FMGE Microbiology Sample Paper – 3

Duration: 60 Minutes **Maximum Marks: 40**

Instructions

- This paper contains **40 Multiple Choice Questions** modelled on the **Microbiology** section of FMGE (Bacteriology, Immunology, Virology, Parasitology, and Mycology).
- Each correct answer carries **+1 mark**. There is **no negative marking** – attempt all questions.
- Questions follow the clinically integrated pattern of the FMGE examination.
- Click a question number in the answer key to navigate to its solution.

FMGE Microbiology – Q1 to Q40

Q1. A 10-year-old boy presents with migratory polyarthritis and low-grade fever 3 weeks after an episode of sore throat. Echocardiography reveals mitral regurgitation with leaflet thickening. Laboratory investigations show ASO titre of 320 IU/mL. Which organism is most likely responsible for the preceding pharyngeal infection?

(A) Staphylococcus aureus

(B) Streptococcus pneumoniae

(C) Streptococcus pyogenes

(D) Enterococcus faecalis

Q2. A 68-year-old man in the ICU develops fever and dysuria on day 12 of vancomycin therapy. Urine culture grows Gram-positive cocci in pairs and chains. The organism is identified as *Enterococcus faecalis*. Susceptibility testing shows high-level vancomycin resistance (MIC >512 μg/mL). Which gene is most likely responsible for this resistance pattern?

(A) mecA

(B) blaZ

(C) vanB

(D) vanA

Q3. A 54-year-old chronic alcoholic presents with sudden onset high fever, productive cough with thick, dark-red "currant jelly" sputum, and right upper lobe consolidation with early cavitation on chest X-ray. Sputum culture grows mucoid, lactose-fermenting colonies on MacConkey agar with a positive string test. What is the most likely causative organism?

(A) *Klebsiella pneumoniae*

(B) *Pseudomonas aeruginosa*

(C) *Streptococcus pneumoniae*

(D) *Haemophilus influenzae*

Q4. A 45-year-old woman presents with recurrent urinary tract infections and staghorn calculi on CT KUB. Urine pH is 8.2 (alkaline). Urine culture shows a Gram-negative rod that produces concentric swarming rings on blood agar and gives a strongly positive urease test. Which organism is responsible?

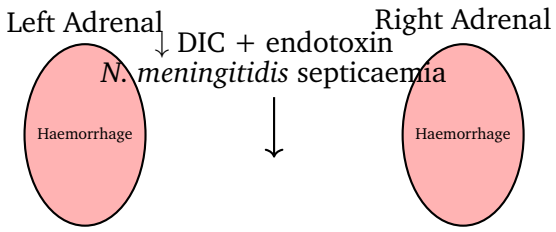
(A) *Escherichia coli*

(B) *Proteus mirabilis*

(C) *Pseudomonas aeruginosa*

(D) *Klebsiella pneumoniae*

Q5. A 19-year-old college student presents with sudden high fever, non-blanching petechial rash, hypotension, and altered sensorium. He deteriorates rapidly and dies within 24 hours. Autopsy reveals bilateral adrenal haemorrhage as shown in the diagram below.



Waterhouse-Friderichsen Syndrome
Bilateral adrenal haemorrhagic infarction

What is the most likely causative organism?

(A) *Streptococcus pneumoniae*

(B) *Haemophilus influenzae*

(C) *Neisseria meningitidis*

(D) *Staphylococcus aureus*

Q6. A 35-year-old man presents with progressive ascending weakness of all four limbs over 10 days. He had an episode of bloody diarrhoea 2 weeks prior that resolved spontaneously. Nerve conduction studies show demyelinating polyneuropathy. CSF reveals albuminocytological dissociation. Which preceding infection is most likely responsible through molecular mimicry with GM1 gangliosides?

(A) *Salmonella typhi*

(B) *Shigella dysenteriae*

(C) *Escherichia coli* O157:H7

(D) *Campylobacter jejuni*

Q7. A 40-year-old farmer sustains a deep laceration of his right thigh in an agricultural accident. Two days later he develops severe pain, tense oedema, bronze discolouration of skin, and crepitus on palpation. X-ray shows gas in soft tissues. Culture on egg-yolk agar shows a zone of opalescence around colonies that is inhibited by specific antitoxin (Nagler reaction). What is the causative organism?

(A) *Clostridium perfringens*

(B) *Clostridium tetani*

(C) *Clostridium difficile*

(D) *Bacteroides fragilis*

Q8. A 38-year-old HIV-positive patient (CD4 count 80 cells/mm^3) presents with headache, right-sided weakness, and multiple ring-enhancing brain lesions on MRI. CT chest shows bilateral lung nodules. Sputum staining shows branching filamentous Gram-positive rods that are partially (weakly) acid-fast on modified Ziehl-Neelsen staining. What is the most likely diagnosis?

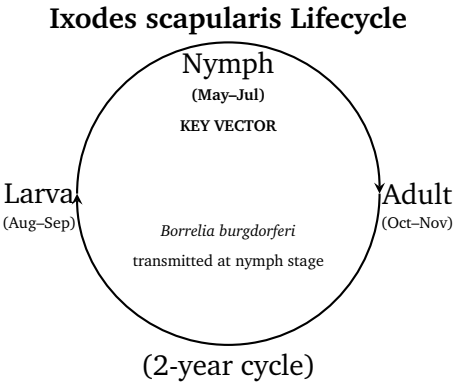
(A) *Actinomyces israelii* infection

(B) *Nocardia asteroides* infection

(C) Cryptococcal meningitis

(D) Cerebral toxoplasmosis

Q9. A 28-year-old hiker returning from a forested area in the northeastern United States develops a 12-cm expanding annular erythematous rash with central clearing on his right thigh. Three weeks later he develops migratory arthritis and second-degree heart block. The lifecycle of the vector is depicted below.



What is the most likely causative organism?

(A) *Rickettsia rickettsii*

(B) *Ehrlichia chaffeensis*

(C) *Borrelia burgdorferi*

(D) *Anaplasma phagocytophilum*

Q10. A 52-year-old rabbit hunter presents with sudden-onset fever (39.5°C), a painful ulcer with a raised border at the site of a tick bite on his forearm, and tender ipsilateral axillary lymphadenopathy (ulceroglandular form). He is in septic shock. The organism is a tiny Gram-negative coccobacillus that requires cysteine for growth and is a Biosafety Level 3 agent. What is the most likely diagnosis?

(A) Bubonic plague (*Yersinia pestis*)

(B) Cat scratch disease (*Bartonella henselae*)

(C) Brucellosis (*Brucella abortus*)

(D) Tularemia (*Francisella tularensis*)

Q11. A 48-year-old veterinarian presents with prolonged fever, severe headache, and atypical pneumonia (non-productive cough, patchy infiltrates on CXR) after assisting with animal deliveries at a sheep farm. He does not recall any tick bite. Blood cultures are negative. Serology reveals high Phase II IgG antibody titres. Which organism is responsible?

(A) *Coxiella burnetii*

(B) *Rickettsia conorii*

(C) *Chlamydia psittaci*

(D) *Legionella pneumophila*

Q12. A 55-year-old rice-paddy farmer from Thailand presents with prolonged fever, pneumonia with upper-lobe cavitation, and hepatosplenomegaly. Blood culture grows a Gram-negative rod showing bipolar “safety-pin” staining on Giemsa. The organism is resistant to trimethoprim-sulfamethoxazole and grows with a metallic sheen on Ashdown’s medium. What is the diagnosis?

(A) Glanders (*Burkholderia mallei*)

(B) Melioidosis (*Burkholderia pseudomallei*)

(C) Plague (*Yersinia pestis*)

(D) Brucellosis (*Brucella melitensis*)

Q13. A 70-year-old male on mechanical ventilation in the ICU for 14 days develops new fever, purulent tracheal secretions, and worsening oxygenation. Bronchoalveolar lavage culture grows a non-fermenting, oxidase-negative Gram-negative coccobacillus. The isolate is resistant to imipenem, meropenem, and all beta-lactams. Molecular testing reveals OXA-23 carbapenemase gene. Which organism is responsible?

(A) *Pseudomonas aeruginosa*

(B) *Stenotrophomonas maltophilia*

(C) *Acinetobacter baumannii*

(D) *Burkholderia cepacia*

Q14. A 35-year-old woman presents with rapidly progressing cellulitis, erythema, and purulent discharge from a cat bite wound on her right hand, occurring within 12 hours of injury. Gram stain shows small Gram-negative coccobacilli. Culture on blood agar grows grey colonies with a "mousy" odour. The isolate is sensitive to penicillin. What is the most likely causative organism?

(A) *Eikenella corrodens*

(B) *Capnocytophaga canimorsus*

(C) *Staphylococcus aureus*

(D) *Pasteurella multocida*

Q15. A 25-year-old woman develops sudden urticaria, angioedema, bronchospasm, and hypotension within 5 minutes of receiving penicillin injection. Her oxygen saturation drops to 82%. Which immunological mechanism and first-line treatment are most appropriate?

(A) IgE-mediated mast cell degranulation; immediate intramuscular epinephrine

(B) IgG-mediated complement activation; intravenous corticosteroids

(C) Immune complex deposition; antihistamines

(D) Cell-mediated cytotoxicity; IV immunoglobulin

Q16. A patient with severe sepsis has marked neutrophilia with extensive neutrophil migration to the site of infection. A complement split product is identified as the most potent chemotactic factor for neutrophils and also causes mast cell degranulation. Which complement component is responsible?

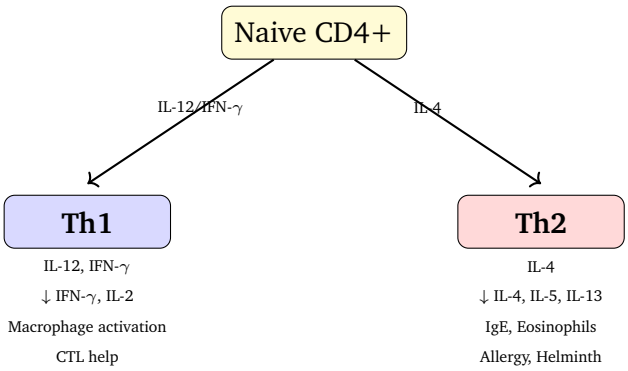
(A) C3b

(B) C5a

(C) C1q

(D) C4b

Q17. A researcher is studying T-helper cell differentiation. The diagram below shows the pathways from naive CD4+ T cells. Which combination of cytokines and effector functions correctly identifies the Th1 pathway and its role in intracellular infections?



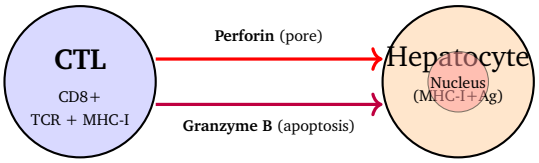
(A) Th1: IL-4, IL-5; promotes IgE production

(B) Th1: IL-10, TGF-β; suppresses inflammation

(C) Th1: IFN-γ, IL-2; activates macrophages and CTLs against intracellular pathogens

(D) Th1: IL-13, IL-17; promotes eosinophil recruitment

Q18. A patient with active viral hepatitis B has hepatocyte destruction mediated by cytotoxic T lymphocytes (CTLs). The immunological synapse between CTL and infected hepatocyte is depicted below.



