FMGE Pathology Sample Paper – 4

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 68-year-old hypertensive man presents with sudden onset left hemiplegia. MRI brain shows an ischaemic infarct in the right middle cerebral artery territory. Twenty days later the infarcted area appears as a fluid-filled cyst. Which property of brain tissue best explains why cerebral infarction undergoes **liquefactive** rather than coagulative necrosis?

(A) High water and lipid content with abundant proteolytic enzymes

(B) Rich collagen framework that resists enzymatic digestion

(C) Absence of blood supply from the circle of Willis

(D) Predominance of fibrous astrocytes over neurons

Q2. A pathologist examining a liver biopsy from a patient with viral hepatitis notes that individual hepatocytes are shrunken, with deeply eosinophilic, homogeneous cytoplasm and condensed nuclear chromatin, without an inflammatory infiltrate around them. These cells represent:

(A) Coagulative necrosis with ghost-cell outlines

(B) Caseous necrosis with loss of cellular architecture

(C) Cell shrinkage with chromatin condensation (apoptosis)

(D) Liquefactive necrosis secondary to proteolytic enzymes

Q3. A 65-year-old man presents with nocturia, hesitancy, and a post-void residual urine volume of 180 mL. Rectal examination reveals a smooth, symmetrically enlarged prostate. Histology shows glandular and stromal hyperplasia predominantly in the periurethral transition zone. Which hormone most directly drives this hyperplastic process?

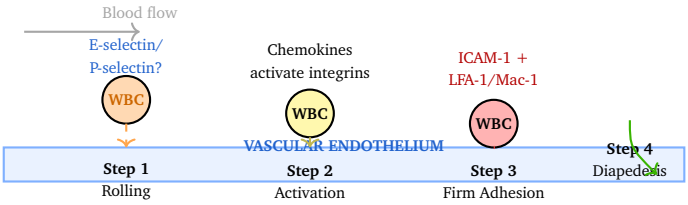
(A) Testosterone (unmetabolised)

(B) Dihydrotestosterone (DHT)

(C) Oestradiol

(D) Follicle-stimulating hormone (FSH)

Q4. The diagram below illustrates the multistep process of leukocyte adhesion to the vascular endothelium during acute inflammation. Which molecule on the endothelial surface mediates the initial **rolling** step (Step 1) shown in the figure?



(A) ICAM-1 (intercellular adhesion molecule-1)

(B) VCAM-1 (vascular cell adhesion molecule-1)

(C) Interleukin-8 (IL-8 / CXCL8)

(D) E-selectin

Q5. A 25-year-old man presents with acute onset of severe, generalised abdominal pain with board-like rigidity. Emergency laparotomy reveals

perforation of the appendix with a thick, yellowish-brown material coating all peritoneal surfaces. Histologically this exudate consists predominantly of fibrin with entrapped neutrophils. This type of inflammation is best classified as:

(A) Fibrinous peritonitis

(B) Serous pleuritis

(C) Pseudomembranous colitis

(D) Granulomatous appendicitis

Q6. A 35-year-old Afro-Caribbean woman presents with dyspnoea, bilateral hilar adenopathy on chest X-ray, skin lesions (erythema nodosum), and elevated serum ACE. Bronchoalveolar lavage reveals CD4+ lymphocytes. Lung biopsy shows non-caseating granulomas containing large macrophages with laminated, concentrically calcified cytoplasmic inclusions. These inclusions are called:

(A) Michaelis-Gutmann bodies

(B) Asteroid bodies only (no calcification)

(C) Schaumann bodies (conchoidal calcifications)

(D) Aschoff bodies

Q7. A 40-year-old woman undergoes abdominal surgery. During secondary intention wound healing, the wound edges gradually approximate over two weeks. The cell type primarily responsible for wound **contraction** that expresses alpha-smooth muscle actin (α-SMA) is:

(A) Type I collagen-secreting fibroblasts

(B) Myofibroblasts (α-SMA positive)

(C) Endothelial cells forming new capillaries

(D) Mast cells releasing histamine

Q8. A patient with bacterial sepsis develops fever of 39.5°C. Macrophages release IL-1 and TNF-α as endogenous pyrogens. These cytokines act

on the **vascular organ of the lamina terminalis (OVLT)** at the blood-brain barrier to stimulate synthesis of which mediator that resets the hypothalamic temperature set-point?

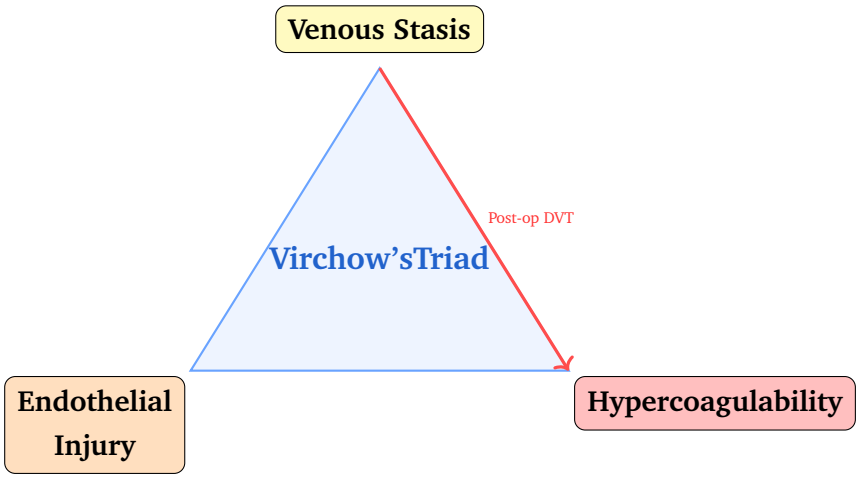
(A) Leukotriene B4 (LTB4)

(B) Platelet-activating factor (PAF)

(C) Interleukin-6 (IL-6) alone

(D) Prostaglandin E2 (PGE2) in the hypothalamus

Q9. A 72-year-old man who underwent elective hip replacement 5 days ago now develops calf pain and oedema. Doppler ultrasound confirms deep vein thrombosis (DVT). The diagram below shows the components of Virchow's triad. Which two factors are **most prominently** responsible for post-operative DVT formation?



(A) Venous stasis and hypercoagulability

(B) Endothelial injury and venous stasis only

(C) Hypercoagulability alone (Factor V Leiden)

(D) Arterial turbulence and endothelial injury

Q10. A 28-year-old woman suddenly develops severe dyspnoea, cyanosis, and cardiovascular collapse immediately after a precipitous delivery. She rapidly progresses to DIC. At autopsy, pulmonary vessel sections show squamous cells, lanugo hairs, and mucin within the pulmonary capillaries and arterioles. The diagnosis is:

(A) Pulmonary thromboembolism (saddle embolus)

(B) Fat embolism syndrome

(C) Amniotic fluid embolism

(D) Air embolism from IV line

Q11. A 22-year-old motorcyclist sustains a C5 spinal cord transection. In the emergency department he has BP 80/50 mmHg and heart rate 52 bpm (bradycardia), with warm, dry, flushed skin and no diaphoresis. This haemodynamic pattern is most consistent with:

(A) Hypovolaemic shock (haemorrhage)

(B) Neurogenic shock (loss of vasomotor tone)

(C) Septic shock (distributive)

(D) Obstructive shock (tension pneumothorax)

Q12. A 58-year-old man undergoes right hemicolectomy for a moderately differentiated adenocarcinoma of the ascending colon (pT3N1M0). Postoperatively, which serum tumour marker is most useful for monitoring recurrence and response to chemotherapy?

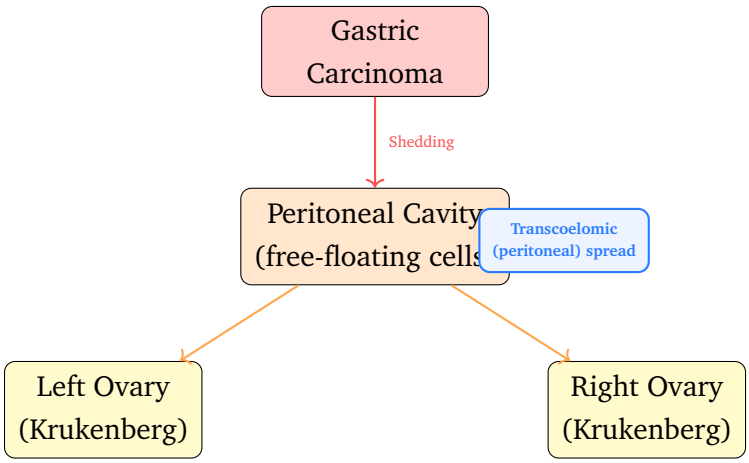
(A) CA-125 (ovarian cancer marker)

(B) PSA (prostate-specific antigen)

(C) AFP (α-fetoprotein)

(D) Carcinoembryonic antigen (CEA)

Q13. A 55-year-old woman with a known gastric carcinoma develops bilateral solid ovarian masses. Biopsy of the ovaries shows signet-ring cells filled with mucin within an abundant cellular stroma. The route by which the gastric tumour reached the ovaries, and the histological name given to these ovarian metastases, is best described in the diagram below. What is this route of spread?



(A) Transcoelomic (peritoneal) spread

(B) Haematogenous spread via portal vein

(C) Lymphatic spread to para-aortic nodes then ovary

(D) Direct invasion through the retroperitoneum

Q14. A 52-year-old man presents with episodic flushing, severe diarrhoea, and right-sided heart failure with tricuspid regurgitation. CT abdomen reveals a 2.5 cm mass at the terminal ileum with multiple hepatic metastases. Which investigation best **confirms** the diagnosis of carcinoid syndrome?

