

CLOSTRIDIAL DISEASES



This disease
is known to
occur in
Afghanistan.



1. Definition

Clostridial diseases are caused by bacteria affecting cattle, sheep, goats and horses. The diseases go by many names depending on the specific bacteria causing the disease, or the clinical signs associated with it: blackleg (black quarter); bacillary hemoglobinuria (red water); enterotoxemia (overeating disease, pulpy kidney disease); infectious necrotic hepatitis (black disease); malignant edema, big head (swelled head); tetanus (lock jaw).

2. Etiology

Clostridia are relatively large, anaerobic, spore forming, rod-shaped organisms. Associations with disease are as follows: *Cl. chauvoei* - blackleg; *Cl. haemolyticum* - bacillary hemoglobinuria; *Cl. perfringens* types B, C and D – enterotoxemia; *Cl. novyi* - infectious necrotic hepatitis; *Cl. septicum* - malignant edema; *Cl. sordellii* - big head; and *Cl. tetani* – tetanus.

3. Transmission

Clostridial bacteria are common in soil and the intestinal tract of animals, and are usually harmless. Under the right conditions, however, the bacteria grow rapidly and release toxins, quickly destroying tissue and often causing death. Flooding of low lying pasture may also bring the bacteria to the surface and increase the risk of exposure. These diseases are not contagious, meaning they do not spread from animal to animal.

4. Species affected

- blackleg: cattle and sheep
- bacillary hemoglobinuria: cattle and sheep
- enterotoxemia: sheep, cattle and goats
- infectious necrotic hepatitis: Sheep mainly, and sometimes cattle
- malignant edema: sheep mainly, cattle and goats
- big head: rams
- tetanus: horses are most susceptible, goats, sheep and cattle

5. Clinical signs

Blackleg: Incidence of this disease is high in Afghanistan in summer and fall, and often strikes the biggest and healthiest cattle and sheep in the flock. The onset of the disease is sudden, and a few animals may be found dead without signs. Acute lameness and marked depression are common. Initially, there is a fever but, by the time clinical signs are obvious, body temperature may be normal or subnormal. Characteristic edematous and crepitant swellings develop in the hip, shoulder, chest, back, neck, or elsewhere. At first, the swelling is small, hot, and painful. As the disease rapidly progresses, the swelling enlarges, there is crepitation on palpation, and the skin becomes cold and insensitive as the blood supply to the area diminishes. Death occurs in 12-48 hours. Most cases of blackleg in cattle occur from 6 months to 2 years of age, and in sheep the disease more often occurs following some form of injury or wounds.

Bacillary hemoglobinuria: Cattle may be found dead without any signs. Usually, there is a sudden onset of severe depression, fever, abdominal pain, dyspnea, dysentery, and hemoglobinuria. Anemia and jaundice are present in varying degrees.

Enterotoxemia: Infection with *Cl. perfringens* types B and C causes severe enteritis, dysentery, toxemia, and high mortality in young lambs, kids, and calves. Sudden death is often the first or only sign in lambs and kids. Some young animals may show additional signs before death, such as crying out and teeth grinding, muscular tremors, frothing at the mouth, yellowish or bloody diarrhea, and convulsions. High levels of starchy food in the diet and slowing of gut movement are predisposing factors. In calves, there is acute diarrhea, dysentery, abdominal pain, convulsions, and opisthotonus. Death may occur in a few hours, but less severe cases survive for a few days, and recovery over a period of several days is possible.

Cl. perfringens type D causes pulpy kidney disease, a classic enterotoxemia of lambs that are either <2 wk old or weaned in feedlots and on a high-carbohydrate diet or, less often, on lush green pastures, seen less frequently in goats and rarely in cattle. Usually, sudden deaths in the best-conditioned lambs are the first sign. In some cases, excitement, incoordination, and convulsions occur before death. Opisthotonus, circling, and pushing the head against fixed objects are common signs of CNS involvement; frequently, hyperglycemia or glucosuria is seen. Diarrhea may or may not develop.

Infectious necrotic hepatitis: There is sudden death in sheep with no well-defined signs. Affected animals tend to lag behind the flock, assume sternal recumbency, and die within a few hours. Most cases occur in the summer and early fall when liver fluke infection is at its peak. The disease is most prevalent in 1 to 4 year old sheep and is limited to animals infected with liver flukes.

Malignant edema: General signs, such as anorexia, intoxication, and high fever, as well as local lesions, develop within a few hours to a few days after predisposing injury. The local lesions are soft swellings that pit on pressure and extend rapidly because of the formation of large

quantities of exudates that infiltrate the subcutaneous and intramuscular connective tissue of the affected areas. The muscle in such areas is dark brown to black. Accumulations of gas are uncommon. Severe edema of the head of rams develops after infection of wounds inflicted by fighting. Malignant edema associated with lacerations of the vulva at parturition is characterized by marked edema of the vulva, severe toxemia, and death in 24-48 hours.

