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Bacillary Hemoglobinuria

(Red Water Disease)

ETIOLOGY

C. haemolyticum* (*C. novyi* type D)** is a soil borne anaerobe. The longevity of the spores in soil is unknown, but 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 **phospholipase C (β-toxin**), a **necrotoxic and hemolytic toxin responsible for the clinical disease.** Damage to the liver by ***F. necrophorum, and **fasciolosis** have been suggested as precipitating causes.

EPIDEMIOLOGY

Animal Risk Factors

Cattle are the usual species involved, although occasional cases occur in sheep and rare cases in pigs. As is the case in many clostridial diseases, animals **in good condition are more susceptible.**

Environmental Risk Factors

Bacillary hemoglobinuria is a disease of the **summer and autumn** months. A primary association occurs with pastures that also are associated with the occurrence of **liver fluke** although other, less determined risk factors obtain. The highest incidence of bacillary hemoglobinuria is on irrigated or poorly drained pasture, especially if the soil is alkaline in reaction.

Source of infection

-The disease is **spread** from infected to non-infected 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 affect spread of the infection.

- **Contamination of pasture** may occur from feces or from decomposing cadavers

PATHOGENESIS

under natural conditions in endemic areas invasion occurs from the alimentary tract after ingestion of contaminated material. As in black disease of sheep, 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.

Migrating flukes are thought to be predisposed to clinical disease by leading to liver necrosis and the establishment of anaerobic conditions in the liver that will lead to the multiplication of the causative organism.

Invasion of the liver by *Cysticercus tenuicollis* and other causes of liver damage can also lead to the disease. Once *C. haemolyticum* returns to its vegetative state in an anaerobic environment and replicates, it also **produces phospholipase C (β-toxin)**. **This β -toxin causes hemolysis, necrosis of hepatocytes**, and damage to capillary endothelium, all of which lead to **hemoglobinuria** and **loss of vascular fluid into tissues and serous cavities**. The development of an organized thrombus in a sub terminal 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 β-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 obvious signs of the disease. More often there is a **sudden onset**, with complete

cessation of rumination, feeding, lactation, and defecation. Abdominal pain is evidenced by reluctance to move and an arched-back posture. Grunting may be evident on walking. Respiration is shallow and labored and the pulse is weak and rapid. Fever (39.5–41° C) is evident in the early stages, but the temperature subsides to subnormal before death. **Edema of the brisket** is a common finding. The feces are dark brown; there may be diarrhea with a great deal of mucus and some blood. The **urine** is **dark red. Jaundice** is present but may not be very obvious. The duration of the illness varies from 12 hours in dairy cows in advanced pregnancy to 4 days in dry stock. Pregnant cows often abort. Severe dyspnea is evident just before death. The disease in sheep presents with similar signs.

CLINICAL PATHOLOGY

The **red color of the urine** is caused by the presence of hemoglobin; there are no free red cells. In the later stages there is **anemia**, characterized by a marked decline of packed cell volume and red blood cell counts. Leukocyte counts tend to be mildly to markedly elevated with the presence of toxic granulocytes.

Most prominent changes in blood biochemistry analysis are **elevated enzyme** activity of **AST and GGT, as** well as mild to moderate elevation of serum bilirubin concentrations, which are reflective of liver damage .**Blood cultures** during the acute stages of the disease may be positive. Serum agglutinins against *C. haemolyticum* may be detectable at low levels (1 : 25 or 1 : 50) during the clinical illness and, if the animal recovers, rise to appreciable levels (1 : 50–1 : 800) a week later. Titers greater than 1 : 400 are usual at this time. A positive agglutination test is not conclusive evidence of the presence

of the disease.

NECROPSY FINDINGS

Rigor mortis develops quickly. The perineum is soiled with bloodstained urine and feces.

Subcutaneous, gelatinous edema, which tends to become crepitant in a few hours, and extensive petechial or **diffuse hemorrhages**

in subcutaneous tissue are characteristic. There is a variable degree of jaundice. Excessive amounts of fluid, varying from clear to bloodstained and turbid, are

present in the pleural, pericardial, and peritoneal cavities. Generalized subserous hemorrhages are also present. Similar hemorrhages appear under the endocardium. Hemorrhagic abomasitis and enteritis are accompanied by the presence of bloodstained ingesta or free blood. The **characteristic lesion** of bacillary hemoglobinuria is an **ischemic infarct in the liver**.

DIFFERENTIAL DIAGNOSIS

The diagnosis of bacillary hemoglobinuria is largely a question of differentiation 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. Differentiating between hemoglobinuria and hematuria by spinning urine will allow the discrimination of those differentials not associated with intravascular hemolysis.

- **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 **penicillin or Tetracyclines at** high doses and **antitoxic serum if available.**

Prompt treatment is essential. If the serum is administered in the early stages of the disease, then hemoglobinuria may disappear within 12 hours.

Supportive treatment, including blood transfusion, parenteral fluid, and electrolyte solutions, is of considerable importance. Care is required during treatment and examination, because undue excitement or exercise may cause sudden death.

Bulls should not be used for service until at least 3 weeks after recovery because of the danger of liver rupture. Convalescence is often prolonged, and

animals should be protected from nutritional and climatic stress until they are fully recovered.

CONTROL

-**A formalin-killed whole culture adsorbed on aluminum hydroxide gives good protection for a year in cattle. Vaccination** is performed 4 to 6 weeks before the expected occurrence of the disease. **Annual revaccination** of all animals **over 6 months of age** is necessary in enzootic areas. In some **locations of extreme risk** a second vaccination during the grazing season is recommended. Modern vaccines prepared to avoid these local reactions lack immunogenicity and need to be administered twice a year.

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

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References:

- Constable PD, Hinchcliff KW, Done SH, et al. (2017). Veterinary Medicine: A Textbook of the Diseases of Cattle, Horses, Sheep, Pigs, and Goats. 11th ed. Elsevier, St. Louis, Missouri, USA.