MEDICINE • INFECTIOUS DISEASES

CROSS-CHAPTER PRINCIPLES

IN INFECTIOUS DISEASES

Antimicrobial Stewardship • Antimicrobial Resistance

Infection Control • Public Health • Prognostic Assessment

With Master Lists of Essential Tables & Figures

MedMentor EDU

Medicine Study Notes | MBBS • NEET-PG • INI-CET • DNB • FMGE

Reference texts: Harrison | Davidson | Kumar & Clark | API Textbook of Medicine

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SECTION 1 | Antimicrobial Stewardship

1.1 Definition and Core Concept

Antimicrobial stewardship is a coordinated set of interventions designed to improve and measure the appropriate use of antimicrobials by promoting the optimal drug, dose, route and duration of therapy — with the aim of achieving the best clinical outcome while minimising toxicity, cost and the emergence of resistance.

Simplified: give the right antibiotic, to the right patient, at the right dose, by the right route, for the right duration — and stop it at the right time.

The 5 Ds of Stewardship

- Right Diagnosis — is this truly an infection?
- Right Drug — narrowest effective agent

- Right Dose — PK/PD-guided, weight- and organ-adjusted
- Right Duration — shortest evidence-based course
- Right De-escalation — narrow or stop on culture data

Exam Pearl

- Stewardship is NOT simply "use fewer antibiotics" — it is "use antibiotics appropriately".
- In septic shock, stewardship demands EARLY and BROAD therapy first, then rapid de-escalation.
- Two pillars: **Stewardship** (right use) + **Infection Control** (prevent spread) — both are needed to contain resistance. **[VERY HIG**

1.2 Indication for Therapy

- The first stewardship question is always: is antimicrobial therapy indicated at all?
- Distinguish infection from colonization and contamination before prescribing.
- Fever alone is NOT an indication for antibiotics — many fevers are non-infectious or viral.

Situations where antibiotics are commonly given but NOT indicated

- Asymptomatic bacteriuria (except pregnancy and before urological procedures with mucosal breach)
- Positive respiratory culture without clinical/radiological pneumonia (tracheal colonization)
- Colonized chronic wounds and ulcers without cellulitis or systemic features
- Upper respiratory tract infection, acute bronchitis and most sore throats (viral)
- Simple viral gastroenteritis
- Single positive blood culture growing coagulase-negative Staphylococcus in a patient without a device

Common Error

- Treating a positive culture rather than treating the patient.
- Every positive microbiological result must be interpreted against the clinical picture.

1.3 Empirical Therapy

Empirical therapy is antimicrobial treatment started before the causative organism and its susceptibility are known, based on the most likely pathogens for that clinical syndrome.

Determinants of empirical choice

- Clinical syndrome and probable anatomical source
- Most likely pathogens for that site
- Severity of illness (sepsis / septic shock ? broader initial cover)
- Community-acquired versus healthcare-associated infection
- Local antibiogram and unit-specific resistance patterns
- Prior antimicrobial exposure in the preceding 90 days
- Prior colonization or infection with resistant organisms
- Host factors — age, pregnancy, renal and hepatic function, immunosuppression, allergy
- Tissue penetration required (CNS, bone, prostate, abscess, vegetations)
- Drug interactions and toxicity profile

Clinical Note — Timing

- Septic shock: first dose within 1 hour of recognition.
- Bacterial meningitis: antibiotics within 1 hour; do not delay for CT or LP.
- Neutropenic fever: antipseudomonal beta-lactam within 1 hour.
- Necrotizing fasciitis: immediate broad therapy + emergency surgical exploration.

1.4 Culture Before Treatment

- Obtain all relevant microbiological specimens BEFORE the first antimicrobial dose whenever feasible.
- Two sets of blood cultures (aerobic + anaerobic) from separate venepuncture sites; adequate volume is the single most important determinant of yield (8–10 mL per adult bottle).
- Site-directed specimens: urine, sputum, CSF, pus, aspirates, tissue, stool, swabs from the deep part of a wound.
- Specimens should be transported promptly; CSF and specimens for Neisseria must not be refrigerated.
- Antibiotics must NOT be delayed beyond 45 minutes in septic shock while awaiting specimen collection.

Exam Pearl

- Cultures before antibiotics — but never let culture collection delay therapy in septic shock, meningitis, severe malaria or neutrop fever. **[VERY HIGH-YIELD]**
- Blood cultures become sterile within hours of the first dose; a single missed opportunity may make the diagnosis unprovable for admission.

1.5 Local Antibiogram

An antibiogram is a periodic (usually annual) cumulative summary of the in-vitro susceptibility rates of organisms isolated in a defined population or unit.

- Guides rational empirical therapy for each institution and each unit (ICU antibiograms differ from ward antibiograms).
- Should be unit-specific, specimen-specific and updated at least annually.
- Traditional rule of thumb: an agent is suitable for empirical use if local susceptibility exceeds approximately 80–90% for that syndrome.
- Detects emerging resistance trends and informs formulary restriction policies.

1.6 Appropriate Spectrum

- Use the narrowest agent that reliably covers the likely pathogens.
- Broad-spectrum therapy is justified initially in severe sepsis, neutropenia and known resistant colonization — but must be reviewed within 48–72 hours.
- Unnecessary broad spectrum drives Clostridioides difficile infection, candidiasis and multidrug resistance.
- Highest C. difficile risk: clindamycin, third/fourth-generation cephalosporins, fluoroquinolones, broad-spectrum penicillins ("the 4 Cs" — clindamycin, cephalosporins, co-amoxiclav, ciprofloxacin).

Viva Point

- Q: Why is a narrow-spectrum agent preferred once cultures return?
- A: Equal efficacy, less collateral damage to commensal flora, lower risk of C. difficile and resistance, lower cost and toxicity.

1.7 Loading Dose

A loading dose is a larger initial dose given to rapidly achieve therapeutic plasma and tissue concentrations; it is determined by the volume of distribution, NOT by clearance.

- Loading dose is NOT reduced in renal or hepatic impairment — only maintenance doses are adjusted.
- Critically ill and septic patients have an expanded volume of distribution (capillary leak, aggressive fluid resuscitation) and often need HIGHER loading doses.
- Drugs commonly requiring a loading dose: vancomycin, teicoplanin, colistin (colistimethate), amikacin, caspofungin, voriconazole, artesunate, anti-tubercular therapy is an exception (no loading).

Exam Pearl

- Loading dose depends on volume of distribution; maintenance dose depends on clearance. Therefore the loading dose is unchang renal failure. **[FAVORITE EXAM QUESTION]**

1.8 Renal Adjustment

- Adjust maintenance dose or dosing interval according to estimated creatinine clearance (Cockcroft–Gault preferred for drug dosing).
- Adjustment may be by dose reduction, interval extension, or both.
- Remember augmented renal clearance in young trauma, burns and post-partum patients — these patients may be UNDER-dosed at standard doses.
- Account for renal replacement therapy: many agents are cleared by haemodialysis and require post-dialysis dosing.

Renal handling	Representative agents

Require dose reduction	• Aminoglycosides • Vancomycin • Most beta-lactams • Fluconazole • Acyclovir • Colistin • Ethambutol • Levofloxacin • Cotrimoxazole
Usually no adjustment	• Ceftriaxone • Azithromycin • Doxycycline • Clindamycin • Linezolid • Moxifloxacin • Rifampicin • Isoniazid • Metronidazole (mild-moderate)
Avoid / caution in renal failure	• Nitrofurantoin (ineffective, neurotoxic) • Tetracycline • Aminoglycosides where alternatives exist

Nephrotoxicity Watch

- Aminoglycosides, vancomycin (especially with piperacillin–tazobactam), amphotericin B deoxycholate, colistin, high-dose acyclovir (crystal nephropathy), cotrimoxazole, tenofovir.
- Monitor creatinine, urine output and electrolytes; ensure adequate hydration.

1.9 Hepatic Adjustment

- No validated formula equivalent to creatinine clearance exists; use Child–Pugh class and clinical judgement.
- Agents predominantly cleared hepatically or biliary excreted require caution: rifampicin, isoniazid, pyrazinamide, macrolides (erythromycin, clarithromycin), metronidazole, clindamycin, chloramphenicol, tigecycline, caspofungin, voriconazole, ceftriaxone (biliary sludging).
- Hepatotoxic antimicrobials: anti-tubercular drugs (INH, rifampicin, pyrazinamide), co-amoxiclav (cholestatic), flucloxacillin, azoles, nitrofurantoin, tetracyclines.
- Monitor liver enzymes where prolonged therapy with hepatotoxic drugs is planned.

1.10 Pharmacokinetics

Pharmacokinetics (PK) describes what the body does to the drug — absorption, distribution, metabolism and excretion.

- Absorption — oral bioavailability; impaired by ileus, vomiting, shock and gut oedema; chelation of fluoroquinolones and tetracyclines by calcium, iron, magnesium and antacids.
- Distribution — volume of distribution; protein binding (only free drug is active); tissue penetration.
- Metabolism — hepatic; cytochrome P450 interactions (rifampicin is a potent inducer; azoles and macrolides are inhibitors).
- Excretion — predominantly renal for beta-lactams, aminoglycosides, glycopeptides.

Sanctuary sites with poor antimicrobial penetration

- Central nervous system — requires agents crossing the blood–brain barrier at high dose (ceftriaxone, meropenem, vancomycin, linezolid, metronidazole, fluconazole)
- Abscess cavities — low pH, low oxygen tension and high bacterial inoculum; drainage is essential
- Prostate, bone, vegetations, biofilm on prosthetic material and vitreous cavity

1.11 Pharmacodynamics

Pharmacodynamics (PD) describes what the drug does to the organism — the relationship between drug concentration and antimicrobial killing.

PD pattern	Key index	Representative drugs

Time-dependent	T > MIC (aim 40–70% of the dosing interval)	• Penicillins • Cephalosporins • Carbapenems • Aztreonam • Linezolid
Concentration-dependent	Cmax / MIC (aim ? 8–10)	• Aminoglycosides • Daptomycin • Metronidazole • Polymyxins
Concentration-dependent with time dependence (AUC-driven)	AUC24 / MIC	• Vancomycin • Fluoroquinolones • Azithromycin • Tetracyclines

- Practical consequence of time dependence: beta-lactams benefit from more frequent dosing, extended (3–4 h) or continuous infusion in severe sepsis.
- Practical consequence of concentration dependence: aminoglycosides are given as a single high daily dose, which also reduces nephrotoxicity.
- Post-antibiotic effect — persistent suppression of growth after concentrations fall below the MIC; prominent with aminoglycosides and carbapenems against Gram-negative bacilli.

Exam Pearl

- Beta-lactams = time-dependent (T > MIC). Aminoglycosides = concentration-dependent (Cmax/MIC). Vancomycin and fluoroq = AUC/MIC. **[COMMON MCQ]**

1.12 Therapeutic Drug Monitoring (TDM)

TDM is measurement of drug concentrations to individualise dosing where the therapeutic window is narrow, toxicity is concentration-related, or PK is unpredictable.

Drug	What is monitored / target
Vancomycin	• AUC24/MIC 400–600 preferred; trough 15–20 mg/L if trough-based • Sample immediately before the 4th dose
Aminoglycosides	• Trough (predicts nephro/ototoxicity): gentamicin < 1 mg/L • Peak predicts efficacy
Voriconazole	Trough 1–5.5 mg/L — wide interpatient variability; toxicity = visual and neu
Teicoplanin	Trough ? 15–20 mg/L (higher in endocarditis)
Flucytosine	Peak < 100 mg/L — marrow toxicity
Colistin, linezolid	Increasingly monitored in critical illness and renal impairment

1.13 De-escalation

De-escalation is the deliberate narrowing or stopping of empirical broad-spectrum therapy once microbiological and clinical data become available — usually at 48–72 hours.

De-escalation Pathway

Empirical broad-spectrum therapy started

?

Review at 48–72 hours ("antibiotic time-out")

?

Culture positive? Clinical improvement?

- *Organism identified with susceptibilities ? switch to narrowest effective agent*
- *Cultures negative + patient improved + infection unlikely ? STOP antibiotics*
- *Cultures negative but infection likely ? continue targeted therapy, seek the source*
- *Not improving ? reconsider diagnosis, resistant organism, undrained focus, non-bacterial cause*

Document decision, agent, and planned stop date

- De-escalation has been shown to be safe and does not increase mortality.
- Procalcitonin-guided algorithms may support earlier discontinuation in respiratory infection and sepsis, but must never override clinical judgement.

