Post Parturient Hemoglobinuria of Cows Hemoglobinuria in Buffaloes (Red Water)

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Fig. 3: Red colour urine on 3rd day of treatment

Definition

It is a metabolic hemolytic disease of cattle and buffaloes characterized clinically by:

- ❖ hemoglobinuria
- ❖anemia
- ❖biochemically by **intravascular hemolysis and hypophosphatemia.**

Incidence and occurrence

(1) It is usually **occur 2-4 weeks after parturition** in cows and in mid-term of gestation in buffaloes.
(2) Animals are susceptible between fifth and tenth calving.

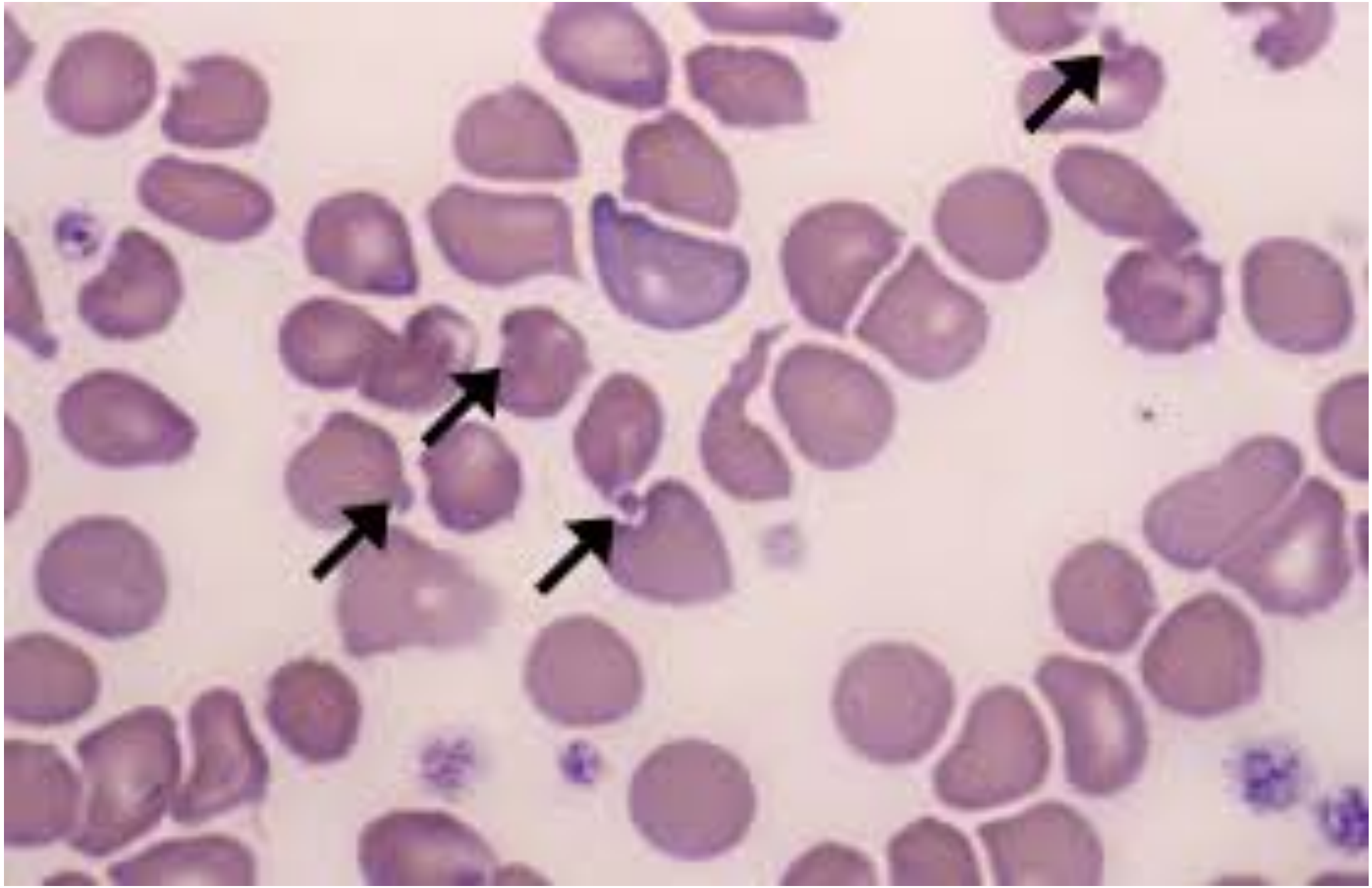
Causes

(1) Diets **low in phosphorus or un supplemented with ph.**
(2) Feeding of **cruciferous plants** such as **Berseem, grass, turnips, kale** (contain thiocyanites, nitrates and sulphoxides as toxins) and carbage (contains thiouracil as toxins).

Pathogenesis

(1) The cruciferous plants may be rich in Ca and poor in ph so excessive Ca needs more ph to metabolism and excreted (in urine and feces) resulting in ph deficiency.

(2) The toxins of these plants cause irreversible oxidative changes in hemoglobin leading to the formation of Heinz bodies in the RBCs. As soon as Heinz bodies are formed, the erythrocytes become foreign to the circulatory system.



