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An Update on Milk Fever and Its Economic Consequences

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A B S T R A C T

Nutritional deficiencies, imbalances or erratic management of feeding programs for dairy cows can create various types of health problems generally referred as metabolic diseases. High producing lactating cows are the most susceptible to metabolic diseases during the periparturient period. During this period the animal is tremendously challenged to maintain calcium homeostasis because of physiological and pathological factors. Milk fever is an important metabolic disorder of dairy cattle around the time of calving. Several factors have been consistently associated with increased incidence of milk fever, including parturition and initiation of lactation, advancing age, breed, and diet. Clinical symptoms of this disease include in appetence, tetany, inhibition of urination and defecation, lateral recumbency, and eventual coma and death if left untreated. Parturient paresis is treated intravenously with calcium borogluconate. Of the various methods used in attempts to control the disease, the most progress has been made in dietary management. Until recently, most attention has focused on manipulating the levels of dietary calcium to control milk fever. Furthermore, as the dairy profitability is determined by the biological cycles of milk production and reproduction emphasis should be given on the nutritional management of dairy cows in the dry period as well as at parturition.

Introduction

Periparturient hypocalcemia is a common metabolic disorder in dairy cows that leads to an increased risk of detrimental health and production outcomes and in severe cases can be life threatening.

Physiologically, serum calcium concentration in the adult cow is maintained above 2.0 mmol/L (Martin-Tereso and Martens, 2014). Due to the start of colostrum production and consequently increasing calcium demand, the nadir of serum calcium concentration occurs 12 to 24 h after parturition (Kimura *et al.*, 2006; Goff, 2008). For example, a cow producing 10 L of colostrum, it loses about 23 g of Ca in a single milking. This amount is about nine times as much Ca as is present in the entire plasma Ca pool of the cow.

Calcium lost from the plasma pool must be replaced by increasing intestinal Ca absorption, or increasing bone Ca resorption, or both. As a consequence of this sudden Ca requirement, nearly all cows experience some degree of hypocalcemia during the 1st d after calving as the intestine and bone adapt to the Ca demands of lactation. In some cows, the mammary drain of Ca causes extracellular

and plasma Ca concentrations to decline to levels that disrupt neuromuscular function, resulting in the clinical syndrome of milk fever.

Predisposing factors of hypocalcaemia

Breed

From Several study it have been suggest that certain breeds of cattle are more susceptible to hypocalcaemia, particularly Channel Island, Swedish Red and White, and Jersey cattle (Kusumanti *et al.,* 1993). The intestinal receptors for 1, 25 $(OH)_2D_3$ are lower in Jerseys than in aged-matched Holsteins. Lower receptors would result in a loss of target tissue sensitivity to 1,25 $(OH)_2D_3$. At parturition, plasma 1,25 $(OH)_2D_3$ is elevated as the cow becomes hypocalcemic. Normally, the elevated $1,25(OH)_2D_3$ would result in enhanced bone Ca resorption and intestinal Ca absorption. However, with a reduced number of $1,25(OH)_2D_3$ receptors, the activation of genomic events by $1,25(OH)_2D_3$ is less efficient, resulting in increased susceptibility to hypocalcaemia.

Age

The risk of a cow developing milk fever increases with age (NRC, 2001; Rezac, 2010). From the third lactation onwards, dairy cows produce more milk, resulting in a higher calcium demand. In addition to increased milk production, ageing also results in a diminished ability to mobilize calcium from bone stores and a decline in the active transport of calcium in the intestine, as well as impaired production of $1,25(OH)_2D_3$. Increased age also causes a decrease in the number of $1,25(OH)_2D_3$ receptors (Rezac, D.J., 2010). The hypocalcaemia at calving is age related and most marked in cows from third to seventh parturition; it is infrequent at the first parturition.

Body condition score

According to Ostergaard *et al.,* (2003) high BCS enhance the risk of milk fever. Dairy cows that are over conditioned at calving are up to four more likely to develop milk fever.

This is due to dairy cows with higher BCS at calving have a higher calcium output in milk, making them more prone to milk fever and over conditioning results in decreased feed intake during; gestation period, this is caused by reduced appetite in the critical period around calving which predisposes them to the development of hypocalcaemia (Harris *et al.*, 1999).

