CASE REPORT

Bacillary Hemoglobinuria Associated with Hepatic Necrobacillosis in a Yearling Feedlot Heifer

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Summary

Bacillary hemoglobinuria was diagnosed in a yearling heifer in a large western Canadian feedlot. The animal had come through a terminal market in another province and arrived in the feedlot three weeks previously. Predisposing hepatic injury may have been caused by *Fusobacterium* infection secondary to the rumenitis of grain overload.

Résumé

Hémoglobinurie bacillaire associée à la nécrobacillose hépatique, chez une taure d'un parc d'engraissement

Les auteurs ont diagnostiqué l'hémoglobinurie bacillaire, chez une taure d'un parc d'engraissement de l'Ouest canadien. Elle provenait d'un marché terminal d'une autre province et vivait dans ce parc d'engraissement, depuis trois semaines. Les lésions hépatiques prédisposantes semblèrent imputables à une infection à *Fusobacterium*, secondaire à une ruménite attribuable à une ingestion excessive de grain.

Introduction

Immunization of beef cattle for clostridiosis is a routine management procedure in western Canada. While bacillary hemoglobinuria occurs only sporadically, it should be considered whenever hemoglobinuria is encountered in cattle. Bacillary hemoglobinuria was first described in California in 1916 (5). The causative bacterium was isolated and named *Clostridium hemolyticum* in 1926 (10). In North America bacillary hemoglobinuria was first associated with the liver damage caused by *Fasciola hepatica* in 1954 (8). At that time, healthy cattle were found to be carrying *Clostridium hemolyticum* in their livers and kidneys (7, 8).

In the accepted pathogenesis of this disease, predisposing liver necrosis from migrating young flukes, intoxicants or severe hepatic congestion allows dormant bacteria present to proliferate and produce toxins under the influence of these hypoxic conditions. The beta toxin (phospholipase C) is a hepatotoxin and a hemolysin. Liver necrosis is followed by hemoglobinemia, anemia and hemoglobinuria. Respiratory failure is attributed to a combination of hypoxia and toxemia (1, 2, 4).

Seven cattle have been diagnosed as having bacillary hemoglobinuria at the Western College of Veterinary Medicine during the last 12 years. In four cases the diagnosis was confirmed by the identification of *Clostridium novyi Type D (Clostridium hemolyticum)* by culture or the fluorescent antibody technique (FAT). The other three diagnoses were based on the typical gross and microscopic lesions. The reported incidence of the disease in feedlots is low (1, 4, 9), however, increased losses are often associated with movement of cattle from infected alkaline pastures to uninfected premises (1, 3, 4, 8, 9).

The purpose of this report is to suggest a possible association between hepatic necrobacillosis secondary to subclinical rumenitis, and the development of bacillary hemoglobinuria.

History

During the first week of October, a truckload of yearling cattle arrived at a Saskatchewan feedlot from Edmonton, Alberta. By October 19 these cattle were consuming a 92% concentrate ration. A Charolais crossbred heifer, about ten months old, was noticed anorexic and depressed. Although her rectal temperature was normal, the treatment personnel administered penicillin at 46200 i.u. per kg. But in spite of this treatment, she was found dead the next day.

The feedyard foreman in charge of treating sick cattle reported other cattle in the same pen were scouring and ataxic after being placed on a 92% concentrate ration. Two other heifers from this pen that had died with respiratory disease were coincidentally found to have rumenitis and embolic mycotic hepatitis.

Necropsy Findings

A necropsy was done on this heifer two days later. The carcass was in good physical condition. The tissues were extremely icteric and the bladder was full of dark brown to black urine. The kidneys were extremely dark, and had undergone severe autolysis. The liver was enlarged and yellow. The most striking liver lesions were several focal areas of necrosis 10-20 cm in diameter (Figure 1). These large firm circumscribed areas, randomly distributed in the liver, were raised above the surface. The affected areas were dark with small, central, pale foci up to 2 cm diameter.

