INI CET Pathology

Sample Paper – 1

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 characteristically seen in the lung and lymph nodes in tuberculosis is:

(A) Coagulative necrosis

(B) Liquefactive necrosis

(C) Caseous necrosis

(D) Fat necrosis

Q2. Which change marks the transition from reversible to irreversible cell injury?

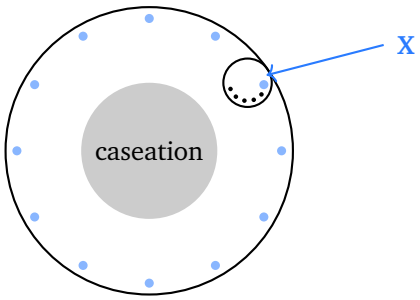
(A) Severe mitochondrial damage with membrane permeability defects

(B) Cellular (hydropic) swelling

(C) Detachment of ribosomes from the endoplasmic reticulum

(D) Fatty change (steatosis)

Q3. In the diagram of a tuberculous granuloma, the multinucleate giant cell with nuclei arranged in a peripheral horseshoe (marked **X**) is a:



(A) Foreign body giant cell

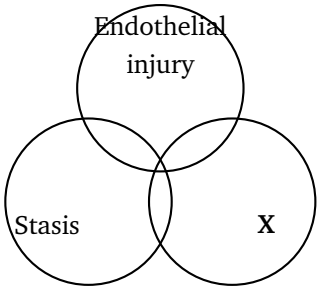
(B) Touton giant cell

(C) Langhans giant cell

(D) Reed-Sternberg cell

Hemodynamic Disorders

Q4. The diagram shows Virchow's triad, the three factors predisposing to thrombosis. Two are endothelial injury and abnormal blood flow (stasis). The third, marked **X**, is:



(A) Hypercoagulability

(B) Hypocalcaemia

(C) Hyperlipidaemia

(D) Anaemia

Q5. The most common source of a pulmonary thromboembolism is the:

(A) Left atrium

(B) Deep veins of the lower limbs

(C) Superior vena cava

(D) Portal vein

Neoplasia

Q6. Which tumour suppressor gene, mutated in Li-Fraumeni syndrome and in most human cancers, is called the "guardian of the genome"?

(A) RB1

(B) APC

(C) BRCA1

(D) TP53 (p53)

Q7. Carcinomas (epithelial malignancies) most characteristically spread first by which route?

(A) Haematogenous spread

(B) Lymphatic spread

(C) Transcoelomic spread

(D) Perineural spread

Q8. Which tumour marker is most strongly associated with hepatocellular carcinoma?

(A) CA-125

(B) Carcinoembryonic antigen (CEA)

(C) Prostate-specific antigen (PSA)

(D) Alpha-fetoprotein (AFP)

Immunopathology

Q9. Anaphylaxis and atopic allergy are mediated by which type of hypersensitivity reaction?

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

(B) Type II (antibody-mediated cytotoxic)

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

(D) Type III (immune-complex)

Q10. Which special stain demonstrates amyloid, giving an apple-green birefringence under polarised light?

(A) Congo red

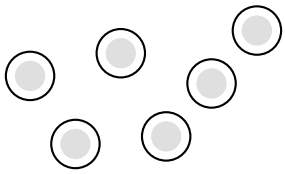
(B) Prussian blue

(C) Periodic acid-Schiff (PAS)

(D) Ziehl-Neelsen

Hematology

Q11. The peripheral smear shown, with small red cells and a widened central pale area, is typical of which anaemia?



small cells, wide central pallor

(A) Macrocytic anaemia

(B) Normocytic normochromic anaemia

(C) Microcytic hypochromic anaemia

(D) Macrocytic anaemia with hypersegmented neutrophils

Q12. The Philadelphia chromosome, a t(9;22) translocation producing the BCR-ABL fusion gene, is the hallmark of:

(A) Acute lymphoblastic leukaemia

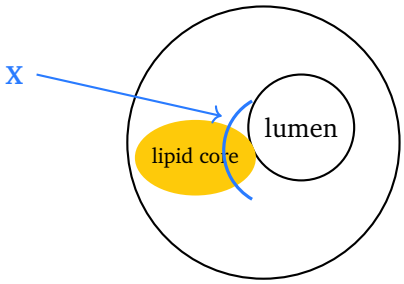
(B) Hodgkin lymphoma

(C) Multiple myeloma

(D) Chronic myeloid leukaemia

Systemic Pathology

Q13. In the cross-section of an atherosclerotic plaque shown, the component marked **X**, whose rupture triggers acute coronary thrombosis, is the:



