INI CET Pathology

Sample Paper – 3

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 type of necrosis is characteristically seen in infarcts of the brain and in abscesses, where the dead tissue is digested to a soft, liquid mass?

(A) Liquefactive necrosis

(B) Coagulative necrosis

(C) Fibrinoid necrosis

(D) Fat necrosis

Q2. Deposition of calcium salts in dead or degenerated tissue while the serum calcium level remains normal is termed:

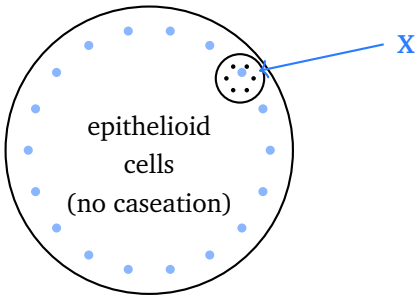
(A) Metastatic calcification

(B) Psammomatous calcification

(C) Dystrophic calcification

(D) Calciphylaxis

Q3. The schematic shows a granuloma made of epithelioid cells with a multinucleate giant cell (marked **X**) but with **no central caseation**. This non-caseating granuloma is most characteristic of:



(A) Tuberculosis

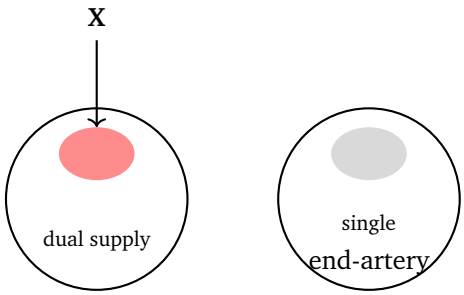
(B) Syphilitic gumma

(C) Lepromatous leprosy

(D) Sarcoidosis

Hemodynamic Disorders

Q4. In the schematic, organ **X** develops a red (haemorrhagic) infarct, whereas the organ on the right develops a pale (anaemic) infarct. Red infarcts characteristically occur in tissues that:



(A) Are solid with a single end-arterial supply, such as the kidney

(B) Are highly resistant to hypoxia

(C) Are supplied by end arteries in dense, compact organs

(D) Have a dual or collateral blood supply, such as lung and intestine

Q5. A transudate producing generalised oedema, as in congestive heart failure and the nephrotic syndrome, results mainly from:

(A) Increased vascular permeability from acute inflammation

(B) Lymphatic obstruction alone

(C) Raised hydrostatic pressure and/or reduced plasma oncotic (colloid osmotic) pressure

(D) A protein-rich exudate laden with fibrin

Neoplasia

Q6. A malignant tumour arising from epithelium is called a carcinoma. A malignant tumour arising from mesenchymal (connective) tissue is called a:

(A) Sarcoma

(B) Adenoma

(C) Papilloma

(D) Teratoma

Q7. The Knudson "two-hit" hypothesis, requiring inactivation of both alleles of a tumour-suppressor gene before a tumour develops, was proposed to explain the inheritance of:

(A) Retinoblastoma (RB1)

(B) Chronic myeloid leukaemia

(C) Burkitt lymphoma

(D) Follicular lymphoma

Q8. Which serum tumour marker is most useful for monitoring epithelial (serous) carcinoma of the ovary?

(A) Prostate-specific antigen (PSA)

(B) CA-125

(C) Carcinoembryonic antigen (CEA)

(D) Beta-human chorionic gonadotropin (β-hCG)

Immunopathology

Q9. Serum sickness, post-streptococcal glomerulonephritis and the Arthus reaction are all mediated by which type of hypersensitivity?