These foreign RBC are carried to the spleen where they are erythrophagocytosed by the spleen and other reticuloendothelial system.

So, increased destruction of RBC leads to

- ❖hemolytic anemia
- ❖hemoglobinuria.

- Some of the sulphoxides as S-alkyl-crystalline sulphoxid are broken down in the rumen by bacterial lyase to form dimethyl sulphide which is the acute cause for hemolysis of R.B.C.
- likewise, nitrate is broken down in the rumen to nitrite which cause oxidative hemolysis. In nitrite poisoning there is also formation of Heinz bodies.

(3) Phosphorus deficiency results in:

A. Decrease of RBC glycolysis (due to a decrease in glucose -6-phosphate dehydrogenase).

B. Decrease of adenosine triphosphate synthesis (ATP).

C. Decrease of phospholipid in the wall of RBC.

The previous points result in loss of normal RBC deformability, increase its fragility and IV hemolysis resulting in hemoglobinemia and hemoglobinuria.

(4) Copper and selenium provide some protection against the effect of orally hemolytic agents in cruciferous plants (previous toxins) so **that copper and selenium deficiency may be a predisposing cause of ph deficiency.**

(5) The clinical findings are those of acute hemolytic anemia and in fatal cases death occurs due to anemic anoxia.

Clinical findings

(1) Color of urine may vary from **light brown in mild cases to deep red or coffee colored in severe cases.**