(A) Serum chromogranin A (elevated non-specifically)

(B) Plasma serotonin level

(C) 24-hour urinary 5-HIAA (5-hydroxyindoleacetic acid)

(D) Octreotide scan (localises tumour only)

Q15. A 35-year-old HIV-positive man presents with cervical and axillary lymphadenopathy, B symptoms, and an elevated eosinophil count. Lymph node biopsy shows Reed-Sternberg cells against a background of numerous eosinophils, plasma cells, lymphocytes, and histiocytes. No fibrous bands are seen. Which subtype of classical Hodgkin lymphoma is this?

(A) Nodular sclerosis (most common subtype overall)

(B) Mixed cellularity (prominent eosinophilia, EBV-associated)

(C) Lymphocyte-rich classical HL

(D) Lymphocyte-depleted (worst prognosis)

Q16. A 48-year-old woman's screening mammogram is unremarkable, but core biopsy of a vague thickening reveals lobular carcinoma *in situ* (LCIS). The small, uniform, discohesive neoplastic cells fill and distend the lobular acini. Immunohistochemistry demonstrates complete loss of membranous staining for which adhesion molecule, distinguishing LCIS from ductal CIS?

(A) HER-2/neu (c-erbB2)

(B) ER (oestrogen receptor)

(C) Ki-67 (proliferation index)

(D) E-cadherin (CDH1)

Q17. A 50-year-old man on long-term isoniazid therapy presents with fatigue, microcytic anaemia, and serum iron studies showing elevated serum ferritin with elevated transferrin saturation. Bone marrow aspirate stained with Perls' Prussian blue shows iron-laden mitochondria forming a ring around the erythroblast nucleus. These abnormal cells are called:

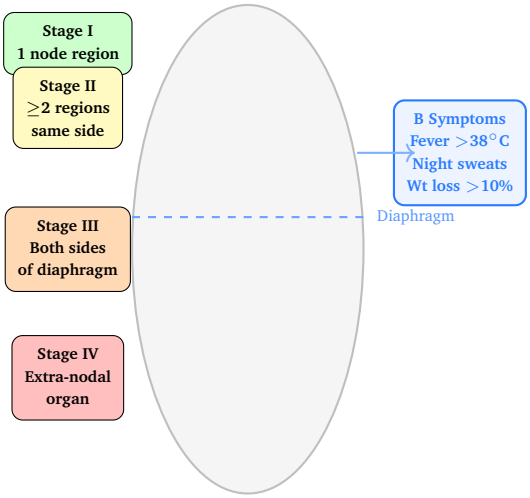
(A) Ringed sideroblasts

(B) Siderocytes (mature RBC with iron granules)

(C) Pappenheimer bodies on peripheral smear

(D) Basophilic stippling erythroblasts

Q18. A 26-year-old woman is newly diagnosed with Hodgkin lymphoma. Her staging workup includes assessment for "B symptoms", which affect the Ann Arbor stage designation (e.g., IIA vs IIB). The diagram below shows the Ann Arbor staging schema. Which constellation of findings defines **B symptoms**?



(A) Pruritus, fatigue, and bone pain

(B) Hepatosplenomegaly only

(C) Fever >38°C, drenching night sweats, and weight loss >10% of body weight

(D) Elevated ESR and serum LDH

Q19. A 32-year-old woman presents with the pentad of fever, microangiopathic haemolytic anaemia, thrombocytopenia, renal impairment, and fluctuating neurological deficits. Peripheral blood smear shows schistocytes. Coagulation studies (PT, aPTT, fibrinogen) are normal. Which mechanism best explains this condition?

(A) Factor VIII antibody causing haemophilia A

(B) ADAMTS13 deficiency leading to ultra-large vWF multimers (TTP)

(C) Complement-mediated red cell destruction (PNH)

(D) DIC with consumption of all clotting factors

Q20. An 8-year-old boy presents with haemarthrosis of the right knee following minor trauma. His maternal uncle had similar bleeding problems. Lab results show: prolonged aPTT, normal PT and bleeding time, platelet count normal. Mixing study corrects the aPTT. Factor assay reveals markedly reduced activity of:

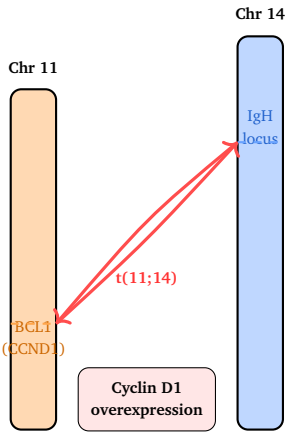
(A) Factor VIII (Haemophilia A)

(B) von Willebrand factor (vWD)

(C) Factor XI (Haemophilia C)

(D) Factor IX (Haemophilia B / Christmas disease)

Q21. A 62-year-old man presents with generalised lymphadenopathy, splenomegaly, and a lymphocyte count of 45,000/μL. Bone marrow biopsy reveals diffuse infiltration by small-to-medium lymphocytes. Immunophenotype: CD5+, CD19+, CD23–, FMC7+. Cyclin D1 is strongly overexpressed. The specific chromosomal translocation responsible for cyclin D1 overexpression is shown below. What is it?



(A) t(11;14)(q13;q32) – BCL1/IgH (cyclin D1 overexpression)

(B) t(14;18)(q32;q21) – BCL2/IgH (follicular lymphoma)

(C) t(8;14)(q24;q32) – MYC/IgH (Burkitt lymphoma)

(D) t(9;22)(q34;q11) – BCR-ABL (CML / Ph chromosome)

Q22. A 12-year-old girl presents with anaemia, intermittent jaundice, and splenomegaly. Her father has a similar history. Blood smear shows spherocytes without central pallor. Osmotic fragility test is increased. Mean corpuscular haemoglobin concentration (MCHC) is elevated (38 g/dL). The primary molecular defect in the commonest form of hereditary spherocytosis is:

(A) Deficiency of glucose-6-phosphate dehydrogenase (G6PD)

(B) Pyruvate kinase deficiency

(C) Spectrin or ankyrin deficiency (cytoskeletal protein)

(D) Haemoglobin S polymerisation

Q23. A 6-year-old child from an endemic area is found to have a calcified subpleural nodule in the lower zone/mid-zone of the right lung parenchyma on chest X-ray, with ipsilateral calcified hilar lymph nodes. This combination constitutes the Ghon complex of primary tuberculosis. Where is the **Ghon focus** (parenchymal component) typically located in primary pulmonary TB?

(A) Upper lobe apex (Simon's focus – secondary TB reactivation site)

(B) Lower lobe or mid-zone (subpleural) – primary TB Ghon focus

(C) Right main bronchus (endobronchial TB)

(D) Paravertebral soft tissue (Pott's disease)

Q24. A 30-year-old college student develops fever, dry cough, and mild dyspnoea two weeks after returning from a university dormitory. Chest X-ray shows bilateral patchy interstitial infiltrates more extensive than clinical signs suggest. Sputum Gram stain shows no organisms; cultures are negative at 48 hours. Cold agglutinin test is positive. The histological pattern in such cases shows:

(A) Alveolar exudate of fibrin and neutrophils (lobar pneumonia)

(B) Bronchopneumonia with focal bronchiolar plugging

(C) Caseous necrosis with granuloma formation

(D) Interstitial pneumonitis with mononuclear cell infiltrate (atypical pneumonia pattern)

Q25. A 60-year-old heavy smoker presents with a large peripheral lung mass (8 cm) on CT. Bronchoscopic biopsy shows sheets of large pleomorphic cells with vesicular nuclei and prominent nucleoli. No squamous pearls, no glandular formation, and TTF-1 / p40 / CK5-6 are all negative on IHC. The diagnosis is:

(A) Large cell undifferentiated carcinoma

(B) Squamous cell carcinoma (poorly differentiated)

(C) Adenocarcinoma (solid subtype)

(D) Mesothelioma (epithelioid variant)

Q26. A 45-year-old aerospace engineer who worked with rocket nozzles develops progressive dyspnoea, weight loss, and bilateral infiltrates on CT. Serum ACE is normal. Bronchoalveolar lavage lymphocyte transformation test using beryllium salts is **positive**. Lung biopsy shows non-caseating granulomas indistinguishable from sarcoidosis. This occupational lung disease is:

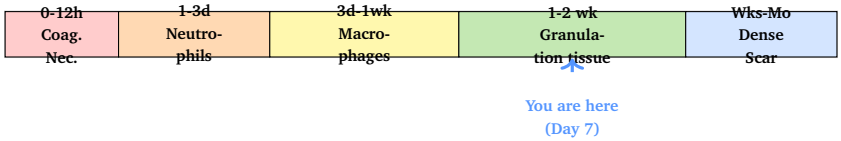
(A) Hypersensitivity pneumonitis (farmer's lung)

(B) Silicosis (silica inhalation)

(C) Berylliosis (chronic beryllium disease)

(D) Coal worker's pneumoconiosis (CWP)

Q27. A 55-year-old man suffers an acute ST-elevation MI. The diagram below summarises the sequential histopathological changes in the infarcted myocardium over time. At **one week** after the MI, which finding is most characteristic of the healing zone?



(A) Coagulative necrosis with preserved architecture (ghost fibres)

(B) Granulation tissue (macrophages clearing debris, fibroblasts, new capillaries)

(C) Dense collagenous scar (acellular fibrosis)

(D) Neutrophilic infiltrate at the infarct margins

Q28. A 50-year-old man with coronary artery disease dies suddenly after a fatal MI. At autopsy the culprit coronary plaque is found to have a thin fibrous cap, a large lipid-rich core, and abundant macrophage infiltration

at the plaque "shoulder". The most important mechanism by which these plaque macrophages contribute to **plaque instability and rupture** is:

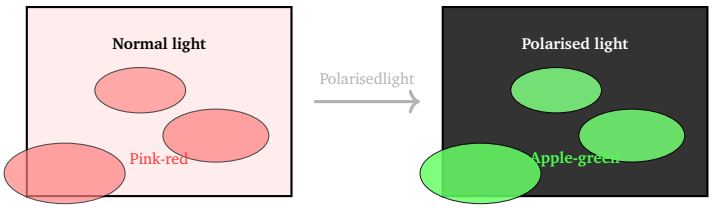
(A) Smooth muscle cell proliferation and collagen deposition

(B) Platelet adhesion to the exposed lipid core

(C) Foam cell formation from LDL oxidation

(D) Matrix metalloproteinase (MMP) secretion degrading the fibrous cap

Q29. A 72-year-old man with multiple myeloma develops progressive exertional dyspnoea and a restrictive pattern on echocardiography with "sparkling" granular myocardial texture. Endomyocardial biopsy is performed. The diagram below shows the appearance of the biopsy under two different light conditions. Which stain and finding confirm amyloid deposition?



