



Milk Fever in Dairy Cows: Causes, Pathophysiology, Prevention Strategies and Treatment

Mohanapriya Thangarajan¹, Ashvini Bansod², Biswa Ranjan Jena³, Abhilash Jadhao¹

¹Department of Veterinary Pathology

²Animal Nutrition Division, IVRI, Bareilly (UP)

³Department of Veterinary Medicine

College of Veterinary Science, Guru Angad Dev Veterinary and Animal Sciences, Ludhiana, Punjab- 141 002

<https://doi.org/10.5281/zenodo.7300224>

Introduction

The welfare and economic impact of milk fever in dairy cows is significant. Milk fever is a metabolic disease of adult females, occurring most commonly at the time of parturition and characterized by hypocalcemia, severe muscular weakness, sternal and lateral recumbency, circulatory collapse and depression of consciousness (DeGaris and Lean, 2008). Milk fever is a sporadic disease. In cattle, it occurs at three main stages in the lactation cycle - a) **Prepartum stage**: About 3% cases occur few days before calving, b) **At calving**: About 6% cases occur just few hours before or at the time of parturition, c) **Post-partum stage**: About 91% cases occur in this stage. Distribution of these 91% cases is as follows - i) 75% occur within 24 hours after calving. ii) 12% cases occur between 24-48 hours after calving. iii) 4% cases occur 48 hours after calving (Chakrabarti, 2012).

The cows and buffaloes are mostly affected and sometimes ewes, does, sows, mare and bitches are also affected. It is most common in mature dairy cows and buffaloes about 5-10 years of age. First calvers rarely develop milk fever because they are able to adapt rapidly to high demands of Ca for lactation. Older animals and high yielding animals are mostly affected because of excess drain of Ca through milk (1.2 g / kg) or colostrum (2.3 g / kg). It occurs in cows in their 3rd to 7th calving because they reach to peak of their milk yield in this stage. The disease mostly occurs in the calving season i.e. winter season (September to December in India). 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 (Thilising-Hansen *et al.*, 2002).

Causes of Milk fever (DeGaris and Lean, 2008)

The principal cause of milk fever is hypocalcemia, and which is due to -

- Excessive drain / loss of Ca through colostrum and milk



- Decreased absorption of Ca from the intestine at the time of parturition

The decreased absorption of Ca is due to -

- i) Starvation / reduced feed intake at parturition or during pregnancy.
 - ii) Digestive disturbances such as ruminal atony / indigestion
 - iii) Deficiency of Vitamin D.
 - iv) Improper calcium – phosphorous ratio in diet.
 - v) Alkaline diet (legume diet).
 - vi) Excess of oxalate and phytates in diet
- Slow mobilization of Ca from bone / skeleton:
 - i) Parathormone deficiency
 - ii) High calcitonin in blood
 - iii) Vitamin D3 deficiency
 - Dietary risk factors:
 - i. **Dietary Ca:** Excessive Ca intake (>100gm/day) during dry period is associated with increased incidence of milk fever. Feeding low Ca diet prevents milk fever by activating Ca transport mechanism in the intestine and bone prior to parturition.
 - ii. **Dietary P:** High levels of dietary P (>80 gm / day) also increases incidence of milk fever as it inhibits renal enzymes that catalyzes production of 1, 25 (OH)₂ D.
 - iii. **Dietary anion – cation balance:** Prepartum diet high in cations such as Na & K is associated with an increase in incidence of milk fever while diet high in anions viz. chlorides and Sulphur is associated with decrease in the incidence of milk fever. High anion diet increases the plasma levels of 1, 25 (OH)₂ D prior to parturition. Most forages viz. legumes and grasses are high in potassium and are alkaline.

Other causes

- Long distance transportation, forced exercise and excitement.
- Sudden deprivation of food / starvation for 48 hours
- Grazing on oxalate rich plants or green cereal crops
- Digestive disturbance / anorexia
- Oestrus – increased susceptibility at oestrus may be due to a) Depression of appetite and b) Depression of ionization of Ca

Clinical signs: The clinical signs develop into three stages.

- I) **First stage or stage of excitement:** This stage is very brief and may go unnoticed.
 - ✓ Animal is still standing



- ✓ Tetany
- ✓ Hypersensitiveness
- ✓ Muscle tremors of hind limbs
- ✓ Ataxia
- ✓ Protrusion of tongue
- ✓ Grinding of teeth
- ✓ Rectal temperature – Normal to slightly above normal

(II) Second stage or stage of sternal recumbency:

- ✓ Animal is unable to stand and rests on sternum with head turned towards shoulder or flank
- ✓ Drowsiness
- ✓ Cold skin and extremities, dry muzzle
- ✓ Dilatation of pupil with absence of papillary light reflex
- ✓ Relaxation of anus with loss of anal reflex
- ✓ Tachycardia (80 bpm) with muffled heart sounds
- ✓ Subnormal body temperature (97 – 101⁰ F)
- ✓ Suspended rumination, defaecation and urination
- ✓ Low venous pressure
- ✓ Secondary bloat / atony of rumen / constipation.

(III) Third stage or stage of lateral recumbency:

- ✓ Animal is unable to stand or sit and remains in lying down condition
- ✓ Marked subnormal body temperature
- ✓ Extremely cold skin and body extremities
- ✓ Severe tachycardia (120 bpm) with inaudible heart sounds
- ✓ Bloat
- ✓ Difficult to raise the vein (venous pressure is very low)
- ✓ Pulse is impalpable
- ✓ Coma and death

Treatment (Thilsing-Hansen *et al.*, 2002)

1) **Calcium therapy:** Parenteral injection of calcium salt is the standard practice. The treatment is very effective. Calcium borogluconate is the preparation of choice. Calcium gluconate is generally used because it causes less tissue irritation. Addition of boric acid increases the solubility of Ca gluconate. Dose and route: Calcium borogluconate 25% @ 400-800 ml or 1 ml / kg body weight I/V or 1 gm / 45 kg body weight I/V. The various workers have found that an ARD system of dose fixation is convenient.



The word A denotes Appetite R denotes Ruminal motility D denotes defaecation.

Prevention Measures

- ✓ Remove mineral supplements, feed low quality hay (no alfalfa as it is high in Ca) beginning about two weeks prior to parturition.
- ✓ This allows the animal's endocrine system to become more active in mobilizing Ca from bone to blood and increases intestinal absorption of Ca.
- ✓ Feed rations with a high P to Ca ratio during late pregnancy.

Conclusion

The milk fever is not only economically important, but also it causes loss of animals as it occurs at the most productive period of a lactating cow. Economic loss due to milk fever happens because of reduction in quantity of milk as well as expenditure on treatment of disease-affected animal. As milk fever is a metabolic disease directly related with feeding management, adopting appropriate feeding strategy during the pregnancy period and immediately after calving can prevent the occurrence of milk fever. Feeding of proper balanced ration considering the factors predisposing milk fever will help the farmers to avoid this menace successfully.

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