REVIEW ARTICLE

Listeriosis: A Farm-to-Table Foodborne Disease

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Abstract

Foodborne diseases have emerged as a global public health concern. *Listeria monocytogenes* is a foodborne bacterium that causes listeriosis in animals and humans. Listeriosis is mainly contracted through the consumption of contaminated food. This significantly impacts newborns, pregnant women, and immunosuppressed and elderly individuals, putting them at higher risk of severe illness or death. Listeriosis symptoms can vary among patients depending on the part of the infected body. Diarrhea and fever are the common symptoms of listeriosis, but it is rarely diagnosed. Several *L. monocytogenes* outbreaks have been associated with contaminated unpasteurized cheese and fresh raw milk consumption. *L. monocytogenes* environmental presence is ubiquitous in grazing pastures, silage, farmyards, haylage, water, and crop fields. Wild animal feces are essential pathogen vectors of farm animal infections. Milking lines are also prone to bacterial contamination. This review elaborates on the virulence factors, pathogenesis, and the infection cycle of *L. monocytogenes*. Besides, we will display the various sources of *L. monocytogenes* contaminations in farm ruminants and transmission vectors and analyze applicable therapeutic and control measures at different stages.

Keywords

L. monocytogenes, Farm animals, Foodborne, Humans, Listeriosis

INTRODUCTION

Foodborne diseases (FBD) have emerged as a global public health concern (Vallejo et al., 2022; Pires et al., 2021). WHO defines FBD as a "disease of infectious or toxic nature caused by the consumption of food or water" (O'Shea et al., 2019). The average incidence rate of listeriosis caused by Listeria monocytogenes (*L. monocytogenes*) is only 0.3/100,000 individuals with over 2,300 annual cases. However, 92% of cases require hospitalization, with a death 16% toll (CDC, 2022; Scallan et al., 2011).

The bacterium *L. monocytogenes* is a well-known listeriosis pathogen of animals and humans. The main transmission route in humans is through consuming contaminated food. Most at-risk groups are newborns, pregnant women, and individuals with weakened immune systems. Listeriosis symptoms can vary among patients depending on the part of the infected body. Diarrhea and fever are the common symptoms of listeriosis, but it is rarely diagnosed (CDC, 2022). Invasive listeriosis is more common, where the bacterium reaches beyond the gut. The symptoms can also vary during pregnancy, and the patient can experience flu-like symptoms. However, the infection could lead to complications such as premature delivery, miscarriage, stillbirth, and fatal disease of the newborn. Other invasive listeriosis patients could feel headaches, loss of balance, stiff neck, convulsions, confusion, muscle aches, and fever. The symptoms generally appear after 1–4 weeks of infection (CDC, 2022).

Several *L. monocytogenes* outbreaks have been associated with contaminated unpasteurized cheese and fresh raw milk consumption. Therefore, good farm hygiene is necessary to avoid milk contamination. *L. monocytogenes* environmental presence is ubiquitous in grazing pastures, silage, farmyards, haylage, water, and crop fields (Rodriguez et al., 2021). Wild animal feces are essential pathogen vectors of farm animal infections and contamination of soils, animal bedding, and feed bunk tanks, especially during indoor months. Milking lines are also prone to bacterial infections. This review elaborates on various sources of *L. monocytogenes* contaminations in farm ruminants, identifies transmission vectors, explores bacterial virulence, and analyzes applicable control measures at different stages.

LISTERIOSIS

Organism

L. monocytogenes is a rod-shaped, facultatively anaerobic, Gram-positive listeriosis-causing bacterium of humans and animals (Schoder et al., 2022). Its environmental presence is ubiquitous, and it flourishes as a saprophyte of decomposing plant material under optimal humidity and temperatures (Pizarro-Cerdá & Cossart, 2019). The genus Listeria belongs to the family Listeriaceae, and it currently contains 20 species that naturally inhabit plants, soil, and water (Rodriguez et al., 2021; Parte et al., 2020). The antigens-based (flagellar & somatic) serotype classification of *L. monocytogenes* is categorized into 3 major well-conserved evolutionary divisions. Currently, 14 serotypes are reported, with serotype 4b (lineage I) being more virulent than other serotypes. Together with serotypes 1/2a, 1/2b, and 1/2c, these serotypes are largely associated with human cases of listeriosis (Muchaamba et al., 2022).