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

(B) Type II (antibody-mediated cytotoxic)

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

(D) Type III (immune-complex)

Q10. In primary amyloidosis linked to a plasma-cell dyscrasia, the amyloid fibril protein (AL type) is derived from:

(A) Serum amyloid-associated (SAA) protein

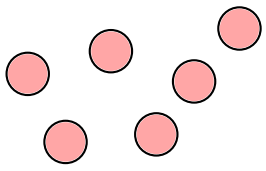
(B) Immunoglobulin light chains

(C) Transthyretin

(D) Beta-2 microglobulin

Hematology

Q11. The peripheral smear shown has small, densely staining red cells that lack the normal central pale zone. Together with increased osmotic fragility and splenomegaly, this points to:



dense cells, no central pallor

(A) Iron deficiency anaemia

(B) Hereditary spherocytosis

(C) Megaloblastic anaemia

(D) Aplastic anaemia

Q12. A large binucleate "owl-eye" cell with two mirror-image nuclei and prominent inclusion-like nucleoli (the Reed-Sternberg cell) is the diagnostic hallmark of:

(A) Burkitt lymphoma

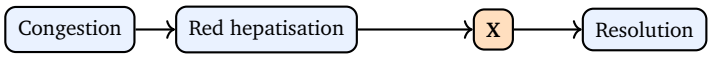
(B) Chronic lymphocytic leukaemia

(C) Hodgkin lymphoma

(D) Multiple myeloma

Systemic Pathology

Q13. The flow diagram shows the four sequential stages of lobar pneumonia. Stage **X**, in which the lung is dry, firm and grey-brown with a fibrinosuppurative exudate and disintegrating red cells, is:



(A) Congestion

(B) Red hepatisation

(C) Grey hepatisation

(D) Resolution

Q14. Which feature favours a diagnosis of Crohn disease over ulcerative colitis?

(A) Continuous mucosal disease beginning in the rectum

(B) Transmural inflammation with skip lesions and non-caseating granulomas

(C) Inflammation confined to mucosa and submucosa

(D) Disease limited to the colon with no small-bowel involvement

Q15. Optically clear "Orphan-Annie-eye" nuclei, nuclear grooves and psammoma bodies are the diagnostic microscopic features of:

(A) Papillary carcinoma of the thyroid

(B) Follicular carcinoma of the thyroid

(C) Medullary carcinoma of the thyroid

(D) Anaplastic carcinoma of the thyroid

Detailed Solutions

Q1.

Solution

Concept – patterns of necrosis: The morphology of dead tissue points to the organ and mechanism.

Step 1 – match brain and abscess: Enzymatic digestion turns the dead tissue into a soft, liquid mass called **liquefactive necrosis**.

Step 2 – why here: The brain is rich in lipid and hydrolytic enzymes, and abscesses are full of neutrophil enzymes, so both liquefy.

Why the other options are wrong:

- B, coagulative: seen in ischaemic infarcts of solid organs like the kidney and heart.
- C, fibrinoid: seen in vessel walls in malignant hypertension and vasculitis.
- D, fat necrosis: seen in acute pancreatitis and in breast trauma.

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

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – pathological calcification: There are two types, sorted by the serum calcium level.

Step 1 – read the clue: Calcium is laid down in dead or damaged tissue while serum calcium is **normal**.

Step 2 – name it: Calcification in injured tissue with normal serum calcium is **dystrophic calcification**.

Why the other options are wrong:

- A, metastatic calcification: occurs in normal tissue when serum calcium is *raised* (hypercalcaemia).
- B, psammomatous calcification: a laminated pattern (psammoma body), a subtype, not the general term asked.
- D, calciphylaxis: a specific vascular calcification syndrome in renal failure, not this definition.

Final Answer: Dystrophic calcification ⇒ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – caseating vs non-caseating granuloma: The presence or absence of central caseation separates the causes.

Step 1 – read the figure: Epithelioid cells and a giant cell form the granuloma, but there is **no central caseation**.

Step 2 – match the cause: A non-caseating granuloma is the classic finding of **sarcoidosis**.

Why the other options are wrong:

- A, tuberculosis: gives a *caseating* granuloma.
- B, syphilitic gumma: shows central coagulative-type necrosis.
- C, lepromatous leprosy: sheets of foamy macrophages full of bacilli, not tight non-caseating granulomas.

Final Answer: Sarcoidosis ⇒ D

Answer: (D) Go Back to Q3

Q4.

Solution

Concept – red vs white infarcts: The colour of an infarct depends on the tissue's blood supply.

Step 1 – identify X: Organ X bleeds into the dead area, giving a **red (haemorrhagic) infarct**.

Step 2 – which tissues: Red infarcts occur where a **dual or collateral blood supply** lets blood seep back in, as in the **lung and intestine**, and after venous occlusion.

