



ISSN (E): 2277- 7695

ISSN (P): 2349-8242

NAAS Rating: 5.03

TPI 2019; 8(5): 228-230

© 2019 TPI

www.thepharmajournal.com

Received: 12-03-2019

Accepted: 13-04-2019

K Sethy

Assistant Professor, Department of Animal Nutrition, CV. Sc & AH, OUAT, Bhubaneswar, Odisha, India

V Dhaigude

MVSc. Scholar, Department of Animal Nutrition, CVSc. & AH, OUAT, Bhubaneswar, Odisha, India

RD Mukherjee

MVSc. Scholar, Department of Animal Nutrition, CVSc. & AH, OUAT, Bhubaneswar, Odisha, India

P Dwibedy

MVSc. Scholar, Department of Animal Nutrition, CVSc. & AH, OUAT, Bhubaneswar, Odisha, India

M Nayak

MVSc Scholar, Department of Animal Nutrition, CVSc. & AH, OUAT, Bhubaneswar, Odisha, India

P Priyadarshinee

MVSc Scholar, Department of Animal Nutrition, CVSc. & AH, OUAT, Bhubaneswar, Odisha, India

Correspondence

K Sethy

Assistant Professor, Department of Animal Nutrition, CVSc. & AH, OUAT, Bhubaneswar, Odisha, India

Calcium homeostasis in transition cows

K Sethy, V Dhaigude, RD Mukherjee, P Dwibedy, M Nayak, and P Priyadarshinee

Abstract

Milk fever is a metabolic disease of cows occurring around the time of parturition, generally within 48 hr after calving, but also occurs several weeks before or after calving. A decreased level of blood calcium is responsible for milk fever in transition cows. This is due to imbalance between calcium output in the colostrums and intake of calcium through feed. A cow producing 10 kg of colostrums will lose around 23g of calcium in single milking. This is about nine times as much as calcium present in the entire plasma calcium pool of the cow. The feedstuffs high in calcium and potassium can increase the occurrence of milk fever by reducing the mobilisation of calcium from the bones. Feeding anionic salt or manipulating the dietary cation and anion difference of the diet has become a common approach for maintaining calcium homeostasis in transition cows.

Keywords: Cow, calcium, homeostasis, milk fever

Introduction

Milk fever, a complex metabolic disease, affects high producing dairy animals usually within one or two days after calving, resulting in a huge reduction in milk production and thus becomes economically most important. Although treatment with intravenous infusion of calcium salt solutions cure most clinical cases of hypocalcaemia, such cows are later more susceptible to other metabolic and infectious diseases (Curtis *et al.*, 1985) ^[1]. Clinical symptoms of this disease include inappetence, tetany, inhibition of urination and defecation, lateral recumbency, and eventual coma and death if left untreated. It is also known as parturient paresis. It is estimated that this disease affects 3 to 8% of cows with some herds having prevalence as high as 25 to 30%. Symptoms appear when blood calcium levels are low; hence the synonym of milk fever is hypocalcaemia. Incidence of milk fever is increased with cow age. Economic losses due to medicines, veterinary services and reduced production increased significantly in a commercial farm with occurrence of milk fever (Mandali, 2004) ^[2]. Losses are also associated with increased incidence of secondary diseases, such as ketosis, mastitis, retained placenta, displacement of abomasum, uterine prolapse, limb injuries, and pneumonia can further inflate losses. Milk fever occurs in dairy cattle after calving because of low blood calcium levels as a result of calcium moving into milk (Chamberlain and Wilkinson, 1996) ^[3]. There are about 23 grams of calcium in 10 liters of colostrums, and when this is added to the normal amount of calcium needed for maintenance, the needs of the cow can be more than 10 times the supply of calcium in her 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 (Westerhuis, 1975) ^[4].

