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Stepan Gzhytskyi National University of Veterinary Medicine and Biotechnologies Lviv

Department of Epizootology

EDUCATIONAL AND METHODOLOGICAL MANUAL FOR CODUCTING LABORATORY CLASSES ON SPECIAL EPIZOOTOLOGY ON INFECTIOUS DISEASES OF RUMINANTS AND PIGS

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Recommended by the Lviv National University of Veterinary Medicine and Biotechnology named after Stepan Gzhitskyi as a teaching and methodical manual for students - master's degree in specialty 211 "Veterinary Medicine"
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The manual covers material on 39 infectious diseases, including: 12 infectious diseases of cattle (leukemia, emphysematous carbuncle, paratuberculosis, campylobacteriosis, contagious pleuropneumonia, plague, malignant catarrhal fever, infectious rhinotracheitis, parainfluenza, viral diarrhea, spongiform encephalopathy, nodular dermatitis) , 11 infectious diseases of small cattle (bradzot, infectious enterotoxemia, infectious agalactia, infectious catarrhal fever, hoof rot, contagious pustular dermatitis, infectious epididymitis, smallpox, visna-maedi, scrapie, malignant edema), 16 infectious diseases of pigs (classic and African swine fever, erysipelas, reproductive and respiratory syndrome, influenza, infectious atrophic rhinitis, vesicular disease, vesicular exanthema, enzootic encephalomyelitis, transmissible gastroenteritis of pigs, colienterotoxemia, dysentery, enzootic pneumonia, hemophilic polyserositis, hemophilic pleuropneumonia of pigs, chlamydia). Issues of diagnosis, treatment, immunoprophylaxis, general prevention and control measures are highlighted.

For students of institutions of higher education in specialty 211 "Veterinary Medicine". Can be useful for postgraduate students and veterinary medicine specialists.

Gratitude for the cooperation to the head of the Department of Ukrainian and Foreign Languages named after Yakym Yarema LNUVMBT named after S.Z. Gzhytsky
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DISEASES OF CATTLE

Topic: Bovine leukemia

(diagnosis, prevention and control measures).

Bovine leukemia is a chronic infectious disease of cattle, which is characterized by hemoblastosis (malignant growth of hematopoietic and lymphoid tissue), as well as a violation of the maturation process of blood cell elements.

Leukemia manifests itself in two main forms:

- *actually leukemia* (lympholeukosis, myeloleukosis, hemocytoblastosis), which are characterized by systemic damage to hematopoietic organs and leukemic changes in the pattern of peripheral blood.
- *reticulosis* (lymphoreticulosarcoma, systemic reticulosis, lymphogranulomatosis), which are characterized by focal or generalized tumor growths of reticular elements of hematopoietic tissue and the absence of deviations from the norm in hematological indicators.

The causative agent of leukemia. Classified as an RNA-genomic oncornavirus that belongs to the family Retroviridae, subfamily Oncoviridae, to the genus C oncornaviruses.

Leukemia virus shows tropism to lymphocytes, agglutinates erythrocytes of mice. It is reproduced in leukocytes. It′s found in colostrum and milk cells of spontaneously diseased animals. The virus cannot be isolated from saliva, nasal mucus, urine, semen. Cattle infected with the leukemia virus remain infected for life, despite the presence of specific antibodies in their bodies. It′s cultivated in the leukocytes of a cattle leukemia patient, in the culture of cells of the spleen of a sheep embryo, in the culture of cells of the lungs of a cow. Unstable in the external environment - it is quickly inactivated by boiling and under the influence of 2% caustic sodium, 3% formaldehyde, a solution of chlorinated lime with a content of 2% active chlorine.

The diagnosis of leukemia is made by a complex method, which includes: epizootological data, clinical signs, patho-anatomical changes and mandatory laboratory tests.

Epizootological data. Leukemias are most common among cattle. Both young and adult animals are affected, but leukemia is most often registered in animals 4-8 years old. The source of the causative agent of leukemia is a sick animal. Already in the early stages of the disease, the oncornavirus is released from the body of cows with colostrum and milk. The leukemic process proceeds slowly and is characterized by stages, which are characterized by successive periods of the course, such as incubation, prodromal and clinical.

The incubation period is considered the time from the moment of infection of the animal to the appearance of specific immunological reactions (registration of

antibodies), which can last from 15 days to 4-6 months. The period from the appearance of immunobiological reactions to changes in the blood picture, which indicate the beginning of a pathological process, is considered a prodromal period, during which the absolute content of leukocytes in sick animals reaches 15.0-25.0 G/l, and lymphocytes 75-80%. The duration of which can be three or more years. The clinical stage of leukemia is the period of full development of the disease, which is characterized by the appearance of hematological changes and the main clinical signs.

The occurrence of leukemia in prosperous farms is associated with the importation of sick animals from farms that are not prosperous in terms of the disease. The causative agent of leukemia can be transmitted to a healthy animal in two ways: horizontally - from one animal to another and vertically - from the mother to the fetus, with the help of various factors. One of the important factors in the transmission of this disease is milk and colostrum, which contain a large number of lymphocytes. The factor of transmission of the leukemia virus, which causes the horizontal route of transmission, is the blood of sick and infected animals, medicinal and prophylactic drugs made from it, and veterinary and zootechnical manipulations, as a result of which the mucous membrane and skin of animals are disturbed and bleeding occurs. It′s possible to infect cattle through tools during mass veterinary activities, which prove that to infect cows with leukemia, it′s enough to inject them subcutaneously with 2,500 lymphocytes from an infected animal (this number of cells is contained in 0.0005 ml of blood). About 10% of calves from mothers infected with leukemia can be infected vertically in the womb.

Clinical signs and course of the disease. The incubation period for experimental infection lasts from 60 to 750 days, and for spontaneous infection - 2-6 years. In the development of the leukemic process, *pre-leukemic, initial, advanced and terminal stages* are distinguished. With the development of the pathological process, the indicated stages follow one after the other.

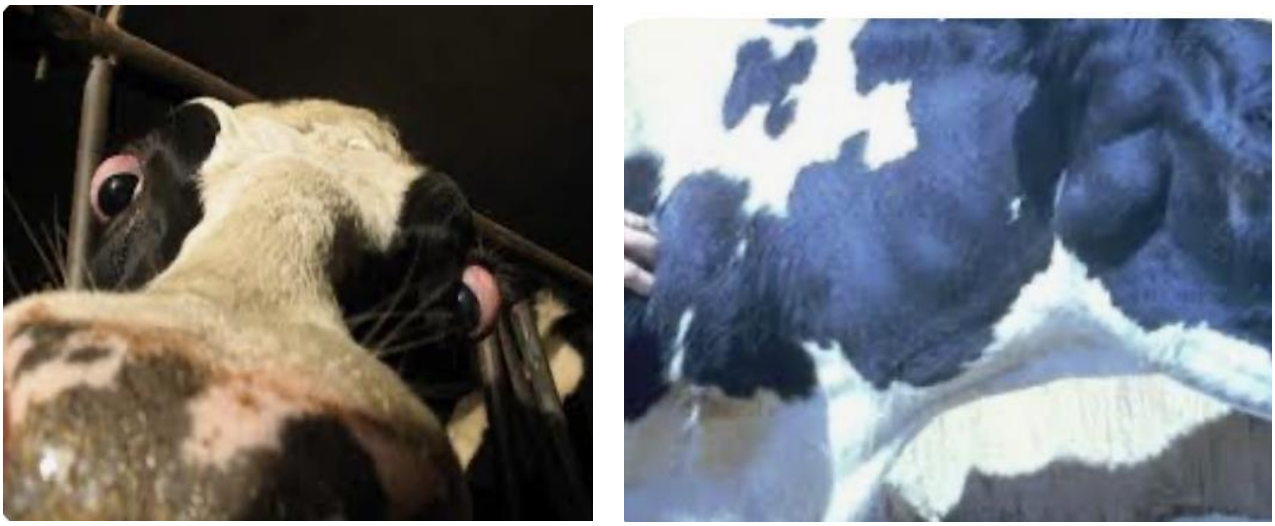
The pre-leukemic stage is diagnosed with the help of serological and virological studies, while no hematological changes are yet detected. *The initial stage* is characterized by systemic or focal damage to the organs of the hematopoietic system (spleen, lymph nodes, bone marrow). At this stage, there are no clinical signs of leukemia. During a hematological examination, quantitative changes in the cellular composition of the blood are noted - an increase in leukocytes, an increase in the percentage of lymphocytes, the appearance of immature and pathologically altered forms of cells.

The advanced stage is characterized, in addition to hematological changes, by various non-specific and specific clinical signs. Its manifestation depends on the morphological forms of leukemia and the location of the pathological process. The general condition of animals worsens, rapid fatigue appears, feed is poorly digested, milk production decreases, exhaustion progresses, digestion is disturbed (diarrhea, constipation, atony, tympany of the antrum), cardiac activity weakens, cyanosis and

jaundice of the mucous membranes are observed, breathing worsens, swellings appear in in the area of the breast, abdomen, udder, lameness on one or both hind limbs, difficulty urinating, abortions occur, infertility occurs, one or more lobes of the udder get bigger.

Specific signs of leukemia are an get bigger in superficial (prescapular, parotid, patellar, submaxillary, suprapubic) and internal (accessible for rectal examination) lymph nodes; the appearance of tumor growths in different parts of the body; exophthalmos - bulging eyes; enlargement of the spleen and liver. Lymph nodes are the size of a walnut to a child's head, they are painless, mobile, elastic and dense (Fig. 1).

In young animals, tumor growths in the lower part of the neck are also noted, the thymus is affected and infiltrative skin lesions appear.



Exophthalmos Enlargement of parotid lymph nodes.

Fig. 1. Clinical signs of bovine leukemia.
(https://dpsscn.gov.ua/.html)

The terminal stage is characterized by the rapid development of the pathological process. Nonspecific symptoms appear. The number of leukocytes in the peripheral blood sometimes decreases, while their pathological forms prevail. This leads to exhaustion of hematopoietic organs, blockade of the immune system and ends in the death of the animal (rupture of the spleen and sudden death due to internal hemorrhage are possible. In such animals, changes in the general condition and productivity are not always detected.

Pathological anatomical changes. In animals that died from leukemia, as well as those killed in the terminal stage, pathological changes are detected in all organs of the hematopoietic system - *lymph nodes, spleen, bone marrow*.

Lymph nodes are enlarged, have a soft elastic consistency, the surface of the section is wet, grayish or yellowish-white with a marble pattern, often reveal bleeding of various sizes.

The size of the spleen is significantly increased, sometimes 8-10 times; the pulp is loose, crimson in color; the cut surface is grainy due to enlarged follicles; there are nodular growths of leukemic tissue of different sizes.

Focal growth of lymphoid and reticular cells in the form of gray and yellowish-green nests of a soft consistency are found in the bone marrow, which displace the red bone marrow and are also located in its fatty part.

In addition to the organs of the hematopoietic system, pathological anatomical changes are found in the heart, liver, kidneys, spleen, intestines, ovaries, and uterus. All of them are quite similar and are characterized by diffuse infiltration by lymphoid cells (increase in the size and mass of the affected organ, change in color) or tumor growth with the formation of limited leukemic foci (round growths of gray or yellow-gray color, protruding above the surface of the organ).

In skeletal muscles, macroscopic changes are rarely detected, they are characterized by infiltrative growth of leukemic tissue, which permeate all or part of it in the form of gray nests and cords.

Laboratory studies. They include hematological, serological and histological studies.

Lifetime diagnosis of leukemia is carried out by a serological method, which consists the detection of specific antibodies in the blood serum, which are products of the reaction - the body's response to the action of a disease-causing agent. The immunodiffusion reaction and enzyme-linked immunosorbent assay are used for serological tests. Polymerase chain reaction is used for the study of especially valuable animals and for arbitration conclusions. The immunodiffusion reaction is the most accessible and simple.

To perform an immunodiffusion reaction, the biological industry produces a set of components, which includes: antigen, positive control serum, agar-salt mixture, antigen and control serum solvent.

The setting of the immunodiffusion reaction is carried out in the following sequence:

1. Agar-gel is prepared and poured into Petri dishes.
2. Punches are used to cut holes in the gel.
3. Fill the holes. After filling the wells, the Petri dishes are placed in a hermetic chamber (desiccator), where with the help of water poured with a layer of 2-3 cm between the bottom of the chamber and the stand for the Petri dishes, a relative humidity of 98-100% is created.
4. Evaluation of the results of the immunodiffusion reaction. Conducted after 24 and 48 hours. The results of the reaction with the tested sera are evaluated against the background of the reaction of the antigen and the control serum. A positive reaction with the tested serum infected with the animal leukemia virus is characterized by the formation of a precipitation line that connects with the precipitation line formed between the wells with the antigen and the control serum. A negative reaction with the

tested serum is characterized by the absence of a precipitation line between the wells with the antigen and the serum.

Hematological tests for leukemia mainly include quantitative and qualitative analysis of white blood cells. Various criteria called "leukemia keys" have been proposed for diagnostic evaluation of the results of quantitative analysis of blood-forming elements (leukocytes, lymphocytes). The limited possibilities of quantitative analysis of formed blood elements are due to the extreme variability of hematological indicators in leukemia. Thus, with the help of "leukemia keys" it is practically possible to detect only 50-60% of animals with leukemia. This makes it necessary to conduct repeated repeated (after 3-5 months) clinical and hematological studies.

In histological sections of leukemia, mature lymphocytes are detected, to a lesser extent prolymphocytes, lymphoblasts and sometimes reticular cells.

Therefore, the main method of lifelong diagnosis of leukemia is serological - immunodiffusion reaction. Hematological and clinical research methods are used to determine the stage of full development of the disease in seropositive animals. Pathological-anatomical studies reveal the degree of damage to organs by malignant neoplasms, and histological studies determine the morphological forms of leukemia.

The diagnosis of leukemia is considered to be established in the presence of one of the following indicators: a positive result of a serological test with an immunodiffusion reaction; typical pathological signs of the disease; a positive result of histological studies of patmaterial. An animal is considered sick if there is a positive result of a serological test based on the immunodiffusion reaction.

Differential diagnosis. When considering the differential diagnosis, it is necessary to take into account that many acute and chronic diseases such as tuberculosis, brucellosis, paratuberculosis, actinomycosis, some invasive diseases, traumatic pericarditis, reticulitis, metritis, mastitis, hepatitis are characterized by significant changes in the blood, which have a protective nature and are defined as a leucomoid reaction of the body, which characterizes functional-reactive changes. This reaction is temporary and disappears when the animal's condition improves.

Treatment. No treatment has been developed for bovine leukemia.

Immunity. Not studied enough.

Prevention and control measures. Comprehensive veterinary and sanitary, organizational and economic and special anti-leukemia measures are included. Herds in which antibodies to the leukemia virus are not detected during serological tests are considered to be positive for bovine leukemia. In such farms, serological research of animals is carried out starting from the age of 6 months. In publicly owned herds, cows are tested for leukemia once a year. Breeding bulls in breeding enterprises and blood-producing animals in biofactories are examined every 6 months. Animals brought to the farm for breeding and use purposes are tested for leukemia serologically during the quarantine period. It is strictly forbidden to import serologically positive animals into

healthy herds. Formation of farms, rental and individual farms is carried out only from serologically negative animals.

A farm in which leukemia has been diagnosed according to the results of two serological tests with an interval of 30-45 days is declared unfavorable for leukemia and quarantine restrictions are introduced in it. When pathomorphological changes characteristic of leukemia are detected in individual animals, a two-time serological examination is carried out with an interval of 30-45 days of all livestock older than 6 months of age. Based on the negative results of the research, the farm is considered prosperous. In case of detection of animals reacting positively to the immunodiffusion reaction, they are isolated in separate rooms. In a dysfunctional herd, it is prohibited to use milk without prior disinfection for public consumption, feeding it to animals, selling it to the state and on markets.

Herds (farms) affected by leukosis are rehabilitated:

1. By simultaneous complete replacement of the unhealthy herd, when more than 30 percent of it is infected, with animals from farms that are healthy in relation to leukemia.

2. By carrying out systematic research with the selection of diseased animals from the herd. Seropositive animals are examined clinically and hematologically within 15 days after the separation of the herd, and thereafter once a year. Animals with hematological or clinical and hematological signs of leukemia are slaughtered no later than 15 days after their detection.

All serologically positive animals are kept and operated in a separate room. Calves are fed mother's milk up to 7 days of age, and then pasteurized milk. In individual farms and farms, animals infected with the leukemia virus are slaughtered. It is forbidden to graze them in common herds and sell the milk obtained from them.

After each examination and isolation of sick animals, rooms and equipment are disinfected with a 2% solution of caustic soda, a 2% solution of chlorinated lime, a 2% suspension of freshly slaked lime, a 5% solution of soda ash, a 2% solution of formalin, and paraformaldehyde. Milk from serologically positive animals, which are kept in isolation from serologically negative herds, is pasteurized on the farm at a temperature of at least 80°C, after which it is used for feeding calves or handed over to a dairy. Milk from serologically negative cows is sold without prior pasteurization. Milk from cows with clinical (hematological) signs of leukemia is forbidden to be used as food and fed to animals. Such milk is denatured by adding a 5% solution of formaldehyde, creolin or another disinfectant to it. Animals with hematological and clinical signs of leukemia are slaughtered at a sanitary slaughterhouse. Manure and wastewater are disposed of on a general basis.

Farm, herd is considered to be healthy after all sick animals have been removed and two consecutive negative results (with an interval of 30-45 days) of serological testing of livestock older than 6 months have been obtained. In the first year after recovery, serological tests are carried out quarterly.

Control questions and tasks.

1. The causative agent of leukemia and its characteristics.
2. Epizootological features of the disease.
3. Stages of the clinical course of the disease and their main clinical signs.
4. Characteristic patho-anatomical changes.
5. Laboratory studies and their essence
6. When is the diagnosis of leukemia considered established?
7. Describe preventive and health measures for leukemia.

Topic: Emphysematous carbuncle

(diagnosis, prevention, control measures).

Emphysematous carbuncle (Gangraena emphysematosa, emkar) is an acute non-contagious infectious disease of large and small cattle, characterized by the development of crepitative inflammatory swellings in the muscles of the body. People can′t get sick.

Emkar is registered in all countries of the world. The specific gravity of this disease among other clostidiosis of cattle in Ukraine is 40%. In the structure of economic losses from emkar, direct losses amount to 11.6%, prophylactic vaccination accounts for 88.4%.

The causative agent of the disease. Cl. Chauvoei is an anaerobic motile polymorphic spore-forming bacillus. It does not form a capsule. It is well stained with aniline dyes, and in preparations from young cultures and from pat material it is positively stained by Gram.

Cultivated only in anaerobic conditions at 36-38°C. The causative agent grows well on Kitt-Tarozzi broth and Marten broth, produces a complex exotoxin with lethal and necrotic properties. On serum agar, the growth of Cl. chauvoei is accompanied by the formation of lenticular or rounded colonies with delicate appendages. In Petri dishes with glucose-blood agar, after 24-48 hours, characteristic colonies of the pathogen appear in the form of mother-of-pearl buttons or grape leaves, surrounded by a zone of transparent hemolysis. In the corpses of animals killed by emkar, including those that have not been dissected, the pathogen forms spores that are extremely stable in the external environment. Spores are stored in the soil for up to 20-25 years, in rotting corpses - up to 3 months, in rotting muscles - up to 6 months, at the bottom of stagnant water bodies - for more than 10 years, in salted meat - for more than 2 years. Direct solar radiation destroys spores only after 24 hours, running steam - after 5-6 hours, boiling - after 2 hours, autoclaving - after 30-40 minutes.

The diagnosis is made by a complex method on the basis of epizootological data, characteristic clinical signs and the results of laboratory studies.

Epizootological data. In natural conditions, cattle and buffaloes, sheep, goats, elk, and deer get sick from emkar. Guinea pigs are extremely sensitive laboratory animals. Horses, pigs, dogs, cats, birds, mice, rats and rabbits are not susceptible to the disease. Animals of any age, mostly meat breeds and well-fed, are sick. However, in stationary centers, the insensitivity of young cattle up to 3 months of age and adult animals over 4 years of age is clearly expressed. Immunity in young animals is due to passive immunity, in older animals it is a consequence of immunizing subinfection. The source of the pathogen is sick animals. In case of untimely or careless cleaning of corpses, emkara spores contaminate soil, water, flooded meadows, pastures, and create long-term (for tens of years) stationary foci of infection. Cattle are infected through the alimentary route, with fodder when grazing on dry grass or prickly stubble, when the mucous membranes of the oral cavity are injured and the causative agent enters the blood without obstacles. Sheep can become infected through skin damaged during shearing, during castration or during difficult births. The disease has a clearly defined summer-autumn seasonality, it becomes widespread in the hot pasture period, when favorable conditions for infection are created. Sporadic cases of emkar disease in animals during the stall period are caused by feeding fodder collected from unhealthy meadows. Emkar passes mainly sporadically, in epidemic centers - in the form of enzootia. Lethality can reach 85-95%.

Clinical signs and course of the disease. The incubation period lasts 1-2 days, sometimes up to 5 days. The course of the disease in cattle is always acute. The disease is manifested by a sudden increase in body temperature to 41-42°C, severe depression, loss of appetite, and cessation of chewing gum. The pulse is frequent, the breathing is accelerated, the mucous membranes are cyanotic. At the same time or before the appearance of general symptoms of the disease, movement disorders, lameness, stiffness of the joints, and dragging of the limbs are observed in animals. Inflammatory swellings appear in the lumbar area, shoulders, lower back, less often the chest, neck, submaxillary area, which quickly increase in size. At first, swellings are limited, dense, hot and painful, later they become cold and painless. A tympanic sound occurs during percussion of the swelling, and crepitation is detected during palpation. The skin on the surface of the edema loses its elasticity, becomes dry, acquires a dark brown or black color. Regional lymph nodes increase in size. In the case of an accidental dissection of the swelling, a characteristic foamy liquid of dark red and later black color with a specific smell of rancid oil is revealed. The disease lasts 12-24 hours, sometimes it lasts for 3-6 days. Recovery is very rare.

In sheep, the course of the disease was subacute. Swellings are voluminous, dough-like consistency, crepitate. Stiffness of gait, lameness is observed. The death of the animal occurs after 6-24 hours.

Pathological anatomical changes. During the external examination of the corpse, severe swelling due to the formation of gases, the outflow of foamy bloody liquid from the nostrils, vagina, and rectum is determined. The skin is tense, has a gray-

bluish color, sometimes necrotic. If there is no doubt about the correctness of the diagnosis, the corpse, due to the possibility of significant spread of spores, is not dissected, but burned together with the skin. If necessary, only a partial autopsy is performed, the pathological material for research is taken near a pit specially prepared for burning a corpse. At the autopsy, large, irregularly shaped gas edemas are found under the skin and in the muscles in the area of the croup, chest, neck, shoulders, and sometimes in the submaxillary space, which contain a gelatinous bloody transudate with gas bubbles (Fig. 1)..

Fig. 1. Gas edema.
(http://milkua.info/uk/post/klostridiozi-na-molocnij-fermi)

Affected muscles are dry, porous, permeated with gas bubbles, have a black-red color, crackle when pressed, and smell of rancid oil. Accumulation of cloudy serous fluid in the chest and abdominal cavities. Regional lymph nodes are enlarged, hyperemic, and permeated with hemorrhages. The lungs are swollen, the spleen is flaccid, the liver is enlarged, it contains centers of necrosis. Hemorrhages on the serous and mucous membranes (Fig. 2).

Fig. 2. Hemorrhages on serous and mucous membranes of internal organs.

(http://milkua.info/uk/post/klostridiozi-na-molocnij-fermi)

Laboratory diagnostics. It involves microscopic studies of swabs-imprints from pathological material, selection of a pure culture of the pathogen on nutrient media, conducting a bioassay on guinea pigs. Exudate from edema and pieces of affected muscles measuring 3×3×3 cm are sent to the laboratory with all precautions to prevent the dispersion of spores no later than 4 hours after the death of the animal, and in the case of a partial autopsy, pieces of the liver, spleen, and blood are also taken from the heart.

The diagnosis of emkar is considered established if one of the following indicators is obtained:

separation from pathological material of a culture with properties characteristic of the causative agent of the disease;

the death of at least one guinea pig out of two infected with the original pathological material, if it has pathological anatomical changes typical for this disease and the isolation of the culture of the pathogen from its organs, if even in cultures from the original pathological material, the culture of the pathogen is not isolated. The term of laboratory research is up to 8 days.

Differential diagnosis. Presupposes the need to distinguish emkar from anthrax and malignant edema. Emkar is registered mainly in cattle, and anthrax - in all types of animals. With emkara, there is a crepitation of edema, which is not characteristic of anthrax. Malignant edema is caused by a wound infection and appears sporadically.

Treatment. Due to the rapid course of the disease, treatment should be started as early as possible. Various antibiotics are used intramuscularly (penicillin, bicillin, streptomycin, tetracycline, baytril, clomaxil, enroxyl), 3-5% solutions of carbolic acid, lysol, 1-2% solutions of hydrogen peroxide, 0.1% solution of potassium permanganate. Symptomatic therapy is carried out. Serum against emphysematous carbuncle is used.

Immunity. In stationarily unfavorable points with regard to emkar, every spring, no later than 14 days before the beginning of the grazing period, all cattle aged from 3 months (sheep from 6 months) to 4 years old are vaccinated with a concentrated hydroxydaluminum formol vaccine against emkar. The vaccine is injected once into the muscles of the hind limb in a dose of 2-3 ml. Immunity is formed 14 days after vaccination and lasts for 6 months.

Prevention and control measures. In unfavorable points with respect to emkar, the presence and location of old burials of animals and livestock cemeteries, unfavorable pastures and water bodies are found out. They are fenced off, neutralized. Pastures and watering places are regularly cleaned from the remains of corpses. Every spring, no later than 2-4 weeks before cattle are driven to pastures, preventive vaccination of cattle aged from 3 months (sheep over 6 months) to 4 years with concentrated hydroxydaluminum formol vaccine is carried out. In the summer, to protect livestock from stinging insects, they practice night grazing and keeping them under sheds during the day.

When the disease appears, the sources of the causative agent of the infection are found out, livestock are transferred to other areas of pastures, and drinking water from infected reservoirs is not allowed. In unfavorable points and unfavorable farms, restrictions are introduced, under which it is prohibited to import, export and regroup cattle, export bulky fodder collected from infected pastures, forced slaughter of sick and suspected cow diseases, burying animal corpses, use of milk from sick cows. Cleaning and disinfection of livestock premises and animal care items are carried out regularly. Sick and suspected animal diseases are isolated and treated. Suspected animals are vaccinated against emkar. Slaughter of animals suffering from emkar is strictly prohibited. The corpses are burned along with the skin. Fodder, bedding and manure contaminated with secretions of sick animals are burned. Milk from sick animals is disinfected by boiling or dry chlorinated lime.

Restrictions on a dysfunctional farm are lifted 14 days after the last case of death or recovery of an animal, vaccination against emkar of all sensitive livestock and final disinfection. For disinfection, use a 10% hot solution of caustic soda, a 4% solution of formaldehyde, various solutions of chlorine-containing preparations with a content of at least 5% active chlorine in the solution.

Control questions and tasks.

1. The causative agent of emphysematous carbuncle and its characteristics
2. Epizootological data on emphysematous carbuncle.
3. Characteristic clinical signs of emphysematous carbuncle:
4. Pathological-anatomical changes in emphysematous carbuncle.
5. On the basis of what is the diagnosis of emphysematous carbuncle?
6. Does the differential diagnosis of emphysematous carbuncle involve exclusion?
7. How do you deal with animals suffering from emphysematous carbuncle?
8. What disinfectants are recommended for disinfection of premises with emphysematous carbuncle?
9. Under what conditions is the farm declared safe from emphysematous carbuncle of cattle?

Topic: Paratuberculosis

(diagnosis, prevention and control measures).

Paratuberculosis (Ione's disease) –chronic infectious disease of ruminants, which is characterized by a specific productive enteritis, periodic diarrhea, and progressive exhaustion. The disease causes significant damage to livestock farms, which consists of the cost of dead or forcibly slaughtered livestock, disruption of breeding work, reduction of animal productivity, costs of health and quarantine-restrictive measures.

The causative agent of the disease. Mycobacterium paratuberculosis is a small, immobile acid-alcohol-resistant bacillus that stains well with Ziel-Nielsen. It does not form spores or capsules. Smears from pathological material have the appearance of small clusters of bacteria, diplococci, and individual rods painted in red. Isolation of a pure culture of the pathogen and its further cultivation is associated with significant difficulties. For this purpose, special nutrient media of Dubo-Smith, Alikaeva, Dunkin, Ghon, etc. are used. with the addition of various growth factors. On dense nutrient media, the growth of primary cultures of the pathogen appears 15-180 days after sowing and is manifested in the form of whitish-gray, shrunken, dry colonies with uneven edges and a bumpy surface. In the process of growth in liquid media, the causative agent produces a toxic substance - paratuberculin, which causes an allergic reaction in infected animals. Paratuberculosis mycobacteria are not pathogenic for laboratory animals.

The causative agent of paratuberculosis is extremely resistant in the external environment. Pastures remain contaminated for 2-3 seasons. It is stored in manure and soil for more than 11 months, in fodder and water of non-flowing reservoirs - 9-12 months, in urine - 7 days. Heating to 80°C inactivates bacteria after 5 minutes, in milk - after 1-5 minutes. Effective disinfectants are 3-5% carbolic acid solution, 3-4% formalin solution, 5% lysol solution, 20% chloride-lime milk, 10% sulfur-carbolic mixture, 5% xylonaphtha emulsion.

The diagnosis is made by a complex method based on the analysis of the epizootological situation, clinical signs of the disease, data from the pathological and anatomical autopsy of sick animals slaughtered for diagnostic purposes, as well as laboratory studies.

Epizootological data. In natural conditions, paratuberculosis most often affects cattle aged 3-6 years, sheep aged 2-3 years, buffaloes and camels aged 2-3 years, rarely reindeer aged 2 years and older. Calves are infected from the first days of life until the age of 6 months through the mother's colostrum or collected milk from infected cows.