Dietary factor

Boda and Cole (2003) indicated that diets providing dry cows a high daily intake of calcium are associated with an increased incidence of parturient paresis. At this level the maintenance requirement of calcium can be met predominantly by passive absorption since active absorption of dietary calcium and bone resorption are then suppressed. Cows in this condition are not able to quickly replace plasma calcium lost in milk and become severely hypocalcaemic.

Excessive dietary phosphorus intake (>80 g day) during late gestation can also induce milk fever and the severity of hypocalcaemia by raising blood phosphorus concentrations to the point that phosphorus directly inhibits renal synthesis of 1,25-$(OH)_2D_3$ and thus reduces the intestinal calcium absorption mechanisms (Oetzel *et al.,* 2011).

Pre-partum diets high in cations like sodium and potassium are associated with an increased incidence of milk fever while diets of high in anion, especially chlorides and sulphides are associated with decreased incidence of the disease

Clinical symptom of hypocalcaemia

Hypocalcaemia can be clinical or subclinical based on whether an animal may or may not show clinical signs. Clinical milk fever (hypocalcaemia) is the most severe hypocalcaemia results in a cow that is unable to rise (from lying to stand position) and is the most easily recognized form of hypocalcaemia with blood Ca^{++} concentration less than 5 mg/dl. Subclinical hypocalcaemia results in less severe disturbances in blood Ca and does not have any outward signs of milk fever. During subclinical hypocalcaemia, blood Ca^{++} concentration ranges between 5.5 and 8.0 mg/dl. Clinical milk fever has very serious economic point of view because if not rapidly controlled may lead to loss of the affected animal; on the other hand, subclinical hypocalcaemia assumes a more insidious role leading to loss of production and fertility.

According to Oetzel (2011) the degree of hypocalcaemia and time of occurrence the clinical sign of milk fever in dairy cattle around calving can be divided into three stages;

Stage I

Stage I milk fever is early signs without recumbency. It may go unnoticed because its signs are subtle and transient. Affected cattle may appear excitable, nervous, or weak. Some may shift their weight frequently and shuffle their hind feet.

Stage II (Sternal recumbency)

Cows in Stage II milk fever are down but not flat out on their side. They exhibit moderate to severe depression, partial paralysis and typically lie with their head turned into their flank (Oetzel, *et al.*, 2011). The clinical signs of stage II milk fever can last from 1 to 12 hours. This is frequently seen with lateral kink or S-shape neck curvature in which the cow tends to lie with her head tucked into her flank.

Stage III (Lateral recumbency)

In this stage hypocalcemic cows are flat out on their side, completely paralyzed, typically bloated and are severely depressed (to the point of coma). They will die within a few hours without treatment (Oetzel *et al.*, 2011). Generally this stage is characterized by inability to stand and a progressive loss of consciousness leading to coma. Cows will not survive for more than a few hours without treatment in this stage (Radostits *et al.*, 2007).

Treatment

The treatment should be carried out as quickly as possible. Administration of calcium borogluconate by oral route is the best approach to hypocalcemic cows that are still standing, but the intravenous (IV) calcium administration is not recommended for the treatment of cows that are still standing, since this application if not done correctly can result in dead animal by cardiac complication. The prognosis is excellent if cows are treated early and properly. The prognosis of milk fever depends on the stage of the condition; stage 1 is less severe and the animal is able to stand but staggering. In stage 2, the cow is recumbent on sterna decumbency, while in stage 3, there is progressive muscular paralysis that may lead to coma and death if prolonged (Huntjens *et al.*, 2005).

Prevention and control of hypocalcaemia

Multiple strategies have been utilized to prevent hypocalcemia and mobilize Ca in dairy cows through nutritional management including: feeding anionic salts, low calcium ion diets, low potassium forages and vitamin D supplementation (Amaral-Phillips, D.,

2014). Thilsing-Hansen (2002) indicated as several principles for milk fever control. However, due to a variety of reasons only four of these are widely used on commercial dairy farms today. These are:

Oral drenching around calving with a supplement of easily absorbed calcium.

The feeding of acidifying rations by anionic salt supplementation during the last weeks of pregnancy.

Feeding low calcium rations during the last weeks of pregnancy.

Prepartum administration of vitamin D, vitamin D metabolites and analogues.