Source of Foodborne Infection

L. monocytogenes-associated listeriosis is a severe foodborne disease. Listeriosis is relatively rare (0.1 to 10 cases/1 million individuals/year) in various regions of the world. However, a high mortality rate is associated with listeriosis, which makes it a serious public health concern (Osek et al., 2022). Unlike other foodborne bacteria, *L. monocytogenes* can survive and grow at low temperatures in refrigerators. Infection mainly occurs through the consumption of *L. monocytogenes*-contaminated food. Listeriosis can also be transmitted between humans, particularly from pregnant mothers to unborn babies (Osek et al., 2022). *L. monocytogenes* is found in the animal digestive tract, soil, and water. The contaminated soil and use of infected manure as fertilizer could further pollute the vegetables. Ready-to-eat (RTE) food could be contaminated during processing, and bacteria can rapidly flourish during storage and distribution (Rodriguez et al., 2021). Listeriosis-associated food mainly includes (a) refrigerated long shelf-life products; and (b) products that are consumed raw or without cooking. Ready-to-eat meat products, dairy products, prepared salads, and fresh fruits and vegetables are known sources of previous listeriosis outbreaks (Buchanan et al., 2017).

Listeriosis is of two types: invasive and non-invasive. Noninvasive listeriosis is a mild form with a short incubation period of a few days, and it mainly affects healthy individuals. Fever, muscle pain, headache, and diarrhea are common symptoms of this infection. The ingestion of foods highly infected with *L. monocytogenes* has caused outbreaks of this disease (Osek et al., 2022). Invasive listeriosis is severe and affects high-risk groups, including pregnant women, infants, elderly individuals, and patients with AIDS, organ transplants, and cancer. Symptoms (meningitis, septicemia, muscle pain, and fever) of this disease are severe, with a higher mortality rate (20%–30%). The incubation period of invasive listeriosis is generally 1-2 weeks but could vary from a few days up to 90 days (Osek et al., 2022). Pregnant women are 20 times more prone to listeriosis than healthy adults. Listeriosis in pregnant women can lead to stillbirth or miscarriage, whereas newborn infections are characterized by meningitis, septicemia, and low birth weight. (Jeffs et al., 2020).

L. Monocytogenes Virulence Factors

L. monocytogenes have various human and animal-associated virulence factors (Table 1). *L. monocytogenes* contains different soluble and surface proteins, which facilitate target cell adhesion, intracellular multiplication, internalization, and dissemination to other hosts (Quereda et al., 2021). The encoding of virulence factors occurs either as clusters on pathogenicity islands or as separate loci across the bacterial genome (Disson et al., 2021). The genomes of all the *L. monocytogenes* strains contain conserved core virulence-related genes (*mpl*, *hly*, *prfA*, *actA*, *plcB,* and *plcA*) (Disson et al., 2021). Moreover, several other separate loci-encoded virulence factors (internalin A/Internalin B (*inlAB*) operon) are also conserved in all *L. monocytogenes* strains (de las Heras et al., 2011).

Listeria adhesion protein (LAP)

LAP (104-kDa) is a cell wall protein that is ubiquitous to all *Listeria* species (Jagadeesan et al., 2011). Pandiripally et al. (1999) reported it for the first time as protein p104, which was later identified as alcohol acetaldehyde dehydrogenase (Burkholder and Bhunia, 2010). LAP essential enzyme is primarily produced as a cytosolic protein in all *Listeria* species. LAP epithelial receptor is a constitutively expressing mitochondrial heat shock protein 60 (Hsp60) that facilitates the pathogenic translocation across the intestinal epithelium (Drolia et al., 2018; Jagadeesan et al., 2011).

Fibronectin binding protein (FbpA)

Fibronectin binding proteins (Fbp) are cell wall-anchored proteins that are common in Gram-positive bacteria (Hymes and Klaenhammer, 2016). Fbp identifies and binds to human extracellular matrix component fibronectin for further inter-cellular interactions and adhesion (Henderson et al., 2011). A three-component bridge is formed through Fbps and fibronectin molecules interaction that helps bacterial cell adhesion with the host cells (Hymes and Klaenhammer, 2016; Dramsi et al. (2004).