Big head: The disease is characterized by a nongaseous, nonhemorrhagic, edematous swelling of the head, face, and neck of young rams. This infection is initiated in young rams by their continual butting of one another. The bruised and battered subcutaneous tissues provide conditions suitable for growth of pathogenic clostridia, and the breaks in the skin offer an opportunity for their entrance.

Tetanus: The incubation period varies from one to several weeks but usually averages 10-14 days. Localized stiffness, often involving the masseter muscles and muscles of the neck, the hind limbs, and the region of the infected wound, is seen first; general stiffness becomes pronounced ~1 day later, and tonic spasms and hyperesthesia become evident. The animal is easily excited into more violent, general spasms by sudden movement or noise. Spasms of head muscles cause difficulty in prehension and mastication of food, hence the common name, lockjaw. In horses, the ears are erect, the tail stiff and extended, the nares dilated, and the third eyelid prolapsed. Walking, turning, and backing are difficult. Spasms of the neck and back muscles cause extension of the head and neck, while stiffness of the leg muscles causes the animal to assume a “sawhorse” stance. Sweating is common. General spasms disturb circulation and respiration, which results in increased heart rate, rapid breathing, and congestion of mucous membranes. Sheep and goats often fall to the ground and exhibit opisthotonus when startled. Consciousness is not affected.

6. Pathologic findings

Blackleg: Edematous and crepitant swellings develop in the hip, shoulder, chest, back, and neck muscles. The affected muscle is dark red to black and dry and spongy; it has a sweetish odor and is infiltrated with small bubbles but with little edema. In sheep, because the lesions of the spontaneously occurring type are often small and deep, they may be overlooked.

Bacillary hemoglobinuria: Dehydration, anemia, and sometimes subcutaneous edema are present. There is bloody fluid in the abdominal and thoracic cavities. The lungs are not grossly affected, and the trachea contains bloody froth with hemorrhages in the mucosa. The small intestine and occasionally the large intestine are hemorrhagic; their contents often contain free or clotted blood. An anemic infarct in the liver is virtually pathognomonic; it is slightly elevated, lighter in color than the surrounding tissue, and outlined by a bluish red zone of congestion. The kidneys are dark, friable, and usually studded with petechiae. The bladder contains dark urine.

Enterotoxemia: Hemorrhagic enteritis with ulceration of the mucosa is the major lesion in all species. Grossly, the affected portion of the intestine is deep blue-purple and appears at first glance to be an infarction associated with mesenteric torsion. In young lambs, necropsy may reveal only a few hyperemic areas on the intestine and a fluid-filled pericardial sac. In older animals, hemorrhagic areas on the myocardium may be found as well as petechiae and ecchymoses of the abdominal muscles and serosa of the intestine. Rapid postmortem autolysis of the kidneys has led to the popular name, pulpy kidney disease; however, pulpy kidneys are by no means always found in affected young lambs and are seldom found in affected goats or cattle. Hemorrhagic or necrotic enterocolitis may be seen in goats.

Infectious necrotic hepatitis: The most characteristic lesions are the grayish yellow, necrotic foci in the liver that often follow the migratory tracks of the young flukes. Other common findings are an enlarged pericardial sac filled with straw-colored fluid, and excess fluid in the peritoneal and thoracic cavities. Usually, there is extensive rupture of the capillaries in the subcutaneous tissue, which causes the adjacent skin to turn black (hence the common name, black disease).

Malignant edema: The local lesions consist of large quantities of serous exudates that infiltrate the subcutaneous and intramuscular connective tissue of the affected areas. The muscle in such areas is dark brown to black. Accumulations of gas are uncommon.

Big head: There are areas of bruised and battered subcutaneous tissues of the head.

Tetanus: The point of entry of organism cannot be found because the wound itself may be minor or healed. There are no remarkable lesions.

7. Diagnosis

The most important thing to do when sudden deaths of stock occur is to get an accurate diagnosis. Anthrax should be considered as differential diagnosis since many of the clostridial diseases can look like anthrax. Animals suspected of dying from anthrax should not be moved or cut up in any way. Clostridial diseases are usually fatal. Death occurs rapidly with pulpy kidney, black disease and blackleg, but takes several days to weeks with tetanus. Laboratory testing should be considered to identify the bacteria or the toxin. Sample must be collected as soon as possible after death.

8. Treatment

Clostridial diseases are difficult to treat because they progress so rapidly. Prevention, through proper management and vaccination, is far more

effective. Antitoxins should be used when available in conjunction with antibiotic therapy, such as penicillin.

9. Prevention and control

The major factor in controlling these diseases is to develop satisfactory immunity within the animal at risk through vaccination. Passive immunity is transferred from the mother to the offspring in the first milk (colostrum). If the mother is boosted with a multi-valent colostral vaccine about one month before the offspring is due to be born the level of protection and period of time for which the young animal is protected is increased.

Active immunity requires a primary course of two doses of vaccine 4 to 6 weeks apart to give a reasonable period of protection. The first dose is usually given at 8 weeks of age or weaning time, when the protection from the mother's milk is starting to decline. Annual boosters are required to maintain the protection, as well as providing antibodies in the colostrum to protect the young until they are old enough to be vaccinated. Boosters should be given strategically before high-risk periods.