

Review Article

APPLIED NUTRITIONAL MANAGEMENT OF CLINICAL MASTITIS IN DAIRY CATTLE: A REVIEW

Saroj¹, Subha Ganguly^{2*} and Tanvi Mahajan³

¹Assistant Professor, Department of Animal Nutrition, ²Associate Professor & Head,
Department of Veterinary Microbiology, ³Assistant Professor, Department of Veterinary
Anatomy & Histology, Arawali Veterinary College (Affiliated with Rajasthan University of
Veterinary and Animal Sciences, Bikaner), N.H. – 11 Jaipur Road, V.P.O. Bajor, Dist. Sikar,
Pin – 332001, Rajasthan, India

Email: ganguly38@gmail.com (**Corresponding Author*)

Abstract: Mastitis is a disease which affects a large number of dairy animals worldwide. In a survey conducted in the major milk-producing countries, it has been seen that each year clinical mastitis afflicts 15% to 20% of cows. Mastitis means inflammation of mammary gland or udder tissue. Inflammatory response is due to injury to the body and is provoked by the infection of bacteria. Non-infectious causes like mechanical injury to teats and udder causes inflammation much like redness, swelling, and pain. Further mechanical injury may open the way for bacterial infection. Inflammation, the response of the body may be visible or invisible and in case of visible response mastitis is clinical.

Keywords: Clinical mastitis, Management, Nutrition.

Introduction

The clinical form may be mild, moderate or severe. In mild cases, visible abnormality is limited to the milk only i.e clots, flakes or watery milk. Of course, one has to look to see such changes. If cows are not fore-stripped before the milking unit is attached, mild clinical mastitis will go unnoticed. In the case of moderate clinical mastitis, both milk and udder show abnormalities. In severe cases milk, udder, and cow are affected [1]. The animal may have a fever, be off-feed, depressed, and down. Severe clinical mastitis is often called acute mastitis. Strictly speaking, acute refers to the duration of the mastitis, rather than to the severity. Clinical mastitis can be acute (just started) or chronic (e.g. chronic *E. coli* mastitis) [2, 3].

When no signs of clinical mastitis are visible, this mastitis is of subclinical type. Because subclinical mastitis often goes unnoticed, the duration of subclinical mastitis is usually longer than the duration of clinical mastitis. For many people chronic mastitis and subclinical

mastitis are more or less synonymous, although the first term refers to duration and the latter term to severity. Subclinical mastitis can be a source of infection to other animals in the herd and the cow has high somatic cell count (SCC) and production losses [4]. Many subclinical mastitis cases turn to clinical mastitis cases when a cow is in heat or when there is a change in the weather. If cow has high SCC it gives lower milk and cheese yields, shorter shelf life of processed milk, hence lower milk prices [5]. Usually, 200,000 cells/ml is used as a threshold between normal and abnormal milk, but any cow level SCC above 50,000 cells/ml is associated with a decrease in production relative to the cow's genetic potential [6]. From an economic point of view, diagnosis of subclinical mastitis is more important than clinical mastitis, even though it cannot be seen in the milking parlor. To ascertain subclinical mastitis, additional tests are available such as SCC measurement, CMT (California Mastitis Test or *paddle test*), bacterial culture, and conductivity measurements.

Nutritional factors contributing to mastitis

There are many factors responsible for causing mastitis such as climate, housing, bedding, quality of indoor air, stress, genetics and nutrition [7, 8]. Nutrition can affect the outcome of mastitis via modulation of immune system of animal. At the onset of lactation nutritional, management, and metabolic stresses, a protein or energy deprived heifer may suffer from depressed immune system in specific situations [9].

Despite a lot of studies on the nutrition, the links between diet and mastitis still raise questions in scientific circles. But it has been agreed that rapid changes in diet and excess or imbalance in the different components of rations increase the risks of mastitis [10]. Antioxidants and trace minerals influence the immune function and some aspects of health in transition dairy cows. Vitamin A and Zn affect the epithelial health, physical defense barriers of the udder, and alter the quality and quantity of keratin plug. Cu, Zn, Se, and vitamins A and E influence the phagocytic cells functions in cattle. The killing ability of immune cells is shown to be increased by nutritional supplementation with Vitamin E, which has consistently been shown to improve neutrophil function in dairy cows [11]. If there is deficiency or excess of essential nutrients in the transition cow ration can induce metabolic diseases [12, 13].

