Lecture 1

Bacteriology

Systemic Bacteriology

Objective : Pyogenic cocci

Pyogenic cocci : Pyogenic means “pus forming”

Cocci means “spherical bacteria”

They include:

1. Staphylococcus (Gram positive)
2. Streptococcus (Gram positive)
3. Pneumococcus (Gram positive)
4. Neisseria (Gram Negative)

The pathogenic cocci are often called pyogenic cocci because of their ability to form pus (suppuration).

Genus Staphylococcus

This microorganism is widely distributed in our environment some of them are members of the normal flora of humans, the
normal flora include all microorganism which are normally found on the skin and mucous membrane of human beings. Others are important human pathogens like Staphylococcus aureus, Staphylococcus epidermidis, Staphylococcus saprophyticus.

Staphylococci are spherical in shape usually arranged in grape like clusters, grow rapidly on many types of ordinary bacterial media ferment CHO and produced pigments varying from white to deep yellow.

**Resistance to environmental condition:**

Staphylococcus aureus considered one of the hardest of all non spore forming bacteria most strain are relatively heat stable, with stand a temperature as high as 60 c for 30 min. also resist a high concentration of salt (7.5% - 9% Nacl).
Antigenic structure of *Staphylococcus aureus*:  
1. **Peptidoglycan**: a polysaccharide polymer provides rigid exoskeleton of the cell wall characterized by:
   * Can be destroyed by exposure to strong acids and lysozymes.
   * Stimulate the production of IL-1.
   * Stimulate the production of opsonic antibodies.
   * The peptidoglycan has endotoxin activity.

2. **Teichoic acids**:  
   They are polymers of glycerol or ribitol phosphate. These polymers are linked to the peptidoglycan and can be antigenic. Antiteichoic acid antibodies may be found in patients with active endocarditis caused by *Staphylococcus aureus*.

3. **Protein A**:  
   Is a cell wall component of *Staphylococcus aureus* strains that binds to the FC portion of IgG molecule.

4. **Extracellular substances**: (enzymes and toxin)  
   Most extracellular substances produced by *Staphylococcus aureus* are antigenic (stimulate the production of antibodies).

These are:-
1. Catalase:

Is an enzyme that breaks hydrogen peroxide into O₂ and H₂O so the benefit of catalase is that H₂O₂ is toxic to bacteria so they need catalase enzyme to get rid of H₂O₂.

It’s useful in differentiation between members of genus *Staphylococcus aureus* and genus *Streptococcus*.

2. Coagulase:

It’s an enzyme like protein; it clots citrated or oxalated human or rabbit plasma in the presence of serum factors. The serum factor reacts with coagulase generating clotting activity.

This enzyme converts fibrinogen (soluble) into fibrin (insoluble). Fibrin can be deposited on the surface of bacteria forming a wall around the bacteria which has an important role in:

A) Protection of bacteria from phagocytosis.
B) Preventing the action of antibiotics.
Coagulase occurs in two forms the first one is soluble coagulase elaborated or produced outside the bacteria cell. The second one is bound coagulase attached to bacteria cell (clumping factor).

3) Proteinase:

Is an enzyme which breaks down protein material.
4) Lipase:
   Is an enzyme which breaks down lipid in the skin, lipoprotein in blood.

5) Hyaluronidase:
   Enzyme breaks down hyaluronic acid (is important substance in binding tissue cells together therefore this enzyme helps the bacteria to spread in the tissues. some time it is called (spreading factor).

6) β - Lactamase:
   break down β -lactam ring of penicillin.

**TOXINS**

7) Hemolysins (exotoxins):
   Include several toxins act on cell membrane of RBCs of Varity of animal species.

8) Leukocidin :
   Toxic substance causes degradation of WBC leading to cell death.

9) Exfoliative toxins:
   Produced by certain strains of *Staphylococcus aureus*. It includes at least 2 protein that yield the generalized desequamation
of the skin in (scaled skin syndrome) this syndrome is common among small children.

