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Microbiology

Doctor 2017 | Medicine | JU

Sheet Slides

Done By

Contributed in the Scientific Correction

Contributed in the Grammatical Correction

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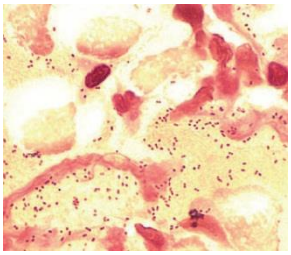
Doctor

Let's start our journey!

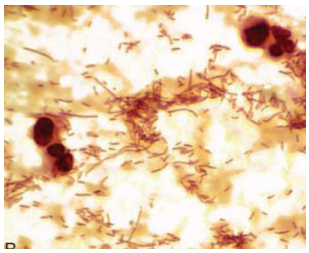


Firstly:

Haemophilus: -as the name implies- they're blood lovers

❖ General information:

1- Haemophilus species are present in almost all individuals, primarily colonizing the *mucosal membranes of the respiratory tract*, H. influenzae (particularly serotype b [biotype I]) is uncommon in the upper respiratory tract.



2- The surface of many Haemophilus is covered by a polysaccharide capsule, and *six antigenic serotypes (a-f)* on which the typing depends.

❖ Features and morphology:

Small, Pleomorphic (variant shapes like: rods and coccobacilli) & Gram negative.

❖ growth requirements:

- They're considered fastidious (they need special nutrients on the agar to grow properly)
- Nutrients requirements: NAD (factor V), and hemin (factor X) which are both in RBCs.
- Blood agar *by itself isn't enough*, since the bacteria needs lysed RBCs to get factors X and X, to by-pass this we use special type of blood agar called chocolate agar (don't be fooled by the name, hence it contains no actual chocolate just broken down (heated) RBCS that have brown-chocolate like color).
- Sometimes, Haemophilus colonies surround the staph colonies, since the staph colonies lyse RBCs releasing factors V and X.

-Note: the name H. influenzae was attributed in the past to the influenza disease (then viruses were discovered to be the actual behind it).

❖ Identification and detection:

Identifying Haemophilus strains is by: 1) under the microscope, if you see small pleomorphic gram negative 2) they only grow on chocolate agar.

Detection occurs through: ***PRP capsular antigen*** (poly ribitol phosphate)

❖ Prevention:

1) Due to vaccines (purified PRP) dependent on the polysaccharide capsule antigens, and this is important since the capsule won't resist phagocytosis, which means antibodies can attack it facilitating phagocytosis.
2) Anti-capsular antibodies will develop too if you were infected (natural infection).

3) Passive Transfer of the antibodies -from mother to fetus- via the placenta.

❖ Epidemiology:

- In the past it used to be an important pathogen, but nowadays thanks to vaccines, it is no longer as important.
- The problem is now due to non-encapsulated/nontypeable H. influenzae since vaccines developed were to the encapsulated strains.
- In countries with poor vaccination programs, H. influenzae is a major pathogen, mainly type b in children.

❖ Pathogenesis:

- The danger behind it, emerges from its ability to disseminate to blood after avoiding phagocytosis.
- Diseases it causes:

a) Meningitis (in unvaccinated children): inflammation of the meninges.
b) Epiglottis: inflammation of the epiglottis which closes the airway to prevent the food from going to the respiratory tract (regulating breathing and eating), and it's characterized by difficulty in breathing that may progress to total obstruction and fatality

** The uncapsulated H. influenzae commensally colonize within the nasal cavity and the nasopharynx.

c) Pneumonia: mostly in the elderly by nontypeable strains (if immunocompromised or got infected by nontypeable strains -even if vaccinated -you will get the disease).
d) Acute and chronic otitis (infection of the ear) and sinusitis: the most common cause along with S. pneumoniae.
e) Cellulitis & bacteremia

*Risk factors: if you're unvaccinated, depleted of complement -opsonin- or underwent splenectomy (since it has a lot of WBCs as a part of reticular endothelial system), increasing susceptibility to capsulated pathogens.

