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Prevalence, incidence and risk factors of heifer mastitis

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ABSTRACT

Traditionally heifers, as calves and as primiparae, have been thought of as a group as free of mastitis. Without appreciable lacteal secretion, there is reduced nutrient fluid available to support growth of intramammary pathogens. Contagious mastitis is primarily transmitted at milking time and the milking process affects the patency of the teat orifice which can increase the risk of development of environmental mastitis. Logically therefore prepartum heifers should be free of intramammary infections. During the last 20 years there have been numerous investigations describing the nature of mastitis in heifers and thus the dogma that heifers are free of this disease has been challenged. The purpose of this manuscript is to review that literature describing heifer intramammary infections that cause both subclinical and clinical disease. Mammary quarter infection prevalence ranges between 28.9–74.6% prepartum, and 12.3–45.5% at parturition. Generally, the pathogens that cause mastitis in heifers are the same as those that cause infections in the older cows. In all but one study reviewed, coagulase-negative staphylococci (CNS) are the most prevalent cause of subclinical intramammary infections in heifers. Coagulase-positive staphylococci (CPS) in some studies are the second most prevalent pathogens, while in other studies the environmental mastitis pathogens are more prevalent. The risk factors for subclinical mastitis appear to be season, herd location, and trimester of pregnancy; all suggesting that management can have an impact in control of this disease prepartum. With respect to clinical mastitis, the most prevalent mastitis pathogen has been reported to be CNS in one study and CPS, or environmental mastitis pathogens, in other studies. The heifer is most at risk for clinical mastitis during the periparturient period. Risk factors found are related to diet, mammary gland factors such as edema and leaking of milk, and factors associated with the change in management and introduction of the heifer to the milking herd.

1. Introduction

It is common to introduce the subject of heifer mastitis by indicating that primiparae should in theory be free of intramammary infections (IMI) at first parturition. These animals have not experienced the rigors of multiple daily milkings, and thus have had less exposure to contagious pathogens that could be transmitted during milking time. Additionally, heifers have not been challenged with milking vacuums that have been associated with a deleterious effect on the structure of the teat end. For most of the heifer's life, the mammary gland has been immature and it would seem less likely to be in close physical contact to the environment, as contrasted with multiparous cattle. However, mastitis in heifers is not uncommon, although tending to be less prevalent than mastitis in older cows. The focus of this manuscript is the description of pre- and postpartum prevalence and incidence of clinical and subclinical mastitis in heifers and a discussion of possible risk factors associated with heifer mastitis.

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2. Subclinical mastitis in heifers

Mastitis is defined as inflammation of mammary gland and not as IMI. Yet IMI and mastitis are often used interchangeably. The measure of milk somatic cell count (SCC) is an often used measure of mammary inflammation and an increase in SCC is strongly correlated with increased probability of IMI (Eberhart et al., 1979; Dohoo and Leslie, 1991). The milk SCC threshold of 200,000 cells/ml is used to distinguish milk secretion from a mammary quarter with (>200,000 cells/ml) or without (<200,000 cells/ml) subclinical mastitis as this has support as an optimal threshold level (Dohoo and Leslie, 1991). De Vliegher et al. (2004) examined the SCC records of more than 14,000 heifers. During the first 14 days postpartum, they report that SCC from a milk sample taken at day 5 of lactation are on average higher than one taken on day 6, decreases over time and were lowest on day 14. They stratified the data set by SCC in early lactation, those with SCC up to 50,000 cells/ml, 51–200,000 cells/ml, 201–500,000 cells/ml, 500,001–1,000,000 cells/ml, and those >1,000,000 cells/ml. When SCC was plotted against days in milk (DIM) by stratification, SCC over time for those heifers with initial SCC < 200,000 cells/ml increased linearly, but the geometric mean SCC by 365 DIM was not greater than 100,000 cell/ml. Moreover, SCC over time was always distinctly lower for the lowest SCC strata. The graphic depictions of SCC by DIM for the other strata were different in that the SCC for the first time period plotted (day 15–day 45 DIM) was elevated relative to the SCC for the second time period plotted (day 46–day 75 DIM). Thereafter, the SCC plots for the highest 3 strata converged and increased linearly with a geometric mean SCC approaching 200,000 cells/ml at the last time period. Taken together these data demonstrate that heifers with a peripartum SCC of <200,000 cells/ml had distinctly lower SCC during their first lactation. This would indicate that subclinical mastitis at parturition will likely result in higher lactation average SCC. De Vliegher et al. (2004) could not determine whether heifers in the three highest strata were more at risk for a new case of subclinical mastitis or if they had a persistent case of subclinical mastitis acquired prior to or shortly after parturition. But data from this study would concur with a previous report that heifers with low initial SCC are at less risk for subclinical mastitis during lactation (Coffey et al., 1986).