Specific antibodies for the exfoliative toxin product against the exfoliative action of the skin (it’s antigenic, stimulate production of antitoxins.

10) Enterotoxins:
*they are 6 soluble toxins (A, B, C, D, E, and F).
* Heat stable.
* Resistant to the gut enzymes and acidity.
* are the proteins that cause nausea, vomiting and diarrhea.
* an important cause of food poisoning.
* produced by Staphylococcus aureus growing in meat and dairy products.

11) Toxic shock syndrome toxin 1: (TSST-1)
Associated with toxic shock syndrome TSS. Produced by some strains of Staphylococcus aureus in human this toxin cause TSS which is a systemic infection characterized by high fever, hypotension and shock with multisystem involvement.
Staphylococci are variably sensitive to many antimicrobial drug and resistance falls into several classes:

1) β- Lactamase production:

Is common and its under plasmid control makes the organism resistant to many penicillin like penicillin G, Ampicillin.

β -Lactamase enzyme break down β -Lactam ring of penicillin and render the antibiotic inactive the gene coding for production of this enzyme is located probably on plasmid (extrachromosomal genetic material).

This type of bacteria can be found in hospitals because above 90% of hospitals strains of *Staphylococcus aureus* are β-lactamase producer so antibiotics are useless when giving to the patients.

2) Resistance to naphcillin, methicillin and oxacillin:

Is independent of β -lactamase production the genes are probably located on chromosomes the mechanism for naphcillin resistance is based on the lack of penicillin binding proteins in the organism.

3) Tolerance:

Indicates that Staphylococci are inhibited by the drug but not killed (i.e. no growth, no multiplication and when removing the drug the bacteria will grow and multiply).
Tolerance is due to the lack of the activation of autolytic enzymes in the cell wall.

4) Plasmid can also carry genes for resistance to tetracycline, erythromycins and aminoglycosides.

**Pathogenecity of *Staphylococcus aureus***:

*Staphylococcus aureus* produces human infection by its capability to invade and spread into tissue and by production of certain enzymes and toxins.

Disease spectrum: from moderate to severe.

**Moderate infection:**

Minor skin infection (acne, furuncle, localized abscess, impetigo which is infection in the superficial layer of skin, common in small children).

**Sever infection:** (Bacteremia)

A. Osteomyelitis: Infection reaches the bone by hematogenous route.
B. Pneumonia: In post operative patients or fallowing viral infection.
C. Arthritis.
D. Deep organ abscess as brain or lung abscess.
Diseases associated with toxin production

* Staphylococcus food poisoning due to ingestion of enterotoxin.
* Scaled skin syndrome SSS (retter’s disease) associated with exfoliative toxin.
* Toxic shock syndrome toxin associated with TSST-1.

**Staphylococcus epidermidis**

All humans carry this bacteria in the deep layer of skin its usually associated with nosocomial infections and with foreign objects (i.e. catheters, shunts, pacemaker wires, heart valves replacement).

Virulence factors is production of an exopolysaccharide “slime”

The slime layer around the bacterial cell wall:

- promotes adherence to plastic surfaces
- increase resistance to phagocytosis
- inhibits entrance of antibiotics to the cell.

**Staphylococcus saprophytics:**

Normal flora when become pathogenic, it causes upper and lower urinary tract infection in young women.

**Phage typing**

Used to detect the source of infection when there is out break of food poisoning ex. In hospital.
Lecture 2

Bacteriology

Genus Streptococcus

Objective: morphology

Antigenic structure

Pathogenicity

Members of this genus are widely distributed in environment, some are non pathogenic and could be a part of normal flora present in pharynx, mouth, intestine and female genital tract. Others are pathogenic from simple to complex.

Membrane of this group are non motile, non spore forming, some are capsulated.

Culture characters:

Growth occur only in enriched media, majority are facultative i.e. can grow under aerobic or anaerobic conditions grow at 37c but is aided by 5-10% CO₂. Few are obligatory anaerobic e.g peptostreptococcus.

