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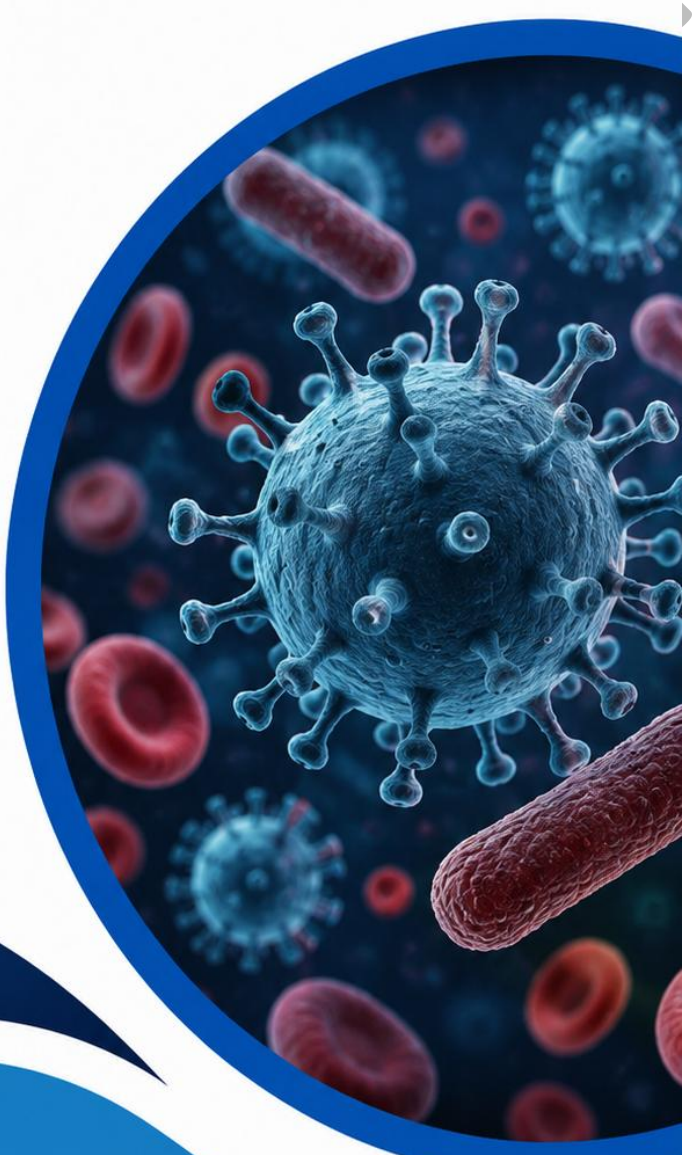
MED 614

INFECTIOUS DISEASES II



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Course code: MED 614

Course title: Infectious Diseases II

Course credit load: 2 Units

Series 1: Host-Pathogen Interactions

- **Part 1.1: Principles of Host Defence**
    - Sub Part 1.1.1: Innate immunity
    - Sub Part 1.1.2: Adaptive immunity
    - Sub Part 1.1.3: The microbiome and colonisation resistance
- **Part 1.2: Microbial Virulence and Pathogenicity**
    - Sub Part 1.2.1: Mechanisms of microbial virulence
    - Sub Part 1.2.2: Adhesion and invasion
    - Sub Part 1.2.3: Microbial evasion of host defence
- **Part 1.3: The Host-Pathogen Relationship**
    - Sub Part 1.3.1: Commensalism, colonisation, and infection
    - Sub Part 1.3.2: Factors determining disease outcome
    - Sub Part 1.3.3: The compromised host
- **Part 1.4: Principles of Diagnosis and Antimicrobial Therapy**
    - Sub Part 1.4.1: Principles of microbiological diagnosis
    - Sub Part 1.4.2: Principles of antimicrobial therapy
    - Sub Part 1.4.3: Antimicrobial resistance

Introduction

This opening Series of **Infectious Diseases II** builds the foundational, conceptual framework the entire course rests on, working through how the human host defends itself against microbial challenge, how microorganisms in turn attempt to overcome that defence, and the genuinely wide spectrum of outcomes, from harmless colonisation to life-threatening infection, this ongoing interaction can produce.

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The aim of this Series is to equip you with an understanding of innate and adaptive immunity, the microbiome and colonisation resistance, the mechanisms of microbial virulence including adhesion, invasion, and immune evasion, the spectrum from commensalism to infection and the factors determining outcome, including the compromised host, and the principles of microbiological diagnosis, antimicrobial therapy, and antimicrobial resistance.

This Series opens with the **principles of host defence**, moves through **microbial virulence and pathogenicity** and **the host-pathogen relationship**, and closes with **principles of diagnosis and antimicrobial therapy**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 1.1:** describe innate and adaptive immunity
- **SLO 1.2:** describe the microbiome and colonisation resistance
- **SLO 1.3:** describe mechanisms of microbial virulence, including adhesion and invasion
- **SLO 1.4:** discuss microbial evasion of host defence
- **SLO 1.5:** describe the spectrum from commensalism to infection and the factors determining disease outcome
- **SLO 1.6:** describe the compromised host
- **SLO 1.7:** describe the principles of microbiological diagnosis and antimicrobial therapy
- **SLO 1.8:** discuss antimicrobial resistance

1.1 Principles of Host Defence

1.1.1 innate immunity

A genuinely rapid, non-specific first line of defence

Innate immunity, the body's immediate, genuinely non-specific first line of defence, includes physical barriers such as intact skin and mucosal surfaces, chemical barriers including gastric acid and antimicrobial peptides, and cellular components, neutrophils and macrophages, capable of recognising broad, genuinely conserved microbial patterns and mounting a rapid response without requiring prior exposure to the specific organism involved.

Pattern recognition receptors, including toll-like receptors, allow innate immune cells to recognise genuinely broad classes of microbial structure shared across many different pathogens,

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triggering inflammatory signalling and recruiting further immune cells to the site of infection, and this rapid, though genuinely non-specific, response provides essential early containment while the more specific adaptive response, discussed in the following sub-Part, develops.

Fill in the blank: Innate immunity provides a rapid response without requiring prior _____ to the specific organism involved.

1.1.2 adaptive immunity

A genuinely specific, memory-forming defence developing over days

Adaptive immunity, developing over days following initial exposure, provides a genuinely highly specific response tailored to the particular pathogen encountered, mediated by **B lymphocytes**, producing specific antibodies, and **T lymphocytes**, including cytotoxic T cells directly destroying infected cells and helper T cells coordinating the broader immune response, working together to mount a genuinely targeted attack against the specific invading organism.

Immunological memory, the genuinely defining feature distinguishing adaptive from innate immunity, allows a considerably faster, more effective response upon subsequent re-exposure to the same specific pathogen, and this memory-based mechanism directly underlies the principle of vaccination, deliberately generating protective immunological memory without the risk of genuine natural infection.

Fill in the blank: Immunological memory allows a considerably faster, more effective response upon subsequent re-exposure to the same specific _____.

1.1.3 the microbiome and colonisation resistance

The body's resident microbial community as a genuine defensive asset

The **microbiome**, the genuinely vast community of microorganisms normally resident on and within the human body, particularly the gastrointestinal tract, provides genuine protective benefit through **colonisation resistance**, the normal microbiota's capacity to competitively exclude potentially pathogenic organisms from establishing themselves, whether through direct competition for nutrients and attachment sites or production of inhibitory substances.

Disruption of the normal microbiome, most commonly through antibiotic therapy already introduced conceptually here and discussed further later in this Series, can genuinely reduce colonisation resistance, allowing potentially pathogenic organisms, including antibiotic-resistant organisms discussed later in this Series, or Clostridioides difficile, to establish themselves and cause genuine clinical infection where the normal, undisturbed microbiome would otherwise have prevented this.

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Fill in the blank: Colonisation resistance is the normal microbiota's capacity to competitively exclude potentially _____ organisms.

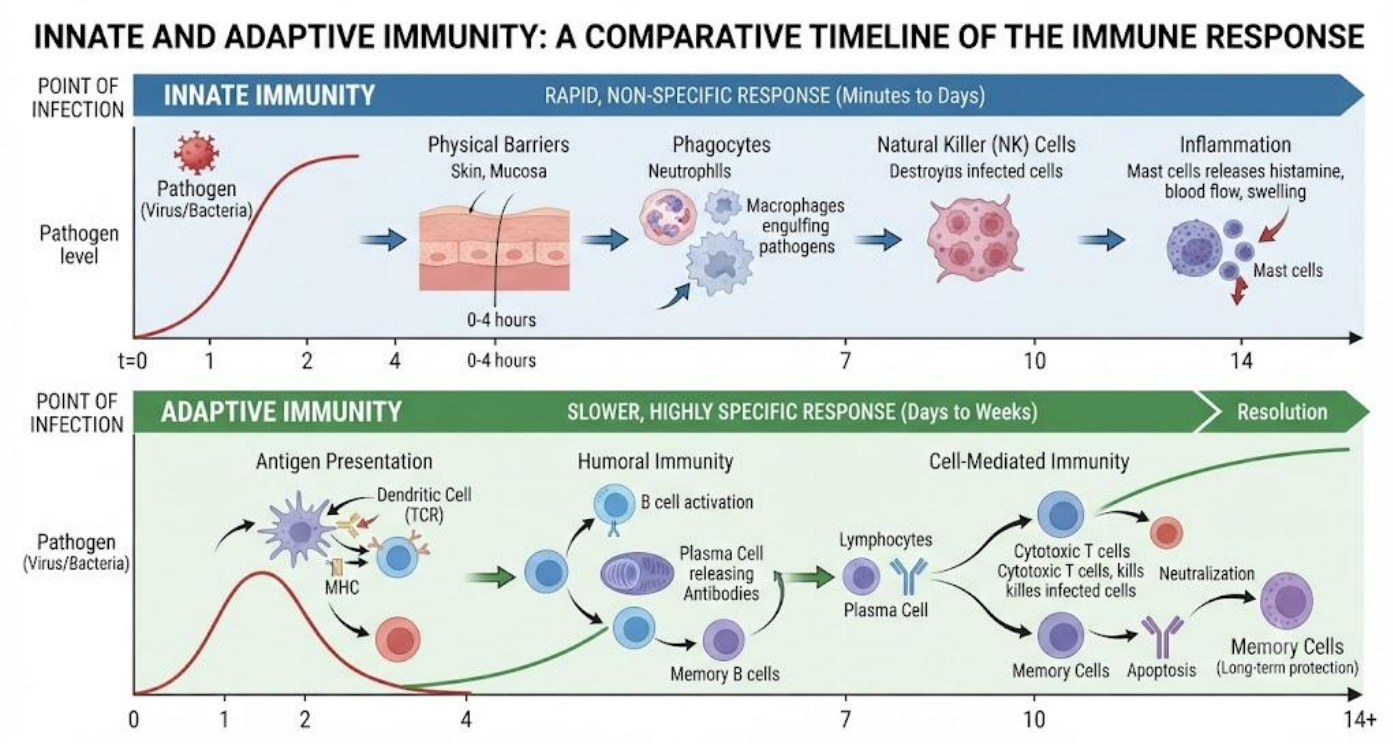

Figure 1.1: A labelled diagram comparing innate and adaptive immune response timelines and characteristics.

Pilot questions 1.1

1. Describe the components of innate immunity. (Linked to SLO 1.1)
2. Describe pattern recognition receptors. (Linked to SLO 1.1)
3. Describe the roles of B lymphocytes and T lymphocytes in adaptive immunity. (Linked to SLO 1.1)
4. Explain immunological memory and its relevance to vaccination. (Linked to SLO 1.1)
5. Describe colonisation resistance. (Linked to SLO 1.2)

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1.2 Microbial Virulence and Pathogenicity

1.2.1 mechanisms of microbial virulence

The genuine tools a pathogen uses to cause disease

Virulence factors, genuinely specific molecular tools a microorganism possesses enabling it to cause disease, include **toxins**, directly damaging host tissue or disrupting normal cellular function, **enzymes**, facilitating tissue invasion or degrading host defensive molecules, and **capsules**, physically resisting phagocytosis by host immune cells, and the specific combination of virulence factors a given organism possesses directly determines its genuine disease-causing potential.

Pathogenicity, an organism's genuine capacity to cause disease, varies considerably across different microorganisms, from genuinely obligate pathogens causing disease in essentially every host they infect, to **opportunistic pathogens**, causing disease only in a genuinely compromised host, a distinction with direct relevance to the compromised host discussion later in this Series, illustrating how genuinely context-dependent the outcome of host-pathogen interaction can be.

Fill in the blank: Capsules physically resist _____ by host immune cells.

1.2.2 adhesion and invasion

The genuinely essential first steps of establishing infection

Adhesion, a microorganism's genuine capacity to attach to host cells or tissue surfaces, typically mediated by specific surface structures including pili and adhesins binding to specific host cell receptors, represents a genuinely essential first step in establishing infection, since organisms unable to adhere are typically cleared by normal host mechanical defences, including mucociliary clearance already introduced in relation to innate immunity.

Invasion, a microorganism's genuine capacity to penetrate host epithelial barriers and disseminate to deeper tissue, whether through direct cellular invasion or exploitation of normal host transport mechanisms, follows successful adhesion in genuinely pathogenic organisms, and understanding this specific, sequential adhesion-then-invasion process directly informs both diagnostic sampling strategy and, in some cases, specific therapeutic approaches targeting these particular steps.

Fill in the blank: Adhesion is typically mediated by specific surface structures including pili and adhesins binding to specific host cell _____.

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1.2.3 microbial evasion of host defence

A genuine evolutionary arms race between pathogen and host

Microorganisms employ genuinely diverse strategies to evade host defence, including **antigenic variation**, altering surface antigens to avoid recognition by existing immune memory, direct interference with phagocytic killing mechanisms, and, in some cases, genuine intracellular survival, hiding within host cells to avoid extracellular immune surveillance entirely, each strategy directly countering a genuinely specific component of the host defence system already discussed earlier in this Series.

This genuine evolutionary arms race between host defence and microbial evasion strategy directly explains why certain infections, despite an apparently intact host immune system, can genuinely persist or recur, and understanding the specific evasion mechanism a given pathogen employs directly informs both the expected natural history of infection and, in some cases, the specific therapeutic or preventive approach most likely to succeed against it.

Fill in the blank: Antigenic variation involves altering surface antigens to avoid recognition by existing immune _____.

Table 1.1: Comparing microbial virulence mechanisms

Mechanism	Genuine function	Example
Toxin production	Direct tissue damage or cellular disruption	Exotoxin release
Capsule formation	Resistance to phagocytosis	Encapsulated bacteria
Antigenic variation	Evasion of existing immune memory	Surface antigen alteration

Pilot questions 1.2

1. Describe virulence factors and give examples. (Linked to SLO 1.3)
2. Distinguish obligate from opportunistic pathogens. (Linked to SLO 1.3)
3. Describe adhesion as a step in establishing infection. (Linked to SLO 1.3)
4. Describe invasion following successful adhesion. (Linked to SLO 1.3)
5. Describe microbial strategies for evading host defence. (Linked to SLO 1.4)

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1.3 The Host-Pathogen Relationship

1.3.1 commensalism, colonisation, and infection

A genuine spectrum, not three sharply separate categories

The relationship between microorganism and host exists along a genuinely continuous spectrum, from **commensalism**, a genuinely mutually neutral or beneficial relationship causing no host harm, through **colonisation**, microbial presence without genuine tissue invasion or clinical symptoms, to **infection**, genuine tissue invasion producing a clinically apparent host response, and the same specific organism can genuinely occupy different points along this spectrum depending on the specific host and circumstances involved.

This genuinely spectrum-based understanding, rather than treating microbial presence as simply present or absent, directly explains why identifying a specific organism through microbiological testing, discussed further later in this Series, does not automatically confirm that organism as the genuine cause of a patient's clinical presentation, since the same organism might genuinely represent harmless colonisation rather than active infection in a given specific clinical context.

Fill in the blank: Colonisation is microbial presence without genuine tissue invasion or clinical _____.

1.3.2 factors determining disease outcome

A genuine balance between pathogen virulence and host defence

Disease outcome following microbial exposure reflects a genuine balance between **pathogen factors**, including the specific virulence characteristics already discussed earlier in this Series and the genuine inoculum size, the quantity of organism the host is exposed to, and **host factors**, including the integrity of the host's innate and adaptive immune defences already discussed, and specific host genetic susceptibility to a particular pathogen.

Environmental and behavioural factors, including nutritional status, coexisting illness, and specific exposure circumstances, further modulate this genuine host-pathogen balance, and understanding this genuinely multifactorial determination of disease outcome, rather than attributing outcome to pathogen virulence or host defence in isolation, provides a genuinely complete framework for understanding why the same organism can produce genuinely different clinical outcomes in different individuals.

Fill in the blank: Environmental and behavioural factors, including nutritional status, further modulate the genuine host-pathogen _____.

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1.3.3 the compromised host

A genuinely important, growing patient population requiring specific consideration

The **compromised host**, a patient with genuinely impaired defence against infection, whether from an underlying medical condition, immunosuppressive medication, or a specific structural or anatomical vulnerability, is genuinely susceptible to infection with organisms that would cause little or no disease in an immunocompetent individual, the opportunistic pathogens already introduced earlier in this Series.

Actively identifying a compromised host, and adjusting the specific differential diagnosis and diagnostic approach accordingly, genuinely matters clinically, since infection in this specific patient population can present atypically, progress more rapidly, and involve organisms genuinely rarely encountered in an immunocompetent host, making a heightened index of suspicion and broader initial differential diagnosis genuinely essential when assessing a compromised patient with suspected infection.

Fill in the blank: Infection in a compromised host can present atypically, progress more rapidly, and involve organisms genuinely rarely encountered in an immunocompetent _____.

Figure 1.2: A clinician carefully assessing an immunocompromised patient for signs of infection.

Pilot questions 1.3

1. Describe the spectrum from commensalism to infection. (Linked to SLO 1.5)
2. Explain why identifying an organism does not automatically confirm it as the cause of disease. (Linked to SLO 1.5)
3. Describe the pathogen and host factors determining disease outcome. (Linked to SLO 1.5)
4. Describe the compromised host. (Linked to SLO 1.6)
5. Explain why a heightened index of suspicion is genuinely essential when assessing a compromised patient. (Linked to SLO 1.6)

1.4 Principles of Diagnosis and Antimicrobial Therapy

1.4.1 principles of microbiological diagnosis

Confirming the specific organism before, or alongside, empirical treatment

Microbiological diagnosis combines specimen collection from the genuinely suspected site of infection, ideally before antimicrobial therapy has begun to maximise diagnostic yield, with **culture**, growing the organism to allow identification and susceptibility testing, and, increasingly, **molecular diagnostic techniques**, directly detecting specific microbial genetic material, offering considerably faster results than traditional culture in many clinical contexts.

