Foot Rot- An Emerging Issue in Sheep Husbandry

Asif Iqbal, A.K.Tripathi, P.U.Peer, V.S.Wazir

Division of Veterinary Epidemiology and Preventive Medicine, FVSc & AH, SKUAST-J, R S Pura-Jammu—181102 India

Abstract

Foot rot is a highly contagious and economically important disease of the feet of ruminants especially sheep and goats and is characterized by the separation of keratinous hoof from the underlying epidermal tissue resulting in severe lameness, loss of body condition and reduced wool production. Although foot rot infection has been reported in cattle, horses, pigs, deer and mouflon, the infection is specific to sheep and goats. The main predisposing factors to foot rot infection include muddy pastures, frequent rains and injury to feet. Other factors include lowland farms, high stocking densities, winter housing, routine foot trimming, and inadequate treatment and failure to isolate the affected sheep. Outbreak of diseases affects the sheep population in a huge way. Among the diseases, this may be bacterial, viral or parasitic origin in J&K. The foot rot is the emerging disease which affects the sheep population in a big way.

Key words: Foot rot, Etiology, clinical forms, Treatment and Vaccination

Introduction

Foot rot is caused by a synergistic reaction between two organisms Fusobacteria necrophorum and Bacteroides nodosa in the foot. One of them, Fusobacteria necrophorum, is nearly always present in the environment. The other, Bacteroides nodosa, can survive a maximum of two weeks if not residing in the hoof. Bacteroides nodosa produces a powerful enzyme that destroys the tissues of the hoof by migrating through the soft tissues to areas under the horn. One or two weeks of wet feet and hot humid conditions creates an ideal environment for foot rot-causing bacteria to grow. Foot injury caused by rough concrete and other hard surfaces may also wear the hoof down, leading to easy bacteria entry and growth.

The disease is dependent on a mixed bacterial infection, but Dichelobacter nodosus (D. nodosus) is the main causative agent. Dichelobacter nodosus is a slow growing, anaerobic, Gram-negative, rod shaped bacterium, with characteristic knobs at each end, and is often heavily
fimbriated with several hundreds fimbriae (10 µm) arranged in a polar distribution.

**Serogroups of *Dichelobacter nodosus***

There are 10 serogroups of *D. nodosus* designated as A, B, C, D, E, F, G, H, I and M based on the surface (K) antigen, now found to be identical with the fimbriae (Claxton, 1989; Ghimire et al, 1998). There is no cross protection between the serogroups. Virulence is proportionately associated with the number of fimbriae on a *D. nodosus* bacterium (Billington et al., 1996) which in turn is associated with the fimA gene, which encodes a fimbrial subunit protein (Kennan et al., 2001). The fimbriae of *D. nodosus* are classified as type IV because of their highly conserved amino-terminal region, polar location, association with twitching motility, and presence of an N-methyl phenylalanine residue as the N-terminal amino acid (Strom and Lory, 1993). Based on the general sequence homology, *D. nodosus* fimbriae can be classified into two distinct fimbrial classes, namely Class I or A set fimAI and Class II or D set fimAII. Fimbriae of serogroups A, B, C, E, F, G and I belong to Class I whilst those of groups D and H belong to Class II (Hobbs et al., 1991; Mattick et al., 1991). Besides serving as basis for classification, the type IV fimbriae of *D. nodosus* are recognized as a major virulence factors and are highly immunogenic.

**Prevalence over the Globe**

Although foot rot disease is prevalent throughout the world, but it has significant economic impact in those sheep farming countries that have temperate climates and moderate to heavy rainfall, such as Australia and New Zealand (Stewart, 1989). The disease has become enzootic throughout the state of Jammu and Kashmir for the last 18 years or more. The disease has been found to cause a 10 per cent production loss in body weight and wool growth in affected animals, in addition to increased treatment and control costs (Marshall et al., 1991; Glynn, 1993). Egerton and Raadsma (1991) and Marshall et al. (1991) estimated that foot rot resulted in a loss of A$ 43 million per annum in New South Wales (Australia) in terms of loss of production, treatment, control, prevention and eradication of virulent foot rot. In Great Britain more than 3 million sheep become lame annually, with more than 1 million cases attributed to foot rot (Grogono and Johnston, 1997) and farmers have recently rated lameness as the condition in their flock about which they are most concerned (Goddard et al., 2006). Nieuwhof and Bishop (2005) reported
that the most prevalent cause of lameness in sheep in United Kingdom is foot rot and results in economic loss of £24 million per annum. All breeds of sheep are able to contract foot rot; however, some of the more primitive British sheep breeds, such as Soays, are less susceptible while, popular breeds in Australia such as Merinos are highly susceptible (Emery et al., 1984).

In India, Darzi et al. (2002) were first to report the occurrence of ovine foot rot in Kashmir. However, Wani et al. (2004) were first to describe the molecular detection and characterization of \textit{D. nodosus} in ovine footrot and reported the presence of serogroup B. Very recently, Hussain et al. (2009) reported the occurrence of B, E and I serogroups of \textit{D. nodosus} in Kashmir with predominant prevalence of serogroup B.

