Haemoglobinuria in Dairy Animals

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Haemoglobinuria is a common presenting clinical sign in dairy animals of all age groups. Haemoglobinuria refers to presence of free hemoglobin in urine. Invariably the cause is excessive intravascular hemolysis. However the cause of intravascular hemolysis vary according to age and species in bovines. For rational treatment the underlying cause has to be identified. This document focuses experience on the diagnostic approach to be adopted for rational treatment and experience on the treatments in vogue.

CAUSES:

Causes	Susceptibility	Occurrence
1. Infectious disease		
● Babesiosis	Adult cattle Buffaloes	Common Very rare
● Leptospirosis	Cattle-buffalo	Not common
● Bacillary haemoglobinuria	Adult bovines	Not common
2. Nutritional		
● Phosphorus and/or Copper deficieny	Adult buffaloes and cattle	Common
● Water intoxication	Calves	Common
3. Poisoning		
● Copper	All ages	Rare
● Plants (Barseem, Onion, Brassica family, Tumip, Cabbage, Beets)	All ages	Less common
4. Drugs		
● Intravenous infusion of undiluted oxytetracycline	Cattle	Very rare
● NSAIDS	Cattle	Very rare

Diagnostic approach: For confirmatory diagnosis a minimum database is required which include examination of blood smear, plasma inorganic phosphorus and physical and chemical examination of urine. The susceptibility varies according to:

Signalment: species and age

Clinical signs: differentiate febrile and non febrile diseases. Observe for anemia and jaundice.

Differentiate between haemoglobinuria, hematuria and myoglobinuria. In some cases color of urine may be helpful. Bright red color suggests hematuria. In case of confusion microscopic examination of urine should be carried out. Brown or brown black color is indicative of haemoglobinuria, myoglobinuria. For differentiation of myoglobinuria chemical examination is essential. Add 10g of ammonium sulphate to 10ml of urine and shake. Disappearance of color suggests haemoglobinuria.

Babesiosis:

Babesiosis is blood protozoan infection often observed in cross bred cattle and very rarely in buffaloes. Local breeds are generally resistant to this disease. Calves below 6 months are resistant to infection. Even if calves are infected disease is mild and short lived. Sudden onset of moderate to high fever (104-106^{0}F) along with attendant signs of anorexia, depression, loss of rumination are the early signs. Later on, pallor mucosae and dark red to brown red or deep brown colored urine are the prominent signs. Terminally severe jaundice is evident. Many severely affected animals die precipitously within 24 hours.

Treatment: effective drugs are available. Diminazine aceturate (Berenil) is effective, well tolerated safe drug. Sterilization with this drug may not be complete. Therapeutic dose is 3.5 mg/kg deep intra muscularly.

Imidocarb propionate is effective @ 1mg/kg body weight subcutaneously. It also maintains residual activity for few weeks.

Disease is generally acute and if treatment is delayed animal may succumb to anemia inspite of elimination of infection. Blood transfusion is essential in severely anemic animals.

Nutritional haemoglobinuria:

This is associated with low blood phosphorus levels. A prolonged hypophosphatemia is a major predisposing factor. Concurrent copper deficiency potentiates this disease. Buffaloes are more susceptible to this condition. Often disease appears during lactation and sometimes in late pregnancy.

Haemoglobinuria is the first sign. Urine color ranges from deep brown to black frothy. Animal lack other systemic signs and illness goes unnoticed till anemia becomes profound. At this stage appetite and milk production is reduced. Some animals show signs of severe constipation and strain to defecate. Terminally recumbancy occurs and on examination mucous membranes are pale icteric. These are bad prognostic signs. Diagnosis can be confirmed by determining plasma inorganic phosphorus (Pi). Plasma Pi is below 3.0 mg/dl.

