

## Listeriosis in Small Ruminants: A Review

Tewodros Fentahun and Atsedewoyne Fresebehat

Unit of Basic Veterinary Science, Faculty of Veterinary Medicine,  
University of Gondar, P.O. Box 196, Gondar, Ethiopia

**Abstract:** Listeriosis is a bacterial disease caused by different *Listeria* species; among these *Listeria monocytogenes* being the most pathogenic species to small ruminants. The other two species, the so called *Listeria ivanovii* and *Listeria innocua* are less frequently implicated in disease of animals. Listeriosis is most prevalent during spring and winter seasons. Soil contamination and ingestion of contaminated silage are the primary modes of transmission. Poor quality silage, a pH greater than 5.5 is commonly implicated and accounts for listeriosis often being referred to as “silage disease”. Both host management and the pathogen itself has different contributions for the occurrence of the disease. The clinical manifestations of listeriosis in small ruminants are usually severe and include abortion, meningoencephalitis and neonatal septicemia. The great effect of listeriosis in small ruminants and cattle is due to the consequence of severe damage on brain. Those diseases affecting the nervous system such as rabies, gid (coenurosis or sturdy), scrapie and pregnancy toxemia should be differentiated from listeriosis. Silage feeding should be discontinued if an outbreak of listeriosis is confirmed. In the case of abortion, isolation of aborting does and ewes from their offspring reduces further infection of the dam. The recommended drugs used for treatment of listeriosis include Oxytetracycline, penicillin-G and Dexamethasone.

**Key words:** Encephalitis • *Listeria Monocytogenes* • Listeriosis • Small Ruminants

### INTRODUCTION

Listeriosis is a serious and life threatening disease caused by the *Listeria monocytogenes*. *Listeria* species are Gram positive, facultative anaerobic intracellular bacteria that are ubiquitously distributed in the environment and grow over a wide range of pH and temperature. The genus is composed of six species; three of which are pathogenic. *Listeria monocytogenes*, the most important of these three pathogens, has been implicated worldwide in diseases of many animal species and human [1].

The organism can grow over a wide range of temperatures from 4 to 45°C and can tolerate pH values between 5.5 and 9.6. In such circumstances, *L. monocytogenes* may reach 1<sup>07</sup> colony forming units (CFU) per kilogram of silage. The other two pathogens, *Listeria ivanovii* and *Listeria innocua* are less frequently implicated in disease of animals. The bacteria have a group of genes, which allow invasion, survival, multiplication and mobility in the intracellular

environment. The organism resists freezing and thawing and is able to survive for several years in feces, straw, silage and soils [2].

Listeriosis is a disease that is most frequently encountered in sheep, goats and cattle. Clinical manifestations of invasive listeriosis in ruminants are usually severe and include abortion, neonatal septicemia and meningoencephalitis [3]. Listerial encephalitis in small ruminants is more commonly found in sheep than goats with higher mortality rate than that observed in cattle. Micro abscesses, focal gliosis and perivascular cuffing characterize listerial encephalitis. Severe lesions are confined to the brain stem, especially pons and medulla oblongata. The great effect of listeriosis in small ruminants and cattle is the severe influence on brain. The first signs of encephalitic listeriosis are the changes in the animal behavior, depression or even hypersensitivity to rumor or other stress. Depending on the nucleus affected, symptoms may be facial hypalgesia, paralysis and ataxia [4]. Especially in sheep listeriosis leads to head tilt and permanent circling movement [2].

Listeriosis is most prevalent during spring and winter seasons which suggests that the prevalence of *L. monocytogenes* in ruminant farm is seasonal. Listeriosis in goats is transmitted via the oral-fecal route, usually when animals ingest contaminated water or feed, or by fecal shedding of *L. monocytogenes*. Infection can also occur by inhalation. Infected animals could die if improperly treated [5]. Therefore, the main objective of this paper is to review imperative points on the small ruminant listeriosis.

