INI CET Pathology

Sample Paper – 2

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. Ischaemic infarction of a solid organ such as the kidney or spleen produces an area of necrosis in which the basic tissue outline (architecture) is preserved for several days. This pattern of necrosis is:

(A) Liquefactive necrosis

(B) Coagulative necrosis

(C) Caseous necrosis

(D) Fat necrosis

Q2. Energy-dependent, caspase-mediated death of single cells that leaves the cell membrane intact, forms apoptotic bodies, and characteristically provokes no surrounding inflammation is called:

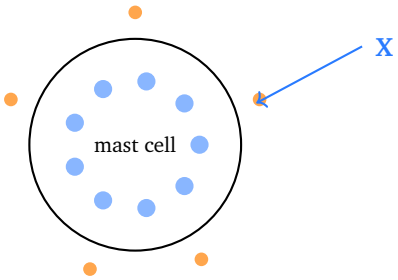
(A) Apoptosis

(B) Coagulative necrosis

(C) Liquefactive necrosis

(D) Postmortem autolysis

Q3. The diagram shows a mast cell degranulating during acute inflammation. The preformed vasoactive amine released from its granules (marked **X**), which is an early mediator causing arteriolar vasodilation and increased venular permeability, is:



(A) Histamine

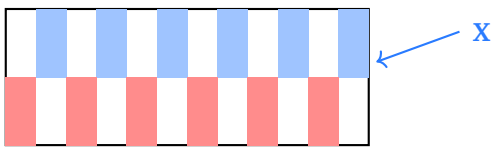
(B) Bradykinin

(C) Interferon-gamma

(D) Collagenase

Hemodynamic Disorders

Q4. A thrombus recovered from an artery shows alternating pale (platelet and fibrin) and dark red (red-cell) laminations, the lines of Zahn marked **X**. Their presence tells the pathologist that the thrombus:



alternating pale and red laminations

(A) Formed after death as a postmortem clot

(B) Formed before death as an antemortem thrombus

(C) Is an embolus carried from a distant site

(D) Is composed purely of red cells

Q5. Two days after fracturing his femur and tibia, a young man suddenly develops breathlessness, confusion and a petechial rash over the chest. The most likely embolic syndrome is:

(A) Air (gas) embolism

(B) Fat embolism syndrome

(C) Amniotic fluid embolism

(D) Tumour embolism

Neoplasia

Q6. Which single property, when present, unequivocally defines a tumour as malignant, given that benign tumours never display it?

(A) Rapid growth rate

(B) A large size

(C) Metastasis to distant sites

(D) Nuclear pleomorphism

Q7. Which proto-oncogene, when activated by a point mutation, encodes a small GTPase locked in its active GTP-bound state and thereby drives continuous cell-growth signalling?

(A) MYC

(B) HER2/neu (ERBB2)

(C) BCL2

(D) RAS

Q8. In the TNM system used to stage a malignant tumour, the three letters T, N and M denote respectively:

T	N	M
?	?	?

(A) Type, Number, Mitoses

(B) Thickness, Necrosis, Margin

(C) Tumour size, Nodal spread, Metastasis

(D) Tissue, Nucleus, Membrane

Immunopathology

Q9. Autoimmune haemolytic anaemia, in which antibodies bind red-cell surface antigens and lead to their destruction, is a classic example of which hypersensitivity reaction?

(A) Type II (antibody-mediated cytotoxic)

(B) Type I (immediate, IgE-mediated)

(C) Type III (immune-complex)

(D) Type IV (delayed, cell-mediated)

Q10. Which autoantibody is highly specific for systemic lupus erythematosus (SLE) and correlates with disease activity, especially lupus nephritis?