Which statement BEST describes cytotoxic T lymphocyte (CTL) function?

(A) CTLs are CD4+ cells restricted by MHC class II

(B) CTLs kill via antibody-dependent cellular cytotoxicity

(C) CTLs require complement activation for target cell killing

(D) CTLs are CD8+ cells; perforin creates membrane pores and granzymes induce apoptosis; restricted by MHC class I

Q19. A patient with pulmonary tuberculosis has poorly formed granulomas with central caseation. Immunological studies reveal a deficiency of macrophage activation. Which cytokine, secreted primarily by Th1 cells, is MOST important for macrophage activation and killing of intracellular *Mycobacterium tuberculosis*?

(A) IFN-γ

(B) IL-4

(C) IL-10

(D) IL-13

Q20. Following vaccination with hepatitis B surface antigen, a patient mounts a robust secondary immune response upon re-exposure to the antigen 10 years later, with rapidly rising high-affinity IgG antibody titres. Which cell type is primarily responsible for this secondary (anamnestic) response, and where does affinity maturation occur?

(A) Naive B cells; bone marrow

(B) Memory B cells; germinal centres of secondary lymphoid organs

(C) Plasma cells; spleen red pulp

(D) Regulatory T cells; thymus

Q21. During severe *Staphylococcus aureus* bacteraemia, neutrophils are observed to extrude web-like structures composed of DNA, histones, and granule proteins (elastase, myeloperoxidase) that trap and kill bacteria extracellularly. What is this neutrophil killing mechanism called?

(A) Antibody-dependent cellular cytotoxicity (ADCC)

(B) Respiratory burst (oxidative killing)

(C) Neutrophil extracellular traps (NETs)

(D) Degranulation (exocytosis of azurophilic granules)

Q22. A patient with sepsis has a paradoxical immunosuppressive phase following the initial pro-inflammatory surge. Regulatory T cells and macrophages produce a cytokine that inhibits TNF-α, IL-12, and IFN-γ production, dampening the inflammatory response. Elevated levels of this cytokine correlate with increased susceptibility to secondary infections. Which cytokine is described?

(A) IL-1β

(B) IL-6

(C) TNF-α

(D) IL-10

Q23. A 28-year-old male presents with chronic lower back pain and stiffness, worse in the morning and improving with exercise, for 18 months. X-ray shows sacroiliitis and bamboo spine. RF is negative. HLA typing would most likely reveal which antigen, and with which group of disorders is it associated?

(A) HLA-B27; seronegative spondyloarthropathies (ankylosing spondylitis, reactive arthritis, psoriatic arthritis)

(B) HLA-DR4; seropositive rheumatoid arthritis

(C) HLA-DR3; Graves' disease and myasthenia gravis

(D) HLA-B8; systemic lupus erythematosus

Q24. A 55-year-old unvaccinated patient presents with a deep puncture wound sustained 6 hours ago. He is given tetanus antitoxin (human tetanus immunoglobulin). Which statement best describes this form of immunisation?

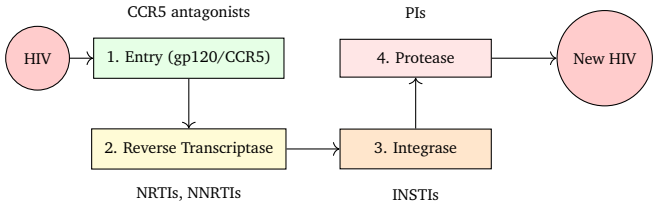
(A) Active immunisation; provides long-lasting protection with immunological memory

(B) Passive immunisation; preformed antibodies provide immediate but temporary protection without generating memory

(C) Herd immunisation; protects the community by reducing transmission

(D) Adoptive immunisation; transfer of sensitised T lymphocytes

Q25. A 32-year-old HIV-positive patient is being initiated on HAART. The HIV replication cycle and sites of drug action are shown below.



A patient on tenofovir, emtricitabine (NRTIs) + efavirenz (NNRTI) develops virological failure. Which target should be added for a second-line regimen?

(A) Nucleocapsid protein

(B) Surface glycoprotein gp41 only

(C) Integrase (using an INSTI such as dolutegravir)

(D) Host cell CD4 receptor

Q26. A 42-year-old male with a history of intravenous drug use presents with fatigue and elevated liver enzymes. Anti-HCV antibody is positive. HCV

RNA PCR confirms viremia with genotype 1. He is initiated on a direct-acting antiviral (DAA) regimen. Which of the following is the most accurate statement about HCV management in India?

(A) HCV is a DNA virus; interferon-based therapy remains the standard of care

(B) HCV genotype 2 is most prevalent in India

(C) Genotype 1 infection has a sustained virological response (SVR) rate of less than 50% with DAAs

(D) HCV genotype 1 is most common in India; sofosbuvir-based DAA regimens achieve SVR >95%

Q27. A neonate born to a mother with active genital lesions at the time of delivery presents on day 7 with vesicular skin rash, seizures, and hepatomegaly. HSV PCR from skin lesion swab is positive for HSV-2. Which statement best describes neonatal herpes?

(A) Neonatal HSV-2 causes disseminated disease with encephalitis; mortality exceeds 80% without IV acyclovir treatment

(B) HSV-2 is transmitted exclusively via blood transfusion to neonates

(C) Neonatal HSV-2 infection is self-limiting and requires no antiviral therapy

(D) HSV-2 neonatal infection always presents with the classic genital ulcer pattern

Q28. An outbreak of acute follicular conjunctivitis (red, painful eyes with preauricular lymphadenopathy) occurs among competitive swimmers after using a shared pool. Several members also develop pharyngitis and fever. The causative virus is non-enveloped with a double-stranded DNA genome and is detected in pooled conjunctival swabs by PCR. What is the most likely causative agent?

(A) Enterovirus 70

(B) Adenovirus

(C) Herpes simplex virus type 1

(D) Cytomegalovirus

Q29. A 7-year-old child presents with a characteristic "slapped-cheek" facial rash followed by a lace-like reticular rash on the trunk and extremities. Meanwhile, a sibling with known sickle-cell disease develops severe aplastic crisis requiring transfusion. The mother, who is pregnant (24 weeks), is concerned about fetal risk. Which virus is responsible for this cluster?

(A) Measles virus (rubeola)

(B) Rubella virus

(C) Parvovirus B19

(D) Human herpesvirus 6 (roseola)

Q30. A 44-year-old AIDS patient (CD4 count 45 cells/mm^3) presents with progressive cognitive decline, visual field defects, and asymmetric limb weakness over 8 weeks. MRI brain reveals multiple non-enhancing, non-space-occupying white matter lesions without mass effect. CSF PCR is positive for a polyomavirus. Which virus is responsible and what is the condition?

(A) CMV — cytomegalovirus retinitis

(B) EBV — primary CNS lymphoma

(C) HSV-1 — herpetic encephalitis

(D) JC virus — progressive multifocal leukoencephalopathy (PML)

Q31. A traveller returning from sub-Saharan Africa presents with high fever, jaundice, haematemesis ("black vomit"), and acute liver failure. He had not received any travel vaccines. Liver biopsy shows midzonal hepatic necrosis with Councilman bodies. Which pathogen is responsible and what is the correct vaccine type?

(A) Yellow fever virus (Flavivirus); 17D live attenuated vaccine, single dose

(B) Dengue virus; no licensed vaccine for travellers

(C) Hepatitis E virus; recombinant subunit vaccine (HEV 239)

(D) Leptospira icterohemorrhagiae; killed whole-cell vaccine

Q32. A 67-year-old immunocompromised transplant recipient develops acute febrile meningoencephalitis in late summer. He recalls multiple mosquito bites while gardening. CSF shows lymphocytic pleocytosis. Serology reveals IgM antibodies against a Flavivirus. Dead crows have been reported in the neighbourhood. Which virus is most likely responsible?

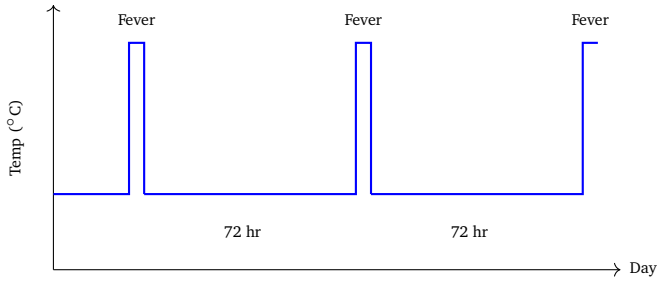
(A) Chikungunya virus

(B) West Nile virus

(C) Japanese encephalitis virus

(D) St. Louis encephalitis virus

Q33. A 9-year-old child from a malaria-endemic region presents with generalised oedema, massive proteinuria, and hypoalbuminaemia (nephrotic syndrome). His mother reports he has had recurring high fevers every 72 hours (quartan pattern) for several months as illustrated below.



Tertian = 48 hr (P. vivax/P. ovale)
Quartan = 72 hr (P. malariae)

Which *Plasmodium* species is most likely responsible and what is a characteristic feature?

(A) *Plasmodium vivax*; causes tertian fever with relapses due to hypnozoites

(B) *Plasmodium falciparum*; causes malignant tertian fever, cerebral malaria

(C) *Plasmodium malariae*; quartan fever (72-hour cycle), causes nephrotic syndrome, no relapse

(D) *Plasmodium ovale*; causes tertian fever, Schüffner's dots in RBCs

Q34. A 36-year-old AIDS patient (CD4 count 60 cells/mm^3) presents with headache, fever, and right-sided hemiparesis. MRI brain reveals multiple ring-enhancing lesions with surrounding oedema in the basal ganglia. Serology shows positive anti-*Toxoplasma* IgG. He owns two cats. He responds to empirical pyrimethamine + sulfadiazine treatment. Which statement about *Toxoplasma gondii* is correct?

(A) Dogs are the definitive host; sexual cycle occurs in dogs

(B) Oocysts are shed in human urine

(C) Primary infection always causes symptomatic disease in immunocompetent adults

(D) Cats are the definitive host (sexual cycle occurs in cats); bradyzoites in tissue cysts reactivate in immunosuppressed hosts

Q35. A 45-year-old man from a filariasis-endemic coastal district presents with massive lymphoedema of the left lower limb (elephantiasis) and scrotal swelling. Midnight blood smear reveals sheathed microfilariae. The vector is a *Culex* mosquito with nocturnal biting habits. What is the causative organism and drug of choice?

(A) *Wuchereria bancrofti*; diethylcarbamazine (DEC)

(B) *Brugia timori*; ivermectin

(C) *Loa loa*; mebendazole

(D) *Mansonella perstans*; albendazole

Q36. A 30-year-old traveller returning from Egypt develops acute fever, urticaria, eosinophilia, and hepatosplenomegaly 6 weeks after wading in freshwater (Katayama fever). Stool microscopy later reveals eggs with a prominent lateral spine. Years later, he develops portal hypertension with periportal ("pipestem") fibrosis. Which *Schistosoma* species is responsible?

