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 - 2025-2026

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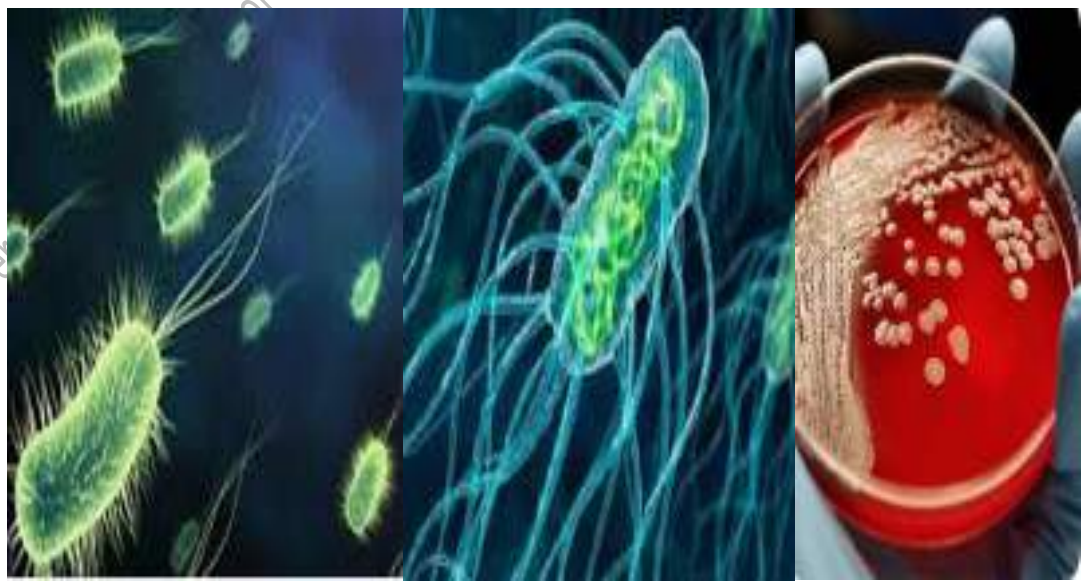


Gastrointestinal gram negative rods

These G- rods belong to diverse taxonomic groups, are facultative organisms constitute only a fraction of the total microbial flora of the GIT. They contain LPS which is both antigenic and an important virulence factor (endotoxin). Different enteric G- rods cause diseases within GIT , outside the GIT or in both location .

- *Escherichia coli :*

E. coli is part of the normal flora of the colon in human and other animals , but can be pathogenic both within and outside of the GIT . Has fimbriae or pili that are important for adherence to host mucosal surface .

-Structural and physiology :

E. coli share many properties with the other enterobacteriaceae , they are all facultative anaerobes . They all ferment glucose , and they all can generate energy by aerobic or anaerobic respiratory , they are oxidase negative .

-Clinical significance : Intestinal disease

Transmission of Intestinal disease is commonly by fecal – oral route , food and water serving as vehicles for transmission . At least five types of intestinal infections that differ in pathogenic mechanisms have been identified

1- Enterotoxigenic E. coli (ETEC): Is a common cause of travelers diarrhea colonize the small intestine mucosa (pili facilitate the binding of the organism to the mucosa). ETEC has

a- Enterotoxin include : a heat – stable toxin (ST)and heat- labile toxin (LT)it's like cholera toxin.

b-Prolonged hyper secretion of chloride ions and water by intestinal mucosal cells , while inhibiting the reabsorption of sodium.

2- Enteropathogenic E. coli (EPEC) : Are important cause of diarrhea in infants . The EPEC attach to mucosal cells in the small intestine by use of bundle – forming pili (BFpA). In addition to destruction of microvilli , are caused by injection of effector proteins in to the host cell by way of a type III secretion system .EPEC are not invasive and , thus do not cause bloody diarrhea .

3- Enterohemorrahagic E. coli (EHEC): Bind to the cells of large intestine , similar to EPEC produce characteristic lesions called attaching and affecting lesions (A/E). EHEC produce one of two exotoxin (shiga-like toxin 1 or 2) , resulting in sever form of copious , bloody diarrhea (hemorrhagic colitis)in the absence of mucosal invasion or inflammation . serotype O157:H7 is the most common strain of E. coli that produce shigalike toxin . The primary reservoir of this

bacteria is cattle .Therefore the possibility of infection can be greatly decreased by thoroughly cooking ground beef and pasteurizing milk .

4- Enteroinvasive E. coli (EIEC): cause a dysentery like syndrome with fever and bloody stools. Plasmid encoded virulence factors are nearly identical to those of shigella species in addition EIEC strains produce a hemolysin (HlyA).

5- Enteroaggregative E. coli (EAEC): Is also cause Traveler's diarrhea and persistence diarrhea in young children . Adherence to small intestine is mediated by aggregative adherence .EAEC produce a heat – stable toxin that is plasmid encoded .

-Clinical significance:

Extraintestinal disease The source of infection is frequently the patient own flora

1-Urinary tract infection (UTI)

2- Neonatal meningitis

3- Nosocomial (hospital- acquired) infections these include sepsis / bacteremia – endotoxic shock , and pneumonia .

