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OPEN ACCESS WORLD ELEPHANT DAY - 12TH AUGUST CASE STUDY

A case report on Post-Parturient Haemoglobunuria (PPH) in Buffalo

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Abstract:

An 8 year buffalo with third parity was presented to Veterinary Clinical complex at Sri Ganganagar Veterinary College, Tantia University with clinical sign including coffee coloured color urine, anorectic, reduced milk production, and impaction. The laboratory examination revealed haemoglobinuria associated with hypophosphatemia (low blood phosphorus levels). A course of five days phosphorus supplementation (sodium-acid-phosphate powder) and supportive therapy was given based on the diagnosis. PPH affected buffalo treated with sodium acid phosphate, inj. ascorbic acid and inj. Vitamin E and selenium was recovered completely.

Keywords: Buffalo, anaemia, Postpartum haemoglobinuria, hypophosphatemia.

Introduction:

Postpartum Hemoglobinuria (PPH) is also known as “Lahu mutna” and it is a metabolic disorder of high producing dairy animals potentially life-threatening condition affecting high-yielding dairy animals, particularly buffaloes, during the early postpartum period or just after parturition. It is characterized by intravascular hemolysis, leading to the excretion of free hemoglobin in the urine, giving it a characteristic dark red or coffee-colored appearance (Bhat, 2010[1]. The condition typically manifests within one month after parturition and is most prevalent in multiparous, high-producing animals under intensive management systems. (Moore et al., 1997 and Whitaker et al., 1999) [6, 11]. The condition is common among animals in their third to sixth lactation (Radostitis et al., 2000) [7].

PPH represents a significant economic concern due to treatment costs, reduced productivity, and possible loss of valuable dairy animals. Early diagnosis and prompt phosphorus supplementation, along with supportive care, are critical for successful recovery. Preventive strategies, including balanced mineral nutrition and proper postpartum management, are essential to reduce the incidence of the disease in susceptible herds.

Case History and Laboratory Observations:

An 8-year-old multiparous Murrah buffalo, 08 days postpartum, was presented to the veterinary clinic complex of Sri Ganganagar Veterinary College, Tantia University with complaints of dark red coffee colored urine, sudden drop in milk yield, anorexia, weakness, and mild icterus. The buffalo had calved normally and was previously in good health. On the basis of clinical findings, the condition was tentatively diagnosed as post parturient haemoglobinuria (Wang XL et al., 1985[10] associated to dietary phosphorus deficiency and hypophosphatemia.

To confirm the PPH, a series of laboratory investigations were carried out, focusing on hematology, serum biochemistry, and urinalysis. The aim was to detect intravascular hemolysis, hypophosphatemia.

In Urinalysis with help of microscopic examination was presence of free hemoglobin in the urine that's why in case of buffalo confirmed for intravascular hemolysis.

A complete Hematological analysis was performed to assess the severity of anemia and to evaluate red blood cell integrity. Blood was collected aseptically from the jugular vein into EDTA tubes for hematological evaluation. The hematological profile revealed:

- Moderate to severe anemia, as evidenced by low hemoglobin and PCV levels.
- No signs of infection or inflammation, as the leukogram were within normal limits.
- Absence of hemoparasites on blood smear ruled out infectious hemolytic conditions like babesiosis or anaplasmosis.
- The findings supported the diagnosis of Postpartum Hemoglobinuria, secondary to non-infectious intravascular hemolysis, most likely due to hypophosphatemia.