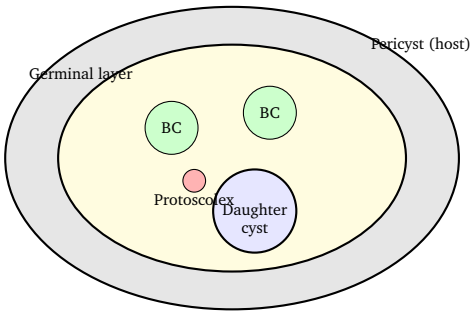
(A) *Schistosoma haematobium* (terminal-spined eggs; bladder involvement)

(B) *Schistosoma mansoni* (lateral-spined eggs; portal hypertension)

(C) *Schistosoma japonicum* (small lateral spine; associated with hepatic fibrosis)

(D) *Schistosoma intercalatum* (terminal spine; rectal involvement)

Q37. A 50-year-old shepherd presents with a slowly enlarging painless cystic lesion in the right lobe of the liver. CT abdomen shows a large unilocular cyst with internal daughter cysts and calcified wall. Casoni intradermal test is positive. The lifecycle involves dogs as definitive hosts and sheep as intermediate hosts. The cyst cross-section is depicted below.



Hydatid cyst: E. granulosus

What is the most likely diagnosis?

(A) Amoebic liver abscess

(B) Simple hepatic cyst

(C) Hydatid disease (*Echinococcus granulosus*)

(D) Polycystic liver disease

Q38. A 40-year-old AIDS patient (CD4 count 75 cells/mm^3) presents with profuse, watery, non-bloody diarrhoea (>10 litres/day) for 3 weeks, leading to severe dehydration and weight loss. Stool microscopy with modified acid-fast stain shows small (4–6 μm) bright-red staining oocysts. There is no effective antiparasitic treatment; management is primarily supportive with HAART to restore immunity. What is the causative organism?

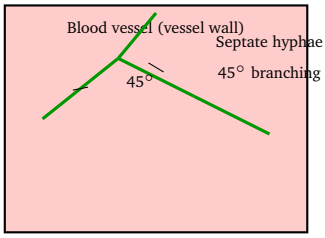
(A) *Giardia lamblia*

(B) *Entamoeba histolytica*

(C) *Isospora belli*

(D) *Cryptosporidium parvum*

Q39. A 58-year-old leukaemia patient (neutrophil count 200 cells/mm^3 on day 18 of chemotherapy) develops persistent fever unresponsive to broad-spectrum antibiotics, productive cough, and haemoptysis. CT chest shows a nodular lesion with surrounding ground-glass opacity ("halo sign"). Serum galactomannan is positive (index 2.4). Microscopy of BAL reveals acute-angle (45°) branching septate hyphae as shown below.



Angioinvasive *Aspergillus*: vessel invasion

What is the most likely causative organism?

(A) *Aspergillus fumigatus*

(B) *Mucor* (Rhizopus) species

(C) *Candida albicans*

(D) *Cryptococcus neoformans*

Q40. A 60-year-old man with uncontrolled diabetes mellitus (blood glucose 480 mg/dL, pH 7.1 — diabetic ketoacidosis) presents with rapidly progressive periorbital swelling, proptosis, black necrotic nasal discharge, ptosis, and ophthalmoplegia. Nasal biopsy shows broad, ribbon-like, aseptate (non-septate) hyphae with near 90° branching on PAS stain. What is the diagnosis?

(A) Invasive aspergillosis

(B) Rhinocerebral mucormycosis (*Mucor/Rhizopus* spp.)

(C) Cryptococcal sinusitis

(D) Fusarium infection

Detailed Solutions

Q1.

Solution

Concept – Streptococcus pyogenes (Group A Streptococcus): *S. pyogenes* causes pharyngitis that triggers autoimmune sequelae via molecular mimicry between streptococcal antigens (M protein) and cardiac, joint, and CNS tissue. ASO (anti-streptolysin O) titre rises 1–3 weeks after streptococcal pharyngitis and is the serological hallmark of preceding Group A Streptococcal (GAS) infection. Rheumatic fever (carditis, migratory polyarthritis, Sydenham's chorea, subcutaneous nodules, erythema marginatum) follows pharyngitis, NOT skin infection.

Key Point: ASO titre >200 IU/mL confirms recent streptococcal pharyngitis. Anti-DNase B is the better marker for post-streptococcal glomerulonephritis (which follows both pharyngitis and skin infection).

Why other options are wrong:

- Option A: *S. aureus* produces alpha-toxin, not streptolysin O; does not cause rheumatic fever.
- Option B: *S. pneumoniae* causes lobar pneumonia, meningitis, and otitis media; not rheumatic fever.
- Option D: *Enterococcus faecalis* is a normal gut commensal; not associated with rheumatic fever.

Final Answer: ASO titre elevation + migratory polyarthritis + carditis after pharyngitis $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q1

Q2.

Solution

Concept – Vancomycin-Resistant Enterococci (VRE): Vancomycin resistance in enterococci is mediated by alterations in the peptidoglycan precursor target. The **vanA** gene encodes a ligase that substitutes D-Ala-D-Lac for D-Ala-D-Ala in peptidoglycan synthesis, preventing vancomycin from binding. vanA confers high-level resistance to both vancomycin AND teicoplanin (MIC >64 μg/mL). vanB confers resistance to vancomycin but remains susceptible to teicoplanin.

Key Point: vanA (plasmid-mediated, transferable) = HIGH-level resistance to vancomycin AND teicoplanin. vanB = resistance to vancomycin only. mecA encodes

PBP2a (MRSA); blaZ encodes penicillinase.

Why other options are wrong:

- Option A: mecA encodes altered PBP2a causing methicillin/oxacillin resistance in *S. aureus* (MRSA), not vancomycin resistance in enterococci.
- Option B: blaZ encodes penicillinase (beta-lactamase) causing penicillin resistance; not relevant to vancomycin resistance.
- Option C: vanB also confers vancomycin resistance but is susceptible to teicoplanin; MIC is lower than vanA.

Final Answer: High-level vancomycin + teicoplanin resistance in *Enterococcus* = vanA gene $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q2

Q3.

Solution

Concept – Klebsiella pneumoniae (Friedlander's Pneumonia): *K. pneumoniae* causes a distinctive community-acquired pneumonia in alcoholics and diabetics characterised by: (1) lobar consolidation with bulging fissure sign, (2) upper lobe predilection with early cavitation, (3) "currant jelly" sputum (blood mixed with mucus from tissue necrosis), and (4) high mortality. Microbiologically: mucoid lactose-fermenting Gram-negative rod, positive string test (mucoviscosity), growth on MacConkey agar.

Key Point: "Currant jelly" sputum + alcoholic + upper lobe cavitation = *Klebsiella pneumoniae*. The capsule (K antigen) is the primary virulence factor, inhibiting phagocytosis.

Why other options are wrong:

- Option B: *P. aeruginosa* causes pneumonia in CF and ICU patients; produces blue-green pigment (pyocyanin); oxidase-positive.
- Option C: *S. pneumoniae* causes lobar pneumonia with rusty sputum; no cavitation typically; Gram-positive lanceolate diplococci.
- Option D: *H. influenzae* causes COPD exacerbations and paediatric pneumonia; small pleomorphic Gram-negative coccobacilli; no currant jelly sputum.

Final Answer: Alcoholic + currant jelly sputum + upper lobe cavitation + mucoid colonies = *Klebsiella pneumoniae* $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – Proteus mirabilis (Swarming, Urease, Struvite Stones): *P. mirabilis* is a Gram-negative rod from the Enterobacteriaceae family with three hallmark features: (1) **Swarming motility** — periodic waves of migration producing concentric rings on solid agar due to hyperflagellation; (2) **Urease-positive** — splits urea to produce ammonia, alkalinising urine (pH >7); (3) **Struvite (triple phosphate) stone formation** — alkaline urine allows precipitation of magnesium ammonium phosphate (struvite) and carbonate apatite, forming staghorn calculi.

Key Point: Recurrent UTI + alkaline urine + staghorn calculi + swarming on agar + urease-positive = *Proteus mirabilis*.

Why other options are wrong:

- Option A: *E. coli* is the commonest cause of UTI but does NOT swarm and is urease-negative.
- Option C: *P. aeruginosa* causes UTI in catheterised patients; oxidase-positive; pyocyanin pigment; does NOT produce struvite stones classically.
- Option D: *K. pneumoniae* can cause UTI; urease-positive but does NOT show swarming motility.

Final Answer: Recurrent UTI + alkaline urine + swarming + urease-positive + staghorn calculi = *Proteus mirabilis* $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q4

Q5.

Solution

Concept – Waterhouse-Friderichsen Syndrome (Neisseria meningitidis): Fulminant meningococcaemia (Group A, B, C, W, Y) can cause Waterhouse-Friderichsen syndrome — bilateral adrenal haemorrhage leading to acute adrenal insufficiency. Pathogenesis: endotoxin (LPS) + DIC → haemorrhagic infarction of adrenal cortex. Clinical triad: (1) sudden high fever, (2) petechial/purpuric non-blanching rash (meningococcaemia), (3) cardiovascular collapse. Treatment: immediate penicillin G or ceftriaxone + ICU support. **Gram stain**: Gram-negative diplococci (kidney-bean shaped), oxidase-positive, maltose-fermenting (distinguishes from *N. gonorrhoeae*).

Key Point: Non-blanching petechial rash + rapid deterioration + bilateral adrenal haemorrhage = Waterhouse-Friderichsen syndrome = *Neisseria meningitidis*. Do

NOT wait for investigations — give penicillin immediately.

Why other options are wrong:

- Option A: *S. pneumoniae* causes meningitis but rarely causes Waterhouse-Friderichsen syndrome; no petechial rash typically.
- Option B: *H. influenzae* type b causes meningitis, mainly in children; petechial rash not characteristic.
- Option D: *S. aureus* causes toxic shock syndrome with diffuse erythroderma (not petechiae) and is non-encapsulated.

Final Answer: Petechial rash + fulminant shock + bilateral adrenal haemorrhage = *Neisseria meningitidis* $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q5

Q6.

Solution

Concept – Campylobacter jejuni and Guillain-Barré Syndrome (GBS): *C. jejuni* is the most common bacterial cause preceding GBS. The mechanism is **molecular mimicry**: *C. jejuni* lipooligosaccharide (LOS) shares structural homology with GM1 and GD1a gangliosides on peripheral nerve myelin. Antibodies generated against LOS cross-react with gangliosides, causing demyelination (AIDP) or axonal damage (AMAN variant). GBS presents 1–3 weeks after infection as ascending flaccid paralysis with albuminocytological dissociation in CSF (elevated protein, normal cells). *C. jejuni* microbiology: spiral/comma-shaped Gram-negative rod, oxidase-positive, microaerophilic, grows at 42°C.

