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Journal of Veterinary Medicine and Research

Review Article

Calcium Requirement in Relation to Milk Fever and its Economic Impact in Dairy Cattle

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Abstract

The goal of this research was to compile and consolidate the disparate data on calcium requirements in connection to milk fever and its economic impact in dairy cattle. The most frequent mineral-related metabolic condition affecting dairy cows at parturition is milk fever, which occurs most commonly in adult dairy cows two to three days following parturition .It is caused by a severe lack of metabolizable calcium ions in the circulation (hypocalcaemia), and its primarily affects high-producing and high-performing dairy cows that are nearing their maximal output capability. Milk yield, parity, cow breed, and a lack of awareness of basic dairy management and ration formulation are all variables that contribute to milk fever. It reduces milk yield and fertility, which leads to the culling of high-producing dairy cows from a herd. It also raises the expense of animal treatment and the danger of additional parturient disorders such retained placenta, ketosis, displaced abomasum, and environmental mastitis. Intravenous calcium salts, such as borogluconate, at a rate of 2g/100kg body weight, as well as oral calcium solutions, are two methods for treating milk fever in dairy cows. Cases of milk fever, on the other hand, can be minimized with good management, notably through the use of proper feeds and feeding systems .To summarize, management of milk fever is very important because it should be considered as a gateway disease that greatly reduce the chance for full productivity and reproduction of dairy cows. The key to prevention of milk fever is management of a close-up dry cow or management during late pregnancy .Therefore, milk fever management is economically most important, as a result, raising dairy farmers' awareness of milk fever and optimal ration compositions for their dairy cows is a critical instrument in the fight against milk fever.

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Submitted: 21 September 2021

Accepted: 18 October 2021

Published: 20 October 2021

ISSN: 2379-948X

DOI: 10.47739/veterinarymedicine-1215

Keywords

- Calcium requirement
- Dairy cattle
- Economic impact
- Milk fever

INTRODUCTION

Milk fever is the most prevalent mineral-related metabolic illness affecting dairy cows at parturition. It occurs most commonly in adult dairy cows two to three days after parturition [1] Hypocalcaemia is a metabolic disease that arises soon after or shortly after calving as a result of a low level of calcium (Ca) in the blood. A transient blood calcium deficit (hypocalcaemia) causes milk fever, which usually occurs around the time of calving [2].

Calcium demand is relatively low during the dry period. As a result, intestinal calcium absorption and bone resorption are moderately inactive at this time. Lactation begins with a sudden loss of calcium through milk. Hypocalcaemia develops when the calcium homeostatic mechanism is unable to supply the calcium demand for milk production. Parturient paresis is a type of cow affliction that develops soon after birthing and is marked by hypocalcaemia, motor paralysis, sensory nerve devices, overall muscular weakness, circulatory collapse, and awareness depression [3].

In a small proportion of cows, hypocalcaemia becomes severe and results in paresis, recumbency and occasionally death. Most commonly milk fever affects high producing and well performing dairy cows about to reach their maximum production potential [4].

Milk fever is intermittent in nature, but on certain farms, it can affect as many as 25% to 30% of susceptible cows. Milk fever is more common as the cow gets older and produces more milk [5]. After calving, blood calcium concentrations in around half of dairy cows in their second lactation and higher fall below the threshold for subclinical hypocalcaemia [6].

The disease's tremendous economic effects, milk fever are a serious concern in many countries. The most significant economic impact of milk fever is the decline in milk output, as well as the loss of animals owing to culling and mortality, as well as the cost of animal care [7].

It is critical to have well-organized and compiled information on calcium requirements in relation to milk fever and its economic impact in dairy cattle to solve this problem. As a result, this review was started with the goal of encapsulating and synthesizing the disparate information on calcium requirements in relation to milk fever and its economic impact in dairy cattle, which lays the groundwork for dairy cow improvement.

ECONOMIC IMPACTS OF MILK FEVER

Milk fever can put dairy farmers in a lot of trouble if important management practices are not put in place. It can shorten the productive life of dairy cows by 3.4 years.60-70 percent of cows will die if milk fever is not treated. Clinical cases of milk fever result in significant economic losses, which include losses from deaths (around 8% of affected cows), premature culling (around 12% of affected cows), treatment costs, and costs of additives required for affected cows to regain production, as well as decreased milk production in subsequent lactations [1]. Furthermore, each episode of clinical milk fever raises the chance of other parturient illnesses such retained placenta, ketosis, displaced abomasum, and environmental mastitis (Oetzel, 2011). It has a number of indirect expenses in addition to its direct effect. The immunological suppression experienced by per parturient dairy cattle is exacerbated by milk fever and subclinical hypocalcaemia [8].

