, the lesion dimples inward (positive "dimple sign"). Biopsy shows a dermal proliferation of bland fibroblasts and histiocytes with overlying epidermal hyperplasia and basal hyperpigmentation. Immunohistochemistry is positive for Factor XIIIa and negative for CD34. What is the diagnosis?

(A) Dermatofibrosarcoma protuberans (DFSP) – CD34 positive, infiltrative

(B) Lipoma – mature adipocytes, deep soft tissue

(C) Dermatofibroma (fibrous histiocytoma) – Factor XIIIa positive, dimple sign

(D) Epidermal inclusion cyst – keratin-filled cyst, no solid proliferation

Detailed Solutions

Q1.

Solution

Concept – Caseous necrosis in tuberculosis: Caseous necrosis is the hallmark of tuberculosis. Grossly it resembles soft white cheese; microscopically it appears as amorphous, granular, eosinophilic material with complete loss of tissue architecture – no ghost cell outlines are retained. It is surrounded by a granulomatous reaction comprising epithelioid macrophages and Langhans-type giant cells.

Key Point: Caseous necrosis is unique among necrosis types because it combines features of both coagulative and liquefactive necrosis; it is seen in TB, other mycobacterial infections, and fungal infections such as histoplasmosis.

Why other options are wrong:

- Option A: Coagulative necrosis preserves cell outlines ("ghost cells") – seen in ischaemic infarcts of most organs.
- Option C: Liquefactive necrosis produces a fluid-filled cavity with neutrophils – typical of brain infarcts and abscesses.
- Option D: Fat necrosis involves chalky-white saponification deposits from lipase action – seen in acute pancreatitis and breast trauma.

Final Answer: Caseous necrosis – amorphous eosinophilic material, no tissue architecture $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q1

Q2.

Solution

Concept – Executioner caspases in intrinsic apoptosis: The intrinsic (mitochondrial) pathway involves release of cytochrome c from mitochondria (triggered by pro-apoptotic Bcl-2 family members such as Bax/Bak). Cytochrome c binds Apaf-1 to form the apoptosome, which activates initiator caspase-9. Caspase-9 then cleaves and activates **caspase-3**, the key executioner caspase that cleaves cellular proteins (including ICAD, leading to DNA fragmentation) and produces the morphological features of apoptosis.

Key Point: Caspase-3 is the central executioner caspase; it is downstream of both intrinsic (caspase-9) and extrinsic (caspase-8) initiator pathways.

Why other options are wrong:

- Option A: Bcl-2 is anti-apoptotic (inhibits cytochrome c release) – not the executioner.
- Option B: Cytochrome c is the upstream activator of the apoptosome, not the executioner.
- Option C: Apaf-1 is the scaffold protein of the apoptosome (activates caspase-9), not the direct executioner.

Final Answer: Caspase-3 (executioner caspase) $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q2

Q3.

Solution

Concept – Necrosis triggers inflammation; apoptosis does not: Necrosis is a pathological cell death involving membrane disruption and release of intracellular contents (DAMPs – damage-associated molecular patterns) that trigger an acute inflammatory response. Apoptosis is a programmed, energy-dependent process: apoptotic cells form membrane-bound bodies that are rapidly phagocytosed by macrophages without membrane disruption, so cellular contents are not released and **no inflammation is triggered**.

Key Point: This distinction is of fundamental clinical importance – apoptosis is physiological (embryogenesis, immune regulation, tumour therapy), while necrosis is pathological and drives tissue destruction through inflammation.

Why other options are wrong:

- Option B: Apoptosis does NOT trigger inflammation – this is incorrect.
- Option C: The two processes have opposite effects on inflammation.
- Option D: Necrosis clearly triggers inflammation via DAMP release.

Final Answer: Necrosis triggers inflammation; apoptosis does not $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – Leukocyte extravasation – margination and rolling: The sequential steps of leukocyte migration out of blood vessels are: (1) **Margination** – redistribution from central flow to the periphery as blood flow slows; (2) **Rolling** –

loose, reversible binding via selectins (P-selectin, E-selectin on endothelium; L-selectin on leukocytes) interacting with sialylated carbohydrate ligands; (3) **Firm adhesion** – integrins (LFA-1, Mac-1) bind ICAM-1/ICAM-2 (upregulated by TNF, IL-1); (4) **Transmigration/diapedesis** through intercellular junctions (PECAM-1/CD31); (5) **Chemotaxis** toward the injury site.

Key Point: Margination and rolling is the FIRST step. It is mediated by selectins and their ligands (PSGL-1). Histamine and thrombin rapidly mobilise P-selectin from Weibel-Palade bodies to the endothelial surface within minutes.

Why other options are wrong:

- Option A: Transmigration is the third/fourth step, not the first.
- Option B: Firm adhesion via integrins occurs after rolling (step 3).
- Option D: Chemotaxis occurs after the leukocyte has crossed the vessel wall.

Final Answer: Margination and rolling along the endothelium $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q4

Q5.

Solution

Concept – Pseudomembranous colitis from Clostridium difficile: *Clostridium difficile* is an anaerobic, gram-positive, spore-forming bacillus. It colonises the colon after disruption of normal flora by broad-spectrum antibiotics (clindamycin, ampicillin, cephalosporins, fluoroquinolones). Toxins A (enterotoxin) and B (cytotoxin) damage enterocytes, causing exudation of fibrin, mucus, necrotic cells, and neutrophils that coalesce into the characteristic yellow-white plaques (pseudomembranes). Diagnosis is confirmed by stool toxin PCR or EIA.

Key Point: Treatment is oral vancomycin or fidaxomicin (first-line); metronidazole for mild cases. For recurrent disease, faecal microbiota transplantation (FMT) is effective.

Why other options are wrong:

- Option A: *S. aureus* can cause food poisoning but not pseudomembranous colitis.
- Option C: *Campylobacter* causes bloody diarrhoea, not pseudomembranes.
- Option D: *E. coli* O157:H7 causes haemorrhagic colitis and HUS, not pseudomembranes.

Final Answer: Clostridium difficile $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – Sarcoidosis and non-caseating granuloma: Sarcoidosis is a systemic granulomatous disease of unknown aetiology (likely an exaggerated immune response to an antigen in genetically susceptible individuals). The hallmark is **non-caseating epithelioid granuloma** – compact aggregates of epithelioid macrophages and CD4+ T cells without central necrosis (unlike TB granulomata which caseate). Multinucleated giant cells may contain Schaumann bodies and asteroid bodies. Serum ACE is elevated.

