**Short notes (summary) of lecture:**

***Scarlet fever , H. flu , Pertussis &Tetanus***

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# Scarlet Fever

Infectious disease caused by group A streptococcus,which is gram – positive cocci, aerobic bacteria.

The bacteria infects the throat, produces a toxin → scarlet fever.

Most common in children between two and ten y.o., it was very serious before, but now easily treatable.

- Streptococcus usually enters the body through the mouth or nose traveling through the respiratory tract.
- It travels through the body and lays between the cells and the skin tissues in most cases.
- Then the bacteria produce a toxin that causes several infections to occur.
- In rare cases, scarlet fever may develop from a streptococcal skin infection like impetigo. In these cases, the person may not get a sore throat.

## Transmitting Streptococcus:

- Direct contact from person to person: droplets of spray from the infected person or holding hands.

- Indirect contact: touching something that the infected person has touched and used like silverware.

## The incubation period:

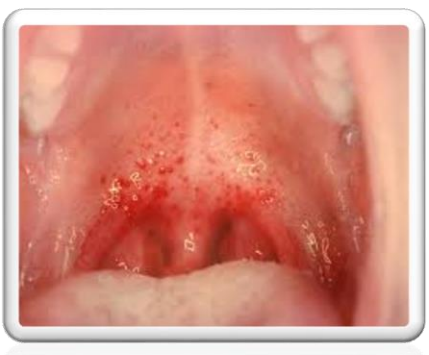
It is 1–4 days.

Asymptomatic carriage may occur in 15–20% of school-age children.

## Symptoms & signs:

- Fever
- Sore throat
- White strawberry tongue
- Peeling of the skin around the finger tips
- Forchheimer spots: fleeting small,red spots on the soft palate

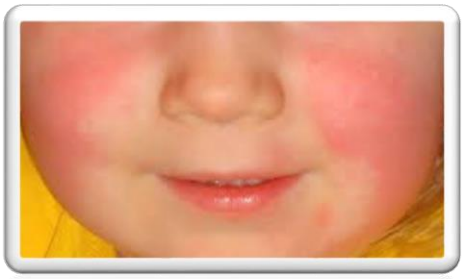
White strawberry tongue



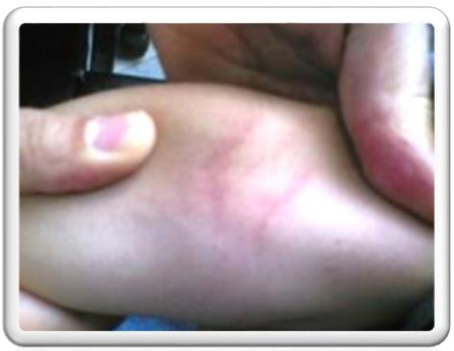
Forchheimer spots

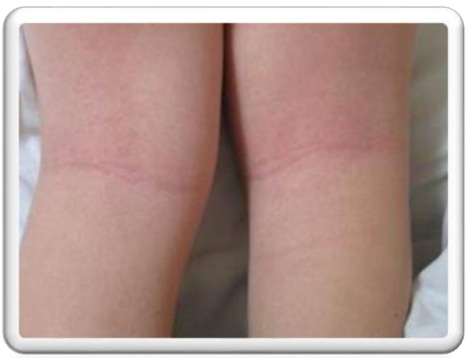
- Rash; which appears 12 days after the toxin is released into the body and 12-48 hours after the fever.

  - The rash starts on the neck and chest and spreads out over the body , Fine, tiny, red bumps.
  - blanches upon pressure.
  - on the face, often shows as red cheeks with a characteristic pale area around the mouth (circumoral pallor).



  - The rash worse in the skin folds (so-called Pastia's lines, where the rash runs together in the armpits and groin, can persist after the rash is gone).