Energy Status

It is suggested to feed reduced quantities of concentrates to a cow suffering from mastitis. It appears this is also true for preventing mastitis according to a German study conducted on 1038 first lactation cows and 572 cows of successive lactations. When the cow rations

contained 25% concentrates rather than 40%, the incidence of mastitis was 7% compared to 36% for first lactation cows and 19% in comparison to 37% for other cows.

The same study also compared different energy levels in rations. A ration with high energy content had increased chances of mastitis in first lactation while it had the opposite effect on the dry cows.

Calcium-Phosphorus ratio

An inadequate calcium to phosphorus ratio in rations results in problems with milk fever at calving. If the ration is lacking calcium, up to 50% of animals will develop coliform mastitis a few hours after calving. This hypocalcemia which develops during dry periods generally results from inadequate calcium to phosphorous ratio in rations.

Silage and hay

Poor quality silage negatively affects the immune system of animal. The overheated proteins and sugars may kill the white blood cells protecting the udder. Cows which are fed on hay and grain have greater resistance to several pathogens than cows fed on silage. *Pseudomonas* and *Proteus* can survive even the high temperatures produced in silage. Silage contaminated with these microbes may then be a source of mastitis. Even moldy hay and mycotoxins destroy white blood cells and therefore weaken the immune system.

Alfalfa and other legumes

Legumes, particularly alfalfa, contain high content of estrogenic substances whose concentration varies depending on plant maturity. Even turning of these legumes into silage does not reduce their estrogenic properties which is responsible for fostering mastitis. Intake of a large quantity of estrogenic legume encourages premature development of the udder tissue and also increases the incidence of environmental mastitis.

Vitamin A and β -Carotene

The role of vitamin A and β -carotene on mastitis is still inconsistent. Positive effects has been seen on neutrophil and lymphocyte function when cows are supplemented with approximately 70,000 IU/d of vitamin A or 300 to 600 mg of β -carotene [14, 15], but clinically when similar treatments were given no effect on mammary gland health was seen [14]. Jukola *et al.* [16] suggested >3 mg/L plasma concentrations of β -carotene in dairy cows to be to optimize for udder health. Currently available data do not support feeding vitamin A in excess of the current NRC requirement (approximately 70,000 IU/d) to improve mammary gland health. Supplemental B-carotene may have some benefit if cows are in low β -carotene status (i.e., fed a diet based largely on weathered, low quality hay).

Zinc and Copper

Zinc helps in maintaining the health and integrity of skin due to its role in cellular repair, wear and tear and also increases the speed of wound healing [17, 18]. Along with its healing effect, Zn reduces SCC due to its role in keratin formation. Zinc plays a critical role in function and effectiveness of some immune components. It is an essential component of several enzymes involved in the synthesis of DNA and RNA, and has an antioxidant role by being part of a group of elements that induces the synthesis of metallothionein, which binds to free radicals [19]. As a component of the enzyme superoxide dismutase, it can stabilize cell membrane structures [20]. According to Goff and Stable [21], Zn levels in dairy cows decrease at parturition due to a decrease in DMI, transfer of Zn to colostrum, increased stress at this time, and return to baseline levels within 3-5 days postpartum. In addition, during *Escherichia coli* induced mastitis, the blood concentration of Zn declines, suggesting an antibacterial mechanism by which Zn is made less available for bacterial growth [19]. However, there are very few studies on Zn supplementation with very limited clinical data on the effect of dietary Zn on mammary gland health. Copper has also been associated with immune function. It is a component of the enzyme ceruloplasmin, which is synthesized in the liver that assists in iron absorption and transport. Furthermore, Cu is an important part of superoxide dismutase, an enzyme that protects cells from the toxic effects of oxygen metabolites released during phagocytosis. Both functions may be important in reducing the incidence of mastitis during the periparturient period [21]. Copper supplemented to heifers starting 60 d pre-calving and continuing to 30 d postpartum decreases the severity of *Escherichia coli* mastitis cases [22, 23].

