

Outbreak of teat lesions in a herd in Northland

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An outbreak of teat lesions with an acute onset occurred in a herd of 300 milking cows, three and a half months into the calving season of 2003. Nearly 40% of the herd was affected. Affected cows all had similar lesions and the number of infected cows increased over a 1-2 month. Teat skin changes ranged from papules through vesicles and ulcerations to scabs that developed quickly. Lesions were primarily localised on the teat skin, but were evident on the udder skin in about 30% of cases. The final diagnosis was not established. A presumptive diagnosis of bovine herpes mammillitis was made based on the incidence of lesions and clinical findings. Virus isolation and serology were not attempted.

Introduction

Teat lesions are common in dairy cattle in New Zealand. Most of the infectious cases are caused by pseudocowpox virus, but lesions similar to those caused by bovine herpesvirus (BHV)-2 have been described clinically and confirmed serologically in New Zealand (Horner and Raynel, 1988). Presumptive diagnosis of the disease has been reported previously by Daniel (1970). Since then there have been no articles published in this country on this disease, so a review article and clinical case presentation may be timely, if the disease has reappeared here. Bovine herpes mammillitis (or bovine ulcerative mammillitis) is an acute viral disease in cattle caused by BHV-2 and BHV-4 (Hillerton et al., 2001). Bovine herpes viruses are members of the genus Simplex virus and the subfamily Alphaherpesvirinae within the Herpesviridae family. Bovine herpes viruses 2 and 4 often infect young heifers and young cows at first parity or in their first lactation. Lesions are clinically and epidemiologically similar for the two BHV types, which are distinguished serologically (Hillerton et al., 2001). Bovine herpes virus-2 is closely related, if not identical, to the Allerton virus, the causative agent of pseudo-lumpy skin disease. Outbreaks of bovine herpes mammillitis in the Northern Hemisphere usually occur during the autumn (Gibbs et al., 1972) and winter months. Disease may be epidemic or endemic, and is usually self-limiting. Outbreaks may last between 6 and 15 weeks and immunity lasts for at least 1-2 years after an outbreak. In a completely susceptible herd, the disease spreads quickly and nearly all cows become infected over 1 to 2 months. Introduction of BHV into a naïve herd via replacement stock can result in lesions in cows of any age. Latent infections may occur (Hillerton et al., 2001), and latently infected older cows are the most likely source of infection for recently-calved heifers (Turner et al., 1976; Letchworth et al., 1982). BHV may be spread from cow to cow by milking equipment or on the hands of milkers (Gibbs et al., 1972), or via contaminated salves and ointments. Biting flies have also been incriminated as a possible vector. Experimentally, deep inoculation of virus into the teat wall is required for

reproduction of the disease.

BHV-s produce intranuclear inclusions and syncytia in infected cows. The incubation period is 5-10 days and there is no systemic illness. Disease may be sub-clinical or relatively mild, but can also be severe. Bovine herpes mammillitis is initially characterised by a painful and oedematous swelling and erythema of the teat skin (Turner et al., 1976; Hillerton et al., 2001). Recently-calved heifers are usually most severely affected (Letchworth et al., 1982). Initial lesions are followed by the development of irregularly-shaped vesicles (0.5 to 5 cm in diameter) which usually rupture within 24 h, probably aided by the milking process (Letchworth et al 1982), leaving an ulcerated surface (Hillerton et al., 2001). Confluence of vesicles or ulcers is common leading to extensive sloughing of skin. There is often secondary bacterial infection of the affected area (Hillerton et al., 2001). Lesions can range from vesicles and ulcerations of large (up to 10 cm) areas of teat skin to single small plaques of oedema 2 to 3 cm wide (Letchworth et al., 1982). In uncomplicated cases, ulcers heal in 2 to 6 weeks by granulation and epithelisation, with extensive scab formation and temporary depigmentation. Some cows become slow milkers, but show no physical lesions. The function of the keratin lining of the teat canal may be impaired, leading to increased susceptibility to mastitis. Calves suckling an infected dam often develop lesions in the mouth (Gibbs et al., 1972). Diagnosis is by virus isolation or serology (Gibbs et al., 1972; Letchworth et al., 1982; Turner et al., 1976). As far as the author is aware, neither BHV-2 nor BHV-4 have yet been isolated in New Zealand. Samples for virus isolation are best obtained by aspiration from early vesicles but virus isolation in many cases is unsuccessful. However, nowadays, virus identification nowadays can be conducted using a PCR test. Demonstration of high serum neutralisation titers, historically, appears to be a more successful and practical method of diagnosis (Letchworth, 1982) because recovered animals show high antibody titres. Bovine herpes viruses-2 and -4 do not appear to cross-react to any significant extent, so serological testing for both virus types should be conducted. Serological testing is not routinely available in New Zealand. The virus is held at the National Centre for Disease Investigation (NCDI) at Wallaceville, Upper Hutt, and it may be possible to arrange for virus neutralisation assays to be carried out on cases in such a severe outbreak. The failure to diagnose the causative agent in this case was due to a lack of information and communication, and the author strongly advises veterinarians or laboratory staff investigating any similar cases in the future to contact virologists at Massey University or the NCDI to arrange for definitive diagnostic testing. The main differential diagnosis is pseudocowpox, which is often also present in herds affected with bovine herpes mammillitis.