Calcium regulation mechanism

Cattle can absorb Ca from gut according to their needs. They are able to alter the absorption efficiency to meet changes in Ca requirement. When cattle consume more Ca than needed, the proportion of Ca absorbed is decreased (Horst, 1986) ^[5]. Ca is regulated by parathyroid hormone (PTH) and calcitonin, which are secreted from parathyroid gland and thyroid gland respectively. The decrease in Ca intake stimulates the secretion of parathyroid hormone (PTH) from the parathyroid gland. PTH enhances renal reabsorption of Ca (Capen and Rosol, 1989) ^[6] and promotes the synthesis of 1,25-dihydroxycholecalciferol from 25-hydroxycholecalciferol in the kidney (Allen and Sansom, 1985) ^[7]. As result of stimulated 1,25-dihydroxycholecalciferol, bone Ca resorption and intestinal Ca absorption increase (Horst *et al.*, 1997) ^[8].

The action of PTH hormone is counteracted by calcitonin, which is secreted by thyroid C cells. Calcitonin decreases the concentration of Ca in blood plasma by reducing the rate of bone resorption (Allen and Sansom, 1985) ^[7]. 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) ^[9]. Recommended

optimal Ca: P ratio to reduce the incidence of parturient paresis were approximately 2.3:1. When the Ca: P ratio decreased from 2.3:1 to 1.1:1 the incidence of parturient paresis increases. A nutritional approach to manage milk fever involves monitoring specific elements in the diet. The nutritional factors responsible for milk fever were presented in Table 1.

Table 1: Nutritional factors responsible for milk fever

S. No.	Factor	Effect
1	General Nutritional status at Prepartum period	Increase K - pasture, Over fat cow and very thin cows
2	Reduce feed intake on day of calving	Reduce Ca intake and absorption
3	Increase Ca intake prepartum	Increase passive absorption and quiescence of Parathyroid gland
4	Increase P intake prepartum (>80 g P/d)	Inhibits Vit-D metabolism
5	Reduce Mg concentration at prepartum	Reduce production and secretion of PTH
6	Dietary cation anion differences	Increase DCAD balance

Strategies to prevent milk fever

The common strategies to prevent milk fever were summarized below

1) Low calcium feeding (often with relatively high phosphorus)

The principle of feeding rations with low calcium is highly significant in milk fever prevention provided the calcium intake in the dry period is kept below 20 g per day (Thilising-Hansen and Jorgensen, 2001) ^[10]. The main problem in implementing the low Ca principle is difficulties in formulating rations sufficiently low in calcium when using commonly available feeds. It works by ensuring that the dry cow is mobilising calcium, so that when it switches to milk production it is better able to cope (Wiggers *et al.*, 1975) ^[11]. A useful analogy is to think of the lactating cow as an athlete, it's much more likely to do better if it has a bit of training before it needs to compete. Low calcium in the dry period provides that training. But due to low palatability and difficulty in formulating low calcium diets, economic conditions and time-consuming processes are the major constraints in this aspect. Lack of organized dairy farms is also a reason for not implementing milk fever control procedures in India. These issues should be thoroughly considered before selecting the method for milk fever prevention in a farm.

2) DCAD (Dietary cation anion difference) strategy

Dietary cation-anion balancing is a new concept that has received much attention recently as a nutritional tool for reducing milk fever in early lactation as well as improving health and production (Hemsley, 1957) ^[12]. The dietary electrolytes are balanced according to the charges they contain. Because cations are positively charged and anions are negatively charged, the ration is balanced to be either negative or positive. A negatively balanced ration favours prepartum dry cows and reduced incidence of milk fever, whereas a positively balanced ration favors lactating cows and increased levels of milk production (Julien *et al.*, 1977) ^[13]. That is, lactating cows do better with a positively balanced ration and prepartum cows with a negatively balanced ration. Mineral elements considered in cation-anion balancing are sodium, potassium, chlorine and sulfur. To create a well-balanced ration using the cation-anion approach, about 150 to 250 total grams of a combination of compounds such as ammonium chloride, ammonium sulfate, calcium chloride, calcium sulfate and magnesium sulfate are needed daily per cow

(Kichura *et al.*, 1982) ^[14]. The amount needed will vary with the concentration of potassium and sodium in the ration. Because anionic diets stimulate greater mobilization of calcium from bone, they should not be fed the entire dry period. The anionic salts used in the prevention of milk fever are quite unpalatable to dairy cows. As a result, they should be used with caution and mixed thoroughly in the dry cow ration.

