Alternatives To Antibiotic Therapy Of Clinical Mastitis

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Mastitis is the most common cause of antibiotic use in adult dairy cows. In surveys of well-managed herds with somatic cell counts (SCC) under 150,000 and virtually no mastitis due to coagulase-positive Staphylococcus aureus (Staph.) or Streptococcus agalactiae (Strep. ag.), 35-55% of lactations had one or more incidents of clinical mastitis (2,4,7,8,9). In such herds, 15-40% of the clinical cases had no bacteria isolated from the milk, 21-43% had coliforms, and 9-32% had environmental streptococci (1,2,4,7). This contrasts with high SCC herds with a significant prevalance of Staph. and Strep. ag., where most of the clinical mastitis is caused by those organisms (4). As more herds respond to quality incentives and stricter SCC standards by controlling the contagious pathogens, we can expect the relative importance of the environmental pathogens to continue to increase.

Antibiotic treatment of clinical mastitis incurs the cost of the drugs used, the discarded milk, and the loss of the option to cull the cow until after the withdrawal time has elapsed if treatment fails. It also incurs the risk of contaminating the bulk tank with antibiotics, and all of the expensive regulatory consequences of violative antibiotic residues under the revised Pasteurized Milk Ordinance, including a significant loss of revenue from milk. Those who design treatment protocols should be sure that the benefit of antibiotic treatment outweighs these very real economic costs.

Some dairymen and veterinarians have already decided that the risks of antibiotic use in most clinical mastitis cases exceed the benefits and have stopped treating clinical mastitis cows with antibiotics in herds with a low prevalence of the contagious organisms. They emphasize protocols of frequent milkout aided by oxytocin injections and anti-inflammatory drugs, along with heightened attention to management of housing, bedding, and premilking hygiene to prevent infection with environmental pathogens. While the anecdotal reports about such programs are favorable, there is no published data about the rate of chronic or recurring infections in such herds compared to herds using antibiotics, nor on the effects of these infections on bulk tank SCC or subsequent milk yield.

There is no published evidence that the benefits of antibiotic treatment of mild clinical mastitis outweigh the risks and costs. There are no published studies on the antibiotic treatment of mild clinical mastitis (mastitis in which the cow does not become systemically ill) under field conditions that include untreated controls. Chamings (3) reported an 87% clinical cure rate in cows that were not treated with antibiotics for mild clinical mastitis caused by Staph. and Streptococcus uberis. The bacteriological cure rate for both organisms was 19-20% This study did not have a positive control group for comparison. This type of mastitis is treated on most dairies with mastitis tubes, possibly in conjunction with extra-label parenteral antibiotics or antiinflammatory drugs. All of the approved intramammary mastitis preparations on the market in the United States as of June, 1992 were tested against subclinical infections with gram-positive organisms.

Only one has a label claim for mastitis caused by Escherichia coli, which is the most frequently isolated udder pathogen in many outbreaks of clinical mastitis in herds with low SCC.

The pharmacology of mastitis therapy has recently been reviewed (6,13,14). Reasons why antibiotic therapy might fail are summarized in Table 1. The underlying assumption of research on mastitis to date has been that the primary aim of therapy is to kill bacteria, and that the normal state of milk in the udder is sterility. Yet subclinical infections with environmental and contagious pathogens probably exist in every herd (4). Clinical mastitis may be due to the flareup of subclinical infection in a stressed cow. In the short run, the economically important clinical outcome in the treatment of clinical mastitis is not the absence of bacteria, but rather the return of milk and udder to their normal state, so that the cow's milk can once again be sold.

Only one common pathogen, Streptococcus agalactiae, is highly sensitive to and easily cured by approved intramammary antibiotics used according to the label. In most herds with low SCC the prevalence of Strep. ag. is low or zero. Many such herds have no Strep. ag. isolated from bulk tank samples or clinical cows for years. In herds with Strep. ag. infected cows, use of intramammary antibiotics is easily justified on medical, if not economic grounds because it stops the shedding of bacteria by the cow with clinical mastitis and because Strep. ag. is very sensitive to all of the antibiotic tubes on the market. Treatment of clinical mastitis in lactating cows is not effective, however, in reducing prevalence in the herd unless it is part of a total control program (11). Only an integrated program of teat dipping, milking machine maintenance, milking hygiene, and dry cow treatment can bring about a long-term reduction in prevalence.

