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Vishaka Sharma

Department of veterinary
Gynaecology and obstetrics,
College of Veterinary & Animal
Sciences, Pantnagar,
Uttarakhand, India

Himanshu Kumar

Department of veterinary
Gynaecology and obstetrics,
College of Veterinary & Animal
Sciences, Pantnagar,
Uttarakhand, India

Satish Kumar

Department of Veterinary
Clinics, College of Veterinary &
Animal Sciences, Pantnagar,
Uttarakhand, India

Mrigakshi Yadav

Department of veterinary
physiology and biochemistry,
College of Veterinary & Animal
Sciences, Pantnagar,
Uttarakhand, India

Preeti Bisht

Department of pharmacology
and toxicology, College of
Veterinary & Animal Sciences,
Pantnagar, Uttarakhand, India

Alok Mishra

Department of Veterinary
Clinics, College of Veterinary &
Animal Sciences, Pantnagar,
Uttarakhand, India

Corresponding Author:

Satish Kumar

Department of Veterinary
Clinics, College of Veterinary &
Animal Sciences, Pantnagar,
Uttarakhand, India

Therapeutic management of post-parturient haemoglobinuria in buffalo: A case report

Vishaka Sharma, Himanshu Kumar, Satish Kumar, Mrigakshi Yadav, Preeti Bisht and Alok Mishra

Abstract

A 6 year old buffalo with third parity was presented to Veterinary Clinical complex at College of veterinary and animal sciences, Pantnagar with reddish brown color urine, inappetance, reduced milk production, and constipation. The laboratory examination revealed anemia haemoglobinuria and hypophosphatemia. A phosphorus deficiency is associated with predominantly forage based diet and inadequate mineral supplementation is suspected for probable underlying cause. A course of five days phosphorus supplementation and non specific supportive therapy was immediately initiated based on the tentative diagnosis. It effectively reversed the clinical course of the disease and brought significant improvement. The case report indicated that phosphorus supplementation may improve the haematological and serum biochemical parameters and successful therapeutic management of post parturient haemoglobinuria (PPH) in buffaloes.

Keywords: Buffalo, anaemia, haemoglobinuria, hypophosphatemia, post-parturient

Introduction

The Postparturient haemoglobinuria (also known as “Lahu mutna” or “Rakth mutna”) is a metabolic disorder of high producing dairy animals and an endemic disease in buffalo, which is characterized by intravascular haemolysis, haemoglobinuria and anemia (Bhat, 2010^[1]). The condition is common among animals in their third to sixth lactation (Radostitis *et al.*, 2000)^[8]. The transition between late pregnancy and early lactation, from calving upto 4 week postpartum, is a high-risk period for the occurrence of the disease in the dairy animal. The risk is high during the period of parturition (Moore *et al.*, 1997 and Whitaker *et al.*, 1999)^[7, 12]. The inorganic phosphate is a potential indicator of the bioavailability of feed phosphorus in dairy cows (Montiel *et al.*, 2007)^[6]. It is believed that inorganic phosphate level in the blood reveals an indication of the dietary phosphate intake and haemoglobin was measured to access anaemia (Grunwaldt *et al.*, 2005)^[2]. The hypophosphatemia inhibits erythrocyte ATP production, causes a loss of normal deformability and predispose cells to increased fragility (Wang *et al.*, 1985)^[11]. The pathogenesis of erythrocyte destruction leading to anaemia and haemoglobinuria in PPH is unknown. Otherwise, bacterial haemolysis and blood parasites (Babesia, Anaplasma etc.) in cows may cause red blood cell destruction (Macwillims *et al.*, 1982)^[5]. Prevention of clinical disease requires the proper nutritional management of pregnant and lactating buffaloes. The present case report describes changes of some biochemical and haematological parameters with successful treatment in a buffalo for PPH.

Case history and observations

A 6 year old buffalo with third parity was presented to Veterinary Clinical complex at College of veterinary and animal sciences, Pantnagar with a history of parturition 3 month back and was reported suffering from a generalized illness with reddish brown discoloration of urine starting three days back. The other prominent observations viz. inappetance, reduced milk production and constipation. The animal was kept tied in stall and fed on a predominantly green fodder specially sugarcane pulp and cabbage. Clinical examination showed reddish brown colored urine (Figure 1), slightly pale mucus membrane, heart rate 98 per minute, bit difficulty in respiration and normal temperature (100°F). The posture, gait and skin coat condition of the animal was normal. On the basis of clinical findings, the condition was tentatively diagnosed as postparturient haemoglobinuria associated to dietary phosphorus deficiency and hypophosphatemia. A course of therapy comprising of phosphorus

supplementation and non specific supportive therapy was immediately initiated based on the tentative diagnosis. For confirmative diagnosis blood samples were collected before treatment. An aliquot of blood was placed into an EDTA-containing tube for haematological examination. The rest of the blood samples were placed into glass tubes for the serum. These tubes were centrifuged and harvested serum was immediately frozen at -20 °C until analysis. The serum total protein (TP), albumin (Alb), globulin, calcium (Ca), phosphorus (P) and glucose, were measured by an autoanalyser. The blood smears were stained by Giemsa's stain and then examined for blood parasites in erythrocytes under a light microscope.

