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PATHOLOGICAL ANATOMICAL CHANGES IN SAIGAS INFECTED WITH COENUROSIS CEREBRALIS

ANNOTATION

This article describes an autopsy of an animal infected with coenurosis, a parasitic disease caused by the larval stage of the tapeworm. A cadaver infected with cenosis showed moderate hardening, the abdomen was swollen and the eyes were closed. The spleen of all sick animals is softened and the tissue structure is disturbed. Examination of the subcutaneous fat showed signs of body fat, but they were mottled with red dots. The lymph nodes were also swollen, dark red, moist, mottled with tiny red dots, with red fluid running along the surface. The lungs were enlarged, the consistency was loose, the serous membrane had many dark red dots, and bloody fluid was leaking from the surface. The heart was found to be loose, enlarged, with soft consistency. The liver was whole-blooded, enlarged, with many dark red dots on the surface, and the gallbladder was moderately full, without stones or parasites.

On examination of the digestive tract we saw that the stomach was swollen due to a brown fluid content, the intestinal mucosa was damaged and swollen due to the fluid content within, and the intestinal mucosa was swollen, dark red and thickened in places. The brain tissue is hyperemic and oncospheres are found in tubes twisted across the surface and depth of the brain. In the early stages of cenurotic disease, newly formed haemorrhages and hyperaemia were observed in some parts of the cerebral membranes. A detailed description of the pathological anatomical changes that occurred as a result of the disease is given in the article.

Key words: *coenurosis cerebrialis, saiga, Ural population, internal organs, autopsy.*

Introduction. Coenurosis cerebrialis is a dangerous invasive disease mainly of sheep, less often goats, cattle, camels, domestic yaks, buffalo, horses, and in isolated cases even humans [1-8]. The causative agent of this disease is Coenurosis cerebrialis, the larval stage of the cestode *Multiceps multiceps* [1,2,3]. The parasite is localized in the brain and less frequently in the spinal cord of animals, and in its final stage of development it takes the form of a large bladder filled with fluid and pressing on the organ tissue. The clinical symptoms of the disease are various manifestations of nervous reactions, such as blindness, spinning syndrome, paralysis, paresis, loss of locomotor control and, later on, coma and death. The tapeworm stage of the cestode is parasitic in the small intestine of wild and domestic dogs [4]. The tapeworm stage of cestode is localized in the small intestine of the definitive host. In its final stage of development, it takes the form of a large bladder filled with fluid and pressing on organ tissues [5]. Most often it affects young animals under two years of age. The tapeworm enters the animal body through grass, water, and hay. Most often, the definitive host is dogs guarding flocks of sheep. Dogs can excrete up to 500,000 eggs daily. The disease develops from inadequate sheep housing conditions. Improper feeding reduces immunity and allows the pathogen to enter the body. The disease has four stages. During the first stage there are no visible signs of the disease, infection can be determined by allergic reaction from the 12th day. During the second stage the animals show jitteriness, tonic and

clinical convulsions, forced postures. During this time, oncospheres penetrate the brain and bladder growth is observed. The third stage is due to the increased growth of the coenurosis, which presses on the surrounding tissue. The final fourth stage is characterized by intensive growth of the bladder and lasts 1-2 months and ends with the death of the animal [5,6,7].

The disease is widespread throughout the world in regions with intensively developed sheep breeding. The circle of intermediate hosts in the wild is very wide and depends on the area of infestation distribution. In the northern regions of the globe, they are deer, argali, roe deer, mouflon, yaks, hares; in the southern regions - antelope, chamois, rabbits and other animal species [7,89].

In the CIS, coenurosis is distributed unevenly. In the territory of the Caucasus, coenurosis affects 10-20% of livestock, in Karachay-Cherkessia 15.8% (2013), in Kalmykia 10-12% (2014)[3], in Dagestan 22-46%. In Bashkiriya, up to 47.3% (1991) of the population; in Buryatia, 50% of the population (1982). In Turkmenistan - 18.7% (1998). In Uzbekistan - 11.4% (2001). Such unevenness in the spread of infestation is often caused by grazing sheep on the same pastures for many years. Where there is a change of pastures, the infestation does not exceed 3% of the flock.

If all cases of coenurosis in CIS are considered as 100%, Kazakhstan accounts for 40%, Central Asia - 10%, North Caucasus - 18%, Middle and Lower Volga - 15%, and other southern regions for the remaining 17% [9].

Often, the diagnosis of acute coenurosis is difficult due to the implicit symptoms or complete absence of clinical signs of the disease. During this period of the disease, lamb mortality is not taken into account and, in fact, losses from coenurosis may be much higher. Chronic coenurosis has a very characteristic course and is usually easily diagnosed [10,11,12].

