🦠 *Streptococcus pneumoniae* (Pneumococcus)

🔹 Diseases Caused

Most Common Cause of ⭐

- Community-acquired pneumonia (CAP) 🌬️ → Classically lobar pneumonia with consolidation
- Acute bacterial meningitis 🧠 (esp. adults)
- Sepsis in splenectomized patients 🚨 (OPSI)
- Otitis media 👂 & sinusitis 👃 (especially in children)

Other Infections

- Mastoiditis
- Conjunctivitis (pediatric predominance)
- Pericarditis ❤️
- Purulent bronchitis

📌 Exam Pearl: *S. pneumoniae* = leading cause of pneumonia + meningitis + OPSI

🔹 Important Properties

Morphology

- Gram-positive
- Lancet-shaped diplococci (oval with pointed ends) 🔍
- Occasionally short chains

📌 Exam tip: "Lancet-shaped diplococci" is a classic spotter phrase

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Culture Characteristics

- Grows on blood agar
- Produces α-hemolysis → greenish discoloration due to partial hemoglobin breakdown

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Key Laboratory Differentiation 🔬

Feature	*S. pneumoniae*	Viridans streptococci

Optochin	Sensitive ✅	Resistant ❌
Bile solubility	Soluble	Insoluble
Capsule	Present	Absent

📌 High-yield line:

👉 *Optochin sensitivity + bile solubility = Pneumococcus*

Capsule (Major Virulence Factor) 🛡️

- Composed of polysaccharide
- 91 antigenically distinct serotypes
- Protects against phagocytosis
- Basis of protective immunity

Identification

- Quellung reaction → capsule appears swollen when mixed with type-specific antiserum

📌 Exam Pearl: Anticapsular antibodies = opsonization + protection

C-Substance (C-Polysaccharide)

- Cell wall teichoic acid
- Reacts with C-reactive protein (CRP)

CRP Facts 💡

- Acute-phase reactant
- Nonspecific marker of inflammation
- Can rise up to 1000×
- Clinically: Better predictor of myocardial infarction risk than cholesterol

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🔹 Transmission

- Humans = only natural host (no animal reservoir)
- Carrier state: → 5–50% of healthy individuals harbor organism in oropharynx
- Disease is usually not communicable

- Infection occurs when host defenses are compromised

📌 Exam line: Pneumococcus = endogenous infection

🔹 Pathogenesis

Major Virulence Factor

- Capsular polysaccharide → Resists phagocytosis → Requires anticapsular IgG for opsonization

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Other Virulence Factors

Factor	Action
Lipoteichoic acid	Activates complement → ↑ cytokines → septic shock
Pneumolysin	Hemolysin → α-hemolysis, tissue damage
IgA protease	Cleaves IgA → enhances respiratory colonization

Mechanism of Pneumococcal Pneumonia

Inhalation or aspiration → Organisms reach alveoli → Rapid multiplication → Exudation of fluid + RBCs + WBCs → Lobar consolidation → Macrophage-mediated clearance → Resolution of consolidation

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🔄 Flowchart: Pathogenesis of Pneumococcal Infection

Colonization of oropharynx → IgA protease prevents mucosal clearance → Aspiration into lungs / spread to meninges, ear, sinuses → Capsular polysaccharide resists phagocytosis → Lipoteichoic acid activates complement → inflammation → Pneumolysin damages host tissues (α-hemolysis) → Clinical disease (Pneumonia / Meningitis / Otitis media / Bacteremia / sepsis)

🔹 Predisposing Factors

1. Impaired Defense Mechanisms

- Alcohol / drug intoxication 🍷💊 → ↓ cough reflex → aspiration
- CNS disorders → impaired clearance

2. Respiratory Abnormalities

- Viral infections → mucosal damage
- Bronchial obstruction
- Impaired mucociliary clearance

3. Cardiovascular Disease

- Pulmonary congestion
- Heart failure

4. Splenectomy / Functional Asplenia 🚨

- Sickle cell disease
- Nephrotic syndrome
- High risk of OPSI

5. Trauma

- Skull fracture with CSF leak → Predisposes to meningitis

📌 Exam Pearl: Pneumococcus = most dangerous pathogen in asplenic patients

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🧾 Clinical Findings

- Sudden onset chills + high fever 🌡️
- Cough with pleuritic chest pain
- Rusty sputum (RBC breakdown)
- Bacteremia: 15–25% cases
- Recovery: 5–10 days with antibody formation

Associated Infections

- Otitis media 👂
- Sinusitis
- Mastoiditis
- Conjunctivitis 👁️
- Pericarditis ❤️
- Meningitis 🧠
- Sepsis (leading cause in asplenic patients)

🔄 Flowchart: Clinical Course

Inhalation of pneumococcus → Alveolar infection (Fever, cough, pleuritic pain) → Rusty sputum production → Bacteremia (15–25%) → Local or systemic complications → Anticapsular antibody formation → Recovery in 5–10 days

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🧪 Laboratory Diagnosis

Microscopy

- Gram-positive lancet-shaped diplococci in sputum

Culture

- Blood agar → α-hemolytic colonies
- Bile soluble
- Optochin sensitive

Special Tests

- Quellung reaction → capsular swelling
- Blood culture → positive in 15–25%
- CSF culture → meningitis cases

Rapid Tests

- Latex agglutination → capsular antigen in CSF
- Urinary antigen test → C-polysaccharide

📌 Important: Antibiotic sensitivity testing is mandatory

💊 Treatment

Drugs of Choice

- Severe infection: Penicillin G
- Mild infection: Penicillin V (oral)

Alternatives

- Fluoroquinolones (Levofloxacin)
- Macrolides (Azithromycin, Erythromycin)

Resistance

- Low-level resistance (~25%) → altered PBPs
- High-level resistance (15–35%) → multiple PBPs
- ❌ No β-lactamase production

Resistant Strains

- Severe → Vancomycin
- Less severe → Ceftriaxone / Levofloxacin

📊 Table: Antibiotic Choices

Condition	First-Line	Alternatives	Resistant Strains
Severe	Penicillin G	Levofloxacin, Macrolides	Vancomycin
Mild	Penicillin V	Azithromycin, Erythromycin	Ceftriaxone, Levofloxacin

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🛡️ Prevention

High-Risk Groups

- Children <5 years 👶

- Elderly >50 years 👴
- Immunocompromised
- Splenectomized patients

Vaccines

- PCV13 (Prevnar 13)
  → Conjugate vaccine (polysaccharide + diphtheria toxoid)
- PPSV23 (Pneumovax 23) → Polysaccharide vaccine

Boosters

- At 65 years (if vaccinated earlier)
- Age 2–64 if asplenic, HIV+, on chemotherapy

Additional Points

- Protection lasts ~5 years
- Herd immunity via childhood vaccination
- Serotype replacement (e.g., 19A) → addressed by PCV13

🔄 Flowchart: Pneumococcal Prevention

High-risk individuals

→ PCV13 vaccination (children, immunocompromised)

→ PPSV23 in adults >50 years

→ Booster doses when indicated

→ Reduced nasopharyngeal carriage in children

→ Decreased adult disease burden

→ Surveillance for serotype replacement

-> The End <-