INI CET Pathology

Sample Paper – 4

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. In acute pancreatitis, released lipases digest peripancreatic fat and the liberated fatty acids combine with calcium to form chalky-white deposits on the mesentery. This appearance represents:

(A) Coagulative necrosis

(B) Fat necrosis with saponification

(C) Liquefactive necrosis

(D) Fibrinoid necrosis

Q2. During acute inflammation, what is the correct sequence of leucocyte extravasation from the blood into the tissue?

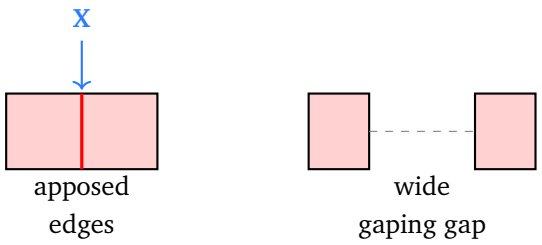
(A) Firm adhesion, then rolling, then margination, then transmigration

(B) Transmigration, then margination, then rolling, then adhesion

(C) Margination, then rolling on selectins, then firm adhesion via integrins, then transmigration

(D) Rolling on selectins, then margination, then transmigration, then firm adhesion

Q3. The diagram compares two healing wounds. Wound **X** has closely apposed edges with minimal tissue loss, scant granulation tissue and a thin scar. This mode of healing is:



(A) Healing by second intention

(B) Healing by first (primary) intention

(C) Healing by wound contraction alone

(D) Keloid formation

Hemodynamic Disorders

Q4. A trauma victim with massive external haemorrhage becomes hypotensive and cold with a rapid thready pulse. Reduced circulating blood volume is driving his collapse. This type of shock is classified as:

(A) Hypovolaemic shock

(B) Cardiogenic shock

(C) Septic shock

(D) Neurogenic shock

Q5. A condition in which widespread activation of coagulation forms microthrombi throughout the small vessels, consuming platelets and clotting factors so that the patient paradoxically bleeds, is:

(A) Immune thrombocytopenic purpura

(B) Disseminated intravascular coagulation

(C) Von Willebrand disease

(D) Thrombotic thrombocytopenic purpura

Neoplasia

Q6. Amplification of which oncogene in a breast carcinoma predicts an aggressive tumour and, importantly, responsiveness to the monoclonal antibody trastuzumab?

(A) KRAS

(B) MYC

(C) BCR-ABL

(D) HER2/neu (ERBB2)

Q7. A patient with small-cell lung carcinoma develops confusion and a serum sodium of 118 mmol/L due to ectopic hormone production by the tumour. Which paraneoplastic syndrome is responsible?

(A) Hypercalcaemia from parathyroid hormone-related peptide

(B) Polycythaemia from ectopic erythropoietin

(C) Carcinoid syndrome from serotonin

(D) Syndrome of inappropriate ADH secretion (SIADH)

Q8. Persistent infection with which virus, especially the high-risk types 16 and 18, is the principal cause of cervical carcinoma?

(A) Epstein-Barr virus

(B) Human papillomavirus (HPV)

(C) Hepatitis B virus

(D) Human herpesvirus-8

Immunopathology

Q9. The tuberculin (Mantoux) skin reaction and contact dermatitis to nickel are classic examples of which type of hypersensitivity?

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

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

(C) Type II (antibody-mediated cytotoxic)

(D) Type III (immune-complex)

Q10. Which pair of autoantibodies is most useful in diagnosing rheumatoid arthritis, the second member being highly specific for the disease?

(A) Rheumatoid factor and anti-CCP (anti-cyclic citrullinated peptide)

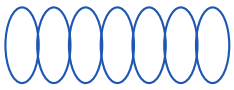
(B) Anti-double-stranded DNA and anti-Smith

(C) Anti-mitochondrial and anti-smooth-muscle

(D) Anti-Scl-70 and anticentromere

Hematology

Q11. A 62-year-old with bone pain has a serum M-band, Bence Jones proteinuria and punched-out lytic skull lesions. The blood smear (shown) has red cells stacked like a pile of coins. The diagnosis is:



red cells stacked like coins (rouleaux)

(A) Chronic lymphocytic leukaemia

(B) Hodgkin lymphoma

(C) Aplastic anaemia

(D) Multiple myeloma

Q12. A young boy has recurrent bleeding into his knee joints (haemarthrosis), a prolonged aPTT with a normal PT and a normal platelet count; the aPTT corrects on mixing with normal plasma. Deficiency of which factor is responsible?