Specimen quality and appropriate collection technique, avoiding contamination with normal commensal flora already discussed earlier in this Series, genuinely matters given the spectrum-based understanding of colonisation versus infection already established, since a poorly collected specimen can produce a genuinely misleading result, either falsely suggesting infection from contaminating commensal organisms or failing to identify the genuine causative pathogen.

Fill in the blank: Molecular diagnostic techniques directly detect specific microbial genetic _____.

1.4.2 principles of antimicrobial therapy

Matching the specific agent to the specific organism and site

Empirical antimicrobial therapy, initiated based on the genuinely most likely causative organisms for a given clinical presentation before definitive microbiological confirmation is

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available, is subsequently refined to **targeted therapy** once specific culture and susceptibility results, already discussed in the previous sub-Part, become available, a genuinely structured, sequential approach balancing the need for prompt treatment against the genuine value of precise, organism-specific therapy.

Antimicrobial selection must genuinely consider the specific organism's known susceptibility pattern, the drug's ability to penetrate the specific infected site, and patient-specific factors including allergy history and organ function affecting drug metabolism, and this genuinely individualised, structured approach to antimicrobial selection directly reflects the same comprehensive clinical reasoning already emphasised throughout this course.

Fill in the blank: Antimicrobial selection must consider the drug's ability to _____ the specific infected site.

1.4.3 antimicrobial resistance

A genuinely growing, global threat to effective infection treatment

Antimicrobial resistance, a microorganism's genuine, acquired capacity to survive exposure to an antimicrobial agent that would previously have been effective against it, arises through genetic mutation or acquisition of resistance genes from other organisms, and represents a genuinely growing global threat, increasingly limiting the effective antimicrobial options available for treating genuinely common infections.

Antimicrobial stewardship, the deliberate, structured effort to optimise antimicrobial use, including avoiding unnecessary prescription, selecting the narrowest genuinely effective agent, and using the shortest genuinely adequate treatment duration, directly aims to slow the further development and spread of resistance, and this genuinely responsible prescribing philosophy, closing this Series's foundational coverage, will be actively applied throughout the remainder of this course as specific infectious conditions are covered.

Fill in the blank: Antimicrobial stewardship directly aims to slow the further development and spread of _____.

Table 1.2: Principles of responsible antimicrobial prescribing

Principle	Genuine rationale	Practical application
Avoid unnecessary prescription	Reduces selection pressure driving resistance	Confirm genuine infection before treating
Select the narrowest effective agent	Limits collateral effect on normal microbiota	Avoid broad-spectrum agents where unnecessary

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Use the shortest adequate duration	Reduces cumulative resistance selection pressure	Follow evidence-based duration guidance

Pilot questions 1.4

1. Describe the principles of specimen collection for microbiological diagnosis. (Linked to SLO 1.7)
2. Distinguish empirical from targeted antimicrobial therapy. (Linked to SLO 1.7)
3. Describe the factors genuinely considered in antimicrobial selection. (Linked to SLO 1.7)
4. Describe antimicrobial resistance and how it arises. (Linked to SLO 1.8)
5. Describe antimicrobial stewardship and its practical principles. (Linked to SLO 1.8)

Series Summary

This opening Series built the conceptual foundation this course rests on. Part 1.1 covered innate and adaptive immunity alongside the microbiome, while Part 1.2 turned to microbial virulence, adhesion, invasion, and evasion. Part 1.3 covered the host-pathogen relationship and the compromised host, and Part 1.4 closed with the principles of diagnosis, antimicrobial therapy, and resistance.

You should now be able to describe host defence, microbial virulence, the host-pathogen relationship, and the principles of diagnosis and antimicrobial therapy across all four Parts of this Series, meeting the outcomes set for this Series. Series 2 turns next to sepsis, systemic inflammatory response syndrome, and multi-organ dysfunction syndrome.

Glossary of Terms

- **Adaptive immunity:** a highly specific, memory-forming immune response developing over days.
- **Antimicrobial resistance:** a microorganism's acquired capacity to survive exposure to a previously effective agent.
- **Antimicrobial stewardship:** a structured effort to optimise antimicrobial use and slow resistance development.
- **Colonisation:** microbial presence without genuine tissue invasion or clinical symptoms.

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- **Colonisation resistance:** the normal microbiota's capacity to competitively exclude potentially pathogenic organisms.
- **Compromised host:** a patient with genuinely impaired defence against infection.
- **Innate immunity:** the body's immediate, non-specific first line of defence.
- **Microbiome:** the community of microorganisms normally resident on and within the human body.
- **Opportunistic pathogen:** an organism causing disease only in a genuinely compromised host.
- **Pathogenicity:** an organism's genuine capacity to cause disease.
- **Pattern recognition receptor:** a receptor allowing immune cells to recognise broad microbial structures.
- **Virulence factor:** a specific molecular tool enabling a microorganism to cause disease.

Concept Check Questions [Series 1]

Objective questions

1. Innate immunity provides a rapid response without requiring prior _____ to the specific organism involved. (Linked to SLO 1.1)
2. Colonisation resistance is the normal microbiota's capacity to competitively exclude potentially _____ organisms. (Linked to SLO 1.2)
3. Capsules physically resist _____ by host immune cells. (Linked to SLO 1.3)
4. Colonisation is microbial presence without genuine tissue invasion or clinical _____. (Linked to SLO 1.5)
5. Antimicrobial stewardship directly aims to slow the further development and spread of _____. (Linked to SLO 1.8)

Short-answer questions

6. Distinguish innate from adaptive immunity. (Linked to SLO 1.1)
7. Distinguish obligate from opportunistic pathogens. (Linked to SLO 1.3)
8. Describe microbial strategies for evading host defence. (Linked to SLO 1.4)
9. Describe the compromised host. (Linked to SLO 1.6)
10. Distinguish empirical from targeted antimicrobial therapy. (Linked to SLO 1.7)

Long-answer questions

11. Discuss innate and adaptive immunity and the microbiome's role in colonisation resistance. (Linked to SLO 1.1, 1.2)

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12. Describe the mechanisms of microbial virulence and evasion of host defence. (Linked to SLO 1.3, 1.4)
13. Discuss the spectrum from commensalism to infection, the factors determining disease outcome, and the compromised host. (Linked to SLO 1.5, 1.6)
14. Describe the principles of microbiological diagnosis and antimicrobial therapy. (Linked to SLO 1.7)
15. Discuss antimicrobial resistance and stewardship. (Linked to SLO 1.8)

Answers to Concept Check Questions [Series 1]

Objective questions

1. Exposure.
2. Pathogenic.
3. Phagocytosis.
4. Symptoms.
5. Resistance.

Short-answer questions

6. Innate immunity is rapid and non-specific, while adaptive immunity is slower but highly specific with memory.
7. Obligate pathogens cause disease in essentially every host, while opportunistic pathogens require a compromised host.
8. Strategies include antigenic variation, phagocytic interference, and intracellular survival.
9. The compromised host has genuinely impaired defence against infection from illness, medication, or vulnerability.
10. Empirical therapy is initiated before confirmation, while targeted therapy is refined once results are available.

Long-answer questions

11. **Basic response:** Innate immunity provides rapid, non-specific defence through barriers and cells; adaptive immunity provides specific, memory-based defence through lymphocytes; the microbiome provides colonisation resistance against pathogenic organisms.

    Value-added response: A value-added response notes that vaccination deliberately exploits immunological memory without the risk of natural infection.

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12. **Basic response:** Virulence factors include toxins, enzymes, and capsules; adhesion and invasion establish infection; evasion strategies include antigenic variation and intracellular survival.

    Value-added response: A value-added response notes this reflects a genuine evolutionary arms race between host defence and microbial evasion.

13. **Basic response:** The host-pathogen relationship spans a spectrum from commensalism through colonisation to infection; outcome depends on pathogen and host factors; the compromised host is susceptible to opportunistic organisms.

    Value-added response: A value-added response notes that infection in a compromised host can present atypically and progress rapidly.

14. **Basic response:** Diagnosis combines specimen collection, culture, and molecular techniques; therapy begins empirically and is refined to targeted treatment once results are available, guided by susceptibility and site penetration.

    Value-added response: A value-added response notes that specimen quality genuinely matters given the spectrum-based understanding of colonisation versus infection.

15. **Basic response:** Antimicrobial resistance arises through mutation or gene acquisition, representing a growing global threat; stewardship optimises antimicrobial use to slow further resistance development.

    Value-added response: A value-added response notes that stewardship principles will be actively applied throughout the remainder of this course.

Answers to Pilot Questions [Series 1]

Part 1.1

1. Components include physical barriers, chemical barriers, and cellular components such as neutrophils and macrophages.
2. Pattern recognition receptors, including toll-like receptors, recognise broad conserved microbial structures.
3. B lymphocytes produce antibodies, while T lymphocytes destroy infected cells and coordinate the immune response.

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4. Immunological memory allows faster response on re-exposure, directly underlying the principle of vaccination.
5. Colonisation resistance is the normal microbiota's capacity to competitively exclude pathogenic organisms.

Part 1.2

1. Virulence factors include toxins, enzymes, and capsules, each enabling a specific disease-causing capability.
2. Obligate pathogens cause disease in essentially every host, while opportunistic pathogens require a compromised host.
3. Adhesion is attachment to host cells via structures such as pili and adhesins, an essential first infection step.
4. Invasion is penetration of host epithelial barriers and dissemination to deeper tissue following adhesion.
5. Strategies include antigenic variation, phagocytic interference, and intracellular survival.

Part 1.3

1. The spectrum spans commensalism, colonisation, and infection, reflecting increasing tissue invasion and clinical impact.
2. The same organism can represent harmless colonisation or genuine infection depending on the specific clinical context.
3. Pathogen factors include virulence and inoculum size; host factors include immune defence integrity and susceptibility.
4. The compromised host has genuinely impaired defence from illness, medication, or structural vulnerability.
5. Infection in this population can present atypically and progress more rapidly, requiring broader initial suspicion.

Part 1.4

1. Specimens should be collected from the suspected infection site, ideally before antimicrobial therapy begins.
2. Empirical therapy is initiated before confirmation, while targeted therapy is refined once results are available.
3. Factors include organism susceptibility, drug site penetration, and patient-specific factors such as allergy.
4. Resistance arises through genetic mutation or acquisition of resistance genes from other organisms.

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5. Stewardship includes avoiding unnecessary prescription, selecting narrow agents, and using adequate but shortest duration.

References and Attributions [Series 1]

- Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
- Murray, P. R., Rosenthal, K. S., & Pfaller, M. A. (2020). Medical Microbiology (9th ed.). Elsevier.
- World Health Organization (2021). Global Action Plan on Antimicrobial Resistance. WHO.
- Mandell, G. L., Bennett, J. E., & Dolin, R. (2019). Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases (9th ed.). Elsevier.

Figures, Plates, Tables, and Boxes

- Figure 1.1: A labelled diagram comparing innate and adaptive immune response timelines and characteristics. AI-generated using the prompt: "Create a clean, accurate labelled educational diagram titled Innate and Adaptive Immunity, showing two parallel timelines from the point of infection, one labelled innate immunity with a rapid, non-specific response, and one labelled adaptive immunity with a slower, highly specific response developing over days. Educational anatomical diagram style, no photographic content."
- Table 1.1: Comparing microbial virulence mechanisms. AI-generated using the prompt: "Create a clean reference table titled Comparing Microbial Virulence Mechanisms, listing mechanism, genuine function, and example. Simple clinical reference table style with outside border."
- Figure 1.2: A clinician carefully assessing an immunocompromised patient for signs of infection. AI-generated using the prompt: "Create a realistic clinical illustration titled Assessing the Immunocompromised Patient, showing a clinician attentively examining a seated patient while reviewing their medical chart, consultation room setting. Clean clinical illustration style, no text."
- Table 1.2: Principles of responsible antimicrobial prescribing. AI-generated using the prompt: "Create a clean reference table titled Principles of Responsible Antimicrobial Prescribing, listing principle, genuine rationale, and practical application. Simple clinical reference table style with outside border."

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Course code: MED 614

Course title: Infectious Diseases II

Course credit load: 2 Units

Series 2: Sepsis, Systemic Inflammatory Response Syndrome, and Multi-Organ Dysfunction Syndrome

- **Part 2.1: Systemic Inflammatory Response Syndrome**
  - Sub Part 2.1.1: Pathophysiology of SIRS
  - Sub Part 2.1.2: Diagnostic criteria for SIRS
  - Sub Part 2.1.3: Causes of SIRS
- **Part 2.2: Sepsis**
  - Sub Part 2.2.1: Definitions and pathophysiology of sepsis
  - Sub Part 2.2.2: Clinical presentation and recognition of sepsis
  - Sub Part 2.2.3: Investigation of sepsis
- **Part 2.3: Septic Shock**
  - Sub Part 2.3.1: Pathophysiology of septic shock
  - Sub Part 2.3.2: Clinical presentation of septic shock
  - Sub Part 2.3.3: Management of sepsis and septic shock
- **Part 2.4: Multi-Organ Dysfunction Syndrome**
  - Sub Part 2.4.1: Pathophysiology of multi-organ dysfunction syndrome
  - Sub Part 2.4.2: Organ-specific manifestations of multi-organ dysfunction syndrome
  - Sub Part 2.4.3: Management and prognosis of multi-organ dysfunction syndrome

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Introduction

This Series builds directly on the host-pathogen framework established in Series 1, working through what happens when the host's own inflammatory response, rather than the invading organism alone, becomes the genuinely dominant threat to survival. **Systemic inflammatory response syndrome**, **sepsis**, **septic shock**, and **multi-organ dysfunction syndrome** together represent a genuine continuum of escalating physiological derangement demanding the same rapid, structured recognition emphasised throughout this course.

The aim of this Series is to equip you with an understanding of the pathophysiology, diagnostic criteria, and causes of systemic inflammatory response syndrome, the definitions, pathophysiology, presentation, and investigation of sepsis, the pathophysiology, presentation, and management of septic shock, and the pathophysiology, organ-specific manifestations, management, and prognosis of multi-organ dysfunction syndrome.

This Series opens with **systemic inflammatory response syndrome**, moves through **sepsis** and **septic shock**, and closes with **multi-organ dysfunction syndrome**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 2.1:** describe the pathophysiology and diagnostic criteria of systemic inflammatory response syndrome
- **SLO 2.2:** describe the causes of systemic inflammatory response syndrome
- **SLO 2.3:** describe the definitions, pathophysiology, and clinical presentation of sepsis
- **SLO 2.4:** discuss the investigation of sepsis
- **SLO 2.5:** describe the pathophysiology and clinical presentation of septic shock
- **SLO 2.6:** discuss the management of sepsis and septic shock
- **SLO 2.7:** describe the pathophysiology and organ-specific manifestations of multi-organ dysfunction syndrome
- **SLO 2.8:** discuss the management and prognosis of multi-organ dysfunction syndrome

2.1 Systemic Inflammatory Response Syndrome

2.1.1 pathophysiology of sirs

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The host's own inflammatory response, genuinely amplified

Systemic inflammatory response syndrome, commonly abbreviated as SIRS, represents a genuinely widespread, systemic activation of the inflammatory response already introduced in the innate immunity discussion of Series 1, producing physiological derangement affecting temperature, heart rate, respiratory rate, and white cell count, and, genuinely importantly, SIRS can result from either infectious or non-infectious triggers, discussed further in the closing sub-Part of this Part.

This genuinely amplified inflammatory response, while originally serving a protective, defensive purpose, can itself become genuinely harmful when sufficiently widespread and sustained, producing the specific physiological derangement discussed in the following sub-Part, and understanding SIRS as a genuine host response pattern, rather than a disease in itself, directly underlies the more specific definitions of sepsis discussed later in this Series.

Fill in the blank: SIRS can result from either infectious or _____ triggers.

2.1.2 diagnostic criteria for sirs

A structured set of physiological parameters

SIRS diagnostic criteria, requiring at least two of four specific physiological parameters, abnormal body temperature, either fever or hypothermia, tachycardia, tachypnoea, and abnormal white cell count, either elevated or reduced, provide a structured, reproducible framework for recognising this genuinely widespread inflammatory state, and this specific combination of parameters directly reflects the systemic nature of the inflammatory process already discussed.

While these specific SIRS criteria have genuinely evolved within more recent sepsis definitions discussed further in the following Part, understanding the underlying physiological derangement they capture remains genuinely essential, since recognising this pattern of abnormal vital signs and laboratory findings, regardless of the specific terminology or scoring system applied, directly prompts the clinician to actively consider an underlying, potentially serious triggering process.

Fill in the blank: SIRS diagnostic criteria require at least two of four specific physiological _____.

2.1.3 causes of sirs

A genuinely wide differential, infectious and non-infectious alike

Infectious causes of SIRS, discussed further in relation to sepsis specifically in the following Part of this Series, include bacterial, viral, and fungal infection, while **non-infectious causes**,

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genuinely important to actively consider given their identical physiological presentation, include severe trauma, burns, pancreatitis already discussed in an earlier course of this programme, and major surgery, each capable of triggering the genuinely same widespread inflammatory response.

This genuinely wide differential diagnosis underlines why the presence of SIRS criteria alone, without further clinical context, cannot reliably distinguish an infectious from a non-infectious trigger, meaning careful, structured clinical assessment for a genuine potential source of infection, discussed further in the following Part, is essential before assuming an infectious cause for any given patient meeting these specific SIRS criteria.

Fill in the blank: Non-infectious causes of SIRS include severe trauma, burns, pancreatitis, and major _____.

