INI CET Medicine

Sample Paper – 9

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

Instructions

- This paper contains **15** Multiple Choice Questions (Single Best Response), modelled on the General Medicine content of **INI CET** (Institutes of National Importance Combined Entrance Test) conducted by AIIMS New Delhi.
- The **15-question** length matches the number of Medicine 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.

Cardiology

Q1. A young man has sharp retrosternal chest pain that eases on sitting forward and worsens on lying flat, with a scratchy triphasic rub on auscultation. Which ECG finding is most characteristic of this acute pericarditis?

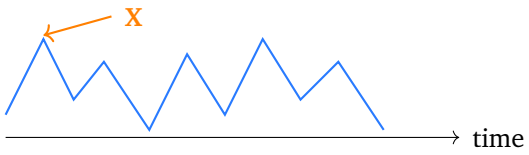
(A) Widespread concave (saddle-shaped) ST-segment elevation with PR-segment depression

(B) ST-segment elevation confined to the anterior leads with reciprocal depression

(C) Tall tented T waves with a widened QRS complex

(D) Deep symmetrical T-wave inversion in the lateral leads only

Q2. The jugular venous pressure (JVP) trace shown, with a preserved systolic descent (marked **X**) and a lost diastolic descent, is from a patient with cardiac tamponade. Besides hypotension and muffled heart sounds, the third component of Beck's triad is:



(A) Raised jugular venous pressure (distended neck veins)

(B) Sinus bradycardia

(C) A loud pansystolic murmur

(D) Bounding peripheral pulses

Q3. A patient with previous tuberculous pericarditis now has a raised JVP, ascites, pedal oedema and a pericardial knock; CT shows a thickened, calcified pericardium and Kussmaul's sign is present. The diagnosis is:

(A) Acute fibrinous pericarditis

(B) Cardiac tamponade

(C) Constrictive pericarditis

(D) Dilated cardiomyopathy

Respiratory Medicine

Q4. Which paraneoplastic syndrome is most characteristically associated with small-cell lung carcinoma?

(A) Hypercalcaemia from parathyroid hormone-related peptide (PTHrP)

(B) Hypertrophic pulmonary osteoarthropathy with finger clubbing

(C) Polycythaemia from ectopic erythropoietin

(D) Syndrome of inappropriate ADH secretion (SIADH) with hyponatraemia

Q5. On CT chest a solitary pulmonary nodule is found. Which single feature most strongly favours a benign lesion?

(A) Spiculated (irregular) margins

(B) Diameter greater than 3 cm

(C) Documented growth over the last three months

(D) Central or popcorn pattern of calcification, stable over two years

Gastroenterology and Hepatology

Q6. Which condition produces a predominantly unconjugated (indirect) hyperbilirubinaemia?

(A) Haemolytic anaemia

(B) Common bile duct stone (choledocholithiasis)

(C) Carcinoma of the head of the pancreas

(D) Primary biliary cholangitis

Q7. A patient has pale (clay-coloured) stools, dark urine, pruritus, a markedly raised conjugated bilirubin and a disproportionately high alkaline phosphatase. This biochemical pattern indicates which type of jaundice?

(A) Prehepatic (haemolytic) jaundice

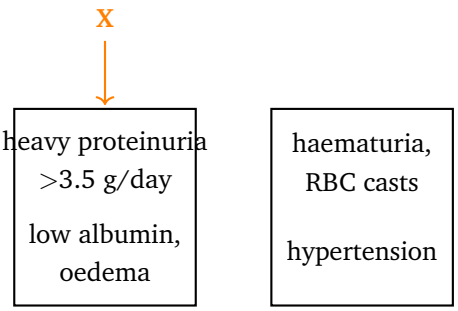
(B) Posthepatic (obstructive) jaundice

(C) Hepatocellular jaundice from acute viral hepatitis

(D) Gilbert syndrome

Endocrinology and Nephrology

Q8. The comparison chart contrasts two glomerular syndromes. The column marked **X** shows heavy proteinuria (greater than 3.5 g/day), a low serum albumin and oedema. This picture defines:



(A) Nephritic syndrome

(B) Acute tubular necrosis

(C) Nephrotic syndrome

(D) Prerenal acute kidney injury

Q9. Which combination of findings best characterises the nephritic syndrome?

