

Recognizing and Managing Infectious Causes of Lameness in Cattle

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Lameness in cattle is a common condition that can result in significant economic loss to a producer through a variety of mechanisms. Lamé cows do not eat as much as healthy cows and thus produce less milk or less gain. They may become anestrous. Furthermore, they may be prematurely culled due to low milk production, delayed conception or emergency slaughter.

In a comparison with the other most common clinical conditions of dairy cattle, lameness was estimated to be the most costly on a herd basis.¹ Estimates were derived from published reports and data from the records of the Ambulatory Clinic at Cornell University. With an average incidence of 30 cases per 100 cows per year; a case fatality rate of 2%; involuntary culling of 20% of cases; average increased days open 28; treatment costs including veterinary fees, drugs and farmer labor of \$23 per case; the total cost per 100 cows per year is estimated to be about \$9,000.

The infectious causes of lameness common in North America comprise 3 distinct entities. The terminology for these conditions may be confusing with many synonyms in common use but footrot, interdigital dermatitis and digital dermatitis are generally understood by most veterinarians. There is often overlap in the clinical picture presented since a cow may be simultaneously infected with the organisms causing each of the disease entities. This paper will describe these 3 diseases to enable practitioners to differentiate them since treatment and prevention options vary with disease.

Footrot

The first written description of a disease of cattle was footrot.² It is easily recognized as an acute problem differentiating it from digital and interdigital dermatitis. The classic signs familiar to all bovine practitioners are symmetrical swelling of the limb above the claws and acute pain. The animals may have a fever and be unwilling to bear weight on the affected limb. Many cases will have a fissure in the interdigital skin with varying degrees of necrosis of the proximal soft tissues and a characteristic "Limburger cheese" odor.³ Several bacte-

rial species are commonly isolated from clinical material. Current evidence supports *Fusobacterium necrophorum* as the primary pathogen with supporting roles played by *Bacteroides melaninogenicus* and *Actinomyces pyogenes*.⁴ *F. necrophorum* is a normal part of the ruminal flora but there may be strain differences from those causing footrot. Conditions predisposing to development of disease are environmental moisture resulting in maceration of the interdigital skin and sand or stones that may cause mechanical skin damage. The treatment of footrot with parenteral antibiotics has long been recognized to be effective.⁵ Additional modes of therapy may include debridement of necrotic material and topical dressings with antibacterial preparations.⁶ Most unattended cases resolve spontaneously in about a week as the immune response overcomes the bacteria and their toxins.⁷ Some neglected cases may progress to irreversible debility if the digital joints or flexor tendons become involved. Prevention has been relatively easy to achieve with routine footbathing in antiseptic solutions (5 to 10% aqueous solution of copper sulfate or zinc sulfate or 5% formalin) or powders (anhydrous lime or lime and copper sulfate mixtures) and restriction of access to mudholes and low lying pasture.⁸ In the United States, there is a vaccine licensed for prevention of footrot (Volar, Miles Laboratories, St. Louis, MO) but little supporting data from clinical trials to assess efficacy. The feeding of EDDI⁹ or tetracyclines¹⁰ have been promoted as preventive measures for control of footrot but are not widely used at the present. Routine footbathing has provided good control under most circumstances for dairy cattle.

Interdigital Dermatitis

A chronic dermatitis of the interdigital skin that often progresses to the development of lesions of the heel bulbs and heel horn is very widespread in adult dairy cattle.¹¹ Surveys of Dutch cattle indicated that the majority of cattle at any point in time have lesions indicative of infection but not necessarily clinically lame. In my experience, this infection is nearly ubiquitous in New

York dairy cattle. The prevalence and severity increase with age. Clinically lame cows are typically in early lactation as with most cases of lameness. The mechanism for this is probably a combination of the stresses of the periparturient period and the documented change in the pattern of weight distribution on the claws of the rear limbs following calving.¹² The dermatitis is manifested by mild local sensitivity of the interdigital skin. There is often some hypertrophy and roughening of the affected skin accompanied by a white to grey exudate with a sour-milk smell. The interdigital skin may become inflamed to the point of detectable lameness. This condition has been referred to as winter foul. Perhaps a more common presentation for a lame cow is with heel cracks. Affected cows may stand with their heels suspended over the manure gutter or noticeably favor one leg when walking. Despite the obvious lameness in the affected digits, both rear limbs or all 4 limbs are frequently involved. You might know the cause of this disease as *Bacteroides nodosus* but DNA homology testing led to the creation of a new genus in 1990 so it is now *Dichelobacter nodosus*.¹³ *Actinomyces pyogenes* is also commonly present in the lesions. Heel cracks may progress to a more severe cause of lameness by undermining of the sole horn with the development of a sole ulcer or to heel horn erosion. Another consequence of chronic heel cracks is hypertrophy of the skin of the heel bulbs or the development of an interdigital fibroma. This was described by Toussaint Raven¹¹ and is easily recognized by the asymmetry of the fibromas; they are more prominent of the side of the claw with the most severe heel crack.

In the herds involved in the Dutch survey and in the herds that I regularly see, footbathing is a common and routine practice. The organisms involved in interdigital dermatitis are relatively sensitive to most footbath compounds. Why is the infection so prevalent? My theories are: that the interdigital skin may not be thoroughly exposed to the compound due to the opposing pressure of the skin folds between the digits, or if a crack is present the antibacterial agent does not penetrate very deeply during the passage through the bath. In many herds, only the lactating cows are footbathed and the dry cows can develop cracks which are deep enough to maintain the infection despite subsequent bathing. Thus routine trimming with appropriate attention to the heels plays an important role in prevention of disease due to interdigital dermatitis. The same footbathing solutions used for footrot have been less effective in controlling lameness due to heel cracks and their sequelae perhaps due to the factors just mentioned. Treatment of lame individuals requires debridement of skin flaps and overrun sole horn and topical treatment with the antibacterial of your choice. In most cases affecting the rear limbs, the lateral claw is more severely

affected including overgrowth due to induced hypertrophy and reduced wear from an altered way of going. Balanced weight bearing of both claws should be reestablished by trimming. As with cases of sole ulcer due to laminitis, hoof blocks can be very beneficial in returning comfort to cows with severe complications.

