Global Veterinaria 17 (1): 52-62, 2016
ISSN 1992-6197

DOI: 10.5829/idosi.gv.2016.17.01.1044

Listeriosis and Its Public Health Importance: A Review

Abdi Gezali, Feyissa Begna and Kula Jilo

School of Veterinary Medicine, College of Agriculture and
Veterinary Medicine Jimma University, Jimma, Ethiopia

Abstract: Listeriosis is a widespread zoonotic disease that becomes an important source of human food borne infections all over the world and also affects cattle herd, sheep and goat flocks. The aim of this manuscript was to review and compile the available literature on listeriosis in animals and its public health importance.There are 7 species of Listeria namely: *L. monocytogenes*, *L. innocua*, *L. welshimeri*, *L. seeligeri*, *L. ivanovii*, *L. murrayi* and *L. grayi*. But *L. monocytogenes* is the only species of Listeria that is pathogenic for both humans and animals. Listeriosis often being referred to as "Silage disease" since poor quality silage is a source for animal infection, but the most commonly considered risk foodstuffs for *L. monocytogenes* is milk and milk by-products for both animal and human. The major clinical forms of listeriosis in cattle are encephalitis, abortion, Mastitis, Ophthalmitis, iritis and keratoconjunctivitis. There are two main forms of illness in human associated with *L. monocytogenes* infection such as invasive (fatal form) and non-invasive forms (mild form). People at risk of invasive listeriosis include pregnant women and their fetuses, newborn babies, the elderly and immunocompromised individuals (such as cancer, transplant and HIV/AIDS patients). Every effort should be made to produce silage of good quality, with early cutting of grass, minimal contamination with soil or feces and ensuring optimal anaerobic fermentation, which will insure that the pH falls below 5.0; at that level, growth of *Listeria* species is inhibited. People susceptible for acquiring listeriosis should not consume unpasteurized milk and milk products.

Key words: *Listeria monocytogenes · Foodborne disease · Zoonotic · Listeriosis*

INTRODUCTION

Listeriosis is an infectious disease of man and animals with a world-wide distribution [1]. The disease primarily affect older adults, pregnant women, newborns and adults with weakened immune systems [2]. Listeriosis is recognized in 1920 for the first time as an infectious disease of rodents and other small animals. Murraz is the person who described a septic disease in rabbits at 1926, which showed peripheral monocytosis so because of this he named microorganism *Bacterium monocytogenes*. Although monocytosis is not a typical finding in human infections, species *monocytogenes* renamed to *Listerella* followed by Listeria, in honor of Lord Lister and the microorganism is now known as *Listeria monocytogenes*. *L. monocytogenes* is mostly responsible of human listeriosis but occasionally infection with *L. seeligeri* and *L. ivanovii* has been reported [2].

The first report of Listeriosis in cattle was by Jones and Little in 1934 where it was implicated in what is now known as "typical Listeriosis", namely meningoencephalitis. The literature on *Listeria* and Listeriosis has grown rapidly since that time [1].

Bovine listeriosis is a sporadic bacterial infection most commonly manifested by encephalitis or meningoencephalitis in adult animals associated with feeding poorly fermented silage during the winter months [3].

The first human case of listeriosis was reported by Nyfeldt in 1929 [1]. Today, listeriosis is regarded as a food-borne disease of serious public health concern due to the great mortality rate (20-30%) [2].

The bacteria have been recognized as causative agents of diseases in humans during World War II. The recognition of food-born listeriosis episodes caused by pre-packaged meat products, sausages, soft cheeses, fine, salads and other foods was recorded [4].

Corresponding Author: Kula Jilo, School of Veterinary Medicine, College of Agriculture and Veterinary Medicine, Jimma University, P.O. Box: 307, Jimma, Ethiopia.

Mortality is high without early antibiotic and supportive therapy. *Listeria monocytogenes* can affect many species and is a public health concern [5].The natural habitat of these bacteria is thought to be decomposing plant matter, in which they live as saprophytes. Domesticated ruminants probably play a key role in the maintenance of Listeria spp. in the rural environment via a continuous fecal-oral enrichment cycle [6]. In addition, it is available in soil, poor quality silage, sewage, slaughterhouse wastes and animal intestines. The intestinal mucosa is the main route of entry for septicemic/abortive listeriosis, after oral ingestion and its incubation period can be as short as 1 day. The oral mucosa is the main route for rhombencephalitis of *L. monocytogenes* which likely invades the brainstem via cranial nerves. The incubation period for meningoencephalitis significantly is longer than in the septicemic form, usually 2–3 weeks. The course of the disease is usually acute in sheep and goats for 1–4 days [7], although it can be more protracted in cattle. Diseased animals show unidirectional circling and display unilateral facial paralysis, but also there is difficulty in swallowing, fever, blindness and head pressings. Paralysis and death follow in 2 to 3 days. *L. monocytogenes* may localize in the placentas and cross over to amniotic fluid in pregnant animals. It also multiplies there and then ingested by the foetus which eventually causing foetal death and abortion. Abortion due to listeriosis is usually occurs in late gestation [5]. The objective of this paper was to review the available literature on listeriosis and its public health importance.

Literature Review

Etiology: According to Pal [8], listeriosis is a serious illness caused by eating food contaminated with the bacterium *Listeria*, which is a Gram-positive, psychotropic, facultative anaerobic, non sporulating, motile, small rod. It displays characteristic tumbling motility that is facilitated by the presence of peritrichous flagella. Motility is temperature dependent, showing high motility at 20-30°C when flagellar expression is maximum. According to Gebretsadik *et al.* [9] there are 7 species of *Listeria* as *L. monocytogenes, L. innocua, L. welshimeri, L. seeligeri, L. ivanovii, L. murrayi* and *L. grayi*. *L. monocytogenes* is the only species of listeria that is pathogenic for both humans and animals. *L. monocytogenes* causes miscarriages and reproductive disorders in ruminants and animals may carry species in their intestines without showing any symptoms [10]. *L. ivanovii* infection is rare in human infection [11, 12].

