INI CET Pathology

Sample Paper – 5

Duration: 14 Minutes **Maximum Marks: 15**

Instructions

- This paper contains **15** Multiple Choice Questions (Single Best Response), modelled on the Pathology content of **INI CET** (Institutes of National Importance Combined Entrance Test) conducted by AIIMS New Delhi.
- The **15-question** length matches the number of Pathology items you actually meet in the real 200-question paper.
- Each correct answer carries **+1 mark**. There is **negative marking of one-third (0.33) mark** for every incorrect answer; unattempted questions carry no penalty.
- Every question has **exactly one best answer**. Choose the single most appropriate option.
- Attempt this practice paper in one timed sitting of about **14 minutes**, the pace the real computer-based test demands.

Cell Injury and Inflammation

Q1. The type of necrosis seen in the walls of small blood vessels in immune-mediated vasculitis and malignant hypertension, where plasma proteins and immune complexes are deposited to produce a bright pink amorphous appearance, is:

(A) Fibrinoid necrosis

(B) Caseous necrosis

(C) Gangrenous necrosis

(D) Liquefactive necrosis

Q2. Deposition of calcium salts in normal, undamaged tissues such as the

gastric mucosa, kidneys and lung septa, occurring secondary to hypercalcaemia, is termed:

(A) Dystrophic calcification

(B) Metastatic calcification

(C) Psammoma body formation

(D) Heterotopic ossification

Q3. During pregnancy the uterus enlarges both by an increase in the size of the individual smooth muscle cells and by an increase in their number. These two cellular adaptations to stress are, respectively:

(A) Hyperplasia and hypertrophy

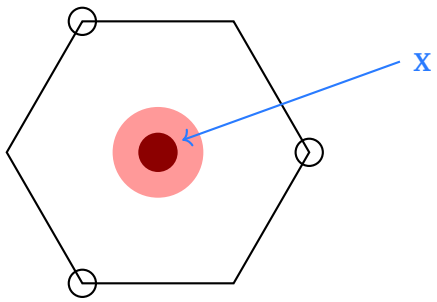
(B) Metaplasia and atrophy

(C) Hypertrophy and hyperplasia

(D) Atrophy and metaplasia

Hemodynamic Disorders

Q4. The schematic below represents a hepatic lobule in chronic passive venous congestion of the liver, which gives the cut surface a mottled "nutmeg" appearance. The congestion begins around the region marked **X**, which is the:



congested centre, paler periphery

(A) Portal tract

(B) Bile canaliculus

(C) Centrilobular (central) vein

(D) Space of Disse

Q5. In chronic pulmonary congestion caused by left-sided heart failure, the alveolar spaces come to contain haemosiderin-laden macrophages. These cells are known as:

(A) Anitschkow cells

(B) Aschoff giant cells

(C) Foam cells

(D) Heart-failure cells

Neoplasia

Q6. Regarding the assessment of a malignant tumour, which statement correctly distinguishes grading from staging?

(A) Grade reflects the extent of local and distant spread

(B) Staging is based on the degree of cellular differentiation

(C) Grade reflects the degree of differentiation, whereas stage reflects the extent of spread

(D) Both grade and stage are determined solely by the mitotic count

Q7. Familial adenomatous polyposis, in which hundreds of colonic adenomatous polyps develop and almost inevitably progress to carcinoma, results from a germline mutation of which tumour-suppressor gene?

(A) RB1

(B) APC

(C) NF1

(D) VHL

Q8. On immunohistochemistry a poorly differentiated tumour stains strongly positive for cytokeratin and negative for vimentin. This staining pattern indicates that the tumour is most likely:

(A) A mesenchymal (connective-tissue) tumour

(B) A lymphoma

(C) A malignant melanoma

(D) An epithelial tumour (carcinoma)

Immunopathology

Q9. Minutes after the vascular clamps are released, a transplanted kidney becomes cyanotic, mottled and flaccid because of preformed recipient antibodies directed against donor endothelial antigens. This reaction is:

(A) Hyperacute rejection

(B) Acute cellular rejection

(C) Chronic rejection

(D) Graft-versus-host disease

Q10. A middle-aged woman with hypothyroidism has a diffusely firm goitre whose histology shows dense lymphocytic infiltration with germinal centres and Hurthle-cell change. Her serum autoantibodies are chiefly directed against:

(A) Thyroid peroxidase (anti-TPO)

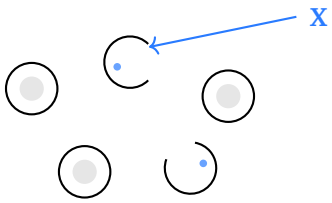
(B) The TSH receptor (stimulating antibody)