(A) Anti-mitochondrial antibody

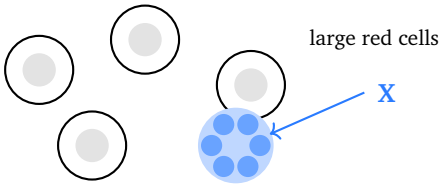
(B) Anti-cyclic citrullinated peptide (anti-CCP)

(C) Anti-topoisomerase (Scl-70)

(D) Anti-double-stranded DNA (anti-dsDNA)

Hematology

Q11. The peripheral smear shown has enlarged red cells and a neutrophil with six nuclear lobes (hypersegmented, marked **X**). With a raised MCV, this picture points to:



(A) Iron deficiency anaemia

(B) Aplastic anaemia

(C) Hereditary spherocytosis

(D) Megaloblastic anaemia

Q12. Pink, needle-shaped azurophilic cytoplasmic inclusions (Auer rods) found within blast cells on a bone-marrow smear are diagnostic of:

(A) Acute myeloid leukaemia

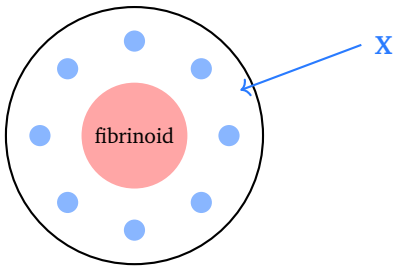
(B) Chronic lymphocytic leukaemia

(C) Hodgkin lymphoma

(D) Multiple myeloma

Systemic Pathology

Q13. The myocardial lesion shown, with a core of fibrinoid necrosis rimmed by plump activated macrophages (Anitschkow/Aschoff cells) and lymphocytes (an Aschoff body, marked **X**), is pathognomonic of a disease that classically progresses to mitral stenosis. It is:



(A) Infective endocarditis

(B) Myocardial infarction

(C) Rheumatic heart disease

(D) Viral myocarditis

Q14. In primary pulmonary tuberculosis, a subpleural parenchymal focus together with enlarged caseating hilar lymph nodes draining it is together known as the:

(A) Rasmussen aneurysm

(B) Assmann focus

(C) Ghon complex

(D) Simon focus

Q15. A child presents with heavy proteinuria (more than 3.5 g per day), hypoalbuminaemia, generalised oedema and hyperlipidaemia. This constellation defines:

(A) Nephritic syndrome

(B) Nephrotic syndrome

(C) Acute tubular injury

(D) Lower urinary tract infection

Detailed Solutions

Q1.

Solution

Concept – patterns of necrosis: Each pattern of necrosis points to a particular cause and location.

Step 1 – read the clue: The stem describes an ischaemic infarct in a solid organ with preserved tissue outline.

Step 2 – match the pattern: This is **coagulative necrosis**, in which enzymatic protein denaturation preserves the cell architecture for days.

Why the other options are wrong:

- A, liquefactive: seen in brain infarcts and abscesses, with complete tissue dissolution.
- C, caseous: cheese-like, characteristic of tuberculosis.
- D, fat necrosis: seen in acute pancreatitis and breast trauma.

Final Answer: Coagulative necrosis $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q1

Q2.

Solution

Concept – apoptosis versus necrosis: Apoptosis is a controlled, programmed form of cell death.

Step 1 – name the process: Caspase-mediated, energy-dependent death of single cells with intact membranes and apoptotic bodies is **apoptosis**.

Step 2 – key contrast: Because the membrane stays intact and contents are packaged, there is no surrounding inflammation.

Why the other options are wrong:

- B, coagulative necrosis: passive death of groups of cells with inflammation.
- C, liquefactive necrosis: enzymatic dissolution of tissue.
- D, postmortem autolysis: self-digestion of the whole body after death.

Final Answer: Apoptosis $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – early mediators of acute inflammation: Preformed mediators act within seconds to minutes.

Step 1 – identify X: The vasoactive amine stored in mast cell granules is **histamine**.

Step 2 – its action: Released on degranulation, histamine causes arteriolar vasodilation and increased venular permeability, the first vascular changes of inflammation.

Why the other options are wrong:

- B, bradykinin: a plasma-derived kinin, not stored in mast cell granules.
- C, interferon-gamma: a cytokine of cell-mediated immunity.
- D, collagenase: a tissue-degrading enzyme, not a vasoactive amine.