(A) Haematuria with red-cell casts, hypertension and mild-to-moderate proteinuria

(B) Massive proteinuria with hypoalbuminaemia and hyperlipidaemia

(C) Glycosuria with a normal blood glucose

(D) Isolated sterile pyuria

Q10. A 4-year-old boy develops periorbital oedema and frothy urine with nephrotic-range proteinuria. Light microscopy of the renal biopsy is normal, but electron microscopy shows diffuse effacement of podocyte foot processes. The diagnosis is:

(A) Post-streptococcal glomerulonephritis

(B) Minimal change disease

(C) Diabetic nephropathy

(D) IgA nephropathy

Neurology

Q11. Which statement best describes the pattern of muscle weakness in myasthenia gravis?

(A) Fixed distal weakness with early wasting and fasciculations

(B) Weakness that improves steadily with repeated effort

(C) Proximal weakness with a raised creatine kinase and no ocular signs

(D) Fatigable weakness that worsens with sustained or repeated use and improves with rest

Q12. In a patient with fatigable ptosis and diplopia, which finding most specifically confirms myasthenia gravis, alongside a positive ice-pack test and edrophonium response?

(A) Anti-GQ1b ganglioside antibodies

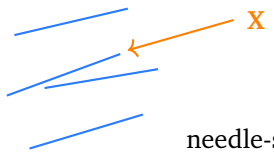
(B) Anti-acetylcholine receptor (anti-AChR) antibodies

(C) Anti-voltage-gated calcium channel antibodies

(D) Anti-glutamic acid decarboxylase (anti-GAD) antibodies

Infectious Diseases, Hematology and Rheumatology

Q13. A joint aspirate viewed under polarised light shows the crystals sketched below (marked **X**): needle-shaped and strongly negatively birefringent. The diagnosis is:



needle-shaped crystals

(A) Pseudogout (calcium pyrophosphate deposition)

(B) Gout (monosodium urate deposition)

(C) Septic arthritis

(D) Rheumatoid arthritis

Q14. In pseudogout, the crystals aspirated from the joint are:

(A) Monosodium urate: needle-shaped, negatively birefringent

(B) Cholesterol: flat rectangular plates with notched corners

(C) Calcium pyrophosphate dihydrate: rhomboid, weakly positively birefringent

(D) Calcium hydroxyapatite: non-birefringent, seen only on electron microscopy

Q15. A 72-year-old woman has a new unilateral temporal headache, jaw claudication on chewing, scalp tenderness and an ESR of 90 mm/hour, with a risk of sudden visual loss. The most likely diagnosis is:

(A) Kawasaki disease

(B) Takayasu arteritis

(C) Giant cell (temporal) arteritis

(D) Buerger disease (thromboangiitis obliterans)

Detailed Solutions

Q1.

Solution

Concept – acute pericarditis: Inflammation of the whole pericardial sac changes the ECG diffusely, not in a single arterial territory.

Step 1 – read the clinical clues: Pleuritic pain relieved by leaning forward and a triphasic (three-component) friction rub point to acute pericarditis.

Step 2 – match the ECG: The classic early change is **widespread concave (saddle-shaped) ST elevation** across most leads, with **PR-segment depression**.

Step 3 – reason it out: Because the inflammation is generalised, the ST changes are not confined to one coronary territory and there is no reciprocal depression.

Why the other options are wrong:

- B, territorial ST elevation with reciprocal change: this is the pattern of acute myocardial infarction.
- C, tall tented T waves with wide QRS: this is hyperkalaemia.
- D, isolated lateral T inversion: a non-specific ischaemic pattern, not diffuse pericarditis.