(C) The acetylcholine receptor

(D) Double-stranded DNA

Hematology

Q11. The peripheral smear shown, taken after exposure to an oxidant drug, contains red cells with a bitten-out edge (**X**) together with membrane-bound inclusions demonstrable on supravital staining. The deficient enzyme is:



bite cells with Heinz-body inclusions

(A) Pyruvate kinase

(B) Hexokinase

(C) Glucose-6-phosphatase

(D) Glucose-6-phosphate dehydrogenase (G6PD)

Q12. A patient's coagulation profile shows a prolonged prothrombin time (PT/INR) with a normal activated partial thromboplastin time (aPTT). This pattern localises the defect to the:

(A) Extrinsic pathway

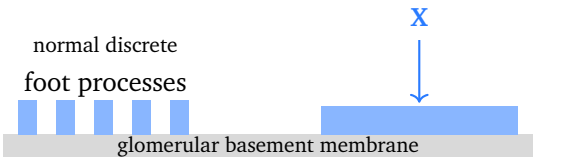
(B) Intrinsic pathway

(C) Common pathway only

(D) Fibrinolytic pathway

Systemic Pathology

Q13. A 4-year-old boy has generalised oedema and heavy proteinuria. Light microscopy of the glomerulus is normal, but electron microscopy shows the change depicted below: uniform effacement of the epithelial foot processes (**X**). The diagnosis is:



effaced (flattened) foot processes

(A) Membranous nephropathy

(B) Focal segmental glomerulosclerosis

(C) Minimal change disease

(D) Diffuse proliferative glomerulonephritis

Q14. A febrile intravenous drug user develops a new heart murmur. Echocardiography reveals friable masses attached to a valve, and histology shows fibrin, platelets and colonies of bacteria. These masses are best described as:

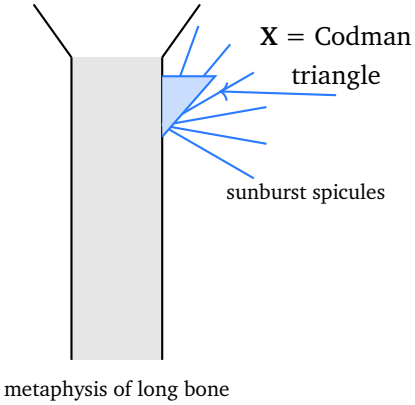
(A) Aschoff bodies

(B) Vegetations of infective endocarditis

(C) Lambl excrescences

(D) Papillary fibroelastomas

Q15. An adolescent has a painful swelling around the knee. The radiograph schematic below shows a metaphyseal lesion that lifts the periosteum to form a Codman triangle (**X**) with radiating "sunburst" spicules. The most likely tumour is:



(A) Ewing sarcoma

(B) Osteosarcoma

(C) Osteochondroma

(D) Giant cell tumour of bone

Detailed Solutions

Q1.

Solution

Concept – patterns of necrosis: Each morphological pattern points to a specific setting.

Step 1 – match the vessel wall: Immune vasculitis and malignant hypertension damage vessel walls, letting plasma proteins and fibrin leak in.

Step 2 – name the pattern: This deposit gives a smudgy, bright pink amorphous look called **fibrinoid necrosis**.

Why the other options are wrong:

- B, caseous: cheese-like necrosis of tuberculosis and other granulomas.
- C, gangrenous: ischaemic coagulative necrosis of a limb, often with superadded infection.
- D, liquefactive: seen in brain infarcts and abscesses.

Final Answer: Fibrinoid necrosis $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – two kinds of pathological calcification: Calcium may deposit in damaged tissue or in normal tissue.

Step 1 – read the clue: The calcium settles in **normal, undamaged** organs, and there is **hypercalcaemia**.

Step 2 – name it: Calcification of normal tissue driven by a high serum calcium is **metastatic calcification**.

Why the other options are wrong:

- A, dystrophic calcification: occurs in already damaged or dead tissue with a normal serum calcium.
- C, psammoma bodies: laminated calcified concretions inside certain tumours.
- D, heterotopic ossification: formation of actual bone in soft tissue, not simple calcium salt deposition.

Final Answer: Metastatic calcification ⇒ B

Answer: (B) Go Back to Q2

Q3.

Solution

Concept – cellular adaptations to stress: Cells adapt by changing size, number or type.

Step 1 – define the two terms: An increase in cell **size** is hypertrophy; an increase in cell **number** is hyperplasia.

Step 2 – apply to the uterus: The pregnant uterus grows by both, so the order asked (size, then number) is **hypertrophy and hyperplasia**.

