



A Monthly e Magazine

ISSN:2583-2212

Jan 2024 Vol.4(1), 141-146

Popular Article

Nutritional management of Milk Fever

Sarita Mahicha¹, Monika karnani², Sheela choudhary³, Manju⁴ and Anisha⁵

Department of Animal Nutrition

Post Graduate Institute of Veterinary Education and Research (PGIVER), Jaipur

¹PhD Scholar, Department of Animal Nutrition, PGIVER, Jaipur

²Assistant professor, Department of Animal Nutrition, PGIVER, Jaipur

³Professor & Head, Department of Animal Nutrition, PGIVER, Jaipur

⁴Assistant professor, Department of Animal Nutrition, PGIVER, Jaipur

⁵PhD Scholar, Department of Animal Nutrition, PGIVER, Jaipur

<https://doi.org/10.5281/zenodo.10466702>

Abstract

Milk fever, a metabolic disorder occurrence increased during parturition due to sudden increase in calcium requirement for production of milk and colostrum. High cation diets can cause milk fever in dairy cows as they induce a metabolic alkalosis reducing the ability of the cow to maintain calcium homeostasis at the onset of lactation. Adding anions to the diet can offset the effect of the high cation forages by inducing a mild metabolic acidosis, restoring the ability to maintain calcium homeostasis. The PTH also play important role in maintaining calcium homeostasis. The calcium homeostasis can be managed by maintaining nutritional management of feeding like oral drenching of calcium, feeding of acidifying ration, feeding of prepartum vitamin D, low calcium diet in advanced pregnancy and also can provide additional supplementation of minerals.

Introduction

High-producing dairy animals are typically affected by milk fever, a metabolic disease, occurring one or two days after calving. This disease causes a significant decrease in milk production, making it the most significant economically. It is also known as periparturient paresis. Milk fever occurs in dairy cattle after calving because of low blood calcium levels as a result of calcium moving into milk. The amount of calcium found in 10 liters of colostrums is around 23 grams. When this is combined with the typical amount of calcium required for maintenance, the amount of calcium required by the cow may exceed ten times the amount found in its bloodstream. When the demand for calcium is greater than the supply in the blood, it causes the problem of milk fever, unless the cow can rapidly mobilize stored calcium in her



body (e.g. in bones) to offset the situation.

Etiology

Hypocalcaemia at parturition is due to the sudden increase in Ca requirements for the production of colostrum and due to the moving milk into colostrum.

There is a higher chance of hypocalcemia and milk fever in cows fed diets high in cations, particularly potassium and sodium, prior to calving (Ender *et al.*, 1971; Goff and Horst, 1997). A diet rich in cations raises blood pH, which leads to a mild metabolic alkalosis. Reduced bone calcium mobilization and decreased production of 1,25-dihydroxyvitamin D, the hormone form of vitamin D, are observed in cows fed diets high in cations (Block, 1984; Goff *et al.*, 1991) and reduced ability to produce the hormonal form of vitamin D, 1,25-dihydroxyvitamin D (Gaynor *et al.*, 1989; Goff *et al.*, 1989; Phillippo *et al.*, 1994).

Calcium Regulation Mechanism

Depending on their demands, cattle can absorb calcium from their stomach. They can adjust the absorption efficiency to accommodate variations in the required amount of Ca. Cattle that eat more than they require reduce the amount of Ca that is absorbed (Horst, 1986). The parathyroid gland secretes both parathyroid hormone and calcitonin, which control calcium levels. The parathyroid gland secretes parathyroid hormone (PTH) in response to a decrease in calcium intake. PTH stimulates the kidney's production of 1,25-dihydroxycholecalciferol (1,25-(OH)₂D) from 25-hydroxycholecalciferol and improves the kidney's reabsorption of calcium (Allen and Sansom, 1985). As result of stimulated 1, 25-(OH) 2D and PTH secretion, bone Ca resorption and intestinal Ca absorption increase (Horst *et al.*, 1994). The action of PTH hormone is counteracted by calcitonin (CT), which is secreted by thyroid C cells. CT decreases the concentration of Ca in blood plasma by reducing the rate of bone resorption (Allen and Sansom, 1985). Interrelationships between minerals can also influence the absorption and utilization of each other Ca has an interrelationship with phosphorus, magnesium, manganese and zinc (Underwood and Suttle, 1999). An ideal Ca:P ratio of roughly 2.3:1 was suggested to lower the prevalence of parturient paresis. Parturient paresis is more common when the Ca: P ratio falls from 2.3:1 to 1.1:1.

