INI CET Pathology

Sample Paper – 9

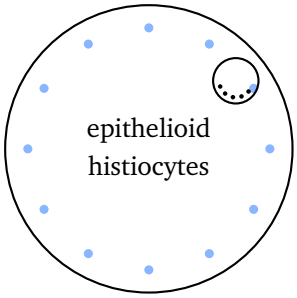
Duration: 14 Minutes **Maximum Marks: 15**

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 schematic shows a granuloma, a focus of activated epithelioid histiocytes with a multinucleate giant cell. Which of the following is a recognised cause of granulomatous inflammation?



A granuloma with a rim of histiocytes and a giant cell

(A) Iron deficiency

(B) Lobar (bacterial) pneumonia

(C) Sarcoidosis

(D) Acute suppurative appendicitis

Q2. The eosinophilic cytoplasmic inclusions seen in ballooned hepatocytes in alcoholic hepatitis, formed from damaged keratin intermediate filaments, are called:

(A) Councilman bodies

(B) Russell bodies

(C) Ferruginous bodies

(D) Mallory (Mallory-Denk) bodies

Q3. During cutaneous wound healing, the collagen of early granulation tissue is predominantly type III. As the wound matures into a scar, this is progressively replaced by which collagen type?

(A) Type II

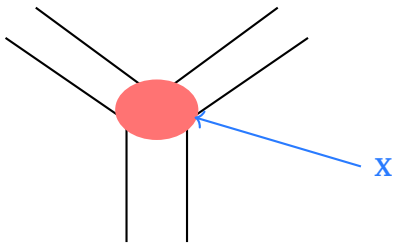
(B) Type IV

(C) Type III (unchanged)

(D) Type I

Hemodynamic Disorders

Q4. The diagram shows a large thromboembolus (marked **X**) lodged astride the bifurcation of the main pulmonary artery. This is termed a:



main pulmonary artery bifurcation

(A) Paradoxical embolus

(B) Saddle embolus

(C) Fat embolus

(D) Septic embolus

Q5. The clinical course of shock classically passes through three sequential stages. In correct order these are:

(A) Irreversible, then progressive, then compensated

(B) Progressive, then compensated, then irreversible

(C) Compensated, then irreversible, then progressive

(D) Non-progressive (compensated), then progressive, then irreversible

Neoplasia

Q6. A normal proto-oncogene is converted into a cancer-driving oncogene by which type of change?

(A) A gain-of-function mutation

(B) A loss-of-function mutation of both alleles

(C) A silent (synonymous) mutation

(D) Complete deletion of the gene

Q7. Which mechanism most directly allows tumour cells to escape replicative senescence and divide without limit ("replicative immortality")?

(A) Loss of contact inhibition

(B) Increased tumour angiogenesis

(C) Reactivation of telomerase

(D) Evasion of the immune response

Q8. Which serum marker is characteristically elevated in gestational trophoblastic disease (hydatidiform mole, choriocarcinoma) and in certain germ-cell tumours?

(A) Beta-human chorionic gonadotropin (beta-hCG)

(B) CA 19-9

(C) Chromogranin A

(D) Calcitonin

Immunopathology

Q9. A middle-aged woman presents with dry eyes and a dry mouth (keratoconjunctivitis sicca and xerostomia). Which autoantibodies are most characteristic of her condition?

(A) Anti-Ro (SSA) and anti-La (SSB)

(B) Anti-Scl-70

(C) Anti-mitochondrial antibody

(D) Anti-glomerular basement membrane antibody

Q10. A male infant develops recurrent bacterial infections after six months of age. Investigations show absent mature B cells and very low serum immunoglobulins, due to a mutation in Bruton tyrosine kinase (BTK). The diagnosis is:

(A) DiGeorge syndrome

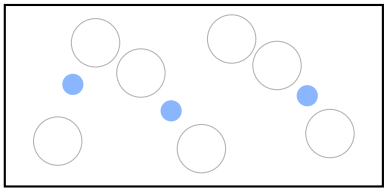
(B) Chronic granulomatous disease

(C) Bruton X-linked agammaglobulinaemia

(D) Chediak-Higashi syndrome

Hematology

Q11. The bone-marrow schematic shown is markedly hypocellular, mostly fat spaces with only a few scattered haematopoietic cells. Combined with peripheral pancytopenia (low red cells, white cells and platelets), this picture is typical of:



Hypocellular marrow: mostly fat spaces, few cells

(A) Chronic myeloid leukaemia

(B) Polycythaemia vera

(C) Aplastic anaemia

(D) Reactive marrow hyperplasia

Q12. A patient has fever, fluctuating neurological signs, renal impairment, thrombocytopenia and a microangiopathic haemolytic anaemia with schistocytes on the smear, all traced to a deficiency of the ADAMTS13 enzyme. The diagnosis is:

(A) Disseminated intravascular coagulation

(B) Thrombotic thrombocytopenic purpura

(C) Immune thrombocytopenic purpura

(D) Haemophilia A

Systemic Pathology

Q13. Chronic rheumatic heart disease, a late sequel of rheumatic fever, most commonly deforms which valve and produces which lesion?

(A) Aortic valve, causing aortic regurgitation

(B) Mitral valve, causing mitral stenosis

(C) Tricuspid valve, causing tricuspid stenosis

(D) Pulmonary valve, causing pulmonary stenosis

Q14. An occupational lung disease showing ferruginous (asbestos) bodies within the lung tissue and carrying a markedly increased risk of malignant mesothelioma is:

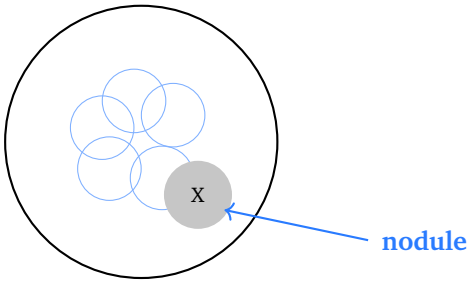
(A) Asbestosis

(B) Silicosis

(C) Coal worker's pneumoconiosis

(D) Berylliosis

Q15. The glomerulus schematic shows a rounded, acellular nodule (marked **X**) expanding the mesangium. Such nodular (Kimmelstiel-Wilson) glomerulosclerosis is the hallmark renal lesion of:



Glomerulus with a rounded mesangial nodule (X)

(A) Minimal change disease

(B) Diabetic nephropathy

(C) Post-streptococcal glomerulonephritis

(D) Thin basement membrane disease

Detailed Solutions

Q1.

Solution

Concept – causes of granulomatous inflammation: A granuloma is a focus of epithelioid histiocytes, often with giant cells, formed against poorly degradable agents.

Step 1 – list the classic causes: Tuberculosis, leprosy, fungal infection, sarcoidosis and foreign-body reactions all produce granulomas.

Step 2 – pick from the options: Sarcoidosis is a recognised non-caseating granulomatous disease.

Why the other options are wrong:

- A, iron deficiency: an anaemia, not an inflammatory granuloma.
- B, lobar pneumonia: an acute neutrophilic exudate, not granulomatous.
- D, acute appendicitis: acute suppurative (neutrophilic) inflammation.

Final Answer: Sarcoidosis $\Rightarrow$ C

Answer: (C) Go Back to Q1

Q2.

Solution

Concept – hepatocyte inclusions in alcohol: Alcohol damages the cytoskeleton of hepatocytes.

Step 1 – name the inclusion: Mallory (Mallory-Denk) bodies are clumped, damaged keratin intermediate filaments.

Step 2 – appearance: They are eosinophilic, irregular cytoplasmic aggregates in ballooned hepatocytes in alcoholic hepatitis.

Why the other options are wrong:

- A, Councilman bodies: apoptotic hepatocytes in viral hepatitis.
- B, Russell bodies: immunoglobulin-stuffed plasma cells.
- C, ferruginous bodies: iron-coated fibres such as asbestos bodies.

Final Answer: Mallory (Mallory-Denk) bodies $\Rightarrow$ D

Answer: (D) Go Back to Q2

Q3.

Solution

Concept – collagen remodelling in scars: Wound collagen changes type as it matures.

Step 1 – early granulation tissue: Early granulation tissue lays down mainly **type III** collagen, which is thin and weak.

