

DIETARY CATION – ANIONIC BALANCE (DCAB) TO PREVENT MILK FEVER

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Abstract: Milk fever is an economically important metabolic disease, which not only reduces the productive life of a cow, but also increases the cost of milk production. In dry period, calcium requirements are minimal, but just after parturition these requirements increase and plasma calcium concentration declines due to calcium loss in each milking. As a consequence of this sudden change, nearly all cows experience some degree of hypocalcaemia, during first day after calving. Plasma calcium is controlled by the calcitropic hormone: (parathyroid hormone (PTH) and 1, 25-dihydroxyvitamin D₃), which are produced in response to hypocalcaemia and act to increase the entry of Ca²⁺ into the plasma Ca²⁺ pool. Major underlying cause of milk fever is metabolic alkalosis. Incidence of milk fever depends on the abundance of the cations, relative to the anions which is generally referred to as the dietary cation-anion balance (DCAB). Anion addition reduces both blood and urine pH and is associated with reduction in milk fever as opposed to cations, which increases blood and urine pH associated with increased the incidence of milk fever. Low DCAB prevents metabolic alkalosis by increasing target tissue responsiveness to PTH, which controls renal 1- α -hydroxylase and reabsorption of bone calcium. Mild metabolic acidosis induction with the addition of anions can increase calcium mobilization from bone by releasing calcium from bone surfaces, increase orthoclastic bone resorption and synthesis of 1, 25-dihydroxyvitamin D₃ in cows. Elemental sulphur can be used to raise dietary DCAB, but no evidence exists that elemental sulphur (as opposed to sulphate) acidifies the blood. High dietary calcium increased the risk of milk fever. During prepartum period, low calcium diets stimulate PTH and 1, 25-dihydroxyvitamin D₃ production which at the time of parturition helps to maintain the calcium level but currently it has been noted that high dietary calcium is required to enhance the efficacy of anionic diets.

Keywords: DCAD, Parturient Paresis, Milk Fever, Calcium, Economics.

PATHOPHYSIOLOGY OF MILK FEVER

Calcium is a macro-mineral that has important functions in the body. Among them are the bone matrix, the process of muscle contractor and transmission of nerve impulses. Ionized calcium is also necessary for vital cellular functions such as signalling, neurotransmission, muscle contraction, metabolism, growth and proliferation and activation of immune (osteoclast activation) responses. Normal blood calcium in the adult cows is maintained

between 8.5 and 10 mg/dl. Maintenance of blood calcium within the acceptable range is a balancing act between the calcium demand of milk production and the cow's homeostatic mechanisms to maintain blood calcium. During the dry period, the supply of calcium through the diet is usually more than adequate to maintain homeostasis without activating the calcium mobilization system, which is thus usually not activated until parturition. Therefore, dry period is the phase most important in the development of milk fever.

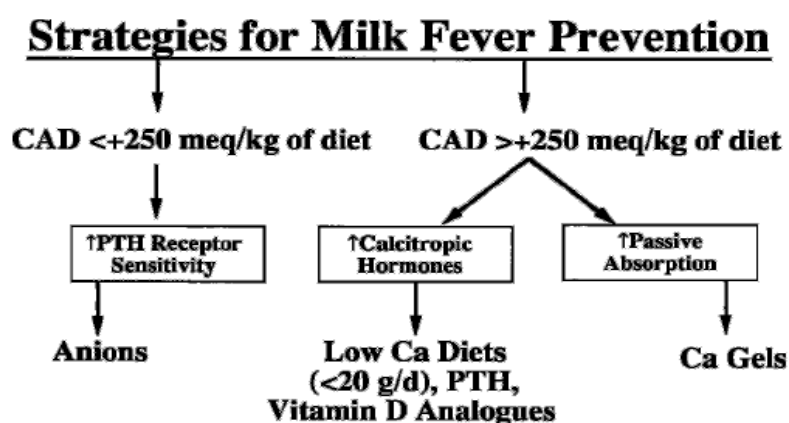
When intake and homeostatic mechanisms cannot meet the increased demands for calcium metabolism, the pathogenesis of hypocalcaemia is initiated. The level of calcium in plasma is well regulated and when the level decreases, the parathyroid gland will excrete parathyroid hormone (PTH). This increases the mobilization of calcium from the skeleton and also raises the renal threshold for calcium in the kidneys. The regulation of serum calcium is controlled by three potent calcitropic hormones, parathyroid hormone (PTH) secreted from the parathyroid gland, $1, 25\text{-(OH)}_2\text{D}_3$, a metabolite of vitamin D produced in the kidney and calcitonin, while calcitonin plays a valuable feedback relationship with hypocalcaemia, or managing blood calcium concentrations after an intravenous calcium treatment, it has a lesser impact on calcium homeostasis. Factors such as the production of milk, age and breed are predisposing cows to have the metabolic disturbance, since cows for producing more secreted calcium should have efficient metabolism to meet increased demand. The great demand of calcium in early lactation to produce 10 litres of colostrum, cow losses 23g of calcium in a single milking (2.3 g/L) which is about nine times more present in the plasma compartment.