1.14 Intravenous-to-Oral Switch

COMS criteria for switching

- C — Clinical improvement observed
- O — Oral route not compromised (able to swallow, no vomiting, no malabsorption, no ileus)
- M — Markers trending towards normal (afebrile > 24–48 h, falling WCC and CRP, haemodynamic stability)
- S — Specific indication for continued IV therapy absent

Infections requiring prolonged IV therapy (do not switch early)

- Infective endocarditis, Staphylococcus aureus bacteraemia
- Bacterial meningitis and brain abscess
- Osteomyelitis and septic arthritis (early phase)
- Necrotizing soft-tissue infection, mediastinitis

- Undrained empyema or abscess; prosthetic device infection
- Neutropenic fever until count recovery and clinical stability

Highly bioavailable oral agents suitable for switch

- Fluoroquinolones (levofloxacin, ciprofloxacin, moxifloxacin)
- Linezolid, metronidazole, fluconazole, voriconazole
- Cotrimoxazole, doxycycline, clindamycin, rifampicin, isoniazid

Exam Pearl

- Linezolid, metronidazole, fluconazole and fluoroquinolones have oral bioavailability approaching 100% — oral dose equals IV **[VERY HIGH-YIELD]**
- Benefits of switching: removes line-related bacteraemia risk, allows earlier discharge, reduces cost and nursing workload.

1.15 Duration of Therapy

- Modern evidence consistently supports SHORTER courses — "shorter is better" for most uncomplicated infections.
- Duration should be defined and documented at the time of prescribing.
- Prolonged therapy increases toxicity, C. difficile infection and resistance without improving outcome.

Infection	Typical duration
Community-acquired pneumonia	5 days (if afebrile 48 h and clinically stable)
Hospital-acquired / ventilator-associated pneumonia	7 days
Uncomplicated cystitis (female)	3 days (nitrofurantoin 5 days)
Acute pyelonephritis	7 days (fluoroquinolone) to 10–14 days

Cellulitis	5–6 days if responding
Intra-abdominal infection with adequate source control	4–7 days
Gram-negative bacteraemia	7 days (if source controlled and responding)
Staphylococcus aureus bacteraemia	14 days uncomplicated; 4–6 weeks if complicated
Native-valve infective endocarditis	4–6 weeks
Osteomyelitis	6 weeks
Bacterial meningitis	• Meningococcal 5–7 d • Pneumococcal 10–14 d • Listeria 21 d
Neutropenic fever	Until neutrophil recovery and clinical resolution

Exam Pearl

- 7 days is non-inferior to 14 days for Gram-negative bacteraemia with source control. **[COMMON MCQ]**
- Staphylococcus aureus bacteraemia is the classic exception — never treat as a short course; always seek metastatic foci and obtain echocardiography.

1.16 Avoidance of Duplicate Coverage

Duplicate (redundant) therapy is the concurrent use of two or more agents with overlapping spectra where the combination provides no additional benefit.

Classic examples of redundant combinations

- Double anaerobic cover — piperacillin–tazobactam or a carbapenem PLUS metronidazole
- Double Gram-negative cover — a carbapenem PLUS a fluoroquinolone in a stable patient
- Double anti-MRSA cover — vancomycin PLUS linezolid or daptomycin
- Double atypical cover — a macrolide PLUS a respiratory fluoroquinolone
- Metronidazole added to co-amoxiclav or to a carbapenem

When combination therapy IS justified

- To broaden empirical cover in severe sepsis of unknown source
- To achieve synergy — e.g. beta-lactam plus aminoglycoside in enterococcal endocarditis
- To prevent emergence of resistance — tuberculosis, HIV, Helicobacter pylori
- Polymicrobial infection where no single agent covers all pathogens
- Toxin suppression — clindamycin added in streptococcal toxic shock and necrotizing fasciitis

1.17 Documentation

Every antimicrobial prescription must record:

- Indication (clinical syndrome or organism)
- Drug, dose, route and frequency
- Date and time of the first dose
- Planned duration or stop date
- Allergy status and the nature of the reaction
- Cultures sent and results awaited
- Whether the prescription complies with local guidelines, and the reason if it does not

Viva Point

- Q: Why is documenting the indication so important?
- A: It permits meaningful review at 48–72 hours, prevents indefinite continuation by successive teams, and is the single most reli quality metric in stewardship audit.

1.18 Review Date

- A mandatory prescription review ("antibiotic time-out") is performed at 48–72 hours.
- Automatic stop orders and prescription-chart expiry dates prevent inadvertent continuation.
- The Start Smart – Then Focus framework defines five possible outcomes at review.

Start Smart – Then Focus: Five Decisions at 48–72 Hours

STOP — no evidence of infection

?

SWITCH — intravenous to oral

?

CHANGE — narrow (de-escalate) or broaden per culture

?

CONTINUE — with documented review date

?

OPAT — outpatient parenteral antimicrobial therapy

1.19 Source Control

Source control refers to all physical measures used to eliminate a focus of infection, control ongoing contamination, and restore anatomy and function.

- Antimicrobials CANNOT sterilise an undrained collection, necrotic tissue, or an infected foreign body.
- Failure to improve on appropriate antibiotics should immediately raise suspicion of inadequate source control.
- Should be achieved as early as feasible — generally within 6–12 hours of diagnosis in sepsis.

Modality	Examples
Drainage	Percutaneous or surgical drainage of abscess, empyema, pyonephrosis, cholangitis (ERCP)
Debridement	Necrotizing fasciitis, infected pancreatic necrosis, diabetic foot, osteomyelitis sequestrectomy
Device removal	Infected central venous catheter, urinary catheter, prosthetic joint, pacemaker lead, ventriculoperitoneal shunt
Definitive control	Bowel resection for perforation, cholecystectomy, amputation, repair of anastomotic leak
Diversion / decompression	Stoma formation, ureteric stent or nephrostomy, biliary stent

Critical Concept

- Source control is a central component of sepsis management — without it, no antibiotic regimen will succeed. **[VERY HIGH-YIELD]**

1.20 Measuring the Impact of Stewardship

- Process measures — guideline compliance, documentation of indication, timeliness of first dose, de-escalation and IV-to-oral switch rates
- Consumption measures — defined daily doses (DDD) or days of therapy (DOT) per 1000 patient-days
- Outcome measures — mortality, length of stay, readmission, C. difficile rates, resistance rates
- AWaRe classification (WHO) — Access, Watch and Reserve antibiotics; the target is that at least 60% of consumption comes from the Access group

Exam Pearl — WHO AWaRe

- ACCESS — first-line, narrow spectrum, low resistance potential (amoxicillin, cotrimoxazole, gentamicin, doxycycline)
- WATCH — higher resistance potential, restricted (fluoroquinolones, third-generation cephalosporins, macrolides, carbapenems, glycopeptides)
- RESERVE — last resort for MDR infections (colistin, linezolid, ceftazidime–avibactam, daptomycin, tigecycline)

SECTION 2 | Antimicrobial Resistance

2.1 Definitions and Terminology

Antimicrobial resistance (AMR) is the ability of a micro-organism to survive and multiply in the presence of an antimicrobial agent at concentrations that would normally inhibit or kill it.

Term	Definition
MDR (Multidrug-resistant)	Non-susceptible to at least one agent in ? 3 antimicrobial classes
XDR (Extensively drug-resistant)	Non-susceptible to at least one agent in all but ? 2 classes
PDR (Pandrug-resistant)	Non-susceptible to all agents in all classes
Intrinsic resistance	Natural, species-wide (e.g. Enterococcus to cephalosporins; Klebsiella to am
Acquired resistance	Mutation or horizontal gene transfer (plasmids, transposons, integrons)
Tolerance	Organism survives but does not grow — MIC unchanged, MBC greatly raise
Breakpoint	MIC value separating susceptible from resistant (CLSI / EUCAST)

Mechanisms of Resistance — Four Broad Groups

- Enzymatic inactivation — beta-lactamases, aminoglycoside-modifying enzymes, chloramphenicol acetyltransferase

- Target modification — altered PBP (MRSA), altered ribosome (macrolide erm methylase), altered DNA gyrase (quinolones), altered peptidoglycan terminus (VRE)
- Reduced permeability — loss of outer-membrane porins (OprD loss in Pseudomonas causing carbapenem resistance)
- Active efflux — MexAB-OprM in Pseudomonas, tet efflux pumps

Viva Point — Global Drivers of AMR

- Inappropriate and over-prescription in humans
- Over-the-counter availability and incomplete courses
- Agricultural and veterinary use as growth promoters
- Poor infection control, overcrowding and inadequate sanitation
- International travel and medical tourism
- Dry antibiotic development pipeline

2.2 MRSA — Methicillin-Resistant Staphylococcus aureus

Mechanism

- mecA gene (rarely mecC) carried on the SCCmec cassette encodes an altered penicillin-binding protein PBP2a with low affinity for beta-lactams.
- Confers resistance to ALL beta-lactams (except the anti-MRSA cephalosporins ceftaroline and ceftobiprole).
- Detected by cefoxitin disc screening, mecA PCR or PBP2a latex agglutination.

Hospital-acquired versus community-acquired MRSA

Feature	HA-MRSA	CA-MRSA
SCCmec type	I, II, III (large)	IV, V (small, mobile)
Risk group	Elderly, devices, prior antibiotics, ICU	Young, healthy, athletes, prisons, IDU

Typical disease	Bacteraemia, surgical site, pneumonia, line sepsis	Skin and soft-tissue abscess, necrotizing p
PVL toxin	Usually absent	Often present
Resistance	Multidrug-resistant	Often susceptible to clindamycin, cotrimo

Treatment

- Severe / bacteraemia — vancomycin or daptomycin (NOT daptomycin for pneumonia: inactivated by surfactant); alternatives teicoplanin, linezolid, ceftaroline
- Skin and soft tissue — clindamycin, cotrimoxazole, doxycycline, linezolid; drainage is essential
- Adjunct — clindamycin or linezolid suppress toxin production in PVL-associated and toxic-shock disease
- Decolonization — nasal mupirocin 5 days + chlorhexidine body wash

Exam Pearl

- MRSA resistance = mecA ? PBP2a. Daptomycin must NOT be used for pneumonia because pulmonary surfactant inactivates it. **HIGH-YIELD]**
- VISA / hVISA — thickened cell wall traps vancomycin. VRSA (rare) — acquisition of vanA from enterococci.

2.3 VRE — Vancomycin-Resistant Enterococci

- Mechanism: van gene clusters alter the peptidoglycan precursor terminus from D-alanyl-D-alanine to D-alanyl-D-lactate (vanA, vanB) or D-alanyl-D-serine (vanC), reducing glycopeptide binding.
- Enterococcus faecium accounts for the majority of VRE; E. faecalis is more often susceptible.
- Enterococci are intrinsically resistant to cephalosporins, cotrimoxazole (in vivo), clindamycin and low-dose aminoglycosides.

Phenotype	Key features

vanA	• High-level resistance to vancomycin AND teicoplanin • Plasmid-mediated, transferable • Inducible
vanB	Variable vancomycin resistance, teicoplanin usually SUSCEPTIBLE
vanC	• Intrinsic, low-level; E. gallinarum and E. casseliflavus • Not transferable

- Treatment options: linezolid, daptomycin (high dose), tigecycline, quinupristin–dalfopristin (E. faecium only), nitrofurantoin or fosfomycin for lower urinary tract infection.
- Risk factors: prolonged hospitalisation, prior vancomycin or cephalosporin use, renal failure, neutropenia, transplant.
- Control: contact precautions, cohorting, environmental cleaning — VRE survives on surfaces for weeks.

Exam Pearl

- vanA — resistant to both vancomycin and teicoplanin. vanB — teicoplanin usually remains active. **[COMMON MCQ]**

2.4 ESBL — Extended-Spectrum Beta-Lactamases

ESBLs are plasmid-mediated beta-lactamases that hydrolyse penicillins, and first-, second- and third-generation cephalosporins and aztreonam, but are inhibited by clavulanic acid and are inactive against carbapenems and cephamycins.

- Predominant enzymes: CTX-M (now the commonest worldwide, especially CTX-M-15), plus TEM and SHV derivatives; Ambler class A.
- Principal organisms: Escherichia coli, Klebsiella pneumoniae, Proteus mirabilis.
- Plasmids frequently co-carry resistance to fluoroquinolones, aminoglycosides and cotrimoxazole — hence multidrug resistance.
- Risk factors: prior cephalosporin or fluoroquinolone exposure, recurrent urinary tract infection, indwelling catheter, healthcare exposure, travel to high-prevalence regions including South Asia.

