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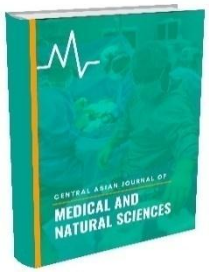

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Infectious Anaerobic Enterotoxemia Disease of Sheep

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Abstract: The article describes the types of the causative agent of infectious anaerobic enterotoxemia of sheep, the history of the development of the disease, ways of transmission of the causative agent to animals, epizootic status, timely identification of the causative agent of the disease, the causative agent of the disease Information about the clinical manifestations of the pathogen, identification of characteristic pathological changes, diagnosis of the disease, prevention of the disease and countermeasures is given.

Key words: Infectious anaerobic enterotoxemia of sheep, infected sheep, epizootology of the causative agent, economic damage, Cl. Types of perfringens, ways of spreading the disease, diagnosis, differential diagnosis, treatment, immunity.

Enterotoxemia (lat. - Enterotoxemia infectiosa anaerobica; visual. - Struck, Pulpy kidney disease) - "kidney emptying" - is an acute severe infectious disease of sheep, hemorrhagic enteritis, characterized by a nervous condition, severe kidney damage and poisoning by the toxin released by the pathogen.

Historical information. The disease was diagnosed in 1910. Gilruch called it by such names as "soft kidney", "kidney emptying", bilious disease, Bradzot-like disease. 1926 in Australia. Beinets found toxin and Cl from the small intestine of a lamb that died of this disease. Perfringens isolated type D and prepared a vaccine from it. He writes that in Australia, this disease occurs as a result of intoxication of sheep by grazing on grasses. 1931 Ewen (England) from a young sick sheep Cl. Perfringens isolated the species S.

Economic damage. Many sheep die as a result of severe disease. In some farms, 15-20 percent of sheep get sick, and in severe cases, up to 100 percent die. It is necessary to spend a lot of money for mandatory measures to eliminate the disease. If the disease is observed during the period of lambing, abortion is observed in many ewes, the lambs are born prematurely and they also die. Dead sheep are disposed of with their skin and wool. Additional costs are also spent on vaccination and health measures.

Trigger. The causative agent is anaerobic, belongs to the genus Clostridia, and in sheep Cl. Perfringens D, S and, in rare cases, type A, and in other animals, types A, V, E and G cause disease.

Each of them causes a specific disease. Cl. These 6 types of Perfringens have similar morphological, cultural, and biochemical characteristics, but they differ from each other in terms of pathogenicity, disease-causing ability, and toxin release. 15 different combinations of toxins were isolated from the causative filtrate.

Cl. Perfringens is gram-positive in young culture, gram-negative in old culture, non-motile (4-8x1-1.5μm, size), slightly bent rods with coiled ends, coccoidal and filamentous forms. In the external environment, the bacterium forms a spore in the center or near the tip. It forms a capsule in the body and in the nutrient medium. Kitt-Tarotsi produces gas and turbid broth when grown in glucose medium. 3 types of colonies grow in agar medium: S-smooth, R-smooth, and M-smooth forms. Cl. Perfringens type C alpha and betatoxins, Cl. Perfringens type D secretes alpha and epsilon toxins. These toxins are separated from the pathogen in the form of inactive prototoxins, prototoxins are activated under the influence of trypsin, pancreatin and other enzymes in the body.

In the territories of Australia, New Zealand, USA, Canada, France, Peru, Angola, the disease is Sl Perfrigens type D, and in some cases type C, have been identified. Some literature shows that type A is the cause in the USA and France. In Greece, Cyprus, Bulgaria Sl. Types C, D of Perfrigens, type D in Iran, type C in Turkmenistan, type D in Kazakhstan, type C in the northern regions of Russia, and type D in Dagestan, Kyrgyzstan, and in some farms, both types cause disease. it provokes.

Resistance of the trigger. Spore forms of clostridia are highly resistant to physical and chemical effects. It remains active in soil and water for 16-20 months, and in wool and skin for more than 2 years. It is kept active in the soil for 16-20 months at 35-40°C, and up to 40 months at 15-20°C. It can live in water for 20 months. It is stored for up to 3 days in dried form, up to 2 years in leather and wool at 10-20 °C. When dried, it dies in 1-2 days. Boiling kills the pathogen in 15-20 minutes. 3-5% active chlorine lime, 5% hot caustic soda, 15% hot sulfuric-corbolic acid mixture, 5-10% formaldehyde solutions inactivate the pathogen in 10-15 minutes, and therefore in disinfection is used. And the pathogen in the vegetative state is resistant to the effects of the external environment.