The calcium lost from the plasma compartment should be replaced by intestinal calcium absorbed and bone reabsorption. During the dry season these mechanisms are inactive and all cows undergo hypocalcaemia in the first days after birth until the intestines and bones are adapted. The adaptation starts with increased PTH and $1, 25\text{-(OH)}_2\text{D}_3$ at the beginning of hypocalcaemia. About 24 hours of stimulation of $1, 25\text{-(OH)}_2\text{D}_3$ is required for intestinal calcium transport increase significantly. Bone reabsorption (osteoclast recruitment and activation) is not increased until 48 hours after the stimulation of PTH. When these compensatory mechanisms are prolonged, clinical hypocalcaemia or milk fever develops. Consequently, most cows with clinical hypocalcaemia have higher levels of PTH and $1, 25\text{-(OH)}_2\text{D}_3$.

Mechanisms that may explain the detrimental effects of hypocalcaemia include impaired energy balance, which is reflected in higher serum NEFA concentrations and direct

impairment of immune cell responses to an activating stimulus. According to Iggo Hypocalcaemia affects muscular contraction mainly in three ways. Firstly, calcium has a membrane stabilizing effect on the peripheral nerves. Hyperesthesia and mild tetany seen in early stages of milk fever are due to lack of nerve cell membrane stabilization. Secondly, calcium is required for the release of acetylcholine at the neuromuscular junction. The inability to release acetylcholine, due to hypocalcaemia causes paralysis by blocking the transmission of nerve impulse to the muscle fibres. Thirdly, calcium is directly required by muscle cells for contraction. Paralysis of various muscle types results in the clinical signs of parturient paresis. Currently it is known that the pathogenesis of the disease is much more associated with the action of PTH on cells responsible for demineralization (osteoclasts), cells of the intestine responsible for absorption and kidney cells responsible for the reabsorption of calcium in the tubules.

PREVENTION OF MILK FEVER



Formulation of anionic salt

Acidifying diets (either by addition of anionic salts or mineral acids) are commonly used to prevent milk fever in dairy cattle. The ability of different diets to cause acidification of the cow can be explained by dietary cation-anion difference (anion-cation balance, dietary electrolyte balance, strong ion difference). The DCAD of diets can be manipulated with relative ease, simply by adding relatively more anions or cations.

Understanding how DCAD affects calcium metabolism first requires a short review of chemistry. Dietary electrolytes can be classified as either anions or cations. Anions have a negative charge; cations have a positive charge. The charge carried by these electrolytes affects acid-base balance and ultimately calcium metabolism. Important dietary cations are sodium, potassium, calcium, and magnesium; important dietary anions are chloride, sulfur,

and phosphorus. Sodium, potassium, sulfur, and chloride (the monovalent ions) are thought to exert the strongest ionic effects on acid-base balance and are referred to as the "strong ions".

Dietary cation-anion difference can be used to determine the relationship between strong cations and anions and thus predict whether a diet will evoke an acidic or alkaline response when fed to a dairy cow. Several equations for calculating DCAD of a diet have been proposed:

$$\text{DCAD (meq)} = (\text{Na} + \text{K} + \text{Ca} + \text{Mg}) - (\text{Cl} + \text{SO}_4 + \text{H}_2\text{PO}_4 + \text{HPO}_4)$$

$$\text{DCAD (meq)} = (\text{Na} + \text{K} + \text{Ca} + \text{Mg}) - (\text{Cl} + \text{S} + \text{P})$$

$$\text{DCAD (meq)} = (\text{Na} + \text{K} + 0.15 \text{ Ca} + 0.15 \text{ Mg}) - (\text{Cl} + 0.20 \text{ S} + 0.30 \text{ P})$$

$$\text{DCAD (meq)} = (\text{Na} + \text{K}) - (\text{Cl} + \text{S})$$

$$\text{DCAD (meq)} = (\text{Na} + \text{K}) - (\text{Cl})$$

Calculation of the DCAD of a diet, regardless of the equation employed, requires using the equivalent weights of the electrolytes. This is necessary because acid-base balance is affected by electrical charge rather than mass. The equivalent weight is equal to the molecular weight divided by the valency (electrical charge strength). The term mill equivalent (meq) is used to express equivalent weights in milligram; one mill equivalent equals 1/1000th of an equivalent.