Milk fever lowers the efficiency with which inputs are turned into outputs in dairy production, resulting in lower total productivity. The cost of veterinary treatments and the time spent by the herdsman dealing with the affected animals are both directly related to the disease. The increased risk of associated health problems is the indirect cost of milk fever [9].

PHYSIOLOGY OF CA METABOLISM

Pathophysiology

Calcium demand is relatively low during the dry period. As a result, intestinal calcium absorption and bone resorption are moderately inactive at this time. For the formation of milk, parturition is accompanied by a rapid increase in calcium sequestration. Calcium requirements increase by 2 to 5 times during the lactation [10].

Reduced plasma calcium causes nervous system hyper excitability and decreased muscular contractions, resulting in tetany and paresis [11]. The calcium homeostatic mechanism is stimulated by a decrease in blood calcium levels, which improves intestine absorption and bone resorption. A decrease in plasma calcium produces a rise in parathyroid hormone and calcitriol to compensate, however this isn't always the case.

It takes at least a week for parathyroid hormone to mobilize calcium from the bones, and a day or two for calcitriol to boost calcium absorption efficiency. As a result, nearly all animals have hypocalcaemia after parturition, although high yielders are more likely to develop milk fever. The disease's pathophysiology is more closely linked to the activity of parathyroid hormone (PTH), which is responsible for calcium homeostasis regulation).

Muscle contraction is affected by hypocalcaemia in three ways. Calcium, for starters, has a membrane-stabilizing action on peripheral nerves. In the early stages of milk fever, hyperesthesia and mild tetany are common. Second, calcium is needed for acetylcholine release at the neuromuscular junction. The inability to release acetylcholine produces paralysis by preventing nerve impulses from reaching the brain [12].

The contractility of cardiac muscle is reduced, and the stroke volume is reduced, resulting in a drop in arterial blood flow. Hypothermia and consciousness depression ensued as a result of the diminished peripheral perfusion [13].

Hypocalcaemia impairs gastrointestinal (GI) function as well. Serum calcium levels below 5 mg/dLdecrease abomasal motility and rumen function, lowering end levels. Immune suppression is also experienced by per parturient cows. Because intracellular calcium signaling is a vital early aspect in immune cell activation, the higher calcium demand in these cows affects immune cell intracellular calcium storage, resulting in immunological suppression [8].

Control of calcium homeostasis

A complicated homeostatic mechanism involving calcium fluxes between the extracellular fluid (ECF) and the kidney, bone, and gut closely controls the extracellular fluid (or plasma) calcium content. Three primary hormones control these fluxes: parathyroid hormone (PTH), calcitonin, and 1, 25-dihydroxyvitamin D [1,25(OH) 2D3].A number of crucial cellular activities are reliant on the maintenance of the extracellular calcium concentration within a narrow range [14]. In most cases, skeletal mineral accretion equals skeletal mineral resorption, and urinary calcium content approximates net intestine absorption. One gram of elemental calcium per day is provided by a typical western diet. In most cases, only 30% (300 mg) of the dose is absorbed; the rest is excreted. For a healthy adult in proper calcium balance, net calcium absorption is 150mg per day since intestinal calcium output is pretty consistent at 150mg per day. Calcium from the intestine enters the bloodstream and is filtered by the kidneys. Only 150mg per day is expelled in healthy people because the majority of filtered calcium (98%) is reabsorbed in the proximal renal tubules [15]. The skeleton is where the majority of calcium is stored in the body. A healthy adult cow has 11.3 kg of calcium in its skeleton, with 99 percent of it in the form of hydroxyapatite [16].

Pth and pth-related peptide

PTH (parathyroid hormone) is an 84-amino-acid peptide produced by the parathyroid gland's main cells.PTH secretion is largely dependent on the concentration of ionized calcium, and it is a simple negative feedback loop. Although PTH secretion is not completely suppressed, serum PTH concentration drops as serum calcium concentration rises [17]. When total adjusted serum calcium is 2.9mmol/L (11.5 mg/dL) or 2.1mmol/L (8.5 mg/dL), there is a rather small range of regulation of PTH secretion by extracellular calcium, with no further influence. The calcium-sensing receptor that mediates negative feedback in bovine parathyroid cells was recently cloned [18].

