Microbiology

Medical bacteriology

Objectives of *Listeria*

Listeria monocytogenes

Morphology and Identification

- Pathogenesis

Pathogenesis

- Pathology

- Clinical Findings

- Immunity

-Diagnostic Laboratory Tests

- Treatment

Listeria monocytogenes

- There are several species in the genus *Listeria*. Of these, *L monocytogenes* is important as a cause of a wide spectrum of disease in animals and humans.
- *L monocytogenes* is capable of growing and surviving over a wide range of environmental conditions. It can survive at refrigerator temperatures (4°C), under conditions of low pH and high salt conditions. Therefore, it is able to overcome food preservation and safety barriers, making it an important foodborne pathogen.
- *Listeria* had the ability to easily contaminate a variety of foods during any stage of the food handling process.

Morphology and Identification

- *L monocytogenes* is a short, **gram-positive**, non–spore-forming rod.
- It is catalase positive and has a tumbling end-over-end motility at 22–28°C but not at 37°C;

- the motility test rapidly differentiates *Listeria* from diphtheroids that are members of the normal microbiota of the skin.

Culture and Growth Characteristics

- *Listeria* grows well on media such as 5% sheep blood agar on which it exhibits the characteristic small zone of hemolysis around and under colonies.
- The organism is a facultative anaerobe and is catalase positive, esculin hydrolysis positive, and motile.
- Listeria produces acid but not gas from utilization of a variety of carbohydrates.
- The motility at room temperature and hemolysin production are primary findings that help differentiate *Listeria* from coryneform bacteria.

Antigenic Classification

- There are 13 known serovars based on O (somatic) and H (flagellar) antigens.
- Serotypes 1/2a, 1/2b, and 4b make up more than 95% of the isolates from humans.
- Serotype 4b causes most of the foodborne outbreaks.

Pathogenesis and Immunity

- *L monocytogenes* enters the body through the gastrointestinal tract after ingestion of contaminated foods such as cheese, fruit, or vegetables.
- The organism has several adhesin proteins (Ami, Fbp A, and flagellin proteins) that facilitate bacterial binding to the host cells and that contribute to virulence.
- It has cell wall surface proteins called internalins A and B that interact with E-cadherin, a receptor on epithelial cells, promoting phagocytosis into the epithelial cells. After phagocytosis, the bacterium is enclosed in a phagolysosome, where the low pH activates the bacterium to produce listeriolysin O. This enzyme, along with two phospholipases, lyses the

membrane of the phagolysosome and allows the listeriae to escape into the cytoplasm of the epithelial cell.

- The organisms proliferate, and ActA, another listerial surface protein, induces host cell actin polymerization, which propels them to the cell membrane.
- Pushing against the host cell membrane, they cause formation of elongated protrusions called filopods. These filopods are ingested by adjacent epithelial cells, macrophages, and hepatocytes, the listeriae are released, and the cycle begins again.
- *L monocytogenes* can move from cell to cell without being exposed to antibodies, complement, or polymorphonuclear cells.
- *Shigella flexneri* and rickettsiae also usurp the host cells' actin and contractile system to spread their infections.
- Iron is an important virulence factor.
- Listeriae produce siderophores and are able to obtain iron from transferrin.
- Immunity to *L monocytogenes* is primarily cell mediated, as demonstrated by the intracellular location of infection and by the marked association of infection with conditions of impaired cell-mediated immunity such as pregnancy, advanced age, AIDS, lymphoma, and organ transplantation. Immunity can be transferred by sensitized lymphocytes but not by antibodies.

Clinical Findings

- There are two forms of perinatal human listeriosis. Early onset syndrome (granulomatosis infantiseptica) is the result of infection in utero and is a disseminated form of the disease characterized by neonatal sepsis, pustular lesions, and granulomas containing *L monocytogenes* in multiple organs. Death may occur before or after delivery.

- The late-onset syndrome causes the development of meningitis between birth and the third week of life; it is often caused by serotype 4b and has a significant mortality rate.
- Healthy persons exposed to *L monocytogenes* in food may not become ill or may develop a mild, self-limiting febrile gastroenteritis lasting 1–3 days. This develops after an incubation period of 6–48 hours. Symptoms include fever, chills, headache, myalgias, abdominal pain, and diarrhea.
- Immunocompromised individuals can develop *Listeria* meningoencephalitis, bacteremia, and (rarely) focal infections.
- *Listeria* is one of the more common causes of meningitis in this group of patients.
- Clinical presentation of *Listeria* meningitis varies from insidious to fulminate and is nonspecific. Most clinical laboratories do not routinely culture for *Listeria* from routine stool samples. The diagnosis of systemic listeriosis rests on isolation of the organism in cultures of blood and spinal fluid.
- Spontaneous infection occurs in many domestic and wild animals. In ruminants (eg, sheep), *Listeria* may cause meningoencephalitis with or without bacteremia. In smaller animals (eg, rabbits, chickens), there is septicemia with focal abscesses in the liver and heart muscle and marked monocytosis.
- Many antimicrobial drugs inhibit *Listeria* species in vitro. Clinical cures have been obtained with ampicillin, erythromycin, or intravenous trimethoprim–sulfamethoxazole.
- Cephalosporins and fluoroquinolones are not active against *L monocytogenes*. Ampicillin plus gentamicin is often recommended for therapy, but gentamicin does not enter host cells and may not help treat the *Listeria* infection. Trimethoprim–sulfamethoxazole is the drug of choice

for central nervous system infections in patients who are allergic to penicillin.