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Sheep lameness: causes, types and treatment options

Abstract

This article will give an overview of the main causes of lameness in sheep, focusing on the three most common diseases present in the UK, which are all of infectious nature: interdigital dermatitis, footrot and contagious ovine digital dermatitis (CODD). The most recent and relevant findings regarding the aetiological agents are discussed. A refresh on the options available for diagnosis and control, specifically on the role of the vet in dealing with lameness at a flock level, is also presented. Finally a summary of the choices available for treatment is given.

Key words:

Lameness, sheep, footrot, CODD, disease control.

Sheep lameness is rightly recognised as one of the major health, welfare and production limiting condition. The main causes of sheep lameness in the UK are infectious diseases. Interdigital dermatitis (ID) and footrot have been on the podium for quite some time, although contagious ovine digital dermatitis (CODD) seem to have become more prevalent over the years [1]. These three conditions alone account for over 90% of cases of lameness in the UK [2] and are almost always described at a flock level. Other causes of lameness which are commonly encountered in clinical practice, but either have much lower prevalence or account for individual problems, are white line disease (shelly hoof), toe abscess and toe granuloma. Table 1 gives an overview of other causes of sheep lameness currently identify in the UK.

Causes

The dogma we have become familiar with through lectures and textbooks, is that ID is caused by *Fusobacterium necrophorum* and benign footrot, although clinically indistinguishable from ID, is caused by *Dichelobacter nodosus*, with virulent strains causing a more severe clinical presentation, with under-running of the sole and exposure of sensitive tissue. Recent evidence, though, seem to suggest that interdigital dermatitis and footrot are, in fact, two stages of the same disease. The causative agent, in both cases, is *D. nodosus*, with *F. necrophorum* acting as opportunistic pathogen [3].

What is important to remember about *D. nodosus* is that without the right conditions, such as a wet and warm environment, high stocking rate and initial damage to the interdigital skin, it is not able to cause disease. Furthermore, the bacteria can only survive on ruminant feet, and can survive in the environment for up to 14 days. This has important implications for control of the disease, as without introducing sheep carrying the bacteria, there should not be any footrot/ID in the flock. Furthermore, it is also important to remember that not all strains present the same virulence and therefore the same severity in clinical signs.

Regarding CODD, the general consent seem to be that *Treponema* sp. are the causative agent of the disease. Again, *D. nodosus* has also been isolated from cases of CODD [4] as well as *Treponema* been isolated in cases of footrot [3], which is suggesting that what we describe as footrot might actually

be a multifactorial disease with multiple microorganisms involved having a synergistic role and possibly different clinical presentations.

Diagnosis

Regardless of the causative agent, one of the main challenges is the prompt and effective recognition of lame sheep to allow for immediate treatment of as many cases as possible as well as being able to identify those that reoccur after treatment.

Although footrot is considerably the biggest problem, it is worth knowing that is also the most common lesion incorrectly named by farmers, as shown in a survey were many farmers would tend to name any hoof horn lesion as footrot and white line disease being the most misdiagnosed condition [2]. This suggests that veterinary involvement should be seek out to confirm diagnosis and obviously to implement a treatment and control plan. Clinical aspect will usually be sufficient for a diagnosis (Figure 1-3), but for definitive confirmation the best option is to submit a swab from the lesion for PCR or bacterial culture.

Another fundamental point is knowing the extent of the problem. Before embarking in any control program, it is necessary to have a clear idea of the prevalence of lameness. Number of sheep affected, age group, speed of onset and degree of lameness are all important questions that need addressed. Prevalence of lameness seems to have dropped from 10% to 5% in the last few years [1], which is in line with the 2011 Farm Animal Welfare Council Opinion and probably a good indicator of an achievable target for commercial flocks. A further reduction to 2% was set as a 10-year goal. Recently there has also been a growing interest in using modern technologies to improve farming practice, with methods like infra-red technologies [5] and radar sensors [6] exploited to aid in recognition of lameness.

Control

Once a clinical diagnosis is reached and the extent of the problem is clear, some practical questions should be considered. How often should sheep be observed for lameness and therefore gathered for treatment? How can all lame sheep be identified at each single observation and how practical it is to catch them? What degree of lameness is “acceptable” before prompting intervention? And is this going to be a flock treatment or a targeted individual one? The scientific evidence suggest that catching mildly lame sheep within 3 days of first becoming lame is associated with a decrease prevalence of lameness [1]. It also suggest that targeted individual treatment is considerably better than flock treatment using foot bath [7]. From this, it would be recommended to regularly (e.g. once to twice a week) inspect sheep for lameness, at each observation catch every sheep even showing mild degree of lameness (score 1 and above) and individually treat them. While this might be the gold standard of a lameness control program, it might not work for all farmers. The key, as always, is to work with them in setting achievable goals and propose solutions that are practical and can fit with their existing system.