Final Answer: Widespread concave ST elevation with PR depression $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – cardiac tamponade and Beck's triad: Fluid under pressure in the pericardial sac compresses the heart and impairs filling.

Step 1 – recall Beck's triad: The three signs are **hypotension**, **muffled (soft) heart sounds** and **raised jugular venous pressure**.

Step 2 – pick the missing one: Hypotension and muffled sounds are already given, so the third is a **raised JVP with distended neck veins**.

Step 3 – read the trace: On the JVP the systolic x descent (marked X) is preserved while the diastolic y descent is lost, because filling can only occur in systole.

Why the other options are wrong:

- B, bradycardia: tamponade usually causes a compensatory tachycardia, not

bradycardia.
- C, pansystolic murmur: a valve-lesion sign, not part of tamponade.
- D, bounding pulses: tamponade gives a weak pulse with pulsus paradoxus, not bounding pulses.

Final Answer: Raised jugular venous pressure ⇒ A

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – constrictive pericarditis: A rigid, often calcified pericardium limits ventricular filling in diastole.

Step 1 – gather the signs: Raised JVP, ascites, oedema, a pericardial knock and pericardial calcification after TB all fit constriction.

Step 2 – confirm with Kussmaul's sign: A paradoxical rise in JVP on inspiration (Kussmaul's sign) is characteristic of constrictive pericarditis.

Step 3 – name it: The thickened, calcified, non-compliant pericardium defines **constrictive pericarditis**.

Why the other options are wrong:

- A, acute fibrinous pericarditis: presents with acute pleuritic pain and a rub, not chronic calcification.
- B, cardiac tamponade: an acute effusion with pulsus paradoxus, without a calcified pericardium or a knock.
- D, dilated cardiomyopathy: a dilated, poorly contracting ventricle, not a rigid pericardium.

Final Answer: Constrictive pericarditis ⇒ C

Answer: (C) Go Back to Q3

Q4.

Solution

Concept – paraneoplastic syndromes of lung cancer: Different cell types secrete different ectopic hormones.

Step 1 – match small-cell carcinoma: Small-cell lung carcinoma, of neuroendocrine origin, most characteristically causes **SIADH** with hyponatraemia (and

also ectopic ACTH).

Step 2 – mechanism: Tumour cells secrete antidiuretic hormone, driving water retention and dilutional hyponatraemia.

Why the other options are wrong:

- A, PTHrP hypercalcaemia: this is typical of squamous-cell carcinoma.
- B, hypertrophic osteoarthropathy: usually linked to non-small-cell (adenocarcinoma), not small-cell.
- C, ectopic erythropoietin: a feature of renal and hepatic tumours, not small-cell lung cancer.

Final Answer: SIADH with hyponatraemia $\Rightarrow$ D

Answer: (D) Go Back to Q4

Q5.

Solution

Concept – solitary pulmonary nodule: Certain features predict a benign nodule and reassure against cancer.

Step 1 – name the benign clue: A **central, laminated or popcorn pattern of calcification that is stable over two years** strongly favours a benign lesion (old granuloma or hamartoma).

Step 2 – reason it out: Long-term size stability plus benign calcification make malignant growth very unlikely.

Why the other options are wrong:

- A, spiculated margins: a classic sign of malignancy.
- B, size over 3 cm: larger nodules carry a higher cancer risk.
- C, recent growth: interval enlargement raises concern for malignancy.

Final Answer: Popcorn calcification, stable over two years $\Rightarrow$ D

Answer: (D) Go Back to Q5

Q6.

Solution

Concept – classifying jaundice by bilirubin type: Unconjugated (indirect) bilirubin rises when production outpaces or precedes hepatic conjugation.

Step 1 – match the cause: Haemolytic anaemia increases the breakdown of red cells, flooding the liver with bilirubin and raising the unconjugated fraction.

Step 2 – confirm: The urine shows no bilirubin (unconjugated bilirubin is not water soluble), but urinary urobilinogen is raised.