Nutritionally streptococci are fastidious m.o. and only grow on enriched media for example blood agar, chocolate agar and
can’t grow on simple media like nutrient agar, it arranged in chains or in pairs.
Classification of Streptococcus:
Specific criteria are used in classification of Streptococci
1. Type of growth on blood agar medium.
2. Serologic specificity.

Type of growth on blood agar
1. β-hemolytic Streptococci: clear zones of hemolysis around the colonies.
2. α – hemolytic Streptococci : partial or incomplete hemolysis around the colony ex. Viridans streptococci , Streptococcus pneumoniae.
3. γ type of hemolysis ( Non hemolytic) streptococci : no change on surface of blood agar.

Group β streptococci is the most important group of streptococci

Antigenic structure of hemolytic streptococci:-
1. Carbohydrate antigen ( C antigen):
   Its group specific cell wall antigen which is carbohydrate in nature and it’s located in the cell wall of many streptococci and form the basis of serologic grouping (Lancefield grouping). On
basic of this antigen β-hemolytic Streptococci are divided in to 19 groups from A-U except I and J
A B C D E F G H   K L M N O P Q R S T U (Lancefield groups)

**Group A streptococci** (**streptococcus pyogenes**)  
Is the most important human pathogen it can cause pharyngitis, tonsillitis.

This antigen can be extracted from bacterial cells by one of the three methods:
1. by treatment with hot Hcl.
2. by enzymatic lysis of cell (with trypsin or pepsin).
3. by autoclaving.

**Group B streptococci** (**Streptococcus agalaectiae**)  
Normal flora of female genital tract and can cause neonatal sepsis and meningitis.

**Group D streptococci include:** - Enterococci  
Non Enterococci

**Enterococcus** e.g **Enterococcus faecalis** it resist bile, 6.5% Nacl and penicillin G.

**Non Enterococcus** e.g **Streptococcus bovis** it is inhibited by 6.5% Nacl and killed by penicillin G.
2. **M-protein:**

It’s associated with virulence of group A streptococci. It’s type specific and on basis of this antigen, group A streptococci are subdivided into 60 types. It appears as hair like projection of the cell wall, it help the bacteria to resist phagocytosis.

Immunity to infection with group A streptococci is related to the presence of special Ab. But because there are approximately 80 type of M proteins, explain the repeatative infection with group A streptococci having different types of M proteins.

3. **T substance:**

This antigen has no relation to the virulence of streptococci. T-substance is acid labile and heat labile and it’s obtained from streptococci by proteolytic digestion. On basis of this antigen certain types of streptococci are differentiated by agglutination. It’s type specific, permits differentiation of certain types of streptococci. It’s used in epidemiological studies as “typing marker”.

4. **Nucleoprotein:**

Antigens associated with streptococcal cell body.
Toxins and enzymes of group A streptococci (Extracellular product):

- Hemolysins (streptolysins).
- Streptokinase (fibrinolysin).
- Streptodornase (DNase).
- Hyaluronidase (spreading factor).
- Erythrogenic toxin.

More than 20 extracellular products that are antigenic are elaborated by group A streptococci including:

Hemolysins: β- hemolytic streptococci elaborate 2 hemolysins:

1. Streptolysin O *sensitive to oxygen
   * antigenic ASO

   Streptolysin O is a protein that is hemolytically active in the reduced state but rapidly inactivated in the presence of oxygen its antigenic and combines quantitively with antistreptolysin O (ASO) this antibody blocks hemolysis by streptolysin O.

Streptolysin O +RBCs = hemolysis
Streptolysin O+ASO+ RBCs = no hemolysis

The ASO titer can be applied in the diagnosis of streptococcal infections.

Serum titer >200 I.U. is considered abnormal.
2. streptolysin S * Not sensitive to oxygen * Not an antigenic

It’s responsible for hemolysis around streptococcal colonies on blood agar surface.

