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CLINICAL SYMPTOMS, DIAGNOSTICS AND PREVENTIVE MEASURES OF MONIEZIOSIS IN SMALL-HORNED ANIMALS

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Abstract: In this article, the etiology, pathogenesis, clinical signs of the disease, pathologoanatomical changes, diagnosis, treatment and preventive measures of moniezosis in small horned animals were studied.
Key words: moniesiosis, etiology, pathogenesis, enzootic, metabolism, intoxication, diarrhea, macroscopic, microscopic, clinical, morphological, helminthoscopy, helmintoovoscopy, helminto-caprological, laboratory, pathomorphology, epizootological data, pathologoanatomical, antiparasitic, prevention, disinfection, disinsection .

Relevance of the topic: Monieziosis is an acute and chronic cestodosis disease of ruminants, which is caused by the parasitism of pathogens belonging to the genus Moniezia in the small intestines of animals, the disease is weakening, digestive organs It is characterized by functional impairment, severe diarrhea, sometimes nervous system failure, retardation of growth and development, a sharp decrease in productivity and death of young animals (especially young lambs and goats). The disease is usually enzootic. more lambs, goats and calves die than infected animals. Monieziosis occurs in 20-21% of sheep herds. According to experts, 5-7% of sheep infected with this disease will die. Due to the severity of the disease in young animals and the specificity of the causative agent, a sharp decrease in productivity in infected animals and stunted growth and development in young animals, cases of death from the disease occur, and large quantities are used to control the disease. the spending of funds is causing great economic damage to farms.[1,3]

Causative agent: Moniezia expansa is a milky-white cestode, up to 10 m long, up to 1.6 cm wide, adapted to parasitize animals up to 6 months old. **M. venedeni** is a whitish-yellow cestode, up to 4 m long and 2.6 cm wide, parasitizing animals older than 6 months. **M.autumnalia** is also a yellowish-white cestode, up to 3 meters long and 8 mm wide. A parasitic biohelminth, the main hosts are ruminants, mainly sheep, goats and cattle, and sometimes other ruminants, and the intermediate hosts are oribatid (soil) mites.[1,2]

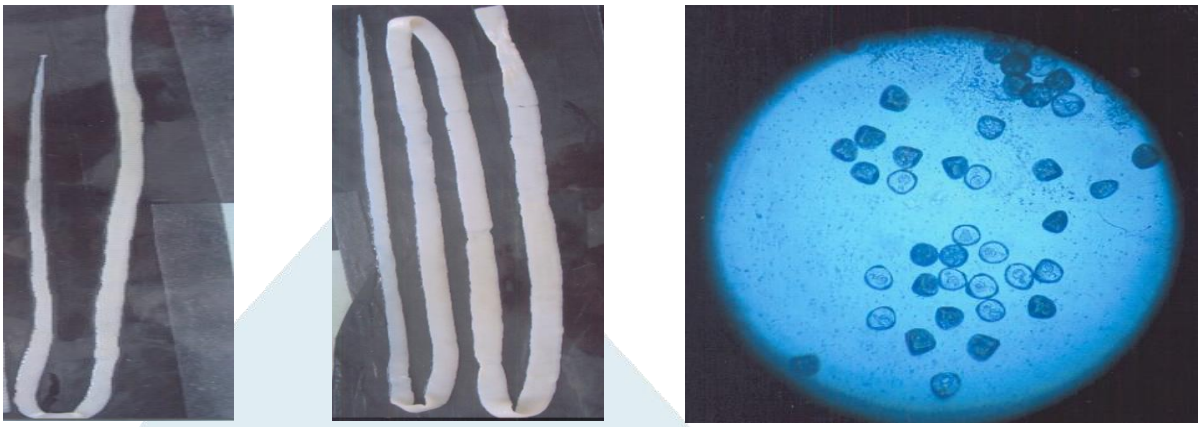

Fig. 1. Macroscopic and microscopic appearance of the causative agent.

Epizootology. The spread of monieziosis among animals depends on their age, especially in lambs, goats and calves aged 1.5-8 months. For example, 59% of one-year-old cattle were infected, 31% of one- to two-year-old cattle, and only 15% of cattle over two years old were infected with monieziosis. Lambs are infected first with M. expanza and then with M. benedeni. Adult cattle are rarely infected with monieziosis, M. benedeni is more common in them. Monieziosis is a widespread disease in the conditions of Uzbekistan, which mainly affects young cattle.[1,3] Clinical signs: the working capacity of the digestive (metabolism) organs of an animal infected with monieziosis is disturbed. Animal dung changes and loses its color. Mole is often aggravated, has diarrhea (diarrhea). The area around the rear exit hole (anus) of sick cattle becomes polluted, the animal loses appetite, loses weight (atrophy) and stops growing despite being given nutritious food. Most often, swellings appear in the chest and abdomen. Their wool loses its luster, becomes brittle and breaks and sheds.