Other options that should be considered are included in the “Five point plan” [8]. One is the always fundamental quarantine of all incoming stock (which includes both purchased and returning stock) and which should be applied anyway as a baseline biosecurity measure in every flock. A licensed vaccine for footrot is also available, with recommendation of vaccination of all stock implemented at critical time of the year (e.g. housing). Culling repeated and worst offenders as well as breeding from more resistant animals is another option to reduce incidence of lameness in the flock. Regarding foot bathing, if best practice is followed, it can definitely play a role in the control of lameness [7]. Handling facilities (race and penning) have to be excellent, all sheep should remain in the bath for

the recommended length of time (which depends on the chemicals used and on the addition of a surfactant) and all sheep are required to stand on a clean, dry and possibly hard area for at least half an hour after footbathing. If all these measures are not in place, then this practice can actually make the problem worse, by increasing spread of the disease. The frequency of foot bathing is another point to consider, with suggestions of a weekly frequency being a solution for elimination/treatment of the disease [9] and regular (few times a year, at housing and gathering) for prevention. Finally, chemicals that can be used are either formalin or zinc sulphate.

Treatment

There is now strong evidence that early treatment of individual sheep affected by footrot with parenteral antibiotics is the best and possibly the most cost-effective option [10]. The active principles that have shown efficacy are oxytetracycline (20mg/kg), gamithromycin (6 mg/kg), tilmicosin (10mg/kg) and florfenicol. Topical antibiotic sprays containing oxytetracycline, are also an option for mild cases and to reduce further environmental contamination.

At the same time, the absolute ban on trimming feet as a treatment option for footrot [11] is now well established, with a very positive farmer uptake on this message [1]. Although research has focused mainly on footrot, it is reasonable to assume that similar sound advice would be applicable for CODD. Avoiding trimming affected feet to limit the spread of the disease and aim instead for targeted parenteral antibiotics is likely to be the best option. CODD cases do not seem to respond to either formalin or zinc sulphate foot bathing, while long-acting amoxicillin (15mg/kg), oxytetracycline (20mg/kg) and tilmicosin (10 mg/kg) have all been shown efficacy.

Conclusions

As for any other disease to tackle, first of all you need to know your enemy. Veterinary involvement is crucial in dealing with lameness, both for definitive diagnosis as well as tailored advice. As usual, the need for a health plan with clear guidelines of first line treatment and control measures (from biosecurity for infectious diseases to specific lameness control) is a must have. We also need to remember we are working with our clients and whatever advice will be given has to fit with an existing system and a busy schedule. Data collection (diagnosis, prevalence of lameness and lameness scoring), a clear idea of the resources (routine treatments, handling facilities and available labour) and a set of sensible and achievable goals are the basis for a successful collaboration and for the control of this significant production-limiting condition.

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Table 1 – Other causes of sheep lameness currently identify in the UK.

Name	Cause	Flock prevalence	Treatment	Lameness
White line lesions (shelly hoof)	Non-infectious (nutritional?)	Variable	Trimming	Mild
Toe abscess	Infectious (F. necrophorum, T. pyogenes)	Low	Trimming, (antibiotics?)	Severe
Toe granuloma	Non-infectious (over-trimming)	Low	Removal and bandage/cauterization	Variable
Septic pedal arthritis	Infectious	Low	Arthrodesis, amputation or joint lavage	Severe
Joint ill	Infectious (S. dysgalactiae)	Variable	Management and hygiene	Moderate to severe
Osteoarthritis	Non-infectious	Low	Pain management, culling	Variable
Infectious polyarthritis and post-dipping lameness	Infectious (Erysipelothrix rhusiopathiae)	High	Antibiotics	Severe
Strawberry footrot	Infectious (<i>Dermatophilus congolensis</i> and ORF)	Variable	Vaccination, antibiotics	Mild
Interdigital hyperplasia	Non-infectious (hereditary)	Low	Breeding/culling	Variable
Interdigital foreign body/soil manure bailing	Non-infectious	Low	Removal	Variable

White muscle disease (delayed)	Non-infectious (selenium deficiency)	Variable	Selenium supplementation	Variable
Rickets	Non-infectious (vit D deficiency)	High	Vit D supplementation	Moderate to severe
Horn grooves and cracks	Non-infectious (stress or poor nutrition)	High	Management	Mild
Long bone fracture	Non-infectious	Low	Cast	Severe

Figure 1. Interdigital dermatitis. There is inflammation and sloughing of the superficial layers of the skin at the interdigital space.

Figure 2: Virulent footrot. There is progression of the disease into under-running of the sole and separation of the hoof wall from the underlying tissue.

Figure 3: CODD. A) Lesions start with inflammation and ulceration at the coronary band. B) Lesions progress to separation at the skin-horn junction of the coronary band and under-running of the horn down to the sole.