**Listeriosis in Small Ruminants:** Listeriosis in small ruminants may present as encephalitis, abortion, septicemia or endophthalmitis, but mainly takes the form of meningoencephalitis, called circling disease in its most common form. Affected animals circle, in one direction only and display unilateral facial paralysis, difficulty in swallowing, fever, blindness and head pressings. Paralysis and death follow in 2 to 3 days [6]. Usually, only one form of the disease occurs in a group of affected animals. Septicemia is often encountered in neonates and can also occur in adult sheep [1].

In pregnant animals, *L. monocytogenes* may localize in the placentas and cross over to amniotic fluid. It multiplies there and is ingested by the fetus, eventually causing fetal death and abortion. Listerial abortion usually occurs in late gestation [6].

**Genus *Listeria*:** The genus *Listeria* is composed of six species; three of which are pathogenic. *L. monocytogenes*, the most important of these pathogens, has been implicated worldwide in diseases of many animal species and human. The other two pathogens, *L. ivanovii* and *L. innocua*, are less frequently implicated in diseases of animals [6]. *L. ivanovii* is mildly pathogenic and is an occasional cause of abortion in sheep and cattle. It is occasionally associated with encephalitis in ruminants that is clinically and pathogenically similar to that associated with *L. monocytogenes* [2].

### 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 a 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 [8, 9].

*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* [8].

### Differentiation of *Listeria* Species Affecting Small Ruminants:

The patterns of hemolysis on sheep blood agar, CAMP test and acid production from a short range of sugars are useful differentiating laboratory method for *Listeria* species (Table 1) [1]. *L. monocytogene* is CAMP-positive when cross streaked with a beta-toxin producing *Staphylococcus aureus* (SA) on blood agar. A similar phenomenon is observed when *L. ivanovii* (LIV) is cross streaked with *Rhodococcus equi* (RE). A week CAMP like reaction is observed between *L. monocytogenes* and RE. *L. innocua* is not hemolytic, whereas both *L. monocytogenes* and LIV are hemolytic. In semisolid motility media incubated at room temperature (25°C), a characteristic umbrella pattern of motility develops 3 to 4 mm below the surface, due to the microaerophilic nature of *Listeria* [7]. On sheep blood agar, most strains of *L. monocytogenes* produce a narrow zone of hemolysis. Colonies are usually 1 to 2mm in diameter and appear blue-green in obliquely transmitted light on solid media such as tryptose agar. Colonies of *L. ivanovii* produce a larger and more intense zone of hemolysis [8].

Table 1: Laboratory methods for differentiating *Listeria* species affecting small ruminants

<i>Listeria</i> species	Hemolysis on sheep blood agar	CAMP test		Acid production from sugars		
		SA	RE	D-mannitol	L-rhamnose	D-xylose
<i>L. monocytogenes</i>	+	+	-	-	+	-
<i>L. ivanovii</i>	++	-	+	-	V	+
<i>L. innocua</i>	-	-	-	-	-	-

Where, V = Variable reaction  
 + = Positive reaction  
 -- = negative reaction;  
 SA= *Staphylococcus aureus*  
 RE= *Rhodococcus equi*  
 Source: Quinn *et al.* [1].

**Epidemiology of Listeriosis Occurrence:** Geographical location: Although the organism is wide spread in nature, clinical diseases in animals occur mainly in the northern and southern latitudes and are much less common in tropical and subtropical than in temperate climates [2, 5]. In the northern hemispheres, listeriosis has a distinct seasonal occurrence, probably associated with seasonal feeding of silage, with the highest prevalence in the months of December through May [2, 7].

**Host Factor:** listeriosis is primarily a disease of ruminants, particularly sheep and the major diseases associated with *L. monocytogenes* are encephalitis and abortion. It also produces syndromes of septicemia, spinal myelitis, uveitis, gastroenteritis and mastitis [2].

**Source of Infection:** The organism is truly ubiquitous in the environment and can be 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. The ability to form bio-film may assist in its survival in the environment and in perpetuating its presence in water troughs on infected farms [1, 2].