Key Point: Bilateral hilar lymphadenopathy on chest X-ray + elevated ACE + non-caseating granuloma on biopsy = sarcoidosis until proven otherwise.

Why other options are wrong:

- Option A: Caseating granuloma is the hallmark of tuberculosis, not sarcoidosis.
- Option B: Suppurative granuloma (with neutrophils in the centre) is seen in cat-scratch disease, lymphogranuloma venereum.
- Option C: Touton giant cells with foamy cytoplasm are seen in fat necrosis and xanthogranulomas.

Final Answer: Non-caseating epithelioid granuloma $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – Myofibroblasts in wound contraction (secondary intention): In healing by secondary intention, the wound defect is filled with granulation tissue. Key cells include myofibroblasts – modified fibroblasts that express **α-smooth muscle actin (α-SMA)**, generating contractile force. Contraction reduces the wound area by up to 80%, closing the defect. Contraction is driven by the cytoskeletal machinery of myofibroblasts interacting with the extracellular matrix via fibronectin-rich fibronexi. TGF-β is the key growth factor inducing fibroblast-to-myofibroblast differentiation.

Key Point: Excessive myofibroblast activity leads to contractures (e.g.,

Dupuytren's contracture, hypertrophic scars). Myofibroblasts are also the key cells in organ fibrosis.

Why other options are wrong:

- Option B: Type I collagen-secreting fibroblasts contribute to scar strength but do not mediate contraction.
- Option C: Neutrophils debride the wound in the early (inflammatory) phase; they do not cause contraction.
- Option D: Macrophages orchestrate healing by releasing growth factors (TGF-β, PDGF, FGF) but do not directly contract the wound.

Final Answer: Myofibroblast contraction $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – PGE2 as a mediator of fever and pain: Prostaglandins are derived from arachidonic acid via cyclooxygenase (COX-1 and COX-2). **PGE2** (prostaglandin E2) is the principal prostaglandin responsible for: (i) **Fever** – acts on the hypothalamus (preoptic area) to raise the temperature set-point via cAMP-mediated mechanisms; (ii) **Pain sensitisation** (hyperalgesia) – sensitises peripheral nociceptors to bradykinin and other pain mediators. NSAIDs (aspirin, ibuprofen) inhibit COX enzymes, reducing PGE2 and thereby reducing fever and pain.

Key Point: IL-1 and TNF-α (endogenous pyrogens) stimulate macrophages and endothelial cells to produce PGE2, which crosses the blood-brain barrier to act on the hypothalamus.

Why other options are wrong:

- Option A: LTB4 is a potent neutrophil chemotactic factor – not a mediator of fever or hyperalgesia.
- Option B: TXA2 causes platelet aggregation and vasoconstriction – its role in fever/pain is minimal.
- Option D: PAF activates platelets and increases vascular permeability but is not the primary fever/pain mediator.

Final Answer: Prostaglandin E2 (PGE2) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q8

Q9.

Solution

Concept – Arterial thrombus (platelet-rich, white): Thrombus composition depends on blood flow. In **arteries** (high-flow, high-shear environment), thrombi form at sites of endothelial injury or plaque rupture. Platelets are the primary component because high shear activates platelets and von Willebrand factor (vWF) promotes platelet adhesion. These are called **"white thrombi"** or platelet-rich thrombi; they show **lines of Zahn** (pale platelet/fibrin layers alternating with red cell layers) on histology. In veins (low-flow), stasis predominates and fibrin/RBC-rich "red thrombi" form.

Key Point: The lines of Zahn are a useful morphological marker distinguishing ante-mortem thrombus from post-mortem clot. Antiplatelet drugs (aspirin, clopidogrel) are effective for arterial thrombosis; anticoagulants for venous thrombosis.

Why other options are wrong:

- Option A: Fibrin-rich red thrombus is characteristic of venous thrombosis (stasis).
- Option C: Mixed thrombi occur in large veins or at the boundary of flow zones, not typically arterial.
- Option D: Hyaline (fibrin) thrombi of microvasculature are seen in DIC and TTP/HUS.

Final Answer: Platelet-rich (white) thrombus with lines of Zahn $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q9

Q10.

Solution

Concept – Fat embolism syndrome: Fat embolism syndrome occurs typically 24–72 hours after long bone or pelvic fractures (also seen in sickle cell crisis, liposuction, bone marrow transplantation). Fat droplets enter the venous sinusoids and embolise to the lungs, brain, and skin. The classic **triad** is: (1) **Respiratory failure/ARDS** (dyspnoea, hypoxaemia); (2) **Neurological dysfunction** (confusion, seizures); (3) **Petechial rash** (axillae, chest, conjunctivae). Fat globules can be detected in urine and sputum. The pathogenesis involves mechanical obstruction AND toxic effects of free fatty acids on endothelium.

Key Point: Fat embolism has a specific timing (24–72 h post-fracture) and the petechial rash in the axilla/chest is pathognomonic. Treatment is supportive (oxy-

gen, respiratory support).

Why other options are wrong:

- Option A: Pulmonary thromboembolism presents with pleuritic chest pain and does not cause petechiae or mental status changes in the same context.
- Option B: Air embolism follows procedures (central line, surgery) with air entering venous system – no petechiae.
- Option C: Amniotic fluid embolism occurs during or immediately after delivery, not after orthopaedic trauma.

Final Answer: Fat embolism syndrome $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q10

Q11.

Solution

Concept – Septic shock (distributive): Gram-negative LPS activates macrophages/monocytes to release TNF-α, IL-1, and IL-6. These cytokines cause massive peripheral vasodilation (distributive shock) via excess nitric oxide (iNOS-derived) and prostacyclin. Despite adequate (or supranormal) cardiac output, tissue perfusion is impaired. Clinically: warm, flushed skin (high-output, low SVR state), bounding pulse, fever, lactic acidosis. This contrasts with cardiogenic shock (cold clammy skin, elevated SVR, low CO).

Key Point: In early septic shock, cardiac output is HIGH (hyperdynamic); in late septic shock, myocardial depression leads to low output. Serum lactate $>$2 mmol/L defines sepsis-induced tissue hypoperfusion.

Why other options are wrong:

- Option B: Hypovolaemic shock: cold/clammy skin, high SVR, precipitated by haemorrhage/fluid loss.
- Option C: Cardiogenic shock: cold extremities, elevated JVP/CVP, pulmonary oedema, precipitated by MI.
- Option D: Obstructive shock: elevated CVP, absent breath sounds/distended neck veins in tension pneumothorax.

Final Answer: Septic shock (distributive) $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q11

Q12.

