

AN OUTBREAK OF *STREPTOCOCCUS UBERIS* AS A CONSEQUENCE OF ADOPTING A PROTOCOL OF NO ANTIBIOTIC THERAPY FOR CLINICAL MASTITIS

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Introduction

In recent years, the approach to clinical mastitis treatment and control has undergone several changes. Questions have been raised about the value of intramammary therapy for the treatment of clinical mastitis caused by the majority of pathogens. Research has shown that intramammary antibiotic treatment (IMM) of coliform mastitis cases is not efficacious, not cost effective and possibly detrimental to the animal's health (5,8). Field studies comparing treatment of any clinical mastitis with IMM antibiotics to oxytocin alone showed no difference in short term case outcome as assessed by pathogen clearance or clinical cure (7). Some mastitis experts have concluded that "except for the treatment of *Streptococcus agalactiae*, there is no evidence that antibiotic use is cost-effective in treating mastitis during lactation" (12) and "neither dry cow therapy nor lactational therapy have any significant effect on the reservoir of environmental pathogens" (14).

On a practical level, large herds are frequently managed with standardized treatment protocols that are applied uniformly to all clinical cases. In a herd where contagious pathogens are controlled or eliminated and regular monitoring is conducted, cessation of intramammary lactational therapy may be warranted. The danger of implementing this strategy is illustrated by this case example from a large commercial herd.

Mastitis management

This outbreak will be discussed in the framework of mastitis management recommendations. This case illustrates that it is possible to follow guidelines for management and have problems that do not fit the typical contagious or environmental outbreak description.

Contagious pathogen control

First let's review the components of the "5 point plan" for contagious mastitis control (2,10), how it works and how it was implemented in this particular herd. The program consists of the following:

1. post milking teat dipping
2. dry cow therapy
3. culling
4. therapy of clinical mastitis
5. proper maintenance of milking equipment

Herd implementation prior to the outbreak consisted of the following:

1. post milking teat dipping--regularly performed
2. dry cow therapy--Dry-Clox

3. culling--*Staphylococcus aureus*, *Mycoplasma* spp.
4. therapy of clinical mastitis--*Streptococcus agalactia*
5. proper maintenance of milking equipment--regularly performed

The success of this program relies on reduction of the number of infected quarters in the herd and reduction of the frequency of exposure of non-infected quarters by milk contaminated fomites (6,13). Because these practices, as well as screening of purchased heifers, had been in place for several years, contagious pathogens were isolated from less than 1% of clinical cases in this herd. If a pathogen of this class was identified in pooled weekly samples from the bulk tank or the fresh pen, additional samples were collected to identify the shedding cow as quickly as possible. Identification of contagious pathogens in pooled samples was an infrequent occurrence.

Environmental pathogen control

Control of environmental mastitis also involves measures that reduce teat end exposure to pathogens. This is accomplished by reducing the magnitude of environmental contamination and exposure. Control measures include:

1. clean, dry environment
2. pre-dipping milking hygiene
3. vaccination with core-antigen

Environmental mastitis control measures in place at the beginning of the outbreak included:

1. clean, dry environment--marginal, straw bedding
2. pre-dipping milking hygiene--regularly performed, no fore-stripping
3. vaccination with core-antigen--J-5

Herds experiencing environmental mastitis problems commonly are well managed modern herds with bulk tank somatic cell counts <200,000 cells/ml and effective control of contagious pathogens (9,16). In the example herd, somatic cell counts were consistently below 200,000 cells/ml, except for short periods of time following precipitation (figure 1.--Bulk tank somatic cell counts). Clinical mastitis incidence remained below 3% of the lactating herd per month, except after severe weather when coliform incidence transiently increased (figure 2.--Clinical mastitis incidence). The outbreak occurred under these conditions.

The Outbreak

"Control of contagious pathogens and improvements in prevention of environmental mastitis have altered the pathogen profile but have not significantly reduced the overall incidence of mastitis" (4,11). This principle is supported by the epidemiology of this herd outbreak (Table 1.--Proportion of clinical mastitis isolates by species, Table 2.--Proportion of bulk tank isolates by species). The first warning signs of the impending clinical mastitis outbreak were trends in the ratios of *Streptococcus* to coliform spp. isolated from bulk tank samples, fresh pen samples and clinical mastitis cases. *Streptococcus* spp. were typically 20% or less or represented organisms before and after the outbreak, but reached up to 48% of bulk tank isolates and 73% of clinical mastitis isolates at peak. Following a long-term reduction in the incidence of clinical mastitis caused by contagious and coliform pathogens, the overall incidence of mastitis acutely increased. Bulk tank somatic cell counts concurrently increased to unacceptable levels. Herd screening with the California Mastitis Test and examination of milk from

fore-stripping to find mild mastitis cases, identified a group of potentially infected animals for culture. *Streptococcus uberis* was isolated from the majority of clinical cases and cows with CMT scores greater than 2 during herd screening. The sensitivity of environmental *Streptococcus* spp. isolates to currently available intramammary antimicrobials showed that the majority was resistant to the dry cow therapy in use at the time (Table 3.--*In vitro* sensitivity of 93 *Streptococcus* spp. isolates).