Listeria can be divided into 16 serovars on the basis of somatic and flagellar antigens and there is considerable genetic diversity between serovars [1]. *Listeria monocytogenes* has thirteen serotypes, but, only three serotypes; serotype 4b, 1/2a and 1/2b are responsible for the majority of veterinary and human listeriosis cases. Serotype 4b has been identified as the cause of most human listeriosis cases whereas serotypes 4a and 4c are most of the time limited to animals [13]. However, Bundrant *et al*. [14] indicated that the outbreak was caused by an unusual *L. monocytogenes* serotype 4b strain, which was classified into lineage III with clinical signs of listeriosis in cattle.

L. monocytogenes has three distinct lineages, with differing evolutionary histories and pathogenic potentials [15]. Lineage I strains contain the majority of human clinical isolates and all human epidemic clones, but are underrepresented in animal clinical isolates [15]. Lineage II strains are overrepresented in animal cases and underrepresented in human clinical cases and are more prevalent in environmental and food samples [16]. Lineage III isolates is very rare, but significantly more common in animal than human isolates [15].

Morphology and Growth Characteristics of Listeria: Listeria species are small, Gram-positive, non- acid fast, non-spore forming and non-capsulate coccobacilli measuring 0.5 to 2 mm x 0.4 to 0.5 mm. *Listeria* has typical Gram positive cell wall. They are facultative anaerobes that grow best under reduced oxygen and increased carbon dioxide concentration. Growth occurs at 4 to 45°C, with an optimum temperature of 30 to 37°C. Simple laboratory media support growth preferably at an alkaline or neutral pH. *Listeria* tolerates 0.04% potassium tellurite, 0.025% thallium acetate, 3.75% potassium thiocyanate, 10% NaCl and 40% bile in media. Most strains grow over a pH range of 5.5 to 9.6. It has greater heat tolerance than other non-spore forming bacteria; however, short-time high temperature pasteurization is effective for killing listeria [17].

Epidemiology

Occurrence and Distribution: Listeriosis is believed to be a sporadic disease, predominantly of ruminants and found worldwide. Although Listeriosis is of worldwide distribution it has been an important problem in North America, Europe, Britain, New Zealand and Australia [18, 19, 20]. In the northern hemisphere, Listeriosis has a distinct seasonal occurrence with the highest prevalence in the winter months (between December and May) [1].

Abortion is obviously associated with pregnancy, the majority of encephalitis cases occur in February and March [21, 22]. The reason for this seasonal distribution is obscure but the peak of infection is coincidental with late pregnancy when there are fundamental changes in the animals' immune status.

Cattle show a similar age incidence to sheep with the majority of cases affected two to three year-olds, although cattle appear much less susceptible to listeriosis than sheep [23].

The organism was found to survive for 13 years in milk, 16 years in a brain sample, 12 years in feces and 12 years in silage. It is reported to persist for 2 years in dry soil, 11.5 months in damp soil, 2 years in dry feces, 3 months in sheep feces, 16.5 months in cattle feces and up to 7 months on dry straw [19]. In humans, age and pregnancy-related defects in cell-mediated immunity have been described [24] which may explain the prevalence of listeric infections in neonates and during pregnancy. Similar mechanisms may be involved in listeric infections of ruminants.

Source of Infection: The organism is commonly isolated from animal feces, human feces, farm slurry, sewerage sludge, soil, farm water troughs, surface water, plants, animal feed and the walls, floors, drains etc of farms and other environments since *listeria* has ubiquitous nature in the environment [19, 25]. *L. monocytogenes* have the potential to be found in most feeds like hays, grains and formulated feeds but, its multiplication is restricted due to low level of available water in food. In ruminants, *L. monocytogenes* can be isolated from the feces and nasal secretions of healthy animals known as asymptomatic carriers of germs and therefore constitute an important reservoir of germs. The main source of *L. monocytogenes* in raw milk is mostly the gastrointestinal tract of animals and the environment, skin of the teats, in particular shedding of *Listeria* into milk due to mastitis [17].

L. monocytogenes is commonly available in the silage, but it doesn't multiply to any significant extent in effectively preserved silage which is characterized by anaerobic storage, high density, a high concentration of organic acids and a pH below 4.5. It may be present in silage which is poorly fermented. When silage contains soil its risk to contaminate by *Listeria* is higher than non-contaminated silage. Moist preserved feeds other than grass silage are at risk for *Listeria* growth. Infected animals can also serve as a source of infection from their urine, feces, aborted fetuses, uterine discharges and the milk [17].

Transmission: The primary mode of transmission for Listeria is through soil contamination and ingestion of contaminated feed. Calves which develop septicemic disease may acquire infection from contamination on the cow teat, from the ingestion of milk containing the organism or from cow with subclinical bacteremia, through the navel from the environment and also as a congenital infection [17]. The encephalitic form of the disease results from infection of the terminals of the trigeminal nerve consequent to abrasion of the buccal mucosa from feed or browse or from infection of tooth cavities. Spinal myelitis is believed to result from growth up to spinal nerves subsequent to body area infections [18, 19].

Risk Factors: A number of predisposing factors have been observed or proposed, as risk factors for the disease. These include factors that cause a lowering of the host animal's resistance and factors that increase the infection pressure of the organism. In farms the latter appears the most important as risk factor to the occurrence of outbreaks of listeriosis. There are three most common risk factors for listeriosis [17, 19].

Host Risk Factors: Regarding sensitivity, virtually all wild or domestic animals are susceptible to infection caused by *Listeria* [20]. Listeriosis in cattle may develop sporadically or enzootic, having a stationary character, without tendency to disseminate in the outbreak [17]. Morbidity may vary between 5-15%, sometimes 20-25% and mortality varies according to clinical form, being very high in nervous form (over 90%) and much lower in the abortive form [21].