Key Point: Ascending paralysis 1–3 weeks after **watery or bloody diarrhoea** = GBS due to *C. jejuni*. *C. jejuni* is the commonest infection preceding GBS.

Why other options are wrong:

- Option A: *S. typhi* causes enteric fever; associated with reactive arthritis, not GBS.
- Option B: *Shigella* causes dysentery; associated with HUS (in *S. dysenteriae* type 1), not GBS.
- Option C: *E. coli* O157:H7 causes HUS (haemolytic uraemic syndrome), not GBS.

Final Answer: Diarrhoea → ascending paralysis + albuminocytological dissociation = GBS from *C. jejuni* $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – Clostridium perfringens (Gas Gangrene, Nagler Reaction): *C. perfringens* (Type A) causes gas gangrene (clostridial myonecrosis) through the alpha-toxin (**lecithinase/phospholipase C**), which cleaves lecithin (phosphatidylcholine) in cell membranes, causing massive tissue destruction. Clinical features: rapidly progressive severe pain, oedema, bronze/dark skin, gas in tissues (crepitus on palpation/X-ray), sweet/offensive odour, systemic toxaemia. **Nagler Reaction**: alpha-toxin produces lecithinase activity detected as an opalescent precipitate around colonies on egg-yolk agar, inhibited by specific anti-alpha-toxin antiserum.

Key Point: Deep agricultural wound + crepitus (gas in soft tissue) + Nagler reaction positive + opalescence on egg-yolk agar = *Clostridium perfringens*. Treatment: immediate surgical debridement + penicillin G + hyperbaric oxygen.

Why other options are wrong:

- Option B: *C. tetani* causes tetanus (spastic paralysis), not gas gangrene; produces tetanospasmin.
- Option C: *C. difficile* causes pseudomembranous colitis (antibiotic-associated diarrhoea), not gas gangrene.
- Option D: *B. fragilis* causes intra-abdominal anaerobic infections; does not produce alpha-toxin; Nagler-negative.

Final Answer: Agricultural wound + gas + Nagler reaction = *Clostridium perfringens* $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – Nocardia asteroides (Weakly Acid-Fast, Immunocompromised): *Nocardia* spp. are aerobic, filamentous, Gram-positive bacteria that are partially (weakly) acid-fast on modified ZN stain (using 1% H_2SO_4 as decolouriser instead of 3% HCl). This distinguishes *Nocardia* from *Actinomyces* (not acid-fast) and *Mycobacterium* (strongly acid-fast). *Nocardia* causes disseminated infections in immunocompromised hosts: pulmonary nodules/cavities + **brain abscess** is the

classic combination. Treatment: TMP-SMX (first-line, long-term 6–12 months).

Key Point: Immunocompromised (HIV, transplant) + lung nodules + brain abscess + **weakly acid-fast branching filaments** = *Nocardia asteroides*. Gram-positive AND partially acid-fast = Nocardia.

Why other options are wrong:

- Option A: *Actinomyces israelii* is NOT acid-fast, causes cervicofacial actinomycosis (lumpy jaw), forms sulphur granules; anaerobic.
- Option C: Cryptococcal meningitis presents with headache, meningismus; CSF shows encapsulated yeast with India ink; no lung nodules typically.
- Option D: Cerebral toxoplasmosis also causes ring-enhancing lesions but the organism is a protozoan parasite (not acid-fast filaments); responds to pyrimethamine + sulfadiazine.

Final Answer: HIV + lung nodules + brain abscess + weakly acid-fast branching filaments = *Nocardia asteroides* $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q8

Q9.

Solution

Concept – Borrelia burgdorferi (Lyme Disease, Ixodes Tick): Lyme disease is caused by *Borrelia burgdorferi* (USA), *B. garinii*, and *B. afzelii* (Europe). Vector: *Ixodes scapularis* (deer tick) in the northeastern USA. Transmission occurs at the **nymph stage** (May–July), because nymphs are small and feed for longer. Stages of Lyme disease: (1) Early localised: erythema migrans (bull's-eye rash, >5 cm); (2) Early disseminated: multiple EM, cranial nerve palsy (Bell's palsy), AV block, migratory arthritis; (3) Late: chronic arthritis, encephalopathy. Treatment: doxycycline (early), ceftriaxone (CNS/cardiac).

Key Point: Erythema migrans (expanding annular rash) + migratory arthritis + heart block + forested area northeastern USA = Lyme disease = *Borrelia burgdorferi*, transmitted by *Ixodes* nymph.

Why other options are wrong:

- Option A: *R. rickettsii* causes Rocky Mountain Spotted Fever (petechial rash starting at wrists/ankles spreading centrally); transmitted by *Dermacentor* tick.
- Option B: *Ehrlichia chaffeensis* causes monocytic ehrlichiosis; fever, leucope-

nia, thrombocytopenia; *Amblyomma* tick vector.
- Option D: *Anaplasma phagocytophilum* causes granulocytic anaplasmosis; morulae in neutrophils; *Ixodes* tick but no erythema migrans.

Final Answer: Bull's-eye rash + arthritis + heart block + northeastern USA = Lyme disease = *Borrelia burgdorferi* $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q9

Q10.

Solution

Concept – Francisella tularensis (Tularemia): *F. tularensis* is a tiny, fastidious, Gram-negative coccobacillus that requires cysteine and glucose for growth. It is a Biosafety Level 3 (BSL-3) agent and potential bioterrorism agent. Transmission: contact with infected animals (rabbits, hares, squirrels), tick/deerfly bites, inhalation of aerosols. **Clinical forms**: ulceroglandular (most common, 75%) — skin ulcer at inoculation site + regional lymphadenopathy (buboes); pneumonic (most severe, >30% mortality if untreated); typhoidal. Treatment: streptomycin (first-line) or gentamicin; alternatives: doxycycline, ciprofloxacin.

Key Point: Rabbit/tick exposure + painless ulcer at bite site + regional lymphadenopathy + septic shock + cysteine-requiring tiny Gram-negative coccobacillus = *Francisella tularensis* (tularemia).

Why other options are wrong:

- Option A: *Y. pestis* (plague) also causes buboes but transmitted by rat flea (*Xenopsylla cheopis*); safety-pin bipolar staining; no skin ulcer.
- Option B: *Bartonella henselae* causes cat scratch disease (papule + regional lymphadenopathy); transmitted by cat scratch, not tick; not causing septic shock.
- Option C: *Brucella* causes undulant fever, hepatosplenomegaly; associated with unpasteurised dairy or animal contact; no skin ulcer.

Final Answer: Rabbit hunter + tick bite + skin ulcer + lymphadenopathy + cysteine-requiring organism = *Francisella tularensis* $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q10

Q11.

Solution

Concept – Coxiella burnetii (Q Fever): *C. burnetii* is a unique obligate intracellular organism (formerly classified as Rickettsia, now in Legionellales). Key distinguishing features: (1) **No arthropod vector required** for transmission — primarily aerosol transmission from parturient animals (placenta, amniotic fluid, urine of sheep, goats, cattle); (2) Highly infectious (ID_{50} = 1 organism); (3) Forms spore-like variants that survive in the environment. Serology: Phase II IgG elevated in acute Q fever; Phase I IgG elevated in chronic Q fever (endocarditis). Complications: Q fever endocarditis (culture-negative, requires anti-phase I IgG titre >1:800 for diagnosis). Treatment: doxycycline.

Key Point: Atypical pneumonia + contact with parturient animals + **NO tick bite** + high Phase II antibody titre = *Coxiella burnetii* (Q fever). The "Q" stands for "Query" (unknown aetiology when first described).

Why other options are wrong:

- Option B: *R. conorii* (Mediterranean spotted fever) requires tick bite; maculopapular rash with eschar; does not cause atypical pneumonia typically.
- Option C: *C. psittaci* causes psittacosis from bird (parrot) exposure; not farm animals.
- Option D: *L. pneumophila* causes Legionnaire's disease from contaminated water aerosols (cooling towers, AC); no animal contact.

Final Answer: Atypical pneumonia + farm animal (sheep) contact + no tick bite + Phase II IgG positive = *Coxiella burnetii* Q fever $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q11

Q12.

Solution

Concept – Burkholderia pseudomallei (Melioidosis): Melioidosis is endemic in Southeast Asia and northern Australia (especially Thailand, Vietnam). *B. pseudomallei* is a Gram-negative rod in the soil and water of rice paddies. Characteristic features: (1) **Bipolar "safety-pin" staining** on Giemsa/methylene blue; (2) Growth on Ashdown's medium (purple metallic colonies); (3) Oxidase-positive; (4) TMP-SMX RESISTANT (unusual — used to treat *Nocardia*); (5) Pulmonary form mimics TB (upper lobe cavitation). Treatment: intensive phase IV meropenem or ceftazidime; eradication phase oral TMP-SMX (despite intrinsic resistance, this is the eradication regimen — based on clinical evidence). Melioidosis

is NOT to be confused with glanders (*B. mallei* — horse-to-human).

Key Point: Rice-paddy farmer from SE Asia + upper lobe cavitation + bipolar safety-pin Gram stain + Ashdown's medium = *Burkholderia pseudomallei* (melioidosis).

Why other options are wrong:

- Option A: *B. mallei* causes glanders in horses (rare in humans); not rice-paddy associated.
- Option C: *Y. pestis* (plague) shows bipolar staining but causes bubonic/pneumonic plague; rat flea vector; not rice-paddy association.
- Option D: *Brucella melitensis* shows bipolar staining but causes undulant fever, hepatosplenomegaly; associated with goats/unpasteurised milk.

Final Answer: Rice-paddy farmer + bipolar safety-pin + Ashdown's medium + upper lobe cavitation = melioidosis (*B. pseudomallei*) $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q12

Q13.

Solution

Concept – Acinetobacter baumannii (MDR, Ventilator-Associated Pneumonia): *A. baumannii* is a non-fermenting, oxidase-negative, Gram-negative coccobacillus that is notorious for hospital-acquired and ICU infections. Key features: (1) Ventilator-associated pneumonia (VAP) is the most common presentation; (2) MDR/XDR due to OXA-type carbapenemases (OXA-23, OXA-24, OXA-58) which are most prevalent carbapenem resistance mechanism in *Acinetobacter*; (3) Also metallobetalactamases (NDM-1) possible; (4) Treatment options for MDR: colistin (polymyxin E), tigecycline, sulbactam (has intrinsic activity). Epidemiological control requires strict contact precautions.

Key Point: ICU + mechanical ventilation + oxidase-negative non-fermenter + OXA carbapenemase = *Acinetobacter baumannii*. Colistin is the "last resort" treatment.

Why other options are wrong:

- Option A: *P. aeruginosa* is also oxidase-positive (key difference); also MDR but uses VIM, IMP, or GES metallo-beta-lactamases and AmpC; not OXA-23.
- Option B: *Stenotrophomonas maltophilia* is TMP-SMX sensitive; oxidase-negative but intrinsically resistant to carbapenems (different mechanism —

L1 metallo-beta-lactamase); associated with CF.
- Option D: *B. cepacia* is oxidase-positive; mainly causes infection in CF patients; rare VAP.

