FMGE Microbiology Sample Paper – 2

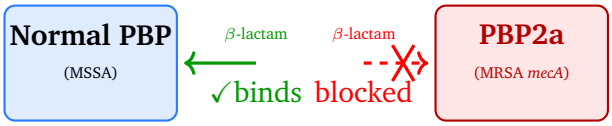
Duration: 60 Minutes **Maximum Marks: 40**

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 55-year-old diabetic patient is admitted with a non-healing wound infection that fails to respond to oxacillin therapy. Blood cultures grow gram-positive cocci in clusters. PCR of the isolate detects the *mecA* gene, which encodes a modified penicillin-binding protein (PBP2a) with low affinity for all beta-lactam antibiotics. The diagram below illustrates drug binding to normal versus altered PBP.



Which of the following best describes this organism?

(A) Methicillin-sensitive *Staphylococcus aureus* (MSSA)

(B) Methicillin-resistant *Staphylococcus aureus* (MRSA)

(C) Extended-spectrum beta-lactamase-producing *E. coli*

(D) Vancomycin-resistant *Enterococcus* (VRE)

Q2. A 68-year-old man with COPD presents with high fever, productive cough with rust-coloured sputum, and right lower lobe consolidation on chest X-ray. Gram stain of sputum reveals lancet-shaped gram-positive diplococci. When the sputum is mixed with type-specific antiserum and methylene blue, the capsule of the organisms appears to swell visibly under the microscope. Which of the following best describes the diagnostic test performed?

(A) Coagulase test

(B) CAMP test

(C) Quellung (Neufeld) reaction

(D) Quellung-negative capsule detection by India ink

Q3. A 4-year-old child in a rural area presents with bloody mucoid diarrhoea, severe abdominal cramps, and high fever. Stool culture on MacConkey agar shows non-lactose-fermenting colonies. Serotyping identifies the isolate as serotype 1 of a *Shigella* species. The organism's major virulence factor cleaves the 28S rRNA of the 60S ribosomal subunit by its RNA N-glycosidase activity, halting protein synthesis and causing endothelial and epithelial cell death. Which organism and toxin best describe this case?

(A) *Shigella sonnei* — endotoxin

(B) *Clostridium perfringens* — alpha toxin

(C) *Shigella flexneri* — Shiga-like toxin (Stx2)

(D) *Shigella dysenteriae* — Shiga toxin (Stx1)

Q4. A 35-year-old man from a leprosy-endemic region presents with diffuse skin infiltration, loss of eyebrows (madarosis), and bilateral symmetrical anaesthetic patches. Slit-skin smear stained with Ziehl-Neelsen shows numerous acid-fast bacilli within macrophages (foamy Virchow cells). Lepromin test is negative. Which of the following best describes the host immunological profile in this patient compared to the tuberculoid form?

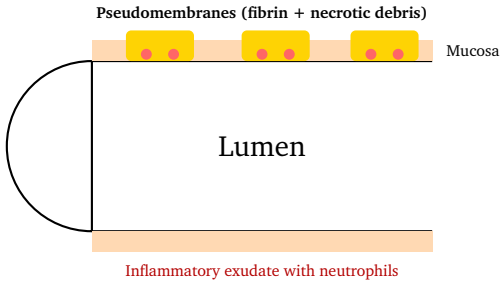
(A) Lepromatous: Th2 response, low CMI, high bacillary load (multibacillary); Tuberculoid: Th1 response, high CMI, paucibacillary

(B) Lepromatous: Th1 response, high CMI, paucibacillary; Tuberculoid: Th2 response, low CMI

(C) Both forms show equal cellular immunity but differ only in bacillary load

(D) Lepromatous leprosy is caused by a different species of *Mycobacterium* than tuberculoid leprosy

Q5. A 72-year-old woman on long-term clindamycin for a dental infection develops profuse watery diarrhoea with fever and leucocytosis. Colonoscopy reveals yellowish plaques covering the colonic mucosa. The diagram below shows a cross-section of the affected bowel. Stool enzyme immunoassay is positive for toxin A and toxin B. Which organism is responsible and what is the mechanism of toxin B?



(A) *Staphylococcus aureus* — TSST-1 superantigen

(B) *Clostridioides difficile* — Toxin B (cytotoxin) inactivates Rho GTPases, disrupting actin cytoskeleton

(C) *Clostridium perfringens* — alpha toxin (lecithinase)

(D) *Bacillus cereus* — cereulide emetic toxin

Q6. A 58-year-old immunosuppressed patient presents with high-grade fever, dry cough, confusion, and hyponatraemia following a stay in a hotel with a large air-conditioning system. Chest X-ray shows patchy consolidation. Sputum Gram stain shows few organisms despite heavy growth on charcoal yeast extract agar supplemented with L-cysteine and iron (L-BCYE).

Urinary antigen test is positive. Which of the following best describes the pathogen?

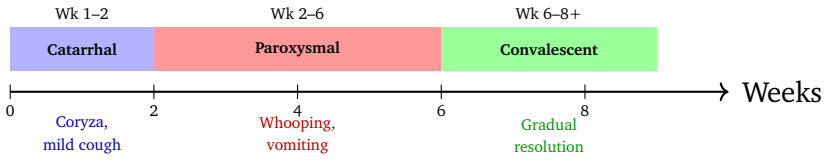
(A) *Mycoplasma pneumoniae* — no cell wall, Eaton agar

(B) *Chlamydophila pneumoniae* — obligate intracellular, MIF test

(C) *Legionella pneumophila* — intracellular in alveolar macrophages, L-BCYE, silver stain

(D) *Coxiella burnetii* — Q fever, no arthropod vector

Q7. A 6-month-old unimmunised infant presents with 3 weeks of severe paroxysmal coughing followed by an inspiratory whoop, post-tussive vomiting, and lymphocytosis. The attending physician describes three distinct clinical stages. The following timeline represents the clinical course of the disease.



Which of the following correctly identifies the causative organism and its virulence factor responsible for lymphocytosis?

(A) *Bordetella parapertussis* — filamentous haemagglutinin

(B) *Haemophilus influenzae* — IgA protease

(C) *Moraxella catarrhalis* — beta-lactamase

(D) *Bordetella pertussis* — pertussis toxin (causes lymphocytosis by blocking lymphocyte recirculation)

Q8. A 45-year-old man presents with recurrent epigastric pain that is relieved by meals and antacids. Upper GI endoscopy shows a duodenal ulcer. Rapid urease test (CLO test) of the biopsy sample turns the medium pink/magenta within 1 hour. The patient is a non-smoker with no NSAID use. Which of the following best describes the causative organism and the gold-standard non-invasive test for confirming eradication?

(A) *Helicobacter pylori* — Urea breath test (^{13}C or ^{14}C-urea) is the gold standard for confirming eradication

(B) *Helicobacter pylori* — Serology (IgG) is the best test for confirming eradication

(C) *Campylobacter jejuni* — Stool antigen test

(D) *Helicobacter pylori* — Rapid urease test is the gold standard for eradication confirmation

Q9. A 24-year-old sexually active woman presents with mucopurulent cervical discharge and dysuria. Her male partner has a urethral discharge. A separate patient from the same clinic has bilateral inguinal lymphadenopathy (buboes) with a groove sign. A third patient has bilateral follicular conjunctivitis with corneal pannus leading to blindness. All three are infected by the same genus. Which of the following correctly matches the *Chlamydia trachomatis* serovars to their associated diseases?

(A) Serovars A–C: urogenital; D–K: trachoma; L1–L3: LGV

(B) Serovars A–C: trachoma (blindness); D–K: urogenital; L1–L3: lymphogranuloma venereum (LGV)

(C) Serovars A–C: LGV; D–K: trachoma; L1–L3: urogenital

(D) Serovars A–K: all cause the same urogenital disease; L1–L3: trachoma

Q10. A 30-year-old hiker returns from the Appalachian Mountains with fever, severe headache, and a rash that began on the wrists and ankles and spread centripetally to the trunk. The rash involves the palms and soles. Laboratory testing shows thrombocytopaenia and elevated transaminases. The Weil-Felix test is positive with OX-19 and OX-2 *Proteus* agglutination. Which of the following correctly identifies the pathogen and its vector?

(A) *Rickettsia typhi* — *Xenopsylla cheopis* rat flea

(B) *Borrelia burgdorferi* — *Ixodes* tick

(C) *Rickettsia rickettsii* — *Dermacentor* tick (Rocky Mountain spotted fever)

(D) *Rickettsia prowazekii* — *Pediculus* body louse

Q11. A 28-year-old farmer presents with abrupt onset fever, severe myalgia (especially calf muscles), conjunctival suffusion, jaundice, oliguria, and petechial haemorrhages. He recalls wading through floodwaters 10 days earlier. Serum creatinine is markedly elevated. Darkfield microscopy of urine reveals motile, hooked, thin spirochaetes. Which of the following best describes this condition?

(A) *Borrelia recurrentis* — relapsing fever

(B) *Treponema pallidum* — congenital syphilis

(C) *Leptospira biflexa* — saprophytic, non-pathogenic form

(D) *Leptospira interrogans* — Weil's disease (icterohaemorrhagic leptospirosis): jaundice + acute kidney injury + haemorrhage

Q12. A 25-year-old man presents with a painless genital ulcer that healed spontaneously, followed 6 weeks later by generalised maculopapular rash involving the palms and soles, condylomata lata, and oral mucous patches. Serological testing is performed. VDRL titre is 1:128. FTA-ABS is reactive. Which of the following correctly interprets the serological tests and their roles in syphilis diagnosis?

(A) VDRL is a non-treponemal test used for screening and monitoring treatment response; FTA-ABS is a treponemal test used for confirmation and remains positive for life

(B) VDRL is a treponemal test; FTA-ABS is a non-treponemal test used for screening

(C) Both VDRL and FTA-ABS are non-treponemal tests that become negative after treatment

(D) FTA-ABS is used for screening; VDRL is used for confirmation

Q13. A 40-year-old man from a rural area presents with sudden-onset high fever, chills, and a markedly swollen, extremely tender lymph node in the right inguinal region (bubo). He reports numerous dead rats near

his home. Blood culture on MacConkey agar at 28°C grows colonies with a characteristic "safety pin" bipolar staining on Wayson stain. The F1 antigen of the causative organism inhibits phagocytosis. Which of the following correctly identifies the organism and its vector?

(A) *Francisella tularensis* — *Dermacentor* tick

(B) *Yersinia pestis* — *Xenopsylla cheopis* (Oriental rat flea)

(C) *Brucella melitensis* — unpasteurised goat's milk

(D) *Bartonella henselae* — *Ctenocephalides felis* cat flea

Q14. A 38-year-old man presents with a slowly progressive, painless jaw swelling following dental extraction 2 months ago. The swelling is indurated with multiple draining sinuses producing yellowish granular material. Microscopy of the discharge shows lobulated granules surrounded by a rosette of Gram-positive, club-shaped peripheral filaments. Culture under anaerobic conditions on blood agar produces "molar tooth" colonies. Which of the following best identifies the organism?

(A) *Nocardia asteroides* — aerobic, partially acid-fast, pulmonary

(B) *Streptomyces griseus* — aerobic soil organism, streptomycin producer

(C) *Actinomyces israelii* — anaerobic gram-positive, sulfur granules, cervicofacial actinomycosis

(D) *Aspergillus fumigatus* — fungal, septate hyphae with V-shaped branching

Q15. A 52-year-old man with blood group A receives a transfusion of blood group B blood due to a clerical error. Within 30 minutes he develops fever, rigors, hypotension, haemoglobinuria, and flank pain. The mechanism involves pre-formed IgM and IgG antibodies against ABO antigens on donor erythrocytes activating complement (C3b opsonisation and MAC lysis) and ADCC. Which type of hypersensitivity reaction is responsible?

(A) Type I (IgE-mediated) — mast cell degranulation

(B) Type III — immune complex deposition with complement activation

(C) Type IV — delayed type, T-cell mediated

(D) Type II — antibody-mediated cytotoxicity (complement activation + ADCC against cell-surface antigens)

Q16. A 22-year-old woman with SLE presents with arthralgia, haematuria, and low serum complement (C3, C4). Renal biopsy shows granular ("lumpy-bumpy") immune complex deposits along the glomerular basement membrane. Her serum contains anti-dsDNA antibodies. Another patient, given horse antivenom 10 days earlier, develops fever, urticaria, and arthralgia. Both conditions share the same pathogenic mechanism. Which of the following best describes this mechanism?

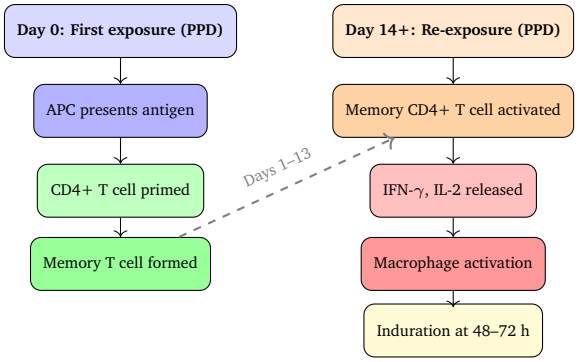
(A) Soluble antigen-antibody immune complex deposition in vessel walls and tissue, complement activation (C3a, C5a), and neutrophil-mediated tissue injury (Type III hypersensitivity)

(B) Direct antibody binding to cell-surface antigens causing complement-mediated lysis

(C) IgE-mediated mast cell degranulation releasing histamine

(D) Sensitised CD4+ T cells releasing interferon-gamma and activating macrophages

Q17. A 35-year-old healthcare worker undergoes a Mantoux tuberculin skin test. Purified protein derivative (PPD) is injected intradermally. At 48–72 hours, she develops a 15 mm area of induration. The diagram below shows the mechanism of Type IV delayed-type hypersensitivity (DTH).



