



Postparturient Haemoglobinuria in A Buffalo - A Case Report

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Abstract

A 7-year-old buffalo, calved 28 days ago, was referred to the Veterinary Clinical Complex of NTR College of Veterinary Science, Gannavaram with a history of reddish-brown discolouration of urine, inappetence and reduced milk yield for the past 4 days. Physical examination revealed significantly increased heart and respiratory rates, along with decreased ruminal motility. Hematobiochemical examination revealed anaemia and hypophosphatemia. Phosphorus deficiency, often associated with a predominantly forage-based diet and inadequate mineral supplementation, is suspected to be the probable underlying cause. A specific therapy with inorganic phosphorus was administered parenterally for five days, along with supportive care. This treatment effectively reversed the clinical course of the disease and resulted in significant improvement. This case report emphasizes the significance of phosphorus supplementation in improving haematological and serum biochemical parameters, highlighting its pivotal role in the successful therapeutic management of post parturient haemoglobinuria in buffaloes.

Keywords: Anaemia, Buffalo, Hypophosphatemia, Post Parturient Haemoglobinuria.

Introduction

Postparturient haemoglobinuria (PPH) is an acute metabolic disorder which is sporadic and non-infectious disease of high yielding dairy animals characterised by intravascular haemolysis, hemoglobinemia, haemoglobinuria, anaemia, drop in milk yield and death due to anaemic anoxia (Sharma *et al.*, 2020). It is less frequently observed in beef cattle compared to dairy cows (Rahmati *et al.*, 2021). The main reason for this difference is the increased loss of phosphates through milk in dairy cattle, particularly during times of low phosphorus intake (Yadav *et al.*, 2023). The considerable risk factors include feeding of cruciferous plants especially, Brassica sp. (Cabbage), mustard, lucerne, berseem, sorghum straw, sugarcane tops and beet reduces the phosphorus level in the body. A higher incidence has been recorded in buffaloes compared to cattle (Bhikane and Syed, 2014). Large ruminants are most susceptible to PPH from the third to sixth gestation period (Almubarak *et al.*, 2023). The transition period from late pregnancy to early lactation, from calving up to four weeks postpartum, represents a high-risk period for the onset of the disease in dairy animals (Sharma *et al.*, 2020). Hypophosphatemia might induce haemoglobinemia and haemoglobinuria by rendering red blood cells more fragile and prone to structural and functional changes (Resum *et al.*, 2017). The present case report describes successful therapeutic management of PPH in a buffalo.

Case History and Clinical Observations

A 7-year-old buffalo with third parity, calved 28 days' back was referred to Veterinary Clinical Complex at NTR college of Veterinary Science, Gannavaram with history of reddish-brown discolouration of urine, inappetence, reduced milk yield and dullness for the past 4 days. The animal was stall-fed on predominantly dry fodder, which is deficient in phosphorus. Clinical examination revealed reddish-brown discolouration of urine (Fig. 1), slightly pale mucous membranes, reduced ruminal motility, tachycardia and normal rectal temperature (101.6°F). Based on the clinical observations, the condition was tentatively diagnosed as post parturient haemoglobinuria associated to dietary phosphorus deficiency. For confirmative diagnosis blood samples were collected in both EDTA and serum vials before initiation of treatment for haemato-biochemical analysis. Prior to treatment the urine sample was centrifuged to confirm haemoglobinuria. Additionally, a urine dipstick test was conducted to perform qualitative analysis of the urine. The blood smears were stained with Giemsa stain and examined for haemoprotezoans under a light microscope.



Figure 1: Reddish brown discolouration of urine

Treatment

Based on clinical and laboratory findings, the case was tentatively diagnosed as of PPH and a course of specific therapy consisting of inorganic phosphorus supplementation and supportive therapy was immediately initiated. Phosphorus supplementation involved the use of buffered inorganic phosphorus solution with ATP stimulators (Novizac®) at a dosage of 50ml intravenously on the first day, along with 500 ml of DNS (Dextrose Normal Saline), followed by 25ml intravenously for the next four days.

Supportive therapy included supplementation of Ethamsylate (10 ml IV, twice daily) and Vitamin B-complex (10 ml IV, once daily) for three days. Oral haematinic syrup and probiotics were administered twice daily for five days. Additionally, a combination of Calcium borogluconate, magnesium and phosphorus with dextrose (Mifex®) was

given at 300 ml IV, with the remainder subcutaneously, and mineral mixture (50gms PO, twice daily) to address mineral deficiencies.

Results and Discussion

Analysis of samples collected prior to treatment revealed haemoglobinuria, evident from the uniform reddish-brown erythrocyte-free urine upon centrifugation. Haematological analysis indicated reduced haemoglobin, packed cell volume and total erythrocyte count, as outlined in Table 1. The current clinical, haematological and urinalysis findings were in accordance with Kumar *et al.* (2019) and Wankhede *et al.* (2021). Increased erythrocyte fragility and haemolysis in PPH have been attributed to impaired glycolytic pathway and depletion of ATP in erythrocytes, predisposing red blood cells to altered functions and structure (Constable *et al.*, 2017). Urine dipstick test was positive for protein, blood cells and the pH of the urine is 8.0. Similar findings were reported by Resum *et al.* (2017).

Table 1: Haemato-biochemical profile of the affected buffalo before and after treatment

Parameter	Prior treatment	Post treatment	Reference range
Haemoglobin (gm/dL)	6.7	8.2	15-Aug
PCV(%)	23	28	25.3-38.5
TEC($\times 10^6/\text{mm}^3$)	4.6	5.3	5.12-9.30
TLC($\times 10^3/\text{mm}^3$)	5.1	6.4	5.80-15.5
Neutrophils(%)	38	32	15-35
Lymphocytes(%)	61	67	60-65
Eosinophils(%)	1	1	0-8
Glucose (mg/dL)	51.4	54.2	42-75
Creatinine (mg/dL)	1.6	1.1	2-Jan
BUN (mg/dL)	21	15	7.8-25
Total Protein (gm/dL)	5.8	6.2	5.7-8.1
Albumin (gm/dL)	3	2.8	2.1-3.6
Globulin (gm/dL)	2.8	3.4	3.2-4.9
Phosphorus (mg/dL)	2.6	5.9	4.6-9.0
Calcium (mg/dL)	8.8	10.2	7.9-10



Figure 2a. Before treatment **Figure 2b.** After treatment

Serum biochemical findings revealed low level of inorganic phosphorus (2.6mg/dl) (Table 1). Hypophosphatemia leads to slowing down of Embden Meyerhoff pathway which subsequently leads to building of oxidative stress on erythrocytes leading to precipitation of haemoglobin and Heinz body formation. Inosine and sodium pyruvate in buffered phosphorus preparation, plays pivotal role in RBC glycolysis and ATP synthesis which reduces oxidative

stress on erythrocytes and prevents them from being fragile (Bhikane *et al.*, 2011). Thus, to replenish the inorganic phosphorus level of blood and to correct metabolic defect, buffered phosphorus solution with ATP stimulators (Novizac®) was used. The animal showed an uneventful recovery, and the urine colour returned to normal five days after treatment (Fig. 2).

Conclusion

In brief, hypophosphatemia might be responsible for the cause of PPH in buffaloes. Early phosphorus supplementation with buffered inorganic phosphorus solution with ATP stimulators can mitigate hypophosphatemia and can improve the clinical course of the disease in affected buffaloes.

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Contribution by Authors

Each co-author contributes equally.

Conflict of Interests

There is no conflict of interest.

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