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Successful management of milk fever in a Jersey Cow: A case report

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Abstract
A seven year old Jersey cow on her 4th lactation weighing approximately 300 kg body weight was presented with a history of prolonged recumbency and decreased appetite. The cow had calved 4 days ago and was semi-intensively managed. The daily production of milk was about 12 litres. The temperature and pulse rate were found within the normal range, but there was increase in respiratory rate, nasal discharge with mild dehydration. Auscultation on cardiac region revealed decreased intensity of the heart sound. There was no change in the haematology except higher total leucocyte count ($10.73X10^3$µl). Serum biochemical study revealed decreased Calcium (6.14g/dl) and Phosphorus (5.29g/dl) along with increased level of Magnesium (2.18 g/dl). Based on the Clinico-haematological findings, the Jersey cow was diagnosed with milk fever and the treatment was rendered by slow intravenous infusion of Calcium Magnesium Borogluconate (@ 2g of calcium/100 kg body weight), oral calcium gel (@300 ml daily) along with intravenous infusion of 0.9% NaCl and Ringer's Lactate with 5% Glucose (@20ml/Kg body weight/ hour), NSAID (@8.0 ml/100 kg body weight intramuscularly), oral multivitamins (@100 ml orally daily) and probiotics (@200 ml rally b.i.d) to restore the calcium-blood glucose level and also to correct dehydration and restoration of appetite. The prognosis was good and the cow showed prompt recovery after 2 days of treatment.

Keywords: Milk fever, Jersey cow, effective treatment, calcium

Introduction
Milk fever or parturient paresis is an acute to Peracute, afebrile metabolic disease of high producing dairy cows that usually occurs within 48 to 72 hours of calving (Radostits *et al.*, 2007) [16] causing huge reduction in milk production. The disease leads to appearance of other associated conditions like acidosis, Ketosis, Mastitis etc. Several studies have claimed that Milk fever is not a disease of single etiology but rather a complex interrelated disorders (DeGaris & Lean 2009; Drackley *et al.*, 2005; Mulligan and Doherty, 2008) [6, 7, 12]. Various predisposing factors irrespective of age, breed, parturition stage, number of parity, milk production level and genetic predisposition have been implicated in the pathogenesis of the disease (Horst *et al.*, 1997; Roche and Berry, 2006; Mulligan *et al.*, 2006b) [10, 17, 13]. Based on the clinical presentation, three stages of milk fever are identified in ailing animals. In stage I, the cow is able to stand but staggers due to weakness of the muscle. In stage II, the cow is on sternal recumbency with twisting or curving of the neck towards the flank, while in stage III, the cow suffers from paralysis leading to coma and death (Hutjens and Aalseth, 2005; Abdullah *et al.*, 2014) [11, 1]. Both stage I and stage II can be managed easily while treatment of stage III often difficult because of development of secondary complications and muscle paralysis. Cows affected with milk fever can be managed by slow intravenous infusion of calcium borogluconate into the jugular vein. Prevention of milk fever involves several key factors including dietary calcium restriction in the dry period, maintenance of Ca: P ratio in the ration, incorporation of different mineral salts, metabolites or synthetic analogues, intramuscular and intravenous parathyroid hormone (PTH) applications, feeding of calcium rich rations 3 to 4 days before parturition, vitamin D supplementation and magnesium supplementation in late gestation (Goff, 2008; Mulligan *et al.*, 2006b; Patel *et al.*, 2011; Chiwome *et al.* 2017; Nese, 2018) [8, 13, 15, 3, 14]. High yielding dairy cows are more susceptible to milk fever due to the high demands of blood calcium, resulting in metabolic diseases in them. Milk fever mostly occurs shortly after calving and extends up to the peak of lactation within 6 to 8 week of post-partum (Hutjens and Aalseth, 2005) [11]. The prognosis depends on the clinical stage of the disease and promptness in delivery of treatment.