Treatment

- Carbapenems are the treatment of choice for serious infection (meropenem, imipenem, ertapenem).
- Piperacillin–tazobactam is inferior to meropenem for ESBL bacteraemia — the MERINO trial showed higher mortality.
- Uncomplicated cystitis — nitrofurantoin, fosfomycin, or oral aminoglycoside-sparing options may be used to spare carbapenems.
- Ceftazidime–avibactam and ceftolozane–tazobactam are effective carbapenem-sparing alternatives.

Exam Pearl

- ESBL is INHIBITED by clavulanate; AmpC is NOT. This is the key laboratory and conceptual discriminator. **[FAVORITE EXAM QUESTION]**

2.5 AmpC Beta-Lactamases

- Ambler class C cephalosporinases; may be chromosomal and inducible, or plasmid-mediated.
- Hydrolyse penicillins, cephamycins (cefoxitin) and third-generation cephalosporins; NOT inhibited by clavulanate, sulbactam or tazobactam.
- Cefepime (fourth generation) and carbapenems are stable against AmpC.
- De-repression during therapy causes emergent resistance in vivo — an organism initially reported susceptible to ceftriaxone becomes resistant during treatment.

Mnemonic — Inducible Chromosomal AmpC Producers

- ESCPM: Enterobacter, Serratia, Citrobacter freundii, Providencia, Morganella morganii
- Also: Pseudomonas aeruginosa, Hafnia, Aeromonas
- Alternative mnemonic: SPICE / SPACE organisms

- Practical rule: avoid third-generation cephalosporins for serious ESCPM infections even if reported susceptible; use cefepime or a carbapenem.

2.6 CRE — Carbapenem-Resistant Enterobacterales

- Among the most urgent AMR threats; mortality in bacteraemia may reach 40–50%.
- Mechanisms: carbapenemase production, or ESBL/AmpC combined with porin loss.

Carbapenemase	Class & features
KPC	• Ambler class A serine carbapenemase • Inhibited by avibactam and vaborbactam
NDM-1, VIM, IMP	• Class B metallo-beta-lactamases (zinc-dependent) • NOT inhibited by avibactam • Inhibited in vitro by EDTA • Aztreonam is stable but often co-resistance present
OXA-48 and OXA-181	• Class D oxacillinase • Weak carbapenem hydrolysis • Common in South Asia and the Middle East

Treatment options

- Ceftazidime–avibactam — active against KPC and OXA-48, NOT against metallo-enzymes
- Ceftazidime–avibactam PLUS aztreonam — for metallo-beta-lactamase producers such as NDM-1
- Meropenem–vaborbactam, imipenem–relebactam — for KPC
- Cefiderocol — siderophore cephalosporin, broad activity including metallo-enzymes
- Colistin (polymyxin E), tigecycline, fosfomycin, high-dose prolonged-infusion meropenem where MIC is low — usually in combination

Exam Pearl

- NDM-1 (New Delhi metallo-beta-lactamase) is a metallo-enzyme: avibactam does NOT restore activity; aztreonam plus ceftazidime–avibactam is the standard workaround. **[VERY HIGH-YIELD]**

2.7 MDR Pseudomonas aeruginosa

- Intrinsically resistant to many agents because of low outer-membrane permeability, constitutive efflux and inducible AmpC.
- Acquired mechanisms: loss of the OprD porin (specific carbapenem resistance), upregulated MexAB-OprM efflux, derepressed AmpC, acquired carbapenemases (VIM, IMP, NDM), aminoglycoside-modifying enzymes, target mutations in gyrA/parC.
- Risk factors: ICU stay, mechanical ventilation, bronchiectasis and cystic fibrosis, burns, neutropenia, prior carbapenem or fluoroquinolone therapy.

Antipseudomonal agents

- Beta-lactams — piperacillin–tazobactam, ceftazidime, cefepime, meropenem, imipenem, aztreonam
- Newer agents — ceftolozane–tazobactam (drug of choice for MDR Pseudomonas), ceftazidime–avibactam, imipenem–relebactam, cefiderocol
- Others — aminoglycosides (amikacin, tobramycin), ciprofloxacin, levofloxacin, colistin
- Note: ertapenem has NO antipseudomonal activity

Exam Pearl

- OprD porin loss = selective carbapenem (imipenem) resistance in Pseudomonas with preserved susceptibility to other beta-lactar **[COMMON MCQ]**
- Ertapenem is the only carbapenem WITHOUT antipseudomonal activity.

2.8 Carbapenem-Resistant Acinetobacter baumannii

- A WHO critical-priority pathogen; predominantly ICU-acquired and highly environmentally persistent.

- Main mechanism: OXA-type carbapenemases, especially OXA-23; also OXA-24/40, OXA-58 and metallo-enzymes (NDM).
- Additional mechanisms: efflux pumps, porin loss, aminoglycoside-modifying enzymes.
- Distinguishing colonization of the respiratory tract from true ventilator-associated pneumonia is a persistent clinical challenge.

Treatment

- Sulbactam-based regimens — sulbactam has intrinsic activity against Acinetobacter; sulbactam–durlobactam is a newer targeted option
- Polymyxins (colistin, polymyxin B), high-dose ampicillin–sulbactam, minocycline, tigecycline, cefiderocol
- Combination therapy is often used given limited monotherapy options
- Environmental control is essential — the organism survives on dry surfaces for weeks to months

2.9 Drug-Resistant Tuberculosis

Category	Definition
Mono-resistant TB	Resistance to a single first-line drug
Poly-resistant TB	Resistance to more than one first-line drug but NOT to both isoniazid and rif
RR-TB	Rifampicin-resistant TB (with or without other resistance)
MDR-TB	Resistant to at least isoniazid AND rifampicin
Pre-XDR TB	MDR/RR-TB plus resistance to any fluoroquinolone
XDR TB (2021 WHO definition)	MDR/RR-TB plus fluoroquinolone resistance plus resistance to at least one G (bedaquiline or linezolid)

WHO drug grouping for MDR-TB regimens

- Group A — levofloxacin/moxifloxacin, bedaquiline, linezolid (include all three)
- Group B — clofazimine, cycloserine/terizidone (add one or both)
- Group C — ethambutol, delamanid, pyrazinamide, imipenem–cilastatin or meropenem, amikacin, ethionamide, para-aminosalicylic acid

Diagnosis and current regimens

- Rapid molecular diagnostics: Xpert MTB/RIF and Xpert Ultra (rifampicin resistance), Truenat, line probe assays (first- and second-line).
- BPaLM regimen — bedaquiline, pretomanid, linezolid, moxifloxacin for 6 months in eligible pre-XDR/MDR-TB.
- Shorter all-oral regimens have replaced injectable-containing regimens; kanamycin and capreomycin are no longer recommended.
- Key monitoring: QT prolongation (bedaquiline, moxifloxacin, clofazimine), linezolid myelosuppression and peripheral/optic neuropathy.

Exam Pearl

- Rifampicin resistance is a surrogate marker for MDR-TB because more than 90% of rifampicin-resistant isolates are also isoniaz resistant. **[VERY HIGH-YIELD]**
- All diagnosed TB in India must be notified through the Nikshay portal — notification is legally mandatory.

2.10 Antifungal Resistance

Organism	Resistance pattern
Candida krusei	INTRINSICALLY resistant to fluconazole
Candida glabrata	• Dose-dependent azole susceptibility • Increasing echinocandin resistance via FKS mutations

Candida auris	• Multidrug-resistant, often pan-azole resistant • Environmentally persistent • Causes hospital outbreaks • Difficult to identify on conventional systems
Candida parapsilosis	Higher echinocandin MICs (natural FKS polymorphism)
Aspergillus fumigatus	Azole resistance via cyp51A mutations TR34/L98H — driven by agricultura fungicides
Cryptococcus	Flucytosine resistance may emerge on monotherapy

Mechanisms of antifungal resistance

- Azoles — ERG11/cyp51A target mutation, target overexpression, efflux pumps (CDR, MDR)
- Echinocandins — FKS1/FKS2 mutations in glucan synthase
- Polyenes — reduced ergosterol content in the cell membrane
- Flucytosine — loss of cytosine permease or cytosine deaminase

Exam Pearl

- Candida auris is the emerging multidrug-resistant yeast requiring contact precautions and outbreak-level infection control. **[IMPO**
- Echinocandins are first-line for invasive candidiasis and candidaemia pending species identification.

2.11 Infection-Control Implications of Resistance

- All multidrug-resistant organisms require contact precautions with single-room isolation or cohorting.
- Active surveillance screening on admission for high-risk patients — nasal swab for MRSA, rectal or perianal swab for VRE, CRE and ESBL.
- Flagging of the electronic record so that precautions restart on any readmission.

- Environmental decontamination — sporicidal agents for C. difficile; chlorine-based disinfectants for Acinetobacter and C. auris.
- Dedicated (single-patient) equipment: stethoscope, thermometer, sphygmomanometer cuff.
- Decolonization where evidence supports it — mupirocin plus chlorhexidine for MRSA; there is NO effective decolonization for VRE or CRE.
- Antimicrobial stewardship reduces the selection pressure that maintains resistant flora.
- Notification to the infection-control team and, for novel or epidemiologically important organisms, to public-health authorities.

Clinical Note

- Two interventions consistently reduce MDRO transmission: hand hygiene and antimicrobial stewardship.
- Both must operate together — stewardship reduces selection, infection control reduces spread.

SECTION 3 | Infection-Control Precautions

3.1 Principles and the Chain of Infection

All infection-control measures act by interrupting one or more links in the chain of infection.

Chain of Infection

Infectious agent

?

Reservoir

?

Portal of exit

?

Mode of transmission

?

Portal of entry

?

Susceptible host

Two-tier system of precautions

- Tier 1 — Standard precautions: applied to ALL patients at ALL times, regardless of diagnosis
- Tier 2 — Transmission-based precautions: contact, droplet and airborne, added according to the suspected route of spread

3.2 Standard Precautions

Standard precautions are the minimum infection-prevention practices applied to the care of all patients, based on the principle that all blood, body fluids, secretions, excretions (except sweat), non-intact skin and mucous membranes may contain transmissible agents.

- Hand hygiene
- Personal protective equipment based on anticipated exposure
- Respiratory hygiene and cough etiquette
- Safe injection practice — one needle, one syringe, one time
- Safe handling and disposal of sharps; no recapping of needles
- Safe handling of contaminated linen and clinical waste (colour-coded segregation)
- Cleaning and disinfection of the patient environment and shared equipment
- Aseptic technique for invasive procedures
- Patient placement to minimise transmission risk

Exam Pearl

- Standard precautions apply to every patient regardless of known infection status — this is the foundation of all infection control. **[IMPORTANT]**
- Sweat is the only body fluid excluded from standard precautions.

3.3 Contact Precautions

- Indicated for organisms spread by direct or indirect contact.
- Gloves and gown on entering the room; removed before exit.
- Single room preferred; cohorting acceptable when a single room is unavailable.
- Dedicated patient-care equipment.
- Limit patient movement and transport.

Conditions requiring contact precautions

- MRSA, VRE, ESBL, CRE, MDR Pseudomonas and Acinetobacter, Candida auris
- Clostridioides difficile infection (soap and water hand washing — alcohol does not kill spores)
- Scabies, pediculosis, impetigo, herpes simplex (neonatal, disseminated), zoster (localised in immunocompromised)
- Enteric infections in incontinent patients — rotavirus, norovirus, Shigella, hepatitis A
- Viral haemorrhagic fevers and diphtheria (cutaneous) — with additional precautions

- RSV and parainfluenza in children

Critical Point — Clostridioides difficile

- Alcohol-based hand rub does NOT kill C. difficile spores. Wash hands with soap and water, and use a sporicidal (chlorine-based for the environment. **[VERY HIGH-YIELD]**

3.4 Droplet Precautions

- Droplets are large respiratory particles (> 5 micrometres) that travel short distances — approximately 1 to 2 metres — and do not remain suspended in air.
- Surgical (medical) mask on entering the room or when within 1–2 metres of the patient.
- Single room preferred; if unavailable, maintain spatial separation of at least 1 metre with a curtain between beds.
- Patient wears a surgical mask during transport.
- Special air handling and ventilation are NOT required.