Most feed hays, grains and formulated feeds have the potential to contain *L. monocytogenes* but, with most low level of available water restrict its multiplication. In ruminants, *L. monocytogenes* can be isolated from the feces and nasal secretions of healthy animals. In temperate climates, the prevalence of *L. monocytogenes* in the feces of ruminants appears to vary with the season, being higher in the winter period. It is also increased during periods of environmental stress and in association with the stress of lambing and transport. The presence in feces and secretions can also be influenced by the number of the organism in feeds fed to the animals [2, 6].

*L. monocytogenes* is commonly present in silage, but it doesn't multiply to any significant extent in effectively preserved silage which is characterized by an aerobic 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. The risk for contamination of silage with *Listeria* is higher when it contains soil. Moist preserved feeds other than grass silage are at risk for *Listeria* growth; listeriosis is recorded, for example, in association with the feeding of moist brewers' grains, wet spoiled hay bales and silage made from commodity by-products such as orange and artichoke waste. Infected

animals can also serve as a source of infection from their urine, feces, aborted fetuses, uterine discharges and the milk. Woody browse may be a risk factor for goats [2].

**Transmission:** Soil contamination and ingestion of contaminated feed are the primary modes of transmission of *Listeria*. Poor quality silage, with a pH greater than 5.5, is commonly implicated and accounts for listeriosis often being referred to as "Silage disease" [8]. Lambs which develop septicemic disease may acquire infection from contamination on the ewe's teat, from the ingestion of milk containing the organism from ewes or does with subclinical bacteremia, through the navel from the environment and also as a congenital infection [2]. 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 [2, 7].

**Risk Factors:** It is apparent that many animals are exposed to *L. monocytogenes*; carry it in their feces as a normal bowel inhabitant, but that only a small proportion of animals develop clinical disease. 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 [2].

**Host Management Risk Factors Include:** poor nutritional status, sudden changes of weather to very cold and wet, late pregnancy and parturition stresses, transport, long periods of flooding with resulting poor access to pasture and overcrowding and insanitary conditions with poor access to feed supplies [7].

**Pathogen Risk Factors:** That increase the infection pressure largely involve a massive multiplication of *L. monocytogenes* in the feed or environment. The feeding of grass or corn silage as a major risk factor for the occurrence of listeriosis has been recognized for many decades. The increase in use of silage for feed in ruminants may be the reason for the apparent increase in the prevalence of the disease in recent years. Introduction of virulent strains to the flock may also occur via a carrier

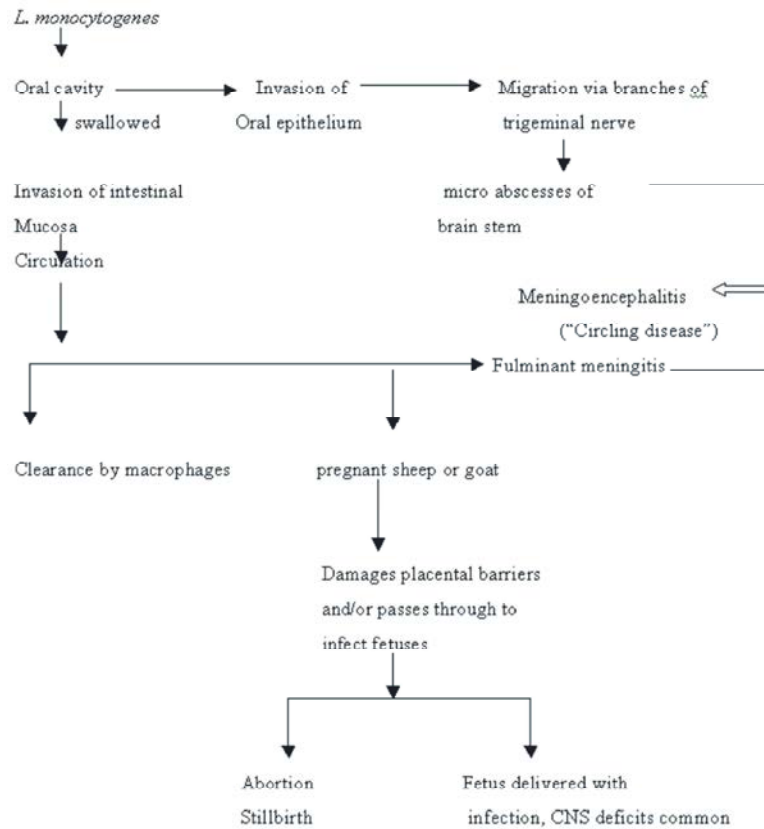


Fig. 1: Natural history of *Listeria monocytogenes* infections. Source: Songer and Post [6].