(A) Factor IX

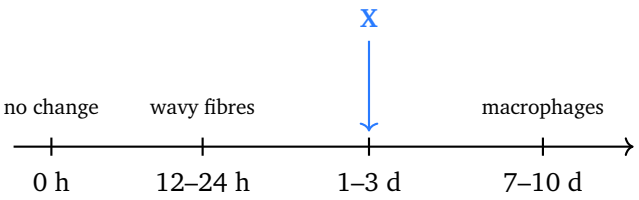
(B) Von Willebrand factor

(C) Factor VIII

(D) Factor VII

Systemic Pathology

Q13. The timeline shows the histological evolution of a myocardial infarct. The interval marked **X**, when a dense neutrophilic infiltrate dominates the necrotic myocardium, corresponds to:



(A) The first 4 hours (no visible change)

(B) 12 to 24 hours (early coagulative necrosis with wavy fibres)

(C) 1 to 3 days (dense neutrophilic infiltrate)

(D) 7 to 10 days (macrophages and early granulation tissue)

Q14. Which feature best distinguishes emphysema from chronic bronchitis in chronic obstructive pulmonary disease?

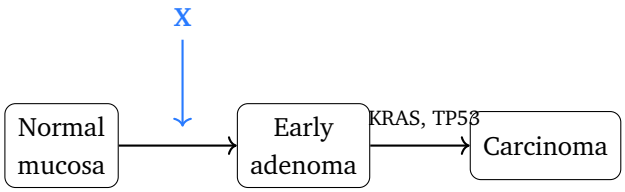
(A) Permanent enlargement of air spaces distal to the terminal bronchiole with destruction of alveolar walls

(B) A chronic productive cough for at least 3 months in 2 consecutive years

(C) An increased Reid index from mucous gland hyperplasia

(D) Hyperplasia of bronchial goblet cells with excess mucus

Q15. The flow diagram shows the adenoma-carcinoma sequence of colorectal cancer. The earliest, initiating mutation marked **X**, which drives normal mucosa to form an early adenoma, is inactivation of which gene?



(A) TP53

(B) KRAS

(C) APC

(D) HER2/neu

Detailed Solutions

Q1.

Solution

Concept – necrosis in acute pancreatitis: Released enzymes produce a distinctive pattern.

Step 1 – name the process: Lipases digest fat and free fatty acids bind calcium to form chalky soaps, called **fat necrosis with saponification**.

Step 2 – appearance: The deposits look like white flecks on the mesentery and omentum.

Why the other options are wrong:

- A, coagulative: typical of ischaemic infarcts in solid organs.
- C, liquefactive: seen in brain infarcts and abscesses.
- D, fibrinoid: seen in vessel walls in immune-mediated injury.

Final Answer: Fat necrosis with saponification $\Rightarrow$ B

Answer: (B) Go Back to Q1

Q2.

Solution

Concept – leucocyte extravasation: Cells leave the vessel in an ordered cascade.

Step 1 – lay out the order: **Margination**, then **rolling** on selectins, then **firm adhesion** via integrins, then **transmigration** through the wall.

Step 2 – molecular basis: Selectins mediate the loose rolling and integrins the tight arrest before diapedesis.

Why the other options are wrong:

- A: puts firm adhesion before rolling, reversing the true order.
- B: begins with transmigration, which is the last step.
- D: places firm adhesion after transmigration, which is impossible.

Final Answer: Margination, rolling, adhesion, transmigration $\Rightarrow$ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – modes of wound healing: Edge apposition decides the pattern.

Step 1 – read wound X: Closely apposed edges with minimal tissue loss heal by **first (primary) intention**, leaving a thin scar.

Step 2 – contrast: A wide gaping defect heals by second intention, with abundant granulation tissue and contraction.

