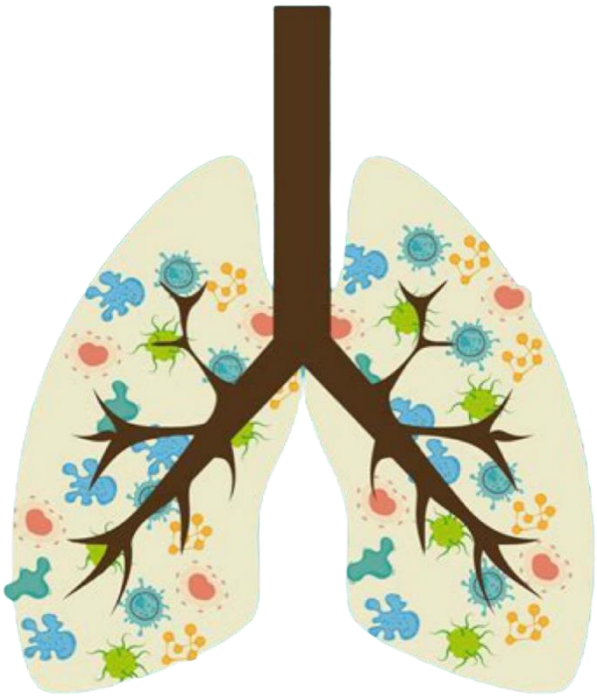
Doctor 2020 - wateen - medicine - MU



Microbiology sheet

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Respiratory Bacterial Infections

Pseudomonas and related organisms

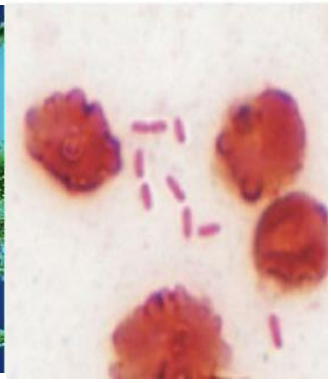
Aerobic gram-negative non fermentative rods

Pseudomonas aeruginosa: extremely opportunistic infections of multiple sites.

Moraxella catarrhalis: opportunistic RT infections.

Opportunistic : the patient loss their immunity, then this microb come, replicate and do a disease .

Structure and Physiology



- Gram-negative rods.
- Motile with polar flagella.
- Obligate aerobe.
- Oxidase-positive.
- Encapsulated.

Do not ferment carbohydrates.

Resistant to multiple drugs.

Aerobe : The microbe infect the lungs or the surfaces outside organs like (skin) . So, this microbe need oxygen to come and replicate .

They know oxidizer (they use oxygen to produce energy).

They are not fermenter (do not ferment glucose) → Psudomonas and moraxella.

They found in different sites which cause different disease and clinical symptoms.

Motile with Polar flagella → flagella in the end at one side, which get it move forward .

Encapsulated → * antiphagocytic

* stick to surfaces → this microbe attached and stick in plastic things like (inhaler, catheter, cannula) →use its capsule in this process (it has lipopolysaccharides in the surface. So, they can stick to this places.)

So, encapsulated is kind of virulence factors which use it just to stick to the surface.

They are resistant to multiple drugs : multiple drugs means the drug not act on one site , some act on cell wall, proteins ,nucleic acid . So, this microbe use multiple defense mechanisms against the antibiotics.

For example need to go inside the cell using the pores, this microbe closed this pores. So, they stop the action of these drugs , or they produce enzymes (betalactames enzymes) penicillin and sefalosporin have betalactam ring. This ring destroy cell wall. If microbe produce betalactames enzyme , this ring destroy so, no drug action.

P. aeruginosa

Forms round colonies with afluorescent greenish color, fruityodor, and □-hemolysis.

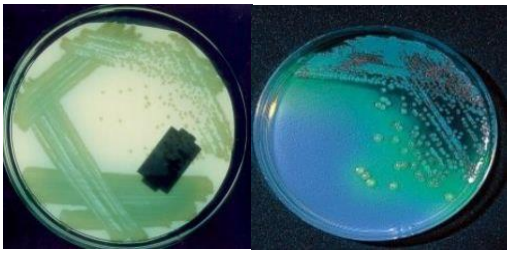
Pyocyanin- nonfluorescent

bluish pigment;

pyoverdin- fluorescent

greenish pigment;

pyorubin, andpyomelanin



pyomelanin→(black).

In this microbe if the infection in skin we know from the color.

