Subclinical Hypocalcemia or Milk Fever in Dairy Cows - Why all the Fuss?

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With the initiation of lactation and continued milk production, tremendous adaptations occur in the dairy cow because of the increased need for nutrients to support milk synthesis. Aside from the increased energy and amino acid requirements for colostrum and, later, milk production, the calcium need increases two to three times that of a dairy cow before calving. A dairy cow deposits 8 to 10 g/d of calcium into her foetus just before calving, but when she gives birth, she secretes 20 to 30 g/d in colostrum and milk. As a result, metabolic changes are required to meet the increased calcium requirement. If they don't happen soon enough or in large enough amounts, the blood calcium concentration decreases below a key threshold, resulting in clinical and subclinical hypocalcemia, or milk fever.

Role of Calcium

Calcium is required for skeletal tissue, smooth muscle and neuron function, as well as gastrointestinal motility and muscle strength. Within 12 to 24 hours following calving, the lowest concentration of blood calcium occurs, and in healthy cows, it rebounds to normal within 2 to 3 days. Clinical hypocalcemia is the most well-known condition among dairy producers, with a 5-percentage-point incidence rate. Cattle from Jersey and Guernsey are particularly sensitive to the disease. Jersey cattle have less vitamin D receptors than Holstein cattle, which is one reason for this. With increased milk supply and subsequent lactation, the incidence rises. Because they produce less colostrum and milk and can mobilise calcium from bone in their expanding skeleton, first-calf heifers seldom develop clinical hypocalcemia. Clinical hypocalcemia was reported to be present in 1% of first-lactation Holstein cows, 4% of second-lactation Holstein cows, 7% of third-lactation Holstein cows, and 10% of fourth-lactation Holstein cows in a recent study of 1,462 cows.

The absorption of dietary calcium and the release or uptake of calcium from bone are closely regulated, resulting in a high concentration of calcium in the blood. These processes are regulated by two hormones: parathyroid hormone (also known as PTH) and 1,25-dihydroxy vitamin D₃. PTH is released in response to a drop in calcium content in the blood, and it acts on the kidneys to reduce calcium excretion in the urine. Only little changes in blood calcium content are possible as a result of this alteration. When more calcium is required, such as during the onset and maintenance of lactation, PTH acts on the bone, causing calcium to be reabsorbed and released into the bloodstream. PTH also affects the kidney, causing a vitamin D metabolite to be converted into 1,25-dihydroxy vitamin D₃. The active transport of calcium from the small intestine can then be regulated by 1,25-dihydroxy vitamin D₃. Appropriate magnesium and a slightly less alkaline blood pH (known as metabolic acidosis) are required for PTH to be secreted and effectively bind to its receptor, illustrating the need to provide adequate magnesium in pre-fresh diets and balance these diets to provide a negative cation-to-anion difference (DCAD) in order to prevent hypocalcemia.
Hypocalcemia

Subclinical hypocalcemia affects dairy cows who do not display clinical symptoms yet have a low calcium blood content within 24 hours of calving. As a result, the only method to tell if dairy cows are suffering from subclinical hypocalcemia is to test their blood for calcium levels within the first 1 to 2 days following calving. Excitability, anxiety, weight shifting, and shuffling of the hind feet are common early clinical signs (stage 1: the cow is still able to stand). Subclinical hypocalcemia does not cause these symptoms.

Subclinically hypocalcemic dairy cows have blood calcium readings of 8.0 mg/dl (2.0 mmol/l) or less but show no clinical indications. In a study of 1,462 dairy cows, Reinhardt and colleagues discovered that 50% of mature dairy cows and 25% of first-calf heifers had subclinical hypocalcemia at this cut-off point. According to Oetzel of the University of Wisconsin, the economic cost of subclinical hypocalcemia in a dairy herd is four times that of clinical instances, resulting in a significant impact on dairy profitability. The higher economic cost is due to the higher number of cows with subclinical hypocalcemia compared to clinical hypocalcemia, despite the fact that a subclinical case costs 40% less than a clinical case.

Martinez and colleagues at the University of Florida recently proposed raising the cut-off to 8.5 mg/dl (2.1 mmol/l) since cows with concentrations below this level were more likely to suffer metritis or metabolic problems. Reinhardt and coworkers found that over 65 percent of mature cows and 51 percent of first-calf heifers fell below this requirement when using this higher standard.

Impact of Hypocalcemia on Performance of Animal

Hypocalcemia has an influence on the health of fresh cows, future milk output, and reproductive success. Low blood calcium concentrations in dairy cows have also been demonstrated to affect immunological function in studies. Cows with decreased blood calcium levels the day after calving are more likely to have a displaced abomasum, ketosis (including fatty liver), retained placenta and metritis, and mastitis. After calving, several studies have found a reduction in feed intake and rumination, as well as greater non-esterified fatty acid (NEFA) concentrations.