**Disease caused by streptococcus pyogenes:**

Can be divided into two groups:
1. Suppurative infection (pus forming).
2. Non suppurative sequelae.

**Suppurative infection include:**

A) Strep. Pharyngitis most common in infants and small children, there is extension to the surrounding tissue causing otitis media, mastoiditis, meningitis. The tonsils are usually enlarged with enlarged tender cervical lymph nodes.

B) Strep. Infection can involve URT causing pneumonia which most commonly occur after viral infection.

C) Streptococcus pyoderma.

D) Erysipelas: which is spreading infection of the skin or mucous membrane usually seen on the face.

E) Surgical wound infection.

F) purpural sepsis: streptococcus enter the uterus after delivery causing purpural fever.

G) Scarlet fever.

H) Streptococcus toxic shock syndrome.
Non suppurative infection
Post Streptococcus disease
1. Acute rheumatic fever.
2. Acute glomerulonephritis.

Rheumatic fever

Certain strain of group A hemolytic streptococci with cell membrane antigen, this cross react with human heart muscles producing AB which react with human heart muscles and valves, it is a hypersensitivity response.

Acute glomerulonephritis

After skin infection or after pharyngitis by two to three weeks, certain GAHS type 2,4,12,49,59,61 known as nephritogenic strain causing Nephritis initiated by (antigen- antibody) complex on the glomerular basement membrane.
Bacteriology

α – hemolytic Streptococci

It includes:

1. *Viridans* streptococci
2. *Streptococcus pneumoniae*

*Viridans streptococci*

This microorganism has low virulence and often colonize in the URT. They considered as commensal m.o of the mouth and they can act as opportunistic pathogen and attack tissue. It is associated with dental caries and they are the leading cause of
subacute bacterial endocarditis (SBE) following dental extraction it include:

Streptococcus mitis
Streptococcus mutans
Streptococcus sangius
Streptococcus salivaris

These m.o. lack group specific carbohydrate in their cell wall.

Streptococcus pneumoniae

Also called Diplococcus pneumoniae or pneumococcus.
- Normal inhabitant of the upper respiratory tract (URT) of human i.e. 40%-70% of normal individuals are carriers of these bacteria.
- No animal reservoir, i.e. transmission is from infected to normal persons by direct route.
- Sometimes may cause important human diseases such as pneumonia, bronchitis, sinusitis, otitis media and less frequently it invades blood stream producing bacteremia, and the most important complication of bacteremia includes: meningitis and septic arthritis.

Characteristics
- morphological
  - Gram positive young culture

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- Lancet shaped diplococci, the adjacent sides are blunt, while the distal sides (ends) are pointed.
- Capsulated possessing a polysaccharide capsule.
- In sputum or pus, the bacteria are found singly sometimes, or forming short chains not exceeding 3 pairs in each chain.
- Gram negative in old culture and later on spontaneously will lyse due to autolytic activity and finally we can find gram negative debris, no intact cells are found in the culture.
Such lysis can be enhanced by surface active agent (bile salts). This phenomenon can be used to differentiate it from other bacteria by adding 10% bile salts to young culture, well notice that the culture will become clear and the turbidity is gone, this is called bile solubility test.

**Cultural:**
- Facultative anaerobic.
- Don’t grow on simple laboratory media (fastidious) i.e. requires enriched media.
- On blood agar they produce small colonies surrounding by α type of hemolysis.
- After few hours of incubation colonies from a dome on the culture media converted upon continuous incubation into central plateau and raised rims (botton shaped colonies) (draughman’s colony).
Differentiation between *Diplococcus pneumoniae* and *α*-haemolytic streptococci

<table>
<thead>
<tr>
<th>Test</th>
<th><em>Diplococcus pneumoniae</em></th>
<th><em>α</em>-haemolytic streptococci</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bile solubility test</td>
<td>Soluble</td>
<td>Insoluble</td>
</tr>
<tr>
<td>Optochin sensitivity test</td>
<td>Sensitive</td>
<td>Resistant</td>
</tr>
<tr>
<td>Inulin fermentation test</td>
<td>Positive</td>
<td>Negative</td>
</tr>
<tr>
<td>Capsule swelling (Quellung reaction)</td>
<td>Positive</td>
<td>Negative</td>
</tr>
<tr>
<td>Animal pathogenicity</td>
<td>Virulent for white mouse</td>
<td>Avirulent</td>
</tr>
</tbody>
</table>

**Note:**

Optochin is ethylhydrocupreine hydrochloride.

