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Milk Fever: A Comprehensive Overview

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Abstract

Now a day's metabolic disorder is a major cause of concern in high yielding livestock. Out of many metabolic disorders, milk fever is one of the most important disorders which not only reduces the production performance of dairy animals but also create problems for other metabolic disease occurrence and infertility or repeat breeding like reproductive disorders in future. So, proper feeding management is prerequisite during advance pregnancy to reduce risk of milk fever. Metabolic profiling tests, which are using specific parameters known to be responsive to dietary intake, can be used to complement dietary evaluation of current feeding programme adequacy or a response to a feeding programme change. Blood Ca^{+2} and serum Ca^{+2} in all (normal parturient and recumbent) cattle should be within the range of 0.22–1.22 mmol/L and 2.81–10.91 mg/dl, respectively.

Key words: Milk Fever, Hypercalcemia, Parturient Apoplexy, Metabolic Disorder

Introduction

Clinical hypocalcaemia, commonly referred to as milk fever, was first described in 1793, which coincided with the provision of additional nutrition to cows to increase milk production. Milk fever is arguably the classic veterinary disease of dairy cattle, with stories of recumbent cows soon after calving recounted for centuries. The enormous and immediate requirement of calcium (Ca) for colostrum and milk production, paired with a normal decrease in dry matter intake around parturition, creates a large Ca deficit that takes days to rectify. Although a detailed discussion of Ca metabolism is beyond the scope of this article, multiple hormones and minerals are important for appropriate Ca regulation. To maintain Ca concentrations at a level required for life (eg, thermoregulation, muscle contraction) while transferring a large quantity to colostrum and milk, cows increase the secretion of parathyroid hormone which increases release of Ca from bone, decreases Ca excretion by the kidney, and indirectly increases intestinal absorption of Ca via vitamin D-dependent means. As sufficient

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magnesium (Mg) concentrations are required for proper interaction of parathyroid hormone with its receptor, adequate blood Mg concentration is also important for Ca regulation. Appropriate and timely coordination of these Ca regulation mediators is essential, as milk fever results when blood and extracellular Ca concentrations fall faster than homeostatic mechanisms can adapt to the demands of lactation, resulting in insufficient Ca for normal bodily functions and weakness that can progress to recumbency and death (Sabine Mann et al.2019).

Etiology

Dairy cows are at considerable risk for hypocalcaemia at the onset of lactation, when daily calcium excretion suddenly increases from about 10 g to 30 g per day. This stresses calcium homeostasis and may cause blood calcium concentrations to fall well below the normal lower reference range of approximately 8.5 mg/dL. Blood calcium concentrations typically decrease around the onset of parturition but recover quickly. Cows with parturient paresis have a more profound decrease in blood calcium concentration—typically below 5.5 mg/dL.

Clinical signs

The main clinical manifestations are divided into three stages**.**

1. **First stage or Stage of excitement**: Anorexia (decreased appetite), nervousness or hypersensitivity, mixed excitement or tetany without recumbency, weakness or weight shifting, stiffness of hind legs, rapid heart rate, rectal temperature is usually normal or above normal (>102.2 F)
2. **Second stage or Stage of sternal recumbency**: Sternal recumbency comprising down on chest and drowsiness, characteristic “S” shaped posture- sitting with lateral kink in neck or head turned to lateral flank, depression, fine muscle tremors, rapid heart rate with decreased intensity of heart sounds, cold extremities, decreased rectal temperature (97 F to 98F), decreased gastrointestinal activity, pupils dilated and unresponsive to light
3. **Third stage or Stage of lateral recumbency**: Lateral recumbency, comprising of almost comatose condition, progressing to loss of consciousness, severe bloat, flaccid muscles profound gastrointestinal atony, rapid heart rate, impalpable pulse and almost inaudible heart sounds. (Dr. R.C. Ramteke et al. 2023)

Clinical Diagnosis:

A combination of information gathering, clinical examination, and laboratory testing is used to diagnose milk fever. Cow's age, breed, lactation stage, milk yield, calving day, and response to intravenous calcium borogluconate solution were obtained during history tracing. Milk fever is common in mature dairy cows (>5 years old) within 72 hours of parturition. It occurs more frequently in dairy cows that produce much volume of milk (Radostits et al., 2007). Paresis and weakness depression in cows that have recently given birth to young are diagnostic indications of the condition. Whereas laboratory investigation of blood calcium levels and a positive response to intravenous

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calcium solutions is the most accurate technique to diagnose a case of milk fever. The blood calcium level should be between 8 and 10 mg/dL (Thirunavukkarasu et al., 2010). Cows having blood calcium levels less than 7.5 mg/dL are classified as hypocalcaemic. Stage I Hypocalcaemic cows have a blood Ca concentration of < 7.5 mg/dl. Stage II hypocalcemia cows have blood Ca level ranging from 3.5 to 6.5 mg/dl, whereas stage III has concentrations as low as 2 mg/dl. Bradford, (1996) states that prolonged recumbency causes ischemic muscle necrosis as well as an elevation in the serum muscle enzymes CPK and AST. CPK levels typically vary between 105 and 409 IU/L, with values larger than 1000 IU/L indicating serious muscle injury from being low, and AST levels greater than 500 IU/L indicating severe muscular damage. Recently a study group from Germany diagnosed subclinical hypocalcemia by taking the historical clinical impression of "cold ears" and using an infrared thermometer to determine the skin temperature of ears in fresh cows. However, diagnosis of subclinical hypocalcemia by ear temperature is an unreliable method, because the results were greatly affected by the ambient temperature (Nurye, M et al. 2022).

