JUST AGRICULTURE
multidisciplinary e-Newsletter

Diagnosis and Treatment of Post Paturient Hemoglobinuria

Chaithra S N, Manish Arya and Aakanksha
PhD Scholar, Division of Surgery, ICAR- Indian Veterinary Research Institute Izatnagar, Bareilly, UP – 243122

ARTICLE ID: 15

Abstract

Postparturient haemoglobinuria is a metabolic disease of dairy cows, it is more commonly observed in cows at postpartum. It is characterized by haemolysis, haemoglobinuria and anaemia. Occurs due to the phosphorus deficiency in diet mainly and shows clinical signs such as haemoglobinuria, inappetence, anaemia, weakness develop suddenly, and severe depression of milk yield. Diagnosis can be made by history, clinical signs and laboratory findings. Treatment includes blood transfusion, fluid therapy, intravenous sodium acid phosphate injection and dietary supplementation of dicalcium phosphate.

Introduction

- Postparturient haemoglobinuria of dairy cows is characterized by intravascular haemolysis, haemoglobinuria & anaemia.
- Also called as nutritional haemoglobinuria.

Etiology

- Diet deficient in phosphorus.
- Feeding of cruciferous plants.
- Cu and Se deficiency.

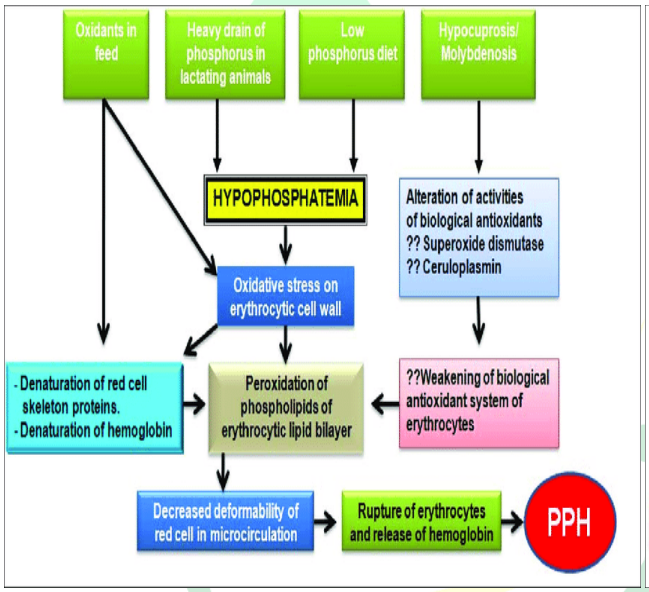
Epidemiology

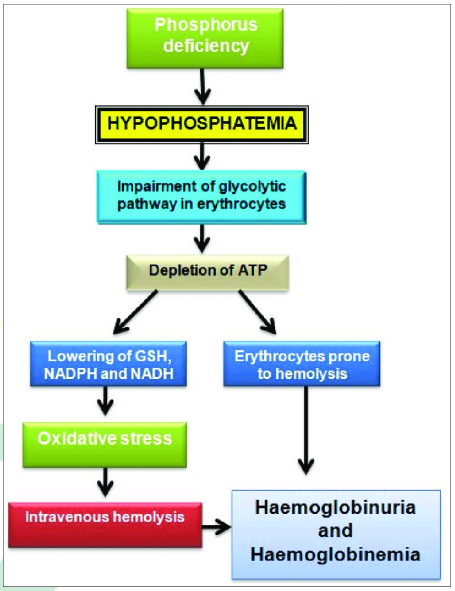
- Only adult cows develop the typical haemolytic syndrome, usually in the period 2-4 wks after calving.
- Phosphorus deficient soils & drought conditions.
- When the cows graze rape, turnips or other cruciferous plants or when large quantities of beet pulp are fed.
- Copper deficiency - excessive application of Mo fertilizer & lime to pasture.
- The ingestion of cold water or exposure to extremely cold weather.