Internalin A (InlA)

InlA (80 kDa protein) is a principal virulence factor of *L. monocytogenes*, as first reported by Gaillard et al. (1991). It anchors to the cell wall peptidoglycan via the C-terminal LPXTG motif (Ireton et al., 2021). InlA mediates pathogen adhesion and internalization into

Table 1. *L. monocytogenes* virulence factors (Source: Disson et al., 2021).

Gene	Protein	Function	Role in Virulence
hly	β-hemolysin (Lysteriolysin)	Pore-forming cytolysin, secreted protein	Escape from phagocytic vacuoles
actA	ActA	Actin polymerization, surface protein	Cell-to-cell spread
plcA	Phospholipase C (PI-PLC)	Phosphatidyl-inositol-specific, secreted protein	Accessory factor in vacuole escape
plcB	Phospholipase C (PC-PLC)	Phosphatidyl-choline-specific secreted protein	Accessory factor in vacuole escape
mpl	Metalloprotease	Protease	Accessory factor in vacuole escape
inlA	Internalin A	Surface protein	Attachment invasion
inlB	Internalin B	Surface protein	Attachment invasion
iap	Invasion-associated protein Protein p60	Secreted/surface protein	Invasion factor
prfA	Positive regulatory factor A	Cytoplasmic protein	Regulates transcription of plcA, hly, mpl, actA, and plcB

enterocytes for the invasion of the intestinal barrier (Drolia and Bhunia, 2019).

Internalin B (InlB)

InlB is also an adhesion protein that facilitates *L. monocytogenes* binding to enterocytes followed by intestinal barrier invasion (Ireton et al., 2021). InlB uses glycine and tryptophan (GW) modules, which non-covalently interact with cell wall teichoic acids to anchor on it (Braun et al., 1997). The LRR domain serves as the recognition and binding site for a tyrosine kinase host receptor known as mesenchymal-epithelial transition factor (Met) (Drolia and Bhunia, 2019). *L. monocytogenes* produces several LRR proteins belonging to the internalin family (Bierne et al., 2007).

Listeriolysin O (LLO)

LLO (56 kDa), belonging to the cholesterol-dependent cytolysins (CDCs) family, is a pore-forming *hly* gene-encoded cytotoxin (Hamon et al., 2012; Milohanic et al., 2003). LLO is among the initially identified virulence factors of *L. monocytogenes* involving blood agar hemolysis (Hof, 1984). Later studies reported hemolysin as a sulfhydryl-activated toxin associated with *L. monocytogenes* intracellular growth in human enterocyte-like Caco-2 cells (Geoffroy et al., 1987; Kuhn et al., 1988). LLO mainly participates in internalization and vacuole lysis, leading to the pathogenic release into host cell cytosol (Phelps et al., 2018).

Actin-polymerizing protein ActA

ActA is an *actA* gene-encoded surface protein, which mediates actin polymerization-based bacterial motility inside the cells of an infected host (Suárez et al., 2001). The protein anchors on the bacterial cell membrane through the hydrophobic C-terminal domain, whereas the functional N-terminal domain is exposed to the cytoplasm of the host cell (Suárez et al., 2001). Intracellular motility is facilitated through ActA-based filament formation and actin nucleation (Skoble et al., 2001).

Phospholipases

L. monocytogenes contains two types of phospholipases [*plcA* gene-encoded Phosphatidylinositol-specific phospholipase C (PI-PLC) and *plcB* gene-encoded phosphatidylcholine phospholipase C (PC-PLC)] (Smith et al., 1995; Camilli et al., 1991). The combination of PI-PLC and LLO participates in primary and secondary vacuole lysis followed by pathogen internalization (Pizarro-Cerdá et al., 2012). It catalyzes membrane phosphatidylinositol cleavage into diacylglycerol and inositol phosphate (Poussin and Goldfine, 2005). PC-PLC, a broad-range phospholipase, is necessarily needed for the lysis of primary vacuole under LLO deficiency and double-membrane secondary vacuole (Gründling et al., 2003; Coffey et al., 2000).

