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Medical Bacteriology – Lecture 15

Gram Negative Rods

Pseudomonadaceae

Pseudomonas

Burkholderia

Vibrionaceae

Vibrio

Pseudomonas

- Gram-negative rods
- Motile
- Aerobic (respiratory metabolism, never fermentative)
- Versatile
- Catalase + Oxidase +
- Found in water, soil, sewage, vegetable, human and animal intestine and skin.

Medical importance species: P. aeruginosa

P. aeruginosa

- Part of normal flora in human and animal intestine, water, soil and moist environment in hospitals.
- Nosocomial pathogen- Opportunistic pathogens
- very simple growth requirement. It is often observed "growing in distilled water", evidence of minimal nutritional needs).
- Its optimum temperature for growth is 37 C, and it is able to grow at temperatures as high as 42C.
- Invasive and toxigenic infections in patients with abnormal host defenses
- Produce soluble **fluorescent pyocyanin pigment**, refers to **''blue pus''**, which is a characteristic of suppurative infections caused by *P. aeruginosa*.
- Causes UTI (introduced by catheter)- Wound infection of burn sites, Septicemia, Otitis externa (Malignant external ear infection in poorly treated diabetic patients) – Pneumonia- Eye infection (injury or surgery)- Endocarditis- bacteremiameningitis- brain abscesses.
- Identification is based on colony morphology, oxidase-positive, growth at 42C-Non lactose fermintor - Bluish-green pigmented large colonies with "fruity" odor on culture media.
- It is resistant to high concentrations of salts and dyes, weak antiseptics, and many antibiotics

P. aeruginosa Virulence Factors

1- Pilli (Adhere to epithelial cells)

2- Exopolysaccharide capsule (protected from opsonization by antibodies, complement & phagocytosis)

3- Biofilm (Biofilm mucoid strains of *Pseudomonas* are also less susceptible to antibiotics).

4- Motility: spread through tissues

5- Fluorescent pigment pyocyanin (impairs normal function of human nasal cilia, disrupts respiratory epithelium).

6- Enzymes: Two extracellular proteases associated with invasive stage: elastase & alkaline protease

• Elastases: Digests protein (elastin, collagen, IgG, lyse fibronectin for bacterial attachment on the mucosa of the lung, disrupts the respiratory epithelium and interferes with cilia function)





• Alkaline protease lyses fibrin.

7- Toxins:

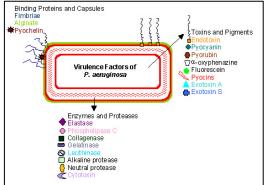
• Haemolysins (beta haemolysis)

• Phospholipase & lecithinase (destroy lipid and lecithin)

• Exdotoxin A (Cytotoxic by blocking protein synthesis- pore forming protein), same mechanism of action as the **diphtheria toxin**

- Toxin S: interfere with membrane permeability
- •Lipd A (Endotoxin): tissue necrosis
- Enterotoxins: food poising
- 8- Iron acquiring systems

9- Antibiotics resistance due to outer membrane changes



P. alkaligenes

Very rarely human pathogen- Nosocomial pathogen, wounds, urinary tract infection

P. fluorescens

unusual cause of disease in humans (usually affects patients with compromised immune system)

Produce yellow pyoveriden pigment, which is soluble in water but not chloroform

Burkholderia

•Burkholderia mallei (pseudomonas mallei) Human & animal pathogen causing Glanders

•Burkholderia pseudomallei Human & animal pathogen causing Melioidosis

•Burkholderia cepacia (Onion bulb rot, Foot rot of man)

- Septicemia, urinary tract infection, wounds, endocarditis pneumonia in immunocompromised individuals.

Curved Bacilli (Vibrio)

- Actively motile
- Vibrios are distinguished from **enterics** by being **oxidase-positive** and motile by means of polar flagella.
- Vibrios are distinguished from **pseudomonads** by being **fermentative and oxidative** in their metabolism
- Vibrios are one of the most common organisms in surface waters of the world.



•Medical importance species:

V. cholerae and V. parahaemolyticus

- Both are pathogens of humans
- •Both produce diarrhea, but in different ways.
- V. parahaemolyticus is an invasive the colon

• *V. cholerae* is **noninvasive** (**not reach the blood**), only act locally, affecting the **small intestine** through secretion of an enterotoxin.

