Respiratory Bacterial Infections

RESPIRATORY SYSTEM
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Pseudomonas and related organisms

Aerobic gram-negative non fermentative rods

Pseudomonas aeruginosa: extremely opportunistic infections of multiple sites

Moraxella catarrhalis: opportunistic RT infections

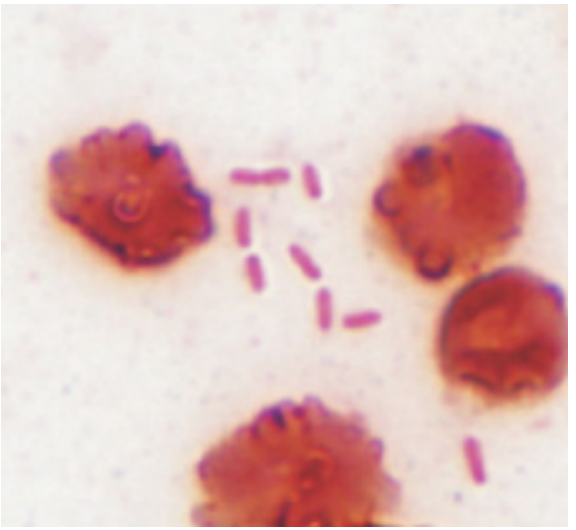
Pseudomonas

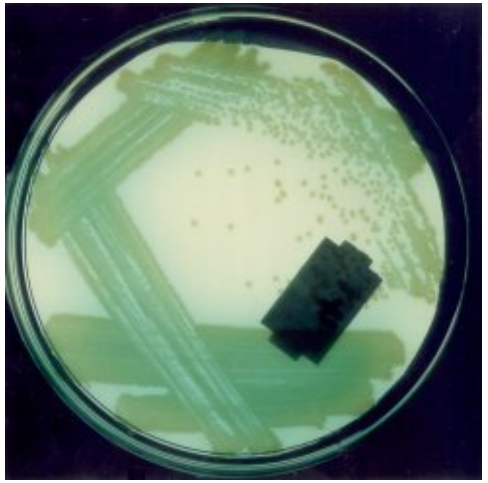
Structure and Physiology

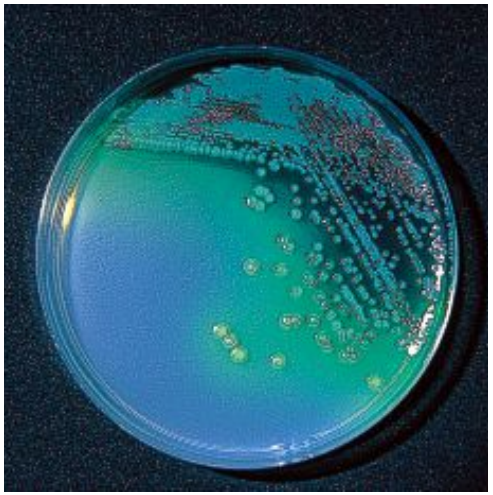
- Gram-negative rods.
- Motile with polar flagella.
- Obligate aerobe.
- Oxidase-positive.
- Encapsulated.

Do not ferment carbohydrates.

Resistant to multiple drugs.







P. aeruginosa

Forms round colonies with a fluorescent greenish color, fruity odor, and β-hemolysis.

Pyocyanin- nonfluorescent bluish pigment;

pyoverdin- fluorescent greenish pigment;

pyorubin, and pyomelanin

Identification of *P. aeruginosa* is usually based on oxidase test and its colonial morphology: β-hemolysis, the presence of characteristic pigments, sweet odor, and growth at 42 ^{o}C.

P. aeruginosa: Pathogenesis and Immunity

This organism is widely distributed in nature and is commonly present in moist environments in hospitals. It is pathogenic only when 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 colonizes the mucous membrane or skin, invade locally, and produces systemic diseases and septicemia.

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

P. aeruginosa

Virulence Factors

Antigenic structure, enzymes, and toxins

Pili and nonpilus adhesions.

Capsule seen in cultures from patients with cystic fibrosis.

LPS- endotoxin, multiple immunotypes.