COLONIZATION OF GASTROINTESTINAL TRACT HOST CELL

L. monocytogenes is one of several human gastroenteritis-associated bacteria (Halbedel et al., 2019). The pathogen causes self-limited and acute febrile gastroenteritis in healthy individuals (Halbedel et al., 2019). Invasive listeriosis is focused on because of the high severity and fatality rates. However, outbreaks of non-invasive listeria febrile gastroenteritis are also commonly reported (Maurella et al., 2018; Jacks et al., 2016; Ooi and Lorber, 2005; Sim et al., 2002). Non-invasive listeriosis is characterized by fever, nausea, vomiting, and non-bloody diarrhea within 24 h of taking contaminated food (Halbedel et al., 2019; Ooi and Lorber, 2005). Non-invasive listeriosis-related pathogenesis mechanisms remain unclear (Halbedel et al., 2019). A few recent studies have elucidated the colonization mechanisms of *L. monocytogenes* in the gastrointestinal tract (Halbedel et al., 2019; Travier et al., 2013). Travier et al. (2013) used *actA* gene mutants in orally infected mice and noticed actA-mediated *L. monocytogenes* aggregation in the gut lumen. ActA-mediated aggregation mechanism is considered to engage C-terminal regions (not participating in polymerization) to direct ActA-ActA interactions (Travier et al., 2013). They also noticed that ActA-dependent aggregation enhanced the ability of *L. monocytogenes* to persist in mice's colon lumen and cecum. Halbedel et al. (2019) also noted a genetic correlation between listeriosis (non-invasive or invasive) and the absence/presence of a functional chitinase gene (*chiB*).

InlA-mediated Transcytosis

This is the primary route of *L. monocytogenes* intestinal cell invasion. The cell wall anchored protein (InlA) facilitates the *L. monocytogenes* uptake into non-phagocytic cells via receptor-mediated endocytosis (Radoshevich and Cossart, 2018). InlA enhances pathogenic adhesion and intestinal epithelium

invasion by interacting with its E-cadherin receptor (a component of adherens junctions) (Pizarro-Cerdá et al., 2012). InlA and receptor interaction occur at sites of E-cadherin transient exposure to the intestinal lumen (Nikitas et al., 2011; Pentecost et al., 2006). InlA binding initiates the acquisition of other junctional proteins (β-catenin, α-catenin, p120 catenin, and actin), leading to E-cadherin clustering at the bacterial entry site (Bonazzi et al., 2009). Then, a post-translational E-cadherin modification results in endocytosis through clathrin or caveolin (Drolia and Bhunia, 2019; Bonazzi et al., 2009). Finally, InlA/E-cadherin-mediated endocytosis involves host cytoskeleton components for host cell membrane protrusions, which form endocytic vesicles around adhering bacterial cells (Pizarro-Cerdá et al., 2012; Ireton et al., 2021; Saila et al., 2020).

Contrarily, InlB does not participate in intestinal cell invasion (Ireton et al., 2021). However, it contributes to other tissue invasions (placenta, liver, CNS, and spleen) along with InlA (Tang and Sails, 2014). InlB receptor is a ubiquitous tyrosine kinase Met, whereas HGF (Hepatocyte Growth Factor) is its normal ligand (Pizarro-Cerdá et al., 2012). InlB binding to Met leads to Met proteins' cytoplasmic tail autophosphorylation for initiating a reaction cascade, which culminates in the localized actin polymerization and bacterial cell internalization similar to InlA (Radoshevich and Cossart, 2018).

LAP-mediated Translocation

The pathway mediated by inlA is a recognized way that *L. monocytogenes* uses to pass through the intestine epithelium (Nikitas et al., 2011; Bonazzi et al., 2009; Pentecost et al., 2006). Subsequently, it was observed that strains lacking functional InlA could cause infections in guinea pigs and mice that were given the bacteria orally. These findings reveal that *L. monocytogenes* could also use other mechanisms for intestinal invasion (Drolia et al., 2018). The adhesive role of LAP surface protein was initially detected for *L. monocytogenes* binding to enterocytes, but it is also known to translocate the pathogens across intestinal epithelium (Drolia et al., 2018). Drolia et al. (2018) employed a mouse model and Caco-2 cell line to elucidate the LAP-mediated invasion pathway. They reported that LAP induces intestinal epithelial barrier dysfunction to enhance bacterial translocation. LAP binding to luminal receptor protein Hsp60 activates MLCK (myosin light-chain kinase), which opens the intestinal barrier by redistributing E-cadherin, occludin, and junctional proteins (Drolia et al., 2018). These reactions further open the tight junctions between neighboring enterocytes for *L. monocytogenes* translocation (Drolia and Bhunia, 2019; Drolia et al., 2018).