Management Risk Factors: The most common management risk factor that reduce host immune response includes poor nutritional status, transport, sudden changes of weather to very cold and wet, late pregnancy and parturition stresses, long periods of flooding with resulting poor access to pasture and overcrowding and insanitary conditions with poor access to feed supplies [17, 18].

Pathogen Risk Factors: The pathogenic risk factor of infection increases due to a massive multiplication of *L. monocytogenes* in the feed or environment. The most important aspect in food hygiene is the ability of the bacteria to survive in a wide range of temperatures and to make biofilms on solid surfaces, including food processing facilities, which are more resistant to disinfectants and sanitizing agents [22].

The organism is a facultative intracellular pathogen that can infect cells, including intestinal cells, by directed endocytosis. Bacterial superoxide dismutase protects against the bactericidal activity of the respiratory burst of the phagocyte and listeriolysin O disrupts lysosomal membranes' allowing the organism to grow in the cytoplasm [19].

Host Range: *Listeria monocytogenes* is a widespread zoonotic pathogen that infects variety of species including mammals (Cattle, Cats, Rabbits, Sheep, Deer, Guinea-pigs, Goats, Pigs, Horses, Dogs, Foxes, Humans etc.), birds, fish and crustaceans. The most clinical listeriosis cases occur in ruminants; pigs rarely develop disease and birds are generally subclinical carriers of the organism [26].

Disease Status in Ethiopia: Listeriosis, a bacterial disease in humans and animals, is mostly caused by ingestion of *L. monocytogenes* via contaminated food and/or water, or by a zoonotic infection [8, 27]. In Ethiopia, a study has shown the presence and distribution of *L. monocytogenes* and other *Listeria* species in a variety of raw and ready-to-eat food products in Addis Ababa with a prevalence of 5.1% [28]. Derra *et al*. [27] described 4.1% of prevalence from raw meat and dairy products like raw milk, cottage cheese and cream cake collected from the capital and five neighboring towns in Ethiopia. The serotypes of *Listeria monocytogenes* identified belonged to 1/2b, 4b and 4e.

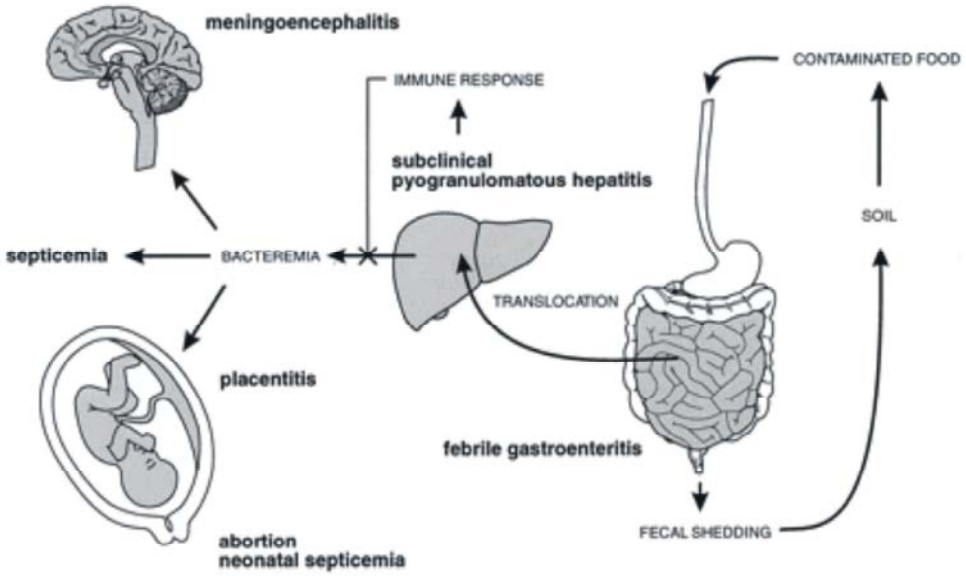
Pathogenesis of *L. monocytogenes*: Once organism is ingested then penetrates to the intestinal mucosa and results in a clinically in-apparent infection with localization of bacteria in various organs, or a fatal septicemia. According to Coetzer and Tustin [29] listeriae localize themselves in the uterus of pregnant animals and usually cause abortion if infection takes place early in pregnancy. It is not known precisely how bacteria reach the brain in animals developing meningoencephalitis; however, they probably gain entrance through wounds in the mucosa of the oral cavity.

The common mechanisms by which organisms reach the brain are proposed as follows: According to Drevets and Bronze [30] *Listeria monocytogenes* transports across the blood-brain or blood-choroid barriers within parasitized leukocytes and by direct invasion of endothelial cells by extracellular blood-borne bacteria. Retro-grade migration into the brain within the axons of cranial nerves (e.g. trigeminal nerve) can occur. The development of unilateral micro-abscesses and the perivascular infiltration of lymphocytes largely restricted to the brain stem (pons and medulla oblongata) are typically seen which seems to support this assumption. Recent studies in different models indicated non-neuronal cells take up bacteria more avidly than neurons, with microglia being the most easily infected. A more reliable means for *L. monocytogenes* to infect neurons seems to be through cell-to-cell spread from an infected macrophage [29, 30]

Several molecular virulence determinants have been identified that play a role in the cellular infection by *L. monocytogenes* and the unravelling of their mechanism of action has made of *L. monocytogenes* one of the most exciting models of host–pathogen interaction at the cellular and molecular levels. These virulence determinants include, among others, the internalins, listeriolysin O (LLO), ActA protein, two phospholipases, a metalloprotease, Vip protein, a bile exclusion system (BilE) and a bile salt hydrolase [31-33]. Although there is polymorphism among different strains of *L. monocytogenes* for some of these virulence determinants, it cannot be correlated with the ability or inability of the organism to produce disease [34]. Nevertheless, internalin A or B mediates entry of *L. monocytogenes* into some human-cultured cell lines and crossing of the intestinal in gerbil or transgenic mice expressing its receptor, human E-cadherin, in enterocytes.