Bus either blue or green, have a good odour (fruity odour)in the place.

This due to beta-hemolytic action in blood agar . (complete hemolysis)

Identification of P. aeruginosa is usually based on oxidase testand its colonial morphology: □-hemolysis, the presence ofcharacteristic pigments, sweet odor, and growth at 42C.

42C growth temperature not normal for microbes→ which prefer(36.5 -37-38)

This characteristic give you evidences this microbe is p. aeruginosa.

P. aeruginosa: Pathogenesis and Immunity

- This organism is widely distributed in nature and is commonlypresent in moist environments in hospitals. It is pathogenic onlywhen introduced into areas devoid of normal defenses, e.g.,

1. Disruption of mucous membrane and skin.

2. Usage of intravenous or urinary catheters.

3. Neutropenia (as in cancer therapy).

- P. aeruginosa can infect almost any external site or organ.

- P. aeruginosa is invasive and toxigenic. It attaches to and colonizesthe mucous membrane or skin, invade locally, and producessystemic diseases andsepticemia.

- P. aeruginosa is resistant to many antibiotics. It becomes dominantwhen more susceptible bacteria of the normal flora are suppressed.

It has multi virulence factors to cause a disease.

In hospitals it found on respirator , shower, conditioners, inhalers.

Immundeficiency patients→ * take corticosteroids drugs *AIDS *burnings and injuries *chronic disease *cancer patients (target to p.aeruginosa) * devices

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Virulence Factors

Microbe infected the body by two ways :

1) Direct invasion OR 2)toxin production / Or both two

It spread by direct contact in (water, devices, doctor stethoscope with out sterilization , respirators, beds ….)

-Antigenic structure,enzymes, and toxins:

Pili and nonpilus adhesions.→to attachment

Capsule seen in cultures frompatients with cystic fibrosis.→antiphagocytics

* in cystic fibrosis (genetic disease (inherited) which is distraction of cell in lung ,p.aeruginosa is problem for cystic fibrosis patient due to it is attach and increase cell death.)

LPS- endotoxin, multipleimmunotypes.

Pyocyanin: catalyzesproduction of toxic forms ofoxygen that cause tissuedamage. It also induces IL-8production.

O3 (Ozone)→ which causes tissue damage so help in microbe spread

we filter water with ozone due to inhibition of pyocyanin in some microbes and can kill them .

IL-8 which recruit inflammatory cells (inflammatory response) →to stop infection , but it is harmful mechanism (distraction locally in this response)

So, increase effect of disease

Pyoverdin: asiderophore.

Proteases: protease cause tissue damage and help bacteria spread.

Phospholipase C: a hemolysin

Exotoxin A: causes tissue necrosis and is lethal for animals(disrupts protein synthesis);immunosuppressive.

Exoenzyme S and T: cytotoxic tohost cells.

All cause cell distraction .

Clinical Diseases

- Infection of wounds and burns, (blue-green pus). Patients with severe burns may develop intobacteremia→ if it reach blood.

- Skin and nail infections.

- Meningitis: (when introduced bylumbar puncture).

Dr. use contaminated needle.

*- Pulmonary infection: Tracheobronchitis

*- Necrotizing pneumonia in CFpatients: diffuse, bilateralbronchopneumonia withmicroabscess and necrosis.

- Eye infections.

- Ear infections.→With people who swim

- Otitis externa: mild inswimmers; malignant (invasive)in diabetic patients.

Chronic otitis media.

- Osteochondritis of the foot.

- Urinary tract infection.

- Gastrointestinal infection.

- Sepsis.

Laboratory Diagnosis

Specimen: skin lesions, pus, urine, blood, spinal fluid, sputum , nails , hair.

Multisite of infection.

Culture: blood agar plate and differential media.

Treatment

Combined antibiotic therapy is generally required to avoid resistance that develops rapidly when single drugs areemployed. Aminoglycoside, antipseudomonal B-lactam or aquinolone.

Antibiotic sensitivity test is important before give any drug.

If we give antibiotics haphazardly, we kill the normal flora. Which is good for body.

Empirical treatment → treatment without diagnosis and test (in severe cases / life threatening) as meningitis in children (cause disability)

- in ER take lumber puncture(before give any drug if after –ve result) to lab then give broad spectrum antibiotic until you see test result -1-2 days-

Combination of two different mode of actions.

Prevention and Control

Spread is mainly via contaminated sterile equipments and crosscontamination of patients by medical personnel.

