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Dr. V Arul
M.V.Sc Veterinary Assistant Surgeon, Veterinary Dispensary, Vanapuram, Tamil Nadu, India

Dr. C Inbaraj
M.V.Sc Veterinary Assistant Surgeon, Veterinary Dispensary, Mekkalur, Tamil Nadu, India

V Brindha
MSc. MPhil. M. Ed, Assistant Professor, Department of Physical Science AKT Educational College Kallakurichi, Tamil Nadu, India

Corresponding Author:
Dr. V Arul
M.V.Sc Veterinary Assistant Surgeon, Veterinary Dispensary, Vanapuram, Tamil Nadu, India

Successful management of downer cow syndrome in a cow: A case report

Dr. V Arul, Dr. C Inbaraj and V Brindha

Abstract
A bovine 8 years old crossbred jersey cow was presented with sternal recumbency with the history of calved yesterday drenched in rain feeding with a high concentrated diet & low bran diet. Physical examination was done. Complete blood profile, serum biochemistry and rumen fluid examination was done. Based on history and diagnosis the animal was treated with fluids antibiotic, and antacids.

Keywords: Downer cow, hypo calcemia, acidosis

Introduction
Milk fever (MF) is one of the most common mineral-related metabolic conditions affecting dairy cows at parturition, a disorder that occurs immediately after or close to calving as a result of a low level of calcium in the blood (hypocalcaemia). A mild degree of hypocalcaemia develops in the majority of cows during the peripartum period and has been linked to calving problems, retained placenta, uterine prolapse, metritis, mastitis, ruminal stasis, depression of the immune system and generally reduced reproductive performance (Wubishet *et al.*, 2016) [1]. Total blood calcium (Ca) concentration in the adult cow is maintained between 8.5 and 10.0 mg/dL (2.1 and 2.5 mmol/L). Nearly all cows will experience some degree of hypocalcemia at the onset of lactation; however, the severity and duration of the hypocalcemia experienced depends on the integrity of the cow's Ca homeostasis mechanisms (Jesse P, 2014) [2].

History and clinical findings
The animal was presented with (day 1 & 2) Sternal recumbency, Regurgitation of food material through nostril, Lateral kink of the neck physical examination revealed rectal temperature 104.2^0 F, congested mucous membrane, dry muzzle, heart rate - 100 bpm and high intensity of heart sound were observed. Rumen -bloated, hypomotility, dilated pupil, respiratory distress and diarrhea were also observed. Day 3 - Lateral recumbency Temp: 37.7, H/R; 70/min, Bloated abdomen AND Diarrhea. Rumen fluid: offensive odour milky white in colour, pH: 5, Live small sized protozoa and Large number of dead protozoa were noticed. Haematology: Increased PCV, Neutrophilia. Serum Bio chemistry: Glucose: 92 mg/dl, Calcium: 4.35 mmol/dl, Phosphorous: 7 mmol/dl. Day 4 - Sternal recumbency Heart rate: 85/min, Bruxism noticed, Scanty dung voided and rectal examination: scanty mucus coated dung. Day 5 - Animal was taking water and hay Concentrate about 1 kg intake, Lateral kink of the neck, D/U: voided normally, animal trying to get up, Standing while giving support, H/R:73/min and R/R:24/min.

Result and Discussion
Day 1 & 2 - Treated with Inj. melonex 15ml i/m, Inj. OTC 60ml i/v, Inj. NS 500ml i/v, Inj. CPM 15ml i/m, Inj. Neurovet 10 ml i/m and Bol. Comin forte-y 4-0-4 PO. Day 3 - Treated with Inj. DNS 500ml 2 SACHT i/v, Inj. Sodium bicarbonate, after 6 hours Inj. Cal. Boro gluconate (25%) 5g i/v were given to avoid chelation, Inj. CPM 10 ml i/m, Inj. Neurovet 10ml i/m and Bol. OTC 1g 4-0-4 PO. Day 4 - The animal was treated with Inj. DNS 500ml 2 sachet i/v, Inj. Cal. Boro gluconate (25%) 5g i/v, Inj. CPM 10 ml i/m, Inj. Neurovet 10ml i/m and Bol. OTC 1g 4-0-4 PO. Day 5 - the animal was treated with Inj.DNS 500 ml i/v, Inj. OTC 60 ml i/v, Inj. CPM 15 ml i/v, Inj. Neurobion forte 20 ml i/v, Advised to owner put the animal in sling and after 5^{th} day animal was standing and walking normally.

Three main preventive principles against milk fever were evaluated in this literature review, and the efficacy of each principle was estimated from the results of controlled investigations. Oral calcium drenching around calving apparently has a mean efficacy of 50%–60% in terms of milk fever prevention as well as prevention of milk fever relapse after intravenous treatment with calcium solutions. However, some drenches have been shown to cause lesions in the fore stomach. When using the DCAD (dietary cation -anion difference) principle, feeding rations with a negative DCAD (measured as (Na + K) – (Cl + S)) significantly reduce the milk fever incidence. Calculating the relative risk (RR) of developing milk fever from controlled experiments results in a mean RR between 0.19 and 0.35 when rations with a negative versus positive DCAD are compared. The main drawback from the DCAD principle is a palatability problem. The principle of feeding rations low in calcium is highly efficient in milk fever prevention provided the calcium intake in the dry period is kept below 20 g per day. Calculating the relative risk (RR) of developing milk fever from controlled experiments results in a very low mean RR (between 0 and 0.20) (daily calcium intake below versus above 20 g/d). The main problem in implementing the low-Ca principle is difficulties in formulating rations sufficiently low in calcium when using commonly available feeds. The use of large doses of vitamin D metabolites and analogues for milk fever prevention is controversial. Due to toxicity problems and an almost total lack of recent studies on the subject this principle is not described in detail. A few management related issues were discussed briefly, and the following conclusions were made: It is important to supply the periparturient cow with sufficient magnesium to fulfil its needs, and to prevent the dry cows from being too fat. Available information on the influence of carbohydrate intake, and on the effect of the length of the dry period and prepartum milking, is at present insufficient to include these factors in control programmes (Thilsing *et al.*, 2002) [3].

Another major risk factor for milk fever is hypomagnesemia, which is observed when animals are fed inadequate amounts of magnesium, or some factor is present in the diet that prevents adequate absorption of magnesium. Moderate hypomagnesemia impairs the ability of the cow to maintain calcium homeostasis and hypocalcaemia occurs (Jesse P, 2014) [4].

Conclusion

- This study concluded that early treatment of the recumbent cow suffering from milk fever is essential to prevent necrosis of the tissues of the side on which the animal is lying, which is known to commence within 4 hours of recumbency. The goal of treatment in milk fever is to restore the serum concentration of calcium sufficiently to support cellular function. It is achieved by intravenous administration of calcium salts such as borogluconate at a rate of 2 g/100 kg body weight
- Prevention of milk fever involves several strategies including 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 (Goff, 2008) [5].

References

1. Wubishet F, Tegegne D, Abdela N, Ahmed WM. Milk Fever and its Economic Consequences in Dairy Cows: A Review. Global Veterinaria. 2016; 16**(5)**:441-452.
2. Jesse P. The monitoring, prevention, and treatment of milk fever and subclinical hypocalcemia in dairy cows. The vet. Jour. 2014; 176(1):50-57.
3. Thilsing TH, Jorgensen RJ, Ostergaard S. Milk Fever Control Principles: A Review. Acta Veterinaria Scandinavica. 2002; 43:1
4. Jesse P. Calcium and Magnesium Disorders. Vet Clin Food Anim. 2014; 30:359-381.