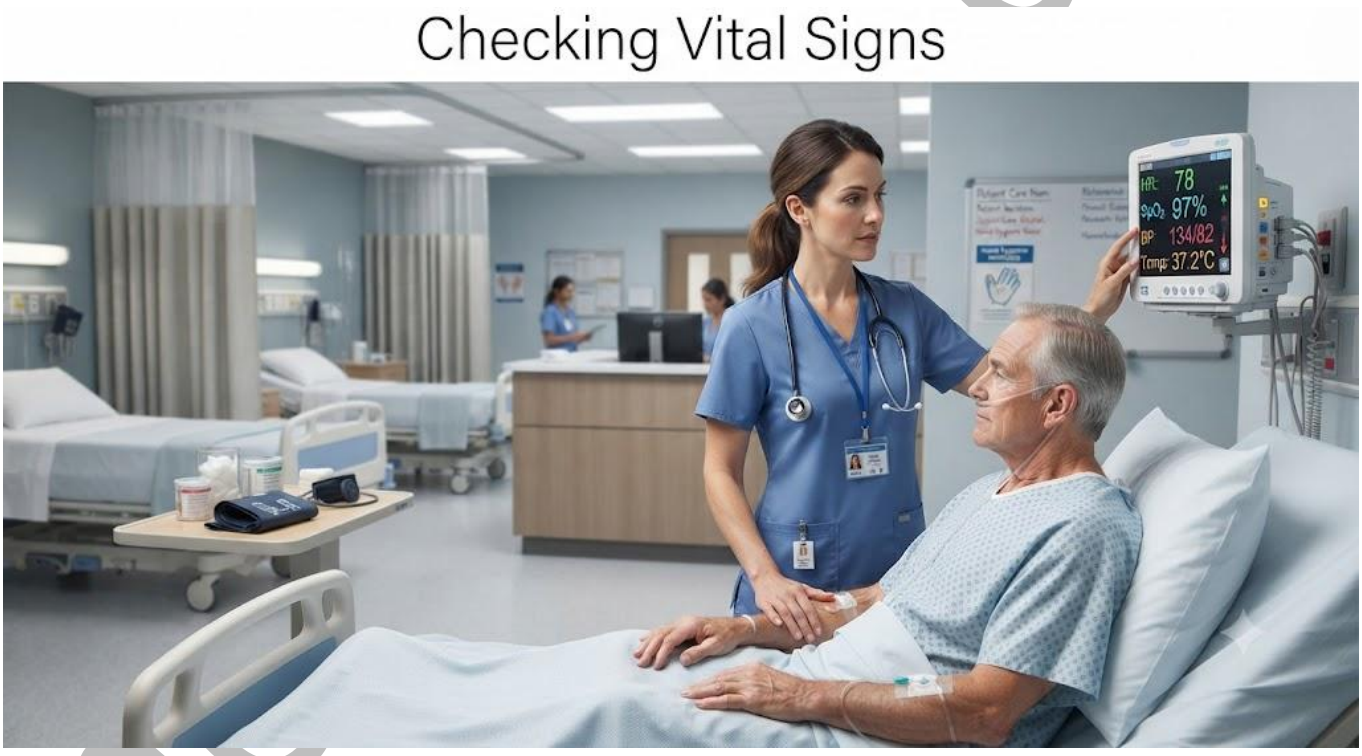

Figure 2.1: A clinician checking a patient's vital signs at the bedside.

Pilot questions 2.1

- Describe the pathophysiology of SIRS. (Linked to SLO 2.1)
- List the four SIRS diagnostic criteria. (Linked to SLO 2.1)
- Explain why understanding the underlying physiological derangement remains essential despite evolving terminology. (Linked to SLO 2.1)
- List infectious and non-infectious causes of SIRS. (Linked to SLO 2.2)

- Explain why SIRS criteria alone cannot reliably distinguish infectious from non-infectious causes. (Linked to SLO 2.2)

2.2 Sepsis

2.2.1 definitions and pathophysiology of sepsis

Life-threatening organ dysfunction from a genuinely dysregulated response

Sepsis, currently defined as life-threatening organ dysfunction resulting from a genuinely dysregulated host response to infection, represents infection combined with genuine evidence of organ dysfunction, a definition that, unlike the earlier SIRS-based framework already discussed, directly emphasises the genuinely harmful, dysregulated nature of the host response itself, rather than simply the presence of widespread inflammation.

The underlying pathophysiology involves genuinely widespread endothelial dysfunction, activation of the coagulation cascade already discussed in an earlier course of this programme, and microvascular dysfunction, together impairing normal tissue oxygen delivery and utilisation despite, in many cases, a genuinely preserved or even increased overall cardiac output, a specific pathophysiological pattern directly explaining the organ dysfunction sepsis produces.

Fill in the blank: Sepsis is currently defined as life-threatening organ dysfunction resulting from a genuinely dysregulated host response to _____.

2.2.2 clinical presentation and recognition of sepsis

A genuinely wide-ranging presentation demanding active vigilance

Sepsis presentation genuinely varies considerably depending on the specific underlying source of infection and the individual patient's baseline physiological reserve, already discussed in relation to the older patient in an earlier course of this programme, and can range from subtle, non-specific deterioration, particularly in older or immunocompromised patients, to genuinely dramatic, rapid physiological collapse.

Structured screening tools, including quick sequential organ failure assessment, a simple bedside score using respiratory rate, mental status, and blood pressure, allow rapid identification of patients genuinely at higher risk of poor outcome from suspected infection, and active, deliberate use of such structured screening, rather than relying on clinical gestalt alone,

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genuinely improves the reliability of sepsis recognition across a wide range of clinical settings and practitioner experience levels.

Fill in the blank: Structured screening tools allow rapid identification of patients genuinely at higher risk of poor outcome from suspected _____.

2.2.3 investigation of sepsis

Confirming the source while never delaying treatment

Investigation of suspected sepsis combines **blood cultures**, ideally taken before antimicrobial therapy begins to maximise diagnostic yield, already discussed as a general principle in Series 1, with **lactate measurement**, a genuinely valuable marker of tissue hypoperfusion, and specific site-directed investigation guided by the suspected source of infection, whether urinary, respiratory, or another anatomical source.

Genuinely importantly, and mirroring the same principle already established for other time-critical infections earlier in this course, obtaining these investigations should never meaningfully delay the initiation of empirical antimicrobial therapy, discussed further in the following Part of this Series, since every hour of delay in appropriate antimicrobial treatment for sepsis measurably worsens patient outcome, making prompt treatment a genuine, immediate clinical priority.

Fill in the blank: Lactate measurement is a genuinely valuable marker of tissue _____.

Table 2.1: Comparing SIRS criteria and the quick sequential organ failure assessment score

Feature	SIRS criteria	Quick sequential organ failure assessment
Parameters assessed	Temperature, heart rate, respiratory rate, white cell count	Respiratory rate, mental status, blood pressure
Genuine focus	Widespread inflammation	Risk of poor outcome from infection
Threshold for concern	Two or more criteria met	Two or more parameters abnormal

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Pilot questions 2.2

1. Describe the current definition of sepsis. (Linked to SLO 2.3)
2. Describe the underlying pathophysiology of sepsis. (Linked to SLO 2.3)
3. Explain why sepsis presentation can be subtle in older or immunocompromised patients. (Linked to SLO 2.3)
4. Describe the quick sequential organ failure assessment score. (Linked to SLO 2.4)
5. Explain why investigation should never delay antimicrobial treatment in suspected sepsis. (Linked to SLO 2.4)

2.3 Septic Shock

2.3.1 pathophysiology of septic shock

A genuine subset of sepsis with profound circulatory failure

Septic shock, a genuine subset of sepsis with profound circulatory, cellular, and metabolic abnormalities associated with a considerably higher mortality risk than sepsis alone, results from genuinely severe vasodilation, reduced systemic vascular resistance, combined with the impaired tissue oxygen utilisation already discussed for sepsis generally, producing genuine, significant hypotension despite adequate or even increased cardiac output.

This specific combination of vasodilation and metabolic derangement genuinely distinguishes septic shock from other shock categories already discussed elsewhere in this programme, including cardiogenic and hypovolaemic shock, and understanding this specific pathophysiological distinction directly informs the genuinely different management approach septic shock requires compared with these other shock categories.

Fill in the blank: Septic shock results from genuinely severe vasodilation, reduced systemic vascular _____.

2.3.2 clinical presentation of septic shock

Persistent hypotension despite adequate fluid resuscitation

Septic shock presents with persistent hypotension genuinely requiring vasopressor medication to maintain an adequate mean arterial pressure despite adequate fluid resuscitation already discussed in relation to management, alongside elevated serum lactate reflecting genuine tissue

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hypoperfusion, and this specific combination of findings, rather than hypotension or elevated lactate in isolation, defines this genuinely more severe subset of sepsis requiring correspondingly more intensive management.

Additional clinical features reflecting the widespread physiological derangement include altered mental status, reduced urine output reflecting renal hypoperfusion, and, in genuinely severe cases, mottled, poorly perfused skin, and prompt recognition of this specific clinical pattern is genuinely essential given the considerably higher mortality risk septic shock carries compared with sepsis without shock.

Fill in the blank: Septic shock presents with persistent hypotension genuinely requiring _____ medication despite adequate fluid resuscitation.

2.3.3 management of sepsis and septic shock

A structured, time-critical bundle of interventions

Structured sepsis management, delivered as a coordinated bundle of interventions within a genuinely narrow, time-critical window following recognition, includes prompt blood cultures already discussed, empirical broad-spectrum antimicrobial therapy, intravenous fluid resuscitation, and serial lactate measurement, with this structured, bundled approach genuinely, measurably improving patient outcome compared with an unstructured, delayed response.

For septic shock specifically, **vasopressor therapy**, most commonly noradrenaline, is added where fluid resuscitation alone genuinely fails to restore adequate blood pressure, and ongoing, structured reassessment of the patient's response to treatment, adjusting the specific intervention intensity accordingly, closes this Part's coverage and reflects the same theme of dynamic, responsive clinical management already emphasised throughout this whole course.

Fill in the blank: Vasopressor therapy, most commonly noradrenaline, is added where fluid resuscitation alone genuinely fails to restore adequate blood _____.

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Figure 2.2: A clinical team delivering a structured sepsis resuscitation bundle.

Pilot questions 2.3

1. Describe the pathophysiology of septic shock. (Linked to SLO 2.5)
2. Distinguish septic shock from other shock categories. (Linked to SLO 2.5)
3. Describe the clinical presentation of septic shock. (Linked to SLO 2.5)
4. Describe the structured sepsis management bundle. (Linked to SLO 2.6)
5. Describe the role of vasopressor therapy in septic shock. (Linked to SLO 2.6)

2.4 Multi-Organ Dysfunction Syndrome

2.4.1 pathophysiology of multi-organ dysfunction syndrome

Progressive dysfunction across genuinely multiple organ systems

Multi-organ dysfunction syndrome, progressive, genuinely simultaneous dysfunction of two or more organ systems, represents the genuinely most severe endpoint of the inflammatory continuum this Series has traced from SIRS through sepsis and septic shock, resulting from the

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combined effects of the widespread inflammation, microvascular dysfunction, and tissue hypoperfusion already discussed across the preceding Parts of this Series.

This genuinely progressive process can affect essentially any organ system, discussed further in the following sub-Part, and the number of organ systems involved, alongside the specific degree of dysfunction within each affected system, directly correlates with overall prognosis, discussed further in the closing sub-Part of this Part, underlining why active, structured monitoring for emerging organ dysfunction genuinely matters throughout the management of any critically unwell septic patient.

Fill in the blank: Multi-organ dysfunction syndrome represents progressive, genuinely simultaneous dysfunction of two or more organ _____.

2.4.2 organ-specific manifestations of multi-organ dysfunction syndrome

A genuinely wide-ranging pattern across affected systems

Respiratory dysfunction, presenting as acute respiratory distress syndrome, and **renal dysfunction**, presenting as acute kidney injury, are among the genuinely most commonly affected organ systems, alongside **cardiovascular dysfunction**, reflecting the septic shock already discussed earlier in this Series, and **hepatic dysfunction**, presenting with deranged liver function tests and, in severe cases, coagulopathy, each reflecting the widespread microvascular and inflammatory process already established as the underlying mechanism.

Neurological dysfunction, presenting as encephalopathy with altered mental status, and **haematological dysfunction**, presenting with thrombocytopenia and, in severe cases, disseminated intravascular coagulation already discussed in an earlier course of this programme, complete the genuinely wide-ranging pattern of organ involvement multi-organ dysfunction syndrome can produce, and active, structured screening for dysfunction across each of these systems forms an essential part of comprehensive assessment.

Fill in the blank: Respiratory dysfunction in multi-organ dysfunction syndrome presents as acute respiratory _____ syndrome.

2.4.3 management and prognosis of multi-organ dysfunction syndrome

Comprehensive organ support, alongside treating the underlying trigger

Management combines the sepsis-directed treatment already discussed earlier in this Series with **organ-specific supportive care**, including mechanical ventilation for respiratory failure, renal replacement therapy for significant acute kidney injury, and vasopressor support for

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cardiovascular dysfunction, delivered within an intensive care setting given the genuinely intensive level of monitoring and intervention this severe condition requires.

Prognosis genuinely correlates directly with the number of organ systems involved and the specific degree of dysfunction within each, and, while mortality from multi-organ dysfunction syndrome remains genuinely significant despite modern intensive care advances, prompt recognition and treatment of the underlying sepsis, closing this Series and reflecting its opening themes, genuinely represents the most effective strategy for preventing progression to this severe, life-threatening endpoint in the first place.

Fill in the blank: Prognosis genuinely correlates directly with the number of organ systems involved and the specific degree of dysfunction within _____.

Table 2.2: Organ-specific manifestations of multi-organ dysfunction syndrome

Organ system	Manifestation	Key management approach
Respiratory	Acute respiratory distress syndrome	Mechanical ventilation
Renal	Acute kidney injury	Renal replacement therapy
Neurological	Encephalopathy, altered mental status	Supportive care, treat underlying cause

Pilot questions 2.4

1. Describe multi-organ dysfunction syndrome. (Linked to SLO 2.7)
2. List organ systems commonly affected by multi-organ dysfunction syndrome. (Linked to SLO 2.7)
3. Describe the respiratory and renal manifestations of multi-organ dysfunction syndrome. (Linked to SLO 2.7)
4. Describe the general management approach for multi-organ dysfunction syndrome. (Linked to SLO 2.8)
5. Describe the relationship between organ involvement and prognosis. (Linked to SLO 2.8)

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Series Summary

This Series traced the genuine continuum from systemic inflammation to life-threatening multi-organ failure. Part 2.1 covered the pathophysiology, criteria, and causes of systemic inflammatory response syndrome, while Part 2.2 turned to the definitions, presentation, and investigation of sepsis. Part 2.3 covered septic shock and the management of sepsis and septic shock, and Part 2.4 closed with multi-organ dysfunction syndrome.

You should now be able to recognise, investigate, and manage the continuum from SIRS through sepsis and septic shock to multi-organ dysfunction syndrome across all four Parts of this Series, meeting the outcomes set for this Series. Series 3 turns next to viral infectious syndromes.

Glossary of Terms

1. **Acute respiratory distress syndrome:** respiratory dysfunction, a common manifestation of multi-organ dysfunction syndrome.
2. **Endothelial dysfunction:** impaired blood vessel lining function contributing to the pathophysiology of sepsis.
3. **Lactate:** a marker of tissue hypoperfusion, genuinely valuable in sepsis investigation.
4. **Multi-organ dysfunction syndrome:** progressive, simultaneous dysfunction of two or more organ systems.
5. **Quick sequential organ failure assessment:** a bedside score identifying patients at higher risk from suspected infection.
6. **Renal replacement therapy:** supportive treatment for significant acute kidney injury.
7. **Sepsis:** life-threatening organ dysfunction from a dysregulated host response to infection.
8. **Septic shock:** a subset of sepsis with profound circulatory, cellular, and metabolic abnormalities.
9. **Structured sepsis management bundle:** a coordinated set of time-critical interventions for suspected sepsis.
10. **Systemic inflammatory response syndrome:** widespread activation of the inflammatory response from infectious or non-infectious triggers.
11. **Tachypnoea:** abnormally rapid breathing, a component of SIRS diagnostic criteria.
12. **Vasopressor:** medication used to restore adequate blood pressure in septic shock.

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Concept Check Questions [Series 2]

Objective questions

- SIRS can result from either infectious or _____ triggers. (Linked to SLO 2.1)
- Sepsis is currently defined as life-threatening organ dysfunction resulting from a genuinely dysregulated host response to _____. (Linked to SLO 2.3)
- Septic shock results from genuinely severe vasodilation, reduced systemic vascular _____. (Linked to SLO 2.5)
- Multi-organ dysfunction syndrome represents progressive, genuinely simultaneous dysfunction of two or more organ _____. (Linked to SLO 2.7)
- Prognosis genuinely correlates directly with the number of organ systems involved and the specific degree of dysfunction within _____. (Linked to SLO 2.8)

Short-answer questions

1. List the four SIRS diagnostic criteria. (Linked to SLO 2.1)
2. Describe the quick sequential organ failure assessment score. (Linked to SLO 2.4)
3. Distinguish septic shock from other shock categories. (Linked to SLO 2.5)
4. Describe the structured sepsis management bundle. (Linked to SLO 2.6)
5. List organ systems commonly affected by multi-organ dysfunction syndrome. (Linked to SLO 2.7)

Long-answer questions

6. Discuss the pathophysiology, diagnostic criteria, and causes of systemic inflammatory response syndrome. (Linked to SLO 2.1, 2.2)
7. Describe the definitions, pathophysiology, presentation, and investigation of sepsis. (Linked to SLO 2.3, 2.4)
8. Discuss the pathophysiology, presentation, and management of septic shock. (Linked to SLO 2.5, 2.6)
9. Describe the pathophysiology and organ-specific manifestations of multi-organ dysfunction syndrome. (Linked to SLO 2.7)
10. Discuss the management and prognosis of multi-organ dysfunction syndrome. (Linked to SLO 2.8)

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Answers to Concept Check Questions [Series 2]

Objective questions

11. Non-infectious.
12. Infection.
13. Resistance.
14. Systems.
15. Each.

Short-answer questions

16. The criteria are abnormal temperature, tachycardia, tachypnoea, and abnormal white cell count.
17. It is a bedside score using respiratory rate, mental status, and blood pressure to identify higher-risk patients.
18. Septic shock involves severe vasodilation and metabolic derangement, distinct from cardiogenic or hypovolaemic shock.
19. The bundle includes blood cultures, empirical antibiotics, fluid resuscitation, and serial lactate measurement.
20. Systems commonly affected include respiratory, renal, cardiovascular, hepatic, neurological, and haematological.

Long-answer questions

21. **Basic response:** SIRS represents widespread inflammatory activation, diagnosed using temperature, heart rate, respiratory rate, and white cell count criteria; causes include both infectious and non-infectious triggers such as trauma and pancreatitis.

    Value-added response: A value-added response notes that SIRS criteria alone cannot reliably distinguish infectious from non-infectious causes.

22. **Basic response:** Sepsis is life-threatening organ dysfunction from a dysregulated host response, involving endothelial and microvascular dysfunction; presentation varies widely; investigation combines blood cultures and lactate measurement without delaying treatment.

    Value-added response: A value-added response notes that structured screening tools improve reliability of sepsis recognition across settings.

23. **Basic response:** Septic shock involves severe vasodilation and metabolic derangement, presenting with persistent hypotension requiring vasopressors despite fluid resuscitation;

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management follows a structured bundle including fluids, antibiotics, and vasopressor therapy.

Value-added response: A value-added response notes that septic shock carries considerably higher mortality risk than sepsis without shock.

24. **Basic response:** Multi-organ dysfunction syndrome results from widespread inflammation and microvascular dysfunction, affecting respiratory, renal, cardiovascular, hepatic, neurological, and haematological systems.