Why the other options are wrong:

- A and C, single end-arterial supply in a compact organ: produce pale (white) infarcts, as in kidney, spleen and heart.
- B, resistance to hypoxia: does not determine infarct colour.

Final Answer: Dual or collateral blood supply (lung, intestine) ⇒ D

Answer: (D) Go Back to Q4

Q5.

Solution

Concept – transudate vs exudate: A transudate is a low-protein fluid driven by pressure forces.

Step 1 – name the mechanism: Transudative oedema comes from **raised hydrostatic pressure and/or low plasma oncotic pressure**.

Step 2 – link to the examples: Heart failure raises hydrostatic pressure; the nephrotic syndrome loses albumin, lowering oncotic pressure.

Why the other options are wrong:

- A, increased permeability: produces an *exudate* of inflammation, not a transudate.
- B, lymphatic obstruction: causes lymphoedema, a separate mechanism.
- D, protein-rich fibrin fluid: describes an exudate, the opposite of a transudate.

Final Answer: Raised hydrostatic and/or low oncotic pressure ⇒ C

Answer: (C) Go Back to Q5

Q6.

Solution

Concept – tumour nomenclature: The name follows the tissue of origin and whether it is benign or malignant.

Step 1 – apply the rule: Malignant epithelial tumour = carcinoma; malignant mesenchymal tumour = **sarcoma**.

Step 2 – example: A malignant tumour of bone is an osteosarcoma; of fat, a liposarcoma.

Why the other options are wrong:

- B, adenoma: a *benign* glandular epithelial tumour.
- C, papilloma: a benign epithelial tumour with finger-like fronds.
- D, teratoma: a germ-cell tumour with tissues from more than one germ layer.

Final Answer: Sarcoma $\Rightarrow$ A

Answer: (A) Go Back to Q6

Q7.

Solution

Concept – Knudson two-hit hypothesis: Both copies of a tumour-suppressor gene must be lost for a tumour to form.

Step 1 – match the classic example: The hypothesis was framed for **retinoblastoma**, from loss of both RB1 alleles.

Step 2 – inherited vs sporadic: Familial cases inherit one defective allele (first hit) and need only one more somatic hit.

Why the other options are wrong:

- B, chronic myeloid leukaemia: driven by the BCR-ABL fusion (an oncogene), not two-hit loss.
- C, Burkitt lymphoma: driven by MYC translocation, an activated oncogene.
- D, follicular lymphoma: driven by BCL2 translocation, again an oncogene mechanism.

Final Answer: Retinoblastoma (RB1) $\Rightarrow$ A

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – tumour markers: Each marker maps best to one tumour for monitoring.

Step 1 – match ovarian carcinoma: CA-125 is the serum marker used to follow epithelial (serous) ovarian carcinoma.

Step 2 – use: Rising CA-125 after surgery suggests recurrence.

Why the other options are wrong:

- A, PSA: prostate carcinoma.
- C, CEA: colorectal carcinoma.
- D, β-hCG: gestational trophoblastic disease and some germ cell tumours.

Final Answer: CA-125 $\Rightarrow$ B

Answer: (B) Go Back to Q8

Q9.

Solution

Concept – hypersensitivity types: Immune-complex deposition defines one whole class.

Step 1 – match the examples: Serum sickness, post-streptococcal glomerulonephritis and the Arthus reaction are all **Type III**, immune-complex reactions.

Step 2 – mechanism: Antigen-antibody complexes deposit in vessel walls and tissues, fixing complement and drawing in neutrophils.

Why the other options are wrong:

- A, Type I: immediate, IgE-mediated allergy.
- B, Type II: antibody directed against fixed cell or tissue antigens.
- C, Type IV: delayed, T-cell mediated (e.g. contact dermatitis).

Final Answer: Type III (immune-complex) $\Rightarrow$ D

Answer: (D) Go Back to Q9

Q10.

Solution

Concept – chemical types of amyloid: The two main fibril proteins are AL and AA.

Step 1 – identify AL: In a plasma-cell dyscrasia the **AL** fibril is made of **immunoglobulin light chains**.

Step 2 – contrast with AA: The AA fibril, in chronic inflammation, is derived from serum amyloid-associated (SAA) protein.