Step 2 – maturation: Over weeks this is degraded and replaced by **type I** collagen, which gives the mature scar its tensile strength.

Why the other options are wrong:

- A, type II: found in cartilage, not skin scars.
- B, type IV: found in basement membranes.
- C, type III unchanged: it is replaced, not retained.

Final Answer: Type I $\Rightarrow$ D

Answer: (D) Go Back to Q3

Q4.

Solution

Concept – pulmonary thromboembolism: The site where a large embolus lodges defines the term.

Step 1 – identify X: An embolus straddling the bifurcation of the main pulmonary artery is a **saddle embolus**.

Step 2 – significance: It can obstruct both main branches and cause sudden death.

Why the other options are wrong:

- A, paradoxical embolus: crosses a septal defect into the systemic circulation.
- C, fat embolus: fat globules after long-bone fracture.
- D, septic embolus: infected fragment, e.g. from endocarditis.

Final Answer: Saddle embolus $\Rightarrow$ B

Answer: (B) Go Back to Q4

Q5.

Solution

Concept – stages of shock: Shock evolves through a predictable sequence.

Step 1 – order the stages: First **non-progressive (compensated)**, then **progressive**, then **irreversible**.

Step 2 – what each means: Compensation maintains perfusion; the progressive stage brings tissue hypoxia and acidosis; the irreversible stage brings cell and organ death even if haemodynamics are restored.

Why the other options are wrong:

- A, B, C: all place the stages out of the true physiological order.

Final Answer: Non-progressive, progressive, irreversible $\Rightarrow$ D

Answer: (D) Go Back to Q5

Q6.

Solution

Concept – proto-oncogene to oncogene: Oncogenes are hyperactive versions of normal genes.

Step 1 – name the change: A **gain-of-function mutation** converts a proto-oncogene into an oncogene.

Step 2 – how it acts: The oncogene is dominant, so one activated allele is enough to drive proliferation.

Why the other options are wrong:

- B, loss-of-function of both alleles: the mechanism for tumour suppressor genes.
- C, silent mutation: no change in protein, no effect.
- D, complete deletion: removes function, opposite of activation.

Final Answer: A gain-of-function mutation $\Rightarrow$ A

Answer: (A) Go Back to Q6

Q7.

Solution

Concept – replicative immortality: Normal cells stop dividing as telomeres shorten.

Step 1 – name the mechanism: Tumour cells **reactivate telomerase**, which rebuilds telomeres and prevents senescence.

Step 2 – result: The cells escape the normal division limit and become immortal.

Why the other options are wrong:

- A, loss of contact inhibition: allows piling up, not endless division.
- B, angiogenesis: supplies blood, a separate hallmark.
- D, immune evasion: avoids destruction, not senescence.

Final Answer: Reactivation of telomerase $\Rightarrow$ C

Answer: (C) Go Back to Q7

Q8.

Solution

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

Step 1 – match the disease: Beta-hCG is produced by trophoblast and is raised in gestational trophoblastic disease and germ-cell tumours.

Step 2 – use: Serial levels track a molar pregnancy or choriocarcinoma response.

Why the other options are wrong:

- B, CA 19-9: pancreatic and biliary carcinoma.
- C, chromogranin A: neuroendocrine tumours.
- D, calcitonin: medullary thyroid carcinoma.

Final Answer: Beta-hCG $\Rightarrow$ A

Answer: (A) Go Back to Q8

Q9.

Solution

Concept – Sjogren syndrome: Dry eyes and dry mouth point to lacrimal and salivary gland destruction.

Step 1 – match the antibodies: Anti-Ro (SSA) and anti-La (SSB) are characteristic of Sjogren syndrome.

Step 2 – mechanism: Lymphocytic infiltration destroys the exocrine glands, giving sicca symptoms.

Why the other options are wrong:

- B, anti-Scl-70: systemic sclerosis.
- C, anti-mitochondrial antibody: primary biliary cholangitis.
- D, anti-GBM antibody: Goodpasture syndrome.

Final Answer: Anti-Ro (SSA) and anti-La (SSB) $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – primary B-cell immunodeficiency: Absent B cells with low immunoglobulins is a distinctive pattern.