    Value-added response: A value-added response notes that the number of organs involved directly correlates with overall prognosis.

25. **Basic response:** Management combines sepsis-directed treatment with organ-specific supportive care in an intensive care setting; prognosis correlates with the extent of organ involvement.

    Value-added response: A value-added response notes that prompt recognition and treatment of sepsis remains the most effective strategy for preventing progression.

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Answers to Pilot Questions [Series 2]

Part 2.1

1. SIRS represents widespread activation of the inflammatory response from infectious or non-infectious triggers.
2. The criteria are abnormal temperature, tachycardia, tachypnoea, and abnormal white cell count.
3. Recognising the underlying pattern prompts active consideration of a serious triggering process regardless of terminology.
4. Causes include bacterial, viral, and fungal infection alongside trauma, burns, pancreatitis, and major surgery.
5. The same criteria are produced by infectious and non-infectious triggers, requiring further clinical assessment.

Part 2.2

6. Sepsis is life-threatening organ dysfunction from a dysregulated host response to infection.
7. Sepsis involves endothelial dysfunction, coagulation activation, and microvascular dysfunction impairing tissue oxygenation.
8. Older or immunocompromised patients can show subtle, non-specific deterioration rather than dramatic collapse.
9. The score uses respiratory rate, mental status, and blood pressure to identify higher-risk patients at the bedside.
10. Every hour of delay in antimicrobial treatment for sepsis measurably worsens patient outcome.

Part 2.3

11. Septic shock results from severe vasodilation and impaired tissue oxygen utilisation producing profound hypotension.
12. Septic shock involves vasodilation, unlike cardiogenic shock, from pump failure, or hypovolaemic shock, from volume loss.
13. Presentation includes persistent hypotension requiring vasopressors, elevated lactate, and altered mental status.
14. The bundle includes blood cultures, empirical antibiotics, fluid resuscitation, and serial lactate measurement.
15. Vasopressor therapy, most commonly noradrenaline, is added where fluid resuscitation alone fails to restore blood pressure.

Part 2.4

1. Multi-organ dysfunction syndrome is progressive, simultaneous dysfunction of two or more organ systems.
2. Systems commonly affected include respiratory, renal, cardiovascular, hepatic, neurological, and haematological.
3. Respiratory dysfunction presents as acute respiratory distress syndrome; renal dysfunction presents as acute kidney injury.
4. Management combines sepsis-directed treatment with organ-specific supportive care in intensive care.
5. Prognosis correlates directly with the number of organ systems involved and the degree of dysfunction in each.

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References and Attributions [Series 2]

1. Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
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3. Surviving Sepsis Campaign (2021). International Guidelines for Management of Sepsis and Septic Shock. Critical Care Medicine.
4. Mandell, G. L., Bennett, J. E., & Dolin, R. (2019). Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases (9th ed.). Elsevier.

Figures, Plates, Tables, and Boxes

1. Figure 2.1: A clinician checking a patient's vital signs at the bedside. AI-generated using the prompt: "Create a realistic clinical illustration titled Checking Vital Signs, showing a clinician reading values from a bedside monitor while attending to a patient in a hospital bed, clinical ward setting. Clean clinical illustration style, no text."
2. Table 2.1: Comparing SIRS criteria and the quick sequential organ failure assessment score. AI-generated using the prompt: "Create a clean reference table titled Comparing SIRS Criteria and the Quick Sequential Organ Failure Assessment, listing feature, SIRS criteria, and quick sequential organ failure assessment. Simple clinical reference table style with outside border."
3. Figure 2.2: A clinical team delivering a structured sepsis resuscitation bundle. AI-generated using the prompt: "Create a realistic clinical illustration titled Sepsis Resuscitation Bundle, showing two clinicians attending to a patient in an emergency department bed, one administering intravenous fluids and the other checking medication, urgent but organised atmosphere. Clean clinical illustration style, no text."
4. Table 2.2: Organ-specific manifestations of multi-organ dysfunction syndrome. AI-generated using the prompt: "Create a clean reference table titled Organ-Specific Manifestations of Multi-Organ Dysfunction Syndrome, listing organ system, manifestation, and key management approach. Simple clinical reference table style with outside border."

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Course code: MED 614

Course title: Infectious Diseases II

Course credit load: 2 Units

Series 3: Viral Infectious Syndromes

- **Part 3.1: Principles of Viral Infection**
  - Sub Part 3.1.1: Viral structure and replication
  - Sub Part 3.1.2: Viral pathogenesis and host response
  - Sub Part 3.1.3: Principles of antiviral therapy
- **Part 3.2: Respiratory Viral Syndromes**
  - Sub Part 3.2.1: Influenza
  - Sub Part 3.2.2: Other common respiratory viral infections
  - Sub Part 3.2.3: Management of respiratory viral infections
- **Part 3.3: Viral Exanthems and Herpesvirus Infections**
  - Sub Part 3.3.1: Common viral exanthems
  - Sub Part 3.3.2: Herpes simplex virus infection
  - Sub Part 3.3.3: Varicella-zoster virus infection
- **Part 3.4: Viral Hepatitis and Other Systemic Viral Syndromes**
  - Sub Part 3.4.1: Viral hepatitis overview
  - Sub Part 3.4.2: Infectious mononucleosis
  - Sub Part 3.4.3: Viral haemorrhagic fever

Introduction

This Series turns to **viral infectious syndromes**, working from the fundamental principles of viral structure and replication through to the specific respiratory, dermatological, and systemic viral syndromes genuinely commonly encountered in clinical practice. Building directly on the host-pathogen framework established in Series 1, this Series applies that framework specifically

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to the genuinely distinctive biology viruses demonstrate compared with bacteria and other microorganisms.

The aim of this Series is to equip you with an understanding of viral structure, replication, pathogenesis, and the principles of antiviral therapy, influenza and other respiratory viral syndromes and their management, common viral exanthems and herpesvirus infections and their management, and viral hepatitis, infectious mononucleosis, and viral haemorrhagic fever.

This Series opens with the **principles of viral infection**, moves through **respiratory viral syndromes** and **viral exanthems and herpesvirus infections**, and closes with **viral hepatitis and other systemic viral syndromes**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 3.1:** describe viral structure, replication, and pathogenesis
- **SLO 3.2:** describe the principles of antiviral therapy
- **SLO 3.3:** describe influenza and other common respiratory viral infections
- **SLO 3.4:** discuss the management of respiratory viral infections
- **SLO 3.5:** describe common viral exanthems and herpesvirus infections
- **SLO 3.6:** discuss the management of herpesvirus infections
- **SLO 3.7:** describe viral hepatitis and infectious mononucleosis
- **SLO 3.8:** describe viral haemorrhagic fever

3.1 Principles of Viral Infection

3.1.1 viral structure and replication

Genuinely obligate intracellular parasites

Viruses, genuinely obligate intracellular parasites lacking the independent metabolic machinery bacteria possess, consist of genetic material, either DNA or RNA, enclosed within a protective protein coat, the **capsid**, and, in some viruses, a surrounding lipid **envelope**, and this genuinely minimal structure directly explains why viruses absolutely require a host cell to replicate, in genuine contrast to the bacteria capable of independent replication already discussed in earlier Series of this course.

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Viral replication proceeds through attachment to a specific host cell receptor, genuinely echoing the adhesion principle already discussed for bacteria in Series 1, entry into the host cell, hijacking host cellular machinery to replicate viral genetic material and produce new viral proteins, assembly of new viral particles, and release, either through cell lysis or budding, to infect further host cells, a genuinely distinctive replication cycle directly underlying both viral pathogenesis and antiviral drug targets discussed later in this Part.

Fill in the blank: Viruses are genuinely obligate intracellular parasites absolutely requiring a host cell to _____.

3.1.2 viral pathogenesis and host response

Direct cellular damage, and the host's own immune contribution

Viral pathogenesis results from both **direct cytopathic effect**, genuine cellular damage or death resulting directly from the viral replication process itself, and, genuinely importantly, the host's own immune response to the infection, which, while ultimately protective, can itself contribute meaningfully to tissue damage and clinical symptoms, a genuinely important dual mechanism this Part aims to establish clearly.

The specific **tissue tropism** a given virus demonstrates, its genuine preference for infecting particular cell types or organ systems, directly determined by the specific host cell receptors the virus can bind to, directly explains the genuinely characteristic clinical syndrome each specific virus produces, a principle this Series returns to repeatedly across the specific viral syndromes covered in the remaining Parts.

Fill in the blank: Tissue tropism is a virus's genuine preference for infecting particular cell types, determined by specific host cell _____.

3.1.3 principles of antiviral therapy

Targeting the genuinely distinctive viral replication cycle

Antiviral medications, genuinely targeting specific steps within the viral replication cycle already discussed, including entry inhibitors, viral enzyme inhibitors, and agents interfering with viral assembly or release, offer a genuinely more targeted therapeutic approach than the broader antibacterial agents already discussed in Series 1, though genuinely fewer effective antiviral options exist overall compared with the wide range of antibacterial agents available.

Timing of antiviral therapy genuinely matters considerably, since many antiviral agents demonstrate meaningfully greater effectiveness when administered early in the course of

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infection, before genuinely extensive viral replication and associated tissue damage has occurred, a principle directly relevant to the specific management discussions covered across the remaining Parts of this Series.

Fill in the blank: Timing of antiviral therapy genuinely matters considerably, with meaningfully greater effectiveness when administered _____ in the course of infection.

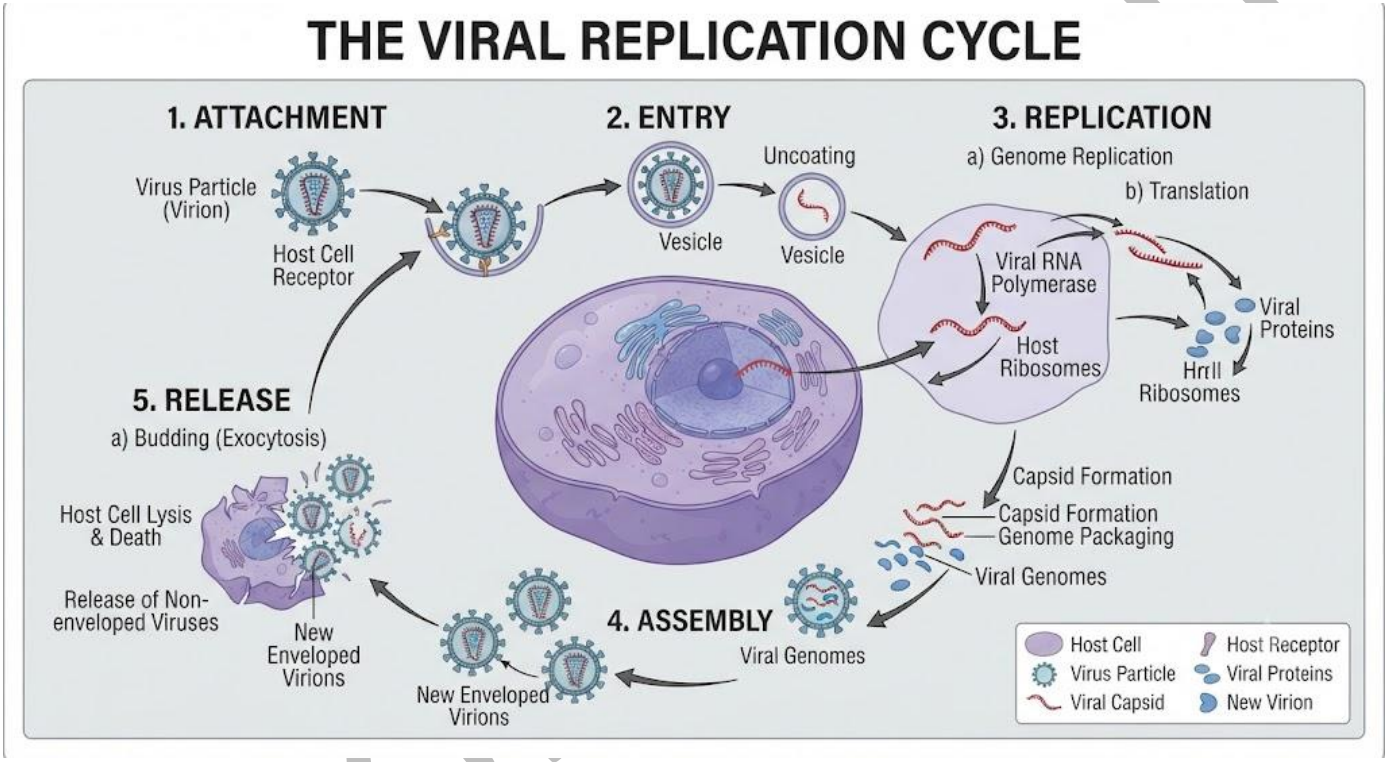

Figure 3.1: A labelled diagram of the viral replication cycle.

Pilot questions 3.1

- Describe the basic structure of a virus. (Linked to SLO 3.1)
- Describe the stages of the viral replication cycle. (Linked to SLO 3.1)
- Describe cytopathic effect and the host's own contribution to viral pathogenesis. (Linked to SLO 3.1)
- Describe tissue tropism. (Linked to SLO 3.1)
- Explain why the timing of antiviral therapy genuinely matters. (Linked to SLO 3.2)

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3.2 Respiratory Viral Syndromes

3.2.1 influenza

A genuinely significant seasonal respiratory pathogen

Influenza, presenting with abrupt-onset fever, myalgia, headache, and respiratory symptoms, typically genuinely more severe in overall systemic impact than the common cold already discussed in the following sub-Part, follows a genuinely characteristic seasonal pattern in temperate climates, and, given influenza's capacity for **antigenic drift**, gradual surface protein change, and, less commonly, **antigenic shift**, sudden, major surface protein change, seasonal vaccination requires genuine annual updating to remain effective.

High-risk groups, including older adults, young children, pregnant women, and patients with chronic underlying illness, carry genuinely elevated risk of severe influenza complications, including secondary bacterial pneumonia, and active identification of these specific high-risk patients directly informs both vaccination prioritisation and the threshold for considering antiviral treatment discussed further later in this Part.

Fill in the blank: Antigenic drift is gradual surface protein change, while antigenic shift is sudden, _____ surface protein change.

3.2.2 other common respiratory viral infections

A genuinely wide range of respiratory pathogens beyond influenza

Rhinovirus, the most common cause of the common cold, produces genuinely milder, predominantly upper respiratory symptoms, while **respiratory syncytial virus**, a genuinely important cause of bronchiolitis in young children and, increasingly recognised, significant respiratory illness in older adults, and **parainfluenza virus**, associated with croup in children, together illustrate the genuinely wide range of respiratory viral pathogens beyond influenza alone.

Coronaviruses, encompassing both genuinely mild, common cold-causing strains and, as more recently and genuinely dramatically demonstrated, strains capable of producing considerably more severe respiratory illness, further illustrate the genuinely wide clinical spectrum respiratory viral infection can span, and confidently distinguishing between these various respiratory viral pathogens, where clinically relevant, relies on specific epidemiological and clinical pattern recognition alongside targeted testing where genuinely indicated.

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Fill in the blank: Respiratory syncytial virus is a genuinely important cause of _____ in young children.

3.2.3 management of respiratory viral infections

Largely supportive, with targeted antiviral treatment in specific circumstances

Management of the great majority of respiratory viral infections is genuinely supportive, adequate hydration, rest, and symptomatic treatment, given the self-limiting natural history most of these infections follow in an otherwise healthy host, and this genuinely conservative default approach directly contrasts with the more aggressive antimicrobial treatment approach already discussed for bacterial infections and sepsis in earlier Series of this course.

Specific antiviral treatment, most notably neuraminidase inhibitors for influenza, already discussed in relation to timing in the previous Part, is genuinely reserved for patients at high risk of severe complications already discussed earlier in this Part, or those presenting with genuinely severe illness, given the modest but genuine clinical benefit these specific antiviral agents provide when administered within the appropriate treatment window.

Fill in the blank: Specific antiviral treatment, most notably neuraminidase inhibitors, is genuinely reserved for patients at high risk of severe _____.

Table 3.1: Comparing influenza with other common respiratory viral infections

Virus	Typical severity	Genuinely characteristic feature
Influenza	Often systemically severe	Abrupt onset, seasonal pattern
Rhinovirus	Generally mild	Predominantly upper respiratory symptoms
Respiratory syncytial virus	Variable, can be significant	Important cause of bronchiolitis in children

Pilot questions 3.2

1. Describe the typical presentation of influenza. (Linked to SLO 3.3)
2. Distinguish antigenic drift from antigenic shift. (Linked to SLO 3.3)
3. List high-risk groups for severe influenza complications. (Linked to SLO 3.3)
4. Describe other common respiratory viral pathogens beyond influenza. (Linked to SLO 3.3)

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5. Describe the general management approach to respiratory viral infections. (Linked to SLO 3.4)

3.3 Viral Exanthems and Herpesvirus Infections

3.3.1 common viral exanthems

Characteristic rash patterns, each with a genuinely distinctive appearance

Viral exanthems, widespread skin eruptions accompanying systemic viral infection, include **measles**, presenting with a characteristic cephalocaudal spreading maculopapular rash alongside fever and the pathognomonic Koplik spots within the oral mucosa, and **rubella**, presenting with a genuinely milder rash and lymphadenopathy, though carrying genuine, significant teratogenic risk if contracted during pregnancy.

Parvovirus B19, causing erythema infectiosum, presenting with a characteristic slapped-cheek facial appearance in children, and **hand, foot, and mouth disease**, most commonly caused by coxsackievirus, presenting with characteristic vesicular lesions across these three specific anatomical sites, further illustrate the genuinely wide range of characteristic viral exanthem presentations this Part aims to establish confident recognition of.

Fill in the blank: Koplik spots are pathognomonic lesions found within the oral mucosa in _____.

3.3.2 herpes simplex virus infection

A genuinely lifelong infection with recurrent reactivation

Herpes simplex virus, existing as type 1, classically causing orolabial infection, and type 2, classically causing genital infection, though genuinely with considerable clinical overlap between the two types, establishes **latent infection** within sensory nerve ganglia following primary infection, allowing genuine, recurrent reactivation and symptomatic recurrence throughout the affected individual's lifetime.

Primary infection is frequently more severe and symptomatic than subsequent recurrent episodes, and, beyond the characteristic mucocutaneous vesicular lesions, herpes simplex virus can genuinely cause more severe manifestations, including herpes encephalitis, already discussed as the most common cause of sporadic viral encephalitis in an earlier course of this programme, underlining the genuinely wide clinical spectrum this common virus can produce.

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Fill in the blank: Herpes simplex virus establishes latent infection within sensory nerve _____ following primary infection.