Case history and clinical symptoms

In the present study, a seven year old Jersey cow on her 4th lactation weighing approximately 300 kg body weight was presented with a history of prolonged recumbency and decreased appetite. The cow had calved 4 days ago and was semi-intensively managed. The daily production of milk was about 12 litres. Deworming and vaccination status was regular. Clinical examination of the cow revealed emaciation, depression and lethargy. Although, rectal temperature (102.2^0F) and pulse rate (56 beats per minute) were found to be within the normal range, but there was an increase in the respiratory rate (74 beats per minute), serous nasal discharge with mild dehydration. Auscultation on cardiac region revealed decreased intensity of the heart sound. The cow was on lateral recumbency and completely unable to stand. About 10 ml of blood was collected through jugular venipuncture before treatment and divided into 5 ml with anticoagulant for haematological study (Hb, PCV, MCV, MCHC and TLC) using haematology analyzer and another 5 ml into sterile vacutainer without anticoagulant and subjected for centrifugation at 3000 rpm for 10 minutes for serum biochemical (Calcium, Phosphorus and Magnesium) estimation using commercial kits.

Diagnosis and Treatment

On clinical examination, the vital parameters (*viz*. temperature and pulse) of the cow were within the normal range. However, auscultation of the heart revealed decreased intensity of the heart sound. The cow was on lateral recumbency (Fig 1) with the neck curved towards the left flank and extension of left hind leg. There was mild dehydration (4-5%). There was no change in the haematology except higher total leucocyte count ($10.73X10^3$µl). The serum biochemical study revealed decreased Calcium (6.14g/dl) and Phosphorus (5.29g/dl) along with increased level of Magnesium (2.18 g/dl) as compared to the reference values in healthy cattle.

Fig 1: Lateral recumbency of the affected cow

Fig 2: Clinical recovery of the cow from Milk fever

Based on the clinical history of high milk yield of the cow, improper nutritional management after interviewing with the cattle farm owner, presented clinical symptoms and detailed clinical examination, haemato-biochemical findings, the case was tentatively diagnosed as milk fever. The cow was treated with slow intravenous infusion of Calcium Magnesium Borogluconate @ 2g of calcium per 100 kg body weight, oral calcium gel @300 ml daily along with intravenous infusion of 0.9% NaCl and Ringer's Lactate with 5% dextrose @20 ml/Kg/hourly, non-steroidal anti-inflammatory drug @ 8.0 ml/100kg body weight intramuscularly, oral multivitamins @ 100 ml daily and probiotics @200 ml orally b.i.d. were prescribed for restoration of serum calcium-blood glucose level, correction of dehydration and appetite. The prognosis was good and on 3rd day, the cow started taking feed and was able to perform normal activities (Fig 2).

Discussion

Milk fever also known as parturient paresis is an economically important disease of dairy cows. Although the cases showed recovery after treatment, but most cows if not treated promptly, become susceptible to other metabolic and infectious diseases (Curtis *et al*., 1983; Curtis *et al*., 1984) [4, 5]. Many developed countries have already given utmost priority to prevent Milk fever through adoption of various control principles and factors described in the database (Swety and Pradeep, 2021 and Halder, 2022) [18, 9]. Specific control program should be applied when the incidence of milk fever increases above 10% among high yielding dairy cows (Allenstein, 1993) [2]. Most recommended management practices are the supplementation of easily absorbed calcium throughout calving followed by feeding of acidifying rations by supplementation of anionic salt during last stage of pregnancy (Hutjens and Aalseth, 2005) [11] with feeding of low calcium rations and vitamin D and also at pre-partum.

In the present case, a Jersey cow showing Stage II Milk fever with clinical manifestations of sternal recumbency with curving of neck to the flank, was effectively treated by intravenous administration of calcium salt along with other supplements and recovered within two days.

Conclusion

Dietary deficiency of calcium in particular, as a result of poor ration formulation is the most probable cause of milk fever in the present case. Hence, the farmer was advised and enlightened about the importance of proper ration formulation and provision of mineral supplements to his dairy cows.

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