The source of the causative agent of the infection is clinically sick animals, which secrete a large number of bacteria with feces, milk, urine, amniotic fluid, and semen. No less dangerous are animals with a latent course of the disease, the number of which in an unhealthy herd can reach 30-50%. Such animals become a dangerous source of the causative agent of infection when they are brought into prosperous farms. Infection passes through the alimentary canal when feeding fodder and drinking from non-flowing reservoirs contaminated with secretions of sick animals. The probability of infection and the spread of the disease in the presence of bacterial carriers increases significantly during the stable period, when in connection with the crowding of animals and their poor feeding, favorable conditions are created for their re-infection. After penetrating the herd, the infection develops slowly and is mostly latent. However, under the influence of various provoking factors that reduce the body's resistance (childbirth, poor nutrition, stressful situations), the infectious process is activated and

causes the clinical manifestation of the disease. Paratuberculosis has been observed for years in stable, dysfunctional farms, mostly in the form of sporadic cases, sometimes enzootic. Lethality is 10-25%.

Clinical signs and course of the disease. The incubation period lasts from 5-12 months to 5-6 years. The course of the disease is latent and chronic. There are asymptomatic and clinical stages of the disease.

In cattle, the asymptomatic stage of the disease can last for years and can be detected only during an allergic, serological or bacteriological examination. The fact that 30-50% of such animals release the pathogen into the external environment, which causes a significant spread of the disease, deserves special attention. The transition from the asymptomatic stage to the clinical stage can occur gradually or suddenly.

The clinical stage of the disease lasts from 2 weeks to 1-2 years. In cows, clinical signs of the disease appear most often after the first or second calving and are manifested by rapid progressive weight loss, despite the presence of appetite, lethargy, pallor of the mucous membranes; the wool becomes matte and easily breaks out. In sick cows, milk yield decreases at first, and later milk secretion stops completely. Body temperature is normal, sometimes it drops before death. The main clinical sign of the disease is profuse diarrhea, which alternates with periods of normal defecation approximately every 10-15-20 days. During defecation, as a result of strong tension, the spine bends in an arc and the stool is released under pressure in a long, curved stream. Fecal masses are liquid, watery, greenish-yellow or brown in color, contain small lumps of mucus, blood veins and gas bubbles, have an unpleasant rotten smell. Some animals develop swelling in the area of the eyelids, intermaxillary space, abdomen, breast, udder (Fig. 1).

Fig. 1. Swelling in the area of the submaxillary space, breastbone. (https://agrotimes.ua/tvarinnitstvo/vid-poyavy-klinichnyh-oznak-do-zagybeli-korovy-vid-paratuberkulozu-mynaye-2-4-misyaczi/**)**

As a result of exhaustion, the flanks sink, fat deposits disappear, the muscles of the croup and hind limbs are extremely reduced, which creates the impression of asymmetry of the animal's body, as if the rear is cut off - "stilt-backed". Rarely, the disease ends with the death of the animal. In young children, the course of the disease is latent, accompanied by a delay in growth and development. Sometimes, characteristic clinical signs of the disease may develop, which are caused by an intense worm infestation or other reasons for a decrease in the body's resistance.

In sheep, the infection proceeds even more covertly than in cattle. In adult sheep and breeding rams, fatness may decrease, swellings appear in the area of the intermaxillary space, large areas of baldness are formed, and diarrhea is very rare. After vomiting, an exacerbation of the disease is often observed.

In camels and buffaloes, the disease is accompanied by exhaustion, first intermittent, and then profuse diarrhea, swelling in the lower abdomen and around the genitals. Death occurs with the phenomena of cachexia.

Pathological anatomical changes. In cattle, they are characterized by lesions of the ileum and jejunum, less often – the cecum and colon and, as an exception, the duodenum and rectum. At the same time, thickening of individual areas of the mucous membrane by 4-10 times, dense longitudinal and transverse folds of a pale color, resembling convolutions of the brain, dotted and striped hemorrhages are observed. Serous membranes and mesentery are swollen, lymphatic vessels are thickened, they look like thick twisted cords. Mesenteric lymph nodes are enlarged, softened, sometimes with grayish-white sarcoma-like cells. In some cases, an autopsy reveals folding of the mucous membrane of the urinary and gall bladders, laxity of the heart muscle, and degenerative changes in the liver, kidneys, and heart.

In sheep and goats, thickening and folds of the intestinal mucosa are less pronounced, and calcified and encapsulated necrosis centers are found in the enlarged lymph nodes and intestinal mucosa. Ascites is often observed. In the latent stage of the disease, pathological changes are noted only in the mesenteric lymph nodes. In camels, catarrhal and later productive inflammation of the small intestines, thickening and folds of the large intestines, a sharp increase in mesenteric, submandibular, or pharyngeal lymph nodes, as well as degenerative lesions of the liver, spleen, and kidneys are detected.

Laboratory diagnostics. Includes microscopic and histological examination of pathological material taken from sick or forcibly slaughtered animals, and, if necessary, isolation of a pure culture of paratuberculosis bacteria. Scrapings from the mucous membrane of the rectum, stool samples with lumps of mucus and blood impurities taken from the rectum, blood serum are sent to the laboratory for intravital diagnosis from sick animals; for postmortem studies - pieces of the affected areas of the ileum and jejunum and separate mesenteric (mesenteric) lymph nodes.

Allergic studies are conducted to detect latently diseased cattle and sheep. Cattle are injected with tuberculin for poultry intradermally in the middle third of the neck:

animals under the age of 2 years - 0.2 ml, from 2 to 3 years - 0.3 ml, over 3 years old - 0.4 ml. Exhausted animals, dams a week before giving birth and a week after giving birth, as well as animals 2 weeks after vaccination are not allowed to be tested by the allergic method. Evaluation of the reaction after the first injection is carried out after 48 hours. Tuberculin is re-injected in the same place and in the same doses to animals that gave a doubtful reaction and to those that did not react. The results of the reaction to repeated administration are taken into account after 24 hours. The reaction is considered positive if there is a diffuse, painful, hot swelling of a dough-like consistency at the site of tuberculin injection, and the skin fold thickens by 7 mm or more. The reaction is considered doubtful with less pronounced inflammatory phenomena and thickening of the skin fold from 5 to 7 mm. The reaction is considered negative in the absence of inflammatory phenomena at the site of tuberculin injection, as well as in the case of the formation of a painless, cold, limited hardening, even if the thickness of the skin fold increases by more than 5-7 mm.

For allergic diagnosis of paratuberculosis in sheep, dry purified (PPD) tuberculin for poultry is used, which is injected once in a dose of 0.2 ml under the skin of the lower eyelid, 1-1.5 cm below its edge. The results of the reaction are taken into account after 48 hours. A reaction is considered positive when inflammatory edema appears at the site of PPD-tuberculin injection. Sheep are subjected to an allergic test for paratuberculosis starting from the age of 3 months, cattle - from the age of 10 months. In infected animals with low fatness or with a clinical manifestation of the disease, the allergic reaction may be weak or absent. A more sensitive method of diagnosing paratuberculosis in the presence of clinical signs of the disease is the examination of blood serum using the complement binding reaction.

Differential diagnosis. It requires the exclusion of tuberculosis, enteritis of non-infectious etiology, coccidiosis, and worm infestations.

In tuberculosis, enteritis is rare, accompanied by simultaneous damage to the lungs and external lymph nodes, differentiated by allergic indicators. Coccidiosis and worm infestations are diagnosed based on the results of coprological studies. Enteritis of non-infectious etiology has a mass character, is diagnosed based on the results of feed analysis.

Treatment. Specific therapy for paratuberculosis of large and small cattle has not been developed. Symptomatic treatment is not effective.

Immunity. In case of paratuberculosis, it is not sterile. No effective vaccine has been proposed to prevent the disease.

Prevention and control measures. Cattle care includes the protection of healthy farms from the introduction of infection, veterinary and sanitary measures aimed at eliminating the disease in unhealthy farms, and measures to improve the health of unhealthy farms. In order to prevent the occurrence of the disease in a healthy farm, a clear control is established over the composition of the herd, it is allowed to import cattle only from farms that are safe with regard to paratuberculosis. During the 30-day

preventive quarantine, imported animals are kept isolated from the main herd and veterinary supervision is organized, and in case of suspicion, allergy tests for paratuberculosis are carried out.

When paratuberculosis is detected, the farm is declared unhealthy and quarantine restrictions are imposed on it. A thorough clinical examination of livestock, isolation of clinically ill animals and their slaughter are carried out. The rest of the animals (over 18 months of age) are examined using the complement binding reaction. Animals that react positively are examined comprehensively after 15-20 days using a complement binding reaction and a double intradermal test with dry purified tuberculin PPD for poultry. Animals that have given a positive allergic reaction and respond positively to complement binding reactions are slaughtered. The rest are left in the herd and examined in the above order twice a year. Animals with clinical signs of the disease are slaughtered regardless of the results of the research. Calves from sick cows are also slaughtered. Calves from healthy animals in a dysfunctional herd are separated from cows and fed with colostrum for 5 days, and then raised on an isolated farm. At the age of 10-12 months, they are examined for paratuberculosis with a double intradermal test, and if negative results are obtained, they are considered healthy.

The young, which reacted positively or doubtfully to tuberculin, are isolated and after 30-45 days are re-examined by the allergic method. The young, which gave a positive or doubtful reaction during the re-examination, are slaughtered, the rest are returned to the herd.

For disinfection, a clarified solution of chlorinated lime containing at least 5% active chlorine, a 10% hot solution of a sulfur-carbolic mixture, a 20% suspension of freshly slaked lime (three times bleaching with an interval of 1 hour), an alkaline formaldehyde solution containing 3% formaldehyde is used for disinfection. and 3% caustic soda, with an exposure of 1 hour, 5% hot emulsion of creolin or 5% hot emulsion of xylonaphtha. Manure from clinically ill animals, as well as animals that respond positively to allergy tests, are burned.

The farm is considered recovered from bovine paratuberculosis 3 years after the last case of detection of a sick animal and the implementation of all measures provided for by the current instructions.

Control questions and tasks.

1. The causative agent of paratuberculosis and its characteristics.
2. Epizootological features of paratuberculosis.
3. Stages of the clinical course of the disease and their main clinical signs.
4. Characteristic patho-anatomical.
5. What is the sequence of diagnostic studies in order to confirm or exclude the diagnosis of paratuberculosis?
6. Draw up a scheme for the rehabilitation of a dysfunctional animal husbandry, taking into account the age and infectious status of the rehabilitation groups.

7. What is the method of allergic research of cattle for paratuberculosis?

Topic: Campylobacteriosis

(diagnosis, prevention and control measures).

Campylobacteriosis (Campulobacteriosos, vibriosis) is an infectious disease of large and small cattle, which is characterized by abortions, frequent grazing, temporary infertility, and the birth of non-viable offspring. People can get sick with campylobacteriosis.

Cattle disease is observed in many countries of the world. It causes significant economic losses to disadvantaged farms, caused by the death of newborn young, morbidity and a decrease in milk productivity of the mother herd, disruption of breeding work, costs for health measures.

The causative agent of the disease is the fetal vibrio Campylobacter fetus, which belongs to the genus Campylobacter, includes two pathogenic species of campylobacter - Campylobacter fetus subspecies fetus with two subspecies - Campylobacter fetus subspecies veneralis and Campylobacter fetus subspecies fetus, as well as Campylobacter yeyuni, which cause cattle campylobacteriosis livestock Campylobacteriosis in sheep is caused by pathogenic campylobacter Campylobacter fetus subspecies fetus. In addition to pathogenic species and subspecies of Campylobacter, there is also a saprophytic species - Campylobacter sputorum bubulus, which is found in the alimentary canal and genitals, as well as in manure, sewage, polluted water bodies.

Morphologically, all species and subspecies of Campylobacter are identical, differing only in pathogenic properties for different species of animals, as well as in serological and some cultural and biochemical parameters. Campylobacter are polymorphic, motile gram-negative microorganisms, which in smears and hanging drops have the appearance of a coma, a seagull in flight or a short spiral with a different number of curls. Capsules and spores do not form. They are dyed with all aniline dyes and according to Romanovsky-Giemza.

Campylobacter contains O- and H-antigens, the differences between which are used during serological typing. 0.15-0.2% semi-liquid meat-peptone liver agar, semi-liquid agar with 10% bile content, semi-liquid liver agar, dense 2-3% meat-peptone liver agar, Marten's agar are used for cultivation of campylobacter. Campylobacter growth occurs at a temperature of 37.5°C, in conditions of reduced oxygen content and the presence of 10-25% carbon dioxide in the desiccator. In semi-liquid agar, after 36-48 hours of cultivation, campylobacter forms a grayish-blue ring of growth 1-4 mm thick. On dense nutrient media, growth is observed after 72-96 hours in the form of smooth, rough and slimy small blue colonies or a delicate dewy coating. Sheep, goats,

guinea pigs, hamsters are susceptible to experimental infection - after subcutaneous, intravaginal, intraperitoneal and oral administration of pathological material.

Campylobacter is not stable in the external environment. In manure, hay, water, soil at 18-20°C they are stored for only up to 20 days, at 6°C - up to 1 month; in the contents of the stomach, liver, cotyledons of aborted fetuses remain viable for up to 20-50 days. When dried, they die after 3 hours, at 55°C - after 10 minutes. They show pronounced resistance to low temperatures, at which they can be stored for 30 days. They are quickly destroyed in rotting material, as well as under the influence of high temperatures and disinfectants.

The diagnosis of campylobacteriosis is made by a complex method, which includes: epizootological data, clinical signs and the results of laboratory examination of pathological material.

Epizootological data. Under natural conditions, sexually mature heifers, cows, ewes, and sometimes goats are affected by campylobacteriosis. In cows, the main source of the causative agent of the disease is infected broodstock-bugai, in the preputial sac of which, less often in semen, campylobacter is stored for years. Campylobacter is released by sick cows and ewes during abortions with the fetus, fetal membranes, amniotic fluid, secretions from the genital organs, as well as with urine and milk. Excretions of sick animals contaminate the premises, bedding, feed, water, animal care items, which become factors of transmission of the pathogen to healthy animals. Infection can occur during mating with a sick breeder, artificial insemination with infected sperm, as well as in the case of joint keeping of sick animals with healthy ones. It is believed that the transmission of the causative agent of campylobacteriosis in cows is carried out during natural mating in 40-90% of cases, through semen - 30-70%, through infected obstetric instruments, hands and clothes of service personnel - 2-5%, bedding - 8-12% cases. Healthy bulls become infected during mating with infected cows and heifers. In prosperous farms, primary outbreaks of campylobacteriosis in cattle are mainly associated with the introduction of microbe-carrying animals or artificial insemination with the semen of infected bulls. In these cases, the infection has an acute course, abortions are observed in 30-68% of cows, infertility in 60-64% of heifers, 20-55% of cows. In the following years, abortions are usually stopped, most of the sick animals recover their reproductive capacity.

In sheep, the main source of the pathogen is sick ewes, in which microbonocarriage lasts 1-1.5 years. Only ewes are infected through contaminated feed and water. The role of rams in the spread of campilobacteriosis has not been proven.

Chickens are sick with campylobacteriosis. There is a significant death among chickens under 1 month of age (3-15%), a decrease in the weight gain of broiler chickens (by 20-47%), a decrease in laying hens (by 15-35%).

Human infection occurs when using campylobacter-contaminated livestock products (meat, raw milk), as well as through water. Mutual re-infection of people with animals and vice versa is possible.

Clinical signs and course of the disease. At the primary occurrence of campylobacteriosis, the main symptom is mass abortions in calving cows and numerous cases of infertility in heifers. Abortions occur in the first or at the beginning of the second half of pregnancy and are almost always complicated by litter retention, vaginitis and metritis. Born calves are weak, non-viable, get sick and die within 2-7 days of life. In heifers, 6-15 days after infection, an increase in body temperature, significant secretion of mucus, redness of the mucous membrane of the vagina is observed (Fig. 1). Later, cervicitis, metritis, salpingitis, oophoritis and granulation vaginitis appear (Fig. 2). After 3-6 months, inflammatory phenomena disappear, reproductive capacity is restored.

Fig. 1. Hyperemia of the mucous membranes of the vagina

Fig. 2. Postpartum metritis with uterine prolapse

In bulls, the disease is asymptomatic, accompanied by long-term campylobacteriosis. In farms that have been unfavorable for campylobacteriosis for a long time, barrenness and frequent wandering of cows are also observed. Periods of sexual rest become longer, up to 30-60 days or more, infertility can occur in 20-50% of cows, and in heifers it can reach 60%. Abortions are rarely observed, mainly in cows in the first or the beginning of the second half of pregnancy. There are early abortions that may go unnoticed. In breeders, the infection is latent, in calves it causes damage to the intestines.

Mass abortions (from 10 to 70% or more) are observed in sheep 1.5-2 months before calving, which are accompanied by an increase in body temperature up to 41°C, mucous, and when complicated by secondary microflora, mucous-purulent discharge from the vagina, metritis . Cases of the birth of dead and non-viable lambs are registered, as well as the death (3-10%) of ewes.

Pathological anatomical changes. The uterus is swollen, blood vessels are injected, numerous foci of inflammation are found on the mucous membrane. Caruncles are enlarged, inflamed, easily separated from the placenta. Foci of necrosis and thickening are determined in the placenta. The fetal membranes are swollen, covered with mucous-purulent viscous exudate, there are small hemorrhages and multiple foci of surface necrosis. At the autopsy of an aborted fetus, a lot of serous effusion with fibrin and blood impurities is revealed, and in the sputum there is a liquid brownish exudate with grayish-white flakes. The subcutaneous tissue and tissues of the aborted fetus are swollen. On the walls of the abdominal and thoracic cavities, as well as on the internal organs, there are fibrinous layers, on the heart, under the capsule of the spleen - small hemorrhages, on the liver - centers of necrosis. In a bird that died from campylobacteriosis, catarrhal inflammation of the intestinal mucosa and necrotic lesions in the liver were found.

Laboratory diagnostics. Includes microscopic, bacteriological and serological studies with isolation and determination of the subspecies of the pathogen. The entire aborted fetus (from large fetuses – head, stomach, liver with gall bladder, lungs) is sent to the laboratory, as well as the placenta or its part, taken during the first day after the abortion. Mucus from the cervix of cows is taken only during the calving period or the first 3-4 days after abortion. Preputial mucus, sperm and secretion of the accessory gonads are sent from bulls, vagina, uterus, lymph nodes of the pelvic cavity, selected from animals slaughtered for diagnostic purposes, from cows. Samples of vaginal mucus from cows, preputial mucus, secretion of gonads and sperm from breeders are taken and sent in a thermos with ice no later than 6 hours. From ewes that have aborted, blood sera taken in the first 20 days after abortion are sent for serological testing.

Microscopic studies involve the preparation of smears from pathological material, their staining with Gram and Ziel-Nielsen fuchsin for examination under a light microscope, as well as bivalent luminescent serum against Campylobacter fetuses of the 1st and 2nd pathogenic subspecies and monovalent luminescent serum of the saprophytic species Sputorum for luminescence microscopy. In case of detection of the luminescence of microbial cells morphologically typical for campylobacter in smears, a positive luminescent diagnosis is made, which is regarded as a signal and is the basis for carrying out measures against campylobacteriosis in the farm until the results of bacteriological studies are obtained.

In order to isolate a pure culture of campylobacter, the pathological material is sown on semi-liquid and dense media, cultivated in a desiccator with a content of 10-15% carbon dioxide, reviewed daily to detect the typical growth of the pathogen. The identification of isolated campylobacter is carried out by studying the pathogenic, cultural, biochemical and serological properties of the culture, as well as by the immunofluorescence method.

Serological differentiation is carried out using an agglutination reaction with monospecific campylobacter agglutinating sera against the 1st and 2nd pathogenic

subspecies of the pathogen and the saprophytic species. For the identification and differentiation of isolated Campylobacter by immunofluorescence method, bivalent luminescent serum against Campylobacter fetus of the 1st and 2nd pathogenic subspecies, as well as monovalent serum of the saprophytic species is used.

In addition to the above laboratory methods of diagnosis, the farm conducts a mass examination of the herd of cows using the agglutination reaction with vaginal mucus. To do this, samples of vaginal mucus are taken from all cows and sexually mature heifers with a sexual cycle disorder and examined in zonal laboratories. Blood is taken from sheep for serological diagnosis and examined by the agglutination reaction using the classic method.

Differential diagnosis. In cattle, brucellosis, trichomoniasis and listeriosis should be ruled out, in sheep - also salmonellosis and chlamydial abortion.

With brucellosis in cows, abortions occur at 5-8 months of pregnancy, heifers often abort, serous bursitis and serofibrinous arthritis occur. In sheep, abortions of brucellosis etiology are more often registered at 4-5 months, and sometimes earlier, orchitis and epididymitis are characteristic of rams. During the bacteriological examination of aborted fetuses, Brucella are isolated, according to the complement binding reaction and the agglutination reaction - specific antibodies. Trichomoniasis in cows is characterized by purulent metritis, barrenness associated with abortions, while in campylobacteriosis heifers become infertile after the first insemination. Trichomonads are easily detected during microscopic examination.

Listeriosis is accompanied by damage to the central nervous system and septic phenomena. Salmonellosis affects sheep of all ages. Its characteristic features are inflammation of the alimentary canal, lungs, and joints. Salmonellosis abortions are often accompanied by the death of ewes. The diagnosis is confirmed by bacteriological and serological studies. Chlamydial abortion of sheep is observed 2-3 weeks before lambing, ewes that have aborted often die. The diagnosis is made on the basis of the detection of elementary bodies, stained according to Romanovsky-Giemsa, in smears-imprints from the parenchymal organs of the aborted fetus and discharge from the vagina. Serological diagnosis is carried out by complement binding reaction, hemagglutination delay reaction, microagglutination reaction.

Treatment. It is effective only in case of simultaneous general and local therapy. A mixture of an emulsion of streptomycin and penicillin is used for the treatment of bugs, which is prepared at the rate of 1,000,000 units of each antibiotic in 40-50 ml of fish oil or oil. The emulsion is injected into the preputial sac, which is gently massaged for 25-30 minutes. Before the introduction of the emulsion, the preputial cavity is thoroughly washed with warm, boiled soapy water, and then with a 3% solution of hydrogen peroxide or a solution of furacilin in a dilution of 1:5000. Treatment is carried out for 4 days in a row. Simultaneously with local treatment, penicillin and streptomycin in a 0.5% solution of novocaine are administered intramuscularly to bulls twice a day at the rate of 5,000 units of each antibiotic per 1 kg of animal weight, also

for 4 days. After 5-6 days, the treatment is repeated. Oxytetracycline in a 0.5% solution of novocaine is injected intramuscularly twice a day at the rate of 5,000 units per 1 kg of animal weight, and a 5% emulsion of furazolidone on fish fat or oil is applied locally. The effectiveness of the treatment is checked after a month with the help of three times with an interval of 10 days bacteriological examination of sperm and preputial mucus. In the case of a negative result, bulls are considered healthy. High-value animals, whose recovery has not been achieved, are re-treated with subsequent bacteriological examination. If campylobacteriosis remains after repeated treatment, the bull is culled.

Cows and heifers with clinical signs of campylobacteriosis (abortion, litter retention, metritis) are also treated locally and generally. Sick animals are injected with 1,000,000 units of penicillin and streptomycin, emulsified in 40-50 ml of sterile oil, fish oil or dissolved in 0,9% NaCl solution, into the uterine cavity every day for 4 days in a row. Solutions of furacilin (1:5000) or rivanol (1:1000) are used for douching the vagina. At the same time, streptomycin in a 0.5% solution of novocaine is injected intramuscularly at the rate of 4000 units/kg of animal weight twice a day for 4 days in a row.

Ewes are treated with streptomycin, penicillin, bicillin 3, tetracycline, which are emulsified in oil or fish oil, at the rate of 2000 units of the antibiotic in 20 ml of oil. An emulsion of one of the specified antibiotics is injected into the uterine cavity every day for 3-5 days in a row. At the same time, streptomycin or oxytetracycline in a 0.5% solution of novocaine is injected intramuscularly at the rate of 4,000 units/kg of animal weight twice a day for 4 days in a row. Rams, which are used to fertilize sheep in a dysfunctional flock, are injected intramuscularly with streptomycin or oxytetracycline in a 0.5% solution of novocaine at the rate of 4,000 units of each antibiotic per 1 kg of animal weight for 4 days in a row.

Immunity. After an illness, it is formed only in sheep, and in cattle, susceptibility to vibriosis remains. Cows that have contracted vibriosis do not abort again. Inactivated emulsin vaccine is used for specific prevention of campylobacteriosis in sheep.

Prevention and control measures. To prevent the introduction of campylobacteriosis when forming a herd, it is necessary to import only healthy animals and only from farms safe from campylobacteriosis. In bulls that have reached sexual maturity, before being removed from the farm, the preputial mucus, sperm or mucus of the accessory gonads are examined once by a bacteriological method. The breeding of healthy heifers and repair bulls that have not reached mating age is carried out without a preliminary examination, however, the veterinary certificate indicates the state of well-being of the farm in relation to campylobacteriosis. Breeding of sheep for breeding and production purposes is carried out without tests for campylobacteriosis. In bulls that have entered the farm for use for breeding or production purposes, during the monthly quarantine, three times (with an interval of 10 days) bacteriological examination of preputial mucus, sperm or secretion of accessory gonads must be carried out. In the future, breeding bulls are examined by the bacteriological method

once every 6 months three times with an interval of 10 days. For insemination of the maternal herd of cattle, the sperm of bulls is used only from safe artificial insemination stations or breeding plants. Rams are not tested for campylobacteriosis when brought to the farm.

When campylobacteriosis occurs among cattle, control measures are carried out taking into account the peculiarities of the direction of the farm's activity. At the artificial insemination station, when the diagnosis is established, quarantine restrictions are introduced and a plan of measures to eliminate campylobacteriosis is approved. The station of artificial insemination of animals is declared safe on the basis of receiving negative results of bacteriological examination of bulls treated with antibiotics, after removal of culled animals and carrying out a full set of veterinary and sanitary measures. After lifting the restrictions at the station during the year, once a quarter, bacteriological tests of sperm and preputial mucus of all bulls are carried out 3 times with an interval of 10 days.

When cattle campylobacteriosis caused by S. fetus subspecies fetus is established, quarantine restrictions are introduced in the commercial economy (farm), according to which it is prohibited to introduce healthy animals to the unhealthy farm for the period until the restrictions are lifted, and to remove animals from the farm for breeding and user purposes in other farms, and from a dysfunctional farm within the farm, it is prohibited to remove animals for various purposes, except for slaughtering for meat; the use of infected and suspected bulls as breeders and the selection of their sperm for artificial insemination is not allowed. Breeding bulls, which are on a dysfunctional farm, are subjected to medical and preventive treatments and are examined by analogy with dysfunctional artificial insemination stations. In the summer, cattle from dysfunctional farms are taken to camps, artificial insemination of cows and heifers is carried out with the sperm of healthy bulls. The farm carries out sanitary cleaning, repairs, disinfection of livestock premises and their sanitation throughout the summer. Cows and heifers are calved only in the maternity ward. Each animal that aborted is isolated. Clean and disinfect the premises and machines where it was placed. Walking areas, yards, bases and other places where infected animals stay are systematically disinfected. Aborted fetuses and amniotic fluid after disinfection in a moisture-proof container are burned or buried in a place specially designated for this purpose. Sick animals are treated, systematic surveillance is established for all breeding stock and prophylactic treatment with antibiotics is carried out. For this purpose, 10-12 hours after the second insemination, 100,000 units of streptomycin and penicillin in 20 ml of warm 0,9% NaCl solution are injected into the uterine cavity of clinically healthy cows and heifers. Pregnant cows and heifers are given 250-300 ml of an aqueous solution of ryvanol (1:1000) orally once a day for 5 days. The farm is declared healthy if, within 12 months after the last case of detection of sick animals, during the examination of sick cows and heifers of a dysfunctional farm and aborted fetuses, the culture of S. fetus subspecies veneralis was not isolated and the entire complex of

veterinary and sanitary measures provided for in the instructions was carried out in the farm.

When a diagnosis of campylobacteriosis is established in sheep, it is forbidden to remove them from unhealthy flocks and to reform flocks. All sheep that have aborted, as well as animals with signs of premature birth, are immediately isolated and treated. Aborted fetuses, fetus shells, droppings, as well as bedding, manure, feed contaminated with secretions of sick animals are burned or buried in the ground after disinfection with disinfectants. Cleaning and disinfection of the place where the abortion took place, as well as dysfunctional pens and walking yards are carried out. When keeping sheep on pasture, they are transferred to other areas, and the places of previous grazing are quarantined for 2 months. To prevent abortions, cats are given chlortetracycline with feed at the rate of 5-8 mg/kg of animal weight for 10-12 days or bigumal at 0.5 g per head for 3 days in a row, 1.5-2 months before farrowing. Rams, which are used for fertilization of sheep in a dysfunctional flock, are injected intramuscularly twice a day with streptomycin and penicillin in a 0.5% solution of novocaine at the rate of 4,000 units of each antibiotic per 1 kg of animal weight for 4 days. A flock is considered to be free from campylobacteriosis in the absence of campylobacterial abortions in ewes for two years.

For the disinfection of livestock premises, walks, pens, equipment, inventory, a clarified solution of chlorinated lime containing 2% active chlorine, 2% hot solution of caustic soda, 3% hot solution of sulfur-carbolic mixture, 20% suspension of freshly slaked lime, 5% emulsion is used disinfecting (phenolic) creolin, 2% formaldehyde solution with exposure for 1 hour. When using a 5% hot solution of soda ash, the exposure should be 3 hours. Manure is disinfected by the biothermal method. Manure from the premises intended for the goat is dried in the sun in a fenced area, and then used as fuel without restrictions.

Control questions and tasks.

1. The causative agent of campylobacteriosis and its characteristics.
2. Epizootological features of campylobacteriosis.
3. Clinical signs and course of campylobacteriosis.
4. When is the diagnosis of campylobacteriosis considered established?
5. Treatment of sick animals with campylobacteriosis.
6. Describe general and specific preventive measures for campylobacteriosis.
7. Describe health measures for campylobacteriosis.

Topic: Contagious pleuropneumonia of cattle

(diagnosis, prevention and control measures).

Contagious pleuropneumonia of cattle (Pleuropneumonia contagiosa bovum, grazing pneumonia, peripneumonia of cattle, CPP) is a chronic highly contagious disease characterized by serous inflammation of the interlobular lymphatic vessels, serous-fibrinous pleurisy, croupous pneumonia with the formation of anemic necrotic foci in the lungs (sequestration).

A high degree of disadvantage regarding the disease of contagious pleuropneumonia is observed in many countries of the African continent, which account for 98.17% of all registered outbreaks in the world, in Asia (0.68%), Australia (0.46%), as well as in Europe (0.68%). The American continent is free of this disease. Economic losses are quite significant and consist of extremely high costs for the organization of quarantine-restrictive and preventive-liquidation measures, as well as in connection with high mortality and forced slaughter of sick and suspected animal diseases.