Conditions requiring droplet precautions

- Neisseria meningitidis (until 24 hours of effective therapy)
- Haemophilus influenzae type b invasive disease
- Bordetella pertussis, diphtheria (pharyngeal)
- Influenza, mumps, rubella, parvovirus B19
- Group A streptococcal pharyngitis and pneumonia (first 24 hours)
- Mycoplasma pneumoniae, plague (pneumonic)

Mnemonic — Droplet Precautions

- Think "SPIDERMAN": Sepsis (meningococcal), Pertussis, Influenza, Diphtheria, Epiglottitis (Hib), Rubella, Mumps, Adenoviru Neisseria meningitidis
- Practical memory anchor: mumps and rubella are DROPLET; measles and varicella are AIRBORNE.

3.5 Airborne Precautions

- Airborne (droplet nuclei) particles are ? 5 micrometres, remain suspended for prolonged periods and travel long distances on air currents.
- Fit-tested N95 (or FFP2/FFP3) respirator must be worn by all entering staff.
- Airborne infection isolation room — single room, negative pressure relative to the corridor, at least 6–12 air changes per hour, air exhausted outside or through a HEPA filter, door kept closed.
- Patient wears a SURGICAL mask (not N95) during transport.
- Susceptible staff should not enter rooms of patients with measles or varicella when immune staff are available.

Conditions requiring airborne precautions

- Pulmonary and laryngeal tuberculosis (also draining TB sinus)
- Measles (rubeola)
- Varicella (chickenpox) and disseminated herpes zoster — airborne PLUS contact
- Localised zoster in an immunocompromised patient
- Smallpox, monkeypox (airborne plus contact)
- SARS, MERS and COVID-19 — particularly during aerosol-generating procedures

Exam Pearl — Mnemonic "My Chicken Has TB"

- Measles — Chickenpox (varicella) — Herpes zoster (disseminated) — Tuberculosis
- Varicella requires airborne AND contact precautions.
- Aerosol-generating procedures: intubation, bronchoscopy, suctioning, nebulisation, non-invasive ventilation, CPR, sputum induction, upper GI endoscopy, dental procedures. **[COMMON MCQ]**

Feature	Droplet	Airborne
Particle size	> 5 micrometres	? 5 micrometres
Distance travelled	1–2 metres	Long distance, remains suspended

Mask for staff	Surgical mask	Fit-tested N95 / FFP3
Room	Single room preferred	Negative-pressure isolation room
Air changes	Not required	? 6–12 per hour
Examples	Meningococcus, influenza, pertussis, mumps	TB, measles, varicella

3.6 Protective (Reverse) Isolation

Protective isolation aims to shield a severely immunocompromised patient from environmental and person-to-person acquisition of pathogens, rather than to prevent spread from the patient.

Indications

- Allogeneic haematopoietic stem-cell transplantation
- Profound prolonged neutropenia (absolute neutrophil count < 0.5 × 10?/L)
- Severe combined immunodeficiency; extensive burns

Measures

- Single room with POSITIVE pressure relative to the corridor and HEPA-filtered air with ? 12 air changes per hour
- Strict hand hygiene; masks for visitors with respiratory symptoms; exclusion of unwell visitors
- No fresh flowers, potted plants or standing water (Aspergillus, Pseudomonas)
- Neutropenic diet principles — avoid unpasteurised dairy, raw eggs, uncooked meat and unwashed salads
- Sealed barriers and portable HEPA units during nearby hospital construction (Aspergillus risk)
- Avoidance of rectal thermometers, suppositories and enemas (mucosal breach and bacteraemia risk)

Exam Pearl

- Protective isolation uses POSITIVE pressure; airborne isolation uses NEGATIVE pressure. A patient with TB who is also neutr requires an anteroom. **[FAVORITE EXAM QUESTION]**

3.7 Personal Protective Equipment (PPE)

Sequence of donning (putting on)

- Hand hygiene
- Gown
- Mask or respirator (perform a fit/seal check for N95)
- Goggles or face shield
- Gloves — last, drawn over the gown cuffs

Sequence of doffing (removing)

- Gloves — first (most contaminated)
- Goggles or face shield
- Gown
- Hand hygiene
- Mask or respirator — removed LAST, outside the room, handling only the ties or elastic
- Hand hygiene again

Exam Pearl

- Gloves are put on LAST and removed FIRST. The respirator is removed LAST and outside the patient room. **[VERY HIGH-YIEL**
- Gloves are never a substitute for hand hygiene; hands must be decontaminated after glove removal.

3.8 Hand Hygiene

WHO "My 5 Moments for Hand Hygiene"

- Before touching a patient
- Before a clean or aseptic procedure
- After body-fluid exposure risk
- After touching a patient
- After touching patient surroundings
- Alcohol-based hand rub is the preferred method when hands are not visibly soiled — faster, more effective, better skin tolerance and higher compliance.
- Soap and water are mandatory when hands are visibly soiled and after caring for patients with C. difficile, norovirus or Bacillus anthracis (spore formers).
- Recommended durations: alcohol rub 20–30 seconds; handwashing 40–60 seconds.
- Surgical hand antisepsis: 2–5 minutes with an antiseptic agent or an alcohol-based surgical rub.
- Bare below the elbows; no rings other than a plain band; nails short and free of artificial extensions.
- Hand hygiene is the single most effective measure for preventing healthcare-associated infection.

3.9 Patient Transport

- Limit transport of isolated patients to medically essential purposes.
- Notify the receiving department in advance so precautions can be prepared.
- Airborne precautions — patient wears a surgical mask; staff need not wear a respirator in an open corridor if the patient is masked.
- Droplet precautions — patient wears a surgical mask.
- Contact precautions — cover infected or draining wounds; clean hands; ensure the trolley and lift surfaces are cleaned afterwards.
- Staff should don fresh PPE at the destination and remove contaminated PPE before leaving the source area.
- Schedule such patients last on operating and endoscopy lists where practicable.

3.10 Environmental Cleaning

- The environment is a genuine reservoir — MRSA, VRE, C. difficile spores, Acinetobacter, C. auris and norovirus persist on dry surfaces for days to months.
- Focus on high-touch surfaces: bed rails, over-bed tables, call bells, door handles, infusion pumps, monitors, taps.
- Terminal cleaning after discharge of an isolated patient must include all surfaces and curtains.
- Disinfectants: sodium hypochlorite 1000 ppm for routine disinfection and 5000 ppm for blood spills and C. difficile; hydrogen peroxide vapour or ultraviolet devices as adjuncts.
- Alcohol is ineffective against bacterial spores and non-enveloped viruses such as norovirus.
- Cleaning must always precede disinfection — organic soiling inactivates disinfectants.

3.11 Equipment Decontamination

Spaulding classification

Category	Risk & process
Critical	• Enters sterile tissue or vasculature • Requires STERILIZATION • Surgical instruments, implants, catheters, arthroscopes
Semi-critical	• Contacts mucous membranes or non-intact skin • Requires HIGH-LEVEL DISINFECTION • Endoscopes, laryngoscope blades, respiratory equipment, vaginal pr
Non-critical	• Contacts intact skin only • Requires LOW/INTERMEDIATE-LEVEL DISINFECTION • Blood-pressure cuffs, stethoscopes, bedpans, ECG leads

- Sterilization methods: steam autoclave (121 °C for 15 min or 134 °C for 3 min) — the method of choice; ethylene oxide, hydrogen peroxide plasma and low-temperature methods for heat-sensitive items.
- High-level disinfection: glutaraldehyde 2%, ortho-phthalaldehyde, peracetic acid.
- Single-use devices must never be reprocessed.
- Endoscopes are a recognised vehicle for outbreaks (Pseudomonas, CRE from duodenoscopes) — meticulous manual cleaning before automated reprocessing is essential.

3.12 Exposure Management

Immediate first aid after a sharps or splash injury

- Encourage free bleeding gently; do NOT squeeze or scrub the wound
- Wash with soap and running water; do not use bleach or caustics on the wound
- Irrigate mucous membranes and conjunctivae copiously with water or saline
- Report immediately to occupational health; document the incident

Risk of transmission from a percutaneous exposure to a positive source

Virus	Approximate risk & action
Hepatitis B	• ~ 30% (up to 30–62% if e-antigen positive) • Vaccine ± HBIG within 24–48 hours for the non-immune
Hepatitis C	• ~ 3% • No prophylaxis • Monitor HCV RNA at 4–6 weeks; treat established acute infection w acting antivirals
HIV	• ~ 0.3% percutaneous, ~ 0.09% mucous membrane • Post-exposure prophylaxis within 72 hours, ideally < 2 hours, for 28

- HIV PEP regimen: tenofovir + emtricitabine (or lamivudine) plus dolutegravir or raltegravir for 28 days.
- Baseline and follow-up testing of the exposed worker at 6 weeks, 12 weeks and 6 months.
- Source patient testing with consent; do not delay PEP awaiting the result.
- Also consider exposure to meningococcus (rifampicin or ciprofloxacin for mouth-to-mouth or intubation without a mask), tuberculosis (contact tracing and latent-infection testing), varicella (VZIG for the non-immune) and rabies.

Do NOT

- Do not squeeze the wound, do not suck it, and do not apply antiseptic caustics — none reduce transmission and all increase tissu

SECTION 4 | Public-Health Response

Every infection has a population dimension. The clinician's duty does not end with the individual patient — it extends to contacts, to the community and to statutory notification.

4.1 Notifiable Diseases

A notifiable disease is one that, by law, must be reported to designated public-health authorities when diagnosed or suspected, so that surveillance, control and outbreak response can be initiated.

- Notification is based on CLINICAL SUSPICION — do not wait for laboratory confirmation.
- Notification is a statutory duty of the attending clinician and cannot be delegated away.
- Notification is not a breach of confidentiality; it is a legally mandated public-health function.

Diseases notifiable under the International Health Regulations (2005)

- Always notifiable to WHO: smallpox, poliomyelitis due to wild-type poliovirus, human influenza caused by a new subtype, SARS
- Historic quarantinable diseases: cholera, plague, yellow fever
- Any event that may constitute a Public Health Emergency of International Concern (PHEIC)

Notification in India

- Integrated Disease Surveillance Programme (IDSP) — P (presumptive), L (laboratory) and S (syndromic) reporting formats; now transitioning to the Integrated Health Information Platform (IHIP).
- Tuberculosis — notification made mandatory for all healthcare providers including the private sector; reported through the Nikshay portal.
- Commonly notified conditions: cholera, acute diarrhoeal disease, typhoid, viral hepatitis, measles, diphtheria, pertussis, tetanus, meningococcal disease, acute flaccid paralysis, malaria, dengue, chikungunya, Japanese encephalitis, kala-azar, leprosy, rabies (animal bites), plague, COVID-19.
- Acute flaccid paralysis surveillance in any child under 15 years is a core polio-eradication activity.

Exam Pearl

- Notify on clinical suspicion, not on laboratory confirmation — delay defeats the purpose of surveillance. **[IMPORTANT]**
- Under IHR 2005 the four always-notifiable conditions are smallpox, wild poliovirus poliomyelitis, novel-subtype human influen SARS.

4.2 Outbreak Recognition

An outbreak is the occurrence of cases of a disease in excess of what would normally be expected in a defined community, geographical area or season.

Related terms

- Endemic — constant baseline presence in a population
- Epidemic — clear excess over the expected baseline
- Pandemic — epidemic crossing international boundaries affecting large numbers
- Sporadic — irregular, scattered single cases
- Cluster — aggregation of cases in place and time, possibly by chance

Steps in Outbreak Investigation

Confirm the existence of an outbreak (compare with the expected baseline)

?

Verify the diagnosis

?

Define and count cases (case definition: confirmed, probable, suspected)

?

Describe by TIME, PLACE and PERSON — construct the epidemic curve and spot map

?

Develop a hypothesis on source and mode of spread

?

Test the hypothesis (case–control or cohort study)

?

Institute control measures (may begin at any point — do not wait)

?

Communicate findings and evaluate; maintain ongoing surveillance

Reading the epidemic curve

- Point-source outbreak — single sharp peak within one incubation period (food poisoning at a single event)
- Continuous common-source — broad plateau (contaminated water supply)
- Propagated (person-to-person) — successive peaks separated by one incubation period (measles, COVID-19)
- Intermittent common-source — irregular peaks

Viva Point

- Q: How do you differentiate a point-source from a propagated outbreak on the epidemic curve?
- A: A point source produces a single sharp peak spanning approximately one incubation period; a propagated outbreak shows pro successive waves each separated by one incubation period.