Epizootological information. In a natural state, sheep are susceptible regardless of breed and age. Cattle, goats, horses, poultry, pigs, camels and wild animals are less susceptible. Guinea pigs, rabbits, pigeons and white mice are prone to laboratory animals. People get sick too. Sheep of all ages are affected, especially ewes, lambs and young 8-10-month-old sheep. Sheep with little movement in the flock, and sheep of fat and fast-growing breeds get sick faster. Cl. The disease caused by Perfringens type D is observed in all ages of sheep: in spring - lambs, in autumn - in older sheep.

Cl. The disease caused by Perfringens S type is mainly observed in large sheep. The disease occurs more often in spring, less often in autumn and winter.

Sick and recovered clostridia are carriers of the disease. A sick animal pollutes the environment with its excrement, especially soil, pastures and water. The causative alimentary tract enters through the mucous membranes of the digestive system through food and water. Mass disease among lambs is observed in spring, late spring, and early summer in rainy years. In natural conditions, animals are infected when they feed on pastures, mainly when they eat soil feed (grass, hay) contaminated with pathogens, or drink water. Disruption of the secretory and motor functions of digestive organs makes it possible for the disease to appear.

This includes, in particular, a rapid change in the quality of feed, the sudden exit of sheep standing in the barn to the pasture or frost and dew, snow on the grass or eating frozen grass, grazing after rain, mineral and caused by a lack of protein.

The disease occurs in many countries of the world, including Australia, New Zealand, the USA, Canada, Argentina, Peru, Angola, Italy, France, Cyprus, Bulgaria, Hungary, Turkey, Iran, the Siberian

regions of the Russian Federation, Transcaucasia, Kazakhstan and registered in the Republics of Turkmenistan, Kyrgyzstan, Uzbekistan.

The disease occurs among sheep of different ages. In some cases, it has been noted among sheep fed on fattening, especially when the diet contains a lot of concentrates or when the pasture is very rich in grass. In Dagestan, all older sheep from 8-10 months of age are infected. According to K. Urgeev (1985), the disease is recorded in 52% of mother sheep, 21% of lambs and 27% of sheep of mixed age. Most ewes get sick in the last months of the period. When 640 sheep died of enterotoxemia were dissected, 489 of them were pregnant, and 276 of them had 2 or more fetuses. In some regions, the incidence of enterotoxemia in lambs was observed more often, and in the regions around Western Siberia and Baikal, the incidence of lambs was also higher. Their age is 1.5-2 months, and they were often found when they were fed in the same place with soft feed. This situation was not observed in sheep raised on pasture. In unhealthy farms and flocks, the disease is acute, sheep of all ages are affected. In many cases, the causative agent of enterotoxemia can be isolated from the organism of healthy sheep. This disease mainly affects sheep, but it can also be found in cattle, goats, horses, pigs, and camels.

Laboratory animals are susceptible to guinea pigs, cats, and white mice. Rabbits and rats are not infected. Factors that disrupt the secretory and motor function of the stomach are of great importance in the origin of the disease. This is especially the case when they are raised by hand or in one place and then suddenly transferred to pasture conditions. In our conditions, the disease occurs mainly in early spring, when new foliage begins to grow. The sheep, which have suffered from winter, are very happy, they are thrown into the new green, and they eat a lot, being greedy. In this case, the stomachs of the sheep, which are not used to green, become swollen and gas accumulates. As a result, an anaerobic environment is created in the gastrointestinal tract, and clostridia develop and multiply. This is especially evident in early spring, when young greens are covered with dew or frost and have not yet evaporated.

Pathogenesis. When clostridia, which are widespread in nature, enter the animal body, due to disruption of the secretory and movement activities of the organs of the gastrointestinal system, in anaerobic conditions, fullness of the stomach with undigested nutrients is considered a favorable condition for the existence of the causative agent of enterotoxemia. they multiply in this organ and release a prototoxin from themselves. Then, under the influence of proteolytic enzymes in the intestine, the prototoxin turns into epsilon-toxin, which is absorbed into the blood, lyses erythrocytes, injures the mucous membranes of the intestines, epithelial cells, kidney parenchyma, liver and central nervous system, and poisons the entire animal organism. Increases the permeability of the endothelium of blood vessels and capillaries, as a result of which hemodynamics is disturbed. These pathological processes disrupt metabolism, especially carbohydrate metabolism in the liver, kidneys, and brain. The heart, kidneys, liver and central nervous system cannot perform their normal functions and death occurs. Conditionally pathogenic bacteria in the organs of the digestive system can contribute to such a severe course of the pathological process.

Course and clinical signs. The latent period of the disease depends on the virulence of the causative agent of enterotoxemia, the resistance of the animal and the state of the organs of the digestive system. It is 2 - 6 hours in case of artificial infection. The disease can be very acute, acute, semi-acute and chronic. Many scientists distinguish between comatose and hemorrhagic forms, which depends on the nature of the toxin released from the pathogen.