(A) Congo red stain showing apple-green birefringence under polarised light

(B) PAS stain for glycogen (magenta positivity)

(C) Masson's trichrome showing collagen fibres (blue)

(D) Prussian blue stain for haemosiderin (iron)

Q30. A 48-year-old man with a 20-year history of heavy alcohol use presents with right upper quadrant pain, jaundice, and tender hepatomegaly. LFTs show AST:ALT ratio $>$ 2:1. Liver biopsy shows hepatocytes swollen with large, clear vacuoles displacing the nucleus to the periphery (macrovesicular steatosis), along with eosinophilic cytoplasmic inclusions (Mallory-Denk bodies) and a neutrophil-rich inflammatory infiltrate. This combination is diagnostic of:

(A) Non-alcoholic fatty liver disease (NAFLD/NASH)

(B) Haemochromatosis with hepatic iron overload

(C) Alcoholic steatohepatitis (macrovesicular steatosis + ballooning + Mallory bodies + PMN)

(D) Primary sclerosing cholangitis (PSC)

Q31. A 28-year-old man presents with recurrent crampy abdominal pain, non-bloody diarrhoea, weight loss, and perianal fistulae. Colonoscopy shows cobblestone mucosa with skip lesions; terminal ileum is thickened and narrowed (string sign on barium). Full-thickness bowel wall biopsy reveals transmural inflammation, fissuring ulcers, and discrete aggregates of epithelioid macrophages without central caseation. These aggregates are:

(A) Caseating granulomas (tuberculosis)

(B) Non-caseating granulomas (Crohn's disease – pathognomonic)

(C) Aschoff nodules (rheumatic fever)

(D) Palisading granulomas (rheumatoid nodule)

Q32. A 50-year-old woman presents with pruritus, fatigue, and gradually rising alkaline phosphatase over 3 years. She is anicteric. Liver biopsy shows granulomatous destruction of small intrahepatic bile ducts with a dense lymphoplasmacytic infiltrate. The most specific serum autoantibody for this condition is:

(A) Antinuclear antibody (ANA) – primary autoimmune hepatitis

(B) Anti-smooth muscle antibody (ASMA) – autoimmune hepatitis type 1

(C) pANCA – primary sclerosing cholangitis

(D) Anti-mitochondrial antibody (AMA) M2 subtype – primary biliary cholangitis

Q33. A 40-year-old man with a history of acute pancreatitis (alcohol-induced) 6 weeks ago now presents with persistent epigastric pain and early satiety. CT abdomen shows a 7 cm fluid collection in the lesser sac with a thick wall but no internal septa or solid components. Endoscopic

ultrasound-guided aspiration yields fluid with amylase $>$ 5,000 U/L. The wall consists of fibrous and granulation tissue with **no epithelial lining**. This lesion is:

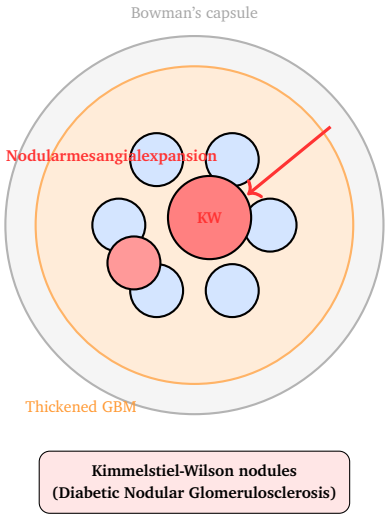
(A) Pancreatic pseudocyst (no epithelial lining, fibrous wall)

(B) Mucinous cystic neoplasm (MCN – with epithelial lining)

(C) Serous cystadenoma (microcystic, glycogen-rich cells)

(D) Intraductal papillary mucinous neoplasm (IPMN)

Q34. A 58-year-old man with type 2 diabetes mellitus of 20 years' duration has proteinuria 4+ and a serum creatinine of 3.5 mg/dL. Renal biopsy is performed. The diagram below shows a glomerulus from this patient. Identify the pathological lesion marked by the arrow.



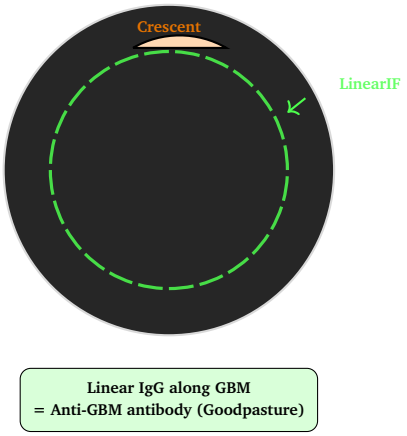
(A) Mesangial IgA deposits (IgA nephropathy – Berger's disease)

(B) Subepithelial immune complex humps (post-streptococcal GN)

(C) Kimmelstiel-Wilson nodules (diabetic nodular glomerulosclerosis)

(D) Wire-loop lesions (lupus nephritis class IV)

Q35. A 22-year-old man presents with haemoptysis and haematuria. Urine microscopy shows red cell casts. Renal biopsy shows crescentic glomerulonephritis. Immunofluorescence of the renal biopsy is shown in the diagram below. The staining pattern and the responsible antibody are:



(A) Granular "lumpy-bumpy" IgG (immune complex deposition – post-strep GN)

(B) Anti-GBM antibody (type II hypersensitivity) with linear IgG on IF – Goodpasture syndrome

(C) pANCA-associated vasculitis (ANCA-associated GN – no immune deposits, "pauci-immune")

(D) Mesangial IgA deposits (IgA nephropathy)

Q36. A 42-year-old man presents with flank pain, haematuria, and hypertension. His father died of a ruptured cerebral aneurysm. Ultrasound shows massively enlarged kidneys bilaterally with innumerable cysts of varying sizes. Liver cysts are also noted. Genetic analysis reveals a mutation in the gene encoding polycystin-1. Which condition and gene mutation does this represent?

(A) Autosomal recessive PKD (ARPKD) – PKHD1 gene mutation

(B) Von Hippel-Lindau disease – VHL gene mutation

(C) Tuberous sclerosis complex – TSC1/TSC2 mutation

(D) Autosomal dominant PKD (ADPKD) – PKD1/PKD2 gene mutation

Q37. A 45-year-old woman with a family history of phaeochromocytoma and hyperparathyroidism presents with a thyroid nodule. Fine-needle aspiration biopsy shows polygonal cells in an amyloid stroma that stains with Congo red. Calcitonin level is markedly elevated. Genetic testing reveals a germline point mutation in which proto-oncogene?

(A) RET proto-oncogene (MEN2A/MEN2B – medullary thyroid carcinoma)

(B) BRAF V600E (papillary thyroid carcinoma)

(C) RAS mutation (follicular thyroid carcinoma)

(D) PAX8-PPARγ fusion (follicular thyroid carcinoma)

Q38. A 40-year-old woman presents with resistant hypertension and persistent hypokalaemia (K^+ 2.8 mEq/L) despite potassium supplementation. She has no oedema. Plasma aldosterone:renin ratio is markedly elevated ($>$ 30). CT adrenal shows a 1.8 cm right adrenal nodule. The most likely diagnosis is:

(A) Secondary hyperaldosteronism (renovascular hypertension)

(B) Phaeochromocytoma (catecholamine excess)

(C) Primary hyperaldosteronism – Conn syndrome (adrenal adenoma)

(D) Cushing syndrome (cortisol excess)

Q39. A 32-year-old HIV-positive man (CD4 count 85 cells/μL) develops multiple violaceous, non-tender plaques on the lower extremities, palate, and gastrointestinal tract. Biopsy shows spindle cell proliferation forming slit-like vascular spaces with extravasated red blood cells and haemosiderin deposits. This AIDS-defining neoplasm is caused by:

(A) Epstein-Barr virus (EBV) – Burkitt lymphoma / DLBCL

(B) Human herpesvirus-8 (HHV-8 / KSHV) – Kaposi sarcoma

(C) HPV types 16/18 – squamous carcinoma

(D) Cytomegalovirus (CMV) – retinitis / colitis

Q40. A 55-year-old fair-skinned man with a history of extensive sun exposure presents with an irregularly pigmented, asymmetric lesion on his back that has grown rapidly over 3 months. Excision biopsy confirms malignant melanoma (Breslow thickness 2.8 mm, Clark level IV). Molecular testing is requested to guide targeted therapy. Which somatic mutation is found in approximately 50% of cutaneous melanomas and predicts response to vemurafenib?

(A) KIT mutation (acral lentiginous / mucosal melanoma)

(B) NRAS Q61K mutation

(C) NF1 loss-of-function mutation

(D) BRAF V600E mutation (serine/threonine kinase)

Detailed Solutions

Q1.

Solution

Concept – Liquefactive necrosis in brain: Brain tissue undergoes **liquefactive** (colliquative) necrosis after ischaemic infarction, unlike other tissues which show coagulative necrosis. This occurs because the brain has an unusually high water content ($\sim$80%), high lipid content (myelin), and abundant hydrolytic (proteolytic and lipolytic) enzymes from resident microglia and recruited macrophages. These enzymes rapidly digest the necrotic parenchyma, converting it into a fluid-filled cavity (cyst) over days to weeks.

