

# Sheep Beard Disease to Develop Share Factors Impact and Against Fight Measure Events

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**Annotation:** More than 200 species of Clostridia are known, but only a few of them are toxic and pathogenic. Bradzot disease is a very dangerous infectious disease that occurs in the regions of our country. This article describes in detail the etiological factors, epizootological data, clinical signs, pathomorphological changes, diagnostic methods, and preventive measures in the development of Bradzot disease in sheep, based on literature data.

**Keywords:** clostridiosis, infection, anaerobic, aerobic, spore, capsule, helminths, polyinvasion, enzyme immunoassay, hyperimmune serum, convalescent serum, neutralization reaction, antigen, hemolysis, urobilin, phagocytosis, vaccination.

**Login.** Livestock breeding occupies a special place in the economy of Uzbekistan, and great importance is attached to the development of this sector. The development and profitability of livestock breeding depend on factors such as increasing the number of livestock in state, farm and private farms, increasing their productivity, obtaining healthy sheep and lambs, their proper care, and protection from various diseases. Sheep band disease is a major threat to sheep breeding. Band disease among sheep It is well known that the economic damage to sheep farming is a pressing problem. The shortage of biological and chemical drugs in the veterinary sector is further complicating the problem and contributing to the wider spread of diseases.

Due to the widespread prevalence of rabies among sheep, it causes significant damage to the economies of many livestock farmers .

Clostridia cause many diseases. More than 200 species of Clostridia are known, but only a few of them are toxic and pathogenic. Some species do not cause disease by themselves, but in combination with other anaerobic bacteria they complicate the infection. Pathogenic species include *C. septicum*, *Cl. Oedematiens*, *C. perfringens*, *C. chauvoei*, *C. botulinum*, *C. fallax*, *C. haemolyticum*, *C. sordelli*, *C. sporogenes*, *C. tetani*, *C. histolyticum*, *C. novyi*, and others.

**Relevance of the topic.** Bradzot (lat. - Hepatitis infectiosa necrotica, Bradzot; eng. - Braxy; bradzot; Russian - бpaдзoт; Danish - lightning-fast death) is an acute infectious disease of sheep and goats, characterized by inflammation of the mucous membranes of the stomach and duodenum and a violation of the properties of the parenchyma of the digestive organs. An analysis of the literature shows that external factors play an important role in the pathogenesis and spread of bradzot. The main factors in the pathogenesis of bradzot are damage to the gastrointestinal tract from substandard feed, internal mechanical damage by helminths, the role of which in the pathogenesis of the disease is the consumption of large amounts of green young grass, poor feed, grazing on frozen pastures, as well as hypothermia or overheating of the animal's body, and many other factors. Animals become infected through the digestive tract when they consume contaminated feed and water containing spores of the pathogen.

As a result of anaerobic conditions in the abomasum and duodenum walls under the influence of adverse factors, the pathogen multiplies rapidly and releases toxins that lead to general intoxication of the body.

The disease was infectious in 1875. Krabbe wrote that bradzot was widespread in Scandinavian countries (Iceland, Norway, Scotland) and Australia and called it black disease, necrotic hepatitis. In 1888. N. Nielsen isolated a bacillus from a sick sheep in Norway, proved that this disease was not anthrax and was an acute infectious disease, and in 1922 it was named *Clostridium septicum*. Later, Geiger proved that in addition to this pathogen, another one, *Cl. Oedematiens*, was also the causative agent of the disease. In 1918-1921, bradzot was called "Black disease" in Australia. The cause of death of sheep was understood to be the result of bacteria and the toxin released from it. Later it was called "Necrotic hepatitis". The disease was recorded in sheep in the Bukhara region of Uzbekistan by KA Andreev (1926). The importance of helminths in the parenchymal organs of animals in the pathogenesis of fascioliasis was first reported in the literature by A. Damm (1971), M. Ardexali, H. Derakchan (1975). They stated that young fasciolae carry clostridia to the liver, which results in the development of fascioliasis, necrotic hepatitis and sudden death of the animal. BG Gariev (1970-1985) also observed cases of sudden death of animals from fascioliasis in sheep farms with problems with fascioliasis. DI. Panasyuk (1984), PP Wiebe (1976), PV Radionov (1971-1990) reported on the prevalence of clostridial infections in ruminants on farms with problems with moniziasis, nematodosis, and trematodosis.