**Quellung reaction test:** Pneumococcus of certain type when mixed with anti-capsular antibodies on microscopic slide, the capsule swells markedly and can be visualized by examination of the slide under low power.
Antigenic structure of *Diplococcus pneumoniae*

1. **Capsular polysaccharide (specific soluble substance SSS)**
   - large polysaccharide polymers (pneumococcal capsule).
   - hydrophilic gel on the surface of the organism.
   - Antigenic (once injected into human body it produces antibodies) and type-specific.
   - Pneumococci are differentiated into 80 serological types on basis of capsular polysaccharide.

2. **Somatic portion of pneumococci:**
   (Somatic means every thing except capsule)
   - Contains M- protein antigen that is characteristic for each type.
   - M-protein antigen of pneumococci play no role in their virulence.
   - Also contains Group specific carbohydrates which:
     - are common in old culture of pneumococcus.
     - Can be precipitated by C- reactive protein which is an abnormal protein can be detected in the sera of patients with certain febrile illness.
**Pathogenicity:**

- In adults:
  - Types 1-8 are the most important because 75% of pneumococcal pneumoniae are caused by these types.
  - 50% of all pneumococcal bacteremia are fatal.

- In children:
  Types: 6, 12, 19, 23 are the most important because they are responsible for all pneumococcal infection in children.

**Virulence Markers** of *Streptococcus pneumoniae* associated with:

- Polysaccharide capsule which may prevent or delay phagocytosis. Non capsulated pneumococci are avirulent (non-pathogenic).
- Production of IgA protease: this enzyme inactivate IgA which is the main secretary Ig in URT.

This enzyme aids the bacteria to colonize in the URT.

- We inhale these bacteria continuously but they are phagocytized this indicates that reparatory mucosa posses a great natural resistant to streptococci. In some individuals such bacteria colonize in the mucosa of URT and these
become carriers. If resistance becomes low, the bacteria invade the lung producing pneumonia.

**Predisposing Factors:**

1. Viral infection.
2. Abnormality of respiratory tract.
3. Trauma of pharynx.
4. Allergy.
5. Cough reflex.
6. Some change in movement of cilia.
7. Deep anaesthesia.
8. Long bed rest.

From lung they may invade the blood stream producing bacteremia.
Lecture 4

**Genus Neisseriae**

The Neisseria species are gram negative cocci that usually occur in pairs, *Neisseria gonorrhea* “gonococci” and *Neisseria meningitidis* “meningococci” are pathogenic for human and typically are found associated with or inside polymorphonuclear leukocytes (PNL).

Some Neisseriae are normally inhabitant of human respiratory tract and occur extracellularly. Gonococci and meningococci are closely related with 70% DNA homology and are differentiated by few LAB tests and specific characteristics:

**Meningococci**: 1) have polysaccharide capsules
   2) Rarely have plasmids
   3) They cause a disease called meningitis.
   4) Ferment glucose and maltose

**Gonococci**: 1) do not have capsules
   2) They have plasmids
   3) They cause genital infection
   4) Ferment glucose only
Neisseria meningitidis within PNC in spinal fluid

The Neisseriae are:

1) Human pathogens
- Neisseria gonorrhoea (gonococcus) cause genital infection.
- Neisseria meningitidis (meningococcus) cause acute meningitis. or subacute septicemia with petechial rash.
2) Non pathogenic:
- Neisseria sicca
- Neisseria subflava
- Neisseria lactamica
- Neisseria mucosa

These are normal inhabitants of human respiratory tract.