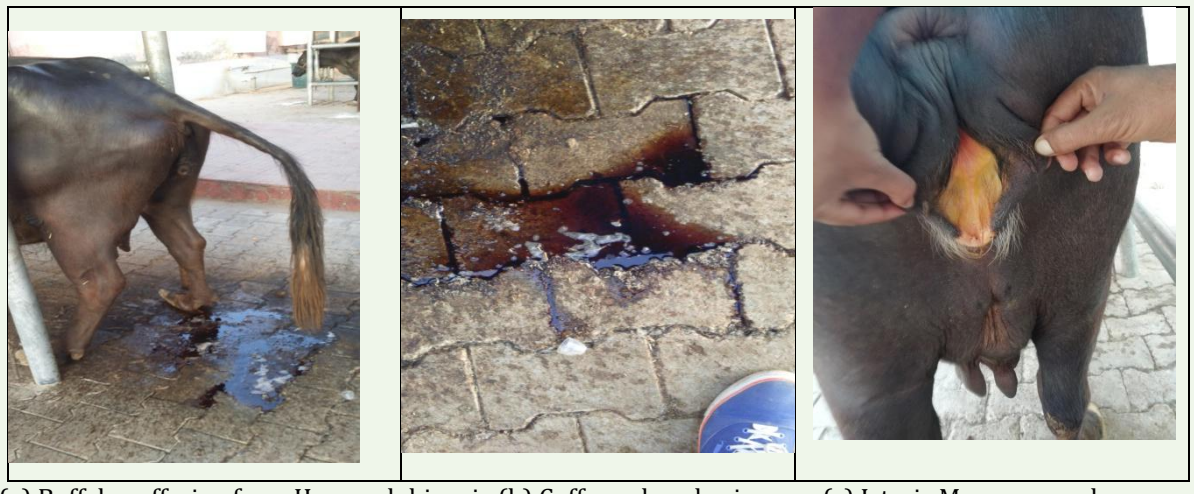

Fig 1: (a) Buffalo suffering from Haemoglobinuria (b) Coffee colored urine (c) Icteric Mucous membrane

Therapeutic Protocol:

The treatment for post-partum hemoglobinuria was the slow intravenous administration of sodium acid phosphate, given at a dose of 60 gm diluted in 500 mL normal saline, once daily for 3 days, same 60 gm dissolve in normal saline use as subcutaneous for 3 to 5 days and 60 gm powder use orally. This helps replenish deficient inorganic phosphorus and restore red blood cell stability. In addition to phosphorus

therapy, antioxidants such as ascorbic acid (20 ml IM daily) for 5 days and vitamin E with selenium (5–10 mL IM every other day for 2–3 doses) are administered to counter oxidative damage that contributes to red blood cell destruction.

Parameter	Result	Reference Range	Interpretation
Hemoglobin (Hb)	5.5 g/dL	8–15 g/dL	Decreased
Packed Cell Volume (PCV)	16%	24–46%	Decreased
Total RBC Count	Decreased	Normal: 5–10 million/µL	Anemia
Total Leukocyte Count (TLC)	Normal	4,000–12,000 /µL	No infection
Blood Smear	No hemoparasites	–	No parasitic cause

Table 1: Hematological analysis

Result and Discussion:

The buffalo presented with classical signs of Postpartum Hemoglobinuria (PPH), including dark red coffee colored urine, reduced milk yield, anorexia, weakness, and mild jaundice, within 08 days postpartum. Hematological and biochemical investigations confirmed moderate anemia (Hb: 5.5 g/dL, PCV: 16%), normocytic normochromic RBCs, (Grünwaldt EG et al., 2005[2] and a marked decrease in serum inorganic phosphorus (1.6 mg/dL), which is consistent with the known pathophysiology of PPH. (Stockdale CR et al., 2005[8], (Jubb TF et al., 1990[3]. Urinalysis revealed hemoglobinuria without the presence of intact red blood cells, confirming intravascular hemolysis rather than hematuria.

The therapeutic protocol, including IV sodium acid phosphate, Ascorbic acid, vitamin E + selenium, IV fluids, and oral phosphorus supplementation, was initiated immediately. The buffalo showed visible improvement within 48 hours, with normalization of urine color, return of appetite, and gradual recovery of milk production. Full clinical recovery was observed within 5 days, with no relapse during a 20-day follow-up period.

Parameter	Pre-treatment	Post-treatment	Reference range
Phosphorus (mg/dl)	1.6	4.2	4-7

Table 2: Phosphorus analysis

The results of this case strongly support the role of hypophosphatemia as the primary etiological factor in PPH. Deficiency impairs membrane function, making erythrocytes more susceptible to oxidative damage and haemolysis. The response to phosphorus supplementation and antioxidant therapy further emphasizes the

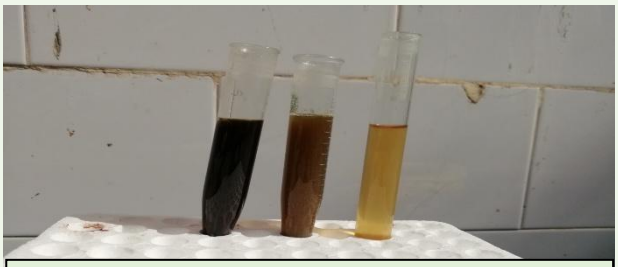

Fig 2: Changes in coffee color to normal color of urine from first day to last day of treatment

importance of timely mineral correction and oxidative stress management.

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