Selenium and vitamin E

One of the earliest studies to address the enhancement of immune function in the dairy cow related to mastitis was done by Smith *et al.* [24] and studied the effect of vitamin E and Se on the incidence and duration of clinical mastitis. Improvements in vitamin E and Se supplementation were associated with improved phagocytic cell activity and function. In studies that followed it was observed that the optimal benefit of these two nutrients was achieved when both were supplemented in the diet of the dry cow to increase the defense against mastitis. In the case of Se, soils in many parts of the world are deficient in this mineral. Consequently, feeds grown in such soils will be Se-deficient. Therefore, heifers fed with typical diets based primarily on stored forage are likely to be prime candidates for Se and vitamin E deficiency unless diets are supplemented [25]. In a recent meta-analysis of

vitamin E supplementation research [26], vitamin E was associated with a 14% reduction in the risk of IMI, reduced somatic cell count (SCC) by a factor of 0.7 and decreased risk of clinical mastitis by 30%. Plasma α -tocopherol has been described to be very important in decreasing the incidence of new IMI at calving even in situations of low Se levels [27]. Based on these and numerous other studies, NRC (2001) increased the vitamin E requirement for dry and milking cows to 80 IU/kg DM and 15–20 IU/kg DM. Currently, suggested feeding levels are 0.3 ppmol Se (as regulated by the USFDA) and 1000 IU/day of vitamin E for dry cows [28]. Little work has been done on the appropriate recommendations for pre-fresh heifers; however, their needs are not likely to be different from adult dry cows on a metabolic body size basis. With the availability of economical feed sources of Se, supplementing this nutrient is more common than in the past. Compared to absorption of Se from inorganic sources, absorption is improved when feeding organic Se sources [30]. However, Weiss *et al.* [26] reported that improved Se absorption from Se-yeast did not enhance immunity. They postulated this lack of response was due to absorption of Se-methionine into proteins other than those involved in the immune system, which generally require Se-cystein. Over a decade, several researchers have suggested the use of supplements and the role of selenium and vitamin E in the prevention and treatment of mastitis. By maintaining an adequate level of selenium in the cow ration, helps to prevent mastitis, reduce the severity of infection and duration is also shortened. Selenium serves to reboot the immune system response by increasing the release of white blood cells and increasing the efficiency and activity of phagocytes. Both Se and vitamin E work together towards aiding the immune system of cow [29]. Thus, a 1000 IU/day of vitamin E supplement alone reduces the somatic cell count but not necessarily the incidence of mastitis.

The role of selenium is also considered to be most significant in the case of treating subclinical mastitis. In case of mastitis caused by *E. coli*, Selenium supplements have significant role. For example, cows administered with selenium supplement of 0.35 mg/kg dry matter are better able to resist *E. coli* mastitis [30]. Even duration of this type of mastitis is reduced when cows receive 2 mg of selenium per day per kilo of ration. Recommended blood levels are 0.2-1.0 g/ml for selenium and more than 4 g/ml for vitamin E. Rations should provide 1000 IU of vitamin E per day for both dry as well as lactating cow [31].

Importance: It is not useful and even harmful to give large doses of selenium only (that is, without vitamin E), because the effect can be toxic. A selenium dose of 16 mg/day results in higher levels of mastitis unless vitamin E supplements are administered at the same time [16].

Supplementation of minerals and vitamins

The level of supplementation of mineral and vitamin required should be determined by amount present naturally in forage and grain sources. If pasture is the primary forage then minerals in the soil will dictate the level in the unsupplemented diet. In periods when stored feeds are fed, heifers commonly receive the poorest forages containing the lowest levels of vitamins and perhaps minerals. Supplementing the diet of heifers with appropriate amount of minerals is very important in allowing these animals to maintain proper complete nutrient status for optimal immunity at or near calving. Since, many of the nutrients can be stored in body tissues (liver, muscle), depletion during the pre-fresh period of the heifer can be large and may create the need for higher supplementation after calving [31]. Therefore, heifer rations should be supplemented where necessary with balanced diet to assure that first lactation animals have adequate stores for themselves and for transfer into colostrums [16].