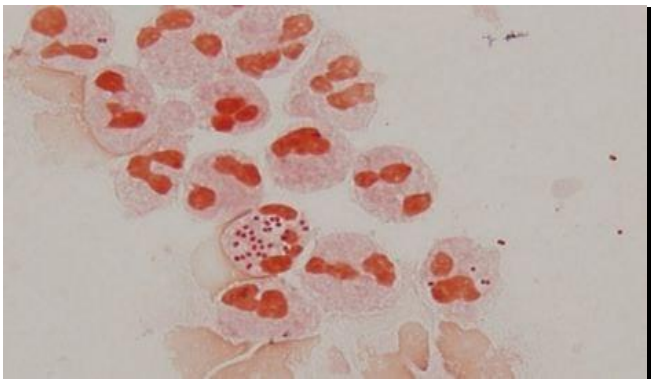
Control:

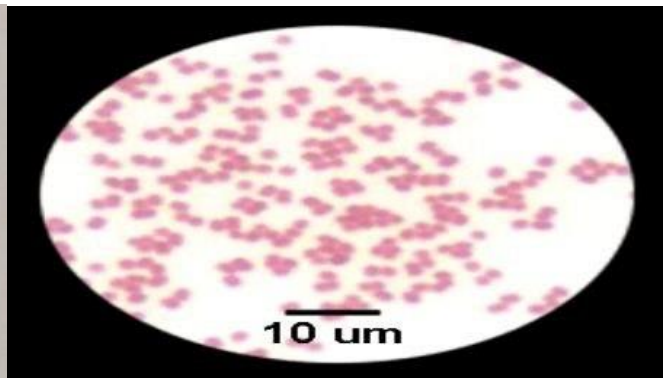
1. Patients at high risk should not be admitted to a ward wherecases of pseudomonas infection are present.

2. Patients infected with P. aeruginosa should be isolated.

3. Sterilize all instruments, apparatus, and dressing

If we have PA patient do not mix him and isolate him away from highly suitable to the disease . And sterilize things and dressings, … .





MORAXELLA CATARRHALIS

General characteristics:

- Aerobic, gram-negative cocci or cocobacilli.
- Diplococci or diplococcibacilli.
- Non motile.
- Oxidase positive.
- They don't ferment carbohydrates.

-Catalase +ve .

Normal commensal of the respiratory tract (humans only).

Has become an important opportunistic pathogen.

It is a normal flora of respiratory tract in human .

Clinical infections

- Pneumonia.
- Sinusitis.
- Otitis media (3rd most common cause).
- Eye, CNS, Joints infection.

Predisposing factors

- Advanced age
- Immunodeficiency
- Neutropenia
- Other debilitating diseases

Laboratory diagnosis

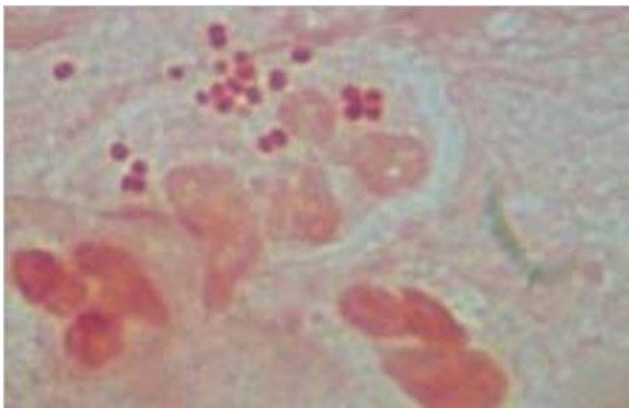
Colonies appear smooth with a grayish- white color.

When colonies pushed with loop, they "scoot" acrossmedia.

*they are not attached firmly to the Surface of the agar

Moraxella catarrhalis growingchocolate agar after 48 hours ofincubation

Direct smear from an otitis media Sampleshowingintracellular gramnegative diplococci.



Laboratory Diagnosis and treatment

- Oxidase positive→ aerobe

- Catalase positive

- All sugar fermentation negative

- Produce beta- lactamase→resistant to betalactam antibiotic

- DNase positive

Treatment: fluoroquinolones, most second and thirdgeneration cephalosporins, erythromycin, andamoxicillin-clavulanate.