When the disease is very acute, they suddenly die in 2-3 hours without clinical signs. Such a course is observed in young and fat sheep. Dead sheep are seen in the morning in the fields and pastures. Affected sheep lag behind without grazing and become a little weak. They have depression, a normal or slight increase in body temperature, the pulse is weak and accelerated. From their nose and mouth

foamy serous, blood-mixed mucus flows, frequent urination is observed. In some cases, bloody diarrhea and cramping are observed. The gait of sick sheep is reminiscent of the swaying posture of a cradle rocker, as they crawl and fall. While lying down, he swims with his legs, trembles, grinds his teeth, his eyes are watery, his mucous membranes are red. Eventually they die.

When the disease is acute, they have general depression, lethargy, anorexia, fever up to 41 0C, bloody mucous diarrhea, ataxia, paralysis of the legs. Symptoms of central nervous system disease are observed in sheep, they walk forward, fall and fall again, swim with their legs, lie unconscious. His head falls back as a result of the contraction of his muscles. A foamy mucous substance flows from the mouth. Anemia in the mucous membranes, blood in the urine. They urinate involuntarily. Gastrointestinal activity slows down, the patient quickly weakens and dies after 2-3 days.

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Chronic shedding is more common in lean sheep. Sick sheep become weak, do not eat anything, the mucous membranes bleed, the sheep becomes silent and sleepy. And the lambs lose their appetite, become lethargic, shiver, have colic, have diarrhea and nervous disorders. Sick sheep usually lose a lot of weight. Some of the sick sheep can sometimes recover.

Cl. In sheep infected with Perfringens type C, a hemorrhagic condition is observed in the intestines and parenchymal organs, which is called hemorrhagic enterotoxemia. Cl. In sheep infected with perfringens D (epsilon - toxin) type, toxic condition is often observed and it is called infectious enterotoxemia. In this case, the disease can be very acute, acute and chronic, and the condition of glucosuria is manifested.

Pathologoanatomical changes. A dead body is opened only for the purpose of making a diagnosis. A dead body rots quickly, and if handled sharply, the smell is noticeable. A dark brown spot is visible in the woolless areas. The dead body immediately fills with air within 2-5 hours and begins to grow. The fur is easily pulled off and the skin has large blue spots. A thick foam mixed with blood flows from the mouth and nose, hemorrhagic swelling and bleeding are observed when the skin is irritated. Mucous reddish fluid accumulates in the chest and abdomen. Hemorrhage is observed in the epicardium. Hemorrhage and inflammation are visible in the mucous membrane of the large abdomen. The lungs are swollen and cystic, the bladder is full of urine mixed with blood, the kidney is cystic, and there is blood under the capsule. The kidney becomes a shapeless mass, a pulpy mass filled with a very loose sac. In old sheep, this condition may not be clearly manifested. When lymph nodes are cut, mucous fluid flows and small necrosis foci are found.

Cl. Perfringens S turi bilan kasallangan qo‘ylarda – ingichka ichaklarda gemorragik yallig‘lanish va buyrakni yumshashi, Cl. Perfringens D turi bilan kasallangan qo‘ylarda esa, o‘zgarish faqat buyrak parenximasini - shaklsiz bo‘tqasimon massaga aylanishi kuzatiladi. Bu holat, ayniqsa qo‘zilarda kuzatiladi.

Diagnosis. The diagnosis of enterotoxemia is made on the basis of epizootological data, clinical signs, pathologoanatomical changes, and, of course, the results of laboratory examination. A dead body can be opened in a special place only for diagnosis. In the laboratory examination, pathogens and their toxins are isolated from parenchymatous organs and small intestines and identified using special blood serum.

At the same time, the presence of a toxin (stimulating product) is detected in the small intestine. The body of a dead sheep, parenchymatous organs or a piece of the small intestine taken in a state where both sides are tied are sent to the laboratory. The examination is carried out by finding the presence of a toxin in the intestine and its trigger. For the first method, a 1:1 or 1:2 physiological suspension is prepared from a piece of intestine, which is filtered. To determine the type of toxin, 1 ml of filtrate is taken in 5 test tubes and 1 ml of antitoxin serum is added. The first test tube contains "A" serum, the second "C", the third "D", the fourth "E", the fifth 1 ml of physiological solution. 0.5 ml of this mixture is injected into the abdomen of 2 white mice or 0.2 ml into the skin of guinea pigs or rabbits. Rabbits get necrosis, guinea pigs die.