4.3 Contact Tracing

Contact tracing is the systematic identification, assessment and management of persons who may have been exposed to an infectious individual.

- Steps: define the infectious period ? identify contacts ? risk-stratify (close versus casual) ? notify ? evaluate ? offer prophylaxis, vaccination, testing or surveillance.
- Contact definition varies by disease — household and intimate contacts for meningococcus; prolonged shared airspace for tuberculosis; sexual and needle-sharing partners for HIV, hepatitis B and C, and syphilis.
- Index case confidentiality must be preserved wherever possible.
- Concentric ("stone in the pond") principle in tuberculosis: begin with the closest household contacts and widen only if the yield of infection is high.

Disease	Contact action
Meningococcal disease	Chemoprophylaxis to household and kissing contacts and to healthcare work unprotected airway exposure
Tuberculosis	• Screen household and close contacts • Test and treat latent infection • Prioritise children < 5 years and those with HIV
Measles	Vaccine within 72 hours or immunoglobulin within 6 days for susceptible co

Diphtheria	Throat swab, antibiotic prophylaxis, booster vaccination of contacts
Pertussis	Macrolide prophylaxis for household contacts, especially infants and pregnar
HIV / syphilis / hepatitis B	Partner notification, testing, vaccination and prophylaxis where indicated
Rabies exposure	Category-based wound care, vaccine ± rabies immunoglobulin

4.4 Isolation

Isolation is the separation of a person KNOWN or STRONGLY SUSPECTED to be infected, for the duration of the period of communicability, in order to prevent transmission.

- Applies to symptomatic or confirmed cases.
- Duration is determined by the infectious period of the specific disease.
- Illustrative durations: pulmonary tuberculosis until clinical improvement plus 2 weeks of effective therapy and negative smears; measles until 4 days after rash onset; varicella until all lesions are crusted; meningococcal disease until 24 hours of effective therapy.
- Consider the psychological burden of isolation — maintain communication and mental-health support.

4.5 Quarantine

Quarantine is the restriction of movement of apparently WELL persons who have been EXPOSED to an infectious disease, for a period equal to the longest usual incubation period, to observe whether they develop illness.

Feature	Isolation	Quarantine
Applies to	Cases (ill or infected)	Exposed but well contacts

Purpose	Prevent spread from a known case	Detect disease early in the exposed
Duration	Period of communicability	Longest incubation period
Setting	Hospital or home	Home or designated facility

Exam Pearl

- Isolation is for the SICK; quarantine is for the EXPOSED but well. Quarantine duration equals the longest incubation period of the disease. **[FAVORITE EXAM QUESTION]**

4.6 Post-Exposure Prophylaxis (PEP)

Exposure	Prophylaxis
Meningococcal disease	• Ciprofloxacin single dose • or Rifampicin 12-hourly for 2 days • or Ceftriaxone IM (preferred in pregnancy)
Haemophilus influenzae type b	Rifampicin for 4 days for household contacts with a vulnerable child

Pertussis	Azithromycin (preferred) or clarithromycin / erythromycin
Diphtheria	Benzathine penicillin or erythromycin plus vaccination
Tuberculosis (latent infection)	• Isoniazid 6–9 months • or Rifampicin–isoniazid 3 months • or Rifapentine–isoniazid weekly for 12 weeks (3HP)
HIV	• Tenofovir + lamivudine/emtricitabine + dolutegravir for 28 days • Start within 72 hours, ideally < 2 hours
Hepatitis B	Hepatitis B vaccine ± HBIG within 24–48 hours for the non-immune
Rabies	• Wound washing 15 min with soap and water • Category II: vaccine • Category III: vaccine + rabies immunoglobulin infiltrated into the wound
Measles	• MMR within 72 hours • or immunoglobulin within 6 days
Varicella	• VZIG within 96 hours for susceptible high-risk contacts • or vaccine within 3–5 days

Tetanus-prone wound	Tetanus toxoid ± human tetanus immunoglobulin per immunisation status
Influenza	Oseltamivir for high-risk contacts within 48 hours

Exam Pearl — Rabies

- Category III exposure (transdermal bite, scratch with bleeding, mucous membrane or bat contact) requires BOTH vaccine and rabies immunoglobulin infiltrated around the wound. **[VERY HIGH-YIELD]**
- Immediate wound washing with soap and running water for 15 minutes is the single most effective first measure.

4.7 Vaccination of Contacts

- Post-exposure vaccination works when the vaccine-induced immune response is faster than the natural incubation period.
- Effective post-exposure: measles (within 72 h), varicella (within 3–5 days), hepatitis A, hepatitis B, rabies, mumps (limited benefit), meningococcal and typhoid vaccination in outbreak settings.
- Ring vaccination — vaccinating contacts and contacts-of-contacts around each case; the strategy that eradicated smallpox and used for Ebola.
- Outbreak response immunisation for measles, cholera and meningococcal disease.
- Live vaccines are contraindicated in pregnancy and significant immunosuppression — use immunoglobulin instead.

4.8 Food and Water Control

- Applies to faeco-orally transmitted disease: cholera, typhoid, hepatitis A and E, dysentery, giardiasis, amoebiasis, polio, foodborne toxin illness.
- Water safety — protected source, chlorination (residual chlorine 0.5 mg/L), filtration, boiling, safe household storage.
- Sanitation — safe excreta disposal, prevention of cross-connection between sewage and drinking-water supply.
- Food safety — the WHO Five Keys: keep clean; separate raw and cooked; cook thoroughly; keep food at safe temperatures; use safe water and raw materials.
- Exclusion of food handlers with diarrhoea, vomiting or confirmed enteric infection until cleared.

- Milk safety — pasteurisation prevents brucellosis, tuberculosis, salmonellosis and listeriosis.
- Health education, handwashing promotion and, in outbreaks, oral cholera vaccine.

4.9 Vector Control

Vector	Disease & control
Anopheles mosquito	• Malaria • Long-lasting insecticidal nets, indoor residual spraying, larval source
Aedes aegypti	• Dengue, chikungunya, Zika, yellow fever • Source reduction of domestic water containers, day-biting so nets are
Culex mosquito	• Japanese encephalitis, lymphatic filariasis, West Nile • Environmental sanitation, larvicides
Sandfly (Phlebotomus)	• Kala-azar • Indoor residual spraying, plastering of cracked walls
Tick	• Kyasanur forest disease, scrub typhus (mite), Lyme disease • Repellents, protective clothing, tick removal
Louse	• Epidemic typhus, relapsing fever, trench fever • Delousing, hygiene
Housefly	• Enteric infections, trachoma • Sanitation and waste management

Rat flea (Xenopsylla)	• Plague • Insecticide FIRST, then rodenticide

Methods of vector control

- Environmental — source reduction, drainage, filling, proper waste disposal (most sustainable)
- Chemical — insecticides, larvicides, indoor residual spraying, insecticide-treated nets, repellents
- Biological — larvivorous fish (Gambusia, Poecilia), Bacillus thuringiensis israelensis, Wolbachia-infected Aedes
- Genetic — sterile insect technique
- Personal protection — nets, screening, full-sleeved clothing, repellents (DEET)

Exam Pearl

- In plague control, insecticide must be applied BEFORE rodenticide — killing rats first drives infected fleas onto humans. **[COMED MCQ]**

4.10 Occupational-Health Notification

- Healthcare workers are both at risk of and a source of infection.
- Notify occupational health for: sharps and mucocutaneous exposures; unprotected exposure to tuberculosis, meningococcus, measles, varicella or pertussis; and any healthcare worker developing a potentially transmissible infection.
- Pre-employment assessment: immunisation status and screening — hepatitis B (with post-vaccination antibody titre), MMR, varicella, tetanus/diphtheria, BCG and tuberculosis screening; annual influenza and COVID-19 vaccination.
- Work restriction for infected staff — acute diarrhoea or vomiting (exclude until 48 hours symptom-free), infectious tuberculosis, measles, varicella, active herpetic whitlow, group A streptococcal infection, conjunctivitis.
- Healthcare workers performing exposure-prone procedures require documented hepatitis B, hepatitis C and HIV status.
- Occupational infections such as tuberculosis or hepatitis B in a healthcare worker may be legally reportable as occupational disease and may attract compensation.
- Fitness-to-work assessment, counselling, confidentiality and non-discrimination are integral to the process.

SECTION 5 | Prognostic Assessment

Prognosis in infection is determined by the interaction of three variables: the HOST, the PATHOGEN and the TREATMENT delivered.

Determinants of Outcome in Infection

HOST — age, frailty, comorbidity, immune status

?

PATHOGEN — virulence, resistance, inoculum, source

?

RESPONSE — timeliness of therapy, source control, organ support

?

OUTCOME — survival, organ recovery, functional status, recurrence

5.1 Age

- Extremes of age carry the highest mortality — neonates and adults over 65 years.
- Immunosenescence: reduced T-cell function, blunted antibody responses, impaired neutrophil chemotaxis.
- Atypical presentation in the elderly — confusion, falls, functional decline, incontinence or anorexia may replace fever.
- Older patients may be AFEBRILE or HYPOTHERMIC despite severe sepsis; hypothermia carries a worse prognosis than fever.
- Age is an independent variable in almost every severity score (CURB-65, PSI, SOFA-related models, qSOFA context).

Clinical Alert

- Absence of fever in an elderly patient does not exclude serious infection — acute confusion may be the only presenting sign. **[VE HIGH-YIELD]**

5.2 Frailty

Frailty is a state of reduced physiological reserve across multiple systems, causing disproportionate vulnerability to minor stressors.

- Assessed with the Clinical Frailty Scale (1 = very fit to 9 = terminally ill) or the Rockwood frailty index.
- Frailty predicts mortality, delirium, prolonged stay, institutionalisation and readmission independently of age and comorbidity.
- Predicts failure to return to baseline function even in survivors.
- Should inform goals-of-care discussions, escalation decisions and ceiling-of-treatment planning.

5.3 Comorbidity

- Diabetes mellitus — impaired neutrophil function; predisposes to malignant otitis externa, emphysematous pyelonephritis and cholecystitis, mucormycosis, foot sepsis, tuberculosis.
- Chronic kidney disease — uraemic immune dysfunction, dialysis access infection, drug accumulation.
- Chronic liver disease — impaired Kupffer-cell clearance, complement deficiency; spontaneous bacterial peritonitis; high risk from Vibrio vulnificus.
- Chronic lung disease — recurrent pneumonia, Pseudomonas colonisation in bronchiectasis.
- Cardiac failure — poor tolerance of fluid resuscitation and of sepsis-related myocardial depression.
- Malignancy — obstruction, neutropenia, mucosal barrier injury, indwelling devices.
- Alcohol dependence and malnutrition — aspiration, tuberculosis, pneumococcal and Klebsiella pneumonia.
- Charlson Comorbidity Index quantifies burden and predicts long-term mortality.

5.4 Immune Status

Prognosis and likely pathogen are both predicted by the SPECIFIC immune defect, not by "immunosuppression" as a general label.

Immune defect	Characteristic pathogens
Neutropenia	• Gram-negative bacilli incl. Pseudomonas • Staphylococci • Aspergillus • Candida
Cell-mediated (T-cell) defect	• Mycobacteria • Listeria • Salmonella • Nocardia • Pneumocystis • Cryptococcus • CMV • HSV/VZV • Toxoplasma
Humoral (B-cell) / hypogammaglobulinaemia	• Encapsulated organisms — pneumococcus, Haemophilus, meningococcus • Giardia • Enteroviruses
Complement deficiency (C5–C9)	Recurrent Neisseria infection
Asplenia / hyposplenism	• Encapsulated organisms • Overwhelming post-splenectomy infection • Babesia • Capnocytophaga
Barrier breach (lines, catheters, burns)	• Staphylococci • Gram-negative bacilli • Candida

Emergency

- A febrile asplenic patient must be treated as a medical emergency — give empirical antibiotics immediately; overwhelming post-splenectomy infection can be fatal within hours. **[VERY HIGH-YIELD]**

5.5 Organ Dysfunction

- The number of failing organs is one of the strongest predictors of mortality in sepsis.
- SOFA score assesses six systems: respiratory (PaO2/FiO2), coagulation (platelets), liver (bilirubin), cardiovascular (mean arterial pressure and vasopressor requirement), central nervous system (Glasgow Coma Scale) and renal (creatinine and urine output).
- Sepsis-3 defines sepsis as an acute rise in SOFA of 2 or more points attributable to infection.
- Septic shock — persisting hypotension requiring vasopressors to maintain a mean arterial pressure ? 65 mmHg PLUS lactate > 2 mmol/L despite adequate fluid resuscitation; mortality exceeds 40%.
- Each additional failing organ system increases mortality substantially.