Conclusion

Mastitis as a whole is a multi-factorial disease. The cow itself, bacteria, management and the environment all collectively play a role in prevention and control of mastitis. In summary, these studies show that nutrition and feeding management can affect the outcome of mastitis and that some of these nutritional components are likely to be more specific to heifers than to older cows due to differences in their nutrient requirements and metabolism. Some specific minerals and vitamins are clear cut documented to influence mastitis, while the impact of feeding system management on mastitis is less clear. Continued research using field studies and controlled studies is needed to further define the role of nutrition in animal health and in affecting specific mastitis organisms.

References

- [1] Ganguly, S. 2014. A comprehensive and illustrious review on clinical and diagnostic aspects of mastitis infection in high yielding lactating cows. *World J. Pharma. Res.* 3(9): 352-360.
- [2] Radostits, O.M. 1961. Coliform mastitis in cattle. *Canadian Veterinary Journal.* 2:201-206.
- [3] Erskine, R.J., Eberhart, R.J., Grasso, P.J. and Scholz, R.W. 1989. Induction of *Escherichia coli* mastitis in cows fed selenium-deficient or selenium-supplemented diets. *Am. J. Vet. Res.* 50:2093-2100.

- [4] Erskine, R.J., Eberhart, R.J., Hutchinson, L.J. and Spencer, S.B. 1987. Herd management and prevalence of mastitis in dairy herds with high and low somatic cell counts. *J. Am. Vet. Med. Assoc.* 190:1411-1416.
- [5] Phelps, A. 1989. Survey shows global extent of mastitis incidence costs. *Feedstuffs*, 61(41):pp. 11.
- [6] Daley, M.J., E.R. Oldham, T.J. Williams, and P.A. Coyle. 1991. Quantitative and qualitative properties of host polymorphonuclear cells during experimentally induced *Staphylococcus aureus* mastitis in cows. *Am. J. Vet. Res.*52:474-479.
- [7] Klastrup, O., Bakken, G., Bramley, J. and Bushnell, R. 1987. Environmental influences on bovine mastitis. *Bulletin of the international dairy federation*, No. 217, pp. 37.
- [8] Klug, F., Franz, H., Bethge, B., Jansch, G. and Lemme, F. 1989. Effects of level of nutrition during early lactation on health and conception rate of group-fed dairy cows. *Tierzucht*, 43(2):56-57.
- [9] Barkema, H.W., Schukken, Y.H., Lam, T.J.G.M., Beiboer, M.L., Benedictus, G., Brand, A., 1999. Management practices associated with the incidence rate of clinical mastitis. *J. Dairy Sci.* 82: 1643–1654.
- [10] Pouden, W.D., Hibbs, J.W. and Edging, B.H. 1952. The activity of *Streptococcus agalactiae* in milk possibly influenced by the ration. *American Journal of Veterinary Research*, 13:486-499.
- [11] Politis, I., Hidioglou, N., White, J.H., Gilmore, J.A., Williams, S.N., Scherf, H. and Frigg, M. 1996. Effects of vitamin E on mammary and blood leukocyte function with emphasis on chemotaxis in periparturient dairy cows. *Am. J. Vet. Res.*57:468-471.
- [12] Grasso, P.J., Scholz, R.W., Erskine R.J. and Eberhart R.J. 1990. Phagocytosis, bactericidal activity, and oxidative metabolism of milk neutrophils from dairy cows fed selenium-supplemented and selenium-deficient diets. *Am. J. Vet. Res.* 51:269-274.
- [13] Van Saun, R.J. 1991. Dry cow nutrition: The key to improving fresh cow performance. *Vet. Clinics N. Amer.: Food Anim. Pract.* 7:599-620.
- [14] Oldham, E.R., Eberhart, R.J. and Muller, L.D.1991. Effects of supplemental vitamin A and B-carotene during the dry period and early lactation on udder health. *J. Dairy Sci.* 74:3775-3781.
- [15] Michal, J.J., Heirman, L.R., Wong, T.S., Chew, B.P., Frigg, M. and Volker, L. 1994. Modulatory effects of dietary B-carotene on blood and mammary leukocyte function in periparturient dairy cows. *J. Dairy Sci.* 77:1408-1421.