Therapeutic protocol

The animal received phosphorus supplementation and supportive treatment.

Phosphorus supplementation: Sodium acid phosphate 40.3% weight/volume (w/v) (equivalent to elemental phosphorus 8 % w/v), 30 ml suspension given in 1 liter Normal Saline as intravenous drip, once a day for 5days.

Supportive therapy

- A liverotropic (Thiamin hydrochloride 30 mg, Pyridinone hydrochloride 13.75 mg and Cyanocobalmine 50 mg), 10 mL, IM, once a day for 5 days
- Anti-inflammatory (Dexamethasone 4.4 mg/ml), 1o ml, IM, once a day for 3 days
- Calcium borogluconate together with Magnesium and Phosphrous in organic combination and Dextrose (Mifex), 300 ml slow IV and rest subcutaneously.
- Niacinamide, Folic acid, Vit B12 and B6 Glycinated iron copper & cobalt, Vitamine -E (RBCrakt). Fluid therapy, Rumenotoric - Biovet Yc- goold

Result and Discussion

Analysis of pretreatment samples like urine, whole blood and serum indicated that haemoglobinuria (uniformly reddish brown erythrocyte free urine) (Figure.1), anemia (reduced Hb), reduced Packed cell Volume (PCV), Total Erythrocyte Count (TEC) total leukocyte count (TLC), and differential leukocyte count (DLC) (Table 1).The serum biochemical parameters (Glucose,Total protein, Albumin and Globulin) are given in Table 2. The blood smear showing negative for haemoprotozoan (Figure.2a) and clumping of erythrocyte (Figure.2 b).

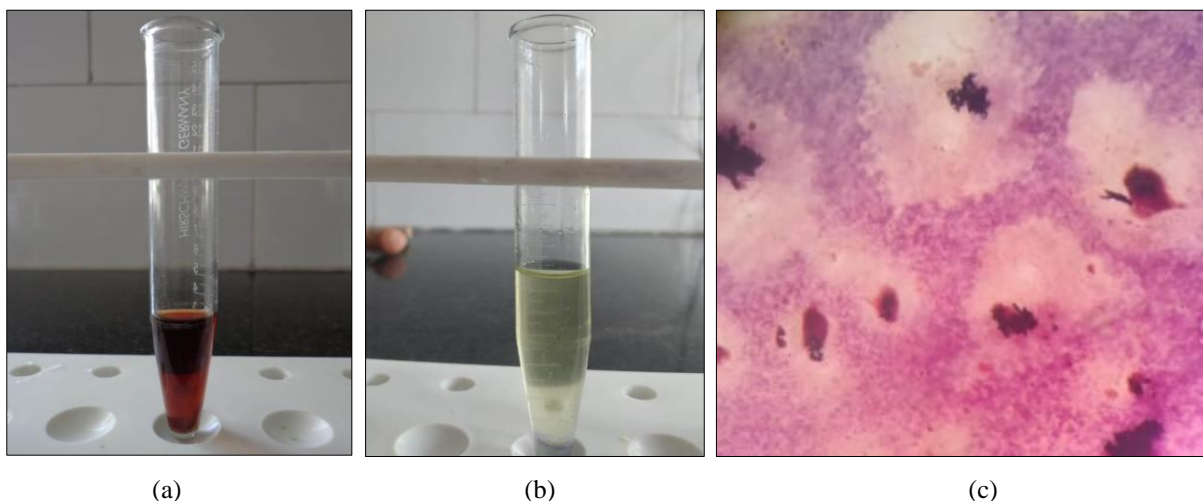


Fig 1: urine examination findings (a) reddish brown color urine (b) post treatment urine color (c) acellular debris and protein cast

The clinical findings of this case revealed for PPH in buffaloes as low level of inorganic phosphorus (1.7 mg/dl). (Table.3). Five day post treatment urine colour was normal (Figure.1b) and urinary protein cast in acellular debris seen in

microscopic examination of stained urine (Figure1.c). Qualitative urine test includes protein (positive), glucose (positive), blood cells (present) ketone bodies (absent) and bile salts (negative), Ph of urine -8.2.