Haemophilus influenzae

Meningitis: a disease primarily of unimmunized children characterized by fever, severe headache, and systemic signs

Epiglottitis: a disease primarily of unimmunized children characterized by initial pharyngitis, fever, and difficulty breathing, and progressing to cellulitis and swelling of the supraglottic tissues, with obstruction of the airways possible

Pneumonia: inflammation and consolidation of the lungs observed primarily in the elderly with underlying chronic pulmonary disease; typically caused by nontypeable strains

Other strains:

- H. aegyptius (Koch-weeks bacillus)
  1- It was common amongst population of ancient Egypt
  2- Causative agent of acute purulent *conjunctivitis.*
- H. ducreyl:
  1- Causes *chancroids*: lesion-like tender papules with red base on the tip of genitalia or perianal areas
  2- After 5 -7 days of exposure
  3- occurs in men via *sexual transmission*.

ϖ Due to active immunization program in the USA, H. influenzae is uncommon unlike the rest of the globe.

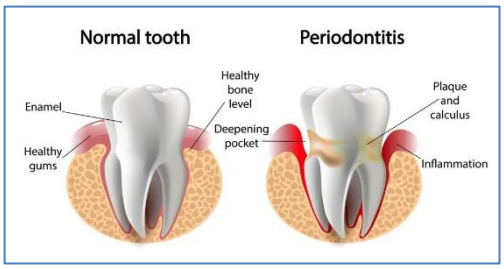
Secondly:

Aggregatibacter

- There are 2 members of this genus: **A. actinomycetemcomitans** and **A. aphrophilus**.
- A. actinomycetemcomitans:

✓ Remember the bacteria in your oral cavity can be pathogenic, especially with poor oral hygiene.

✓ It causes periodontitis – inflammation of the gum adjacent to the tooth, characterized by plaques and calculi on the tooth.

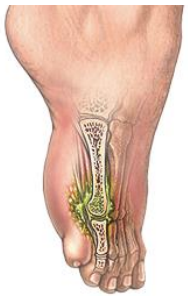


✓ Can colonize the mouth, but also can disseminate to the blood -if a bite or dental procedure took place- attaching to your damaged or artificial heart valves causing endocarditis.

Thirdly:

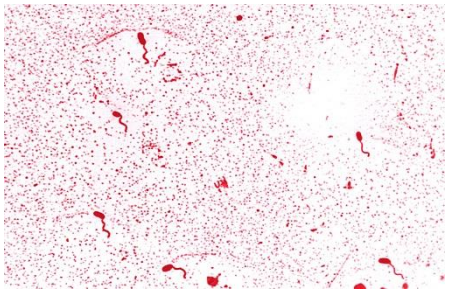
Pasteurella

- Gram-negative, facultatively anaerobe (fermentative) coccobacilli.
- commonly found as *commensals in the oropharynx of healthy animals.*
- Major strains usually pathogens to humans: *P. multocida* and *P. canis*.
- If a dog bites you, a cat scratches you or you share food with your animals; a disease might develop starting as cellulitis ending up as a local chronic infection of deep tissues and osteomyelitis.



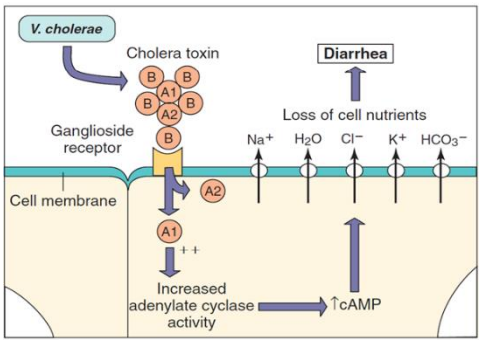
The final type for this lecture:

Vibrio:



- Features ad morphology:
  - *facultatively anaerobic (fermentative)*, *oxidase positive*, *Gram negative curved rods with polar flagellum (on one side) for motility*.
  - These are the human pathogens: V. cholerae, V. parahaemolyticus, and V. vulnificus.
  - V. cholerae is subdivided - using serotyping – to V. cholerae O1 and O139 which make cholera toxin & cause cholera epidemics, other strains of V. cholerae are not toxin producing and epidemic causing – however, they may cause non-epidemic infections.
- Growth:
  - A simple media within a broad temperature range (from 14° C to 40° C)
  - Tolerate a wide range of pH (e.g., pH of 6.5 to 9.0) but are *susceptible to stomach acids*.
  - Halophilic species: require sodium chloride (NaCl) for growth.
  - Live in: oceans, seas and small lakes/estuarine and marine environments, also can live waters with chitinous shellfish (and this relates to its halophilicity and mode of transmission).
- Transmission and epidemiology:
  - V. cholera is spread by contaminated water and food rather than direct person-to-person spread, because a high inoculum (e.g., >108 organisms) is required to establish infection in a person with normal gastric acidity -> so if you get it in any form from a person, your acidic stomach will destroy the small number of cells that were transmitted to you and no infection occurs.
  - Cholera is usually seen in communities with poor sanitation, like in: India and Africa where people get water from anywhere.
  - It is estimated that 3 to 5 million cases of cholera and 120,000 deaths occur worldwide each year.
  - Seven major pandemics of cholera have occurred since 1817, resulting in thousands of deaths and major socioeconomic changes and the most recent outbreak was in Yemen due to war and poor sanitation.

℘ Pathogenesis and Virulence factors:



First: **Cholera toxin**

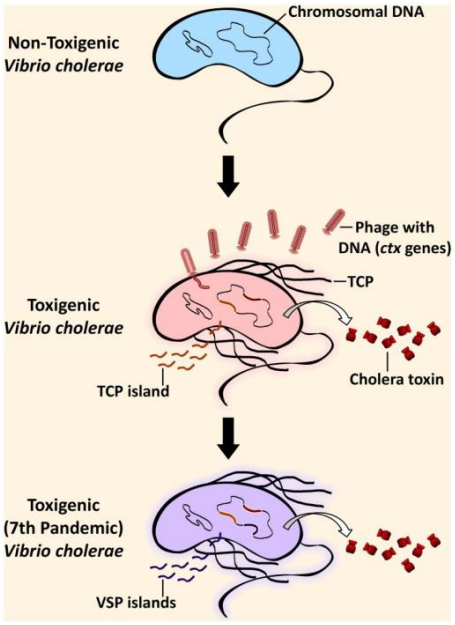
ϕ Once V. cholerae is ingested, *it releases a certain toxin* **– as shown here –** into the intestine

ϕ This toxin is a classic A-B toxin, B subunit helps in binding the ganglioside receptor while the A subunit is internalized causing increase adenylyl cyclase activity which leads to increased amounts of cAMP.

ϕ Increased [cAMP] contributes in the massive secretion of water and electrolytes – like: Na^+, Cl^-, K^+ and HCO_3^- from the epithelium.

Second: **Toxin co-regulated pilus (TCP)** and **chemotaxis proteins**

- Their role isn't only for adhering the bacteria to the enterocytes.
- They contribute in transforming the non-toxigenic V. cholerae to toxigenic, and this happen as follows:



1. Toxigenic cholerae expresses TCP on VPI
2. It's infected by a phage and lysogenic cycle occurs
3. This phage infects a non-toxigenic cholerae and its genome is incorporated with the non-toxigenic cholera rendering it toxigenic.

℘ Clinical manifestation, symptoms and treatment:

ψ It starts showing symptoms at an average of 2 to 3 days after ingestion of the bacteria (can be <12 hours).

ψ Characterized by:

1- abrupt onset vomiting and watery diarrhea (as much as 1L/hr) and within few days the *person is dehydrated* - (here the diarrhea is in the form of rice-water stools which indicates that it's like the rice water turbid with no smell).

2- Painful muscle cramps.

3- Metabolic acidosis – due to bicarbonate (HCO_3^-) loss

4- Hypokalemia with hypovolemic shock (potassium loss) and renal failure.

5- Cardiac arrhythmia (since the heart depends on certain electrolytes).

ψ It's treated by rehydration and replacement of the lost electrolytes (this has decreased the mortality from 70% to less than 1%).