  - It begins to fade three to four days after onset and desquamation (peeling) begins.
  - "This phase begins with flakes peeling from the face. Peeling from the palms and around the fingers occurs about a week later." Peeling also occurs in the axilla, the groin, and the tips of fingers and toes.

- Sandpapery" texture:

  lasts for about a week and then fades slowly - fading may take up to a month.

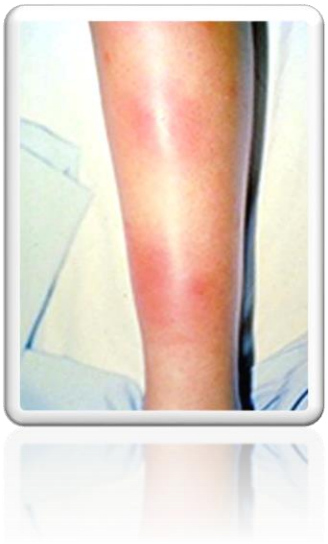
## Diagnosis:

- ❖ Complete blood count (high WBC with neutrophilia and conserved or increased eosinophils).
- ❖ High erythrocyte sedimentation rate and C-reactive protein.
- ❖ Elevation of antistreptolysin O titer.
- ❖ Throat culture is usually most successful.
- ❖ Blood culture is rarely positive.
- ❖ The rash is also important in diagnosis – the texture is more important than the look.

## Complications:

- It is due to spread of streptococci in blood, and immune-mediated complications due to an aberrant immune response
- Septic complications—today rare—include: ear and sinus infection, streptococcal pneumonia, empyema thoracis, meningitis, and full-blown sepsis (malignant scarlet fever).

- Immune complications include:
  - Acute glomerulonephritis.
  - Rheumatic fever.
  - Erythema nodosum

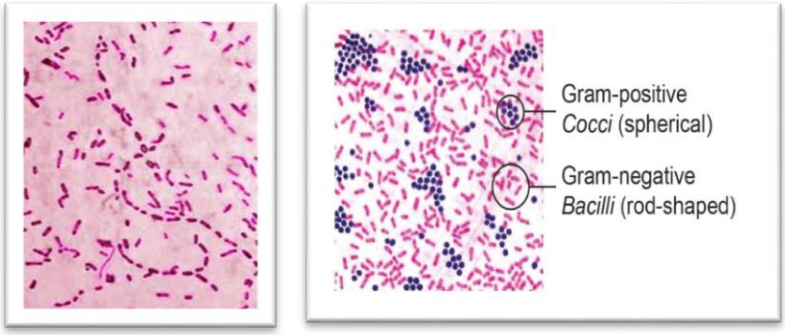


## Treatment:

- George Fredrick Dick developed the vaccine in the 1924 (discontinued due to poor efficacy and the introduction of antibiotics)
- Penicillin was then developed in the 1940's.
- A person with scarlet fever should not be infectious after 24 hours on antibiotics.

# *Haemophilus* influenza

- It is a gram negative rod (coccobacillus).
- A facultative anaerobe which grows best in media enriched with $co_2$.




- **Two major categories of H. influenzae were defined:** the unencapsulated strains and the encapsulated strains.
- Encapsulated strains were classified on the basis of their distinct capsular antigens.
- There are six generally recognized types of encapsulated H. influenzae: a, b, c, d, e, and f.
- Serotype b is most virulent type.
- Organism found only in humans.
- Unencapsulated strains are termed nontypable (NTHi) because they lack capsular serotypes.
- The unencapsulated strains are almost always less invasive.

## The incubation period:

- It is not certain but could be as short as a few days.

## Transmission:

- Haemophilus influenzae bacteria, including Hib, are spread person-to-person by direct contact or through respiratory droplets coughing and sneezing.
- Usually the bacteria remain in the nose and throat ,causing no harm. Sometimes the bacteria can enter the blood and spread, causing serious infection in the individual.