•Vibrio parahaemolyticus Two biotypes (Parahaemolyticus and Alginolyticus)

•Parahaemolyticus causes food poising in Japan

•Gastroenteritis (enterotoxin): Explosive or mild diarrhea

Aeromonas hydrophila

Septicemia (Produce phospholipase + haemolysin)

Pleisiomonas shigelloides

Septicemia, wound infections, and gastroenteritis Haemolysin (kanagawa test)

Vibrio cholera

- Found in fresh water- sea food. most often in communities with poor sewage and water treatment.
- Grow in asparagine (as a sole source of carbon and nitrogen)
- Optimum pH growth range (8.5-9.5)- Sensitive to acidic pH
- Causes Cholera (epidemic cholera) is a severe diarrheal disease
- Transmission to humans is by contaminated water or food- Route of infection is (fecal-oral)
- Incubation period 1-4 days
- Large inoculum (10^8) is required to cause disease because the bacteria are susceptible to acidic stomach environment

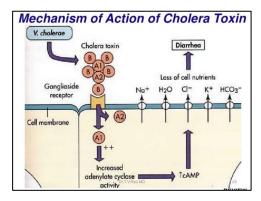
Cholera is one of the most rapidly fatal illnesses. Human may become hypotensive within an hour of the onset of symptoms and may die within 2-3h if no treatment. The disease progresses from the first liquid stool to shock in 4-12 hours, with death following in 18 h to several days.

Abrupt watery diarrhea and vomiting (Rice– water stool is characteristic) contains enormous numbers of vibrios, mucosa, epithelial cells– result in severe fluids and electrolytes loss- dehydration, can lead to coma and death- The loss of potassium ions may result in cardiac complications and circulatory failure. Untreated cholera results in high (50-60%) mortality.

Cholera toxin (heat labile enterotoxin) is the most important virulence factor of V. *cholera*, action on the mucosal epithelium- it is responsible for the characteristic diarrhea of cholera disease.

Mode of action: Cholera toxin activates the adenylate cyclase enzyme of the intestinal mucosa leading to increase secretion of H_2O , Na+, K+, Cl-, and HCO₃- into the lumen of the small intestine.

The bacterium produces an invasion, neuraminidase, during the colonization stage which is degrade N-acetyl- neuromeric acid.



Cultures:

1- TCBS (thiosulphate citrate bile salt sucrose agar) Selective media for primary isolation of *V. cholerae* (Observe large yellow sucrose-fermenting colonies after 18-24 hrs of incubation)

2- Alkaline peptone water: Enrichment media for V. cholerae

Treatment:

- **Give solution containing:** (Glucose + Bicarbonate + Potassium) for fluids and electrolytes replacement
- Tetracycline shorten the duration of diarrhea and reduce fluid loss.

Disease progression

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Ingestion- Pass HCl Barrier of the stomach

(100,000,000 acidic, 10,000 neutral)

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Multiply in the small intestine (predisposes malnutrition, vitamin B drop)

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1 -Motility (for contact)

2 -Envelope (for adhesion)

3-Mucinase (break the mucosal layer to allow attachment to epithelial cells)

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4- Endotoxin

5-Enterotoxin

6-Neuraminidase to break N-acetyl- neuraminic acid

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diarrhea (plasma → lumen)

Loss of fluids (decrease of blood volume + pressure)

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Shock

• Loss of electrolytes: Potassium→ Disturb heart rhythm
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Bicarbonate \rightarrow Acidosis \rightarrow weak heart

Review Questions

What is the causative agent of the following diseases: cholera, Glander, foot rot of man?

What is the major characteristic of *P. aeroginasea- V. cholera*?

What is the different between endotoxins, enetrotoxins and neurotoxins?

Write the Latin name of the bacteria that cause 50% of the food poisoning cases in Japan?

Differentiate between Vibrio cholera and Vibrio parahaemolyticus?

Give six examples of *P. aeruginosa* virulence factors? Give three examples of its toxins, with its roles? Its exotoxin A is similar with other bacteria toxin. What is it?

Why *P. aeruginosa* can grow on distilled water? The bacteria can grow at two different temperature. Explain. What it's the special pigment?

Why V. cholera needs a large numbers of cells to initiate their infections?

Cholera fatality or death may results from severe fluids and electrolytes loss due to dehydration, explain? How can lead the loss of potassium and bicarbonates- How can you treat a patient with severe diarrhea as a result of *V. cholera* infection?

What is the characteristic of cholera diarrhea? Explain the mechanism of the V. cholera enterotoxin?

What is the major virulence factor for *V. cholera*?

Give three examples of *P. aeruginosa* infections? (nosocomial urinary tract infection introduced by catheter- Wound infection of burn sites- eye infection)