INI CET Pathology

Sample Paper – 8

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. Which feature is characteristic of **apoptosis** rather than necrosis?

(A) Swelling of the cell and its organelles

(B) Rupture of the plasma membrane with leakage of contents

(C) Shrinkage of single cells with membrane blebbing and no surrounding inflammation

(D) Karyolysis accompanied by a brisk acute inflammatory response

Q2. Which term describes a disordered, premalignant proliferation of epithelium with loss of the normal orderly maturation, in contrast to orderly hyperplasia?

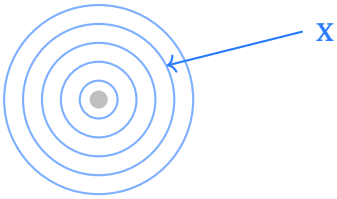
(A) Hyperplasia

(B) Metaplasia

(C) Dysplasia

(D) Hypertrophy

Q3. The laminated, concentric calcified structures (marked **X**) seen in papillary thyroid carcinoma, meningioma and serous ovarian tumours are called:



laminated concentric calcification

(A) Asteroid bodies

(B) Psammoma bodies

(C) Mallory bodies

(D) Councilman bodies

Hemodynamic Disorders

Q4. In the early ("warm") phase of septic shock, the skin is warm and flushed. This is mainly caused by:

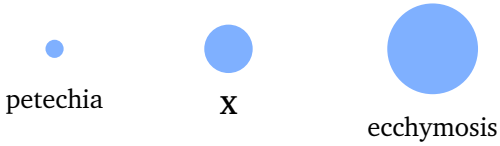
(A) Widespread peripheral vasodilation

(B) Intense peripheral vasoconstriction

(C) Loss of circulating blood volume from haemorrhage

(D) Pump failure of the left ventricle

Q5. Cutaneous haemorrhages are graded by size. In the diagram, the intermediate lesions of about 3 mm to 1 cm (marked **X**) are termed:



(A) Petechiae

(B) Purpura

(C) Ecchymoses

(D) Haematoma

Neoplasia

Q6. Which tumour-suppressor gene is mutated in Wilms tumour (nephroblastoma), the commonest renal malignancy of childhood?

(A) WT1

(B) RB1

(C) VHL

(D) NF1

Q7. The translocation t(14;18), which overexpresses BCL2 and thereby blocks apoptosis, is the characteristic abnormality of:

(A) Burkitt lymphoma

(B) Mantle cell lymphoma

(C) Follicular lymphoma

(D) Chronic myeloid leukaemia

Q8. Which immunohistochemical marker is characteristically positive in melanoma and in nerve-sheath (Schwann cell) tumours?

(A) Cytokeratin

(B) Desmin

(C) S-100 protein

(D) CD20

Immunopathology

Q9. Anti-Scl-70 (anti-topoisomerase I) and anti-centromere antibodies are the characteristic autoantibodies of:

(A) Systemic sclerosis (scleroderma)

(B) Systemic lupus erythematosus

(C) Rheumatoid arthritis

(D) Sjogren syndrome

Q10. A boy has recurrent catalase-positive bacterial and fungal infections, and his nitroblue-tetrazolium (NBT) test is negative, indicating defective NADPH oxidase in his neutrophils. The diagnosis is:

(A) DiGeorge syndrome

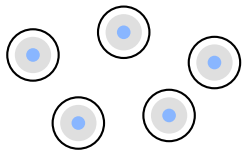
(B) Bruton (X-linked) agammaglobulinaemia

(C) Chediak-Higashi syndrome

(D) Chronic granulomatous disease

Hematology

Q11. The peripheral smear shown, with numerous target cells (central and rim staining), in a child with a microcytic anaemia and imbalanced globin-chain synthesis, suggests:



central + rim staining (target cells)

(A) Hereditary spherocytosis

(B) Thalassaemia

(C) G6PD deficiency

(D) Sickle cell anaemia

Q12. A JAK2 (V617F) mutation together with a raised red-cell mass and a low serum erythropoietin is characteristic of:

(A) Chronic myeloid leukaemia

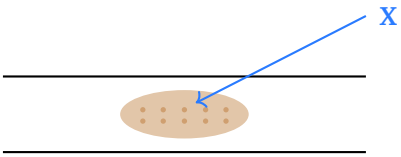
(B) Essential thrombocythaemia

(C) Secondary polycythaemia

(D) Polycythaemia vera

Systemic Pathology

Q13. The schematic shows a renal tubule whose lumen contains the granular cast marked **X**. Such muddy-brown granular casts, in the commonest cause of acute kidney injury, point to:



tubular lumen with granular cast

(A) Acute glomerulonephritis

(B) Acute tubular necrosis

(C) Chronic pyelonephritis

(D) Renal cell carcinoma

Q14. Which serological marker indicates an active hepatitis B infection (acute or chronic)?

(A) HBsAg (hepatitis B surface antigen)

(B) Anti-HBs antibody

(C) Anti-HBc IgG antibody

(D) Anti-HBe antibody

Q15. A young woman presents with a diffuse goitre, exophthalmos and circulating anti-TSH-receptor (stimulating) antibodies. The diagnosis is:

(A) Hashimoto thyroiditis

(B) Toxic multinodular goitre

(C) Subacute (de Quervain) thyroiditis

(D) Graves disease

Detailed Solutions

Q1.

Solution

Concept – apoptosis versus necrosis: The two forms of cell death differ in scale, morphology and host reaction.

Step 1 – features of apoptosis: Apoptosis kills **single cells**, which shrink, condense their chromatin and bud off membrane-bound apoptotic bodies.

Step 2 – the key contrast: The plasma membrane stays intact, so cell contents do not leak and **there is no surrounding inflammation**.

Why the other options are wrong:

- A, cell swelling: a feature of necrosis (and reversible injury), not apoptosis.
- B, membrane rupture with leakage: necrosis, which spills contents and provokes inflammation.
- D, karyolysis with acute inflammation: a necrotic pattern.

Final Answer: Shrinkage of single cells with membrane blebbing and no inflammation $\Rightarrow$ C

Answer: (C) Go Back to Q1

Q2.

Solution

Concept – adaptations versus dysplasia: Cellular responses range from orderly adaptation to disordered premalignant change.

Step 1 – define dysplasia: Dysplasia is a disordered proliferation with loss of uniformity and of normal maturation; it is regarded as premalignant.

Step 2 – contrast with hyperplasia: Hyperplasia is an orderly increase in cell number that keeps normal architecture.

Why the other options are wrong:

- A, hyperplasia: orderly, reversible increase in cell number.
- B, metaplasia: reversible replacement of one mature cell type by another.
- D, hypertrophy: increase in cell size, not disordered growth.

Final Answer: Dysplasia $\Rightarrow$ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – laminated calcifications: Concentric calcified whorls have a specific name and set of associations.

Step 1 – identify X: The laminated, onion-skin concentric calcification is a **psammoma body**.

Step 2 – where they occur: Papillary thyroid carcinoma, meningioma and serous (papillary) ovarian tumours classically contain them.

Why the other options are wrong:

- A, asteroid bodies: star-shaped inclusions in giant cells of sarcoidosis.
- C, Mallory bodies: eosinophilic hepatocyte inclusions in alcoholic hepatitis.
- D, Councilman bodies: apoptotic hepatocytes in viral hepatitis.

Final Answer: Psammoma bodies $\Rightarrow$ B

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – septic (distributive) shock: Sepsis produces a distinctive early haemodynamic picture.

Step 1 – explain the warm skin: Inflammatory mediators cause **widespread peripheral vasodilation**, so the skin is warm and flushed with a bounding pulse.

Step 2 – later course: As shock progresses, perfusion fails and the picture becomes cold and clammy.

Why the other options are wrong:

- B, vasoconstriction: gives the cold skin of hypovolaemic or cardiogenic shock.
- C, blood loss: describes hypovolaemic (haemorrhagic) shock.
- D, pump failure: describes cardiogenic shock.

Final Answer: Widespread peripheral vasodilation $\Rightarrow$ A

Answer: (A) Go Back to Q4

Q5.