Exam Pearl — qSOFA

- Respiratory rate ? 22/min — Altered mentation (GCS < 15) — Systolic blood pressure ? 100 mmHg
- qSOFA is a bedside PROMPT to consider sepsis and to escalate assessment — it is NOT a standalone diagnostic criterion for se has poor sensitivity. **[FAVORITE EXAM QUESTION]**

5.6 Lactate

- Serum lactate is the single most useful biochemical prognostic marker in sepsis.
- Lactate > 2 mmol/L indicates tissue hypoperfusion; > 4 mmol/L defines high risk and mandates aggressive resuscitation regardless of blood pressure ("cryptic shock").
- LACTATE CLEARANCE is more informative than any single value — failure to clear by at least 10–20% within 2–6 hours predicts a poor outcome.
- Non-hypoxic (type B) causes must be considered: liver failure, thiamine deficiency, metformin, beta-agonists, adrenaline, malignancy, seizures, alcohol, HIV drugs.

5.7 Source of Infection

Source	Prognostic significance
Urinary tract	Best prognosis — lowest mortality among septic sources
Intra-abdominal	• Intermediate to high • Depends critically on adequacy of source control
Pneumonia	• Commonest source of sepsis • High mortality
Unknown source	Poorer outcome — reflects diagnostic delay and inadequate source control
Endocarditis / CNS / necrotizing soft tissue	Highest mortality and morbidity

5.8 Pathogen

- Staphylococcus aureus bacteraemia — high mortality; always seek metastatic foci and perform echocardiography.
- Streptococcus pneumoniae — bacteraemic pneumonia and meningitis carry high mortality, especially in asplenia.
- Pseudomonas aeruginosa and Acinetobacter — poor outcome in ventilated and neutropenic patients.
- Candida bloodstream infection — mortality 40–50%; delayed antifungal therapy strongly worsens outcome.
- Neisseria meningitidis — fulminant purpura fulminans; Waterhouse–Friderichsen syndrome.
- Toxin-mediated disease — streptococcal and staphylococcal toxic shock, necrotizing fasciitis, tetanus, diphtheria.
- Falciparum malaria — poor prognostic markers include impaired consciousness, hyperparasitaemia, acidosis, hypoglycaemia, renal failure and pulmonary oedema.

5.9 Resistance

- Infection with a resistant organism roughly doubles the odds of inappropriate empirical therapy, which is itself an independent predictor of death.
- MDR infection is associated with longer hospital stay, higher cost, greater toxicity from salvage agents (colistin, aminoglycosides) and higher mortality.
- Prior colonization with a resistant organism should directly modify empirical therapy.
- Rapid diagnostics that shorten the time to appropriate therapy improve survival.

5.10 Delay in Therapy

- Time to effective antimicrobial therapy is one of the few strongly modifiable determinants of outcome.
- In septic shock, each hour of delay in appropriate antimicrobial therapy is associated with a measurable increase in mortality.
- Similar time dependence applies to bacterial meningitis, necrotizing fasciitis, severe malaria and neutropenic fever.
- Inappropriate initial therapy — an agent to which the organism is resistant — is as harmful as delayed therapy.

Do Not Delay Therapy In

- Septic shock — Bacterial meningitis — Severe falciparum malaria — Neutropenic fever — Necrotizing fasciitis — Asplenic sep

5.11 Source Control (as a prognostic determinant)

- Failure or delay of source control is among the strongest predictors of persistent sepsis and death.
- Achieving source control within 6–12 hours improves survival in intra-abdominal sepsis and necrotizing infection.
- Persistent fever, rising inflammatory markers or continued vasopressor requirement after 48–72 hours of appropriate antibiotics indicates inadequate source control until proven otherwise.
- The least invasive effective procedure should be chosen in the physiologically unstable patient (damage-control principle).

5.12 Recurrence

- Relapse — recurrence with the SAME organism; usually reflects inadequate duration, poor tissue penetration, undrained focus or retained device.
- Reinfection — recurrence with a DIFFERENT organism; reflects persistent host susceptibility or ongoing exposure.
- Classic recurrent infections: C. difficile (15–35% after a first episode), urinary tract infection, cellulitis with lymphoedema, prosthetic device infection, tuberculosis, herpes zoster.
- Recurrence should prompt a search for an anatomical abnormality, foreign body, biofilm, immunodeficiency or unrecognised HIV or diabetes.

5.13 Readmission

- Infection is among the commonest causes of unplanned 30-day readmission.
- Predictors: frailty, multimorbidity, incomplete source control, premature discharge, inadequate outpatient follow-up, poor adherence and social vulnerability.
- Reducing readmission requires clear discharge antibiotic plans, documented stop dates, OPAT arrangements where needed, and early clinical review.

5.14 Functional Recovery

- Survival is not the only outcome — many sepsis survivors do not return to baseline function.
- Post-sepsis syndrome: persistent fatigue, physical deconditioning, ICU-acquired weakness (critical-illness polyneuropathy and myopathy), cognitive impairment, anxiety, depression and post-traumatic stress.
- Older survivors have a substantially increased risk of new cognitive and functional disability and of death in the year following sepsis.
- Management: early mobilisation, nutritional support, rehabilitation, delirium prevention, medication review and structured follow-up.

Clinical Note

- Prognostic assessment must be dynamic — reassess after resuscitation, source control and 48–72 hours of therapy rather than rel single admission score.

SECTION 6 | Master List of Essential Tables

The following tables must be mastered for written papers, viva and rapid revision. Each entry is followed by the single most examinable discriminator it must contain.

6.1 Basic Concepts and Approach

- **1. Infection, colonization and contamination** — Infection = invasion with host response; colonization = presence without response; contamination = organism in the specimen, not in the patient.
- **2. Important infection terminology** — Bacteraemia, septicaemia, sepsis, septic shock, SIRS, pyaemia, toxaemia, latent infection, carrier state, virulence, pathogenicity, inoculum.
- **3. Host–pathogen–environment interaction** — Disease results only when a virulent pathogen meets a susceptible host in a permissive environment (epidemiological triad).
- **4. Epidemiological exposure and likely infection** — Animal, occupational, travel, water, food, sexual, drug-use and healthcare exposures mapped to their characteristic organisms.
- **5. Incubation period as a diagnostic clue** — Short (< 10 days): influenza, meningococcus, dengue. Intermediate: typhoid, malaria. Long (> 21 days): hepatitis A/B/E, TB, HIV, amoebic liver abscess, visceral leishmaniasis.
- **6. Immune defect and likely pathogen** — Neutropenia ? Gram-negatives and moulds; T-cell defect ? intracellular pathogens; B-cell defect and asplenia ? encapsulated organisms; complement C5–C9 ? Neisseria.
- **7. Infection versus non-infectious mimics** — Malignancy, connective-tissue disease, vasculitis, drug fever, thromboembolism, haematoma, pancreatitis, transfusion reaction, adrenal insufficiency.
- **8. Specimen collection and transport** — Correct specimen, adequate volume, before antibiotics, correct transport medium; CSF and Neisseria specimens must NOT be refrigerated.
- **9. Isolation precautions** — Standard for all; plus contact, droplet or airborne. Varicella and disseminated zoster need airborne PLUS contact.

6.2 Fever and Fever of Unknown Origin

- **10. Fever patterns** — Continuous (typhoid, typhus), intermittent (malaria, pyogenic abscess), remittent, relapsing (Pel–Ebstein in Hodgkin, borreliosis), biphasic saddleback (dengue).
- **11. Fever versus hyperthermia** — Fever = raised hypothalamic set point, responds to antipyretics. Hyperthermia = set point normal, thermoregulation overwhelmed; antipyretics ineffective, needs physical cooling.

- **12. Causes of relative bradycardia (Faget sign)** — Typhoid, brucellosis, legionella, psittacosis, leptospirosis, dengue, yellow fever, drug fever, factitious fever.
- **13. FUO classification** — Classical, nosocomial, neutropenic and HIV-associated; the classical definition requires fever > 38.3 °C for more than 3 weeks with no diagnosis after appropriate initial evaluation.

6.3 Bloodstream Infection and Sepsis

- **14. Blood-culture interpretation** — Judged by the organism, the number of positive sets, time to positivity, and clinical context.
- **15. True bacteraemia versus contamination** — True: S. aureus, S. pneumoniae, Enterobacterales, Pseudomonas, Candida — always significant. Likely contaminant: coagulase-negative staphylococci, Bacillus, Corynebacterium, Propionibacterium in a single set.
- **16. Repeat blood-culture indications** — S. aureus and Candida bacteraemia (to document clearance), suspected endocarditis, persistent fever on therapy, intravascular device infection.
- **17. Sepsis-3 definitions** — Sepsis = life-threatening organ dysfunction due to a dysregulated host response to infection (SOFA rise ? 2). Septic shock = vasopressor requirement plus lactate > 2 mmol/L after adequate fluids.
- **18. SOFA and qSOFA** — SOFA scores six organ systems. qSOFA = respiratory rate ? 22, altered mentation, systolic BP ? 100 — a prompt, not a diagnostic criterion.
- **19. Causes of elevated lactate** — Type A (hypoperfusion — sepsis, shock, ischaemia) and type B (liver failure, thiamine deficiency, metformin, beta-agonists, adrenaline, malignancy, seizures).
- **20. Sepsis empirical therapy** — By suspected source and host: community versus healthcare-associated, MRSA risk, Pseudomonas risk, intra-abdominal anaerobic cover, antifungal indications.
- **21. Vasopressors and inotropes** — Noradrenaline first line; add vasopressin as second agent; adrenaline third; dobutamine for myocardial dysfunction; target mean arterial pressure ? 65 mmHg.
- **22. Source-control procedures** — Drainage, debridement, device removal, definitive control and diversion — ideally within 6–12 hours.

6.4 Gastrointestinal Infection

- **23. Watery versus inflammatory diarrhoea** — Watery/non-inflammatory: small bowel, large volume, no blood, no faecal leucocytes (cholera, ETEC, viral, Giardia). Inflammatory: colonic, small volume, blood and mucus, fever, leucocytes present (Shigella, Campylobacter, Salmonella, EHEC, amoebiasis, C. difficile).
- **24. Food exposure and organism** — Reheated rice ? Bacillus cereus; poultry and eggs ? Salmonella; undercooked beef ? EHEC O157:H7; seafood ? Vibrio parahaemolyticus; canned food ? Clostridium botulinum; unpasteurised dairy ? Listeria and Brucella; short-onset vomiting after custards ? Staphylococcus aureus toxin.

- **25. Stool-testing indications** — Bloody diarrhoea, fever, severe dehydration, immunosuppression, duration beyond 7 days, recent antibiotics or hospitalisation, outbreak investigation, recent travel.

6.5 Healthcare-Associated and Special-Population Infection

- **26. Healthcare-associated infections** — Urinary tract infection, surgical site infection, pneumonia, bloodstream infection and C. difficile — the classical "big five".
- **27. Device-associated infections** — CAUTI, CLABSI, ventilator-associated pneumonia and prosthetic device infection — biofilm formation makes device removal essential.
- **28. Postoperative fever** — The 5 Ws by day: Wind (atelectasis/pneumonia, days 1–2), Water (urinary, days 3–5), Wound (days 5–7), Walking (venous thromboembolism, days 5–10), Wonder drugs (drug fever, any time).
- **29. Infections in injection drug users** — Skin and soft-tissue abscess, right-sided (tricuspid) endocarditis due to S. aureus, septic pulmonary emboli, blood-borne viruses, tetanus, botulism, spinal and epidural abscess.

6.6 Tropical and Travel-Related Infection

- **30. Tropical infections by geography** — Sub-Saharan Africa ? falciparum malaria, HIV, viral haemorrhagic fever; South Asia ? enteric fever, dengue, chikungunya, kala-azar, scrub typhus; Southeast Asia ? melioidosis, dengue; Latin America ? Chagas disease, dengue, yellow fever.
- **31. Tropical infections by incubation period** — < 10 days: dengue, chikungunya, rickettsia, enteric bacteria. 10–21 days: malaria, typhoid, leptospirosis, brucellosis. > 21 days: tuberculosis, viral hepatitis, HIV seroconversion, amoebic liver abscess, visceral leishmaniasis.
- **32. Fever with thrombocytopenia** — Dengue, malaria, scrub typhus and other rickettsioses, leptospirosis, enteric fever, sepsis with DIC, HIV, visceral leishmaniasis, haemophagocytic lymphohistiocytosis.
- **33. Fever with jaundice** — Malaria, leptospirosis (Weil disease), viral hepatitis, ascending cholangitis, liver abscess, enteric fever, dengue, yellow fever, sepsis-associated cholestasis.