Histopathology

The renal tubules contained a large amount of orange brown pigment. Areas of coagulative necrosis in the liver were marginated by a zone of bac-

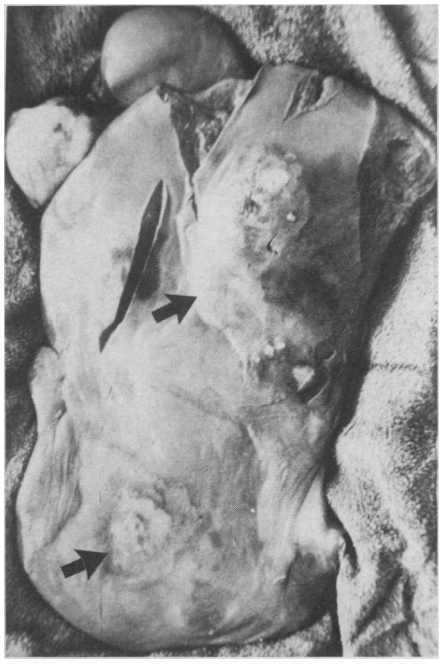

FIGURE 1. Two areas of coagulative necrosis 10-20 cm in diameter on the affected liver (indicated by arrows).

terial proliferation. These bacteria were fusiform Gram-negative bacilli with the typical morphology of species of *Fusobacterium*. More peripheral to these were degenerating neutrophils. These areas were incorporated into larger irregular zones of necrosis with numerous Gram-positive bacilli present and with less clearly defined zones of infiltrating degenerating neutrophils.

Bacteriology

Positive fluorescence for *Clostridium novyi* was found in the liver lesions, and smears contained numerous large Gram-positive bacilli and Gram-negative fusiform bacilli. On anaerobic culture on blood agar and cooked meat broth, however, no *Clostridium novyi* nor *Fusobacterium necrophorum* could be cultured from the liver, probably because of treatment with penicillin.

Discussion

In this case, hepatic injury was probably initiated by hepatic necrosis due to infection with *Fusobacterium necrophorum*. This is a common sequel to rumenitis caused by sudden change to a high grain diet. *Clostridium hemolyticum*, likely dormant in the liver of this heifer, was able to proliferate, when the hypoxic conditions of hepatic necrobacillosis occurred, to cause more acute and more extensive necrosis.

The clostridial vaccination regime in this feedlot involves use of a bacterin that contains *Clostridium chauvoei* and *septicum* only. To provide protection against bacillary hemoglobinuria would mean an almost fourfold increase in cost of clostridial immunization. Because the disease is of such sporadic incidence, this increase in cost in this feedlot probably can not be justified.

This report is an illustration of the benefit of an organized system of mortality surveillance in a commercial feedyard. Even if routine necropsies demonstrate that the occurrence of bacillary hemoglobinuria is increased, the preventive approach might best be aimed at prolonging the period of transition from a high roughage to a high concentrate diet rather than a change in the clostridial vaccination protocol.

Acknowledgments

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ABSTRACT

Mason, T.A., Lavelle, R.B., Skipper, S.C. and Wrigley, W.R. Osteochondrosis of the elbow joint in young dogs. Journal of Small Animal Practice (1980) 21: 641-656. (Dep. Vet. Clin. Sci., Univ. Melbourne, Werribee, Victoria 3030, Australia).

Osteochondrosis is a failure of the normal process of endochondral ossification. In the elbow joint there are three manifestations of osteochondrosis, namely osteochondritis dissecans of the medial condyle of the humerus, ununited coronoid process and ununited anconeal process. This paper describes a series of sixty-eight cases diagnosed at a referral centre over a 10-year period. Ununited coronoid process and OCD of the medial condyle are not readily diagnosed because the lesions are difficult to demonstrate radiographically, but signs of degenerative joint disease of the elbow in young dogs of the medium to large breeds is strongly suggestive. Surgical treatment is indicated and the surgical approach to the medial aspect of the joint is described.

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