Final Answer: ICU VAP + non-fermenter + oxidase-negative + OXA-23 carbapenemase = *Acinetobacter baumannii* $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – Pasteurella multocida (Animal Bite Infections): *P. multocida* is the most common cause of rapidly progressing wound infections following cat bites (>50%) and dog bites (~20–30%). Key features: (1) **Rapid onset** (within 24 hours of bite — faster than other wound pathogens); (2) Small Gram-negative coccobacilli with bipolar staining; (3) Grows on blood agar with "mousy" odour; does NOT grow on MacConkey agar; (4) Oxidase-positive; (5) **Penicillin-sensitive** (important! — MRSA prophylaxis NOT needed; amoxicillin-clavulanate is first-line for animal bites).

Key Point: Cat bite + rapid cellulitis (<24 hours) + penicillin-sensitive Gram-negative coccobacillus = *Pasteurella multocida*. Cat bites have higher infection rate than dog bites due to deep puncture wounds.

Why other options are wrong:

- Option A: *Eikenella corrodens* causes human bite infections ("fight bite"); pit on agar; penicillin-sensitive.
- Option B: *Capnocytophaga canimorsus* causes severe sepsis in asplenic patients after dog bite; Gram-negative fusiform rods; rare.
- Option C: *S. aureus* is Gram-positive; not specifically associated with cat bites; slower onset typically; penicillin-resistant (usually).

Final Answer: Cat bite + rapid cellulitis (<12 hours) + penicillin-sensitive Gram-negative coccobacillus + mousy odour = *Pasteurella multocida* $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q14

Q15.

Solution

Concept – Anaphylaxis (Type I Hypersensitivity): Anaphylaxis is a life-threatening **IgE-mediated (Type I)** hypersensitivity reaction. Mechanism: prior sensitisation → allergen-specific IgE binds to high-affinity FcεRI receptors on mast cells and basophils → re-exposure cross-links IgE → massive degranulation releasing histamine, tryptase, prostaglandins, leukotrienes, and platelet-activating factor → vasodilation, bronchospasm, increased vascular permeability. Clinical: urticaria, angioedema, bronchospasm, hypotension, cardiovascular collapse within minutes. **First-line treatment: intramuscular epinephrine** (adrenaline) 0.5 mg (1:1000) IM into the anterolateral thigh; epinephrine reverses bronchospasm, vasodilation, and prevents further mediator release.

Key Point: Anaphylaxis = IgE + mast cell degranulation (histamine) = **IM epinephrine** (NOT IV steroids or antihistamines as first-line). Tryptase is the best marker to confirm anaphylaxis retrospectively.

Why other options are wrong:

- Option B: IgG + complement (Type II hypersensitivity) causes autoimmune haemolytic anaemia, not anaphylaxis; steroids are not first-line for anaphylaxis.
- Option C: Immune complex deposition (Type III) causes serum sickness, SLE, post-streptococcal GN; presents days to weeks later.
- Option D: Cell-mediated (Type IV) causes contact dermatitis, granuloma; no immediate reaction.

Final Answer: Immediate reaction to penicillin (urticaria, bronchospasm, hypotension) = IgE-mediated anaphylaxis → IM epinephrine $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q15

Q16.

Solution

Concept – Complement C5a (Chemotaxin): The complement system generates several biologically active split products. **C5a** is the most potent chemotactic factor for neutrophils (and monocytes), attracting them to sites of infection/inflammation. C5a also: (1) causes mast cell and basophil degranulation (anaphylatoxin activity); (2) upregulates neutrophil adhesion molecules; (3) triggers neutrophil oxidative burst. Compare with C3a — also an anaphylatoxin but weaker chemotaxin. C5b-9 forms the membrane attack complex (MAC). C3b is

the major opsonin. C4b is a weak opsonin.

Key Point: C5a = most potent chemotaxin + anaphylatoxin. C3a = anaphylatoxin (weaker). C3b = opsonin. C5b-9 = MAC (lysis). Mnemonic: "C5a = Come here" (chemoattractant).

Why other options are wrong:

- Option A: C3b is the most important opsonin (promotes phagocytosis via CR1 on macrophages); weak chemotaxin.
- Option C: C1q initiates the classical pathway by binding antibody-antigen complexes; no direct chemotactic activity.
- Option D: C4b is a weak opsonin formed in the classical and lectin pathways; not a significant chemotaxin.

Final Answer: Most potent complement-derived chemotaxin for neutrophils + mast cell degranulation = C5a $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q16

Q17.

Solution

Concept – Th1 vs Th2 CD4+ T-Helper Cell Differentiation: Naive CD4+ T cells differentiate into functional subsets depending on the cytokine environment: **Th1 pathway**: IL-12 (from macrophages/DC) + IFN-γ $\rightarrow$ T-bet transcription factor $\rightarrow$ secretes IFN-γ, IL-2, TNF-β $\rightarrow$ activates macrophages (intracellular killing), promotes CTL responses, IgG2a antibodies. Key role: defence against **intracellular pathogens** (*Mycobacteria, Salmonella, Leishmania*, viruses). **Th2 pathway**: IL-4 $\rightarrow$ GATA-3 transcription factor $\rightarrow$ secretes IL-4, IL-5, IL-13, IL-10 $\rightarrow$ IgE production, eosinophil activation, mast cell sensitisation. Key role: defence against **helminths**, allergy/atopy.

Key Point: Th1 = IFN-γ + IL-2 (intracellular pathogens, macrophage activation). Th2 = IL-4 + IL-5 + IL-13 (IgE, eosinophils, helminths). IL-12 from macrophages drives Th1. IL-4 drives Th2.

Why other options are wrong:

- Option A: IL-4, IL-5 are Th2 cytokines promoting IgE and eosinophils; these are NOT Th1.
- Option B: IL-10 and TGF-β are produced by Treg (regulatory T) cells; suppress inflammation; not Th1.

- Option D: IL-13 is a Th2 cytokine; IL-17 is produced by Th17 cells (not Th1); promotes neutrophil recruitment.

Final Answer: Th1 cells secrete IFN-γ and IL-2, activating macrophages and CTLs against intracellular pathogens $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q17

Q18.

Solution

Concept – Cytotoxic T Lymphocytes (CD8+, MHC Class I): CTLs (cytotoxic T lymphocytes) are **CD8+** T cells that recognise antigen presented on **MHC class I** molecules (present on all nucleated cells). Mechanism of killing: (1) **Perforin** — polymerises in the target cell membrane to form pores (similar to MAC of complement), allowing entry of granzymes; (2) **Granzyme B** — serine protease entering through perforin pores, activates caspase 3 and 9 cascade $\rightarrow$ apoptosis; (3) **FasL-Fas interaction** — CTL Fas ligand binds Fas (CD95) on target cell $\rightarrow$ apoptosis. CTLs are essential for killing virus-infected cells, tumour cells, and transplant rejection.

Key Point: CTLs = CD8+ + MHC class I restriction. Killing mechanism = perforin (pores) + granzyme B (apoptosis). CD4+ cells = MHC class II restriction (helper function).

Why other options are wrong:

- Option A: CTLs are CD8+ (not CD4+) and restricted by MHC class I (not class II). CD4+ cells are T-helper cells.
- Option B: ADCC is mediated by NK cells and macrophages carrying Fc receptors, not by CTLs (which are antigen-specific).
- Option C: Complement activation is a humoral mechanism; CTLs kill through perforin/granzyme and Fas-FasL, not complement.

Final Answer: CTLs = CD8+ cells, MHC class I restricted; perforin pores + granzyme B apoptosis $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q18

Q19.

Solution

Concept – Macrophage Activation by IFN-γ: *Mycobacterium tuberculosis* survives within macrophages by preventing phagolysosome fusion (via lipoarabinomannan). The key to intracellular killing is **macrophage activation** by **IFN-γ** secreted by Th1 CD4+ T cells and CD8+ T cells. IFN-γ activates macrophages to: (1) enhance phagolysosome fusion; (2) upregulate NADPH oxidase (reactive oxygen species — superoxide, H_2O_2); (3) induce iNOS (inducible nitric oxide synthase) producing reactive nitrogen species (NO, peroxynitrite); (4) increase antigen presentation (MHC class II upregulation). Granuloma formation also requires IFN-γ-activated macrophages to form Langhans giant cells.

Key Point: IFN-γ is the primary cytokine that activates macrophages to kill intracellular pathogens. IFN-γ deficiency leads to disseminated mycobacterial infections (Mendelian susceptibility to mycobacterial disease, MSMD).

Why other options are wrong:

- Option B: IL-4 is a Th2 cytokine; promotes IgE production and mast cell growth; INHIBITS macrophage activation (anti-Th1).
- Option C: IL-10 is anti-inflammatory; produced by Tregs and macrophages; inhibits macrophage activation (suppresses TNF-α, IL-12, IFN-γ).
- Option D: IL-13 is a Th2 cytokine; promotes mucus production and airway hyperresponsiveness; does not activate macrophages for killing.

Final Answer: Macrophage activation for intracellular killing (TB) = IFN-γ (from Th1 cells) $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q19

Q20.

Solution

Concept – Memory B Cells and Affinity Maturation: The secondary (anamnestic) immune response is faster, stronger, and of higher affinity than the primary response due to **memory B cells**. Memory B cells are generated during the primary immune response in the germinal centres (GCs) of secondary lymphoid organs (lymph nodes, spleen). Key processes in germinal centres: (1) **Somatic hypermutation** — point mutations introduced in Ig variable region genes by AID (activation-induced cytidine deaminase); (2) **Affinity maturation** — B cells with higher-affinity BCRs for antigen are positively selected to survive; those with lower affinity undergo apoptosis; (3) **Class switching** — IgM $\rightarrow$ IgG, IgA, or IgE. Mem-

ory B cells persist long-term and rapidly differentiate into plasma cells upon re-exposure.

Key Point: Memory B cells (from GC reactions) produce rapid secondary response with high-affinity IgG. Affinity maturation = somatic hypermutation + selection in germinal centres.

Why other options are wrong:

- Option A: Naive B cells mediate the **primary** immune response; slow onset, lower affinity IgM initially; no affinity maturation occurs in bone marrow.
- Option C: Plasma cells are antibody-secreting effector cells; they are short-lived (after primary response) or long-lived (in bone marrow after secondary response); not responsible for ”recall”.
- Option D: Regulatory T cells (Tregs) suppress immune responses; not responsible for anamnestic antibody responses.

Final Answer: Rapid secondary response with high-affinity IgG = memory B cells; affinity maturation in germinal centres $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q20

Q21.