3.3.3 varicella-zoster virus infection

Chickenpox, then, potentially decades later, shingles

Varicella-zoster virus, causing **chickenpox** on primary infection, presenting with a characteristic widespread vesicular rash appearing in successive crops, similarly establishes latent infection within sensory nerve ganglia following primary infection, mirroring the herpes simplex virus pattern already discussed in the previous sub-Part, and can genuinely reactivate decades later as **herpes zoster**, shingles.

Herpes zoster presents with a characteristic vesicular rash confined to a single dermatome, the specific area of skin supplied by a single affected sensory nerve, reflecting the site of viral reactivation within that specific nerve's ganglion, and prompt antiviral treatment, discussed further in the following sub-Part, genuinely reduces both the severity and duration of the acute episode and the risk of the genuinely troublesome chronic complication of post-herpetic neuralgia.

Fill in the blank: Herpes zoster presents with a characteristic vesicular rash confined to a single _____.

Figure 3.2: A vesicular rash confined to a single dermatome, characteristic of herpes zoster.

Pilot questions 3.3

1. Describe measles and its characteristic features. (Linked to SLO 3.5)
2. Describe erythema infectiosum. (Linked to SLO 3.5)
3. Describe latent infection in herpes simplex virus. (Linked to SLO 3.5)
4. Distinguish chickenpox from herpes zoster. (Linked to SLO 3.5)
5. Describe the benefit of prompt antiviral treatment for herpes zoster. (Linked to SLO 3.6)

3.4 Viral Hepatitis and Other Systemic Viral Syndromes

3.4.1 viral hepatitis overview

Several genuinely distinct viruses sharing a common target organ

Viral hepatitis, already discussed in genuine depth in an earlier course of this programme covering gastroenterology, results from several genuinely distinct hepatotropic viruses, hepatitis A through E, each with a genuinely distinct transmission route, natural history, and chronicity risk, hepatitis A and E typically transmitted faeco-orally causing acute, self-limiting illness, and hepatitis B and C, typically transmitted parenterally, carrying genuine risk of chronic infection.

This Series's coverage of viral hepatitis genuinely complements, rather than repeats, the earlier gastroenterological coverage, emphasising the specific virological principles, structure, replication, and pathogenesis already established earlier in this Series, as they apply to this genuinely important group of hepatotropic viruses, reinforcing how the general viral principles established at the start of this Series apply across genuinely diverse specific clinical syndromes.

Fill in the blank: Hepatitis A and E are typically transmitted faeco-orally, causing acute, _____ illness.

3.4.2 infectious mononucleosis

A genuinely characteristic triad from Epstein-Barr virus infection

Infectious mononucleosis, most commonly resulting from Epstein-Barr virus infection, already introduced in relation to Hodgkin lymphoma in an earlier course of this programme, presents with a genuinely characteristic triad of fever, pharyngitis, and lymphadenopathy, alongside

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genuinely significant fatigue that can persist for weeks beyond resolution of the acute febrile illness, and splenomegaly in a meaningful proportion of affected patients.

Avoiding contact sports during the acute illness and for a genuinely defined period thereafter is specifically recommended given the genuine, though relatively uncommon, risk of splenic rupture from the splenomegaly already discussed, and this specific, practical precaution illustrates how understanding a condition's specific complications directly translates into genuinely concrete, actionable clinical advice for the affected patient.

Fill in the blank: Avoiding contact sports is specifically recommended given the genuine risk of splenic _____ from splenomegaly.

3.4.3 viral haemorrhagic fever

A genuinely rare but genuinely serious infection control priority

Viral haemorrhagic fever, a group of genuinely severe illnesses caused by several distinct virus families, presents with fever, widespread bleeding tendency, and, in genuinely severe cases, shock and multi-organ dysfunction already discussed in the previous Series of this course, and, while genuinely rare in most clinical settings, carries genuine, considerable importance given both its potential severity and its genuine transmissibility between individuals for several specific viruses within this category.

Rapid recognition and immediate isolation of a patient with suspected viral haemorrhagic fever, based on a genuinely relevant travel or exposure history combined with the characteristic clinical presentation, is genuinely essential to prevent onward transmission, and this specific infection control priority, closing this Series and this course's coverage of viral syndromes, underlines why a thorough travel history genuinely forms an essential part of assessing any patient with an unexplained, genuinely severe febrile illness.

Fill in the blank: Rapid recognition and immediate isolation of suspected viral haemorrhagic fever is genuinely essential to prevent onward _____.

Table 3.2: Comparing hepatitis A, B, and C transmission

Virus	Typical transmission route	Chronicity risk
Hepatitis A	Faeco-oral	Does not cause chronic infection
Hepatitis B	Parenteral, sexual, perinatal	Genuine risk of chronic infection

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Hepatitis C	Parenteral	Genuine, meaningful risk of chronic infection

Pilot questions 3.4

1. List the hepatotropic viruses and distinguish their transmission routes. (Linked to SLO 3.7)
2. Describe infectious mononucleosis and its characteristic triad. (Linked to SLO 3.7)
3. Explain why avoiding contact sports is recommended in infectious mononucleosis. (Linked to SLO 3.7)
4. Describe viral haemorrhagic fever. (Linked to SLO 3.8)
5. Explain why rapid recognition and isolation are genuinely essential in suspected viral haemorrhagic fever. (Linked to SLO 3.8)

Series Summary

This Series covered viral infectious syndromes from fundamental principles through to specific clinical presentations. Part 3.1 covered viral structure, replication, pathogenesis, and antiviral therapy, while Part 3.2 turned to respiratory viral syndromes. Part 3.3 covered viral exanthems and herpesvirus infections, and Part 3.4 closed with viral hepatitis and other systemic viral syndromes.

You should now be able to describe and manage the range of viral infectious syndromes covered across all four Parts of this Series, meeting the outcomes set for this Series. Series 4 turns next to anaerobic and non-tuberculous mycobacterial infections.

Glossary of Terms

1. **Antigenic drift:** gradual surface protein change in influenza requiring annual vaccine updating.
2. **Antigenic shift:** sudden, major surface protein change in influenza.
3. **Capsid:** the protective protein coat enclosing viral genetic material.
4. **Cytopathic effect:** direct cellular damage or death from viral replication.

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5. **Herpes zoster:** reactivation of latent varicella-zoster virus, presenting as shingles.
6. **Infectious mononucleosis:** a fever, pharyngitis, and lymphadenopathy syndrome from Epstein-Barr virus.
7. **Koplik spots:** pathognomonic oral mucosal lesions in measles.
8. **Latent infection:** dormant viral infection within sensory nerve ganglia capable of reactivation.
9. **Neuraminidase inhibitor:** a class of antiviral medication used for influenza treatment.
10. **Tissue tropism:** a virus's preference for infecting particular cell types or organs.
11. **Viral exanthem:** a widespread skin eruption accompanying systemic viral infection.
12. **Viral haemorrhagic fever:** a group of severe illnesses causing fever and widespread bleeding tendency.

Concept Check Questions [Series 3]

Objective questions

- Viruses are genuinely obligate intracellular parasites absolutely requiring a host cell to _____. (Linked to SLO 3.1)
- Antigenic drift is gradual surface protein change, while antigenic shift is sudden, _____ surface protein change. (Linked to SLO 3.3)
- Koplik spots are pathognomonic lesions found within the oral mucosa in _____. (Linked to SLO 3.5)
- Hepatitis A and E are typically transmitted faeco-orally, causing acute, _____ illness. (Linked to SLO 3.7)
- Rapid recognition and immediate isolation of suspected viral haemorrhagic fever is genuinely essential to prevent onward _____. (Linked to SLO 3.8)

Short-answer questions

1. Describe the stages of the viral replication cycle. (Linked to SLO 3.1)
2. List high-risk groups for severe influenza complications. (Linked to SLO 3.3)
3. Distinguish chickenpox from herpes zoster. (Linked to SLO 3.5)
4. Describe infectious mononucleosis and its characteristic triad. (Linked to SLO 3.7)
5. Describe viral haemorrhagic fever. (Linked to SLO 3.8)

Long-answer questions

6. Discuss viral structure, replication, pathogenesis, and the principles of antiviral therapy. (Linked to SLO 3.1, 3.2)

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7. Describe influenza and other common respiratory viral infections and their management. (Linked to SLO 3.3, 3.4)
8. Discuss common viral exanthems and herpesvirus infections and their management. (Linked to SLO 3.5, 3.6)
9. Describe viral hepatitis and infectious mononucleosis. (Linked to SLO 3.7)
10. Discuss viral haemorrhagic fever. (Linked to SLO 3.8)

Answers to Concept Check Questions [Series 3]

Objective questions

11. Replicate.
12. Major.
13. Measles.
14. Self-limiting.
15. Transmission.

Short-answer questions

16. Stages include attachment, entry, replication, assembly, and release.
17. High-risk groups include older adults, young children, pregnant women, and those with chronic illness.
18. Chickenpox is primary infection with widespread rash, while shingles is reactivation confined to a single dermatome.
19. Infectious mononucleosis presents with fever, pharyngitis, and lymphadenopathy, alongside fatigue and splenomegaly.
20. Viral haemorrhagic fever is a severe illness group causing fever and widespread bleeding tendency.

Long-answer questions

21. **Basic response:** Viruses are obligate intracellular parasites replicating through attachment, entry, replication, assembly, and release; pathogenesis involves cytopathic effect and host immune response; antiviral therapy targets specific replication steps and works best when timed early.

    Value-added response: A value-added response notes that tissue tropism directly explains the characteristic clinical syndrome each virus produces.

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22. **Basic response:** Influenza presents with abrupt systemic symptoms and undergoes antigenic drift and shift; other viruses include rhinovirus, respiratory syncytial virus, and coronaviruses; management is largely supportive, with antivirals reserved for high-risk patients.

    Value-added response: A value-added response notes that respiratory syncytial virus is an important cause of bronchiolitis in young children.

23. **Basic response:** Exanthems include measles, rubella, erythema infectiosum, and hand-foot-mouth disease; herpes simplex and varicella-zoster viruses establish latent infection with recurrent reactivation.

    Value-added response: A value-added response notes that prompt antiviral treatment for herpes zoster reduces severity and post-herpetic neuralgia risk.

24. **Basic response:** Viral hepatitis involves several hepatotropic viruses with distinct transmission and chronicity risk; infectious mononucleosis presents with a characteristic triad from Epstein-Barr virus, with genuine splenic rupture risk requiring activity precaution.

    Value-added response: A value-added response notes that hepatitis B and C carry genuine chronic infection risk, unlike hepatitis A and E.

25. **Basic response:** Viral haemorrhagic fever is a group of severe illnesses causing fever and bleeding tendency, requiring rapid recognition and isolation given transmissibility.

    Value-added response: A value-added response notes that a thorough travel history is essential when assessing unexplained severe febrile illness.

Answers to Pilot Questions [Series 3]

Part 3.1

1. A virus consists of genetic material enclosed in a protein capsid, with some viruses having a lipid envelope.
2. Stages include attachment, entry, replication, assembly, and release.
3. Cytopathic effect is direct cellular damage from replication; the host's own immune response also contributes to damage.

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4. Tissue tropism is a virus's preference for infecting particular cell types, determined by host cell receptors.
5. Many antivirals show greater effectiveness administered early before extensive replication and tissue damage occurs.

Part 3.2

6. Influenza presents with abrupt-onset fever, myalgia, headache, and respiratory symptoms.
7. Drift is gradual surface protein change; shift is sudden, major surface protein change.
8. High-risk groups include older adults, young children, pregnant women, and those with chronic illness.
9. Other pathogens include rhinovirus, respiratory syncytial virus, parainfluenza virus, and coronaviruses.
10. Management is largely supportive, with hydration, rest, and symptomatic treatment for most patients.

Part 3.3

11. Measles presents with a cephalocaudal maculopapular rash, fever, and pathognomonic Koplik spots.
12. Erythema infectiosum presents with a characteristic slapped-cheek facial appearance in children.
13. Latent infection establishes dormant virus within sensory nerve ganglia, allowing recurrent reactivation.
14. Chickenpox is primary infection with widespread rash; herpes zoster is reactivation confined to a single dermatome.
15. Prompt antiviral treatment reduces severity and duration of the acute episode and post-herpetic neuralgia risk.

Part 3.4

1. Hepatitis A and E are faeco-oral; hepatitis B and C are parenteral, carrying genuine chronic infection risk.
2. Infectious mononucleosis presents with fever, pharyngitis, and lymphadenopathy, plus fatigue and splenomegaly.
3. Splenomegaly carries a genuine, though uncommon, risk of splenic rupture during contact sports.
4. Viral haemorrhagic fever is a group of severe illnesses causing fever and widespread bleeding tendency.
5. Rapid isolation prevents onward transmission given the genuine transmissibility of certain viral haemorrhagic fever viruses.

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References and Attributions [Series 3]

1. Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
2. Murray, P. R., Rosenthal, K. S., & Pfaller, M. A. (2020). Medical Microbiology (9th ed.). Elsevier.
3. Mandell, G. L., Bennett, J. E., & Dolin, R. (2019). Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases (9th ed.). Elsevier.
4. World Health Organization (2023). Influenza Fact Sheet. WHO.

Figures, Plates, Tables, and Boxes

1. Figure 3.1: A labelled diagram of the viral replication cycle. AI-generated using the prompt: "Create a clean, accurate labelled educational diagram titled The Viral Replication Cycle, showing a sequential cycle with labelled stages for attachment, entry, replication, assembly, and release from a host cell. Educational anatomical diagram style, no photographic content."
2. Table 3.1: Comparing influenza with other common respiratory viral infections. AI-generated using the prompt: "Create a clean reference table titled Comparing Influenza with Other Common Respiratory Viral Infections, listing virus, typical severity, and genuinely characteristic feature. Simple clinical reference table style with outside border."
3. Figure 3.2: A vesicular rash confined to a single dermatome, characteristic of herpes zoster. AI-generated using the prompt: "Create a realistic clinical illustration titled Herpes Zoster Dermatomal Rash, showing a patient's torso with a band of small vesicular lesions following a single dermatomal distribution on one side of the body, clean neutral background. Clean clinical illustration style, no text."
4. Table 3.2: Comparing hepatitis A, B, and C transmission. AI-generated using the prompt: "Create a clean reference table titled Comparing Hepatitis A, B, and C Transmission, listing virus, typical transmission route, and chronicity risk. Simple clinical reference table style with outside border."

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Course code: MED 614

Course title: Infectious Diseases II

Course credit load: 2 Units

Series 4: Anaerobic and Non-Tuberculous Mycobacterial Infections

- **Part 4.1: Principles of Anaerobic Infection**
    - Sub Part 4.1.1: Microbiology of anaerobic organisms
    - Sub Part 4.1.2: Clinical settings favouring anaerobic infection
    - Sub Part 4.1.3: Diagnosis of anaerobic infection
- **Part 4.2: Specific Anaerobic Infectious Syndromes**
    - Sub Part 4.2.1: Clostridial infections
    - Sub Part 4.2.2: Anaerobic infections of the head, neck, and chest
    - Sub Part 4.2.3: Management of anaerobic infections
- **Part 4.3: Non-Tuberculous Mycobacteria I: Microbiology and Pulmonary Disease**
    - Sub Part 4.3.1: Microbiology and classification of non-tuberculous mycobacteria
    - Sub Part 4.3.2: Pulmonary non-tuberculous mycobacterial disease
    - Sub Part 4.3.3: Diagnosis of non-tuberculous mycobacterial disease
- **Part 4.4: Non-Tuberculous Mycobacteria II: Other Presentations and Management**
    - Sub Part 4.4.1: Extrapulmonary non-tuberculous mycobacterial disease
    - Sub Part 4.4.2: Non-tuberculous mycobacteria in the immunocompromised host
    - Sub Part 4.4.3: Management of non-tuberculous mycobacterial disease

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Introduction

This Series turns to two genuinely distinct groups of organisms demanding specific microbiological understanding beyond the general host-pathogen and viral principles already established earlier in this course, **anaerobic bacteria**, thriving specifically in low-oxygen environments, and **non-tuberculous mycobacteria**, a genuinely diverse group of organisms related to, though genuinely distinct from, the tuberculosis-causing mycobacterium already covered in an earlier infectious diseases course of this programme.

The aim of this Series is to equip you with an understanding of the microbiology of anaerobic organisms, the clinical settings favouring anaerobic infection, and its diagnosis, clostridial infections and anaerobic infections of the head, neck, and chest, and their management, and the microbiology, classification, pulmonary and extrapulmonary disease, diagnosis, and management of non-tuberculous mycobacteria.

This Series opens with the **principles of anaerobic infection**, moves through **specific anaerobic infectious syndromes** and **non-tuberculous mycobacteria I: microbiology and pulmonary disease**, and closes with **non-tuberculous mycobacteria II: other presentations and management**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 4.1:** describe the microbiology of anaerobic organisms and the clinical settings favouring anaerobic infection
- **SLO 4.2:** discuss the diagnosis of anaerobic infection
- **SLO 4.3:** describe clostridial infections and anaerobic infections of the head, neck, and chest
- **SLO 4.4:** discuss the management of anaerobic infections
- **SLO 4.5:** describe the microbiology, classification, and pulmonary disease of non-tuberculous mycobacteria
- **SLO 4.6:** discuss the diagnosis of non-tuberculous mycobacterial disease
- **SLO 4.7:** describe extrapulmonary non-tuberculous mycobacterial disease and disease in the immunocompromised host
- **SLO 4.8:** discuss the management of non-tuberculous mycobacterial disease

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4.1 Principles of Anaerobic Infection

4.1.1 microbiology of anaerobic organisms

Organisms genuinely thriving where oxygen is scarce or absent

Anaerobic bacteria, organisms genuinely unable to grow, or growing only poorly, in the presence of oxygen, form a genuinely major component of the normal human microbiome already introduced in Series 1, particularly within the gastrointestinal tract, oral cavity, and female genital tract, and this genuinely large normal anaerobic population directly explains why anaerobic infection so frequently arises from disruption of these specific normal anatomical sites.

Obligate anaerobes, genuinely unable to survive in oxygen-containing environments at all, and **facultative anaerobes**, capable of growing in either the presence or absence of oxygen, together comprise this genuinely diverse microbial group, and this specific oxygen requirement directly explains the distinctive clinical settings favouring anaerobic infection discussed in the following sub-Part of this Part.

Fill in the blank: Obligate anaerobes are genuinely unable to survive in _____-containing environments at all.

4.1.2 clinical settings favouring anaerobic infection

Low-oxygen tissue conditions, and specific anatomical breaches

Tissue necrosis, reduced local blood supply, and **foreign body presence**, each genuinely reducing local tissue oxygen tension, favour anaerobic organism proliferation, and clinical settings including deep, penetrating wounds, abscess formation, and infection following disruption of a normally anaerobe-rich anatomical site, the bowel following surgery or perforation already discussed in an earlier course of this programme, characteristically involve anaerobic organisms.

