BACILLARY HEMOGLOBINURIA

**ETIOLOGY**

Clostridium haemolyticum (C *novyi* type D) is a soil-borne anaerobe.

The organism has been isolated from bones a year after the death of an animal from bacillary hemoglobinuria.

In infected areas the organism is often found in the livers of healthy cattle.

Under anaerobic conditions the organism grows and produces

a necrotoxic and hemolytic toxin that is responsible for the clinical disease.

Damage to the liver from telangiectasis, necrobacillosis caused by Fusobacterium necrophorum, and fascioliasis have been suggested as precipitating causes

The disease has been produced experimentally by infecting calves orally and inducing liver damage by liver biopsy, or by implanting the organism in the liver.

**EPIDEMIOLOGY**

**Occurrence**

Bacillary hemoglobinuria has been reportedin many parts of world.

**Risk factors**

**Animal risk factors**

1-Cattle , sheep and rare cases in pigs

2- animals in good condition are more susceptible.

**Environmental risk factors**

Bacillary hemoglobinuria is a disease of the summer and autumn months. that also are associated with the occurrence of liver fluke

The disease is spread from infected to noninfected areas by flooding, natural drainage, contaminated hay from infected areas, or carrier animals. The carriage of bones or meat by dogs or other carnivores could also effect spread of the infection. Contamination of pasture may occur from feces or from decomposing cadavers.

**PATHOGENESIS**

The disease occurs from the alimentary tract after ingestion of contaminated material. Migrating flukes are the primary factor leading to liver necrosis and the establishment of anaerobic conditions in the liver that will lead to the multiplication of the causative organism.

the bacteria are carried to the liver and lodge there until damage to the parenchyma of the liver and the resulting hypoxia create conditions suitable for their proliferation.

The development of an organized thrombus in a subterminal branch of the portal vein produces the large anemic infarct that is characteristic of the disease. Most of the bacteria are to be found in this infarct and, under the anaerobic conditions, the necrotoxic and hemolytic beta toxin is released systemically to result in toxemia, generalized vascular damage and intravascular hemolysis.

**CLINICAL FINDINGS**

The illness is of short duration and cattle at pasture may be found dead without signs having been observed. More often there is

1. A sudden onset, with complete cessation of rumination, feeding, lactation, and defecation.
2. Abdominal pain is evidenced by disinclination to move and an arched back posture.
3. Grunting walking.
4. 4-Respiration is shallow and labored and Severe dyspnea is observed before death.
5. the pulse is weak and rapid.

6- Fever (39.5-41°C) in the early stages but the temperature subsides to subnormal before death.

7-Edema of the brisket is a common finding.

8-The feces are dark brown; may be diarrhea with mucus and some blood.

9-The urine is dark red. Jaundice is present some time

10-The duration of the illness varies from 12 hours in dairy cows in advanced pregnancy to4 days in dry stock

11-Pregnant cows often abort.

12-The disease in sheep presents with similar signs.

**CLINICAL PATHOLOGY**

The red color of the urine is due to the presence of hemoglobin. In the later stages there is anemia, the

1-erythrocyte count being depressed to between 1 and 4 x 10 12/L

2-the hemoglobin to 3- 8 g\L.

3-Leukocyte counts vary considerably from 6700-34800 x 109/L. and neutrophilia in severe cases

4- Serum calcium and phosphorus levels are normal but blood glucose levels may be elevated (100-120 mg/dL).

**NECROPSY FINDINGS**

1-Rigor mortis develops quickly.

2-The perineum is soiled with bloodstained urine and feces, Subcutaneous gelatinous edema, and extensive petechial or diffuse hemorrhages in subcutaneous tissue are characteristic.

3-. There is a variable degree of jaundice

4- Excessive amounts of fluid, varying from clear to bloodstained and turbid, are present in the pleural, pericardial and peritoneal cavities.

5- hemorrhages appear under the endocardium.

6- Hemorrhagic abomasitis and enteritis are accompanied by the presence of bloodstained ingesta or free blood.

7- The characteristic lesion of bacillary hemoglobinuria is an ischemic infarct in the liver, One or more may be vary from5-20 cm in diameter.

8- The infarct is pale, surrounded by a zone of hyperemia, and local necrosis.

9- Red urine is present in the kidneys and bladder and petechiation is evident throughout the kidney.

10- The bacteria can be isolated from the liver infarct from a fresh carcass.

**Diagnosis**

1-Clinical signs

2-Bacteriology - tissue from edge of liver infarct, placed in an airtight container; four air-dried impression smears from lesion border (anaerobic CULT, FAT)

3- Histology - fixed liver lesion, kidney.

**Differential diagnosis**

The diagnosis of bacillary hemoglobinuria is differentiated from other diseases in which hemoglobinuria, myoglobinuria, and hematuria are cardial signs. In an animal found dead differentiation from other clostridial diseases and anthrax may be required.

• Acute leptospirosis

• Postparturient hemoglobinuria

• Hemolytic anemia caused by cruciferous plants

• Babesiosis and anaplasmosis

• Enzootic hematuria

• Chronic copper poisoning (sheep)

**TREATMENT**

Specific treatment includes the immediate use of

1-penicillin or tetracyclines at high doses

2- antitoxic serum if available administered in the early

stages of the disease.

3-Supportive treatment - including blood transfusion, parenteral fluid, and electrolyte solutions

4-Care is required during treatment and examination, as undue excitement or exercise may cause sudden death.

5-Bulls should not be used for service until at least 3 weeks after recovery because of the danger of liver rupture.

6-Convalescence is often prolonged and animals should be protected from nutritional and climatic stress until they are fully recovered.

7-Hemopoiesis should be facilitated by the provision of mineral supplements containing iron, copper, and cobalt.

8-hemoglobinuria may disappear within 12 hours.

**CONTROL**

1-A formalin-killed whole culture adsorbed on aluminum hydroxide gives good protection for a year in cattle.

2-Vaccination is carried out 4-6 weeks before the expected occurrence of the disease.

3-Annual revaccination of all animals over6 months of age is necessary in enzootic areas.

4-The carcasses of animals dying of the disease should be disposed of by burning or deep burial.