6.7 Adolescent, Sexual and Maternal Health

- **34. Adolescent infections** — Infectious mononucleosis (EBV), sexually transmitted infections, meningococcal disease, acne-related soft-tissue infection, vaccine-preventable disease in the incompletely immunised.
- **35. STI syndromic presentation** — Urethral discharge, vaginal discharge, genital ulcer (painful — herpes and chancroid; painless — syphilis and LGV), lower abdominal pain, inguinal bubo, scrotal swelling.
- **36. Maternal infections and fetal effects** — TORCH complex — Toxoplasma, Others (syphilis, varicella, parvovirus B19, listeria, HIV, hepatitis B, Zika), Rubella, Cytomegalovirus, Herpes simplex.

- **37. Antimicrobial safety in pregnancy** — SAFE: penicillins, cephalosporins, erythromycin (not estolate), azithromycin, clindamycin, nitrofurantoin (avoid at term). AVOID: tetracyclines, fluoroquinolones, aminoglycosides, cotrimoxazole (first and third trimesters), chloramphenicol, metronidazole (caution first trimester).
- **38. Prevention of vertical transmission** — Antenatal screening, maternal antiretroviral therapy, hepatitis B birth-dose vaccine plus HBIG, intrapartum antibiotic prophylaxis for group B streptococcus, elective caesarean for active genital herpes, infant prophylaxis and safe infant feeding.

6.8 The Immunocompromised Host

- **39. Opportunistic infections by CD4 count** — CD4 < 200 ? Pneumocystis jirovecii pneumonia; < 100 ? cerebral toxoplasmosis, cryptococcal meningitis; < 50 ? CMV retinitis and disseminated Mycobacterium avium complex. Tuberculosis occurs at ANY CD4 count.
- **40. Neutropenic-fever risk stratification** — MASCC score ? 21 identifies low risk suitable for oral or outpatient therapy; < 21 is high risk requiring admission and intravenous antipseudomonal therapy.
- **41. Post-transplant infection timeline** — Month 1 — nosocomial, surgical and donor-derived infections. Months 1–6 — opportunistic (CMV, Pneumocystis, Aspergillus, reactivation TB). Beyond 6 months — community-acquired infection, and late opportunistic infection in those with chronic rejection.
- **42. Biological therapy and infection risk** — Anti-TNF agents ? tuberculosis reactivation, histoplasmosis, listeria. Rituximab ? hepatitis B reactivation, PML. Eculizumab ? meningococcal disease. JAK inhibitors ? herpes zoster.
- **43. Prophylaxis in immunocompromised hosts** — Cotrimoxazole for Pneumocystis, fluconazole or posaconazole for fungal prophylaxis, acyclovir for herpesviruses, valganciclovir for CMV, isoniazid or 3HP for latent tuberculosis.
- **44. Vaccination in immunocompromised hosts** — LIVE vaccines are contraindicated (BCG, oral polio, MMR, varicella, yellow fever, live typhoid). Inactivated vaccines are safe but less immunogenic — give before planned immunosuppression where possible.

6.9 Outcome

- **45. Prognostic indicators in severe infection** — Age, frailty, comorbidity, immune status, number of failing organs, lactate and its clearance, source, pathogen, resistance, delay in therapy, and adequacy of source control.

How to Use This List

- In written examinations, reproducing the correct table earns more marks than an unstructured paragraph.
- For each table, memorise the COLUMN HEADINGS first — the content then follows logically.
- Tables 1, 6, 11, 17, 23, 28, 39 and 41 are the most frequently examined in NEET-PG and INI-CET. **[VERY HIGH-YIELD]**

SECTION 7 | Master List of Essential Figures

All image and diagram content for this chapter is compiled here. For each figure, the essential labelled components that must be reproducible in an examination answer are specified.

7.1 Important Diagrams and Conceptual Figures

- **1. Chain of infection** — Six links — infectious agent, reservoir, portal of exit, mode of transmission, portal of entry, susceptible host; with the control measure that breaks each link.
- **2. Host–pathogen–environment triangle** — Epidemiological triad showing agent, host and environment with the interacting arrows; label the balance that determines whether disease occurs.
- **3. Approach to suspected infection** — Flow from history and examination ? severity assessment ? specimen collection ? empirical therapy ? review and de-escalation.
- **4. Syndromic approach to fever** — Branching diagram of fever with localising features versus fever without localising signs, subdivided by exposure history and host status.
- **5. Infection source localization** — Body diagram with the common sepsis sources labelled — respiratory, urinary, abdominal, skin and soft tissue, central nervous system, device, cardiac.
- **6. Microbiological specimen pathway** — Collection ? labelling ? transport medium ? laboratory processing ? Gram stain ? culture ? identification ? susceptibility ? clinical interpretation.
- **7. Thermoregulation** — Anterior hypothalamic thermoregulatory centre with heat-loss and heat-gain mechanisms and the peripheral thermoreceptor input.
- **8. Pathogenesis of fever** — Exogenous pyrogen ? macrophage ? endogenous pyrogens (IL-1, IL-6, TNF-alpha, interferons) ? organum vasculosum of the lamina terminalis ? PGE2 ? raised hypothalamic set point ? heat conservation and generation.
- **9. Fever versus hyperthermia** — Paired diagram contrasting a RAISED set point (fever) with an unchanged set point and failed heat dissipation (hyperthermia).
- **10. FUO diagnostic pathway** — Stepwise algorithm — repeat history and examination, first-line tests, targeted imaging including PET-CT, then biopsy; emphasising avoidance of indiscriminate testing.
- **11. Blood-culture interpretation** — Decision tree using organism identity, number of positive sets, time to positivity and presence of a device.
- **12. Catheter-related bloodstream infection** — Diagram of extraluminal (skin-track) and intraluminal (hub) routes of colonization with biofilm on the catheter surface, plus the differential time-to-positivity concept.
- **13. Sepsis pathogenesis** — Pathogen-associated molecular patterns ? pattern-recognition receptors ? cytokine storm ? endothelial injury and glycocalyx shedding ? capillary leak, vasodilatation, microthrombosis and DIC ? tissue hypoperfusion ? multi-organ dysfunction.

- **14. Sepsis resuscitation** — Hour-1 bundle — measure lactate, obtain blood cultures, give broad-spectrum antibiotics, start 30 mL/kg crystalloid for hypotension or lactate ? 4, start vasopressors to maintain MAP ? 65 mmHg.
- **15. Fluid and vasopressor pathway** — Fluid challenge ? assessment of fluid responsiveness ? noradrenaline ? vasopressin ? adrenaline ? dobutamine for myocardial dysfunction ? hydrocortisone in refractory shock.
- **16. Source-control algorithm** — Identification of the focus ? imaging ? choice of least invasive effective procedure ? timing within 6–12 hours ? reassessment if not improving.
- **17. Mechanisms of infectious diarrhoea** — Four mechanisms — secretory (cholera toxin, cAMP), invasive/inflammatory (Shigella), cytotoxic (Shiga toxin), and osmotic/malabsorptive (Giardia, rotavirus villous damage).
- **18. Acute diarrhoea algorithm** — Assessment of dehydration ? watery versus inflammatory ? indications for stool testing ? rehydration ? selective antibiotic use ? explicit avoidance of antibiotics in suspected Shiga-toxin-producing E. coli.
- **19. Healthcare-associated infection pathway** — Reservoir ? hands and equipment ? device or wound ? patient, with the preventive bundle interrupting each step.
- **20. Postoperative fever algorithm** — Timeline-based approach using the 5 Ws with the corresponding investigations at each postoperative day range.
- **21. Injection-drug-user infection pathway** — Injection site ? local abscess and cellulitis ? bacteraemia ? right-sided endocarditis ? septic pulmonary emboli; plus blood-borne viral transmission and spinal infection.
- **22. Returned-traveller fever algorithm** — Exclude malaria urgently ? define geography and incubation period ? identify danger signs ? syndromic grouping (fever with rash, jaundice, thrombocytopenia, diarrhoea, respiratory or neurological features).
- **23. Tropical incubation-period chart** — Horizontal timeline placing each tropical infection at its usual incubation window — under 10 days, 10 to 21 days, and beyond 21 days.
- **24. Vertical-transmission pathway** — Three routes — transplacental (antenatal), intrapartum (perinatal) and postnatal (breast milk) — with the organisms characteristic of each and the corresponding preventive intervention.
- **25. Fever in pregnancy algorithm** — Assessment of both maternal and fetal risk ? exclusion of pyelonephritis, chorioamnionitis, listeria, malaria and influenza ? selection of pregnancy-safe antimicrobials ? fetal monitoring.
- **26. Neutropenic-fever algorithm** — Recognition (single temperature ? 38.3 °C or ? 38.0 °C for 1 hour with ANC < 0.5) ? cultures ? MASCC risk score ? antipseudomonal beta-lactam within 1 hour ? addition of anti-MRSA or antifungal cover ? reassessment at 48–72 and 96 hours.
- **27. Transplant infection timeline** — Horizontal timeline divided at 1 month and 6 months, with the characteristic pathogen groups plotted in each period.
- **28. Immune defect–pathogen relationship** — Matrix linking neutropenia, T-cell defect, B-cell defect, complement deficiency, asplenia and barrier breach to their characteristic organisms.
- **29. Pulmonary infiltrate in the immunocompromised host** — Algorithm by radiological pattern — diffuse interstitial (Pneumocystis, CMV, viral), focal consolidation (bacterial, Nocardia), nodular with halo (invasive Aspergillus), cavitary (TB, Nocardia, fungal) — leading to bronchoalveolar lavage or biopsy.

- **30. CNS lesion in HIV algorithm** — Ring-enhancing lesion ? toxoplasma serology and CD4 count ? empirical anti-toxoplasma therapy with imaging response at 2 weeks ? if no response, consider primary CNS lymphoma (EBV DNA in CSF, thallium SPECT) and biopsy; distinguish from progressive multifocal leukoencephalopathy (non-enhancing white matter) and cryptococcal or tuberculous disease.

7.2 Important Clinical Photographs

- Purpura fulminans and non-blanching petechial rash of meningococcal septicaemia
- Necrotizing fasciitis — dusky discolouration, bullae, crepitus and skin necrosis
- Erythema migrans of Lyme disease; eschar of scrub typhus (tache noire)
- Rose spots of enteric fever; the maculopapular rash and “white islands in a red sea” of dengue
- Koplik spots and the morbilliform rash of measles
- Splinter haemorrhages, Osler nodes, Janeway lesions and Roth spots in infective endocarditis
- Injection-site abscesses and track marks in an injection drug user
- Oral candidiasis, oral hairy leukoplakia and Kaposi sarcoma in advanced HIV
- Herpes zoster in a dermatomal distribution; disseminated zoster
- Tetanus — risus sardonicus and opisthotonus; trismus

7.3 Important Radiology Images

- Chest radiograph — lobar consolidation; bilateral diffuse infiltrates of Pneumocystis pneumonia
- Chest radiograph and CT — upper-lobe cavitation and miliary mottling in tuberculosis
- CT chest — halo sign and air-crescent sign of invasive pulmonary aspergillosis
- CT abdomen — intra-abdominal collection, liver abscess, emphysematous pyelonephritis
- Ultrasound — hepatic abscess, pyonephrosis, ascites for diagnostic tap
- Contrast MRI brain — ring-enhancing lesions of cerebral toxoplasmosis; tuberculoma; non-enhancing white-matter lesions of PML
- MRI spine — vertebral osteomyelitis, discitis and epidural abscess
- Radiograph of soft tissue — subcutaneous gas in necrotizing infection
- PET-CT in the investigation of fever of unknown origin

7.4 Important ECG and Echocardiography Images

- ECG — prolonged PR interval indicating aortic root abscess complicating infective endocarditis
- ECG — diffuse concave ST elevation and PR depression in myopericarditis
- ECG — QT prolongation from antimicrobials (macrolides, fluoroquinolones, bedaquiline, clofazimine)
- ECG — low voltage and electrical alternans in pericardial effusion or tamponade

