Clostridial Diseases

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Take Home Message

The diseases caused by the clostridial group of organisms, such as blackleg caused by Clostridium chauvoei in young, well-fed beef cattle, are characterised by sudden onset, short duration, and high death rate in affected animals. Uncontrolled, these diseases can be a significant loss to livestock production throughout the world. The very rapid progress of these diseases makes them extremely difficult to treat. Fortunately, most clostridial diseases are effectively controlled by vaccination.

Introduction

The widespread use of clostridial vaccines makes it difficult to obtain information on the prevalence and economic significance of clostridial diseases. One report suggested that clostridial diseases were responsible for the deaths of 289 (3%) of nearly 9500 unvaccinated feedlot cattle and in other reports the death rates for blackleg and bacillary hemoglobinuria (red water disease) in unvaccinated animals have varied between 3% and 10%.

Clostridial bacteria are widely distributed in soils throughout the world, surviving for many years as highly resistant, inactive spores which are activated under certain circumstances. They enter the body through wounds and contaminated feed as active bacteria or spores. Indeed, some are normal inhabitants of the intestinal tract; Clostridium perfringens has grown in the gut of newborn calves by 24 hours of age. In addition to wounds and the gut, Clostridium organisms have two other preferred sites in the body, the muscles in the case of Clostridium chauvoei, and the liver for Clostridium hemolyticum and Clostridium novyi B which cause bacillary hemoglobinuria and black disease respectively. From this it can be appreciated that the clostridial diseases are infectious but not contagious i.e. they do not spread directly from animal to animal. However, it should be recognised that many animals may be exposed to the same contaminated environment or the same initiating factors at the same time.
Multiplication of clostridial bacteria occurs only in the absence of oxygen, or, in environments with very low concentrations of oxygen. During active multiplication the clostridial organisms produce a number of toxins, (in this context, toxins are biologically active proteins), but there are three important groups of toxins which are responsible for most of the clinical signs seen in clostridial diseases: 1. Lethal necrotizing toxins which kill cells and destroy tissues; 2. Hemolytic toxins which destroy red blood cells and 3. Neurotoxins which interfere with the transmission of nerve impulses. Some clostridia elaborate toxins which act at the site of production, such as blackleg, while others produce toxins that are absorbed into the blood stream and exert their effects throughout the body, e.g. the gut-inhabiting clostridia.

**Blackleg**

Blackleg is an acute, fatal disease of young, well-fed cattle between 6 and 18 months old, but is occasionally seen in calves as young as 2 months. Muscle bruising associated with fighting, butting, riding, or merely physical exertion may create the conditions favouring spore germination and rapid bacterial multiplication. At the start of an outbreak animals may simply be found dead. Blackleg should be suspected in all cases of ‘sudden death’. If found alive, affected animals are extremely depressed, reluctant to move and obviously lame in one or more legs. Hot, painful swellings of one of the large muscle masses of the hind quarters, shoulders, and neck are invariably present. These swellings often crackle on handling due to the gas present in the muscles. In the early stages a marked fever, 40.5 - 41.5°C (105 - 107°F), is often present, but the temperature quickly becomes subnormal. The disease progresses rapidly, with the development of laboured respiration, muscle tremors and prostration. Death occurs 12 to 24 hours after the onset of clinical signs.

Bloat and putrification (rotting) occur rapidly after death in cases of blackleg and it is important that a post-mortem examination is carried out as soon as possible after death. On incision of the swellings, gas bubbles and gelatinous fluid are found under the skin. In the deeper muscle groups the colour varies from dark red to almost black. When cut, a sweet, rancid odour is noticeable and the affected muscle looks and feels like lung tissue due to the presence of gas bubbles. In fresh carcasses, there is an obvious demarcation between affected and normal muscle groups.
The clinical signs and the post mortem findings of blackleg are sufficiently characteristic to allow an accurate diagnosis, but the diagnosis can be confirmed by laboratory techniques. *Clostridium chauvoei* is the organism most commonly isolated from blackleg cases, but mixed infections with *Cl. septicum* and *Cl. sordelli* also occur.