Which of the following correctly identifies this reaction and the effector cells responsible?

(A) Type I hypersensitivity — IgE-mediated mast cell activation

(B) Type IV (delayed-type) hypersensitivity — sensitised CD4+ Th1 cells activate macrophages via IFN-γ; no antibody involved

(C) Type II hypersensitivity — complement-dependent cytotoxicity

(D) Type III hypersensitivity — Arthus reaction with immune complex deposition

Q18. A newborn presents with hypocalcaemia causing neonatal tetany, an interrupted aortic arch (conotruncal heart defect), and absent thymic shadow on chest X-ray. Immunological workup reveals severely reduced T-cell numbers, normal or elevated B-cell counts, and very low serum IgG due to absent T-cell help. The condition results from failure of development of the 3rd and 4th pharyngeal pouches. Chromosomal microarray detects a deletion at 22q11.2. Which of the following best describes the immunological defect?

(A) Absent B cells with normal T cells (Bruton's agammaglobulinemia)

(B) Absent T and B cells (SCID)

(C) DiGeorge syndrome — thymic aplasia causing absent T cells; B cells normal but dysfunctional due to lack of T-cell help

(D) Hyper-IgM syndrome — absent class switching due to CD40L deficiency

Q19. A 6-month-old boy presents with recurrent bacterial infections (pneumonia, otitis media, meningitis) starting after maternal IgG wanes. Physical examination reveals absent tonsils and lymph nodes. Immunological evaluation shows absent circulating B lymphocytes ($CD19^{-}$) with all immunoglobulin classes severely reduced. T-cell number and function are normal. Family history reveals two maternal uncles with a similar condition. Genetic testing reveals a mutation in a tyrosine kinase essential for B-cell maturation. Which of the following best describes this condition?

(A) Common Variable Immunodeficiency (CVID) — normal B cells, reduced immunoglobulins, X-linked

(B) Hyper-IgM syndrome — elevated IgM, absent IgG/IgA, CD40L defect

(C) IgA deficiency — selective absence of IgA, normal other immunoglobulins

(D) Bruton's X-linked agammaglobulinemia — BTK mutation, absent pre-B to B-cell maturation, no circulating B cells

Q20. A 4-month-old girl presents with failure to thrive, recurrent oral candidiasis, *Pneumocystis jirovecii* pneumonia, and persistent diarrhoea. Her blood counts show marked lymphopaenia. Flow cytometry reveals absent CD3+ T cells, absent B cells, and absent NK cells. Metabolic workup reveals accumulation of toxic deoxyadenosine metabolites damaging lymphocyte precursors. Enzyme replacement therapy with PEG-ADA is initiated while awaiting bone marrow transplantation. Which of the following correctly identifies this condition?

(A) SCID due to adenosine deaminase (ADA) deficiency — autosomal recessive; toxic deoxyadenosine metabolites destroy lymphoid precursors, causing $T^{-}B^{-}NK^{-}$ phenotype

(B) DiGeorge syndrome — 22q11 deletion, absent T cells, normal B cells

(C) Bruton's agammaglobulinemia — absent B cells, normal T and NK cells

(D) Wiskott-Aldrich syndrome — thrombocytopaenia, eczema, immunodeficiency

Q21. A medical student is studying mucosal immunity and reviews the immunoglobulin isotype that is most abundant in secretions such as saliva, tears, breast milk, and intestinal fluid. This isotype is synthesised as a dimer by plasma cells in the lamina propria, acquires a secretory component from epithelial cells during transcytosis across the mucosa, and provides the first line of defence against pathogens at mucosal surfaces. Which immunoglobulin isotype is described, and what is the role of the secretory component?

(A) IgG — Fc-mediated transplacental transfer, most abundant in serum

(B) IgA — secretory component (poly-Ig receptor remnant) protects the dimer from proteolytic degradation in mucosal secretions

(C) IgM — pentameric, first antibody in primary response

(D) IgE — mediates type I hypersensitivity and anti-parasite responses

Q22. During a pathology lecture, the professor discusses how phagocytes and NK cells use surface receptors for the Fc portion of IgG to enhance killing. Macrophages express a high-affinity receptor (K_d $\sim 10^{-9}$ M) that binds monomeric IgG even before antigen stimulation, facilitating phagocytosis and ADCC. NK cells and neutrophils express a lower-affinity receptor (K_d $\sim 10^{-7}$ M) activated mainly by IgG-coated targets, triggering ADCC. Which of the following correctly identifies these receptors?

(A) Macrophages: FcεRI (CD89); NK cells: FcαRI (CD16)

(B) Macrophages: FcγRII (CD32); NK cells: FcγRI (CD64) — high affinity

(C) Macrophages: FcγRI (CD64) — high affinity; NK cells and neutrophils: FcγRIII (CD16) — lower affinity, mediates ADCC

(D) Both macrophages and NK cells express identical FcγRIII (CD16)

Q23. A 65-year-old diabetic patient presents to the ICU with fever (39.8°C), hypotension (BP 80/50), tachycardia, and confusion after a urinary tract procedure. Blood cultures grow gram-negative rods. Serum levels of TNF-α, IL-1, and IL-6 are markedly elevated. Despite antibiotics, he deteriorates with DIC, ARDS, and multi-organ failure. LPS from the gram-negative cell wall has triggered massive macrophage activation. Which of the following best describes the pathogenesis of the multi-organ failure?

(A) IgE-mediated mast cell degranulation causing anaphylaxis

(B) Type II hypersensitivity: antibody against LPS causing complement lysis

(C) Toll-like receptor blockade preventing macrophage activation

(D) Cytokine storm: LPS binds CD14/TLR-4 on macrophages $\rightarrow$ excessive TNF-α, IL-1, IL-6 release $\rightarrow$ vasodilation, DIC, ARDS, multi-organ failure

Q24. An immunologist is studying how B cells in a germinal centre switch from producing IgM to other isotypes. She finds that cytokines from Th2 cells are essential. She notes that IL-4 drives switching to IgE (responsible for allergy and anti-parasite responses), IL-5 promotes IgA switching (with TGF-β at mucosal sites), and IFN-γ from Th1 cells drives switching to IgG subclasses. CD40L on T cells engaging CD40 on B cells provides the co-stimulatory signal required for class switch recombination. Which of the following correctly describes IL-4's primary role in class switching?

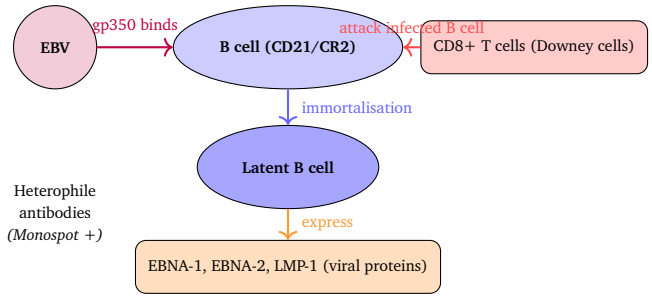
(A) IL-4 drives class switching to IgE (and IgG4 in humans), promoting allergic and anti-parasitic responses

(B) IL-4 promotes switching to IgG3 for opsonisation of bacteria

(C) TGF-β drives IgE switching; IL-4 drives IgA switching

(D) IFN-γ is the primary driver of IgE class switching

Q25. A 19-year-old university student presents with fever, severe sore throat, bilateral cervical lymphadenopathy, and hepatosplenomegaly. Blood film shows atypical lymphocytes (Downey cells). Monospot (Paul-Bunnell) test is positive. The diagram below illustrates the infection cycle of the causative virus.



Which of the following correctly identifies the virus and its cellular receptor?

(A) Cytomegalovirus (CMV) — binds MHC class I on all nucleated cells

(B) Epstein-Barr virus (EBV) — binds CD21 (complement receptor 2, CR2) on B lymphocytes

(C) Herpes simplex virus type 1 — binds herpesvirus entry mediator (HVEM) on epithelial cells

(D) Human herpesvirus 6 (HHV-6) — binds CD46 on T cells

Q26. A 35-year-old man with HIV infection and a CD4 count of 35 cells/μL presents with progressive painless loss of vision starting in the peripheral visual field. Ophthalmoscopy reveals a "pizza pie" or "tomato and cheese" pattern of haemorrhages and exudates along the retinal vessels. A tissue biopsy from a different patient with CMV pneumonitis shows large cells containing prominent intranuclear inclusions surrounded by a clear halo and smaller cytoplasmic inclusions, resembling an owl's eye. Which of the following best identifies the organism and its characteristic histological finding?

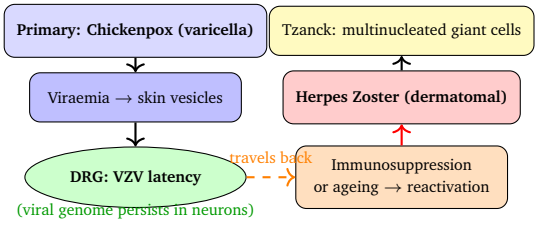
(A) *Toxoplasma gondii* — bradyzoites in tissue cysts

(B) *Herpes simplex virus* — Cowdry type A inclusions without cytoplasmic changes

(C) Cytomegalovirus (CMV) — owl eye intranuclear inclusions; CMV retinitis occurs in AIDS when CD4 count $<$50 cells/μL

(D) *Cryptococcus neoformans* — India ink capsule, no inclusion bodies

Q27. A 70-year-old man on long-term prednisolone presents with a painful vesicular rash distributed in a unilateral dermatomal pattern along the T5 thoracic dermatome. A Tzanck smear of a vesicle scraping shows multinucleated giant cells with intranuclear inclusions. The diagram below shows the pathophysiology of the causative virus.



Which of the following correctly describes the pathophysiology and diagnostic finding?

(A) Herpes simplex virus type 2 — latent in sacral ganglia; Tzanck smear not useful

(B) Epstein-Barr virus — latent in B cells; diagnosed by Monospot test

(C) Cytomegalovirus — latent in monocytes; diagnosed by shell vial culture

(D) Varicella-zoster virus — latent in dorsal root ganglia (DRG); reactivates as herpes zoster; Tzanck smear shows multinucleated giant cells

Q28. A 20-year-old unvaccinated male university student presents with fever, painful parotid swelling, and 3 days later develops severe unilateral testicular pain and swelling. Serum amylase is elevated. CSF analysis reveals 200 lymphocytes/mm^3 with normal glucose, suggesting aseptic meningitis. The causative virus is a paramyxovirus with a single-stranded negative-sense RNA genome. Which of the following correctly identifies the most common extra-salivary complication in post-pubertal males and the risk it poses?

(A) Orchitis in post-pubertal males — the most common severe complication; bilateral orchitis can lead to infertility; unilateral is more common and rarely causes sterility

(B) Oophoritis in post-pubertal females — the most common complication

(C) Sensorineural deafness — the most common complication of mumps overall

(D) Encephalitis — occurs in 1 in 6000 cases; the most common severe complication

Q29. A 24-year-old pregnant woman in her 8th week presents with low-grade fever, posterior cervical and suboccipital lymphadenopathy, and a fine maculopapular rash spreading from face to trunk. IgM antibodies to

rubella virus are detected. At birth, the infant is found to have a patent ductus arteriosus, bilateral cataracts, and sensorineural deafness. The infant also has petechiae giving a "blueberry muffin" appearance due to dermal haematopoiesis. Which of the following correctly describes the teratogenic risk by trimester?

(A) Third trimester infection carries the highest risk of major congenital defects

(B) First trimester infection carries the greatest risk of congenital rubella syndrome (CRS) — up to 85%; classic triad: cardiac defects, cataracts, and sensorineural deafness

(C) Rubella is only dangerous if the mother is symptomatic

(D) Second trimester infection is equally teratogenic as first trimester

Q30. A 3-year-old unvaccinated child in a polio-endemic area develops acute onset fever followed by asymmetric flaccid paralysis of the left lower limb without sensory loss. There is no bladder dysfunction. Stool viral culture yields a small, non-enveloped, single-stranded positive-sense RNA virus. CSF shows mild pleocytosis with normal protein and glucose. Electromyography confirms anterior horn cell (motor neuron) destruction. Which of the following correctly describes the mechanism of paralysis?

(A) Demyelination of peripheral motor nerves (Guillain-Barré pattern)

(B) Toxin-mediated neuromuscular junction blockade (botulism)

(C) Poliovirus (picornavirus, Enterovirus) — replicates in the anterior horn cells of the spinal cord causing lower motor neuron destruction and flaccid paralysis; no sensory involvement

(D) Upper motor neuron destruction causing spastic paralysis

Q31. A 14-month-old child in a developing country presents in winter with sudden-onset non-bloody, non-mucoid, watery diarrhoea (10–12 episodes/day), vomiting, and fever leading to severe dehydration. Electron microscopy of stool shows a "wheel-like" particle with characteristic double-shelled

capsid. The child recovers with oral rehydration therapy. This virus is the leading global cause of severe dehydrating gastroenteritis in children under 5 years. Which of the following correctly identifies the virus and its key epidemiological feature?