When diagnosing, the data on the incidence of coenurosis in a given region are recorded. The study of clinical symptoms in sick animals with comparison of their timing of manifestation with the development cycle of the parasite, laboratory examination of feces of primate dogs according to the method of Füllerborn in order to detect mature multiceps proglottids and tenid eggs are carried out. Blood tests, cerebrospinal fluid of sheep suspected of the disease, ocular fundus examination of affected animals for the presence of congestion in the optic nerve papilla can be informative in detection of early coenurosis [13,14].

Ophthalmoscopic diagnostics: 1-2 months before the appearance of clinical signs of the disease in animals ophthalmoscopy can reveal edema of the eye fundus, smoothing of the optic nerve papilla borders, their gradual fusion with retina, sharp filling of venous vessels, congestion and pinpoint hemorrhages both along the vertical vessels and on the optic nerve papilla. Retinal color becomes yellow with greenish tint [15,16,17].

Coenurosis of sheep should be distinguished from rabies, listeriosis, sarcocystosis, infectious encephalomyelitis, brain polyencephalomalacia with magnesium deficiency, and brain abscesses [18,19].

It is also important to distinguish coenurosis from other larval tenidoses of animals, such as cysticercosis of sheep, which also occurs quite frequently in the brain of animals. As a rule, the cysticercus bladder is much smaller than the cenicerous bladder; in addition, the cenicerous bladder contains several protoscolexes, which distinguishes it from the cysticercus, which contains a single protoscolex [20,21].

In recent years, there has been a significant change in the actual helminth composition of sheep, depending on various factors. One of these factors is wild animals inhabiting the territory of West Kazakhstan region. The maximum of these is saigas. The "Ural population" of saigas mainly inhabits the southern districts of West Kazakhstan region (Kaztalov, Zhanibek).

In recent years, the Saiga "Ural" (*Saiga tatarica*) population in West Kazakhstan region has been unstable, with clinical signs and pathological changes of helminthological diseases among saigas.

The aim of the study was to investigate pathological changes in saigas infected with coenurosis.

Materials and methods of research.

The carcasses of saigas infected with coenurosis in the Kaztalovsky and Zhanibeksky districts were examined by sampling in the field. Official data from veterinary services and Okhotzooptom inspectors were also used.

The research was conducted in the pathology and anatomy laboratory of the Higher School "Veterinary Clinical Sciences" of the Institute of Veterinary Medicine and Animal Husbandry of the Zhangir Khan West Kazakhstan Agrarian and Technical University (Uralsk, Kazakhstan).

Results and discussion. At autopsy, the animal exhibited moderate rigor mortis, the jaws slightly immobile, the abdomen taut and moderately enlarged (Figure 1).

The eyes were closed, there was no ocular effusion, the conjunctiva was anaemic, the cornea was smooth, slightly cloudy, and the pupils were dilated. Nasal oozes were foamy, red, the nose was intact and dry. Oozes from I mouth, anal and preputial orifices are absent, uninjured.

Negative changes in the appearance of dead and killed sick animals, matting of the hair and visible emaciation in varying degrees were noted; dead animals had no fat deposits in the subcutaneous tissue, heart and omentum. Internal organs were normal, but the spleen in all sick animals was soft and tissue turgor was reduced. Examination of subcutaneous fatty tissue revealed that fat deposits were well expressed, the subcutaneous tissue was saturated with red-yellow fluid and streaked with red dots the size of a matchstick. Superficial lymph nodes are enlarged, swollen, dark red, moist, streaked with small red dots, edges not converging, red fluid draining from the surface. In the liver, small whitish-whitish twisted strands and foci are detected at the site of death of oncospheres. The same formations are found in the muscles of the heart, kidneys and other organs. The carcasses of sheep killed by coenurosis in the third and final stage of the disease are emaciated.

Internal examination revealed that the organs in the thoracic and abdominal cavities were correctly positioned. The thoracic and abdominal cavity showed red-brown fluid, mucous membrane moist with red dots; diaphragm intact; pleura dull, opaque, covered by a film. No oral contents detected, mucosa pale, without ulceration or lesions. Laryngeal cartilages and tracheal rings intact. The contents were liquid, white, without blood. The mucous membranes of the trachea and larynx were pale and intact. The lungs are enlarged in volume, not collapsed, flabby consistency, red-brown, the edges do not converge, the serous membrane is covered with multiple dark red dots the size of a matchstick; bloody liquid flows from the surface of the section, pieces of lung float in water. The bronchial and mediastinal lymph nodes are enlarged, dark red, of a loose consistency, with reddish fluid draining from the incision. On examination of the heart and the pouch, blood in the heart was found to be unconverted, tarry thick consistency, dark in colour, the serous membrane of the endocardium, myocardium and epicardium were streaked with multiple black-red small dots. The heart is flabby, enlarged in size, of soft consistency. After opening the heart, thick black-red blood was found in the cavity of the left ventricle; there were no dense connections and rough areas where the clots were in contact with the endocardium. The endocardium is not enlarged, elastic, without overlap, the valves are easily detached. The vascular lumen is dilated, the walls are thin, and there is black-red coagulated blood inside.