Key Point: The combination of high water content, high lipid, and abundant lysosomal enzymes means the brain "digests itself" from within after ischaemia, producing liquefaction rather than a firm coagulum.

Why other options are wrong:

- Option B: A rich collagen framework (found in heart, liver) actually *resists* liquefaction and promotes coagulative necrosis.
- Option C: The circle of Willis and collateral supply are irrelevant to the *type* of necrosis that occurs.
- Option D: Fibrous astrocytes form the glial scar *after* liquefaction – they do not explain the type of necrosis.

Final Answer: High water and lipid content with abundant proteolytic enzymes $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – Apoptosis vs Necrosis: Apoptosis (programmed cell death) is characterised by: (1) cell shrinkage (condensation of cytoplasm), (2) chromatin condensation (pyknosis) and margination, (3) nuclear fragmentation (karyorrhexis), (4) formation of apoptotic bodies, and (5) **absence of inflammation**. In viral hepatitis, "Councilman bodies" (acidophil bodies) are individual apoptotic hepatocytes – deeply eosinophilic with condensed chromatin. Necrosis, by contrast, causes cell swelling, membrane rupture, and inflammation.

Key Point: Absence of surrounding inflammation is a hallmark of apoptosis; the

apoptotic cells are rapidly phagocytosed by neighbouring cells or macrophages without releasing their contents.

Why other options are wrong:

- Option A: Coagulative necrosis preserves ghost outlines but is accompanied by cellular swelling and inflammatory response.
- Option B: Caseous necrosis is a feature of TB granulomas with complete loss of architecture and "cheese-like" gross appearance.
- Option D: Liquefactive necrosis is seen in brain infarcts and bacterial abscesses (suppuration).

Final Answer: Cell shrinkage with chromatin condensation (apoptosis) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – BPH and DHT: Benign prostatic hyperplasia (BPH) involves glandular and stromal hyperplasia predominantly in the **transition zone** of the prostate. The key hormonal mediator is **dihydrotestosterone (DHT)**, produced from testosterone by **5-alpha-reductase** within prostatic stromal cells. DHT binds androgen receptors in both stromal and epithelial cells, driving hyperplasia. 5-alpha-reductase inhibitors (finasteride, dutasteride) are used therapeutically.

Key Point: Men with congenital 5-alpha-reductase deficiency do not develop BPH despite normal testosterone levels, confirming the central role of DHT.

Why other options are wrong:

- Option A: Testosterone itself has a weak effect on the prostate – it is the converted form (DHT) that is active.
- Option C: Oestradiol may contribute to stromal proliferation synergistically but is not the primary driver.
- Option D: FSH acts on the testis (spermatogenesis) and has no direct role in prostatic hyperplasia.

Final Answer: Dihydrotestosterone (DHT) $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – Leukocyte Adhesion Cascade (Selectins): Leukocyte recruitment to sites of inflammation follows a multistep process. **Step 1 (Rolling)** is mediated by **selectins**: **E-selectin** (CD62E) and P-selectin (CD62P) on the endothelium bind sialyl-Lewis^x (CD15s) on leukocytes. E-selectin is induced within hours by IL-1 and TNF-α. P-selectin is rapidly redistributed from Weibel-Palade bodies. Firm adhesion (Step 3) requires integrins (LFA-1/Mac-1) binding ICAM-1.

Key Point: Rolling is the first step; without selectin-mediated rolling, subsequent firm adhesion and diapedesis cannot occur. E-selectin and P-selectin are the endothelial molecules responsible.

Why other options are wrong:

- Option A: ICAM-1 mediates firm adhesion (Step 3), not rolling.
- Option B: VCAM-1 binds VLA-4 on lymphocytes and eosinophils during firm adhesion, not the initial rolling step.
- Option C: IL-8 is a chemokine that activates integrins (Step 2 – activation), not rolling.

Final Answer: E-selectin (endothelial selectin mediates rolling) $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q4

Q5.

Solution

Concept – Fibrinous Peritonitis: When acute inflammation affects serosal surfaces (peritoneum, pleura, pericardium) the exudate is rich in **fibrin**. In perforated appendicitis, bowel contents (bacteria + enzymes) released into the peritoneal cavity stimulate a massive acute inflammatory response with fibrin deposition over all peritoneal surfaces. The exudate consists of fibrin threads entrapping neutrophils – classified as **fibrinous (or fibrinopurulent) peritonitis**. If fibrin is not dissolved, organisation leads to fibrous adhesions.

Key Point: Fibrinous exudate = fibrin is the major component. Serous = protein-rich fluid without much fibrin (e.g., early viral pleuritis). Pseudomembranous = a surface membrane of fibrin + necrotic epithelium (C. difficile colitis).

Why other options are wrong:

- Option B: Serous pleuritis produces a thin, watery exudate, not the thick

fibrin coat described.

- Option C: Pseudomembranous colitis is specific to the colonic mucosa (Clostridioides difficile toxin).
- Option D: Granulomatous appendicitis (Crohn's) shows non-caseating granulomas, not fibrinous exudate.

Final Answer: Fibrinous peritonitis $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – Sarcoidosis and Schaumann Bodies: Sarcoidosis is a systemic granulomatous disease of unknown aetiology characterised by **non-caseating granulomas**. Within the giant cells (Langhans type) of these granulomas, two types of cytoplasmic inclusions may be seen: (1) **Schaumann bodies** – laminated, concentrically calcified (conchoidal) bodies; (2) **Asteroid bodies** – star-shaped eosinophilic inclusions. Neither is pathognomonic alone but both are characteristic. The diagnosis also requires compatible clinical/radiological findings and exclusion of infection.

Key Point: Michaelis-Gutmann bodies are seen in *malakoplakia* (not sarcoidosis); Aschoff bodies are in *rheumatic fever* myocarditis.

Why other options are wrong:

- Option A: Michaelis-Gutmann bodies are basophilic calcium-phosphate concretions in macrophages in malakoplakia of the bladder.
- Option B: Asteroid bodies are the non-calcified, star-shaped inclusions; Schaumann bodies are the calcified conchoidal ones – the question specifies "laminated, concentrically calcified."
- Option D: Aschoff bodies are perivascular granulomas in the myocardium in acute rheumatic fever, not the lung.

Final Answer: Schaumann bodies (conchoidal calcifications in macrophages) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q6

Q7.

Solution

Concept – Wound Contraction (Myofibroblasts): During wound healing by secondary intention, **wound contraction** reduces the wound area by up to 80% within 2 weeks. This is mediated by **myofibroblasts** – modified fibroblasts that express **α-smooth muscle actin (α-SMA)** and develop contractile stress fibres. They are derived from local fibroblasts under TGF-β_1 stimulation. Myofibroblasts also synthesise type I collagen for scar formation.

Key Point: Myofibroblasts = fibroblasts + smooth muscle properties. They are the key cell in wound contraction, keloid formation, and organ fibrosis (liver cirrhosis, renal fibrosis).

Why other options are wrong:

- Option A: Fibroblasts produce collagen but lack the contractile apparatus (α-SMA) needed for wound contraction.
- Option C: Endothelial cells form new capillaries (angiogenesis) – essential for granulation tissue but not contraction.
- Option D: Mast cells release histamine and heparin in early acute inflammation, not wound contraction.

Final Answer: Myofibroblasts (α-SMA positive) $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q7

Q8.

Solution

Concept – Endogenous Pyrogens and Fever: IL-1 and TNF-α are the principal endogenous pyrogens released by activated macrophages. They act on the **hypothalamus** (specifically the circumventricular organ OVLT, which lacks a blood-brain barrier) to stimulate the enzyme **cyclooxygenase-2 (COX-2)**, producing **prostaglandin E2 (PGE2)**. PGE2 acts on hypothalamic neurons via EP3 receptors, raising the thermostat set-point and causing fever. Aspirin and NSAIDs reduce fever by inhibiting COX-2 and thus PGE2 synthesis.

Key Point: The fever cascade: macrophage $\rightarrow$ IL-1/TNF $\rightarrow$ OVLT $\rightarrow$ COX-2 $\rightarrow$ PGE2 $\rightarrow$ hypothalamic set-point $\uparrow$.

Why other options are wrong:

- Option A: LTB4 is a powerful neutrophil chemoattractant but is not the hy-

pothalamic mediator of fever.

- Option B: PAF (platelet-activating factor) is a lipid mediator of platelet aggregation and bronchoconstriction, not the fever mediator.
- Option C: IL-6 also contributes to the acute phase response and is pyrogenic, but the direct hypothalamic mediator is PGE2.

Final Answer: Prostaglandin E2 (PGE2) in the hypothalamus $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q8

Q9.

Solution

Concept – Post-operative DVT (Virchow's Triad): Deep vein thrombosis after surgery is explained by **Virchow's triad**: (1) **Venous stasis** – immobility during and after surgery causes stagnation of blood in the calf veins, promoting thrombus formation; (2) **Hypercoagulability** – surgery activates the coagulation cascade (tissue factor release), increases acute phase proteins including fibrinogen, and reduces natural anticoagulants (protein C, antithrombin). Together these two factors dominate in post-operative DVT. Endothelial injury plays a lesser role unless there is direct venous trauma.

Key Point: Prophylaxis targets both components: LMWH (reduces hypercoagulability) + compression stockings / early mobilisation (reduces stasis).

Why other options are wrong:

- Option B: Endothelial injury alone without stasis/hypercoagulability is insufficient for post-operative DVT; the question asks for the most prominent factors.
- Option C: Factor V Leiden (inherited thrombophilia) increases risk but is not the mechanism operating in all post-operative patients.
- Option D: Arterial turbulence is a risk for arterial thrombosis (atherosclerotic plaques), not venous DVT.