While all mastitis tubes carry a label claim for Staph., the cure rate is so low that dairymen are best advised to consider it negligible (10,11,12). The cure rate in Staph. cows is low because the organism forms microabscesses in the udder tissue outside the ducts, where intramammary drugs can not reach it. It also can survive inside white blood cells, makes L-forms, and can acquire resistance to commonly used antibiotics (10). The best hope for successful antibiotic treatment of Staph-infected cows is in young cows with recent infections. Parenteral treatment may increase the chance of a cure (10). In herds with a high prevalence of Staph. infections, the emphasis should be on teat dipping, culling, milking machine maintenance, milking hygiene, and segregation of infected cows to gradually reduce the prevalence of the infection. Antibiotic treatment may reduce shedding of Staph. by clinical mastitis cows and thus help reduce the spread, but it will not reduce overall prevalence in the herd significantly (11).

In herds with low SCC and low prevalence of contagious pathogens, clinical experience and published surveys (1,2,4,7) show that about 15-40% of pretreatment milk samples from cows with clinical mastitis are negative for bacterial growth on blood agar. We presume that these samples containing too few organisms for a positive culture result reflect the ability of the cow's immune system to rid the affected quarter of pathogens. Antibiotic treatment of these cows is difficult to justify; the problem is that we can not know which cows they are until after treatment has to be initiated. The aim of treatment should be to return the quarter and the milk to normal, not to prevent the spread of infection. Anti-inflammatory drugs or immune modulators would seem indicated, rather than antibiotics.

A fairly large group of so-called "minor" pathogens — minor in prevalence in the industry, not to the infected cow or her owner — are refractory to all antibiotic treatment. This group includes the genera Mycoplasma, Pseudomonas, Pasteurella, Serratia, Prototheca, Mycobacterium, Nocardia, Bacillus, the yeasts and fungi, and Actinomyces pyogenes.

In surveys of clinical mastitis in herds with low SCCs, coliform organisms account for about one-third of isolates from clinical cows. Coliform organisms can cause mastitis of severity rang-

ing from subclinical to peracute. Erskine (5,6) has shown that clinical signs appear in experimental coliform mastitis after bacterial numbers in milk have peaked, and that treatment of these cows with intramammary gentamicin did not affect clinical outcome. Toxic mastitis can be reproduced by infusing endotoxin without living organisms into the udder; most of the clinical signs of coliform mastitis are thought to be due to the effects of endotoxin (5). Treatment should therefore aim primarily at removing endotoxin from the udder with frequent and complete milkout and at counteracting the effects of endotoxin with appropriate antiinflammatory and supportive treatments. The most important part of a treatment protocol for coliform cows is to milk the quarter out completely and often, possibly with the help of oxytocin injections. Unfortunately, treatment must begin before the organisms involved can be identified, and the appearance of the abnormal secretions alone is not a reliable basis for an etiologic diagnosis, except perhaps in the most severe cases.

The environmental streptococci and the coliforms account for the majority of environmental clinical mastitis cases where a diagnosis is obtained. Philpot (11) cited a cure rate for clinical mastitis caused by environmental streptococci of 36%. Erskine (6) states that acceptable cure rates (>75%) are attainable with a combination of intrammary antibiotics and intramuscular procaine penicillin G. Tyler (13) states that response of clinical Streptococcus uberis infections to antibiotic therapy during lactation is poor, although a combination of parenteral and intramammary erythromycin appears to be the most efficacious treatment. More research is needed on these organisms, particularly on any long-range benefit from antibiotic treatment in eliminating chronic infections during lactation.

The challenges in treating clinical mastitis in a herd with low SCC are the impossibility of establishing an etiologic diagnosis at the time of first treatment, the fact that about a third of cows being treated have already cleared the infection, and the fact that in the case of the coliforms at least, the primary aim of treatment has to be to counteract the effects of endotoxin rather than reducing bacterial numbers. This must be accomplished without incurring undue risk of antibiotic contamination of milk, in the absence of clear experimental evidence from controlled trials that antibiotic treatment of mastitis is efficacious or cost-effective. Clearly more research is needed.