A cation-anion balance is calculated by subtracting anion milli equivalents from cation milli equivalents (meq.) (Moodie and Robertson, 1962) ^[15]. While several formulas have been used, the following equation is suggested for dry cows.

Cation-anion balance = $\text{meq}[(\text{Na}+\text{K})-(\text{Cl} + \text{S})] / 100 \text{ gms DM}$
Dietary K plays a significant role in predisposing cows to milk fever. An effective means of offsetting the detrimental effects of K is to increase the anions content of diets (Mullen, 1975) ^[16]. It has the distinct disadvantage of being unpalatable. The current understanding of the cation anion difference concept suggests that milk fever could be managed more effectively if dietary K was reduced (Horst *et al.*, 1994) ^[17]. High concentrations of potassium in rations may lead to a greater incidence of hypocalcemia and possibly milk fever in a dairy herd. A number of studies show less milk fever when cows are fed a ration with a negative cation-anion balance during the late dry period. The reduction in incidence of milk fever appears to be due primarily to the greater mobilization of calcium from bone stores. Research has shown that cows fed anionic diets have higher blood calcium levels at calving (Sampson *et al.*, 1983) ^[18].

Altering the balance of the diet by feeding an excess of strong anions (primarily chloride and sulphide) would change the pH of the blood, if the body's mechanisms didn't prevent it. One of the mechanisms that prevents this is the calcium metabolism, thus feeding an 'anionic diet' is another way of training the cow before it starts milking (Hove and Hilde, 1984) ^[19]. This can be very effective, but it is more difficult to undertake than the low calcium strategy. Potential problems include low palatability of some anionic salts and the requirement for regular urine pH testing. If the DCAD strategy is not done correctly it can lead to significantly increased milk fever levels. When using the DCAD principle, rations feeding with a negative DCAD (measured as $(\text{Na} + \text{K}) - (\text{Cl} + \text{S})$) significantly reduce the milk fever incidence (Thilising-Hansen and Jorgensen, 2001) ^[10]. But the main drawback from the DCAD principle is a palatability problem.

3) Drenching or pasting

Oral drenching of calcium after calving apparently has a mean efficacy of 50%–60% in terms of milk fever prevention as well as prevention of milk fever relapse after intravenous treatment with calcium solutions (Vagnoni and Oetzel, 1998) [20]. However, some drenches have been shown to cause lesions in the fore stomach. Several commercial products are available for boosting blood calcium. Best results are obtained if the first dose is given just before calving (usually around 8 hours before) with a second dose 24 hours later. This obviously requires calving to be accurately predicted. It is probably used in herds suffering from a high level of metabolic disease around calving. (Jönsson and Pehrson, 1970) [21].

4) Vitamin D and milk fever prevention-

Twenty million U.S.P. units of vitamin D fed daily to dairy cows three days prior and one day following parturition sharply reduced the incidence of milk fever. (Green *et al.*, 1981) [22]. Prepartum administration of vitamin D, vitamin D metabolites and analogues also significantly reduced the incidence of milk fever (Thilsing-Hansen and Jorgensen, 2000) [23].

Conclusion

Milk fever is a common problem in high yielding animals, which causes economic losses to the farmers and makes the animal prone to other transitional diseases. Altering the nutritional requirement of the animals can prevent milk fever in a herd.