Treatment

1. Oral calcium supplementation for standing cows
2. Intravenous calcium infusion for recumbent cows
3. Prevention of hypocalcemic relapses in all affected cows

Recumbent cows are at extremely high risk for muscle and nerve damage therefore, treatment of parturient paresis must be prompt. Excessive exogenous calcium administration increases the risk for hypocalcemic relapse. The lowest dose of calcium needed to restore normal blood calcium concentration should be used.

Cows with stage 1 parturient paresis (ie, cows that are still standing) should be treated with an oral calcium supplement which is rapidly absorbed into the bloodstream and poses little risk for subsequent hypercalcemia followed by a rebound hypocalcemia. Bolus formulations of oral calcium are the safest means of providing oral calcium supplementation. Oral paste, gel, or liquid formulations of supplemental calcium are not recommended due to unnecessary risk for aspiration and pharyngeal irritation. Oral calcium boluses should include a coating to protect the cow from mucosal damage should the bolus remain in contact with pharyngeal or esophageal mucosa. The preferred approach to oral calcium supplementation is an acidogenic source of calcium (usually calcium chloride or calcium sulfate) in a bolus form. These are highly available and promote the cow's own calcium homeostasis by enhancing parathyroid hormone (PTH) receptor responsiveness. A standard oral dose provides 40–55 g of elemental calcium. Blood calcium increases to peak concentrations within 30 minutes of oral administration and equals about 4 g of IV calcium. Higher doses could cause uncompensated metabolic acidosis, decreased feed intake, and increased risk for hypocalcemic relapses.

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Non-acidogenic calcium sources (usually calcium propionate) can be used for oral calcium supplementation: however, they are not preferred for cows in stage 1 parturient paresis. Oral calcium propionate requires a higher dose of elemental calcium (100 g or more), does not enhance the cow's own calcium homeostatic mechanisms, and unnecessarily increases blood glucose at a time when many cows are hyperglycaemic. Oral calcium propionate is best reserved for cows that are two or more days post-calving.

Cows with stage 2or3 hypercalcemia (ie, cows that are recumbent) require immediate correction of their hypocalcaemia by intravenous calcium infusion. The standard treatment for an adult dairy cow is 500 mL of a 23% calcium gluconate solution. These solutions must contain boric acid to solubilize the calcium gluconate and stabilize the solution; thus, they may be labelled as calcium borogluconate. This standard treatment provides 10.7 g of elemental calcium, which is more than sufficient to restore normocalcemia for even the largest cows with the most profound hypercalcemia.

Many products marketed for treatment of hypocalcemia include phosphorus, magnesium, glucose, or potassium in addition to calcium. No additional electrolytes are needed to treat parturient paresis, and some could be harmful. Calcium gluconate alone is the best choice for intravenous treatment of parturient paresis.

The jugular vein is the preferred site for intravenous calcium infusion. Asepsis at the injection site and accurate placement of the needle within the lumen of the jugular vein is necessary to lower the risk for thrombosis and perivascular leakage.

The cranial superficial epigastric (mammary) vein may be more accessible than the jugular vein in some recumbent cows but it is prone to thrombosis and phlebitis so should be used only when jugular vein is unavailable.

The response to intravenous calcium infusion is usually immediate and rewarding. Response to intravenous treatment is the main means of confirming the diagnosis. It is an excellent practice to routinely collect a pre-treatment blood sample before starting the intravenous calcium infusion. If the cow does not respond favourably, this sample can be submitted for laboratory confirmation of hypocalcemia. Blood samples collected after intravenous calcium infusion cannot be used for diagnostic purposes, because the calcium concentration has been confounded.

A typical response to intravenous calcium infusion includes declining heart rate and increasing pulse intensity as cardiac contractility is restored. Muscular paresis is reversed, resulting in muscle tremoring and attempts by the cow to rise. About 75% of recumbent cows are able to rise within 2 hours. Pre-existing musculoskeletal and nerve damage is the main reason that cows remain recumbent after successful correction of hypocalcemia. Intravenous infusion of calcium transiently raises blood calcium concentrations to nearly twice the normal upper limit. This puts the cow at risk for fatal cardiac arrhythmia. Therefore, calcium-containing solutions should be administered slowly

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(over 10–20 minutes) while cardiac rhythm is monitored by auscultation or carotid pulse. If severe dysrhythmias or bradycardia develop, administration should be stopped until the heart rhythm has returned to normal. Endotoxic animals are especially prone to dysrhythmias due to intravenous calcium treatment.

Transient hypercalcemia due to intravenous calcium infusion places the cow at risk of a hypercalcaemic relapse. Hypercalcemia shuts down the cow's efforts to mobilize its own calcium stores by halting PTH release and triggering calcitonin (CT) secretion instead. About 25% to 40% of recumbent cows that are able to rise after intravenous calcium infusion become recumbent again (usually in 12 to 24 hours) unless measures are taken to decrease the risk for relapse.

The treatment of choice for preventing hypocalcaemia relapses is oral calcium administration, as described above for treatment of stage 1 cases of parturient paresis. Cows should be standing, alert, and able to swallow before administering an oral calcium bolus.

Subcutaneous calcium is a second choice for the prevention of hypercalcaemic relapses. Subcutaneous calcium alone may not be adequately absorbed because of poor peripheral perfusion during the initial hypocalcemia and should not be the sole route of treatment. Strict asepsis is necessary to prevent infection at the site of the subcutaneous injection. Solutions containing formaldehyde or dextrose should not be administered subcutaneously because they are highly irritating. Calcium gluconate (500 mL, 23% solution [the same as used for IV infusion]) is the best choice for SC calcium administration. This solution is still quite irritating and should be divided into multiple sites with smaller volume. (MSD manuals).

Conclusion

In managing milk fever, a combination of prompt diagnosis, appropriate treatment, and preventive measures is essential to ensure the health and well-being of dairy cows during the critical transition period around calving.

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