Solution

Concept – Neutrophil Extracellular Traps (NETs): NETs are a specialised neutrophil killing mechanism involving the extrusion of decondensed chromatin (DNA + histones) studded with granule proteins (elastase, myeloperoxidase, cathepsin G, defensins) to form extracellular web-like structures that trap and kill bacteria, fungi, and parasites. NET formation (NETosis) can be: (1) **Suicidal NETosis**: NADPH oxidase-dependent, cell dies; (2) **Vital NETosis**: cell survives after NET extrusion. NETs are most important against large extracellular pathogens that cannot be phagocytosed. Citrullination of histones by PAD4 enzyme facilitates chromatin decondensation. Elevated NET levels contribute to pathology in sepsis, lupus, thrombosis.

Key Point: NETs = DNA + histones + granule enzymes extruded extracellularly. Traps and kills bacteria (*S. aureus*, *S. pyogenes*) and fungi that are too large to phagocytose. Marker of NETs: citrullinated histone H3 (CitH3).

Why other options are wrong:

- Option A: ADCC requires antibody coating of target + NK cells or

macrophages with Fc receptors; not a neutrophil extracellular mechanism.
- Option B: Respiratory burst (NADPH oxidase) produces intracellular reactive oxygen species within the phagolysosome; an intracellular killing mechanism.
- Option D: Degranulation releases granule contents (elastase, defensins) outside the cell but does NOT involve chromatin/DNA extrusion; NETs are distinct.

Final Answer: Extracellular DNA-histone webs trapping bacteria = Neutrophil Extracellular Traps (NETs) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q21

Q22.

Solution

Concept – IL-10 (Anti-Inflammatory Cytokine): IL-10 is the major anti-inflammatory (immunosuppressive) cytokine produced by: regulatory T cells (Tregs), macrophages, B cells, and Th2 cells. Actions of IL-10: (1) Inhibits production of pro-inflammatory cytokines: TNF-α, IL-1, IL-6, IL-12, IFN-γ; (2) Downregulates MHC class II expression on antigen-presenting cells; (3) Reduces macrophage activation; (4) Promotes Treg differentiation. In sepsis, IL-10 contributes to the **immunoparalysis phase** (CARS — compensatory anti-inflammatory response syndrome), increasing susceptibility to secondary infections. Paradoxically, IL-10 is also protective against excessive inflammation and organ damage.

Key Point: IL-10 = master anti-inflammatory cytokine. Inhibits TNF-α, IL-12, IFN-γ. Produced by Tregs and macrophages. Contrast: pro-inflammatory cytokines (TNF-α, IL-1β, IL-6, IL-8, IFN-γ).

Why other options are wrong:

- Option A: IL-1β is a potent pro-inflammatory cytokine (fever, acute phase response); produced by macrophages in the SIRS phase.
- Option B: IL-6 is pro-inflammatory; stimulates acute phase protein production (CRP, fibrinogen); not anti-inflammatory.
- Option C: TNF-α is the major pro-inflammatory cytokine; causes fever, cachexia, activation of endothelium; not anti-inflammatory.

Final Answer: Immunosuppressive cytokine inhibiting TNF-α and IL-12 in sepsis = IL-10 $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q22

Q23.

Solution

Concept – HLA-B27 (Seronegative Spondyloarthropathies): HLA-B27 is a class I MHC antigen strongly associated with the seronegative spondyloarthropathies (SpA). The association includes: ankylosing spondylitis (AS, 90–95% HLA-B27 positive), reactive arthritis (formerly Reiter syndrome, 60–80%), psoriatic arthritis (with axial involvement, 60%), inflammatory bowel disease-associated arthritis, and undifferentiated SpA. In AS: chronic sacroiliitis + bamboo spine on X-ray + morning stiffness improving with exercise + negative RF/ANA. HLA-B27 arthritogenic mechanisms: molecular mimicry, misfolded protein accumulation (unfolded protein response), or aberrant peptide presentation to CD8+ T cells.

Key Point: HLA-B27 = seronegative spondyloarthropathies (AS, reactive arthritis, psoriatic arthritis, IBD arthritis). "PAIR" mnemonic: Psoriatic arthritis, Ankylosing spondylitis, IBD arthritis, Reactive arthritis.

Why other options are wrong:

- Option B: HLA-DR4 is associated with seropositive (RF+) rheumatoid arthritis; not with seronegative SpA.
- Option C: HLA-DR3 is associated with Graves' disease, myasthenia gravis, type 1 diabetes, coeliac disease; not AS.
- Option D: HLA-B8/DR3 is associated with SLE, myasthenia gravis, primary Sjögren's; not AS.

Final Answer: Young male + sacroiliitis + bamboo spine + morning stiffness + RF negative = ankylosing spondylitis = HLA-B27 $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q23

Q24.

Solution

Concept – Passive Immunisation (Antitoxin): Immunisation can be active or passive. **Passive immunisation**: administration of **preformed antibodies** (immunoglobulins/antitoxin) from an external source. Features: (1) Immediate protection (no lag period); (2) Temporary (weeks to months, as the antibodies are catabolised); (3) **No immunological memory generated**; (4) Examples: HTIG (human tetanus immunoglobulin), rabies immunoglobulin (RIG), hepatitis B immunoglobulin (HBIG), diphtheria antitoxin, varicella-zoster immunoglobulin (VZIG), palivizumab (anti-RSV monoclonal antibody). Contrast with active immunisation (vaccines) which requires 1–2 weeks to develop immunity but generates

memory.

Key Point: Passive immunisation = preformed antibodies; immediate but temporary; NO memory. Active immunisation = antigen stimulus; delayed (1–2 weeks); long-lasting memory. Neonatal protection from maternal IgG = natural passive immunity.

Why other options are wrong:

- Option A: Active immunisation is vaccination with antigen (dead/live/toxoid); generates memory; not relevant to antitoxin given post-exposure.
- Option C: Herd immunity is a population-level concept where high vaccination coverage protects unvaccinated individuals indirectly; not applicable to a single patient.
- Option D: Adoptive immunisation/transfer involves transferring sensitised T lymphocytes (used experimentally); not the mechanism of antitoxin.

Final Answer: Tetanus antitoxin given post-exposure = passive immunisation (preformed antibodies, temporary, no memory) $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q24

Q25.

Solution

Concept – HIV Replication Cycle and HAART Drug Targets: HIV (Lentivirus, Retroviridae) replication: (1) **Entry**: gp120 binds CD4, then CCR5/CXCR4 co-receptor; blocked by **CCR5 antagonists** (maraviroc) and fusion inhibitors (enfuvirtide); (2) **Reverse transcription**: RNA $\rightarrow$ DNA by reverse transcriptase (RT); blocked by **NRTIs** (tenofovir, abacavir, emtricitabine) and **NNRTIs** (efavirenz, nevirapine); (3) **Integration**: viral DNA integrates into host genome by integrase; blocked by **INSTIs** (dolutegravir, raltegravir, bictegravir); (4) **Maturation**: protease cleaves Gag-Pol polyprotein; blocked by **PIs** (lopinavir, darunavir, atazanavir). Virological failure on TDF/FTC/EFV requires switching to integrase-based regimen (dolutegravir preferred for high barrier to resistance).

Key Point: HAART failure on NRTI + NNRTI backbone $\rightarrow$ add/switch to **INSTI** (dolutegravir). Dolutegravir has the highest genetic barrier to resistance among INSTIs.

Why other options are wrong:

- Option A: Nucleocapsid protein is not a current drug target in clinical use.
- Option B: Targeting gp41 only (enfuvirtide, a fusion inhibitor) is not sufficient for second-line rescue therapy after NNRTI failure.
- Option D: CD4 receptor is a host protein; targeting it would cause severe immunosuppression; not a therapeutic strategy.

Final Answer: NRTI + NNRTI failure $\rightarrow$ add integrase inhibitor (INSTI: dolutegravir) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q25

Q26.

Solution

Concept – Hepatitis C Virus (Genotype 1, DAAs): HCV is a **single-stranded, positive-sense RNA virus** (Flaviviridae family, Hepacivirus genus). Key facts: (1) Most common mode of transmission: intravenous drug use, needlestick injuries, blood transfusion (before 1992); (2) **Genotype 1** is most prevalent globally and in India; (3) **Direct-acting antivirals (DAAs)**: sofosbuvir (NS5B polymerase inhibitor) + daclatasvir or ledipasvir (NS5A inhibitors); achieve **SVR >95%** (essentially curative) in 8–12 weeks; pan-genotypic combinations (sofosbuvir + velpatasvir) effective against all genotypes; (4) No vaccine available for HCV; (5) IFN-based therapy is no longer standard of care.

Key Point: HCV = RNA virus (NOT DNA). Genotype 1 most common in India. DAAs (sofosbuvir-based) achieve SVR >95% = functional cure. No HCV vaccine.

Why other options are wrong:

- Option A: HCV is an RNA virus, NOT DNA. IFN-based therapy is obsolete (replaced by DAAs).
- Option B: HCV genotype 1 (not 2) is most prevalent in India; genotype 3 is also common in India/Pakistan.
- Option C: DAA regimens achieve SVR >95% with genotype 1 — not less than 50%; this was the case with old IFN-based therapy.

Final Answer: HCV genotype 1 most common in India; sofosbuvir-based DAAs achieve SVR >95% $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q26

Q27.

Solution

Concept – Neonatal HSV-2 Infection: Neonatal herpes is acquired primarily at delivery (85%) through exposure to HSV in the birth canal; less commonly transplacental (5%) or postnatal (10%). **HSV-2** is responsible for 75% of neonatal herpes. Presentations: (1) Skin-eye-mouth (SEM) disease — most common (45%), best prognosis; (2) CNS disease — encephalitis, seizures; (3) **Disseminated disease** — liver, adrenal involvement + encephalitis; mortality >80% without treatment. Treatment: **intravenous acyclovir** 60 mg/kg/day in 3 divided doses for 14–21 days. **Without treatment, disseminated neonatal herpes mortality exceeds 80%**; with IV acyclovir, mortality drops to <30%.

Key Point: Neonatal HSV-2 (perinatal transmission) = disseminated disease with encephalitis + hepatitis; mortality >80% without IV acyclovir. Most dangerous neonatal viral infection.

Why other options are wrong:

- Option B: HSV-2 is transmitted perinatally (birth canal exposure), NOT via blood transfusion.
- Option C: Neonatal HSV-2 is NOT self-limiting; it is life-threatening and requires urgent IV acyclovir.
- Option D: The classic genital ulcer pattern is seen in adult primary HSV-2, not in neonates who present with disseminated disease or encephalitis.

Final Answer: Neonatal HSV-2 = disseminated disease with encephalitis; mortality >80% without IV acyclovir $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q27

Q28.

Solution

Concept – Adenovirus (Epidemic Keratoconjunctivitis, No Envelope): Adenoviruses are **non-enveloped, double-stranded DNA viruses** (icosahedral capsid). Adenovirus (serotypes 8, 19, 37) causes epidemic keratoconjunctivitis (EKC, "shipyard eye") — follicular conjunctivitis + keratitis; highly contagious through contaminated swimming pools, shared towels, ophthalmic equipment. Features: (1) Preauricular lymphadenopathy (distinguishes from bacterial conjunctivitis); (2) Pharyngoconjunctival fever (PCF) — serotypes 3, 7: fever + pharyngitis + conjunctivitis in summer camps/swimming pools; (3) Enteric adenovirus (40, 41): diarrhoea in children. Other manifestations: pneumonia (immunocompromised),

haemorrhagic cystitis (serotypes 11, 21 in transplant).