Hypocalcemia is more common in cows with a high body condition at calving. Other research hasn't shown a negative impact on feed intake or milk output. Cows with subclinical hypocalcemia stood 2.6 hours longer in the 24-hour period before calving and produced 5.6 kg more milk throughout weeks 2, 3, and 4 of lactation, according to Jawor and colleagues at the University of British Columbia. Regardless of their blood calcium content, all cows in the third or larger lactation received prophylactic calcium treatment after calving in this trial.

How to prevent it:

Hypocalcemia is usually prevented by making changes to the pre-fresh or close-up diet. These alterations allow the calcium mobilising physiological system to be primed and ready for the increased calcium requirement associated with the synthesis of colostrum and milk.

- Low calcium diets are difficult to apply on the farm, despite the fact that they lower the prevalence of hypocalcemia. Diets must supply fewer than 20 g of accessible calcium to be effective. These diets frequently contain low-quality forages, which may reduce consumption, but a limited intake of pre-fresh forages is not desirable. Low calcium diets may be possible in some grazing circumstances (e.g., depending on forage species and pasture productivity).
Low potassium forages (e.g., corn silage) may reduce the chance of clinical hypocalcemia in pre-fresh dairy cows, but not the frequency of subclinical hypocalcemia. When low potassium forages are fed without further dietary alterations of chlorine and sulphur, changes in the dietary cation-anion difference (DCAD) may not be big enough to generate metabolic acidosis and avoid a subclinical decline in blood calcium concentration. The DCAD affects the cow’s capacity to reabsorb calcium from bone and absorb dietary calcium from the small intestine, as well as the pH of the blood and tissue response to PTH.

Clinical (five-fold decrease) and subclinical hypocalcemia can be prevented by feeding a negative DCAD diet 21 days before fresh. Diets should be designed with the most palatable anionic mineral supplements to achieve a dietary DCAD of -10 to -15 mEq/100g dietary dry matter. These meals can be supplemented with a variety of commercially available anionic mineral or protein-based supplements. The quantity of potassium and sodium given by forages and other feedstuffs should be maintained as low as feasible when constructing diets. To avoid hypocalcemia, close-up meals should contain roughly 1.0 percent calcium and 0.35 percent magnesium. Excess phosphorus (0.4 percent total diet) raises the risk of hypocalcemia, hence close-up meals should have a phosphorus concentration of 0.25 percent to 0.3 percent.

The pH of one's urine should be utilised to determine whether or not DCAD treatment is working. Urine pH, on the other hand, does not suggest a lower risk of hypocalcemia. After cows have been fed the anionic salt diet for at least 48 hours, urine should be collected midstream. There should be no faeces in the urine. Urine pH should be between 6.2 and 6.8 (at least less than 7.0) for Holstein cows and between 5.8 and 6.3 for Jersey cows. Excess anions are being given (from both feed and water sources) if the average urine pH is between 5.0 and 5.5, and the diet has to be adjusted to avoid a decline in dry matter intake.

Anionic salts are often offered for 21 days before to calving and should not be provided for the full dry season. Feeding and maintaining dry cows in two different groups may not be possible in herds maintained for short dry periods (45 days dry), and feeding anionic salts throughout the full dry season may be necessary to accommodate available facilities and personnel. Anionic salts were fed 0 (control no anionic salts pre-fresh), 21, or 42 days before calving in a recent study by Weich and colleagues at the University of Minnesota. When anionic salts were supplied for 21 or 42 days before the projected calving date, no variations in dry matter intake or milk production were observed.

After calving, calcium supplementation given orally (not as part of the food) has been demonstrated to avoid a decline in blood calcium concentration. Many oral supplements are absorbed within 30 minutes of ingestion, resulting in a rise in blood calcium content lasting 4 to 6 hours. Calcium chloride in gel or paste form is commonly used as an oral calcium supplement. Because the calcium chloride in various forms might cause respiratory issues if inhaled, caution should be exercised when using it. More recently, when two doses (one at calving, the second 12 hours later) of a solid bolus comprising calcium chloride and calcium sulphate were administered after calving, it was found to be beneficial in boosting blood calcium concentration. Cows would be less likely to aspirate the product if the boluses were coated. These boluses were examined in conjunction with anionic salts provided pre-calving in another trial, but no changes were seen when compared to administering anionic salts alone.

Take home message

When creating transition cow programmes for maximum post-calving health, reproductive efficiency, and milk production, avoiding hypocalcemia (low blood calcium concentration) around
calving is critical. Calcium is required for the production of colostrum and milk, as well as muscle and nerve function and immunity. Clinical instances of hypocalcemia are simple to identify, and dairy managers should be aware that feeding and management modifications are required to avoid future occurrences. Subclinical hypocalcemia, on the other hand, is difficult to detect and may be a contributing factor in herds with a high occurrence of metabolic diseases. Subclinical hypocalcemia can affect up to 50% of dairy cows, has no symptoms, and can only be recognised when blood samples are taken within the first 1 to 2 days after calving and the blood calcium content is less than 8.5 mEq/dL. The use of anionic salts and other treatment measures may help avoid this metabolic problem, as it is with all metabolic illnesses.