Once the problem was identified three key management changes were implemented:

1. Fore-stripping to detect mild clinical mastitis
2. Change dry cow therapy
3. Re-initiate clinical lactational antibiotic therapy

Within two months, somatic cell counts returned to pre-outbreak levels and continued to decline. Mastitis incidence decreased more slowly, presumably due to subclinical infections in animals not yet eligible for dry cow therapy. The proportion of clinical mastitis isolates gradually returned to a coliform dominant profile.

Discussion

This case history highlights several points that contradict current paradigms. First, generalization of research results beyond the frame of inference can create problems. The erroneous assumption in this situation was that IMM therapy was not justifiable except for the treatment of *S. agalactia* infections. The clinical response to therapy and economic value of treatment are highly dependent upon the population of organisms causing clinical signs. The pathogen profile is herd dependent and changes with time. This outbreak showed that without appropriate lactational and dry cow therapy, the streptococcal clinical mastitis incidence increased over time. This increase was due to chronic and recurrent infections, resulting in increased duration of infection relative to other environmental pathogens. New infections were attributable to both a constant environmental exposure and an increasing organism reservoir in infected quarters. A recently published study comparing two IMM antibiotic treatments to oxytocin alone, found that although clinical cure rates, milk production and time to removal from the herd did not differ among treatment groups, the rate of both relapse and recurrence of clinical mastitis was significantly elevated in untreated cows. This tendency was strongest if environmental *Streptococcus* spp. were isolated prior to treatment (17). *Streptococcus dysgalactia* has been characterized as intermediate between contagious and environmental (14). Because of the potential for the reservoir of *Streptococcus uberis* within infected quarters to be a significant source of exposure, this organism also should be considered to be intermediate between these two categories. The rapid and exponential rise in bulk tank somatic cell counts and mastitis incidence to levels often associated with contagious pathogens provides additional justification for this re-classification.

The second fallacy of mastitis management is that one dry cow therapy is as good as another. Again, the efficacy of therapy depends in part on the pathogen profile of the herd at that time. Dry-treated cows have fewer streptococcal infections at calving than untreated animals (3,15). Environmental *Streptococcus* spp. tend to have more strain specific variability in sensitivity to antimicrobials than *Streptococcus agalactia* (1). To maximize the difference in response to therapy over spontaneous cure, and therefore cost efficiency of lactational or dry treatment, it is useful to know not only the species profile of the herd but the sensitivity pattern of the herd specific strain in the event of an outbreak.

In large herd mastitis management and implicit in mastitis field research, it is assumed that one treatment protocol is adequate and is required for simplicity and consistent compliance. However, a protocol can be designed as a decision tree based not only on systemic signs but on the character of secretion or clinical culture result. If justified by the return on investment attributed to reduced antimicrobial use, reduced rate of recurrence and the long-term effect on bulk tank somatic cell counts, antimicrobial therapy during lactation can be targeted to specific sub-group of infected animals. It is possible to culture all clinical cases but to treat only those with resulting gram positive isolates. This would result in less antimicrobial use and more successful therapy. The optimal approach to coliform mastitis therapy is to treat the symptoms. The optimal approach to gram positive mastitis therapy is to treat the organism.

References

1. pers comm Austin Belschner, Upjohn Dairy Technical Services Consultant
2. Bramley, A.J., F.H. Dodd. 1984. Reviews of progress of dairy science: Mastitis control-progress and prospects. *J. Dairy Res.* 51:481.
3. Eberhart, R.J. 1986. Management of dry cows to reduce mastitis. *J. Dairy Sci.* 69:1721.
4. Erskine, R.J., R.J. Eberhart, L.J. Hutchinson, S.B. Spencer and M.A. Campbell. 1988. Incidence and types of clinical mastitis in dairy herds with high and low somatic cell counts. *J. Am. Vet. Med. Assoc.* 192:761.
5. Erskine, R.J., et al. 1992. Intramammary administration of gentamicin as treatment for experimentally induced *Escherichia coli* mastitis in cows. *Am. J. Vet. Res.* 53:375.
6. Fox, L.K. and J.M. Gay. 1993. Contagious mastitis. In *Update on Bovine Mastitis. The clinics of North America: Food Animal Practice.* 9:475.
7. Guterbock, W.M., A.L. Van Eenennaam, R.J. Anderson, I.A. Gardner, J.S. Cullor and C.A. Holmberg. 1993. Efficacy of intramammary antibiotic therapy for the treatment of clinical mastitis caused by environmental pathogens. *J. Dairy Sci.* 76:3437.
8. Hogan, J.S., K.L. Smith, D.A. Todhunter, et al. 1987. Efficacy of intramammary infusion products for lactational therapy of mastitis caused by environmental pathogens. In *Proceedings of the International Mastitis Symposium, Ste Anne de Bellvue, Quebec, Canada.* 302.
9. Johnson, P.E. 1993. Dealing with environmental mastitis on a daily basis. In *Proceedings of the 32nd Annual Meeting of the National Mastitis Council.* 79.
10. Philpot, W.W. 1979. Control of mastitis by hygiene and therapy. *J. Dairy Sci.* 62:168.
11. Schukken, Y.D., D. van der Geer, F.J. Gommers, J.A.H. Smith and A. Brand. 1989. Intramammary infections and risk factors for clinical mastitis in herds with low somatic cell counts in the bulk tank. *Vet. Rec.* 125:393.
12. Sears, P.M. 1993. *Staphylococcus aureus* mastitis. In *Proceedings of the 32nd Annual Meeting of the National Mastitis Council.* 4.