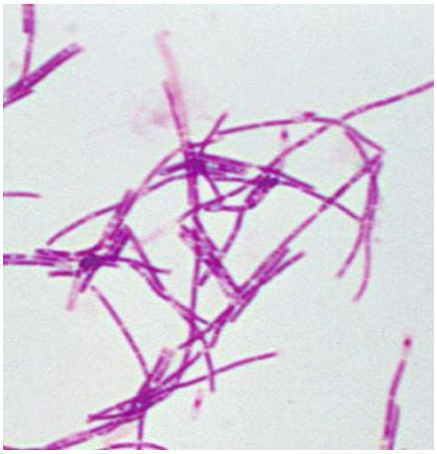
In treatment if the enz. Not enough the antibiotic act in microbe.

In sephalosporin scientists increase NO. of rings.

Bacillus

B. anthracis: anthrax of the animals and humans.

Morphology and Physiology



- Aerobic or facultative anaerobic.

- Large gram-positive rods, havesquare ends, arranged in long chains.

- Spore is located in the center of thecell.

Spore→ once microbe in harmful environment they couldn't to survive they produce spores to adapt with conditions (heat, disinfectants,…) and go around in air, soil. If found good place to regenerate, multiply again. (survival methods and virulence factors).

By autoclaving we kill spores.

- Most are saprophytic (soil, water, air,

and on vegetation.)

-Non hemolytic.

#people at risk to be infected:

*farmers *who have pets *who work at slaghterhouses

Physiology and Structure

- B. anthracis is encapsulated and non-motile.

- The capsule consists of polypeptide (poly-D-glutamic acid)and is an important virulence factor.mutation of this capsule microbe become avirulent.

They produces toxins that can be fatal by causing necrosis

- The spores can withstand dry heat and certain disinfectantsfor moderate periods, and persist for years in dry earth.

Spores protect microbes…if they found a good environment, it will reactive again

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Pathogenesis and Immunity

- Primarily a disease of herbivores (sheep, cattle, horses);humans are rarely affected.

- In animals, portal of entry is mouth and GI tract. In humans, scratches in the skin (95% of infection), ingestion or inhalation lead to infection.

- The spores germinate in the tissue at the site of entry, and growth of the vegetative forms results in gelatinous edema and congestion. Bacillus spread via lymphatics to the blood and other tissues.

Bleeding and sudden death of animals /the farmer infected

* More in GI (ingestion of spores) – in animals

*Cutaneous /skin, inhalation and GI- in human

To cause infection must high load of spores…. (10-20..) not enough.

Anthrax Suspected Carcass Sampling

Spores they come attach cutaneously and they germinate then produce toxins cause ulcers black in skin might go to blood or other organs.

Virulence factors

- Capsule (encoded from a plasmid)→ invasion

- Exotoxins (A-B toxins encoded from another plasmid)

#if the plasmid is not found in the microbe, it would be avirulant

- Edema toxin is composed of protective antigen (B-subunit)and edema factor (EF; an adenylate cyclase). This toxincomplex increases vascular permeability which leads to shock.

- Lethal toxin is composed of protective antigen and lethalfactor (LF; ametalloprotease). This toxin causes cell deathand stimulates macrophages to release proinflammatorycytokines.

Clinical Diseases

- Inhalation anthrax (wool-sorters' disease): long incubationtime (2 months or more).veryrare one.