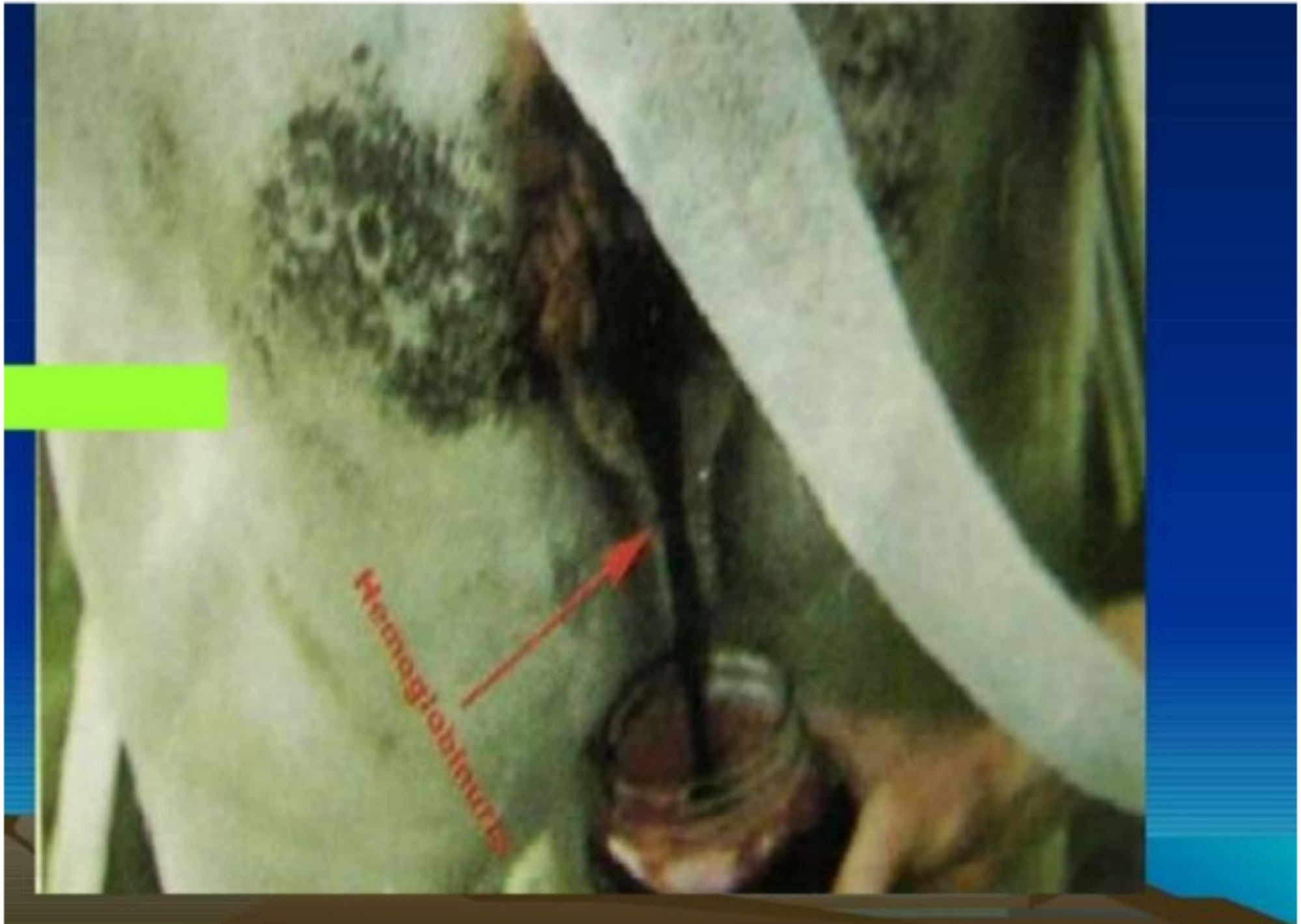
(2) **Inappetence, pica, dull, depression and weakness** develop suddenly.

(3) Severe depression of milk yield (although in some less cases, the cow continues to eat and milk normally for 24 hours after **discoloration of the urine**. Milk may be yellowish or reddish in color.

(4) Dehydration develops quickly.

(5) The mucous membranes are pale and anemic, the cardiac impulse and jugular pulse are much augmented with tachycardia.