animal and birds such as seagulls that scavenge on sewerage areas, may carry a heavy population of the bacteria and can contaminate feed or pasture for silage. The organism persists for as long as 3 months in sheep faeces and has been shown to survive up to 11.5 months in damp soil, up to 16.5 months in cattle feces, up to 207 days on dry straw and for more than 2 years in dry soil and faeces [3].

**Cellular Products of *Listeria* Contributing to its Virulence:** these include Act-A, it is a protein that is important in intracellular movement by acting polymerization and is also thought to play a role in cell tropism (adhesion) and invasion. Internalins, they are surface proteins responsible for adhesion and entry into target cells. Listeriolysin O (LLO), it is a pore-forming cholesterol dependent cytolysin used in the release of *L. monocytogenes* from the phagosome into the cytosol following phagosome acidification; under which conditions LLO is the most active. Other roles include lysis of ferritin vacuoles and perhaps its effect on secondary vesicles formed during *L. monocytogenes*

movement from cell to cell. LLO is also thought to induce apoptosis in hepatocytes. Ivanolysin, it is another cholesterol-dependent cytolysin (a counter part in *L. ivanvii*). Phospholipase C; phosphatidylinositol-specific phospholipase-C and lecithinase are important in mediating membrane lysis and bile salt hydrolase promotes survival and persistence of *Listeria* in the intestinal lumen [7].

**Pathogenesis Mechanism of Pathogenesis:** Infection with *L. monocytogenes* usually follows ingestion of contaminated feed and may result in septicemia, encephalitis, abortion (Figure 1). Most *Listeria* species are destroyed by gastric acids. Use of antacids and H<sub>2</sub>-blockers increases survival rate and are considered as risk factors for developing listeriosis [7]. Organisms probably penetrate the M-cells in Payer's patches in the intestine. Spread, occurs via lymph and blood to various tissues [1]. Internalin, a surface protein and its interaction with host cell receptors mediate entry. An alternative route of entry has been proposed for central nervous system (CNS) infection through damaged oral, nasal or

Table 2: Clinical manifestations of infection with *Listeria* species in small ruminants

Species	Host	Forms of disease
<i>L. monocytogenes</i>	Sheep and goats	Encephalitis (neural form) Abortion septicemia Endophthalmitis (ocular form)
<i>L. ivanovii</i>	Sheep	Abortion
<i>L. innocua</i>	Sheep	Meningoencephalitis (rare)

Source: Songer and Post [6].

ocular mucosal surfaces via the neural sheath of peripheral nerve endings, particularly the trigeminal nerve. It is postulated that centripetal migration along cranial nerves leads to infection of the CNS. In the brain stem, there is unilateral bacterial localization with focal micro-abscess formation [10]. *L. monocytogenes* has the ability to invade both phagocytic and non phagocytic cells, to survive and replicate intracellularly and transfer from cell to cell without exposure to humoral defense mechanisms. Virulent strains also possess a cytolytic toxin, listeriolysin, which destroys the membranes of phagocytic vacuoles allowing *Listeria* to escape into the cytoplasm. In the cytoplasm, the organism utilizes cellular microfilaments to generate tail-like structures which confer motility [1].

**Pathology:** With CNS involvement, the cerebrospinal fluid (CSF) may be cloudy and the meningeal vessels are congested. Occasionally, areas of softening in the medulla are seen [7]. Lesions in the brainstem, often unilateral, are composed of micro-abscesses and perivascular lymphocytic cuffs [1]. In septicemic form, multiple foci of necrosis in the liver and less frequently, the spleen may be noted. In the aborted fetus of ruminants, gross lesions are minimal. Autolysis is usually present as a result of the dead fetus being retained for a period before being expelled [7].

