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Management and Control of Milk Fever in Dairy Cattle in Field Condition

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Abstract

Milk fever is an acute to peracute, afebrile, flaccid paralysis of mature dairy cows that occurs most commonly at or soon after parturition. It is manifested by changes in mentation, generalized paresis, and circulatory collapse. Clinical milk fever is more commonly seen in older animals as they have reduced ability to mobilize calcium from bone. Recommended treatment is IV injection of a calcium gluconate salt. Approximately 75% of cows stand within 2 hr of treatment. Animals not responding by 4-8 hr should be reevaluated. The best way to prevent parturient paresis is by use of the dietary cation-anion difference to decreases the blood pH of cows during the late prepartum and early postpartum period. Administration of vitamin D3 also effectively prevents parturient paresis. The prognosis is generally good. However, some cows can relapse. Without treatment, between 60% and 80% of cows usually die, although death rates as high as 90% have been recorded.

Introduction

Parturient paresis, or milk fever (*Paresis puerperalis*), is a hypocalcemic metabolic disorder that occurs in high producing mature dairy cows (third parity and older), lactating beef cows, sheep, goat, sows and rarely, horses just before, around or after parturition (DeGaris and Lean, 2008). Most vulnerable window in dairy cows is within 48 hours after calving, but also occurs several weeks before or after. This disease is of considerable importance for dairy cow welfare and economy (Goff, 2008). Hypocalcemia is considered as a gateway disease and predisposes the cow to various other metabolic and infectious disorders in early lactation such as metritis and mastitis (Goff, 2008). In many countries prevention of parturient hypocalcaemia is therefore given a high priority (Constable *et al.,* 2017). The incidence of clinical hypocalcaemia in the field generally ranges from 0-10%, but may exceed 25% of cows calving (DeGaris and Lean, 2008). However, incidence of subclinical hypocalcaemia can go up to 50% of multiparous periparturient dairy cows. It has been proposed that a specific control program is relevant when the incidence of milk fever increases to above 10% among high-risk cows, *i.e.*, cows entering third or later lactations (Constable *et al.,* 2017).

Clinical milk fever is more commonly seen in older animals as they have reduced ability to mobilize calcium from bone and in certain breeds such as Jersey, Swedish Red and White and Norwegian Red breeds (because of differences in the number of intestinal vitamin D receptors regulating the intestinal Ca uptake compared to other breeds) (Constable *et al.,* 2017).

Etiology

During the late gestation, dairy cattle have relatively low calcium requirements, approximately 30 g day^{-1}. At parturition, the requirement for calcium is greatly increased due to initiation of lactation, when mammary drainage of calcium may exceed 50 g day^{-1} (DeGaris and Lean, 2008). Most cows adapt within 48 hours after calving by increase in plasma concentrations of parathyroid hormone (PTH) and 1,25-$(OH)_2D$ (biologically active form of vitamin D_3. However, cows unable to develop counter-regulatory mechanisms within that period develop clinical hypocalcemia (Constable *et al.,* 2017).

Clinical Signs

Three stages of milk fever in cattle are commonly recognized.

Stage 1

In the first stage, the cow is still standing, nervousness, excitement, tetany with hypersensitivity, muscle tremor of the head and limbs, disinclined to move/ ataxic gait, protrusion of the tongue, rectal temperature normal/ slightly elevated, cool skin/ extremities, heart and respiration rate may be normal/ slightly elevated.

Stage 2

The second stage is characterized by sternal recumbency with depressed consciousness, head turned into the flank (Figure 1), absence of tetany, cool skin/ extremities, subnormal rectal temperature (97-101 °F), increased heart rate (about 80 bpm) with marked decrease in the intensity of the heart sounds, weak arterial pulse and venous pressure, ruminal stasis and bloat, relaxation of the anus, dry and staring eye, pupillary light reflex (PLR) is incomplete/ absent, pupillary dilatation from normal to maximum (Figure 2).