Clinical Signs: Clinical outcome depends on the number of organisms ingested, pathogenic properties of the strains of Listeria and the immune status of the host. Although listeriosis is manifested by three major clinical signs; meningoencephalitis, abortion and septicaemia, only one clinical form of the disease usually occurs in a group of animals or an individual animal. However an overlap of clinical forms of disease has been reported. In addition to these three major signs of disease, mild mastitis, uveitis, ophthalmitis, iritis and/or keratoconjunctivitis have also been associated with *L. monocytogenes* [1].

Symptoms of Disease with Listeria Species in Human: There are two main forms of illness associated with *L. monocytogenes* infection such as invasive and non-invasive forms. Non-invasive form of listeriosis is the mild form of disease, while invasive listeriosis is the severe form of disease and can be fatal [36]. The likelihood that invasive listeriosis will develop depends upon a number of factors, including host susceptibility, the number of organisms consumed and the virulence of the particular strain [37].



Source: [35]

Fig. 1: Schematic representation of the pathophysiology of listeria infection

Symptoms of non-invasive listeriosis can include fever, diarrhea, muscle aches, nausea, vomiting, drowsiness and fatigue. The incubation period is usually 1 day (range 6 hours to 10 days) [36, 38]. Non-invasive listeriosis is also known as listerial gastroenteritis or febrile listeriosis.

Invasive listeriosis is characterized by the presence of *L. monocytogenes* in the blood, in the fluid of the central nervous system (leading to bacterial meningitis) or infection of the uterus of pregnant women. The latter may result in spontaneous abortion or stillbirth (20% of cases) or neonatal infection (63% of cases). Influenza-like symptoms, fever and gastrointestinal symptoms often occur in pregnant women with invasive listeriosis. In non-pregnant adults, invasive listeriosis presents in the form of bacterial meningitis with a fatality rate of 30%. Symptoms of invasive listeriosis include fever, malaise, ataxia, seizures and altered mental status [38] and the incubation period occurs before onset of invasive listeriosis and ranges from 3 days to 3 months [36].

The invasive forms of listeriosis in humans include septicemia, meningitis (or meningoencephalitis) and encephalitis (rhombencephalitis). Gastro enteric manifestations with fever also occur. Although the morbidity of listeriosis is relatively low, the mortality can reach values around 30%. In pregnant women, infection may result in abortion, stillbirth or premature birth and may be preceded by influenza-like signs including fever [38].

Necropsy Findings: Aborted fetuses of ruminants show very few gross lesions, but autolysis may be present if the fetus was retained before being expelled [39]. In abortion the pathological picture depends on the stage of pregnancy. Metritis usually occurs and results in retention of the foetal membranes. Gross lesions are tiny pin-point yellow foci in the liver. Similar foci but visible only microscopically are seen in the lung, myocardium, kidney, spleen and brain [1] Aborted fetuses are usually edematous and autolysed, with very large numbers of bacteria visible microscopically in a variety of tissues. Aborted fetuses due to *L. ivanovii* have suppurative bronchopneumonia and lack the multifocal hepatocellular necrosis commonly seen in abortions associated with *L. monocytogenes* [19].

There are usually no remarkable gross lesions in the brain of affected animals, but occasionally slight clouding or pin-point greyish-white foci of the meninges may be observed. Microscopic lesions are always confined to the pons, medulla and anterior spinal cord. Both white and grey matter may be involved. In the brain substance and sometimes in the meninges a remarkable perivascular cuffing with varying degrees of focal necrosis develops which is typical of Listeria encephalitis [1].

In the encephalitic form, the cerebrospinal fluid may be cloudy and the meningeal vessels congested. Gross lesions are generally subtle and characterized by vascular congestion and mild tan discoloration of the brainstem.

On occasion, the medulla shows areas of softening (malacia) and abscessation. The medulla and pons are most severely involved [39].

Diagnostic Techniques:

Tentative Diagnosis: The diseases can be tentatively diagnosed based on history, clinical signs and epidemiological findings and Post mortem findings [17].

Laboratory Diagnosis: Confirmatory diagnosis can be achieved by isolating the pathogen from appropriate specimens. Appropriate specimens for laboratory examination depend on the form of the disease. Cerebrospinal fluid (CSF) and tissue from the medulla and pons of animals with neurological signs should be sampled. Fresh tissue is required for isolation of organisms and fixed tissue for histopathological examination. Specimens for cases of abortion should include cotyledons, fetal abomasal contents and uterine discharges. Suitable samples from septicemic cases include fresh liver, spleen or blood [17].

Direct Microscopy: Smears from cotyledons or from liver lesions may reveal Gram positive coco bacillary bacteria. Histopathological examination of fixed (10% formalin) brain tissue can often give a presumptive diagnosis of neural listeriosis. Micro abscesses in the brain stem usually unilateral together with perivascular cuffing are very characteristics of listeriosis [22].

Isolation and Identification: Specimens from cases of abortion and septicemia can be inoculated directly onto blood agar, selective blood agar containing 0.05% potassium tellurite (inhibitory to Gram-negative bacteria) and MacConkey agar. The plates are incubated aerobically at 37°C for 24 to 48 hours. Commercial selective and indicator media are available, such as *Listeria* selective agar (Oxoid) and these are designed mainly for the isolation of *Listeria* from human food stuffs [15].

Cold-enrichment procedure is necessary for brain tissue. Small pieces of spinal cord and medulla are homogenized and a 10% suspension is made in a nutrient broth. The suspension is held at 4°C in the refrigerator and sub cultured onto blood agar once weekly for up to 12 weeks. Small transparent colonies with smooth borders appear on blood agar in 24 hours, becoming grayish white in 48 hours. All the *Listeria* species hydrolyze esculin (esculin broth). *L. monocytogenes*, particularly, shows the characteristic 'tumbling motility' when a 2 - 4 hour broth is cultured, incubated at 25°C and examined by the hanging-drop method. Catalase test is positive for *Listeria* species [17].