(A) Norovirus — most common cause of epidemic non-bacterial gastroenteritis in all ages; cruise-ship outbreaks

(B) Astrovirus — causes mild, self-limited diarrhoea predominantly in children

(C) Adenovirus serotypes 40/41 — second most common cause of viral gastroenteritis in infants

(D) Rotavirus — double-stranded segmented RNA virus; most common cause of severe dehydrating diarrhoea in children <5 years worldwide; prevented by oral vaccines (Rotarix, RotaTeq)

Q32. A cruise ship with 1200 passengers experiences an outbreak of vomiting and diarrhoea affecting 400 people within 48 hours. The illness is characterised by sudden-onset projectile vomiting, mild watery diarrhoea, nausea, and low-grade fever lasting 24–48 hours. Most passengers recover spontaneously. Epidemiological investigation finds a common meal and identifies virus particles by RT-PCR from environmental surfaces (fomites), vomitus, and stool. The causative agent is a single-stranded positive-sense RNA, non-enveloped virus. Which of the following best identifies the pathogen and its characteristic features?

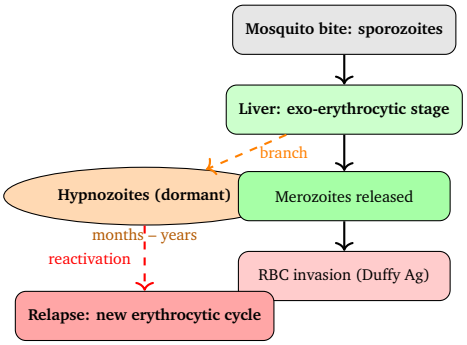
(A) Norovirus (calicivirus) — vomiting-predominant illness, 24–48 hour duration, fomite and droplet transmission; most common cause of epidemic acute gastroenteritis in all age groups

(B) Rotavirus — causes dehydrating diarrhoea predominantly in infants

(C) Hepatitis E virus — enteric hepatitis, icteric illness, faecal-oral route

(D) Astrovirus — mild self-limited diarrhoea in children; not a common cause of large outbreaks

Q33. A 28-year-old man returns from a trip to India with fever, chills, and rigors occurring every 48 hours (tertian pattern). Peripheral blood smear shows enlarged erythrocytes with Schüffner's stippling and a young ring-form trophozoite with an ameboid appearance. Six months after treatment, he develops another episode of fever. The diagram below shows the lifecycle of the causative *Plasmodium* species with emphasis on the mechanism of relapse.



Which of the following correctly identifies the organism and the mechanism of relapse?

(A) *Plasmodium falciparum* — knob formation, no relapse, most lethal

(B) *Plasmodium vivax* — hypnozoites remain dormant in hepatocytes; reactivate to cause relapse; Duffy antigen (DARC) required for RBC invasion; primaquine needed to eliminate hypnozoites

(C) *Plasmodium malariae* — quartan (72-hour) fever, recrudesces but does not relapse via hypnozoites

(D) *Plasmodium ovale* — also relapses, but without Schüffner's stippling or enlarged RBCs

Q34. A 32-year-old man returning from Mexico presents with bloody diarrhoea, severe abdominal cramps, and right upper quadrant pain. Stool microscopy reveals cysts containing 4 nuclei with peripheral chromatin and chromatoid bodies with blunt, cigar-shaped ends. An ultrasound shows a liver abscess with "anchovy sauce" contents. The organism is morphologically identical to a non-pathogenic species. Which of the following correctly distinguishes the pathogen from its non-pathogenic look-alike?

(A) *E. histolytica* has 8-nucleated cysts; *E. dispar* has 4-nucleated cysts

(B) *E. histolytica* produces cysts with pointed chromatoid bodies; *E. dispar* produces cysts with blunt chromatoid bodies

(C) *E. histolytica* (pathogenic) vs *E. dispar* (non-pathogenic) — morphologically identical; distinguished only by PCR or antigen detection (ELISA); *E. histolytica* produces invasive disease and liver abscess; *E. dispar* does not

(D) *E. dispar* is the pathogenic species responsible for liver abscess

Q35. A 45-year-old safari guide from sub-Saharan Africa presents with a chancre at the site of an insect bite, followed by fever, lymphadenopathy, and skin rash. Months later he develops daytime somnolence, night-time insomnia, confusion, and behavioural changes. CSF shows pleocytosis and the presence of motile trypomastigotes. Blood film reveals characteristic kinetoplasts and undulating membranes in flagellated organisms. Which of the following correctly identifies the pathogen, its vector, and the CNS stage?

(A) *Trypanosoma cruzi* — *Triatoma* (reduviid bug); Chagas disease, cardiac involvement

(B) *Leishmania donovani* — sand fly; visceral leishmaniasis (kala-azar)

(C) *Trypanosoma brucei gambiense* — *Glossina* (tsetse fly); West African sleeping sickness (slowly progressive)

(D) *Trypanosoma brucei rhodesiense* — *Glossina* (tsetse fly); East African sleeping sickness; CNS stage (meningoencephalitis) with somnolence and behavioural changes; fatal if untreated

Q36. A 10-year-old boy from Bihar, India, presents with prolonged irregular fever for 6 months, massive splenomegaly, hepatomegaly, weight loss, and progressive wasting. He appears darkened (hyperpigmentation) and has a markedly elevated serum globulin. Bone marrow biopsy reveals oval, non-flagellated organisms (2–4 μm) within macrophages with a characteristic lateral bar (kinetoplast). The aldehyde test (formal-gel

test) is positive due to hypergammaglobulinaemia. Which of the following correctly identifies the organism and its vector?

(A) *Leishmania donovani* — transmitted by female *Phlebotomus* sand fly; amastigotes in macrophages; visceral leishmaniasis (kala-azar); hyperpigmentation gives the Hindi name

(B) *Leishmania tropica* — cutaneous leishmaniasis (oriental sore), sand fly

(C) *Leishmania braziliensis* — mucocutaneous leishmaniasis, *Lutzomyia* sand fly

(D) *Trypanosoma brucei* — *Glossina* tsetse fly; sleeping sickness

Q37. A 55-year-old man on long-term prednisolone for rheumatoid arthritis presents with epigastric pain, diarrhoea, urticarial skin rashes (larva currens), and eosinophilia. Stool examination shows rhabditiform larvae. He subsequently develops high fever, Gram-negative septicaemia, and larval invasion of the lungs and CNS. Duodenal biopsy reveals filariform larvae and adults embedded in the crypts. Which of the following correctly describes the unique life cycle feature responsible for this patient's severe disease?

(A) Encystation in muscle tissue (cysticercosis) causing mass lesions

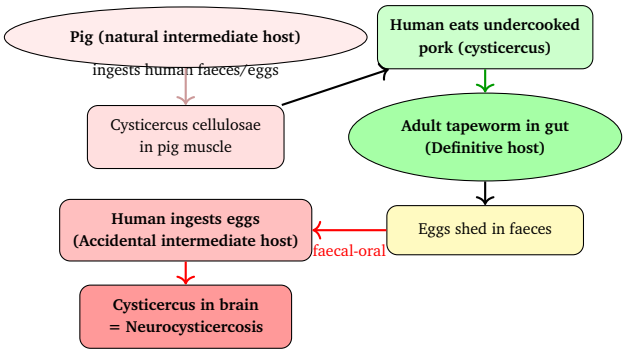
(B) Autoinfection: *Strongyloides stercoralis* rhabditiform larvae transform into infective filariform larvae within the gut; in immunocompromised patients, massive hyperinfection occurs with filariform larvae penetrating the gut wall, carrying gut bacteria and causing septicaemia, and disseminating to lungs, CNS, and other organs

(C) Migration through the liver (Loeffler's syndrome) with peripheral eosinophilia only

(D) Adult worm migration to the lymphatics causing lymphoedema (filariasis)

Q38. A 30-year-old man from a rural pig-farming community presents with new-onset seizures. MRI brain shows multiple ring-enhancing cystic le-

sions with scolices. He also reports occasional passage of proglottids in his stool. The diagram below shows the lifecycle of the causative cestode, highlighting how humans can serve as both definitive and accidental intermediate hosts.



Which of the following correctly identifies the condition causing this patient's seizures?

(A) *Taenia saginata* — neurocysticercosis from eating undercooked beef; identical to *T. solium*

(B) *Echinococcus granulosus* — hydatid cyst in liver; daughter cysts within mother cyst

(C) *Taenia solium* — human ingests eggs (faecal-oral/autoinfection) $\rightarrow$ cysticerci (larvae) lodge in brain causing neurocysticercosis; distinct from taeniasis (adult worm in gut from eating pork)

(D) *Taenia solium* — hydatid disease; liver is the only organ affected

Q39. A 38-year-old man with HIV infection and CD4 count of 40 cells/μL presents with gradual onset headache, fever, neck stiffness, and confusion over 2 weeks. CSF opening pressure is markedly elevated. CSF Indian ink preparation shows yeast cells surrounded by a wide, clear polysaccharide capsule. CSF cryptococcal antigen (CrAg) latex agglutination test is strongly positive. The organism is commonly associated with pigeon droppings. Which of the following correctly identifies the organism and its primary virulence factor?

(A) *Candida albicans* — germ tube formation, pseudohyphae, thrush

(B) *Aspergillus fumigatus* — septate hyphae with 45° branching, angio-invasion

(C) *Histoplasma capsulatum* — intracellular in macrophages, endemic (Ohio/Mississippi Valley)

(D) *Cryptococcus neoformans* — polysaccharide capsule (anti-phagocytic); seen in pigeon droppings; meningitis in AIDS; India ink shows capsule; treated with amphotericin B + flucytosine, then fluconazole maintenance

Q40. A 35-year-old cave explorer from the Ohio River Valley presents with fever, night sweats, weight loss, and bilateral hilar lymphadenopathy on chest X-ray mimicking primary tuberculosis. He recalls disturbing a large bat colony in a cave 3 weeks earlier. Bone marrow biopsy shows small (2–4 μm) oval yeast forms packed within macrophages. Urine antigen test for a dimorphic fungus is positive. Culture at 25°C reveals cottony white mould with characteristic tuberculate macroconidia. Which of the following correctly identifies this organism and its epidemiological niche?

(A) *Histoplasma capsulatum* — dimorphic fungus; endemic to Ohio and Mississippi River Valleys; yeast phase (37°C) grows intracellularly in macrophages; mould phase (25°C) produces tuberculate macroconidia; found in soil enriched by bat or bird (starling) droppings

(B) *Blastomyces dermatitidis* — large (8–15 μm) yeast with broad-based budding; Great Lakes region

(C) *Coccidioides immitis* — San Joaquin Valley fever; spherules with endospores; South-western USA and Latin America

(D) *Paracoccidioides brasiliensis* — "captain's wheel" pattern; South America; coffee farmers

Detailed Solutions

Q1.

Solution

Concept – MRSA and mecA Gene: *Staphylococcus aureus* becomes methicillin-resistant when it acquires the *mecA* gene on the staphylococcal cassette chromosome *mec* (SCCmec). The *mecA* gene encodes a modified penicillin-binding protein, PBP2a, with a drastically reduced affinity for all beta-lactam antibiotics. Because PBP2a can still catalyse cell wall transpeptidation even when all normal PBPs are inhibited, the organism survives exposure to penicillins, cephalosporins, and carbapenems.

Key Points:

- Normal PBPs: the bacterial enzymes that cross-link peptidoglycan; their active site serine is acylated and permanently inhibited by beta-lactams.
- PBP2a: the *mecA*-encoded substitute; steric hindrance in the active site prevents beta-lactam binding; transpeptidation continues.
- Treatment of MRSA: vancomycin (inhibits lipid II, not PBPs), daptomycin, linezolid, or telavancin.
- MRSA screening: PCR for *mecA* gene or CHROMagar MRSA selective medium.

Why other options are wrong:

- Option A: MSSA lacks *mecA*; PBP2 is inhibited normally by oxacillin and other beta-lactams.
- Option C: ESBL-producing *E. coli* is gram-negative; ESBL hydrolyses beta-lactams rather than using an alternative PBP.
- Option D: VRE is *Enterococcus* spp. with altered peptidoglycan precursor (D-Ala-D-Lac instead of D-Ala-D-Ala) conferring vancomycin resistance, unrelated to *mecA*.

Final Answer: oxacillin failure + *mecA* PCR + gram-positive cocci clusters $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q1

Q2.

Solution

Concept – Quellung (Neufeld) Reaction: The Quellung (German: "swelling") reaction, also called the Neufeld test, is a specific method for identifying encapsulated bacteria, particularly *Streptococcus pneumoniae*. When the organism is mixed with type-specific anti-capsular antiserum and a vital dye (methylene blue), antibodies bind the polysaccharide capsule, causing it to appear to swell and become sharply refractile under the microscope due to the antibody-antigen complex changing capsule optical properties. This is the classical method for pneumococcal serotyping.

Key Points:

- *S. pneumoniae*: lancet-shaped gram-positive diplococci; bile soluble; optochin-sensitive.
- **92 capsular serotypes** identified by Quellung; determines vaccine coverage.
- Quellung differentiates pneumococcal serotypes; India ink simply detects capsule presence without specificity.
- Clinical context: rust-coloured sputum + lobar consolidation + gram-positive diplococci = classic pneumococcal pneumonia.