PTH stimulates osteoclastic bone resorption and calcium and phosphate release from bone, inhibits phosphate reabsorption from the renal tubules, and stimulates renal synthesis of 1, 25(OH) 2D3, which promotes intestine calcium and phosphate absorption. To induce these physiological responses, the PTH molecule's amino-terminal end interacts to the PTH receptor. The PTH receptor was recently cloned and discovered to be a member of a vast family of receptors with a seven Trans membrane-spanning domain that activates G-proteins [19].

PTH metabolism is complicated, resulting in a variety of

biological and immunologically reactive fragments. In the circulation, the intact and physiologically active peptide has a half-life of 4 minutes [20].

CAUSES OF MILK FEVER AND PATHOPHYSIOLOGY

Cause of milk fever

A severe deficit of metabolizable calcium ion in the blood causes milk fever [2]. Calcium from milk is frequently the primary source of calcium absorption. In a single milking, a cow producing 10 liters of colostrum loses roughly 23 gram of calcium [21].

The ability of cows to maintain calcium homeostasis is tested when lactation begins. Colostrum and milk production increase around parturition, while dry mater intake falls temporarily, resulting in a brief period of hypocalcaemia [6].

Calcium is removed from the cow by endogenous fecal calcium, glomerular filtration clearance, placental calcium transport to the fetus, bone deposition, and mammary gland calcium production [22].Milk fever is likely to occur when around 50% of the circulating blood calcium is lost [23].

During a rapid and severe calcium outflow, the dairy animal's intricate processes for maintaining calcium homeostasis fail, resulting in hypocalcaemia. Milk fever is caused by a delay in the operation of the calcium homeostatic system [24].

In general, three major factors can affect calcium homeostasis: excessive calcium loss in colostrum beyond the capacity of absorption from the intestines and mobilization from the bones to replace it; impairment of calcium absorption from the intestine at parturition; and insufficient mobilization of calcium from skeleton storage., Since the gland is mostly dormant during the dry season, this could be due to parathyroid insufficiency [25]. As a result, parathyroid hormone (PTH) deficiency is caused by the parathyroid gland's inability to operate properly [26].

SYMPTOMS AND DIAGNOSIS

Symptoms of milk fever

Metabolic diseases showing symptoms resembling milk fever include hypoglycaemia, hypomagnesaemia and hypophosphatemia. Toxic conditions that can be confused with milk fever include acute toxic mastitis and acute diffuse peritonitis. Traumatic conditions may cause symptoms resembling milk fever includes maternal obstetrical paralysis and musculoskeletal injury including downer cow's syndrome due to pressure damage to muscles and nerves. Most cases can be differentiated from milk fever, as hypocalcaemia has a rapid response and good recovery to administration of calcium borogluconate [27].

Hypocalcaemia can be classified as clinical or subclinical depending on whether or not an animal exhibits clinical signs. Clinical milk fever is the most severe form of hypocalcaemia, characterized by a cow's inability to rise (from a lying to a standing position) and is the most easily recognized form of hypocalcaemia, with a blood calcium concentration of less than 5 mg/dL [29].

Subclinical hypocalcaemia causes less severe changes in blood calcium levels and has no visible symptoms. Blood calcium concentrations in subclinical hypocalcaemia range from 5.5 to 8.0 mg/dL [29].

Milk fever causes the animal to become ataxic, anxious, and energetic at first. Poor appetite, rumen motility, bloating, low body temperature, slow respiration, impalpable pulse, weak but rapid heartbeats (80-100 per minute) with very difficult to hear due to reduced ability of muscles to contract, dilated pupils, and a dry muzzle are all common symptoms.

The clinical indications of milk fever are split into three stages based on the degree of hypocalcaemia and the time of manifestation. Stage I milk fever is characterized by early symptoms that do not require recumbency. Its symptoms are modest and temporary, therefore it may go unnoticed. Cattle that have been affected may appear agitated, nervous, or feeble. Stage II milk fever causes sternal recumbency in cows. They show signs of depression ranging from mild to severe. The body temperature is low, the muzzle is dry, and the heart beat is fast [9].

Stage III hypocalcaemia cows are fully paralyzed, swollen, in lateral recumbency, and gradually lose consciousness, eventually resulting in coma. There is a significant drop in temperature, an increase in heart rate, and animals will die if not handled [30].