Animal Inoculation: Most isolates of animal origin are virulent, a characteristic which can be confirmed by animal inoculation. Anton Test is performed by inoculating a drop of broth culture into conjunctiva of rabbit or guinea pig. Only *L. monocytogenes* causes purulent keratoconjunctivitis within 24 - 36 hours of inoculation. Both *L. monocytogenes* and *L .ivanovii* are pathogenic for mice. Intraperitoneal inoculation of mice with a 24 hour broth culture results of their death within 5 days with necrotic lesions in the liver [17].

Molecular Methods: As molecular methods are accurate, sensitive and specific, they are increasingly used in Identification of *L. monocytogenes* from foods. Various molecular methods used are DNA hybridization, polymerase chain reaction and real time PCR (RT PCR). Among these, PCR and real time PCR are now established methods for identification of *Listeria monocytogenes* from other non-virulent Listeria spp. from foods. The real-time PCR is a very sensitive and quantitative method for direction of pathogen and thus has emerged as most important tool for *L. monocytogenes* detection and quantitation in foods [40].

Public Healthy Importance: Listeria is an opportunistic intracellular pathogen that has become an important cause of human foodborne infections worldwide [28].

Although *L. monocytogenes* is infective to all human population groups, it has a propensity to cause especially severe problems in pregnant women, neonates, the elderly and immunosuppressed individuals [41].

Direct transmission is possible especially among veterinarians performing gynecological interventions with aborted animals. Animals may be diseased or asymptomatic carriers of *L. Monocytogenes* shedding the organism in their feces. Thus, earlier it was believed that *L. monocytogenes* was causing disease by direct transmission from animals to humans. Indirect transmission may occur simply by consumption of food products from diseased animals, for example, Danielsson-Tham *et al.* [42] reported that on-farm manufactured raw milk cheese made from cattle with subclinical infection caused an outbreak with febrile gastrointestinal listeriosis involving 120 people. Raw or contaminated milk, vegetables and ready-to-eat meat have been implicated in overseas outbreaks. Contamination

could be during preparation and it then multiplies during the storage process. Unlike some other foodborne pathogens, *Listeria monocytogenes* can multiply in contaminated refrigerated food [43]. For example, soft cheese is considered as a high risk product for listeriosis because the bacteria may grow to significant numbers during refrigeration [44]. An outbreak was involved consumption of contaminated unpasteurized milk and other dairy products such as cheese and yoghurt [45]. Transmission of *L. monocytogenes* from mother to foetus or neonates has been frequently reported but cross-infection postpartum is also possible [46].

In neonatal infection, the pathogen is transmitted from the infected mother to her fetus transplacentally following maternal bacteremia. Some infections may also occur through ascending spread from vaginal colonization with the fetus acquiring infection during passage through the birth canal. Other routes of transmission, e.g. outbreaks attributed to contaminated equipment or materials in delivery room and hospital have also been reported [47, 48].

People at Risk: The risk of listeriosis for different ages and conditions found different categories that increased the risk [49].

Age: The major age at risk of listeriosis is newborn babies and the elderly with age, starting around age 65 or 70 years. In elderly as age increase risk of infection also increase [49].

Pregnancy: The risk of maternal listeriosis increases during pregnancy, particularly in the third trimester. As compared to the general population, pregnancy increases the risk of listeriosis by 2 to 17-fold. Most infected pregnant women have mild illness unless they have another underlying illness. However, about 20% of cases result in spontaneous abortion or neonatal death. About 2/3 of surviving infants develop neonatal listeriosis (presenting as sepsis or meningitis) [49]. Cell-mediated immunity decreases during pregnancy, so pregnant woman are at higher risk of getting *L. monocytogenes* infection. Listeriosis is most common in the third trimester but listeriosis cases have been reported in all stages of pregnancy. Pregnant women may also be more prone to listeriosis due to the tropism of internalin for E-cadherin molecules present on the syncytiotrophoblasts [50].

Immunocompromised and Medications: Usually individuals having weakened cell-mediated immunity are more susceptible to *L. monocytogenes* [31]. Medical conditions and medications that decrease T-cell mediated immunity increase the risk of listeriosis. Transplants and blood-related cancers confer the greatest risk. HIV/AIDS seems to rarely lead to listeriosis since the advent of Highly Active Anti-Retroviral Therapy (HAART) and trimethoprim-sulfamethoxazole prophylaxis. Pre-HAART, HIV/AIDS increased the risk of listeriosis by 865-fold. Other cancers, dialysis, liver disease and diabetes all confer a moderate risk of infection, greater than that caused by advanced age and pregnancy [49].

Treatment, Prevention and Control

Treatment: Although the optimal antibiotic treatment regimens for the various forms of listeriosis have not been established in experimental and clinical trial, cases with non-nervous signs (abortion, septicaemia and iritis) respond well to antibiotic treatments. The treatment is more effective in cattle because the course of the disease is longer and less severe in cattle. Animals that remain ambulatory are likely to recover but recumbent or comatose animals rarely survive and spontaneous recovery rarely occurs [1].

The difficulty in treating encephalitic Listeriosis has resulted in several *in-vitro* and *in-vivo* experimental studies to determine the best possible treatment regiments. In vitro studies have shown that the majority of antibiotics; penicillin, ampicillin, erythromycin, rifampicin, chloramphenicol, tetracycline and the aminoglycosides, with the exception of cephalosporin are effective against *L. monocytogenes*. However, *in vivo* use has proved controversial. Several drugs and their combinations were used in experimental Listeriosis in laboratory animals. A combination of trimethoprim and tetracycline was more effective than a combination of trimethoprim and penicillin and these combinations were better than the use of each antibiotic alone [1].

Cases of spinal myelitis are poorly responsive to treatment. Treatment of Listeria iritis is with systemic antibiotics in the early stages coupled with sub palpebral corticosteroid and atropine to dilate the pupil. In vitro resistance to the tetracycline group of antimicrobials is reported [19]. Treatment of uveitis includes the use of systemic antibiosis, but subconjunctival corticosteroids and topical atropine are essential for effective resolution [23]. The animals recovered after treatment with parenteral ampicillin and topical ceprovin in case of ocular listeriosis [51].