The liver is full-blooded, enlarged in volume, the edges are blunt, not convergent, reddish-brown in colour on the cut, lobular structure is flattened, under the capsule and on the surface of the cut there are multiple dark red dots the size of a matchbox, the consistency is flabby, dull. The gallbladder was moderately full, the wall was not thickened; no stones or parasites were found in the gallbladder or bile ducts. The pancreas is not enlarged, there are no stones, cysts or impositions. The kidneys are enlarged in size, dark brown in colour, of dense consistency, the capsule is easily removed, there are red dots on the surface, under the mucosa in the pelvis there is grayish-red fluid, the border between the cortical and cerebral layers is smoothed, the edges of the incision do not coincide. The bladder mucosa is pale, not thickened, with cloudy yellow contents of an unpleasant odour.



Figure1 – Saiga autopsy

Stomach enlarged, mucous membrane swollen, with coffee-coloured liquid contents inside. The reticulum is reddish, enlarged, with red small dots; the mucosa of the abomasum is folded, swollen, with liquid contents inside. Duodenal mucosa swollen throughout, dark red, thickened in places and impregnated with jelly-like mass of brown. The mucous membrane of the jejunum is swollen and bloody. The mucous membrane of the ileum was swollen throughout, dark red, thickened in places and impregnated with a jelly-like mass of brown. The contents of the small intestine were liquid and coffee-coloured. The examination of the large intestine revealed that the mucosa of the cecum and colon were swollen with small, dark red dots. The mucosa of the rectum was dark red with small cracks, swollen, much of the rectum was bulging; the contents were a frothy black-red fluid.

In the initial (acute) period of coenurosis the tissue and membranes of the brain are hyperemic.

On the surface and in the depth of the brain tortuous ducts (traces of migration of oncospheres) are found. At the ends of these passages, cenocentruses in the initial stage of development can be detected. In the brain and spinal cord, solitary cenurids are usually found cenuruses. Cenurus found in the spinal cord are usually elongated in shape. When animals are slaughtered at the beginning of the disease, traces of oncosphere migration and intense hyperaemia with fresh haemorrhages are noted on the dura mater in some areas. The brain tissue at the locations of the parasite is atrophied. In case of location of coenurosis on the surface of the brain, the skull bones above the developed bladder are thinned or pierced.

Due to the lack of illustrative material on saiga coenurosis, without which it is very difficult to interpret the characteristic signs of the disease correctly. There is little detail in the literature on the nature of ophthalmoscopic examination in sheep coenurosis, and ophthalmoscopy provides a full opportunity to differentiate coenurosis from other diseases.

In scientific studies, coenurosis in saigas was manifested by visual disturbances, congestion in the fundus of the eye, blood overflow, and haemorrhages on the retina and nipple of the eyeball.

For an accurate diagnosis of saiga coenurosis, the clinical and pathomorphological manifestations of the disease, irrespective of its form, were taken into account at an early stage.

At post mortem examination of saiga carcasses, close attention was paid to the detection of elongated coenurosis up to 4 cm long in the brain, and the connective tissue of the brain was atrophic at the helminth sites.

On the surface and in the substance of the brain, sinuous passages were found - traces of oncosphere migration - in which rudiments of cenocentruses the size of a pinhead were found, and the skull bones as a whole above the cenotic bladder were thinned. For an accurate diagnosis of saiga coenurosis, additional records of pathomorphological changes in the organs were made:

- ophthalmoscopic examination of the optic nipple, as patients with saiga coenurosis showed visual disturbance, congestion in the fundus of the eye, blood overflow, haemorrhages on the retina and nipple of the eyeball, and colour changes;

- liver pathological changes in the form of granular dystrophy, detection of small whitish-yellow necrotic foci;

- overfilling of the gall bladder.

Consideration of these features of saiga coenurosis greatly increases the efficiency of pathomorphological diagnosis of the disease, and makes it possible to recognize invasion by its pathognomonic signs at autopsy of saiga carcasses.

To confirm the characteristic pathanatomical changes, we present (Figures 2-4).