(A) Fibrous cap

(B) Lipid (necrotic) core

(C) Tunica adventitia

(D) Internal elastic lamina

Q14. The irreversible end stage of chronic liver disease, marked by diffuse fibrosis and regenerative nodules, is:

(A) Fatty liver (steatosis)

(B) Cirrhosis

(C) Acute hepatitis

(D) Hepatic adenoma

Q15. The most common chemical composition of a renal (kidney) stone is:

(A) Uric acid

(B) Calcium oxalate

(C) Struvite (magnesium ammonium phosphate)

(D) Cystine

Detailed Solutions

Q1.

Solution

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

Step 1 – match tuberculosis: TB produces **caseous necrosis**, a cheese-like mix of coagulative and liquefactive change.

Step 2 – microscopy: It shows amorphous granular debris rimmed by a granuloma.

Why the other options are wrong:

- A, coagulative: typical of ischaemic infarcts in solid organs.
- B, liquefactive: seen in brain infarcts and abscesses.
- D, fat necrosis: seen in acute pancreatitis.

Final Answer: Caseous necrosis ⇒ C

Answer: (C) Go Back to Q1

Q2.

Solution

Concept – reversible vs irreversible injury: A point of no return is crossed when key structures fail.

Step 1 – name the change: Severe mitochondrial damage with membrane defects marks irreversibility.

Step 2 – consequence: Loss of ATP and massive calcium influx trigger cell death.

Why the other options are wrong:

- B and C, swelling and ribosomal detachment: features of reversible injury.
- D, fatty change: a reversible accumulation.

Final Answer: Severe mitochondrial and membrane damage ⇒ A

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – giant cells in granulomas: Nuclear arrangement names the cell.

Step 1 – identify X: Peripheral horseshoe-arranged nuclei define a **Langhans giant cell**, typical of TB.

Step 2 – contrast: Foreign body giant cells have haphazard nuclei.

Why the other options are wrong:

- A, foreign body giant cell: random nuclei.
- B, Touton giant cell: ring of nuclei with foamy periphery (fat lesions).
- D, Reed-Sternberg cell: an owl-eye cell of Hodgkin lymphoma, not a giant cell of granulomas.

Final Answer: Langhans giant cell $\Rightarrow$ C

Answer: (C) Go Back to Q3

Q4.

Solution

Concept – Virchow's triad: Three factors promote thrombosis.

Step 1 – name the third: With endothelial injury and stasis, the third is **hypercoagulability**.

Step 2 – examples: Inherited (factor V Leiden) or acquired (malignancy, pregnancy) states.

Why the other options are wrong:

- B, C, D: hypocalcaemia, hyperlipidaemia and anaemia are not components of the triad.

Final Answer: Hypercoagulability $\Rightarrow$ A

Answer: (A) Go Back to Q4

Q5.

Solution

Concept – pulmonary embolism source: Most emboli come from leg veins.

Step 1 – name the source: The **deep veins of the lower limbs** (deep vein thrombosis) are the commonest source.

Step 2 – pathway: Thrombi travel through the IVC and right heart to the pulmonary arteries.

Why the other options are wrong:

- A, left atrium: gives systemic (arterial) emboli.
- C and D, SVC and portal vein: uncommon sources of pulmonary emboli.

Final Answer: Deep veins of the lower limbs $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – key tumour suppressor: One gene guards genomic integrity.

Step 1 – name it: TP53 (p53) arrests the cycle for repair or triggers apoptosis; it is mutated in most cancers.

Step 2 – syndrome link: Germline loss causes Li-Fraumeni syndrome.

Why the other options are wrong:

- A, RB1: retinoblastoma gene, a different checkpoint.
- B, APC: colorectal (familial adenomatous polyposis).
- C, BRCA1: hereditary breast and ovarian cancer.

Final Answer: TP53 (p53) $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – spread of tumours: Carcinomas and sarcomas favour different routes.

Step 1 – name the route: Carcinomas spread first by the **lymphatics** to regional nodes.

Step 2 – contrast: Sarcomas favour haematogenous (blood) spread.

Why the other options are wrong:

- A, haematogenous: more typical of sarcomas.
- C, transcoelomic: seen in ovarian and GI cancers, but not the general first route.
- D, perineural: characteristic of a few tumours (e.g. prostate, pancreas), not the rule.

Final Answer: Lymphatic spread $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q7

Q8.