Final Answer: Histamine $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – lines of Zahn: Laminations reveal when a thrombus formed.

Step 1 – interpret X: Alternating pale platelet-fibrin and dark red-cell layers (lines of Zahn) form only in flowing blood.

Step 2 – conclusion: Their presence means the thrombus **formed before death (antemortem)**, distinguishing it from a postmortem clot.

Why the other options are wrong:

- A, postmortem clot: gelatinous, lacks lines of Zahn (chicken-fat and currant-jelly layers only).
- C, embolus: a fragment that has travelled; lines of Zahn indicate site of formation, not embolism.
- D, pure red-cell mass: lines of Zahn specifically contain layered platelets and fibrin.

Final Answer: Formed before death (antemortem thrombus) $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q4

Q5.

Solution

Concept – fat embolism syndrome: Long-bone fractures release marrow fat into the circulation.

Step 1 – read the triad: Respiratory distress, neurological change and a petechial rash 1–3 days after fracture form the classic triad.

Step 2 – name the syndrome: This is **fat embolism syndrome**, following fracture of the femur and tibia.

Why the other options are wrong:

- A, air embolism: follows vascular procedures or diving, not fractures.
- C, amniotic fluid embolism: a peripartum catastrophe in labour.
- D, tumour embolism: seen with disseminated malignancy.

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

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – defining malignancy: Only one feature is absolutely diagnostic of malignancy.

Step 1 – name it: Metastasis, spread to a discontinuous distant site, unequivocally marks a tumour as malignant.

Step 2 – why it is decisive: Benign tumours never metastasise, so its presence removes all doubt.

Why the other options are wrong:

- A, rapid growth: some benign tumours grow fast; some malignant ones grow slowly.
- B, large size: size alone does not indicate behaviour.
- D, nuclear pleomorphism: a cytological clue to malignancy, but not absolute.

Final Answer: Metastasis $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q6

Q7.

Solution

Concept – oncogene activation: Point mutation can lock a signalling protein on.

Step 1 – identify the gene: RAS encodes a small GTPase; a point mutation impairs its GTP hydrolysis, trapping it in the active GTP-bound state.

Step 2 – effect: The active RAS drives continuous downstream growth signalling. It is the most commonly mutated oncogene in human cancers.

Why the other options are wrong:

- A, MYC: a transcription factor, usually activated by amplification or translocation.
- B, HER2/neu: a receptor tyrosine kinase, typically activated by amplification.
- C, BCL2: an anti-apoptotic gene, not a GTPase.

Final Answer: RAS $\Rightarrow$ D

Answer: (D) Go Back to Q7

Q8.

Solution

Concept – TNM staging: Staging measures the anatomical extent of a tumour.

Step 1 – decode the letters: **T** = tumour size and local extent, **N** = regional nodal spread, **M** = distant metastasis.

Step 2 – use: Stage guides prognosis and treatment more powerfully than grade.

Why the other options are wrong:

- A, B and D: none matches the accepted meaning of T, N and M in the TNM system.

Final Answer: Tumour size, Nodal spread, Metastasis $\Rightarrow$ C

Answer: (C) Go Back to Q8

Q9.

Solution

Concept – hypersensitivity types: Each type has a defining mechanism.

Step 1 – match the disease: In autoimmune haemolytic anaemia, antibodies bind red-cell antigens and cause destruction, which is **Type II** (antibody-mediated cytotoxic) hypersensitivity.

Step 2 – mechanism: Cells are cleared by complement and by phagocytes in the spleen.

Why the other options are wrong:

- B, Type I: IgE-mediated immediate allergy such as anaphylaxis.
- C, Type III: circulating immune complexes deposit in tissue (e.g. serum sickness).
- D, Type IV: delayed T-cell mediated (e.g. contact dermatitis).

Final Answer: Type II (antibody-mediated cytotoxic) $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – autoantibodies in SLE: Some autoantibodies are highly specific markers.