M-cell Mediated Transcytosis

The specialized microfold (M) epithelial cells exert a mucosal immune response by searching for antigens in the intestinal mucosa. M-cells rapidly uptake the antigens from the intestinal mucosa to transcytose them through the intestinal epithelium into Peyer patches' lymphoid tissues (Hase et al., 2009). This is also a passive route of pathogen transcytosis into follicle-associated epithelium's basolateral side (Rey et al., 2020). M-cells-oriented *L. monocytogenes* transcytosis is well known. However, the mechanism of pathogenic interaction with M-cells requires further elucidation (Rey et al., 2020). Orally infected and *in vitro* mice models have revealed that *L. monocytogenes* could rapidly aggregate in Peyer patches in the absence of InlA (Corr et al., 2006; Marco et al., 1997). Currently, transcytosis across M cells is supposed to occur through a vacuole (Drolia and Bhunia, 2019; Tang and Sails, 2014).

INTRACELLULAR SURVIVAL AND DISSEMINATION

L. monocytogenes is an efficient intracellular organism, which flourishes rapidly inside cells (Charlier et al., 2020). Neonates, immunosuppressed patients, pregnant women, and elderly individuals are susceptible to *L. monocytogenes*-related bacteremia. *L. monocytogenes* mainly enter the bloodstream by passing through the intestinal barrier (Charlier et al., 2020). Due to its preference for the CNS (Central nervous system) and placenta in pregnant women, neurolisteriosis, septicemia, and maternofetal infection are the major outcomes of invasive listeriosis (Charlier et al., 2020). The reasons for *L. monocytogenes* high tropism toward these tissues remain unclear. However, the presence of InlB-related Met and InlA-related E-cadherin might contribute to this phenomenon (Disson and Lecuit, 2012). *L. monocytogenes* follows an InlB-dependent mechanism to invade the human placenta in the presence of Met in HUVEC (human umbilical vein endothelial cells) (Disson et al., 2008). Both InlB and InlA mechanisms facilitate CNS invasion (Disson and Lecuit, 2012).

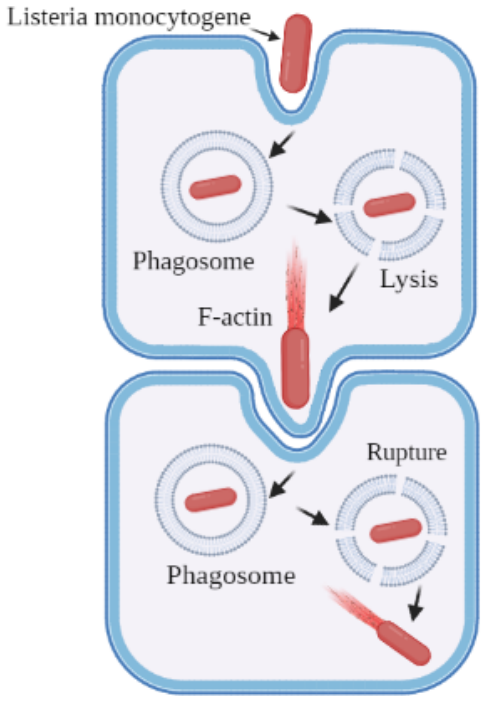

Figure 1. Intracellular survival and dissemination mechanism of *L. monocytogenes*.