Pyocyanin: catalyzes production of toxic forms of oxygen that cause tissue damage. It also induces IL-8 production. Pyoverdin: a siderophore.

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 to host cells.

P. aeruginosa

Clinical Diseases

Infection of wounds and burns (blue-green pus). Patients with severe burns may develop into bacteremia.

Skin and nail infections

Meningitis (when introduced by lumbar puncture).

Pulmonary infection

Tracheobronchitis

Necrotizing pneumonia in CF patients: diffuse, bilateral bronchopneumonia with microabscess and necrosis.

Eye infections

Ear infections

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

Chronic otitis media

Osteochondritis of the foot.

Urinary tract infection

Gastrointestinal infection

Sepsis

P. aeruginosa

Laboratory Diagnosis

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

Culture: blood agar plate and differential media.

Treatment

Combined antibiotic therapy is generally required to avoid resistance that develops rapidly when single drugs are employed. Aminoglycoside, antipseudomonal B-lactam or a quinolone

P. aeruginosa

Prevention and Control

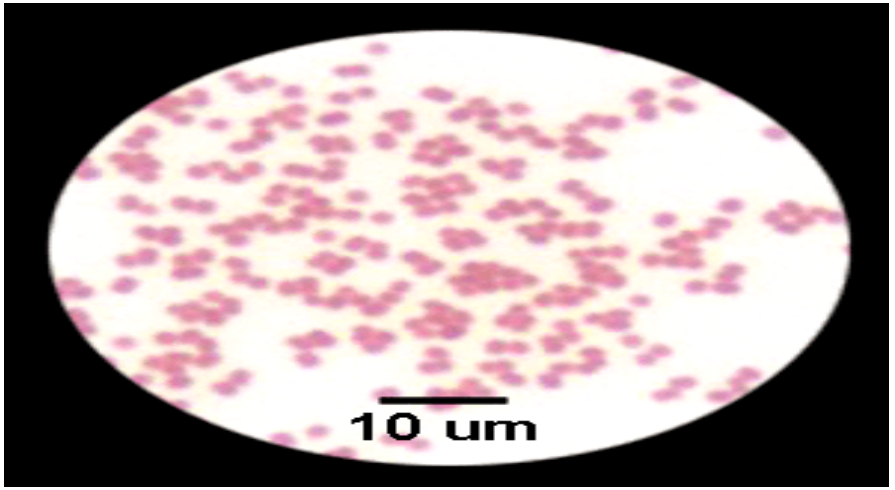
Spread is mainly via contaminated sterile equipments and cross-contamination of patients by medical personnel.

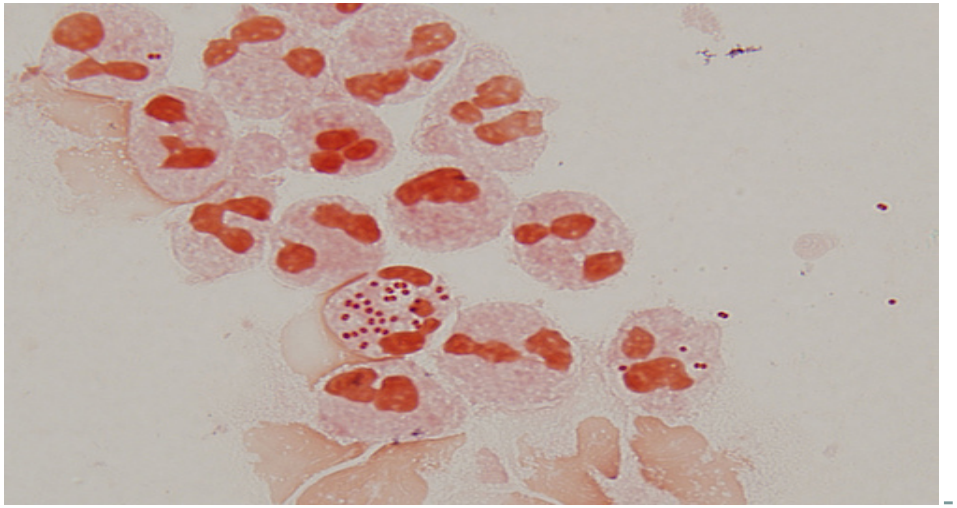
Control:

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

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

3. Sterilize all instruments, apparatus, and dressing





MORAXELLA CATARRHALIS

Moraxella catarrhalis

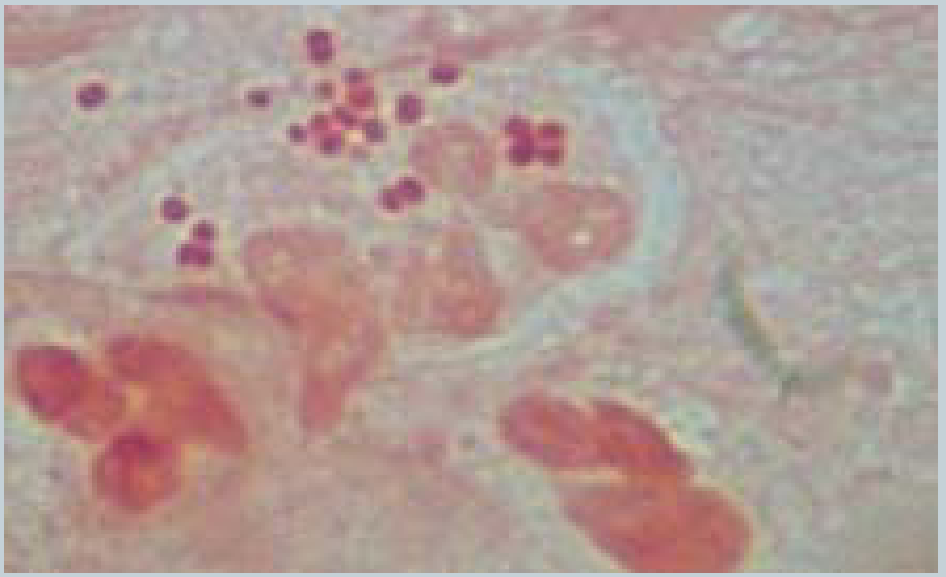
- General characteristics
  - Aerobic, gram-negative cocci or cocobacilli
  - Diplococci or diplococcibacilli
  - Non motile
  - Oxidase positive
  - They don't ferment carbohydrates
- Normal commensal of the respiratory tract (humans only)
- Has become an important opportunistic pathogen

Clinical infections

- 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” across media



Direct smear from an otitis media sample showing intracellular gram-negative diplococci

Moraxella catarrhalis growing on chocolate agar after 48 hours of incubation

Laboratory Diagnosis and treatment:

- Oxidase positive
- Catalase positive
- All sugar fermentation negative
- Produce beta- lactamase
- DNase positive

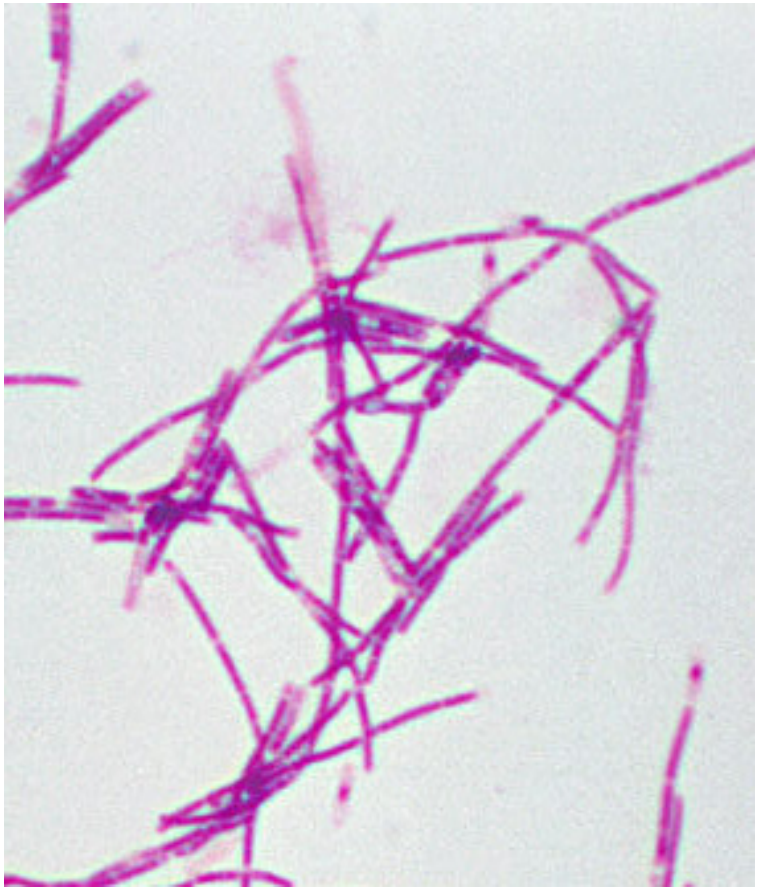
Treatment: fluoroquinolones, most second and third generation cephalosporins, erythromycin, and amoxicillin-clavulanate.