Other possible but less specific control measures for the prevention of milk fever include management practices such as: Dietary magnesium level control peripartum, Body condition control, controlling dietary carbohydrate Body condition control, controlling dietary carbohydrate milking and reduced milking in early lactation (Thilsing-Hansen *et al.*, 2002)

Dietary Cation Anion Balance (DCAB) and potassium

One common prevention strategy is supplementing anionic salts to reduce diet cation-anion difference (Overton *et al.*, 2004). The goal of this type of supplementation is to reduce absorbable cations (Na^+ and K^+), while increasing available anions (Cl^- and SO_4^{2-}) in the diet (Goff, 2008). The concept of dietary cation-anion balance (Na^++ K^+) – (Cl and SO_4^{2-}) has focused attention on the level of potassium (K^+) that is contained in the feed of pre-calving dairy cattle. It is now widely accepted that the homeostatic mechanisms that result in milk fever prevention work more efficiently when DCAB is negative. The most common strategy employed to achieve this negative DCAB is the addition of anionic salts to the diet of pre-calving cattle (Goff, 2008). He has stated also that it is very difficult to control hypocalcaemia if total ration of K is >1.8%. Since high potassium diets usually induce milk fever, pre-calving potassium levels should be kept as low as possible. As dry fodder contains more potassium, feeding of dairy animals with higher amount of dry fodder should be discouraged to prevent milk fever. Inclusion of silage and succulent / green fodder as a major portion of the dry cow's diet is essential, as it has lower potassium content (Thirunavukkarasu *et al.*, 2010). A useful method for determining whether an animal is responding to added dietary anions is to monitor urine pH. A urinary pH within the range 5.5 to 6.2 is accepted as an indicator of successful administration of anions (Horst *et al.*, 1997). Several methods for calculating the DCAD of the diet have been utilized, including the following equations:

DCAD (meq) = (Na + K + Ca + Mg) – (Cl + SO 4 + H2PO4 + HPO4)

DCAD (meq) = (Na + K + Ca + Mg) – (Cl + S + P)

DCAD (meq) = (Na + K +.38 Ca +.30 Mg) – (Cl +.60 S +.50 P)

DCAD (meq) = (Na + K) – (Cl + S) DCAD (meq) = (Na + K) – (Cl)

The equation most often used by dairy nutritionists is, however, the one considering (Na +K) – (Cl + S) (Oetzel *et al.*, 1996).

Side effect of use of DCAD

The use of anions to reduce the DCAD is limited by problems with palatability of the anionic salts most commonly used (Oetzel

and Barmore, 1993). It may be added that the rather unnatural acidosis induced by the DCAD principle could possibly also contribute to a reduced feed intake. Several studies have shown a negative effect on the dry matter intake (DMI) when adding anionic salts to the ration (Gaynor *et al.,* 1989; Oetzel and Barmore, 1993; Goff and Horst, 1997; Taurianen *et al.,* 1998; Moore *et al.,* 2000), whereas others found no effect on DMI (Oetzel *et al.,* 1988).

Calcium restriction and milk fever prevention

One of the classical strategies often proposed for milk fever prevention is the restriction of calcium rich feed intake pre-calving. This has the effect of making sure that parathyroid hormone and the active form of vitamin-D3 are in higher concentrations in circulation on the day of parturition when Ca export in colostrums increases suddenly. This strategy does work and recent data where Ca binders were used to block Ca uptake from the gut have shown a reduced milk fever incidence on several farms.

Vitamin D Supplementation for prevention of hypocalcaemia

A practice by some farms is supplementing high amounts of vitamin D to prepartum dry cows either in the feed or parenterally. Supplementation requires that up to 10 million IU of vitamin D must be injected or fed daily for 10-14 days before calving. These vitamin D doses pharmacologically increased intestinal Ca absorption and sometimes prevented milk fever (Goff, 2008).

Economic consequences of milk fever

Economically, milk fever is important diseases that can reduce dairy cow's productive life by 3.4 years as well affect reproductive performance. Mostly in untreated cases of milk fever, 60-70% cows die (McDowel *et al.,* 2002). Economic losses due to clinical cases of milk fever are substantial and include losses from deaths (~8% of affected cows), premature culling (~12% of affected cows), treatment costs and decreased milk production in the subsequent lactation (Khan *et al.,* 2015*).* In addition, each episode of clinical milk fever increases the risk for other parturient diseases such as retained placenta, ketosis, displaced abomasums and environmental mastitis (Oetzel, *et al.,* 2011). More recently it has been reported subclinical hypocalcaemia exacerbate the level of immuno-suppression experienced by peri-parturient dairy cattle (Kimura *et al.,* 2006).