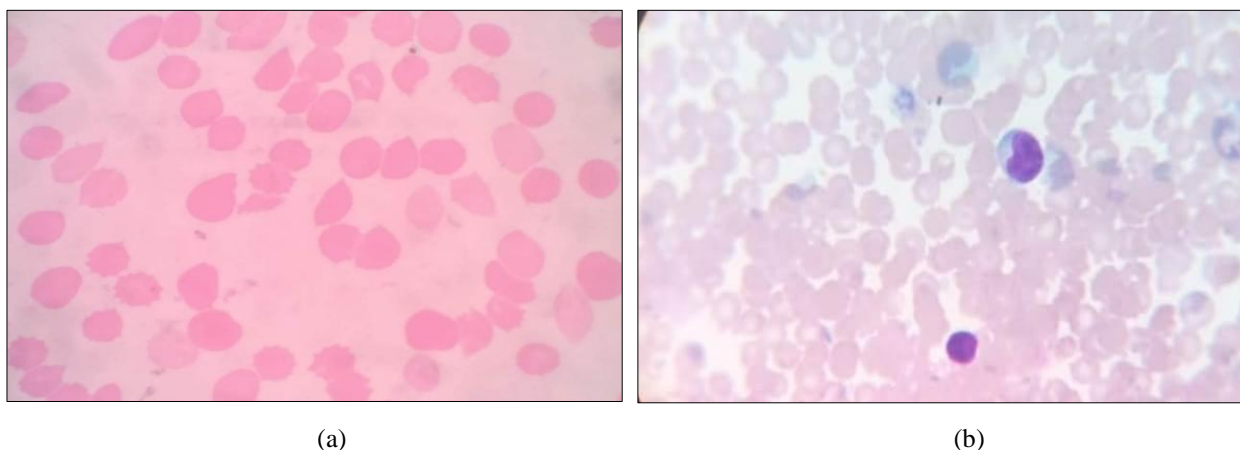


Fig 2: (a) blood smear negative for haemoprotozoan (b) leishman stained blood smear showing erythrocytic clumping

Table 1: Hematological analysis

Parameters	Pre-treatment	Post -treatment	Reference range
Hemoglobin (gm %)	7.8	10.8	8-15
PCV (%)	27	34	44
TLC (10 ³ /cumm)	6.2	6.7	6-7
TEC(10 ⁶ /cumm)	5.4	6.6	6-8
Differential leukocyte count (DLC)			
Neutrophils (%)	38	32	28-30
Lymphocytes (%)	58	63	60-65
Eosiniphils (%)	01	01	2-9

Table 2: Serum biochemical examination parameters pre and post treatment

Parameters	Pre-treatment	Post -treatment	Reference range
Glucose (mg/dl)	52.16	54.12	50-55
Total protein (gm/dl)	4.82	6.9	6-7
Albumin (gm/dl)	3.2	4.1	2.7-4.3
Globulin (gm/dl)	2.7	2.9	3.3

Table 3: Mineral profile

Parameters	Pre-treatment	Post-treatment	Reference range
Phosphorus (mg/dl)	1.7	4.2	4.6-9.0
Calcium ((mg/dl)	8.6	10.0	7.9-10

The phosphorus requirements of buffalo may vary depending on the various stages of growth, lactation, and pregnancy. It is well known that phosphorus deficiency can reduce the adenosine triphosphate (ATP) content in red blood cells, which influence the structure and function of the cell, thereby increasing fragility and haemolysis, which may lead to acute haemoglobinuria (Wang *et al.*, 1985) ^[11]. In this case report, anorexia reduced milk production, moderate tachycardia, pale mucosa membrane and haemoglobinuria were detected in the buffalo. Moreover, the serum phosphorus was very low (1.7 mg/dl). Jubb *et al.* (1990) ^[3] reported that circulating oxidants cause erythrocyte damage, which may be predisposed to hypophosphataemia. Similarly Stockdale *et al.* (2005) ^[9] reported that marginal dietary phosphorus deficiency may lead to acute haemoglobinuria. Thompson and Badger (1999) ^[10] found that the serum phosphorus level in the cattle with haemoglobinuria was markedly decreased. In addition, Karapınar *et al.* (2006) ^[4] also detected that the serum phosphorus level in cows with haemoglobinuria was very low (0.5 and 1.5 mg/dl).

Conclusion

From this case report we can conclude that phosphorus deficiency may be responsible for the development of post-parturient haemoglobinuria in buffaloes. Early phosphorus replacement therapy can alleviate hypophosphatemia and improve the clinical condition in affected buffaloes.

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