Bacillus

***B. anthracis*: anthrax of the animals and humans.**

Morphology and Physiology

- Aerobic or facultative anaerobic.
- Large gram-positive rods, have square ends, arranged in long chains.
- Spore is located in the center of the cell.
- Most are saprophytic (soil, water, air, and on vegetation.)



B. anthracis

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

B. anthracis

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.

Anthrax Suspected Carcass Sampling

B. anthracis

Pathogenesis and Immunity

Virulence factors

- Capsule (encoded from a plasmid)
- Exotoxins (A-B toxins encoded from another plasmid)
  - Edema toxin is composed of protective antigen (B-subunit) and edema factor (EF; an adenylate cyclase). This toxin complex increases vascular permeability which leads to shock.
  - Lethal toxin is composed of protective antigen and lethal factor (LF; a metalloprotease). This toxin causes cell death and stimulates macrophages to release proinflammatory cytokines.

B. anthracis

Clinical Diseases

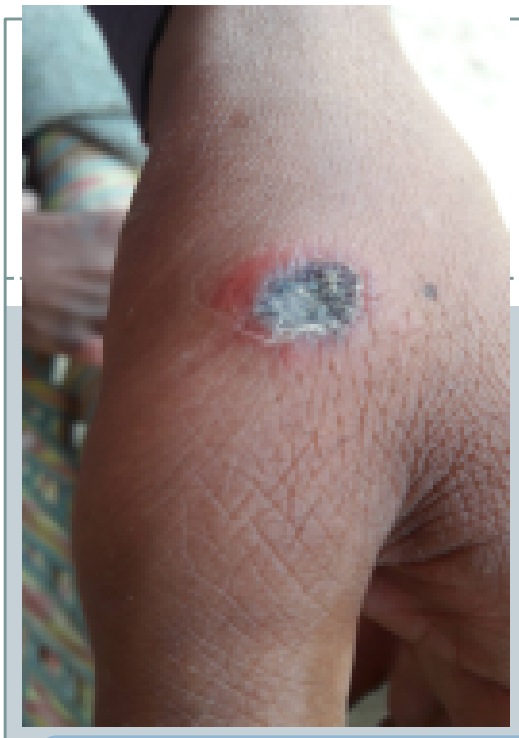
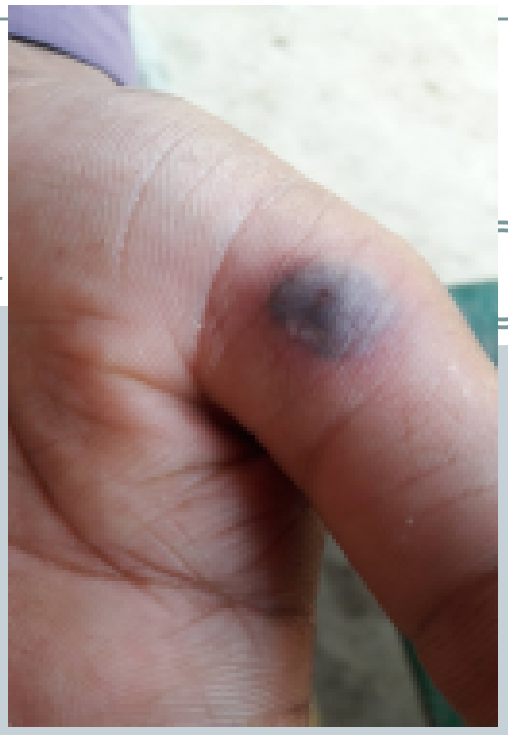
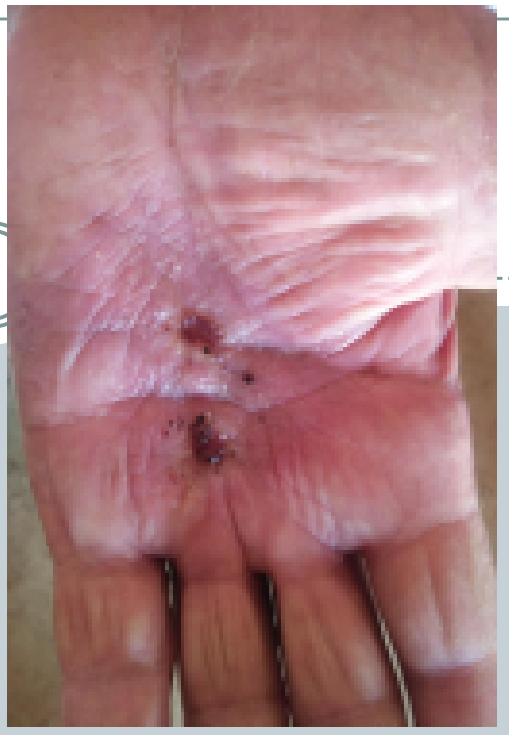
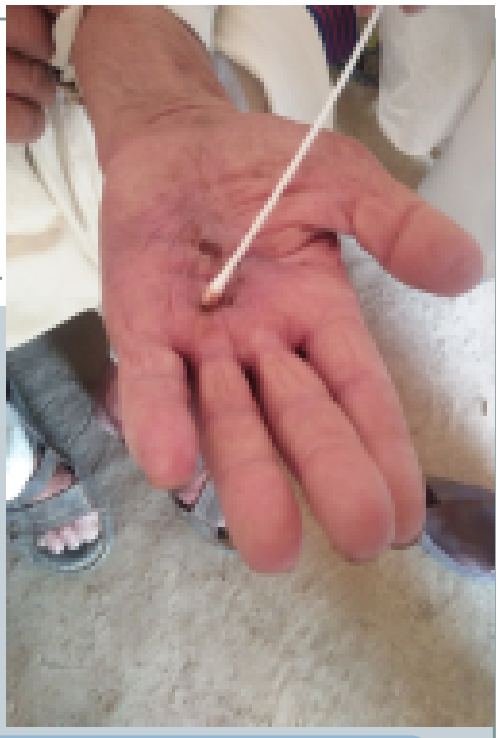
Inhalation anthrax (wool-sorters' disease): long incubation time (2 months or more).

