

Magnesium deficiency in young calves and its management

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Introduction

Hypomagnesemic tetany of ruminants is a non-infectious metabolic disorder that occurs in a wide range of nutritional and managerial conditions (Smith and Edward, 1988). This condition occurred due to magnesium deficiency resulting from a reduced ability of the calves to absorb dietary magnesium as they become older (Smith, 1961).

Magnesium is an essential mineral component, is involved in many of the enzymatic activities in the body, a major intracellular divalent cation and also with many physiological and biochemical functions. There is no feedback regulation to control magnesium concentration in the body of the ruminants as reported by Radostits et al, (2000); Martens and Schwigel, (2000). Therefore, the magnesium concentration in blood and extra cellular fluid are essentially determined by the balance between dietary intake of magnesium, loss in feces and milk and the modulating effect of magnesium homeostasis by the kidney (Radostits et al, 2000).

The magnesium homeostasis depends on absorption from the gastrointestinal tract and endogenous secretions, milk production, uptake by tissues. Conversely, if the outflow (mainly milk secretion and endogenous loss) exceeds inflow, hypomagnesemia occurs because of the lack of hormonal mechanisms of homeostasis. The most prominent signs of hypomagnesemia are excitations and muscle cramps, which are closely correlated with the magnesium concentration in the cerebro spinal fluid. It is suggested that the clinical signs are caused by spontaneous activation of neurons in the cerebro spinal fluid at low magnesium concentrations, which leads to tetany (Martens and Schwigel, 2000).

The calves affected have concurrent hypocalcemia, as they fed milk with low magnesium concentration. The calves fed with only milk, will be insufficient to maintain the balance of magnesium and calcium. The efficiency of absorption during the intestinal transit is reduced in cases of diarrhoea (Radostits et al, 2000). The present study describes the successful management of magnesium tetany in

young calves.

History and observation

The five young calves with a history of staggering gait and an inability to stand up were presented to the peripheral veterinary hospital, shimoga. The calves were reared mostly on mother's milk only without exogenous dietary supply.

The calves were examined clinically; they showed hyperesthesia to touch, ataxia, fine muscular tremors appear, kicking of belly, frothing at the mouth and calf try to get up but falls.

Since the animals did not show any signs of distress like labored breathing, fever, diarrhea etc. It was assumed that the origin of the clinical sign was not infectious. Since such signs could also be observed in tetanus, the animal was thoroughly examined for presence of wounds on the body. Since, there were no external injuries in the calves. It was arrived that the clinical signs exhibited could be due to managerial defect, probable nutritional deficiency. Careful investigation into the clinical signs of the calves indicated towards magnesium deficiency. Hence, the blood samples were collected with EDTA for magnesium level estimation.

Treatment and discussion

Among five calves, the blood magnesium level was in the range of 1.1 to 1.3 mg/100ml. According to Sastry and Rao, (2001) the normal magnesium level is in the range of 2-4 mg/100ml. Based on the blood magnesium level in the present study, the cases were diagnosed as magnesium tetany.

Magnesium tetany in young calves might be due to reduced absorption of magnesium in the intestine up to about 3 months of age and this was the age where maximum susceptibility was observed. It occurs in calves aged 2-4 months or older, fed solely on the diet of whole milk. The calves receiving the large quantity of milk and growing rapidly are more likely to be affected, because of their greater need for magnesium for incorporation into developing soft tissues (Radostits et al, 2000). The calves fed only on milk for long time will

usually, sooner or later, become hypomagnesaemic (Smith, 1961). The sudden death occurs in calves about 4 month of age with clinical signs of muscular dysfunction, including tetanic spasms reported by Haggard et al., (1978).

Hence, the young calves were treated with the calcium magnesium borogluconate injection (Novartis India Limited, Gujarat) 50 ml by slow intravenous route along with 50 ml subcutaneously in neck region for 5 days. Consequently, 1-2 g of magnesium sulphate was given orally per day. The calves were recovered and became normal after the completion of treatment. Radostits et al. (2000) reported that, the oral supplementation of magnesium oxide in calves at the rate of 1g per day was found to be effective. However in the present study the calcium magnesium borogluconate intravenous injection along with oral supplementation of magnesium sulphate in calves given promising result and can be effectively used in the field conditions.

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