Solution

Concept – Alpha-fetoprotein (AFP) as a tumour marker: AFP is a glycoprotein normally produced by the fetal yolk sac, liver, and GI tract. It falls to adult levels ($<$10 ng/mL) after birth. Marked elevation in adults indicates: (i) **Hepatocellular carcinoma (HCC)** – AFP $>$400 ng/mL is diagnostic in the right clinical context; (ii) **Yolk sac tumour** (endodermal sinus tumour) of the gonads; (iii) Mixed germ cell tumours. Mild AFP elevation may occur in liver regeneration (cirrhosis, viral hepatitis).

Key Point: AFP + HCC: levels correlate with tumour burden and prognosis. AFP is also used for prenatal screening (elevated in neural tube defects, low in Down syndrome with MSAFP). AFP is NOT elevated in cholangiocarcinoma.

Why other options are wrong:

- Option A: CEA is elevated in colorectal, gastric, pancreatic, and lung adenocarcinoma – not specifically HCC.
- Option B: CA 19-9 is elevated in pancreatic and biliary cancers.
- Option D: CA-125 is the ovarian epithelial tumour marker.

Final Answer: Alpha-fetoprotein (AFP) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q12

Q13.

Solution

Concept – Perineural invasion in adenoid cystic carcinoma: Adenoid cystic carcinoma (AdCC) is the most common malignant tumour of minor salivary glands and the second most common of parotid gland. Its histological hallmark is the **cribriform (Swiss-cheese) pattern** of small basaloid cells arranged around pseudo-cystic spaces containing mucoid material. It has a notorious predilection for **perineural invasion (PNI)**, spreading along nerve sheaths for long distances – explaining pain radiating along nerve distributions and the high local recurrence rate even after seemingly complete resection.

Key Point: Perineural invasion in AdCC means that surgical margins must be assessed for neural involvement; recurrence rates are high even decades later. Distant metastases (especially to lungs) are common.

Why other options are wrong:

- Option A: Haematogenous spread to the lungs occurs late; the primary route is perineural, not haematogenous.
- Option C: Lymph node metastasis is less common in AdCC compared to mucoepidermoid carcinoma.
- Option D: Direct capsular invasion occurs but is not the characteristic route of spread that distinguishes AdCC.

Final Answer: Perineural spread (perineural invasion) $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – VIPoma (WDHA syndrome): VIPoma is a pancreatic neuroendocrine tumour that secretes **Vasoactive Intestinal Peptide (VIP)**. VIP activates adenylyl cyclase in enterocytes, causing massive secretory diarrhoea (cholera-like). The resulting WDHA syndrome comprises: **W**atery diarrhoea (>3 L/day), **H**ypokalaemia (from faecal potassium loss), **A**chlorhydria (VIP inhibits gastric acid secretion). This is also called Verner-Morrison syndrome or pancreatic cholera.

Key Point: VIPoma is one of the APUDomas – tumours of amine precursor uptake and decarboxylation cells. Treatment: somatostatin analogues (octreotide) control symptoms; surgical resection if localised.

Why other options are wrong:

- Option A: Insulinoma causes hypoglycaemia, sweating, Whipple's triad – not massive diarrhoea.
- Option B: Glucagonoma causes the 4Ds: Diabetes, Dermatitis (necrolytic migratory erythema), DVT, Depression – not secretory diarrhoea.
- Option C: Gastrinoma (Zollinger-Ellison) causes peptic ulcers, elevated gastric acid – not achlorhydria.

Final Answer: VIPoma (WDHA – Watery Diarrhoea, Hypokalaemia, Achlorhydria) $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q14

Q15.

Solution

Concept – Anaplastic large cell lymphoma (ALCL) and ALK rearrangement: ALCL is a T-cell lymphoma characterised by large pleomorphic cells with horseshoe or kidney-shaped nuclei ("hallmark cells"), CD30 positivity, and in ALK-positive cases, the **t(2;5)(p23;q35)** translocation fusing the **ALK** gene (encoding anaplastic lymphoma kinase on chromosome 2) to the **NPM1** gene (nucleophosmin on chromosome 5). This creates NPM-ALK fusion protein with constitutive kinase activity. ALK-positive ALCL affects young adults and has a **better prognosis** than ALK-negative ALCL. ALK inhibitors (crizotinib) are effective.

Key Point: CD30 positivity is shared by Hodgkin lymphoma (Reed-Sternberg cells) – distinction: RS cells are also CD15+; ALCL cells are T-cell lineage markers positive.

Why other options are wrong:

- Option B: BCL-2 rearrangement t(14;18) is the hallmark of follicular lymphoma.
- Option C: BCL-6 rearrangement t(3;14) is seen in diffuse large B-cell lymphoma (DLBCL).
- Option D: c-MYC translocation t(8;14) is the hallmark of Burkitt lymphoma.

Final Answer: ALK gene rearrangement [t(2;5)(p23;q35)] $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q15

Q16.

Solution

Concept – Ductal carcinoma in situ (DCIS) – comedo subtype: DCIS is a non-invasive breast malignancy confined within the ductal-lobular system (intact basement membrane). The **comedo subtype** is the most aggressive pattern of DCIS: malignant cells fill and distend ducts with **central necrosis** and **dystrophic calcification** (which appears as casting-type calcifications on mammography). No stromal invasion means it is not invasive carcinoma. DCIS is a precursor lesion – untreated comedo DCIS has a high rate of progression to invasive carcinoma.

Key Point: DCIS is typically detected on mammographic screening. Treatment depends on grade and extent: lumpectomy $\pm$ radiotherapy $\pm$ endocrine therapy for ER+ cases.

Why other options are wrong:

- Option A: Invasive ductal carcinoma (IDC) would show tumour cells beyond the basement membrane into stroma.
- Option B: LCIS is a lobular lesion with small cells filling lobular units; it does not form ducts and is often multifocal/bilateral.
- Option D: Phyllodes tumour has a biphasic stroma + epithelial component with leaf-like architecture.

Final Answer: Ductal carcinoma in situ (DCIS), comedo subtype $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q16

Q17.

Solution

Concept – Aplastic anaemia and hypocellular bone marrow: Aplastic anaemia is characterised by **pancytopenia** due to destruction or failure of haematopoietic stem cells, resulting in a **hypocellular bone marrow** with replacement by fat cells. Most cases are immune-mediated (autoreactive T cells destroy HSCs; antigen is unknown). Causes include idiopathic, drugs (chloramphenicol, chemotherapy), viral infections (EBV, hepatitis), radiation, and inherited (Fanconi anaemia). Reticulocytes are markedly reduced (hypoproliferative anaemia).