For bacteriological examination, a smear is prepared from the mass of the small intestine or parenchymatous organs, stained by the gram method, and from parechymatous organs, bone marrow is cultured in Kitt-Tarotsi, GPB, GPA, and Kitt-Tarotsi media from the intestine and grown at 37-38 °C. will be deleted. Cl. One of the important diagnostic signs of perfringens type D is the presence of sugar in the urine (glucosuria).

Differential diagnosis. It is necessary to distinguish enterotoxemia from bradzot, anthrax, pasteurellosis, listeriosis and food poisoning, piroplasmosis. In Bradzot, hemorrhagic inflammation, ulcer pus, necrotic cells are observed in the duodenum, liver, and there is no toxin in the intestines, and the kidney is not emptied. In case of anthrax, the spleen enlarges, the pulp becomes soft, and when cut, the pulp becomes similar to degt. When pasteurellosis is semi-acute and chronic, septic process and pneumonia are observed. In listeriosis - there is no toxin in the intestine. Many animals are affected by food poisoning. In piroplasmosis, the parasite is seen in erythrocytes. In all cases, laboratory methods - bacteriological, serological and toxicological tests are the basis for making a final diagnosis.

Treatment. Enterotoxemia is difficult to treat because it is acute and acute, and in chronic cases, bivalent hyperimmune blood serum is administered (antibiotics are also added). Some experts have obtained good results by injecting 2-2.5 mg/kg of biomycin intramuscularly 4-5 times. Sintomycin was given to large sheep at 0.5-1 mg/kg, and to lambs at 0.2 mg/kg. K. Riskulov (1983) recommends prolonged antibiotics for the treatment and prevention of the disease. He states that it is very appropriate to add it with bivalent blood serum. When mixing 0.25-1 kg of cormogrizin, 0.5-1 kg of biovit, 1-1.5 kg of baxicillin in 1 s of feed from antibiotics, lambs were protected from disease (the disease rate decreased by 4.2 times). Intramuscular injection of dibiomycin and tetracycline works well.

Immunity. For active immunity, the concentrated GOA formalvaccine (F.Kagan, A.P. Kolesova) is used to vaccinate sheep and goats against polyvalent bradzot, enterotoxemia, malignant tumor and dysentery. The vaccine is administered 2 times with an interval of 12-14 days and preventive vaccination with an interval of 20-30 days. Immunity appears in 12-14 days and lasts for 6 months. Polyanatoxin is also used against sheep clostridiosis (L.V. Kirillov, F.I. Kagan). If this drug is vaccinated 2 times with an interval of 30-45 days, the immunity lasts for 10 months. If the anatoxin vaccine against Cl. Perfringens type D (A. Volkova) is vaccinated subcutaneously 2 times with an interval of 12-28 days, immunity is maintained for 4-5 months. It is also possible to use the method of complex vaccination of sheep against bradzot, enterotoxemia, anthrax and smallpox.

Prevention and countermeasures. For prevention, it is necessary to control the veterinary-sanitary condition of cattle, pastures and drinking places. Factors contributing to the development of the disease are eliminated. Sheep should not be driven to pasture in early spring when the grasses appear. At this time, it is necessary to give dry hay. If it is not possible, the sheep can be put out to pasture after the frost or dew has lifted. It is necessary to consider all areas of enterotoxemia and to vaccinate sheep in the spring, 30-45 days before putting them on pasture.

If this disease is detected clinically, pathologically, anatomically, bacteriologically among sheep, the herd or farm is declared unhealthy and a restriction is placed on it under the Veterinary Regulations. At the unhealthy point, all containment measures and measures to prevent the spread of the disease are taken. Entry and exit of new sheep to the farm, shearing, and mixing of sheep with other groups are prohibited.

After the disease is detected in the herd, it is transferred to another pasture, it is not driven to far places. Sheep in the flock are clinically examined. Diseased and suspected animals are isolated and kept in pens and treated with special, hyperimmune blood serum, symptomatic and antibiotics. Clinically healthy sheep are left in the pen, vaccinated, given coarse hay and mineral salts. The cage is disinfected weekly with 3% chlorinated lime, 3-5% caustic soda, 10% formaldehyde, and monochlorinated iodine. Dead bodies are burned without removing skin and fur. Sick sheep are not slaughtered for meat, wool and milk are not taken. The ban from the farm is removed after 20 days of the end of the outbreak and recovery, after all measures and final disinfection have been carried out.

Summary

1. The causative agent of infectious anaerobic enterotoxemia of sheep is highly contagious, as the causative agent of the disease spreads in an epizootic form.
2. Infectious anaerobic enterotoxemia of sheep occurs in our country in all seasons of the year, many susceptible animals die as a result of the disease.
3. Timely identification of the causative agent of infectious anaerobic enterotoxemia of sheep, study of the epizootology of the disease, and prevention of economic damage caused by it are one of our important tasks.

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