Why other options are wrong:

- Option A: Coagulase test distinguishes *S. aureus* (coagulase-positive) from coagulase-negative staphylococci; not used for pneumococcus.
- Option B: CAMP test identifies group B streptococci (*S. agalactiae*); irrelevant here.
- Option D: India ink detects capsule presence (used for *Cryptococcus*) but does not provide serotype-specific identification.

Final Answer: antiserum + capsular swelling + lancet-shaped diplococci $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – Shiga Toxin of *Shigella dysenteriae*: *Shigella dysenteriae* serotype 1 produces Shiga toxin (Stx1), the prototype AB5 toxin. The A subunit has RNA N-glycosidase activity: it cleaves the N-glycosidic bond of a specific adenine residue in the 28S rRNA of the 60S ribosomal subunit. This irreversibly halts protein

synthesis and causes endothelial and epithelial cell death, leading to haemorrhagic colitis and, in severe cases, haemolytic uraemic syndrome (HUS).

Key Points:

- **Shiga toxin mechanism**: AB5; B subunits bind Gb3 (globotriaosylceramide) on host cells; A subunit cleaves 28S rRNA (N-glycosidase); ribosome inactivation $\rightarrow$ cell death.
- *Shiga-like toxins* (Stx1, Stx2) of EHEC (*E. coli* O157:H7) use identical mechanism.
- *S. dysenteriae* serotype 1: non-lactose-fermenting on MacConkey; non-motile; non-gas-producing.
- Clinical: bloody mucoid dysentery + severe cramps + fever = bacillary dysentery.

Why other options are wrong:

- Option A: *S. sonnei* produces only mild diarrhoea; its toxin production is minimal; serotype 1 is specifically *S. dysenteriae*.
- Option B: *C. perfringens* alpha toxin (lecithinase/phospholipase C) degrades cell membrane phospholipids; not an RNA N-glycosidase.
- Option C: Stx2 is from EHEC, not *Shigella flexneri*; *S. flexneri* is serotype 2–6.

Final Answer: RNA N-glycosidase on 60S + non-lactose-fermenting + serotype 1 $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q3

Q4.

Solution

Concept – Lepromatous vs Tuberculoid Leprosy: The clinical spectrum of leprosy reflects the host immune response to *Mycobacterium leprae*. Lepromatous leprosy (LL) results from a polarised Th2 response with low cell-mediated immunity (CMI): abundant IL-4, IL-5, and IL-10 suppress macrophage activation, permitting unrestricted bacterial multiplication (multibacillary, $>$5 lesions). Tuberculoid leprosy (TT) reflects a strong Th1 response: IFN-γ activates macrophages to kill bacilli, resulting in paucibacillary disease ($\leq$5 lesions) with high CMI but tissue damage from granuloma formation.

Comparison Table:

- LL: Th2, IL-4/IL-10 dominant, lepromin −, many bacilli, diffuse infiltration, madarosis, foamy macrophages (Virchow cells).
- TT: Th1, IFN-γ dominant, lepromin +, few bacilli, well-defined anaesthetic patches, granulomas with epithelioid cells.
- **Mnemonic**: Lepromatous = Low immunity; Tuberculoid = T-cell (strong); Both caused by the same organism (*M. leprae*).

Why other options are wrong:

- Option B: Reverses the correct immunological profiles of the two forms.
- Option C: Cellular immunity differs markedly between the two polar forms; the Ridley-Jopling spectrum spans TT $\rightarrow$ BT $\rightarrow$ BB $\rightarrow$ BL $\rightarrow$ LL.
- Option D: Both forms are caused by *M. leprae*; no other species is responsible.

Final Answer: multibacillary + Th2 + lepromin negative = lepromatous; paucibacillary + Th1 = tuberculoid $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q4

Q5.

Solution

Concept – *Clostridioides difficile* and Pseudomembranous Colitis: After clindamycin (or any broad-spectrum antibiotic) disrupts normal colonic flora, *C. difficile* spores germinate and overgrow. The organism produces two major toxins: Toxin A (enterotoxin) causes fluid secretion and mucosal damage; Toxin B (cytotoxin) is the primary virulence factor — it inactivates Rho GTPases (Rho, Rac, Cdc42) by glucosylation, disrupting the actin cytoskeleton and causing cell rounding, tight junction dissolution, epithelial cell death, and the characteristic pseudomembrane (fibrin + mucus + necrotic debris + neutrophils).

Key Points:

- **Risk factors**: clindamycin, fluoroquinolones, cephalosporins, PPIs, hospitalisation, age >65.
- Diagnosis: stool EIA for toxin A and B; NAAT (PCR) for *tcdB* gene (most sensitive).
- Treatment: mild/moderate — oral vancomycin or fidaxomicin; severe — IV metronidazole adjunct; recurrence — faecal microbiota transplant (FMT).
- Hypervirulent NAP1/BI/027 strain: binary toxin + increased Toxin A and B production.

Why other options are wrong:

- Option A: TSST-1 is from *S. aureus*; causes toxic shock syndrome, not pseudomembranous colitis.
- Option C: *C. perfringens* alpha toxin causes gas gangrene and food poisoning (epsilon toxin); not PMC.
- Option D: *Bacillus cereus* cereulide causes emetic syndrome (starchy foods); diarrhoeal form involves heat-labile enterotoxin; not PMC.

Final Answer: clindamycin + yellowish plaques + toxin A and B EIA positive $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – *Legionella pneumophila* and Legionnaires' Disease: *Legionella pneumophila* is a fastidious gram-negative bacillus that requires L-cysteine and iron for growth, hence requiring L-BCYE (buffered charcoal yeast extract) agar. It is an obligate intracellular pathogen of alveolar macrophages, hijacking the phagosome to form a Legionella-containing vacuole (LCV) that evades lysosomal fusion. Silver stain (Dieterle) is used for tissue sections. Sputum Gram stain is often negative despite heavy infection. Urinary antigen test detects serogroup 1 antigen.

Key Distinguishing Features:

- Source: aerosolised contaminated water (cooling towers, air-conditioning systems, hot tubs, hospital water).
- Systemic features: hyponatraemia, elevated liver enzymes, confusion (Pontiac fever = milder form without pneumonia).
- Culture: L-BCYE; no growth on blood agar or MacConkey.
- Silver stain (Dieterle): identifies organisms in tissue; Gram stain: poorly staining, often missed.
- Treatment: azithromycin or fluoroquinolone (intracellular penetration required).

Why other options are wrong:

- Option A: *Mycoplasma pneumoniae* is the most common atypical pneumonia but grows on Eaton agar; no urinary antigen test available; associated with cold agglutinins.

- Option B: *Chlamydophila pneumoniae* is intracellular but grows in cell culture; no urinary antigen; requires MIF serology.
- Option D: *Coxiella burnetii* causes Q fever; no urinary antigen; associated with livestock (cattle, sheep, goats); no L-BCYE requirement.

Final Answer: L-BCYE + urinary antigen + hotel air conditioning + hyponatraemia + intracellular $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q6

Q7.

Solution

Concept – *Bordetella pertussis* and Whooping Cough: *B. pertussis* causes pertussis (whooping cough) in three stages. **(1) Catarrhal stage** (1–2 weeks): mild coryza, low-grade fever, and mild cough; most infectious. **(2) Paroxysmal stage** (2–6 weeks): severe paroxysmal coughing fits followed by an inspiratory whoop; post-tussive vomiting; lymphocytosis. **(3) Convalescent stage** (weeks 6–8+): gradual resolution of cough. Pertussis toxin (PT) is the key virulence factor: it ADP-ribosylates Gi proteins, increasing cAMP and causing lymphocytosis by blocking lymphocyte recirculation from blood to lymph nodes.

Key Points:

- Pertussis toxin: A-B type; ADP-ribosylates Gi (inhibitory G protein) $\rightarrow$ adenylate cyclase constitutively active $\rightarrow$ raised cAMP; causes lymphocytosis by preventing CXCR5-mediated lymphocyte homing.
- Other virulence factors: filamentous haemagglutinin (FHA) — adherence; adenylate cyclase toxin — impairs phagocyte killing; tracheal cytotoxin — ciliary damage.
- Culture: Bordet-Gengou (potato-blood) agar or BCYE-like charcoal agar (Regan-Lowe).
- Diagnosis: nasopharyngeal PCR (most sensitive); DFA; serology (anti-PT IgG).
- Vaccine: acellular pertussis (DTaP/Tdap) — PT, FHA, pertactin, fimbriae.

Why other options are wrong:

- Option A: *B. parapertussis* causes a milder pertussis-like illness and does not produce pertussis toxin; FHA alone.
- Option B: *H. influenzae* causes epiglottitis and otitis media; not whooping cough; IgA protease is relevant to mucosal colonisation.

- Option C: *M. catarrhalis* causes otitis media and sinusitis; produces beta-lactamase; unrelated to whooping cough.

Final Answer: three-stage illness + inspiratory whoop + lymphocytosis + pertussis toxin blocks lymphocyte recirculation $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q7

Q8.

Solution

Concept – *Helicobacter pylori* and Eradication Testing: *H. pylori* is a gram-negative, microaerophilic, spiral-shaped bacterium with potent urease activity. It colonises the gastric antrum and body, disrupting the mucous barrier and causing peptic ulcer disease. The **urea breath test** (^{13}C- or ^{14}C-urea) is the gold standard non-invasive test for confirming eradication (performed $\geq$4 weeks after completing therapy and $\geq$2 weeks after stopping PPIs). The patient ingests labelled urea; active urease from living *H. pylori* hydrolyses it to labelled CO_2, detected in exhaled breath.

Diagnostic Methods for *H. pylori*:

- Invasive (endoscopy required): rapid urease test (CLO) — diagnosis; histology — gold standard for diagnosis; culture — sensitivity testing.
- Non-invasive: urea breath test (UBT) — gold standard for eradication confirmation; stool antigen test (HpSA) — alternative for eradication; serology (IgG) — screening only; cannot confirm eradication (antibodies persist for years).

Why other options are wrong:

- Option B: Serology (IgG) remains positive for months to years after eradication; it cannot confirm successful treatment.
- Option C: *Campylobacter jejuni* causes acute bloody diarrhoea; does not cause duodenal ulcers.
- Option D: CLO (rapid urease) test requires endoscopic biopsy; it is invasive and is not used to confirm eradication.

Final Answer: CLO test positive + duodenal ulcer + eradication confirmation $\rightarrow$ ^{13}C urea breath test $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q8

Q9.

Solution

Concept – *Chlamydia trachomatis* Serovars: *C. trachomatis* serovars are classified by major outer membrane protein (MOMP) antigenic variation into three disease groups:

- **Serovars A, B, Ba, C**: trachoma (chronic follicular conjunctivitis → corneal pannus → blindness); transmitted by flies and fomites in endemic areas.
- **Serovars D–K**: urogenital infections (NGU in males; cervicitis, PID, salpingitis in females); neonatal ophthalmia neonatorum; sexually transmitted.
- **Serovars L1, L2, L3**: lymphogranuloma venereum (LGV); invades lymphatics; genital ulcer → inguinal buboes with groove sign → chronic scarring; more invasive than D–K.

Key Pathobiological Notes:

- *C. trachomatis*: obligate intracellular; two forms — elementary body (EB, infectious) and reticulate body (RB, replicating intracellularly).
- Diagnosis: NAAT (PCR) on urogenital swabs or first-void urine — most sensitive; culture in McCoy cells.
- Treatment: azithromycin (single dose) or doxycycline 7 days for uncomplicated; doxycycline 21 days for LGV.

Why other options are wrong:

- Option A: Reverses the serovars — A–C are trachoma, not urogenital.
- Option C: Incorrectly assigns A–C to LGV and L1–L3 to urogenital.
- Option D: Incorrect; serovars are clearly distinguished by disease.

Final Answer: A–C trachoma, D–K urogenital, L1–L3 LGV $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q9

Q10.

Solution

Concept – Rocky Mountain Spotted Fever (*Rickettsia rickettsii*): RMSF is caused by *R. rickettsii*, transmitted by *Dermacentor variabilis* (American dog tick) and *D. andersoni* (Rocky Mountain wood tick). The classic clinical triad is fever + headache + rash (centrifugal, starting on wrists and ankles spreading to trunk, involving palms and soles). *Rickettsia* invades endothelial cells, causing vasculi-

tis, thrombocytopaenia, and multi-organ involvement. The Weil-Felix test uses cross-reactive antibodies: anti-*R. rickettsii* antibodies agglutinate *Proteus* OX-19 and OX-2 (but not OX-K).

Weil-Felix Test Summary:

- OX-19 + OX-2 positive: *R. rickettsii* (RMSF), *R. conorii*, *R. prowazekii*.
- OX-K positive only: *R. tsutsugamushi* (scrub typhus).
- Negative: *R. typhi* (murine typhus) — OX-19 weakly positive.
- Gold standard: IFA serology; PCR; Weil-Felix is non-specific but useful in resource-limited settings.

Why other options are wrong:

- Option A: *R. typhi* causes murine typhus (rat-flea-borne); rash is centripetal (trunk first); milder disease.
- Option B: *Borrelia burgdorferi* causes Lyme disease; Ixodes tick; erythema migrans; Weil-Felix negative.
- Option D: *R. prowazekii* causes epidemic typhus; louse-borne; no palm/sole rash.

Final Answer: wrist-ankle rash + palms/soles + *Dermacentor* + OX-19/OX-2 positive $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q10

Q11.