Mixed aerobic-anaerobic infection, rather than purely anaerobic infection alone, is genuinely more common in clinical practice, since the initial aerobic organism often genuinely consumes local oxygen, creating the low-oxygen conditions that then favour subsequent, or simultaneous, anaerobic organism proliferation, a genuinely important synergistic relationship directly relevant to the empirical antimicrobial coverage discussed later in this Series.

Fill in the blank: Mixed aerobic-anaerobic infection is genuinely more common than purely _____ infection alone.

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4.1.3 diagnosis of anaerobic infection

A genuinely distinctive clinical clue, and specific laboratory requirements

Clinical clues suggesting anaerobic infection include a genuinely characteristic foul odour from the infected site, gas formation within tissue, palpable or radiographically visible, and infection genuinely arising in proximity to a mucosal surface rich in normal anaerobic flora already discussed earlier in this Part, and recognising these specific clinical features directly prompts consideration of anaerobic involvement even before microbiological confirmation is available.

Specimen collection and transport for suspected anaerobic infection requires genuine, specific care, avoiding air exposure that would kill obligate anaerobic organisms already discussed, using specific anaerobic transport media, and this genuinely specific technical requirement, distinct from standard specimen handling already discussed in Series 1, directly explains why anaerobic culture can be genuinely more technically demanding than standard aerobic bacterial culture.

Fill in the blank: Specimen collection for suspected anaerobic infection requires avoiding air exposure that would kill obligate _____ organisms.

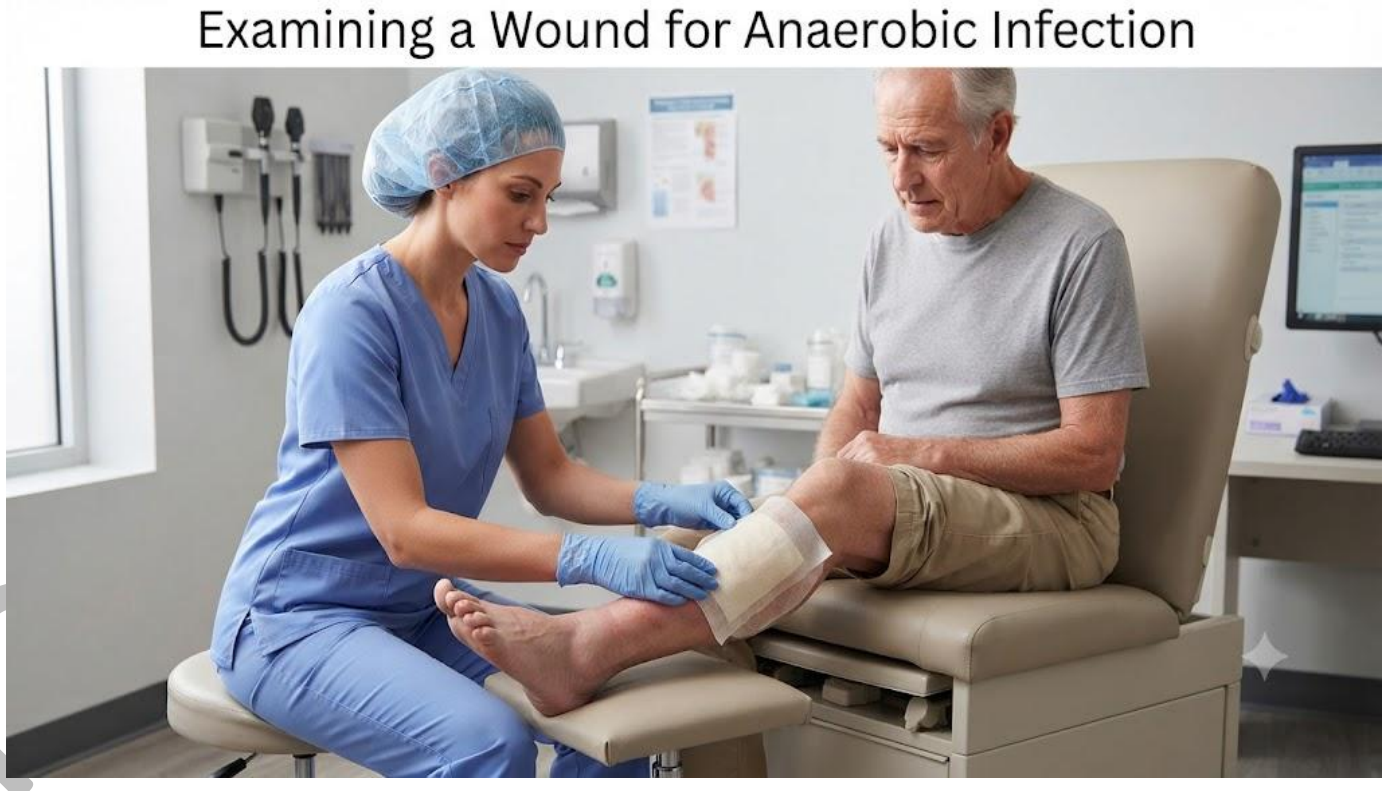

Figure 4.1: A clinician examining a wound for signs suggestive of anaerobic infection.

Pilot questions 4.1

- Describe anaerobic bacteria and their normal distribution within the body. (Linked to SLO 4.1)
- Distinguish obligate from facultative anaerobes. (Linked to SLO 4.1)
- Describe clinical settings favouring anaerobic infection. (Linked to SLO 4.1)
- Explain why mixed aerobic-anaerobic infection is genuinely more common than purely anaerobic infection. (Linked to SLO 4.1)
- Describe the clinical clues suggesting anaerobic infection and specific specimen handling requirements. (Linked to SLO 4.2)

4.2 Specific Anaerobic Infectious Syndromes

4.2.1 clostridial infections

A genus producing genuinely some of the most potent toxins known

Clostridium perfringens, causing gas gangrene, genuinely rapidly progressive, life-threatening tissue necrosis with characteristic gas formation, requires urgent surgical debridement alongside antimicrobial therapy given its genuinely aggressive natural history, and this specific condition represents a genuine surgical emergency demanding the same urgent recognition and response already emphasised for other time-critical conditions throughout this course.

Clostridium tetani, causing tetanus through a genuinely potent neurotoxin producing muscle rigidity and spasm, and **Clostridium botulinum**, causing botulism through a genuinely distinct neurotoxin producing flaccid paralysis, illustrate the genuinely wide-ranging, toxin-mediated disease this clostridial genus can produce, each requiring specific, distinct management including, where genuinely available and indicated, specific antitoxin therapy.

Fill in the blank: Clostridium perfringens causes gas gangrene, genuinely rapidly progressive tissue _____.

4.2.2 anaerobic infections of the head, neck, and chest

Infections arising from the genuinely anaerobe-rich oral cavity

Dental and oropharyngeal infection, given the genuinely dense anaerobic flora normally resident within the oral cavity already discussed earlier in this Series, can spread to produce deep

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neck space infection, and **Lemierre's syndrome**, a genuinely serious complication involving septic thrombophlebitis of the internal jugular vein following oropharyngeal infection, illustrates the genuine potential severity anaerobic head and neck infection can reach if inadequately treated.

Aspiration pneumonia, resulting from aspiration of oropharyngeal secretions genuinely rich in anaerobic organisms, particularly in patients with impaired swallowing or reduced consciousness, and its potential complication of **lung abscess**, already discussed in principle for cerebral abscess in an earlier course of this programme though affecting a genuinely distinct anatomical site, complete this Part's coverage of anaerobic infection affecting the head, neck, and chest.

Fill in the blank: Lemierre's syndrome involves septic thrombophlebitis of the internal jugular _____ following oropharyngeal infection.

4.2.3 management of anaerobic infections

Source control alongside genuinely targeted antimicrobial therapy

Source control, surgical drainage of an abscess or debridement of necrotic tissue already discussed in relation to gas gangrene, is genuinely essential for effective management of anaerobic infection, since antimicrobial therapy alone frequently fails to adequately penetrate and clear infection within necrotic tissue or an undrained abscess collection, mirroring the same source control principle already emphasised for other infections throughout this course.

Antimicrobial therapy for anaerobic infection requires genuinely specific agent selection given the distinct susceptibility pattern anaerobic organisms demonstrate compared with the aerobic organisms already discussed extensively elsewhere in this course, and, given the mixed aerobic-anaerobic infection pattern already discussed earlier in this Series, empirical treatment frequently requires genuinely combined coverage addressing both organism categories simultaneously.

Fill in the blank: Empirical treatment frequently requires genuinely combined coverage addressing both aerobic and anaerobic organism _____.

Table 4.1: Comparing clostridial infections

Organism	Condition	Key clinical feature
Clostridium perfringens	Gas gangrene	Rapidly progressive necrosis with gas formation
Clostridium tetani	Tetanus	Muscle rigidity and spasm

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Clostridium botulinum	Botulism	Flaccid paralysis

Pilot questions 4.2

1. Describe gas gangrene and its management priority. (Linked to SLO 4.3)
2. Distinguish tetanus from botulism by clinical presentation. (Linked to SLO 4.3)
3. Describe Lemierre's syndrome. (Linked to SLO 4.3)
4. Describe aspiration pneumonia and its relationship to anaerobic organisms. (Linked to SLO 4.3)
5. Explain why source control is genuinely essential in managing anaerobic infection. (Linked to SLO 4.4)

4.3 Non-Tuberculous Mycobacteria I: Microbiology and Pulmonary Disease

4.3.1 microbiology and classification of non-tuberculous mycobacteria

A genuinely diverse, environmentally widespread group of organisms

Non-tuberculous mycobacteria, a genuinely diverse group of mycobacterial species distinct from Mycobacterium tuberculosis and Mycobacterium leprae, are genuinely widely distributed within the environment, including soil and water sources, in genuine contrast to the person-to-person transmission characteristic of tuberculosis already discussed in an earlier infectious diseases course of this programme.

Classification includes **rapidly growing** species, producing visible culture growth within days, and **slowly growing** species, requiring genuinely considerably longer culture incubation periods similar to Mycobacterium tuberculosis itself, and this specific growth rate classification directly informs the practical laboratory approach to identification discussed further later in this Part.

Fill in the blank: Non-tuberculous mycobacteria are genuinely widely distributed within the environment, including soil and _____ sources.

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4.3.2 pulmonary non-tuberculous mycobacterial disease

A genuinely chronic, indolent respiratory presentation

Pulmonary non-tuberculous mycobacterial disease, most commonly affecting patients with pre-existing structural lung disease, discussed further in principle for chronic respiratory conditions in an earlier course of this programme, presents with a genuinely chronic, indolent course, chronic cough, fatigue, and weight loss, developing over months rather than the more acute presentation characteristic of many other respiratory infections already discussed elsewhere in this course.

This genuinely chronic, indolent presentation, combined with the genuinely wide environmental distribution of these organisms already discussed in the previous sub-Part, means a positive culture result alone does not automatically confirm genuine clinical disease, since the same colonisation-versus-infection spectrum already established in Series 1 applies genuinely directly to non-tuberculous mycobacteria, requiring the formal diagnostic criteria discussed further in the following sub-Part.

Fill in the blank: A positive culture result alone does not automatically confirm genuine clinical disease, given the colonisation-versus-infection _____.

4.3.3 diagnosis of non-tuberculous mycobacterial disease

Combining clinical, radiological, and microbiological criteria

Formal diagnostic criteria, requiring the combination of genuinely compatible clinical symptoms, characteristic radiological findings on chest imaging, and positive microbiological confirmation, typically from repeated respiratory specimens given the genuine risk of a single positive result representing transient colonisation rather than genuine infection, together confirm the diagnosis of clinically significant pulmonary non-tuberculous mycobacterial disease.

Species-level identification, distinguishing the specific non-tuberculous mycobacterial species involved, genuinely matters clinically given the meaningfully differing clinical behaviour, treatment approach, and prognosis different species demonstrate, and this specific requirement for species-level, rather than simply genus-level, identification directly parallels the immunophenotyping and cytogenetic subtyping already discussed for leukaemia in an earlier course of this programme.

Fill in the blank: Species-level identification genuinely matters clinically given the meaningfully differing clinical behaviour and treatment _____ different species demonstrate.

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Figure 4.2: A clinician reviewing a chest radiograph as part of diagnostic assessment.

Pilot questions 4.3

1. Describe non-tuberculous mycobacteria and their environmental distribution. (Linked to SLO 4.5)
2. Distinguish rapidly growing from slowly growing non-tuberculous mycobacteria. (Linked to SLO 4.5)
3. Describe the typical presentation of pulmonary non-tuberculous mycobacterial disease. (Linked to SLO 4.5)
4. Explain why a positive culture does not automatically confirm genuine disease. (Linked to SLO 4.6)
5. Describe the formal diagnostic criteria for pulmonary non-tuberculous mycobacterial disease. (Linked to SLO 4.6)

4.4 Non-Tuberculous Mycobacteria II: Other Presentations and Management

4.4.1 extrapulmonary non-tuberculous mycobacterial disease

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Beyond the lungs, a genuinely wide-ranging presentation

Lymphadenitis, most commonly cervical, particularly in young children, **skin and soft tissue infection**, often following direct inoculation through trauma or a specific environmental exposure, and, less commonly, **disseminated disease**, genuinely widespread infection affecting multiple organ systems, together illustrate the genuinely wide-ranging extrapulmonary presentation non-tuberculous mycobacterial disease can produce beyond the pulmonary disease already discussed in the previous Part.

Specific environmental exposure history, including recent aquarium or fish tank contact for certain species genuinely associated with skin and soft tissue infection, or recent tattooing or cosmetic procedures where infection control has been genuinely inadequate, provides genuinely valuable diagnostic clues directly relevant to identifying the specific likely causative species and appropriately targeted treatment approach.

Fill in the blank: Specific environmental exposure history provides genuinely valuable diagnostic clues relevant to identifying the specific likely causative _____.

4.4.2 non-tuberculous mycobacteria in the immunocompromised host

A genuinely more severe, disseminated pattern in this specific population

The **compromised host**, already introduced as a specific, genuinely important category in Series 1, carries genuinely elevated risk of disseminated non-tuberculous mycobacterial disease, particularly patients with advanced immunosuppression, and this specific patient population, given their genuinely reduced capacity to contain infection to a localised site, requires a genuinely heightened index of suspicion when presenting with unexplained systemic symptoms.

Disseminated disease in this specific population presents with genuinely non-specific systemic symptoms, fever, weight loss, and fatigue, alongside specific organ involvement depending on the particular pattern of dissemination, and prompt recognition and treatment genuinely matters given the significant associated morbidity this specific presentation carries in an already genuinely vulnerable patient population.

Fill in the blank: Disseminated non-tuberculous mycobacterial disease presents with genuinely non-specific systemic symptoms, fever, weight loss, and _____.

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4.4.3 management of non-tuberculous mycobacterial disease

A genuinely prolonged, multi-drug treatment approach

Multi-drug antimicrobial therapy, typically continued for a genuinely prolonged treatment course, often many months, given the generally slower response and genuine risk of treatment failure or relapse with shorter courses, forms the mainstay of management for confirmed non-tuberculous mycobacterial disease, with the specific drug combination genuinely tailored to the particular species identified given the meaningfully differing susceptibility patterns already discussed earlier in this Series.

Surgical management, resection of localised disease, offers a further treatment option in appropriately selected cases, particularly for extrapulmonary disease genuinely amenable to complete surgical excision, and this Series closes on the same theme of individualised, structured, species-specific treatment planning that has run throughout the whole of this course's coverage of infectious disease management.

Fill in the blank: Multi-drug antimicrobial therapy is typically continued for a genuinely prolonged treatment course, often many _____.

Table 4.2: Comparing pulmonary and extrapulmonary non-tuberculous mycobacterial disease

Feature	Pulmonary disease	Extrapulmonary disease
Typical population	Pre-existing structural lung disease	Varies by presentation type
Common presentation	Chronic cough, fatigue, weight loss	Lymphadenitis, skin and soft tissue infection
Diagnostic approach	Formal clinical, radiological, and microbiological criteria	Culture confirmation and exposure history

Pilot questions 4.4

1. Describe extrapulmonary presentations of non-tuberculous mycobacterial disease. (Linked to SLO 4.7)
2. Describe specific environmental exposures relevant to non-tuberculous mycobacterial diagnosis. (Linked to SLO 4.7)

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3. Describe disseminated non-tuberculous mycobacterial disease in the immunocompromised host. (Linked to SLO 4.7)
4. Describe the general management approach for non-tuberculous mycobacterial disease. (Linked to SLO 4.8)
5. Describe the role of surgical management for non-tuberculous mycobacterial disease. (Linked to SLO 4.8)

Series Summary

This Series covered anaerobic and non-tuberculous mycobacterial infections. Part 4.1 covered the microbiology, clinical settings, and diagnosis of anaerobic infection, while Part 4.2 turned to specific anaerobic infectious syndromes and their management. Part 4.3 covered the microbiology and pulmonary disease of non-tuberculous mycobacteria, and Part 4.4 closed with extrapulmonary disease and management.

You should now be able to describe and manage anaerobic and non-tuberculous mycobacterial infections across all four Parts of this Series, meeting the outcomes set for this Series. Series 5 turns next to fungal infections and infections complicating animal bites.

Glossary of Terms

1. **Anaerobic bacteria:** organisms genuinely unable to grow, or growing only poorly, in the presence of oxygen.
2. **Botulism:** flaccid paralysis from Clostridium botulinum neurotoxin.
3. **Facultative anaerobe:** an organism capable of growing in either the presence or absence of oxygen.
4. **Gas gangrene:** rapidly progressive tissue necrosis with gas formation from Clostridium perfringens.
5. **Lemierre's syndrome:** septic thrombophlebitis of the internal jugular vein following oropharyngeal infection.
6. **Multi-drug antimicrobial therapy:** prolonged combined treatment forming the mainstay of NTM disease management.
7. **Non-tuberculous mycobacteria:** mycobacterial species distinct from Mycobacterium tuberculosis and leprae.

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8. **Obligate anaerobe:** an organism genuinely unable to survive in oxygen-containing environments.
9. **Rapidly growing mycobacteria:** NTM species producing visible culture growth within days.
10. **Slowly growing mycobacteria:** NTM species requiring considerably longer culture incubation periods.
11. **Source control:** surgical drainage or debridement essential for effective infection management.
12. **Tetanus:** muscle rigidity and spasm from Clostridium tetani neurotoxin.

