Briefly discuss about the Streptococcus pyogenes and its diagnosis?

Classification

1. Gram positive bacteria arranged in chains of varying length
2. each cell is approximately 1 micrometre in diameter
3. non-motile
4. non-sporing
5. may be capsulate
6. most are facultatively anaerobes, but there are species that are strictly aerobic
7. Catalase negative
8. Classification depends on the type of haemolysis seen on the blood agar.
   Strains which have soluble haemolysins (streptolysins O and S) will produce a clear zone of haemolysis on fresh blood agar media. This is known as beta-haemolysis and the organism is classified as beta-haemolysis streptococci. Those that cause a narrow zone of partial clearing and green coloration are called alpha-haemolysis streptococci. Those that produce no obvious changes around the colonies on blood agar are called non-haemolytic or gamma-haemolytic

**Streptococcus pyogenes**

a) Pathogenesis

The most common route of entry of *Streptococcus pyogenes* is the upper respiratory tract, where the primary infection is established, usually in the throat

i) Structural Components

The virulence of *Strep. pyogenes* is closely related to the surface antigen, the M protein. The fimbriae on the surface of streptococci enable attachment to epithelial cells, contain M protein. Two other surface proteins, T and R protein, do not play any part in virulence but are useful in identification of infecting serotypes

ii) Extracellular products

These includes :-
- Erythrogenic agents - erythrogenic toxins A, B and C are superantigens, T cell mitogens that induce lymphocytes to synthesize and release cytokines such as TNF, interleukin-1beta, interleukin-6, with the subsequent production of fever, shock and tissue damage.

- Streptolysin O - a kind of haemolysin which lyse red blood cells by linking to bound cholesterol in the cell membrane, causing holes to form in this structure. It is also cytotoxic for other cells, including neutrophils, platelets and cardiac tissue. It is inactivated by oxygen. *Streptococcus pyogenes* produce this kind of haemolysin.

- Streptolysin S - this haemolysin is not inactivated by oxygen and is responsible for the haemolysis produced on the surface of an aerobic blood agar plate.

- Streptokinase (fibrinolysin) - is protein in nature and is antigenic. Role is not clear but because of its action in preventing the formation of an effective fibrin barrier, it may well influence the character of the lesions by interfering with the localization of the infection.

- Deoxyribonuclease (DNAases) - at least four of these enzymes, designated types A, B (*Streptococcus pyogenes*), C and D and are all antigenic. They hydrolyse nucleic acids and nucleoproteins.

- Hyaluronidase

- Nicotinamide adenine dinucleotidase (NADase) - antigenic

- Serum opacity factor (lipoproteinase)

b) Laboratory Diagnosis

1. Examination of patient's sera for a rising titre of antibodies to one or more streptococcal antigens are the means of diagnosis.
2. Swabs are taken from patients or from suspected carriers as.
3. Microscopy is of little help in the diagnosis.
4. Using bacitracin sensitivity test as beta-haemolytic streplococci, being group A organism, are more sensitive than other groups to bacitracin but the test is not totally reliable as some non-group A strains may also be sensitive.
5. Antistreptolysin O (ASO) test is most common performed, and the anti-DNAase B test may also be useful.

c) Epidemiology

Besides the acute case of sore throat and the nasal or saliva carrier, other dangerous sources of infection are patients with streptococcal otitis media, vulvovaginitis or infected skin lesions.

d) Chemotherapy
Str. pyogenes is highly sensitive to a wide range of antibacterial drugs, including penicillin and erythromycin but strains resistant to sulphonamides and tetracyclines are common. Benzylpenicillin (penicillin G) or oral phenoxymethylpenicillin (penicillin V) are the drugs of choice. Treatment for 3 - 5 days will limit the effect of severe attacks of streptococcal infection and prevent suppurative complications such as otitis media, but studies have been shown that only if treatment is continued for 10 days will the streptococci be eliminated from the infected area. If the use of any other antimicrobial agent is contemplated, as in cases of hypersensitivity to penicillin, antimicrobial sensitivity testing must be done because of dangers of resistance to the drug.

e) Clinical Infection

The most common and typical infection caused by Str. pyogenes is an acute sore throat. If the infecting streptococcus is capable of producing an erythrogenic toxin and the host has not developed antibodies to this toxin, the sore throat may be accompanied by a generalized punctate erythema or rash. This syndrome is called scarlet fever. Local extension of the infection from the throat may result rarely in such complications as peritonsillar abscess (quinsy), sinusitis, otitis media or mastoiditis. Puerperal sepsis or child-bed fever is traditionally associated with infection by Str. pyogenes. Besides local inflammation of uterine tissues, infection may spread to the adnexa (pelvic cellulitis or peritonitis). Wounds, burns and chronic skin lesions (eczema, psoriasis) may become infected with Str. pyogenes; these superficial infections may extend in the local tissues (cellulitis) or be carried by lymphatics to regional lymph glands (lymphadenitis) or get into the bloodstream and become generalized (septicaemia).

f) Non-Suppurative Complications

i) Rheumatic Fever

- An attack of rheumatic fever is related to antecedent streptococcal throat infection occurring 1 - 5 weeks earlier. Diagnosis may be difficult since throat swabs may or may not yield growth of Str. pyogenes, thus, the most important investigations are serological. Two samples of serum must be assayed to check for a rising titre of antibody to Str. pyogenes. Titres become detectable in the 2nd week after the onset if infection and are maximal by the 6th week and thereafter decrease. Penicillin prophylaxis after a primary attack of rheumatic fever will reduce substantially the risk of a second attack by preventing further streptococcal infection of the throat. However, prevention of primary attacks is
virtually impossible and penicillin treatment of streptococcal sore throats plays little or no part

ii) Acute Glomerulonephritis

- almost always produced by Group A streptococci but group C streptococci may also be involved
- acute glomerulonephritis is produced by a much narrower range as compared to rheumatic fever which can be caused by a wide range of serotypes of *Str.pyogenes*
- Acute Glomerulonephritis following streptococcal throat infection occurs at the colder times of the year and mostly in children and younger adults
- flies of the genus *Hippelates* is of important vectors of streptococci in hot humid weather
- post-streptococcal glomerulonephritis arises because components of the glomerular basement membrane are immunologically similar to the cell membranes of nephritogenic beta-haemolytic streptococcus