Key Point: Bone marrow cellularity $<$25% with pancytopenia = aplastic anaemia. Treatment: immunosuppression (ATG + cyclosporin) for non-severe cases; allogeneic haematopoietic stem cell transplant for severe aplastic anaemia in young patients.

Why other options are wrong:

- Option A: Hypercellular marrow with $>$20% blasts defines acute leukaemia (AML/ALL).
- Option C: Reed-Sternberg cells are the pathognomonic feature of Hodgkin lymphoma – not aplastic anaemia.
- Option D: "Wrinkled tissue paper" macrophages (Gaucher cells) indicate Gaucher disease – a storage disorder.

Final Answer: Hypocellular marrow with fat cell replacement $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q17

Q18.

Solution

Concept – CLL and smudge cells (basket cells): Chronic lymphocytic leukaemia (CLL) is the most common leukaemia in adults >60 years in Western countries. The peripheral blood smear shows small mature-appearing lymphocytes with scant cytoplasm and clumped ("soccer ball") chromatin. A characteristic feature is **smudge cells (basket cells)** – disrupted lymphocytes that were fragile and lysed during blood smear preparation due to their reduced cytoskeletal integrity. Immunophenotype: **CD5+, CD19+, CD23+, CD20 dim, surface Ig dim** (weak) – the CD5/CD19 co-expression is characteristic.

Key Point: CLL is characterised by: lymphocytosis >5,000/mm^3 of clonal B-lymphocytes, smudge cells on smear, and indolent course. Staging (Rai/Binet) guides treatment.

Why other options are wrong:

- Option A: Auer rods (crystallised MPO granules) are seen in AML blasts.
- Option B: Reed-Sternberg cells (owl-eye nuclei, binucleated) are pathognomonic of Hodgkin lymphoma.
- Option C: Flame cells (IgA-containing plasma cells with fiery cytoplasm) are seen in IgA myeloma.

Final Answer: Smudge cells (basket cells) $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q18

Q19.

Solution

Concept – Mucin-secreting adenocarcinoma and DIC (Trousseau syndrome): Mucin-secreting adenocarcinomas (particularly pancreatic, gastric, lung, and ovarian) express tissue factor and shed tumour-derived mucins. Mucins can directly activate factor X and prothrombin, triggering the coagulation cascade independently of normal initiating factors. This leads to **Trousseau syndrome**: migratory thrombophlebitis and hypercoagulability associated with malignancy, which can progress to frank DIC. The mechanism is distinct from the consumptive coagulopathy of sepsis-induced DIC.

Key Point: Trousseau himself died of the migratory thrombophlebitis he described in cancer patients. Any patient with unexplained recurrent DVT should be investigated for occult malignancy.

Why other options are wrong:

- Option B: Tumour-related angiogenesis increases tPA locally but does not cause systemic DIC via this mechanism.
- Option C: Bone marrow infiltration causes thrombocytopaenia (reduced platelet production) but not the hypercoagulable DIC mechanism.
- Option D: Liver metastases can reduce clotting factor synthesis, causing hypocoagulability, not the hypercoagulable Trousseau mechanism.

Final Answer: Mucin secretion activating coagulation (Trousseau syndrome) $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q19

Q20.

Solution

Concept – Factor V Leiden mutation (most common inherited thrombophilia): **Factor V Leiden** is a point mutation (R506Q) in the factor V gene, making factor Va resistant to cleavage by activated protein C (APC). Normal APC inactivates factors Va and VIIIa to limit coagulation. With APC resistance, factor Va remains active, leading to excess thrombin generation and a hypercoagulable state. **Factor V Leiden is the most common inherited thrombophilia** (prevalence $\sim$5% in Caucasians, heterozygous; $>$3-fold increase in DVT risk).

Key Point: APC resistance assay (APTT fails to prolong with added APC) is the screening test; confirmed by Factor V Leiden gene mutation analysis. Pregnancy, oestrogen, and surgery are trigger factors.

Why other options are wrong:

- Option A: Protein C deficiency is an inherited thrombophilia but less common; it reduces inactivation of factors Va and VIIIa.
- Option B: Antithrombin III deficiency reduces inhibition of thrombin and Xa – another inherited thrombophilia, but less prevalent than Factor V Leiden.
- Option D: Prothrombin G20210A mutation is the second most common inherited thrombophilia (increases prothrombin levels).

Final Answer: Factor V Leiden mutation (APC resistance) $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q20

Q21.

Solution

Concept – Hairy cell leukaemia and TRAP positivity: Hairy cell leukaemia (HCL) is a rare, indolent mature B-cell lymphoproliferative disorder. The neoplastic cells have characteristic cytoplasmic projections ("hairy" cells) visible on peripheral blood smear and are strongly positive for **TRAP (tartrate-resistant acid phosphatase isoenzyme 5b)** – a staining reaction that persists even after adding tartrate (which inhibits acid phosphatase isoenzymes 1–4). TRAP positivity is the classic cytochemical marker of HCL. Immunophenotype: CD19+, CD20+, CD22+, CD11c+, CD25+, CD103+, CD123+.

Key Point: HCL presents with splenomegaly, pancytopenia, and "dry tap" on bone marrow aspiration (due to reticulin fibrosis). First-line treatment is cladribine (2-CDA) with high cure rates.

Why other options are wrong:

- Option A: PAS positivity (block positivity) is seen in ALL and some AML subtypes.
- Option C: Sudan black B stains lipids/phospholipids in myeloid leukaemia (AML) granules.
- Option D: Myeloperoxidase (MPO) positivity indicates myeloid lineage (AML) – hairy cells are B-lymphoid.

Final Answer: TRAP (tartrate-resistant acid phosphatase) positivity $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q21

Q22.

Solution

Concept – Heinz bodies in G6PD deficiency: G6PD (glucose-6-phosphate dehydrogenase) deficiency is the most common enzymopathy (X-linked recessive). G6PD is essential in the hexose monophosphate shunt for producing NADPH, which maintains glutathione in its reduced form. Without NADPH, oxidative stress (from primaquine, dapsone, fava beans, infections) causes haemoglobin denaturation and precipitation as **Heinz bodies** – dark inclusions seen with supravital stain (brilliant cresyl blue, methylene blue) attached to the inner membrane. Splenic macrophages remove Heinz body-containing erythrocytes, creating "bite cells" on the routine smear.