Step 1 – name the disease: A BTK mutation blocking B-cell maturation causes **Bruton X-linked agammaglobulinaemia**.

Step 2 – clinical clue: Infections begin after about six months as maternal antibody wanes; there are no mature B cells and no germinal centres.

Why the other options are wrong:

- A, DiGeorge syndrome: a T-cell defect from thymic aplasia.
- B, chronic granulomatous disease: a phagocyte oxidative-burst defect.
- D, Chediak-Higashi syndrome: defective phagocyte granules.

Final Answer: Bruton X-linked agammaglobulinaemia $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q10

Q11.

Solution

Concept – pancytopenia with an empty marrow: Marrow cellularity separates the causes of low blood counts.

Step 1 – read the picture: A **hypocellular** marrow (mostly fat) with pancytopenia defines **aplastic anaemia**.

Step 2 – mechanism: Failure or destruction of haematopoietic stem cells reduces all cell lines.

Why the other options are wrong:

- A, CML: a hypercellular marrow with high white counts.
- B, polycythaemia vera: raised red-cell mass, hypercellular marrow.
- D, reactive hyperplasia: a hypercellular, not empty, marrow.

Final Answer: Aplastic anaemia ⇒ C

Answer: (C) Go Back to Q11

Q12.

Solution

Concept – thrombotic microangiopathy: An enzyme deficiency drives platelet-rich microthrombi.

Step 1 – name the disease: Deficiency of **ADAMTS13** causes **thrombotic thrombocytopenic purpura**.

Step 2 – how it happens: Large von Willebrand multimers are not cleaved, so platelet microthrombi form, consuming platelets and shearing red cells into schistocytes.

Why the other options are wrong:

- A, DIC: consumes clotting factors, so PT and aPTT are prolonged and D-dimer high (normal in TTP).
- C, ITP: isolated low platelets from antibody, no schistocytes.
- D, haemophilia A: factor VIII deficiency causing bleeding, not microangiopathy.

Final Answer: Thrombotic thrombocytopenic purpura ⇒ B

Answer: (B) Go Back to Q12

Q13.

Solution

Concept – chronic rheumatic heart disease: Rheumatic fever scars heart valves years later.

Step 1 – name the lesion: The **mitral valve** is most often affected, producing **mitral stenosis**.

Step 2 – morphology: Fibrous thickening, commissural fusion and a fish-mouth or buttonhole orifice with thickened, fused chordae.

Why the other options are wrong:

- A, aortic valve: often involved too, but mitral stenosis is the classic single lesion.
- C and D, tricuspid and pulmonary: rarely the dominant rheumatic lesions.

Final Answer: Mitral valve, mitral stenosis $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – pneumoconioses: Inhaled mineral dusts give distinct lung diseases.

Step 1 – match the clues: Ferruginous (asbestos) bodies and a raised risk of malignant mesothelioma point to **asbestosis**.

Step 2 – morphology: Diffuse interstitial fibrosis, pleural plaques and asbestos bodies (iron-coated fibres) are seen; both mesothelioma and bronchogenic carcinoma risk rise.

Why the other options are wrong:

- B, silicosis: silicotic nodules with whorled collagen, not asbestos bodies.
- C, coal worker's pneumoconiosis: coal macules and anthracotic pigment.
- D, berylliosis: non-caseating granulomas resembling sarcoidosis.

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

Answer: (A) Go Back to Q14

Q15.

Solution

Concept – nodular glomerulosclerosis: The pattern of mesangial expansion names the disease.

Step 1 – identify X: Rounded, acellular **Kimmelstiel-Wilson nodules** expanding the mesangium are the hallmark of **diabetic nephropathy**.

Step 2 – background: Chronic hyperglycaemia thickens the basement membrane and drives mesangial matrix accumulation.

Why the other options are wrong:

- A, minimal change disease: normal light microscopy, foot-process effacement only.
- C, post-streptococcal GN: hypercellular glomeruli with subepithelial humps.
- D, thin basement membrane disease: uniformly thin basement membrane, no nodules.

Final Answer: Diabetic nephropathy $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q15

Answer Key

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