Specific therapy is intravenous administration of 60g of sodium hydrogen ortho phosphate ($NaH_2PO_4.2H_2O$) dissolved in 5% dextrose solution. Treatment is continued at 24 hour interval unlit abnormal urine color disappears. Concomitantly oral phosphorus supplementation in the form of dicalcium phosphate/ mineral mixture @ 60-80g till recovery. To minimize the risk of haemoglobinuric nephrosis fluid therapy with normal saline is advisable till recovery. These are studies which substantiate that recovery can be enhanced by simultaneous intra venous administration of copper glycinate (120 mg) in fluid. Intravenous administration of 40-50 ml of 10% ascorbic acid also reduces recovery period. There are reports that ascorbic acid is effective in treating less severe cases. Blood transfusion required in severely anemic cases @ 10-15 ml/kg body weight.

Haemoglobinuria due to plant toxicities is generally less severe. Intravenous infusion of ascorbic acid is treatment of choice.

Bacterial causes: Leptospirosis and bacillary haemoglobinuria are the outstanding causes. Acute septicemic form of Leptospirosis characterized by causing intra vascular hemolysis, haemoglobinuria and fever. Primarily effect calves during first month of age. Clinical signs of septicemia are high fever, and petechiation of mucosa accompanied by signs of intravascular hemolysis.

Adults suffer from sub acute and chronic form. Only sub acute form may produce intravascular hemolysis but of less severity. A consistent accompanying sign is blood stained milk with any physical change in udder. Abortion in adult pregnant females may

be another evidence of Leptospirosis. Treatment of Leptospirosis is best done by streptomycin @ 12mg/kg BID for 3-5 days. For complete sterilization single injection @ 25mg/kg is recommended.

Bacillary haemoglobinuria: very acute, toxemic and endemic disease characterized by diarrhea, fever, abdominal pain and sudden death. Differentiation is done by blood film examination. Feces are dark brown or there may be diarrhea with much mucous and some blood. Differentiation from Leptospirosis clinically is impossible. Specific therapy includes use of penicillin @ 20,000 to 30,000 units/kg or tetracycline @ 10-15 mg/kg body weight.

Paroxysmal haemoglobinuria:

It is a common condition observed in calves of 2-10 months of age. Often the sign appear few hours after drinking water.

Clinical signs may vary and in most of the cases haemoglobinuria is the only clinical sign. Animal recover in 3-4 hours. In more severe form, nervous signs may accompany. There is hyperesthesia, muscular tremors, nystagmus and lethargy. Very rarely calves may develop hypothermia, edema of eyelids, arrhythmia and ruminal tympany. Diagnosis is based on age, history of haemoglobinuria after water intake. Treatment with hypertonic saline (5%) @ 0.5-1ml/kg i.v. may be used in mild to moderate form of cases. In severe cases combination of 5% dextrose and hypertonic saline achieve better response. If required tranquilizers may be used.

Diseases causing Hematuria

1. Pyelonephritis
2. Idiopathic hematuria
3. Enzootic bovine hematuria

Pyelonephritis: Refers to purulent inflammation of urinary bladder, ureters and renal pelvis. Clinical presentation varies from case to case. Passing of blood stained urine may be only early presenting sign. In other animals episodic colic along with painful frequent urination may be present. Rectal examination may reveal enlargement of ureter and kidneys with loss of lobulation (pyuria, hematuria).

Confirmation of pyuria, hematuria on urine analysis is sufficient for diagnosis. Treatment with antibiotics viz. ampicillin, potentiated sulphonamides, penicillin for 7-10

days is effective. Penicillin in high doses (15000 u/kg) is the preferred drug.

Idiopathic hematuria: occurrence is most common in crossbred cattle and often appears in recurrent form. There are no systemic effect except for mild reduction in milk production and weight loss. Blood examination show mild to moderate anemia. Treatment is difficult however response to homeopathic treatment is possible in 50 per cent of cases.

Enzootic bovine hematuria: It is a region specific disease often occurring in hilly areas where moist conditions persist. Likely cause is ingestion of bracken fern. It mostly occurs in late summer when other feed is scarce or when animals are fed hay containing bracken fern. Poisoning requires prolonged exposure (several weeks to months). Affected cattle suffer intermittent hematuria and anemia. Affected cattle are weak, rapidly lose weight and develop pyrexia (106^0F). Once animals develop clinical diseases, poisoning is almost always fatal.