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Popular Article

Developments in dietary cation-anion difference (DCAD) concept as an effective tool for preventing milk fever in dairy cows

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Introduction

Milk fever or parturient paresis is one of the most commonly occurring metabolic diseases of high yielding dairy cows. Milk fever occurs immediately after calving for acute deficiency of calcium as a result of huge drainage of calcium through colostrum, and subsequently through milk, unless the cow is supplemented with enough calcium to meet her requirement or she can rapidly mobilize calcium from her body stores mainly from bones.

Maintenance requirement of calcium of cows varies from 0.25 to 0.35% in daily ration on dry matter (DM) basis. Normal cow's ration may not be able to provide sufficient calcium to meet her daily requirement during the early lactation period. Bovine colostrum contains 2.6 to 4.7 g calcium/kg of colostrum; whereas, milk contains 1.2 to 1.3 g calcium/kg of milk (Playford and Weiser, 2021) produced and these quantities must be provided extra over and above the daily maintenance requirement of calcium. For production of every 10 liters of colostrum and milk, there will be excretion of 26 to 47 g and 12 to 13 g of calcium, respectively from the body of the cow. This indicates requirements of around 78 to 131 g calcium during the 1st 3-4 days of lactation and around 36 to 39 g of calcium in the subsequent period of lactation. Therefore, if the cow's daily feed consumption can not meet her calcium demand, there will be acute calcium deficiency and the cow will suffer from milk fever or parturient paresis.

Milk fever usually occurs 48 hours after calving. As this condition results acute to peracute afebrile, flaccid paralysis soon after calving, it is also called parturient paresis. The

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initial signs of milk fever are restlessness, excitability and anorexia which will be followed by three different stages – stage 1 characterized by the cow still will be able to stand, stage 2 characterized by lateral recumbency for hindquarter paralysis and stage 3 characterized by unresponsiveness, coma and death.

There are many nutritional strategies which can be adopted to prevent milk fever in high yielding dairy cows. Amongst these, maintenance of optimum dietary cation-anion difference (DCAD) is one of the effective preventive measures to prevent the occurrence of milk fever in dairy cows. As reported by Razzaghi *et al.* (2012), feeding ration with negative DCAD in late gestation period and high DCAD during early lactation improved performance and productivity of dairy cows. The DCAD and milk production are interrelated through acid-base regulation (Sanchez and Beede, 1994; Hu and Murphy, 2004). Reducing DCAD by feeding anionic salts causes an influx of negatively charged ions leading to increased hydrogen ion concentration which induces a mild metabolic acidosis to increase bone resorption, blood calcium and intestinal calcium absorption (Horst *et al*., 1997).

Calcium homeostasis mechanism in dairy cows

Calcium present in the blood may be in ionic form (Ca^{2+}), free form, in bound form with proteins and as complexes with anions. Ionic form of calcium is only biologically active. When blood calcium level decreases, it stimulates parathyroid gland to secrete parathyroid hormone (PTH). PTH binds to its hormone receptors in kidneys and bone tissues. The PTH increases renal reabsorption of calcium by increasing the production of 1,25-dihydroxycholecalciferol (Vitamin D_3). Vitamin D_3 also stimulates the intestinal epithelial cells to increase absorption of calcium. If calcium in the ration is not sufficient, the mechanism is directed to bone tissues. There will be reabsorption of calcium from bone tissues and the calcium level in the blood will be maintained.

Dietary cation-anion difference (DCAD) concept

The DCAD of ration is directly related with incidence of milk fever. The DCAD is related with mineral homeostasis and acid-base balance. It is a measurement of strong electrolyte concentration. The DCAD of ration can easily be calculated if percentages of concentrations of sodium, potassium, chlorine and sulphur ions of the ration are known.

$$DCAD = (Na + K) – (Cl + S).$$

DCAD can be determined by evaluating the cation-anion status of the feed ingredients or complete ration. While evaluating cation-anion content, cations and anions present in the drinking water should also be considered. After estimating the cations and anions, their levels

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have to be converted into milliequivalents (mEq) to calculate DCAD of the ingredients or ration. This can be done by multiplying the level of each cation and anion of the ingredients or ration by a conversion factor. The conversion factors for calculating mEq/kg DM of Na, K, Cl and S are 435, 256, 282 and 624, respectively.