Key Point: Swimming pool + acute follicular conjunctivitis + preauricular lymphadenopathy + non-enveloped dsDNA virus = Adenovirus. The non-enveloped structure allows survival in chlorinated pools.

Why other options are wrong:

- Option A: Enterovirus 70 causes acute haemorrhagic conjunctivitis (different presentation — haemorrhagic, no keratitis, no swimming pool association).
- Option C: HSV-1 causes keratoconjunctivitis (dendritic ulcer on fluorescein staining) but not in outbreak form associated with swimming pools; enveloped virus.
- Option D: CMV causes retinitis in AIDS (not external conjunctivitis); enveloped virus.

Final Answer: Swimming pool outbreak + follicular conjunctivitis + preauricular lymphadenopathy + non-enveloped dsDNA virus = Adenovirus $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q28

Q29.

Solution

Concept – Parvovirus B19 (Fifth Disease, Aplastic Crisis, Hydrops Fetalis): Parvovirus B19 is a **single-stranded DNA virus** (non-enveloped, smallest DNA virus to infect humans). It targets erythroid progenitor cells (via P antigen/globoside receptor). Clinical manifestations: (1) **Erythema infectiosum (Fifth disease)** in children: "slapped-cheek" facial rash + lace-like reticular rash on trunk; (2) **Aplastic crisis** in patients with haemolytic anaemia (sickle-cell disease, thalassaemia, spherocytosis) — virus destroys RBC precursors $\rightarrow$ severe anaemia; (3) **Hydrops fetalis** — fetal infection causes severe anaemia, high-output cardiac failure, generalised oedema in the fetus; (4) Arthropathy in adults. No antiviral treatment; supportive care.

Key Point: Slapped-cheek rash in child + aplastic crisis in sickle-cell sibling + hydrops fetalis risk in pregnancy = Parvovirus B19. P antigen (globoside) is the receptor.

Why other options are wrong:

- Option A: Measles (rubeola) causes koplik spots + morbilliform rash (head-to-toe spread); cough, coryza, conjunctivitis (3Cs); no slapped-cheek pat-

tern.

- Option B: Rubella causes pinkish maculopapular rash (head-to-toe in 24 hours); suboccipital lymphadenopathy; different clinical picture; no aplastic crisis.
- Option D: HHV-6 causes roseola (exanthem subitum) — high fever 3–4 days then rose-pink macular rash as fever breaks; no slapped-cheek.

Final Answer: Slapped-cheek rash + aplastic crisis in sickle-cell + hydrops fetalis risk = Parvovirus B19 $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q29

Q30.

Solution

Concept – JC Virus and Progressive Multifocal Leukoencephalopathy (PML): JC virus (John Cunningham virus) is a **polyomavirus** (non-enveloped, dsDNA) that establishes latent infection in the kidneys, bone marrow, and B cells of 70–80% of adults. In severe immunosuppression (HIV/AIDS CD4 $<$100, haematological malignancy, natalizumab treatment for MS), JC virus reactivates and infects oligodendrocytes in the CNS $\rightarrow$ demyelination of white matter $\rightarrow$ **PML**. MRI: multiple non-enhancing (no blood-brain barrier disruption), non-space-occupying, asymmetric white matter lesions. CSF: JC virus PCR positive. No specific antiviral therapy; treatment is immune restoration (HAART for HIV).

Key Point: AIDS + CD4 $<$100 + non-enhancing white matter lesions (multifocal) + CSF polyomavirus PCR = JC virus = PML. Natalizumab (anti-VLA-4 for MS) is another important risk factor.

Why other options are wrong:

- Option A: CMV retinitis presents with visual loss, "pizza-pie" fundoscopic appearance; not white matter lesions on MRI; not a polyomavirus.
- Option B: CNS lymphoma (EBV-related in AIDS) presents as ring-enhancing lesion with mass effect; FDG-PET positive; different from non-enhancing PML lesions.
- Option C: HSV-1 encephalitis causes temporal lobe haemorrhagic lesions with enhancement; acute onset fever; different MRI pattern.

Final Answer: AIDS + non-enhancing multifocal white matter lesions + CSF polyomavirus PCR = JC virus = PML $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q30

Q31.

Solution

Concept – Yellow Fever Virus (17D Vaccine, Aedes, Hepatic Necrosis): Yellow fever is caused by Yellow fever virus (Flavivirus, positive-sense ssRNA, **Aedes aegypti** mosquito vector). Clinical: incubation 3–6 days; **Three phases**: (1) Infection phase — fever, headache, myalgia; (2) Remission — 24–48 hours; (3) Intoxication phase (in 15%) — jaundice, **black vomit** (haematemesis of digested blood), albuminuria, haemorrhage, liver failure, death. Pathology: midzonal hepatic necrosis with **Councilman bodies** (acidophilic bodies = apoptotic hepatocytes). **17D vaccine**: live attenuated; single dose; lifelong immunity; mandatory for travel to endemic areas; contraindicated in immunocompromised and infants $<$6 months.

Key Point: Africa/S. America travel + black vomit + jaundice + midzonal hepatic necrosis + Councilman bodies = Yellow fever. 17D = live attenuated vaccine, single dose, lifelong protection.

Why other options are wrong:

- Option B: Dengue (Flavivirus) causes dengue haemorrhagic fever but NO hepatic midzonal necrosis; "black vomit" is not typical; different mosquito season.
- Option C: Hepatitis E causes acute hepatitis (especially in pregnancy); fecooral transmission; no black vomit; HEV 239 vaccine available in China only.
- Option D: Leptospirosis (Weil's disease) causes jaundice + renal failure + uveitis; bacterial spirochaete; not a virus with Councilman bodies.

Final Answer: Africa travel + black vomit + midzonal necrosis + Councilman bodies = Yellow fever; 17D live attenuated vaccine $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q31

Q32.

Solution

Concept – West Nile Virus (Culex Mosquito, Neuroinvasive Disease): West Nile virus (WNV) is a Flavivirus (positive-sense ssRNA) transmitted by **Culex** mosquitoes. The primary vertebrate hosts are birds; humans are dead-end hosts. **Sentinel surveillance**: dead crow mortality is an epidemiological indicator of WNV activity. Spectrum of disease: (1) Asymptomatic (80%); (2) West Nile fever (20%) — mild febrile illness; (3) **West Nile neuroinvasive disease** ($<$1%) — meningitis, encephalitis, acute flaccid paralysis; more severe in elderly and im-

munocompromised. No specific antiviral; supportive care. No licensed human vaccine. CSF: lymphocytic pleocytosis, elevated protein, normal glucose.

Key Point: Late summer + *Culex* mosquito + dead crows (bird mortality) + meningoencephalitis in elderly/immunocompromised + Flavivirus IgM = West Nile virus.

Why other options are wrong:

- Option A: Chikungunya (alphavirus) causes fever + severe polyarthralgia/arthritis; *Aedes* vector; rarely causes encephalitis; no bird association.
- Option C: Japanese encephalitis virus (JEV) is a Flavivirus (*Culex* vector) but is endemic in Asia; pigs/birds as reservoir; no dead crow sentinel in the Americas.
- Option D: St. Louis encephalitis (SLEV) is also a Flavivirus with *Culex* vector but less common than WNV since its emergence; no bird mortality sentinel.

Final Answer: Culex mosquito + dead crows + meningoencephalitis in summer = West Nile virus $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q32

Q33.

Solution

Concept – Plasmodium malariae (Quartan Fever, Nephrotic Syndrome): *P. malariae* has a **72-hour (quartan) erythrocytic cycle** — fever on days 1 and 4 (every third day). Distinctive features: (1) **No hypnozoites** in liver — no true relapses (unlike *P. vivax* and *P. ovale*); however, low-grade parasitaemia can persist for decades ("recrudescence"); (2) **Nephrotic syndrome** in children — immune complex deposition (malarial antigen–antibody complexes) in glomeruli; causes membranoproliferative pattern; (3) Band trophozoite (band-form across RBC) and rosette schizont on blood smear; (4) Invades older RBCs (normal size); (5) No Schüffner's dots. Treatment: chloroquine (for non-falciparum malaria).

Key Point: Quartan (72-hour) fever + nephrotic syndrome in child + no hypnozoites/relapse + band trophozoite = *Plasmodium malariae*.

Why other options are wrong:

- Option A: *P. vivax* causes tertian (48-hour) fever; has hypnozoites (relapses); Schüffner's dots; enlarged RBCs; no nephrotic syndrome.
- Option B: *P. falciparum* causes malignant tertian fever (irregular); cerebral

malaria; falciparum does NOT cause nephrotic syndrome; no band trophozoites.
- Option D: *P. ovale* causes tertian fever; has hypnozoites; fimbriated (oval) RBCs with Schüffner's dots; rarest species.

Final Answer: Quartan (72-hour) fever + nephrotic syndrome + no relapse (no hypnozoites) = *Plasmodium malariae* $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q33

Q34.

Solution

Concept – Toxoplasma gondii (Cat = Definitive Host, Reactivation in AIDS): *Toxoplasma gondii* life cycle: **Cats are the ONLY definitive host** (sexual reproduction occurs in cat intestinal epithelium); oocysts shed in cat faeces. Intermediate hosts (humans, sheep, pigs, rodents): bradyzoites in tissue cysts (muscle, brain). Transmission: ingestion of oocysts (from cat faeces/contaminated soil), undercooked meat (bradyzoites), or transplacental. In AIDS (CD4 $<$100): tissue cysts (bradyzoites) reactivate $\rightarrow$ **cerebral toxoplasmosis** (multiple ring-enhancing lesions in basal ganglia/corticomedullary junction). Treatment: pyrimethamine + sulfadiazine + folinic acid (leucovorin). Prophylaxis when CD4 $<$100: TMP-SMX.

Key Point: Cat = definitive host (sexual cycle). Human = intermediate host (bradyzoites in cysts). AIDS + ring-enhancing brain lesions + cat ownership = cerebral toxoplasmosis.

Why other options are wrong:

- Option A: Dogs are NOT the definitive host of *Toxoplasma*. Only cats (Felidae) support the sexual cycle.
- Option B: Oocysts are shed in **cat** faeces, not human urine. Human infection is from environmental oocysts or tissue cysts in meat.
- Option C: Primary infection in immunocompetent adults is asymptomatic or causes mild lymphadenopathy (90% asymptomatic). Severe disease occurs in immunocompromised or congenital infection.

Final Answer: Cat = definitive host; bradyzoites reactivate in AIDS $\rightarrow$ cerebral toxoplasmosis (ring-enhancing lesions) $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q34

Q35.