4. Sepsis

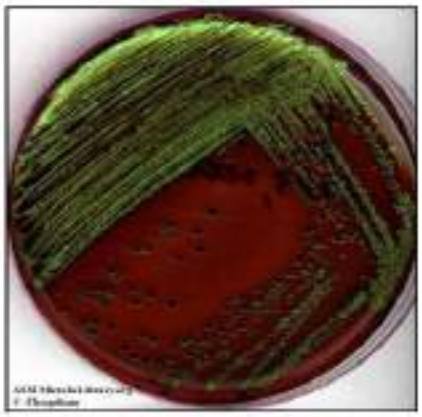
When normal host defenses are inadequate, E coli may reach the bloodstream and cause sepsis. Newborns may be highly susceptible to E coli sepsis because they lack IgM antibodies. Sepsis may occur secondary to urinary tract infection and often the major clone associated with invasion is E coli O25b/ST131.

5. Meningitis : E.coli and group B streptococci are the leading causes of meningitis in infants. Approximately 80% of E coli from meningitis cases have the K1 antigen.

-Lab. identification :

1- Culturing the sample on macConkey and macConkey sorbitol agars.

2-Molecular techniques such as PCR.



E. coli on Eosin methylene blue agar (EMB) (Metallic green sheen)

E. coli on MacConkey agar (Lactose fermenter)

Prevention

1-Its prevented by care in selection , preparation and consumption of food and water .

2-Maintance of fluid and electrolytes balance is of primary importance in treatment .

3-Suitable antibiotics (According to antibiotic sensitivity).

Vibrio

Vibrios are curved, Gram-negative rods commonly found in saltwater. Cells may be linked end to end, forming S shapes and spirals. They are highly motile with a single polar flagellum, non–spore-forming, and oxidase-positive, and they can grow under aerobic or anaerobic conditions. The cell envelope structure is similar to that of other Gram-negative bacteria. Vibrio cholerae is the prototype cause of a water-loss diarrhea called cholera

Growth And Structure

Vibrio cholerae has a low tolerance for acid, but grows under alkaline (pH 8.0-9.5) conditions that inhibit many other Gram-negative bacteria. It is distinguished from other vibrios by biochemical reactions, lipopolysaccharide (LPS) O antigenic structure, and productionof cholera toxin (CT). There are over 200 O antigen serotypes, only two of which (O1 and O139) cause cholera. Vibrio cholerae biogroup El Tor, an O1 variant, is a biotype of the classic strain. The O139 strains phenotypically resemble O1 El Tor strains but also produce a polysaccharide capsule. Vibrio cholerae possess long filamentous pili that form bundles on the bacterial surface and belong to a family of pili whose chemical structure is similar to those of the gonococcus and a number of other bacterial pathogens. All strains capable of causing cholera produce a colonizing factor known as the toxin-coregulated pilus (TCP) because its expression is regulated together with CT. In aquatic environments V cholerae produces polysaccharide biofilms, which contain carbohydrate moieties mediating cell–cell adhesion and attachment to surfaces.

MANIFESTATIONS

Typical cholera has a rapid onset, beginning with abdominal fullness and discomfort, rushes of peristalsis, and loose stools. Vomiting may also occur. The stools quickly become watery, voluminous, almost odorless, and contain mucus flecks, giving it an appearance called rice-water stools. Neither white blood cells nor blood are in the stools, and the patient is afebrile. Clinical features of cholera result from the extensive fluid loss and electrolyte imbalance, which can lead to extreme dehydration, hypotension, and death within hours if untreated. No other disease produces dehydration as rapidly as cholera.

DIAGNOSIS

The initial suspicion of cholera depends on recognition of the typical clinical features in an appropriate epidemiologic setting. A bacteriologic diagnosis is accomplished by isolation of V cholerae from the stool. The organism grows on common clinical laboratory media such as blood agar and MacConkey agar, but its isolation is enhanced by a selective medium (thiosulfate–citrate–bile salt–sucrose agar). Once isolated, the organism is readily identified by biochemical reactions. Outside cholera-endemic areas, the selective medium is not routinely used for stool cultures, so clinical laboratories must be alerted to the suspicion of cholera.

TREATMENT

The outcome of cholera depends on balancing the diarrheal fluid and ionic losses with adequate fluid and electrolyte replacement. This is accomplished by oral and/or intravenous administration of solutions of glucose with near physiologic concentrations of sodium and chloride and higher than physiologic concentrations of potassium and bicarbonate. Exact formulas are available as dried packets to which a given volume of water is added. Oral replacement, particularly if begun early, is sufficient for all but the most severe cases and has substantially reduced the mortality from cholera. Antimicrobial therapy plays a secondary role to fluid replacement by shortening the duration of diarrhea and magnitude of fluid loss. A single dose of azithromycin provides optimal antimicrobial therapy, but doxycycline, a fluoroquinolone, or trimethoprim-sulfamethoxazole are also effective agents.

PREVENTION

Epidemic cholera, a disease of poor sanitation, does not persist where treatment and disposal of human waste are adequate. Because good sanitary conditions do not exist in much of the world, secondary local measures such as boiling and chlorination of water during epidemics are required. Cholera associated with ingestion of crabs and shrimp can be prevented by adequate cooking (10 minutes) and avoidance of recontamination from containers and surfaces. Vaccines prepared from whole cells, lipopolysaccharide, and CT B subunit have been disappointing, providing protection that is not longlasting. Current interest includes live attenuated vaccine strains because of their potential to stimulate the local sIgA immune response.