Microbiology lecture 3

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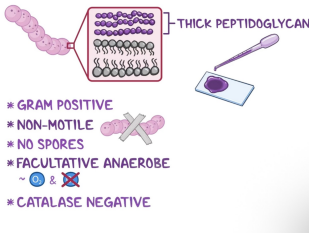
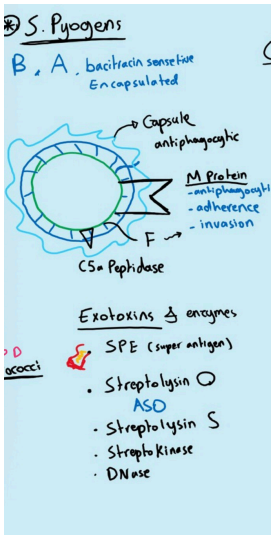
1. Streptococcus pyogenes (Group A Streptococcus - GAS)



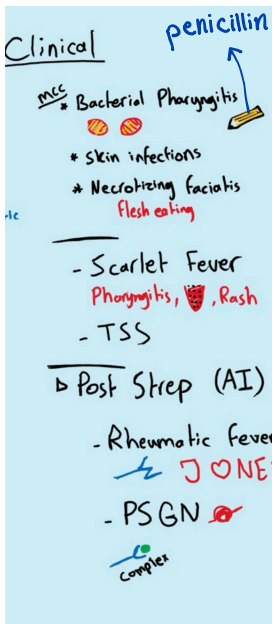
• General Features and Identification: S. pyogenes is a Gram-positive coccus arranged in chains. It is characterized by beta-hemolysis on blood agar and is sensitive to the antibiotic bacitracin.



• Virulence Factors:

o Hyaluronic acid capsule: This structure mimics host tissue to inhibit phagocytosis.
• M protein: A major anti-phagocytic factor, it is also responsible for molecular mimicry, which can lead to immune complications like rheumatic fever.

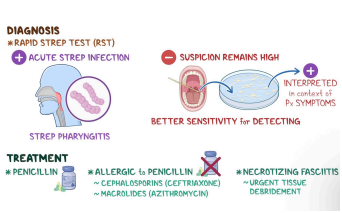
• Pili (fimbriae) and Protein F: These mediate adherence to epithelial cells in the oropharynx, which is a critical step for colonization.



• Clinical Presentation: GAS is the only pathogen causing acute pharyngitis that requires specific etiologic diagnosis and therapy. The clinical syndrome typically involves abrupt onset of intense sore throat, fever, chills, and pharyngeal/tonsillar exudates. Patients often exhibit tender anterior cervical lymphadenopathy.

• Post-Infectious Complications: Inadequate treatment can lead to delayed immune sequelae, including acute rheumatic fever (affecting the heart, joints, skin, and brain) and post-streptococcal glomerulonephritis.

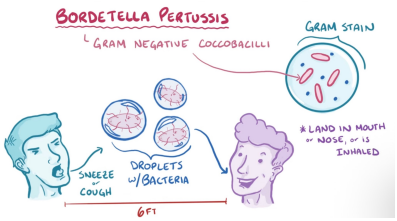
• Diagnosis: Diagnosis can be made via a Rapid Antigen Detection Test (RADT), which has high specificity. A throat culture remains the "gold standard," especially if an RADT is negative in children. Clinical tools like the Centor Score or Molsaac Guide help estimate the probability of infection.



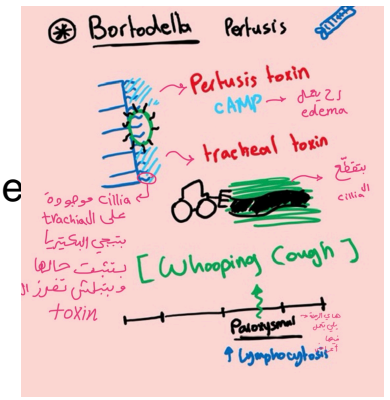
• Management: Penicillin V or Amoxicillin are the first-line therapies, as GAS remains universally susceptible to penicillin. For patients with penicillin allergies, macrolides like azithromycin are used. There is currently no approved commercial vaccine for GAS.

Tx long-term Antibiotics (e.g. Penicillin)

2. Bordetella pertussis

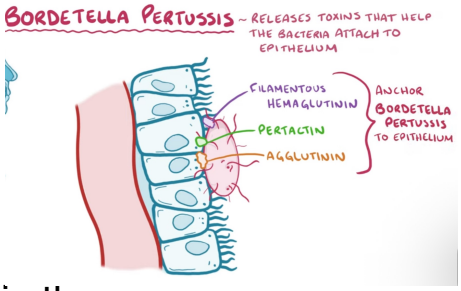


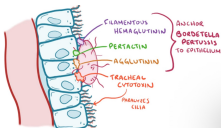
• General Features: This bacterium is a small Gram-negative coccobacillus. It is a strict aerobe and highly fastidious, requiring specific media like Regan-Lowe or Bordet-Gengou agar for culture.



• Epidemiology and Transmission: It is highly contagious and spreads via respiratory droplets. While vaccination led to a massive decline in cases, the transition to acellular vaccines (aP) has seen a resurgencedue to waning immunity.