Final Answer: Venous stasis and hypercoagulability $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – Amniotic Fluid Embolism (AFE): AFE is a rare but catastrophic complication occurring during labour, delivery, or immediately postpartum. Amniotic fluid (containing fetal squamous cells, lanugo hair, vernix caseosa, mucin, meconium) enters the maternal circulation through disrupted uterine vessels, lodging in pulmonary capillaries and triggering: (1) acute respiratory failure, (2) cardiovascular collapse, and (3) DIC. The pathognomonic autopsy finding is the presence of **squamous cells, lanugo hairs, and mucin in pulmonary vessels**.

Key Point: AFE = squamous cells/lanugo in pulmonary vessels + DIC. Mortality is extremely high (>50%).

Why other options are wrong:

- Option A: Pulmonary thromboembolism shows fibrin-platelet thrombus – no squamous cells or lanugo.
- Option B: Fat embolism occurs after long bone fractures and shows fat globules in pulmonary vessels (Oil Red O positive), not squamous cells.
- Option D: Air embolism shows gas bubbles in cardiac chambers/vessels, not squamous cells or lanugo.

Final Answer: Amniotic fluid embolism $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q10

Q11.

Solution

Concept – Neurogenic Shock: Neurogenic shock results from sudden loss of **sympathetic vasomotor tone** following spinal cord injury above T6. Without sympathetic input, peripheral arterioles dilate massively, causing hypotension. Uniquely, the heart rate does **not** increase (no compensatory tachycardia) because cardiac sympathetics are also interrupted – leading to **bradycardia with hypotension**. Skin is warm, dry, and flushed (vasodilated) – the opposite of hypovolaemic shock (cold, clammy, tachycardic).

Key Point: Neurogenic shock = hypotension + bradycardia + warm/flushed skin. This triad distinguishes it from all other shock types (which cause tachycardia).

Why other options are wrong:

- Option A: Hypovolaemic shock causes tachycardia, cold clammy skin, and

peripheral vasoconstriction.

- Option C: Septic shock (distributive) has tachycardia with warm skin initially (hyperdynamic phase) – but not bradycardia.
- Option D: Obstructive shock (tension pneumothorax) causes tachycardia and deviated trachea, not bradycardia.

Final Answer: Neurogenic shock (loss of vasomotor tone) $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – CEA in Colorectal Cancer: Carcinoembryonic antigen (CEA) is a glycoprotein normally expressed during fetal development that is re-expressed in adenocarcinomas of the colon, lung, breast, and pancreas. It is the principal tumour marker used for: (1) monitoring response to chemotherapy, and (2) early detection of recurrence after curative resection. A rising CEA post-operatively mandates imaging to look for liver metastases or local recurrence. CEA is not useful for *screening* (low sensitivity/specificity) but is invaluable for *surveillance*.

Key Point: Pre-operative CEA level also has prognostic value – elevated pre-op CEA correlates with worse prognosis.

Why other options are wrong:

- Option A: CA-125 is the marker for epithelial ovarian carcinoma.
- Option B: PSA is specific to prostate cancer.
- Option C: AFP is elevated in hepatocellular carcinoma, testicular germ cell tumours, and yolk sac tumours.

Final Answer: Carcinoembryonic antigen (CEA) $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q12

Q13.

Solution

Concept – Transcoelomic (Peritoneal) Spread and Krukenberg Tumour: Some carcinomas spread by **transcoelomic** seeding – cells exfoliate from the primary tumour surface, float freely in a body cavity fluid (peritoneal/pleural), and implant on distant serosal surfaces. The classic example is **Krukenberg tumour**: bilat-

eral ovarian metastases from a primary gastric (most common), colonic, or breast carcinoma, characterised histologically by **signet-ring cells** (mucin-filled, nucleus pushed to periphery) in a cellular (desmoplastic) ovarian stroma.

Key Point: Bilateral solid ovarian tumours in a patient with GI malignancy = Krukenberg tumour = transcoelomic spread.

Why other options are wrong:

- Option B: Haematogenous spread via portal vein would give *hepatic* metastases first, not bilateral ovarian masses directly.
- Option C: Lymphatic spread to para-aortic nodes is the primary lymphatic route; reaching the ovary in this way requires retrograde flow.
- Option D: Direct invasion cannot explain bilateral ovarian involvement from a stomach primary.

Final Answer: Transcoelomic (peritoneal) spread $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q13

Q14.

Solution

Concept – Carcinoid Syndrome and 5-HIAA: Carcinoid tumours of the GI tract secrete **serotonin (5-HT)**, kinins, and prostaglandins. While serotonin from intestinal carcinoids is normally inactivated in the liver (first-pass effect), **hepatic metastases** allow serotonin to bypass hepatic metabolism and enter the systemic circulation, causing carcinoid syndrome: episodic flushing, diarrhoea, bronchospasm, and **right-sided cardiac fibrosis** (tricuspid + pulmonary valves). Serotonin is metabolised to **5-hydroxyindoleacetic acid (5-HIAA)**, excreted in urine. A **24-hour urinary 5-HIAA** $>$ 30 mg/day confirms the diagnosis.

Key Point: 5-HIAA is the gold-standard *biochemical* confirmatory test for carcinoid syndrome; chromogranin A is used for monitoring tumour burden.

Why other options are wrong:

- Option A: Chromogranin A is a neuroendocrine marker elevated in many GEP-NEN tumours but is less specific for carcinoid *syndrome*.
- Option B: Plasma serotonin is labile and difficult to measure accurately; 24h urinary 5-HIAA is more reliable.
- Option D: Octreotide scan localises tumour but does not biochemically confirm carcinoid *syndrome*.

Final Answer: 24-hour urinary 5-HIAA $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q14

Q15.

Solution

Concept – Mixed Cellularity Hodgkin Lymphoma: Classical Hodgkin lymphoma (cHL) has four subtypes. **Mixed cellularity (MCCHL)** is characterised by: (1) Reed-Sternberg cells against a mixed cellular background including **prominent eosinophils**, plasma cells, lymphocytes, and histiocytes; (2) No fibrous bands (distinguishing it from nodular sclerosis); (3) Strong association with **EBV** (∼75%) and HIV; (4) More common in developing countries and older/immunocompromised patients. It is the second most common subtype overall (after nodular sclerosis).

Key Point: Mixed cellularity = no bands + eosinophilia + EBV/HIV association. Nodular sclerosis = bands of fibrosis + lacunar cells.

Why other options are wrong:

- Option A: Nodular sclerosis shows fibrous bands dividing lymph node into nodules and lacunar RS cells – no fibrous bands seen here.
- Option C: Lymphocyte-rich cHL has a background mainly of lymphocytes with few RS cells and minimal eosinophils.
- Option D: Lymphocyte-depleted has paucity of lymphocytes with many RS cells; worst prognosis and very rare.

Final Answer: Mixed cellularity subtype (prominent eosinophilia, EBV-associated) $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q15

Q16.

Solution

Concept – LCIS and E-cadherin Loss: Lobular carcinoma *in situ* (LCIS) and invasive lobular carcinoma (ILC) are characterised at the molecular level by **loss of E-cadherin (CDH1)** expression, a transmembrane glycoprotein responsible for cell-cell adhesion. Loss of E-cadherin results in the characteristic discohesive, "single-file" (Indian-file) growth pattern. On IHC, LCIS cells are **E-cadherin negative** (no membranous staining), whereas ductal lesions are E-cadherin positive.

This distinction is diagnostically important.

Key Point: E-cadherin IHC is the key test to distinguish LCIS (negative) from ductal CIS (positive) when morphology is ambiguous.

Why other options are wrong:

- Option A: HER-2/neu overexpression is more characteristic of ductal (luminal B / HER2-enriched) carcinomas, not LCIS.
- Option B: LCIS is typically ER-positive, but ER expression is not the distinguishing marker from DCIS.
- Option C: Ki-67 is a proliferation marker that does not distinguish LCIS from DCIS.

Final Answer: Loss of E-cadherin (CDH1) expression $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q16

Q17.

Solution

Concept – Sideroblastic Anaemia and Ringed Sideroblasts: Sideroblastic anaemia is characterised by iron-laden mitochondria that form a ring around the erythroblast nucleus, visible as **ringed sideroblasts** on Perls' Prussian blue-stained bone marrow aspirate ($\geq$5 iron granules encircling $>$1/3 of the nucleus). Causes include: acquired (alcohol, isoniazid – inhibits pyridoxal phosphate, lead poisoning) and hereditary (X-linked, ALAS2 mutation). Despite iron overload (elevated ferritin, transferrin saturation), haemoglobin synthesis is defective because protoporphyrin IX synthesis is impaired.

Key Point: Isoniazid causes sideroblastic anaemia by inhibiting vitamin B6 (pyridoxal phosphate), a cofactor for the enzyme ALA synthase in haem synthesis. Treatment: pyridoxine supplementation.

Why other options are wrong:

- Option B: Siderocytes are mature RBCs with scattered (non-ringed) iron granules (Pappenheimer bodies on Romanowsky stain).
- Option C: Pappenheimer bodies are the Romanowsky-stained equivalent of siderotic granules in RBCs, not the bone marrow erythroblast pathology.
- Option D: Basophilic stippling in RBCs reflects ribosomal RNA aggregation (lead poisoning, thalassaemia) – not ringed sideroblasts.

Final Answer: Ringed sideroblasts $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q17

Q18.

Solution

Concept – Hodgkin Lymphoma B Symptoms: In the **Ann Arbor staging** system for Hodgkin lymphoma, the suffix **"B"** is added when any of the following systemic symptoms are present: (1) **Fever** $> 38^{\circ}$C (unexplained, recurrent); (2) **Drenching night sweats** (requiring change of clothes/bedding); (3) **Unexplained weight loss** $>$ 10% of body weight in the preceding 6 months. B symptoms indicate a worse prognosis and influence treatment intensity. Their absence = suffix "A."

