***Title of Lecture:***

***streptococci, enterococci, and gram +ve bacilli bacteria***

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***Refer to slide no. : 7,8***

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- Infection in infants(by streptococcus agalatiae) younger than 1week of age is called early onset disease, while that occuring 1week-3months after birth is called late onset disease.

**Early onset disease:**

- Bacteremia, pneumonia or meningitis

-15-30% of survivors have neurological sequelae.

**Late onset disease :**

- Exogenous source

- Bacteremia with meningitis

- **Other beta hemolytic streptococci**

- groups C, F and G

- group D was initially classified as a streptococcus ,but during DNA studies scientists found that its different that the other streptococci groups, therefore group D was assigned a seperate group of bacteria called Enterococcus.

so, Entercoccus was previously known as group D streptococcus.

**Viridian Sterptococci:**

- A heterogeneous collection of alpha – hemolytic and non hemolytic streptococci (viridians = produce green discoloration around the colonies)

- Confusing taxonomy because of a lack of consensus

- Fastidious growth requirements (blood +5-10% CO2), the presence of CO2 promotes the growth of the bacteria .

- Inhabit oropharynx, gastrointestinal and uroginital tracts.

- Most frequently associated with dental caries, subacute endocarditis and suppurative intraabdominal infection

**- viridian sterptococcus is very important as part of our dental career , for its associated with dental caries ,first of all it changes glucose to dextrin which sticks to the tooth enamel ,produces acid as by-product ,acids dissolve the tooth enamel ,causing dental caries .**

***- Streptococcus mutans* and *Streptococcus sanguis* are most important.**

**- Most common cause of subacute endocarditis.**

**-subacute bacterial endocarditis ,this takes place when u do procedures in the oral cavity, for example : tooth brushing and excessive chewing, bacteria from the oral cavity enters the blood stream ,settles on the diseased heart valves and stick to them causing subacute bacterial endocarditis .**

**- in normal individual tooth brushing or excessive chewing and the entry of the viridian streptococcus to the blood stream will not progress to subacute bacterial endocarditis ,because once the bacteria enters into the blood stream it will be cleared by the immune system, which is not the case in individuals with diseased heart valves.**

**Streptococcus pneumonia:**

**- we have another genus of streptococcus which is the streptococcus pneumonia, previously known as pneumococcal bacteria.**

**- it is alpha hemolytic, optochin sensitive, bile soluble and quelling reaction positive.( quelling rxn is associated with capsulated strains of bacteria,where antibodies react with the capsule of the baceria (antigenic) producing such rxn)**

**- in streptococcus pyogenes the capsule is not antigenic ,for its made up of hyaluronic acid.**

**- Encapsulated (84 types), lancet shaped, and arranged in pairs or short chains.**

**- its usually arranged in pairs (diplococcus),or short chains (not commonly found as long strips or chains)**

**-optochin sensitive =streptococcus pneumonia**

**-bacitracin sensitive= streptococcus pyogenes .**

**-bacitracin sensitivity is associated with streptococcus pyogenes , and optochin sensitivity is associated with streptococcus pneumonia .**

**Exposed teichoic acid is called C substance and it can precipitate a serum globulin fraction (CRP) in the presence of calcium.**

**-C-reactive proteins (act as Abs ), they were first discovered in conjunction with the streptococcus pneumonia, they are lgs that actually attack the capsule of the streptococcus pneumonia through the c-substance of the teichoic acid, they can attack other bacteria as well, since these Abs were first discovered through their rxn with the c-substance of the streptococcus pneumonia they were given the name (C-reactive poteins).**

**-C-reactive protein =it's a protein that reacts with the c-component of the polysaccharide (capsule) of the streptococcus pnemonia and components of other bacteria as well .**

**- Lower serotypes (1-8) are responsible for about 75% of cases(responsible for most of the infections).**

**Pathogenesis :**

- Involves the capsule, adhesion factor, IgA protease, pneumolysin, teichoic acid and peptidoglycan.

-capsule is a pathogenic factor, lgA protease breaks down lgA present in mucus and other secretions causing infections,teichioc acid for adhesion.

- Pneumococcal disease originates from spread of organisms colonizing the nasopharynx and oropharynx to distal loci.

- streptococcus pneumonia is usually found in the upper respiratory tract (nasopharynx and the oropharynx), bacteria can spread downward into the lungs causing what so- called Lobar pneumonia , pneumonia involving one of the lung lobes .( this can be seen in elderly people and in children as well )

- streptococcus pneumonia can cause bacteremia ,which might end up with meningitis, sometimes infection goes up to the Eustachian tube ( in the middle ear) leading to middle ear infection or Otitis media.

- alcoholism is an important risk factor of developing pneumonia.

-there is a vaccine against streptococcus pneumonia, which is derived from the capsular antigen ,its given by injection, induces Abs production , immunity against pneumococcal infection.

-elderly people and children should take this vaccine.( it's part of the childrens' program of vaccination )

-vaccination prevents the occurrence of pneumonia as well as meningitis .

