Biological Times

Volume No. 3 | Issue No. 11 | Pages 21-22

Post parturient hemoglobinuria in lactating Buffaloes

Assam Ali, Habiba Asif, Waseem Abbas, Faisal Ramzan and Muhammad Mahboob Ali Hamid*

Institute of Animal & Dairy Sciences, Faculty of Animal Husbandry, University of Agriculture Faisalabad, Pakistan

*Corresponding Author: **dr.mmahboob@uaf.edu.pk**

ABSTRACT

Postpartum Hemoglobinuria (PPH) is a metabolic disorder seen around the world, which affects the high producing of buffaloes and cows mostly in their 3rd to 6th lactation or in pregnancy. This disease is critically dangerous, if left untreated it becomes highly lethal causing intravascular hemolysis and anemia. Along with phosphorous deficiency, low serum copper level and hemolytic toxin containing crops like cruciferous plants can trigger its progression. The soil low in phosphorous give rise to phosphorous deficient plants which further results into hypophosphatemia. Elevated sulfur and lack of copper also raise the risk of PPH. The clinical signs include diminished appetite, red to dark brown color of urine (Red water name given because of this), pale mucous membrane, and reduced milk production. The buffaloes that are affected severally require blood transfusion and supportive therapies. Buffaloes can be treated with parental phosphorous supplements, IV administration of sodium acid phosphate, complemented by oral di-calcium phosphate, is quite effective.

Keywords: Post parturient haemoglobinuria, buffaloes, lactating buffaloes

To cite this article: Ali A, H Asif, W Abbas, F Ramzan & MMA Hamid. Post parturient hemoglobinuria in lactating Buffaloes. Biological Times. 2024 November 3(11): 21-22.

Introduction

Post Parturient hemoglobinuria (PPH) also known as "Red water" is an intense metabolic disorder with detrimental economic losses seen globally primarily impacting high milk producing buffaloes and cows shortly after parturition [1]. Marked by the destruction of red blood cells, Anemia and hemoglobinuria are often accompanied by symptoms like brownish urine, whitish or pale mucosal appearance, fatigue, accelerated breathing and significant declines in milk production due to significant phosphorous loss [2]. Animals in their third to sixth lactation are mostly susceptible to this disease, resulting in economic loss due to lower milk production, medical costs and high mortality rates in critical conditions. In South Asia's dairy sector, it remains a challenge as it sporadically affects 1-2 animals per herd [3]. Compared to Cows, Buffaloes are more at risk due to their high phosphorous demands, inefficient mineral absorption and stress-sensitive red blood cells.

Etiology

Etiology of PPH is still not completely understood but phosphorous deficiency is commonly recognized as a leading factor. Calcium stress triggers parathyroid hormone secretions which heighten phosphorous excretions through urine and saliva potentially causing risk of hypophosphatemia in calcium-stressed animals. Thus, proper cation-anion balance in diets is essential as the serum balance of calcium and phosphorous depends on it. This explains why hypo calcemic animals often develop hypophosphatemia. Copper deficiency further exacerbates the condition by inhibiting superoxide dismutase, a copper dependent enzyme which diminishes the oxidative stress mechanism of erythrocytes. However, this disrupts the normal balance phosphorous concentration leaving calcium deficient animals prone to phosphorous deficiency [4]. PPH is more common in regions like India, Pakistan where diets of animals don't have enough phosphorous. Feeds like sugar cane tops, sugar beets, mustard, cabbage, lucerne, berseem, gram, sugar beet pulp can cause phosphorous deficiency. Also, the areas where soil is deficient in phosphorous crops that grow there will likewise be deficient in phosphorous [5]. Ingestion of cruciferous plants, decreased selenium and a rise in molybdenum level is also considered as a risk factor for PPH, high molybdenum concentrations in soil or fodder reduces phosphorous level in body by hindering its absorption in GIT [6]. Although phosphorous deficiency is a major factor, the detailed causes of PPH remain to be fully understood.

Nutritional and Biochemical Deficiencies in PPH

PPH has been associated with several nutritional and biochemical deficiencies in buffalo, mainly linked to phosphorus and copper levels, A persistently low serum phosphorus concentration in affected buffalo reflects phosphorus deficiency as a possible underlying factor for PPH [7]. Phosphorus deficiency can arise from certain feed ingredients, including sugar cane tops, sugar beet, kale, mustard, cabbage, lucerne either because of their low phosphorus levels or due to inhibitory factors, such as metallic ions, which obstruct the absorption and utilization of dietary phosphorus. Although berseem is commonly used forage due to its nutritional value, it carries the risk of PPH under certain feeding practices. The risk arises when it is fed excessively, high protein levels from excessive berseem can create nitrogen imbalances, increasing the chances of metabolic challenges associated with this condition. The soil environment in which Berseem grows plays a role in its mineral content. Elevated sulfur and a lack of copper can significantly raise the risk of PPH, as copper is important for maintaining red blood cell health. The addition of maize husk and barley grit has been linked to outbreaks of nutritional hemoglobinuria in recently calved buffaloes [8]. Excessive salt in drinking water, high level of molybdenum or potassium and low level of copper in feed can also cause PPH. In addition, the hormonal changes especially release of parathyroid hormone during calcium stress cause increase of phosphorous losses through kidneys.