2.1. Risk factors associated with subclinical mastitis

Svensson et al. (2006) reported the risk of an elevate SCC in early lactation were: (1) increasing the feeding of concentrate to heifers 11–16 months of age; (2) movement to confined housing the day of calving; (3) proportion of cows in the herd likely to have mastitis; and (4) use of restraint measures during milking time. The authors discuss the possible implications of over- and underfeeding grain and its effect on health of the animal, but recognize it is not clear as to the causative nature. Additionally, the authors recognize the relationship between elevated SCC and the movement of the heifer the day calving, as well as restraint of heifers, may only be linked to a general syndrome of a stressful event. The authors acknowledge the need to define more precisely the causal nature of the risk. Yet in support of some of these findings, another recent report (Kalmus et al., 2006) indicated housing had an impact on heifer mastitis, where a longer adaptation period to new housing prior to parturition was related to a decreased risk of clinical mastitis. Also, housing cattle in short stalls was more likely to result in more clinical mastitis at calving.

It is a common belief that the risk of mastitis varies with season and climate. A seasonal effect related to elevated SCC in heifers postpartum has been reported (Olde Riekerink et al., 2007). In their report they found that SCC spikes were most frequent in heifers during the summer months, but of a different pattern than was seen in multiparae which had a bimodal change in frequency in subclinical mastitis spikes as measured by SCC > 200,000 cells/ml. Whist et al. (2007) also indicated that SCC was highest in summer, but they did not specify how parity affected the seasonal effect on subclinical mastitis as measured by SCC.

Additionally, Whist et al. (2007) examined the milk SCC dynamics with heifers having *Streptococcus dysgalactiae* IMI, as opposed to those uninfected. In non-infected glands the results indicate that SCC was highest during the immediate postpartum period, within 5 days of calving, with counts between 50,000 and 100,000 cells/ml. Milk SCC decreased to a nadir at approximately 85 DIM and increased gradually with increasing DIM. The SCC in *S. dysgalactiae* cows followed a similar pattern except that the initial SCC was more than 5-fold greater at the immediate postpartum sampling with a rapid drop to approximately 100,000 cells/ml followed by a gradual increase in SCC as lactation progressed. The dynamics of milk SCC in multiparae was similar except the rise in SCC with lactation was steeper both for uninfected and infected glands. The data discussed herein indicate that subclinical mastitis in heifers is noticeably less severe in heifers than in multiparae as the SCC in both infected and uninfected glands are less than in comparable multiparous glands. The work of De Vliegher et al. (2004) clearly demonstrates the importance in reducing subclinical mastitis during the immediate peripartum period in terms of SCC over the lactation. Yet it is very important to understand the incidence and prevalence of IMI by pathogen type. Knowledge of the etiologic agents causing the disease can be used more specifically to develop control programs that lead to mastitis abatement.

2.2. Pathogens

The more prevalent mastitis pathogens associated with subclinical mastitis in lactating cows are the coagulase-negative and positive staphylococci, and the environmental streptococci and coliforms (Hogan, 1997). These are the same pathogens that cause IMI in heifers, both pre- and postpartum. Summaries of the prevalence of IMI before and after parturition are in Tables 1 and 2. Prevalence of IMI in mammary quarters in heifers both pre-and postpartum varies widely between studies. The range was 39.0–74.6% of quarters with IMI prior to parturition (Table 1). The immediate postpartum prevalence ranged from 12.3% to over 57% with IMI (Table 2). The