Figure 2 – Blood capillaries in the saiga brain injected

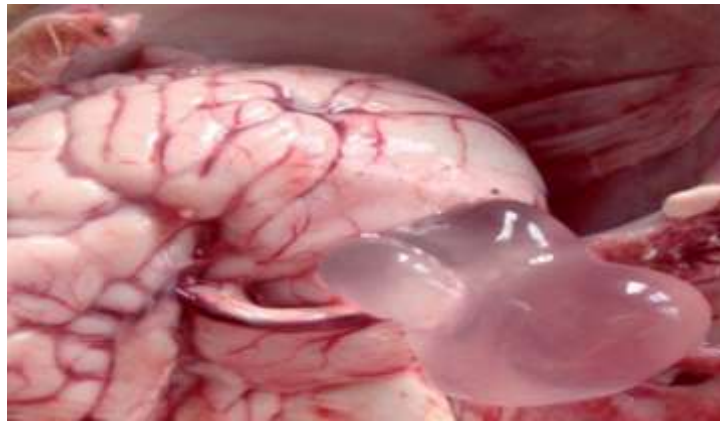


Figure 3 – Single, elongated cenocentrus in the brain



Figure 4 – Ophthalmoscopic examination of the eye

Conclusions. Saiga coenurosis occurs in the Ural population of saigas inhabiting the West Kazakhstan region, and occurs in acute and chronic forms. The acute form of the disease can be distinguished from other similar diseases by pathological anatomical examination. The chronic form is characterised by the formation of vesicles in the brain.

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ТҮЙІН

Мақалада таспа құрттың дернәсілдік сатысынан туындаған паразиттік ауру – ценуроз жұқтырған жануардың жарып-союы сипатталған. Өлекседе орташа сіресу байқалды, іші ісініп, көздері жұмылған. Барлық ауру жануарлардың көкбауыры жұмсарған, тіндердің құрылысы бұзылған. Тері астындағы майды тексеру кезінде дене майының белгілері анықталды, бірақ олар қызыл нүктелермен дақталған. Лимфа түйіндері де ісінген, қою қызыл, дымқыл, ұсақ қызыл нүктелермен дақталған, олардың бетінен қызыл сұйықтық аққан. Өкпе үлкейген, консистенциясы бос, серозды қабығында көптеген қара-қызыл нүктелер көрінеді, бетінен қанды сұйықтық аққаны байқалды. Жүрек босаған, үлкейген, жұмсақ консистенциялы екені анықталды. Бауыр толық қанды, үлкейген, бетінде көптеген қою қызыл нүктелер бар, ал өт қабы орташа мөлшерде толтырылған, тастар мен паразиттер жоқ.

Ас қорыту жолын тексеру кезінде асқазанның қоңыр түсті сұйық құрамымен ісінгенін, ішектің шырышты қабаты бүлінгенін және ішіндегі сұйық құрамымен ісінгенін, ал ішектің шырышты қабаты ісінген, қою қызыл және кейбір жерлерде қалыңдатылғанын көрдік. Ми тіндері гиперемияланған, ал онкосфералар мидың бетінде және тереңдігінде бұралған түтіктерде табылған. Ценуроз ауруының бастапқы кезеңінде ми қабығының кейбір бөліктерінде жаңадан

түзілген қан кетулер мен гиперемия байқалды. Мақалада ауру нәтижесінде болған патологиялық-анатомиялық өзгерістердің толық сипаттамасы берілген.

РЕЗЮМЕ

В статье описывается картина вскрытия трупа животного, инфицированного паразитарным заболеванием - ценурозом, вызванного личиночной стадией ленточного червя. В зараженном ценурозом трупе животного наблюдалось умеренное окоченение, живот вздут, глаза запавшие. Селезенка у всех больных животных размягчена, нарушена структура тканей. При осмотре подкожной клетчатки были обнаружены признаки скопления жира и были пропитаны красными точечными кровоизлияниями. Лимфатические узлы увеличены, темно-красной окраски, влажные, точечные кровоизлияния, с поверхности которых стекает красноватая жидкость. Легкие увеличены, консистенция рыхлая, серозная оболочка имеет множество темно-красных точек, на разрезе с поверхности стекает красноватый экссудат. Сердце дряблой консистенции, незначительно увеличен. Печень увеличен в размере с множественными геморагиями на поверхности, желчный пузырь умеренно заполнен желчью без камней и паразитов.

Во время осмотра пищеварительного тракта, желудок умеренно заполнен кормовыми массами коричневого цвета, слизистая оболочка кишечника повреждена, сочная набухшая из-за содержания жидкости внутри, местами утолщена. Мозговые ткани гиперемированы, а онкосферы обнаружены в трубках, скрученных по поверхности и глубине мозга. На ранних стадиях ценурозного заболевания, в некоторых частях мозговых оболочек наблюдались вновь образовавшиеся кровоизлияния и гиперемия. В данной статье описывается подробное изложение патологоанатомических изменений в результате развития ценурозного заболевания.