## Pathogenecity:

- Enters the body through respiratory tract ;

**Two types of behaviors:**

1. Asymptomatic colonization
2. Infections such as sinusitis, otitis media or pneumonia.

- Organism produces IgA protease which neutralizes respiratory mucosal IgA, this helps in its attachment to respiratory mucosa.

- After attachment to respiratory mucosa it can enter blood stream And cause: Bacteremia and meningitis.

- 95%of encapsulated forms(type b) responsible for these diseases

- Non capsulated forms are responsible for otitis media, sinusitis and pneumonia.

- In children the age group 6 months -6 years is most prone to infection by the organism.
- Peak incidence is from 6 months - 1 year.

## Risk Factors for Invasive Disease:

- **Exposure factors:**
  - household crowding
  - large household size
  - child care attendance
  - low socioeconomic status
  - low parental education
  - school-aged siblings
- **Host factors:**
  - race/ethnicity
  - chronic disease

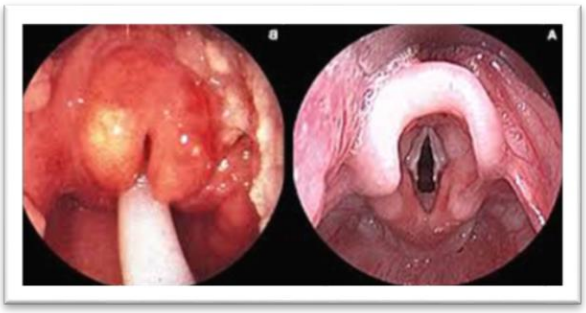
## Clinical features:

1. Meningitis:
   - Accounted for approximately 50%-65% of cases in the prevaccine era.
   - Hearing impairment or neurologic sequelae in 15%-30%.
   - Case-fatality rate 2%-5% despite of effective antimicrobial therapy

2. Otitis media and sinusitis cause pain in affected areas and redness and bulging of tympanic membrane.

3. Septic arthritis, cellulitis and sepsis (specially in splenectomized

4.Rarely epiglotitis in young children.



5.Pneumonia in elderly specially those with chronic respiratory diseases.

## Diagnosis:

- Diagnosis is considered confirmed when the organism is isolated from a sterile body site (cerebrospinal fluid or blood) which indicate H. influenzae infection.
- In this respect, H. influenzae cultured from the nasopharyngeal cavity or sputum would not indicate H. influenzae disease (because these sites are colonized in disease-free individuals).
- Gram staining.
- Organism is grown on chocolate agar.
- Definitive diagnosis can be made with Quellung test.
- Additional means of identifying encapsulated strains include fluorescent-antibody staining of the organism and latex agglutination tests, which detect the capsular polysaccharide.

## Treatment:

- Ceftriaxone is drug of choice in meningitis and other serious infections.

- Otitis media and sinusitis are treated with Augmentin.

## Prevention:

- It is by vacination.
- The vaccine given is called Hib
- It is in conjugated form. Conjugated with a carrier protein.
- Given in between 2-15 months.
- Conjugated is more effective than unconjugated one.
- Incidence has fallen 99% since prevaccine era.
- Most recent cases reported are unvaccinated or incompletely vaccinated children.

## Polysaccharide Conjugate Vaccines:

- Stimulates T-dependent immunity.
- Enhanced antibody production, especially in young children
- Repeat doses elicit booster response.

## Contraindications and Precautions:

- Severe allergic reaction to vaccine component or following a prior dose.
- Moderate or severe acute illness.
- Age  less than 6 weeks.

