INI CET Pathology

Sample Paper – 10

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. During ischaemia-reperfusion injury, a rise in one cytosolic ion in irreversibly injured cells activates phospholipases, proteases and endonucleases and drives cell death. This overloaded ion is:

(A) Potassium

(B) Magnesium

(C) Calcium

(D) Chloride

Q2. Which complement fragment acts both as an anaphylatoxin and as the most potent chemoattractant that recruits neutrophils to a site of acute inflammation?

(A) C1q

(B) C3b

(C) C5a

(D) C9

Q3. A raised, firm scar that continues to grow and extends beyond the original wound margins because of excess deposition of type I collagen is termed a:

(A) Keloid

(B) Hypertrophic scar

(C) Wound contracture

(D) Pyogenic granuloma

Hemodynamic Disorders

Q4. A pleural effusion is found to have a high protein content and a specific gravity above 1.020. By the concept underlying Light's criteria, this fluid is best classified as a(n):

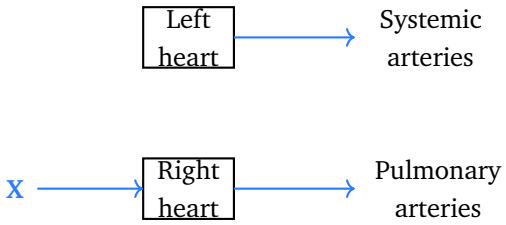
(A) Exudate

(B) Transudate

(C) Chylous effusion

(D) Simple lymphatic filtrate

Q5. The diagram contrasts two embolic pathways. A mural thrombus in the left ventricle throws off systemic (arterial) emboli, whereas an embolus that lodges in the pulmonary arteries almost always originates from the site marked **X**, namely the:



(A) Left atrial appendage

(B) Deep veins of the systemic (lower-limb) circulation

(C) Aortic arch

(D) Coronary arteries

Neoplasia

Q6. Which combination of features best defines anaplasia, the morphological hallmark of malignant transformation?

(A) Uniform cells with a low nuclear-to-cytoplasmic ratio

(B) A fibrous capsule with slow, expansile growth

(C) Well-formed glands closely resembling the tissue of origin

(D) Marked pleomorphism, a high nuclear-to-cytoplasmic ratio and abnormal mitotic figures

Q7. Neurofibromatosis type 1 is caused by loss of a tumour-suppressor gene whose product, neurofibromin, is a GTPase-activating protein that switches off Ras signalling. This gene is:

(A) NF1

(B) RB1

(C) VHL

(D) APC

Q8. Which serum tumour marker is most useful for detecting recurrence during follow-up of a patient treated for colorectal carcinoma?

(A) Prostate-specific antigen (PSA)

(B) Carcinoembryonic antigen (CEA)

(C) CA-125

(D) Alpha-fetoprotein (AFP)

Immunopathology

Q9. A renal transplant slowly loses function over two years and biopsy shows dense vascular intimal fibrosis with narrowing of graft arteries. This pattern is characteristic of:

(A) Hyperacute rejection

(B) Acute cellular (interstitial) rejection

(C) Acute humoral rejection

(D) Chronic rejection

Q10. A young adult presents with repeated episodes of disseminated Neisseria (meningococcal) infection. The most likely underlying immune defect is a deficiency of:

(A) Secretory IgA

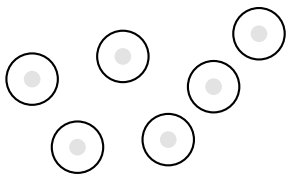
(B) C1 esterase inhibitor

(C) C3

(D) Terminal complement components C5-C9 (the membrane attack complex)

Hematology

Q11. A woman with long-standing rheumatoid arthritis has anaemia with a low serum iron, a raised serum ferritin and a high serum hepcidin. Her smear (below) shows normocytic normochromic red cells with a normal central pallor. The diagnosis is:



normal size, normal (thin) central pallor

(A) Iron deficiency anaemia

(B) Anaemia of chronic disease

(C) Sideroblastic anaemia

(D) Megaloblastic anaemia

Q12. The commonest leukaemia of childhood, whose lymphoblasts are typically positive for terminal deoxynucleotidyl transferase (TdT), is:

(A) Chronic myeloid leukaemia

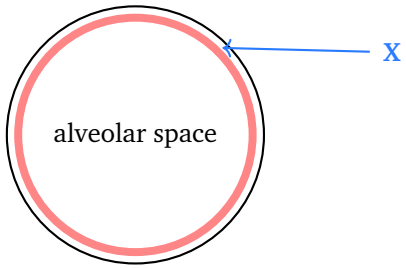
(B) Acute lymphoblastic leukaemia

(C) Chronic lymphocytic leukaemia

(D) Acute myeloid leukaemia

Systemic Pathology

Q13. The diagram shows an alveolus lined by a thick, glassy eosinophilic membrane (marked **X**). Together with diffuse alveolar damage, this hyaline membrane is the defining lesion of:



thick hyaline membrane lining the alveolus

(A) Acute respiratory distress syndrome (ARDS)

(B) Lobar pneumonia

(C) Centriacinar emphysema

(D) Simple cardiogenic pulmonary oedema

Q14. Which of the following is a well-established risk factor for the development of hepatocellular carcinoma?

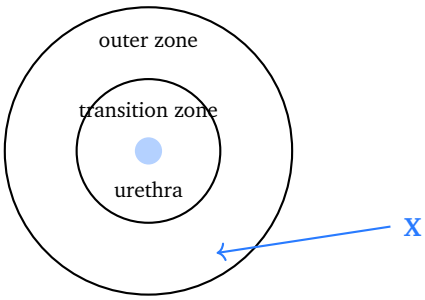
(A) Hepatitis A infection

(B) Gilbert syndrome

(C) Aflatoxin B_1 exposure

(D) Autoimmune haemolytic anaemia

Q15. In the prostate cross-section shown, benign prostatic hyperplasia arises in the transition zone around the urethra, whereas prostatic adenocarcinoma most often arises in the outer region marked **X**, the:



(A) Transition zone

(B) Central zone

(C) Peripheral zone

(D) Periurethral zone

Detailed Solutions

Q1.

Solution

Concept – final common pathway of cell death: One ion, when it floods the cytosol, executes the cell.

Step 1 – name the ion: Sustained cytosolic **calcium** overload marks irreversible injury in ischaemia-reperfusion.

Step 2 – mechanism: Calcium activates phospholipases, proteases, endonucleases and ATPases, damaging membranes, proteins and DNA.

Why the other options are wrong:

- A, potassium: it leaks out of the injured cell, it does not overload the cytosol.
- B, magnesium: not the executioner ion of cell death.
- D, chloride: shifts with water in swelling but does not trigger the death enzymes.

Final Answer: Calcium $\Rightarrow$ C

Answer: (C) Go Back to Q1

Q2.

Solution

Concept – complement anaphylatoxins: C3a and C5a are anaphylatoxins, but one does more.

Step 1 – name the fragment: **C5a** is an anaphylatoxin and the most potent chemoattractant for neutrophils.

Step 2 – actions: It also activates the respiratory burst and up-regulates leukocyte adhesion molecules.

Why the other options are wrong:

- A, C1q: recognises antibody and starts the classical pathway; not a chemoattractant.
- B, C3b: the major opsonin, not an anaphylatoxin.
- D, C9: polymerises to form the membrane attack complex; it does not recruit neutrophils.

Final Answer: C5a $\Rightarrow$ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – aberrant wound healing: Scar that overshoots the wound is a specific entity.

Step 1 – name it: A **keloid** is excess type I collagen that grows beyond the original wound margins.

Step 2 – clinical note: Keloids are commoner in darker skin and tend to recur after excision.

Why the other options are wrong:

- B, hypertrophic scar: raised but stays within the wound margins and may regress.
- C, wound contracture: shrinkage of a healing wound by myofibroblasts, not excess collagen beyond the edge.
- D, pyogenic granuloma: a capillary proliferation, not a collagen scar.

Final Answer: Keloid $\Rightarrow$ A

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – exudate versus transudate: Protein content and specific gravity separate the two.

Step 1 – classify the fluid: High protein with specific gravity above 1.020 defines an **exudate**.

Step 2 – why: An exudate arises from increased vascular permeability in inflammation, so protein-rich fluid escapes (the basis of Light's criteria).