JUST AGRICULTURE
multidisciplinary e-Newsletter

Pathogenesis

- The reason for the sudden development of intravascular haemolysis is unknown.
- The haemolytic factors present in some diets may produce oxidative damage to Hb.
- Erythrocytic fragility is not increased & haemolysis does not always occur in cases of hypophosphatemia.
- Hypophosphatemia is directly related to low phosphorus intake and lot of phosphorus drained during lactation.
- Hypophosphatemia will result in decreased RBC and ATP synthesis.

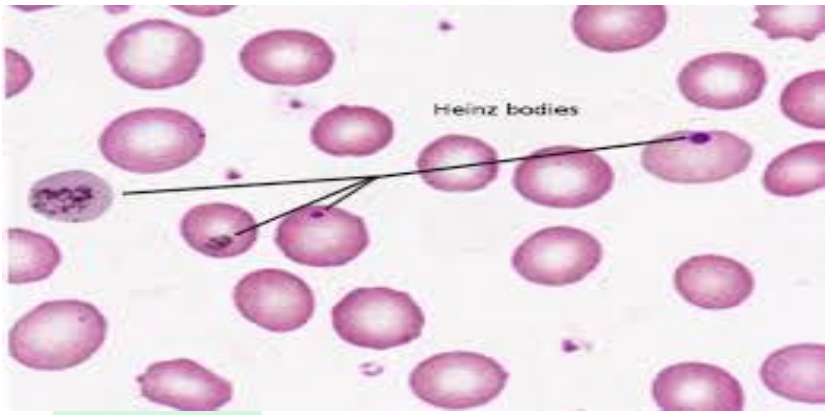




- Decreased ATP predisposes RBC to alter function and normal deformability, which leads to increased fragility of RBC.
- Haemolysis haemoglobinemia haemoglobinuria.
- Haemolytic anaemia of kale poisoning: s-methyl cysteine sulfoxide is converted in the rumen into secondary haemolysin dimethyl disulfide.
- The onset of haemolytic crisis is indicated by increased Heinz body counts & erythrocyte osmotic fragility and decreased Hb.
- Heinz-body anaemia - main feature of postparturient haemoglobinuria associated with Mo induced Cu deficiency.
- Heinz bodies are breakdown products formed as a result of oxidative damage to Hb that bind to the erythrocyte membranes.
- Macrocytic hypochromic anaemia occur following depletion of copper reserves.

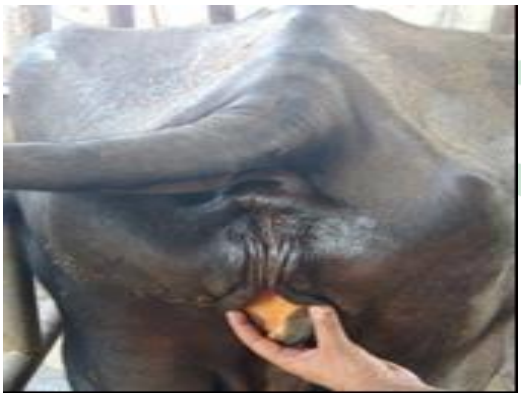
JUST AGRICULTURE
multidisciplinary e-Newsletter

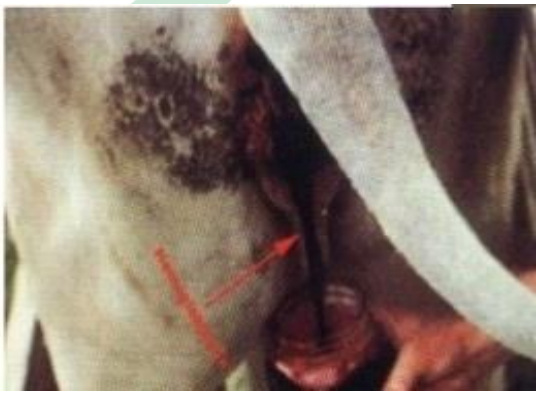
- These changes in RBC are irreversible and rigid cell wall gives rise to reduced lifespan of RBC.