Key Point: G6PD deficiency is protective against malaria (heterozygote advan-

tage). The gene is on chromosome Xq28. Females may be affected if homozygous or with extreme lyonisation.

Why other options are wrong:

- Option A: Howell-Jolly bodies are basophilic DNA remnants seen after splenectomy or in hyposplenism – appear as single purple dots on routine Giemsa stain, not peripheral hairy inclusions.
- Option B: Basophilic stippling shows evenly distributed blue dots throughout the RBC (ribosomal aggregates) – seen in lead poisoning, thalassaemia, megaloblastic anaemia.
- Option C: Pappenheimer bodies are iron-containing granules (siderosomes) in sideroblastic anaemia – Perls' stain positive.

Final Answer: Heinz bodies – denatured haemoglobin from oxidative stress (G6PD deficiency) $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q22

Q23.

Solution

Concept – Miliary tuberculosis (haematogenous dissemination): Miliary TB results from haematogenous dissemination of *Mycobacterium tuberculosis* from a primary or reactivation focus, seeding multiple organs (lungs, liver, spleen, meninges, kidneys, bone marrow) with innumerable small (1–2 mm) granulomata resembling millet seeds. This pattern occurs when host immunity is overwhelmed (HIV/AIDS, immunosuppression, malnutrition, extremes of age). Chest X-ray shows a diffuse micronodular ("snowstorm") pattern; CT shows innumerable bilateral nodules uniformly distributed (random pattern on HRCT).

Key Point: Miliary TB is a medical emergency. Sputum culture is often negative; bronchoalveolar lavage or tissue biopsy (bone marrow, liver) may be required. All anti-TB drugs (HRZE regimen) for 6–9 months.

Why other options are wrong:

- Option B: Direct lymphatic spread from the Ghon focus to hilar nodes describes primary TB – not miliary disease.
- Option C: Reactivation TB typically involves the apical lung segment with cavitation, not diffuse miliary spread.
- Option D: Aspiration pneumonia causes lobar/segmental consolidation, not the diffuse micronodular pattern.

Final Answer: Haematogenous dissemination producing millet-seed (miliary) lesions throughout the lungs and other organs $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q23

Q24.

Solution

Concept – Organising pneumonia (Masson bodies): Organising pneumonia (OP) is a clinicopathological syndrome in which alveolar injury is followed by organisation of the exudate. The pathological hallmark is **Masson bodies** – plugs of loose granulation tissue (myofibroblasts embedded in myxoid matrix) filling alveolar ducts, alveoli, and small bronchioles. The overall lung architecture is preserved (no honeycombing, no destruction). OP can be cryptogenic (COP/BOOP) or secondary to infections, drugs, connective tissue disease, radiation. HRCT shows bilateral patchy consolidation (peribronchovascular/subpleural distribution). **Excellent response to corticosteroids** distinguishes it from UIP/IPF.

Key Point: Preserved lung architecture + Masson bodies + response to steroids = organising pneumonia. UIP (IPF) shows honeycombing, fibroblastic foci, and poor steroid response.

Why other options are wrong:

- Option A: UIP (usual interstitial pneumonia) shows honeycombing, fibroblastic foci, spatially heterogeneous fibrosis – the worst prognosis ILD.
- Option B: DAD (diffuse alveolar damage) shows hyaline membrane formation (exudative phase) followed by fibrosis – the histological pattern of ARDS.
- Option D: RB-ILD shows pigmented (smoker's) macrophages filling respiratory bronchioles – a smoking-related ILD.

Final Answer: Organising pneumonia (OP) with Masson bodies (fibroblastic plugs) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q24

Q25.

Solution

Concept – Lung adenocarcinoma in non-smokers: Adenocarcinoma is the most common lung cancer in non-smokers and in women. It typically arises in the **pe-**

ripheral lung parenchyma, grows in a **lepidic pattern** (spread along pre-existing alveolar walls without destroying architecture – formerly called bronchioloalveolar carcinoma), and can form acinar, papillary, micropapillary, or solid patterns. **EGFR mutations** (exon 19 deletions, exon 21 L858R) are present in ∼15% of Western and ∼50% of East Asian adenocarcinomas – targeted by EGFR-TKIs (gefitinib, erlotinib, osimertinib). **KRAS** mutations occur in smokers.

Key Point: Peripheral location + non-smoker + EGFR mutation + lepidic growth = adenocarcinoma. All newly diagnosed advanced lung adenocarcinoma should undergo molecular testing (EGFR, ALK, ROS1, KRAS, BRAF, NTRK, MET, PD-L1).

Why other options are wrong:

- Option A: Small cell carcinoma arises centrally, in heavy smokers; oat cells with neuroendocrine features; EGFR/KRAS mutations absent.
- Option C: Squamous cell carcinoma is central, in smokers; keratin pearls, intercellular bridges; not associated with EGFR mutations.
- Option D: Large cell neuroendocrine carcinoma lacks glandular differentiation; neuroendocrine markers positive; no lepidic growth.

Final Answer: Adenocarcinoma of the lung $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q25

Q26.

Solution

Concept – Coal workers' pneumoconiosis (CWP): CWP is caused by inhalation of coal dust (a mixture of carbon and silica). Macrophages phagocytose coal particles, forming **coal macules** (coal dust + macrophages in respiratory bronchioles) – simple CWP. Progressive accumulation leads to nodule formation; in a minority, **progressive massive fibrosis (PMF)** develops (>1 cm coalescent masses in upper lobes). Unlike silicosis, CWP **does not increase susceptibility to tuberculosis**. CWP may co-exist with rheumatoid arthritis (Caplan syndrome).

Key Point: Silicosis (birefringent crystalline silica) carries a high TB risk ("silicotuberculosis"); CWP does not. Both show upper lobe predominant nodules.

Why other options are wrong:

- Option A: Silicosis – caused by crystalline SiO_2 (quartz); **associated with TB**; whorled collagenous nodules; eggshell calcification of hilar nodes.
- Option B: Asbestosis – lower lobe, diffuse interstitial fibrosis; pleural

plaques; ferruginous bodies; associated with mesothelioma and lung carcinoma.
- Option C: Berylliosis – non-caseating granulomata histologically identical to sarcoidosis; caused by beryllium exposure (aerospace industry).

Final Answer: Coal workers' pneumoconiosis (CWP – coal dust macules) $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q26

Q27.