**Bacillary Hemoglobinuria (Red Water Disease)**

Bacillary hemoglobinuria (red water) is an acute, highly fatal, but fortunately sporadic, disease of cattle and occasionally sheep. It is caused by *Clostridium hemolyticum* which multiplies in the liver following damage to the liver by migrating liver flukes, liver abscesses, or liver necrosis caused by *Fusobacterium necrophorum* secondary to a rumenitis of grain overload in feedlot animals. The multiplying bacteria produce several toxins, one of which is hemolytic, i.e. destroys red blood cells. The destroyed red cells release hemoglobin which is the red pigment that carries oxygen. This red pigment is excreted in the urine (hemoglobinuria). Older, adult cattle are more likely to be affected, but it has been recognised in feedlot animals. Clinically, animals may be found dead with no warning signs. Animals found alive are extremely ill with severe depression, complete inappetence, laboured respirations, red coloured urine, and occasionally dysentery (bloody diarrhea). The clinical signs last for 12 hours. At post-mortem examination, the presence of a circular area of dead tissue, 5-6 cm in diameter, in the liver (infarct), together with port-wine coloured urine in the bladder and an excess of bloody fluid in the body cavities is indicative of bacillary hemoglobinuria.

*Black disease*, caused by *Clostridium novyi B*, has a very similar development to bacillary hemoglobinuria in that the bacterial spores are activated in the liver following injury to the liver. This species of *Clostridium* does not produce a hemolytic toxin and there is no hemoglobinuria in this disease. It is most often seen in sheep as a result of liver fluke migration, but it is occasionally recognised in cattle. Affected sheep are found dead.

**The Enterotoxemias**

Enterotoxemia is any condition in which toxins produced in the intestines are found in the blood and in veterinary medicine it is generally accepted that the enterotoxemias are associated with the *Clostridium perfringens* group of organisms. This group of organisms is widely distributed in soil, and readily colonises the gut. Six types (A-F) are recognised on the
basis of toxin production. Under certain conditions in the intestines the bacteria multiply and produce toxins which are absorbed into the blood stream and have severe, deleterious effects on the major organs, the liver, the heart, the lungs and the brain. Other toxins act locally on the gut wall. This group of organisms produces a range of diseases in man and animals. Diagnosis is confirmed by the demonstration of specific toxins in the gut contents by biological assay.

_Clostridium perfringens_ type D is the most important species of this group. It causes _enterotoxemia_ or _pulpy kidney disease_ (overeating disease) in sheep throughout the world. It is most often seen when fast growing lambs are introduced to diets high in carbohydrate, either lush pasture or grain-based diets. The duration of clinical signs is very short, from 30 minutes to 2 hours, and most affected animals are simply found dead. Fortunately, although cattle are often managed similarly, with sudden introduction to lush pastures and planned, or accidental, introduction to high carbohydrate diets, type D enterotoxemia is very rare in cattle.

Two other _Cl. perfringens_ species are of interest to cattle producers. _Cl. perfringens_ type C causes a severe, bloody diarrhea in calves (and lambs, piglets and foals) less than a week old and has been recognised in Alberta. _Cl. perfringens_ type A has been associated with abomasal tympany and abomasal ulceration in young beef calves, but a recent study at the Western College of Veterinary Medicine failed to confirm an association between _Cl. perfringens_ type A and ulceration of the abomasum.