Prognosis: The animals that were recumbent, excited, with an absent or weak menace reflex, nystagmus, high numbers of leucocytes in the CSF, high serum

concentrations of urea and calcium and high serum activities of aspartate aminotransferase and creatine kinase and an acid-base deficit, had a smaller chance of surviving. When a logistic regression model was constructed, only recumbency, excitement and a weak or absent menace reflex remained significant factors affecting the likelihood of survival [52].

Prevention and Control: Prevention of the disease in animals includes provision of good quality silage and elimination of stressful conditions such as dense stocking rates and minimizing ingestion of soil-contaminated pastures. Spoiled silage should be avoided and Corn ensiled before being too mature and grass silage containing additives are likely to have a more acid pH, which discourages multiplication of *L. monocytogenes* should be used [53].Vaccines are available in some countries, however results are questionable, which leads to questions about the cost-benefit of vaccination.

Epidemiologic investigations have demonstrated that nearly all types of food can transmit *Listeria*. Most sporadic cases and all large outbreaks have been associated with manufactured foods [54]. Pasteurization eliminates *Listeria* from dairy products and most dairy-associated outbreaks are from items that are inadequately pasteurized or contaminated after pasteurization. Women should wash all utensils and surfaces well after preparing meat dishes or cutting prepared foods. Patients should know to contact their provider if they have any of the common symptoms listed in. Providers should then maintain enough suspicion for listeria infection to draw blood cultures for any woman at risk [55].

The control of Listeria in foods relies largely on a HACCP approach and the establishment of effective critical control points in the process. The careful design and layout of processing equipment in conjunction with the implementation of regular, thorough cleaning regimes of the processing environment can significantly reduce the level of Listeria contamination in many processed foods. However, because of its ubiquitous nature it is virtually impossible to totally eliminate the pathogen from many food products. Vulnerable individuals, especially pregnant women, the elderly and the immunosuppressed are advised to avoid consuming unpasteurized dairy products to reduce the risk from listeriosis [23].

Early detection of a listeriosis outbreak and efficient intervention are important in preventing the epidemic from continuing. Typing of food isolates and comparison with clinical isolates may also lead authorities to contaminated food processing plants. However, in addition to typing results, epidemiological evidence is needed for the incrimination of a food or a food processing plant [55]. Standards/legislation for the pasteurization of ice cream/frozen desserts adapted in various countries has an importance in reducing listeriosis [49].

CONCLUSIONS AND RECOMMENDATIONS

L. monocytogenes has gained recognition as a global human pathogen because of the increasing incidence, diagnosis of infections and also, it is widespread in nature and lives naturally in plants and soil environments and has potential to introduce food plant. It can grow in a wide range of temperature and pH. Milk and milk products are important vehicles of *L. monocytogenes*, regularly causing listeriosis outbreaks in different countries of the world.

Good manufacturing and hygiene practices, particularly maintaining hygiene of processing machines, are the keys in preventing *L .monocytogenes* contamination. It is also equally important to notice that products, which may be subjected to post processing contamination, should be properly reheated before consumption by highly immune compromised persons in order to eliminate possible contamination. A food safety management system based on the principles of HACCP with regular reviews should be developed and implemented in dairy plant.

Based on the fact and information mentioned in the review the following recommendations are forwarded.

- There should be proper disposal of aborted fetus and feces of infected animal to avoid spread of the disease.
- Public health learning through Mass-media, radio and teaching livestock holder's and people who are at risk to *Listeria monocytogenes* is important.
- People susceptible for acquiring listeriosis should not consume unpasteurized milk and milk products.
- Every effort should be made to produce silage of good quality, with early cutting of grass, minimal contamination with soil or feces and ensuring optimal anaerobic fermentation, which will insure that the pH falls below 5.0; at that level, growth of *Listeria* spp. is inhibited.
- Meat products should be treated with heat before consumption which can kill Listeria species or reduce them to undetectable level.
- There should be wear gloves when handling fetuses and specimens from aborted cow.