The causative agent of the disease. Mycoplasma mycoides var. Mycoides is a small, immobile gram-negative extremely polymorphic microorganism, has cocoon-like, thread-like, branched and star-like forms. Passes through bacterial filters, stains well according to Romanovsky-Giemsa, according to Gram - negative. It is antigenically homogeneous. Does not grow on normal nutrient media. On dense nutrient media, it forms very small, dewy colonies with a solid center that grows into the agar and smooth edges. On blood agar, it causes a change in the color of the medium from red to green. In Marten's broth with blood serum, there is first a weak opalescence, then slight turbidity. It also reproduces in chicken embryos.

Unstable in the external environment: stored in rotting material for 9 days, in frozen lungs - up to 1 year. When heated to 55°C, it is inactivated after 5 minutes, when dried and under the influence of solar radiation - after 5 hours. It is quickly destroyed under the action of all disinfectants.

The diagnosis is made by a complex method based on epizootological, clinical, patho-anatomical data and the results of laboratory studies.

Epizootological data. In natural conditions, only ruminants - cattle, buffaloes, yaks, bisons, zebu, camels - get sick with contagious pleuropneumonia. The source of the causative agent of infection is clearly sick animals, chronically ill animals with unclear clinical signs, and livestock with a latent course of infection. The causative agent is released with discharge from the nose, drops of mucus during coughing, sometimes with milk, urine, feces, amniotic fluid during abortions. Animals with a chronic and latent course of the disease are especially dangerous, in which the causative agent in a virulent state can persist for years in the encapsulated lung tissue, and when the process is aggravated, it can be released into the environment. Such animals often become a source of introduction of the causative agent of infection into prosperous farms.

The causative agent of infection is transmitted from sick to healthy animals by air and contact, and even short-term joint housing is enough. There are described cases of livestock infection through feed contaminated with the pathogen, sexually; a transmissible way of mycoplasma transmission is also not excluded. Contagious pleuropneumonia is characterized by a slow, gradual development of infection, especially when animals are kept on pasture. An epizootic with variable intensity can last for many years. The incidence of contagious pleuropneumonia is 25-70%, the mortality rate is 20%. These indicators depend to a large extent on the conditions of keeping animals - when kept in stables on a limited territory, they are much higher than on pastures. In stationary dysfunctional farms, the majority of animals may not have specific lung lesions, and the infection of livestock is determined only by serological indicators.

Clinical signs and course of the disease. The incubation period lasts 2-4 weeks. The course of the disease is hyperacute (very rarely), acute, subacute and chronic. There are typical, atypical and latent forms of the disease.

In the case of a hyperacute course, an increase in body temperature up to 41°C, cessation of chewing, severe depression, heavy breathing, and sometimes diarrhea are observed. Inflammation of the pleura and lungs develops very quickly, which leads to the death of the animal within 2-8 days.

With an acute course, at the beginning of the disease, a dry, short, painful cough is detected, as well as a slight (0.5-1°C) increase in body temperature, depression. Over time, the pulse accelerates to 80-100, breathing - to 55 per minute, the temperature rises sharply to 41-42°C and remains at this level until death. The general condition of the animal deteriorates sharply, breathing becomes superficial, the cough is strong, wet, mucous-purulent discharges are released from the nose. Percussion in the area of the lungs reveals a muffled or dull sound, auscultation reveals harsh vesicular or bronchial breathing, wet wheezing. When the pleura is affected, friction sounds appear, and in the presence of cavities in the lungs, the sound of a drop can be heard. Sick animals stand with their legs wide apart, with their necks stretched forward, their mouths open. They respond painfully to pressure in the area of the intercostal spaces and the spine. Swelling appears in the area of the chest, the lower part of the abdomen, and on the limbs. In cows, lactation decreases, abortions are possible. In the future, mediastinal and bronchial lymph nodes and the pleura are involved in the lymphogenic process. Animals die within 10-15 days. Mortality in the acute course of the disease reaches 50-74%. Sometimes recovery is noted. However, a relapse is possible at any time.

In the subacute course, fever, runny cough, and diarrhea are noted, which may disappear periodically. In the chronic course, exhaustion, lethargy, change in appetite, pallor of the mucous membranes, and periodic bowel dysfunction are observed. A characteristic clinical sign is a persistent cough, during which purulent masses are sometimes released (Fig. 1). Swelling often occurs in the extremities, neck, and abdomen. An increase in body temperature, worsening of the general condition,

diarrhea is observed. Percussion and auscultation reveal blunting of the percussion sound, absence of respiratory sounds. The disease lasts about 6 months, recovery occurs only in 30% of sick animals.

The atypical form of the disease is manifested by short-term fever, lethargy, cough, and decreased appetite. With this form of the disease, the animal often recovers completely. The latent form of the disease is characterized by the absence of any clinical signs and is detected during serological tests

Fig. 1. Purulent exudate in the nostrils after coughing.
(https://veterinary.lenobl.ru/ru/news/41780/)

Pathological anatomical changes. In case of contagious pleuropneumonia of cattle, they are found in the lungs and chest cavity and are so characteristic that they are of essential importance for making a diagnosis. At the autopsy of the chest cavity in the middle and main lobes of the lungs, hard to the touch, enlarged lobar lesions are found, which, depending on the age of the inflammation, are at different stages of hepatization and are colored in dark red, gray-red or gray color, which gives a characteristic the appearance of variegated marble. On the surface of the cut of the lungs, wide connective tissue strands of gray and white color stand out, which divide the affected tissue into separate areas, in which there are wide cells of dead tissue surrounded by a capsule - sequestra. In the swollen and thickened connective tissue layers, greatly expanded lymphatic vessels are located, overflowing with liquid transparent lymph that drains on the incision.

The lymph nodes of the thoracic cavity are swollen, enlarged, and penetrated by small centers of necrosis. Fibrinous layering, serous-fibrinous inflammation are observed on the pleura; a significant accumulation (up to 10 l) of serous-fibrinous transudate is noted in the pleural cavity. Sometimes connective tissue adhesions form between the lungs and the pleura, pericardium and epicardium. The specified patho-anatomical changes are found only in the chronic course of the disease. In acute cases, the changes in the lungs are atypical and are characterized only by numerous

pneumonic foci in the stage of red and gray hepatization, a sharp expansion of the interlobular connective tissue.

Laboratory diagnostics. Includes bioassay on calves, bacteriological and serological studies. Mediastinal lymph nodes preserved in glycerol or frozen, pleural and pulmonary inflammatory fluid, as well as pieces (4-5 cm) of affected lungs and sequestrations are sent to the laboratory. Samples of pathological material fixed with 10% formalin solution are sent for histological examination, blood serum for serological examination. To conduct a bioassay, calves from farms safe from contagious pleuropneumonia are infected subcutaneously with lung lymph or exudate from the chest cavity, which are taken from sick animals that died or were slaughtered for diagnostic purposes. With positive results, significant inflammation of the subcutaneous connective tissue (phlegmon), damage to regional lymph nodes, general intoxication of the body, and death of test calves are observed 15-17 days after infection. Bacteriological studies are rarely carried out due to the difficulties that arise during the isolation of a pure culture of the causative agent of the disease. Cultures of pleural exudate and lymph, taken from the affected areas of the lungs, are carried out on special nutrient media. In the acute period of the disease, blood cultures are performed. Serological studies involve the detection of animals with a latent form of infection using the complement binding reaction (diagnostic titer 1:10). Immunodiffusion reaction, indirect hemagglutination reaction, immunofluorescence reaction, conglutination reaction, lamellar agglutination reaction are also used. In some African countries, to identify infected animals, blood serum tests are conducted based on the complement binding reaction in the recesses of plastic plates, as well as allergy studies. The allergen is injected intradermally in a dose of 0.1 ml in the middle part of the neck. An increase in the skin fold of 4 mm or more after 24 hours is considered a positive result.

Differential diagnosis. Presupposes the exclusion of pasteurellosis, tuberculosis, croup pneumonia of non-infectious origin.

Pasteurellosis has an acute, rapid course, phenomena of hemorrhagic diathesis, damage to the intestines are observed. Bacteriological and biological studies make it possible to quickly and accurately identify the causative agent of an infection. Tuberculosis is diagnosed on the basis of an intradermal allegric test, detection of the pathogen in the pathological material and its identification. Croupous pneumonia of non-infectious origin is characterized by sporadicity, a more acute course, the absence of sequestrations and the marble pattern characteristic only of contagious pleuropneumonia on autopsy of the affected lungs.

Treatment. Due to the danger of spreading the infection, it is prohibited. Sick animals are slaughtered. For the treatment of post-vaccination complications, neosalvarsan (2-3 g intravenously in the form of a 10% solution 2-3 times every 2 days), sulfamesate sodium (intravenously or subcutaneously in the form of a 33.3% solution), bronchocillin (100,000 IU for 3 days in a row) is used), tylosin

(intramuscular 7.5-15 mg/kg 2 times a day), chloramphenicol (11-13 mg/kg 3 days in a row).

Immunity has not been studied enough. In vaccinated animals, complement-binding antibodies are detected within 8 months. For immunization, a live culture of M. mycoides is used, which is injected subcutaneously on the inner surface of the tip of the tail. Vaccines from live weakened mycoplasmas (avianized, attenuated or naturally weakened strains of the causative agent) are preferred. In the countries of Africa and Australia, vaccines are made from KH3J, Tl and T3, V5 strains, which are injected into cattle subcutaneously in the tip of the tail or intradermally in the upper half of the outer surface of the auricle, in zebu - in the depth of the nasal speculum by 1.5 cm. After the use of live vaccines, complement-binding antibodies are detected in animals for 12 months. In this regard, before vaccination, control serological tests of the entire herd are carried out for the timely detection and removal of spontaneously infected animals.

Prevention and control measures. Ukraine is safe with CPP, therefore measures should be aimed at protecting the borders from the introduction of the causative agent of contagious pleuropneumonia by cattle coming from abroad, which is carried out by the border service of veterinary medicine. When animals latently infected with mycoplasmas are detected, they are immediately slaughtered at slaughterhouses specially designated for this purpose, with subsequent disposal of the carcasses together with the skin. The territory and premises where latently infected animals temporarily stayed during the quarantine are carefully cleaned and disinfected. For disinfection of premises, equipment, cars and other vehicles, a clarified solution of chlorinated lime containing at least 4% active chlorine or sodium hypochlorite containing at least 2% active chlorine is used, as well as a 2% solution of caustic soda, 2% formaldehyde solution. Clothes and shoes are disinfected in a paraformalin chamber, manure - by a biothermal method. All preventive measures must be carried out in accordance with the instructional documents in force in our country, under the control and with the direct participation of the state commission, which in such cases is created specially.

Control questions and tasks.

1. The causative agent of contagious pleuropneumonia of cattle and its characteristics
2. Epizootological data on contagious pleuropneumonia.
3. Clinical signs of contagious pleuropneumonia.
4. Pathological-anatomical changes in contagious pleuropneumonia
5. On the basis of what is the diagnosis of contagious pleuropneumonia made?
6. Does differential diagnosis of contagious pleuropneumonia involve exclusion?
7. How do you treat animals with contagious pleuropneumonia?
8. What disinfectants are recommended for disinfecting premises with contagious pleuropneumonia?

Topic: Plague of cattle

(diagnosis, prevention and control measures).

Plague of cattle (rinderpest ,Pestis bovum) is an acute contagious disease of ruminants, which is characterized by septicemia, hemorrhagic diathesis, inflammatory-necrotic lesions of the mucous membranes of the alimentary canal, diarrhea, high mortality.

In many countries of Africa and Asia, the disease appears periodically, spreads rapidly, causing extremely large losses to animal husbandry. Ukraine is safe from rinderpest. However, the presence of the disease in countries with which the state maintains business and trade-economic ties poses a constant threat of its introduction from abroad.

The causative agent of the disease. RNA-genomic virus from the Paramyxoviridae family. Virions are mostly spherical in shape. They are covered with an outer shell, on the surface of which characteristic protrusions (cilia) are visible. The rinderpest virus is antigenically homogeneous and is related to rinderpest, human measles, and canine distemper viruses.

The rinderpest virus is pantropic, in high titers it is found in the blood, lymph nodes, tears, mucous membrane of the rennet, lungs, and kidneys. The virus can be present in rennet ulcers up to 140 days after the clinical recovery of cattle. Among laboratory animals, rabbits, guinea pigs, mice, hamsters and ferrets are susceptible to the virus.

The virus is cultivated in the body of calves, rabbits, and chicken embryos, as well as in the primary culture of cattle leukocytes, calf kidneys, and cow embryo kidneys. Virus reproduction is accompanied by cell rounding and refractoriness, the appearance of stellate cells, the formation of giant multinucleated cells, cell syncytiums, symplasts, cytoplasmic and intranuclear eosinophilic inclusion bodies. Cell fragmentation occurs 6-12 days after infection. The persistence of the virus is insignificant. The virus is inactivated in urine, feces and pus - after 30 hours, at room temperature - after 3-4 days, at 60°C - after a few minutes, when boiling - instantly. It dies in the pasture after 36 hours. It is quickly destroyed by rotting, thanks to which in tropical countries, animal corpses are disinfected after only a few hours. Inactivated by all disinfectants; ultraviolet and solar radiation inactivates the virus within 40 minutes. In dried skins, the virus loses its pathogenicity after 24-48 hours. In a lyophilized state at plus temperatures, it is stored for 1.5 years, at 20°C – 5 years. In fresh meat, it does not die for 4-6 hours, in salted - up to 28 days. Citrate blood containing the virus remains active at room temperature for 4-6 days, at 5°C for 7 days, at 0°C for several weeks.

The diagnosis is made by a complex method on the basis of epizootological data, clinical signs and nature of the course of the disease, pathoanatomical changes, results of virological and serological studies of pathological material. In doubtful cases, a bioassay is performed on calves. The asymptomatic course of the disease in long-term infected herds is revealed only by the results of serological studies.

Epizootological data. From domestic animals, cattle, zebu, yaks, and buffaloes get sick under natural conditions. Young children up to 1 year are more susceptible, however, in stationary centers, they acquire colostral immunity for a period of up to 8-11 months. In epizootic centers, rinderpest can affect about 60 species of wild equids, primarily Kaffir buffalo, giraffes, and warthogs. Striped wildebeest, antelopes are less susceptible to this infection, small gazelles and half-breed riding horses are less sensitive. The latter do not get sick in a clinically expressed form, but can become a source of the pathogen for other animals, moving to a long distance from the source of infection. African sheep and goats are sensitive when experimentally infected, but do not play any role in the spread of the virus among cattle in tropical Africa. Camels are never included in the epizootic chain. African and European pigs do not get rinderpest. Among Asian breeds of pigs, the rinderpest virus causes a fatal infection.

The source of the causative agent of infection for cattle is clinically and latently sick animals, from whose body the virus is released with nasal secretions (2 days before fever and up to 9-10 days of illness), feces (3-8 days of illness), urine (1- 8 days of illness), milk, saliva, conjunctival mucus, vaginal discharge, with blood during bleeding (12-48 hours before the onset of fever and up to the 8th day of illness). Asymptomatic plague-stricken domestic and wild animals pose a significant danger as a source of the causative agent of infection. Factors of pathogen transmission can be corpses of dead animals, meat of forcibly slaughtered livestock and raw materials of animal origin (bones, horns, hooves, wool, skins, intestines, etc.). Mechanical transmission of the pathogen can occur through contaminated feed, water, litter, care items, transport, as well as when dogs, birds, and predators eat infected corpses. The transmissible way of transmission of the pathogen does not play a significant role in this disease. Natural infection occurs through the digestive tract when eating feed contaminated with secretions of sick or diseased animals, by contact - when keeping or grazing healthy and sick livestock together, possibly also through the conjunctiva of the eyes and respiratory tract. The plague is spread during contact with nomadic animals, in the case of drinking from sources contaminated with the pathogen, when wild susceptible ungulates are included in the epizootic chain.

Epizootics of rinderpest can occur at any time of the year. A characteristic feature of the infection is extremely high contagiousness, rapid spread over very long distances, high morbidity and mortality. In fresh epizootic centers, the plague is devastating, causing 90-100% mortality in animals of various ages. In hospital-affected areas, the disease has a limited character, it is found only in young animals from 10 months to 2 years of age. The mortality rate among them does not exceed 5-20%.

Rinderpest can be complicated by secondary infections or accompanied by piroplasmosis, trypanosomosis, eimeriosis and other diseases.

Clinical signs and course of the disease. The incubation period lasts 3-17 days. The course of the disease is acute, subacute, rarely superacute. The disease manifests itself in typical, abortive and latent (asymptomatic) forms.

With an acute course, the disease passes in a typical form. A sudden, sharp rise in body temperature to 40-42.2°C, acceleration of the pulse and breathing, thirst, slight redness of the mucous membranes, slight excitement, decreased lactation, leukopenia are observed. Photophobia, catarrhal rhinitis are detected. From the 3-4th day of the disease, signs of inflammatory-necrotic lesions of the mucous membranes of the digestive tract begin to dominate. On the inner surface of the lips and cheeks, on the gums of the incisors, tongue, palate and pharynx, there is diffuse hyperemia, numerous small centers of necrosis in the form of gray and light yellow nodules, as if covered with bran, in the place of which erosions and ulcers with uneven edges and bright - with a red bottom (Fig. 1). Purulent conjunctivitis, mucopurulent rhinitis and vaginitis develop. Damage to the mucous membranes is accompanied by significant drooling, secretion of mucous-purulent secretion with blood impurities from the nose, and purulent exudate from the vulva. The eyelids are swollen in the form of a roller, hyperemic, covered with hemorrhages, often glued with pus (Fig. 2). Coughing and sneezing are noted in sick animals, abortions are possible in pregnant cows. The temperature on the surface of the body is unevenly distributed, the horns and ears at their base are hot, the mirror is dry. Sometimes exanthema with the formation of nodules, crusts and focal baldness appears on tender areas of the skin. In the following days, the condition of the animals worsens significantly, diarrhea occurs with an involuntary act of defecation. Fecal masses are watery, gray-yellow or dirty-brown in color, contain mucus, blood, pieces of necrotized intestinal epithelium. Body temperature drops below normal, abdominal breathing, painful, with moaning. Dehydration of the body occurs, rapid weight loss, weakness. The animal lies with its limbs spread out on the ground, its head tucked under itself, and quickly dies. The duration of the disease is 4-10 days.

Fig. 1. Hemorrhagic-necrotic centers on the gums

Fig. 2. Swelling and hyperemia of the eyelids

(https://ru.scribd.com/presentation/529702266/%D1%87%D1%83%D0%BC%D0%B0-%D0%B2%D1%80%D1%85)

The subacute course of the disease is characteristic of hospital-affected areas. It is accompanied by fever, inflammation of the mucous membranes of the eyes, nasal and oral cavities, digestive tract, usually without necrotic lesions. On the 6-7th day, the body temperature decreases, ulcers and erosions on the mucous membrane of the mouth begin to heal, diarrhea stops. However, disorders of the functions of the digestive tract in the form of flatulence and periodic diarrhea remain for a long time. The duration of the disease is 2-3 weeks. Most of the animals recover, only the young die.

The acute course of the disease is accompanied by fever, septicemia, hemorrhagic diathesis phenomena. The death of the animal occurs within 1-2 days. With the abortive form of the disease, short-term fever, moderate diarrhea without damage to the mucous membrane of the oral cavity is observed. The prognosis is favorable. In the latent form of the disease, there are no clinical signs. Infection is detected by serological tests. In sheep, goats and camels, the course of the plague is atypical or latent (asymptomatic).

Pathological anatomical changes. The corpses of animals killed by the plague are emaciated, the skin in the area of the thighs and tail is contaminated with feces. The blood does not coagulate well, the mucous membranes of the lips, cheeks, gums, root of the tongue and hard palate are covered with gray-yellow brittle layers, under which significant erosions and ulcers are found.

The mucous membranes of the small intestines are diffusely hyperemic, permeated with band-shaped and diffuse hemorrhages, centers of necrosis, covered with gray-yellow nodules and diphtheritic layers with ulcers. Solitary follicles and Peyer's patches are greatly enlarged, hemorrhagically inflamed, with yellowish-cheesy scabs, after their removal ulcers. The esophagus and foreskin are usually unchanged. Mesenteric lymph nodes are sharply enlarged, juicy, hyperemic, with hemorrhages and

foci of necrosis. The lungs are full of blood, sometimes with foci of lobular catarrhal or croupous pneumonia. Edema, interstitial emphysema is often noted. The mucous membrane of the respiratory tract is swollen, reddened, with striped and speckled hemorrhages, covered with fibrinous plaques. The liver is flaccid, clay or saffron in color. The kidneys are swollen, hyperemic, covered with hemorrhages. The bladder is filled with cloudy urine mixed with blood, the mucous membrane is covered with hemorrhages. The spleen is slightly enlarged, with subcapsular hemorrhages. The gall bladder is very full of thick dark brown bile, the mucous membrane is hyperemic, with small ulcers. The heart muscle is weak, small hemorrhages are found under the epicardium and endocardium.

During a histological examination, the most characteristic and permanent changes are found in the lymph nodes, spleen, and lymphoid tissue of the intestines. At the beginning of the disease, a serous-catarrhal lymphadenitis is observed, which is accompanied by hyperplasia of cells of the reticulohistiocyte system and necrosis of lymphoblasts in the germinal centers of lymphofollicles. During the development of the clinical picture of the disease, total cell necrosis, atrophy of follicles, and serous-hemorrhagic lymphadenitis occur. In the liver and kidneys, granular and fatty dystrophy of parenchymal cells is determined, in the brain - non-purulent encephalitis and small hemorrhages.

Laboratory diagnostics. It is carried out in specialized diagnostic laboratories for particularly dangerous infectious diseases of animals in accordance with current methodological guidelines. For research, pathological material taken from sick animals during the period of the greatest manifestation of clinical signs of the disease (high temperature, serous-purulent discharge from the eyes and nasal cavity, presence of erosions on the mucous membrane of the nasal cavity, diarrhea) or from specially slaughtered animals is sent to the laboratory for research. for the diagnostic purpose of sick animals, or from dead animals no later than 4-6 hours after the moment of death. Blood and lymph node punctures are taken from sick animals, prescapular and mesenteric lymph nodes, pieces of spleen and liver are taken from corpses.

Laboratory studies involve isolation of the virus in the primary culture of leukocyte cells of cattle, primary cultures of kidney cells of a cow embryo or calf kidney, identification of the isolated virus using a neutralization reaction with a specific anti-plague serum, indication of the viral antigen in the organs and tissues of dead or forcibly slaughtered animals for diagnostic purposes according to the reaction of neutralization, reaction of binding of complement and reaction of immunofluorescence. Detection of specific antibodies in paired sera of infected animals is carried out by neutralization reaction, complement binding reaction, indirect hemagglutination reaction, delayed hemagglutination reaction (using the hemagglutinin of the human measles virus and monkey erythrocytes), as well as the ELISA method. In necessary cases, a bioassay is performed on three unvaccinated and two vaccinated (control) calves aged 6-12 months, which are injected subcutaneously

with 10 ml of undiluted blood taken from sick animals in the first days of the disease, or a 10-20% suspension of the spleen and lymph nodes from slaughtered sick animals. After 5-7 days, unvaccinated calves develop a typical clinical picture of the disease, which in 90% of cases ends in death after 5-12 days. In the pathological material of the dead animals, the viral antigen is detected by the complement binding reaction, the diffuse precipitation reaction, and the immunofluorescence reaction. Vaccinated calves remain healthy.

Differential diagnosis. It provides for the exclusion of malignant catarrhal fever, foot-and-mouth disease, viral diarrhea, pasteurellosis, hemosporidiosis.

Malignant catarrhal fever is characterized by the absence of contagiousness, the sporadic nature of the disease, diffuse keratitis, opacification of the cornea, and specific damage to the nasal and accessory cavities of the skull. Foot-and-mouth disease is characterized by aphthous lesions of the oral cavity, the skin of the inter-hoof gap, and the udder. The course of the disease is benign, without damage to hematopoietic organs, with low mortality. Positive bioassay on guinea pigs and rabbits. Viral diarrhea differs from the plague in that it is less contagious, the slow development of enzootic disease, a milder course, and low mortality. Pasteurellosis is differentiated by the absence of lesions of the mucous membrane of the oral cavity, the detection of pasteurella during bacteriological examination. Haemosporidiosis is ruled out on the basis of microscopic examination of peripheral blood smears. The final diagnosis is made based on the results of isolation and identification of the causative agent of the corresponding disease.

Treatment. Forbidden. Animals suffering from the plague are to be destroyed by a bloodless method followed by burning the corpses together with the skin.

Immunity. As a result of contracting plague in cattle, stable immunity is formed for a period of 5 years or more.

For active immunization of livestock against plague in the threatened zone, a dry virus-vaccine from the LT strain is used. Vaccinate all animals from the age of one month and older, including calving cows, regardless of the term of their calving. The vaccine is administered once, subcutaneously in the area of the middle third of the neck, in a dose of 1 ml. Immunity comes after 5 days and lasts for 2 years; in young animals up to 2 years of age - 1 year.

Prevention and control measures. They are based on the implementation of a whole set of organizational, anti-epizootic, and veterinary-sanitary measures aimed at protecting the territory of Ukraine from the introduction of the causative agent of rinderpest from disadvantaged countries, in the event of the appearance of the disease - on the organization and implementation of urgent actions in order to prevent its spread and eliminate it as soon as possible. In order to prevent the introduction of the plague from abroad, permanently operating regional services of state veterinary control at the border and transport were established, which are responsible for the control of all animals, products and raw materials of animal origin, feed for animals and poultry,

coming from abroad It is allowed to import animals and products of animal origin only from countries safe in terms of infectious diseases, in the presence of veterinary documents on the health of the animals and carrying out the prescribed preventive vaccinations. During the 21-day quarantine, permanent veterinary supervision of the condition of the imported animals is carried out, daily head temperature measurements and clinical and diagnostic examinations are carried out. In case of suspicion of hidden infection of animals with the plague virus, special serological and virological studies are carried out.

When rinderpest is detected, the entire point is declared plague-free, a quarantine is established, a dangerous zone is determined, security and quarantine posts are set up, barriers are equipped, and containers with disinfectant solution are installed. Measures are being taken to block all public roads and footpaths leading from the quarantined territory. Special announcements with the inscription "Quarantine. Passage, exit, entry is prohibited".To indicate detours and detours, clear signs are installed at all road intersections. Immediate measures are taken to ensure that unfavorable, threatening areas with respect to the introduction of the causative agent are supplied with a sufficient amount of rinderpest vaccine, tools, disinfectants, and special machines for disinfection. If fodder, products or goods need to be delivered to a disadvantaged area, transshipment points are organized. Cattle in a disadvantaged area are slaughtered by a bloodless method at a temporary slaughterhouse specially equipped for this purpose under the direct supervision of the chief veterinarian of the district. Carcasses and carcasses of slaughtered animals are burned together with skins. The territory of the slaughterhouse is thoroughly disinfected. All plague-susceptible animals of the unfavorable point and the threatened zone are vaccinated at the same time. They carry out thorough cleaning and disinfection of all livestock premises, as well as the entire territory of the unfavorable point. They burn wooden floors, partitions, equipment, care items, garbage, manure, feed residues.

Disinfection is carried out three times with an interval of one day. For disinfection of premises, livestock yards, corrals, equipment, cars, use a 2% solution of caustic soda at the rate of 1.5 liters of solution per 1 m2 of area, a clarified solution of chlorinated lime containing at least 4% active chlorine, sodium hypochlorite containing at least 2% active chlorine. Walls, fences and various fences are disinfected with a freshly prepared solution of quicklime or chlorinated lime, clothes, underwear, shoes - with formalin in a paraformalin chamber. Skins, skins, feathers and other raw materials obtained from healthy animals before the introduction of quarantine are treated in a decontamination chamber for 30 minutes at 60°C before being sent for processing.

Quarantine from an unfavorable point is lifted 21 days after the destruction of the last sick animal and the completion of appropriate final measures. After the lifting of the quarantine, 2-3 healthy 8-10-month-old bulls, which have not been vaccinated against plague, are introduced into the premises where sick quarantined animals were

temporarily kept for the purpose of biological testing. If the animals do not get sick within 30 days, it is allowed to place animals of other species here. New cattle that enter the farm and are susceptible to rinderpest are vaccinated and kept in isolation for 15 days. In the future, in the entire territory of the former unfavorable point, every year for 3 years, the entire cattle population must be vaccinated against plague.

Control questions and tasks.

1. How you can explain the widespread spread of rinderpest across the globe in the past and the steady unhappiness of the countries of Africa and Asia at the present time?
2. What are the epizootological features of the plague?
3. Forms of clinical manifestation of the disease and their main clinical signs.
4. What patho-anatomical changes are most characteristic of this disease?
5. Methods of diagnosis and from which diseases it is necessary to differentiate rinderpest?
6. Name the general and specific measures to prevent the introduction of the causative agent of the disease from abroad into the territory of Ukraine.

Topic: Malignant catarrhal fever of cattle

(diagnostics, prevention and elimination measures).

Malignant catarrhal fever of cattle (Coryza gangrenosa bovum, MCF) is an acute non-contagious disease of cattle and buffaloes, characterized by fever, inflammation of the mucous membranes in the head and intestines, damage to the eyes and severe nervous symptoms.

Malignant catarrhal fever is registered in many countries of Europe, Asia, Africa and America in the form of sporadic cases and limited enzootic outbreaks. The disease does not occur in Ukraine. Economic losses are insignificant due to the limited spread of the disease.

The causative agent of the disease is a DNA virus from the herpesvirus family. There are two types of virions: 140-220 nm in diameter with an outer envelope and a central capsid, and 100 nm in diameter with a net capsid. In the body of sick animals, the virus is contained in the blood, lymph nodes, parenchymal organs and brain. The antigenic structure of the virus has not been studied. Infection of animals is accompanied by the formation of virus-neutralizing and complement-binding and precipitating antibodies. The virus reproduces in primary cultures of cells of the thyroid gland of calves and sheep, bull testes, kidneys of rabbits, being mainly in a cell-bound form. The virus can also be cultivated in chicken embryos. The causative agent of the disease is unstable in the external environment and against physical and chemical influences. In natural conditions, it retains its activity for up to 35 days, in blood at

room temperature – 24 hours, at 4°C – 14 days. The virus is sensitive to ether and chloroform, inactivated by freezing. The long-term persistence of the virus in the infected body of animals against the background of the presence of specific antibodies has been established.