Clinical signs

- It runs an acute course of 3-5 days.
- Haemoglobinuria, inappetence, anaemia & weakness develop suddenly.
- Severe depression of milk yield.
- In less acute cases, the cow continues to eat & milk normally for 24 hours after discolouration of the urine is evident.
- In severe cases, H/R increased (50-130beats/min), milk production ceases.
- jugular pulse more pronounced.
- R/R increased, rectal temperature increased (103.5^0 C).
- lose weight & dehydrated.
- Visible mucus membrane is pale & anemic.
- Gangrene & sloughing of the teats, the tip of the tail & digits.





- Intercurrent disease - Mastitis & forced exercise is often associated with fatal diseases.

JUST AGRICULTURE
multidisciplinary e-Newsletter

- Non fatal disease - ketosis & convalescence takes from 3 wks to 2 months.

Haemoglobinuria

Recumbent stage

Clinical pathology

- Haematological examination: Heinz bodies may be demonstrated, low Hb value(<8gm /dL) and low RBC count (<5*10^6/micro litre).
- In phosphorus deficient areas, lactating cows have low serum concentrations of inorganic phosphate (2-3mg/dL) and in extreme situations it can drop up to 0.4-1.5 mg/dL.
- Normal phosphorus level in cattle is 5.6 – 8 mg/dL.
- When Cu deficiency - blood Cu concentration is less than 30 microgram/dL. Liver Cu concentration is 5mg/kg on a DM basis.
- Dark red-brown to black colour urine with moderate turbidity.
- Elevated serum bilirubin & blood urea.

Necropsy findings

- Jaundiced condition of the carcass.
- Pale swollen liver with centrilobular necrosis.
- Haemoglobinuric nephrosis of the kidney.
- Discoloured urine is present in the bladder.

Diagnosis

- By history
- By clinical signs
- By clinical pathology
- laboratory examination

Urinalysis: +ve for Hb and albumin.

JUST AGRICULTURE
multidisciplinary e-Newsletter

Haematology: decreased Hb, PCV & RBC

Biochemical: Hypophosphatemia, hypocupremia and increased molybdenum in few cases.

Differential diagnosis

- Leptospirosis
- Babesiosis
- Bacillary haemoglobinuria
- Anaplasmosis
- Cu poisoning
- Cold water haemolytic anaemia of calves
- Pyelonephritis and cystitis
- Myoglobinuria
- Some plants contain phenolic compounds. These plants eaten by cows leads to the formation of red, brown or black urine.

Treatment

- Whole blood transfusion: a minimum of 5 lts of blood to a 450kg animal is recommended.
- Fluid therapy: as both supportive and specific therapy after blood transfusion to prevent kidney damage andto minimize the danger of haemoglobinuric nephrosis.
- I/V injection of 60gm of sodium acid phosphate in 300ml of sterile distilled water & a similar dose S/C followed by further injections at 12hrs intervals for 3 treatments, that animal drenched with 30gm sodium acid phosphate.
- 120gm of bone meal orally twice daily or Dicalcium phosphate administered orally for 5 days.
- Haematinics
- Ca hypophosphite (30gm in 100ml of 10% glucose) has been used to treat affected cows.
- Glucorticoids (20mg dexamethasone I/M) & give oral treatment for ketosis.
- Cu deficient areas, cows are given Cu glycinate S/C to provide 120mg available Cu.
- Cruciferous plants should be removed from the diet, the cows should be fed good quality hay.

JUST AGRICULTURE
multidisciplinary e-Newsletter

Prevention and control

- Supplementation with Na acid phosphate (30gm/cow/day), bone meal / bone meal licks(100gm/cow/day).
- Cu - deficient areas, pastures can be top dressed with Cu or cows can be given copper glycinate S/C to provide 120mg of available Cu in the month before parturition.
- Restrict the intake of cruciferous plants.
- Adequate phosphorus intake in early lactation.

Reference

- Veterinary Medicine – Otto M Radostits, Clive C Gay, Kenneth W Hinchcliff, Peter D Constable (10th EDITION)
- Veterinary Medicine –D C Blood (6th EDITION)
- Current Veterinary Therapy-[food animal practice-(vol-2)]-Jimmy L Howard