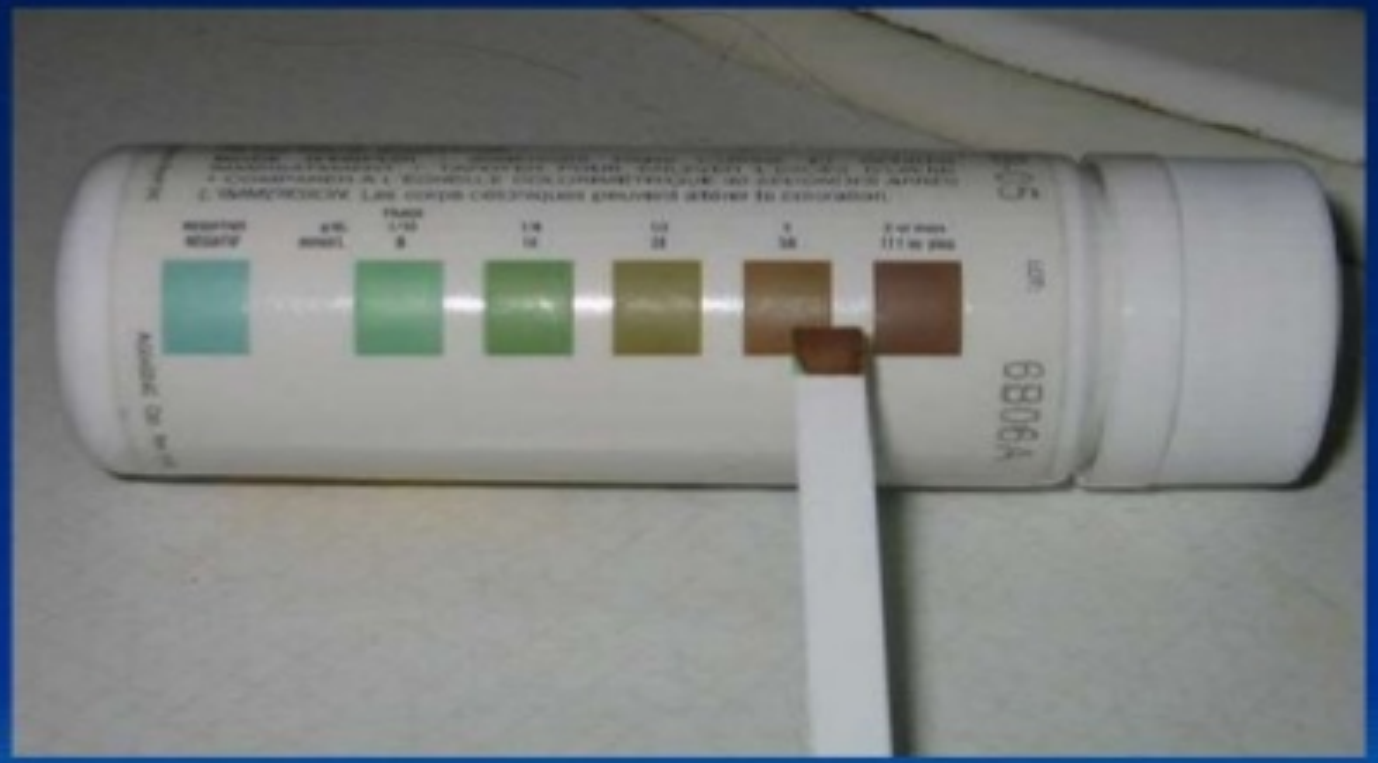
(6) Cessation of rumination and constipation.
(7) The feces are usually dry and firm.
(8) Dyspnea due to anemic anoxia.
(9) Hemolytic jaundice may be apparent in the late stages.
(10) The course of the acute disease extends from 3-5 days, the cow becomes weak and stagger and finally recumbent (Downer).
(11) Gangrene and sloughing of the tips of the tail or teat or ear or the digits.
(12) Ketosis commonly occurs.
(13) Death may occur within a few hours or days, in non-fatal cases, convalescence requires about 3 weeks and recovering animals often show pica.


Hemoglobinuria

Diagnosis

(1) History (pregnancy in buffaloes, parturition in cows or green season).
(2) Clinical symptoms (hemoglobinuria, hemolytic anemia, dehydration, pica).
(3) Laboratory:

A. Serum inorganic phosphorus decreased from 4-7 (normal) to 2-3 or even to 0.4-1.5 mg/dl.
B. RBC dropped from 5.5-6.0 (normal) to 2 million /c.mm with presence of Heinz bodies in RBC.
C. Low copper level of the blood and liver of affected' cows.
D. PCV dropped from 25-35 (normal) to 25%.
E. The urine is dark, red-brown to black in color and usually moderately turbid without RBC.
F. Benzidine test is positive blue for detection of blood in urine.
G. Centrifugation of urine sample or leave urine sample in test tube for long period. No changes occur (Hemoglobinuria).