If a ration contains 0.25% sodium, 1.2% potassium, 0.4% chloride and 0.5% sulfur, their level (mEq/kg DM) will be 108.75, 307.20, 112.80 and 312.00, respectively and thus -

$$\text{DCAD (mEq/kg)} = [(0.25 \times 435) + (1.2 \times 256)] - [(0.4 \times 282) + (0.5 \times 624)]$$
$$= (108.75 + 307.20) - (112.80 + 312.00)$$
$$= -8.85 \text{ i.e., Negative DCAD}$$

Relationship between milk fever and DCAD level of the ration

Cattle fed ration with a high DCAD tends to cause milk fever, whereas negative DCAD tends to prevent milk fever.

Positive DCAD diets reduce tissue sensitivity to parathyroid hormone. The metabolic alkalosis associated with high DCAD diets induce a state of pseudo hypothyroidism at the onset of lactation resulting is hypocalcemia and milk fever. Excesses of absorbed cations (on high DCAD diets) causes metabolic alkalosis in dairy cows. This is directly related with diminished parathyroid hormone stimulation of renal vitamin D activation resulting in reduced calcium absorption and reabsorption.

Negative DCAD diets cause compensated metabolic acidosis in prepartum dairy cows. It is related with decreased urine pH and increased urinary calcium excretion. Calcium homeostasis is maintained with simultaneous increased bone resorption of calcium, increased absorption of calcium from the rumen and small intestine and mobilization of calcium from bone. This calcium flux helps to meet high calcium demand at the initial lactation period. When excess dietary Cl is excreted in the urine, it requires a corresponding cation to maintain a neutral charge. Low K diets stimulate hydrogen ion (low pH) secretion and increase calcium excretion in the urine. Increased loss of Ca in the urine increases the cow's metabolic mechanisms for increased resorption of Ca from bone and increased intestinal absorption of dietary Ca so that the cow is able to regulate blood Ca level more effectively when there is increased demand for Ca at the time of calving.

Dietary manipulation of DCAD

Reducing calcium content in the ration of prepartum cows has become a choice for the dairy farmers to prevent milk fever. This strategy helps in maintaining the parathyroid gland

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active during the dry period. However, this strategy has some disadvantages also. Feeding of more concentrates and cereal silages to reduce calcium intake may predispose some other metabolic conditions like fatty liver syndrome, ketosis, abomasal displacement etc. High energy density ration may also induce acidosis or sub-acute ruminal acidosis (SARA) which might affect productivity in the subsequent lactation. Therefore, to prevent milk fever in high producing dairy cows, reduction of DCAD in the ration rather than calcium content has become a choice during the prepartum (before calving) in dairy cows. The DCAD in the ration can be reduced by adding anionic salts (i.e., salts of chloride, sulphur or phosphorus) to the ration. Typically, DCAD ranges from +100 to +200 mEq/ kg DM of the ration (1 mEq is equal to $1/1000^{th}$ of equivalent weight). Addition of anionic salts (minerals high in Cl and S relative to Na and K) or mineral acids to the ration lowers DCAD in the ration and thereby reduces the chances of occurring milk fever in dairy cows. It is known that by adding three equivalents of anions to 12 kg ration can lowers DCAD by 250 mEq/kg.

Cations like sodium (Na), potassium (K), calcium (Ca) and magnesium (Mg) increase pH of the blood, thereby creating an alkaline metabolic state; whereas, anions like chloride (Cl), sulfur (S) and phosphorus (P) lower pH of the blood thereby creating an acidic metabolic state in the body. However, cows can maintain acid-base balance (Homeostasis) by buffering of the blood by mobilizing calcium phosphate from the bones. Therefore, by feeding more anions will lower blood pH causing cows to mobilize calcium from calcium stores of her body which can prepare her better for her subsequent lactation when calcium will be lost in the milk.

DCAD during transition period

The dairy cows during their late pregnancy period should be provided with a ration with negative DCAD or DCAD less than +50 mEq/kg DM of the ration. Reduced or negative DCAD will not only prevent milk fever, but also incidences of other associated metabolic conditions like fatty liver, retained fetal membrane, abomasal displacement, udder edema and metritis. Dietary DCAD should be reduced to -5 to -10 mEq/kg DM to improve cow health and lactation performance. This can be done by either reducing the dietary Na and K level or by increasing intake of Cl and S by supplementation of anionic salts like ammonium chloride, calcium chloride, magnesium chloride, ammonium sulfate, calcium sulfate, magnesium sulfate etc. To negate palatability issue, molasses may be supplemented in the ration. With DCAD of -50 to -100 meq/kg of dietary DM, and urine pH of 6.0 to 6.7 indicates the effectiveness of anionic salt supplementation.