Diagnosis of milk fever

Milk fever is diagnosed using a combination of history collection, clinical examination, and laboratory testing. During the history, all detailed information about the cow should be obtained, including age, breed lactation stage, milk yield, and calving day. Within 72 hours following parturition, milk fever is frequent in mature dairy cows, usually 5 years old. It is more common in high-producing dairy cows [26]. The most accurate way for diagnosing a case of milk fever is a laboratory evaluation of blood calcium levels and a favorable response to intravenous calcium solutions.Ca levels in the blood should be between 8 and 10 mg/dL [7]. Milk fever is diagnosed by a history of recent calving, clinical signs of progressive ataxia, hypersensitivity and excitability to sternal recumbency, depression, dehydration, and anorexia, which can lead to lateral recumbency, loss of consciousness, coma, and even death if left untreated [26, 10]

Cows with serum calcium lower than 7.5 mg/dL are as considered as hypocalcaemia. Animals with serum calcium level of 5.5 to 7.5 mg/dL show sign of stage I hypocalcaemia.

Stage II hypocalcaemia seen with calcium levels of 3.5 to 6.5 mg/dL and stage III seen when calcium concentration falls below 3.0 mg/dL. Blood samples are often taken later if there has been no improvement [31].

Treatment of milk fever

The goal of treatment in milk fever is to restore the serum concentration of calcium sufficiently to support cellular function. Treatment of milk fever should be done as early as possible, especially if recumbency is present, as recumbency can cause severe musculoskeletal damage [24]. It is achieved by intravenous administration of calcium salts such as borogluconate at a rate of 2 g/100 kg body weight; and oral calcium solutions and intravenous (IV) calcium borogluconate [28].Supplementation of calcium borogluconate by oral route is the best approach

to hypocalcaemic cows that are still standing, such as cows in stage I hypocalcaemia or which have undetected subclinical hypocalcaemia [24].

The fastest way to restore normal plasma calcium concentration is to administer an IV injection of calcium salts. For cows in stage II and III of milk fever should be treated immediately with a slow IV administration of 500 ml of 23% calcium borogluconate [32].

Extremely high dose of calcium may cause fatal cardiac complications [33]. The prognosis depends on the stage of the condition; cows in severe stage may present several complications and poor prognosis [34]. Subcutaneous calcium administration can also be used to support blood calcium concentrations around calving Subcutaneous calcium injections are irritating causes tissue necrosis; administration should be limited to no more than 75 ml of a 23% calcium borogluconate.

Prevention of milk fever

Strategically prevention of milk fever is economically important to the dairy farmers because of minimize production loss, death loss and veterinary costs associated with milk fever. In order to understand how to prevent this condition, one must understand why it becomes a problem [9].

Management of milk fever is very important because it should be considered as gateway diseases that greatly reduce the chance for full productivity and reproduction of dairy cows [8]. Specific management program is relevant to control the incidence of milk fever among high risk cows. The key to prevention of milk fever is management of a close-up dry cow or management during late pregnancy; to limit calcium intake during the dry period to adapt to calcium deficiency and able to respond to milk calcium demand in early lactation [5]. nutritional management including feeding anionic salts, low calcium ion diets and vitamin D supplementation [35]. Moreover, prevention of milk fever involves feeding of calcium-deficient diets in the late dry period, feeding of calcium-rich rations 3-4 days before parturition, vitamin D supplementation, reducing the dietary cation anion difference and magnesium supplementation in the late gestation period [25]; and drenching of calcium borogluconate near calving.

CONCLUSION AND RECOMMENDATION

Milk fever is the most prevalent mineral-related metabolic disease that affects dairy cows during parturition and is caused by a severe shortage of metabolizable calcium ions in the blood. It is most typically a metabolic problem in dairy cows caused by hypocalcaemia that arises around calving in adult high-producing dairy cows. It is uncommon before the third parturition and the incidence is highest from the third to sixth parturition. Besides, low understanding of proper dairy management, ration formulation and the occurrence of production diseases accompanied by low access to proper hypocalcaemia treatment contribute to the incidence and economic effect of the disease. Economically, it reduces milk yield and fertility that leads to culling of high producing dairy cows from a herd and cost of treatment of the animals. Therefore, based on the above conclusive statements, the following recommendations are forwarded:

- It may be necessary to create awareness for dairy farmers about milk fever and proper ration formulations for their dairy cows.
- Commercial dairy farmers should have an integrated herd health program to control metabolic diseases through optimal feeding and management regimens.
- Further study should be made on the prevalence of milk fever and its economic impact in the dairy industry.

ACKNOWLEDGEMENT

The authors are extraordinarily obliged to the researchers conducted their research on Calcium Requirement in Relation to Milk Fever and Its Economic Impact in Dairy Cattle, because their findings are valuable sources for this review paper.

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Cite this article

Fekata A (2021) Calcium Requirement in Relation to Milk Fever and its Economic Impact in Dairy Cattle. J Vet Med Res 8(3): 1215.