For the first time Yu.F. Petrov, IB Sorokina (1984, 1986), IB Sorokina (1982, 1985, 1987), VV Kuzmichev (1997) experimentally proved that young fasciola in the gastrointestinal tract of domestic animals under conditions of superinvasion are intensively poisoned by clostridia. Fasciola and clostridia accumulate in the liver, as a result of which the animal dies from sepsis and poisoning with anaerobic toxins. Later, BG Abalikhin (1996) experimentally showed that during the migration of dicrocodile, lesions appear in the liver of animals and the development of the bradzot and determined that the intoxication of the animal organism leads to its death. Thus, from the literature, the author convincingly proves the correctness of KI Scriabin's statement that "helminths open the gates of infection." The causative agent is anaerobic bacteria *Clostridium septicum*, *Cl. Oedematiens* type A are also often isolated from patients, which increase the pathogenicity of the above-mentioned pathogens. *Cl. septicum* is a polymorphic, gram-positive, motile, spore-forming rod (4-5  $\mu\text{m}$  in size). It appears in a filamentous form in liver smears. At 37 ° C, it hemolyzes on blood agar, turbidizes the broth in Kitt-Tarossi and produces gas. The turbidity settles to a sediment within 48 hours. It grows on Seyssler's medium, showing hemolysis. The bacilli ferment glucose, maltose, fructose, releasing gas. They do not

decompose glycerol and mannitol. In rare cases, they decompose sucrose. This feature is used to distinguish it from *Cl. chavoiei*. Because *Cl. chavoiei* always ferments sucrose. *Cl. C. septicum* secretes four very potent exotoxins: alpha, beta, gamma and delta toxins. This is especially evident in Marten broth. These toxins have necrotic and hemolytic effects (hemolysis of sheep erythrocytes). *C. l. septicum* contains O and There are H antigens, which in turn form agglutinins, precipitins, hemagglutinins in the body. *Cl. oedematiens* is a large straight or slightly curved gram-positive, motile rod (4 8x1-1.5 µm), forms spores. In the smear, 3-5 or more bacteria are arranged in chains. It grows in Kitt-Tarossi for 48 s, produces less gas. There are 4 types of *Cl. oedematiens* : A ( *Cl. novyi* ), B ( *Cl. gigas* ), S ( *Cl. bubalorum* ) and D ( *Cl. haemolyticum* ). Each type produces its own soluble antigen - toxin.

In nature, sheep are infected with bradzet, regardless of breed and age. More often, sheep under 2 years old are infected. Epizootics are observed in them. In one case, only lambs are infected, in another, only older sheep. Sheep that move less and are fatter are infected first. Older sheep are more susceptible to the disease when fed on pasture, and young lambs in a pen or at home. The disease occurs in all seasons of the year, but is more common in cold seasons (autumn, winter, spring). The occurrence of bradzet is facilitated by feeding sheep in snow, frost and dew, infection with helminths, lack of protein and mineral substances in the body, and low resistance. *Bacillus septicum* produces a very strong poison that poisons the whole organism and quickly kills infected animals. The source of the disease is sick and recovered clostridia. The pathogen enters the body through the alimentary - mucous membranes of the digestive system. A sick animal contaminates the external environment with its feces, especially soil, pastures and water. In natural conditions, animals are infected when grazing on pastures, mainly when eating soil-based feed (hay), grass or drinking water contaminated with the pathogen. Bradzet usually occurs very quickly in sheep and lambs. A clinically healthy animal in the evening is dead in the morning. In some cases, a seemingly healthy sheep, when being driven to pasture, immediately lies down, shivers, gnashes its teeth and dies within a few minutes. At the time of the disease, the incidence is 30-35%, and the mortality rate is 90-100%. Some observers describe Bradzet as occurring more often at the confluence of rivers and along the banks of lakes. It is suggested that this is caused by sheep eating grass mixed with mud from infected areas. If we analyze some data, special attention is paid to the occurrence of bradzet in pastures and in the case of grazing in one place. Lambs are most often affected when grazing in one place, and adult sheep are most often affected when grazing in pastures. There is also a lot of evidence in the literature that bradzet occurs in sheep that eat frozen hay. The fact that the pasture is infected with the pathogen is of great importance in the origin of the disease. Often bradzet and enterotoxemia can occur together in the spring.

**Conclusion**, for the prevention of sheep's fur coat disease, it is necessary to monitor the veterinary and sanitary condition of the flocks, pastures and drinking places. Factors contributing to the development of the disease are eliminated. All fur coat areas should be taken into account and healthy sheep should be vaccinated in early spring, 30-45 days before being released to pasture. All restrictive measures should be taken at the unhealthy point and measures should be taken to prevent the spread of the disease. According to the restrictive requirements, sheep, their products, strangers, vehicles are prohibited from entering or leaving the farm, shearing on the farm, mixing sheep with other groups. The flock is disinfected weekly with 5% active chlorine lime, 3-5% caustic soda or 10% formaldehyde. Dead bodies are burned without removing the skin and wool. Sick sheep are not slaughtered for meat, wool, or milk is not collected. Manure and remaining hay contaminated with feces and urine of sick animals are burned. The farm is closed 20 days after the end of the disease, recovery, and after all measures and final disinfection have been carried out.

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