Step 1 – name the antibody: Anti-double-stranded DNA (anti-dsDNA) is highly specific for SLE and tracks disease activity, especially lupus nephritis.

Step 2 – context: Anti-Smith is the other highly specific SLE antibody; ANA is sensitive but not specific.

Why the other options are wrong:

- A, anti-mitochondrial: primary biliary cholangitis.
- B, anti-CCP: rheumatoid arthritis.
- C, Scl-70: systemic sclerosis.

Final Answer: Anti-double-stranded DNA (anti-dsDNA) $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q10

Q11.

Solution

Concept – macrocytic red-cell morphology: Large cells plus hypersegmented neutrophils have a specific cause.

Step 1 – read the smear: Enlarged red cells with a hypersegmented (six-lobed) neutrophil and a raised MCV indicate **megaloblastic anaemia**.

Step 2 – cause: This results from vitamin B12 or folate deficiency impairing DNA synthesis.

Why the other options are wrong:

- A, iron deficiency: microcytic hypochromic, small cells.
- B, aplastic anaemia: pancytopenia with a hypocellular marrow, cells normal in size.
- C, hereditary spherocytosis: small round spherocytes without central pallor.

Final Answer: Megaloblastic anaemia ⇒ D

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – Auer rods: A cytoplasmic inclusion pins the lineage.

Step 1 – interpret the finding: Needle-shaped azurophilic **Auer rods** in blasts identify a myeloid lineage, so the diagnosis is **acute myeloid leukaemia**.

Step 2 – note: Auer rods are especially prominent in acute promyelocytic leukaemia.

Why the other options are wrong:

- B, CLL: mature small lymphocytes and smudge cells, no Auer rods.
- C, Hodgkin lymphoma: Reed-Sternberg cells in nodes, not blasts with Auer rods.
- D, multiple myeloma: sheets of plasma cells, no Auer rods.

Final Answer: Acute myeloid leukaemia ⇒ A

Answer: (A) Go Back to Q12

Q13.

Solution

Concept – Aschoff body: A specific myocardial granuloma names the disease.

Step 1 – identify X: Central fibrinoid necrosis rimmed by Anitschkow/Aschoff cells and lymphocytes is an **Aschoff body**, pathognomonic of **rheumatic heart disease**.

Step 2 – consequence: Repeated rheumatic activity scars the mitral valve and classically causes mitral stenosis.

Why the other options are wrong:

- A, infective endocarditis: valve vegetations with bacteria, no Aschoff bodies.
- B, myocardial infarction: coagulative necrosis with neutrophils, not a granuloma.
- D, viral myocarditis: a lymphocytic interstitial infiltrate without Aschoff bodies.

Final Answer: Rheumatic heart disease $\Rightarrow$ C

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – primary tuberculosis: The first exposure leaves a characteristic complex.

Step 1 – name it: A subpleural parenchymal focus plus caseating hilar nodes draining it is the **Ghon complex**.

Step 2 – note: When it heals by calcification it is called the Ranke complex.

Why the other options are wrong:

- A, Rasmussen aneurysm: a dilated artery inside a TB cavity.
- B, Assmann focus: an early apical focus of reinfection (adult) TB.
- D, Simon focus: an apical focus seeded during primary infection.

Final Answer: Ghon complex $\Rightarrow$ C

Answer: (C) Go Back to Q14

Q15.

Solution

Concept – nephrotic syndrome: Heavy protein loss defines the picture.

Step 1 – assemble the features: Proteinuria above 3.5 g per day, hypoalbuminaemia, oedema and hyperlipidaemia together define **nephrotic syndrome**.

Step 2 – mechanism: Damage to the glomerular filtration barrier allows massive albumin loss into the urine.

Why the other options are wrong:

- A, nephritic syndrome: haematuria, hypertension and mild proteinuria.
- C, acute tubular injury: rising creatinine with granular casts, not massive proteinuria.
- D, urinary tract infection: dysuria and pyuria, no heavy proteinuria.

Final Answer: Nephrotic syndrome $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q15

Answer Key

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