Why the other options are wrong:

- A: correct pair but in the reversed order.
- B and D: metaplasia (change of cell type) and atrophy (shrinkage) do not describe uterine growth.

Final Answer: Hypertrophy and hyperplasia ⇒ C

Answer: (C) Go Back to Q3

Q4.

Solution

Concept – nutmeg liver: Right heart failure backs blood up into the liver.

Step 1 – read the figure: Congestion starts at the centre of the lobule, marked **X**.

Step 2 – name X: The centre of the lobule drains into the **centrilobular (central) vein**; congestion here reddens the centre while the periphery stays pale, giving the nutmeg pattern.

Why the other options are wrong:

- A, portal tract: sits at the periphery (the corners), not the congested centre.
- B, bile canaliculus: a tiny channel between hepatocytes, not a venous drainage point.
- D, space of Disse: the perisinusoidal space, not the vessel that congestion centres on.

Final Answer: Centrilobular (central) vein ⇒ C

Answer: (C) Go Back to Q4

Q5.

Solution

Concept – chronic pulmonary congestion: Left heart failure raises pressure in the lung capillaries.

Step 1 – trace the events: Red cells leak into alveoli and are engulfed by macrophages, which store the iron as haemosiderin.

Step 2 – name the cell: These haemosiderin-laden alveolar macrophages are **heart-failure cells**.

Why the other options are wrong:

- A, Anitschkow cells: caterpillar-nucleus cells of rheumatic myocarditis.
- B, Aschoff giant cells: multinucleate cells of the rheumatic Aschoff body.
- C, foam cells: lipid-laden macrophages of atherosclerosis and xanthomas.

Final Answer: Heart-failure cells $\Rightarrow$ D

Answer: (D) Go Back to Q5

Q6.

Solution

Concept – grade versus stage: Both describe a cancer but measure different things.

Step 1 – define grade: Grade reflects how well the tumour cells are **differentiated** (how closely they resemble normal tissue).

Step 2 – define stage: Stage reflects the **extent of spread** (size, nodal involvement, distant metastasis; the TNM system).

Why the other options are wrong:

- A: reverses the definitions (spread is stage, not grade).
- B: differentiation is grade, not stage.
- D: mitotic count is only one input into grade and does not determine stage.

Final Answer: Grade reflects differentiation, stage reflects spread $\Rightarrow$ C

Answer: (C) Go Back to Q6

Q7.

Solution

Concept – familial adenomatous polyposis: An inherited polyposis syndrome with a specific gene.

Step 1 – identify the gene: FAP is caused by germline mutation of the **APC** tumour-suppressor gene on chromosome 5q.

Step 2 – consequence: Loss of APC removes a brake on the Wnt pathway, so hundreds of adenomas form and progress to colorectal carcinoma.

Why the other options are wrong:

- A, RB1: retinoblastoma and osteosarcoma.
- C, NF1: neurofibromatosis type 1.
- D, VHL: von Hippel-Lindau disease (renal cell carcinoma, haemangioblastoma).

Final Answer: APC $\Rightarrow$ B

Answer: (B) Go Back to Q7

Q8.

Solution

Concept – immunohistochemical lineage markers: Intermediate filaments reveal a tumour's cell of origin.

Step 1 – read the panel: Cytokeratin positivity marks epithelial cells; vimentin negativity argues against a mesenchymal origin.

Step 2 – conclude: A cytokeratin-positive, vimentin-negative tumour is an **epithelial tumour (carcinoma)**.

Why the other options are wrong:

- A, mesenchymal tumour: would be vimentin-positive, cytokeratin-negative.
- B, lymphoma: expresses CD45 (leukocyte common antigen), not cytokeratin.
- C, melanoma: expresses S-100, HMB-45 and Melan-A, not cytokeratin.

Final Answer: Epithelial tumour (carcinoma) $\Rightarrow$ D

Answer: (D) Go Back to Q8

Q9.

Solution

Concept – graft rejection timing: The tempo of rejection points to its mechanism.

Step 1 – read the clue: Failure within **minutes** due to **preformed antibodies** against donor endothelium.

Step 2 – name it: This is **hyperacute rejection**, in which antibody and complement thrombose the graft vessels at once.

Why the other options are wrong:

- B, acute cellular rejection: develops over days to weeks and is T-cell mediated.
- C, chronic rejection: months to years, with vascular fibrointimal thickening.
- D, graft-versus-host disease: donor lymphocytes attack the host, seen after marrow transplant, not solid organs.

Final Answer: Hyperacute rejection $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – Hashimoto thyroiditis: An autoimmune cause of hypothyroidism.