في القدم كان يصيب الصوافين في القرى

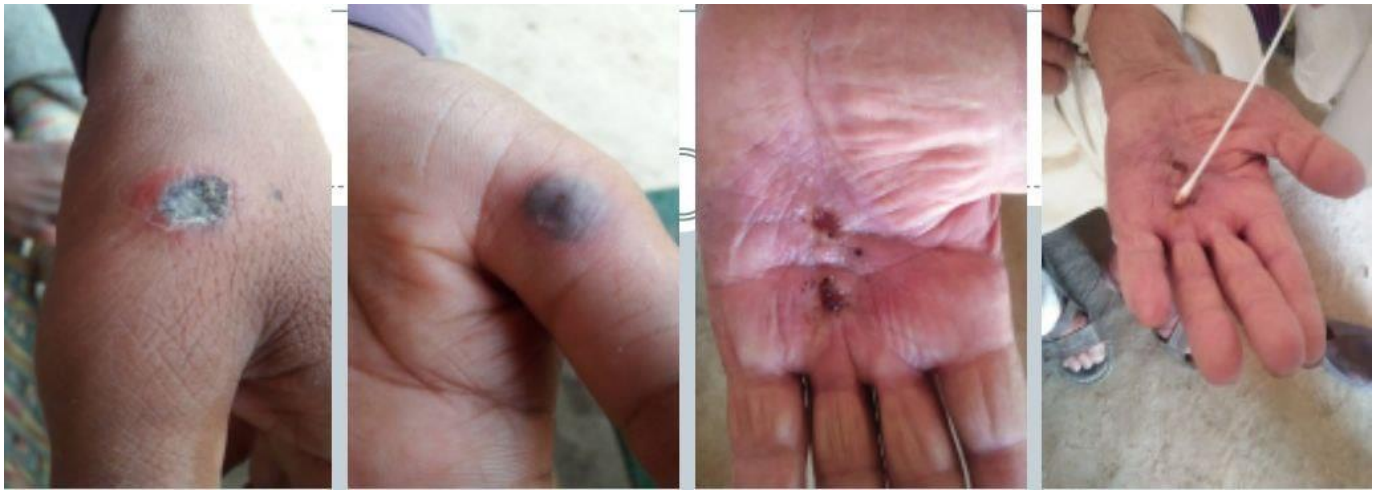
Inhaled the spores then go to his lung and replicate , infect lymph node, enlargement .

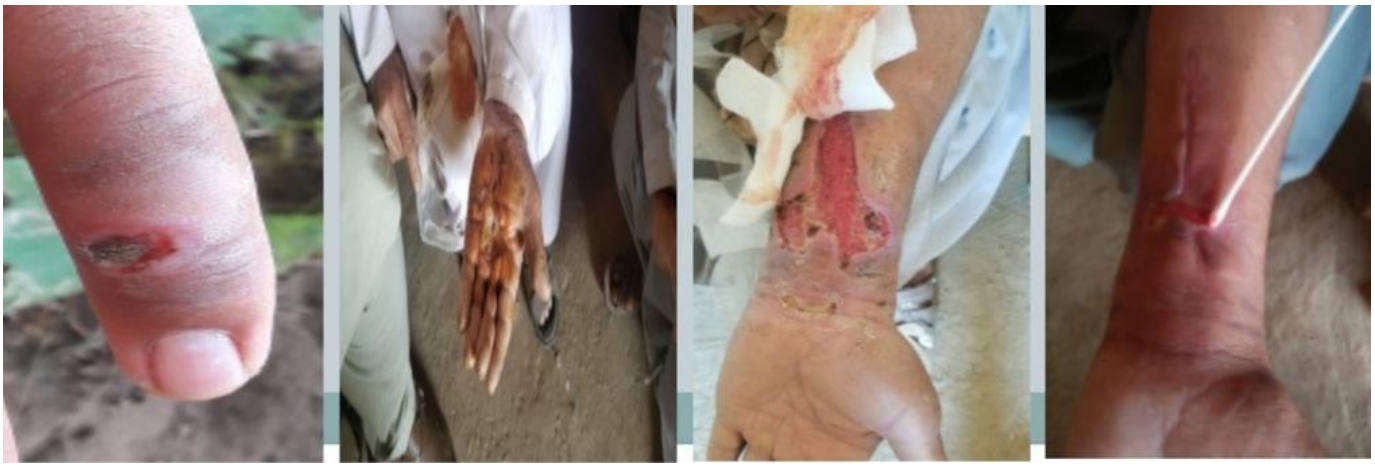
Progressive hemorrhagic lymphadenitis /Mediastinitis(enlargement of mediastinal lymph nodes), sepsis, andmeningitis (50% patients).

Pulmonary disease rarely develops. Fatal if untreated 100%

- Cutaneous anthrax most common

- Gastrointestinal anthrax (very rare)





Human Cutaneous Anthrax Sampling (Suspected)