References

1. Curtis CR, Erb EH, Snioen CJ, Smith RD, Kronfeld DS. Path analysis of dry period nutrition, postpartum metabolic and reproductive disorders, and mastitis in Holstein cows. *Journal of Dairy Science*. 1985; 68:2347-2360.
2. Mandali GC, Patel PR, Dhami AJ, Raval SK. Epidemiological surveillance on effect of housing, hygiene and nutritional status of periparturient disorders in buffaloes. *Indian Journal of Dairy Science*. 2004; 57:132-136.
3. Chamberlain AT, Wilkinson JM. Minerals and vitamins. In: *Feeding the dairy cow*. Chalcombe publications, Great Britain, 1996, 79-94.
4. Westerhuis JH. Parturient hypocalcaemia prevention in parturient cows prone to milk fever by dietary measures. *Tijdschr. Diergeneeskd*. 1975; 100(4):213-220.
5. Horst RL. Regulation calcium and phosphorus homeostasis in the dairy cow. *Journal of Dairy Science*. 1986; 69:604-616.
6. Capen CC, Rosol TJ. Calcium regulating hormones and diseases of abnormal mineral metabolism. In: *Clinical biochemistry of domestic animals*. Kaneko JJ. (ed.). 4th ed. Academic Press, Inc. San Diego, California, USA, 1989, 678-752.
7. Allen WM, Sansom BF. Milk fever and calcium metabolism. *Journal of Veterinary Pharmacology and Therapeutics*. 1985; 8:19-29.
8. Horst RL, Goff JP, Reinhardt TA, Buxton DR. Strategies for preventing milk fever in dairy cattle. *Journal of Dairy Science*. 1997; 80:1269-1280.
9. Underwood EJ, Suttle NF. *The mineral nutrition of livestock*. 3rd ed. CAB International, Wallingford, UK: 614 (<http://ethesis.helsinki.fi>), 1999.
10. Thilsing-Hansen T, Jørgensen RJ. Prevention of parturient paresis and subclinical hypocalcemia in dairy cows by Zeolite A administration in the dryperiod. *Journal of Dairy Science*. 2001; 84:691-693.
11. Wiggers KD, Nelson DK, Jacobson NL. Prevention of parturient paresis by low-calcium diet prepartum: A field study. *Journal of Dairy Science*. 1975; 58:430-431.
12. Hemsley LA. Some observations on milk fever. *Veterinary Record*. 1957; 69:464-468.
13. Julien WE, Conrad HR, Hibbs JW, Crist WL. Milk fever in dairy cows. Effect of injected vitamin D3 and calcium and phosphorus intake on incidence. *Journal of Dairy Science*. 1977; 60:431-436.
14. Kichura TS, Horst RL, Beitz DC, Littledike ET. Relationships between prepartal dietary calcium and phosphorus, vitamin D metabolism and parturient paresis in dairy cows. *Journal of Nutrition*. 1982; 112:480-487.
15. Moodie EW, Robertson A. Some aspects of calcium metabolism in the dairy cow. *Research in Veterinary Science*. 1962; 3:470-484.
16. Mullen PA. Clinical and biochemical responses to the treatment of milk fever. *Veterinary Record*. 1975; 97:87-92.
17. Horst RL, Goff JP, Reinhardt TA. Calcium and vitamin D Metabolism in the dairy cow. Symposium: calcium metabolism and utilization. *Journal of Dairy Science*. 1994; 77:1936-1951.
18. Sampson BF, Manston R, Vagg MJ. Magnesium and milk fever. *Veterinary Record*. 1983; 112:447-449.
19. Hove K, Hilde BL. Plasma calcium in the lactating cow: Dependence on continuous intestinal absorption. *Can. Journal Animal Science*. 1984; 64:227-228.
20. Vagnoni DB, Oetzel GR. Effects of dietary cation-anion difference on the acid-base status of dry cows. *Journal of Dairy Science*. 1998; 81:1643-1652.
21. Jönsson G, Pehrson B. Trials with prophylactic treatment of parturient paresis. *The Vet Record*. 1970; 87:575-583.
22. Green HB, Horst RL, Beitz DC, Littledike ET. Vitamin D metabolites in plasma of cows fed a prepartum low-calcium diet for prevention of parturient hypocalcemia. *Journal of Dairy Science*. 1981; 64:217-226.
23. Thilsing-Hansen T, Jørgensen RJ. A novel hypothesis for the prevention of parturient hypocalcaemia: 2. Proof of concept. *Proc. World Buiatrics Congress, Punta del Este, Uruguay*. 2000; 21:s6481-6482.