Key Point: The three B symptoms are the "3 W's": Warming (fever), Wetting (night sweats), Wasting (weight loss >10%).

Why other options are wrong:

- Option A: Pruritus is common in HL but is NOT an official B symptom by Ann Arbor criteria; fatigue and bone pain also do not qualify.
- Option B: Hepatosplenomegaly denotes E-site or extranodal involvement, not B symptoms per se.
- Option D: Elevated ESR and LDH are adverse prognostic markers in the International Prognostic Score (IPS) but are laboratory values, not clinical B symptoms.

Final Answer: Fever >38°C, drenching night sweats, and weight loss >10% $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q18

Q19.

Solution

Concept – TTP and ADAMTS13 Deficiency: Thrombotic thrombocytopenic purpura (TTP) presents with the classic pentad: fever, microangiopathic haemolytic anaemia (MAHA), thrombocytopenia, renal dysfunction, and **neurological symptoms**. The underlying mechanism is deficiency of **ADAMTS13**, a metalloprotease that normally cleaves ultra-large von Willebrand factor (UL-vWF) multimers. Without ADAMTS13, UL-vWF accumulates, causing spontaneous platelet aggregation and microthrombi in small vessels. Crucially, PT, aPTT, and fibrinogen are **normal** (differentiating TTP from DIC).

Key Point: TTP vs HUS: TTP has predominantly neurological features and low ADAMTS13; HUS is predominantly renal (children post-STEC infection, complement dysregulation).

Why other options are wrong:

- Option A: Factor VIII antibody causes haemophilia A (prolonged aPTT, no microangiopathic haemolysis).
- Option C: PNH (paroxysmal nocturnal haemoglobinuria) shows complement-mediated intravascular haemolysis + venous thrombosis but not the full pentad of TTP.
- Option D: DIC consumes all clotting factors, producing prolonged PT/aPTT, low fibrinogen – not seen here.

Final Answer: ADAMTS13 deficiency leading to ultra-large vWF multimers (TTP) $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q19

Q20.

Solution

Concept – Haemophilia B (Factor IX Deficiency): Haemophilia B (Christmas disease) is an **X-linked recessive** disorder caused by deficiency of **Factor IX** (plasma thromboplastin component). Like Haemophilia A (Factor VIII deficiency), it presents with haemarthroses, deep muscle haematomas, and prolonged aPTT with normal PT and platelet count. The mixing study corrects the aPTT (factor deficiency, not inhibitor). Family history of maternal uncle affected is classic X-linked inheritance. Haemophilia B is 5 times less common than Haemophilia A.

Key Point: Both Haemophilia A and B: X-linked recessive, prolonged aPTT, normal PT/BT/platelets, haemarthrosis. Distinguished only by factor assay.

Why other options are wrong:

- Option A: Factor VIII deficiency = Haemophilia A (same clinical picture, 5× more common).
- Option B: vWD (especially type 1) causes prolonged bleeding time and mucocutaneous bleeding, not haemarthrosis.
- Option C: Factor XI deficiency (Haemophilia C) is autosomal recessive, mild, and more common in Ashkenazi Jews; haemarthrosis is uncommon.

Final Answer: Factor IX deficiency (Haemophilia B / Christmas disease) $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q20

Q21.

Solution

Concept – Mantle Cell Lymphoma and t(11;14): Mantle cell lymphoma (MCL) is an aggressive B-cell lymphoma with a pathognomonic **t(11;14)(q13;q32)** translocation that juxtaposes the **BCL1 (CCND1/cyclin D1)** gene on chromosome 11 with the **IgH enhancer** on chromosome 14, causing constitutive **cyclin D1 overexpression**. Cyclin D1 promotes G1→S cell cycle progression. Immunophenotype: $CD5^+$, $CD19^+$, $CD23^-$, $FMC7^+$, cyclin $D1^+$. MCL typically presents at an advanced stage and has a median survival of 3–5 years.

Key Point: t(11;14) + cyclin D1 = Mantle cell lymphoma. $CD5^+CD23^-$ distinguishes MCL from CLL ($CD5^+CD23^+$).

Why other options are wrong:

- Option B: t(14;18) BCL2/IgH → follicular lymphoma (overexpresses BCL-2, anti-apoptotic).
- Option C: t(8;14) MYC/IgH → Burkitt lymphoma (c-MYC overexpression, starry-sky pattern).
- Option D: t(9;22) BCR-ABL → CML (Philadelphia chromosome – a myeloid neoplasm, not lymphoma).

Final Answer: t(11;14)(q13;q32) – BCL1/IgH (cyclin D1 overexpression) $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q21

Q22.

Solution

Concept – Hereditary Spherocytosis (Spectrin/Ankyrin Deficiency): Hereditary spherocytosis (HS) is the most common inherited haemolytic anaemia in Northern Europeans (autosomal dominant in ∼75%). The primary defect is in cytoskeletal membrane proteins, most commonly **spectrin** (alone or with ankyrin or protein 4.2). Loss of cytoskeletal support causes the RBC to lose membrane surface area while retaining volume, forming a sphere with no central pallor and **elevated MCHC**. Spherocytes are trapped and destroyed in the splenic sinusoids.

Key Point: HS triad: spherocytes on smear + elevated osmotic fragility + splenomegaly + elevated MCHC. Negative direct Coombs (distinguishes from au-

toimmune haemolytic anaemia).

Why other options are wrong:

- Option A: G6PD deficiency causes episodic, oxidant-triggered haemolysis with bite cells and Heinz bodies, not spherocytes.
- Option B: Pyruvate kinase deficiency is autosomal recessive and causes haemolysis with echinocytes/burr cells, elevated 2,3-BPG.
- Option D: HbS polymerisation (sickle cell disease) produces sickle-shaped cells, not spherocytes.

Final Answer: Spectrin (or ankyrin) deficiency $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q22

Q23.

Solution

Concept – Primary TB Ghon Complex: In **primary pulmonary tuberculosis**, inhaled *M. tuberculosis* bacilli reach the well-ventilated **lower lobe or mid-zone subpleural parenchyma** (where airflow is greatest), producing the **Ghon focus** – a small (1–1.5 cm) subpleural caseous granuloma. Draining lymphatics carry bacilli to the ipsilateral hilar/paratracheal lymph nodes (Ghon complex = Ghon focus + draining lymph nodes). In secondary (reactivation) TB, the lesion is characteristically in the **upper lobe apex** (Simon's foci – haematogenously seeded during primary infection).

Key Point: Primary TB = lower/mid-zone subpleural Ghon focus + hilar nodes. Secondary TB = upper lobe apical fibrocaseous cavitary disease.

Why other options are wrong:

- Option A: Upper lobe apex is the site of Simon's foci (haematogenous seeding) and secondary/reactivation TB, not the primary Ghon focus.
- Option C: Endobronchial TB is a complication of primary TB due to lymph node erosion into bronchi – not the primary parenchymal focus.
- Option D: Paravertebral soft tissue involvement (Pott's disease) is skeletal TB from haematogenous spread.

Final Answer: Lower lobe or mid-zone (subpleural) – primary TB Ghon focus $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q23

Q24.

Solution

Concept – Atypical/Viral Pneumonia (Interstitial Pattern): Atypical pneumonia (caused by *Mycoplasma pneumoniae*, respiratory viruses, *Chlamydophila*, *Legionella*) shows an **interstitial pneumonitis pattern**: inflammation primarily in the alveolar walls and interstitium, with a **mononuclear cell** (lymphocytic/histiocytic) infiltrate rather than alveolar neutrophilic exudate. No bacteria are seen on Gram stain; cold agglutinins (>1:64) are positive in *Mycoplasma* infection. This "walking pneumonia" is clinically milder than typical lobar pneumonia.

Key Point: Atypical pneumonia = interstitial pattern + mononuclear cells + positive cold agglutinin test (Mycoplasma). Classic/typical pneumonia = alveolar exudate + neutrophils + consolidation.

Why other options are wrong:

- Option A: Alveolar fibrin + neutrophil exudate describes lobar pneumonia (pneumococcal – red/grey hepatisation).
- Option B: Bronchopneumonia has patchy bronchiolar/peribronchiolar neutrophilic plugging (staphylococcal, gram-negative).
- Option C: Caseous necrosis with granulomas is TB, not atypical pneumonia.

Final Answer: Interstitial pneumonitis with mononuclear cell infiltrate (atypical pneumonia pattern) $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q24

Q25.

Solution

Concept – Large Cell Undifferentiated Carcinoma of Lung: Large cell carcinoma (LCC) is a diagnosis of exclusion among non-small cell lung carcinomas (NSCLC). It is defined by: (1) large pleomorphic cells with vesicular nuclei and prominent nucleoli; (2) **no squamous differentiation** (no keratinisation, no intercellular bridges – p40/CK5-6 negative); (3) **no glandular differentiation** (no acini, no mucin – TTF-1 negative); (4) not neuroendocrine (synaptophysin/chromogranin negative). It tends to be peripheral, bulky, and rapidly growing.

Key Point: LCC = all IHC markers negative ($p40^-$, $TTF\text{-}1^-$, $CK5\text{-}6^-$, $synaptophysin^-$). It carries a poor prognosis and has the highest mitotic rate among NSCLC subtypes.

Why other options are wrong:

- Option B: Poorly differentiated SCC would show p40 and/or CK5-6 positivity even focally.
- Option C: Adenocarcinoma solid subtype is TTF-1 or Napsin A positive.
- Option D: Mesothelioma is positive for WT1, calretinin, and CK5/6 – and is pleural-based, not a lung parenchymal mass.

Final Answer: Large cell undifferentiated carcinoma $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q25

Q26.