Why the other options are wrong:

- A, second intention: applies to the wide gaping wound, not wound X.
- C, contraction alone: a feature of large open wounds, not clean incisions.
- D, keloid: an abnormal excess of scar collagen, not the normal path shown.

Final Answer: Healing by first (primary) intention $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – classification of shock: Shock is grouped by its underlying cause.

Step 1 – match the case: Massive blood loss lowers circulating volume, which defines **hypovolaemic shock**.

Step 2 – physiology: Reduced preload cuts cardiac output, giving hypotension and a compensatory tachycardia.

Why the other options are wrong:

- B, cardiogenic: pump failure, as after a large infarct.
- C, septic: vasodilation and maldistribution from infection.
- D, neurogenic: loss of vascular tone after spinal injury.

Final Answer: Hypovolaemic shock $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q4

Q5.

Solution

Concept – consumptive coagulopathy: Uncontrolled clotting can end in bleeding.

Step 1 – name it: Disseminated intravascular coagulation lays down microthrombi throughout small vessels.

Step 2 – paradox: Platelets and clotting factors are consumed, so the patient then bleeds and D-dimer rises.

Why the other options are wrong:

- A, ITP: isolated antibody-mediated platelet destruction, no microthrombi.
- C, von Willebrand disease: an inherited bleeding disorder, not consumptive.
- D, TTP: microthrombi occur but from ADAMTS13 deficiency, without the broad factor consumption of DIC.

Final Answer: Disseminated intravascular coagulation $\Rightarrow$ B

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – oncogene amplification in breast cancer: One gene guides targeted therapy.

Step 1 – name it: Amplification of **HER2/neu (ERBB2)** marks an aggressive breast carcinoma.

Step 2 – therapy link: Such tumours respond to trastuzumab, a monoclonal antibody against the HER2 receptor.

Why the other options are wrong:

- A, KRAS: a signalling oncogene of colon, lung and pancreatic cancers.
- B, MYC: amplified in Burkitt lymphoma and others, not the trastuzumab target.
- C, BCR-ABL: the fusion gene of chronic myeloid leukaemia.

Final Answer: HER2/neu (ERBB2) $\Rightarrow$ D

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – paraneoplastic syndromes of lung cancer: Small-cell tumours secrete ectopic hormones.

Step 1 – match the case: Hyponatraemia with confusion from ectopic ADH is the **syndrome of inappropriate ADH secretion (SIADH)**.

Step 2 – mechanism: Excess ADH causes water retention, diluting serum sodium.

Why the other options are wrong:

- A, PTHrP hypercalcaemia: classic of squamous-cell lung carcinoma, and it raises calcium, not lowers sodium.
- B, erythropoietin: causes polycythaemia, typically in renal-cell carcinoma.
- C, carcinoid syndrome: flushing and diarrhoea from serotonin, not hyponatraemia.

Final Answer: SIADH $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q7

Q8.

Solution

Concept – viral carcinogenesis: A specific virus drives cervical cancer.

Step 1 – name it: Human papillomavirus, mainly types 16 and 18, causes cervical carcinoma.

Step 2 – mechanism: Its E6 and E7 proteins inactivate p53 and Rb, releasing the cell cycle.

Why the other options are wrong:

- A, EBV: linked to Burkitt lymphoma and nasopharyngeal carcinoma.
- C, hepatitis B: linked to hepatocellular carcinoma.
- D, HHV-8: linked to Kaposi sarcoma.

Final Answer: Human papillomavirus (HPV) $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q8

Q9.

Solution

Concept – hypersensitivity types: The mechanism names the type.

Step 1 – match the examples: The tuberculin test and contact dermatitis are **Type IV**, delayed and T-cell mediated.

Step 2 – timing: The reaction peaks at 48 to 72 hours as sensitised T cells recruit macrophages.

Why the other options are wrong:

- B, Type I: immediate IgE reaction, as in anaphylaxis.
- C, Type II: antibody against cell-surface antigens.
- D, Type III: immune-complex deposition, as in serum sickness.