Concept Check Questions [Series 4]

Objective questions

- Obligate anaerobes are genuinely unable to survive in _____-containing environments at all. (Linked to SLO 4.1)
- Clostridium perfringens causes gas gangrene, genuinely rapidly progressive tissue _____. (Linked to SLO 4.3)
- Non-tuberculous mycobacteria are genuinely widely distributed within the environment, including soil and _____ sources. (Linked to SLO 4.5)
- Specific environmental exposure history provides genuinely valuable diagnostic clues relevant to identifying the specific likely causative _____. (Linked to SLO 4.7)
- Multi-drug antimicrobial therapy is typically continued for a genuinely prolonged treatment course, often many _____. (Linked to SLO 4.8)

Short-answer questions

1. Describe clinical settings favouring anaerobic infection. (Linked to SLO 4.1)
2. Distinguish tetanus from botulism by clinical presentation. (Linked to SLO 4.3)
3. Distinguish rapidly growing from slowly growing non-tuberculous mycobacteria. (Linked to SLO 4.5)
4. Describe the formal diagnostic criteria for pulmonary non-tuberculous mycobacterial disease. (Linked to SLO 4.6)
5. Describe disseminated non-tuberculous mycobacterial disease in the immunocompromised host. (Linked to SLO 4.7)

Long-answer questions

6. Discuss the microbiology of anaerobic organisms, clinical settings favouring anaerobic infection, and its diagnosis. (Linked to SLO 4.1, 4.2)

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7. Describe clostridial infections and anaerobic infections of the head, neck, and chest, and their management. (Linked to SLO 4.3, 4.4)
8. Discuss the microbiology, classification, pulmonary disease, and diagnosis of non-tuberculous mycobacteria. (Linked to SLO 4.5, 4.6)
9. Describe extrapulmonary non-tuberculous mycobacterial disease and disease in the immunocompromised host. (Linked to SLO 4.7)
10. Discuss the management of non-tuberculous mycobacterial disease. (Linked to SLO 4.8)

Answers to Concept Check Questions [Series 4]

Objective questions

11. Oxygen.
12. Necrosis.
13. Water.
14. Species.
15. Months.

Short-answer questions

16. Settings include tissue necrosis, foreign body presence, and disruption of anaerobe-rich anatomical sites.
17. Tetanus produces muscle rigidity and spasm, while botulism produces flaccid paralysis.
18. Rapidly growing species show visible growth within days, while slowly growing species require considerably longer.
19. Criteria require compatible symptoms, characteristic radiological findings, and positive microbiological confirmation.
20. Disseminated disease presents with non-specific systemic symptoms alongside specific organ involvement.

Long-answer questions

21. **Basic response:** Anaerobic bacteria are a major component of the normal microbiome, favoured by tissue necrosis and foreign body presence; diagnosis relies on clinical clues such as foul odour and gas formation, alongside specific anaerobic specimen handling.

    Value-added response: A value-added response notes that mixed aerobic-anaerobic infection is genuinely more common than purely anaerobic infection alone.

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22. **Basic response:** Clostridial infections include gas gangrene, tetanus, and botulism; head, neck, and chest anaerobic infections include Lemierre's syndrome and aspiration pneumonia; management requires source control alongside targeted antimicrobial therapy.

    Value-added response: A value-added response notes that antimicrobial therapy alone frequently fails without adequate source control.

23. **Basic response:** Non-tuberculous mycobacteria are environmentally widespread, classified as rapidly or slowly growing; pulmonary disease presents chronically; diagnosis requires combined clinical, radiological, and microbiological criteria.

    Value-added response: A value-added response notes that species-level identification genuinely matters given differing clinical behaviour.

24. **Basic response:** Extrapulmonary disease includes lymphadenitis, skin and soft tissue infection, and disseminated disease; the compromised host carries elevated risk of disseminated disease with non-specific systemic symptoms.

    Value-added response: A value-added response notes that specific environmental exposure history provides valuable diagnostic clues.

25. **Basic response:** Management uses prolonged multi-drug antimicrobial therapy tailored to the specific species, with surgical resection an option for appropriately selected localised disease.

    Value-added response: A value-added response notes that this reflects the same individualised, species-specific treatment planning theme running throughout this course.

Answers to Pilot Questions [Series 4]

Part 4.1

1. Anaerobic bacteria are unable to grow well with oxygen, forming a major component of the normal microbiome.
2. Obligate anaerobes cannot survive with oxygen at all, while facultative anaerobes can grow with or without it.

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3. Settings include tissue necrosis, foreign body presence, and disruption of anaerobe-rich anatomical sites.
4. The initial aerobic organism consumes local oxygen, creating conditions favouring subsequent anaerobic proliferation.
5. Clues include foul odour and gas formation; specimens require anaerobic transport media avoiding air exposure.

Part 4.2

6. Gas gangrene is rapidly progressive necrosis requiring urgent surgical debridement and antimicrobial therapy.
7. Tetanus produces muscle rigidity and spasm, while botulism produces flaccid paralysis.
8. Lemierre's syndrome is septic thrombophlebitis of the internal jugular vein following oropharyngeal infection.
9. Aspiration pneumonia results from aspirating anaerobe-rich oropharyngeal secretions, particularly with impaired swallowing.
10. Antimicrobial therapy alone often fails to penetrate necrotic tissue or an undrained abscess collection.

Part 4.3

11. Non-tuberculous mycobacteria are widely distributed in the environment, including soil and water sources.
12. Rapidly growing species show visible growth within days, while slowly growing species take considerably longer.
13. Presentation is chronic and indolent, with chronic cough, fatigue, and weight loss developing over months.
14. The same colonisation-versus-infection spectrum applies, meaning a positive culture alone is not diagnostic.
15. Criteria combine compatible symptoms, characteristic radiological findings, and repeated positive cultures.

Part 4.4

1. Presentations include lymphadenitis, skin and soft tissue infection, and disseminated disease.
2. Exposures include aquarium or fish tank contact and inadequately controlled tattooing or cosmetic procedures.
3. Disseminated disease presents with non-specific systemic symptoms alongside specific organ involvement.

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4. Management uses prolonged multi-drug antimicrobial therapy tailored to the specific species identified.
5. Surgical resection offers a further option, particularly for localised extrapulmonary disease amenable to excision.

References and Attributions [Series 4]

1. Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
2. Murray, P. R., Rosenthal, K. S., & Pfaller, M. A. (2020). Medical Microbiology (9th ed.). Elsevier.
3. Mandell, G. L., Bennett, J. E., & Dolin, R. (2019). Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases (9th ed.). Elsevier.
4. American Thoracic Society (2020). Treatment of Nontuberculous Mycobacterial Pulmonary Disease. American Journal of Respiratory and Critical Care Medicine.

Figures, Plates, Tables, and Boxes

1. Figure 4.1: A clinician examining a wound for signs suggestive of anaerobic infection. AI-generated using the prompt: "Create a realistic clinical illustration titled Examining a Wound for Anaerobic Infection, showing a clinician wearing gloves carefully inspecting and gently palpating a patient's wound dressing area, clinical examination room setting. Clean clinical illustration style, no text."
2. Table 4.1: Comparing clostridial infections. AI-generated using the prompt: "Create a clean reference table titled Comparing Clostridial Infections, listing organism, condition, and key clinical feature. Simple clinical reference table style with outside border."
3. Figure 4.2: A clinician reviewing a chest radiograph as part of diagnostic assessment. AI-generated using the prompt: "Create a realistic clinical illustration titled Reviewing a Chest Radiograph, showing a clinician examining a chest X-ray displayed on a viewing screen in a consultation room. Clean clinical illustration style, no text."
4. Table 4.2: Comparing pulmonary and extrapulmonary non-tuberculous mycobacterial disease. AI-generated using the prompt: "Create a clean reference table titled Comparing Pulmonary and Extrapulmonary Non-Tuberculous Mycobacterial Disease, listing feature, pulmonary disease, and extrapulmonary disease. Simple clinical reference table style with outside border."

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Course code: MED 614

Course title: Infectious Diseases II

Course credit load: 2 Units

Series 5: Fungal Infections and Infections Complicating Animal Bites

- **Part 5.1: Principles of Fungal Infection**
  - Sub Part 5.1.1: Microbiology and classification of fungi
  - Sub Part 5.1.2: Host defence against fungal infection
  - Sub Part 5.1.3: Diagnosis of fungal infection
- **Part 5.2: Superficial and Mucocutaneous Fungal Infections**
  - Sub Part 5.2.1: Dermatophyte infections
  - Sub Part 5.2.2: Candidiasis
  - Sub Part 5.2.3: Management of superficial fungal infections
- **Part 5.3: Invasive Fungal Infections**
  - Sub Part 5.3.1: Invasive candidiasis
  - Sub Part 5.3.2: Invasive aspergillosis
  - Sub Part 5.3.3: Other invasive fungal infections
- **Part 5.4: Treatment of Fungal Infections and Infections Complicating Animal Bites**
  - Sub Part 5.4.1: Principles of antifungal therapy
  - Sub Part 5.4.2: Microbiology and assessment of animal bite wounds
  - Sub Part 5.4.3: Management of infections complicating animal bites

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Introduction

This final Series closes the course with **fungal infections**, spanning superficial, mucocutaneous, and genuinely life-threatening invasive disease, and **infections complicating animal bites**, a genuinely distinct clinical scenario demanding specific microbiological understanding of the particular organisms these wounds characteristically introduce. Both topics apply the general host-pathogen and diagnostic principles established across this whole course to two genuinely specific, clinically important scenarios.

The aim of this Series is to equip you with an understanding of the microbiology, classification, host defence, and diagnosis relevant to fungal infection, dermatophyte infections and candidiasis and their management, invasive candidiasis, invasive aspergillosis, and other invasive fungal infections, and the principles of antifungal therapy alongside the microbiology, assessment, and management of infections complicating animal bites.

This Series opens with the **principles of fungal infection**, moves through **superficial and mucocutaneous fungal infections** and **invasive fungal infections**, and closes with **treatment of fungal infections and infections complicating animal bites**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 5.1:** describe the microbiology, classification, and host defence relevant to fungal infection
- **SLO 5.2:** discuss the diagnosis of fungal infection
- **SLO 5.3:** describe dermatophyte infections and candidiasis
- **SLO 5.4:** discuss the management of superficial fungal infections
- **SLO 5.5:** describe invasive candidiasis and invasive aspergillosis
- **SLO 5.6:** describe other invasive fungal infections
- **SLO 5.7:** discuss the principles of antifungal therapy
- **SLO 5.8:** discuss the microbiology, assessment, and management of infections complicating animal bites

5.1 Principles of Fungal Infection

5.1.1 microbiology and classification of fungi

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A genuinely distinct kingdom, sharing some biology with the human host

Fungi, genuinely eukaryotic organisms sharing a considerably closer biological relationship with human cells than the bacteria already discussed extensively in earlier Series of this course, are classified broadly as **yeasts**, single-celled, budding organisms, and **moulds**, filamentous organisms growing as branching hyphae, and this genuinely fundamental biological similarity to human cells directly explains why antifungal therapy, discussed later in this Series, presents genuinely distinct challenges compared with antibacterial therapy.

Dimorphic fungi, capable of existing as either yeast or mould form depending on the specific environmental temperature and conditions, illustrate the genuinely wide-ranging biological diversity within this kingdom, and understanding this basic classification framework directly underlies the specific fungal syndromes discussed across the remainder of this Series, from superficial skin infection through to genuinely life-threatening invasive disease.

Fill in the blank: Fungi share a considerably closer biological relationship with human cells than _____, already discussed extensively earlier in this course.

5.1.2 host defence against fungal infection

A genuinely important, though sometimes underappreciated, defensive role

Neutrophils, already discussed in relation to innate immunity in Series 1, provide genuinely essential defence against invasive fungal infection, particularly the invasive mould infections discussed later in this Series, and **cell-mediated immunity**, T lymphocyte-directed defence already introduced in Series 1, provides genuinely essential defence against certain other fungal infections, meaning specific patterns of immune compromise carry genuinely distinct fungal infection risk profiles.

Neutropenia, already discussed extensively in relation to aplastic anaemia and leukaemia treatment in an earlier course of this programme, carries genuinely elevated risk of invasive mould infection specifically, while conditions producing genuine cell-mediated immune compromise carry genuinely elevated risk of a somewhat different pattern of fungal infection, and recognising this specific distinction genuinely matters when assessing a compromised patient's particular fungal infection risk profile.

Fill in the blank: Neutropenia carries genuinely elevated risk of invasive _____ infection specifically.

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5.1.3 diagnosis of fungal infection

A genuinely challenging diagnostic area demanding a high index of suspicion

Microscopy and culture, direct visualisation and growth of the causative fungal organism, remain genuinely important diagnostic tools, though fungal culture frequently requires considerably longer incubation periods than standard bacterial culture already discussed in Series 1, and specific fungal biomarkers, including **galactomannan** and **beta-D-glucan**, offer genuinely valuable, more rapid supportive evidence for certain invasive fungal infections discussed further later in this Series.

Tissue biopsy, providing direct histological confirmation of fungal invasion into host tissue, is genuinely often required for confident diagnosis of invasive fungal infection, given the genuine diagnostic challenge distinguishing true invasive infection from mere colonisation, mirroring the colonisation-versus-infection spectrum already established in Series 1 and applied to non-tuberculous mycobacteria in the previous Series of this course.

Fill in the blank: Tissue biopsy provides direct histological confirmation of fungal invasion into host _____.

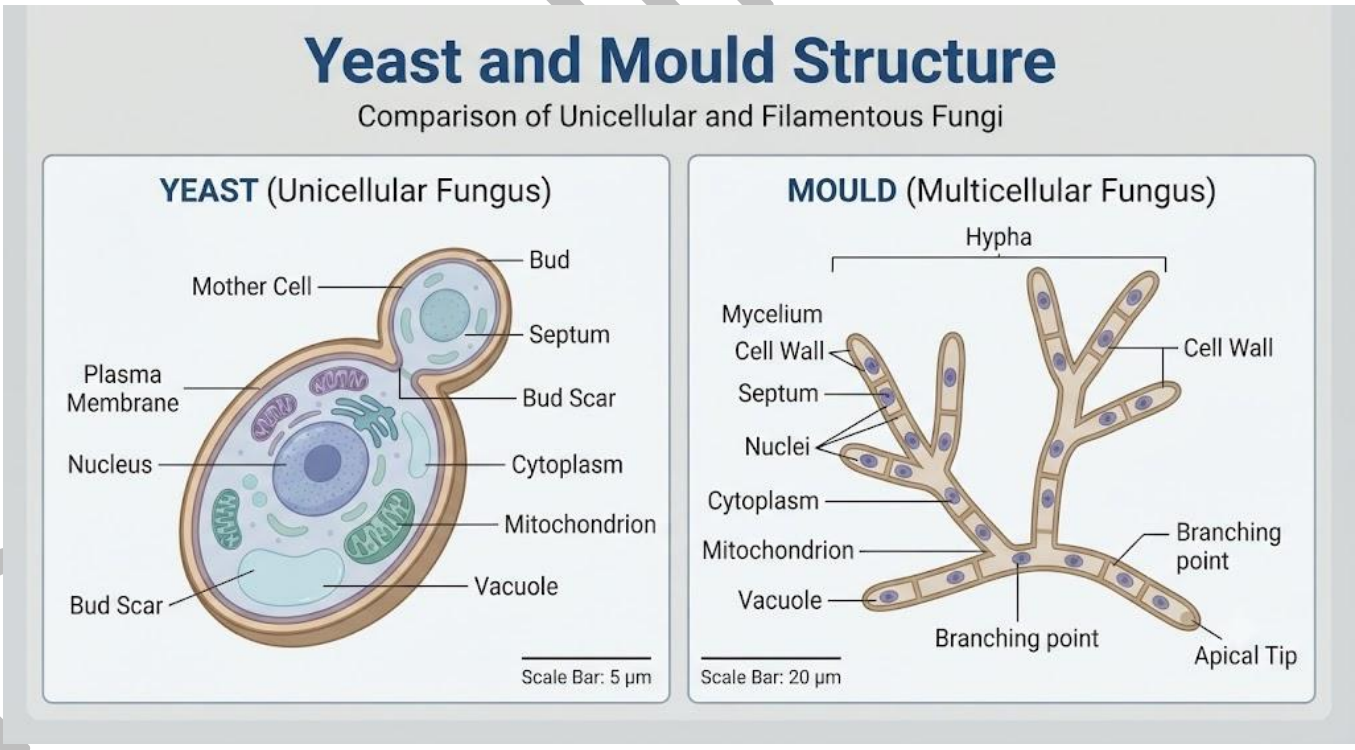

Figure 5.1: A labelled diagram comparing yeast and mould fungal structure.

Pilot questions 5.1

- Distinguish yeasts from moulds. (Linked to SLO 5.1)
- Describe dimorphic fungi. (Linked to SLO 5.1)
- Describe the roles of neutrophils and cell-mediated immunity in fungal host defence. (Linked to SLO 5.1)
- Explain why neutropenia carries elevated risk of invasive mould infection specifically. (Linked to SLO 5.1)
- Describe the diagnostic approach to suspected fungal infection. (Linked to SLO 5.2)

5.2 Superficial and Mucocutaneous Fungal Infections

5.2.1 dermatophyte infections

Fungi with a genuine preference for keratinised tissue

Dermatophyte infections, commonly termed **tinea**, or ringworm, result from fungi with a genuine preference for keratinised tissue, skin, hair, and nails, producing characteristic annular, scaling skin lesions with genuinely central clearing, and the specific clinical terminology used, tinea corporis for body involvement, tinea pedis for the feet, and tinea capitis for the scalp, directly reflects the specific anatomical site affected.

Diagnosis combines the characteristic clinical appearance already discussed with, where genuinely needed, direct microscopy of skin scrapings and fungal culture, and dermatophyte infections, while genuinely common and generally benign, can occasionally prove genuinely persistent or extensive, particularly in immunocompromised patients already discussed earlier in this Series, warranting more structured investigation and treatment in this specific context.

Fill in the blank: Dermatophyte infections produce characteristic annular, scaling skin lesions with genuinely central _____.

5.2.2 candidiasis

A genuinely common commensal organism turning pathogenic

Candida, a genus of yeast normally resident as commensal flora, already discussed in principle in Series 1, within the mouth, gastrointestinal tract, and genital tract, causes **mucocutaneous candidiasis** when this normal commensal-host balance is genuinely disrupted, including oral

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thrush, presenting with characteristic white, adherent plaques, and vulvovaginal candidiasis, presenting with itching and characteristic discharge.

Risk factors for mucocutaneous candidiasis include antibiotic therapy, already discussed in relation to disrupted colonisation resistance in Series 1, diabetes mellitus, and immunosuppression, and recognising these specific risk factors directly informs both the index of clinical suspicion and, in some cases, the identification of a previously unrecognised underlying predisposing condition when a patient presents with genuinely recurrent or unusually extensive candidiasis.