Predisposing Factors

- ❖ Reduced ability to mobilize calcium from the skeleton in older cows.
- ❖ High levels of oestrogen during parturition inhibit calcium mobilization.
- ❖ Low Ca intake due to reduced feed intake around parturition.



- ❖ Inhibition of calcium bone resorption in cows fed high K or high Na diets due to metabolic alkalosis.
- ❖ Slow response to increased calcium demand from high Ca intake during the dry period and reduced gut absorption of calcium due to low magnesium intake affects adaptation process and resulted in hypocalcaemia.

The normal range for the concentration of calcium in plasma is 8.8–10.4 mg/dl. At parturition, levels will drop to less than 6.0 mg/dl in 30–40% of cows.

Clinical Signs: -

Clinical manifestations of milk fever often occur when blood calcium levels are between 2.0 and 7.0 mg/dl.

The first stage is marked by an immediate onset, short duration (less than an hour), and a decrease in appetite as well as hypersensitivity, agitation and excitability.

The symptoms of the second stage, which can last anywhere from one to twelve hours, include muscle tremors, weakness, cold extremities, and a dry nose.

The third stage of the disease causes the animal to become unconscious and die within a few hours, as a result of neurological and muscle collapse. Cows with milk fever frequently lose their ability to stand because calcium is necessary for muscle contraction. The animal falls on its side, apparently comatose, its head drooping over its flank etc.

Nutritional management of milk fever

Prevention of milk fever is economically important to the dairy farmer. Following 5 principles are widely used on commercial dairy farms to control milk fever.

- Oral drenching around calving with a supplement of easily absorbed Ca salts such as calcium chloride, providing 40-50 g of Ca per dose either as bolus, gel, paste or as liquid in order to promote passive Ca transport into the extracellular fluids, oral Ca therapy likely raises luminal Ca concentration above 1 mM. The capacity of the passive transport of Ca in principle is unlimited and independent of stimulation by 1, 25-dihydroxyvitamin D. Thus, the net absorption of Ca ions increases linearly with the increasing luminal Ca concentrations. Supplying the cow with CaCl_2 , salts may furthermore, stimulate the oesophageal groove reflex due to the osmotic effect, permitting rumen bypass. A strong concentration gradient would be obtained in the abomasum, favoring passive Ca transport, and the Ca solution would avoid dilution inside the rumen as an outcome. Within five minutes of injection, there was a noticeable increase in serum total calcium; after twenty-



four hours, it returned to baseline. This makes it possible to quickly achieve a considerable rise in serum calcium, which helps to avoid milk fever during or after calving.

- B. Providing acidifying diets by supplementing them with anionic salts in the final week of pregnancy. Feeding an anionic or acidic diet may aid in the control and prevention of milk fever if it is a herd issue. By acidifying the blood and restoring tissue response to PTH, ionic salts help avoid milk fever. Dairy animals should be fed anionic salts, such as ammonium chloride, at least three to four weeks before calving. Nonetheless, a minimum of ten days of prepartum nutrition are necessary. The dietary cation anion difference (DCAD) of the prepartum ration determines the necessary amount of anionic salts, which can vary from about 50 to 500g according on the kind of salt. Urine pH should be between 5.5 and 6.5 to track the efficiency of the anionic ration.
- C. Providing low-calcium diet in the final weeks of pregnancy. Intestinal Ca absorption and bone resorption will both rise in response to lower plasma Ca, which will also activate the PTH and 1-hydroxylase system. Ca may be mobilized from bone far more quickly than from feed when the requirement for Ca rises at calving, avoiding milk fever.
- D. The management of milk fever can also be aided by the prepartum injection of vitamin D, its metabolites, and analogs; however, the timing of the treatments is crucial. It may be beneficial to inject 10 million IU of vitamin D two to eight days before to calving. The incidence is decreased when large doses of vitamin D (20–30 million U) are fed in feed for five to seven days before to parturition. The cow becomes more vulnerable if the medication is withdrawn more than four days before to calving.
- E. A sufficient intake of magnesium (Mg) supplements is essential in preventing milk fever because it is a key component in the metabolism of calcium. In accordance to research, the incidence of milk fever was lowered by 62% when magnesium supplementation was increased from 0.3 to 0.4% of diet DM.