Solution

Concept – Weil's Disease (*Leptospira interrogans*): Leptospirosis is caused by pathogenic *Leptospira interrogans* serovars (e.g., icterohaemorrhagiae). Humans are infected through contact with water or soil contaminated by urine of infected animals (rats, cattle, dogs). The classic severe form, Weil's disease, presents with the triad: **jaundice** (hepatocellular + haemolytic) + **acute kidney injury** + **haemorrhage** (pulmonary, conjunctival, petechiae). Leptospires are thin, tightly coiled spirochaetes with hooked ends, visible by darkfield microscopy in blood (week 1) and urine (week 2 onward).

Key Diagnostic Features:

- Darkfield microscopy: motile, hooked spirochaetes in urine.
- Serology: MAT (microscopic agglutination test) — gold standard; ELISA IgM

for early screening.
- Culture: EMJH medium at 28–30°C (slow-growing, 6–8 weeks).
- Weil-Felix: NEGATIVE (Leptospira not a rickettsia).
- Treatment: penicillin or doxycycline; doxycycline prophylaxis for exposure.

Why other options are wrong:

- Option A: *Borrelia recurrentis* causes relapsing fever (tick/louse-borne); characterised by multiple febrile episodes; no jaundice or renal failure.
- Option B: *T. pallidum* congenital syphilis presents in neonates, not adults; no acute renal failure or haemorrhage.
- Option C: *Leptospira biflexa* is the saprophytic, non-pathogenic environmental species; does not cause human disease.

Final Answer: floodwater exposure + jaundice + AKI + haemorrhage + hooked spirochaetes in urine $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – Syphilis Serology (VDRL vs FTA-ABS): Two categories of serological tests are used in syphilis:

- **Non-treponemal tests** (VDRL, RPR): detect anti-cardiolipin (reagin) antibodies; used for **screening** and **monitoring treatment** (titre falls with successful therapy; a fourfold fall = treatment success); can give **biological false positives** (SLE, pregnancy, viral infections, malaria).
- **Treponemal tests** (FTA-ABS, TPHA, TPPA, EIA): detect specific anti-treponemal antibodies; used for **confirmation**; **remain positive for life** even after treatment (scar serology); cannot be used to monitor therapy.

Secondary Syphilis Clues:

- Generalised maculopapular rash including palms and soles (classic distinguishing feature).
- Condylomata lata (flat, moist, infectious genital warts) — different from condylomata acuminata (HPV).
- Oral mucous patches; generalised lymphadenopathy.
- VDRL titre highest in secondary syphilis (often >1:32).

Why other options are wrong:

- Option B: Reverses the roles — FTA-ABS is treponemal (confirmatory), not non-treponemal.
- Option C: Only non-treponemal tests (VDRL, RPR) become negative after treatment; treponemal tests (FTA-ABS) remain positive for life.
- Option D: Reverses the screening/confirmation roles.

Final Answer: VDRL = non-treponemal screening/treatment monitoring; FTA-ABS = treponemal confirmatory, lifelong positive $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q12

Q13.

Solution

Concept – Bubonic Plague (*Yersinia pestis*): *Y. pestis* causes bubonic plague, transmitted by the bite of the Oriental rat flea *Xenopsylla cheopis*. Bacilli are injected into the skin and drain to regional lymph nodes, causing a massively inflamed, haemorrhagic bubo. Key virulence factors: **F1 antigen** (capsule-like, anti-phagocytic at 37°C), **V antigen** (anti-phagocytic, immunosuppressive), LcrV, and Yops (Yersinia outer proteins disrupting phagocyte signalling). The organism stains with characteristic bipolar ("safety pin") appearance with Wayson or Giemsa stain due to bipolar distribution of cytoplasmic contents.

Key Microbiological Features:

- Gram-negative coccobacillus; facultative anaerobe; grows at 28°C (flea temperature) better than 37°C.
- Culture: MacConkey (non-lactose fermenting, slow growth); blood agar at 28°C; BCYE-like media.
- Three forms: bubonic (lymph node); septicaemic (bloodstream); pneumonic (lungs, most dangerous, person-to-person).
- Diagnosis: Gram stain and culture of bubo aspirate; PCR; serology (anti-F1 antibodies).
- Treatment: streptomycin (traditional), doxycycline, or ciprofloxacin.

Why other options are wrong:

- Option A: *Francisella tularensis* causes tularaemia (rabbit fever); ulceroglandular form; *Dermacentor* tick or direct contact; no safety-pin bipolar staining.
- Option C: *Brucella melitensis* causes brucellosis (undulant fever); no massive bubo; unpasteurised goat milk.

- Option D: *Bartonella henselae* causes cat-scratch disease; cat flea; typically self-limiting lymphadenopathy without systemic toxicity.

Final Answer: bubo + dead rats + bipolar staining + F1 antigen + *Xenopsylla cheopis* $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – Cervicofacial Actinomycosis (*Actinomyces israelii*): *A. israelii* is a gram-positive, non-acid-fast, anaerobic-to-microaerophilic branching filamentous bacterium that is part of normal oral flora. After disruption of mucosal barriers (dental extractions, jaw trauma), it causes slowly progressive cervicofacial actinomycosis characterised by: indurated jaw mass with draining sinuses, production of **sulfur granules** (yellow granules = colonies of organisms surrounded by a Splendore-Hoeppli eosinophilic matrix and club-shaped peripheral filaments). Culture on blood agar under anaerobic conditions produces "molar tooth" or "breadcrumb" colonies after >5 days.

Key Distinguishing Features from Nocardia:

- *Actinomyces*: anaerobic, NOT acid-fast, gram-positive; sulfur granules; responds to penicillin (long courses 6–12 months).
- *Nocardia*: aerobic, weakly/partially acid-fast, gram-positive; pulmonary/disseminated; responds to sulfonamides (TMP-SMX).
- *Actinomyces*: normal flora (mouth, GI, vagina) – endogenous pathogen, NOT acquired from environment.

Why other options are wrong:

- Option A: *Nocardia asteroides* is aerobic, partially acid-fast, and causes pulmonary or CNS nocardiosis; cervicofacial disease is rare.
- Option B: *Streptomyces* species are aerobic soil organisms that rarely cause human disease (actinomycetoma in tropics); they produce antibiotics.
- Option D: *Aspergillus fumigatus* is a fungus with septate hyphae branching at 45° angles; not gram-positive; causes invasive aspergillosis, not cervicofacial disease.

Final Answer: jaw swelling post-dental + draining sinuses + sulfur granules + anaerobic + molar tooth colonies $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q14

Q15.

Solution

Concept – Type II Hypersensitivity (Antibody-Mediated Cytotoxicity): Type II hypersensitivity involves IgG and IgM antibodies directed against antigens on the surface of cells or extracellular matrix. Three mechanisms of tissue damage:

- **Complement-mediated lysis**: Ab + Ag $\rightarrow$ classical complement pathway $\rightarrow$ MAC formation $\rightarrow$ cell lysis.
- **Opsonisation and phagocytosis**: C3b and Fc-receptor-mediated uptake by macrophages and neutrophils.
- **ADCC**: NK cells bind IgG-coated target cells via FcγRIII (CD16) $\rightarrow$ cytotoxic granule release.

Classic Examples of Type II Hypersensitivity:

- Acute haemolytic transfusion reaction (ABO incompatibility) — as in this question.
- Autoimmune haemolytic anaemia; immune thrombocytopaenia (ITP).
- Goodpasture's disease (anti-GBM antibodies against type IV collagen).
- Myasthenia gravis (anti-AChR antibodies — functional block/destruction).
- Graves' disease (stimulatory — TSH receptor antibodies).
- Haemolytic disease of the newborn (anti-Rh D IgG crossing placenta).

Why other options are wrong:

- Option A: Type I (IgE-mediated) involves mast cell degranulation; no complement classical pathway; occurs within minutes; e.g., anaphylaxis, asthma.
- Option B: Type III involves soluble antigen-antibody immune complexes depositing in tissue; Arthus reaction, serum sickness.
- Option C: Type IV is T-cell mediated; no antibody; delayed 48–72 hours; e.g., tuberculin test, contact dermatitis.

Final Answer: pre-formed Ab against RBC surface Ag + complement + ADCC + haemoglobinuria $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q15

Q16.

Solution

Concept – Type III Hypersensitivity (Immune Complex-Mediated): Type III hypersensitivity occurs when soluble antigen-antibody complexes form and deposit in blood vessel walls or tissues. These complexes fix complement (C1q binding) generating C3a and C5a (anaphylatoxins and chemotaxins). Neutrophil recruitment and frustrated phagocytosis releases lysosomal enzymes causing local tissue destruction. Complement consumption causes low C3 and C4 levels.

Classic Examples:

- SLE: anti-dsDNA + dsDNA complexes in glomeruli $\rightarrow$ proliferative GN; low C3/C4; ANA and anti-dsDNA positive.
- Post-streptococcal GN: streptococcal antigen–antibody complexes in glomeruli; "lumpy-bumpy" immunofluorescence; elevated ASO titre.
- Serum sickness: foreign protein (horse antivenom, murine MAb) + antibodies $\rightarrow$ fever, urticaria, arthralgia, lymphadenopathy 7–14 days after exposure.
- Arthus reaction: local Type III reaction after intradermal antigen injection in pre-immunised individual.

Why other options are wrong:

- Option B: Direct cell-surface antibody binding (complement lysis, ADCC) = Type II.
- Option C: IgE + mast cell + histamine = Type I (immediate hypersensitivity, allergy).
- Option D: CD4+ T cell + IFN-γ + macrophage = Type IV (delayed-type, no antibody).

Final Answer: immune complex deposition + low complement + granular GBM deposits + serum sickness $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q16

Q17.

Solution

Concept – Type IV Delayed-Type Hypersensitivity (DTH) and Mantoux Test: Type IV (cell-mediated) hypersensitivity is mediated exclusively by T cells, with no antibody involvement. It consists of two phases:

- **Sensitisation phase** (first exposure, days 1–13): antigen-presenting cells (dendritic cells and macrophages) process and present PPD peptides on MHC II to naive CD4+ T cells $\rightarrow$ clonal expansion and formation of memory Th1 cells.
- **Elicitation phase** (re-exposure, from day 14 onward): memory Th1 cells recognise PPD-MHC II complexes on APCs $\rightarrow$ release IFN-γ, IL-2, TNF-β $\rightarrow$ macrophage activation $\rightarrow$ induration (firm swelling from mononuclear cell infiltrate) at 48–72 hours.

Mantoux Test Interpretation:

- $\geq$15 mm: healthy individuals with no risk factors (positive).
- $\geq$10 mm: healthcare workers, immigrants, children $<$4 years, prison populations.
- $\geq$5 mm: HIV-positive, immunosuppressed, close contacts of TB, abnormal chest X-ray.
- False positive: BCG vaccination, NTM infection.
- False negative: severe immunosuppression (anergy), recent TB or viral infection, incorrect technique.

Why other options are wrong:

- Option A: Type I = IgE + mast cells; immediate (minutes); urticaria and anaphylaxis.
- Option C: Type II = cell-surface antibody + complement; ADCC; haemolysis, cytotoxicity.
- Option D: Arthus reaction = local Type III; neutrophil-mediated; occurs at antigen injection site in pre-immunised individuals; distinct from DTH.

Final Answer: Mantoux 48–72 h induration + CD4+ Th1 + IFN-γ + macrophage activation + no antibody $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q17

Q18.

Solution

Concept – DiGeorge Syndrome (22q11 Deletion): DiGeorge syndrome results from failure of development of the 3rd and 4th pharyngeal pouches (derived from neural crest cells), caused by a microdeletion at chromosome 22q11.2. This leads to thymic aplasia or hypoplasia (absent T-cell development), hypoparathyroidism

(hypocalcaemia, tetany), and conotruncal cardiac defects (interrupted aortic arch, truncus arteriosus, tetralogy of Fallot). The immunological result: severely reduced or absent CD3+ T cells; B cells are present but cannot receive T-cell help, causing secondary antibody deficiency (reduced IgG, IgA, IgE despite normal B-cell numbers).

Key Clinical Features Mnemonic – CATCH-22:

- **C**ardiac defects (conotruncal)
- **A**bnormal facies
- **T**hymic aplasia (absent T cells)
- **C**left palate
- **H**ypocalcaemia (hypoparathyroidism)
- **22**q11 deletion

Why other options are wrong:

- Option A: Bruton's agammaglobulinemia = absent B cells, normal T cells; X-linked; BTK mutation; presents at 6 months.
- Option B: SCID = absent T and B (and often NK) cells; ADA deficiency or γc-chain mutation; more severe than DiGeorge.
- Option D: Hyper-IgM syndrome = elevated IgM, absent IgG/IgA/IgE due to CD40L (CD154) mutation on T cells preventing B-cell class switching; T cells are present and functional.

Final Answer: 22q11 deletion + thymus aplasia + absent T cells + normal B cells + hypocalcaemia + conotruncal heart defect $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q18

Q19.

Solution

Concept – Bruton's X-linked Agammaglobulinemia (XLA): XLA is caused by mutations in the **BTK (Bruton's tyrosine kinase)** gene on chromosome Xq22. BTK is essential for signalling from the pre-B-cell receptor (pre-BCR), enabling maturation from pro-B to pre-B cells. Without functional BTK, B-cell development arrests at the pre-B cell stage in bone marrow: no mature B cells enter circulation; no immunoglobulins (IgG, IgM, IgA, IgE, IgD) are produced. T cells are unaffected. Clinical features: recurrent pyogenic bacterial infections (encapsulated bacteria: *S. pneumoniae*, *H. influenzae*, *Neisseria*) starting at 5–6 months when maternal

IgG wanes; absent tonsils and lymph nodes (no germinal centres); susceptibility to *Giardia* and enteroviruses.