Physiological and life-cycle risk factors

PPH mainly occurs during the transition period from pregnancy to lactation. With the progression of pregnancy, the fetus calcium and phosphorous demands grow and inability to meet these demands leads to hypophosphatemia. There is a significant correlation between PPH and Buffalo that have reached a parity of Four or more. Buffaloes with 3 or more parity have a 6.39 times higher chances of experiencing PPH than those with 2 or less parity. During the periods of peak milk production from the third to sixth lactation the increased stress on mineral imbalance of buffalo may be a contributing factor in developing PPH. Blood calcium level and phosphorous level are strongly connected to each other. Thus, majority cases of hypophosphatemia are driven by hypocalcemia [9]. Buffaloes with a prior history of PPH significantly increase their risk of experiencing the condition again, as they are 21 times more likely to present with low serum phosphorus levels, which compounds the challenges in managing their health [10].

The physiological stressors associated with advanced pregnancy and lactation further heightens the risk of PPH.

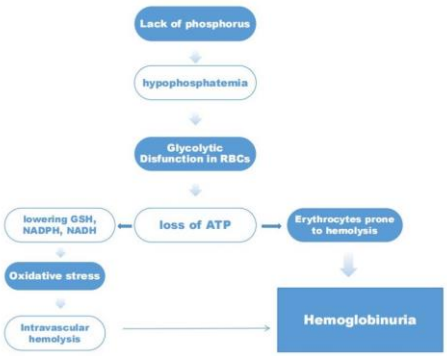

Fig. 1: The Mechanism behind Hemoglobinuria Induced by Phosphorus Deficiency

Environmental interactions

PPH poses a significant risk to buffaloes due to species-specific vulnerabilities and agro-environmental factors due to unique physiological traits and specific nutrient needs that are often unmet under various environmental conditions. A vital aspect influencing PPH is the availability

of phosphorus; in areas with phosphorus-deficient soils, crops tend to have low phosphorus content, increasing the risk for buffaloes consuming these crops. Additionally, their dietary preferences, particularly the consumption of cruciferous plants and other high-inhibitory-factor fodder, contribute to phosphorus deficiency. Environmental stressors, such as elevated molybdenum and potassium levels in feeds or drinking water, further exacerbate phosphorus deficiency, increasing the likelihood of PPH [11]. Regions experiencing heavy rainfall often suffer phosphorus loss due to soil leaching, leading to an increase in PPH cases.

Clinical Signs & Symptoms

In buffaloes suffering from hemoglobinuria, the urine color ranged significantly, with 28% exhibiting red, 20% dark red, and 52% coffee-colored urine linked to illness severity and duration. Milk production is high before the disease, then drop substantially post-onset. In cases of illness, respiration and pulse rates are considerably accelerated, while ruminal motility is often weak and diminished. At more advanced stages, anorexia is commonly seen, with symptoms such as ruminal stasis and severe straining during defecation. Some animals initially continue normal eating following the appearance of discolored urine, with appetite loss emerging later. Feces consistency may be normal, hard, or in some cases, loose [12]. Enlarged lymph nodes, pale mucous membranes, decreased capillary refill time (1-2 per sec), dyspnea, weak pulse, tachycardia, panting and mild salivation are also clinical signs shown by PPH animals. The pH of urine in haemoglobinuric buffaloes is significantly higher than in healthy buffaloes (7.86 ± 0.20).

Treatment and Supportive Therapies for PPH

An early diagnosis proves crucial, and parenteral phosphorus supplementation serves as a key element in successful treatment, greatly enhancing clinical outcomes like temperament, food intake, and urine color. Research findings suggest that the IV administration of sodium acid phosphate, complemented by oral di-calcium phosphate, is an appropriate treatment for PPH in cattle and buffaloes. Administering sodium acid phosphate for seven days as part of a regimen, together with supportive therapy, has proven to be quite effective. Supplementing acid inorganic phosphorus is effective in managing PPH in crossbred dairy animals. Moreover, Positive treatment outcomes in buffaloes have been observed with the IV infusion of copper glycinate and the administration of oral di-calcium phosphate [13]. Evidence shows that treatment outcomes improve when sodium acid phosphate is combined with ascorbic acid, compared to using sodium acid phosphate by itself [14]. It is essential to include supportive therapy as part of the treatment for PPH-affected animals. Some commonly used strategies in therapy are Administration of Liver tropic injection of thiamine hydrochloride, Pyridoxine, Cyanocobalamin intramuscularly continuously for 5 days. And for inflammation control providing of 10ml of Dexamethasone for 3 days, slow IV infusion of 300ml of Mifex (Calcium Boro gluconate together with Magnesium and Phosphorus and Dextrose) followed by subcutaneous administration to ensure the therapeutic effect. To support recovery process Niacinamide, folic acid, vitamin B12, B6, glycinated iron, copper, cobalt and Vitamin E are used [15].