Digital Dermatitis

There has been considerable discussion recently of a disorder causing lameness in cattle that is of as yet unresolved etiology. The condition goes by the name of hairy heel warts, strawberry foot, verrucous dermatitis, digital warts, interdigital papillomatosis and probably most correctly digital dermatitis. The condition is identical to that described by various authors in Europe and North America and is known as Mortellaro after one of the Italian writers describing it in 1974.¹⁴ We saw the condition as a few isolated cases in 1979¹⁵ and then it disappeared until the mid eighties. Since then it has developed into a very common condition involving most freestall housed herds in New York. Within the last 3 years the disease has developed to epidemic proportions in northern Europe and spread throughout the United States. One wonders why a disease that was reported originally in 1974 suddenly spread worldwide in dairy cattle in the last few years.

The earliest lesion recognizable as digital dermatitis is a reddened circumscribed area typically just above the interdigital cleft on the plantar aspect of the pastern, the strawberry lesion. The most striking feature of the lesion is the degree of pain expressed by the cow. Hairs at the periphery of the lesion are often erect and matted in exudate to form a rim. As the lesion progresses focal hypertrophy of the dermis and epidermis leads to raised conical projections appearing much like wet, grey terrycloth. In even later stages papilliform projections of blackened keratin may extend 10 to 15 mm from the surface, the hairy wart stage. Many cows have simultaneous infection with *Dichelobacter nodosus* leading to significant erosion of the horn of the heels in a hemispherical pattern surrounding the axial space. The hoof may be noticeable overgrown from reduced wear caused by the altered use of the limb. Interdigital fibromas regardless of cause are commonly infected with digital dermatitis in endemic herds. In my experience, after digital dermatitis has been present in a herd for a year or so most cases of lameness are found in the first lactation animals even though lesions may be seen on the digits of older cows during routine hoof trimming.

Dutch researchers observed a spirochete in specially stained samples of lesions in 1981.¹⁶ There were reports in 1992 from Deryck Read of the San Bernardino Diagnostic Laboratory in California that a spirochete had been identified in material from cases there.¹⁷

Researchers in Ontario have found a spirochete on the digits of healthy cows in affected herds and in herds without evidence of digital dermatitis.¹⁸ I cannot offer any insights into this problem of etiology since our efforts to identify viral pathogens over the last few years have all turned up negative. After early histological and ultrastructural studies revealed a lesion similar to that caused by a papilloma virus we employed bovine papilloma virus gene probes on tissue specimens from many cases and found no evidence of papilloma virus DNA in any.¹⁹ This work has been repeated by Deryck Read and collaborators with the same negative results.¹⁷

After a sabbatical leave in Utrecht were Mortellaro was commonly seen and routinely treated with a topical spray consisting of tetracycline and gentian violet I evaluated topical therapy in New York herds. I was skeptical since I had not been in a position to follow up on cases seen in Utrecht and none of the cases I saw or heard described were proliferative to the hairy wart stage. Additionally, most of my experimental efforts at therapy resulted in return of the lesion if the cow was followed for sufficient time. I tried various disinfectants and caustic chemicals, cryosurgery, electrocautery and excision. Some of the procedures gave immediate pain relief but the lesion recurred after all of them in from 3 to 6 weeks. We had been recommending topical treatment with formaldehyde solutions and routine footbathing with 5% formalin as an acceptable control program with generally good results. No one really likes working with formalin and my colleagues in Vermont informed me that it had been forbidden for footbathing by their state environmental regulatory officials.

Going back to my experiences in the Netherlands, I employed topical oxytetracycline in the form of 5 to 15cc of injectable oxytetracycline applied on a cotton dressing with a flimsy wrap. I have examined many of these cows after 2 to 5 days and have been amazed at the regression of the lesion and complete elimination of pain. Since the response is so rapid I am using less and less of a bandage so that the cotton will fall off in a few days. Secondly, I have been recommending that my clients use a 0.1% oxytetracycline foot bath on a twice weekly basis and have found herd level control to be adequate. In the United States this is an extra label drug use and must be undertaken with veterinary involvement. The cheapest current source for us is a poultry water medication that is 70% active ingredient and goes by various names including "324" (324 grams per pound). In addition, another once weekly foot bath of 5 to 10% copper sulfate seems to provide acceptable control of footrot and interdigital dermatitis. Herds that have been using this program for almost 2 years describe mixed success at present. Some report continued excellent control unless circumstances force a lapse in footbathing. Others claim that control seems to be less effective now than when

the program was begun. Those instances of reported failure that I have investigated personally seem to have been a failure of implementation rather than true resistance of the pathogens to the process. Perhaps complacency led to less effective programs. I have discussed problems with some dairymen who have increased the footbath concentration to 0.4% and found this effective. Individuals treated with topical tetracycline although improved immediately may relapse to lameness in 5 to 7 weeks if no herd preventative program is in place.

These results do not prove the precise etiology but seem to provide a reasonable means of treatment and control. I have discussed the food safety concerns with representatives from manufacturers of oxytetracycline and we agree that likelihood of residues is negligible. There remain many unanswered questions regarding digital dermatitis but I hope these remarks help others in dealing with this increasingly prevalent problem.

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