**Clinical Signs:** The clinical outcomes of listeriosis depends on the number of organisms ingested, pathogenic properties of the strains and the immune status of the host. There are three major clinical forms of listeriosis (Table 2). These are septicemia, encephalitic and abortion forms [7, 9].

**Septicemic Form:** This form is known in the formation of septicemia. The septicemic form is marked by depression, in appetite, fever and death. The septicemic disease in sheep and goats usually occurs within 2 days of introduction to silage and abortions 6 - 13 days later [2]. Acute septicemia due to *L. monocytogenes* is not common

in adult ruminants but does occur in monogastric animals and in newborn lambs and calves. There are no signs suggestive of nervous system involvement, the syndrome being a general one comprising weakness, emaciation, diarrhea in some cases; with hepatic necrosis and gastroenteritis at necropsy [10].

**Encephalitic Form:** The encephalitic form, sometimes called “circling disease”, is the most common form in ruminants. The course of the disease is more acute and frequently fatal in sheep and goats, but subacute to chronic in cattle [8]. The outbreaks of the encephalitis which occurs in sheep after introduction to silage usually commence about 3 - 4 weeks later. This delay reflects the time for ascending infection [2, 11].

Signs vary between individual sheep but incoordination, head deviation sometimes with head tilt, walking in circles, unilateral facial hypalgesia and facial paralysis are usually present. Facial hypalgesia can be detected with pressure from hemostat and facial paralysis is manifested with drooping of ear, paralysis of the lips and ptosis on the same side of the face as the hypalgesia. This may be accompanied by exposure keratitis, often severe enough to cause corneal ulceration. There is a paresis of the muscle of the jaw, with poor tone or a dropped jaw, in which case prehension and mastication are slow and the animal may stand for long periods drooling saliva and with food hanging from its mouth. Death is due to dehydration and starvation [10].

The position of heads varies In many cases, there is deviation of the head to one side with the poll-nose relationship undisturbed without rotation, but in others there is also head tilt. The head may be retroflexed or ventro-flexed depending on the localization of the lesion and in some cases may be in a normal position. The deviation of the head cannot be corrected actively by the animal and if it is corrected passively the head returns to its previous position as soon as it is released. There is ataxia, often with consistent falling to one side and an affected sheep may lean against the examiner or a fence. The affected animal becomes recumbent and is unable to

rise, although often still able to move its legs. Death is due to respiratory failure. In goats, the disease is similar to that of sheep, but in the young goat, the onset is very sudden and the course is short, with death occurring in 2 - 3 days [6].

**Abortion:** Abortion is common in ruminants usually late term-after 12 weeks in sheep. The fetus may be macerated or delivered weak and moribund. Retained placenta and metritis may be resulted [8]. Outbreaks of abortion occur more commonly in sheep and goats and there will be a blood stained vaginal discharge for several days. There may be some deaths of ewes from septicemia if the fetus is retained. In both species, the rates of abortion in a group are low but may reach as high as 15%. On some farms, abortions recur each year [10].

**Diagnosis:** The diseases can be tentatively diagnosed based on clinical signs and its confirmation is achieved by isolating the pathogen from appropriate specimens. Characteristic neurological signs or abortion in association with silage feeding may suggest listeriosis [1].

**Laboratory Diagnosis:** 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 [1, 12, 13].

**Direct Microscopy:** Smears from cotyledons or from liver lesions may reveal Gram positive coccobacillary 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 [12].

**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 [1, 14]. 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 [14].

A '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 [1, 14]. 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 [12].

**Animal Inoculation:** Most isolates of animal origin are virulent, a characteristic which can be confirmed by animal inoculation [14].

**Anton Test:** Inoculation of a drop of broth culture into conjunctiva of rabbit or guinea pig. Only *L. monocytogenes* causes purulent keratoconjunctivitis within 24 - 36 hours of inoculation [14, 15]. 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 [15].