Fill in the blank: Candidiasis develops when the normal commensal-host balance is genuinely disrupted, allowing overgrowth of the resident _____.

5.2.3 management of superficial fungal infections

Topical treatment for most, with systemic therapy reserved for specific cases

Topical antifungal therapy, applied directly to the affected skin, nail, or mucosal surface, is genuinely effective for the great majority of uncomplicated superficial fungal infections already discussed in this Part, and this genuinely conservative, localised treatment approach reflects the generally limited, superficial nature of these infections compared with the invasive fungal disease discussed later in this Series.

Systemic antifungal therapy, reserved for genuinely extensive, treatment-refractory, or specific site involvement, including tinea capitis or nail infection genuinely poorly responsive to topical treatment given limited topical drug penetration into these specific sites, offers a further treatment option in appropriately selected cases, and addressing any identified underlying risk factor, already discussed in the previous sub-Part for candidiasis specifically, remains a genuinely essential complementary component of comprehensive management.

Fill in the blank: Systemic antifungal therapy is reserved for genuinely extensive, treatment-refractory, or specific site _____.

Table 5.1: Comparing dermatophyte infection and candidiasis

Feature	Dermatophyte infection	Candidiasis
Organism origin	Environmental or person-to-person acquisition	Normal commensal flora
Characteristic appearance	Annular lesion with central clearing	White adherent plaques or discharge

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Key risk factor	Warm, moist environments	Antibiotic use, diabetes, immunosuppression

Pilot questions 5.2

1. Describe dermatophyte infections and their characteristic appearance. (Linked to SLO 5.3)
2. Describe candidiasis and its underlying mechanism. (Linked to SLO 5.3)
3. List risk factors for mucocutaneous candidiasis. (Linked to SLO 5.3)
4. Describe the general management approach to superficial fungal infections. (Linked to SLO 5.4)
5. Explain when systemic antifungal therapy is genuinely indicated for superficial infection. (Linked to SLO 5.4)

5.3 Invasive Fungal Infections

5.3.1 invasive candidiasis

Bloodstream and deep tissue invasion by a normally commensal organism

Invasive candidiasis, encompassing **candidaemia**, bloodstream infection, and deep-seated organ involvement, genuinely occurs predominantly in critically unwell or significantly immunocompromised patients, particularly those with central venous catheters, already discussed in principle in relation to healthcare-associated infection risk elsewhere in this programme, providing a genuine portal of entry for this normally commensal organism already discussed earlier in this Series.

Clinical presentation genuinely mirrors bacterial sepsis, already discussed extensively in the previous Series of this course, with fever and haemodynamic instability, meaning invasive candidiasis should be actively considered in a critically unwell patient with genuinely relevant risk factors failing to improve despite appropriate antibacterial therapy, a genuinely important diagnostic consideration this Part aims to establish firmly.

Fill in the blank: Candidaemia is bloodstream infection by Candida, genuinely occurring predominantly in critically unwell or significantly _____ patients.

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5.3.2 invasive aspergillosis

A genuinely serious mould infection in the vulnerable host

Invasive aspergillosis, most commonly affecting the lungs, occurs predominantly in genuinely severely immunocompromised patients, particularly those with prolonged, significant neutropenia already discussed earlier in this Series, and presents with fever, cough, and chest pain, though genuinely often with a subtler, less specific presentation than bacterial pneumonia, making a genuinely high index of suspicion essential in this specific, vulnerable patient population.

Diagnosis combines characteristic radiological findings on chest imaging with the galactomannan biomarker already introduced earlier in this Series, and, where genuinely feasible, tissue or bronchoalveolar lavage sampling for microscopy and culture, and prompt diagnosis and treatment genuinely matters given invasive aspergillosis's considerable, genuine mortality risk in this already vulnerable patient population.

Fill in the blank: Invasive aspergillosis occurs predominantly in genuinely severely immunocompromised patients, particularly those with prolonged, significant _____.

5.3.3 other invasive fungal infections

A genuinely wide differential beyond candida and aspergillus

Cryptococcal infection, particularly cryptococcal meningitis, occurs predominantly in patients with genuinely significant cell-mediated immune compromise already discussed earlier in this Series, and **mucormycosis**, a genuinely aggressive, rapidly progressive mould infection, occurs predominantly in patients with poorly controlled diabetes or significant immunosuppression, each requiring genuinely prompt recognition given their considerable potential severity.

Recognising this genuinely wide differential of invasive fungal pathogens, rather than considering only candida and aspergillus already discussed earlier in this Part, and actively matching the specific pattern of immune compromise a patient demonstrates to the genuinely characteristic fungal infection risk that specific compromise carries, closes this Part's coverage and directly reinforces the compromised host framework already established in Series 1 of this course.

Fill in the blank: Mucormycosis is a genuinely aggressive, rapidly progressive mould infection occurring predominantly in patients with poorly controlled _____.

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Figure 5.2: A clinician examining a patient for a suspicious skin lesion in suspected invasive fungal infection.

Pilot questions 5.3

1. Describe invasive candidiasis and its typical risk factors. (Linked to SLO 5.5)
2. Describe the typical presentation of invasive aspergillosis. (Linked to SLO 5.5)
3. Describe the diagnostic approach to suspected invasive aspergillosis. (Linked to SLO 5.5)
4. Describe cryptococcal infection and its typical patient population. (Linked to SLO 5.6)
5. Describe mucormycosis. (Linked to SLO 5.6)

5.4 Treatment of Fungal Infections and Infections Complicating Animal Bites

5.4.1 principles of antifungal therapy

Genuinely fewer options, reflecting the shared host-pathogen biology

Antifungal drug classes, including azoles, echinocandins, and polyenes, each target genuinely distinct components of fungal cell structure or metabolism, and the genuinely fewer available antifungal options compared with the wide range of antibacterial agents already discussed extensively throughout this course directly reflects the closer biological relationship between

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fungal and human cells already established earlier in this Series, limiting the number of genuinely fungal-specific therapeutic targets available.

Antifungal selection requires genuine consideration of the specific fungal organism identified, its site of infection, given differing tissue penetration between antifungal classes, and patient-specific factors including organ function and potential drug interactions, and this genuinely individualised approach to antifungal selection directly mirrors the same structured antimicrobial selection principles already established for antibacterial therapy in Series 1 of this course.

Fill in the blank: Antifungal drug classes include azoles, echinocandins, and _____.

5.4.2 microbiology and assessment of animal bite wounds

A genuinely predictable, though wide-ranging, polymicrobial flora

Animal bite wounds, whether from dogs, cats, or other animals, introduce a genuinely predictable, though wide-ranging, polymicrobial flora reflecting the specific animal's normal oral microbiome, including **Pasteurella species**, particularly common following cat bites, and both aerobic and anaerobic organisms already discussed extensively earlier in this Series, given the genuinely dense anaerobic flora many animal oral cavities harbour.

Wound assessment, evaluating depth, location, and the specific mechanism of injury, genuinely matters clinically, since **puncture wounds**, particularly common following cat bites given their genuinely narrow, deep tooth structure, carry meaningfully higher infection risk than more superficial lacerations, since the deep, narrow puncture tract genuinely limits adequate wound irrigation and cleaning compared with a more open, accessible laceration.

Fill in the blank: Puncture wounds carry meaningfully higher infection risk than superficial lacerations given limited adequate wound _____.

5.4.3 management of infections complicating animal bites

Wound care, targeted antimicrobial coverage, and specific preventive measures

Wound management, including thorough irrigation and cleaning already discussed as genuinely more challenging for puncture wounds specifically, and careful clinical judgement regarding primary wound closure, given the genuinely elevated infection risk closure can carry for certain higher-risk bite wounds, forms the initial management priority, alongside prophylactic or, where infection is already established, targeted antimicrobial therapy covering the genuinely predictable polymicrobial flora already discussed.

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Tetanus prophylaxis, already discussed in relation to Clostridium tetani in the previous Series of this course, and **rabies risk assessment**, given the genuine, though geographically variable, risk this specific viral infection carries following certain animal bites, complete the genuinely comprehensive management approach animal bite wounds require, closing this Series, and the entire course, on the same theme of comprehensive, structured, multi-faceted infectious disease management that has run consistently throughout this whole programme.

Fill in the blank: Rabies risk assessment carries genuine, though geographically variable, risk following certain animal _____.

Table 5.2: Comparing higher-risk and lower-risk animal bite wounds

Feature	Higher-risk wound	Lower-risk wound
Wound type	Deep puncture wound	Superficial laceration
Typical source	Cat bite	Dog bite with clean edges
Management approach	Prophylactic antibiotics, caution with closure	Thorough cleaning, closure often appropriate

Pilot questions 5.4

1. Describe the antifungal drug classes and why fewer options exist compared with antibacterial agents. (Linked to SLO 5.7)
2. Describe the factors genuinely considered in antifungal selection. (Linked to SLO 5.7)
3. Describe the typical microbiology of animal bite wounds. (Linked to SLO 5.8)
4. Explain why puncture wounds carry meaningfully higher infection risk. (Linked to SLO 5.8)
5. Describe the comprehensive management approach for animal bite wounds. (Linked to SLO 5.8)

Series Summary

This final Series closed the course with fungal infections and infections complicating animal bites. Part 5.1 covered the microbiology, host defence, and diagnosis of fungal infection, while Part 5.2 turned to superficial and mucocutaneous fungal infections. Part 5.3 covered invasive

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fungal infections, and Part 5.4 closed with antifungal therapy and infections complicating animal bites.

You should now be able to describe and manage fungal infections and infections complicating animal bites across all four Parts of this Series, meeting the outcomes set for this Series. Across the full course, Series 1 covered host-pathogen interactions, Series 2 covered sepsis, systemic inflammatory response syndrome, and multi-organ dysfunction syndrome, Series 3 covered viral infectious syndromes, Series 4 covered anaerobic and non-tuberculous mycobacterial infections, and this Series has closed the course with fungal infections and infections complicating animal bites, together forming a comprehensive foundation in Infectious Diseases II.

Glossary of Terms

1. **Candidaemia:** bloodstream infection with Candida yeast.
2. **Candidiasis:** infection from overgrowth of the normally commensal Candida yeast.
3. **Dermatophyte infection:** fungal infection with a preference for keratinised skin, hair, and nail tissue.
4. **Dimorphic fungi:** fungi capable of existing as either yeast or mould form.
5. **Galactomannan:** a fungal biomarker supporting diagnosis of invasive aspergillosis.
6. **Invasive aspergillosis:** serious mould infection occurring predominantly in severely immunocompromised patients.
7. **Mucormycosis:** an aggressive, rapidly progressive mould infection in vulnerable hosts.
8. **Pasteurella species:** organisms particularly common following cat bite wound infection.
9. **Puncture wound:** a deep, narrow wound type carrying meaningfully higher infection risk.
10. **Rabies risk assessment:** evaluation of viral infection risk following certain animal bites.
11. **Tinea:** the common clinical term for dermatophyte infection, also called ringworm.
12. **Yeast:** single-celled, budding fungal organism.

Concept Check Questions [Series 5]

Objective questions

- Fungi share a considerably closer biological relationship with human cells than _____, already discussed extensively earlier in this course. (Linked to SLO 5.1)
- Dermatophyte infections produce characteristic annular, scaling skin lesions with genuinely central _____. (Linked to SLO 5.3)

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- Candidaemia is bloodstream infection by Candida, genuinely occurring predominantly in critically unwell or significantly _____ patients. (Linked to SLO 5.5)
- Antifungal drug classes include azoles, echinocandins, and _____. (Linked to SLO 5.7)
- Puncture wounds carry meaningfully higher infection risk than superficial lacerations given limited adequate wound _____. (Linked to SLO 5.8)

Short-answer questions

1. Distinguish yeasts from moulds. (Linked to SLO 5.1)
2. List risk factors for mucocutaneous candidiasis. (Linked to SLO 5.3)
3. Describe the typical presentation of invasive aspergillosis. (Linked to SLO 5.5)
4. Describe cryptococcal infection and its typical patient population. (Linked to SLO 5.6)
5. Describe the typical microbiology of animal bite wounds. (Linked to SLO 5.8)

Long-answer questions

6. Discuss the microbiology, classification, host defence, and diagnosis relevant to fungal infection. (Linked to SLO 5.1, 5.2)
7. Describe dermatophyte infections and candidiasis, and their management. (Linked to SLO 5.3, 5.4)
8. Discuss invasive candidiasis, invasive aspergillosis, and other invasive fungal infections. (Linked to SLO 5.5, 5.6)
9. Describe the principles of antifungal therapy. (Linked to SLO 5.7)
10. Discuss the microbiology, assessment, and management of infections complicating animal bites. (Linked to SLO 5.8)

Answers to Concept Check Questions [Series 5]

Objective questions

11. Bacteria.
12. Clearing.
13. Immunocompromised.
14. Polyenes.
15. Irrigation.

Short-answer questions

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16. Yeasts are single-celled, budding organisms, while moulds are filamentous organisms growing as branching hyphae.
17. Risk factors include antibiotic therapy, diabetes mellitus, and immunosuppression.
18. Presentation includes fever, cough, and chest pain, often subtler than bacterial pneumonia.
19. Cryptococcal infection occurs predominantly in patients with significant cell-mediated immune compromise.
20. Animal bite wounds introduce a predictable, wide-ranging polymicrobial flora including Pasteurella species.

Long-answer questions

21. **Basic response:** Fungi are classified as yeasts or moulds; neutrophils and cell-mediated immunity provide distinct host defence; diagnosis combines microscopy, culture, biomarkers, and tissue biopsy given the diagnostic challenge of colonisation versus infection.

    Value-added response: A value-added response notes that neutropenia and cell-mediated immune compromise carry genuinely distinct fungal infection risk profiles.

22. **Basic response:** Dermatophyte infections cause annular skin lesions from keratinised tissue infection; candidiasis results from disrupted commensal balance; management is largely topical, with systemic therapy reserved for extensive or refractory disease.

    Value-added response: A value-added response notes that identifying underlying risk factors is genuinely important for recurrent or extensive candidiasis.

23. **Basic response:** Invasive candidiasis mirrors bacterial sepsis in critically unwell patients; invasive aspergillosis affects severely neutropenic patients; cryptococcal infection and mucormycosis affect distinct vulnerable populations.

    Value-added response: A value-added response notes that matching the pattern of immune compromise to the characteristic fungal risk profile aids diagnosis.

24. **Basic response:** Antifungal classes include azoles, echinocandins, and polyenes, targeting distinct fungal structures; fewer options exist given closer fungal-human cell biological similarity; selection considers organism, site, and patient factors.

    Value-added response: A value-added response notes that this individualised selection approach mirrors antibacterial selection principles established earlier in the course.

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25. **Basic response:** Animal bites introduce predictable polymicrobial flora including Pasteurella species; assessment evaluates wound depth and mechanism; management includes wound care, targeted antimicrobials, tetanus prophylaxis, and rabies risk assessment.

    Value-added response: A value-added response notes that puncture wounds carry higher infection risk given limited wound irrigation capacity.

Answers to Pilot Questions [Series 5]

Part 5.1

1. Yeasts are single-celled, budding organisms, while moulds are filamentous organisms with branching hyphae.
2. Dimorphic fungi can exist as either yeast or mould form depending on environmental conditions.
3. Neutrophils defend against invasive mould infection, while cell-mediated immunity defends against other fungal infections.
4. Neutrophils provide essential defence against mould infection, so their deficiency elevates this specific risk.
5. Diagnosis uses microscopy, culture, biomarkers such as galactomannan, and tissue biopsy.

Part 5.2

6. Dermatophyte infections produce annular, scaling lesions with central clearing on keratinised tissue.
7. Candidiasis develops when normal commensal-host balance is disrupted, allowing yeast overgrowth.
8. Risk factors include antibiotic therapy, diabetes mellitus, and immunosuppression.
9. Management uses topical antifungal therapy for most uncomplicated superficial infections.
10. Systemic therapy is indicated for extensive, treatment-refractory, or specific site involvement such as nails.

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Part 5.3

11. Invasive candidiasis includes candidaemia and deep organ involvement, with central venous catheters a key risk factor.
12. Presentation includes fever, cough, and chest pain, often subtler than bacterial pneumonia.
13. Diagnosis combines chest imaging, galactomannan testing, and tissue or lavage sampling.
14. Cryptococcal infection occurs predominantly in patients with significant cell-mediated immune compromise.
15. Mucormycosis is an aggressive, rapidly progressive mould infection in patients with diabetes or immunosuppression.

Part 5.4

1. Classes include azoles, echinocandins, and polyenes; fewer options exist given closer fungal-human biological similarity.
2. Selection considers the specific organism, site penetration, and patient-specific factors.
3. Bite wounds introduce a predictable polymicrobial flora including Pasteurella species and anaerobic organisms.
4. Puncture wounds limit adequate irrigation and cleaning compared with more open, accessible lacerations.
5. Management includes wound care, targeted antimicrobials, tetanus prophylaxis, and rabies risk assessment.

References and Attributions [Series 5]

1. Kumar, P., & Clark, M. (2020). Kumar and Clark's Clinical Medicine (10th ed.). Elsevier.
2. Murray, P. R., Rosenthal, K. S., & Pfaller, M. A. (2020). Medical Microbiology (9th ed.). Elsevier.
3. Pappas, P. G., Kauffman, C. A., Andes, D. R., et al. (2016). Clinical Practice Guideline for the Management of Candidiasis. Clinical Infectious Diseases.
4. Mandell, G. L., Bennett, J. E., & Dolin, R. (2019). Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases (9th ed.). Elsevier.

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Figures, Plates, Tables, and Boxes

1. Figure 5.1: A labelled diagram comparing yeast and mould fungal structure. AI-generated using the prompt: "Create a clean, accurate labelled educational diagram titled Yeast and Mould Structure, showing a single, budding yeast cell beside a branching filamentous mould hypha, both clearly labelled. Educational anatomical diagram style, no photographic content."
2. Table 5.1: Comparing dermatophyte infection and candidiasis. AI-generated using the prompt: "Create a clean reference table titled Comparing Dermatophyte Infection and Candidiasis, listing feature, dermatophyte infection, and candidiasis. Simple clinical reference table style with outside border."
3. Figure 5.2: A clinician examining a patient for a suspicious skin lesion in suspected invasive fungal infection. AI-generated using the prompt: "Create a realistic clinical illustration titled Examining a Suspicious Skin Lesion, showing a clinician wearing gloves carefully examining a patient's forearm skin under good lighting, clinical examination room setting. Clean clinical illustration style, no text."
4. Table 5.2: Comparing higher-risk and lower-risk animal bite wounds. AI-generated using the prompt: "Create a clean reference table titled Comparing Higher-Risk and Lower-Risk Animal Bite Wounds, listing feature, higher-risk wound, and lower-risk wound. Simple clinical reference table style with outside border."

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