Poster 4

SERUM AMYLOID A IN SERUM AND MILK IN EXPERIMENTALLY INDUCED BOVINE CNS MASTITIS

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Introduction

A new experimental mastitis model was developed, in which a strain of coagulase-negative staphylococci (CNS), S. chromogenes was used to induce udder infection. The aim of this study was to investigate development and inflammatory reaction of mastitis caused by a minor pathogen, CNS. The results are compared with those from a serious experimental infection model by a major pathogen, *E. coli.*

Materials and Methods

Six first-lactating, clinically healthy dairy cows of Holstein-Friesian breed with a low somatic cell count in their milk were used in the study. The experimental challenge was done in early lactation. One udder quarter of each cow was inoculated using a CNS strain (S. chromogenes SL37-2/01) isolated from subclinical mastitis. The infection dose was approximately 2x106 CFU per quarter in 5 ml of saline. Infection dose was selected based on pilot challenge studies with different doses of CNS. One cow was excluded after challenge, due to mastitis in another quarter. Cows did not receive any treatment. Before the challenge and at regular intervals thereafter the cows were examined clinically using a scoring system. Clinical findings consisted of general attitude, appetite, temperature, rumen function, udder palpation and milk appearance. Milk samples were taken for bacteriological culturing, SCC, NAGase, and acute phase protein determination, and blood samples were taken for acute phase protein determination. Serum amyloid A (SAA) was determined from serum and milk using a commercial kit (Phase SAA kit, Tridelta Ltd, Ireland).

Results

All cows became infected with *S. chromogenes*. Clinical signs were mild and none of the cows showed systemic signs such as fever. Only mild local signs in the inoculated quarter such as slight swelling and a few clots in the milk could be seen, which disappeared within a few days. Infection was eliminated within 38 h post-challenge in all cows except one which developed chronic mastitis. Somatic cell count increased in the affected quarters, peaking at 24-32 h (mean peak value 3.68 million cells/ml). The concentration of SAA increased in serum, and the peak levels which were seen by 48 h post-challenge varied between the cows (19-131 mg/l). SAA concentration in the milk before challenge was under the detection limit but slightly increased at 24 h after challenge (mean 2.5 mg/l) and fluctuated during the following 3 days. The maximal SAA concentrations in serum of the cows with CNS mastitis were about one fifth and SAA concentrations in the milk about 1/100 of that seen in *E. coli* mastitis (data not shown).

Conclusions

Bovine experimental CNS mastitis has not been described before, and there certainly are differences between different species in this respect. A large inoculum dose was necessary to induce mastitis. Results from this study confirmed the mild character of CNS mastitis, however detectable changes in SAA could be seen in serum and milk, which could be used to diagnose mastitis.