- Transthoracic and transoesophageal echocardiography — valvular vegetation, perivalvular abscess, valve perforation and regurgitation
- Tricuspid vegetation in right-sided endocarditis of an injection drug user
- Echocardiographic assessment of sepsis-induced cardiomyopathy and of fluid responsiveness (IVC collapsibility)

7.5 Important Microbiology and Histopathology Slides

Slide / stain	What must be visible & why it matters
Gram stain — Gram-positive cocci in clusters	• Staphylococcus aureus • Immediate guide to empirical anti-staphylococcal therapy
Gram stain — Gram-positive lancet-shaped diplococci	• Streptococcus pneumoniae • Commonest cause of bacterial pneumonia and meningitis
Gram stain — Gram-negative intracellular diplococci	• Neisseria meningitidis / gonorrhoeae • Rapid diagnosis in CSF or urethral smear
Ziehl–Neelsen stain	• Acid-fast bacilli, red on blue background • Diagnosis of tuberculosis and other mycobacteria
India ink / mucicarmine of CSF	• Encapsulated budding yeast with a clear halo • Cryptococcus neoformans
Silver (GMS) or immunofluorescence of BAL	• Cup- or crushed-ping-pong-ball-shaped cysts • Pneumocystis jirovecii

KOH mount and calcofluor white	Septate acute-angle branching hyphae (Aspergillus) versus broad aseptate ribbon-like hyphae (Mucorales)
Peripheral blood smear — thick and thin films	• Ring forms, banana-shaped gametocytes and multiply infected cells of Plasmodium falciparum • Species identification and parasite density
Peripheral smear — atypical lymphocytes	Infectious mononucleosis (EBV), CMV, acute HIV, toxoplasmosis
Bone-marrow aspirate	• Amastigotes of Leishmania (LD bodies) • Granulomas in disseminated tuberculosis • Haemophagocytosis in HLH
Histopathology — caseating granuloma	• Central caseous necrosis with epithelioid cells and Langhans giant cells • Tuberculosis
Histopathology — non-caseating granuloma	• Sarcoidosis, fungal and other causes • Key differential from tuberculosis
Histopathology — owl-eye intranuclear inclusion	Cytomegalovirus infection in tissue
Histopathology — Negri body	• Eosinophilic cytoplasmic inclusion in hippocampal neurons • Rabies

Stool microscopy	• Trophozoites with ingested red cells (Entamoeba histolytica) • Pear-shaped flagellate (Giardia) • Modified acid-fast oocysts (Cryptosporidium)
Cytology — Tzanck smear	• Multinucleated giant cells with intranuclear inclusions • HSV and VZV

Presentation Tip

- In examinations, always name the stain first, then the finding, then the organism — marks are awarded for the sequence, not merely the final diagnosis.

SECTION 8 | Final Essential Examination Points

These seventeen statements represent the highest-yield conceptual points of the entire infectious-diseases block. Each is followed by the reasoning an examiner expects.

1. Infection must be distinguished from colonization and contamination.

Infection implies invasion with a host inflammatory response; colonization is presence without response; contamination is introduction of organisms during sampling. Treating colonization or contamination causes unnecessary toxicity, C. difficile infection and resistance.

2. Clinical severity assessment should precede detailed etiological evaluation.

Recognising sepsis and shock determines the urgency of therapy. Resuscitate and treat first; refine the microbiological diagnosis afterwards.

3. Microbiological specimens should be obtained before antibiotics whenever feasible.

Blood cultures sterilise within hours of the first dose. A missed opportunity may make the diagnosis unprovable for the entire admission — but collection must never delay therapy in the critically ill.

4. Treatment must not be delayed in septic shock, meningitis, severe malaria or neutropenic fever.

In each of these, mortality rises measurably with every hour of delay. Give the first dose within one hour of recognition.

5. Blood-culture interpretation depends on organism, number of positive sets, time to positivity and clinical context.

S. aureus, S. pneumoniae, Enterobacterales, Pseudomonas and Candida are always significant. Coagulase-negative staphylococci in a single set without a device usually represent contamination. Short time to positivity and multiple positive sets favour true bacteraemia.

6. Fever and hyperthermia are pathophysiologically different.

Fever is a REGULATED rise in the hypothalamic set point mediated by prostaglandin E2 and responds to antipyretics. Hyperthermia is an UNREGULATED rise with a normal set point; antipyretics are ineffective and physical cooling plus specific therapy (dantrolene, discontinuation of the offending drug) is required.

7. FUO requires repeated history and examination rather than indiscriminate testing.

The diagnostic yield of serial clinical reassessment exceeds that of untargeted investigation. Look for new physical clues, review drugs, and stop unnecessary medications before ordering further tests.

8. qSOFA is not a standalone diagnostic criterion for sepsis.

qSOFA is a bedside prompt to consider sepsis and escalate assessment. It has poor sensitivity and must not be used to rule sepsis out or to define it.

9. Source control is a central component of sepsis management.

No antimicrobial regimen will sterilise an undrained abscess, necrotic tissue or an infected device. Failure to improve on appropriate antibiotics means inadequate source control until proven otherwise.

10. Avoid antibiotics in suspected Shiga-toxin-producing E. coli diarrhoea.

Antibiotics increase toxin release and the risk of haemolytic uraemic syndrome. Suspect STEC in bloody diarrhoea without high fever, particularly in children after undercooked beef exposure. Antimotility agents are also contraindicated.

11. Malaria must be urgently excluded in a febrile traveller returning from an endemic area.

Falciparum malaria can kill within 24 hours. Perform thick and thin films or a rapid diagnostic test immediately, and repeat at 12–24 hour intervals for three sets before excluding the diagnosis.

12. Infection may present atypically in older, pregnant, neutropenic and immunocompromised patients.

Fever may be absent or replaced by hypothermia, confusion, falls or functional decline. Neutropenic patients cannot mount pus, so classical signs of localised infection are muted or absent.

13. A febrile asplenic patient should be treated as a medical emergency.

Overwhelming post-splenectomy infection, usually pneumococcal, may progress to death within hours. Give immediate empirical antibiotics, and ensure lifelong vaccination against encapsulated organisms plus a standby antibiotic supply.

14. Neutropenic fever requires immediate antipseudomonal antibiotic therapy.

Administer an antipseudomonal beta-lactam (piperacillin–tazobactam, cefepime or meropenem) within one hour of recognition, after taking cultures from both peripheral and central lines.

15. Opportunistic infections should be predicted according to the specific immune defect.

The pattern of immunodeficiency — neutropenia, T-cell, B-cell, complement, splenic or barrier — predicts the pathogen far more reliably than the label "immunosuppressed".

16. Pregnancy-related infection management must address both maternal and fetal risk.

Consider maternal severity, fetal effects of the infection, teratogenicity of the antimicrobial and the risk of vertical transmission. Untreated maternal infection is usually more dangerous to the fetus than a carefully selected safe antimicrobial.

17. Antimicrobial stewardship, infection control and public-health notification should be integrated throughout all chapters.

These are not administrative add-ons: they determine individual outcome, institutional resistance rates and community transmission. Every prescription is simultaneously a clinical and a public-health act.

The Six Infectious-Disease Emergencies — Treat Before Confirming

- Septic shock
- Bacterial meningitis
- Severe falciparum malaria
- Neutropenic fever
- Necrotizing fasciitis
- Overwhelming post-splenectomy infection

SECTION 9 | Rapid Revision — High-Yield One-Liners

STEWARDSHIP — MUST-KNOW FACTS

- The 5 Ds: right Diagnosis, Drug, Dose, Duration, De-escalation
- Loading dose depends on volume of distribution — NOT reduced in renal failure
- Beta-lactams are time-dependent (T > MIC); aminoglycosides are concentration-dependent (Cmax/MIC); vancomycin and fluoroquinolones are AUC/MIC-driven
- Vancomycin target AUC24/MIC = 400–600
- Antibiotic time-out at 48–72 hours — Start Smart, Then Focus
- IV-to-oral switch uses the COMS criteria
- 100% oral bioavailability: linezolid, metronidazole, fluconazole, fluoroquinolones
- 7 days equals 14 days for Gram-negative bacteraemia with source control
- S. aureus bacteraemia is never treated as a short course — minimum 14 days
- The 4 Cs cause C. difficile: clindamycin, cephalosporins, co-amoxiclav, ciprofloxacin
- WHO AWaRe: Access – Watch – Reserve; target ? 60% Access

RESISTANCE — MUST-KNOW FACTS

- MRSA: mecA gene ? altered PBP2a
- Daptomycin must NEVER be used for pneumonia — inactivated by surfactant
- vanA = vancomycin AND teicoplanin resistant; vanB = teicoplanin usually susceptible
- ESBL is inhibited by clavulanate; AmpC is NOT
- CTX-M is the commonest ESBL worldwide
- Carbapenem is the drug of choice for serious ESBL infection (MERINO trial)
- ESCPM organisms carry inducible chromosomal AmpC
- NDM-1 is a metallo-beta-lactamase — avibactam ineffective; use aztreonam plus ceftazidime–avibactam
- OprD porin loss causes selective carbapenem resistance in Pseudomonas
- Ertapenem has NO antipseudomonal activity
- OXA-23 is the main carbapenemase of Acinetobacter baumannii
- MDR-TB = resistance to isoniazid AND rifampicin; XDR adds fluoroquinolone plus a Group A drug
- Group A anti-TB drugs: levofloxacin/moxifloxacin, bedaquiline, linezolid
- Candida krusei is intrinsically fluconazole-resistant; Candida auris is multidrug-resistant

INFECTION CONTROL — MUST-KNOW FACTS

- Standard precautions apply to ALL patients; sweat is the only excluded body fluid
- Alcohol rub does NOT kill C. difficile spores — soap and water plus chlorine-based disinfectant
- Airborne: measles, chickenpox, disseminated zoster, tuberculosis ("My Chicken Has TB")
- Varicella needs airborne PLUS contact precautions
- Droplet particles > 5 micrometres travel 1–2 metres; airborne particles ? 5 micrometres remain suspended
- Airborne isolation = NEGATIVE pressure; protective isolation = POSITIVE pressure
- Airborne isolation room requires ? 6–12 air changes per hour
- Gloves on LAST, off FIRST; respirator removed LAST and outside the room
- The patient wears a SURGICAL mask during transport, never an N95
- Hand hygiene is the single most effective infection-control measure — WHO 5 Moments
- Spaulding: critical ? sterilization; semi-critical ? high-level disinfection; non-critical ? low-level disinfection
- Needlestick transmission risk: hepatitis B ~30%, hepatitis C ~3%, HIV ~0.3%
- HIV PEP within 72 hours (ideally < 2 hours) for 28 days

PUBLIC HEALTH — MUST-KNOW FACTS

- Notify on clinical SUSPICION, not on laboratory confirmation
- IHR 2005 always-notifiable: smallpox, wild poliovirus polio, novel-subtype influenza, SARS
- TB notification in India is mandatory through the Nikshay portal
- Isolation is for the SICK; quarantine is for the EXPOSED but well
- Quarantine duration = longest incubation period of the disease
- Point-source outbreak ? single sharp peak; propagated outbreak ? successive peaks one incubation period apart
- Measles PEP: vaccine within 72 hours, immunoglobulin within 6 days
- Varicella PEP: VZIG within 96 hours
- Rabies Category III requires vaccine PLUS immunoglobulin infiltrated into the wound
- Meningococcal prophylaxis: ciprofloxacin, rifampicin, or ceftriaxone in pregnancy
- Plague control: insecticide BEFORE rodenticide
- Ring vaccination eradicated smallpox

PROGNOSIS — MUST-KNOW FACTS

- Sepsis-3: sepsis = infection plus SOFA rise ? 2
- Septic shock = vasopressors for MAP ? 65 mmHg plus lactate > 2 mmol/L despite fluids; mortality > 40%
- qSOFA = respiratory rate ? 22, altered mentation, systolic BP ? 100 — a prompt, not a diagnosis
- Lactate > 4 mmol/L = high risk even if normotensive (cryptic shock)
- Lactate CLEARANCE matters more than any single value
- Noradrenaline is the first-line vasopressor in septic shock
- Urinary source has the best prognosis; unknown source and endocarditis the worst
- Every hour of delayed appropriate therapy increases mortality in septic shock
- Source control should be achieved within 6–12 hours
- A febrile asplenic patient is a medical emergency
- MASCC score ? 21 identifies low-risk neutropenic fever
- Post-sepsis syndrome: physical, cognitive and psychological sequelae in survivors

End of Cross-Chapter Principles in Infectious Diseases

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