INI CET Pathology

Sample Paper – 6

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. Gangrenous necrosis of a limb may be dry or wet. Compared with dry gangrene, **wet** gangrene is distinguished by:

(A) Superimposed bacterial infection with liquefaction and a poor line of demarcation

(B) Complete absence of any bacterial infection

(C) Slow spread with a sharp, well-demarcated margin

(D) A consistently better prognosis than dry gangrene

Q2. The brown, finely granular "wear-and-tear" pigment that accumulates in ageing cells of the heart and liver, representing indigestible residue of lipid peroxidation, is:

(A) Haemosiderin

(B) Melanin

(C) Lipofuscin

(D) Bilirubin

Q3. An inflammatory **exudate** differs from a **transudate** chiefly in that an exudate has:

(A) Low protein content and very few cells

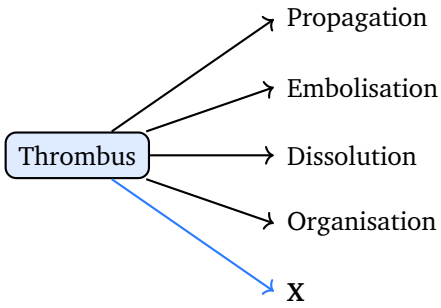
(B) High protein content and many inflammatory cells

(C) A specific gravity below 1.012

(D) No fibrinogen and a low cell count

Hemodynamic Disorders

Q4. The diagram shows the possible fates of a thrombus. Four are propagation, embolisation, dissolution and organisation. The fate marked **X**, in which new vascular channels re-establish blood flow through an organised thrombus, is:



(A) Recanalisation

(B) Calcification

(C) Infarction

(D) Haemorrhage

Q5. Decompression sickness (“the bends”), seen in deep-sea divers who ascend too rapidly, results from which type of embolism?

(A) Fat embolism

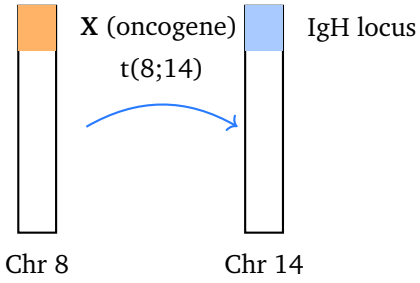
(B) Gas (nitrogen) embolism

(C) Amniotic fluid embolism

(D) Tumour embolism

Neoplasia

Q6. The diagram shows the t(8;14) translocation of Burkitt lymphoma, which places an oncogene from chromosome 8 next to the immunoglobulin heavy-chain (IgH) locus on chromosome 14, driving its over-expression. The oncogene marked **X** is:



(A) BCL2

(B) ABL1

(C) RB1

(D) MYC (c-MYC)

Q7. A solid tumour cannot grow beyond about 1–2 mm without recruiting its own blood supply. Which growth factor most directly drives this tumour angiogenesis?

(A) Epidermal growth factor (EGF)

(B) Platelet-derived growth factor (PDGF)

(C) Transforming growth factor-beta (TGF-β)

(D) Vascular endothelial growth factor (VEGF)

Q8. Bilateral ovarian metastases composed of mucin-filled signet-ring cells, classically arising from a gastric carcinoma, are known as a:

(A) Brenner tumour

(B) Krukenberg tumour

(C) Meigs syndrome

(D) Sertoli–Leydig cell tumour

Immunopathology

Q9. Type II hypersensitivity antibodies directed against the glomerular and alveolar basement membranes, causing combined lung haemorrhage and rapidly progressive glomerulonephritis, characterise:

(A) Granulomatosis with polyangiitis (Wegener)

(B) Systemic lupus erythematosus

(C) Goodpasture syndrome

(D) Henoch–Schonlein purpura

Q10. Failure of development of the third and fourth pharyngeal pouches, producing thymic aplasia with a profound T-cell deficiency and hypocalcaemia, is characteristic of:

(A) DiGeorge syndrome

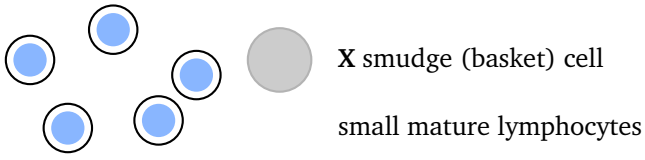
(B) Bruton (X-linked) agammaglobulinaemia

(C) Chronic granulomatous disease

(D) Wiskott–Aldrich syndrome

Hematology

Q11. The peripheral smear from an elderly patient, shown below, is packed with small mature lymphocytes and fragile ruptured cells (marked **X**, the "smudge" or basket cells). The most likely diagnosis is:



(A) Hairy cell leukaemia

(B) Acute myeloid leukaemia

(C) Infectious mononucleosis

(D) Chronic lymphocytic leukaemia

Q12. The commonest inherited bleeding disorder, caused by defective platelet adhesion and marked by a prolonged bleeding time with a normal platelet count, is:

(A) Von Willebrand disease

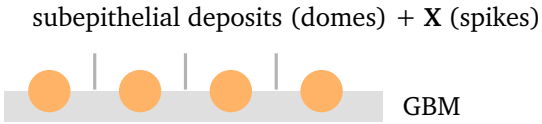
(B) Haemophilia A

(C) Immune thrombocytopenic purpura

(D) Glanzmann thrombasthenia

Systemic Pathology

Q13. The electron-microscopy schematic shows subepithelial immune-complex deposits (domes) with basement-membrane projections (**X**, spikes) growing up between them, the "spike-and-dome" pattern. In an adult presenting with nephrotic syndrome this indicates:



(A) Minimal change disease

(B) Membranous glomerulonephritis

(C) IgA nephropathy

(D) Post-streptococcal glomerulonephritis

Q14. Replacement of the normal squamous epithelium of the lower oesophagus by columnar epithelium with goblet cells, driven by chronic acid reflux and carrying a risk of adenocarcinoma, is:

(A) Squamous metaplasia

(B) Achalasia cardia

(C) Barrett oesophagus

(D) Plummer–Vinson syndrome

Q15. A brain at autopsy shows cortical atrophy with extracellular neuritic (amyloid) plaques and intracellular neurofibrillary tangles of hyperphosphorylated tau. The diagnosis is:

(A) Parkinson disease

(B) Huntington disease

(C) Alzheimer disease

(D) Pick disease (frontotemporal dementia)

Detailed Solutions

Q1.

Solution

Concept – dry versus wet gangrene: Gangrene is coagulative necrosis of a limb, later modified by infection.

Step 1 – define wet gangrene: In wet gangrene, **superimposed bacterial infection** adds liquefactive change.

Step 2 – consequences: Spread is rapid, the margin is poorly demarcated and the prognosis is worse than dry gangrene.

Why the other options are wrong:

- B, no infection: describes dry gangrene, not wet.
- C, sharp demarcation: a feature of dry gangrene.
- D, better prognosis: wet gangrene is more dangerous, with a risk of septicaemia.

Final Answer: Superimposed bacterial infection with liquefaction $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – endogenous pigments: Each pigment has a characteristic colour and origin.

Step 1 – name the pigment: Lipofuscin is the brown "wear-and-tear" pigment of ageing cells.

Step 2 – origin: It is the indigestible residue of lipid peroxidation of membranes, collecting in heart, liver and neurons.

Why the other options are wrong:

- A, haemosiderin: a golden-brown iron-storage pigment from red-cell breakdown.
- B, melanin: a brown-black pigment made by melanocytes.
- D, bilirubin: a bile pigment causing jaundice.

Final Answer: Lipofuscin $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – exudate versus transudate: Protein and cell content separate the two fluids.

Step 1 – define an exudate: An **exudate** is protein-rich (specific gravity above 1.020) with many inflammatory cells.

Step 2 – mechanism: Increased vascular permeability in inflammation lets protein and leukocytes escape.

Why the other options are wrong:

- A, low protein and few cells: describes a transudate.
- C, specific gravity below 1.012: a transudate value.
- D, no fibrinogen: an exudate is rich in fibrinogen, hence fibrin formation.