Solution

Concept – grading cutaneous haemorrhage by size: The names change with the diameter of the bleed.

Step 1 – identify X: Lesions of roughly 3 mm to 1 cm are **purpura**, the intermediate grade.

Step 2 – the full scale: Petechiae are 1–2 mm pinpoint bleeds; ecchymoses are larger than 1–2 cm bruises.

Why the other options are wrong:

- A, petechiae: the smallest (1–2 mm) pinpoint haemorrhages.
- C, ecchymoses: the largest cutaneous bruises.
- D, haematoma: a localised collection of blood within tissue, not a size grade of surface bleeding.

Final Answer: Purpura $\Rightarrow$ B

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – gene of Wilms tumour: A childhood renal cancer is tied to a specific tumour suppressor.

Step 1 – name the gene: WT1 on chromosome 11p13 is the tumour-suppressor gene mutated in Wilms tumour (nephroblastoma).

Step 2 – clinical link: It is the commonest renal malignancy of childhood and part of the WAGR and Denys-Drash syndromes.

Why the other options are wrong:

- B, RB1: retinoblastoma and osteosarcoma.
- C, VHL: von Hippel-Lindau disease and clear-cell renal carcinoma of adults.
- D, NF1: neurofibromatosis type 1.

Final Answer: WT1 $\Rightarrow$ A

Answer: (A) Go Back to Q6

Q7.

Solution

Concept – lymphoma translocations: Each translocation drives a specific lymphoma through a named gene.

Step 1 – match t(14;18): t(14;18) juxtaposes BCL2 with the IgH locus, overexpressing **BCL2**, which blocks apoptosis; this defines **follicular lymphoma**.

Step 2 – mechanism: The surplus anti-apoptotic BCL2 lets malignant follicle-centre B cells accumulate.

Why the other options are wrong:

- A, Burkitt lymphoma: t(8;14) with MYC overexpression.
- B, mantle cell lymphoma: t(11;14) with cyclin D1 overexpression.
- D, chronic myeloid leukaemia: t(9;22), BCR-ABL, not a lymphoma.

Final Answer: Follicular lymphoma $\Rightarrow$ C

Answer: (C) Go Back to Q7

Q8.

Solution

Concept – immunohistochemical markers: Each marker maps to a lineage of tumours.

Step 1 – match the description: S-100 protein is positive in melanoma and in nerve-sheath (Schwann cell) tumours such as schwannoma and neurofibroma.

Step 2 – use: It helps confirm neural crest and melanocytic origin.

Why the other options are wrong:

- A, cytokeratin: marks epithelial tumours (carcinomas).
- B, desmin: marks muscle tumours.
- D, CD20: marks B-cell lymphomas.

Final Answer: S-100 protein $\Rightarrow$ C

Answer: (C) Go Back to Q8

Q9.

Solution

Concept – autoantibody profiles: Specific antibodies point to specific connective-tissue diseases.

Step 1 – match the antibodies: Anti-Scl-70 (diffuse disease) and anti-centromere (limited/CREST disease) are the markers of **systemic sclerosis (scleroderma)**.

Step 2 – clinical picture: Progressive skin and visceral fibrosis with Raynaud phenomenon.

Why the other options are wrong:

- B, SLE: anti-dsDNA and anti-Smith antibodies.
- C, rheumatoid arthritis: rheumatoid factor and anti-CCP antibodies.
- D, Sjogren syndrome: anti-Ro (SS-A) and anti-La (SS-B) antibodies.

Final Answer: Systemic sclerosis (scleroderma) ⇒ A

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – neutrophil killing defects: A failed respiratory burst gives recurrent infection with a specific test result.

Step 1 – interpret the NBT test: A negative nitroblue-tetrazolium test means neutrophils cannot generate the oxidative burst, that is defective **NADPH oxidase**.

Step 2 – name the disease: This is **chronic granulomatous disease**, with recurrent catalase-positive infections and granuloma formation.

Why the other options are wrong:

- A, DiGeorge syndrome: thymic aplasia with T-cell deficiency (normal NBT).
- B, Bruton agammaglobulinaemia: absent B cells and antibodies, not an oxidase defect.
- C, Chediak-Higashi syndrome: giant lysosomal granules and a fusion defect, with a normal NADPH oxidase.