In addition to above, management of the diet can be a valuable aid preventing milk fever. Feed a balanced ration supplemented with recommended level of vitamin D, (NRC, 2001) throughout the gestation period. If the physical state of cows needs to be improved to enhance milk production, offer them a diet high in energy but low in calcium, such as oat hay or cereal grains. Diets high in sodium or potassium alkalinize the blood, reduce the amount of calcium reabsorbed from the bone and the amount of vitamin D produced by the kidneys; they also raise the risk of milk fever. Avoid giving cows which will soon give birth a diet heavy in fodders high in calcium and phosphorus, such as alfalfa. Modify fertilization techniques to avoid high



K levels in feed (legumes and grasses) used in the close-up dry cow program in order to maintain low K levels. Give hay from sources that haven't had potash fertilizers treated recently. Provide hay from sources that not recently been treated with potash fertilizers. Insufficient magnesium during the dry season can hinder a cow's capacity to mobilize calcium after parturition.

Treatment

- i. Ca (often Ca borogluconate solution @ 300–600 ml of 40% solution) injections should be administered to cows suffering from milk fever, ideally early in the illness. To prevent heart block and prevent relapse, it can be given subcutaneously or in combination with intramuscular or intravenous injections for gradual release.
- ii. Composite solutions including Ca, Mg, P, and glucose may be advised in cases of classical or complex milk fever, where risks of milk fever other than Ca are present.
- iii. When treating recumbent cows who are not improving, immerse them in a solution containing 900 g of Epsom salts ($MgSO_4 \cdot 7H_2O$) in 3.75 L of water. Within two to four hours, this will assist remove toxins from the lower gastrointestinal tract and allow the cows to stand. Reluctant cows should be encouraged to stand up if they react to therapy; prolonged lying down can exacerbate existing conditions.

References

- Allen, W. M., and Sansom, B. F. (1985). Milk fever and calcium metabolism. *Journal of veterinary pharmacology and therapeutics*, 8(1): 19-29.
- Block, E. (1984). Manipulating dietary anions and cations for prepartum dairy cows to reduce incidence of milk fever. *Journal of dairy science*, 67(12): 2939-2948.
- Ender, F., Dishington, I. W., and Helge-bostad, A. (1971). Calcium balance studies in dairy cows under experimental induction and prevention of hypo-calcaemic paresis puerperalis. The solution of the aetiology and the prevention of milk fever by dietary means. *Zeitschrift fur Tierphysiologie, Tierernahrung und Futtermittelkunde*, 28(5): 233-256.
- Gaynor, P. J., Mueller, F. J., Miller, J. K., Ramsey, N., Goff, J. P., and Horst, R. L. (1989). Parturient hypocalcemia in Jersey cows fed alfalfa haylage-based diets with different cation to anion ratios. *Journal of Dairy Science*, 72(10): 2525-2531.
- Goff, J. P., and Horst, R. L. (1997). Effects of the addition of potassium or sodium, but not calcium, to prepartum rations on milk fever in dairy cows. *Journal of Dairy Science*, 80(1): 176-186.
- Goff, J. P., Horst, R. L., Mueller, F. J., Miller, J. K., Kiess, G. A., and Dowlen, H. H. (1991). Addition of chloride to a prepartal diet high in cations increases 1, 25-dihydroxyvitamin D response to hypocalcemia preventing milk fever. *Journal of dairy science*, 74(11): 3863-3871.
- GOFF, J. P., REINHARDT, T. A., and HORST, R. L. (1989). Recurring hypocalcemia of bovine parturient paresis is associated with failure to produce 1, 25-dihydroxyvitamin D. *Endocrinology*, 125(1): 49-53.



- Horst, R. L., Goff, J. P., and Reinhardt, T. A. (1994). Calcium and vitamin D metabolism in the dairy cow. *Journal of dairy science*, 77(7): 1936-1951.
- National Research Council. (2001). Nutrient Requirements of Dairy Cattle 7th revised edition The National Academies Press. *Washington, DC*.
- Phillippo, M., Reid, G. W., and Nevison, I. M. (1994). Parturient hypocalcaemia in dairy cows: effects of dietary acidity on plasma minerals and calciotropic hormones. *Research in Veterinary Science*, 56(3): 303-309.
- Underwood, E. J., and Suttle, N. F. (1999). *The mineral nutrition of livestock*. CABI publishing.