- Urine strip Dipstick is positive for Hb

- **Necropsy findings**
- (1) The blood is thin, Icterus is widespread in the body.
- (2) The liver is swollen and fatty infiltration and degeneration.
- (3) Discolored urine is present in the bladder.

Differential diagnosis

(1) Leptospirosis: All ages of cattle are affected. There is acute fever, anemia, red colored milk. Abortion, *leptospira* titers and leucopenia. Animal may die in 24-48 hours, response to antibiotic therapy.

(2) Bacillary hemoglobinuria: Acute fever and abdominal pain. No changes in milk, marked hemglobinuria and death in 2-4 days. In anaerobic culture examination Cl. hemolyticum organisms are found, there may be Leucocytosis or leucopenia.

- (3) Babesiosis: History of tick infestation, occur in enzootic form, young animals are mostly susceptible acute in onset, fever, jaundice, abortion, marked hemoglobinuria.
- Course of disease is 2-3 weeks. Identification of organisms in blood smear. Response to specific antiprotozoal drug i.e. Berenil.
- (4) Anaplasmosis: Common in yearling and mature cattle in summer season. No hemoglobinuria; jaundice is common; fever. Blood smear and CFT reveal the organism. Response to specific drug.
- (5) Drug induced such as phenothiazine: No hemolytic anemia. History will detect the case.
- (6) Euzootic hematuria: Sommon in hilly areas. Intact RBC found in the urine. Occur due to certain vesicular changes in bladder (carcinogenic).
- (7) Blood transfusion reaction: Sudden onset, dyspnea, cough, trembling. Responds to adrenaline.
- (8) Pyelonephritis: Hematuria, pus and cast in urine, enlarged kidney.
- (9) Myoglobinuria: occurs in young cattle affected with enzootic nutritional muscular dystrophy and may be confused with hemoglobinurea. History of grain engorgement.
- (10) Chronic copper poisoning: severe jaundice, no fever, hemoglobinurea present, toxic levels of copper in blood, liver and feces.
- (11) Water intoxication: History

- **Treatment**

1. Blood transfusion: 5L of whole blood to a 450 kg cow is indicated in severe cases. This will usually sufficient for up to 48 h then an additional transfusion is necessary if cow is weak and mucous membranes are pale.
2. Sources of phosphorus:

- 1) IV slowly administration of 60 or 80 g of sodium acid phosphate in 300 or 400 ml of distilled water on the first day followed by further SC injections of similar doses at 12-hours intervals till recovery. Similar daily doses (60-80 g) by mouth till 3 days after disappearance of red urine (complete recovery).

2) Oral dosing with bone meal (120 g) twice daily or dicalcium phosphate daily for 5 days or till recovery.
3) In severe cases of red urine, inject also other patent phosphorus preparation as catozal, phospho 2o, tonophosphan 10-15 ml IM or 20-50 ml dissolved in 500 ml glucose slowly IV daily, till recovery.
4) Stop feeding of barseem but add bran to ration together with 100-200 gm Mg sulphate to avoid indigestion.

1. Supportive therapy (glucose 10-20%) to minimize the danger of hemoglobinuric nephritis.
2. Hematinic preparation: iron, copper, cobalt, vitamin B12.

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Control:

(1) An adequate dietary intake of phosphorus.

(2) For pregnant aged buffaloes or lactating senile cows add to their normal ration in rate of 30 gm Na acid phosphate or 60 gm bone meal or daily use of bran.

(3) Copper supplementation in copper deficient area.