A controlled study of intramammary treatment for mild clinical mastitis caused by environmental bacteria was recently completed at the Veterinary Medicine Teaching and Research Center of the University of California, Davis. We compared the efficacy of cephapirin and amoxicillin mastitis tubes to that of oxytocin alone in the treatment of mild clinical environmental mastitis. Both tubes were used according to label instructions. Oxytocin cows received 50 units of oxytocin intramuscularly just before milking. No other treatments were used on cows in the study. No contagious pathogens were isolated from any of the clinical cases. Cows treated in the study had mild mastitis, that is, abnormal milk with or without udder swelling, and no signs of systemic illness, and were randomly assigned to one of the three treatments. Cows that did not improve or got worse during the observation period were called treatment failures and withdrawn from the trial. A clinical cure was the return of the affected quarter and milk to normal at the eighth milking after initial diagnosis and treatment. A bacteriologic cure was the failure to isolate the primary pathogen present at the first milking at the eighth milking and at 20 days after initial treatment. Results are shown in in tables 2, 3 and 4. Herds 1 and 3 were located in San Joaquin County and Herd 2 in Kings County.

In the past, the standard recommendation was to treat all cows with clinical mastitis with antibiotic tubes used according to the label. In herds with low SCC where all clinical mastitis is

caused by environmental bacteria, we can design better treatment protocols that minimize antibiotic use, reduce the risk of residues, and still allow flexibility to beef affected cows if treatment does not work. A responsible treatment protocol requires that permanent records of clinical mastitis be kept so that a cow's past history can be consulted before treatment is initiated.

Clinical mastitis should be classified before treatment as mild or severe. Mild mastitis would be characterized by abnormal milk and slight udder swelling, while severe mastitis would include abnormal milk, severe swelling, the risk of losing the quarter, and systemic illness (fever, off feed, diarrhea).

Before a protocol is put in place, the veterinarian should collect and analyze the results of sampling of clinical mastitis cows to determine the pathogens generally involved on the particular farm in different seasons. On a farm with a significant incidence of clinical mastitis caused by Strep. ag., for example, antibiotic tubes should probably be used on most clinical cases, while on a farm where a third of the clinical samples show no growth and another third yield E. coli antibiotic use is hard to justify.

Dairy personnel should be trained to look at the cow's record before beginning a course of lactating cow treatment. The people making the treatment decisions, usually milkers or herdsmen, need to be trained and trusted to make these decisions properly. The veterinarian and the owner should develop a treatment protocol based on the known past history of pathogens in the herd, age of the cow, reproductive status, milk yield, relative value in the herd, past mastitis history, other unsoundnesses (locomotor problems, poor udder conformation, etc.), and the severity of clinical signs. For example, a cow that is below the herd average, open, and late in lactation will most likely be culled eventually anyway and might as well be culled now that she has mastitis. An average first-lactation cow that is late in gestation should be dried off early, since dry cow preparations are stronger, stay in the udder longer, are more likely to clear up the infection than lactating cow tubes, and present less risk of contaminating the bulk tank with antibiotics. Cows with persistent or recurring infections despite past treatment are unlikely to respond to a repetition of the same treatment protocol. The risky approach on these cows is to turn to extralabel use of parenteral antibiotics, with all of the risk of illegal residues it entails. A safer approach is to evaluate the cow's record and the severity of the infection and decide either to cull the cow, dry her off, treat her, or to let her recover on her own. A young, high-yielding cow in early lactation with mild mastitis might be treated aggressively, with an emphasis on frequent and complete milkout.

Treatment protocols should be modified to fit the culling philosophy and goals of each dairyman. A dairyman who is trying to build up herd numbers, for example, may be more inclined to dry off a clinical mastitis cow than one whose facility is overcrowded and is looking for room for a new heifer. A dairyman may be unwilling to cull his purebred cows under any circumstances.

On large dairies an aid in the management of clinical mastitis is to have a designated mastitis string, which is milked last, just before the hospital or antibiotic string. The mastitis string is milked into the bulk tank. It contains all cows that have had clinical mastitis during the current lacatation, chronic high SCC cows, and cows known to be infected with Staph. that the owner does not want to cull. On some dairies it might include slow-milking cows and cows with poor udder shape that require extra attention at milking time. On others the slow cows are in a separate group. Cows in the mastitis string are generally not to be treated with antibiotics when they get clinical mastitis again. They are either culled, or milked out with the aid of oxytocin injections until their milk is normal. Since abnormal milk may not be put into the bulk tank, these cows with clinical mastitis must either be milked into a separate bucket or put in the hospital

string until their milk is normal. Cows may leave the mastitis pen only to be dried-off or culled, or if their individual SCC remains below 200,000 for three consecutive test days and they are not known to be infected with a contagious pathogen.