Why the other options are wrong:

- B, transudate: low protein, specific gravity below 1.012, driven by pressure changes.
- C, chylous effusion: milky, lymph-rich fluid from lymphatic obstruction, a distinct category.
- D, simple lymphatic filtrate: not a standard pleural-fluid class defined by

these criteria.

Final Answer: Exudate ⇒ A

Answer: (A) Go Back to Q4

Q5.

Solution

Concept – systemic versus pulmonary emboli: Where an embolus lands depends on where it starts.

Step 1 – identify X: A pulmonary embolus originates from the **deep veins of the systemic circulation**, chiefly the lower-limb veins.

Step 2 – pathway: Venous thrombi pass through the right heart into the pulmonary arteries, whereas left-heart thrombi seed the systemic arteries.

Why the other options are wrong:

- A, left atrial appendage: a source of systemic (arterial) emboli, as in atrial fibrillation.
- C, aortic arch: feeds the systemic arterial tree, not the lungs.
- D, coronary arteries: supply the heart; they are not a source of pulmonary emboli.

Final Answer: Deep systemic veins ⇒ B

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – anaplasia: Malignancy shows loss of differentiation.

Step 1 – pick the features: Anaplasia means **marked pleomorphism, a high nuclear-to-cytoplasmic ratio and abnormal mitotic figures**.

Step 2 – add-ons: Hyperchromatic nuclei, prominent nucleoli and loss of polarity complete the picture.

Why the other options are wrong:

- A, uniform cells with a low N:C ratio: features of benign or normal tissue.
- B, capsule with slow expansile growth: a hallmark of a benign tumour.

- C, well-formed glands resembling the origin: describes a well-differentiated, not anaplastic, tumour.

Final Answer: Pleomorphism, high N:C ratio, abnormal mitoses $\Rightarrow$ D

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – tumour-suppressor genes and syndromes: Each inherited cancer syndrome maps to a gene.

Step 1 – name the gene: NF1 encodes neurofibromin and is lost in neurofibromatosis type 1.

Step 2 – function: Neurofibromin is a GTPase-activating protein that inactivates Ras; its loss leaves Ras signalling switched on.

Why the other options are wrong:

- B, RB1: retinoblastoma and osteosarcoma, a cell-cycle checkpoint gene.
- C, VHL: von Hippel-Lindau disease with renal cell carcinoma and haemangioblastomas.
- D, APC: familial adenomatous polyposis and colorectal cancer.

Final Answer: NF1 $\Rightarrow$ A

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – tumour markers in follow-up: Markers monitor recurrence more than they diagnose.

Step 1 – match colorectal carcinoma: Carcinoembryonic antigen (CEA) is used to detect recurrence after resection.

Step 2 – how: A falling CEA after surgery, then a rising level on follow-up, flags recurrent disease.

Why the other options are wrong:

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

- D, AFP: hepatocellular and germ cell tumours.

Final Answer: Carcinoembryonic antigen (CEA) $\Rightarrow$ B

Answer: (B) Go Back to Q8

Q9.

Solution

Concept – timing and morphology of graft rejection: Each type of rejection has its own tempo and lesion.

Step 1 – read the biopsy: Slow loss over years with vascular intimal fibrosis is **chronic rejection**.

Step 2 – contrast: Acute rejection shows an interstitial cellular infiltrate or acute vasculitis, not established fibrosis.

Why the other options are wrong:

- A, hyperacute: minutes to hours, from preformed antibody and thrombosis.
- B, acute cellular: days to weeks, T-cell interstitial infiltrate.
- C, acute humoral: antibody-mediated acute vasculitis, again not slow intimal fibrosis.

Final Answer: Chronic rejection $\Rightarrow$ D

Answer: (D) Go Back to Q9

Q10.

Solution

Concept – complement deficiency and infection: The defect predicts the organism.

Step 1 – name the defect: Recurrent Neisseria infection points to deficiency of the **terminal complement components C5-C9**, the membrane attack complex.

Step 2 – why Neisseria: Killing of Neisseria depends heavily on membrane-attack-complex lysis, so losing it leaves these organisms free to disseminate.

