Post parturient hemoglobinuria in cattle

Etiology:

Diets low in phosphorus or UN supplemented with phosphorus are usually associated with the disease in dairy cattle.

The feeding of cruciferous plants has been associated with the disease, but many cases occur unassociated with such diets and their role as a cause is uncertain.

The current hypothesis is that ingested hemolytic agents, one of them identified for example in rape, some of them not, cause erythrocyte lysis in some circumstances.

Epidemiology

Only adult cows develop the typical hemolytic syndrome, usually in the period 2-4 weeks after calving.

High – producing dairy cows in their third to sixth lactations are most commonly affected.

The disease does not occur commonly in beef cattle.

Phosphorus – deficient soils and drought conditions are considered predisposing causes, and the disease is often a problem on particular farms.

In areas of severe phosphorus deficiency the diseases occur at pasture. It is more common during prolong4ed periods of housing.

Although this disease has been observed in many countries its relatively low incidence makes it a minor disease.

The case – fatality rate may be high as 50% but only one or two animals in a herd are affected at a time.

Cases may also occur when cows graze rape, turnips, or other cruciferous plant or when large quantities of beet pulp are fed. These diets are normally low in phosphorus.

The ingestion of cold water or exposure to extremely cold weather may precipitate an episode of hemoglobinuria.

A similar condition accompanies by hypophostaemia has been observed in late pregnancy in Egyptian buffalo and in the postpaturient period in Indian buffalo.

Pathogenesis:

There is an association with hypophosphatemias and a low dietary intake of phosphorus, and it is presumed that the drain of lactation causes further depletion of phosphorus reserves.

The dependence of mammalian red blood cells on glucose metabolism for the main source of energy for viable function and structure makes them highly vulnerable to factors inhibitory to the glycolytic pathways.

Hypophosphatemia results in a decrease in red blood cell glycolysis and adenosine triphosphare CATP synthesis .

Sub normal concentrations of ATP predispose red blood cells to altered function and structure, a loss of normal deformability, and an increase inn fragility and hemolysis with result ant hemoglobinemia and hemoglobinuria.

The changes in the red blood cells are irreversible and the life span of the cells is probably diminished because they are unable to regain their previous structure and function.

Copper and selenium may be important because they are commonly deficient in feed stuffs. Both copper and selenium may also provide some protection against the effects of orally acquired hemolytic agents in cruciferous plants.

The clinical findings are those of acute hemolytic anemia and in fatal cases death is due to anemic anoxia.

Clinical findings:

Hemoglobinuria, in appetence and weakness develop suddenly and there is a severe depression of the milk yield.

Although in some less acute cases, the cow continues to eat and milk normally for 24 h after discoloration of the urine ids evident.

Dehydration develops quickly, the mucous membranes are pallid, and the cardiac impulse and jugular pulse are much augmented.

A moderate temperature rise (40 oc) often occurs.

The feces are usually dry and firm, dyspnea may be obvious and tachycardia is common. Jaundice may be apparent in the late stages.

Pica ay is observed in the other animals in the group.

The course of the acute disease extends from 3 – 5 d.

The cow becomes weak and staggery and finally recumbent.

Gangrene and sloughing of the tip of the tail or the digits has been observed occasionally.

Death may occur within a few days.

In non – fatal cases, convalescence requires about 3 weeks and recovering animals often show pica.

Ketosis commonly occurs coincidentally

In a herd where the disease occurs, there may be additional signs of phosphorus deficiency, although when the deficiency is marginal the general conditions of the herd may be excellent.

Clinical pathology

In marginal phosphorus deficiency areas normal non – lactating animals in an affected herd may have serum inorganic phosphorus levels within the normal range.

Lactating cows in an affected herd may have moderately low levels of 2-3 mg / dl and affected animal's extremely low levels of 0.4 – 1.5 mg / dl.

Erythrocyte counts and hemoglobin levels are also greatly reduced.

Heinz bodies may be present in erythrocytes.

The urine is dark red – brown to black in color and usually moderately turbid.

A low copper status of the blood and liver of affected cows and the pasture grazed is also recorded.

Differential diagnosis

Post parturient hemoglobinuria is characterized by an acute hemolytic anemia in cows calved within the preceding 4 weeks.

Other causes of acute hemolytic anemia are not confined to the post calving period.

Laboratory examination to confirm the diagnosis.

Treatment

A transfusion of whole blood is indicated in severe cases.

A delay of 12 h often seems to lead to an irreversible state.

A minimum of 5 l. of blood to 450 kg cow is recommended.

This will usually suffice for up to 48 h by which time an additional transfusion may be necessary if the cow is weak and the mucous membranes pale.

Follow blood transfusion, fluid therapy is recommended as both supportive therapy and to minimize the danger of hemoglobinuric nephrosis.

The administration of phosphorus to acutely ill animas should include the IV administration of 60 g of sodium acid phosphate in 300 ml of distill water and a similar dose SC , followed by further SC injections at 12 hourly intervals on three occasions and similar daily doses by mouth .

Oral dosing with bone meal (120 g twice daily) or dl calcium phosphate or a suitable source of calcium and phosphorus daily for 5 d, is recommended followed by inclusion in the ration

Hematinics during convalescence are recommended.