Final Answer: Chronic granulomatous disease ⇒ D

Answer: (D) Go Back to Q10

Q11.

Solution

Concept – target cells and globin synthesis: Red-cell shape and the underlying defect give the diagnosis.

Step 1 – read the smear: Numerous **target cells** with a microcytic anaemia and imbalanced globin-chain synthesis point to **thalassaemia**.

Step 2 – confirm: Haemoglobin electrophoresis (for example a raised HbA_2 in beta-thalassaemia trait) confirms it.

Why the other options are wrong:

- A, hereditary spherocytosis: spherocytes, not target cells; a membrane protein defect.
- C, G6PD deficiency: bite cells and Heinz bodies after oxidant stress.
- D, sickle cell anaemia: sickle cells; a structural haemoglobin defect, not a chain-imbalance anaemia.

Final Answer: Thalassaemia $\Rightarrow$ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – myeloproliferative neoplasms: A driver mutation plus laboratory values names the disorder.

Step 1 – combine the clues: A **JAK2 (V617F)** mutation with a raised red-cell mass and a low erythropoietin defines **polycythaemia vera**.

Step 2 – mechanism: The mutated JAK2 drives erythropoietin-independent red-cell production, suppressing endogenous erythropoietin.

Why the other options are wrong:

- A, chronic myeloid leukaemia: BCR-ABL, not JAK2, with a raised white-cell count.
- B, essential thrombocythaemia: dominated by a very high platelet count.
- C, secondary polycythaemia: erythropoietin is high (hypoxia or tumour driven), and there is no JAK2 mutation.

Final Answer: Polycythaemia vera $\Rightarrow$ D

Answer: (D) Go Back to Q12

Q13.

Solution

Concept – acute kidney injury: Urinary casts localise the site of renal damage.

Step 1 – identify the lesion: Muddy-brown granular casts of sloughed tubular epithelium indicate **acute tubular necrosis**, the commonest cause of acute kidney injury.

Step 2 – causes: Ischaemia (hypotension) and nephrotoxins (drugs, myoglobin) are the usual triggers.

Why the other options are wrong:

- A, acute glomerulonephritis: red-cell casts and dysmorphic red cells, not granular casts.
- C, chronic pyelonephritis: coarse scarring and broad waxy casts, a chronic process.
- D, renal cell carcinoma: a solid tumour, not a cause of granular casts.

Final Answer: Acute tubular necrosis $\Rightarrow$ B

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – hepatitis B serology: Each serological marker signals a different phase of infection.

Step 1 – pick the marker of active infection: HBsAg (hepatitis B surface antigen) is present in active infection, whether acute or chronic.

Step 2 – interpretation: Persistence of HBsAg beyond six months defines chronic hepatitis B.

Why the other options are wrong:

- B, anti-HBs: indicates recovery or vaccination (immunity), not active infection.
- C, anti-HBc IgG: marks past or ongoing exposure but is not the antigen of active infection.
- D, anti-HBe: marks reduced infectivity as the virus is cleared.

Final Answer: HBsAg $\Rightarrow$ A

Answer: (A) Go Back to Q14

Q15.

Solution

Concept – causes of hyperthyroidism: The autoantibody and physical signs identify the disease.

Step 1 – combine the clues: A diffuse goitre, exophthalmos and **anti-TSH-receptor stimulating antibodies** define **Graves disease**.

Step 2 – mechanism: The antibodies switch on the TSH receptor, driving diffuse gland hyperfunction and enlargement.

Why the other options are wrong:

- A, Hashimoto thyroiditis: anti-TPO antibodies with hypothyroidism, not stimulating antibodies.
- B, toxic multinodular goitre: autonomous nodules, no TSH-receptor antibodies or eye signs.
- C, subacute (de Quervain) thyroiditis: a painful, post-viral, self-limiting thyroiditis.

Final Answer: Graves disease $\Rightarrow$ D

Answer: (D) Go Back to Q15

Answer Key

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