

Case Study

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Clinical Management of Hypophosphatemia Induced Post Parturient Hemoglobinuria in a Buffalo

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ABSTRACT

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A Female Buffalo was presented with hemoglobinuria and reduced milk yield with history of calving before one month. The buffalo was diagnosed to have hypophosphatemia through serum biochemistry and treated with phosphorus and calcium supplements and found better improvement over 5 days of treatment. Clinical advises were given to the animal owner and the buffalo recovered uneventfully.

Introduction

Chronic phosphorus deficiency is commonly caused by inadequate feed intake or inadequate phosphorus content in the ration over an extended time and also in grazing animals in arid regions with low phosphorus content in soil. The sudden onset of phosphorus losses through the mammary gland at the onset of lactation and the decreased feed intake around parturition are believed to be the major contributors to postparturient hypophosphatemia of dairy buffalo (Cohrs and Grünberg, 2018). Hypophosphatemia in dairy buffalo is thought

to be associated with an increased risk of disease and impaired productivity particularly in early lactation. Postparturient haemoglobinuria (PPHb) is a clinical syndrome of bovines characterized by intravascular haemolysis, haemoglobinuria and anemia (Bhat, 2010; Mac Williams *et al.*, 1982). The condition is commonly noticed in third to sixth lactation of dairy animals (Radostitis *et al.*, 2000) during the period from calving to fifth week of postpartum (MacWilliams *et al.*, 1982). The etiology of PPHb is believed to involve hypophosphataemia associated to primary dietary deficiency (Chugh *et al.*, 1998;

Samad, 1997). The present case report explains the diagnosis and successful treatment of PPHb in a 6 year old buffalo.

History

A 6 Years old female buffalo weighing around 300 Kg was presented in Thangam Memorial Trust Hospital, Tirupur with the condition of coffee colored urine, partial feeding for past three days, reduced milk yield from 3.5 lit to 1.5 lit and calved one month back. The same episode was reported during last calving and treated by local veterinarian.

Diagnosis

On clinical examination, the buffalo had high temperature 39.8° C, hemoglobinuria (Fig. 1), swollen lymph nodes, tachycardia, pale mucous membrane, polypnea-38/minute, high pulse rate-78/min and doughy rumen with reduced motility. Urine sample was tested positive for glucose and bile salts and the supernatant remains in dark red color on

centrifugation. Blood sample had reduced Hemoglobin-4.5g/dl, PCV-16%,RBC- 2.5×10^6 /ml. The serum biochemistry reveals hypophosphatemia- 1.9 mg/dl and negative for hemoprotozoa.

Treatment

The animal was treated with Inj. Oxytetracycline - 60 ml, Inj. Normal saline - 1000 ml intravenously, Inj. Zobid-M - 10 ml and Inj. Tribivet -10 ml intramuscularly in first day.

Treatment repeated for 4 days along with Inj. Alphas - 20 ml, Calcium borogluconate - 450 ml and Inj. Ringers lactate - 1000 ml intravenously, Inj. Iron sorbital (Feritas) - 6 ml intra muscularly and bolus. Styphon - 8 boli (2-0-2). Clinical status of the animal improved on 5th day of treatment with improved serum phosphorus level of 4 mg/dl, Hemoglobin - 7.1g/dl, PCV - 26%, RBC - 4.5×10^6 /ml and urine colored changed to light yellow.

Fig.1 Clinical symptom of hemoglobinuria in buffalo



Acute phosphorus losses associated with hypophosphatemia are a well-recognized problem in high-yielding dairy cows at the onset of lactation. Phosphorus depletion can also result from chronic renal tubular disease

due to impaired renal reabsorption of phosphorus.

Chugh *et al.*, (1998) reported the severe hypophosphataemia leads to post-parturient

haemoglobinuria in buffaloes. The initial serum phosphorus level in the present reported case was 1.8 mg/dL. This is lower than the normal range (2 mg/dL) indicating phosphorus deficiency in bovines (Samad, 1997).

Increased fragility associated with hypophosphatemia is suggested to subject erythrocytes to destruction by circulating oxidants (Jubb *et al.*, 1990). Phosphorus deficiency is usually primary i.e. involving an absolute deficiency in the diet (Radostitis *et al.*, 2000).

Symptoms includes pale mucous membrane, anemia, increased heart rate ,increased pulse rate, dyspnea, difficult in defecation, hemoglobinuria, decreased serum phosphorus. Young animals grow slowly, develop rickets, and tend to have a rough hair coat, whereas adult animals in early stages may become lethargic, anorectic, and lose weight (MSD vet manual). The phosphorus deficiency, leading to hypophosphatemia, may be a mechanism of post parturient and related syndromes of haemoglobinuria by decreasing red cell glycolysis and resultant ATP synthesis. Subnormal concentrations of ATP would predispose red cells to altered structure and function, a loss of normal deformability, and an increase in fragility and haemolysis with resultant haemoglobinuria (Ogawa *et al.*, 1987) Chronic phosphorus depletion and hypophosphatemia is most effectively treated by providing sufficient amounts of feed with adequate phosphorus content. Intravenous administration of phosphorus-containing solutions is the most appropriate approach. In cattle, rapid administration of sodium phosphate salt solutions is commonly practiced. Mono or dibasic phosphate salts (either Na_2HPO_4 or NaH_2PO_4) infused IV rapidly increase the serum Pi concentration. Tribasic phosphate (Na_3PO_4) is a caustic detergent that cannot be used under any

circumstances for PO or IV phosphorus supplementation. (MSD vet manual).

In conclusion a single injection of acid sodium phosphate 20 ml intravenous route is giving correction for acute hypophosphatemia in Buffaloes. The treatment has a rapid onset of action and a sustained effect. Severe hypophosphatemia condition is an indicator for intravenous rather than oral phosphorus supplementation. Prevention of hypophosphatemia is the best therapy. So these treatments should be used only if indicated and carefully monitored.

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