REFERENCES

1. Erdogan, H.M., 1998. An epidemiological study of listeriosis in dairy cattle. Ph.D. Thesis, Division of Animal Health and Husbandry, Department of Veterinary Clinical Science, University of Bristol.
2. Painter, J. and L. Slutsker, 2007. Listeriosis in humans, listeriosis and Food Safety, 3rd ed. Eds., Ryser, E.T. and E.H. Marth. CRC Press, Taylor & Francis Group, Boca Raton, Florida, USA, pp: 85-110.
3. Cahn, CM. and S. Line, 2005. The Merck Veterinary Manual. 9th edition. Merck & Co; INC. White house Station, N.J., USA, pp: 531-533
4. Yan, H., S.B. Neogi, Z. Mo, W. Guan, Z. Shen, S. Zhang, L. Li, S. Yamasaki, L. Shi and N. Zong, 2010. Prevalence and characterization of antimicrobial resistance of foodborne *Listeria monocytogenes* isolates in Hebei province of Northern China, 2005-2007. International Journal of Food and Microbiology, 144: 310-316.
5. Woo-Sam, 1999. Listeriosis in a Holstein cow. The Canadian Veterinary Journal, 40: 506.
6. Fenlon, D.R., 1999. Listeria monocytogenes in the natural environment. In: Ryser E T, Marth E H, editors. Listeria, listeriosis and food safety. 2nd ed. New York, N.Y: Marcel Dekker Inc., pp: 21-37.
7. Robertsa, J. and Wiedemannm , 2003. Pathogen, host and environmental factors contributing to the pathogenesis of listeriosis. Cellular an Molecular Life Sciences, 60: 904-918.
8. Pal, M., 2007. Zoonoses.Second edition.Satyam Publishers, Jaipur, India, pp: 118-119.
9. Gebretsadik, S., T. Kass, Alemayehu, K. Huruy and N. Kebede, 2010. Isolation and characterization of Listeria monocytogenes and other Listeria species in foods of animal origin in Addis Ababa, Ethiopia. Journal of Infection and Public Health, 4: 22-29.
10. Esteban, J.I., B. Oporto, G. Aduriz, R.A. Juste and A. Hurtado, 2009. Faecal shedding and strain diversity of *Listeria monocytogenes* in healthy ruminants and swine in Northern Spain. Biomedical Central Veterinary Research, 5(2).
11. Bibek, R. and B. Arun, 2008. Fundamental Food Microbiology. CRC Press, United States, 4th ed, USA, pp: 288-294.
12. Chakraborty, T. and T. Hain, 2013. Comparative genomics: The genus Listeria. In The Compendium of International Symposium on Problems of Listeriosis (ISOPOL XVIII), 19-22.Goa India. Abstract No-BIO-22.
13. Molla, B., R. Yilma and D.Alemayehu, 2004. *Listeria monocytogenes* and other *Listeria* Species in Retail Meat And Milk Products In Addis Ababa, Ethiopia. Ethiopian Journal of Health Development, 18(3): 208-212.
14. Bundrant, N.B., T. Hutchins, H.C. Bakker, E. Fortes and M. Wiedmann, 2006. Listeriosis outbreak in dairy cattle caused by an unusual *Listeria monocytogenes*serotype 4b strain. Journal of Veterinary Diagnostic Investigation, 23: 155-158.
15. Wiedmann, M., G. T. Jeffers, J. L.Bruce, P.L. McDonough, J. Scarlett and K.J. Boor, 2001. Comparative genetic characterization of Listeria monocytogenes isolates from human and animal listeriosis cases. Microbiology, 147(Pt 5): 1095-1104.
16. Scott, V.N., M.J. Gray, R.N. Zadoks, E.D. Fortes, B. Dogan, S. Cai and Y. Chen, 2004. Listeria monocytogenes Isolates from Foods and Humans Form Distinct butOverlapping Populations. Applied and Environmental Microbiology, 70(10): 5833-5841
17. Tewodros, F. and F. Atsedewoyne, 2012. Listeriosis in Small Ruminants: Advance In Biological Research, 6(6): 202-209.
18. Hirsh, C.D., J.N. Maclachlan and L.R. Walklers, 2004. Veterinary Microbiology. 2nded, Blackwell publishing, USA, pp: 185-189.
19. Radostits, O.M., C.C. Gay, K.W. Hincheliff and P.D. Constable, 2007. Diseases associated with Listeria species: Veterinary Medicine, a Textbook of the disease of Cattle, Sheep, Pigs, Goats and Horses. 10th edition Saunders Elsevier Published Ltd., London, pp: 805-810.
20. Larpent, J.P., 2000. Listeria, 2nd Edition, Ed. Tec et DOC, Paris.
21. Perianu, T. and Bolile, 2004. Infecþious Animal or Domestic, Vol. I, Bacterioze, Ed. Venus, Iaşi, pp: 431-443
22. Anon., 1992. Veterinary Investigation Diagnosis Analysis III. Ministry of Agriculture, Fisheries and Food. Central Veterinary Laboratory, Weybridge.
23. Andrew, A.H., R.W. Blowey, H. Boyd and R.H. Eddy, 2004. Bovine Medicine Diseases and Husbandry of cattle. 2nd Edition. Blackwell Science Ltd a Blackwell Publishing Company pp: 156, 251, 581-2582, 789 and 904-906.
24. Bortolussi, R., D.D. McGregor, P.A.L. Kongshavn, S. Galsworthy, W. Albritton, J.W. Davies and H.P.R. Seeliger, 1984. Host defense mechanisms to perinatal and neonatal Listeria monocytogenes infection. Pathology and Immunopathology Res., 3(4): 311-332.