Treatment of clinical mastitis is the most common use of antibiotics on dairy farms and the most common cause of illegal antibiotic residues. On well-managed dairy farms most mastitis is caused by the environmental pathogens. There is no data from well-controlled studies demonstrating the efficacy of antibiotic treatment of clinical mastitis caused by the environmental pathogens, nor on any benefit of antibiotic treatment on chronic or persistent infections. However even in the absence of data the veterinarian can be very helpful in developing treatment protocols that greatly reduce the use of antibiotics and decrease the risk of violative residues.

Table 1: Reasons For Failure Of Antibiotic Therapy Of Clinical Mastitis.

A.Drug can not reach all sites of infection.
1. Microabscess formation (Staph)
2. Blockage of ducts with clots of denatured milk.
3. Poor distribution of drug in udder, due to swelling, edema, or intrinsic properties of drug.
4. Abscessation
5. Fibrosis
6. Intracellular bacteria (Staph.)

B. Bacteria already killed by cow's immune system before therapy begins.

C. Inadequate concentration of drug to effect killing.
1. Poor distribution of drug in udder.
2. Absorption of drug from milk into systemic circulation.
3. Failure of drug to be absorbed by affected tissues
4. Drug milked out at subsequent milking.
5. Failure of parenteral drug to cross blood-milk barrier.
6. Failure of client or veterinarian to repeat treatments in time to maintain MIC in tissue long enough to effect killing.

D. Bacteria refractory to killing by drug.
1. Bacteria not in rapid growth phase required for drug to act.
2. Organism is resistant to usable antibiotics (e.g., Pseudomonas, Mycoplasma, yeasts, etc.)
3. Drug with gram-positive spectrum used on gram-negative infection.
4. Acquired resistance by organism.
5. Emergence of L-forms, "naked" acapsular forms that resist beta-lactam antibiotics.

E. Reinfection of affected quarter.

Table 2: Pretreatment bacterial isolates of 3 treatment groups in randomized field trials of therapies for mild clinical mastitis, California, 1991-1992 (%)*.

Variable	Treatment			P value
	Oxytocin	Amoxi-mast	Cefa-lak	
Coliform	33.3	41.9	37.3	0.93
Streptococcus sp.	26.7	23.0	26.7	

Other	15.2	10.8	13.3
Negative	24.8	24.3	22.7
Number of cows	105	74	75

(* : Of the 94 coliforms, 81 (86%) were E.coli. Of the 65 Streptococcus sp., 27 (42%) were S.uberis, 19 (29%) were S.dysgalactiae, and 14 (22%) were S.viridans. Of the 34 "Other" bacteria, 14 (41%) were Staphylococcus sp. (primarily S.hyicus), 9 (26%) were mixed infections, 3 (9%) were Bacillus sp., and 3 (9%) were Corynebacterium sp.

Table 3: Bacterial and clinical cure (%) by treatment group and herd in randomized field trial of therapies for mild clinical mastitis, California, 1991-1992.

Herd	Treatment						P value
	Oxytocin		Amoxi-mast		Cefa-lak		
Bacterial cure %*							
Herd 1 (n=64)	10/26	(38.5)	9/20	(45.0)	11/18	(61.1)	0.33
Herd 2 (n=31)	6/10	(60.0)	6/10	(60 0)	6/11	(54.5)	0.96
Herd 3 (n=43)	12/21	(57.1)	3/11	(27.3)	5/11	(45.5)	0.27
Total (n=138)	28/57	(49.1)	18/41	(43.9)	2/40	(55.0)	0.61
Clinical cure %							
Herd 1 (n=82)	23/33	(69.7)	20/24	(83.3)	17/25	(68.0)	0.41
Herd 2 (n=86)	19/36	(52.8)	12/25	(48.0)	16/25	(64.0)	0.50
Herd 3 (n=86)	28/36	(77.8)	18/25	(72.0)	7/25	(68.0)	0.69
Total (n=254)	70/105	(66.7)	50/74	(67.6)	50/75	(66.7)	0.99

(*: Of 254 cases, 61 were culture negative prior to first treatment, 43 were given additional treatment prior to 9th milking, 2 were treated between 9th milking and 21 days, 2 were dried prior to 21 days, 4 were culled before 9th milking, and 4 were culled before 21-day sample.)