Milk fever, dystocia and uterine prolapse

It has been recognized that subclinical hypocalcaemia reduce the ability of the transition cow to effect smooth and skeletal muscle contraction (Kimura *et al.,* 2006). Loss of uterine muscle tone due to hypocalcemia in cows suffering from hypocalcaemia is a major cause of uterine prolapse.

Cows with milk fever are developing dystocia six times more than that of normal cows. This is because of a reduced ability of smooth and skeletal muscle contraction causes for cow's long period in labour, which predisposes to dystocia (Tadesse *et al.*, 2015).

Milk fever and fertility

From many study it was reported that cows with clinical hypocalcaemia had a greater diameter of the gravid uterine horn and non-gravid uterine horn between 15 and 45 days post-partum (indicative of slower uterine involution) and a significantly reduced likelihood of having a corpus luteum

(indicative of ovulation since parturition) than normal cows. These results in reduced fertility in dairy cows due to its effect on uterine muscle function, slower uterine involution and reduced blood flow to the ovaries.

There are also indirect effects of milk fever on fertility, which is mediated through dystocia, endometritis and ROP (Mulligan *et al.,* 2006).

Milk fever and mastitis

Both milk fever and subclinical hypocalcaemia cause an increase in the normal cortisol response at parturition. Cortisol is believed to be an important component of the suppressed immunity experienced by periparturient dairy cattle. Cows that have suffered from clinical hypocalcaemia are 8 times more likely to develop mastitis than normal cows.

This phenomenon is mainly due to a reduction in smooth muscle function at the teat sphincter and hence an easy routine for infection after milking and an exacerbated suppression of immunity in milk fever cows when compared with normal cows.

Milk fever and GIT function

There is a reduction in the motility of rumen and abomasum in clinically hypocalcaemic cows. This reduction in ruminal and abomasal motility will likely cause a reduction in feed intake (Whiteford *et al.,* 2006). Furthermore, Goff (2003) has indicated that low plasma Ca concentration around calving will result in reduced motility and strength of abomasal contractions and hence abomasal atony and distension of the abomasum. Therefore, milk fever has been implicated as a predisposing factor for many other transition cow disorders.

Retained placenta

Several studies indicated that increased risk for the occurrence of retained placenta following milk fever, with milk fever cows being up to three times more likely to experience retained placenta than normal cows (Houe *et al.,* 2001).The direct effect of milk fever on the occurrence of retained placenta (excluding any interaction for the effect of milk fever on dystocia) has been reported to double the odds of retained placenta occurring (Mulligan *et al.,* 2006). Furthermore there is also a large indirect effect of milk fever on retained placenta, as milk fever is a risk factor for dystocia and dystocia is a risk factor for retained placenta. Melendez *et al.,* (2004) have reported a significantly lower plasma Ca concentration in cows with retained foetal membranes in comparison to cows with normal placental expulsion. The point should also be made that, in this case, the hypocalcaemia experienced by cows with retained foetal membranes was subclinical not clinical. There is, therefore, a clear link between milk fever and the occurrence of retained placenta.

Endometritis

The link between milk fever and periparturient immuno-suppression, provide a strong basis for the suggested association between milk fever and endometritis (Kimura *et al.,* 2006). In support of this, White ford and Sheldon (2005) observed a significantly higher incidence rate of endometritis in UK cows that suffered clinical hypocalcaemia in comparison to normocalcaemic cows.

Metabolic diseases are of great economic impact; it usually affects the animals about to reach their maximum potential production. Dietary deficiencies as a result of poor ration formulation is the most probable cause of metabolic disorder. Milk fever is a common

metabolic disturbance in dairy cows resulting from hypocalcaemia that occurs in older, third to sixth lactation, high producing dairy cows that are near calving or have recently calved. It is mainly characterized by progressive muscle weakness and depression that progresses into coma if not treated promptly. Hypocalcaemic cows will begin trembling and will no longer be able to stand. Subsequently the cow becomes recumbent, first in the sternal position and then laterally. Parturient paresis is favorable to early treatment with intravenous calcium supplementation. Economically, it reduces milk yield and fertility. Hypocalcaemic cow are more sensitive to various reproductive disorder like ROP, endometritis and mastitis. Dietary calcium levels must be low in the weeks leading up to calving. Management practices like body condition score management and shortening the dry period are also critical for the prevention of the disease.

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