University of Tikrit \College of science - Lectures of Pathogenic Bacteria \ 2024-2025 - Dr. Bushra Ali Kadhim





Ministery of Higher education and scientific research

University of Tikrit

College of science

Department of Biology

Lectures of Pathogenic Bacteria

For Diploma students – Pathological analyses - 2024-2025

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Shigella

Shigella species cause shigellosis (bacillary dysentery) Shigella are non motile , unencapsulated , and Lac -. Most strain do not produce gas in a mixed – acid fermentation of glucose . Human are the only natural host for shigella species.

Epidemiology :

1- Spread from person to person, with contaminated stools.

2- Flies and contaminated food or water can also transmitted the disease . 3- 10-100 viable organisms are sufficient to cause disease .

4- 40 serotypes organized in to four groups(A,B,C,and D) based on the polysaccharide O antigens.

The most prevalence species are

1- Shigella dysenteriae type 1 produce shiga toxin which is similar to shiga – like toxins 1 and 2 produced by E. coli . Shiga and shiga- like toxins are capable of resulting in susceptible individuals.

2- Shigella sonni (group D)

3- Shigella flexneri

Pathogenesis and clinical significance

Shigella invade and destroy the mucosa of the large intestine . Plasmid – encoded virulence genes that encode a type III secretion system . This plasmid encodes proteins that allow the shigella to polymerize actin at one pole .

This virulence plasmid is also possessed by EIEC. An exotoxin (shiga toxin)with enterotoxic and cytotoxic properties. It's may cause

1- Hemorrhagic colitis

2- Classic bacillary dysentery characterized by diarrhea with blood, mucus.

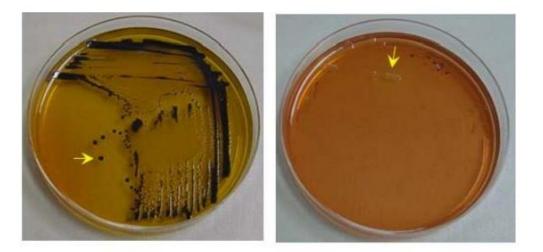
3- May lead to severe dehydration and sometime death.

Laboratory identification

Organisms can be cultured from stools using differential, selective Hektoen agar, s-s agar

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Shigella



Salmonella

Treatment:

- 1- Antibiotics (Ciprofloxacin or azithromycin)
- 2- Water and food supply and personal hygiene. nic Bacte
- 3-Vaccination.

Pseudomonas

The genus Pseudomonas comprises more than 140 species but only one of these is pathogenic to man .i.e Pseudomonas pyocynea (Ps. aeruginosa). P aeruginosa is widely distributed in nature and is commonly present in moist environments in hospitals. It can colonize normal humans, in whom it is a saprophyte. It causes disease in humans with abnormal host defenses, especially in individuals with neutropenia.

Pseudomonas aeruginosa

causes infections (e.g., sepsis, pneumonia, and urinary tract infections) primarily in patients with lowered host defenses. It also causes chronic lower respiratory tract infections in patients with cystic fibrosis, wound infections (cellulitis) in burn patients and malignant otitis externa in diabetic patients. It is the most common cause of ventilator associated pneumonia.

Morphology and Identification

A. Typical Organisms

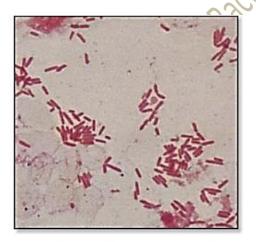
P. aeruginosa is motile and rod shaped, measuring about $0.6 \times 2 \mu m$ It is Gram-negative and occurs as single bacteria, in pairs, and occasionally in short chains.

