Non-Lactating Cattle Mastitis: Prevalence, Pathogenesis, and Prevention: A Review

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Introduction

Mastitis is the most costly disease of dairy cattle. This disease occurs when bacteria penetrate the teat orifice, colonize teat canal keratin and gain entry into the mammary gland.[8,13] More and more researchers are finding that heifers that haven't even calved yet are an important reservoir for many of the contagious mastitis organisms which are found in most dairy herds.[13] At the International Symposium on Bovine Mastitis which was held in September of 1992, three papers addressed heifer mastitis and the kinds of organisms in the udders before first calving.[14,13,15] Heifer mastitis is defined as an intramammary infection at first freshening, either subclinical or clinical in nature.[14] The udder health of heifers upon entering the milking herd is very important.[2,5,13] These animals impact future production and milk quality and it is imperative that heifers begin lactation with a low prevalence of intramammary infection. A summary of several recent studies exploring the prevalence of intramammary infection at freshening in first calf heifers is listed in Table 1 below.[7,14]

Table 1. Intramammary Infection (IMI) at First Calving

Reference	Number Quarters	Number Heifers	% IMI By Pathogen--- Neg	CNS	Sa	Env	Other
Nickerson,*et al*; 1992				34	12.3	20.1	
Cook, *et al*; 1992		525	43	43	6		8
Bray, *et al*; 1989[2]		265	23.2	23.2	5.4	3.8	
Pankey*et al*; 1991		382	54.4	22.8	2.6	14.9	5.2
Oliver and	128		68.8	18.8	.8	13.5	.8
Gross Approximate Averages				28.4	5.4	13.0	4.6

Neg= No IMI; CNS = Coagulase negative staphylococci; Sa = *Staphylococcus aureus*; Env = environmental pathogens, streptococci other than agalactiae and coliforms; other = All other pathogens. Samples collected 3 weeks prior to parturition.

From the table it would appear that almost 30% of all heifers freshen with coagulase negative staphylococcal infection and approximately 20% freshen with a major mastitis pathogen. Another study found 96% of heifers were culture positive at first calving.[13] What are the dynamics of heifer mastitis, or in other words, what could be the pathogenesis for heifer mastitis; and, is there any similarity between this mastitis complex and other mastitis complexes affecting dairy cattle worldwide?

Theories of Pathogenesis

The introduction of mastitic organisms into primigravid heifers has a very similar temporal relationship to the summer mastitis complex which is prevalent throughout most of the dairying areas of the world.[2] There are reports from Sweden, Canada, Ireland, England and Japan which describe mastitis in heifers and in dry cows which occurs during the summer and is caused by a very similar range of organisms to those which have been reported in studies in the United States in heifers in Tennessee, Kentucky, Louisiana, Vermont, and Washington state.[1,2,3,4,5,6,7,8,9] Another striking characteristic of this heifer mastitis is that the prevalence of the mastitis increases with the length of the summer. This fact is ascertained from the increase in prevalence in southern studies in the United States versus studies done in the northern United States.[13,2]

The literature reveals that the types of organisms present in the udder in both the heifer mastitis complex and summer mastitis complex are organisms which are commonly found on the skin and in the environment of the animals.[2,5,6,4,12,9] Something then must be transferring these organisms from the skin surfaces to the teat ends where the organisms is able to enter the teat end. When one looks at the temporal relationship between summer mastitis and heifer mastitis, it becomes apparent that the time of transmission correlates well with the highest numbers of biting flies present in the environment.[11] It has been proposed by many other authors and is being proposed by this author that biting flies transmit heifer mastitis and summer mastitis when they feed on the teat ends by contaminating the teat end with various skin organisms and by lacerating the teat end providing a media for bacterial growth (when they feed on the teat ends).[10,8,11,6]

Whether flies and other biting insects are merely

mechanical vectors or whether they provide a more important role as both mechanical and biological vectors is still up for debate. It does appear that it is necessary for the teat end to be injured in order for the bacteria to fully colonize the teat end prior to invading the teat sphincter.[10] In a brief report by Terry and others in 1978, summer mastitis was successfully transmitted by making needle punctures at the end of the teats of uninfected animals.[10] Nevertheless, the conditions under which the infections became established remained unclear because they were unaware of how the bacteria which induced the mastitis could have reached the teat ends. This question may have been answered by Hillerton and All when they showed that a common biting fly is able to harbor the bacteria in its gut and the bacteria involved were even able to stay viable on the surfaces of the flies for extended periods of time.[6,11] It may not be clear as to what type of vector role the biting fly plays, but many reports substantiate that flies play an important part in these disease processes by feeding on the teat ends.[6,11]