The diagnosis is made by a complex method on the basis of characteristic clinical signs (high temperature, damage to the mucous membranes, keratitis), pathological anatomical changes (necrotic lesions of the skin and mucous membranes, numerous hemorrhages, erosions and fibrinous layers on the mucous membranes of the intestines) and epizootological data (confinement to a certain territory , contact with sheep).

Epizootological data. Cattle and buffaloes are susceptible to the disease, mainly between the ages of 1 and 4 years. Adult animals, especially bulls, are more sensitive, calves rarely get sick. Individual cases of disease in sheep, goats, pigs, bison, moose, roe deer, giraffes, and antelopes are described. The source of the pathogen is sick cattle, the reservoir of the virus is sheep, goats, as well as various wild equids, in which the infection is asymptomatic. The possibility of endogenous infection in the event of a sharp decrease in the body's resistance has been established. The ways of isolating the pathogen from the body of sick animals and the mechanism of its transmission to healthy animals have not been fully elucidated. Along with this, the absence of contagiousness and transplacental infection in this disease was established. The participation of blood-sucking insects in the transmission of the virus to animals is also excluded. The disease occurs mainly in the form of sporadic cases, sometimes small enzootics with the daily appearance for 1.5-2 months of 1-2 of sick animals.

Clinical signs and course of the disease. The incubation period lasts from several weeks to 3-4 months. The course of the disease is acute, subacute and abortive.

With an acute course, a sharp increase in body temperature to 41-42°C and above, chills, lack of appetite, strong thirst, and cessation of chewing gum are observed. Alertness, sometimes, on the contrary, increased excitability of the sick animal is noted. Fever is of a constant type, breathing is accelerated, heavy, the pulse in the early stages of the disease is frequent, intense, and towards the end it becomes thread-like. Milk secretion stops. A generalized lesion of lymph nodes, fibrillary tremors of individual muscle groups are revealed. Within 1-2 days, lacrimation, swelling of the eyelids, photophobia appear, later keratitis develops, clouding of the cornea occurs (Fig. 1). In severe cases, ulcers appear on the cornea, the sclera breaks through, and the iris with the lens capsule falls out. Cataracts and blindness in one or both eyes often develop. At the same time, the mucous membranes of the nasal and oral cavities, accessory cavities of the skull are affected. The inflammatory process from the frontal sinuses can spread to the base of the horns, causing them to fall off. The mucous membrane of the nose is inflamed, covered with fibrinous layers, under which there are often ulcers (Fig. 2).

Fig. 1. Keratitis. Opacity of the cornea

Fig.2. Purulent rhinitis. Ulcers on the nasolabial mirror.

(https://www.nadis.org.uk/disease-a-z/cattle/malignant-catarrhal-fever-mcf/)

Narrowing of the nasal passages, first serous-mucous, and then purulent discharges with admixture of fibrin, which have a putrid smell, are observed. The mucous membrane of the mouth is dry, hot, sometimes there are erosions and ulcers on it, swallowing is difficult. There is a strong salivation, an unpleasant smell from the oral cavity. Cough, shortness of breath, wheezing in the lungs appear. Over time, diarrhea with blood and fibrinous films develops. Sometimes the genital organs are affected, fibrinous films and ulcers form on the mucous membrane of the vagina, abortions are possible. The inflammatory process sometimes spreads to the mucous membrane of the bladder, to the kidneys, causing cystitis and nephritis. In sick animals, urination is difficult and painful, the urine has an acidic reaction, protein, blood, cylinders, renal epithelium are found in it. The acute course lasts 4-10 days and ends fatally in 90-100% of cases.

In the subacute course of the disease, the same symptoms appear, but they are less pronounced and develop more slowly. The disease lasts 14-21 days and in 50-90% of cases ends with the death of animals.

In the case of an abortive form of the disease, the fever has a short-term nature, clinical signs are weakly expressed, sometimes completely erased. Diseased animals develop serious complications - blindness, agalactia. Relapses with a fatal outcome are possible.

Pathological anatomical changes. The corpses are exhausted and rapidly decomposing, the blood is dark and thick. A liquid with a putrid smell oozes from the nasal and oral cavities. The most characteristic are inflammatory changes in the mucous membranes of the head, especially the nasal cavity, and damage to the eyes. The eyelids are hyperemic, swollen, the cornea is cloudy, with ulcers, the conjunctiva with small hemorrhages. With an acute course, the mucous membrane of the nasopharynx, paranasal sinuses, larynx, and trachea is hyperemic, swollen, covered with numerous hemorrhages, purulent exudate, croupous-diphtheritic films, erosions

and ulcers in places. A similar picture is revealed during the examination of the pharynx, gums, palate, and tongue. Hemorrhages, catarrhal-hemorrhagic, croupous-necrotic inflammation of the mucous membrane, deep ulcers, erosions are observed in the alimentary canal. The cerebral ventricles contain a large amount of reddish cloudy fluid. Lymph nodes, especially mesenteric ones, are enlarged, juicy, sometimes with hemorrhages. Focal bronchopneumonia is observed in the front parts of the lungs, acute interstitial emphysema in the back. Spleen without pathological changes, liver and kidneys enlarged in volume, with hemorrhages. The mucous membrane of the bladder is hyperemic, thickened, covered with small hemorrhages. In the subacute course, similar changes are found, but the phenomena of hemorrhagic diathesis are much less pronounced. During the histological examination, ballooning dystrophy of the epithelium of the mucous membranes, especially of the oral cavity and pharynx, erosions and ulcers, perivascular tissue infiltration, mainly of the lymphocytic type, is revealed. Changes typical for non-purulent disseminated meningoencephalitis are observed in the brain - infiltration of soft meninges and brain matter with serous-fibrinous exudate, hemorrhages.

Differential diagnosis. It provides for the exclusion of plague, foot-and-mouth disease, rabies, and viral diarrhea. Rinderpest and foot-and-mouth disease are highly contagious and are not accompanied by meningoencephalitis and keratitis. During foot-and-mouth disease, aphthous and erosive lesions of the mucous membranes are combined with lesions on the extremities. Rabies is manifested by aggressiveness and paralysis, it is diagnosed based on the detection of specific Babesh-Negri bodies in brain cells, a bioassay on white mice, and the immunodiffusion reaction. Viral diarrhea is a contagious disease, it takes the form of enzootic disease, during an outbreak it affects up to 50% of the herd, mainly animals 5-6 months old, the main symptom of the disease is diarrhea.

Immunity. Not studied. Sick animals can get sick again after some time and die. No vaccine against malignant catarrhal fever is offered.

Treatment. No specific therapy has been developed. Heart drugs, 10% calcium chloride solution (200-300 ml intravenously), autohemotherapy (80-100 ml of blood injected subcutaneously twice in 48 hours), 40% alcohol (300 ml intravenously twice in 48 hours) are used as symptomatic agents. sulfonamide preparations (50-100 ml of 10% solution of norsulfazole in 10% glucose solution), broad-spectrum antibiotics. The mucous membranes of the oral and nasal cavities are washed with various antiseptic solutions, wounds are treated with antiseptic ointments. In case of significant difficulty in breathing, tracheotomy is performed, in case of accumulation of pus in the frontal sinuses, trepanation is performed.

Prevention and control measures. In order to prevent the disease, it is necessary to strictly follow the veterinary and sanitary rules for stocking, maintenance and operation of cattle and buffaloes. It is not allowed to keep cows together with potential carriers of the virus - sheep and goats. In the event of an outbreak of the disease, the

household or individual farm is declared to be unfavorable for malignant catarrhal fever and quarantine restrictions are introduced. In a dysfunctional farm, it is forbidden to breed and take out small and large cattle, use raw milk from sick and suspected animal diseases. Milk can be used in the household only after disinfection by boiling. It is forbidden to keep, graze and water cattle together with sheep and goats. In a dysfunctional farm, daily clinical examination and animal thermometry are carried out. Sick animals are isolated and treated. Systematic cleaning and disinfection of premises, inventory, animal care items, and vehicles are carried out. Animals are slaughtered only at a sanitary slaughterhouse or a site specially designated for this purpose under the supervision of a veterinary specialist. The corpses are buried in biothermal pits. Manure, bedding and feed residues are neutralized by the biothermal method. The farm is declared free of malignant catarrhal fever 2 months after the last isolation of the sick animal and the final disinfection. For disinfection, a chlorinated lime suspension containing 4% active chlorine, 5% xylonaphtha emulsion, a hot 2% solution of caustic soda, and a 20% suspension of freshly slaked lime are used when applied twice with an interval of 1 hour.

Control questions and tasks.

1. The causative agent of malignant catarrhal fever and its characteristics
2. Epizootological data on malignant catarrhal fever.
3. Clinical signs of malignant catarrhal fever:
4. Characteristic pathological changes in malignant catarrhal fever:
5. On the basis of what the diagnosis of malignant catarrhal fever is made.
6. What diseases does the differential diagnosis exclude?
7. How do they treat animals suffering from malignant catarrhal fever?
8. What disinfectants are recommended for disinfection of premises in case of malignant catarrhal fever?
9. Under what conditions is the farm declared safe from malignant catarrhal fever.

Topic: Infectious rhinotracheitis of cattle

(diagnosis, prevention and control measures).

Infectious rhinotracheitis of cattle (Rhinotracheitis infectiosa bovum, vesicular rash, "red nose", infectious rhinitis, infectious catarrh of the upper respiratory tract, infectious vulvovaginitis, vesicular disease of the genital organs, pustular vulvovaginitis, balanoposthitis) is an acute highly contagious disease characterized by fever , catarrhal-necrotic inflammation of the mucous membranes of the upper respiratory tract, keratoconjunctivitis and damage to the genitals.

Initially, infectious rhinotracheitis and pustular vulvovaginitis were considered separate diseases. At the same time, the genital form of bovine herpes called "vesicular

rash" was first described by Buchner (1841) in Central Europe. In the middle of the 20th century, vesicular exanthema with imported virus-carrying animals spread all over the world. F. M. Ponomarenko first described the respiratory disease in 1940 in Ukraine under the name "infectious catarrh of the upper respiratory tract". In the following decades, this form of the disease became widespread among young cattle in many countries with developed industrial cattle breeding. In 1954, the disease was diagnosed in the USA and, at the suggestion of Mac Kercher, in 1955, it was named "infectious rhinotracheitis". Infectious rhinotracheitis is registered in many countries of Europe, Africa, North America, Japan, Australia, and New Zealand. Favorable countries are Switzerland, Denmark, Sweden, Finland and Australia. In Ukraine, the disease was studied and described in detail by E. V. Andreev, V. S. Bilokon, O. O. Kucheryavenko (1975), V. A. Atamas (1986). The disease causes significant economic losses, which are determined by high morbidity, forced slaughter of sick animals, lethality (up to 12%), significant weight loss, reduction in milk yield (by 25%), abortions, reproductive function disorders in cows and bulls, costs of treatment and preventive measures.

The causative agent of the disease. A DNA-genomic virus belonging to the Herpesviridae family. Has a well-defined tropism to the epithelial cells of the mucous membranes of the upper respiratory tract and genitals.

In sick calves, the virus is detected in nasal secretions, conjunctival contents, tracheal mucus, saliva, blood, urine; in infected cows - in the aborted fetus, placenta, vaginal secretions; in infected bulls – in semen and urine. To isolate the virus, primary cultures of kidney or spleen cells of an embryo of a cow, kidneys and testicles of calves are used. The cytopathogenic effect appears 48-96 hours after infection in the form of rounding and granularity of cells, the appearance of syncytium and clusters of rounded cells in the shape of grapes, the formation of intranuclear oxyphilic inclusion bodies. Laboratory animals are not sensitive to the infectious rhinotracheitis virus.

The virus is stable in the external environment: it easily withstands repeated freezing and thawing, but quickly loses its activity in an acidic environment. Boiling kills the virus instantly, at a temperature of 56°C it dies within 60 minutes, solar radiation destroys the virus after 48 hours, ether, chloroform, acetone inactivate the virus at 4°C for 18-20 hours, at 37°C – 15 minutes . Solutions of formalin (1-2%), caustic soda (2%), phenol (3%) destroy the virus within 5 minutes.

The diagnosis is made by a complex method on the basis of epizootological data, clinical signs, patho-anatomical changes and the results of laboratory studies.

Epizootological data. In natural conditions, only cattle get sick, especially severely - 1-20-day-old calves and young animals on fattening. The source of the causative agent of the infection is sick and diseased virus-carrying animals. Infected breeding bulls, which contain the virus in their semen for a long time and infect cows during mating, as well as during artificial insemination, are very dangerous. The virus is released from the body of infected animals with discharge from the nose, eyes, and genitals, as well as with semen, milk, urine, and feces. Infection occurs by aerogenic

and contact routes and during steaming. Factors of transmission of the causative agent of infection can be contaminated fodder, bedding, animal care items, clothes and hands of service personnel, tools. Crowded housing and free mating of animals contribute to the spread of the disease. The disease has no pronounced seasonality and occurs at any time when the causative agent appears in the herd. In dysfunctional farms with industrial technology, the disease periodically appears 3-5 days after the next import of animals for stocking. At first, individual animals get sick, then the number of animals increases rapidly and reaches a maximum in 10-12 days. The fetus of a cow is very sensitive to the infectious rhinotracheitis virus, infection and death of which leads to abortion.

Clinical signs and course of the disease. There are respiratory and genital forms of the disease.

The respiratory form is observed mainly in calves 10-20 days old, which are imported for stocking farms operating on an industrial basis, as well as among fattening young animals. The course of the disease is acute, subacute and chronic. An acute course is observed during the initial outbreak of infection in a healthy farm when covertly infected animals are introduced into the herd, as well as in industrial-type enterprises in the case of mixing of imported calves during the period of stocking of "collective" livestock. The incubation period lasts 3-5 days. The disease begins with a sharp rise in body temperature to 41-42°C, lacrimation, salivation, and serous secretions from the nasal cavity. Accelerated superficial breathing, depression, decrease or loss of appetite, cough are noted. Over time, severe shortness of breath develops, the animal stands with its legs wide apart or lies with its neck stretched forward and its mouth open, from which a swollen tongue often falls out, foamy saliva is released. Sometimes there is sudden death from suffocation due to blockage of the lumen of the bronchi with a viscous exudate. Along with the respiratory syndrome, some animals have conjunctivitis and photophobia. With an acute course, from 10 to 20% of calves die within 2-5 days of the disease.

The subacute course is accompanied by an increase in body temperature to 41-42°C, hyperemia of the nasal mucosa, redness of the nasal speculum ("red nose"), depression, serous discharge from the nose, foamy salivation (Fig. 1). With the development of the disease, small necrosis centers and superficial ulcers appear on the mucous membrane of the nose and speculum. Leaks from the nasal cavity become mucous-purulent, smelly. Breathing is accelerated, shallow, pronounced shortness of breath. There is a dry cough, first short, and later loud, wet, conjunctivitis, sometimes diarrhea. Appetite worsens or completely disappears, exhaustion sets in, sick animals lie down. The duration of the disease is 7-10 days. When complicated by secondary microflora, bronchopneumonia often occurs.

Fig. 1. Serous discharge from the nose.
(https://uk.wikipedia.org/wiki/%D0%86%D0%BD%D1%84%D0%B5%D0%BA%D1%86%D1%96%D0%B9%D0%BD%D0%B8%D0%B9_%D1%80%D0%B8%D0%BD%D0%BE%D1%82%D1%80%D0%B0%D1%85%D0%B5%D1%97%D1%82)

The chronic course is observed at the end of enzootic disease, as a consequence of the acute and subacute course. The disease can last more than a month, often being complicated by a secondary infection.

With the genital form in cows, a short-term increase in body temperature, a decrease in appetite, a decrease in lactation, and frequent urination are noted. The mucous membrane of the vulva and the wall of the vagina are swollen, hyperemic, covered with numerous dark red nodules the size of a millet grain, which are surrounded by a bright red zone of inflammation. Over time, vesicles, pustules, diphtheritic films develop, after peeling of which ulcers are exposed. The back is curved, mucous-purulent exudate is released from the vagina. After 2-3 weeks, the general condition of the sick animal improves, recovery occurs. Calf cows have vulvovaginitis and abortions, which are accompanied by metritis and litter retention. Vulvovaginitis can also occur subclinically, which causes long-term virus-carrying (up to 570 days).

With the genital form in bulls, the disease is accompanied by fever, depression, decreased appetite, inability to mate. At the transition of the folds of the mucous membrane from the head of the penis to the prepuce, as well as on the mucous membrane of the preputial sac, small pink nodules are found, which burst on 4-5 days, forming ulcers and erosions. Pus is released from the preputial sac. On the 6-8th day, ulcers and erosions begin to heal without the formation of scars. After 12-14 days, the animals recover. There are cases of subclinical asymptomatic disease in bulls, which is accompanied by latent secretion of the virus with semen for up to 626 days.

Pathological anatomical changes. In the respiratory form of the disease, catarrhal inflammation and swelling of the mucous membrane of the nasal cavity, larynx, trachea, bronchi, accumulation of viscous mucous exudate in the nasal passages

and trachea are detected. The conjunctiva is swollen, hyperemic, sometimes there is gluing of the eyelids with purulent dried crusts. In complicated cases, the lumen of the trachea is filled with purulent exudate, on the mucous membrane of the nasal cavity and trachea, centers of necrosis and diphtheritic films are revealed. Interstitial emphysema of the lungs, hyperplasia of the mediastinal and bronchial lymph nodes, sometimes purulent catarrhal bronchopneumonia are often observed..

Laboratory diagnostics. It involves the detection of viral antigen in pathological material and in the semen of bulls using immunoenzymatic analysis, immunodiffusion reaction, diffuse precipitation reaction, polymerase chain reaction; isolation of the virus in cell culture followed by its identification by immunofluorescence, enzyme immunoassay, polymerase chain reaction, indirect hemagglutination and diffuse precipitation reactions; establishment of a 4-fold increase in titers of specific antibodies in paired samples of sera by neutralization reaction, agglutination reaction, indirect hemagglutination reaction, enzyme immunoassay, or determination of the level of accumulation of specific antibodies during simultaneous blood sampling in different age groups of cattle (representative sampling method). Detection of specific antibodies in blood sera in titers for neutralization reactions - 1:16 and higher, for neutralization reactions - 1:4 and higher, for enzyme immunoassay - 1:100 and higher, for indirect hemagglutination reactions - 1:16 and higher gives a reason for suspicion of infectious rhinotracheitis and necessitates further diagnostic studies in full.

Serous secretions from the nose, scrapings from the mucous membranes of the nasal cavity, vagina, prepuce, which are taken from sick animals during the period of maximum manifestation of clinical signs, are sent to the laboratory for intravital diagnosis. For postmortem diagnosis, in the case of forced or diagnostic slaughter of animals, scrapings or impressions are taken from the mucous membrane of the nose, larynx, trachea, vulva, bladder, as well as small (5x5 cm) pieces of lungs, liver, kidneys, spleen, no later than 2 hours later. lymph nodes, tonsils and affected areas of intestines. Pieces of liver, lungs, kidneys, spleen, abdominal and chest fluid are sent from aborted fetuses, and pieces of placenta and uterine cotyledons are sent from cows. Paired blood sera collected at the beginning of the disease and after 21 days are sent for serological examination of animals. Semen samples and washings from the foreskin are also sent from bulls for virological studies.

The diagnosis of infectious rhinotracheitis is considered established when positive results are obtained in one of the following cases: virus isolation from pathological material or sperm in cell culture and its identification in one of the reactions - neutralization reaction, immunofluorescence reaction, enzyme immunoassay, polymerase chain reaction; detection of infectious rhinotracheitis virus antigen in pathological material and semen using one of the reactions - immunofluorescence reaction, enzyme immunoassay, polymerase chain reaction, diffuse precipitation reaction; establishment of a 4-fold increase in the titers of specific

antibodies in paired blood serum samples or by the method of a representative sample (retrospective method).

Differential diagnosis. Presupposes the need to exclude malignant catarrhal fever, rinderpest, viral diarrhea, campylobacteriosis.

Malignant catarrhal fever occurs sporadically, it is not contagious, characteristic eye damage is noted - diffuse keratitis and fibrinous iritis. Rinderpest is accompanied by high contagiousness and lethality. With viral diarrhea, the main clinical sign is diarrhea, the disease covers up to 50% of livestock. With campylobacteriosis in cows, along with pustular vulvovaginitis, abortions are observed. The decisive role in the differentiation of these diseases belongs to the results of laboratory studies.

Treatment. Conduct hyperimmune serum or serum of convalescents, which are applied by aerosol. Serums are also used subcutaneously or intramuscularly at the rate of 2 ml per 1 kg of weight. Aerosols of thymol, 40% solution of lactic acid, iodine, chloroturpentine are recommended. They use expectorants and tonics. With the genital form, antibiotics of a wide spectrum of action are used. The microclimate is improved in livestock premises, and sick animals are provided with full rations.

Immunity. After the illness, the animals acquire strong immunity for at least 6 months. For active prevention, a dry virus vaccine against infectious rhinotracheitis and a dry culture-associated vaccine against infectious rhinotracheitis and parainfluenza-3 are used, and at breeding enterprises - an inactivated vaccine against infectious rhinotracheitis. Dry virus vaccine against infectious rhinotracheitis is used for preventive and compulsory vaccination of cattle in disadvantaged farms. Clinically healthy animals are vaccinated, the vaccine is not allowed for weak animals and fat cows. The vaccine is administered to young animals aged from 10 days to 4 months in 2 ml doses twice: the first - intranasal 1 ml in each nasal passage, the second - subcutaneous after 14 days in a dose of 2 ml. Animals older than 4 months of age are injected with the vaccine subcutaneously once in 3 ml in the area of the upper third of the neck. Immunity occurs 5-7 days after vaccination and lasts at least one year. Dry culture-associated vaccine against parainfluenza-3 and infectious rhinotracheitis of cattle is used in farms unfavorable for infectious rhinotracheitis and parainfluenza-3. The vaccine is administered to calves under the age of 3 months twice: the first - intranasally, 1 ml in each nostril, the second - after 14 days, 2 ml subcutaneously; young animals older than 3 months - intranasally 1 ml in each nostril, again after 14 days subcutaneously 3 ml. Immunity is formed 14 days after vaccination and lasts at least 3 months. Only inactivated vaccine is used in breeding farms.

Prevention and control measures. Depending on the production orientation of the farm, they include: measures to prevent infectious rhinotracheitis in commodity and farm farms; preventive measures at breeding enterprises; measures to eliminate infectious rhinotracheitis of cattle in commercial, breeding and farming farms; measures to eliminate infectious rhinotracheitis at breeding enterprises.

Measures to prevent infectious rhinotracheitis in commercial and agricultural farms. Ensuring compliance with veterinary and sanitary requirements regarding keeping, growing and feeding cattle in farms of various forms of ownership and subordination. Veterinary and sanitary requirements include: protection of the farm against the introduction of the causative agent of the disease, measures to increase the general resistance of animals, timely diagnosis of the disease, identification and isolation of sick animals, neutralization of the virus in the environment. Cattle should be brought to the farm only from IRT-safe farms. Only animals vaccinated with an inactivated vaccine can be imported from disadvantaged farms and only to farms with a similar epizootic situation. During the 30-day quarantine period, it is mandatory to conduct research on IRT. Beef cows are allowed to be imported no later than 3-4 months old and kept in a 30-day quarantine separately, under constant veterinary supervision. It is not allowed to import vaccinated cattle into IRT-free regions of the country. Bugaev-breeders are examined weekly by IRT by clinical methods and quarterly by serological methods. If the disease is suspected, serological tests of paired blood sera and virological tests of sperm and washings from the foreskin are carried out. In case of obtaining positive results, bulls are culled. When importing to farms where livestock vaccination against IRT is not carried out, animals are not vaccinated and after receiving negative results of serological tests, they are allowed to import. Imported batches of sperm are subject to mandatory virological testing, which, in case of positive results, are destroyed.

Measures to prevent infectious rhinotracheitis in breeding enterprises. In order to prevent the introduction of IRT in artificial insemination stations or in breeding farms, it is prohibited to purchase animals, sperm and embryos from farms that are unfavorable for this disease. During the 30-day quarantine period, clinical examination of the imported animals, thermometry and two serological tests, with an interval of at least 21 days, and, if necessary, virological tests of sperm and mucus from the foreskin are carried out. When receiving positive results of serological and virological studies, bulls are culled. If only positive serological results are obtained, bulls are vaccinated with an inactivated vaccine against IRT. All bulls are examined every quarter by serological and clinical methods to determine the state of the genitals and the quality of sperm.

Cows - egg donors and zygote recipients are examined clinically, gynecologically and serologically. In animals with inflammatory processes in the genital organs, pathological material is taken and examined by IRT using virological and serological methods. In the case of receiving positive virological results, they are culled, if positive results of only serological studies - they are vaccinated with an inactivated vaccine against IRT.

Measures to eliminate infectious rhinotracheitis in commercial, breeding and farming farms. When a diagnosis is established on the IRT, the farm is recognized as unfavorable for this disease, quarantine restrictions are introduced in it, and a plan of

measures to eliminate the disease is approved. In a dysfunctional farm, the purchase and sale of cattle, the regrouping of animals, the removal of fodder, care items and milk products from sick animals without prior disinfection are prohibited. A separate service staff is assigned to sick animals. In farms with an acute course of IRT, all healthy animals are immediately vaccinated with a live vaccine. Young animals from vaccinated cows are grown in isolation and when they reach 1.5 months of age, they are vaccinated with an inactivated vaccine against IRT. In stationary unwell farms, animals are vaccinated with a live vaccine, and after 6 months, in the absence of clinical manifestations of the disease, they switch to the use of inactivated vaccines. In disadvantaged farms, carcasses of slaughtered animals and milk from clinically healthy animals are used without restrictions. Milk from clinically ill animals is pasteurized at 70°C for 30 minutes. Restrictions on the farm are removed after the recovery of all animals and the implementation of veterinary and sanitary measures, but not earlier than 30 days after the last vaccination.

Measures to eliminate infectious rhinotracheitis at breeding enterprises. Quarantine restrictions are introduced at breeding enterprises that are unfavorable in relation to IRT, according to which it is prohibited to buy and sell animals, use them for reproduction, regrouping without the permission of the main veterinarian of the farm, the sale of sperm and embryos without virological control and the permission of the chief state inspector of veterinary medicine of the district. Clinically ill bulls are culled, and especially valuable bulls whose sperm has the IRT viral antigen are isolated and vaccinated with a live vaccine. The collection of sperm from them is stopped, and the sperm obtained in the last 90 days is destroyed. Bugai is isolated and treated with special chemotherapy drugs with antiviral and anti-inflammatory properties. After the clinical recovery of bulls, their semen is subjected to virological control twice with an interval of 30 days, and in the future - every quarter. In case of virus release during the year, they are culled, and other bulls are vaccinated with an inactivated vaccine twice a year. Semen from clinically healthy bulls are stored separately in Dewar vessels and used without restrictions. The semen of bulls that have been in contact with sick animals is examined once a quarter during the year. Twice a year with an interval of 6 months, they are vaccinated with an inactivated vaccine against IRT. In farms where seropositive animals are kept, the semen of all bulls is examined by virological methods at least twice a year. In stationary dysfunctional farms, animals are vaccinated with a live vaccine, and after 6 months, in the absence of clinical manifestations and virus secretion, they begin to use an inactivated vaccine. When specific antibodies in diagnostic titers are detected in bulls, they are isolated, a clinical examination is carried out and they are examined for the presence of a specific viral antigen. In the case of negative results of the virological examination of the semen, bulls are transported to a common room. In the future, their sperm will be examined every quarter throughout the entire period of operation. Restrictions on breeding enterprises are lifted after the

recovery of animals and the completion of veterinary and sanitary measures, but not earlier than 30 days after the last vaccination.

For disinfection of livestock premises and their adjacent territories, a 1% solution of formaldehyde, a solution of sodium hypochlorite with an active chlorine content of 1.5%, a clarified solution of chlorinated lime with an active chlorine content of 2% with exposure for 3 hours, and a 2% hot solution of caustic soda are used. Disinfection can also be carried out by the aerosol method, using formalin at the rate of 20 ml/m3 with exposure for 24 hours. The skins of dead and forcibly slaughtered animals are disinfected by soaking in a disinfectant solution: 50 g of aluminum alums, 200 g of common salt per 1 liter of water at 16-18°C for 48 hours. Milk obtained from sick and suspected cow diseases after pasteurization for 30 minutes. at 70°C they are used as food for humans and animal feed without restrictions.

Control questions and tasks.

1. The causative agent of infectious rhinotracheitis and its characteristics.
2. Epizootological features of infectious rhinotracheitis.
3. Forms of the clinical course of infectious rhinotracheitis and their main signs.
4. Pathological-anatomical changes in infectious rhinotracheitis.
5. When is the diagnosis of infectious rhinotracheitis considered established?
6. How is the treatment of sick animals with infectious rhinotracheitis carried out?
7. Means of specific prevention of infectious rhinotracheitis.
8. Describe the measures to prevent infectious rhinotracheitis in commercial, breeding and farming farms.
9. Measures to eliminate infectious rhinotracheitis in commercial, breeding and farming farms.

Topic: Bovine parainfluenza

(diagnosis, prevention and control measures).

Parainfluenza (Paragrippus bovum, transport fever, parainfluenza-3) is an acute contagious disease of young cattle, which is characterized by fever and damage to the respiratory organs.

The disease is registered in many countries of the world, especially with developed animal husbandry. In Ukraine, the disease was discovered and studied by V. I. Stetsenko (1975), E. V. Andreev (1979), and V. A. Atamas (1980). The economic losses caused by parainfluenza-3 are quite significant and are caused by high morbidity (up to 90%), reduction in animal weight gains (by 30-40%) and mortality (up to 20%).

The causative agent of the disease is an RNA-genomic virus that belongs to the family Paramyxoviridae, genus Paramyxovirus. The virus contains the neuraminidase

enzyme, hemagglutinin, as well as F-factor, which causes hemolysis and cell fusion. The high hemagglutinating and hemadsorbing activity of the virus is used during diagnosis and differential diagnosis of the disease. The close antigenic affinity of the bovine parainfluenza virus and the human parainfluenza-3 virus indicates the possibility of mutual infection. The PG-3 virus reproduces in primary cultures of kidney or lung cells of cow embryos, kidneys or testes of calves, as well as in transplantable lines Hela and Hep-2. The virus can multiply when it infects the amniotic cavity of 6-10-day-old chicken embryos. The virus is not resistant to the action of various factors of the external environment, ether, chloroform, acids, alkalis, heating, ultraviolet radiation. At room temperature, the virus dies in 2-3 hours, at +56°C - in 30-60 minutes, at +100°C - instantly. It quickly breaks down during freezing and thawing. A solution of formaldehyde (1-2%), caustic soda (0.5%), perchloric lime (1%) kills the virus after 5 minutes. The virus is well stored in a lyophilized state (up to 4 years), at sub-zero temperatures (at -60°C – several months) and at +4°C (up to 30 days).