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

Pulmonary disease rarely develops. Fatal if untreated 100%

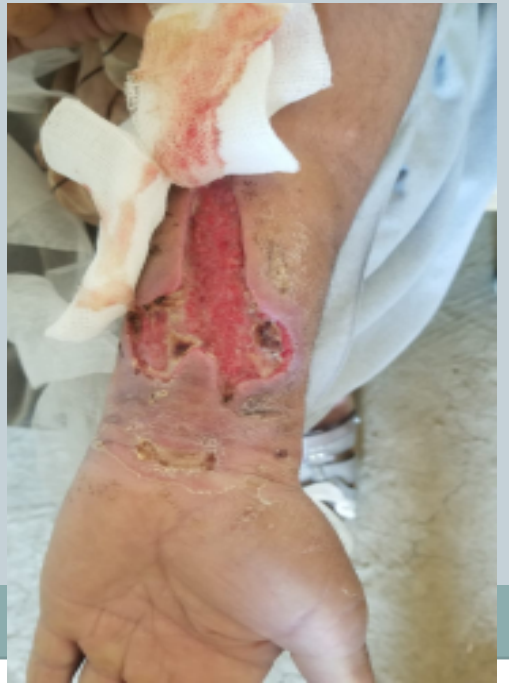
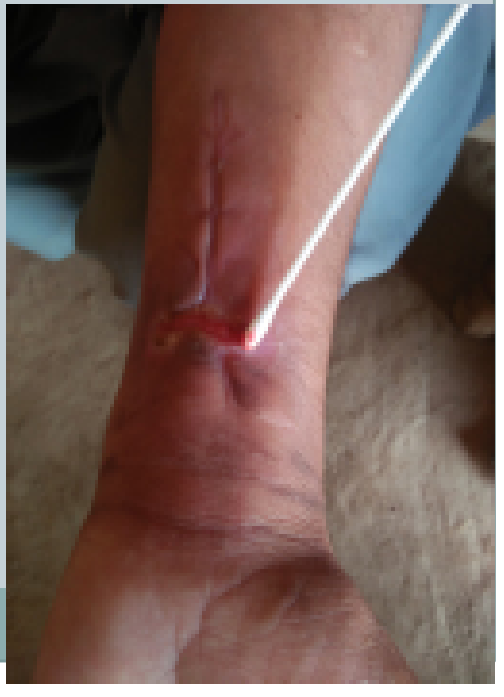
Cutaneous anthrax

Gastrointestinal anthrax (very rare)

Human Cutaneous Anthrax Sampling (Suspected)