MANIPULATING DCAD TO PREVENT MILK FEVER

The traditional method of preventing milk fever has been to restrict calcium intake during the dry period. In theory, this helps condition the cow to calcium deficiency and makes her better able to respond to the acute, intense calcium demands which occur when lactation commences. Calcium intake during the dry period is usually restricted by replacing some or all of the alfalfa hay in a dry cow diet with grass hay and using additional corn silage and concentrates. This feeding practice does help to reduce the incidence of milk fever, but it has several drawbacks.

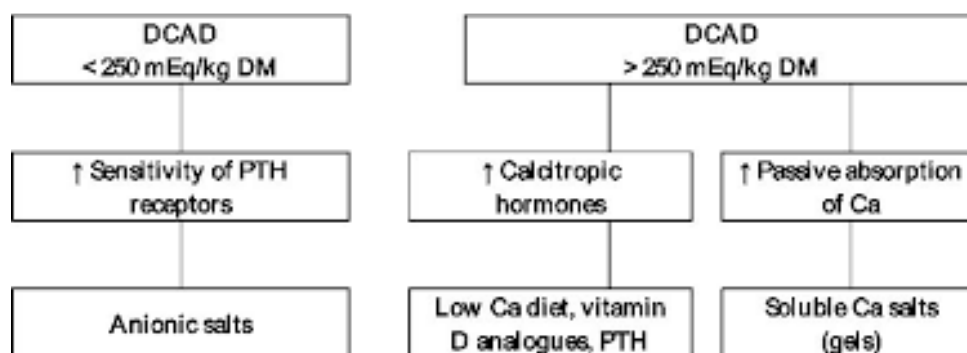
Feeding concentrates and/or corn silage to dry cows may be expensive and may predispose cows to over-conditioning and subsequent fatty liver syndrome, ketosis, and/or abomasal displacements because of their high energy density. Diets consisting of grass hay and corn silage alone have been advocated; however, such diets usually contain sub-optimal levels of protein. Avoidance of alfalfa in the prepartum diet because it is too high in calcium is unfortunate because alfalfa is usually the least expensive and most readily available source of forage protein. Avoidance of alfalfa in the dry period also forces a detrimental forage switch at the time of calving, since lactating rations are usually based on alfalfa forage.

DCAD is more important in controlling milk fever than calcium intake. Dietary calcium does somewhat influence the incidence of milk fever; however, it does so in a non-linear fashion. Both high and low dietary calcium were associated with slightly lower incidence rates of milk fever. High concentrations of dietary potassium (a strong cation and dietary alkalize) caused milk fever but differing levels of dietary calcium had no effect on milk fever incidence.

Dairy cattle diets with a high DCAD (alkaline diets) tend to cause milk fever, while a low or negative DCAD (acidic diets) tends to prevent milk fever. Acidic diets promote bone mobilization (osteocytic resorption) since bone (along with the kidney) acts as a buffer against excessive systemic acidity. Acidic diets have minimal effect on intestinal absorption of calcium. Additionally, low DCAD diets have been shown to increase the amount of 1,25 dihydroxyvitamin D₃ produced per unit increase in parathyroid hormone. This increases osteoclastic bone resorption, which is probably the most important calciotropic effect of acidic diets. Most typical diets fed to dry cows will have an DCAD [using the formula $(Na + K) - (Cl + S)$] of about +100 to +250 meq/kg dry matter. Addition of a cationic salt (such as sodium bicarbonate) to the dry cow diet increases DCAD and increases the incidence of milk fever. Adding anionic salt(s) (minerals high in Cl and S relative to Na and K) to lowers DCAD and reduces the incidence of milk fever

Anionic salt

Concept of DCAD



It is known that force-feeding large amounts of anionic salts or mineral acids can be detrimental. However, lack of palatability limits the likelihood of toxicity if the salts are overdosed. When anionic salts are used, a combination of salts is best. This decreases the potential of toxicity from the cation (Mg, NH₄, Al, etc.) that must necessarily accompany each salt. It is possible to exceed NRC maximum tolerable amounts of sulfur (0.40%), magnesium (0.50%) and NPN (0.50%) by feeding large amounts of any single anionic salt.

