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Research Article

CLINICO-BIOCHEMICAL AND THERAPEUTIC STUDIES IN POST-PARTURIENT HAEMOGLOBINURIA IN BUFFALOES

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ABSTRACT

The study was conducted on 50 clinical cases of post-parturient haemoglobinuria in buffaloes along with 15 healthy animals each from villages and organized farms. Post-parturient haemoglobinuria was recorded mostly in milch buffaloes kept under village conditions. No incidence of disease could be recorded from organized dairy farms. The disease was mostly found in lactating buffaloes (84%) between 3rd to 6th lactation (94%). Some animals (16%) were in advanced pregnancy. Haemoglobinuric buffaloes passed red or coffee colored urine and manifested partial or complete anorexia, straining during defecation and anemia. The diseased animals had significantly low haemoglobin, packed cell volume and inorganic phosphorus.Therapy with sodium acid phosphate along with ascorbic acid was found to be highly effective as compared to sodium acid phosphate alone.

Key words: Phosphorus, haemoglobin, PCV, haemoglobinuria, buffaloes

Post-parturient haemoglobinuria (PPH) is an acute disease of cattle and buffaloes characterized by severe intravascular haemolysis, haemoglobinaemia, haemoglobinuria, anaemia and death due to anaemic anoxia. It is emerging as a potent threat to buffaloes in India and other buffalo rearing countries of the world. Predisposing factors for this condition are thought to be advanced pregnancy, recent parturition, high milk yield, presence of oxidants in feed and other stress factors. Deficiency of inorganic phosphorus in blood is a constant finding and cases respond to phosphate therapy (Nagpal *et al.*, 1968, Malik and Goutam, 1971, Gahlawat, 1998). Apart from phosphorus deficiency, oxidative stress is also responsible for red blood cell membrane alterations and subsequently to haemolysis. Therefore, vitamin C can be the part of therapy to reduce the oxidative stress.

MATERIALS AND METHODS

The present study was conducted on 50 clinical cases of PPH in buffaloes from Haryana and adjoining areas. For comparison, apparently healthy lactating, non-pregnant buffaloes maintained under village conditions and in organized farms were kept as controls. Therapeutic trial in diseased buffaloes was conducted in two groups. Animals in group I (n=25) were given sodium acid phosphate 80 gm i/v as 20% solution and 80 gm orally once daily, while group II animals (n=25) were administered 50 gm sodium acid phosphate plus 7.5 gm ascorbic acid i/v as 20% solution and 50 gm sodium acid phosphate orally once daily till the recovery. Blood samples were collected by jugular venipucture in sterilized vials containing heparin as anticoagulant on day zero (before treatment) and 24, 48 and 72 h after start of treatment. Haematological parameters (haemoglobin and packed cell volume) were estimated as per the method described by Schalm *et al.* (1975). Inorganic phosphorus (Pi) levels in serum were estimated as per the method of Taussky and Shorr (1953).

RESULTS AND DISCUSSION

Post-parturient haemoglobinuria was recorded mostly in milch buffaloes kept under village conditions. No incidence of disease could

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be recorded from any of the organized dairy farms. It could be due to the fact that a well balanced and nutritionally adequate ration supplemented with minerals is being given to the animals which effectively prevents PPH. Clinical manifestations of PPH recorded were red or coffee colored urine, anemia, partial or complete anorexia, straining during defecation, recombancy in advanced stage and death due to anaemic-anoxia. The affected buffaloes were in various lactations ranging from 2nd to 8th, however, majority of the cases (94%) were seen in 3rd to 6th lactation. The incidence of this disease can be correlated with the peak milk yield period, which is usual in these lactations. Most of the cases (84%) were of post-parturient animals while 16% cases were in advanced pregnancy. Eight per cent of the animals had the history of previous occurence of PPH. The above observations were in close agreement with those of Nagpal *et al.* (1968), Singh (1976) and Sridhar (1995).

Mean haemoglobin level in the affected buffaloes were found to be significantly low (7.09 ± 1.23 gm%) as compared with healthy ones of village (9.65 ± 1.27g%) (Table 1). However, the levels (12.23 ± 1.36 g%) in farm healthy buffaloes were comparable with village healthy animals and the difference was insignificant. A steady gradual fall in haemoglobin level was the direct result of severe haemolysis seen in this disease. Similar findings have been reported by Nagpal *et al.* (1968), Singh (1989) and Gahlawat (1998).