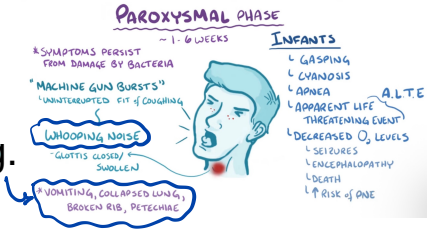
• Virulence Factors:
• Adhesins: Filamentous hemagglutinin (FHA), pertactin, and fimbriae facilitate attachment to ciliated respiratory epithelium.





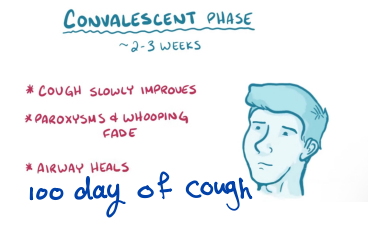
• Toxins: Includes Pertussis toxin (PT), which causes lymphocytosis, Adenylate cyclase toxin, Tracheal cytotoxin, and Dermonecrotic toxin.

• Clinical Stages of "Whooping Cough":
Catarrhal Phase: (1-2 weeks) Non-specific symptoms; this is the most contagious stage where antibiotics are most effective.



Paroxysmal Phase: (2-6 weeks) Characterized by severe coughing fits, an inspiratory "whoop," and post-tussive vomiting.

Convalescent Phase: (Weeks to months) A gradual decrease in cough, often called the "100-day cough".

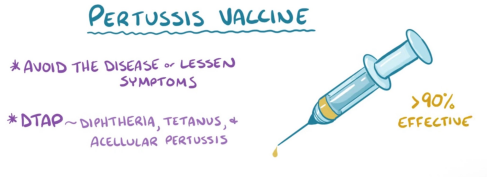


• Diagnosis: PCR of a nasopharyngeal swab is the most sensitive method early in the disease. Absolute lymphocytosis is a classic laboratory finding.

- Pneumonia (most common cause of death). Usually secondary bacterial pneumonia.
- Apnea leading to hypoxia. Vagal reflexes triggered by airway irritation
- Encephalopathy / seizures. From hypoxia, hypoglycemia.
- Weight loss, dehydration. Post-tussive vomiting, poor feeding and increased metabolic demand
- Mortality especially in young infants.

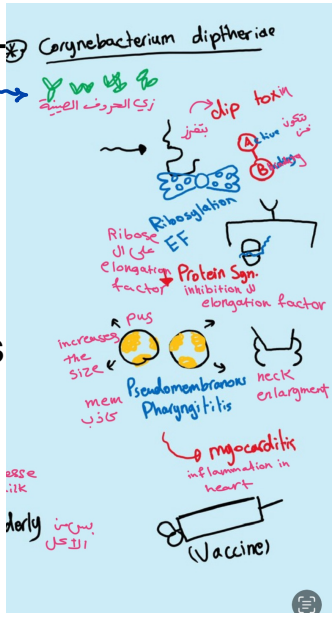
• Management: Macrolides (azithromycin or clarithromycin) are first-line to reduce transmission, though they do not alter the clinical course if started late. Supportive care (oxygen, fluids, monitoring for apnea) is the mainstay of treatment, especially for infants.

• Prevention: Routine immunization involves the DTaP (infants/ children) or Tdap (adults) vaccines.



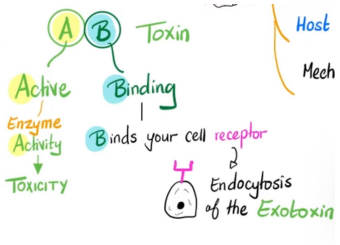
3. Corynebacterium diphtheriae

• General Features: These are Gram-positive, pleomorphic, club-shaped bacilli often seen in V or L shapes resembling "Chinese letters".



• Pathogenesis and Toxin: Only strains carrying a beta-phage (lysogenic conversion) produce the Diphtheria toxin. This A-B exotoxin inhibits host protein synthesis by ADP-ribosylating elongation factor-2 (EF-2).

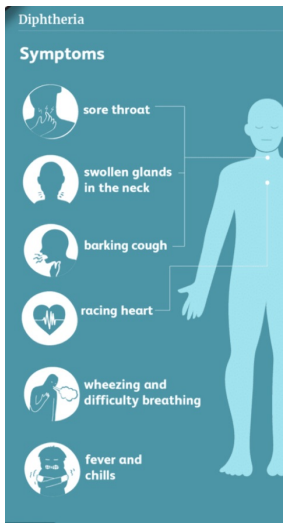
• Clinical Presentation:
° Respiratory Diphtheria: Gradual onset of sore throat and the formation of a thick, gray, adherent pseudomembrane over the tonsils and pharynx. This membrane bleeds if removal is attempted and can cause airway obstruction.



• Systemic Effects: Toxin dissemination can lead to myocarditis (arrhythmias, heart failure) and peripheral neuropathy.
• "Bull Neck": Caused by severe cervical lymphadenitis and soft-tissue edema.

• Diagnosis: Diagnosis is primarily clinical; treatment should not be delayed for laboratory results. Confirmation involves culture on tellurite agar (producing black colonies) and the Elek test to detect toxin production.

• Management: Diphtheria is a medical emergency. Management requires immediate administration of diphtheria antitoxin to neutralize unbound toxin, along with antibiotics (Penicillin or Erythromycin) to stop further toxin production. Patients require strict isolation.



• Prevention: Vaccination utilizes a toxoid that induces neutralizing antibodies. Booster doses are required every 10 years for adults.