Solution

Concept – Myocardial infarction – temporal sequence of histological changes: The sequence of cellular events after MI follows a predictable timeline: **0–4 hours**: No changes on H&E (only seen by electron microscopy or special stains); **4–12 h**: Early coagulation necrosis, wavy fibers; **12–24 h**: Coagulation necrosis, oedema, haemorrhage; **1–3 days**: **Neutrophil infiltration** (acute inflammation – peak at day 2–3); **3–7 days**: Macrophages begin removing debris; **1–3 weeks**: Granulation tissue and new blood vessels; **>6 weeks**: Dense fibrous scar (white scar).

Key Point: At day 3 post-MI, the predominant cellular infiltrate is **neutrophils**. This is clinically relevant because papillary muscle rupture (day 3–5) and free wall rupture are risks during the neutrophil-mediated lytic phase.

Why other options are wrong:

- Option B: Macrophages become the dominant cell from day 3–7, peaking at days 5–10.
- Option C: Lymphocytes and plasma cells appear during the healing/remodelling phase (weeks 2–3).
- Option D: Granulation tissue with new capillaries replaces necrotic muscle from weeks 1–2 onward.

Final Answer: Neutrophils (predominant at days 1–3) $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q27

Q28.

Solution

Concept – Vulnerable atherosclerotic plaque and plaque rupture: Plaque rupture (responsible for ~70% of acute MI and sudden cardiac death) occurs at **vul-**

nerable plaques: those with a **thin fibrous cap** ($<$65 μm), a **large necrotic lipid core**, and **macrophage-rich (foam cell) shoulders**. Matrix metalloproteinases (MMPs) released by macrophages degrade collagen in the fibrous cap, weakening it. Rupture exposes the thrombogenic lipid core and subendothelial matrix to flowing blood, triggering platelet activation and thrombus formation.

Key Point: Paradoxically, severely stenotic "stable" plaques with thick fibrous caps rarely rupture. Most fatal MI occur from rupture of "mild-to-moderate" plaques that were not haemodynamically significant. Statin therapy stabilises plaques by thickening the fibrous cap and reducing lipid content.

Why other options are wrong:

- Option A: A thick fibrous cap with a small lipid core is a stable plaque – low rupture risk.
- Option B: Dense calcification may paradoxically stabilise the plaque (rigid cap less likely to rupture than thin cap).
- Option D: Smooth muscle cell proliferation forms the fibrous cap and contributes to plaque stability.

Final Answer: Thin fibrous cap over a large lipid core with macrophage-rich shoulders $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q28

Q29.

Solution

Concept – Restrictive cardiomyopathy from amyloid: Restrictive cardiomyopathy is characterised by a stiff, non-compliant ventricle with **diastolic dysfunction** and preserved systolic function. The most common cause in adults is **cardiac amyloidosis**: light chain (AL) amyloid in multiple myeloma, or transthyretin (ATTR) amyloid. Amyloid deposits in the myocardium appear as extracellular hyaline deposits; Congo red staining shows apple-green birefringence under polarised light. Echocardiography shows increased wall thickness with a "granular sparkling" appearance.

Key Point: Other causes of restrictive cardiomyopathy: haemochromatosis, sarcoidosis, endomyocardial fibrosis (tropical, eosinophil-related). Constrictive pericarditis mimics restrictive CMP clinically but is pericardial disease.

Why other options are wrong:

- Option A: Dilated cardiomyopathy (DCM) has systolic dysfunction, dilated ventricles, and thin walls – caused by doxorubicin toxicity, viral myocarditis, alcohol.
- Option C: HOCM has asymmetric septal hypertrophy, dynamic LVOT obstruction, systolic anterior motion of the mitral valve (SAM) – autosomal dominant (MYH7, MYBPC3 mutations).
- Option D: ARVC is characterised by fibro-fatty replacement of the RV myocardium, right-sided arrhythmias, and sudden death in young athletes.

Final Answer: Restrictive cardiomyopathy from amyloid deposition $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q29

Q30.

Solution

Concept – Signet ring cell carcinoma of the stomach (diffuse type – linitis plastica): The Lauren classification divides gastric adenocarcinoma into **intestinal type** (gland-forming, H. pylori-related, older patients, better prognosis) and **diffuse type** (poorly cohesive cells due to loss of E-cadherin expression, CDH1 mutation, infiltrates diffusely without forming glands). **Signet ring cells** have abundant intracytoplasmic mucin that compresses and displaces the nucleus to the cell periphery. Diffuse infiltration thickens the entire gastric wall (**linitis plastica – "leather bottle stomach"**). Very poor prognosis.

Key Point: Loss of E-cadherin (CDH1 germline mutation) is pathognomonic of hereditary diffuse gastric cancer (HDGC). Prophylactic total gastrectomy is recommended for CDH1 mutation carriers.

Why other options are wrong:

- Option A: Intestinal-type gastric adenocarcinoma forms discrete gland-like structures; associated with H. pylori; does not cause diffuse wall thickening.
- Option B: MALT lymphoma is a B-cell lymphoma arising from mucosa-associated lymphoid tissue; associated with H. pylori; small lymphocytes with lymphoepithelial lesions.
- Option C: GIST arises from interstitial cells of Cajal; spindle/epithelioid morphology; c-KIT/DOG-1 positive; not mucin-producing.

Final Answer: Signet ring cell carcinoma (diffuse-type – linitis plastica) $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q30

Q31.

Solution

Concept – False diverticula in diverticulosis: Colonic diverticula are **false (pseudo) diverticula** because they contain only **mucosa and submucosa** that herniate through defects in the muscularis propria at sites where blood vessels (vasa recta) penetrate the colonic wall. True diverticula (e.g., Meckel's diverticulum, pharyngeal diverticulum) contain all layers of the wall. Diverticulosis affects the **sigmoid colon** predominantly and is linked to a **low-fibre diet**. Complications: diverticulitis, haemorrhage (most common cause of massive lower GI bleeding in adults), perforation, abscess, fistula.

Key Point: Diverticulosis = presence of diverticula (usually asymptomatic). Diverticulitis = inflammation/infection of a diverticulum (left iliac fossa pain, fever, raised WBC, "left-sided appendicitis").

Why other options are wrong:

- Option B: True diverticula contain all layers – found in Meckel's diverticulum and pharyngeal (Zenker's) diverticulum.
- Option C: Meckel's diverticulum is a true diverticulum (congenital, all layers) at 2 feet from the ileocaecal valve; not sigmoid colon.
- Option D: Duplication cysts are congenital anomalies adjacent to bowel; they are separate from the bowel lumen.

Final Answer: False diverticula (mucosa and submucosa only – no muscularis propria) $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q31

Q32.

