

Clinical and Subclinical Diseases Predisposing to Johne's Disease in Dairy Cattle

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Introduction

Paratuberculosis or Johne's disease (JD) is a chronic and progressive intestinal disease in ruminants caused by *Mycobacterium avium* subsp *paratuberculosis* (MAP). The usual route of infection is fecal-oral, with young cattle becoming infected by exposure to infected adults or their environment. The factors that trigger MAP fecal shedding or JD clinical disease are not understood and little information is provided about it in the literature; why do some cows that already shed the organism in the feces suddenly break into the clinical phase, whereas other shedding herdmates continue in the subclinical phase? The objectives of this study were to identify associations between clinical or subclinical diseases and risk for subsequent occurrence of clinical Johne's disease (JD) and onset of fecal shedding after 305 days in milk (DIM).

Materials and Methods

A total of 1297 cows from two Minnesota dairies were enrolled in the study after fecal samples were obtained during the closeup period. A second fecal sample was obtained from cows after at least 305 days after calving (days in milk; DIM) or at time of leaving the herd (sold/dead). Between 3-21 DIM, blood samples were obtained for beta hydroxy butyrate (BHB) and serum total protein testing. Body condition score (BCS) was evaluated during the closeup period, within 21 DIM and at end of lactation. Clinical disease (milk fever, retained placenta, metritis, ketosis, displaced abomasum, lameness, mastitis, and JD clinical signs) was recorded from a computerized dairy herd management software program (Dairy Comp305®; Valley Agricultural Software, Tulare, CA). Logistic regression was used to assess the association between clinical or subclinical disease and JD clinical signs or the onset of fecal shedding at the end of the lactation.

Fecal samples were processed using the bacterial culture method containing Herrold's egg yolk medium. Colony counts were recorded from weekly for 16 weeks, and final results were reported as negative, light (mean of one to nine colonies/tube [CPT]), moderate (mean of 10 to 49 CPT), and heavy (mean of > 50 CPT) fecal shedding. It was assumed that the fecal culture method had 100% specificity.

Results

Average DIM when cows with JD clinical signs (JDCS) were culled (n=66) was 209. From multivariable analysis, occurrence of JDCS was associated with occurrence of pneumonia (OR=2.6, 95% CI= 1.2-6.0) and level of fecal shedding (light: OR=13.0, 95% CI=5.3-30.0; moderate: OR= 33.0, 95% CI=13.0-85.0; heavy: OR=63.0, 95% CI=25.0-162.0). From multivariable analysis, onset of fecal shedding at the end of lactation in 79 cows was associated only with occurrence of pneumonia (OR=2.2, 95% CI=1.1-4.2).

Significance

The results provide insights into the role of other diseases on JD clinical signs and fecal shedding, which may enable us to better manage the disease early in the lactation. Furthermore, the statistical model suggests that it is possible to predict early in the lactation which cows are more likely to be culled due to JD clinical signs or to have an onset of fecal shedding. It is possible that beyond the epidemiological aspect of these diseases, there is a physiological mechanism that is needed to be better explained. Therefore, the conjunction of both epidemiological and physiological studies is further necessary to better understand the role of these diseases in the occurrence of subclinical and clinical JD.