Control

Control of this heifer mastitis and summer mastitis complex will revolve around control of the vector, the biting insect. A study by Trinidad, *et al.* revealed that intramammary treatment of primigravid heifers greatly reduced the prevalence of mastitis in these heifers at calving.[7] However, intramammary treatment must be begun early enough to prevent damage to the udder and it must be administered with enough frequency to prevent udder damage if reinfection should occur at a later date during gestation. An additional problem with intramammary treatment of heifers is that these heifers could freshen and have levels of antibiotics in the milk which could contaminate the milk supply. Also, it is very difficult to introduce a treatment cannula into the small teat orifice on an unbred heifer. Treatment for this problem may need to begin at a time before the heifer is even bred. Table 2 shows the prevalence of intramammary infections at breeding age and calving in primigravid heifers on seven Vermont dairy farms. This information leads one to believe that proper prevention of damage to the young heifers' mammary glands by the use of antibiotics must begin before the teat and mammary gland have begun to enlarge prepartum.[7,13,14]

It is apparent from these data that control of the vector may be the only real preventive measure which is practical under current dairy management conditions in the United States and Europe. Fly control programs are already in effect for lactating animals but rarely are in effect for dry animals or unbred heifers. Since most of the dry animals and unbred animals are housed in remote locations, it becomes very difficult to apply topical insecticides to the areas which will need them for prevention of mastitis. At this time there is no adequate methodology available at most farms for controlling the vector in these animal groups.

Table 2. Prevalence of intramammary infections at breeding age and at calving in primigravid dairy heifers on seven Vermont dairy herds.

Pathogen	Infection Status By Mammary Quarter At Breeding Age	At Calving
S. aureus	.2	1.5
Coagulase negative staphylococci	21.0	22.5
Streptococci other than agalactiae	2.0	5.7
Coliforms	1.6	2.0
Negative	76.0	70.4

(Adopted from Pankey, *et al;* 1992)

A patent has recently been granted which describes an insecticide dispensing device which is glued to the tails of heifers and is used specifically for prevention of heifer mastitis through its ability to apply a repelling and insecticidal dose of pesticide to the udder area of the animals at risk. It is suggested that animals have these tags attached from the age of ten months until the time of calving and that the tags be freshly applied particularly during the summer when the highest numbers of insect vectors are present. In some areas of the United States it may not be necessary to apply these tags except during mid-summer. Several studies will be undertaken during the summer of 1994 to develop and refine recommendations for the effective use of this new prevention device. It is expected that recommendations for the use of this device will be formulated for dairy heifers, dry dairy cows, beef heifers, and dry beef cows. All of these animal groups are at risk for these mastitis complexes.

The results of the use of the insecticide dispensing device will be published later.

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Inter-relationships of periparturient diseases in dairy cows.

E. J. Peeler, M. J. Otte, R. J. Esslemont.
Veterinary Record (1994) 134, 129-132.

The associations between periparturient diseases in 3603 lactations over three calving seasons were assessed on 10 dairy farms in the south west of England by using logistic regression. Calf mortality and dystocia were strongly associated. Twinning and dystocia were important predictors of calf mortality. Twinning was also a significant predictor for retained fetal membranes. Retained fetal membranes, twins, calf mortality and dystocia, in that order of importance, were risk factors for vulval discharge. Twinning, dystocia, retained fetal membranes and lameness before service increased the risk of mastitis before service. Similarly, mastitis and dystocia before service increased the risk of lameness before service. Oestrus was less likely to be observed in cows that had twinned or suffered lameness before service, the latter having a significantly greater influence in first calvers than older cows.

A study of some pathogenetic aspects of bovine viral diarrhea virus infections.

G. Castrucci, F. Frigeri, B.I. Osburn, M. Ferrari, M. M. Sawyer, and V. Aldovrandi.
Comp. Immun, Microb. Infect. Dis., 1990, 13, 41-49.

The cytopathic (CP) TVM-2 strain of bovine viral diarrhea virus (BVDV) induced in calves a severe disease, characterized by the clinical picture which is usually reported for the acute primary infections observed under natural conditions. In contrast, the calves inoculated with a different biotype of BVDV, the non-cytopathic (NCP) New York-1 strain remained clinically normal with the only evidence of virus replication in these calves being the recovery of the virus from their pharyngeal swabbings and blood and also the detections of specific neutralizing antibody in their serums. When the calves were immunosuppressed with dexamethasone (DMS), they underwent an overt systemic disease of such severity that in most of the cases it ended with the death of the animals. This result was obtained with either the CP and the NCP strains of BVDV. Finally, the mixed infections that were obtained in the calves with the CP and NCP BVDV did not result in any particular unexpected pathological situation. It was speculated that the immunosuppressive activity of BVDV could be a property peculiar to certain isolates of the virus.