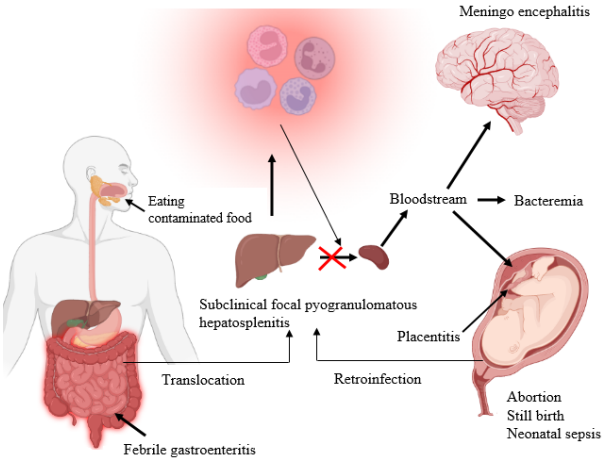

Figure 2. Illustrative depiction of the infection process of *L. monocytogenes* in humans. The consumption of tainted food precedes the invasion of the intestinal barrier by the bacteria, allowing entry into the bloodstream. Subsequently, the bacteria travel via the portal circulation to the spleen and liver, replicating before spreading throughout the bloodstream. Finally, bacteria infect the brain and placenta/fetus in pregnant women. Schem (https://smart.servier.com; accessed on 1 March 2022) is licensed under a Creative Commons Attribution 3.0 Updated License.

During the infection cycle, the internalization is followed by the LLO-mediated escape from the primary vacuole to enter the cell cytosol (Kortebi et al., 2017; Pizarro-Cerdá and Cossart, 2007). The cholesterol-dependent pore-forming LLO cytotoxin ruptures the vacuole to release bacteria into the host cell cytosol (Schnupf and Portnoy, 2007). Moreover, *L. monocytogenes* also involves phospholipases (PI-PLC) to promote primary vacuole lysis (Smith et al., 1995). After the intracellular replication in the infected cells, ActA production leads to actin comet tails formation, which enhances bacterial motility in the cells and facilitates membrane protrusions for further spread to uninfected cells (Cossart and Lebreton, 2014) (Figure 1).

INFECTION CYCLE

Multiple steps of the *L. monocytogenes* infection cycle involve several virulence factors. After entering the human cell, bacteria cross the intestinal epithelial barrier and translocate to mesenteric lymph nodes to reach the primary target organs (spleen and liver). Infectious foci are established in the target organs, which are efficiently removed in immunocompetent individuals through cellular immunity, and the infection mostly remains subclinical. However, high infective doses might also cause febrile gastroenteritis or invasive disease in such individuals (rare cases). On the other hand, in immunocompromised and elderly individuals who lack a strong immune response mediated by T-cells, the initial sites of infection are not effectively cleared. This leads to the bacteria being released into the bloodstream. This condition leads to febrile bacteremia followed by an invasive brain infection. *L. monocytogenes* also colonizes pregnant women's uterus, spleen, and liver. The infection is controlled in the spleen and liver, whereas immune tolerance mechanisms of the placenta allow *L. monocytogenes* proliferation. Bacteria released from the placental reservoir into the bloodstream could re-infect the spleen and liver for the persistence and amplification of the infection (Domínguez et al., 2023). Transplacental *L. monocytogenes* dissemination to the fetus could cause neonatal sepsis, abortion, and stillbirth. Later on, neonates could also face congenital forms accompanied by septicemia, meningitis, and encephalitis (Figure 2).

LISTERIOSIS IN ANIMAL FARMS

L. monocytogenes is commonly detected in animal products, but its foodborne outbreaks are mainly associated with raw, unpasteurized milk and dairy products (Shamloo et al., 2019). Dairy cattle farms can harbor *L. monocytogenes* genotypes that are associated with human listeriosis outbreaks (Castro et al., 2018). Animal bedding, feed bunks, and water troughs that come into direct contact with farm animals, along with animal feed, are considered significant carriers of listeriosis. Moreover, the presence of various *L. monocytogenes* serogroups in farms could be introduced by domestic or wild animals, contaminated machines or vehicles, and farm visitors (Castro et al., 2018). Therefore, specific sanitary measures are needed in the immediate space of farm animals to avoid bacterial infections (Youssef et al., 2021).

Animals can disseminate the bacterium through feces (Castro et al., 2018). The reservoir animals must be carefully supervised, as their fecal excretions could contaminate the bulk tank and raw milk. Soil contaminations can facilitate bacterial spread through farms and wild animals, which serve as vectors for further transmission. The access of birds to farm feed storage could also lead to the contamination of grains, straws, and cereals (Konosonoka et al., 2012). *L. monocytogenes* presence in milk has been linked to its presence in animal feces and directly correlates with poor animal housing (Rodriguez et al., 2021).