25. Quinn, P.J., B K. Markey, W.J. Donnelly, A. Carter and F.C. Leonard, 2002. Veterinary Microbiology and Microbial Disease. 1st published Blackwell publishing; UK (Great Britain), pp: 162-165.
26. Wesley, 2007. Listeriosis in animals. *Listeria*, Listeriosis and Food Safety, Third Edition, Ryser E.T. &Marth E.H., eds. CRC Press, Taylor & Francis Group, Boca Raton, Florida, USA, pp: 55-84.
27. Derra, F.A., S. Karlsmose, D.P. Monga, A. Mache, C.A. Svendsen, B. Félix and R.S. Hendriksen, 2013. Occurrence of Listeria spp. in retail meat and dairy products in the area of Addis Ababa, Ethiopia. Foodborne Pathogens and Disease, 10(6): 577-579.
28. Selamawit, M., 2014. The Prevalence, Risk Factors, Public Health Implication And Antibiogram Of Listeria Monocytogenes In Sheep Meat Collected From Municipal Abattoir And Butcher Shops In Addis Ababa.
29. Coetzer, J.W. and R.C. Tustin, 2004. Infectious Diseases of Livestock.Second edition. Oxford University Press Southern Africa, Cape Town, pp: 46-56.
30. Drevets, D.A. and M.S. Bronze, 2008. *Listeria monocytogenes*: epidemiology, human disease and mechanisms of brain invasion. Immunology and Medical Microbiology, 53: 151-165.
31. Dussurget, O., D. Cabanes, P. Dehoux, M. Lecuit, C. Buchrieser, P. Glaser and P. Cossart, 2002. *Listeria monocytogenes* bile salt hydrolase is a PrfA-regulated virulence factor involvedin the intestinal and hepaticphases of listeriosis. Molecular Microbiology, 45: 1095-1106.
32. Cabanes, D., S. Sousa, A. Cebriá, M. Lecuit, F. García-Del Portillo and P. Cossart, 2005. Gp96 is a receptor for aNovel *Listeria monocytogenes* virulence factor, Vip, a surface protein. The EMBO Journal, 24(15): 2827-2838.
33. Cossart, P. and A. Toledo-Arana, 2008. *Listeria monocytogenes*, a unique model in infection biology: an overview. Microbes and Infection, 10: 1041-1050.
34. Jacquet, C., E. Gouin, D. Jeannel, P. Cossart and J. Rocourt, 2002. Expression of ActA, Ami, InlB, andlisteriolysin O in *Listeria monocytogenes* of human and food origin. Applied and Environmental Microbiology, 68: 616-622.
35. Pron, B., C. Boumaila, F. Jaubert, P. Berche, G. Milon, F. Geissmann and J.L. Gaillard, 2001. Dendritic cells are early cellular targets of Listeria monocytogenes after intestinal delivery and are involved in bacterial spread in the host. Cellular Microbiology, 3: 331-340.
36. FDA., 2012. Bad bug book: Foodborne pathogenic microorganisms and natural toxins handbook, 2nd ed. US Food and Drug Administration, Silver Spring, pp: 100-104.
37. WHO/FAO, 2004. Risk assessment of *Listeria monocytogenes* in ready-to-eat foods. World Health Organization and Food and Agriculture Organization of the United Nations, Geneva.
38. Painter, J. and L. Slutsker, 2007. Listeriosis in humans, listeriosis and Food Safety, Third Edition, Ryser E.T. &Marth E.H., eds. CRC Press, Taylor & Francis Group, Boca Raton, Florida, USA, pp: 85-110.
39. OIE 2014. Version adopted by the World Assembly of Delegates of the OIE in 2014OIE *Terrestrial Manual* 2014 1C H A P T E R 2 . 9. 7. Listeria Monocytogenes, pp: 1-18
40. Hough, A.J., S.A. Harbison, M.G. Savill, L.D. Melton and G. Fletcher, 2002. Rapid enumeration of Listeria monocytogenes in artificially contaminated cabbage using real-time polymerase chain reaction. Journal of Food Protection, 65: 1329-1332.
41. Liu, D., 2006. Identification, subtyping and virulence determination of *Listeria monocytogenes*: An important food borne pathogen. Journal of Medical Microbiology, 55: 645-659.
42. Danielsson-Tham, M.L., E. Eriksson, S. Helmersson, M. Leffler, L. Ludtke, M. Steen, S. Sorgjerd and W. Tham., 2004. Causes behind a human cheese-borne outbreak of Gastro-intestinallisteriosis. Food Born Pathogenes and Disease, 1: 153.
43. Heymann, D.L., 2004. Control of communicable diseases manual. 18th ed. Washington: American Public Health Association.
44. Martin p de Valk, H., C. Jacquet, V. Goulet, V. Vaillant, A. Perra, F. Simon and J.C. Desenclos, 2005. Surveillance of Listeria infections in Europe. European Surveillance, 10: 251-255.
45. Fleming, D.W., S.L. Cochi, K.L. MacDonald, J. Brondum, P.S. Hayes, B.D. Plikaytis, M.B. Holmes, A. Audurier, C.V. Broome and A.L. Reingold, 1985. Pasteurized milk as a vehicle of infection in an outbreak of listeriosis. New England Journal of Medicine, 312: 404-7.
46. Lecuit, M., D.M. Nelson, S.D. Smith, H. Khun, M. Huerre, M.C. Vacher-Lavenu, J.I. Gordon and P. Cossart, 2004. Targeting and crossing of the human materno-fetal barrier by *Listeria monocytogenes*: role of internalin interaction with trophoblast E-cadherin. Procedings of the National Academy of Sciences of the United States of America, 101: 6152-6157.

47. Nelson, K.E., D. Warren, A.M. Tomasi, T.N. Raju and D. Vidyasagar, 1985. Transmission of neonatal listeriosis in a delivery room. American Journal of Diseases of Children, 139(9): 903-905.
48. Kevin, A. Schuchat, C. Lizano, C.V. Broome, B. Swaminathan, C. Kim and K. Winn, 1991. Outbreak of neonatal listeriosis associated with mineral oil. The Pediatric Infectious Disease Journal, 10(3): 183-9.
49. Galanis, Eleni and David Patrick, 2008. Risk of Listeriosis Associated with Age, Pregnancy and Various Conditions. Prepared by BCCDC for the BC Food Safety in Facilities Advisory Group.
50. Cossart, P., 2004 The infection by Listeria monocytogenes towards a complete understanding of human listeriosis. XV International Symposium on Problems of Listeriosis., pp: 131.
51. Staric, J., F. Kri_anec and T. Zadnik, 2008. Listeria monocytogenes keratoconjunctivitis and uveitis in dairy cattle. University of Ljubljana, Veterinary Faculty, Clinic for Ruminants, 1000 Ljubljana, SloveniaivinozdravniškaambulantaKri&Za, 2326 Cirkovce, Slovenia.Veterinary Record, 158: 588-592.
52. Schweizer, G., F. Ehrensperger, P.R. Torgerson and U. Braun, 2006. Clinical findings and treatment of 94 cattle presumptively diagnosed with Listeriosis. Department of Farm Animals. Institute of Veterinary Pathology, Institute of Parasitology, University of Zurich, Winterthurerstrasse 260, CH-8057 Zurich, Switzerland. Veterinary Record, 158: 588-592
53. Clark, R.G., J.M. Gill and S. Swanney, 2004. *Listeria monocytogenes gastroenteritis* in sheep. New Zealand Veterinary Journal, 52: 46-47.
54. Heikkinen, T., K. Laine, P.J. Neuvonen and U. Ekbald, 2000. The transplacental transfer of the macrolide antibiotics erythromycin, roxithromycin and azithromycin. International Journal of Ostetrics and Gyenaecology, 107: 770-775.
55. Cherubin, C.E., M.D. Appleman and P.N.R. Heseltine, 1991. Epidemiological spectrum and current. Review of Infectious Disease, 13: 1108-1114.