Bacillary and post parturient hemoglobinuria biology essay

Post parturient hemoglobinuria is a sporadic disease of multiparous, high producing dairy cows and buffaloes characterized by red blood cells breakdown in the muscles, hemoglobinuria and anemia. The prevalence of the disease in the overall cattle population is very little with a case fatality rate ranging from 10 to 50% (1). Parturient hemoglobinuria was formerly known as milk fever complex, post- parturient hemoglobinuria, puerperal hemoglobinuria and nutritional hemoglobinuria, etc.

History

Parturient hemoglobinuria was first reported in 1939 in buffaloes in the Indian sub-continent in the Lyallpur district, now Faisalabad (Aslam and Haq, 1967). The first known reference of its occurrence and possible cause in Pakistan was by Hussain (1955). Subsequent reviews on the issue recommended that phosphorus insufficiency may be a possible reason of the disease (Pirzada et al., 1989; Pirzada and Ali, 1990). However, so far its true etiology remains doubtful.

Etiology

Consequent papers concluded that PPH is neither infectious nor contagious derived from negative serological and bacteriological verification for pathogenic bacteria and failure to identify erythrocyte parasites (1, 6, 7, 8, 9, 10). Different causes, e. g. protein and mineral insufficiency, Saponin from cruciferous plants, competition for mineral assimilation and, hypophosphataemia because of phosphorus insufficiency. Pirzada et al. (1989) and Cheema et al. (1980) reported that no contributory pathogen could be secluded from the affected animals. Copper insufficiency has been recommended as a possible cause of PPH by personnel in New Zealand.

animals from dairy farms with a high occurrence of PPH had low levels of copper in serum and liver (12). A disease in Ontario named as “ red water” was linked with several predisposing factors which included: a) fresh parturition, b) intense milk production, c) dietetic phosphorus insufficiency and d) eating of turnips, rape, kale, green alfalfa and sugar beet pulp (13, 14, 15). In addition, many cows were hypophosphataemia (1, 16, and 17).

Susceptible animals

A first round report recommended that parturient hemoglobinuria frequently affected high producing buffalo cows in the area of the Punjab province of Pakistan wherever there is a insufficiency of minerals, chiefly in the districts of Faisalabad, Jhelum, Attock and Rawalpindi. At these points soil tended to be deprived in minerals and buffaloes were affected more than cattle (Husain, 1955). The incidence of this syndrome in bulls, cows prior to parturation, heifers under two years old, or beef cows is remarkable but has been reported (23, 22). A disease with many similarities has been described in sheep (13), Egyptian and Indian buffaloes (24, 16) and a goat (17).

Clinical signs

Clinical signs of syndrome can bee seen 20+,-10 days, before or after parturition at what time affected Animals pass red to coffee colored urine and rectal temperature ranges from 38. 38C to 39. 48C, (Pirzada et al., 1989). On the other hand, with the advancement of disease, rectal temperature declines. Additional clinical signs are jaundice, anemia, recumbency, in appetence, labored breathing, and constipation (Razz et al., 1988). Cheema et al. (1980) reported that there was a considerable increase in the rectal temperature of affected animals. The disease affects buffaloes

more than cattle (Pirzada et al., 1989) which is a judgment in close agreement with that of Raz et al. (1988). Pirzada et al. (1989) additional observed that phosphorus administration yielded effective outcome suggestive of hypophosphataemia as a causal issue and that, in the affected areas from August to January, animals in advanced stages of pregnancy should be supplemented with dicalcium phosphate or bone meal which should contain 13% and 17% phosphorus correspondingly (Amir and Kripsch-eer, 1989). This agreed with the findings of Akram et al. (1990) and Raz et al. (1988). Earlier, Sadiq et al. (1965) found that even though cases of parturient hemoglobinuria occurred all the way through the year, the occurrence increased in winter. On the other hand, Cheema et al. (1980) reported that frequently stall-fed animals were affected, and that most cases were occurring in July and September. The acute disease (three to five days) can come to an end in death or be followed by extended convalescence (two to eight weeks). Gangrene and sloughing of the extremities are reported sequelae. Recovered animals get back their former body condition and milk production gradually.

Diagnosis

It can On the basis of clinical signs and history Urinalysis can be helpful in the diagnosis of this syndrome. Microscopic inspection of the urine sediment is imperative to distinguish hematuria from hemoglobinuria.

Treatment

The recommended treatment for Post parturient hemoglobinuria in North America includes: 1) intravenous infusion of sodium acid phosphate (60 g in 300 mL of water), 2) 100 g of bone meal administered as a drench two times

a day, 3) transfusion of fresh blood as needed and 4) intravenous fluids to sustain hydration (1, 29, 30). Improvement of any phosphorus insufficiency or disproportion in the ration together with removal of incriminated feeds might prevent further cases. Because of incompatible results with phosphate therapy and the copper-deficient status of affected cows, personnel in New Zealand recommend parenteral copper (120 mg available copper per cow) as the favored treatment (31).

A study on treatment trials of hemoglobinuria in buffalo cows and cattle indicated that subsequent treatment with sodium acid phosphate (20% sol) administered concurrently by i. v., s. c. and oral routes, approximately 100% of affected animals recovered in 1-3 days (Shah et al., 1988). Blood transfusion also shows a good result in this regard (Rauf, 1989; Raz et al., 1991). A blood coagulant Botrophase prepared from the venom of the snake Bothrops jararaca, seems to have anti fibrinolytic action and was productively used to treat buffaloes facing parturient hemoglobinuria (Goel et al., 1988). Parenteral copper (120 mg available copper per cow) as the favored treatment (31).

Treatment with toldimfos sodium and tea leaves and sodium acid phosphate

Result of this treatment was based on the recovery of urine discoloration; the efficacy of toldimfos sodium was 85% followed by tea leaves 56%, and sodium acid phosphate 18%. (Zameer et. al 2010). Fallowing treatment with tae leaves and toldimofos sodium urine was clear next day, and with treatment with sodium acid phosphate urine was clear on third dy. (Zameer et. al 2010)

Prevention

Parenteral administration of copper has been effectual in dairy herds with previous histories of PPH (36, 37). For prevention of the disease, supplementation with dicalcium phosphate has been suggested (Pirzada et al., 1989; Pirzada and Ali, 1990). The occurrence of PPH was considerably lower (5. 18% versus 25. 51%; P < 0. 01) in cows treated with copper previous to calving. moreover, a top dressing of copper sulfate to pastures four months previous to calving was followed by an increase in pasture, blood and liver concentrations of copper and a noticeable decrease in the occurrence of Post parturient hemoglobinuria. Pick up the soil phosphorus level by using phosphorus fertilizers for the reason that a study in India shows the connection between soil and this disease. (Nag pal et al., 1968) One study which was conducted by Mohamed and El-Bagoury (1990), shows that there is a connection between berseem feeding and parturient hemoglobinuria for the reason that berseem is deficient in phosphorus thus avoid high quantity berseem feeding by feeding with some other fodders. Berseem also has Saponin which causes hemolysis causing hemoglobinuria. (Mohamed, 1980 Mohamed et al., 1988).