Why the other options are wrong:

- A, IgA deficiency: mucosal and sinopulmonary infections, not disseminated Neisseria.
- B, C1 esterase inhibitor: causes hereditary angio-oedema, not this infection

pattern.
- C, C3 deficiency: severe pyogenic infections with encapsulated bacteria, a broader picture.

Final Answer: Terminal complement C5-C9 (MAC) $\Rightarrow$ D

Answer: (D) Go Back to Q10

Q11.

Solution

Concept – iron-restricted anaemias: Iron studies separate two look-alikes.

Step 1 – read the numbers: Low serum iron with **raised ferritin** and high hepcidin, plus normocytic normochromic cells, is **anaemia of chronic disease**.

Step 2 – mechanism: Inflammatory hepcidin traps iron in macrophages, so serum iron falls while stored ferritin stays high.

Why the other options are wrong:

- A, iron deficiency: ferritin is low and cells are microcytic hypochromic.
- C, sideroblastic anaemia: ring sideroblasts with a raised iron and often dimorphic cells.
- D, megaloblastic anaemia: macrocytic cells with hypersegmented neutrophils.

Final Answer: Anaemia of chronic disease $\Rightarrow$ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – childhood leukaemia: Age and a marker fix the diagnosis.

Step 1 – name it: Acute lymphoblastic leukaemia is the commonest leukaemia of childhood, and its blasts are TdT positive.

Step 2 – confirm: TdT, a nuclear DNA polymerase, marks immature lymphoblasts and is negative in myeloid blasts.

Why the other options are wrong:

- A, chronic myeloid leukaemia: an adult disease with the Philadelphia chromosome.

- C, chronic lymphocytic leukaemia: elderly patients, mature B cells, TdT negative.
- D, acute myeloid leukaemia: myeloid blasts with Auer rods, TdT negative.

Final Answer: Acute lymphoblastic leukaemia $\Rightarrow$ B

Answer: (B) Go Back to Q12

Q13.

Solution

Concept – diffuse alveolar damage: The hyaline membrane is the signature lesion.

Step 1 – identify X: The glassy eosinophilic lining is a **hyaline membrane**; with diffuse alveolar damage it defines **ARDS**.

Step 2 – make-up: The membrane is fibrin and necrotic epithelial debris left by injured alveolar walls.

Why the other options are wrong:

- B, lobar pneumonia: alveoli filled with neutrophil-rich exudate, not hyaline membranes.
- C, centriacinar emphysema: destroyed, dilated air spaces without membranes.
- D, cardiogenic pulmonary oedema: watery transudate in alveoli, no hyaline membranes or diffuse alveolar damage.

Final Answer: Acute respiratory distress syndrome $\Rightarrow$ A

Answer: (A) Go Back to Q13

Q14.

Solution

Concept – risk factors for hepatocellular carcinoma: Chronic liver injury and a specific toxin drive HCC.

Step 1 – pick the factor: Aflatoxin B_1, from Aspergillus-contaminated grain, is a recognised HCC risk factor.

Step 2 – company it keeps: Cirrhosis and chronic hepatitis B and C are the other major risks; aflatoxin causes a specific TP53 mutation.

Why the other options are wrong:

- A, hepatitis A: an acute, self-limiting infection that does not cause HCC.
- B, Gilbert syndrome: a benign unconjugated hyperbilirubinaemia, no cancer risk.
- D, autoimmune haemolytic anaemia: a red-cell disorder, unrelated to HCC.

Final Answer: Aflatoxin B_1 exposure $\Rightarrow$ C

Answer: (C) Go Back to Q14

Q15.

Solution

Concept – zonal anatomy of the prostate: Benign and malignant disease favour different zones.

Step 1 – identify X: Prostatic adenocarcinoma arises mainly in the **peripheral zone**, the outer region marked X.

Step 2 – contrast: Benign prostatic hyperplasia arises in the transition zone around the urethra, causing obstructive symptoms; peripheral tumours are felt on digital rectal examination.

Why the other options are wrong:

- A, transition zone: the site of benign prostatic hyperplasia, not carcinoma.
- B, central zone: surrounds the ejaculatory ducts and is an uncommon site of carcinoma.
- D, periurethral zone: a small region near the urethra, not the usual site of adenocarcinoma.

Final Answer: Peripheral zone $\Rightarrow$ C

Answer: (C) Go Back to Q15

Answer Key

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