A significant decrease in the mean packed cell volume (PCV) values was observed in haemoglobinuric buffaloes (20.60 ± 3.97%) as compared to village healthy control (29.67 ± 2.33%). There was no significant difference between the mean packed cell volume of farm healthy (33.60 ± 2.97%) and village healthy buffaloes. As evident from the data, the PCV in affected buffaloes was approximately 30.57% of the values of village healthy buffaloes and about 9.07 per cent erythrocytes had lysed by the time treatment was started. The fall in PCV in affected animals is inspite of the fact that a high degree of spherocytosis has been reported in PPH by Rana (1987) and Singh (1989) which should have at least partially compensated the decrease in PCV due to loss of erythocytes. Low levels of PCV in this disease have also been reported by Gautam *et al.* (1972), Singh (1989) and Gahlawat (1998).

During the present investigation, the serum inorganic phosphorus in diseased animals was found to be significantly low (1.72 ± 0.23 mg%) as compared with the levels in village healthy buffaloes (3.85 ± 0.67 mg%). There has been a steady stream of reports of hypophosphataemia in this disease in buffaloes (Nagpal *et al.*, 1968, Malik and Gautam, 1971, Chugh *et al.*, 1986, Sridhar, 1994, Gahlawat, 1998). The findings of present study are in conformity with these reports.

In the present study, the administration of sodium acid phosphate 80 gm i/v as 20% solution and 80 gm orally once daily resulted in raising the Pi levels from 1.72 ± 0.23 mg% to 2.88 ± 0.38 mg% in four days in PPH buffaloes (Table 2). This showed that the adopted therapy was a success in buffaloes. Though the role of phosphorus deficiency in PPH buffaloes had been recognized long back and the therapy with sodium acid phosphate has been in practice, yet the extent of restoration of serum phosphorus levels

Table 1

Means levels of haemoglobin (g%), packed cell volume (%) and serum inorganic phosphorus (mg%) in farm healthy, village healthy and haemoglobinuric buffaloes

Category of animals		Hb (g%)	PCV	Pi
Farm healthy	Untreated	12.23^{a}	33.60^{a}	3.98^{a}
Village healthy	Untreated	11.91^{a}	29.67^{b}	3.85^{a}
Group I	Pre-treatment	07.09^{b}	20.60^{c}	1.72^{b}
	Post-treatment	05.72^{c}	17.94^{c}	2.88^{c}
Group II	Pre-treatment	07.16^{b}	20.74^{c}	1.71^{b}
	Post-treatment	05.62^{c}	18.14^{c}	2.90^{c}

Means with different superscripts within coulmn differ significantly ($P < 0.05$).

Table 2

Mean levels of haemoglobin (g%), packed cell volume (%) and serum inorganic phosphorus (mg%) in farm healthy, village healthy and haemoglobinuric buffaloes

Parameter	Group	Day 0	Day 1	Day 2	Day 3	Mean
Hb	Group 1	07.09	06.62	05.87	05.72	06.33
	Group II	07.16	06.53	05.78	05.62	06.24
	Mean	07.13^{a}	06.57^{b}	05.83^{c}	05.67^{c}	
PCV	Group 1	20.60	19.04	16.94	17.94	18.63
	Group II	20.74	18.87	17.60	18.14	18.84
	Mean	20.67^{a}	18.95^{b}	17.27^{c}	18.03^{bc}	
Pi	Group 1	01.72	01.93	02.39	02.88	02.24
	Group II	01.71	01.82	02.29	02.90	02.18
	Mean	01.71^{a}	01.90^{b}	02.34^{c}	02.89^{d}	

Means with different superscripts within row differ significantly ($P < 0.05$).

following this therapy had not yet been extensively studied. Wang *et al.* (1985) concluded that dietary phosphorus deficiency leading to hypophosphataemia, may be a mechanism of post parturient and related syndromes of haemoglobinuria by decreasing red cell glycolysis, altered red cell structure and function, a loss of normal deformability and increase in fragility and haemolysis with resultant haemoglobinuria.

It can be inferred from the study that when serum Pi reaches within normal range, haemolysis start decreasing and the animals recovered from the haemolytic syndrome depending upon the loss which has already occurred. Mean serum Pi levels were 1.72 ± 0.23 mg% when the PPH buffaloes brought to our notice and on the day of complete recovery was 2.88 ± 0.38 mg% suggesting inorganic phosphorus deficiency as the main cause of erythrolysis in this disease and sodium acid phosphate is the best suited therapy. Similarly, in group II buffaloes which received vitamin C along with sodium acid phosphate as therapy, had mean Pi levels on 0 h as 1.71± 0.29 mg% which increased to 2.90 ± 0.38 mg% after 72 h of treatment.

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