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 mammmilitis 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 (Horne 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 herpesvirus (BHV) (or bovine ulcerative mammmilitis) is an acute viral disease in cattle caused by BHV-2 and BHV-4 (Hillerton et al., 2001). Bovine herpesviruses are members of the genus Simplex virus and the subfamily Alphaherpesvirinae within the Herpesviridae family. Bovine herpesviruses 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 herpesvirus 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 1-2 months.

Introduction of BHV into a naïve herd via replacement stock can result in lesions in cows of any age. Latent infection 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 sleeves and cointments. 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 mammmilitis 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 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 titres, historically, appears to be a more successful and practical method of diagnosis (Letchworth, 1982) because recovered animals show high antibody titres. Bovine herpes-virus-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 mammmilitis.
Dual infections of individual cattle have been reported (Gibbs et al., 1972).
No specific treatment is available. Supportive therapy includes
iodine sanitisers 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 test lesions affecting 39% of
the herd over a period of 3 months, of what was presumed,
but not confirmed, bovine herpes papillomas 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
continued 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 test skin
and signs of previous test sores, but none showed any active
test lesions. All cows calved on the farm before the middle of
October 2003.

A hydrolysed lincensed fatty-acid-based test spray with 10-15%
added emollient was used as a test spray and routinely applied
to the teats of all cows immediately after milking. The bulk tank
milk somatic cell count (BTSCCC) from July 2003 to the end of
September 2003 averaged 35,000. At the beginning of October
2003, milking frequency was reduced from twice to once per
day as feed supplies were limited. The BTSCCC at the end of
October 2003 was 145,000.

The first test 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 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 test 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 90% 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
test 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 test spray used after each milking was changed to an
iodine-based product with added emollient, mixed at above
normal concentration.
4. Test lesions were treated with the topical application of an
artesipate 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 instituted. In total,
113/300 (37%) 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 2006)
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 test lesions caused by
BHV in New Zealand.

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