**immunity can occur in different ways :**

1- natural immunity: once u get the disease its unlikely to have it again , since ur immune system will destroy the microrganism before it spreads and cause infection.(natural immunization)

2-vaccination by either:

a- killing the bacteria (microorganism) and injecting it into the human's body , in this case it wont cause the disease ,for the microorganism is dead,but its still antigenic (its dead but the antigens are still there), induces Ab production (immunization) .

b-attenuation ( attenuation occurs by 1-treating the toxins with formalin forming toxoids which are not toxic but antigenic or by 2- growing the bacteria under abnormal conditions , in this case bacteria becomes nonpathogenic ,nontoxic, but still antigenic,can induce our immune system )

c- we can use other bacteria having the same antigens ,but less or nonpathogenic than the original one ,so it wont cause the disease but will induce the production of Abs (immunity) against the bacterial antigens.

**Enterococcus:**

- its classified into a separate genus (previously group D streptococci) .

- they are highly resistant bacteria , they grow in 6.5% NaCl (they're resistant to high conc. of NaCl ), resistant to bile thats why they're found mainly in the GI tract as part of the normal flora.

- they're associated with many urinary tract infections , subacute bacterial endocarditis, septicemia, and meningitis mainly in neonates.

-The genus consists of 12 species, 2 of which are responsible for more than 95% of infections; *Enterococcus fecalis* (85-90%), related to feces as the name implies, and *Enterococcus facium* (5-10%).

- they are difficult to treat since they are resistant to many antibiotics.

-They have intrinsic low level resistance to aminoglycosides and intermediate resistance to fluoroquinolones .

***Aerobic Gram Positive Bacilli :***

-The genus Bacillus includes gram positive, aerobic, spore-forming, rod shaped organisms.

- 1 micron in diameter,3-5 micrometer in length, they are arranged singly or in chains( when grown in culture media they usually assume a chain-like structure).

- widely spread in nature, found in water,soil,vegetation, few are present in the GI tract of humans and animals.

**Bacillus anthracis**

- Pathogenesis depends on two plasmid encoded virulence factors:

1- capsule

2-toxins,it produces three types of toxins: a-protective antigen, b- lethal factor, c-edema factor.

usually two of these toxins are enough to cause tissue damage and destruction.

- bacterial capsule prevents phagocytosis.

- tissue damage is caused by the toxin production.

- its believed that if we have one protective antigen with one of the toxin factors (either lethal or edema factor), we will get tissue damage and destruction.

- Anthrax is a disease of herbivores and humans are accidentally infected by exposure to infected animals or animal products.

- there are 3 forms of the disease : a- cutaneous anthrax (most common) ,b-pulmonary anthrax ,c- GI anthrax (rare)

**Cutaneous Anthrax:**

-It usually occurs through contamination of a cut wound or abrasion although in some countries biting flies may also transmit the disease.

- bacteria enters the skin through an abrasion or cut wound , produces toxins ,toxins kill the underlying tissues , scar is formed and ulcer, the area turns black since the tissue down there is necrotic (dead damaged tissue).

- After 2 to 3 days of incubation a small pimple or papule appears at the site of entry. A surrounding ring of vesicles develops, the central papule ulcerates, dries and blackens to form the characteristic eschar.

- sometimes we might have septicemia.

- Lesions on the face or neck are dangerous and fulminating septicemia may develop in 20% of cases.

**Pulmonary anthrax:**

- Inhaled spores are transported by alveolar macrophages to the mediastinal lymph nodes where they germinate and multiply to initiate systemic disease.

- if someone for example inhaled the spores, they will go to the lymph nodes around the lungs and will produce there toxins up there , which will cause septicemia, multiorgan failure and the person might die.

- sometimes called **wool sorter disease**.

**-It is highly fatal (> 95%) because it is not suspected until the course is irreversible.**

**GI anthrax:**

- Very rare with varied clinical presentation (mesenteric adenopathy, hemorrhage and ascites) and high mortality rate (95%).

**- mortility is more than 90% in GI and pulmonary antharx , while in cutaneous anthrax its 15-20% .**

**- cutaneous anthrax is the most common , GI is the least .**

- Clinical diagnosis of anthrax is confirmed by directly visualizing or culturing the anthrax bacilli.

*- Bacillus anthracis* is susceptible to penicillin and almost all other broad spectrum antibodies.( the treatment of choice is the penicillin)

**Bacillus cereus:**

- Large, motile, spore forming bacteria , produces two types of toxins:

1-heat acid stable toxin ( causes emetic syndrome)

2- heat labile enterotoxin (causes diarrhoeal disease)

**1-** **Emetic type** (heat stable toxin)

- Nausea and vomiting begin 1 to 5 hours after consumption of contaminated food.

- Boiled rice that is held for prolonged periods at ambient temperature and then quickly fried before serving is the usual offender.

**2- Diarrheal type** (heat labile toxin).

- Characterized by diarrhea and abdominal pain occurring

8-16 hours after consumption of contaminated food.

- It is associated with a variety of foods including meat and vegetable dishes, sauces, pastas, deserts and dairy products

*- B. cereus* cause panophthalmitis by an incompletely defined mechanism

- It is a post traumatic disease which is rapidly progressive that almost universally ends in complete loss of light perception within 48 hours.