# Pertussis

-A highly contagious acute bacterial infection caused by the bacilli ***Bordetella pertussis***
-Currently worldwide prevalence is diminished due to active immunization
-However it remains a public health problem among older children & adults

**It continues to be an important respiratory disease afflicting unvaccinated infants and previously vaccinated children and adults (waning immunity)**

**Transmission:** through the respiratory route (droplet infection )

- Adolescents and adults are the reservoir. No animal or insect reservoir
- A highly communicable disease. 80% among households contacts
- In the catarrhal stage and 2 weeks after the onset of cough

## Etiology:

*Bordetella pertussis* – aerobic gram-negative coccobacilli

- Produces **toxins** namely pertussis toxin, filamentous hemagglutinin, hemolysin, adenylate cyclase toxin, dermonecrotic toxin and tracheal cytotoxin- responsible for clinical features (toxin mediated disease) and the immunity

## Pathogenesis:

The organism get attached to the respiratory cilia and toxin causes paralysis of cilia

muocopurulent-sanguineous exudate forms in the respiratory tract

This exudate predisposes to atelectasis, cough, cyanosis and pneumonia -Organism causes local tissue damage and systemic effects mediated through its toxin

## CLINICAL MANIFESTATIONS:

**Incubation period**: 7-10 days

- Infection lasts for 6 weeks – 10 weeks

- **Stage I (catarrhal stage; 1-2 weeks):** insidious onset of coryza, sneezing, low grade fever and occasional cough

- **Stage II (paroxysmal cough stage; 1-6 weeks):** due to difficulty in expelling the thick mucous form the tracheobronchial tree
- At the end of paroxysm long inspiratory effort is followed by a whoop
- In between episodes child look well. During episode of cough the child may become cyanosed, followed by vomiting, exhaustion and seizures

- Cough increase for next 2-3 weeks and decreases over next 10 weeks
- Absence of whoop and/or post-tussive vomiting does not rule out clinical diagnosis of pertussis

**paroxysmal cough>2 weeks with or without whoop and/or post-tussive vomiting is the hallmark feature of pertussis**

- **Stage III (convalecence stage):** period of gradual recovery even up to 6 months

## COMPLICATIONS:

1. Secondary pneumonia (1 in 5) and apneic spells (50%; neonates and infant<6 months of age)
2. Neurological complications: seizures (1 in 100) and encephalopathy (1 in 300) due to the toxin or hypoxia or cerebral hemorrhage
3. Otitis media, anorexia and dehydration, rib frcture, pneumothorax, subdural hematoma, hernia and rectal prolapse

### Differential diagnosis:

1. B. parapertussis, adenovirus, mycoplasma pneumonia, and chlamydia trachomatis
2. **Foreign body aspiration**, endobronchial tuberculosis and a mass pressing on the airway

## DIAGNOSIS:

1. Suspected on the basis of history and clinical examination and is confirmed by culture, genomics or serology
2. Elevated WBC count with lymphocytosis. The absolute lymphocyte count of **≥20,000** is highly suggestive
3. Culture: gold standard specially in the catarrhal stage. A saline nasal swab or swab from the posterior pharynx is preferred and the swab should be taken using dacron or calcium alginate and has to be plated on to the selective medium

However culture are not recommended in clinical practice as the yield is poor because of previous vaccination, antibiotic use, diluted specimen and faulty collection and transportation of specimen.

4. PCR: most sensitive to diagnose; can be done even after antibiotic exposure. It should always be used in addition with cultures
5. Direct fluorescent antibody testing:low sensitivity & variable specificity

## TREATMENT:

1. Avoidance of irritants, smoke, noise and other cough promoting factors
2. Antibiotics: effective only if started early in the course of illness.

**Erythromycin** (40-50 mg/kg/day 6 hr orally for **2 weeks** or

**Azithromycin** 10 mg/kg for **5 days** in children <6 months

and for children>6 months 10 mg/kg on day 1, followed by 5mg/kg from day2-5 or **Clarithromycin** 15 mg/kg 12 hrly for **7 days**

3. Supplemental oxygen, hydration, cough mixtures and bronchodilators (in individual cases)

## PREVENTION:

- All household contacts should be given erythromycin for 2 weeks
- Children <7 years of age not completed the four primary dose should complete the same at the earliest
- Children <7 years of age completed primary vaccination but not received booster in the last 3 years have to be given a single booster dose
- VACCINE