Key Immunological Profile:

- B cells: absent ($CD19^-$, $CD20^-$).
- All immunoglobulin classes: markedly reduced or absent.
- T cells: normal number and function.
- Treatment: IVIG replacement every 3–4 weeks.

Why other options are wrong:

- Option A: CVID has normal or near-normal B-cell numbers but reduced immunoglobulins; affects adults; complex genetics; NOT X-linked.
- Option B: Hyper-IgM syndrome: elevated IgM, absent IgG/IgA; CD40L mutation on T cells; T cells present and functional; B cells present.
- Option C: Selective IgA deficiency: IgA absent, all other immunoglobulins normal; B cells present; most common primary immunodeficiency (1:300–700).

Final Answer: BTK mutation + absent B cells (all Ig) + normal T cells + X-linked + absent tonsils/nodes $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q19

Q20.

Solution

Concept – SCID due to ADA Deficiency: Adenosine deaminase (ADA) deficiency accounts for approximately 15–20% of all SCID cases and is autosomal recessive. ADA normally converts adenosine to inosine and deoxyadenosine to deoxyinosine. Without ADA, **deoxyadenosine** and **deoxyadenosine triphosphate (dATP)** accumulate. These are highly toxic to lymphocyte precursors (especially T cells), leading to pan-lymphopaenia ($T^-B^-NK^-$) — the most severe immunodeficiency phenotype. Patients present in the first months of life with PCP, recurrent viral and fungal infections, failure to thrive, and absent thymic shadow on chest X-ray.

Treatment Options:

- Haematopoietic stem cell transplantation (HSCT): curative.
- PEG-ADA (polyethylene glycol-conjugated ADA): enzyme replacement; not curative.

- Gene therapy: ADA gene insertion into autologous stem cells; increasingly successful.

SCID Phenotypes and Causes:

- $T^-B^-NK^-$: ADA deficiency (AR); reticular dysgenesis.
- $T^-B^-NK^+$: Rag1/2 mutations; Omenn syndrome.
- $T^-B^+NK^-$: γc-chain (γc; IL2RG) mutation — X-linked SCID (most common).
- $T^-B^+NK^+$: JAK3 deficiency (AR).

Why other options are wrong:

- Option B: DiGeorge = 22q11 deletion; absent T cells only; normal B cells; hypocalcaemia; conotruncal heart defects.
- Option C: Bruton's agammaglobulinemia = absent B cells; normal T and NK cells; X-linked.
- Option D: Wiskott-Aldrich syndrome = WASp gene mutation; eczema + thrombocytopaenia + immunodeficiency; $T^-B^+NK^-$ (progressive T-cell decline).

Final Answer: PCP + candidiasis + $T^-B^-NK^-$ lymphopaenia + dATP toxic accumulation + PEG-ADA treatment $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q20

Q21.

Solution

Concept – Secretory IgA and Mucosal Immunity: IgA is the most abundant immunoglobulin in external secretions (saliva, tears, colostrum, intestinal fluid, respiratory secretions). Mucosal IgA is produced as a dimer by plasma cells in the lamina propria, linked by the J-chain. The dimer binds the **poly-immunoglobulin receptor (pIgR)** on the basolateral surface of epithelial cells, is transcytosed across the cell, and is released into the lumen as **secretory IgA (sIgA)**. The extracellular portion of pIgR remaining attached is called the **secretory component (SC)**, which: (a) facilitates transcytosis, (b) protects the IgA dimer from proteolytic degradation in luminal secretions, and (c) anchors sIgA at the mucosal surface.

Functions of sIgA:

- Immune exclusion: prevents pathogen adherence to mucosal epithelium.

- Neutralises toxins, viruses, and bacteria in the lumen.
- Does NOT fix complement (avoids inflammatory tissue damage at mucosal surfaces).
- Breast milk sIgA passively protects neonates.

Why other options are wrong:

- Option A: IgG is the most abundant serum immunoglobulin; transplacental transfer provides passive neonatal immunity; not the dominant secretory antibody.
- Option C: IgM (pentameric) is the first antibody in primary immune response; activates complement; not the dominant secretory form.
- Option D: IgE mediates Type I hypersensitivity; anti-parasite responses; extremely low serum concentrations; not a mucosal secretory antibody.

Final Answer: dimer + J-chain + secretory component from epithelial pIgR + protects from proteolysis + mucosal defence $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q21

Q22.

Solution

Concept – Fcγ Receptors: FcγRI (CD64) and FcγRIII (CD16): Fc receptors bind the Fc (constant) region of immunoglobulins, linking humoral and cellular immunity. Key Fcγ receptors for IgG:

- **FcγRI (CD64)** — high affinity (K_d $\sim 10^{-9}$ M); binds monomeric IgG; expressed on monocytes, macrophages, and dendritic cells; mediates phagocytosis, ADCC, and antigen presentation.
- **FcγRII (CD32)** — medium affinity; expressed on most leucocytes (neutrophils, macrophages, B cells); inhibitory (FcγRIIB on B cells) or activating forms.
- **FcγRIII (CD16)** — low-to-medium affinity; expressed on NK cells (CD16a, transmembrane), neutrophils (CD16b, GPI-anchored); binds IgG-coated targets; primary mediator of **ADCC by NK cells**; also expressed on macrophages (FcγRIIIa).

Clinical Relevance:

- ADCC: NK cells kill IgG-coated targets (tumour cells, virus-infected cells, antibody-coated parasites) via CD16 engagement.

- CD16 on neutrophils is shed upon activation, releasing soluble CD16 (useful serum marker of neutrophil activation).
- Therapeutic monoclonal antibodies (e.g., rituximab, trastuzumab) exploit CD16-mediated ADCC.

Why other options are wrong:

- Option A: FcεRI (CD89 = FcαRI, not FcεRI) binds IgE on mast cells and basophils; CD89 is FcαRI (IgA receptor); not relevant here.
- Option B: Reverses the affinities — FcγRI (CD64) has the highest affinity, not FcγRII.
- Option D: NK cells and macrophages express different isoforms of CD16; they are not identical.

Final Answer: high-affinity CD64 on macrophages; low-affinity CD16 on NK cells (ADCC) $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q22

Q23.

Solution

Concept – Cytokine Storm in Gram-Negative Septic Shock: Gram-negative bacterial LPS (lipopolysaccharide/endotoxin) binds LPS-binding protein (LBP) in serum; the complex engages **CD14** on macrophages and monocytes, which signals through **TLR-4 and MD-2**. This triggers NF-κB activation and a massive release of pro-inflammatory cytokines:

- **TNF-α**: early mediator; causes fever, systemic vasodilation, increased vascular permeability, myocardial depression, and activates coagulation cascade $\rightarrow$ DIC.
- **IL-1**: synergises with TNF-α; fever (PGE2 in hypothalamus) and hypotension.
- **IL-6**: acute-phase protein synthesis (CRP, fibrinogen, ferritin); fever; thrombocytosis.

The combined effect causes refractory hypotension, ARDS, multi-organ failure, and DIC — the hallmarks of septic shock.

Key Pathophysiology Points:

- LPS $\rightarrow$ TLR-4/MD-2/CD14 complex $\rightarrow$ NF-κB $\rightarrow$ TNF-α, IL-1, IL-6, IL-8, IL-12.

- TNF-α activates vascular endothelium (E-selectin, ICAM-1 upregulation) $\rightarrow$ neutrophil adhesion and migration.
- DIC: cytokine-mediated tissue factor expression $\rightarrow$ fibrin thrombi $\rightarrow$ microangiopathic haemolysis + consumption coagulopathy.

Why other options are wrong:

- Option A: IgE-mast cell degranulation = anaphylaxis (Type I); triggered by allergen, not LPS; responds to epinephrine.
- Option B: Type II hypersensitivity requires antibodies against cell-surface antigens; LPS does not cause this mechanism.
- Option C: TLR-4 is activated (not blocked) by LPS; TLR blockade would reduce, not cause, septic shock.

Final Answer: LPS + CD14/TLR-4 + TNF-α/IL-1/IL-6 cytokine storm + DIC + ARDS + multi-organ failure $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q23

Q24.

Solution

Concept – Class Switch Recombination and IL-4: Class switch recombination (CSR) is the process by which B cells replace the IgM constant region gene with another isotype constant region (Cγ, Cα, or Cε) while maintaining the same antigen-binding V(D)J region. Two signals are required: (1) **CD40-CD40L interaction** (T cell help); (2) **cytokine signal** (determines which class is switched to). Key cytokine-to-isotype relationships in humans:

- **IL-4**: $\rightarrow$ IgE (and IgG4); promotes Th2 responses; anti-parasitic; allergy/atopy.
- **IL-5 + TGF-β**: $\rightarrow$ IgA (mucosal immunity).
- **TGF-β**: $\rightarrow$ IgA at mucosal sites (Peyer's patches).
- **IFN-γ** (Th1): $\rightarrow$ IgG1 and IgG3 (opsonisation, complement fixation).
- **IL-10**: supports IgG and IgA production; regulatory.

Clinical Relevance:

- Allergic disease: Th2 polarisation + high IL-4 $\rightarrow$ class switch to IgE $\rightarrow$ mast cell sensitisation.
- Parasitic infections: IgE-mediated eosinophil ADCC against helminths.

- Anti-IL-4 therapy (dupilumab: anti-IL-4Rα): blocks both IL-4 and IL-13 signalling; used in severe atopic dermatitis, asthma.

Why other options are wrong:

- Option B: IgG3 switching is driven by IFN-γ (Th1), not IL-4.
- Option C: TGF-β drives IgA switching (at mucosal sites); IL-4 drives IgE, not IgA.
- Option D: IFN-γ (from Th1 cells) drives IgG subclass switching; not IgE.

Final Answer: IL-4 $\rightarrow$ IgE class switching (Th2) $\rightarrow$ allergy and anti-parasite responses $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q24

Q25.

Solution

Concept – Epstein-Barr Virus (EBV) and Infectious Mononucleosis: EBV (HHV-4) is a double-stranded DNA herpesvirus. The viral envelope glycoprotein **gp350** binds **CD21 (complement receptor 2, CR2)** on B lymphocytes, mediating viral entry. EBV immortalises B cells, driving them to proliferate. CD8+ cytotoxic T cells (CTLs) expand massively to control infected B cells — these are the atypical lymphocytes (Downey cells) seen on blood film. Heterophile antibodies (detected by Monospot/Paul-Bunnell test) are IgM antibodies that agglutinate sheep or horse red blood cells; they are non-specific to EBV antigens but are a reliable early marker of infectious mononucleosis.

Key EBV Latency Proteins:

- EBNA-1: maintains viral episome in latent B cells.
- EBNA-2: drives B-cell proliferation (resembles Notch signalling).
- LMP-1: viral oncogene; mimics CD40 signalling; anti-apoptotic (upregulates BCL-2); drives cell growth.

EBV-Associated Malignancies:

- Burkitt's lymphoma (t[8;14], c-Myc) — endemic (African children) and sporadic forms.
- Hodgkin's lymphoma (mixed cellularity subtype).
- Nasopharyngeal carcinoma (Southeast Asia).
- Post-transplant lymphoproliferative disorder (PTLD).

Why other options are wrong:

- Option A: CMV does not use CD21; it binds multiple cell surface molecules and causes a Monospot-negative mononucleosis syndrome.
- Option C: HSV-1 uses HVEM (herpesvirus entry mediator) or nectin; infects epithelial cells, not B cells.
- Option D: HHV-6 infects T cells and causes roseola infantum; receptor is CD46, not CD21.

Final Answer: Monospot positive + atypical lymphocytes + B-cell tropism + gp350–CD21 binding $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q25

Q26.

Solution

Concept – Cytomegalovirus (CMV) and Owl Eye Inclusions: CMV (HHV-5) is the largest human herpesvirus. It infects a wide variety of cells (monocytes, endothelial cells, epithelial cells, neurons, retinal cells). In immunocompromised patients (AIDS, transplant recipients), CMV causes end-organ disease. The **pathognomonic histological finding** is the **owl eye inclusion**: large intranuclear inclusion surrounded by a clear halo (displacement of chromatin to nuclear rim) + smaller cytoplasmic inclusions — creating the appearance of an owl's eye. CMV retinitis is the most common cause of infectious blindness in AIDS patients and typically occurs when CD4 count falls **below 50 cells/μL**. It presents as a painless, progressive visual field loss with the characteristic "pizza pie" appearance on fundoscopy.

CMV Congenital Infection:

- Most common congenital viral infection worldwide.
- Triad: sensorineural hearing loss (most common sequela), chorioretinitis, and periventricular calcifications.
- "Blueberry muffin" rash — also seen in congenital rubella.

Treatment:

- Ganciclovir (first-line; CMV UL97 kinase phosphorylates it) and valganciclovir (oral prodrug).
- Foscarnet: for ganciclovir-resistant CMV.
- Cidofovir: nephrotoxic, used for severe resistant cases.

Why other options are wrong:

- Option A: *T. gondii* shows bradyzoites in tissue cysts; no owl eye inclusion bodies; different histological picture.
- Option B: HSV shows Cowdry type A inclusions (intranuclear, eosinophilic); no characteristic cytoplasmic inclusions.
- Option D: *C. neoformans* shows encapsulated yeast on India ink; no intranuclear inclusions.