Why the other options are wrong:

- B, bile duct stone: causes obstructive, conjugated hyperbilirubinaemia.
- C, pancreatic head carcinoma: obstructs the bile duct, raising conjugated bilirubin.
- D, primary biliary cholangitis: a cholestatic disease with conjugated hyperbilirubinaemia.

Final Answer: Haemolytic anaemia $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q6

Q7.

Solution

Concept – obstructive (posthepatic) jaundice: Blockage of bile flow backs conjugated bilirubin into the blood and stops bile reaching the gut.

Step 1 – read the pattern: Pale stools (no stercobilin), dark urine (conjugated bilirubin is water soluble and spills into urine) and a disproportionately raised alkaline phosphatase are the signature of cholestasis.

Step 2 – classify: This is **posthepatic (obstructive) jaundice**, from a blocked biliary tree.

Why the other options are wrong:

- A, prehepatic: gives unconjugated bilirubinaemia with normal-coloured stools and no bilirubinuria.
- C, hepatocellular: transaminases rise more than alkaline phosphatase, with a mixed bilirubin picture.
- D, Gilbert syndrome: a mild unconjugated hyperbilirubinaemia with normal stools and urine.

Final Answer: Posthepatic (obstructive) jaundice $\Rightarrow$ B

Answer: (B) Go Back to Q7

Q8.

Solution

Concept – nephrotic versus nephritic syndrome: Heavy protein loss defines one, an inflamed haematuric glomerulus the other.

Step 1 – read column X: Column X lists heavy proteinuria over 3.5 g/day, low albumin and oedema.

Step 2 – name it: This tetrad (proteinuria, hypoalbuminaemia, oedema and hyperlipidaemia) defines the **nephrotic syndrome**.

Why the other options are wrong:

- A, nephritic syndrome: the second column (haematuria, red-cell casts, hypertension), not column X.
- B, acute tubular necrosis: causes acute kidney injury with muddy-brown casts, not heavy proteinuria.
- D, prerenal AKI: from underperfusion, with a bland urine and no nephrotic-range proteinuria.

Final Answer: Nephrotic syndrome $\Rightarrow$ C

Answer: (C) Go Back to Q8

Q9.

Solution

Concept – the nephritic picture: Glomerular inflammation lets red cells and casts leak while raising blood pressure.

Step 1 – list the features: Nephritic syndrome shows **haematuria with red-cell casts, hypertension and mild-to-moderate proteinuria**, often with some renal impairment.

Step 2 – contrast: Unlike the nephrotic syndrome, protein loss is not massive and the hallmark is an active urinary sediment.

Why the other options are wrong:

- B, massive proteinuria with hypoalbuminaemia: this is the nephrotic, not

nephritic, syndrome.

- C, glycosuria with normal glucose: this is renal glycosuria, a tubular problem.
- D, isolated sterile pyuria: suggests interstitial or partially treated infection, not glomerulonephritis.

Final Answer: Haematuria with red-cell casts and hypertension ⇒ A

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – minimal change disease: The commonest cause of nephrotic syndrome in children looks normal on light microscopy.

Step 1 – read the biopsy: A normal light microscopy picture with diffuse podocyte foot-process effacement on electron microscopy is diagnostic of **minimal change disease**.

Step 2 – clinical fit: A young child with sudden nephrotic-range proteinuria that is highly steroid-responsive supports this.

Why the other options are wrong:

- A, post-streptococcal GN: a nephritic illness with haematuria and low complement, not pure nephrosis.
- C, diabetic nephropathy: shows mesangial expansion and Kimmelstiel-Wilson nodules, and does not fit a healthy child.
- D, IgA nephropathy: presents with visible haematuria after infection and mesangial IgA deposits.

Final Answer: Minimal change disease ⇒ B

Answer: (B) Go Back to Q10

Q11.

Solution

Concept – neuromuscular junction failure: In myasthenia gravis antibodies reduce working acetylcholine receptors, so transmission fades with use.