Solution

Concept – Wuchereria bancrofti (Lymphatic Filariasis, DEC): *W. bancrofti* causes lymphatic filariasis (elephantiasis), transmitted by **Culex quinquefasciatus** in urban areas and India. Key features: (1) **Nocturnal periodicity** of microfilariae — microfilariae appear in peripheral blood at night (migrating to lung capillaries during the day), coinciding with *Culex* nocturnal feeding habit; (2) Sheathed microfilariae; (3) Lymphatic obstruction $\rightarrow$ lymphoedema (elephantiasis of leg, scrotum, breast); (4) Tropical pulmonary eosinophilia (TPE) in some patients; (5) Blood smear collected at midnight. Treatment: **diethylcarbamazine (DEC)** (macrofilaricidal + microfilaricidal) or ivermectin + albendazole (MDA programme).

Key Point: Lymphoedema + sheathed microfilariae with nocturnal periodicity + *Culex* vector = *Wuchereria bancrofti*. DEC is the drug of choice.

Why other options are wrong:

- Option B: *Brugia timori* also causes lymphatic filariasis but is restricted to lesser Sunda islands of Indonesia; DEC is the treatment (not ivermectin alone).
- Option C: *Loa loa* causes loiasis (African eye worm) with subconjunctival migration; *Chrysops* deerfly vector; DEC is used (not mebendazole).
- Option D: *Mansonella perstans* is non-pathogenic; treatment is mebendazole or doxycycline; not associated with elephantiasis.

Final Answer: Elephantiasis + nocturnal sheathed microfilariae + *Culex* vector = *Wuchereria bancrofti*; treat with DEC $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q35

Q36.

Solution

Concept – Schistosoma mansoni (Lateral Spine, Portal Hypertension): *Schistosoma* spp. are blood flukes (trematodes) with a complex lifecycle involving freshwater snails (intermediate hosts). Key distinguishing features by species: ***S. mansoni***: lateral-spined eggs, inhabits inferior mesenteric veins, causes portal hypertension (Symmers' pipestem fibrosis), associated with colon cancer; ***S. haematobium***: terminal-spined eggs, inhabits vesical plexus, causes haematuria and bladder cancer (squamous cell); ***S. japonicum***: very small lateral spine, eggs found in clusters, hepatic fibrosis, brain involvement. **Katayama fever**: acute

phase (weeks after cercarial penetration) — fever, urticaria, eosinophilia, hepatosplenomegaly (hypersensitivity to migrating schistosomula). Treatment: **praziquantel**.

Key Point: Freshwater exposure in Egypt/Africa + Katayama fever (acute) + lateral-spined eggs in stool + portal hypertension (chronic) = *Schistosoma mansoni*. Praziquantel is curative.

Why other options are wrong:

- Option A: *S. haematobium* has terminal-spined eggs in urine (not stool); causes bladder carcinoma, not portal hypertension.
- Option C: *S. japonicum* also causes hepatic fibrosis but has small lateral spine; eggs found in clusters in stool; endemic in East Asia (not Egypt); no significant Katayama fever typically.
- Option D: *S. intercalatum* is rare, found in Central Africa; terminal-spined eggs in stool; causes rectal pathology.

Final Answer: Freshwater Egypt + Katayama fever + lateral-spined eggs + portal hypertension = *Schistosoma mansoni* $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q36

Q37.

Solution

Concept – Echinococcus granulosus (Hydatid Disease, Dog-Sheep Lifecycle): *E. granulosus* (cestode/tapeworm) causes cystic echinococcosis (hydatid disease). **Lifecycle**: dogs (definitive host, adult tapeworm in small intestine) $\rightarrow$ egg shedding in faeces $\rightarrow$ sheep/cattle/humans (intermediate hosts) ingest eggs $\rightarrow$ hexacanth embryo released in duodenum $\rightarrow$ penetrates portal circulation $\rightarrow$ lodges in liver (most common, 60–70%), lung (20–30%), or other organs $\rightarrow$ develops into hydatid cyst. Cyst structure: **pericyst** (host fibrous layer) + **ectocyst** (laminated, outer parasite layer) + **germinal layer** (inner, produces protoscolices, brood capsules, daughter cysts). **Casoni test**: intradermal antigen injection (old, non-specific). Treatment: PAIR (Puncture, Aspiration, Injection of hypertonic saline, Re-aspiration) + albendazole, or surgery.

Key Point: Shepherd + liver cyst with daughter cysts + calcified wall + Casoni positive + dog-sheep lifecycle = hydatid disease (*E. granulosus*). Never aspirate without albendazole cover (risk of anaphylaxis + peritoneal seeding).

Why other options are wrong:

- Option A: Amoebic liver abscess is unilocular, no daughter cysts, no calcification; single cavity with "anchovy paste" pus; responds to metronidazole.
- Option B: Simple hepatic cysts are asymptomatic, no internal structures, no calcification, Casoni-negative.
- Option D: Polycystic liver disease is hereditary (PRKCSH or SEC63 mutations); multiple small cysts throughout both lobes; no daughter cysts or Casoni test.

Final Answer: Shepherd + liver cyst with daughter cysts + Casoni positive + dog-sheep lifecycle = hydatid disease (*E. granulosus*) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q37

Q38.

Solution

Concept – Cryptosporidium parvum (AIDS, Acid-Fast Oocysts): *Cryptosporidium parvum* is an intracellular apicomplexan parasite that infects intestinal epithelial cells (brush border). Key features: (1) **Acid-fast oocysts** (4–6 μm) on modified ZN stain (rose-pink against blue background); (2) Transmission: fecooral, waterborne (resistant to chlorination), person-to-person; (3) In immunocompetent: self-limiting watery diarrhoea (1–2 weeks); (4) In AIDS (CD4 $<$100): **profuse cholera-like watery diarrhoea** ($>$3 litres/day), chronic, wasting, can involve biliary tract (AIDS cholangiopathy); (5) **No reliably effective antiparasitic**: nitazoxanide helps in immunocompetent; no treatment in AIDS except **HAART to restore CD4** count.

Key Point: AIDS + CD4 $<$100 + profuse watery diarrhoea + **acid-fast oocysts** (4–6 μm) = *Cryptosporidium parvum*. Restore immunity with HAART = only effective strategy.

Why other options are wrong:

- Option A: *Giardia lamblia* causes malabsorptive steatorrhoea (fatty, foul-smelling stools), not profuse watery diarrhoea; cysts and trophozoites on stool exam; NOT acid-fast; treated with metronidazole.
- Option B: *E. histolytica* causes dysentery (bloody) or liver abscess; trophozoites with ingested RBCs; not acid-fast; treated with metronidazole.
- Option C: *Isospora belli* (now *Cystoisospora belli*) also causes AIDS diarrhoea; larger oocysts (20–33 μm); acid-fast; but responds to TMP-SMX (effective treatment available).

Final Answer: AIDS + profuse watery diarrhoea + acid-fast tiny oocysts (4–6

μm) + no effective antiparasitic = *Cryptosporidium parvum* $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q38

Q39.

Solution

Concept – Aspergillus fumigatus (Invasive Aspergillosis, Halo Sign, Galactomannan): *A. fumigatus* is the commonest cause of **invasive aspergillosis** (IA) in neutropenic patients (<500 neutrophils/mm^3). Pathology: **angioinvasion** — hyphae invade blood vessel walls $\rightarrow$ thrombosis and infarction $\rightarrow$ haemoptysis. Radiology: **"halo sign"** on CT chest (ground-glass opacity surrounding a nodule = haemorrhagic infarction surrounding the fungal lesion); later: "air crescent sign" as neutrophils recover. Diagnostics: (1) **Serum galactomannan** (Aspergillus cell wall antigen) — sensitivity 70–80% in IA; (2) CT-guided biopsy: septate hyphae with **45° branching**. Treatment: **voriconazole** (first-line); alternatives: isavuconazole, liposomal amphotericin B.

Key Point: Neutropenic patient + halo sign on CT + positive galactomannan + septate hyphae branching at 45° = *Aspergillus fumigatus*. Voriconazole is first-line treatment.

Why other options are wrong:

- Option B: *Mucor/Rhizopus* also cause invasive fungal infection in neutropenic/diabetic hosts; but hyphae are **broad, ribbon-like, aseptate** with 90° branching; galactomannan is NEGATIVE; halo sign is less common; occurs more in DKA.
- Option C: *Candida* causes invasive candidiasis (bloodstream, hepatosplenic); hyphae not seen on CT; beta-D-glucan positive; galactomannan negative.
- Option D: *Cryptococcus neoformans* (encapsulated yeast) causes meningitis in AIDS; not angioinvasive; India ink positive; cryptococcal antigen test (not galactomannan).

Final Answer: Neutropenic + halo sign CT + galactomannan positive + 45° septate hyphae = *Aspergillus fumigatus* $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q39

Q40.

Solution

Concept – Mucor/Rhizopus (Rhinocerebral Mucormycosis, Diabetic Ketoacidosis): Mucormycosis (formerly zygomycosis) is caused by *Mucor, Rhizopus, Lichtheimia* (Absidia) spp. (order Mucorales). Key features: (1) **Risk factors**: uncontrolled diabetes (especially DKA), haematological malignancy with neutropenia, deferoxamine therapy, solid organ/stem cell transplant, COVID-19 with corticosteroid use; (2) **Rhinocerebral form** (most common in DKA): starts in nasal turbinates $\rightarrow$ spreads to palate, orbit, brain; **black eschar** (necrosis) on palate/nasal mucosa; (3) Histology: **broad, ribbon-like, aseptate (non-septate) hyphae with near 90° branching**; (4) Angioinvasion $\rightarrow$ haematogenous dissemination; (5) Treatment: urgent surgical debridement + **liposomal amphotericin B**; voriconazole has NO activity against Mucorales.

Key Point: Uncontrolled DKA + periorbital swelling + black nasal eschar + broad aseptate hyphae (90° branching) = rhinocerebral mucormycosis. High-dose liposomal amphotericin B + urgent surgery. Voriconazole must NOT be used (no activity against Mucorales).

Why other options are wrong:

- Option A: Invasive aspergillosis occurs in neutropenic patients (not DKA); septate hyphae with 45° branching; halo sign on CT chest; galactomannan positive; voriconazole is first-line.
- Option C: Cryptococcal sinusitis is extremely rare; *Cryptococcus* is an encapsulated yeast (not aseptate hyphae); occurs in AIDS (not DKA); India ink/cryptococcal antigen test.
- Option D: *Fusarium* is a rare mould infection (septate hyphae similar to *Aspergillus*); causes disseminated infection in neutropenic patients; no black eschar; not associated with DKA primarily.

Final Answer: DKA + black nasal eschar + aseptate hyphae (90° branching) = rhinocerebral mucormycosis (*Mucor/Rhizopus*) $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q40

Answer Key – FMGE Microbiology Sample Paper 3

1	C	2	D	3	A	4	B	5	C
6	D	7	A	8	B	9	C	10	D
11	A	12	B	13	C	14	D	15	A
16	B	17	C	18	D	19	A	20	B
21	C	22	D	23	A	24	B	25	C
26	D	27	A	28	B	29	C	30	D
31	A	32	B	33	C	34	D	35	A
36	B	37	C	38	D	39	A	40	B