Conclusion

Postpartum Hemoglobinuria (PPH) is a serious metabolic disorder seen around different areas of the world including Pakistan and India which is the major threat for the dairy farmers. This disease can severely affect the high yielding buffaloes if left untreated. This metabolic disorder in buffaloes is mainly influenced by phosphorus deficiency in ration and causing stress due to malnutrition. Apparently, it is seen at postpartum period in high producing buffaloes. To mitigate this problem prevention and efficient management of PPH is needed, which includes phosphorus supplementation, precision nutrition strategies for regulating body homeostasis during postpartum period and balanced mineral intake. To avoid its risks early diagnosis and timely interventions should be done. Cooperation of farmers with experienced veterinarians for optimized herd health management can reduce this risk.

References

[1] Akhtar MZ, Khan A, Khan MZ, Muhammad G. Haemato-biochemical aspects of parturient haemoglobinuria in buffalo. Turkish Journal of Veterinary & Animal Sciences. 2007;31(2):119-23.

[2] Bhikane AU, Syed AM. Recent trends in management of metabolic disorders of transition buffaloes and buffaloes. Intas Polivet. 2014;15(2):485-96.

[3] Abdul Samad AS. Host and environmental factors associated with phosphorus deficiency haemoglobinuria in buffaloes

[4] Purohit GN, Trilok G, Amit K, Atul S, Mitesh G, Chandra S. Perspectives of parturient hemoglobinuria (pph) in buffaloes. International Journal of Development Research. 2018;2(10):23513-20.

[5] Kumar CP, Praveena G, Sundar NS. Clinical management of acute postparturient haemoglobinuria in a graded murrah buffalo. Intas Polivet. 2014;15(2):531-4.

[6] Tewari D, Singh VK, Gautam S. Clinical management of parturient haemoglobinuria in a buffalo (Bubalus bubalis). Intas Polivet. 2014;15(2):528-30.

[7] Dalir-Naghadeh B, Seifi HA, Asri-Rezaei S, Pilevary N. Post-parturient haemoglobinuria in Iranian river buffaloes: a preliminary study. Comparative Clinical Pathology. 2006 Mar; 14:221-5.

[8] Chhabra S, Randhawa CS, Sethi AP. Nutritional haemoglobinuria in a buffalo herd fed with maize husk and barley grit. Buffalo Bulletin. 2015 Dec 1;34(4):465-8.

[9] van den Brink LM, Cohrs I, Golbeck L, Wächter S, Dobbelaar P, Teske E, Grünberg W. Effect of Dietary Phosphate Deprivation on Red Blood Cell Parameters of Periparturient Dairy Cows. Animals. 2023 Jan 25;13(3):404.

[10] Heuer C, Bode E. Variation of serum inorganic phosphorus and association with haemoglobinuria and osteomalacia in female water buffaloes in Pakistan. Preventive veterinary medicine. 1998 Jan 1;33(1-4):69-81.

[11] Patel SR, Hasan SR, Aghav PD. 2002. Epidemiology, clinic pathology and treatment of hemoglobinuria in buffaloes. Proc National Symp XX Conv Indian Soc Vet Med, Bikaner, Rajasthan, India Abstr, 135.

[12] Khan A, Akhtar MZ. Hemato-biochemical andclinico-epidemiological aspects of parturient hemoglobinuria in Nili-Ravi buffaloes. Italian Journal of Animal Science. 2007 Jan 1;6(sup2):953-6.

[13] Rahmati S, Aziz A, Tawfeeq MM, Zabuli J, Nazhat SA. Clinical features of post-parturient hemoglobinuria in dairy cattle and buffaloes: A review. Open Journal of Veterinary Medicine. 2021 Apr 1;11(4):143-55.

[14] Purohit GN, Trilok G, Amit K, Atul S, Mitesh G, Chandra S. Perspectives of parturient hemoglobinuria (pph) in buffaloes. International Journal of Development Research. 2018;2(10):23513-20.

[15] Sharma V, Kumar H, Kumar S, Yadav M, Bisht P, Mishra A. Therapeutic management of post-parturient haemoglobunuria in buffalo: A case report. The Pharma Innovation Journal. 2020; 9:299-301.