Laboratory Diagnosis

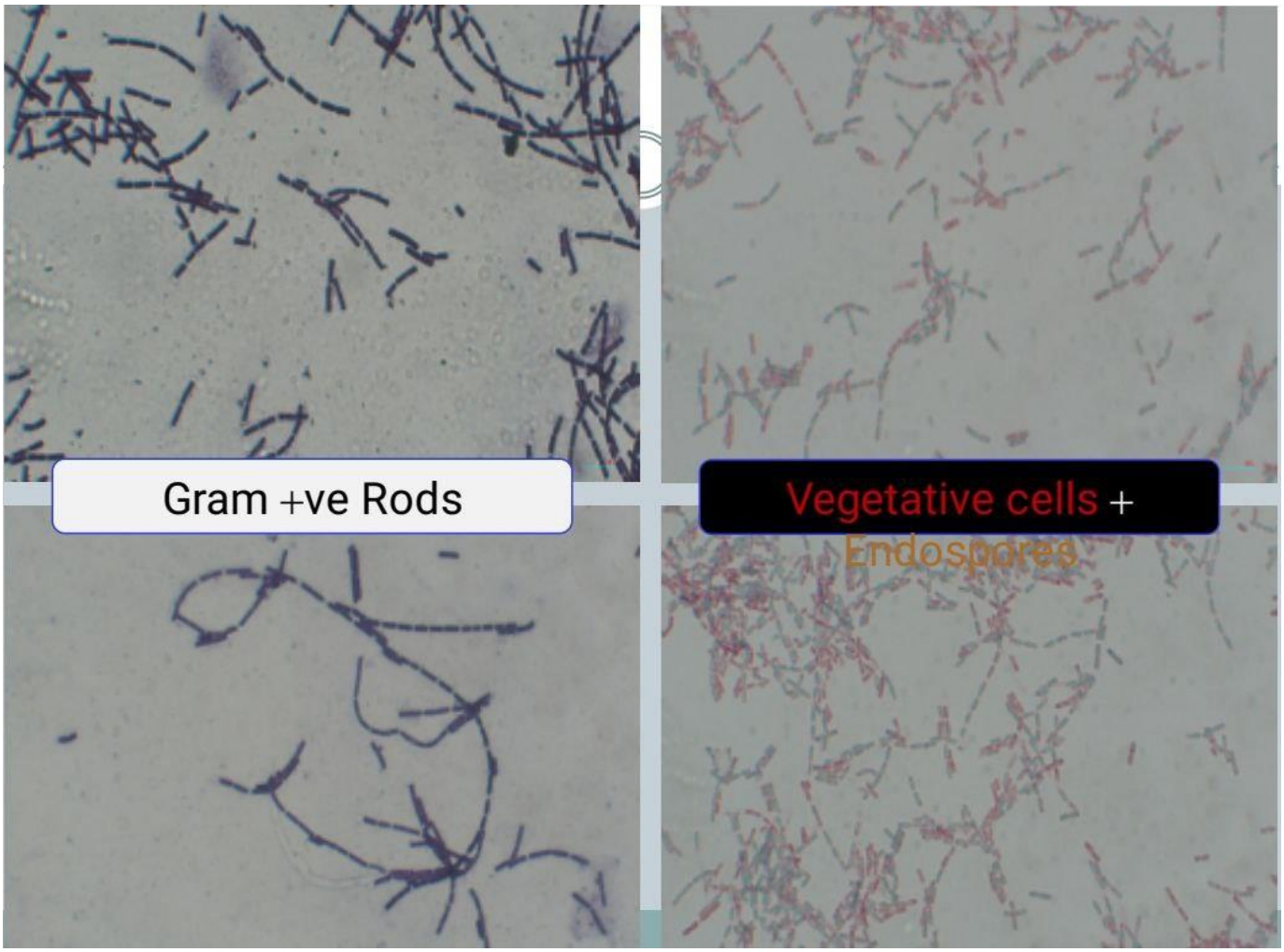
- Specimens: fluid or pus from local lesion, blood, or sputum.

- Smears: long chains (a characteristic of B. anthracis) of large gram-positive rods without spores can be seen. Immuno-fluorescencestain can be used for dried smears.

- Culture: nonhemolytic gray colonies with dry surface on blood agarplates.

- Identification: made in a reference lab by direct fluorescent Ab testagainst capsular polypeptide or PCR test.

- Serological tests: detection of antibodies to lethal toxin and edematoxin.



Long chain spore forming (from center)

Treatment

Multi drug therapy, Ciprofloxacin, rifampin and vancomycin.

Control

- Proper disposal of animal carcasses (burning or deep burial inlime pit).

- Autoclaving of animal products.

- Protective clothing and gloves for handling infected animals.

- Vaccination of domestic animals.2 types of vaccination : for animals and for human →not routine vaccine (is not taken in childhood only if you travel to place have anthrax as soldiers .

- Immunization of persons at high risk with a cell-free vaccinebased on the protective antigen is under investigation.

ليس لك من الأمر شيء ..

ما عليك إلا السعي ..

وما شاء الله كان ..

وما لم يشأ لم يكن ..