Solution

Concept – Berylliosis (Chronic Beryllium Disease): Chronic beryllium disease (CBD) is an occupational granulomatous lung disease caused by beryllium inhalation (aerospace, nuclear, ceramics industries). It is a **delayed-type (type IV) hypersensitivity** reaction. Pathologically, CBD is *indistinguishable* from sarcoidosis (non-caseating granulomas, bilateral infiltrates). The key differentiating test is the **beryllium lymphocyte proliferation test (BeLPT)** – lymphocytes from BAL or blood proliferate in vitro when exposed to beryllium salts (positive = sensitisation/CBD). Serum ACE may be normal (unlike sarcoidosis, where it is often elevated).

Key Point: CBD vs sarcoidosis: identical histology, but CBD has a positive BeLPT and occupational beryllium exposure history. ACE may be normal in CBD.

Why other options are wrong:

- Option A: Hypersensitivity pneumonitis (farmer's lung, bird fancier's lung) shows bronchiolocentric pattern with poorly formed granulomas + lymphocytic bronchiolitis – not identical to sarcoid.
- Option B: Silicosis shows silicotic nodules (birefringent particles under polarised light, "onion-skin" collagen), not sarcoid-like granulomas.
- Option D: CWP shows coal macules with focal emphysema; massive fibrosis in progressive massive fibrosis (PMF).

Final Answer: Berylliosis (chronic beryllium disease) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q26

Q27.

Solution

Concept – Myocardial Infarction Healing Timeline: The sequential histopathological changes after MI follow a predictable timeline: **0–12 hours**: coagulative necrosis (wavy fibres, but architecture preserved – "ghost fibres"); **1–3 days**: neutrophilic infiltration; **3–7 days**: macrophage influx (phagocytosis of necrotic debris); **1–2 weeks**: **granulation tissue** (macrophages clearing debris + fibroblast proliferation + new capillary ingrowth + collagen deposition begins); **weeks–months**: dense fibrous scar (pale, white, acellular collagen – highest risk of cardiac rupture is at 3–7 days).

Key Point: At Day 7 (1 week): granulation tissue is the dominant finding. Granulation tissue = macrophages + fibroblasts + new capillaries (angiogenesis).

Why other options are wrong:

- Option A: Coagulative necrosis (ghost fibres) is seen in the first 12–24 hours, not at 1 week.
- Option C: Dense collagenous scar takes weeks to months to form – not present at Day 7.
- Option D: Neutrophilic infiltrate peaks at 1–3 days and is resolving by Day 7 as macrophages take over.

Final Answer: Granulation tissue formation (macrophages + fibroblasts + new capillaries) $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q27

Q28.

Solution

Concept – Vulnerable Plaque and MMP: A **vulnerable (unstable) plaque** is at high risk of rupture and has three key features: (1) a large lipid core; (2) a thin fibrous cap; (3) a heavy infiltrate of inflammatory cells (macrophages/foam cells) at the plaque **shoulder region**. Activated macrophages secrete **matrix metalloproteinases (MMPs)** – collagenases, gelatinases, stromelysins – which degrade the fibrous cap collagen, thinning and weakening it until rupture occurs. Plaque rupture exposes the thrombogenic lipid core and subendothelial collagen to blood, triggering acute thrombosis.

Key Point: Vulnerable plaque = thin cap + large lipid core + macrophage-derived MMPs at the shoulder. MMPs are the enzymes that cause cap degradation and

rupture.

Why other options are wrong:

- Option A: Smooth muscle cell proliferation and collagen deposition *stabilise* the plaque (stable, fibrous plaques are less likely to rupture).
- Option B: Platelet adhesion occurs *after* plaque rupture – it is the consequence, not the mechanism of rupture.
- Option C: Foam cell formation (LDL oxidation) creates the lipid core – important in plaque *formation*, not the immediate mechanism of cap rupture.

Final Answer: Matrix metalloproteinase (MMP) secretion degrading the fibrous cap $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q28

Q29.

Solution

Concept – Amyloid and Congo Red Staining: Amyloid is an extracellular deposit of misfolded proteins arranged in anti-parallel β-pleated sheet fibrils. In cardiac amyloidosis (AL amyloid from myeloma light chains, or TTR transthyretin amyloid in elderly), the diagnostic stain is **Congo red**: amyloid appears **pink-red** under normal light and shows **apple-green birefringence** under **polarised light** – a pathognomonic finding due to the ordered β-sheet fibril arrangement bending polarised light. This distinguishes amyloid from collagen and other eosinophilic deposits.

Key Point: Congo red + polarised light = apple-green birefringence = amyloid. This is the gold-standard staining method.

Why other options are wrong:

- Option B: PAS stain (magenta) identifies polysaccharides/glycogen – not amyloid.
- Option C: Masson's trichrome stains collagen blue (fibrosis) – amyloid does not stain blue with trichrome.
- Option D: Prussian blue/Perls' stain is for iron (haemosiderin) – used in haemochromatosis and haemosiderosis.

Final Answer: Congo red stain showing apple-green birefringence under polarised light $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q29

Q30.

Solution

Concept – Alcoholic Steatohepatitis (ASH): The histological triad of **alcoholic steatohepatitis** is: (1) **Macrovesicular steatosis** – large lipid vacuoles displacing the nucleus (from impaired VLDL secretion and increased lipogenesis); (2) **Ballooning degeneration** with **Mallory-Denk bodies** (eosinophilic cytoplasmic inclusions of ubiquitinated cytokeratin 8/18); (3) **Neutrophilic (PMN) infiltrate** (unlike NASH which has predominantly mononuclear infiltrate). AST:ALT $>$ 2:1 is a classic biochemical clue due to mitochondrial AST release and B6 depletion impacting ALT synthesis.

Key Point: Mallory-Denk bodies + neutrophilic infiltrate + macrovesicular steatosis = ASH. NASH is morphologically similar but without prominent Mallory bodies and has mononuclear infiltrate.

Why other options are wrong:

- Option A: NAFLD/NASH shows similar steatosis and ballooning but typically lacks Mallory-Denk bodies, has a mononuclear infiltrate, and the clinical history is non-alcoholic.
- Option B: Haemochromatosis shows iron overload (Prussian blue positive) without macrovesicular steatosis as the primary finding.
- Option D: PSC shows onion-skin periductal fibrosis around bile ducts, not steatohepatitis.

Final Answer: Alcoholic steatohepatitis (macrovesicular steatosis + ballooning + Mallory bodies + PMN infiltrate) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q30

Q31.

Solution

Concept – Non-caseating Granulomas in Crohn's Disease: While non-caseating granulomas are found in sarcoidosis, berylliosis, and other conditions, in the context of **gastrointestinal pathology** with transmural inflammation, skip lesions, cobblestone mucosa, and perianal fistulae, non-caseating granulomas are **pathognomonic of Crohn's disease**. They are found in only ~60% of Crohn's biopsies (not always present). The granulomas are small, loosely formed, and can occur in any layer of the bowel wall – unlike TB (caseating) or sarcoidosis (systemic context).

Key Point: In GI pathology: non-caseating granulomas = Crohn's disease. Ulcerative colitis never has granulomas.

Why other options are wrong:

- Option A: Caseating granulomas with central necrosis indicate TB. Intestinal TB can mimic Crohn's but the question specifies non-caseating.
- Option C: Aschoff nodules are perivascular granulomas of the myocardium in acute rheumatic carditis – not GI.
- Option D: Palisading granulomas around central fibrinoid necrosis are characteristic of rheumatoid nodules and necrobiosis lipoidica, not Crohn's.

Final Answer: Non-caseating granulomas (Crohn's disease pathognomonic) $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q31

Q32.

Solution

Concept – Primary Biliary Cholangitis (PBC) and AMA: Primary biliary cholangitis (PBC), formerly primary biliary cirrhosis, is an autoimmune disease causing progressive destruction of small intrahepatic bile ducts by granulomatous inflammation. It predominantly affects middle-aged women (F:M = 9:1). The **antimitochondrial antibody (AMA), specifically M2 subtype** targeting pyruvate dehydrogenase complex (PDC-E2), is positive in $\geq$95% of patients and is highly specific ($>$95%). It is the most specific autoantibody in any autoimmune liver disease.

Key Point: PBC = AMA-M2 positive + cholestatic LFTs + granulomatous bile duct destruction. Treatment: ursodeoxycholic acid.

Why other options are wrong:

- Option A: ANA is positive in autoimmune hepatitis (AIH) type 1 – not the most specific marker for PBC.
- Option B: Anti-smooth muscle antibody (ASMA) is the hallmark of AIH type 1 – not PBC.
- Option C: pANCA (perinuclear anti-neutrophil cytoplasmic antibody) is associated with primary sclerosing cholangitis (PSC) and UC.

Final Answer: Anti-mitochondrial antibody (AMA) M2 subtype $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q32

Q33.

Solution

Concept – Pancreatic Pseudocyst: A **pancreatic pseudocyst** is a fluid collection arising 4–6 weeks after acute pancreatitis (or pancreatic trauma) in the lesser sac or peripancreatic spaces. Key features: (1) wall composed of **fibrous and granulation tissue without an epithelial lining** (hence "pseudo"); (2) fluid is rich in pancreatic enzymes (amylase $\gg$ 1,000 U/L); (3) occurs after disruption of the main pancreatic duct; (4) most resolve spontaneously. Mucinous cystic neoplasms (MCN) and serous cystadenomas have a true **epithelial lining**.

Key Point: Pseudocyst = no epithelial lining + high amylase fluid + post-pancreatitis/trauma history. True cysts and cystic neoplasms all have epithelial linings.

Why other options are wrong:

- Option B: MCN has a tall columnar mucinous epithelial lining (often with ovarian-type stroma) – and arises independently of pancreatitis.
- Option C: Serous cystadenoma has a microcystic pattern with cuboidal glycogen-rich cells lining the cyst – and does not produce high amylase fluid.
- Option D: IPMN arises within the pancreatic duct system with mucin-producing papillary epithelium and communicates with the main duct.