### Clinical Forms of Disease

Although ovine foot rot represents a spectrum of clinical diseases encompassing virulent and benign extremes, in practice, three major forms (i.e. virulent, intermediate and benign footrot) are generally recognized which are caused by corresponding forms of the \textit{D. nodosus} (Stewart, 1989). The parameters for defining the clinical forms of ovine foot rot include the severity of lesions, ability to under-run horn, percentage of flock affected and propensity to self cure (Liu and Yong, 1993). The lesions of ovine foot rot are further graded by a foot score system (Stewart et al., 1982) for which some further modifications have been recently proposed (Whittington and Nicholls, 1995).

**Virulent Foot Rot** (VFR) is highly contagious and is economically one of the most important infections of sheep and goats because of its impact on livestock production and health. It is often the target of expensive control programmes. It represents a more serious and persistent form of the disease, and is characterized by a severe necrotic damage to the epithelial tissue of the hoof and extensive separation of hoof horn in more than one foot in a high percentage of sheep, leading to lameness, inability to feed, loss of body weight and wool quality (Stewart et al., 1984; Stewart, 1989). In many cases, the infection can persist for more than a year if not treated and chronically infected sheep may eventually die of emaciation due to the lameness and severe pain (Stewart, 1989). In contrast, benign foot rot causes little or no production loss (Egerton and Raadsma, 1991), and thus does not warrant intervention.
Benign Foot Rot (also called foot-scald) is a milder and less persistent form of the diseases which is characterized by a moderated inter-digital dermatitis resulting in mild lameness and has a propensity for self cure (Egerton et al., 1969; Glynn, 1993).

Intermediate Foot Rot covers a range of disease syndromes; it may be close to virulent end of the spectrum and resemble virulent foot rot clinically and in laboratory tests (so-called high intermediate foot rot). While at the other extreme it may be close to the benign end of the spectrum and show similar characteristics to benign foot rot (so-called low intermediate foot rot). Like virulent foot rot, high intermediate foot rot may cause significant economic losses to the sheep industry and needs to be dealt with accordingly. However, low intermediate foot rot does not normally result in severe disease and, is not of much economic importance.

Initially elastase test was used to differentiate \textit{D. nodosus} strains into virulent, intermediate and benign strains (Stewart, 1979). Subsequently, Palmer (1993) developed gelatin gel test for strain differentiation of \textit{D. nodosus} based on production of thermo-stable proteases. Subsequently, ELISA involving use of monoclonal antibodies; hybridization probes and PCR using virulence/benign specific primers is being used to differentiate virulent, intermediate and benign strains of organism (Stewart et al., 1990; Katz et al., 1991; Liu and Webber, 1995). Recently, Cheetham et al. (2006) developed a PCR-based assay for the detection of \textit{intA} gene; an integrative genetic element associated with the modulation of virulence (Whittle et al., 1999). There is a high correlation between the presence of the \textit{intA} gene and the ability of the strain to cause virulent foot rot.

**Symptoms**

Lameness will be observed from time to time in all flocks. This may result from foot injury, foot breakage, sheep fighting around feed bunks, rams butting ewes, sharp objects stuck in the hoof, mud, brittle hooves cracking, and injury to other joints in the legs. In more advanced cases, the foot will be red, swollen and appear moist or grayish-yellow when examined.

**Detection**

Initially detection and identification of serogroups of \textit{D. nodosus} in a footrot lesion required
isolation of the organism on trypticase arginine serine (TAS) agar medium, purification by subculturing followed by antigenic analysis by serological methods, which takes at least 3-4 weeks. However, the polymerase chain reaction (PCR)-based method using 16S rDNA specific primers was developed by La Fontaine et al. (1993) for detection of *D. nodosus* without the need to culture the organism. Subsequently, multiplex PCR was developed for determination of serogroup specificity (A-I) by Dhungyel et al. (2002) using serogroup specific primers. A common forward primer was designed from the concerned amino-terminal region of the fimbrial gene (*fimA*) and 9 (A-I) serogroup specific reverse primers were designed from the carboxy-terminal regions of *fimA* of the different serogroups which helped to determine the serological diversity of *D. nodosus* in different parts of the world.

**Treatment**

The earlier foot rot is diagnosed, the quicker it can be treated and cleared up. One method is to use a footbath which can be constructed or purchased. Construction of a footbath from a 4' :4' piece of plywood for the bottom and 2” x 6” boards on the sides will work quite well when placed in strategic spots where sheep must walk to get water. Splashing can be controlled by placing sacks or pieces of carpet or tag wool in the bottom.

Copper sulfate (20% solution) or 10% zinc sulfate can be used daily in the bath. Formalin (10%) can also be used as a bath, but not daily because of irritation to the foot. A 10% solution of formalin can be made by mixing a gallon of 37% formaldebyde to 9 gallons of water. Copper sulfate solution can be prepared by adding 32 Lbs. of copper sulfate to 20 gallons of warm or hot water so that the copper sulfate will dissolve. It has the disadvantages of staining wool and being corrosive to metal. Also, decreased effectiveness is noted when dirt and manure enter the bath. Cross & Parker (1981), most effective treatment in controlling foot rot without trimming the feet is a 10% solution of zinc sulphate. The preparation of a bath containing 8 Lbs. Zinc sulfate in 10 gallons of warm water, stirring until dissolved, results in a 10% solution. Each of these treatments can be used as a precautionary measure. None of them however, should be consumed by the sheep as toxic effects will result. Do not allow them to drink or contaminate other drinking water.