B. Culture

P aeruginosa is an obligate aerobe that grows readily on many types of culture media, sometimes producing a sweet or grape-like or corn taco– like odor. Some strains hemolysis blood. P aeruginosa forms smooth round colonies with a fluorescent greenish color. It often produces the nonfluorescent bluish pigment pyocyanin, which diffuses into the agar. Other Pseudomonas species do not produce pyocyanin. Many strains of P.aeruginosa also produce the fluorescent pigment pyoverdin, which gives a greenish color to the agar. Some strains produce the dark red pigment pyorubin or the black pigment pyomelanin. P aeruginosa in a culture can produce multiple colony types . P aeruginosa from different colony types may also have different biochemical and enzymatic activities and different antimicrobial susceptibility patterns

C. Growth Characteristics

P. aeruginosa grows well at 37–42°C; its growth at 42°C helps differentiate it from other Pseudomonas species that produce fluorescent pigments. It is oxidase positive. It does not ferment carbohydrates, but many strains oxidize glucose. Identification is usually based on colonial morphology, oxidase positivity, the presence of characteristic pigments, and growth at 42°C. Differentiation of P aeruginosa from other pseudomonads on the basis of biochemical activity requires.



Pseudomonas aeruginosa (Gramnegative bacilli)



P. aeruginosa on MacConkey agar



Pseudomonas aeruginosa on Cetrimide agar

Antigenic Structure and Toxins

Pilli (fimbriae) extend from the cell surface and promote attachment to host epithelial cells. An exopolysaccharide, alginate, is responsible for the mucoid colonies seen in cultures from patients with CF. Lipopolysaccharide, which exists in multiple immunotypes, is responsible for many of the endotoxic properties of the organism. P aeruginosa can be typed by lipopolysaccharide immunotype and by pyocin (bacteriocin) susceptibility. Most P aeruginosa isolates from clinical infections produce extracellular enzymes, including elastases, proteases, and two hemolysins (a heat-labile phospholipase and a heat-stable glycolipid). Many strains of P aeruginosa produce exotoxin A, which causes tissue necrosis

Diagnostic Laboratory Tests

A. Specimens

Specimens from skin lesions, pus, urine, blood, spinal fluid, sputum, and other materialshould be obtained as indicated by the type of infection.

B. Culture

Specimens are plated on blood agar and the differential media (MacConkey,s or EMB agar). It is oxidese - positive . A typical metallic sheen of the growth on TSI agar coupled with the blue green pigment on ordinary nutrient agar .P aeruginosa does not ferment lactose and is easily differentiated from the lactose fermenting bacteria. 38 Culture is the specific test for diagnosis of P aeruginosa infection. The diagnosis is confirmed by biochemical reactions.

Epidemiology and Control

P. aeruginosa is found chiefly in soil and water, although approximately 10% of people carry it in the normal flora of the colon. It is found on the skin in moist areas and can colonize the upper respiratory tract of hospitalized patients. Its ability to grow in simple aqueous solutions has resulted in contamination of respiratory therapy and anesthesia equipment, intravenous fluids, and even distilled water. P. aeruginosa is primarily an opportunistic pathogen that causes infections in hospitalized patients (e.g., those with extensive burns), in whom the skin host defenses are destroyed; in those with chronic respiratory disease (e.g., cystic fibrosis), in whom the normal clearance mechanisms are impaired; in those who are immunosuppressed; in those with neutrophil counts of less than 500/mL; and in those with indwelling catheters. It causes 10% to 20% of hospital-acquired infections and, in many hospitals, is the most common cause of gram negative nosocomial pneumonia, especially ventilator-associated pneumonia.

Pathogenesis

Is based on multiple virulence factors: endotoxin, exotoxins, and enzymes. Its endotoxin, like that of other gram-negative bacteria, causes the symptoms of sepsis and septic shock. The best known of the exotoxins is exotoxin A, which causes tissue necrosis. It also produces enzymes, such as elastase and proteases, that are histotoxic and facilitate invasion of the organism into the blood stream. Pyocyanin damages the cilia and mucosal cells of the respiratory tract.

Clinical Findings

P. aeruginosa can cause infections virtually anywhere in the body,

1-urinary tract infections,

2-pneumonia (especially in cystic fibrosis patients),

3-wound infections (especially burns) predominate.

4-It is an important cause of hospital acquired pneumonia

5-External otitis (malignant otitis externa), otitis media

6-skin lesions