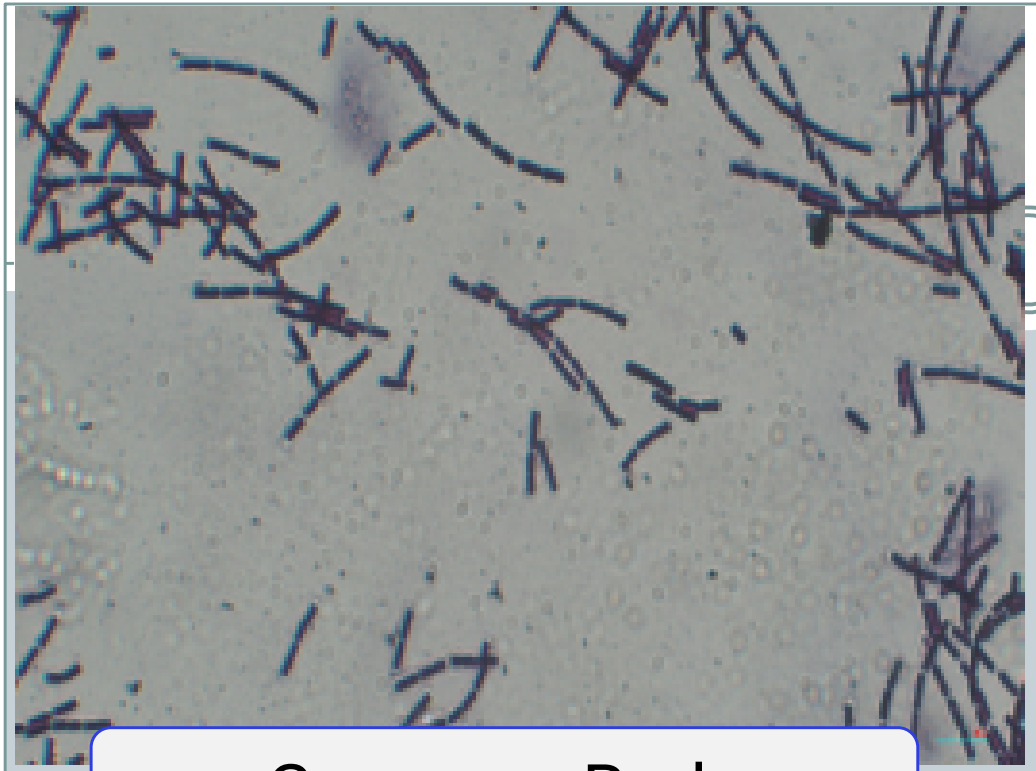


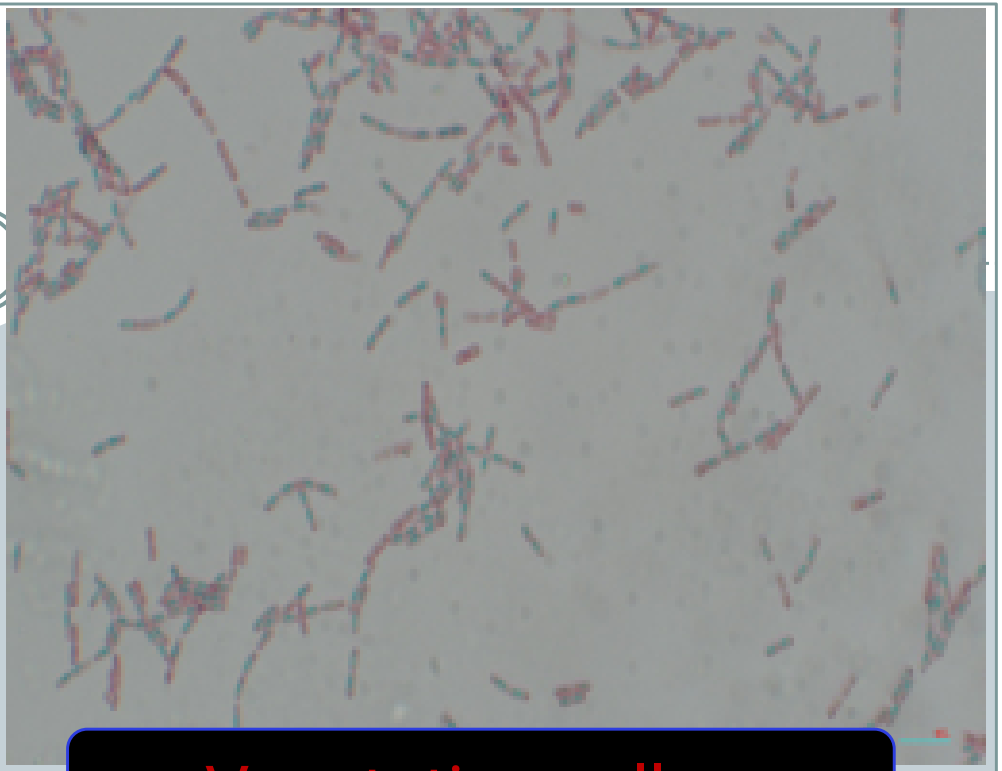
B. anthracis

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-fluorescence stain can be used for dried smears.
- Culture: nonhemolytic gray colonies with dry surface on blood agar plates.
- Identification: made in a reference lab by direct fluorescent Ab test against capsular polypeptide or PCR test.
- Serological tests: detection of antibodies to lethal toxin and edema toxin.