Solution

Concept – tumour markers: Each marker maps to a tumour.

Step 1 – match hepatocellular carcinoma: Alpha-fetoprotein (AFP) is raised in HCC (and in germ cell tumours).

Step 2 – use: It aids diagnosis and monitoring of treatment.

Why the other options are wrong:

- A, CA-125: ovarian carcinoma.
- B, CEA: colorectal carcinoma.
- C, PSA: prostate carcinoma.

Final Answer: Alpha-fetoprotein (AFP) $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q8

Q9.

Solution

Concept – hypersensitivity types: Four classic types, each with a mechanism.

Step 1 – match anaphylaxis: Anaphylaxis and atopy are **Type I**, IgE-mediated on mast cells.

Step 2 – mediators: Histamine and leukotrienes cause the immediate reaction.

Why the other options are wrong:

- A, Type IV: delayed, T-cell mediated (e.g. contact dermatitis).
- B, Type II: antibody against cell antigens (e.g. haemolysis).
- D, Type III: immune-complex disease (e.g. serum sickness).

Final Answer: Type I $\Rightarrow$ C

Answer: (C) Go Back to Q9

Q10.

Solution

Concept – special stains: A stain identifies a deposit.

Step 1 – name the stain: Congo red stains amyloid, giving apple-green birefringence in polarised light.

Step 2 – significance: This confirms amyloid deposition in tissue.

Why the other options are wrong:

- B, Prussian blue: iron (haemosiderin).
- C, PAS: glycogen and basement membranes.
- D, Ziehl-Neelsen: acid-fast bacilli.

Final Answer: Congo red $\Rightarrow$ A

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – red-cell morphology: Size and pallor point to the cause.

Step 1 – read the smear: Small cells with a wide central pallor are **microcytic hypochromic**, typical of iron deficiency anaemia.

Step 2 – confirm: Iron studies show low ferritin and high total iron-binding capacity.

Why the other options are wrong:

- A and D, macrocytic: large cells, seen in B12/folate deficiency.
- B, normocytic normochromic: normal size and colour (e.g. anaemia of chronic disease).

Final Answer: Microcytic hypochromic anaemia $\Rightarrow$ C

Answer: (C) Go Back to Q11

Q12.

Solution

Concept – diagnostic translocations: A translocation pins the diagnosis.

Step 1 – match the Philadelphia chromosome: t(9;22) with BCR-ABL defines **chronic myeloid leukaemia**.

Step 2 – treatment link: The fusion tyrosine kinase is targeted by imatinib.

Why the other options are wrong:

- A, ALL: may rarely carry Ph, but CML is the classic association.
- B, Hodgkin lymphoma: Reed-Sternberg cells, no Ph chromosome.
- C, multiple myeloma: plasma-cell disorder, unrelated translocations.

Final Answer: Chronic myeloid leukaemia $\Rightarrow$ D

Answer: (D) Go Back to Q12

Q13.

Solution

Concept – atherosclerotic plaque: A thin cap over a lipid core is dangerous.

Step 1 – identify X: The **fibrous cap** overlies the lipid core; its rupture exposes thrombogenic material.

Step 2 – consequence: Rupture triggers acute thrombosis and myocardial infarction.

Why the other options are wrong:

- B, lipid core: lies beneath the cap, labelled separately.
- C and D, adventitia and internal elastic lamina: vessel-wall layers, not the cap.

Final Answer: Fibrous cap $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q13

Q14.

Solution

Concept – chronic liver disease: The end stage has a defined architecture.

Step 1 – name it: Cirrhosis shows diffuse fibrosis with regenerative nodules.

Step 2 – consequences: Portal hypertension and hepatic failure follow.

Why the other options are wrong:

- A, steatosis: reversible fat accumulation.
- C, acute hepatitis: inflammation without established nodular fibrosis.
- D, hepatic adenoma: a benign tumour, not diffuse scarring.

Final Answer: Cirrhosis $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q14

Q15.

Solution

Concept – renal calculi: One type dominates.

Step 1 – name it: Calcium oxalate stones are the most common (about 75%).

Step 2 – clue: They are radio-opaque and form in normal or acidic urine.

Why the other options are wrong:

- A, uric acid: radiolucent, in acidic urine and gout.
- C, struvite: staghorn stones with urease-producing infection.
- D, cystine: rare, in cystinuria.

Final Answer: Calcium oxalate $\Rightarrow$ B

Answer: (B) Go Back to Q15

Answer Key

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