Sheep infected with M. expanza are distinguished by four different clinical forms of the disease - severe and mild toxic, intestinal obstruction (obturation) and nervous manifestations. In case of severe toxic symptoms, mainly lambs, and partly also sheep are affected. In this case, the stool becomes liquefied and covered with mucus. After 10-15 days, the clinical signs become more pronounced, the lambs become entrails, become weak and lie down. Their excrement becomes liquefied, dark earth-colored, and mixed with mucus and blood, it contaminates the back of the body, the area around the buttocks, and the inner surface of the thigh.The sick animal becomes anemic, stops growing, and as a result of the deterioration of the heart's function, swellings appear in the lower parts of the body. As a result, lambs begin to die from May-June. A mild toxic clinical manifestation of monieziosis is observed in young cows and lambs of the future, they become pale, emaciated, and the dung becomes liquid. A sick animal suddenly turns around and starts kicking with its legs. The animal dies of self-poisoning (intoxication) as a result of intestinal obstruction during one of these seizures. In the nervous form of the disease, the lambs suddenly become dizzy, fall to the ground and die after one or two hours due to convulsions (tetanic), shivering, as well as in the mixed form of monieziosis.[1,3,4]

Pathologo-anatomical changes: the body of the animal is emaciated, anemic, the interstitial area is contaminated with feces, and it is possible to see moniesian joints. In Bundy's condition, intestinal wall stretching, intestinal intussusception, restlessness, catarrhal inflammation, proliferative-generative changes are formed in the intestinal mucosa and salivary glands, kidneys, spleen, sometimes in the liver. It is determined that there are free-eaters in the cavity of the small intestines. Accumulation of blood fluid in the brain and bleeding is observed, and fluid (transudate) accumulates under the skin, in the abdominal and chest cavities.[1,2,4]

Diagnosis: The disease is diagnosed based on anamnesis data, clinical signs, epizootological data, pathologoanatomical changes and laboratory tests. In particular, examinations were carried out by helminthoscopy and helmintoovoscopy, helminto-caprological and helminthological dissection. [1]

Comparative diagnosis: We must distinguish this disease from Tizanieziosis, anoplocephalosis of ungulates.

Treatment. Sick animals are isolated and the causes of the disease are eliminated, and easily digestible feed should be provided with green grass, crushed tubers, bran or silage, and clean water should be available at all times.

From the first day of the disease, symptomatic treatment should be carried out together with comprehensive measures against parasites.

Disease prevention and prevention: separation of sick animals from the herd, timely deworming, introduction of veterinary sanitary measures (disinfection, disinsection), feeding

animals on a full-value diet to increase resistance, and disease prevention it gives good results in getting. [1,4]

Conclusion and recommendations for practice. Analyzing the epizootology of moniesiosis according to the season of the year, it was noted that lambs are maximally affected by moniesiosis in spring and autumn, and the invasion by its causative agent (M. expansa) is slightly reduced in summer. Moniesiosis is also observed in the winter months, its extent and the course of the disease depends on the abiotic environmental factors (temperature and humidity level) in the area. The most important thing is that there is no infection with moniesia in sheep kept only in severe winter, where the ground is frozen. In most cases, such unfavorable weather is rarely observed during the whole winter (December, January, February), therefore, moniesiosis should not be considered a seasonal disease. Moniesiosis is usually severe in young lambs, and mortality depends on early or late spring season, long or short winter season, long fall season and rainfall. The death of young sheep from this disease occurs mostly in spring, but it can be observed both in winter and autumn. It is advisable to carry out hydrometeorological and phenological observations to determine them. In order to prevent the spread of monieziosis and its causative agents, deworming sheep in winter, spring and autumn months, applying anthelmintic copper and salt mixture to them in these seasons. Use of drugs containing praziver, monizen, nilsash forte, iverpraz, albencloz 12.5% and febendazole in the treatment of moniosis or preventive deworming. After 3-4 hours of deworming, keep the sheep in the pen for at least 12-18 hours.

Fig. 2(A)

Fig. 2(B)

Figure 2. Medicines used in the treatment (A) and prevention (B) of the disease.

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