Final Answer: Pancreatic pseudocyst (no epithelial lining, fibrous wall) $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q33

Q34.

Solution

Concept – Kimmelstiel-Wilson Nodules (Diabetic Nephropathy): Diabetic nodular glomerulosclerosis (Kimmelstiel-Wilson lesion) is pathognomonic of longstanding diabetes mellitus. It consists of **ovoid or spherical nodular deposits of laminated matrix material within the mesangium**, situated at the periphery of the glomerulus, often with compressed capillary loops. This is accompanied by **diffuse glomerulosclerosis** (mesangial expansion throughout all lobules) and **GBM thickening**. Advanced disease causes progressive loss of GFR and nephrotic-range proteinuria.

Key Point: Nodular mesangial expansion in a diabetic patient = Kimmelstiel-Wilson nodule. PAS stain highlights the nodules (PAS positive matrix).

Why other options are wrong:

- Option A: IgA deposits in the mesangium are in IgA nephropathy (Berger's disease) – mesangial, not nodular laminated deposits.
- Option B: Subepithelial humps (between podocytes and GBM) are classic for post-streptococcal GN ("starry sky" on EM).
- Option D: Wire-loop lesions in lupus nephritis class IV are caused by subendothelial immune complex deposits forming thickened, rigid capillary loops.

Final Answer: Kimmelstiel-Wilson nodules (diabetic nodular glomerulosclerosis) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q34

Q35.

Solution

Concept – Goodpasture Syndrome (Anti-GBM Antibody): Goodpasture syndrome is caused by **anti-glomerular basement membrane (anti-GBM) antibodies** targeting the NC1 domain of type IV collagen ($\alpha 3$ chain), present in both renal GBM and pulmonary alveolar BM. This is a **type II hypersensitivity** reaction. On immunofluorescence (IF), the pattern is **linear IgG along the GBM** (in contrast to granular/lumpy-bumpy deposits in immune complex GN). Renal biopsy shows crescentic (rapidly progressive) GN. Plasmapheresis + immunosuppression is the treatment.

Key Point: Linear IF = anti-GBM (Goodpasture). Granular IF = immune complex deposition (post-strep, lupus, IgA). No deposits ("pauci-immune") = ANCA vasculitis.

Why other options are wrong:

- Option A: Granular "lumpy-bumpy" IgG is the pattern in post-streptococcal GN (type III hypersensitivity, immune complex deposition).
- Option C: ANCA-associated GN (Wegener's/MPA) shows NO immunoglobulin deposits (pauci-immune) on IF – not linear IgG.
- Option D: Mesangial IgA deposits give a granular mesangial pattern, not the linear capillary wall pattern of Goodpasture.

Final Answer: Anti-GBM antibody (type II hypersensitivity) with linear IgG on IF – Goodpasture syndrome $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q35

Q36.

Solution

Concept – ADPKD (PKD1/PKD2): Autosomal dominant polycystic kidney disease (ADPKD) is the most common hereditary kidney disease (prevalence 1:400–1:1000). **PKD1** (chromosome 16, encodes polycystin-1; 85% of cases) and **PKD2** (chromosome 4, encodes polycystin-2; 15%) are the causative genes. Clinical features: bilateral enlarged cystic kidneys, hypertension, progressive CKD (ESRD typically in the 4th–6th decade), **berry (saccular) intracranial aneurysms** (10–15% of patients), hepatic cysts, mitral valve prolapse. This is the most common cause of inherited chronic renal failure.

Key Point: ADPKD = bilateral cystic kidneys + family history + berry aneurysms (SAH risk) + polycystin-1/2 mutations.

Why other options are wrong:

- Option A: ARPKD (PKHD1/fibrocystin) presents in neonates with oligohydramnios, bilateral enlarged kidneys, and hepatic fibrosis – not the adult presentation described.
- Option B: Von Hippel-Lindau (VHL) causes haemangioblastomas, clear cell RCC, and phaeochromocytoma – not bilateral polycystic kidneys.
- Option C: Tuberous sclerosis (TSC1/TSC2) causes hamartomas, angiomyolipomas, and occasional simple cysts – not the massively enlarged bilaterally cystic kidneys.

Final Answer: Autosomal dominant PKD (ADPKD) – PKD1/PKD2 gene mutation $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q36

Q37.

Solution

Concept – Medullary Thyroid Carcinoma and RET: Medullary thyroid carcinoma (MTC) arises from parafollicular C-cells (calcitonin-secreting). Key features: (1) **amyloid stroma** (calcitonin deposits – Congo red positive); (2) markedly elevated serum **calcitonin** (tumour marker); (3) 25% are familial as part of **MEN2A** (MTC + phaeochromocytoma + parathyroid hyperplasia) or **MEN2B** (MTC + phaeochromocytoma + mucosal neuromas + Marfanoid habitus); (4) caused by activating point mutations in the **RET proto-oncogene** (chromosome 10). Vandetanib/cabozantinib (RET inhibitors) are used for advanced MTC.

Key Point: MTC = C-cell origin + amyloid + calcitonin elevated + RET mutation. Genetic testing of RET is mandatory in all MTC patients (index case and family).

Why other options are wrong:

- Option B: BRAF V600E is the most common mutation in *papillary* thyroid carcinoma (PTC – psammoma bodies, orphan Annie nuclei).
- Option C: RAS mutations occur in follicular adenoma and follicular carcinoma, not MTC.
- Option D: PAX8-PPARγ fusion is characteristic of follicular thyroid carcinoma.

Final Answer: RET proto-oncogene mutation (MEN2A/MEN2B – medullary thyroid carcinoma) $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q37

Q38.

Solution

Concept – Conn Syndrome (Primary Hyperaldosteronism): Primary hyperaldosteronism (Conn syndrome) is the most common cause of secondary hypertension (5–10% of all hypertensives). Causes: aldosterone-producing adenoma (APA, 35%) or bilateral adrenal hyperplasia (65%). Autonomous aldosterone excess causes: sodium and water retention (hypertension) + urinary potassium wasting (**hypokalaemia**). Crucially, aldosterone is secreted independently of renin, so **plasma renin is suppressed** (low) and **aldosterone:renin ratio (ARR) $>$ 30** is the screening test.

Key Point: Conn syndrome = hypertension + hypokalaemia + elevated ARR (suppressed renin). No oedema (aldosterone escape phenomenon). CT shows adrenal adenoma; confirmed by adrenal vein sampling.

Why other options are wrong:

- Option A: Secondary hyperaldosteronism (renovascular) has *elevated* plasma renin – the ARR would be low/normal.
- Option B: Phaeochromocytoma causes episodic hypertension with headache, diaphoresis, and palpitations; catecholamine excess, not aldosterone.
- Option D: Cushing syndrome causes hypertension via cortisol + increased angiotensinogen, but the typical biochemistry is elevated cortisol/ACTH, not aldosterone; hypokalaemia occurs but the aldosterone:renin ratio is not elevated.

Final Answer: Primary hyperaldosteronism – Conn syndrome (adrenal adenoma) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q38

Q39.

Solution

Concept – Kaposi Sarcoma and HHV-8: Kaposi sarcoma (KS) is a vascular neoplasm of endothelial/mesenchymal origin caused by **Human herpesvirus-8 (HHV-8, also called KSHV – Kaposi sarcoma-associated herpesvirus)**. It is an **AIDS-defining illness** occurring when CD4 count falls below 200/μL. Histology: spindle cells forming slit-like vascular spaces with RBC extravasation and haemosiderin deposits. Four epidemiological forms exist: epidemic (HIV), classic (elderly Mediterranean men), endemic (sub-Saharan Africa), and iatrogenic (transplant recipients). Antiretroviral therapy leads to regression.

Key Point: KS = HHV-8 + spindle cells + slit-like vessels + violaceous lesions + AIDS context. HHV-8 also causes primary effusion lymphoma and Castleman disease.

Why other options are wrong:

- Option A: EBV causes Burkitt lymphoma (t(8;14), starry-sky pattern), DLBCL, nasopharyngeal carcinoma, and infectious mononucleosis – not Kaposi sarcoma.
- Option C: HPV 16/18 cause cervical, anal, oropharyngeal, and penile squamous carcinomas – not Kaposi sarcoma.
- Option D: CMV causes retinitis, colitis, and pneumonitis in AIDS patients – it is not oncogenic.

Final Answer: Human herpesvirus-8 (HHV-8 / KSHV) – Kaposi sarcoma $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q39

Q40.

Solution

Concept – Melanoma and BRAF V600E: BRAF V600E is a point mutation (valine $\rightarrow$ glutamate at codon 600) in the BRAF serine/threonine kinase, found in approximately **50%** of cutaneous melanomas. The mutant BRAF constitutively activates the MAPK/ERK pathway, driving uncontrolled proliferation. **Vemurafenib**

and **dabrafenib** (BRAF inhibitors) produce dramatic responses in BRAF V600E-positive metastatic melanoma. Combined BRAF + MEK inhibition (vemurafenib + cobimetinib, or dabrafenib + trametinib) further delays resistance.

Key Point: BRAF V600E $\approx$ 50% of cutaneous melanoma $\rightarrow$ targeted therapy with BRAF inhibitors (vemurafenib). Preceded by BRAF mutation testing in all advanced melanoma.

Why other options are wrong:

- Option A: KIT mutations occur in acral lentiginous and mucosal melanomas (not UV-induced cutaneous melanoma); targeted by imatinib.
- Option B: NRAS Q61K/R mutations occur in $\sim$20% of melanomas but no approved targeted therapy exists (MEK inhibitors have limited benefit alone).
- Option C: NF1 loss-of-function mutations occur in $\sim$14% of cutaneous melanomas and activate RAS signalling – currently no approved targeted agent for NF1-mutant melanoma.

Final Answer: BRAF V600E mutation (serine/threonine kinase) $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q40

Answer Key – FMGE Pathology Sample Paper 4

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