Final Answer: High protein content and many inflammatory cells $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – fates of a thrombus: A thrombus may propagate, embolise, dissolve, organise or recanalise.

Step 1 – identify X: New channels re-establishing flow through an organised thrombus define **recanalisation**.

Step 2 – mechanism: Ingrowing capillaries within the organised clot fuse into channels that partly restore the lumen.

Why the other options are wrong:

- B, calcification: a late change, not the re-establishment of flow.
- C, infarction: tissue death downstream, a consequence, not a fate of the thrombus itself.
- D, haemorrhage: not a recognised fate of a thrombus.

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

Answer: (A) Go Back to Q4

Q5.

Solution

Concept – gas embolism: Dissolved gas can form intravascular bubbles.

Step 1 – name the cause: Decompression sickness is a **gas (nitrogen) embolism**.

Step 2 – mechanism: Rapid ascent lets dissolved nitrogen come out of solution as bubbles in blood and tissues, causing joint pain (the bends).

Why the other options are wrong:

- A, fat embolism: follows long-bone fractures.
- C, amniotic fluid embolism: a childbirth complication.
- D, tumour embolism: dislodged tumour cells, unrelated to diving.

Final Answer: Gas (nitrogen) embolism $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – Burkitt lymphoma translocation: A translocation drives an oncogene by a strong promoter.

Step 1 – identify X: The t(8;14) moves **MYC (c-MYC)** from chromosome 8 next to the IgH locus on chromosome 14.

Step 2 – effect: The IgH enhancer drives constant MYC over-expression, forcing rapid proliferation (a "starry-sky" histology).

Why the other options are wrong:

- A, BCL2: t(14;18) of follicular lymphoma (anti-apoptosis).
- B, ABL1: t(9;22) of chronic myeloid leukaemia.
- C, RB1: a tumour suppressor, not the driver here.

Final Answer: MYC (c-MYC) $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – tumour angiogenesis: Growth beyond 1–2 mm needs new vessels.

Step 1 – name the factor: VEGF is the main driver of tumour angiogenesis.

Step 2 – clinical link: Anti-VEGF drugs such as bevacizumab exploit this to starve tumours of blood supply.

Why the other options are wrong:

- A, EGF: drives epithelial proliferation, not chiefly angiogenesis.
- B, PDGF: recruits fibroblasts and smooth muscle.
- C, TGF-β: mainly inhibits growth and drives fibrosis.

Final Answer: Vascular endothelial growth factor (VEGF) $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q7

Q8.

Solution

Concept – metastasis to the ovary: A named tumour describes bilateral ovarian secondaries.

Step 1 – name it: Krukenberg tumour is bilateral ovarian metastasis of signet-ring adenocarcinoma, usually gastric.

Step 2 – spread: It reaches the ovaries by transcoelomic and lymphatic routes.

Why the other options are wrong:

- A, Brenner tumour: a primary benign ovarian tumour of transitional epithelium.
- C, Meigs syndrome: ovarian fibroma with ascites and pleural effusion.
- D, Sertoli–Leydig tumour: a primary androgen-secreting sex-cord tumour.

Final Answer: Krukenberg tumour $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q8

Q9.

Solution

Concept – anti-basement-membrane disease: Antibodies against a shared antigen strike two organs.

Step 1 – name the disease: Goodpasture syndrome is a Type II reaction with anti-glomerular-basement-membrane antibodies.

Step 2 – targets: The antibody binds the alpha-3 chain of type IV collagen in lung and kidney, so haemoptysis and glomerulonephritis occur together (linear IgG on immunofluorescence).

Why the other options are wrong:

- A, Wegener (GPA): ANCA-associated granulomatous vasculitis, not anti-GBM.
- B, SLE: immune-complex (Type III) multisystem disease.
- D, Henoch–Schonlein purpura: an IgA small-vessel vasculitis.

Final Answer: Goodpasture syndrome ⇒ C

Answer: (C) Go Back to Q9

Q10.

Solution

Concept – primary T-cell immunodeficiency: A developmental defect explains the pattern.

Step 1 – name it: DiGeorge syndrome follows failed development of the third and fourth pharyngeal pouches.