L. monocytogenes mainly causes uterine infections and encephalitis in ruminants. Uterine infections are associated with late-term abortions and neonate septicemia. Listeriosis-related encephalitis leads to neurological signs such as excessive salivation, unilateral facial paralysis, and circling (Dhama et al., 2015). Ruminants also suffer from *L. monocytogenes*-related keratitis and eye infections, which are linked to direct eye infections from *L. monocytogenes*-infected silage and feeds (Revold et al., 2015). *L. monocytogenes*-infected animals shed the bacteria through feces, whereas healthy animals could also be latent carriers of *L. monocytogenes* (Schoder et al., 2022). Several investigations have reported that 50% of animal fecal (poultry, cattle, pigs, sheep, and goats) samples without listeriosis clinical symptoms might contain *L. monocytogenes* (Schoder et al., 2022). Most of the animal listeriosis reports are associated with the ingestion of *L. monocytogenes* contaminated silage. However, all the cases are not feed-borne (Aydın et al., 2019). Therefore, livestock farms are also considered natural reservoirs of *L. monocytogenes*, which serve as the primary contamination source of food processing plants (Terentjeva et al., 2021). Crops and soil-contaminated partially fermented silage (pH > 5.0 to 5.5) allow *L. monocytogenes* amplification to high numbers. Therefore, silage-feeding represents the common infection route of farm animals (Terentjeva et al., 2021).

L. MONOCYTOGENES CONTROL AND PREVENTION

L. monocytogenes control is a prerequisite at all the stages of the food chain, which requires an integrated mechanism for the prevention of bacterial multiplication in final food products (Schoder et al., 2022). The ubiquitous nature, high resistance, and survivability of this bacterium in the refrigerator (around 5°C) challenge the common preventive measures, including the use of smoke, salt, and acidic conditions (Bondi et al., 2014). Good Manufacturing Practices (GMP) and Good Hygienic Practices (GHP) should be implemented in all food-related sectors. The implementation of an efficient HACCP (Hazard Analysis Critical Control Points) based food safety management system is necessary as well (Martín et al., 2022). Food manufacturers should also compare their HACCP-based mechanisms and hygiene measures against microbiological criteria to verify correct functioning. (Gupta and Adhikari, 2022).

Treatment

Multiple antibiotics are available for treating *L. monocytogenes* infection; however, clinicians prefer ampicillin alone or combined with gentamicin. Some patients may require alternative therapies because of allergies to certain antibiotics or a specific stage of the disease. The second-line agents in such cases include vancomycin, trimethoprim/sulfamethoxazole, fluoroquinolones, and erythromycin. Cephalosporins are ineffective against Listeria (Dos Reis et al., 2022; Young and Thomas, 2018).

Prevention

The cooking process and pasteurization can kill the food bacteria (*L. monocytogenes*). Listeriosis prevention guidelines are generally like other foodborne diseases, which mainly include safe food handling according to FAO and WHO (2022) recommendations. High-risk

individuals should: 1) avoid RTE and deli meat products (sausages, meat spreads, pates, and cold-smoked salmon and other seafood) and unpasteurized milk products; and 2) read the product label and carefully follow the storage temperatures and shelf-life period instructions.

Additionally, the health protection branch of FAO and WHO (2022), has recommended some suggestions to high-risk foodborne listeriosis groups, including: 1) avoid soft cheese consumption; 2) avoid consuming raw animal-based foods; 3) reheat precooked and leftover foods to a temperature of 74°C (165°F) in the center of the food; 4) thoroughly wash and scrub all store-bought and homegrown vegetables with lukewarm water before use; 5) cut and washed vegetables should not be refrigerated for more than 1-2 days; 6) Cooked refrigerated leftovers should be used at the earliest (within 1–2 days); and 7) do not buy foods with expired 'use by' or 'sell by' dates.

Conflict of Interest

The author declared that there is no conflict of interest that is related to this study and this article.

Disclosure

The author did not receive any form of commercial support, including compensation or financial assistance, for this case report. Additionally, the author has no financial interest in any of the products, devices, or drugs mentioned in this article.

Ethical Approval

The study was approved by the Ethics Committee of the KAUH in Jeddah, Kingdom of Saudi Arabia, also known as the Institutional Review Board of Hospitals.

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