The pH of urine of dairy cows fed normal rations ($1/3^{rd}$ of total DM requirement as

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concentrates and 2/3rd of total DM requirement as roughages) ranges between 7.8 and 8.2, which is normal for ruminants. Sometimes without anionic salt supplementation, pH of urine may be within the normal range when DCAD of the ration is +20 mEq or more without any effects on transition performance of cows. Thus, when anionic salts are supplemented to reduce DCAD of the ration, urine pH of the cow can be the indicator of effectiveness of their supplementation for maintaining acid-base homeostasis and Ca status. However, optimum intakes of TMR with DCAD ranging from -5 to -10 mEq/kg during transition period, reduced urine pH may indicate proper anion intake.

DCAD for lactating dairy cows

For lactating dairy cows, dietary DCAD ranging from +250 to +300 mEq/kg DM is effective and sufficient for optimum feed intake and milk yield (Beede, 2005). As high milk yield warrants feeding of high energy-protein diet and nutrient fortification postpartum, increased ruminal fermentation of feeds and systemic metabolism generate higher amounts of acids which demands increased dietary DCAD in lactation ration to neutralize the acids. This can be achieved by reducing high anion-containing ingredients or by supplementation of buffers like sodium bicarbonate or potassium carbonate in the lactation ration. Both Na and K are equally effective for optimum lactation performance if a greater DCAD is required. Thus, under this circumstance, supplementation of cation sources is recommended. However, for optimum nutrient balance, it is recommended to reduce supplementations of K, Na, Cl and sulfate, as the amounts consumed in excess of requirements are excreted and may pose to environmental hazards.

Guidelines to prevent milk fever by maintaining DCAD of the ration

If DCAD level of prepartum ration is found to be +200 mEq/kg dry matter or more, concentrates/forages containing very low potassium level should be introduced. As the anionic salts are bitter in taste, supplementation may reduce feed intake and this may predispose negative energy balance, lower protein intake and other nutrient deficiencies leading metabolic conditions like ketosis and displaced abomasum etc. However, by gradually incorporating anionic salts through total mixed ration (TMR) to dairy cows may reduce the palatability issue.

The DCAD of the transition ration should be between -100mEq/kg to -200 mEq/kg dry matter.

The pH of the urine of the transient cow should be evaluated periodically. As pH of the urine is also an indicator of metabolic pH status, urine pH should range from 6.0 to 6.5 for H & F and 5.5 to 6.0 for Jerseys.

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Feeding feed ingredients with low DCAD can help in reducing incidence of milk fever. Ingredients like soybean meal, lucerne hay, barley, and cereal silages are high in K and therefore contribute to high DCAD of the ration. Silage of maize is intermediate in DCAD content. Protein supplements such as DDGS and canola meal are intermediate in K content and are low DCAD feeds because of their high S content. Most of the forages high in NDF are also high DCAD feeds for their high K content. Thus, ingredient selection is very important while formulating diet for cows during transition and early lactation period.

While restricting calcium intake during prepartum period has been demonstrated to reduce clinical milk fever n dairy cows. However, this approach is very difficult to achieve as commonly utilized feeds (protein, forages and grains) are rich in Ca and thus, all contribute to Ca level in the diet.

Supplementation of Vitamin D before calving is another approach to prevent milk fever in dairy cows.

Reducing dietary K intake during transition period is another approach to reduce incidence of milk fever in dairy cows. Low K level will lower DCAD of the diet.

Calcium is a strong cation and thus addition of Ca to the diet increase the blood pH and may predispose milk fever. Studies indicated that prepartum diets high in Ca is associated with increased incidence of milk fever. When cows are fed low Ca diet prior to calving, negative Ca balance stimulates secretion of PTH and vitamin D_3 production activating bone osteoclasts, stimulating bone Ca resorption and activating renal tubules to resorb urinary Ca. When Ca homeostatic mechanisms are active at the onset of lactation, it prevents a severe decline of plasma Ca concentration of the cow and thus, the occurrence of milk fever.

Conclusion

Cow's calcium requirement increases significantly during transition period (3 weeks before and after calving) and in subsequent lactation. If requirement exceeds dietary supply and ability of the cow to mobilize calcium, hypocalcemia occurs leading to milk fever negatively impacting the productivity of the dairy cows. Dietary DCAD is an important consideration for dairy cows during transition and early lactating period to prevent occurrence of milk fever. The minimal DCAD for lactating cows is about 300 mEq/kg DM of the ration. For reducing both clinical and subclinical hypocalcemia, feeding of negative DCAD diet for at least 21 days pre-partum is recommended in dairy cows. Relationship between dietary DCAD and milk fat content should also be taken into consideration while formulating DCAD diet as this has economic implication for the farmers.

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