Step 2 – consequences: Thymic aplasia gives a T-cell deficiency, and absent parathyroids give hypocalcaemia with tetany.

Why the other options are wrong:

- B, Bruton agammaglobulinaemia: a B-cell defect with absent immunoglobulins.
- C, chronic granulomatous disease: a neutrophil (NADPH oxidase) defect.
- D, Wiskott–Aldrich: eczema, thrombocytopenia and recurrent infection.

Final Answer: DiGeorge syndrome ⇒ A

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – smudge cells on a smear: Fragile lymphocytes rupture during smear preparation.

Step 1 – read the smear: Numerous small mature lymphocytes with **smudge (basket) cells** in an elderly patient indicate **chronic lymphocytic leukaemia**.

Step 2 – confirm: The cells are clonal B lymphocytes (CD5 and CD23 positive), often with lymphocytosis and lymphadenopathy.

Why the other options are wrong:

- A, hairy cell leukaemia: cells with hairy cytoplasmic projections, not smudge cells.
- B, AML: myeloblasts with Auer rods, not mature lymphocytes.
- C, infectious mononucleosis: reactive (atypical) lymphocytes in a young patient, self-limited.

Final Answer: Chronic lymphocytic leukaemia $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – inherited platelet-function disorder: Defective adhesion prolongs the bleeding time.

Step 1 – name it: Von Willebrand disease is the commonest inherited bleeding disorder.

Step 2 – mechanism: Deficient von Willebrand factor impairs platelet adhesion to collagen, giving a prolonged bleeding time with a normal platelet count (and often a mildly raised APTT).

Why the other options are wrong:

- B, haemophilia A: factor VIII deficiency with a raised APTT but a normal bleeding time.
- C, ITP: bleeding from a low platelet count, not defective adhesion.
- D, Glanzmann thrombasthenia: a rare defect of platelet aggregation (GpIIb/IIIa).

Final Answer: Von Willebrand disease $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q12

Q13.

Solution

Concept – spike-and-dome pattern: The deposit location names the glomerular disease.

Step 1 – identify the pattern: Subepithelial deposits with basement-membrane spikes between them (spike-and-dome) define **membranous glomerulonephritis**.

Step 2 – clinical link: It is the commonest cause of nephrotic syndrome in adults; anti-PLA2R antibodies are found in primary cases.

Why the other options are wrong:

- A, minimal change: normal light microscopy with only foot-process effacement.
- C, IgA nephropathy: mesangial IgA deposits, usually with haematuria.
- D, post-streptococcal GN: subepithelial "humps" with an acute nephritic picture, not spike-and-dome.

Final Answer: Membranous glomerulonephritis $\Rightarrow$ B

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – metaplasia in the oesophagus: Chronic reflux switches the epithelial type.

Step 1 – name it: Barrett oesophagus is columnar (intestinal) metaplasia of the lower oesophageal squamous epithelium.

Step 2 – significance: Driven by chronic acid reflux, it carries a raised risk of oesophageal adenocarcinoma and needs surveillance.

Why the other options are wrong:

- A, squamous metaplasia: the reverse change, seen in other sites (e.g. bronchus).
- B, achalasia: a motility disorder from loss of myenteric neurons.
- D, Plummer–Vinson syndrome: oesophageal webs with iron-deficiency

anaemia.

Final Answer: Barrett oesophagus $\Rightarrow$ C

Answer: (C) Go Back to Q14

Q15.

Solution

Concept – neurodegenerative histology: Two hallmark lesions define the disease.

Step 1 – name it: Neuritic amyloid plaques with neurofibrillary tangles of hyperphosphorylated tau define **Alzheimer disease**.

Step 2 – correlate: Cortical atrophy (especially temporal and parietal) matches the progressive dementia.

Why the other options are wrong:

- A, Parkinson disease: Lewy bodies (alpha-synuclein) in the substantia nigra.
- B, Huntington disease: caudate atrophy from a CAG repeat expansion.
- D, Pick disease: tau-positive Pick bodies with frontotemporal atrophy, a different clinical picture.

Final Answer: Alzheimer disease $\Rightarrow$ C

Answer: (C) Go Back to Q15

Answer Key

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