Final Answer: Type IV (delayed, cell-mediated) $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – autoantibodies in rheumatoid arthritis: A sensitive plus a specific marker.

Step 1 – name the pair: Rheumatoid factor and anti-CCP are used together; anti-CCP is highly specific for the disease.

Step 2 – use: Anti-CCP also predicts a more erosive, aggressive course.

Why the other options are wrong:

- B, anti-dsDNA and anti-Smith: markers of systemic lupus erythematosus.
- C, anti-mitochondrial and anti-smooth-muscle: markers of autoimmune liver disease.
- D, anti-Scl-70 and anticentromere: markers of systemic sclerosis.

Final Answer: Rheumatoid factor and anti-CCP $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – plasma-cell malignancy: A cluster of findings points to one tumour.

Step 1 – read the case: An M-band, Bence Jones proteinuria, punched-out lytic lesions and rouleaux define **multiple myeloma**.

Step 2 – mechanism: Clonal plasma cells secrete monoclonal immunoglobulin and activate osteoclasts.

Why the other options are wrong:

- A, CLL: a mature B-lymphocyte leukaemia with smudge cells, no M-band or lytic lesions.
- B, Hodgkin lymphoma: Reed-Sternberg cells and nodal disease.
- C, aplastic anaemia: pancytopenia with an empty marrow, no paraprotein.

Final Answer: Multiple myeloma $\Rightarrow$ D

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – inherited bleeding disorders: The coagulation profile localises the defect.

Step 1 – interpret the tests: Haemarthrosis with a prolonged aPTT, normal PT and normal platelets that corrects on mixing points to **factor VIII** deficiency (haemophilia A).

Step 2 – inheritance: It is X-linked recessive, so it affects boys.

Why the other options are wrong:

- A, factor IX: causes haemophilia B, clinically similar but a different factor and less common.
- B, von Willebrand factor: usually prolongs bleeding time with mucosal bleeding, not deep haemarthrosis.
- D, factor VII: prolongs the PT, not the aPTT.

Final Answer: Factor VIII $\Rightarrow$ C

Answer: (C) Go Back to Q12

Q13.

Solution

Concept – histological timeline of infarction: The infarct changes hour by hour.

Step 1 – identify X: A dense neutrophilic infiltrate is maximal at **1 to 3 days** after the infarct.

Step 2 – sequence: Wavy fibres appear at 12 to 24 hours, and macrophages with early granulation tissue at 7 to 10 days.

Why the other options are wrong:

- A, first 4 hours: no light-microscopic change yet.
- B, 12 to 24 hours: early coagulative necrosis, before the neutrophil peak.
- D, 7 to 10 days: macrophages clear debris, the neutrophils have gone.

Final Answer: 1 to 3 days $\Rightarrow$ C

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – emphysema versus chronic bronchitis: Two faces of COPD with different anatomy.

Step 1 – define emphysema: It is **permanent enlargement of air spaces distal to the terminal bronchiole with destruction of alveolar walls**.

Step 2 – contrast: Chronic bronchitis is a clinical diagnosis based on a productive cough and airway mucous gland changes.

Why the other options are wrong:

- B, chronic productive cough for 3 months over 2 years: the clinical definition of chronic bronchitis.
- C, raised Reid index: a mucous-gland change of chronic bronchitis.
- D, goblet cell hyperplasia: an airway feature of chronic bronchitis.

Final Answer: Air-space enlargement with alveolar wall destruction $\Rightarrow$ A

Answer: (A) Go Back to Q14

Q15.

Solution

Concept – adenoma-carcinoma sequence: Colorectal cancer builds up by step-wise mutation.

Step 1 – identify X: The initiating event is inactivation of the **APC** gene, which starts adenoma formation.

Step 2 – later steps: KRAS activation enlarges the adenoma and TP53 loss allows the shift to carcinoma.

Why the other options are wrong:

- A, TP53: a late event permitting malignant transformation, not the first.
- B, KRAS: an intermediate step, after APC loss.
- D, HER2/neu: a breast and gastric oncogene, not part of this sequence.

Final Answer: APC $\Rightarrow$ C

Answer: (C) Go Back to Q15

Answer Key

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