Step 1 – recognise the picture: Hypothyroidism, a firm goitre, lymphocytic infiltrate with germinal centres and Hurthle cells define **Hashimoto thyroiditis**.

Step 2 – name the antibody: The characteristic autoantibody is against **thyroid peroxidase (anti-TPO)** (also anti-thyroglobulin).

Why the other options are wrong:

- B, TSH-receptor stimulating antibody: causes Graves disease (hyperthyroidism).
- C, acetylcholine receptor: myasthenia gravis.
- D, double-stranded DNA: systemic lupus erythematosus.

Final Answer: Thyroid peroxidase (anti-TPO) $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – oxidant haemolysis: An enzyme defect leaves red cells unable to handle oxidant stress.

Step 1 – read the smear: Bite cells and Heinz-body inclusions after an oxidant drug point to failure of the antioxidant pathway.

Step 2 – name the enzyme: Glucose-6-phosphate dehydrogenase (G6PD) supplies NADPH; its deficiency lets oxidised haemoglobin precipitate as Heinz bodies, which the spleen bites out.

Why the other options are wrong:

- A, pyruvate kinase: deficiency gives chronic haemolysis without Heinz bodies or an oxidant trigger.
- B, hexokinase: a rare glycolytic defect, not the classic bite-cell picture.
- C, glucose-6-phosphatase: its deficiency causes von Gierke glycogen storage disease, not haemolysis.

Final Answer: Glucose-6-phosphate dehydrogenase (G6PD) $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – interpreting PT and aPTT: Each test screens a different limb of the coagulation cascade.

Step 1 – recall the assignments: PT/INR assesses the **extrinsic** pathway (factor VII); aPTT assesses the **intrinsic** pathway (factors VIII, IX, XI, XII).

Step 2 – apply the pattern: An isolated prolonged PT with a normal aPTT localises the defect to the **extrinsic pathway** (classically factor VII deficiency or early warfarin effect).

Why the other options are wrong:

- B, intrinsic pathway: would prolong the aPTT, which is normal here.
- C, common pathway: a defect there prolongs both PT and aPTT together.
- D, fibrinolytic pathway: not measured by PT or aPTT.

Final Answer: Extrinsic pathway $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q12

Q13.

Solution

Concept – childhood nephrotic syndrome: Age and the electron-microscopy finding pin the diagnosis.

Step 1 – read the clues: A young child, heavy proteinuria, a normal light microscopy and diffuse **foot-process effacement** on electron microscopy.

Step 2 – name it: This is **minimal change disease**, the commonest cause of nephrotic syndrome in children, which responds well to steroids.

Why the other options are wrong:

- A, membranous nephropathy: subepithelial deposits and a thick basement membrane; more an adult disease.
- B, focal segmental glomerulosclerosis: segmental scarring on light microscopy.
- D, diffuse proliferative glomerulonephritis: hypercellular glomeruli, usually a nephritic picture.

Final Answer: Minimal change disease ⇒ C

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – infective endocarditis: Bacteraemia seeds a heart valve.

Step 1 – read the picture: An IV drug user, a new murmur, friable valvular masses of fibrin, platelets and bacteria.

Step 2 – name the lesion: These masses are the **vegetations of infective endocarditis**, which can embolise and destroy the valve.

Why the other options are wrong:

- A, Aschoff bodies: granulomatous foci of rheumatic carditis, not friable valve masses.
- C, Lambl excrescences: tiny degenerative valve strands, not infected vegetations.
- D, papillary fibroelastoma: a benign valve tumour, not made of bacteria and

fibrin.

Final Answer: Vegetations of infective endocarditis $\Rightarrow$ B

Answer: (B) Go Back to Q14

Q15.

Solution

Concept – primary bone tumours: Age, site and radiographic signs identify the tumour.

Step 1 – read the figure and clues: An adolescent, a lesion at the **metaphysis** around the knee, a **Codman triangle** and a **sunburst** periosteal reaction.

Step 2 – name it: This triad is classic for **osteosarcoma**, a malignant bone-forming tumour of teenagers.

Why the other options are wrong:

- A, Ewing sarcoma: arises in the diaphysis with an onion-skin periosteal reaction.
- C, osteochondroma: a benign cartilage-capped bony outgrowth, no sunburst.
- D, giant cell tumour: an epiphyseal lytic "soap-bubble" lesion, not sunburst.

Final Answer: Osteosarcoma $\Rightarrow$ B

Answer: (B) Go Back to Q15

Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	A	2	B	3	C	4	C	5	D
6	C	7	B	8	D	9	A	10	A
11	D	12	A	13	C	14	B	15	B