Solution

Concept – Wilson's disease (ATP7B mutation): Wilson's disease is an autosomal recessive disorder of copper metabolism caused by mutations in the **ATP7B gene** (chromosome 13q14.3), which encodes a P-type ATPase copper transporter expressed in hepatocytes. ATP7B normally incorporates copper into ceruloplasmin for secretion and transports excess copper into bile for excretion. In Wilson's disease, ATP7B is dysfunctional, so copper accumulates in the **liver** (cirrhosis), **brain** (basal ganglia – dysarthria, tremor, psychiatric features), **cornea** (Kayser-Fleischer rings), and **kidneys**. Serum ceruloplasmin is low; 24-hour urinary copper is elevated.

Key Point: KF rings are absent in 50% of patients with hepatic-only presentation. Treatment: D-penicillamine or trientine (copper chelators); liver transplantation in fulminant Wilson's.

Why other options are wrong:

- Option A: ATP7A mutation causes Menkes disease (X-linked, defective intestinal copper absorption – copper deficiency, kinky hair).
- Option B: HFE mutation causes hereditary haemochromatosis (iron overload, not copper).
- Option D: ABCB4 mutation causes progressive familial intrahepatic cholestasis type 3 (PFIC3) – phosphatidylcholine secretion defect.

Final Answer: ATP7B gene mutation (Wilson's disease – AR, copper excess) $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q32

Q33.

Solution

Concept – Hepatocellular carcinoma (HCC) and AFP: HCC is the most common primary liver malignancy (global incidence driven by hepatitis B and C cirrhosis). It recapitulates fetal hepatic physiology, producing **alpha-fetoprotein (AFP)** normally made only in fetal life. AFP >400 ng/mL combined with characteristic imaging findings (arterial enhancement with portal/delayed washout on CT/MRI) is diagnostic of HCC without biopsy (Barcelona Clinic Liver Cancer criteria). AFP is also useful for monitoring treatment response and surveillance for recurrence.

Key Point: The "triple-phase CT" enhancement pattern (arterial hyperenhancement + venous washout) is pathognomonic of HCC and is the basis of non-invasive diagnosis. Hepatitis B vaccination is the most effective prevention.

Why other options are wrong:

- Option A: Cholangiocarcinoma (intrahepatic bile duct carcinoma) is characteristically associated with elevated CA 19-9 and CEA – not AFP.
- Option C: Hepatic haemangioma is benign, with characteristic MRI appearance (peripheral nodular enhancement, fill-in on delayed phase); AFP is normal.
- Option D: Metastatic colorectal carcinoma causes elevated CEA; AFP is not raised.

Final Answer: Hepatocellular carcinoma (HCC) – recapitulates fetal liver AFP production $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q33

Q34.

Solution

Concept – Focal segmental glomerulosclerosis (FSGS): FSGS is characterised on light microscopy by **segmental sclerosis** (scarring/hyalinosis) affecting **some** glomeruli (focal – <50%) in **some segments** of the glomerular tuft. Electron microscopy shows **effacement of podocyte foot processes** (diffuse, even in non-sclerotic segments). Immunofluorescence is typically negative or shows non-specific IgM/C3 in sclerotic areas. FSGS is the most common cause of nephrotic syndrome in adults, particularly in **HIV-positive patients** (collapsing variant of FSGS, associated with APOL1 risk alleles in African Americans).

Key Point: FSGS may be primary (idiopathic) or secondary (HIV, heroin, obesity, sickle cell, reflux nephropathy). Primary FSGS often responds to high-dose steroids; secondary FSGS requires treating the underlying cause.

Why other options are wrong:

- Option A: Minimal change disease has normal LM (no sclerosis) and diffuse foot process effacement on EM; responds well to steroids; most common in children.
- Option B: Membranous nephropathy shows diffuse capillary wall thickening; "spike and dome" on silver stain; granular IgG/C3 on IF (subepithelial deposits); anti-PLA2R antibodies.
- Option C: Membranoproliferative GN shows "tram-track" (double contour) capillary walls from mesangial interposition; associated with C3 nephritic factor or monoclonal immunoglobulins.

Final Answer: Focal segmental glomerulosclerosis (FSGS) $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q34

Q35.

Solution

Concept – Wilms tumour and WT1 gene: Wilms tumour (nephroblastoma) is the most common renal malignancy in children (peak age 3–4 years). Sporadic

cases are associated with mutations in the **WT1 tumour suppressor gene** at chromosome **11p13**. WT1 encodes a zinc-finger transcription factor essential for normal renal and gonadal development. Germline WT1 mutations cause WAGR syndrome (Wilms tumour, Aniridia, Genitourinary anomalies, intellectual disability) and Denys-Drash syndrome (nephropathy + pseudohermaphroditism + Wilms). The tumour has a triphasic histology: **blastemal, stromal, and epithelial** components.

Key Point: Anaplastic histology (multipolar mitoses, marked nuclear pleomorphism) in Wilms confers worse prognosis. Overall prognosis is excellent (>90% 5-year survival) with modern treatment (nephrectomy + chemotherapy ± radiotherapy).

Why other options are wrong:

- Option B: RB1 mutation on chromosome 13q14 causes retinoblastoma – the "two-hit" hypothesis paradigm tumour suppressor.
- Option C: TP53 mutation on 17p13 is the most commonly mutated tumour suppressor in human cancer, but is not the specific Wilms gene.
- Option D: VHL mutation on 3p25 causes Von Hippel-Lindau disease (clear cell renal cell carcinoma, haemangioblastoma, phaeochromocytoma) – not Wilms tumour.

Final Answer: WT1 tumour suppressor gene (chromosome 11p13) $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q35

Q36.

Solution

Concept – Lupus nephritis and wire-loop lesion: Systemic lupus erythematosus (SLE) causes immune complex-mediated glomerulonephritis. In **Class IV (diffuse proliferative) lupus nephritis** – the most severe form – massive **subendothelial immune complex deposits** accumulate along the entire glomerular capillary wall, creating the **wire-loop lesion** on light microscopy (the capillary wall appears thickened and "wire-like"). Immunofluorescence shows the classic **"full house" pattern** (all immunoglobulins IgG, IgA, IgM and complement C3, C1q positive) due to deposition of multi-isotype immune complexes.

Key Point: Subendothelial deposits (by EM) produce wire-loop on LM. The WHO/ISN-RPS classification (I–VI) guides treatment: Class III/IV receive induction with cyclophosphamide or MMF + corticosteroids.