Figure 1: A Jersey cow in stage 2 periparturient hypocalcemia with head turned towards flank (Sternal recumbency)

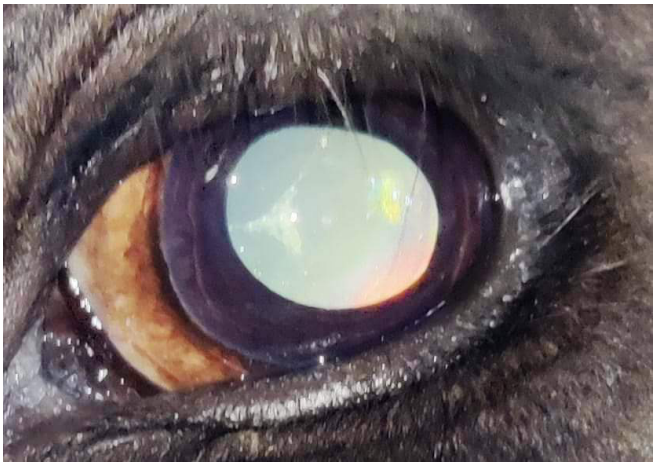

Figure 2: Pupillary dilatation in a Holstein Friesian cow with stage 2 periparturient hypocalcemia

Stage 3

The last stage is characterized by a severe depression or even comatose cow in lateral recumbency, complete flaccidity, can't sit on sternal recumbency on its own, marked subnormal temperature, heart sounds are inaudible, severe rumen bloat, heart rate increased up to 120 bpm, circulatory collapse, coma, and death.

Management

- Treatment with Calcium borogluconate (equivalent to 8-12 g Ca/ cow IV or SC as single dose) during the first stage of the disease, before the cow is recumbent provides uneventful recovery.
- A typical treatment for an adult lactating dairy cow with periparturient hypocalcemia is 500 mL of 23% Ca-borogluconate by slow IV injection with cardiac auscultation which provides 10.7 g of Ca.
- The longer the interval between the time the cow first becomes recumbent and treatment, the greater the incidence of downer-cow syndrome.
- Farmers must be educated to appreciate the importance of early treatment.
- Special dietary care should be given to cows with previous history of hypocalcemia.
- Cows in lateral recumbency (third stage) should be placed in sternal recumbency until treatment is available to facilitate eructation and reduce the risk of aspiration if the cow regurgitates.
- Provide rubber or other mats under the cow to facilitate solid, nonslip footing.
- The cow should be rolled from side to side every few hours and provided with adequate bedding.
- In extreme climatic conditions, erection of a shelter over the

cow is advisable if she cannot be moved to permanent shelter.

• If a cow is recumbent for more than 48 hours, occasional assisted lifting using appropriate cow lifters should be considered.

• Favourable responses to IV Ca is observed in the form of belching, muscle tremor, improvement in the amplitude and pressures of the pulse, decrease heart rate, increase intensity of the heart sounds, sweating of the muzzle and defecation *etc*.

• The best procedure to follow if response does not occur is to revisit the animal at 6-12 hour intervals and check the diagnosis.

• If no other cause of the recumbency can be determined, the initial treatment can be repeated on a maximum of 3 occasions.

Control

There are 4 widely used control strategies on commercial dairy farms today (Constable *et al.,* 2017). These are:

• Feeding low calcium (< 20 g cow day^{-1}), phosphorous (< 40 g cow day^{-1}), and potassium (< 2% in feed DM) rations during the last 2-3 weeks of pregnancy.

• Oral drenching around calving with calcium chloride (equivalent to 50 g Ca cow^{-1}) orally twice a day up to 48 hours post-calving.

• The feeding of acidifying rations by anionic salt supplementation (magnesium sulfate/ ammonium chloride) or commercially available formulation such as METABOLITE® MIX POWDER (Virbac) @ 100 g head^{-1}day^{-1} during the last weeks of pregnancy.

• Prepartum administration of vitamin D_3 @ 10 million IU/ cow IM as single dose 3-7 days before expected calving.

Less commonly followed control measures for the prevention of milk fever include:

• Adequate dietary Mg supplementation in late gestation (~ 0.4% of feed DM).

• Controlling of the body condition.

• Controlling dietary carbohydrate intake peripartum.

• Shortening of the dry period.

• Prepartum milking.

• Partial milking during the first days of lactation (allow calf to suckle).

• Soya bean oil during the last 2-3 weeks of pregnancy (helps Ca absorption).

Conclusion

Even when applied in the most optimal way the preventive effect of the described control principles rarely reaches 100 percent. Combining one or more of the principles is one potential way of improving efficacy.

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