**Necropsy Findings:** Typically, there are no distinctive gross changes associated with listerial encephalitis. Visceral lesions occur as multiple foci of necrosis in the liver, spleen and myocardium in septicemic form and aborted fetuses. Aborted fetuses are usually edematous and autolyzed. In aborting ewes and does, there is placentitis and endometritis in addition to the lesions in the fetus. Sheep with enteritis show ulcerative abomasitis and some also have typhlocolitis at necropsy [2].

**Differential Diagnosis:** Listeriosis must be differentiated from the following common diseases affecting the nervous system: Rabies, it often occurs in a number of animals at one time due to the ease with which a number of sheep and goat can be bitten by a dog or fox. Sexual excitement, vocalization, attacking human being or each other, vigorous wool pulling, aggressiveness, hyper excitability, hyperesthesia can be manifested as a clinical signs [16].

**Gid (Coenurosis or Sturdy):** it is a disease caused by invasion of the brain and spinal cord by the intermediate stage of *Taenia multiceps*. Blindness, deviation of the head with circling in the direction of the blind eye occurs. At necropsy, thin walled cysts may be present anywhere in the brain, but most commonly found on external surface of cerebral hemisphere [2, 16].

**Scrapie:** it is a non-febrile, chronic disease of adult sheep and goats characterized clinically by pruritus and abnormalities of gait and a very long incubation period (1-7 years). It is also characterized by behavioral changes that include withdrawal from the flock, nervousness, aggression toward inanimate objects [17]. Pregnancy toxemia, it is usually suspected in late pregnant ewes and does which show nervous signs and having a history of exertion, stress or sudden deprivation of food. It is readily differentiated by the presence of ketonuria [2, 17].

**Prevention and Control:** The recovery rate is best if treatment is administered early in the course of the disease. Treatment must be administered for a prolonged period of time because recovery may take as long as a month. *L. monocytogenes* is susceptible to most commonly used antimicrobial drugs. Recommended treatments include either oxy tetracycline twice daily or penicillin-G (3 to 4 times per day for 7 days) [17, 4]. Administration of dexamethasone is recommended to treat inflammation in the brain [1].

Although the case attack rate of listeriosis is low, occasional epizootics may occur in cattle, sheep, or goat herds. These are invariably associated with high rates of environmental contamination. In such cases the hay and silage should be examined culturally for *L. monocytogenes* and spoiled feed and hay should be discarded [17]. Sanitation of pens, water supply, pasture and housing should be improved. Wild birds must be kept away from the flock as much as possible as these birds may serve as vectors for the disease. Poor quality silage should not be fed to pregnant ruminants. Silage feeding should be discontinued if an outbreak of listeriosis is confirmed [5].

In the case of abortion, isolate aborting does and ewes and send aborted fetuses and placentas to a diagnosis center for isolation of the causative agent. We have to wear latex gloves when handling placenta membranes. If a doe has listeriosis, fed kid pasteurized colostrums, milk or milk substitute. Vaccination with killed

vaccines, which do not induce an effective cell-mediated response, is not protective because *L. monocytogenes* is an intracellular pathogen. Live, attenuated vaccines, which are available in some countries, are reported to reduce the prevalence of listeriosis in sheep [1].

## CONCLUSIONS

*L. monocytogenes* is an intracellular opportunistic organism found as contaminants of environment and different materials. It can also be isolated from feces of apparently healthy animals. *L. monocytogenes* can multiply at a higher rate in poorly stored silage and rotting vegetation in which these are aerobic conditions and a pH that is higher than 5.4. It can survive at a refrigerator temperature. These contribute as a source of infection to animals so that listerial infection is most prevalent during winter (cold season). Generally, the clinical forms of listeriosis include septicemia of neonates, neonatal death, abortion, septicemia and diarrhea of ewes and neurological diseases. Based on the above conclusions, the following recommendations are forwarded.

- As *L. monocytogenes* does not multiply to any significant extent in effectively preserved silage, which is characterized by anaerobic storage, high concentration of organic acids and a pH below 4.5, the silage should be stored under these characterized conditions to maintain its quality.
- Incorporation of silage into the diet should be gradual and provision of green pasture should be encouraged.
- Susceptible animals should not be exposed to wet, cool and unhygienic environment.
- Silage that is obviously decayed should be avoided from the environment.