The diagnosis is made by a complex method on the basis of epizootological data, clinical signs of the disease, patho-anatomical changes and the results of laboratory tests.

Epizootological data. Only young calves aged from 10 days to 12 months are susceptible to the disease. In adult animals, the course of infection is asymptomatic, accompanied by the formation of specific antibodies. Parainfluenza antibodies are detected in sheep, goats and camels. The source of the causative agent of the disease is sick animals that release the virus with exhaled air, secretions from the nose and eyes, drops of mucus during coughing, vaginal secretions with an aborted fetus and fetal membranes, possibly with feces and milk. Infection takes place when sick animals are kept together with healthy ones, through the airborne route, as well as through feed, water, litter, animal care items contaminated with the pathogen. The appearance and spread of the disease is facilitated by various stress factors that reduce the natural resistance of young animals - overheating, hypothermia, long-term transportation (hence the old name of the disease - "transport fever"), a significant crowding of animals in damp, poorly ventilated rooms, lack of vitamins and trace elements. Parainfluenza-3 takes the form of focal enzootics, is characterized by high contagiousness and a rapid course of the disease. Usually parainfluenza-3 covers up to 70-80% of the available herd of calves within 2-3 weeks, but the mortality does not exceed 20%. The duration of enzootic disease can be prolonged due to complications from secondary microflora (bacteria, mycoplasmas, chlamydia) or co-occurrence with oral, corona, or parvovirus infections.

Clinical signs and course of the disease. The incubation period lasts 2-5 days. The course of the disease is acute, subacute and chronic.

In the acute course, there is an increase in body temperature (up to 41-42°C), depression, superficial and rapid breathing, cough, serous discharge from the nose,

lacrimation, serous-purulent conjunctivitis (Fig. 1). There is increased sensitivity in the area of the larynx and trachea, hyperemia of the mucous membrane of the nasal cavity, later there are foci of dullness and wet wheezing in the lungs. Most animals recover within 1-2 weeks. In severe cases, on the 3rd or 4th day of the illness, discharge becomes purulent, ulcers and erosions appear in the oral cavity. Animals lie or stand with their necks extended forward, front limbs spread wide, often in a state of prostration, very depressed, lack of appetite (Fig. 2).

Fig. 1. Serous-purulent conjunctivitis

Fig. 2. The calf is in a state of prostration

(https://www.cabidigitallibrary.org/doi/10.1079/cabicompendium.91744)

In the subacute course, an increase in body temperature to 40-40.5°C, acceleration of the pulse and breathing, purulent discharge from the nose and eyes, depression, decreased appetite, and sometimes enteritis are noted. Shortness of breath is accompanied by a strong painful cough, wheezing, animals often breathe through their mouths. Pneumonia is determined by auscultation and percussion.

The chronic course, which, as a rule, is the result of secondary infection complications, is accompanied by signs of pleurisy and pneumonia. Lethality ranges from 5-20%. In pregnant cows, intrauterine infection of the fetus, abortions, and the birth of non-viable calves are possible.

Pathological anatomical changes. They are detected mainly in the apical, cardiac and diaphragmatic lobes of the lungs. Affected areas are enlarged, dense, blue-red, dark red or gray in color (red or gray stages of hepatization), often with signs of emphysema on the periphery. The interstitial connective tissue is swollen, the bronchial and mediastinal, less often the nasopharyngeal and cervical lymph nodes are enlarged, hyperemic, sometimes with foci of necrosis. There is also catarrhal inflammation of the mucous membranes of the nasal cavity and trachea with the accumulation of muco-purulent exudate, layering of fibrin on the surface of the pleura, epicardium, pericardium.

Laboratory diagnostics. It provides for the determination of parainfluenza antigen in pathological material by immunofluorescence method; isolation of the pathogen from sick and dead animals in the primary culture of kidney or lung cells of

a cow embryo, kidneys or testicles of calves; indication of the virus by cytopathogenic effect and hemagglutination reaction; identification of the isolated virus by hemagglutination delay reaction, neutralization reaction, immunodiffusion reaction and ELISA method; infection of 6-10-day-old chicken embryos in the amniotic cavity, indication and identification of the virus in extraembryonic fluid by hemagglutination reaction and hemagglutination delay reaction; detection of an increase in virus-neutralizing parainfluenza antibodies in paired blood sera collected on the 4th-5th day of the illness, and then after 14-21 days. Serous secretions from the nose and eyes, scrapings and smears from the mucous membrane of the nasal cavity are sent to the laboratory from sick animals for research, which are taken from the animals from the 2nd to the 5th day of the disease (during the period of the greatest manifestation of clinical signs of the disease). Pieces of the nasal membrane and trachea, lungs, spleen, kidneys, mediastinal and mesenteric lymph nodes, as well as paired blood serums are sent from corpses and slaughtered animals. Due to the low resistance of the parainfluenza virus, the pathological material is collected no later than 2 hours after the death of the animal and transported in a thermos with ice. After delivery to the laboratory, the pathological material is immediately examined, and in case of impossibility, it is immediately frozen at -20-60°C. For retrospective diagnosis, paired blood sera are examined using the neutralization reaction.

Differential diagnosis. Parainfluenza-3 must be distinguished from infectious rhinotracheitis, adenovirus infection, viral diarrhea, chlamydia and pasteurellosis.

Infectious rhinotracheitis is characterized by a slower and gradual development of enzootics, the formation of a vesicular rash and diphtheritic films on the mucous membranes of the respiratory tract and genital organs. The final diagnosis is established based on the results of isolation of the pathogen and its identification by neutralization reaction, immunofluorescence reaction, diffuse precipitation reaction, and enzyme immunoassay. Adenovirus infection is diagnosed based on the results of the complement binding reaction, neutralization reaction, immunofluorescence reaction, diffuse precipitation reaction, and delayed indirect hemagglutination reaction. Viral diarrhea is accompanied by erosive-ulcerative lesions of the mucous membranes of the alimentary canal, virus-infected cells lack intracytoplasmic and intranuclear inclusions. Pasteurellosis and chlamydia are diagnosed based on the results of bacteriological studies (detection of the causative agent in pathological material).

Treatment. It is carried out as early as possible, immediately after the appearance of the first signs of the disease. Hyperimmune serum and serum of convalescents are used. Serums can also be used subcutaneously in a dose of 2 ml per 1 kg of the animal's body weight. For the prevention of secondary infections, various antibiotics are added to the sera, which are selected based on the results of a preliminary determination of the sensitivity to them of the microflora of the respiratory tract of sick calves. Antibiotics of the tetracycline series of prolonged action, which are used for 3-6 days, are especially effective. Various combinations of other drugs are also used:

sulfadimezin and norsulfazole orally at a dose of 0.1 g/kg, penicillin at a dose of 20-30 thousand units/kg and streptomycin - 30 μg/kg subcutaneously, biomycin at a dose of 25 mg/kg, furazolidone - 7 mg/kg, chlortetracycline in a dose of 20 mg/kg, sulfadimezin - 0.2 g/kg, ascorbic acid - 5 mg/kg orally. Symptomatic and strengthening therapy is carried out. For the prevention and treatment of parainfluenza-3 in calves, vetazol, coniferous chlorophyll-carotene paste, and proteflazid are also used, which are drunk with milk or fed with compound feed for 20-30 days at a dose of 1.5 drops of the drug per 1 kg of body weight for preventive purposes and 3 drops per 1 kg of body weight - for therapeutic purposes.

Immunity. Calves infected with parainfluenza-3 become immune to re-infection within 3 months. Calves born from immune cows have colostral antibodies, which are stored in them for 3-9 months and, unfortunately, prevent the formation of immunity during parenteral administration of vaccines, while not protecting them from infection with epizootic strains of the virus. At the same time, intranasal vaccination of calves in the first weeks of life, even against the background of lactogenic immunity, causes active immunobiological restructuring of the body and protection of calves from infection.

For active immunization against parainfluenza-3, live lyophilized vaccine "Paravak" against parainfluenza-3 of cattle and dry culture-associated vaccine "Bivak" against parainfluenza-3 and infectious rhinotracheitis of cattle are proposed. The "Paravak" vaccine is used for preventive immunization against parainfluenza-3 in dangerous and disadvantaged farms. The vaccine is not harmful to calving cows, it can be used for vaccinations of calves from the age of 10 days. Immunity occurs after 7-10 days and lasts at least 6 months after revaccination. The dry culture-associated vaccine "Bivak" is used in farms unfavorable for these diseases. Immunity is formed 14 days after the start of vaccination and lasts at least 6 months.

Prevention and control measures. They include the protection of farms against the introduction of the causative agent of infection, the implementation of complex measures to increase the general resistance of the organism, the strict observance of veterinary and sanitary rules for the cultivation of young cattle, timely diagnosis of the disease, the destruction of the virus in the external environment (prophylactic disinfection). In order to protect the farm from the introduction of the parainfluenza-3 pathogen, it is necessary to stock farms only with healthy animals from breeding farms free of infectious diseases. Animals are delivered to the farm by specially equipped vehicles. The premises should be filled with young animals of the same age in accordance with the principle "everything is free, everything is occupied", without allowing crowding, drafts, dampness and high carbonation. Newly arrived animals are kept in quarantine for 30 days, under constant veterinary supervision. Thermometry and stock inspection are carried out for the timely detection of diseases among imported animals. Disinfection barriers are equipped, preventive disinfection is regularly carried out; visiting the farm by outsiders is prohibited. Passive or active

immunization of all imported livestock should be carried out in disadvantaged farms. During quarantine, calves are treated with aerosols of lactic acid or chloroturpentine, iodine chloride, and iodethylene glycol as a preventive measure. When the disease appears and the diagnosis is established, the farm is declared unhealthy for parainfluenza-3, quarantine restrictions are introduced in it, according to which it is prohibited to take animals to other farms and their regrouping. Sick and suspected animals are immediately isolated and treated. Sick animals are given peace of mind, provided with complete feed and optimal housing conditions without drafts, overcrowding, high humidity and gassiness. All healthy animals are vaccinated. Disinfection is carried out in the premises, sewage gutters are sprinkled with chlorinated lime. The carcasses of forcibly slaughtered animals, in the absence of degenerative changes in the meat, are released without restrictions. When inflammatory and necrotic foci are detected on the mucous membrane of the nasal cavity, trachea, lungs, and alimentary canal, they are subjected to technical disposal.

The farm is declared free of parainfluenza-3 and quarantine restrictions are removed from it 14 days after the last case of recovery or slaughter of a sick animal, as well as the final disinfection. For disinfection, a hot 2% solution of caustic soda, a solution of chlorinated lime containing at least 2% active chlorine, a 1% solution of formaldehyde, and a 20% suspension of freshly slaked lime are used for two applications. Manure is disinfected by the biothermal method.

Control questions and tasks.

1. The causative agent of parainfluenza-3 and its characteristics.
2. Epizootological features of the disease.
3. Forms of the clinical course of the disease and their main clinical signs.
4. Characteristic patho-anatomical changes.
5. When is the diagnosis of parainfluenza-3 considered established?
6. Describe general and specific preventive and health measures for bovine parainfluenza-3.

Topic: Viral diarrhea of cattle

(diagnostics, prevention and elimination measures).

Bovine viral diarrhea (Diarrhea viralis bovum, a disease of the mucous membranes) is a contagious disease of mainly young animals, characterized by fever, profuse bloody diarrhea, damage to the respiratory organs, conjunctivitis, rhinitis, erosive-ulcerative inflammation of the mucous membranes of the alimentary canal. Abortions are possible in pregnant cows.

The disease is widespread in many countries of Europe, America, Africa and Australia. The disease was first diagnosed in Ukraine in 1965.

The economic losses caused by viral diarrhea consist of the cost of dead and forcibly slaughtered animals, a decrease in milk productivity, the birth of non-viable calves, the costs of conducting veterinary and sanitary measures for the prevention and elimination of the disease.

The causative agent of the disease is an RNA-genomic virus belonging to the family Togaviridae, genus Pestivirus. Virions are spherical in shape, 30-40 nm in diameter, covered with an outer lipoprotein shell, which makes them sensitive to ether and chloroform. It is contained in almost all organs and tissues of sick animals, but in high concentration it is found only in the mucous membranes of the intestines, upper respiratory tract, and endothelium of blood vessels. Epizootic strains of the virus differ among themselves in virulence properties, tropism and cytopathogenic effect, but are identical in antigenic respect. At the same time, the antigenic and immunogenic affinity of the diarrhea virus to the classical swine fever virus was established.

Diarrhea virus reproduces in primary cultures of kidney, lung or spleen cells of a cow embryo, calf testicles, macrophages and lymphocytes. It is easy to infect calves 2-18 months old with the diarrhea virus by intravenous or intraperitoneal injection.

The virus is stable in the external environment: in blood, lymph nodes, spleen and other pathological material at 4°C it can be stored for up to 6 months, at minus 30-70°C for several years. In the culture liquid at minus 15°C, the virus is active for up to 1 year. It withstands repeated freezing and thawing well. At 37°C it is inactivated after 5 days, at 56°C - after 1 hour, at 100°C - instantly.

The diagnosis is made by a complex method, established on the basis of clinical-epizootological and patho-anatomical data, as well as the results of virological and serological studies.

Epizootological data. Only cattle are sick, mostly between the ages of 2 months and 2 years. There are reports of massive enteritis in newborn calves and abortions in cows. Buffaloes, deer, roe deer, antelopes, elk, doe can also get sick. Antibodies to the diarrhea virus were detected in sheep and pigs. The main source of the pathogen is sick animals, which for a long time secrete the virus into the external environment with saliva, tears, nasal discharge, urine and feces. Virus-carrying animals with a chronic and latent course of the disease, in whose lymphoid tissue cells the virus can persist for 120 to 200 days, pose a significant danger. Reinfection occurs by airborne droplets, during direct contact of healthy animals with sick ones, as well as in the case of eating feed contaminated with the virus. The possibility of intrauterine infection through the milk of infected mothers has been established. The disease occurs at different times of the year, but most often in late autumn and winter. The emergence and spread of the disease is facilitated by stress factors that reduce the body's resistance - hypothermia, poor nutrition, long-term transportation.

In the case of primary occurrence of diarrhea, it has an "explosive" nature, when almost all animals fall ill within 2-5 days. Mortality in viral diarrhea can reach 40%, and among newborn calves - even 70%. In stationary dysfunctional farms, the course

of the disease is indolent, with erased clinical signs, it is detected only by serological studies. Viral diarrhea can occur simultaneously with other viral diseases - infectious rhinotracheitis, parainfluenza-3, enterovirus or adenovirus infections, as well as be complicated by pathogens of various bacterial diseases.

Clinical signs and course of the disease. The incubation period lasts from 2 to 14 days. The course of the disease is acute, subacute and chronic.

The acute course of the disease is observed at the beginning of enzootic disease and mainly among young animals. The disease is manifested by a sudden increase in body temperature to 42°C, leukopenia (2-3 thousand cells in 1 mm3), depression, loss of appetite, increased breathing (48-90 beats per 1 minute) and heart rate (80-120 beats per 1 minute), mucous, and later mucous-purulent discharge from the nose, lacrimation. Small hyperemic blisters appear on the mucous membrane of the oral cavity and nasal speculum, which quickly turn into papules and vesicles, on the site of which erosions and ulcers are later formed. Ulcers are often found around the mouth and eyes, in the nostrils, vagina, in the area between the hoofs and on the skin of the crown. Salivation and diarrhea are observed. Feces have an unpleasant, fetid smell, are watery, contain a lot of mucus and blood clots.

Coughing, clouding of the cornea, and panophthalmia are observed in some animals. Diseased animals lose weight quickly, stand depressed, hunched over, hang around for a long time, and sometimes they have rectal prolapse. The main clinical sign of the disease is profuse diarrhea that lasts up to 3-4 weeks and leads to exhaustion and death of the animal. Abortions are possible in infected cows, especially in the early period of pregnancy, birth of calves with signs of viral diarrhea, which die in the period from 18 to 96 hours of life.

The subacute course of the disease is determined mostly in calves. It is manifested by low-grade fever, decreased or complete loss of appetite, lesions of the mucous membrane of the oral cavity, mucous-purulent discharge from the nose, short-term diarrhea (14-24 hours). Pregnant cows have abortions. The disease lasts for weeks, recovery is very slow.

The chronic course of the disease rarely occurs and develops as a continuation of the acute or subacute course. In sick animals, there is a decrease in appetite, prolonged moderate fever, exhausting diarrhea, severe cachexia. Erosions and ulcers appear on the mucous membrane of the oral cavity, which do not heal for a long time. The disease lasts 1.5-2 months and ends with the death of the animal. There are cases when the disease in cows is asymptomatic, infection is established only by the presence of specific antibodies.

Pathological anatomical changes. On the nasal speculum, gums, hard palate, on the surface of the tongue, in the nostrils, as well as on the mucous membranes of the entire alimentary canal, especially the jejunum and ileum, erosions and ulcers of various sizes and shapes are found (Fig. 1). The contents of the intestines are watery, mixed with mucus and blood, and have a foul smell. Mucous membranes of the rennet

and the small intestine are swollen, hemorrhagic, catarrhal or fibrinous-necrotically inflamed, with hemorrhages (Fig. 2).

Fig. 1. Erosions and ulcers on the mucous membrane of the ileum

Fig. 2. Hemorrhagic enterocolitis

(https://journals.sagepub.com/doi/full/10.1177/0300985816666610)

Mesenteric lymph nodes, Peyer's patches are enlarged and swollen. Hemorrhages in the gall bladder and under the kidney capsule. The liver is enlarged, with foci of necrosis or fatty degeneration. Bronchitis and bronchopneumonia are also detected. Inflammatory lesions of the mucous membrane of the oral cavity, esophagus, pharynx, trachea, prestomachs, lungs, skin and brain, as well as periamniotic membranes are observed in aborted fetuses of early age.

Laboratory diagnostics. Provides detection of virions by methods of electron and immunoelectron microscopy; determination of viral antigen in pathological material (smears, prints, tissue sections) from sick animals by immunofluorescence reaction; isolation of the pathogen from pathological material in the primary culture of kidney, lung or spleen cells of a cow embryo, testicles of calves with subsequent identification of the virus by neutralization reaction, immunofluorescence reaction, diffuse precipitation reaction, complement binding reaction and immunoenzymatic analysis; conducting a bioassay on 4-6-day-old or 2-6-month-old calves. 4-7 days after infection, the symptoms characteristic of viral diarrhea appear in calves: fever, mucous secretions from the nose, hyperemia, erosions and ulcers on the mucous membrane of the oral cavity, diarrhea with blood impurities. In the pathological material taken from them, the viral antigen is detected by the immunofluorescence reaction, and if necessary, isolation and identification of the virus is carried out; detection of specific antibodies in the blood sera of sick animals (retrospective diagnosis) is carried out by the neutralization reaction and the complement binding reaction and the ELISA method.

Blood samples for determination of antibody titer, as well as smears and scrapings from the mucous membrane of the nasal cavity are sent to the laboratory for

in-life diagnostics from sick animals in the early stages of the disease. The pathological material is placed in sterile vials with a buffer solution or medium for cell cultures with antibiotics, transported in a thermos with ice. Blood for virological studies is taken in the first days of the disease, and for serological studies - at the beginning of the disease and after 3-4 weeks.

Differential diagnosis. Bovine viral diarrhea must be differentiated from rinderpest, malignant catarrhal fever, foot-and-mouth disease, infectious rhinotracheitis, parainfluenza-3, bovine adenovirus, reovirus, and coronavirus infection, paratuberculosis, and necrobacteriosis. Plague is characterized by contagiousness, septicemia, hemorrhagic diathesis, ulcerative-necrotic lesions of mucous membranes, formation of intraplasmic and intranuclear inclusions in cells. It is differentiated by the results of the complement binding reaction, the diffuse precipitation reaction, and the neutralization reaction.

Malignant catarrhal fever is characterized by lack of contagion, occurs sporadically, with high mortality, typical eye damage (diffuse keratitis and fibrinous iritis). Foot-and-mouth disease is characterized by high contagiousness, rapid spread of infection, aphthous lesions on the tongue, udder, and in the area between the hoofs are characteristic. Pigs and sheep also get foot-and-mouth disease. The final diagnosis is established on the basis of the results of the complement binding reaction, diffuse precipitation reaction, neutralization reaction and bioassay on guinea pigs and mice and mammals. Infectious rhinotracheitis is accompanied by a predominant lesion of the upper respiratory tract, there is no diarrhea. Parainfluenza-3 affects the lungs. The diagnosis is easily established with the help of the hemagglutination reaction, the delayed hemagglutination reaction, and a bioassay on white mice. Adenovirus infection is registered mainly in newborn calves. The blood serum of sick animals contains complement-binding antibodies, which, in the reaction of complement binding, cause a delay in the hemolysis of ram erythrocytes. Reovirus infection affects calves up to 5 days of age. The incubation period is 12-13 hours. There is a strong depression of animals, reactivity, absence of any changes at autopsy. The final diagnosis is established on the basis of isolation and identification of the virus by hemagglutination delay reaction, complement binding reaction, diffuse precipitation reaction. Calves at the age of 8-9 days get sick with coronavirus infection. The incubation period is 20 hours, the duration of diarrhea is 5-6 days. Calves die after 48-62 hours. after the onset of the disease. The establishment of the diagnosis is based on the results of an immunofluorescent examination of feces and electron microscopic detection of the virus. Paratuberculosis is established by the allergic method, as well as by means of complement binding reaction and bacteriological studies. Necrobacteriosis is ruled out based on the results of microscopic studies and bioassays on rabbits.

Specific prevention. Live and inactivated vaccines are proposed for the specific prevention of viral diarrhea in cattle. The immunity of infected animals to re-infection lasts from 2-5 months to one year.

Treatment. No specific therapy is offered. Broad-spectrum antibiotics and sulfonamide drugs are used to prevent secondary infection complications. Orally, sick animals are prescribed synthomycin in a dose of 0.03-0.04 g per 1 kg of body weight 3-4 times a day until recovery, furazolidone with milk - 0.3 g (for a calf) three times a day for 3-4 days in a row . Calves are given 2 liters of aqueous solution containing 80 g of the specified mixture three times a day instead of milk. The course of treatment is 3-5 days. Severely ill calves are injected intravenously with a 33% ethanol solution in a 40% glucose solution at the rate of 1 ml/kg of weight. The oral cavity is washed with a 0.1-0.2% solution of potassium permanganate.

Prevention and control measures. Prevention and control measures are based on preventing the introduction of the causative agent of the disease into the farm. The herd must be supplemented with young animals only from prosperous farms, and the animals that come for the herd must be kept in quarantine for 30 days. In the case of an acute outbreak of viral diarrhea in previously prosperous farms, sick animals are immediately slaughtered, the premises, places where animals are kept, and tools for their care are thoroughly disinfected. Healthy animals are kept in isolation under constant veterinary supervision, vaccinated with inactivated vaccines. Strict restrictive measures are introduced in the farm, the importation and export of animals to other farms, the regrouping of animals, as well as the visit of unhealthy premises by outsiders are prohibited. It is allowed to take animals on specially equipped transport only for slaughter to the meat processing plant. In the event of disease in a stable farm, sick animals are isolated and treated. The rest of conditionally healthy animals are vaccinated with live vaccines. Animal carcasses are disposed of. A complex of veterinary and sanitary measures are carried out, aimed at preventing the spread of the causative agent of the disease, including disinfection of premises, adjacent territory, machines, care items, equipment and vehicles. The farm is declared safe from viral diarrhea and quarantine restrictions are lifted 14 days after the last case of recovery or slaughter of a sick animal and final disinfection. For the disinfection of premises, a clarified solution of chlorinated lime containing at least 5% active chlorine, a 20% suspension of freshly slaked lime, a 10% hot solution of a sulfur-carbolic mixture, an alkaline solution of formaldehyde containing 3% formaldehyde and 3% caustic soda is used, 10 % hot solution of caustic soda after exposure for 1 hour.

Control questions and tasks.

1. The causative agent of viral diarrhea and its characteristics.
2. Epizootological data of viral diarrhea.
3. Clinical signs of viral diarrhea.
4. Characteristic pathological changes in viral diarrhea.
5. The diagnosis of viral diarrhea is considered established on the basis.
6. Differential diagnosis of viral diarrhea.
7. In inpatient dysfunctional farms, patients are identified:

8. How to deal with milk from sick animals?
9. How to treat animals with viral diarrhea:
10. How is the premises disinfected in case of viral diarrhea?
11. When the farm is declared safe from viral diarrhea?

Topic: Spongiform encephalopathy of cattle

(diagnosis, prevention and control measures).

Spongiform encephalopathy (transmissible spongiform encephalopathy) belongs to prion infections and is characterized by a long incubation period, a slow course, signs of damage to the central nervous system, and diffuse dystrophic encephalopathy. The disease always ends fatally.

The disease is registered in Ireland, Switzerland, France, Denmark, Portugal, Canada, Germany, Italy, Oman and the Falkland Islands, where covertly infected cattle were imported. Bovine spongiform encephalopathy is not registered in Ukraine.

The causative agent of the disease. Abnormal cellular prion (Pr PCS), which is a protein infectious agent, which differs dramatically in its physical and chemical properties from all known pathogens of infectious diseases. The abnormal prion is created as a result of the transformation of the normal cellular prion protein (Pr Ps/C – Cell), forms clusters in the form of fibrils that can cause the destruction of neurons, spongy changes in the gray matter of the brain, and the formation of vacuoles. The mechanism of the spontaneous transition of the normal cellular prion protein to the abnormal form of the prion remains unclear until now, although there are several hypotheses, including mutational-catalytic. Anomalous cellular prion associated with the plasma membrane of cells, contains a very small amount of nucleic acid, does not cause an immune reaction in the body of an infected animal, therefore it cannot be determined by serological research methods.

The abnormal cellular prion is extremely resistant to the action of chemical and physical factors, including disinfectants. Its complete destruction is not achieved even by long-term autoclaving. It is inactivated at 100°C after only 3 hours, at 180°C – after 30 minutes.

The diagnosis is made by a complex method based on the analysis of the epizootological situation, clinical signs of the disease, characteristic pathohistological changes in the brain and the detection of specific fibrillar formations during an electron microscopic examination of the brain.

Epizootological data. In natural conditions, cattle are sick, more often cows than bulls. In zoos in England, spongiform encephalopathy was found in five species of antelope and two species of deer that were fed meat and bone meal made from the carcasses of sheep killed by scrapie. Spongiform encephalopathy in minks has also occurred after feeding meat from scrapie-affected sheep. Diseases of domestic cats fed canned meat have been established. Today, there is a hypothesis of the probability of transmission of the causative agent of the disease to cattle through insufficiently disinfected feed (meat and bone meal) from waste obtained from slaughtered sheep with scrapie. There is a suspicion that the disease begins to spread horizontally after its occurrence. Evidence of vertical or horizontal ways of transmission of the causative agent of the disease in natural conditions between cattle or between large and small cattle was not found. Despite the long-term use of the same pastures for sheep and cattle, no diseases were observed among cows either. The possibility of infection of calves, sheep and pigs with a specific clinical and pathological manifestation of the disease by intracerebral and intravenous administration of suspension from the brains of sick cows was experimentally proven. In laboratory mice, specific signs of the disease appeared after oral and parenteral infection. The question of the possibility of transmission of the causative agent of the disease from cattle to humans remains unexplored. It is assumed that the spread of spongiform encephalopathy among different species of animals is not related to a sudden mutation of the causative agent of scrapie, but to the penetration of large doses of the protein infectious agent of scrapie into the body and the breakthrough of species immunity. The infectious agent is detected in the brain and spinal cord, as well as in the retina. The causative agent of the disease was not found in milk, blood, bone marrow, digestive tract, heart, tonsils, nerves, lymph nodes, skin, reproductive organs, spleen and trachea.

Clinical signs and course of the disease. The incubation period lasts from 22 months to 8 years or more. Signs of the disease appear only in animals older than 2 years, develop slowly, within 1-4 months. There is no fever, appetite remains, but sick animals lose weight, milk productivity decreases, and complete exhaustion gradually sets in. Almost all sick animals show bradycardia and hyperglobulinemia. The clinical signs of the disease are very similar to those of sheep scrapie and are primarily characterized by a sharp change in the animal's behavior. In sick cows, increased excitability is noted, which is replaced by depression, impaired sensitivity to auditory, light and tactile stimuli. Sharp noises cause fear in animals. Over time, great aggressiveness ("mad cow" syndrome), teeth grinding, lip muscle tremors, and nasal speculum tremors appear. In sick animals, hyperesthesia is strongly expressed in the area of the head and neck, and in some - the entire surface of the body. There are signs of impaired coordination of movements, stumbling, manege movements, abnormal gait in the form of horse-like trotting movements, sudden fall, sometimes muscle tremors in the lower neck and shoulders, convulsions, paresis, dependence (Fig. 1). Sick

animals do not react to obstacles, run into walls, trees, people. The death of the animal occurs after 3 weeks - 6 months after the appearance of clinical signs of the disease.

Fig. 1. Hyperesthesia, impaired coordination of movements.
(https://www.cfs.gov.hk/english/multimedia/multimedia_pub/multimedia_pub_fsf_183_02.html)

Pathological anatomical changes. They are localized only in the brain and are detected during histological, electron microscopic and immunochemical studies. In the brain, dystrophic cells and vacuoles of a rounded or egg-shaped shape are observed, which are located in the gray matter between the large hemispheres and the cerebellum, and in the medulla oblongata - immediately behind the cerebellum. Damage is especially often detected in stem neurons, in the cytoplasm of which large spherical or egg-shaped vacuoles are found. The affected brain resembles a sponge, which is why the disease got the name "spongy encephalopathy".

Laboratory diagnostics. Methods of laboratory research of bacteriological and viral diseases turned out to be unsuitable for the diagnosis of spongiform encephalopathy. Recently, an immunohistological method, a method of immunoenzymatic analysis and a method of detecting scrapie-associated fibrils have been proposed for the indication of prion protein, which, however, due to their complexity, can only be performed in specialized laboratories.