Final Answer: owl eye intranuclear inclusions + AIDS + CD4 <50 + retinal "pizza pie" + painless vision loss $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q26

Q27.

Solution

Concept – Varicella-Zoster Virus (VZV) and Herpes Zoster: VZV (HHV-3) causes two distinct diseases. **Primary infection (chickenpox/varicella)**: generalised vesicular rash in crops, highly contagious, centripetal distribution (trunk $\rightarrow$ extremities). Following primary infection, VZV establishes **latency in dorsal root ganglia (DRG)** and trigeminal ganglia, where viral DNA persists in sensory neuron nuclei. Reactivation (triggered by immunosuppression, ageing, stress) causes **herpes zoster (shingles)**: painful, unilateral, dermatomal vesicular rash corresponding to the sensory distribution of the affected DRG. The **Tzanck smear** (vesicle base scraping stained with Giemsa or Wright stain) shows **multinucleated giant cells with intranuclear inclusions** — diagnostic of HSV and VZV (but cannot distinguish between them without additional testing).

Complications of Herpes Zoster:

- **Post-herpetic neuralgia (PHN)**: most common; severe burning pain persisting >3 months after rash resolution.
- **Ramsay Hunt syndrome**: VZV reactivation in geniculate ganglion (CN VII); facial palsy + ear vesicles + sensorineural hearing loss.
- **Herpes zoster ophthalmicus**: V1 (ophthalmic division) involvement; corneal ulceration, blindness.
- **Disseminated zoster**: pneumonitis, encephalitis in severely immunocompromised.

Why other options are wrong:

- Option A: HSV-2 is latent in sacral ganglia (S2–S4) and causes recurrent genital herpes; Tzanck is positive but VZV causes dermatomal thoracic zoster.
- Option B: EBV is latent in B cells; Monospot test; causes infectious mononucleosis; Tzanck is not used.
- Option C: CMV is latent in monocytes and macrophages; shell vial culture is used; no Tzanck-positive multinucleated giant cells.

Final Answer: unilateral dermatomal rash + Tzanck multinucleated giant cells + DRG latency + immunosuppression trigger $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q27

Q28.

Solution

Concept – Mumps and Orchitis: Mumps is caused by a paramyxovirus (ssRNA−, enveloped) with a single serotype. It is transmitted by respiratory droplets and saliva. Parotitis is the hallmark, but mumps can involve many glands and organs. **Orchitis** is the most important extra-salivary complication in post-pubertal males, occurring in 20–30% of cases. It presents as acute tender testicular swelling, usually unilateral. **Bilateral orchitis** (occurs in 15–30% of orchitis cases) may cause testicular atrophy and impaired spermatogenesis, rarely resulting in complete sterility. **Unilateral orchitis** is more common and rarely causes sterility.

Other Complications of Mumps:

- Aseptic meningitis: most common CNS manifestation (5–10% of cases); lymphocytic pleocytosis, normal glucose.
- Encephalitis: rare (∼1:6,000); most dangerous CNS complication.
- Oophoritis: in post-pubertal females; rarely causes infertility.
- Sensorineural deafness: usually unilateral; less common than orchitis.
- Pancreatitis: elevated amylase and lipase; mid-epigastric pain.

Key Virological Features:

- Paramyxovirus: enveloped, ssRNA−; HN (haemagglutinin-neuraminidase) and F (fusion) surface glycoproteins.
- Prevention: MMR vaccine (live attenuated); 2 doses.
- Diagnosis: serology (mumps IgM/IgG); RT-PCR of saliva/urine/CSF.

Why other options are wrong:

- Option B: Oophoritis is a complication of mumps in females but is not the most common complication in post-pubertal males.
- Option C: Sensorineural deafness occurs but is less frequent than orchitis in adult males.
- Option D: Encephalitis is a severe but rare complication (1:6,000); orchitis is far more common in post-pubertal males.

Final Answer: post-pubertal male + mumps paramyxovirus + orchitis most common extra-salivary complication; bilateral may cause infertility $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q28

Q29.

Solution

Concept – Congenital Rubella Syndrome (CRS) and Trimester Risk: Rubella is caused by a togavirus (ssRNA+, enveloped). Maternal rubella infection during the **first trimester** poses the greatest risk of congenital rubella syndrome (CRS). The classic triad of CRS is:

- **Cardiac defects**: patent ductus arteriosus (PDA) is most common; pulmonary artery stenosis.
- **Cataracts** (and/or glaucoma, retinopathy — "salt-and-pepper" fundus).
- **Sensorineural deafness** (most common single defect in CRS).

Trimester-Specific Risk of CRS:

- First trimester (weeks 1–12): risk of major defects ~85%; all three triad elements can occur.
- Second trimester (weeks 13–24): risk of deafness and retinopathy; cardiac and ocular defects less common.
- Third trimester: infection rarely causes major structural defects (organogenesis complete).

Other CRS Features:

- Blueberry muffin rash: dermal haematopoiesis (extramedullary erythropoiesis in skin) indicating thrombocytopaenic purpura.
- Microcephaly, mental retardation, neonatal hepatosplenomegaly.
- "Blueberry muffin" also seen in congenital CMV and toxoplasmosis.

Why other options are wrong:

- Option A: Third trimester has the lowest risk of major congenital defects; organogenesis is complete.
- Option C: Asymptomatic maternal rubella infection can still cause CRS; symptoms in the mother are irrelevant to fetal risk.
- Option D: Second trimester carries significantly less risk than the first; the statement of equal risk is incorrect.

Final Answer: first trimester rubella + PDA + cataracts + sensorineural deafness + blueberry muffin $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q29

Q30.

Solution

Concept – Poliovirus and Anterior Horn Cell Destruction: Poliovirus is a member of the Picornaviridae family (genus Enterovirus), a non-enveloped, ssRNA+ virus with three serotypes (PV1, PV2, PV3). It enters via the faecal-oral route and replicates in the oropharynx and small intestine. In $<1\%$ of infections, the virus crosses the blood-brain barrier and infects the **anterior horn cells (lower motor neurons)** of the spinal cord (and motor neurons in the brainstem). Destruction of anterior horn cells causes **flaccid paralysis**: asymmetric, affecting lower motor neurons (LMN) features — hypotonia, areflexia, muscle wasting — with **no sensory loss** (sensory neurons not infected).

Clinical Stages of Poliomyelitis:

- Inapparent infection (90–95%): no symptoms.
- Abortive polio (4–8%): fever, sore throat, myalgia; no neurological involvement.
- Non-paralytic (aseptic meningitis): lymphocytic pleocytosis, meningismus.
- Paralytic polio ($<1\%$): LMN flaccid paralysis; bulbar polio (brainstem) $\rightarrow$ respiratory failure.

Post-polio Syndrome:

- New weakness and fatigue 30–40 years after initial paralytic polio; progressive muscle atrophy.

Why other options are wrong:

- Option A: GBS (Guillain-Barré) is demyelinating, usually ascending, symmetric, with sensory involvement; post-infectious (Campylobacter, CMV); no virus in CSF.
- Option B: Botulism toxin blocks ACh release at NMJ $\rightarrow$ descending flaccid paralysis; no anterior horn cell destruction; normal CSF.
- Option D: Upper motor neuron lesions cause spastic (not flaccid) paralysis with hyperreflexia, Babinski sign; poliovirus does not infect UMN.

Final Answer: poliovirus + anterior horn cell LMN destruction + asymmetric flaccid paralysis + no sensory loss $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q30

Q31.

Solution

Concept – Rotavirus and Childhood Gastroenteritis: Rotavirus (family Reoviridae) is a non-enveloped, double-stranded, segmented RNA virus (11 segments) with a characteristic double-shelled "wheel-like" capsid visible on electron microscopy. It is the **leading global cause of severe dehydrating gastroenteritis in children under 5 years**, responsible for $\sim$200,000 deaths annually. Rotavirus infects mature enterocytes of the small intestinal villi, causing: enterocyte destruction $\rightarrow$ malabsorption + osmotic diarrhoea; NSP4 (non-structural protein 4) acts as a viral enterotoxin, increasing intracellular calcium and stimulating Cl^- secretion (secretory component).

Key Epidemiological Features:

- Winter predominance in temperate regions.
- Highly infectious (ID_{50} <10 viral particles); fomite and faecal-oral spread.
- Reinfection common; adults have partial immunity from prior exposure.
- Vaccines: Rotarix (G1P[8], monovalent, 2 doses) and RotaTeq (pentavalent, 3 doses) — dramatically reduce hospitalisation and mortality.

Why other options are wrong:

- Option A: Norovirus is the most common cause of acute gastroenteritis outbreaks in *all ages* (not specifically severe childhood dehydrating disease); vomiting-predominant.
- Option B: Astrovirus causes mild, self-limited diarrhoea; not the most common cause of severe dehydrating diarrhoea.

- Option C: Adenovirus 40/41 is the second most common cause of viral gastroenteritis in infants (year-round), but causes a milder, more prolonged illness.

Final Answer: wheel-like double-shelled dsRNA + winter + children <5 + severe dehydrating diarrhoea + NSP4 enterotoxin $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q31

Q32.

Solution

Concept – Norovirus and Epidemic Gastroenteritis: Norovirus (family Caliciviridae, genus Norovirus, formerly "Norwalk-like virus") is a non-enveloped, positive-sense ssRNA virus. It is the **most common cause of acute epidemic (non-bacterial) gastroenteritis in all age groups worldwide**, causing $\sim$685 million cases annually. Key characteristics:

- **Vomiting-predominant** illness: sudden onset projectile vomiting, watery diarrhoea, low-grade fever.
- **Short duration**: 24–48 hours; self-limiting (rarely fatal, except in the elderly or immunocompromised).
- **Transmission**: fomites (highly stable on surfaces), contaminated food (shellfish, salad), water, droplets from vomiting; cruise ships, nursing homes, schools.
- **Infectious dose**: extremely low ($<$18 viral particles).
- Norovirus does NOT respond to antibiotics; treatment is supportive (oral rehydration).

Diagnosis:

- RT-PCR from stool or environmental swabs (most sensitive).
- EIA (less sensitive, not preferred for outbreaks).
- EM: "Star of David" appearance (calicivirus morphology).

Why other options are wrong:

- Option B: Rotavirus causes severe dehydrating diarrhoea predominantly in infants/toddlers; winter predominance; not the classic cruise-ship/epidemic agent in adults.
- Option C: Hepatitis E virus causes icteric hepatitis (jaundice + elevated liver enzymes); NOT gastroenteritis; waterborne; no vomiting-predominant picture.

- Option D: Astrovirus causes mild self-limited diarrhoea mainly in young children; not responsible for large adult outbreaks on cruise ships.

Final Answer: cruise ship + explosive vomiting + 24–48 h + fomite spread + ssRNA calicivirus $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q32

Q33.

Solution

Concept – *Plasmodium vivax* and Relapsing Malaria: *P. vivax* causes tertian (48-hour) malaria. Its hallmark is the formation of **hypnozoites** — dormant forms in hepatocytes that persist for months to years after primary infection. Reactivation of hypnozoites causes true relapses (distinct from recrudescence, which is caused by persistence of blood-stage parasites). RBC invasion by *P. vivax* merozoites requires the **Duffy antigen receptor for chemokines (DARC, FY blood group)** on red blood cells. Individuals with the Duffy-negative phenotype (common in West Africa: $DARC^{-}DARC^{-}$ homozygous) are naturally protected against *P. vivax* infection.

Distinguishing Features of *P. vivax*:

- Blood smear: enlarged RBCs + Schüffner's stippling (eosinophilic dots) + ameboid trophozoite.
- Fever pattern: tertian (every 48 hours); corresponds to synchronised RBC rupture cycle.
- **Treatment**: chloroquine (blood stage) + **primaquine** or tafenoquine (liver hypnozoites, must test for G6PD deficiency first).

Why other options are wrong:

- Option A: *P. falciparum* causes knob formation (RBC cytoadherence, rosetting), cerebral malaria; no relapse (no hypnozoites); most lethal.
- Option C: *P. malariae* causes quartan (72-hour) fever; recrudesces (no hypnozoites); nephropathy; does not truly relapse.
- Option D: *P. ovale* also forms hypnozoites and relapses; it does show Schüffner's stippling and oval/fimbriated RBCs; Duffy antigen not required.

Final Answer: tertian fever + Schüffner's stippling + enlarged RBCs + relapse 6 months later + hypnozoites + Duffy antigen + primaquine $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q33

Q34.

Solution

Concept – *Entamoeba histolytica* vs *E. dispar*: Both *E. histolytica* and *E. dispar* are morphologically **identical** under light microscopy. Both produce cysts with 4 nuclei and peripheral chromatin with a central karyosome, and chromatoid bodies with **blunt, cigar-shaped (rounded)** ends. Only *E. histolytica* causes invasive disease (amoebic dysentery, amoebic liver abscess with "anchovy sauce" contents). *E. dispar* is non-pathogenic and far more common worldwide. The two species are distinguished by:

- **PCR**: most sensitive and specific for identifying pathogenic species.
- **Antigen detection (ELISA)**: using species-specific monoclonal antibodies against lectin (Gal/GalNAc) or other antigens.
- Trophozoites with ingested RBCs (erythrophagocytosis): characteristic of *E. histolytica* only.