## REFERENCES

1. Quinn, P.J., K.B. Markey, E.M. Carter, C.J.W. Donnelly, C.F. Leonard and D. Maguire, 2002. Veterinary microbiology and microbial diseases. 2<sup>nd</sup>. Blackwell Science, USA. pp: 72-74.
2. Radostits, O.M., C.C. Gay, W.K. Hinchcliff and D.P. Constable, 2007. Veterinary Medicine: A Text Book of the Diseases of Cattle, Sheep, Pigs, Goats and Horses. 10<sup>th</sup> ed, Elsevier Health Science, USA. pp: 805-810.

3. Campero, C.M., A.C. Odeo, A.L. Cipolla, D.P Moore, M. A. Poso and E. Odriozola, 2002. Demonstration of *Listeria monocytogenes* by Immunohistochemistry in Formalin-Fixed Brain Tissues from Natural Cases of Ovine and Bovine Encephalitis. *Global Veterinaria*, 2: 38-40.
4. Wagner, M., D. Melzner, Z. Bago, P. Winter, N.E Gerbacher, F. Schilchar, A. Zangana and D. Schoder, 2005. Outbreak of Clinical Listeriosis in Sheep: Evaluation from possible contamination routes from feed to raw produce and humans. *J. Vet. Med.*, 52: 278-283.
5. Wood, J.S., 1992. Encephalitic listeriosis in a herd of goats. *Advances in Biological Research* 1(3-4): 118-121.
6. Songer and G.J. Post, 2005. *Veterinary Microbiology: Bacterial and fungal agents of animal diseases*. Elsevier Health Science, USA, pp: 88-89.
7. Hirsh, C.D., J.N. MacLachlan and L.R. Walkers, 2004. *Veterinary Microbiology*. 2<sup>nd</sup> ed, Blackwell publishing, USA. pp: 185-189.
8. Hirsh, C.D. and C.Y. Zee, 1999. *Veterinary Microbiology*. Blackwell Science, USA, pp: 255-227.
9. Carter, G.R., 1984. *Diagnostic procedures in Veterinary Bacteriology and Mycology*. 4<sup>th</sup> ed. Blackwell Science: USA, pp: 196.
10. Scanlan, M.C., 1988. *Introduction to Veterinary Bacteriology*. Iowa State University Press, USA. pp: 116-117.
11. Johnson, C.G., H.W Fales, W.C. Maddox and A.J. Romas-vara, 1995. Evaluation of laboratory tests for confirming the diagnosis of encephalitic listeriosis in ruminants. *Global Veterinaria*, 2(2): 46-91.
12. Quinn, J.Q. and K.B. Markey, 2003. *Concise review of Veterinary Microbiology*. 3<sup>rd</sup> ed. Blackwell Science, USA, pp: 26-27.
13. Krauss, H., A. Weber, M. Appel, B. Enders, D.H Isenberg, G.H Schiefer, W. Slenczka, V.A. Graevenitz and H. Zahnor, 2003. *Zoonoses: Infectious Diseases Transmissible from Animals to Humans*. 3<sup>rd</sup> ed. Elsevier Science, USA, pp: 207.
14. Quinn, P.J., M.E. Carter, B. Markey and R.G. Carter, 1994. *Clinical Veterinary Microbiology*. Blackwell publishing Ltd, Spain, pp: 171-174.
15. Carter, G.R. and M.M. Chengappa, 1991. *Essentials of Veterinary Bacteriology and Mycology*. 4<sup>th</sup> ed. Blackwell Science: London, pp: 128.
16. Murphy, F.A., E.P.J Gibbs, M.C. Horzinek and M.J. Studdert, 1999. *Veterinary virology*. 3<sup>rd</sup> ed. Academic press, Elsevier science, USA. pp: 433.
17. Smith, B.B., 1996. *Large Animal Internal Medicine*: Elsevier Health Science, USA. II: 1088-1091.