**Morphology and Identification**

A) Typical organisms: they are Gram negative, non motile, non spore forming diplococcus, 0.8 μm in diameter, and kidney in shape, arranged in pairs. Pathological spp. are intracellular while non pathogenic are extracellular.

B) Culture: in 48 hours on enriched medium gonococci and meningococci form convex elevated mucoid colonies 1-5 mm in diameter colony are opaque, non pigmented and non hemolytic. The non pathogenic spp. can grow in simple media while the pathogenic spp. need enriched media e.g (blood and chocolate agar) selective media for Neisseria called (Modified Thyer martin agar).

C) Growth characteristics: Neisseria grow best under aerobic conditions, ferments carbohydrates producing acid but not gas, they give +ve oxidase test. The microorganism are rapidly killed by drying, sunlight and many disinfectants.
Biochemical test :-

1) Oxidase test

2) Sugar fermentation test

These tests can differentiate the pathogenic Neisseria from the non pathogenic and can also differentiate spp. from each other.

*Neisseria gonorrhoeae* produces acid from oxidation of glucose but not from maltose, sucrose, or lactose. *Neisseria* species produce acid end products from an oxidative pathway rather than from fermentation. The acid turns the pH indicator phenol red from red to yellow. Lactose, not shown here, is not utilized by *N. gonorrhoeae*. 
1) Neisseria gonorrhoea (gonococcus)
One of the pathogenic spp. of Neisseria it’s responsible for a human disease (Gonorroea) which is a sexually transmitted disease (STD) the clinical infection cause by this M.O is divided into 2 types:
1) Localized infection.
2) Systemic infection.

Localized infection:

The microorganism cause localized infection in the genital organs (on the mucous membrane) and to a lesser extent the mucous membrane of the rectum, eye, throat. So when the m.o invade the tissue it will cause suppurative inflammation in the area characterized by pus cell formation it can be followed by chronic infection which is associated with fibrosis. Localized infection can be divided into:-

A) Genital infection: which differ in male than in female.
In male it causes urthritis associated with mucopurulent discharge from anterior urethra and dysurea the infection can extend to the epitedimis leading to epidemitis and if not treated it will be complicated by fibrosis and urethral stricture.
In female the first site of infection is the cervix causing cervicitis the infection is going to spread from cervix to the vagina causing vaginititis and presented by vaginal discharge which is
mucopurulent in nature, if not treated it will spread upward to involve fallopian tube resulting in salpingitis which lead to fibrosis with tube stricture leading to infertility due to tubal damage.

B) Rectal involvement: This is also a localized infection caused by gonococcus called proctitis and the patient presenting symptoms are purulent discharge from anus with tensmus.

C) Eye infection: occur in new born babies during their passage through infected birth canal causing a condition called (ophthalmia – neonatorum) which is a very serious condition and if untreated it will lead to blindness.

Prevention of this condition is by using antimicrobial agent as silver nitrate and erythromycin.

D) Throat infection occur in abnormal person’s suffering from Gonococal pharyngitis it’s common in homosexual person.

2) Systemic infection:

The m.o. spread from the site of infection via the blood stream to the distant organs it will mainly involve the skin, bone, joints causing arthritis or the brain in form of meningitis some time it cause endocardaitis.
**Diagnostic LAB tests:**

a) Specimens:
   
   Pus and secretion are taken from the urethra, cervix, rectum, conjunctiva, throat or synovial fluid for culture and smear.

b) Smears:
   
   Gram stained smears of urethral or endocervical exudate reveal many diplococci within pus cells.

c) Culture:
   
   Immediately after collection pus or mucous is streaked on enriched selective medium.

d) Serology:
   
   Serum and genital fluid contain IgG and IgA antibodies against gonococcal infection, but it is not of great value in the diagnosis because of the antigen diversity of Gonococci and there is delay in the development of these antibodies so it is more important to detect Gonococcal antigens using a technique called ELISA i.e that is Enzyme linked immunosorbent assay or using radioimmuno assay (RIA).