- [16] Jukola, E., Hakkarainen, J., Saloniemi, H. and Sankari, S. 1996. Blood selenium, vitamin E, vitamin A, and B-carotene concentrations and udder health, fertility treatments and fertility. *J. Dairy Sci.* 79:838-845.
- [17] Sordillo, L.M. 2005. Factors affecting mammary gland immunity and mastitis susceptibility. *Liv. Prod. Sci.* 98:89-99.
- [18] Erskine, R.J. and Bartlett, P.C. 1993. Serum concentration of copper, iron, and zinc during *Escherichia coli* induced mastitis. *J. Dairy Sci.* 76:408–413.
- [19] Prasad, A.S., Bao, B., Beck Jr., F.W., Kucuk, O. and Sarkar, F.H. 2004. Antioxidative effect of zinc in humans. *Free Rad. Biol. Med.* 37:1182-1190.
- [20] Reddy, P.G. and Frey, R.A. 1990. Nutritional modulation of immunity in domestic food animals. *Adv. Vet. Sci. Comp. Med.* 35:255–281.
- [21] Goff, J.P. and Stable, J.R. 1990. Decreased plasma retinol, α -tocoferol, and zinc concentration during the periparturient period: effect of milk fever. *J. Dairy. Sci.* 73:3195-3199.
- [22] Maddox, J.F., Reddy, C.C., Eberhart, R.J. and Scholz, R.W. 1991. Dietary selenium effects on milk eicosanoid concentration in dairy cows during coliform mastitis. *Prostaglandins*, 42(4):369-378.
- [23] Scaletti, R.W., Trammel, D.S., Smith, B.A. and Harmon, R.J. 2003. Role of dietary copper in enhancing resistance to *Escherichia coli* mastitis. *J. Dairy Sci.* 86:1240-1249.
- [24] Smith, K. L., Hogan, J. S. and Weiss, W. P. 1997. Dietary vitamin E and selenium affect mastitis and milk quality. *J. Anim. Sci.* 75:1659-1665.
- [25] Smith, K.L., Hogan, J.S. and Weiss, B.P. 1989. Dietary selenium and vitamin E influence the resistance of cows to mastitis. Pages 27 to 32 In: *Proceedings of the British Mastitis Conference*. 1989. The environment and mastitis. Cambridge, UK.
- [26] Weiss, W.P., Hogan, J.S., Smith, K.L. and Hoblet, K.H. 1990. Relationships among Se, vitamin E and mammary gland health in commercial dairy herds. *Journal of Dairy Science*, 73(2):381-390.
- [27] Ndiweni, N. and Finch, J.M. 1991. The relationship between vitamin E-selenium status and the incidence of mastitis in dairy herds near Harare. *Zimbabwe Veterinary Journal*, 22(4):101 -109.
- [28] Hemingway, R.G. 1999. The influence of dietary selenium and vitamin E intakes on milk somatic cell counts and mastitis in cows. *Vet. Res. Commun.* 23:481.

- [29] Erskine, R.J., R.J. Eberhart, P.J. Grosso, R.W. Scholz. 1989. Induction of *E. coli* mastitis in cows fed selenium-deficient or selenium-supplemented diets. *American Journal of Veterinary Research*, 50(12):2093-2100.
- [30] Batra, T.R., Hidioglou M. and Smith, M.W. 1992. Effect of vitamin E on incidence of mastitis in dairy cattle. *Canadian Journal of Animal Science*. 72(2):287-297.
- [31] Kremer, W.D., Noordhuizen-Stassen, E.N., Grommers, F.J., Schukken, Y.H., Heeringa, R., Brand, A. and Burvenich, C. 1993. Severity of experimental *Escherichia coli* mastitis in ketonemic and nonketonemic dairy cows. *J. Dairy Sci.*76:3428-3436.