Anionic salts are not very palatable and are best fed in a total mixed ration (TMR) rather than in a grain or mineral mix alone. Palatability problems are smaller when the salts are added to a TMR. If a TMR is not possible, it is best to hand-mix the anionic salts with wet forage (corn silage or hay silage). If only dry forages or pasture are used, then the salts can be added to a grain mix, but with some difficulty. It appears that the salts must be mixed with more than at least 2 kg of a grain mix, and even then palatability of the grain mix will be impaired. Pelleting a mixture of anionic salts does not appear to increase their palatability, but it may provide advantages in product formulation and in preventing separation of the anionic salts within a concentrate mixture. Pre-mixing loose salts with a carrier that has a strong flavor of its own (eg, dried distillers grains or molasses) may be helpful and is commonly practiced.

Ammonium salts pre-mixed into a concentrate mixture during warm weather may result in release of ammonia gas and feed refusal. In addition, most of the salts are very hygroscopic and attract moisture, which may lead to caking of the product. Thus, anionic feed additives are routinely stored in bags lined with plastic. Among the commonly used anionic salts, $MgSO_4$ is the most palatable and $CaCl_2$ is the least palatable. Sulfates appear to have an advantage in palatability over chlorides; however, the poor acidifying potential of sulfates greatly limits their use. Care must also be taken to avoid exceeding maximum tolerable levels of sulfur in the total diet.

Monitoring urinary pH

DCAD	Urine pH	Acid–base status	Ca status
If more positive	8.0-7.0	Metabolic alkalosis	Low blood Ca
If more negative	6.5-5.5	Metabolic acidosis	Normal blood Ca

Monitoring urinary pH after feeding an acidifying diet may be the most direct and useful approach to establishing the optimal DCAD. Mean urinary pH values in a group of pre-fresh dry cows should be between about 5.5 and 6.5 if anions are correctly dosed and the diet is properly formulated and delivered. There may be significant post-feeding variations in urinary pH; therefore, urine samples should be collected at a consistent time relative to feeding (usually 2 to 4 hours post-feeding).

The beneficial effects of acidifying diets can only be realized when feeding management of the pre-fresh cow is excellent. High quality feed ingredients, proper diet formulation, adequate bunk space, accurate feed mixing, and accurate feed delivery are especially important when acidifying diets are fed. Otherwise, dry matter intake depression may occur and go undetected in the pre-fresh dry cows. This can lead to negative energy balance prior to calving, fatty infiltration of the liver, and significant ketosis problems after calving. It is not acceptable to obtain milk fever prevention at the expense of fatty liver syndrome. Both diseases can be prevented with excellent pre-fresh dry cow nutritional management. Whenever a herd (or individual cow who can be fed separately) has a history of milk fever problems, then use of acidifying diets should be considered. In herds without clinical milk fever problems, acidifying diets may still prevent subclinical hypocalcaemia and thus reduce the incidence of displaced abomasum, improve milk production.

ECONOMICS

Estimated economic losses due to treatment of animals affected by milk fever. (Rs./animal) (Thirunavukkarasu *et al.*, 2010)

Treatment	Cattle	Buffalo
Cost of medicine	304	273
Veterinarian's fee	206	140
Feed supplements	108	75
Total	618	488

Total Economic Losses due to Milk Fever

The average loss per animal due to the treatment of milk fever was higher at Rs. 618/- for a cow than for a buffalo, Rs 488, the average loss being Rs 608. The average loss due to reduction in milk yield per affected animal was also higher for a cow (Rs. 346/-) than a buffalo (Rs. 177/-). The average loss due to mortality and culling was Rs 105 per affected cow and there was no loss due to buffaloes. Thus, the total loss was of Rs. 1,069/- per cow and Rs 666 per buffalo affected with milk fever.

More than half (57.80%) of the total loss was on treatment of the affected cows and around 3/4th (73.34%) of the total loss was on treatment of buffaloes. It needs to be emphasized that treating milk fever affected animals was the first priority of the farmers, since the delay in treatment could further reduce milk yield or even lead to loss of animal. The loss due to milk

yield contributed 32.37 per cent and 26.66 per cent to the total loss in cows and buffaloes, respectively.

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