Key Clinical Features of Amoebiasis (*E. histolytica*):

- Amoebic dysentery: bloody mucoid diarrhoea, crampy pain, flask-shaped ulcers in caecum and ascending colon.
- Amoebic liver abscess: right lobe; single; "anchovy sauce" (brown liquefied necrotic material, odourless).
- Serology: amoebic indirect haemagglutination or ELISA (positive in liver abscess, often negative in intestinal disease).
- Treatment: metronidazole (invasive disease) + diloxanide furoate or iodoquinol (luminal cysts).

Why other options are wrong:

- Option A: Both *E. histolytica* and *E. dispar* have 4-nucleated cysts; an 8-nucleated cyst belongs to *E. coli* (non-pathogenic, unrelated species).
- Option B: Both species have blunt chromatoid bodies — this does not distinguish them from each other.
- Option D: *E. dispar* is the non-pathogenic species; *E. histolytica* causes liver abscess.

Final Answer: 4-nucleated cysts + blunt chromatoid bodies + liver abscess + *E. histolytica* vs *E. dispar* only by PCR/ELISA $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q34

Q35.

Solution

Concept – African Sleeping Sickness (*Trypanosoma brucei*): Human African trypanosomiasis (HAT) is caused by *T. brucei* subspecies transmitted by the bite of the ***Glossina* (tsetse) fly**. Two subspecies:

- ***T. b. gambiense***: West and Central Africa; ∼97% of HAT cases; slow progression; chronic encephalitic stage may develop after months–years.
- ***T. b. rhodesiense***: East Africa; ∼3% of cases; rapid, acute progression; CNS involvement within weeks; more virulent.

Clinical Stages:

- Stage 1 (haemolymphatic): trypanosomal chancre at bite site; fever, lymphadenopathy (Winterbottom's sign: posterior cervical lymphadenopathy in *T. b. gambiense*); skin rash.
- Stage 2 (meningoencephalitic): CNS invasion; daytime somnolence + night-time insomnia (disrupted circadian rhythm); confusion, behavioural changes, seizures, coma; fatal if untreated.

Diagnosis:

- Blood smear: trypomastigotes with kinetoplast, undulating membrane.
- CSF: pleocytosis, morula cells (Mott cells), trypomastigotes confirm Stage 2.
- Card agglutination test (CATT) for *T. b. gambiense* screening.

Why other options are wrong:

- Option A: *T. cruzi* is transmitted by *Triatoma* (reduviid/kissing bug); Chagas disease; cardiac cardiomyopathy and megaoesophagus; no CNS somnolence.
- Option B: *Leishmania donovani* is transmitted by *Phlebotomus* sand fly; kala-azar; amastigotes in macrophages; no trypomastigotes.
- Option C: *T. b. gambiense* causes the slower West African form; the question describes a rapidly progressive East African form but the key identification of *Glossina* vector and CNS stage is correct for both — Option D is more complete and specifically identifies the rhodesiense subspecies matching the acute scenario.

Final Answer: tsetse fly + CNS somnolence + trypomastigotes in CSF + kinetoplast + fatal if untreated $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q35

Q36.

Solution

Concept – Visceral Leishmaniasis / Kala-Azar (*Leishmania donovani*): *L. donovani* causes visceral leishmaniasis (kala-azar, Hindi for "black fever"). It is transmitted by the bite of the female ***Phlebotomus* sand fly** (in Asia and Europe) or *Lutzomyia* (in South America). Promastigotes (flagellated, in sand fly gut) are injected into skin, phagocytosed by macrophages, and transform to **amastigotes** (non-flagellated, oval, with visible kinetoplast — the lateral bar). Amastigotes multiply within macrophages throughout the reticuloendothelial system (spleen, liver, bone marrow, lymph nodes).

Classic Clinical Features:

- Prolonged irregular fever (Pel-Ebstein pattern); massive splenomegaly (most prominent finding); hepatomegaly.
- Progressive wasting (cachexia), anaemia, leucopaenia, thrombocytopaenia (pancytopaenia from bone marrow infiltration).
- **Hyperpigmentation** (darkening of skin, especially in Indians — the origin of the name "kala-azar").
- Markedly elevated serum globulins (IgG polyclonal) $\rightarrow$ positive aldehyde test (formal-gel test): serum solidifies when mixed with 1 drop of formalin.

Why other options are wrong:

- Option B: *L. tropica* causes cutaneous leishmaniasis (oriental sore): dry, self-healing ulcer; no visceral involvement.
- Option C: *L. braziliensis* causes mucocutaneous leishmaniasis (espundia): destructive ulcers of nasal/oral mucosa; South America; *Lutzomyia* vector.
- Option D: *T. brucei* causes sleeping sickness via tsetse fly; entirely different clinical and pathological picture.

Final Answer: Bihar + massive splenomegaly + amastigotes in macrophages + *Phlebotomus* + hyperpigmentation + aldehyde test $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q36

Q37.

Solution

Concept – *Strongyloides stercoralis* Autoinfection and Hyperinfection: *Strongyloides stercoralis* has a unique life cycle feature among human helminths:

autoinfection. Normally, rhabditiform larvae passed in faeces develop into infective filariform larvae in soil and penetrate intact skin. In the autoinfection cycle, some rhabditiform larvae within the gut lumen or perianal skin accelerate development to filariform larvae **before leaving the host**, penetrate the intestinal mucosa or perianal skin, and re-enter circulation. This allows *S. stercoralis* to persist indefinitely within the host without re-exposure.

Hyperinfection Syndrome (in immunocompromised):

- Trigger: HTLV-1 infection, steroid use, haematological malignancy, solid organ transplantation.
- Massive amplification of larval load: filariform larvae penetrate gut wall en masse.
- Larvae carry enteric bacteria (*E. coli*, *Klebsiella*) into mesenteric vessels $\rightarrow$ gram-negative bacteraemia and septicaemia.
- Dissemination to lungs (Loeffler-like pneumonitis), CNS (meningitis), liver, heart.
- Mortality: $>$80% if untreated.

Diagnosis and Treatment:

- Stool examination: rhabditiform larvae (NOT eggs, unlike other helminths).
- Duodenal aspirate or string test; serology (ELISA).
- Treatment: ivermectin (first-line); albendazole (alternative).

Why other options are wrong:

- Option A: Cysticercosis (encystation in muscle/brain) is caused by *T. solium*; not *Strongyloides*.
- Option C: Loeffler's syndrome (pulmonary eosinophilia) occurs during larval lung migration in *Ascaris*, *hookworm*, and *Strongyloides* — but in immunocompromised, hyperinfection with septicaemia is the key concern, not Loeffler's alone.
- Option D: Lymphoedema (elephantiasis) is caused by *Wuchereria bancrofti* or *Brugia* spp. (filarial nematodes); adult worms live in lymphatics.

Final Answer: prednisolone + rhabditiform larvae + gut-wall penetration + gram-negative septicaemia + CNS dissemination + autoinfection/hyperinfection $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q37

Q38.

Solution

Concept – *Taenia solium*: Taeniasis vs Neurocysticercosis: *Taenia solium* (pork tapeworm) can involve humans in two very different ways:

- **Taeniasis** (intestinal): Humans are the definitive host; caused by eating **undercooked pork containing cysticerci**; adult tapeworm (scolex with 4 suckers + double row of hooks) attaches to jejunum; shedding of proglottids in stool; usually asymptomatic or mild GI symptoms.
- **Cysticercosis / Neurocysticercosis**: Humans become the **accidental intermediate host** by ingesting **eggs** (faecal-oral route: auto-infection via ano-oral route, or ingesting food contaminated by an adult tapeworm carrier's faeces); eggs hatch, oncospheres penetrate gut wall, disseminate haematogenously to brain, muscle, eye, and SC; larvae encyst as *Cysticercus cellulosae*; in the brain: ring-enhancing cystic lesions $\rightarrow$ seizures, increased ICP, hydrocephalus.

Diagnosis and Treatment:

- MRI: cystic lesions with scolex ("hole-with-dot" sign); calcified lesions indicate dead larvae.
- Serology: ELISA/western blot for cysticercal antigens.
- Stool exam: proglottids or eggs in carrier (auto-infection risk).
- Treatment: albendazole + dexamethasone (anti-inflammatory); surgery if single accessible lesion.

Why other options are wrong:

- Option A: *T. saginata* (beef tapeworm) has no hooklets and NEVER causes cysticercosis in humans; cysts in cattle only.
- Option B: *Echinococcus granulosus* causes cystic hydatid disease (unilocular cyst in liver/lungs); has daughter cysts within a mother cyst with brood capsules containing protoscolices; no scolex within brain lesion.
- Option D: *T. solium* does not cause hydatid disease; hydatid disease is caused by *Echinococcus*; and it affects multiple organs (brain, eye, spinal cord), not just liver.

Final Answer: seizures + ring-enhancing cysts with scolex + proglottids in stool + human as accidental intermediate host via egg ingestion $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q38

Q39.

Solution

Concept – Cryptococcal Meningitis (*Cryptococcus neoformans*): *C. neoformans* is an encapsulated yeast (not dimorphic) found in pigeon droppings and soil enriched by bird and bat guano. Infection occurs via inhalation of desiccated yeast or basidiospores; primary pulmonary focus often asymptomatic. In immunocompromised individuals (especially AIDS with CD4 $<$200, typically $<$100), haematogenous spread causes **cryptococcal meningoencephalitis** — a subacute, indolent infection with elevated intracranial pressure as the main cause of morbidity and mortality.

Virulence Factors:

- **Polysaccharide capsule** (GXM — glucuronoxylomannan): anti-phagocytic, inhibits complement activation and T-cell proliferation, causes elevated ICP.
- **Melanin production** (phenoloxidase/laccase): protects against oxidative killing by macrophages; tropism for CNS (dopamine-rich environment).
- **Ability to grow at 37°C**: enables systemic infection.

Diagnostic Tests:

- CSF India ink: visualises large polysaccharide capsule (halo effect around yeast cell). Sensitivity 50–80%.
- **Cryptococcal antigen (CrAg)** latex agglutination or LFA: sensitivity $>$99%; can be done on CSF, serum, or urine.
- Culture on Sabouraud agar: cream-coloured mucoid colonies; urease positive; niger seed agar: brown melanin-producing colonies.

Treatment:

- Induction: amphotericin B deoxycholate + flucytosine (2 weeks).
- Consolidation: fluconazole 400 mg/day (8 weeks).
- Maintenance: fluconazole 200 mg/day until immune reconstitution.

Why other options are wrong:

- Option A: *Candida albicans* causes germ tube (Filamentation at 37°C in serum); pseudohyphae; oral thrush and candidaemia; not encapsulated; India ink negative.
- Option B: *Aspergillus fumigatus* has septate hyphae, 45° branching, angioinvasion; not a yeast; no India ink capsule.

- Option C: *Histoplasma capsulatum* is dimorphic; intracellular in macrophages; Ohio/Mississippi River Valley; not encapsulated in the manner detected by India ink.

Final Answer: AIDS + CD4 <50 + pigeon droppings + India ink capsule + CrAg positive + elevated ICP + polysaccharide capsule $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q39

Q40.

Solution

Concept – Histoplasmosis (*Histoplasma capsulatum*): *H. capsulatum* is a thermally dimorphic fungus:

- At **25°C (room temperature/mould phase)**: grows as white-to-brown cottony mould producing characteristic **tuberculate macroconidia** (large, thick-walled, finger-like projections) and microconidia (infectious form).
- At **37°C (body temperature/yeast phase)**: small (2–4 μm), oval yeast cells that are obligately **intracellular in macrophages** (replicating within phagolysosomes by resisting acidification).

Epidemiology:

- Endemic: **Ohio and Mississippi River Valleys** (USA); also Central and South America.
- **Ecological niche**: soil enriched by **bat and bird (especially starling) droppings**; cave exploration, demolition of old buildings, chicken coops.
- Infection: inhalation of microconidia; primary pulmonary focus (resembles TB on CXR: bilateral hilar lymphadenopathy, calcification).

Diagnosis and Treatment:

- Urine and serum Histoplasma polysaccharide antigen (HPA): best for disseminated disease.
- Bone marrow or blood culture (lysis-centrifugation); bone marrow biopsy showing yeast in macrophages.
- Complement fixation (CF) serology: $\geq$1:32 significant; *M* and *H* precipitin bands on immunodiffusion.
- Treatment: mild-moderate — itraconazole; severe disseminated — amphotericin B then itraconazole.

Why other options are wrong:

- Option B: *B. dermatitidis* is dimorphic; yeast phase shows large (8–15 μm) thick-walled cells with broad-based budding; Great Lakes region and Mississippi Valley; lung + skin $\pm$ bone disease; tuberculate macroconidia are absent.
- Option C: *Coccidioides immitis*: spherules (20–60 μm) with endospores in tissue; endemic in South-western USA (San Joaquin Valley) and Latin America; arthroconidia in mould phase; no intracellular yeast in macrophages.
- Option D: *Paracoccidioides brasiliensis*: "captain's wheel" or "steering wheel" multi-budding yeast; South America; coffee and sugarcane farmers; not associated with bat caves in Ohio.

Final Answer: bat cave + Ohio/Mississippi + intracellular yeast in macrophages + tuberculate macroconidia + urine antigen positive $\Rightarrow \boxed{A}$

Answer: (A) **Go Back to Q40**

Answer Key – FMGE Microbiology Sample Paper 2

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