Differential diagnosis. Presupposes the need to rule out rabies, Aujeski's disease, listeriosis, nervous forms of ketosis, hepatic encephalopathy, and hypomagnesemia. For this purpose, epizootological data, clinical signs, results of virological, bacteriological and biological studies are used.

Treatment. Not conducted. Due to the high potential danger to humans, all infected animals are destroyed.

Immunity. Means of specific prevention and chemotherapy have not been developed.

Prevention and control measures. The main method of combating bovine spongiform encephalopathy is the ban on the use of meat and bone or bone meal made

from the carcasses of scrapie-affected sheep for feeding livestock, as well as the slaughter of prion-infected ruminants. Since Ukraine is free of bovine spongiform encephalopathy and sheep scrapie, all measures should be aimed at preventing the introduction of the pathogen from abroad with imported breeding animals, semen, embryos, as well as meat and bone meal and other feed of animal origin. In this regard, it is prohibited to import animals, raw materials and products of livestock origin into Ukraine from countries that are unfavorable for cattle spongiform encephalopathy and sheep scrapie, as well as re-export from other regions of the world, if these products were produced in the specified unfavorable countries. In case of suspicion of spongiform encephalopathy in cattle, veterinary medicine specialists are obliged to immediately take all necessary measures in accordance with the current instructional documents.

An 8% solution of sodium hydroxide, a 20% solution of sodium hypochlorite or concentrated formic acid is recommended for disinfection of places of temporary detention of animals suspected of being infected with prions. Manure, care items, feed residues are burned.

Control questions and tasks.

1. The causative agent of spongiform encephalopathy and its characteristics.
2. Epizootological features of spongiform encephalopathy.
3. Clinical signs and course of the disease.
4. Pathological-anatomical changes.
5. What studies are conducted when diagnosing spongiform encephalopathy?
6. From what diseases is it necessary to differentiate spongiform encephalopathy?
7. What disinfectants are used to disinfect places of temporary detention of animals suspected of being infected with prions?
8. Measures to prevent and combat spongiform encephalopathy.

Topic: Nodular dermatitis of cattle

(diagnosis, prevention and control measures).

Nodular dermatitis of cattle (nodular exanthema, infectious nodular dermatitis, Dermatitis nodulares, Lumpy skin disease) is a contagious infectious disease characterized by fever, damage to the lymphatic system, swelling of the subcutaneous tissue and internal organs, the formation of skin nodules (bumps), damage to the eyes and mucous membranes membrane of the respiratory and digestive organs.

Cattle, sheep, goats and ruminant wild animals are sick. The disease is classified as a particularly dangerous disease of cattle. Nodular dermatitis causes significant economic losses due to the fact that it causes a temporary decrease in milk productivity,

temporary or permanent sterility of breeding bulls, skin lesions, and the death of sick animals due to secondary infections.

Nodular dermatitis of cattle has some similarity to smallpox, but there is no clear staging in the development of skin nodular exanthema. First, the virus reproduces at the point of entry into the body, then it is carried by blood into the sensitive epithelial cells of the skin, mucous membrane of the alimentary canal and respiratory tract, where it causes a typical nodular process. Mass appearance of nodules on the skin is observed 7-20 days after infection.

The causative agent of nodular dermatitis. A DNA-containing virus of the Neethling group, genus Capripoxvirus of the Poxviridae family. The virus remains viable in affected parts of the skin for at least 33 days, in saliva – 11, in blood, urine, milk, semen, secretions from the nasal cavity and eyes, mucous membranes and internal organs of infected animals – 4 days. In samples of affected skin taken after 3 months, viral nucleic acid is detected in the polymerase chain reaction.

Neethling virus is resistant to triple freezing, but sensitive to 20% solution of ether, chloroform, etc. The pathogen is inactivated at a temperature of 55°C for 2 hours, at 60°C for 30 minutes. At 4°C, nodular dermatitis virus remains active for 6 months.

The diagnosis is made by a complex method based on the results of a clinical examination of sick animals and conducting laboratory tests.

Epizootological data. The source of the causative agent of infection is sick, terminally ill and latently infected virus-carrying animals. The virus is transmitted in a transmissible way - by mosquitoes, midges and houseflies. Transmission of the pathogen is possible during direct contact between sick and healthy animals, sexually, in calves - through milk contaminated with the pathogen.

The virus enters the external environment with rejected pieces of affected skin and virus-containing sperm, saliva, and blood. The virus is released with semen within 60 days after the clinical recovery of breeding bulls. In compacted skin nodules, the virus can be detected within 120 days from the moment of their appearance. The disease is registered mainly in lowland areas, in wet and warm weather. Morbidity is 30-75% (rarely 100%), mortality is 10%.

Clinical signs and course of the disease. The incubation period lasts 3-14, more often 7 days. The course of the disease is subacute and chronic. The prodromal period is short, erased, especially during the appearance of the first cases of the disease. Diseased animals have fever (increase in body temperature up to 40°C), hyperemia and swelling of the mucous membranes of the oral and nasal cavities, genitals, discharge from the eyes and nose. Edema develops in the area of the chest, udder, and limbs, and gait becomes stiff. Sick animals refuse feed, lose weight quickly, and their milk production drops sharply. Superficial lymph nodes increase sharply in size, look like tumors, sometimes abscess. Shortly after an increase in body temperature, dense rounded or slightly elongated nodules with a flat surface, 0.5-0.7 cm in diameter, up to

0.5 cm high, appear on the skin in the neck, head, chest, abdomen, and limbs, which are clearly visible in animals with short hair, easily felt by palpation (Fig. 1).

Fig. 1. Round nodules in nodular dermatitis (https://biocor-tech.com/blog/nodulyarnyy-dermatyt-velikoyi-rogatoyi-hudoby-diagnostika-likuvannya)

Also, nodules can appear on the udder of lactating cows, and the milk can acquire a pink tint and a gel-like structure. The number of nodules ranges from several dozen to several hundreds. After some time, the epidermis begins to separate at the edges of the nodules, and characteristic depressions with necrotic dense tissue are formed in the center, which are separated from healthy tissue by a ridge of granulation tissue 1-3 mm wide.

With an acute course, there is an increase in temperature up to 40°C, discharge from the nose, lacrimation. After two days, dense nodules form on the skin and gradual tissue necrosis begins. The necrotized area falls off 7-20 days after the appearance of the tubercle.

If the course of the disease is severe, the animal suffers from prolonged fever and severe damage to the gastrointestinal and respiratory organs. Necrotic nodules fester and turn into ulcers. If the eyes are affected, partial or complete blindness may occur. With swelling of the respiratory tract, dermatitis in cows leads to suffocation.

The atypical form is most often registered in newborn animals and is accompanied by an increase in temperature and diarrhea (there are no signs of skin damage).

Laboratory diagnostics. Affected particles of skin, subcutaneous tissue and mucous membranes are selected for research. Saliva, secretions from the eyes and nose are also examined. Laboratory studies include:

1. Conducting PCR. This makes it possible to identify the causative agent and differentiate it from the related goat and sheep pox viruses.

2. An express method for determining skin dermatitis in cows is electron microscopy. For laboratory research, the virus is isolated in cell culture.

3. Serological control using enzyme immunoassay and neutralization reaction.

It should also be taken into account that the symptoms of nodular dermatitis are similar to a number of other diseases, including urticaria, demodicosis and others. Sometimes this disease is confused even with ordinary insect bites, therefore, with the appearance of any nodules on the skin of animals, it is necessary to carry out laboratory tests.

Differential diagnosis. Nodular dermatitis must be differentiated from dermatophilosis (a chronic skin lesion characterized by the formation of papules associated with the surface layers of the skin, covered with crusts and raised above the skin); tuberculosis (nodules are localized along the lymphatic vessels of the limbs and neck, nodules are subcutaneous and persist much longer); skin reactions to insect bites (clearly visible painful lesions that do not have clear boundaries, nodules are soft and vague).

Treatment. Means of specific therapy have not been developed. Symptomatic treatment is carried out.

Prevention and control measures. Provide for preventive measures against the introduction of nodular dermatitis from countries unfavorable for this disease. Animals can only be brought into the farm with the permission of the State Production and Consumer Service. Taking animals to pasture is allowed after their preliminary treatment with means that protect animals from insect bites. When animals are stabled after grazing, a mandatory clinical examination of the animals is carried out. Preventive disinfestation of livestock premises is carried out. When a disease occurs in previously prosperous regions, all sick and suspected animal diseases are immediately slaughtered, thoroughly disinfected and disinfested, and all quarantine and restrictive veterinary and sanitary measures are carried out. One of the strategies for the prevention of this disease is the vaccination of favorable cattle herds. Animal owners, farm veterinarians, in case of suspicion of nodular dermatitis in animals, are obliged to inform the veterinary medicine service staff without fail. When a diagnosis of nodular dermatitis is established in an epizootic focus and a problem area, restrictive measures are taken, quarantine is established. Sick and infected animals are isolated, removed and destroyed or, with the consent of the animal owners, stamping-out (destruction of all susceptible livestock) is used. The export of milk outside the boundaries of the disadvantaged farm and the protection zone is allowed only after sterilization at a temperature of 132°C for 15 seconds or boiling.

Control questions and tasks.

1. The causative agent of nodular dermatitis and its characteristics.
2. Epizootological features of the disease.
3. Forms of the clinical course of the disease and their main clinical signs.

4. Characteristic patho-anatomical changes.
5. When is the diagnosis of nodular dermatitis considered established?
6. Describe general and specific preventive and health measures for nodular dermatitis.

DISEASES OF SHEEP AND GOATS

Topic: Bradsot

(diagnosis, prevention and control measures).

Bradsot – acute non-contagious toxic infection of sheep, characterized by hemorrhagic inflammation of the mucous membrane of the jejunum and duodenum and degenerative changes of parenchymal organs.

The causative agents of the disease are spore toxin-producing anaerobes - C. septicum, C. novji (C. oedematiens) and C. perfringens. The main role in the etiology of the disease is played by C. Septicum - a thin, polymorphic, gram-positive motile rod that does not form capsules. Spores have an oval shape, placed subterminally. They are well cultivated on all nutrient media for anaerobes. In the Kitt-Tarozzi broth, after 16-20 hours, it causes intense turbidity, gas formation, followed by precipitation and clarification of the broth. On Zeissler's blood agar, it appears as a delicate veil-like plaque or round colonies with a transparent zone of hemolysis.

Among laboratory animals, guinea pigs are very sensitive to all types of clostridia. The causative agent in spore form is extremely resistant in the external environment and to physical and chemical factors. It remains viable in the ground for years, when boiled, it is destroyed after only 60 minutes.

The diagnosis is made by a complex method on the basis of typical clinical signs and epizootological data, confirmed by bacteriological examination of pathological material.

Epizootological data. The disease affects sheep and goats of different breeds and sexes. The disease is more often observed in mothers, as well as in young, well-fed, sedentary animals aged from 3 months to a year. Older sheep get sick from grazing, and lambs 3-7 months old get sick from stables. The emergence and spread of brazot is facilitated by sharp cooling or overheating of animals, eating frozen, frost-covered fodder, grazing on snow, dew in areas of flooded meadows, rivers in lowlands, animals drinking from stagnant water bodies.

The source of the causative agent is sick sheep and goats, as well as bacillus carriers that excrete the causative agent with feces. Factors of transmission of clostridia can be untimely removed corpses, water, fodder, pens, pastures, contaminated with fecal secretions of sick and sick animals, as well as hay harvested on unhealthy meadows, silage and other fodder contaminated with soil. Infection occurs alimentary through the alimentary canal. The disease is registered mainly in autumn, winter and

early spring during a sharp cooling. Bradzot passes in the form of sporadic cases and small enzootics. In many farms, it is observed every year due to the extremely long persistence of the pathogen in the ground, which ensures the stationary nature of the infection. Morbidity in rabies is 15-20%, lethality in hyperacute course can reach 100%.

Clinical signs and course of the disease. The incubation period is extremely short. The course of the disease is most often lightning-fast (hyperacute), hence the name "bradsot" (Danish term - "rapid illness"). An acute course of the disease is much less common.

In a lightning-fast course, sick animals die suddenly, with signs of strong tympany, convulsions, and grinding of teeth. Often healthy sheep are found dead in the morning. In the case of an acute course, fever, depression, rapid breathing, lack of chewing, sudden disordered movements, and the release of frothy bloody saliva from the oral cavity are observed in sick animals. In the area of the head, submandibular space and tongue, swelling is sometimes noted, bloating and pain in the abdomen, bloody diarrhea are quite often observed (Fig. 1). Sick animals die 2-8 hours after the onset of the disease with signs of general weakness and asphyxiation.

Fig. 1. Tympania
(https://www.google.com/url?sa=i&url=https%3A%2F%2Fxn--b1agjaajcft0c.xn--p1ai%2Fbolezni%2Fbradzot%2F&psig=AOvVaw1FbzSmfOgPMywodDw8Fgyj&ust=1690290278431000&source=images&cd=vfe&opi=89978449&ved=0CBMQ3YkBahcKEwiA6p6DtaeAAxUAAAAAHQAAAAAQBA)

Pathological anatomical changes. The corpses of animals killed by brazotos are greatly bloated, the blood does not coagulate well. A characteristic feature is very fast decomposition of the corpse. A frothy bloody liquid is released from the oral and nasal cavities and anus. Bloody infiltrates with small gas bubbles are found in the subcutaneous tissue, in the area of the head, neck and chest. Small hemorrhages are found on the serous and mucous membranes of the pleura, peritoneum, pharynx, trachea, and bronchi. Damage to the mucous membrane of the jejunum and duodenum

is characteristic - hemorrhagic inflammation and ulcers. The lungs are swollen, the liver is full of blood, and small yellowish-gray necrotic cells are found on its surface. The heart muscle is weak, there are hemorrhages on the epicardium and endocardium. The spleen is not changed.

Laboratory diagnostics. Includes microscopy of smears from pathological material, cultures on nutrient media and infection of laboratory animals. For research, pieces of parenchymal organs (if available - necrotic areas of the liver), tubular bone, exudate of the chest and abdominal cavities, swollen tissue, infiltrate of subcutaneous tissue, a part of the duodenum tied at both ends, and altered areas of the jejunum wall are sent to the laboratory for examination. Pathological material is collected only from fresh carcasses, no later than 4 hours after the death of the animal, since in the intestines of sheep after death, anaerobic microorganisms rapidly multiply and penetrate into organs and tissues.

Microscopic examination of pathological material reveals gram-positive bacilli Cl. septicum and Cl. oedematiens with terminally placed round spores.

In cultures from pathological material on the Kitt-Tarozzi medium, uniform turbidity and gas formation are observed, on Zeissler's glucose-blood agar - the formation of veil-like colonies with delicate processes and a zone of hemolysis. Intramuscularly infected guinea pigs die within 16-24 hours. Long filaments of the causative agent of the disease are detected in smears-imprints made from the surface of their livers, and resolution of the original Cl culture is carried out by inoculations on special nutrient media. In case of infection of guinea pigs with S. novji, a gelatinous gelatinous swelling is formed at the place of introduction of the culture.

The diagnosis of brazot is considered to be established when clostridia with properties characteristic of the causative agents of the disease are isolated from the pathological material, and the death of at least one guinea pig out of two, infected with the original pathological material or isolated cultures of clostridia, with a patho-anatomical picture typical of the causative agents, and excretion from its organs culture of the causative agent, the death of at least one guinea pig out of two infected with the original pathological material, if it has a patho-anatomical picture typical for this causative agent and isolation of the culture of the causative agent from it, if even in cultures from the original pathological material the culture of the causative agent is not isolated. The research period is up to 8 days.

Differential diagnosis. It provides for the exclusion of anthrax, infectious enterotoxemia of sheep, babesiosis, pasteurellosis, poisoning with herbs - aconite and ephedra. With anthrax, the spleen is greatly enlarged, softened, thick dark red liquid flows out of the cut. During the microscopic examination of blood smears, the causative agent of the disease is detected. Infectious enterotoxemia is characterized by softening of the kidney. There are no inflammations and ulcers of the mucous membrane of the rennet, liver damage, serous-hemorrhagic infiltrate in the subcutaneous tissue. The selection of the culture of the corresponding disease is

decisive. Piroplasmosis is ruled out by microscopy of peripheral blood smears. Pasteurellosis is accompanied by damage to internal organs and the upper respiratory tract. Pasteurella culture is isolated from pathological material in crops. If herb poisoning is suspected, pastures are examined for the presence of aconite and ephedra. In addition, with aconite poisoning, multiple hemorrhages under the serous membrane of the intestines, characteristic of the disease, are observed.

Treatment. Specific therapy has not been developed, and symptomatic treatment in the case of a hyperacute course of the disease is ineffective due to its rapidity. With an acute course, antibiotics (biomycin, synthomycin, terramycin) are recommended in doses for adult animals - 0.5-1.0 g, for lambs - 0.2 g per 1 kg of weight, as well as symptomatic therapy.

Immunity. Two vaccines have been proposed for active immunization of sheep - a bivalent vaccine against scrapie and infectious enterotoxemia of sheep and a polyvalent concentrated hydroxydaluminum vaccine against these two diseases, as well as malignant edema of sheep and dysentery of lambs. The entire flock of sheep is vaccinated starting from the age of 3 months. The vaccine is administered intramuscularly twice, with an interval between the first and second administrations of 20-30 days. Immunity in vaccinated animals occurs 10-12 days after the second vaccination and lasts 4-5 months. Polyvalent toxoid against sheep clostridiosis is also used.

Prevention and control measures. They consist primarily in observing zooveterinary rules during grazing, feeding, watering, keeping and caring for animals. It is necessary to constantly monitor the sanitary condition of pastures and watering holes, to prevent sheep from eating frozen, frost-covered fodder, and to prevent the use of water from shallow swampy and polluted water bodies. In farms that are unfavorable in terms of brazot, preventive vaccination of sheep should be carried out every year 30-45 days before driving them to pastures with a polyvalent concentrated hydroxydaluminum vaccine against brazot, infectious enterotoxemia, malignant oedema, and lamb dysentery.

When the disease occurs, the farm is declared unhealthy with respect to bradzot, and appropriate restrictions are introduced in it. In unhealthy farms, it is forbidden to introduce, remove and drive sheep, shearing and castration until the disease is stopped. Slaughter and consumption of meat from sick sheep, milking and use of milk from sick sheep, skinning of dead animals are not allowed. Sick and suspected sheep diseases are immediately isolated and treated. Healthy sheep are driven to other pastures or transferred to stables and vaccinated. Sheep carcasses are disposed of or burned together with the skin. Premises and other objects are disinfected twice with an interval of 1 hour with a 5% solution of hot formaldehyde or caustic soda, a clarified solution of chlorinated lime with a content of 3% active chlorine, as well as a 10% hot solution of a sulfur-carbolic mixture, a 10% solution of iodine chloride. Manure and feed residues are burned. Restrictions on the farm are removed 20 days after the last case of

death of animals from the disease of brazot, carrying out the final disinfection and all other measures provided for by the current instructions.

Control questions and tasks.

1. Causative agents of brazot and their characteristics.
2. Epizootological features of brazot.
3. Clinical signs of acute fever.
4. Characteristic features of patho-anatomical changes in brazoti.
5. When is the diagnosis of brazot considered established?
6. From what diseases should brazot be differentiated?
7. How do they treat healthy and sick animals when they become pregnant in a dysfunctional household?
8. What disinfectants are used to disinfect the premises during a fire?
9. Means of specific prevention of brazot.
10. Under what conditions is the economy declared prosperous in relation to brazot?

Topic: Infectious enterotoxemia of sheep

(diagnosis, prevention and control measures).

Infectious enterotoxemia of sheep (Enterotoxaemia infectiosa ovium) is a non-contagious toxic infection of sheep, which is characterized by hemorrhagic enteritis, specific kidney damage and nervous phenomena.

The disease is widespread on all continents of the world. The economic losses caused by the disease are due to significant damage (up to 30%) of the sheep population of unhealthy flocks and very high mortality (80-100%).

The causative agents of the disease are anaerobic microbes from the genus Clostridia - Cl. perfringens types D and C. 6 types of clostridia have been identified - A, B, C, D, E, F, which are similar to each other in terms of morphological and cultural-biochemical properties, but differ in their ability to cause disease in various species of animals and humans, as well as due to the excellent properties of the toxins produced by them.

Clostridia are immobile, thick, short gram-positive rods that form a capsule in the body of animals, and spores in the external environment. In the Kitt-Tarozzi nutrient medium with glucose, the growth of clostridia is accompanied by intense turbidity and gas formation. At first, delicate colonies in the form of dew drops are formed on the surface of the agar, which later acquire a grayish-white color, become round, juicy. Cultures of pathogens produce a strong exotoxin that has hemolytic, lethal, and necrotic properties. Spores of the pathogen are very resistant. They are stored in the ground for 16-20 months, in water - up to 20 months, in dry manure - 3 days, in wet manure - 5 days, on the surface of skin and wool - up to 2 years. Boiling

destroys spores in 15-20 minutes, hot (70-80°C) 10% caustic soda solution, 5-10% formaldehyde solution, chlorinated lime solution with 5% active chlorine - in 10-15 minutes.

The diagnosis is made by a complex method on the basis of epizootological data, clinical examination, patho-anatomical changes and the results of laboratory studies.

Epizootological data. Sheep of different breeds, ages and sexes are susceptible to enterotoxemia. Feline or lactating ewes and young sheep aged 8-10 months are more often affected. Goats, cattle, horses, pigs, camels, wild animals are also sick. Guinea pigs, white mice, and kittens are the most sensitive laboratory animals. The source of the causative agent of the disease is sick animals, as well as microbe-carrying sheep, which secrete clostridia with feces and pollute the environment, land, fodder, and water. Infection occurs through food, through feed and water contaminated with the pathogen. The disease occurs mainly during the grazing period, in the spring, in rainy weather, which is associated with the removal of spores to the surface of the soil and their contamination of intensively growing grasses, as well as the eating by animals of a significant number of juicy, protein-rich plants. Less often, the infection occurs when sheep are kept in stalls in unhealthy pens and pens, lack of exercise and feeding a large amount of protein feed. Disruption of the motor and secretory functions of the digestive tract due to sudden changes in feed during the transition from stable to pasture, eating frozen moldy feed contribute to the emergence and spread of the disease. A characteristic feature of enterotoxemia is stationarity, due to the presence of long-term microbial carriers and long-term persistence of the pathogen in the external environment.

Clinical signs and course of the disease. The incubation period lasts several hours. In the case of infectious enterotoxemia caused by Cl. perfringens type C, hemorrhagic phenomena prevail, especially in the intestines and parenchymal organs. In infectious enterotoxemia caused by Cl. perfringens type D (epsilon toxin), toxic phenomena prevail. The course of the disease is superacute (lightning), acute, subacute and chronic.

With a hyperacute course, animals die suddenly or within 2-3 hours. Clinical signs do not have time to appear, and usually in the morning one or more corpses of well-fed sheep are found in the barn or on the pasture. If it is possible to trace a sick animal, it turns out to be dejected, immobility of movements, the animal stands in one place with its head lowered and its forelimbs spread wide apart or vice versa, sick sheep are excited, constantly spinning in one place, stumbling, falling to the ground and lying with their head back; convulsions often occur. Shortness of breath, drooling, discharge of foamy liquid from the oral and nasal cavities are observed. Body temperature rises by 0.5-1°C or remains normal. Sometimes 30-40% of sheep in the flock die within the first day.

With an acute course, the body temperature in sheep rises to 41°C, sick animals refuse feed, stand for a long time in one place, grind their teeth, move with great

difficulty. Depression observed at the beginning of the disease turns into excitement, clonic muscle spasms occur, animals make involuntary movements, stumble, fall and cannot stand up, lie in a comatose state. The appetite is distorted, the mucous membranes are anemic with a yellowish tint, the pulse and breathing are accelerated. Before death, diarrhea appears, the feces are liquid with impurities of mucus and blood. Most sick animals die within 2-3 days, sometimes after 5-7 days.

The subacute course is observed mostly towards the end of the enzootic, passes independently or as a continuation of the hyperacute or acute course. In animals, digestive disorders are observed, feces are liquid, foul-smelling, with impurities of mucus and blood, urine is brown in color. Sick sheep lose weight quickly, their reflexes are weakened, there is no appetite, hair loss is observed. Feline uteri abort. The disease lasts 10-12 days, sometimes recovery occurs.

The chronic course is found in underfed sheep. They observe depression, refusal of food, drowsiness, nervousness, diarrhea, feces are initially mushy, and later liquid, watery, with mucus admixture. Sheep lose weight to the point of complete exhaustion, sometimes fall into a semi-comatose state and die within 20-30 days. Recovery is very rare.

Pathological anatomical changes. The corpses of sheep that died from entero-toxemia are bloated (Fig. 1), quickly decompose, dark purple spots appear on the hairless areas, and hemorrhages appear under the skin and in the muscles. In the case of death of an animal from enterotoxemia caused by clostridia type D, the accumulation of serous-hemorrhagic transudate is found in the abdominal and thoracic cavities, and hemorrhages are found on the epicardium and endocardium. The mucous membrane of the scar and duodenum is hyperemic, covered with hemorrhages (Fig. 2), the lungs are swollen, hyperemic, the liver is enlarged and reborn. Mesenteric lymph nodes are enlarged, hyperemic, juicy, with foci of necrosis.

Fig. 1. Swelling of a corpse

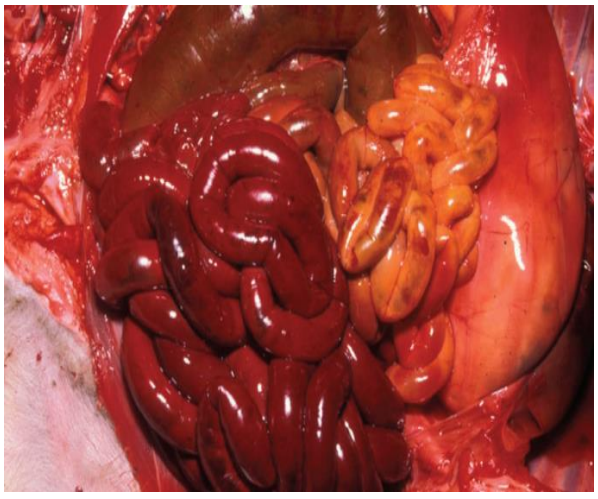


Fig. 2. Hyperemia and hemorrhages in small intestine

(https://tvmdl.tamu.edu/2021/04/19/enterotoxemia-in-sheep-and-goats)

Changes in the kidneys are specific - wateriness, the transformation of the pulp into a soft gelatinous or mushy mass (not always). When animals die from enterotoxemia caused by clostridia type C, hemorrhagic-necrotic inflammation of the mucous membrane of the rennet and small intestines, ulcers, enlargement and hyperemia of the mesenteric lymph nodes are revealed. The parenchyma of one, less often both kidneys is a bloody semi-liquid mass, from which the disease got the name "softened kidney".

Laboratory diagnostics. It is carried out in two directions - the detection of the toxin in the contents of the small intestine and its identification with the help of specific typical serums, as well as the isolation of the causative agent from the pathological material with the subsequent determination of its toxicity.

A whole fresh carcass or a piece of the small intestine with its contents, samples of pieces of the liver, spleen and kidneys, tied at both ends, are sent to the laboratory for examination. Pathological material must be taken no later than 4 hours after the death of the animal.

The diagnosis of infectious enterotoxemia of sheep is considered established when the toxin is detected in the filtrate of the contents of the small intestine by a biological method and its type is determined by a neutralization reaction with type-specific serums; isolation from the contents of the small intestine of a culture with properties characteristic of this pathogen, which produces a toxin, the type of which is established using a neutralization reaction with type-specific sera. The research period is 8 days.

Differential diagnosis. Presupposes the need to exclude bradzot, anthrax, pasteurellosis, piroplasmosis and fodder poisoning by herbs. In brazot, subcutaneous edema, hemorrhagic infiltration of the subcutaneous tissue, acute hemorrhagic inflammation and ulcers of the mucous membrane of the jejunum and duodenum, and necrotic cells in the liver are observed. There is never any softening of the kidney, which is characteristic of enterotoxemia. The results of bacteriological examination of pathological material are of decisive importance. Anthrax is ruled out on the basis of characteristic pathological changes and the results of a bacteriological examination. Pasteurellosis is accompanied by septicemia, hemorrhagic diathesis, lung damage, and is determined by the results of bacteriological studies. Piroplasmosis is ruled out on the basis of studies of peripheral blood smears. Intoxication caused by poisonous herbs is determined by laboratory studies of feed.

Treatment. In connection with the extremely rapid course of the disease, it is ineffective. At the beginning of the disease, bivalent antitoxic serum against anaerobic dysentery and infectious enterotoxemia, antibiotics of the tetracycline series, symptomatic therapy are used.

Immunity. Sheep infected with infectious enterotoxemia acquire type-specific antitoxic immunity lasting up to one year. For specific prevention, a polyvalent concentrated hydroxydaluminum vaccine is used against brazot, infectious

enterotoxemia, malignant oedema, and lamb dysentery. The vaccine is intended for preventive immunization of clinically healthy sheep and lambs from 3 months of age. Sheep are vaccinated intramuscularly twice with an interval of 20-30 days. From the age of 6 months, lambs are revaccinated, also twice. Immunity in animals occurs 10-12 days after the second vaccination and lasts 4-5 months. A toxoid vaccine and a polyvalent toxoid against sheep clostridiosis are also proposed.

Prevention and control measures. In order to prevent the occurrence of the disease in sheep farms, it is necessary to strictly follow the veterinary rules for feeding and keeping sheep, to eliminate all possible causes of digestive disorders in them. Care should be taken to use pastures with a rich grass stand, especially in the morning and after rain. It is necessary to constantly provide sheep with sufficient exercise and complete mineral nutrition, feed them with coarse fodder (straw, hay) and use trace elements (copper and cobalt salts).

Precautionary vaccination of sheep in previously unfavorable farms should be carried out before the start of the grazing season, before the sheep are turned out on the grass and 1-1.5 months before a possible outbreak of infection. When the disease occurs, the farm is declared unhealthy for infectious enterotoxemia of sheep. It prohibits slaughtering and using the meat of sick animals for human consumption, milking sheep and consuming milk for food, grazing on the same pastures of healthy and sick animals, shearing, castration, tail trimming. Sick and suspected sheep are isolated and treated with hyperimmune serum and antibiotics. The corpses are destroyed together with the skin. Healthy animals are transferred to stable housing and vaccinated.