Table 4: Bacterial and clinical cure (%) by treatment group and bacterium isolated at pretreatment sampling in randomized field trial of therapies for mild clinical mastitis, California, 1991-1992.

Herd	Treatment						P value
	Oxytocin		Amoxi-mast		Cefa-lak		
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Coliforms (n=63)	15/26	(57.7)	8/21	(38.1)	8/16	(50.0)	0.41
Strep. sp. (n=49)	10/21	(47.6)	6/13	(46.2)	11/15	(73.3)	0.23
Other bacteria (n=26)	3/10	(30.0)	4/7	(57.1)	3/9	(33.3)	0.48
Pos. culture (n=138)	28/57	(49.1)	18/41	(43 9)	22/40	(55.0)	0.61
Clinical cure %							
Coliforms (n=94)	22/35	(62.9)	21/31	(67.7)	14/28	(50.0)	0.36
Strep. sp. (n=65)	17/28	(60.7)	9/17	(52.9)	14/20	(70.0)	0.56
Other bacteria (n=34)	7/16	(43.7)	7/8	(87.5	9/10	(90.0)	0.02
No bac. isol. (n=61)	24/26	(92.3)	13/18	(72.2)	13/17	(76.5)	0.18
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References:

1. Anderson, K.L., et al., 1982. "Diagnosis and Treatment of Acute Mastitis in a Large Dairy Herd." J. AVMA. 181:690-693.

2. Bennett, R.H., 1990. "Clinical Mastitis from Environmental Pathogens: Analysis of a Large Commercial Dairy." Proc Int. Symp. Bov. Mastitis, National Mastitis Council, 181-185.

3. Chamings, R.J., 1984. "The Effect of Not Treating Milk Cases of Clinical Mastitis in a Dairy Herd." Vet. Rec. 115:499-500.

4. Erskine, R.J., et al., 1988. "Incidence and Types of Clinical Mastitis in Dairy Herds with High and Low Somatic Cell Counts." J. Am. Vet. Med Assn. 192:761-766.

5. Erskine, R.J., et al., 1990. "The Pharmacokinetics and Efficacy of Intramammary Genatamicin for the Treatment of Coliform Mastitis." Proc. Int. Symp. Bov. Mastitis, National Mastitis Council, 256-260.

6. Erskine, R.J., 1991. "Therapy of Clinical Mastitis: Successes and Failures." Proc. Nat. Mastitis Council, 30:40-49.

7. Gonzalez, R.N., et al., 1990. "Clinical Mastitis in Two California Dairy Herds Participating in Contagious Mastitis Control Programs." J. Dairy Sci. 73:648-660.

8. Hogan, J.S., et al., 1989. "Field Survey of Clinical Mastitis in Low Somatic Cell Count Herds." J. Dairy Sci. 72:1547-1556.

9. Morse, D., et al., 1988. "Characterization of Clinical Mastitis Records from One Herd in a Subtropical Environment " J Dairy Sci. 71:1396-1405.

10. Owens, W.E., et al. 1990., "Antibiotic Concentrations in Mammary Tissue and Milk Following Intramammary and/or Intramuscular Therapy." Proc. Int. Symp. Bov. Mastitis, National Mastitis Council, pp. 276-279

11. Philpot, W.N. 1979. "Control of Mastitis for Hygiene and Therapy." J. Dairy Sci. 62:168-176.

12. Soback, S., 1990. "Mastitis Therapy — Past, Present, and Future." Proc. Int. Symp. Bov. Mastitis, National Mastitis Council, pp. 244-251.

13. Tyler, J.W., et al., 1992. "Treatment of Subclinical Mastitis." Vet. Clin. N. Am. (Food Animal Practice) 8:17-28.

14. Ziv, G., 1992. "Treatment of Peracute and Acute Mastitis." Vet. Clin. N. Am. (Food Animal Practice) 8:1-16.

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