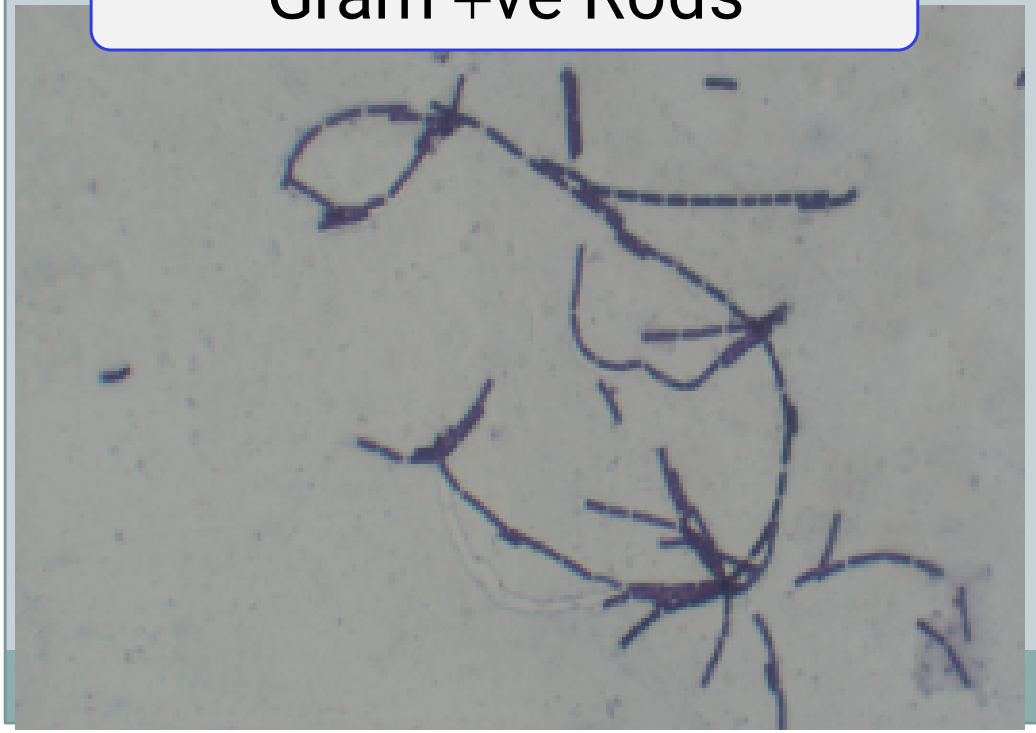


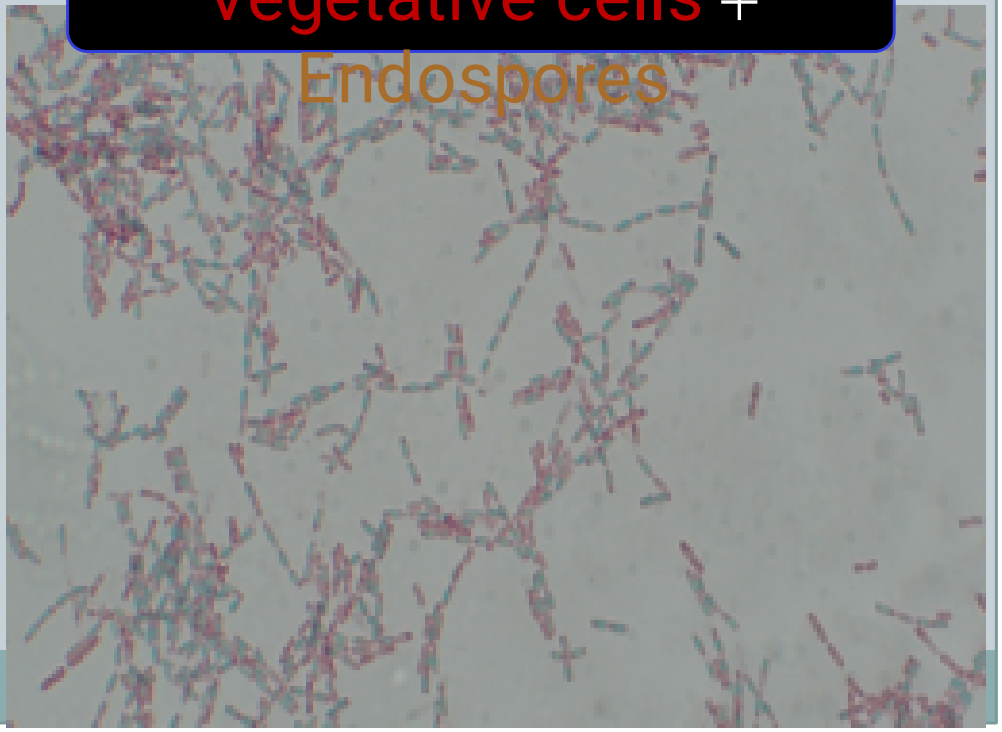
Gram +ve Rods



Vegetative cells +

Endospores





B. anthracis

Treatment

Multi drug therapy, Ciprofloxacin, rifampin and vancomycin

Control

- Proper disposal of animal carcasses (burning or deep burial in lime pit).
- Autoclaving of animal products.
- Protective clothing and gloves for handling infected animals.
- Vaccination of domestic animals.
- Immunization of persons at high risk with a cell-free vaccine based on the protective antigen is under investigation.

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MOLECULAR EPIDEMIOLOGY OF ANTHRAX IN JORDAN

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