Dual infections of individual cattle have been reported (Gibbs et al., 1972).

No specific treatment is available. Supportive therapy includes iodine sanitizers and applications of emollient creams. Products containing methylene blue (that are commonly used to treat wounds on horses) have been helpful in some instances. In severe cases, pre-term drying-off affected cows should be considered. Patient and careful complete milking is necessary to prevent mastitis.

The disease is probably endemic in New Zealand, but is poorly documented and generally considered to be mild and sporadic. This report details the clinical findings, control measures and outcome of an acute outbreak of teat lesions affecting 38% of the herd over a period of 3 months, of what was presumed, but not confirmed, bovine herpes mammillitis on a farm in Northland region of New Zealand.

Case History

The case occurred in a herd of 300 milking cows about three and a half months into the calving season of 2003. Milking commenced in a newly-built milking shed at the end of July 2003 (when calving started). Thirty cows had been introduced into the herd just prior to the start of calving, all purchased from one farm. All of the introduced cows had rough teat skin and signs of previous teat sores, but none showed any active teat lesions. All cows calved on the farm before the middle of October 2003.

A hydrolysed linseed fatty-acid-based teat spray with 10-15% added emollient was used as a teat spray and routinely applied to the teats of all cows immediately after milking. The bulk tank milk somatic cell count (BTSCC) from July 2003 to the end of September 2003 averaged 85,000. At the beginning of October 2003, milking frequency was reduced from twice to once per day as feed supplies were limited. The BTSCC at the end of October 2003 was 145,000.

The first teat skin lesions were detected at the end of September, approximately 9 weeks after the start of calving. Eleven cows were affected in the first 3 weeks and 47 cows were affected over the subsequent 11 days. As a result, there were 58 affected cows (19% of the herd) in total at the time of the first veterinary visit, which took place approximately one month after the outbreak began.

Clinical Findings

Papules and, sometimes, swelling of affected teats were the first signs of infection, followed the next day by the appearance of vesicles with clear fluid or purulent discharge. Vesicles burst during milking and ulcers formed, followed by scab formation beginning at the periphery of lesions within 2-4 days. Lesions were elevated from the teat surface at all times and took 3 weeks or longer to resolve. In the literature elevation is rare; mainly depression of the lesions is registered. Rough skin, small scars and temporary depigmentation remained after resolution of lesions.

The majority of lesions were localised to the teats, but lesions on the skin of the udder were evident in about 30% of cases, and these differed in appearance from those on the teats. On the udder, papules were present for 2 to 3 days before small vesicles formed in the middle, which burst 3 or 4 days later; lesions took longer to recover on the udder than on the teats. Based on the clinical findings, nature, and distribution of the teat lesions, a presumptive diagnosis of bovine herpes mammillitis was made.

Control Measures and Clinical Outcome

My recommendations in this case were as follows:

1. Affected cows were separated from the herd as soon as lesions became apparent and milked last.
2. Milking machine cups and clusters were disinfected using an iodine-based sanitiser after milking each affected cow.
3. The teat spray used after each milking was changed to an iodine-based product with added emollient, mixed at above normal concentration.
4. Teat lesions were treated with the topical application of an antiseptic ointment containing emollient.
5. In addition, the milking machine was tested to ensure efficient operation and that no vacuum fluctuations or retrograde flow of milk occurred.

After the instigation of these control measures, the incidence of new cases decreased. One month following their establishment, 54/58 cases had healed completely and about 45 new cases had occurred, but with milder signs and quicker recovery. The last recorded new case was in the middle of December 2003, approximately 11 weeks after the first case was recorded. Lesions on the udder were not evident in any of the new cases identified after control measures had been instigated. In total, 113/300 (38%) cows were affected. Mastitis was not recorded in any of the affected cows and there was no apparent effect on the health of affected cows or reproductive performance of the herd, which commenced mating at the peak of the outbreak. The report in *New Zealand Veterinary Journal* (Petrovski 2005) is the first I am aware of to document an outbreak of this magnitude of what was presumed to be bovine herpes mammillitis in a dairy herd in New Zealand. Further studies are required to establish the identity and serotype of the causative agent and to define the prevalence of teat lesions caused by BHV in New Zealand.

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