Why other options are wrong:

- Option A: Spike and dome pattern (on silver stain) is the hallmark of membranous nephropathy (subepithelial deposits) – Class V lupus nephritis or idiopathic MN.
- Option B: Crescent formation (with fibrin, parietal cells) indicates rapidly progressive (crescentic) GN – may occur in severe lupus nephritis but is not the "wire-loop."
- Option D: Kimmelstiel-Wilson nodules (nodular glomerulosclerosis) are pathognomonic of diabetic nephropathy, not lupus.

Final Answer: Wire-loop lesion (subendothelial immune deposits on light microscopy) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q36

Q37.

Solution

Concept – Papillary thyroid carcinoma (PTC) – nuclear features: PTC is the most common thyroid malignancy (80–85%). Its diagnosis rests on **nuclear features**: **Orphan Annie eye nuclei** (large, pale, empty-looking nuclei with optically clear chromatin), **nuclear grooves** (longitudinal infoldings of the nuclear membrane), and **intranuclear cytoplasmic pseudoinclusions**. **Psammoma bodies** (laminated calcified concentric rings) are characteristic on cytology and histology. PTC spreads primarily to **regional cervical lymph nodes** (lymphotropic tumour). Molecular alterations: **RET/PTC** rearrangements (especially after radiation exposure), **BRAF V600E** mutation (most common, 60%).

Key Point: Despite lymph node metastases, PTC has an excellent prognosis (10-year survival >95%). Treatment: total thyroidectomy ± radioactive iodine (RAI) ablation ± TSH suppression.

Why other options are wrong:

- Option A: Follicular thyroid carcinoma – diagnosis depends on **capsular and/or vascular invasion** (NOT nuclear features); follicle formation present; FNAC cannot distinguish FTC from follicular adenoma.
- Option C: Medullary thyroid carcinoma arises from parafollicular C-cells; secretes **calcitonin**; amyloid stroma; associated with MEN2A/2B (RET proto-oncogene mutation).
- Option D: Anaplastic thyroid carcinoma – undifferentiated, spindle/giant

cells; rapidly fatal; no nuclear features of PTC.

Final Answer: Papillary thyroid carcinoma (PTC) – Orphan Annie nuclei, psammoma bodies $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q37

Q38.

Solution

Concept – Phaeochromocytoma and urinary VMA (catecholamine metabolites): Phaeochromocytoma is a catecholamine-secreting tumour of the adrenal medulla (chromaffin cells). Episodic release of adrenaline and noradrenaline causes paroxysmal hypertension, palpitations, headache, and diaphoresis (the classic triad). These catecholamines are metabolised to **metanephrines** (normetanephrine, metanephrine) and ultimately to **vanillylmandelic acid (VMA)**. The most sensitive tests are **plasma free metanephrines** and **24-hour urinary metanephrines/VMA**. The "rule of 10s": 10% bilateral, 10% extra-adrenal, 10% malignant, 10% in children, 10% familial.

Key Point: Hereditary phaeochromocytoma: VHL disease, MEN2A (RET), MEN2B (RET), NF1 (neurofibromatosis), SDH mutations (SDHB, SDHD – paraganglioma). Pre-operative α-blockade (phenoxybenzamine) then β-blockade is mandatory before surgery.

Why other options are wrong:

- Option A: Aldosterone-to-renin ratio is used to screen for primary hyperaldosteronism (Conn's syndrome).
- Option B: 24-hour urinary cortisol and ACTH are used to diagnose Cushing's syndrome.
- Option C: Calcitonin and CEA are tumour markers for medullary thyroid carcinoma.

Final Answer: 24-hour urinary vanillylmandelic acid (VMA) and/or plasma metanephrines $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q38

Q39.

Solution

Concept – Squamous cell carcinoma of skin (actinic keratosis precursor): Squamous cell carcinoma (SCC) of the skin arises from keratinocytes in the epidermis. The main precursor lesion is **actinic keratosis (solar keratosis)** – dysplastic keratinocytes in sun-exposed skin from chronic UV (UVB) radiation. SCC is characterised histologically by: malignant keratinocytes invading the dermis, **intercellular bridges** (desmosomes, seen on EM), **individual cell keratinisation**, and **keratin pearls** (concentric whorls of laminated keratin). SCC has significant metastatic potential (unlike BCC).

Key Point: Risk factors for cutaneous SCC: UV exposure, immunosuppression, chronic wounds (Marjolin's ulcer), arsenic, HPV (in perianal/genital SCC), xeroderma pigmentosum. Mucocutaneous SCC (lip, tongue) is more aggressive than skin SCC.

Why other options are wrong:

- Option B: BCC has palisading basal cells at the periphery of tumour islands with retraction artefact; almost never metastasises; pearly rolled margin clinically.
- Option C: Melanoma arises from melanocytes; S100, Melan-A, HMB-45 positive; no keratin pearls or intercellular bridges.
- Option D: Merkel cell carcinoma is a neuroendocrine skin tumour; small blue cells with CK20 paranuclear dot positivity; rare.

Final Answer: Squamous cell carcinoma of the skin $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q39

Q40.

Solution

Concept – Dermatofibroma (fibrous histiocytoma): Dermatofibroma is a benign dermal fibrous proliferation of fibroblasts and histiocytes. Clinical hallmark: **positive dimple sign (Fitzpatrick sign)** – on lateral pinching, the lesion dimples inward because it is tethered to the dermis. Histology shows a dermal nodule of bland spindle cells (fibroblasts and histiocytes) with overlying epidermal hyperplasia and basal layer hyperpigmentation. IHC: **Factor XIIIa positive**, CD34 negative. Contrast with **Dermatofibrosarcoma protuberans (DFSP)**: malignant, CD34 positive, infiltrates subcutis in a storiform pattern, high local recurrence.

Key Point: The positive dimple sign distinguishes dermatofibroma from DFSP (which does NOT dimple, instead protrudes – protuberans). DFSP is treated with wide local excision; Mohs micrographic surgery reduces recurrence.

Why other options are wrong:

- Option A: DFSP is CD34 positive and Factor XIIIa negative – the opposite immunoprofile; infiltrative growth into subcutis.
- Option B: Lipoma is a benign tumour of mature adipocytes located in the subcutaneous tissue; soft, compressible, no dimple sign, no overlying skin change.
- Option D: Epidermal inclusion cyst is a keratin-filled cyst lined by squamous epithelium; it is cystic, not a solid fibrous proliferation.

Final Answer: Dermatofibroma (fibrous histiocytoma) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q40

Answer Key – FMGE Pathology Sample Paper 3

1	B	2	D	3	A	4	C	5	B
6	D	7	A	8	C	9	B	10	D
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