Why the other options are wrong:

- A, SAA protein: the source of AA amyloid, not AL.
- C, transthyretin: gives senile and hereditary (ATTR) amyloid.
- D, beta-2 microglobulin: gives dialysis-associated amyloid.

Final Answer: Immunoglobulin light chains $\Rightarrow$ B

Answer: (B) Go Back to Q10

Q11.

Solution

Concept – red-cell shape on the smear: Spherical cells that have lost their central pallor are spherocytes.

Step 1 – read the smear: Small, dense cells with **no central pale zone** are spherocytes.

Step 2 – put it together: Spherocytes plus increased osmotic fragility and splenomegaly indicate **hereditary spherocytosis**, from a red-cell membrane (spectrin/ankyrin) defect.

Why the other options are wrong:

- A, iron deficiency: small cells but with a *widened* central pallor (hypochromia).
- C, megaloblastic anaemia: large oval cells with hypersegmented neutrophils.
- D, aplastic anaemia: pancytopenia with normal-looking red cells, not spherocytes.

Final Answer: Hereditary spherocytosis $\Rightarrow$ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – the diagnostic cell of Hodgkin lymphoma: One cell defines the disease.

Step 1 – identify the cell: The binucleate "owl-eye" Reed-Sternberg cell is the hallmark of **Hodgkin lymphoma**.

Step 2 – immunophenotype: Classical Reed-Sternberg cells are CD15 and CD30 positive.

Why the other options are wrong:

- A, Burkitt lymphoma: a "starry-sky" pattern of tingible-body macrophages, no Reed-Sternberg cells.
- B, chronic lymphocytic leukaemia: small mature lymphocytes and smudge cells.
- D, multiple myeloma: sheets of plasma cells, not Reed-Sternberg cells.

Final Answer: Hodgkin lymphoma $\Rightarrow$ C

Answer: (C) Go Back to Q12

Q13.

Solution

Concept – stages of lobar pneumonia: The lung passes through four stages in order.

Step 1 – order the stages: Congestion → red hepatisation → **grey hepatisation** → resolution.

Step 2 – identify X: The dry, firm, grey-brown lung with fibrinosuppurative exudate and breaking-down red cells is **grey hepatisation**.

Why the other options are wrong:

- A, congestion: heavy, boggy, red lung with vascular engorgement (stage 1).
- B, red hepatisation: red, firm, liver-like lung packed with red cells and fibrin (stage 2).
- D, resolution: enzymatic clearing of the exudate and return towards normal (stage 4).

Final Answer: Grey hepatisation ⇒ C

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – Crohn disease vs ulcerative colitis: The two inflammatory bowel diseases differ in depth and distribution.

Step 1 – name the Crohn features: Transmural inflammation, skip lesions and non-caseating granulomas favour Crohn disease.

Step 2 – consequences: Full-thickness disease leads to fistulae, strictures and cobblestone mucosa anywhere from mouth to anus.

Why the other options are wrong:

- A, continuous disease from the rectum: this is ulcerative colitis.
- C, mucosa/submucosa only: shallow inflammation typical of ulcerative colitis.
- D, colon only: ulcerative colitis is confined to the colon, whereas Crohn often involves the small bowel.

Final Answer: Transmural inflammation with skip lesions and granulomas $\Rightarrow$ B

Answer: (B) Go Back to Q14

Q15.

Solution

Concept – thyroid carcinoma morphology: Nuclear features, not architecture, define papillary carcinoma.

Step 1 – read the clues: Clear "Orphan-Annie-eye" nuclei, nuclear grooves and psammoma bodies point to **papillary carcinoma of the thyroid**.

Step 2 – behaviour: It is the commonest thyroid cancer and spreads mainly by lymphatics, with a good prognosis.

Why the other options are wrong:

- B, follicular carcinoma: diagnosed by capsular/vascular invasion, spreads by blood, lacks these nuclei.
- C, medullary carcinoma: C-cell tumour with amyloid stroma and raised calcitonin.
- D, anaplastic carcinoma: highly pleomorphic, undifferentiated cells in elderly patients.

Final Answer: Papillary carcinoma of the thyroid $\Rightarrow$ A

Answer: (A) Go Back to Q15

Answer Key

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