Cumulative Cure Rates for the Major Pathogens Causing Mastitis

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Introduction

Approaches to clinical mastitis treatment that utilize elements of a cow's history (Lago *et al*, 2004) and culture information from the affected quarter (Hess *et al*, 2003), have recently been described. Perhaps an optimal approach to treatment would be obtained by combining these two methods. Pathogen-specific somatic cell count (SCC) patterns before and after clinical mastitis have been documented by De Haas *et al* (2002). SCC was low before *Escherichia coli* mastitis cases and cases where bacteria were not isolated, and decreased quickly after a rapid rise. However, SCC was already high before cases associated with *Staphylococcus aureus* and the streptococci, and remained high afterward. The objective of this paper was to document the distribution of clinical cow cases of mastitis due to different pathogens by SCC status prior to clinical mastitis (Premast SCC) and to analyze Cumulative Cure Rate (CCR) for each of these sub-groups.

Materials and Methods

Data from 786 clinical cow cases of mastitis from a 2000-cow dairy using on-farm culture to identify pathogens prior to treatment were analyzed. The herd practiced a no-antibiotic treatment strategy for gram-negative infected quarters and cases where bacteria were not isolated. Cases with gram-positive isolates were treated with cephapirin or pirlimycin. Clinical cow cases were sorted by pathogen (*S. aureus*, coagulase-negative staphylococci (CNS), streptococci, coliforms and no growth) and by Premast SCC status. CCR was calculated using monthly Dairy Herd Improvement Association (DHIA) individual cow SCC as described elsewhere (Lago *et al*, 2004). The data were analyzed using the LOGISTIC procedure of SAS. A significance level of $P<0.05$ was used.

Results

There was a significant difference between pathogens in the distribution of clinical cow cases by Premast SCC group. As expected, 72% of coliform mastitis cases were distributed in the Premast<200 group, compared with less than 46% of gram-positive infected cases. CCR for clinical cow cases infected with *S. aureus* was very poor in each of the Premast SCC categories: 11% for cows not tested prior to clinical mastitis, 17% for cows with SCC <200,000/ml immediately prior to a clinical mastitis event (Premast<200) and 13% for cows with SCC ≥200,000/ml prior to mastitis (Premast≥200). Higher CCR was observed in clinical cow cases due to coliforms (56%), again independent of Premast SCC status. This pattern was repeated for cases with no growth, although cases in the Premast<200 category in this pathogen group had the highest CCR: 79% versus 46% and 52% for fresh cow and Premast≥200 cases, respectively. Cases due to *Streptococcus* spp and CNS had poorer CCR in the Premast≥200 group compared to the other Premast SCC groups. CCR in the Premast≥200 group was 27%, versus 64% for fresh cows and 55% for Premast<200 cases caused by *Streptococcus* spp, and 32 versus 57 and 49%, respectively, when CNS were isolated.

Significance

The distribution of cases by Premast SCC status provides useful information about the main pathogens involved in clinical mastitis within a herd. Documented differences in CCR by Premast SCC status appear to be driven, at least in part, by pathogen type. The relative contribution of one pathogen type over another will influence overall CCR for all cases treated. Individual case treatment decisions on farms where a culture program is implemented should not only be based on pathogen isolated, but also account for Premast SCC status and other elements of a cow's history, which include parity, number of mastitis treatments in a lactation, days in milk and linear score at dry off.