Restrictions on the farm are lifted 20 days after the last case of death of sheep from enterotoxemia and the final disinfection of the premises and the surrounding area. For disinfection, use a 10% solution of caustic soda or a solution of chlorinated lime with a content of 5% active chlorine.

Control questions and tasks.

1. Pathogens of infectious enterotoxemia and their characteristics.
2. Epizootological data of infectious enterotoxemia.
3. Features of the clinical manifestation of the disease depending on the type of Cl. perfringens and their signs.
4. Specific patho-anatomical changes in infectious enterotoxemia.
5. For what purpose are two directions of laboratory research conducted in infectious enterotoxemia?
6. Differential diagnosis of infectious enterotoxemia.
7. Means of specific prevention for infectious enterotoxemia.
8. Prevention and control measures.

Topic: Infectious agalactia of sheep and goats

(diagnosis, prevention and control measures).

Infectious agalactia of sheep and goats (Agalactia ovium et caprarum) is a contagious disease characterized by damage to the udder, joints, eyes and cessation of milk secretion.

According to the International Epizootic Bureau, infectious agalactia of sheep and goats is registered in Syria, India, Sudan, Ecuador, Iran, Israel and Mongolia, as well as in such European countries as Italy, Portugal, France, Spain, Greece. Economic losses consist of the cost of dead and forcibly slaughtered animals, reduced milk productivity, losses from abortions, costs of quarantine and health measures.

The causative agent of the disease. Mycoplasma agalactiae from the Mycoplasmataceae family is a polymorphic immobile gram-negative microorganism visible under a light microscope that passes through bacterial filters. Facultative aerobic, stained according to Romanovsky-Giemsa.

During the acute course of the disease, it is found in the blood, parenchymal organs, all secretions and excreta. Cultivated in Edward's special medium or bovine heart tryptic digest, as well as on Martin's and serum agar and broth. During growth in the broth, opalescence or slight turbidity of the medium is observed without the formation of sediment. On dense nutrient media, during 2-3 days of incubation, it forms characteristic delicate round small colonies with a granular surface and a center embedded in the agar. Forms a zone of hemolysis on blood agar. Small laboratory animals are not susceptible to the causative agent of agalactia, except for rabbits. The causative agent is resistant to various physical and chemical factors. It is stored in the external environment at 0-25°C for up to 4 months, in soil – 25 days, in water – 30 days, in sealed tubes under petroleum jelly at 37°C – up to 2 months, in milk at room temperature – up to 8 days . When heated to 60°C, it is inactivated after 5 minutes, when boiled - instantly. Under the influence of 2% caustic soda, it is destroyed after 1 hour, 2% formalin - 2-4 hours, 2-3% creolin emulsion, lysol - 4 hours, 20% freshly slaked lime - 1 hour.

The diagnosis is made by a complex method directly in the farm on the basis of epizootological data, analysis of the clinical condition of sick animals and patho-anatomical changes. If necessary, laboratory tests are carried out.

Epizootological data. In natural conditions, sheep and goats are susceptible to the disease regardless of breed, sex, and age. Lactating ewes, newborn lambs and kids, as well as young animals up to 1 month of age are more often affected. The source of the causative agent of infection is sick and sick animals, which for 5-7 months secrete mycoplasmas with milk, urine, feces, conjunctival secretions, and during abortion - with secretions from the birth canal, fetus and fetal membranes. Mycoplasma-

contaminated fodder, water, bedding, pastures, corrals, clothes and shoes of milkmaids and shepherds can be factors of pathogen transmission.

Infection of healthy animals occurs through alimentary means, when grazing on lowland pastures and flooded meadows, as well as during watering from stagnant water bodies. The possibility of intrauterine infection has been proven. Lambs and kids get infected from sick mothers who secrete mycoplasmas with milk. In unhealthy herds, infectious agalactia occurs in the form of enzootic outbreaks, which cover 15-50% of animals. Outbreaks of the disease occur in the spring, reach a maximum in the summer and disappear in early autumn. This regularity of the development of enzootic disease is due to the intensive release of the pathogen by infected ewes during birth and lactation, the appearance of a large number of particularly susceptible contingent of newborn lambs and kids, the accumulation of the pathogen in places of long-term housing, grazing and abortions, as well as the negative effect of cold, rainy weather on the resistance of the young. The morbidity of animals during an outbreak of infection is 16-37%, the mortality rate is 15-45%. Pregnant females abort in 15-30% of cases. With the cessation of calving and the herds moving to winter pastures, the enzootic disappears. The stationarity of infectious agalactia is ensured by latent infection of long-term carriers and long-term preservation of the pathogen in the external environment for up to 4 months.

Clinical signs and course of the disease. The incubation period lasts from 2-6 days to 2 months. The course of the disease is acute, subacute, chronic and atypical.

In the case of an acute course, depending on the localization of the pathological process, septic, mastitis, joint, eye and mixed forms of the disease are distinguished.

The septic form is manifested in newborn kids and lambs with a sharp decrease in the body's resistance caused by insufficient milk in sick mothers, as well as in lambs under 2 months of age in very cold, rainy weather and lack of feed. A sudden rise in body temperature up to 42°C, severe depression, complete refusal of food, formation of edema in the subcutaneous tissue is observed. Sick animals die at the end of 2-4 days.

The mastitis form of the disease occurs in lactating sheep and goats and is characterized by fever (41.5-42°C), depression, refusal of feed, and a sharp decrease in milk yield. The udder is affected, more often one of the lobes of the udder, which becomes hot, painful to the touch. Swollen lymph nodes significantly increase in size, milk becomes thick, later watery, acquires a bitter-salty taste. Atrophy and induration of the mammary gland develops, milk secretion gradually stops (Fig. 1).

Joint and ocular forms of the disease predominate in young animals and non-lactating mothers. Fever, depression, decreased appetite are observed. The wrist and hock joints are often affected, less often the elbow and hip joints. Lameness, swelling and pain in the joints, fluctuation in the areas of the joint bags are revealed. In case of eye disease, lacrimation, hyperemia, photophobia, swelling of the eyelids are observed.

Sometimes conjunctivitis is complicated by keratitis, formation of ulcers on the cornea, loss of the lens. The disease lasts 3-5 days and often ends fatally.

Fig. 1. The mastitis form of the disease
(https://www.sciencedirect.com/science/article/abs/pii/S0034528808001501)

With a mixed form of the disease, lactating mothers have lameness, swelling, joint pain, and sometimes conjunctivitis along with udder damage. The disease lasts 5-7 days and often ends fatally.

The atypical form of the disease is characterized by a short-term fever, a slight decrease in milk production, and slightly painful joints. Recovery is possible. Sick animals remain carriers of bacteria for a long time.

The subacute course is characterized by various complications of the main clinical signs, which are caused by the layering of secondary microflora. In female cats and kittens, complications with purulent microflora lead to the development of purulent mastitis, which ends in atrophy of the mammary gland, purulent arthritis that causes partial or complete ankylosis, panophthalmitis, and the formation of deep ulcers on the cornea.

The chronic course of the disease lasts several weeks and even months. Symptoms of the disease are very weak. Ankylosis of individual joints, cataracts, and sometimes abortions are observed.

Pathological anatomical changes. They depend on the nature of the course and the form of the disease. In acute and subacute courses, swellings in the subcutaneous tissue, abscesses under the skin and in the muscles, serous inflammation of the peritoneum and pericardium, and numerous hemorrhages under the epicardium are revealed. Inflammatory changes are also observed in lymph nodes and kidneys. Kidneys have a bumpy surface with necrosis. In the case of a chronic course, pathological changes are local in nature. Chronic inflammatory processes in the joints

and udder are observed. The udder parenchyma is infiltrated, the cistern and milk ducts contain cheesy clots of white or greenish color. When the eyes are affected, conjunctivitis, keratitis, cataracts and other pathological changes are detected. The affected cornea is thickened, has a cone-convex shape. The walls and cartilages of the joints, as well as tendon sheaths are significantly thickened and inflamed. A brownish-red liquid, sometimes pus, is found in the joint bags.

Laboratory diagnostics. Includes microscopic examination of swabs from pathological material, cultures on nutrient media for the isolation of the pathogen and bioassay. Pathological material is sent to the laboratory only fresh, in a thermos with ice, or in frozen form. For intravital diagnostics, samples of blood, milk from the affected parts of the udder, and synovial fluid from the joints are sent to the laboratory from clinically ill animals. Parenchymal organs are sent from dead or forcibly slaughtered animals (kidney, part of liver, spleen, affected part of udder with enlarged lymph nodes, affected eye, synovial fluid from affected joints, cerebrospinal fluid, brain, aborted fetuses). During the microscopic examination of smears-imprints stained according to Romanovsky-Giemsa, with positive results, small polymorphic (cocoon-, thread- or ring-shaped) microorganisms of pink color are revealed. On liquid elective media, the growth of mycoplasma after 5-7 days of incubation at 37-38°C is detected by the characteristic opalescence without the formation of sediment. Small colonies of dewdrops appear on the agar with a light periphery and a dark center embedded in the agar. The bioassay is carried out on rabbits weighing 2.5-3 kg, which are infected in the anterior chamber of the eye with a 10% suspension of pathological material or a 3-4 day culture of the pathogen. With positive results, experimental rabbits develop keratitis 5-12 days after infection.

A laboratory diagnosis of infectious agalactia of sheep and goats is considered to be established after receiving one of the following indicators: separation from the pathological material of a culture with properties characteristic of the causative agent of this disease; a positive bioassay on rabbits, if the pathogen is not isolated even in cultures from the source material; a positive bioassay on sheep and goats followed by the isolation of a culture with properties characteristic of this pathogen, if the pathogen is not isolated even from the source material of the culture. The term of research without a bio sample is 30 days, with a bio sample and the subsequent isolation of the pathogen - 90 days. Diffuse precipitation reaction and complement binding reaction are proposed for serological diagnosis of the disease.

Differential diagnosis. Presupposes the need to rule out infectious sheep mastitis and infectious keratoconjunctivitis. In case of infectious mastitis of sheep, damage to the joints and the cornea of the eye does not occur, the pathological process in the udder is accompanied by gangrenous decay of tissues. The causative agent of the disease is staphylococcus, which is well cultivated on general nutrient media in aerobic and anaerobic conditions. The course of infectious keratoconjunctivitis in sheep is always

benign, the causative agent of the disease - rickettsia - is detected in the pathological material.

Treatment. Specific drugs for the treatment of infectious agalactia in sheep and goats have not been developed. Intravenous administration of a 5% aqueous solution of novarsenol-benzene at the rate of 0.1 g per 1 kg of weight is recommended. The average dose for adult animals weighing 30-40 kg is 0.3-0.5 g, for lambs under the age of 5 months - 0.1-0.15 g, over the age of 5 months - 0.2-0.5 g dry drug After 5-6 days, a 5% novarsenol solution is re-introduced to adult animals - 0.3-0.4 g each, to young animals under the age of 5 months - 0.05-0.1 g, to lambs older than 5 months - 0.14-0.2 g. On the second day after the first use of novarsenol, all animals were injected with a 10% solution of urotropin in sterile saline. Urotropin is injected subcutaneously three times a day, 4 ml with an interval of 4-5 hours for 6-8 days. Sick animals with eye damage are placed in darkened rooms, the eyes are washed with a 1% aqueous solution of boric acid. In case of arthritis in the area of the affected joint, inject Lugol's solution in a dose of 1 ml or 1% solution of copper sulfate in a dose of 2-3 ml for adult animals and 1.0-1.5 ml for lambs and kids. Lactating sick mothers are milked 2-3 times a day. Penicillin solution is injected three times a day into the udder cavity through the teat canal. Intramuscular use of streptomycin, dibiomycin, ditetracycline gives an effective result.

Immunity. Not enough studied. It has been established that goats can get sick again. Hydroxidaluminum formolvaccine is proposed for specific prevention.

Prevention and control measures. First of all, they consist in the implementation of zoo-veterinary rules for stocking farms with small cattle, keeping, grazing and feeding animals. It is impossible to allow contact between animals of different farms and herds during races to summer pastures.

When a disease appears, the household or farm is declared unhealthy, and quarantine restrictions are introduced. All sick and suspected animal diseases, including those that aborted or gave birth to unviable lambs, are isolated in a separate flock and treated. Severely ill animals and animals with a chronic course of infection are culled. Suckling lambs and kids are separated and fed with milk from healthy mothers. Healthy livestock are transferred to new pastures and watering holes, and permanent veterinary supervision is established for them. Vaccination of susceptible animals with live attenuated or inactivated vaccines against agalactia of sheep and goats is carried out in disadvantaged farms and in the threatened zone. Sick animals are kept in isolation for 8 months after recovery, it is advisable to cull them for slaughter for meat. Sick breeders are not allowed to mate and are not used for artificial insemination. Recovered queens are artificially inseminated with the sperm of healthy breeders. Premises and shelters where sick animals were kept are cleaned and disinfected with a 2% solution of freshly slaked lime, a 2% solution of lysol or a 2% solution of caustic soda. Litter, fodder, manure contaminated with secretions of sick animals are burned. Restrictions from the farm are removed 60 days after the removal

or slaughter of the last sick animal. The removal of animals to safe farms for breeding purposes is allowed only after 8 months, provided that during this period no infectious agalactia disease was observed in the farm.

Control questions and tasks.

1. The causative agent of infectious agalactia and its characteristics
2. Epizootological data of infectious agalactia of sheep and goats.
3. Forms of clinical manifestation of the disease and their clinical signs.
4. Pathological-anatomical changes in infectious agalactia.
5. Diagnosis of infectious agalactia of sheep and goats.
6. How to treat sick animals with infectious agalactia?
7. Prevention and control measures.

Topic: Infectious catarrhal fever of sheep

(diagnosis, prevention, control measures).

Infectious catarrhal fever of sheep (Febris catarrhalis infectiosa ovium, blue tongue) is a viral transmissible disease characterized by fever, inflammatory-necrotic lesions of the mucous membranes of the alimentary canal and respiratory tract, as well as degenerative changes in skeletal muscles. In connection with the characteristic lesion of the tongue, the disease received the name "blue tongue" or "black mouth".

Infectious bluetongue fever is classified as a disease of group A, the international epizootic bureau is informed about its occurrence and restrictions are imposed on interstate trade in meat, wool and livestock.

The causative agent of the disease. RNA-genomic virus belonging to the family Reoviridae, genus Orbivirus. The virus is contained in blood, serum and blood plasma, in hematopoietic organs, during fever - in the spleen, liver, and lymph nodes. During the period of pronounced clinical signs, the virus is localized in the mucous membrane of the intestines, epithelial and muscle tissues.

Viremia in sheep continues for 35-49 days after recovery, despite the presence of virus-neutralizing antibodies in the blood. The virus is cultivated on chicken embryos 6-8 days old or 11-13 days old, in the body of newborn white mice, in primary cultures of kidney cells of lambs or cow embryos. Among laboratory animals, 1-4-day-old white mice, as well as hamsters, are susceptible to the virus. The virus is quite stable in the external environment. Resistant to rotting and low temperatures, it is stored for a long time in frozen sperm at -70°C. Heating to 60°C destroys the virus after 5 minutes. Under the influence of a 3% solution of formalin, the virus dies after 48-72 hours, after 5 minutes with a 3% solution of caustic soda or 70° ethyl alcohol.

The diagnosis is made by a complex method on the basis of epizootological data, clinical signs of the disease, patho-anatomical changes and the results of laboratory tests.

Epizootological data. In natural conditions, the causative agent of infectious catarrhal fever is susceptible to sheep and to a lesser extent to cattle, goats, antelopes, elk, American white-tailed deer, and snow sheep. Weaned lambs are especially sensitive. In stationary unsatisfied points, suckling lambs acquire passive immunity and do not get sick. The source of the causative agent of infection are sick sheep, which become virus carriers for 3-4 months. The transmission of the virus from sick animals to healthy ones is carried out by Culicoides lice, which are characterized by a significant distribution and large number, as well as Aedes mosquitoes and Melophagus ovinus mosquitoes. The most significant reservoir of the virus in nature is considered to be cattle, in whose body the virus is stored until the next active period of the development cycle of stinging insects. Wild ruminants and rodents can also be an intermediate link through which the virus can be transmitted to susceptible sheep. The pathogen is not transmitted by contact.

Infectious catarrhal fever of sheep is characterized by seasonality, which coincides with the period of rains and the greatest activity of vector insects. Epizootics of the disease are observed in low-lying areas, downstream of slow rivers, along the banks of stagnant bodies of water. Animals become infected when grazing them at night or early in the morning. In dry years, dry season, hot hours of the day, when sheep are kept indoors, as well as in mountainous areas, the disease is not observed. Infectious catarrhal fever occurs in the form of significant epizootics, with a large coverage of livestock (50-60%), characterized by a severe course and high mortality, which reaches 90% in fresh outbreaks, 30% in stationary ones.

Clinical signs and course of the disease. The incubation period lasts for 2-6 days. The course of the disease is acute and subacute. An abortive form of the disease is also defined.

With an acute course, a sudden rise in body temperature to 40.6-42°C is observed, which is maintained at this level for 6-12 days. At the same time, they note oppression, shaky uncertain gait, sick sheep lag behind the herd. 24-36 hours after the rise in body temperature, conjunctivitis develops, catarrhal inflammation of the mucous membranes of the mouth, tongue, pharynx, and nasal cavity (Fig. 1).

Fig. 1. Clinical symptoms of infectious catarrhal fever.
(https://www.intechopen.com/chapters/64011)

Swelling of the head is detected, the tongue increases in volume, acquires a red-blue color, hence the name of the disease - "blue tongue". However, the blueness of the tongue may not be present, or it may disappear after 1-2 days. At the same time, foamy saliva ("wet muzzle") flows from the oral cavity, which is caused by a kind of incessant movements of the tongue, as well as discharge from the nose, which is initially watery, and then mucous-catarrhal and purulent. Ulcerative or gangrenous stomatitis develops, diarrhea with blood impurities appears.

On the hind limbs, redness and swelling of the corolla, soft parts of the legs, pain when pressed. In many animals, lameness, "tied" gait, exanthematous lesions and hair loss appear on the skin. Animals lose weight and die of the disease in 7-8 days. Lethality can reach 70-90%. Recovery is very slow, no earlier than 10-15 days after the body temperature drops. Lambs develop pneumonia, complications of necrobacteriosis, which in almost 100% of cases end in death.

During the subacute course, the same clinical signs are observed as in the acute course, but their severity is much less pronounced. The duration of the disease is 12-25 days, the mortality rate is 20-60%.

The abortive form of the disease is manifested only by short-term fever, lethargy, superficial catarrhal inflammation of the mucous membranes of the oral and nasal cavities, conjunctivitis. Recovery occurs relatively quickly. At the same time, anorexia, salivation, and hyperemia of the mucous membranes of the oral and nasal cavities are revealed. Ulcers and hemorrhages are found on the nose mirror, lips, gums, udder, and vulva. The tongue swells greatly, protrudes from the mouth, which makes swallowing very difficult, and cattle die of thirst. Sometimes abortions and the birth of non-viable offspring are observed. In endemic areas, the course of this disease in cattle is often asymptomatic, with long-term virus-carrying.

Pathological anatomical changes. The corpses are emaciated, eczematous lesions are observed on the skin. The subcutaneous tissue and intermuscular connective

tissue in the area of the head, neck, and back are swollen and infiltrated with a yellowish liquid. Edemas are also found in the intermaxillary space, pharynx, larynx, tongue and lips. Ulcers and hemorrhages are found on the tongue, lips, and inner surface of the cheeks. The spleen is significantly increased in volume. Lymph nodes, especially pharyngeal, submandibular, cervical and mesenteric, enlarged, hyperemic, swollen. Accumulation of gelatinous exudate with blood impurities in the chest and abdominal cavities. Numerous hemorrhages are found on the surface of the lungs, mucous membranes of the alimentary canal and respiratory tract, under the capsule of the liver and spleen, under the epicardium and endocardium.

Laboratory diagnostics. It involves the isolation of the virus from pathological material, its identification using the neutralization reaction (on chicken embryos and cell culture) by the immunodiffusion reaction, the complement binding reaction, the diffuse precipitation reaction, the study of paired sheep blood sera by the complement binding reaction, the hemagglutination delay reaction, the neutralization reaction and reaction of diffuse precipitation in order to detect specific antibodies. In doubtful cases, a bioassay is carried out on lambs.

Blood samples are sent to the laboratory for intravital research, which are taken during the period of increased body temperature from sick sheep, and for postmortem diagnosis, lymph nodes, pieces of spleen and liver, and aborted fetuses are taken from corpses or dead animals no later than 2 hours after death. To isolate the virus, infection with pathological material is carried out in the yolk sac of 6-8 day old chicken embryos. Identification of the isolated virus is carried out using a neutralization reaction in cell culture or chicken embryos with undiluted specific serum, as well as using an immunofluorescence reaction, a complement binding reaction, and a diffuse precipitation reaction. The bioassay is performed on 1-4-day-old white mice, which are infected intracerebrally, as well as on 3-6-month-old lambs, which are injected with the pathological material intravenously, intradermally, or subcutaneously. Characteristic clinical signs of the disease appear after 6-8 days and are manifested by an increase in body temperature, hyperemia of the mucous membranes of the mouth and nose, swelling of the lips and tongue. Serological and retrospective diagnosis of infectious catarrhal fever of sheep is carried out using the complement binding reaction, diffuse precipitation reaction, hemagglutination delay reaction, neutralization reaction, and, if necessary, also the immunofluorescence reaction and the ELISA method. Most often, the sera of convalescents are examined by the complement binding reaction, which can detect group-specific antibodies against all serotypes of the causative agent of the disease. Complement-binding antibodies are detected in the blood 2 weeks after infection in titers of 1:4-1:16 and reach maximum values (1:60-1:256) on the 30th day.

Differential diagnosis. Presupposes the need to rule out foot-and-mouth disease, contagious ecthyma of sheep and goats, vesicular stomatitis, smallpox, necrobacteriosis, Nairobi disease, Rift Valley fever.

Foot-and-mouth disease, contagious ecthyma of sheep and goats, sheep pox are characterized by high contagiousness, their appearance is not related to the season and mass flight of blood-sucking insects. Foot-and-mouth disease is characterized by aphthous lesions of the oral cavity, udder, and extremities; important results of virological studies. In case of contagious ecthyma, specific pustular lesions of the mucous membranes of the oral cavity and the skin in the area of the scrotum, corolla, and intercostal space are revealed. During microscopic studies of unfixed smears and dry, unpreserved scabs, the causative agent of the disease is revealed. Foot-and-mouth disease and vesicular stomatitis are differentiated on the basis of the results of RCT with antigen from the walls of the vesicles of sick animals. A bioassay is conducted on rabbits and lambs. Smallpox is determined on the basis of microscopic studies of Morozov-stained smears from the inner surface of fresh papules and the detection of single and clustered elementary bodies, stained dark brown, against the yellow background of the preparation. In case of necrobacteriosis, the limbs are mainly affected, the causative agent of the disease, Fusobacterium necrophorum, is detected in the pathological material. Nairobi disease is accompanied by hemorrhagic diathesis and severe gastroenteritis. Rift Valley fever is characterized by liver dystrophy and focal necrosis. The final diagnosis is made based on the results of laboratory tests.

Treatment. Not developed.

Immunity. Sick animals acquire immunity only against the type of virus that caused the disease. Reinfection with another type of virus during the same season or the following year is possible. Active immunity in convalescents is accompanied by the formation of neutralizing and complement-binding antibodies, which are passively transmitted to young animals with colostrum. In disadvantaged countries, live and inactivated vaccines are used for the specific prevention of infectious catarrhal fever in sheep. Immunity and specific antibodies in high titers persist after vaccination for at least one year.

Prevention and control measures. Infectious catarrhal fever is not registered in our country. Therefore, the main attention should be directed to preventing its introduction with imported domestic (sheep, goats, cattle) and wild ruminants.

Preventive quarantine of all imported animals with virological and serological tests, if necessary, is mandatory. In disadvantaged countries, measures to prevent and eliminate this disease are carried out in accordance with their veterinary legislation.

Control questions and tasks.

1. The causative agent of infectious catarrhal fever and its characteristics
2. Epizootological features of infectious catarrhal fever.
3. Clinical signs of an acute course of infectious catarrhal fever.
4. Abortive form of the disease and its clinical signs.
5. Pathological-anatomical changes.
6. Diagnosis of infectious catarrhal fever.

7. What diseases must be differentiated from infectious catarrhal fever of sheep?
8. Prevention and control measures.

Topic: Hoof rot

(diagnosis, prevention and control measures).

Hoof rot (Paronychia contagiosa) is a chronic contagious disease of sheep and goats, which is characterized by lameness, inflammation of the skin of the interspinous cleft, followed by putrefactive decay of the base of the skin, horn tissue, and peeling of the horn shoe of the hooves.

Hoof rot is registered in all countries of the world with developed sheep farming. In Ukraine, the disease occurs in the southern regions in the case of keeping sheep in raw, contaminated pens. Economic losses are determined by the forced slaughter and death of sick animals, a decrease in their productivity, significant costs for carrying out medical and health-improving measures.

The causative agent of the disease. Bacteroides nodosus - belongs to the family Bacteroidaceae, genus Bacteroides. It is a straight or slightly bent, large, mobile gram-negative anaerobic bacillus with thickenings at the ends. It does not form spores or capsules. 11 serotypes of this microbe are known, which cause disease in sheep, but are not pathogenic for laboratory animals. In smears from pathological material, clusters of small gram-negative rods are found, which are located perpendicular to bacterial cells like a picket fence, the so-called "Beveridge phenomenon".Bacteroids are cultivated only on special nutrient media - Baktemirov, Masalek, and Klaubs media. On dense media, it forms large flat colonies with a depressed center and uneven, fringed edges or small colonies with a cone-like raised center and smooth edges. In liver-brain broth, it causes gradual clouding with subsequent enlightenment. In the affected tissues, the causative agent of the disease remains viable for years, it persists in the pasture from several hours to 15 days. Common disinfectants - 2-3% formaldehyde solution, 2% phenol solution, 3% caustic soda solution, 3% creolin solution, chlorinated lime solutions containing 3-5% active chlorine inactivate the pathogen within 10-15 minutes, heating at 90 °C - after 1-2 minutes.

The diagnosis is made by a complex method on the basis of epizootological data, clinical signs of the disease, patho-anatomical changes, as well as the results of laboratory tests.

Epizootological data. Sheep and goats are sick regardless of age and breed, but lambs under the uterus are not sensitive to the pathogen. The disease is more common in areas with lowland wet pastures and high rainfall. In dysfunctional farms, an increase in morbidity is observed in rainy, cold periods of the year (autumn, early spring). The source of the causative agent of the infection is sick animals and micro-carriers that secrete the causative agent with purulent-necrotic secretions from the affected hooves.

Infection of healthy animals occurs through direct contact with sick animals, as well as through litter, manure, soil, pastures contaminated with purulent-necrotic secretions from affected hooves contaminated with the causative agent of the disease. The disease is highly contagious, morbidity in unhealthy flocks reaches 80%, mortality - 5-12%.

Clinical signs and course of the disease. The incubation period lasts 3-6 days. The course of the disease is chronic. There are initial, mild and severe forms of the disease. The main clinical sign of the initial form of the disease is purulent inflammation of the skin of the interspinous cleft, the presence of surface erosions and mucus with the characteristic unpleasant smell of rotten hoof horn. With a mild form of the disease, peeling of the horn of the inner side walls of the hooves occurs in the area of the heels, sometimes part of the sole. In the severe form, there is significant putrefactive decay of the base of the skin, complete detachment from it of the inner side walls of the hoof and the sole on one or more limbs in the absence of abscesses and ulcers in the area of the corolla. A gray-yellow exudate with an unpleasant rotten smell is found under the exfoliated horn. A characteristic clinical sign of hoof rot is great soreness of the hooves and associated lameness. They note the progressive exhaustion of the animal, hair loss. Sick sheep do not come into the flock or give birth to weak, non-viable lambs. Hoof rot is often accompanied by necrobacteriosis, manifested by an increase in body temperature to 40-40.5°C, the formation of abscesses, ulcers and fistulas in the areas of the corolla, foot, and fistulas, damage to joints, ligaments, tendons, as well as necrosis of the mucous membrane of the oral cavity, lips, front part of the head, udder (Fig. 1).

Fig. 1. Hoof rot in a sheep.
(https://www.raisingsheep.net/foot-rot)

Pathological anatomical changes. The base of the skin of the sole, lateral inner walls is in a state of purulent-necrotic decay, the corneous wall is deformed, the corneous layer of the sole is exfoliated almost to the point of separation of the horn shoe of the hooves.

Laboratory diagnostics. Includes microscopic and immunofluorescent examination of pathological material, and, if necessary, a biological test on lambs. In the initial stage of the disease, the purulent exudate covering the interradicular gap, and later the exudate from the depth of the pocket, which was formed as a result of exfoliation of the horn, as well as pieces of tissue taken from freshly affected areas of the base, are sent to the laboratory for examination no later than 24 hours after the time of collection hoof skin, or a hoof from a slaughtered sick animal. A laboratory diagnosis of hoof rot is considered to be established in the case of a positive bioassay on lambs, detection of characteristic rods of the causative agent in Gram-stained smears, positive results of fluorescent microscopy.

Differential diagnosis. Necrobacteriosis, smallpox, foot-and-mouth disease, contagious ecthyma, various hoof diseases of non-infectious etiology must be distinguished from hoof rot.

In case of necrobacteriosis of the limbs, the pathological process is localized mainly on the corolla, it is manifested by damage to the joints, ligaments, tendons, the formation of ulcers, abscesses and fistulas. Lambs in the first days of life, in which there is necrosis of the mucous membrane of the oral cavity, lips, and frontal part of the head, are severely and always fatally ill. An autopsy revealed necrotic cells in the parenchymal organs, on the mucous membrane of the intestines. It is necessary to remember the possibility of a mixed infection of necrobacteriosis and hoof rot.

The course of smallpox is acute, with high body temperature. Characteristic smallpox rashes on the head, lips, walls of the nose, inner surface of the limbs, udder, intercostal space, as well as damage to internal organs are revealed. Smallpox virus is detected during microscopic and virological examination. Foot-and-mouth disease occurs acutely, in the form of epizootics with simultaneous disease of other animal species. Characteristic aphthae and erosions are found on the skin of the inguinal gap and the edges of the corolla. Lambs have diarrhea, high mortality. Virological and serological studies determine the FMD virus. Contagious ecthyma is accompanied by almost 100% damage to livestock, including young animals under one year of age. In animals, together with lameness, damage to the mucous membrane of the mouth, lips, as well as in the area of the nose, ears, eyelids, and genitals is detected. Papules, vesicles, pustules, and crusts are found on the skin of the corolla, interradicular gap. Virological and microscopic examination and bioassay allow reliable differentiation of contagious ecthyma and hoof rot of sheep.