Step 1 – describe the pattern: The weakness is **fatigable**: it worsens with sustained or repeated activity and through the day, and improves with rest.

Step 2 – typical presentation: Ptosis and diplopia that worsen towards evening are classic.

Why the other options are wrong:

- A, fixed distal weakness with fasciculations: points to motor neurone disease, a lower-motor-neurone process.
- B, improvement with repeated effort: this is Lambert-Eaton myasthenic syndrome, the opposite pattern.
- C, high creatine kinase with no ocular signs: suggests a primary myopathy, not myasthenia.

Final Answer: Fatigable weakness worse with use ⇒ D

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – confirming myasthenia gravis: A specific autoantibody clinches the diagnosis at the neuromuscular junction.

Step 1 – name the antibody: Anti-acetylcholine receptor (anti-AChR) antibodies are present in most patients and are highly specific.

Step 2 – supportive bedside tests: A positive ice-pack test (cooling improves ptosis) and an edrophonium (Tensilon) response both fit myasthenia.

Why the other options are wrong:

- A, anti-GQ1b: associated with the Miller Fisher variant of Guillain-Barre syndrome.
- C, anti-voltage-gated calcium channel: found in Lambert-Eaton syndrome, not myasthenia.
- D, anti-GAD: linked to stiff-person syndrome and type 1 diabetes.

Final Answer: Anti-acetylcholine receptor antibodies ⇒ B

Answer: (B) Go Back to Q12

Q13.

Solution

Concept – crystal arthropathy on polarised microscopy: Crystal shape and birefringence separate gout from pseudogout.

Step 1 – read the aspirate: Needle-shaped, strongly negatively birefringent crystals (marked X) are monosodium urate.

Step 2 – name the disease: Monosodium urate crystals define **gout**, typically affecting the first metatarsophalangeal joint.

Why the other options are wrong:

- A, pseudogout: shows rhomboid, weakly positively birefringent calcium pyrophosphate crystals, not needles.
- C, septic arthritis: aspirate shows organisms and a very high neutrophil count, without crystals.
- D, rheumatoid arthritis: an inflammatory arthritis with no diagnostic crystals in the fluid.

Final Answer: Gout (monosodium urate) $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – pseudogout crystals: Pseudogout is caused by calcium pyrophosphate, distinct from urate in shape and optics.

Step 1 – name the crystal: The crystals are **calcium pyrophosphate dihydrate**: rhomboid (brick-shaped) and weakly positively birefringent.

Step 2 – supporting clue: Chondrocalcinosis (cartilage calcification) is often seen on the joint radiograph.

Why the other options are wrong:

- A, monosodium urate: this is gout, needle-shaped and negatively birefringent.
- B, cholesterol plates: seen in chronic effusions, not the cause of pseudogout.
- D, hydroxyapatite: causes calcific periarthritis and is non-birefringent, not the pseudogout crystal.

Final Answer: Calcium pyrophosphate dihydrate, rhomboid, positively birefrin-

gent $\Rightarrow$ C

Answer: (C) Go Back to Q14

Q15.

Solution

Concept – large-vessel vasculitis in the elderly: Giant cell arteritis inflames the temporal and other cranial arteries and threatens sight.

Step 1 – gather the features: Age over 50, a new temporal headache, jaw claudication, scalp tenderness and a very high ESR all point to **giant cell (temporal) arteritis**.

Step 2 – act on it: The danger is anterior ischaemic optic neuropathy, so high-dose steroids are started at once, before biopsy confirmation.

Why the other options are wrong:

- A, Kawasaki disease: a vasculitis of young children with coronary aneurysms.
- B, Takayasu arteritis: affects the aorta and its branches in young women (pulseless disease), without temporal headache.
- D, Buerger disease: affects distal limb vessels in young male smokers, not the temporal artery.

Final Answer: Giant cell (temporal) arteritis $\Rightarrow$ C

Answer: (C) Go Back to Q15

Answer Key

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