**Limitations of Treatment**
Sheep treated in footbaths of zinc sulfate, copper sulfate or formalin for foot rot should not be allowed to walk through the solutions and back through muddy areas or on grass wet with dew. This will dilute the compounds on the feet and render the treatment ineffective. Lame sheep should be separated from healthy sheep to retard the exposure to the bacteria causing foot rot. Sheep not responding to the foot bath in two weeks following treatment should have feet trimmed very closely (bleeding may occur) and a topical spray applied after the bleeding stops. The feet must be trimmed down to the healthy tissue if treatment is expected to be effective.

Sheep, not responding to treatment following footbaths and trimming with topical application, should be culled. Otherwise, they will remain as carriers and affect the healthy flock. Cull all mature animals with deformed feet, they probably are carriers. It only takes one carrier in a flock to continuously spread infection to other sheep in the pasture or dry lot.

Other Effective Measures of Control

Hydrated lime scattered in yards and pens is also effective in destroying footrot-causing bacteria. It will not replace walk-through footbath solutions.

Prevention of Foot rot

When purchasing sheep, never buy from a flock in which you have observed lame sheep. Bring newly purchased sheep home and keep them confined for two or three weeks and observe for lameness. If the animals begin to limp, examine the feet and treat for foot rot by trimming closely and applying a topical spray of 10% formalin, 20% copper sulfate or 10% zinc sulfate. If they continue to show signs of lameness, contact the person from whom you purchased the animals to negotiate with them or sell the animals rather than expose the entire healthy flock.
Quarantine

Sheep introduced to clean flocks should be quarantined for a ‘transmission period’ and their feet inspected for virulent foot rot. For infected flocks it is important that animals are quarantined for at least four to six weeks following arrival or for as long as possible. During this time they should be treated for sheep scab and resistant worms. Every foot should be inspected for foot rot. There is a danger of introducing CODD. Any lame sheep should have a diagnosis made and appropriate treatment given. Walking the quarantined sheep through formalin a number of times, for example, once every five days has shown to be effective. Culling is regarded as an essential control measure for bought-in animals that fail to respond to treatment. If any sheep are found to be lame on arrival, they should be separated and the vendor informed to discuss the diagnosis, the cost of treatment and possible culling.

A Systematic Treatment Plan To Eliminate Foot rot

1. Separate all lame sheep from the flock.
2. Run the non-lame group through a 10% formalin footbath and relocate to a pasture or lot previously unoccupied for 14 days.
3. Treat affected lame sheep by running them through a footbath containing 10% zinc sulfate to reduce the percentage of affected animals when the number is large and labor availability critical.
4. Place the footbath in a strategic area where the infected sheep will walk through it several times a day.
5. When the number of affected animals is small, feet should be trimmed prior to exposure to the footbath.
6. Approximately 75% of the affected feet will be completely healed without trimming within four weeks, if feet remain dry following footbath treatment.
7. Remaining affected animals will require individual treatment of trimming feet to expose diseased areas and subsequent treatment with a topical 10% formalin spray.
8. Run animals through clean 10% zinc sulfate footbath for two more weeks.
9. Animals not responding to treatment should be sold.
10. Purchased animals should have feet trimmed and treated topically with 10% zinc sulfate, and isolated for two weeks before introducing to the existing flock.

**Vaccination stops foot rot**

“Vaccination with Footvax provides effective treatment for infected sheep, as well as long-term protection,” says Schering-Plough veterinary adviser Paul Williams (Jennifer MacKenzie, 2005)

“A single injection of the vaccine can be used to treat foot rot because antibodies are produced against *D.nodosus*. Sheep do not produce a natural antibody response to *D.nodosus*, which means they will never develop a natural immunity to foot rot so will remain susceptible year after year. This is why vaccination is so important.

An initial 1ml injection will stimulate adequate antibody response to treat existing infections and prevent new ones for up to five months. But it is advisable to vaccinate again four to six weeks later for improved cure rates and longer on-going protection. Thereafter, an annual booster should be sufficient to keep foot rot at a manageable level.

Therapeutic vaccination is an alternative to an antibiotic injection and can be used in the face of an outbreak of foot rot. Vaccination of the flock in the autumn is advisable if a high proportion of the flock is infected at that time and there is a history of attempts at foot rot control failing due to wet land, susceptible breeds and inadequate handling facilities. A single dose of vaccine should result in a 20 per cent recovery among sheep breeds and two doses should give 80 per cent recovery. As a clinical response is seen in two to three days, vaccination can be an effective alternative to foot bathing particularly where facilities are poor. Protection is obtained within three to four weeks. Vaccination used in conjunction with other treatments should break the cycle and is part of a good treatment programme.

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