**Tetanus and Botulism**

Tetanus and botulism are two clostridial diseases affecting the nervous system caused by _Clostridium tetani_ and _Clostridium botulinum_ respectively. _Cl. tetani_ is a soilborne organism that contaminates wounds and in low concentrations of oxygen elaborates a toxin which is absorbed into the blood system and subsequently reaches the nervous tissue. Botulism is unusual in that the disease occurs following the ingestion of preformed toxin in contaminated feed. The toxin is produced following multiplication of the organism in decomposing carcases or, less commonly, decaying vegetation (e.g. poorly made big bale silage). The toxins (neurotoxins) of both organisms interfere with the transmission of nerve impulses to the muscles. Although apparent sudden deaths can occur in both diseases, they have a longer duration than other clostridial diseases.
Tetanus (lockjaw) can occur after the contamination of any wound, but the most common cause is the use of rubber rings for castration. Having entered the nervous system, the toxin allows continued, unrestrained transmission of nerve impulses to the muscles resulting in muscle rigidity. A stiff, rigid gait, with fixed, erect ears, and a degree of ruminal tympany are the most prominent clinical signs. Affected animals also have ‘lockjaw’ with rigidity of the facial muscles preventing opening of the mouth and difficulty in eating and even drinking. Death occurs when the respiratory muscles become affected, or through a combination of respiratory paralysis, dehydration and starvation. Less severely affected animals can be treated by intensive nursing, but affected animals may take up to 8 weeks to recover fully.

Botulism is a rare disease in any species. Early reports of the disease in cattle were associated with phosphate deficiency in South Africa in which cattle ate bones contaminated with botulinum toxin. More recently, it has been associated with the incorporation of ensiled poultry waste into feedlot diets or with the spreading of poultry manure contaminated with chicken carcases on pastures. Severe muscle flaccidity, weakness, and an inability to stand are the most obvious clinical signs.

Prevention

As stated in the introduction, treatment of clostridial diseases is very difficult because of their short duration and high death rate. These diseases should be controlled by vaccination. The most important disease in cattle is blackleg, and at a minimum cattle should be vaccinated against this disease. In this area calves are vaccinated with one dose of blackleg vaccine (2 way; *Cl. chauvoei*, *Cl. septicum*) before going to pasture in the spring and revaccinated when they are taken off pasture in the fall. This second vaccination should be a multicomponent clostridial vaccine (7- or 8-way). Replacement heifers should be vaccinated before breeding with a multicomponent vaccine (7- or 8-way). If replacement animals have been vaccinated against clostridial diseases 3 times it is not necessary to vaccinate the adult cow herd again, unless there is a high risk of bacillary hemoglobinuria. If this is the case, the adult herd should be revaccinated annually with a multicomponent vaccine containing *Cl. hemolyticum*. If the vaccination status of the cow herd is unknown, vaccinate all the cows with two doses of multicomponent vaccine (7- or 8-way) 3 to 4 weeks apart and then keep vaccination status of replacements up-to-date.
Where enterotoxemia (\textit{Cl. perfringens} type C) of very young calves is a concern, a 'calf scours' vaccine containing \textit{E. coli}, rotavirus, coronavirus and \textit{Cl. perfringens} type C is available for vaccination of pregnant cows. Alternatively, vaccinate pregnant cows with a multicomponent vaccine (7- or 8-way) containing \textit{Cl. perfringens} type C 3 to 4 weeks before calving.

**Feedlot cattle**

Vaccinate animals on arrival with a multicomponent vaccine (7- or 8-way). Ideally, animals should be revaccinated 3 to 4 weeks after arrival. The vaccine should contain \textit{Cl. tetani} if castration by rubber band is contemplated.

There is some evidence from the United States that the multi component (7-way, 8-way) clostridial vaccines may cause a decrease in feed consumption in the immediate post-vaccination period. For the first four days after vaccination, feed consumption may decline by 10% to 20%. This decreased food intake does not appear to adversely affect weight gains or final finishing weight, but may contribute to feedbunk management problems. In addition, this decline in feed consumption may be more severe after a second vaccination.

All vaccines for clostridial diseases should be given subcutaneously (under the skin) in the neck area because they cause severe muscle damage and scarring which persists until slaughter and causes meat trim and toughness.

**Summary**

Clostridial diseases, especially blackleg and tetanus (lockjaw), are common in this area. They frequently cause 'sudden death' and are extremely difficult, if not impossible, to treat, but, they are preventable with vaccination.