e) Detection of Gonococcal nucleic acid using DNA probe which can detect nucleic acid of the m.o.
Antigenic structure

Gonococci are antigenically heterogeneous both in vivo and in vitro. These antigenic changes help the m.o. to escape from the immune system causing damage to the body tissues these include:

A- Pilli:

Are hair like structures that extend up to several microns from the gonococcal surface. The function of the pilli is by which the m.o. will attached to the host cell m.m. these pilli are antiphagocytic and it will stimulate Abs formation.

B- Por (protein I):

Extends through the gonococcal cell membrane each strain of gonococcus expresses only one type of por but the por of different strains is antigenically different.

C- Opa (protein II)

This protein function is adhesion of gonococci to host cell.

D- RMP (protein III)

It is associated with Opa protein in the formation of pores.

E- Lipo oligosaccharide:

It has Endotoxic effect and it is responsible for toxicity of gonococci.

F- IgA protease:
An enzyme protein produced by gonococci which split IgA making it in non functioning form, so IgA protease is a virulent factor that enhances colonization of bacteria.

Treatment

Uncomplicated urethral or rectal infection can be treated with cefotaxime I.M. as single dose, the other antibiotic is penicillin but 50% of Gonococci are penicillin resistant doxycyclin is recommended but not suitable for pregnant women so erythromycin is used.

2) Neisseria meningitidis (meningococcus)

It is the pathogenic spp. of Neisseria causing C.N.S. in form of meningitis and the m.o. can normally carried in the upper respiratory tract (carrier ) which are the most important clinical source of infection.

Antigenic structure:

1) At least 13 serogroups of meningiococci have been identified by immunologic specificity and capsular polysaccharides the most important serogroups associated with disease in human are A,B,C,Y, W135 ,they are associated with fulminant sepsis with or without meningitis which is used for the preparation of vaccines.
2) Outer membrane protein
3) pilli
Pathogenicity

The source of the infection is either the patient or the carriers which have asymptomatic infection.

The route of entry is through the nasopharynx by droplet infection then the m.o. will attached to the epithelial cell by pilli. From the nasopharynx the meningococci will spread through the blood stream to the target organ causing bacteremia. So the sequelae is either meningitis or suffer from fulminating meningococcemia.

Meningitis is the most common complication it is usually begins suddenly with headache, vomiting and stiff neck, this will progress to coma within few hours.

Fulminating meningococcemia is more sever with high fever and hemorrhagic rash; there may be disseminated intravascular coagulation and circulatory collapse (water- house- friderichsen syndrome).
**Diagnosis by LAB. Tests:**

a) Specimens:

Specimens of blood are taken for culture and specimens of CSF are taken for smear, culture and chemical determination and nasopharyngeal swab are taken for carrier survey’s.

b) Smears:

Gram stain smear of CSF show typical Neisseria within polymorphnuclear leukocytes.

c) Culture:

CSF specimens are placed on heated blood agar (chocolate agar and Thyer martin media) and incubated at 37c.

d) Serology:

Antibodies to meningococcal polysaccharides can be measured by latex agglutination or by immuno electrophoresis.

e) PCR

**Treatment:**

Pencillin G is the drug of choice for treating meningococcal disease. In patient allergic to penicillin chloramphenicol and ceforamime (or ceftriaxone) can be used.
**Prevention:**

1) Irradication of the carrier states (major source).

2) Isolation of the patient.

3) Chemoprophylaxis for contact people.

4) Vaccination.

**Moraxella catarrhalis:**

Previously they are called Neisseria catarrhalis, the name changed to Branhamella and now they are separated genus “Moraxella”.

They are normally found in upper respiratory tract especially among school children. (50% of school children carry this microorganism) it has been reported that this species may cause pneumonia otitis media, sinusitis and other infections.

It is oxidase +ve.