# Tetanus

Tetanus is an acute, fatal, severe **exotoxin** mediated nervous system disorder characterized by muscle spasm

- Caused by the toxin producing anaerobe, ***Clostridium tetani***
- **Tetanus is the only vaccine preventable disease that is infectious but not contagious from person to person**
- C. tetani is a part of the normal flora in human and animal intestines and is disseminated through excreta
- In spore form they are hard and long lasting in soil and dust
- The contamination of wound, unhygienic and improper handling of the umbilical cord in newborns, lack of hygienic habits and aseptic care during and after delivery are the main risk factors for infection

## PATHOGENESIS:

Tetanus occurs when spores of *C.tetani* found in soil gain access to damaged human tissue.

After inoculation, C. tetani transforms into a vegetative rod shaped bacterium and produces the metalloprotease, tetanospasmin.

After reaching the spinal cord and brainstem via retrograde axonal transport and binding tightly and irreversibly to receptor , tetanus toxin blocks neurotransmission .

Net effect is disinhibition of anterior horn cells and autonomic nervous system resulting in increased muscle tone, painful spasms and widespread autonomic instability.

## PREDISPOSING FACTORS:

A penetrating injury – inoculation of *C. tetani* spores

- Coinfection with other bacteria
- Devitalized tissue
- A foreign body
- Localized ischemia
- ***Therefore tetanus develop in these clinical settings***: neonates, obstetric patients, postsurgical patients, patients with dental infection, diabetic patients with infected extremity ulcers, patients who inject illicit and/or contaminated drugs

## CLINICAL MANIFESTATIONS:

- Incubation period: 1-8 days

- **Generalized tetanus:**
  - Presenting feature is trismus
  - Symptoms of autonomic overactivity such as irritability, restlessness, sweating, tachycardia, cardiac arrhythmias, labile hypotension or hypertension and fever
  - Tonic contractions of skeletal muscles (stiff neck, opisthotonus, risus sardonicus, board like rigid abdomen) and intermittent intense muscular spasms with no impairment of consciousness
  - Painful spasms, triggered by loud noises or other sensory stimuli such as physical contact or light
  - Period of apnea and/or upper airway obstruction due to contraction of thoracic muscles and/or glottal or pharyngeal muscle

- **Neonatal tetanus:**
  - Manifested by rigidity, spasms, trismus, inability to suck and seizures

**Diagnosis: mainly clinical**

## TREATMENT:

Best in the ICU as child may need early and aggressive airway management

- The goals of treatment include

1. **Halting toxin production**
   - Wound debridement
   - Antimicrobial therapy: **metronidazole or penicillin** G for **7-10 days**

2. **Neutralization of unbound toxin:**
   - HTIG-3,000-6,000 units i.m.

Equine antitoxin 1,500-3,000 units i.m. or i.v.

3. **Control of muscle spasms**
- Avoidance of sensory stimuli
- Sedatives: diazepam

4. **Management of autonomic dysfunction:**
- Magnesium sulfate, beta blockers, morphine sulfate

5. **Airway management and other supportive measures**
- **Main treatment** as bound tetanus toxin can not be displaced from the
- nervous system
- Endotracheal intubation/tracheostomy, nutritional support, physical
- therapy as soon as spasms have ceased

## PREVENTION:

- Immunization and proper treatment of wounds and traumatic injuries
-

## PROGNOSIS:

- The average mortality of tetanus is 45-55%
- Neonatal tetanus: 60-70%
- Most important factor influencing outcome is supportive care

## VACCINE:

- DTaP vaccine: 3 primary doses starting at 6 weeks of age
  ( 2 months in Saudi Arabia immunization schedule)
- 1st booster at 16-18 months of age, 2nd booster at 5 years of age
- At 10 years of age Tdap/Td followed by Td every 10 years
- Catch-up vaccination:
- Below 7 years: DTaP at 0,1 and 6 months