Treatment. Conducted by group method or individually. For group treatment, foot baths with 5-10% formalin solution for 1.5-2 minutes once every 7 days, 5% paraform solution for 2 minutes every 2-3 days for 2 weeks, copper sulfate solution (5 -30% copper sulfate) for 1-2 minutes once every 7 days, 10-20% solutions of zinc sulfate for 1-2 minutes repeatedly. Before the bath, the hooves should be thoroughly washed, the exfoliated horn should be cut off, and the affected tissues should be

removed. After the bath, the animals are kept on a concrete platform for 1-2 hours, and then they are transferred to a dry pen with fresh bedding or to a safe pasture.

For individual treatment, 5-10% alcohol solutions of antibiotics (levomycetin, chlormycetin, terramycin, penicillin) are used (after a cleaning and thorough surgical treatment of lesions) in the form of irrigations or dressings for 3-5 days, 10-15% emulsion (on fish oil) penicillin, teramycin, tricillin, dibiomycin, neotetramycin in the form of ointments, better with the use of bandages, aerosols of various medicinal products and antibiotics (chloramphenicol, oxytetracycline, teramycin). Drugs based on chloramphenicol are especially effective. Hoofs can be treated with an aqueous emulsion of penicillin in fish oil. In the case of light lesions of the hoof, sheep recover in 3-10 days after 2-3 treatments, in severe cases - in 15-20 days after 4-5 treatments. When the disease is complicated, long-acting antibiotics are used - bicillin-5 (40-50 thousand IU/kg intramuscularly once), dibiomycin (30-50 thousand IU/kg subcutaneously once in the form of a 10% emulsion on 30% sterile glycerin).

Immunity. Not enough studied. Sheep can get sick again after recovery.

Prevention and control measures. In order to prevent the occurrence of hoof rot, it is necessary to import sheep for stocking the flock only from prosperous farms. During the 30-day preventive quarantine, a thorough inspection and cleaning of the hooves, trimming of an overgrown horn is carried out. Before transferring to the main herd, the imported livestock is passed through a disinfection bath with a 5% formalin solution, a 10% copper sulfate solution, and a 5% paraform solution. At least twice a year, cleaning, trimming of hooves, a thorough clinical examination and preventive disinfection are carried out.

In case of detection of hoof rot disease in sheep or goats, the herd is declared unhealthy, and quarantine restrictions are introduced in the farm. Removal of sheep for breeding and economic purposes, their regrouping is prohibited. A thorough clinical examination of the entire herd is carried out, sick animals are isolated in a separate group and treated. After cleaning the hooves, the rest of the animals of the unhealthy herd are passed through a disinfection bath with a 10% solution of formalin or copper sulfate, a 5% solution of paraform at a temperature of 25-35°C, kept on clean, dry litter for 1.5-2 hours, then transferred to a new pasture with equipped with approaches to the watering hole. A thorough inspection of the hooves of conditionally healthy livestock, regular trimming, and preventive disinfection are carried out every day. The corpses of dead animals are burned after skinning. Skins and wool of slaughtered or dead sheep and goats are dried on the farm in an isolated room. Export of skins is allowed only in dried form, and wool - in a container made of thick fabric no earlier than 2 weeks after their removal or shearing. Milk from relatively healthy sheep and goats is allowed to be consumed after boiling, milk obtained from sick animals is destroyed. Dry pastures can be used without restrictions 15 days after sick animals have grazed them. Corrals, walking yards, pens, where sick animals were kept, are cleaned of manure and disinfected. If within a month after the isolation and slaughter of all sick sheep in a

conditionally healthy group, no animals with signs of hoof rot appear, then after carrying out veterinary and sanitary measures, the flock is considered healthy. The farm is considered safe with regard to hoof rot after 1 month after the last case of recovery or slaughter of sick sheep and goats and the final disinfection. For disinfection with exposure for 1 hour, use 2% formaldehyde solution, 2% hot caustic soda solution, 5% disinfectant (phenolic) creolin emulsion, 5% paraform solution, clarified chlorinated lime solution containing 5% active chlorine, 20% slaked lime suspension . A thin layer of slaked lime (pushonki) is sprinkled on the floor of the koshar and walking yards every 3 days. Manure is disinfected by biothermal method.

Control questions and tasks.

1. The causative agent of hoof rot and its characteristics.
2. Epizootological data.
3. Forms of clinical manifestation of the disease and their main clinical signs.
4. On the basis of the analysis of what data is a diagnosis of hoof rot made?
5. Group and individual methods of treatment of sick animals with hoof rot.
6. Prevention and control measures.

Topic: Contagious pustular dermatitis of sheep and goats

(diagnosis, prevention and control measures).

Contagious pustular dermatitis (Dermatitis pustulosa contagiosa, contagious ecthyma of sheep and goats) is an acute viral disease characterized by vesicular-pustular lesions of the mucous membrane of the oral cavity and the skin of the lips, head, udder, genitals and limbs. People are susceptible to the disease.

Breeding is registered in all countries of the world with developed sheep and goat breeding. Economic losses consist of the cost of dead animals (from 0.5 to 90%), reduced productivity, costs of preventive and curative and quarantine and liquidation measures.

The causative agent of the disease is a smallpox-like DNA genomic epiliotropic virus belonging to the family Poxviridae, genus Parapoxvirus. It has an oval shape. It differs from viruses that cause smallpox by antigenic and immunobiological properties. In smears from affected tissues, it appears in the form of elementary bodies, which are well stained according to Pashen and Morozov.

The virus reproduces in primary cultures of cells of kidneys or testicles of lambs, kidneys of embryos of sheep and cows with manifestation of cytopathogenic effect. Extremely stable in the external environment. Retains pathogenicity for 4-15 years in vesicles, pustules, crusts and on wool. In a lyophilized state at room temperature, the cultured virus remains viable for more than 5 years. In livestock premises, the virus remains active for more than 3 years, on pastures and mown grass - up to 300 days, on

the surface of the earth and manure - up to 200 days. Under the influence of direct sunlight, it is destroyed after only 42 hours. It dies relatively quickly in a humid environment: at 64°C – after 2 minutes, at 60°C – 5 minutes, at 56°C – after 30 minutes; in distilled water is inactivated within 24 hours. Rotting destroys the virus within 2 weeks.

After entering the body, the virus reproduces in the genitals, causes proliferation and then degeneration of cells, an exudative process. As a result, vesicles appear, which turn into pustules. Necrosis of the surface epithelium and deposition of fibrin lead to crusts and scabs. The skin under the crusts regenerates without the formation of a scar. In the case of secondary microflora complications, purulent-necrotic foci form under the crusts. Such centers can also appear in internal organs and joints.

The diagnosis is made by a complex method on the basis of epizootological data, the clinical picture of the disease, as well as the results of laboratory tests.

Epizootological data. In natural conditions, sheep and goats get sick regardless of age, sex, breed. However, lambs and kids from 4 days to 10 months of age are more sensitive and more seriously ill. The source of the causative agent of infection is sick animals that secrete the virus into the external environment with leaks from the oral cavity, scabs and crusts of the affected areas of the skin, as well as sick animals that carry the virus. Spontaneous infection occurs through traumatized areas of the skin and mucous membranes, which are formed when eating thorny plants and coarse hay. Keeping animals in damp rooms, long races on stony roads or on frozen snow, as well as staying in low-lying marshy pastures contribute to the emergence of the disease. The disease appears suddenly, spreads quickly and covers all susceptible livestock within 2-3 weeks. Contagious pustular dermatitis due to the high resistance of the causative agent in the external environment often acquires a stationary character. It is registered mainly in spring or autumn. Morbidity is 50%, mortality is 10-20%.

Clinical signs and course of the disease. The incubation period lasts 6-8 days. The course of the disease is acute, subacute and chronic. Depending on the localization of the pathological process, labial, stomatitis, ungulate and genital forms of the disease are distinguished.

At the beginning of the disease, red spots of various sizes are formed on the edges of the lips and in the corners of the mouth, in the center of which nodules appear, and later - vesicles and pustules. After the bursting of the pustules, erosions occur, the secretions from them quickly dry up, forming crusts. The animals have a fever, they are depressed, refuse food. During the benign course of the disease, the wound surface is covered with brownish-black scabs, which fall off after 2-3 weeks, and the animal recovers. In the case of a malignant course, the process can be complicated by secondary microflora, which causes deep purulent-necrotic tissue lesions. Sick animals die at the end of 3-4 weeks of illness.

In the stomatitis form, small red spots first appear on the mucous membrane of the oral cavity, then vesicles, which later burst and erosions, ulcers, and necrosis

centers are formed in their place (Fig. 1). In sick animals, severe depression, a significant increase in body temperature, and an increase in regional lymph nodes are observed. Mortality in this form of the disease can reach 80%.

In the ungulate form, the skin in the area of the foreskin, corolla, interspinous cleft is affected, which is covered with vesicles, first with serous, and later with purulent contents. Lameness, soreness in the area of the legs are observed. Nodules, vesicles, pustules, crusts, scabs are found on the skin of the corolla, hooves, and on the top of the interradicular gap. The addition of a secondary infection can lead to the spread of the inflammatory-necrotic process to the base of the skin of the legs, which separates from the horn shoe and softens. The necrotic process spreads to the joints, tendons, bones of the limbs, which leads to the development of panaritium or necrotic pododermatitis. With this form of the disease, metastases are often formed in the lungs. Most of the sick sheep die.

Fig. 1. Stomatitis form
(http://www.ainfo.inia.uy/digital/bitstream/item/12610/1/Braz-J-Microbiol-2019-Mar-05-Costa.pdf)

In the genital form, which is relatively rare, nodules, vesicles, pustules, crusts appear only on the mucous membrane of the vagina, the skin of the udder, teats (Fig. 2), the inner surface of the thighs, in males - on the skin and mucous membrane of the prepuce. The external genitalia are swollen, purulent discharge is observed from the vagina.

Fig. 2 . Genital form
(https://bmcvetres.biomedcentral.com/articles/10.1186/s12917-018-1339-x)

Pathological anatomical changes. Not typical. The corpses of dead animals are skiny. On the mucous membrane of the mouth, skin, lips, and limbs, erosions, ulcers, and foci of necrosis are found. Necrotic cells are found in the liver and lungs, ulcers are found on the mucous membrane of the alimentary canal, and fibrinous exudate is found in the abdominal cavity. During histological examination, partial or complete destruction of the epidermis, infiltration of the subepidermal layer and intermuscular layers by polymorphonuclear leukocytes, histiocytic-lymphoid cells, rapid proliferation of the epithelium of hair follicles, keratinization of the epidermis are revealed.

Laboratory diagnostics. Includes viroscopy of stained smears, bioassay on clinically healthy lambs, complement fixation reaction.

Vesicles, crusts, scabs, necrotized areas of the skin and mucous membranes, parenchymal organs preserved with glycerin solution are sent to the laboratory. Viroscopy makes it possible to quickly identify the causative agent of the disease in smears from fresh lesions and scabs stained according to Morozov. The bioassay is placed on two lambs, which are infected with a 10% suspension of fresh scabs in physiological solution, which is rubbed into the scarified skin of the inner surface of the thigh. If the causative agent of the disease is present in the pathological material, characteristic signs of the disease appear in infected lambs for 3-5 days.

Differential diagnosis. Presupposes the need to exclude smallpox, foot-and-mouth disease and necrobacteriosis. Sheep pox has the character of a severe general disease, it occurs in the form of a generalized exanthema with the formation of a papulo-pustular rash on hairless and lightly covered parts of the body. The absence of typical vesicles is characteristic, and the nodules that are formed merge with each other and protrude above the surface of the skin in the form of large cross-shaped tubercles. Foot-and-mouth disease is characterized by aphthous damage to the skin of the corolla, soft tissue and the wall of the interradicular fissure. Canker sores on the mucous membrane of the oral cavity in sheep are rare and small in size. With necrobacteriosis,

a purulent-necrotic lesion of the skin of the wall of the interspinous cleft and the base of the skin of the scion develops, which is accompanied by separation from the hoof horn shoe.

Treatment. Specific means of treatment of contagious pustular dermatitis have not been developed. Sick animals are isolated, symptomatic therapy is carried out. The affected mucous membranes of the oral cavity are washed with a 3% solution of hydrogen peroxide, copper sulfate, a weak solution of various astringents and disinfectants; ulcers are lubricated with iodoglycerin. If the skin of the lips, head, or udder is affected, use synthomycin emulsion, zinc, oxytetracycline, polymyxin, dibiomycin, and salicylic ointments, salicylic alcohol, or a mixture of drugs consisting of equal parts of 7% formalin solution and 10% copper sulfate solution. In complicated cases of the disease, especially when it is difficult to accept feed, surgical treatment is performed, antibiotics of a wide spectrum of action are used (biomycin is administered internally at 0.02-0.03 mg/kg for 3 days, biomycin is administered intramuscularly at a dose of 4 mg/kg kg, subcutaneously - 1-2% terramycin solution in a dose of 1-1.5 ml for 3-4 days).

Immunity. Diseased animals become immune to re-infection for two years. Dry culture virus vaccine is used for active immunization of ewes and newborn lambs in disadvantaged farms. The vaccine in a dose of 0.3 ml is applied to the scarified surface of the skin of the upper lip. Immunity is formed after 15 days and lasts up to 8 months.

Prevention and control measures. In order to prevent the disease, sheep and goats for stocking should be imported only from farms that are safe with regard to contagious pustular dermatitis. All imported sheep and goats are quarantined for 30 days. When the disease occurs, the farm is declared unhealthy for contagious pustular dermatitis and quarantine restrictions are introduced. Sick animals are isolated and treated, the rest of the conditionally healthy herd is vaccinated. Unhealthy flocks are driven through disinfection baths, which are filled with an emulsion of creolin (1 part), tar (4 parts) and water (20 parts). Thorough cleaning and disinfection of livestock premises and pens are carried out. For disinfection, a 2% hot solution of caustic sodium or potassium, a 20% suspension of freshly slaked lime, and a 2% formaldehyde solution are used. Manure is disinfected by the biothermal method. Quarantine from a dysfunctional farm is lifted 3 weeks after the last case of death or recovery of the last sick animal and the final disinfection. Pastures where sick animals were grazed are not used for two years.

Control questions and tasks.

1. The causative agent of contagious pustular dermatitis and its characteristics.
2. Epizootological features of the disease.
3. Forms of the clinical course of the disease and their main clinical signs.
4. Characteristic patho-anatomical changes.
5. When is the diagnosis of contagious pustular dermatitis considered established?

6. Describe the general and specific preventive and health measures for contagious pustular dermatitis.

Topic: Infectious epididymitis of sheep

(diagnosis, prevention and control measures).

Infectious epididymitis of rams (Epididymitis infectiosa arietum) is a chronic disease accompanied by proliferative inflammation and atrophy of the testes and appendages in rams, a decrease in their reproductive function.

The disease occurs in many countries of the world. In Ukraine, it was first registered in 1971. Economic losses consist of costs in connection with the violation of breeding work, culling of animals, long-term quarantine, and the implementation of veterinary, sanitary and economic measures to eliminate the disease.

The causative agent of the disease. Brucella ovis - differs from other types of Brucella from the genus Brucella in terms of antigenic and immunogenic properties, it is a resistant R-form. This is a small, immobile gram-negative coccobacillus, which is most often placed in piles of 5-20 cells in pathological material. It does not form spores or capsules. In preparations stained according to Kozlovsky's method, it has a red color, according to Ziel-Nielsen, it is pink.

Brucella are cultivated on special nutrient media - meat-peptone liver broth, meat-peptone liver-glucose-glycerol agar and 2-3% glycerol, potato agar. On the surface of dense environments, brucelli form small, round, transparent (in the form of dew drops) colonies with a blue tint. In semi-liquid media, growth is accompanied by turbidity and the formation of a small sediment. They reproduce well in 7-day-old chicken embryos, which die 42-72 hours after infection. Brucella are resistant to environmental factors: they can be stored in wet soil for more than 70 days, in manure - up to 5 days. At a temperature of 60°C, brucelli die in 30 minutes, at 70°C - in 5-10 minutes, at 90-100°C - instantly. Solar radiation inactivates them within 3-4 hours.

The diagnosis is made by a complex method based on the analysis of the epizootic situation, the characteristic clinical picture of the disease, patho-anatomical changes and the results of serological, bacteriological and allergic studies.

Epizootological data. To the pathogen Br. ovis only sheep are susceptible. Puberty rams aged 2-7 years are most often affected. Lambs do not get sick up to 6 months of age. The main source of the pathogen is infected rams, the use of which for fertilization can lead to abortions in 100% of ewes. In sick rams, the causative agent is excreted from the body with semen (for 2-3 years) and urine, in ewes - with aborted fetuses, fetal membranes and postpartum waters. In the presence of mastitis, the causative agent can be excreted with milk. In sheep farms, the source of the pathogen can also be sentinel dogs suffering from brucellosis, which secrete brucelli with urine. Entering Br. ovis in farms free from this infection occurs in the case of the introduction

of covertly infected breeding rams, as well as the mixing of healthy and unhealthy flocks in pastures and watering places. Ewes become infected during mating with infected rams, in the testicles and appendages of which Brucella can persist and multiply for years, or during artificial insemination with Brucella-contaminated sperm. Unlike rams, most ewes self-recover after 3-4 months, but the pathogen persists in the uterus for 2-3 years or longer. On average, 10% of pregnant ewes abort 2-4 weeks before normal litters. In most ewes, long-term infertility occurs after abortion, during mating they infect healthy rams. The disease occurs during the mating season in ewes in the form of enzootic outbreaks, in lambs 10-15 months old - sporadically. The epizootic process of sheep epididymitis is characterized by stationarity, very slow spread of infection, lack of seasonality and periodicity.

Clinical signs and course of the disease. The incubation period lasts 1-8 weeks. The course of the disease is acute or chronic.

During the acute course of the disease, lambs experience general depression, an increase in body temperature up to 41-42°C, loss of appetite, testicular tenderness, and an increase in their size by 5-7 times. The appendages of the testes also increase, sometimes to the size of a chicken egg (Fig. 1).

Fig. 1. Symptoms of infectious epididymitis of sheep (https://www.researchgate.net/figure/Two-affected-rams-showing-unilateral-left-and-bilateral-right-enlargement-of-scrotum_fig2_11522513)

The skin in the area of the scrotum is hot, tense, painful. After 10-20 days, the acute course of the disease turns into a chronic one. In many lambs, the infection is asymptomatic, but it is accompanied by impaired spermatogenesis, necrospermia and aspermia, which causes low fertility in ewes.

In the chronic course of the disease, rams show asymmetry of the scrotum, one- or two-sided enlargement of the epididymis, fluctuation or dense bumpiness, decrease or absence of their mobility. Abortions in ewes occur easily, without delaying the litter,

but some have complications and death from sepsis. The number of overgrazing increases, and the barrenness can reach 20-60%. 7-14 days after birth, the death of many underdeveloped lambs is observed.

Pathological anatomical changes. In rams, the main lesions are localized in the appendages of the testis, in the testes themselves and cystic glands and are manifested by necrotic and purulent inflammations, sequestrations, petrified cells, growth of fibrinous tissue, fusion of the vaginal membrane with the testis and the appendage. In ewes, a pus-like mass accumulates on the surface of the amniotic membrane and chorionalantois.

Laboratory studies. Includes microscopic and bacteriological studies of pathological material taken from dead animals, as well as serological studies of blood sera of sick and infected sheep. Affected testicles from rams together with appendages are sent to the laboratory, from ewes - aborted fetuses with fetal membranes, affected areas of uterine horns, ovaries, pelvic lymph nodes, as well as secretions from the genital tract, taken in the first 5 days after abortion. For serological studies, paired blood samples are taken after abortion, and then from the same animals after 8-10 days. With positive results of microscopic examination of smears from pathological material stained according to Kozlovsky, small red cocoon-shaped brucelli are found on the green background of the preparation. On dense nutrient media for 7-9 days, the growth of Br culture is observed. ovis in the form of characteristic small gray-white colonies with a blue tint. The morphological and cultural-biochemical properties of the isolated culture are studied, and its serological identification is carried out. To do this, two rabbits are intravenously injected twice with an interval of 7-8 days with 2 daily cultures of Brucella. After 10-12 days, their blood is tested using the long-term binding reaction of complement with oat antigen. The reaction is considered positive, and the diagnosis of sheep epididymitis is established in the case of delayed hemolysis in two or four crosses when diluting sera 1:5-1:10.

Differential diagnosis. Infectious epididymitis must be differentiated from brucellosis, diplococcal infection, pseudotuberculosis, salmonellosis, campylobacteriosis, listeriosis, chlamydia, traumatic epididymitis.

Ewes are more susceptible to brucellosis. Blood sera give positive results by complement binding reaction and agglutination reaction with brucellosis antigen. With diplococcal infection and pseudotuberculosis in ewes, there are no abortions, epididymitis in rams is recorded sporadically. The reaction of long-term binding of complement to oat antigen is negative. Salmonellosis is accompanied by mass abortions in ewes. Rams do not have epididymitis. The results of the agglutination reaction of blood sera of sick animals with salmonellosis antigens are positive. With campylobacteriosis in ewes, abortions are registered 30-45 days before farrowing. The results of the agglutination reaction of blood sera of sick ewes with intestinal campylobacter antigen are positive. Listeriosis manifests itself sporadically, with high mortality in the case of the nervous form of the disease. Blood sera of aborted ewes

give positive results for agglutination reactions and complement binding reactions with listeriosis antigens. In case of chlamydia, ewes have abortions, in animals of other age groups, the course of infection is latent. Traumatic epididymitis is registered in single animals. The results of the reaction of long-term binding of complement to ovis antigen are negative.

Treatment. not developed Sick and reactive rams and ewes reacting to the reaction of long-term complement binding are culled for fattening.

Immunity. For active immunization of rams in farms that are unfavorable for infectious epididymitis, a live dry vaccine from the Rev-1 strain of Brucella melitensis is used. The vaccine is injected subcutaneously in a hairless area behind the elbow joint. Immunity occurs after 3 weeks and lasts at least 2 years. Vaccinated rams are examined serologically (agglutination reaction and long-term complement binding reaction) no earlier than 12 months after vaccination, but necessarily before the mating company. When rams reacting to infectious epididymitis are detected, all animals of this group are tested using the long-term complement fixation reaction every 30 days until two consecutive negative results are obtained per group, and then they are kept under observation for 6 months, during which they are examined twice. In the case of negative results and the absence of clinical signs of the disease, the group is recognized as healthy, and the rams are allowed to be used in reproduction. If animals with positive reactions to brucellosis are found during the research, the group is further examined every 30 days by serological (complement binding reaction) and allergic methods and the animals are treated as indicated above. Animals that serologically react to brucellosis or infectious epididymitis are culled for fattening.

Prevention and control measures. All rams imported from other farms must be quarantined and examined for infectious epididymitis. In breeding farms and artificial insemination stations, once a year, before the start of the mating company, a mandatory diagnostic test for infectious epididymitis of breeding rams is also carried out. Young rams are examined from the age of 12 months. In case of detection of infectious epididymitis in rams, the breeding station is declared unhealthy and quarantine restrictions are introduced. Rams with clinical signs of the disease or those that react positively during the study for prolonged complement binding reactions are slaughtered. The recovery of a dysfunctional herd is carried out either by slaughtering all animals and completely replacing the entire stock of the breeding farm, or by means of systematic serological tests once a month until negative results are obtained twice in a row. For disinfection, use 2% formaldehyde solution, 2-3% creolin emulsion, 5% freshly slaked lime with exposure for 1-3 hours.

Control questions and tasks.

1. The causative agent of infectious epididymitis and its characteristics.
2. Epizootological data.
3. Clinical signs and course of the disease.

4. What investigations does the complex method of diagnosing infectious epididymitis include?
5. Differential diagnosis of infectious epididymitis.
6. Means of specific prevention for active immunization of rams in farms unfavorable for infectious epididymitis.
7. Prevention and control measures

Topic: Sheep pox

(diagnosis, prevention and control measures).

Smallpox (variola) is an acute contagious viral disease of various species of animals and birds, characterized by the development of a specific papular-pustular rash on the skin and mucous membranes, which successively goes through the stages of formation from roseules to scabs and crusts. Human is susceptible to smallpox.

Smallpox in animals is widespread in Africa, Asia, in particular in the Middle East. In dysfunctional farms, the disease causes significant damage in connection with high mortality among lambs (50%) and a decrease in shearing of wool in sheep.

The causative agent of the disease. Epitheliotropic viruses from the Poxviridae family, which are morphologically similar in different species of animals, but differ in pathogenicity, antigenic and immunological properties. Natural cowpox virus and vaccinia virus can cause disease in cattle, pigs, buffalo, camels, mules, and horses; human pox virus in humans and cattle; sheep pox virus - in sheep and goats; goat and fowl pox viruses - only in goats and fowl.

Regardless of their species, smallpox virions have a complex type of symmetry, a rather large size (260×390 - 170×350 nm), which allows them to be detected under a conventional microscope. The cluster of virions was called "elementary bodies", or Paschen bodies, which are well stained by the Morozov silvering method, as well as by the Romanovsky-Giems method. Under an electron microscope, smallpox virions look like short, thick rods, in the central part of which there is a nucleoid with DNA. Smallpox virus is cultivated in primary cultures and subcultures of cells of kidneys and testicles of lambs, calves, goats, where they after 48-96 hours. cause cytopathogenic effect. Of laboratory animals, rabbits are susceptible to the smallpox virus. Experimental infection is also possible in young sheep with intradermal infection with sheep pox virus.

The smallpox virus is quite resistant to environmental factors. It remains viable in lymph at 2-4°C for at least 2 years, in dry crusts - 4-5 years, on pasture - up to 65 days, in sheep's wool - up to 2 months. In a lyophilized state at minus 15-20°C, the smallpox virus remains active for up to 20 years. High temperatures and decay quickly destroy the virus. Virions are also inactivated by 3% formaldehyde solution, 3-5% chloramine solution, 20% slaked lime solution. When boiling - for 2-3 minutes, at 70°C

- 5 minutes, 60°C - 10 minutes, 55°C - 20 minutes, 39°C - 24 hours. Ultraviolet radiation kills the virus for 4 hours.

Pathogenesis. After entering the body, the smallpox virus reproduces in the sensitive cells of the epithelium of the respiratory tract, then enters the blood, causes viremia, is carried throughout the body by blood, settles in the epithelial cells of the mucous membranes and skin, where it causes the exanthem characteristic of smallpox. During the development of viremia, fever, depression, and chills are observed in sick animals.

The course of the pathological process in smallpox depends on the virulence of the causative agent, as well as the state of resistance of the organism, species, age and breed of the animal. In the typical form of smallpox, there is a clear phasing of the development of smallpox exanthema on the mucous membranes and hairless tender areas of the skin, which ends within 12-18 days. In the first 1-2 days, roseolae (red spots) appear, which during the next 1-3 days turn into papules with serous fluid and become vesicles. During this period, the body temperature decreases, the animal's health improves. Then, within 3 days, the vesicles turn into pustules (suppurative stage), in place of which scabs appear after 2-3 days (crusted stage). After 5-6 days, the scabs fall off, the pathological process of exanthema formation stops, recovery occurs.

In complicated cases, pustules merge with each other and form wide foci of suppuration, the so-called "draining smallpox". Sometimes multiple hemorrhages appear inside the pustules, hemorrhagic, or black, smallpox develops. Skin lesions of the smallpox virus can be limited to the formation of nodules, then smallpox is observed. In sheep and goats, the stages of vesicles and pustules pass unnoticed.

The diagnosis is based on the detection of smallpox exanthema characteristic of the disease, the data of the analysis of the epizootic situation, as well as on the results of laboratory studies.

Epizootological data. Sheep, pigs, goats, cattle, camels, horses, rabbits, chickens, turkeys, pigeons are susceptible to smallpox. Young animals are more malignant and with high mortality (20-90%), especially in the case of secondary microflora complications.

The source of the causative agent of infection is sick animals, as well as sick virus carriers, in which the causative agent of the disease persists on fur and skin for up to 8 weeks. The virus is released from the body of infected animals with the contents of smallpox pustules, crusts and falling off scabs, saliva, secretions from the nose and eyes. Factors of transmission of the pathogen can be pastures, pens, paths in places where cattle are run, as well as care items, fodder, wool, skins, manure, clothes and shoes of service personnel contaminated with the virus. In the spread of smallpox, stinging insects, lice, mouse-like rodents, cats and dogs can play a role. Infection occurs by contact when healthy animals are kept together with sick ones, through the respiratory tract, damaged skin, less often - through the digestive tract. Intrauterine

infection is possible in sheep. The disease occurs at any time of the year, but more often appears in the stall period, in cold and rainy weather.

Smallpox is epizootic among sheep and goats. The infection spreads quickly and within 2-3 weeks can cover up to 80-100% of susceptible animals. The emergence and significant spread of infection is facilitated by various stress factors associated with violation of standard conditions for feeding and keeping animals, especially young animals. A very severe course of smallpox in lambs of thin-wooled breeds, in which the morbidity can reach 100%. Epizootic foci of smallpox can turn into stationary ones, in which, against the background of post-infectious immunity, the disease is detected only in young animals, often with an atypical development of the infectious process.

Clinical signs and course of the disease. The incubation period lasts 3-14 days. The course of the disease can be benign or malignant. There are typical, atypical (stone), draining and hemorrhagic forms of smallpox.

In sheep, with the typical form, there is an increase in body temperature to 41-42°C, increased breathing, depression, chills, loss of appetite, cessation of chewing, hyperemia of the nasal mucosa, conjunctivitis, mucous, and later mucous-purulent discharge from the nose and eyes . After 1-4 days, on hairless and sparsely covered areas of the head, around the eyes, on the nose, on the inner surface of the thighs, belly, tail, and udder, smallpox exanthema gradually develops.

With a benign course of the disease, at the end of 3-4 weeks, smallpox crusts fall off, the animals recover. With a malignant course, complications from the respiratory and digestive organs appear. Coughing, wheezing, nasal discharge, clouding of the cornea are observed, pregnant sheep abort.

In the atypical form, red hard nodules (papules) of rounded or oblong shape are formed on the skin, which dry up and disappear without transitioning into vesicles. Sick sheep soon recover. In the confluent form of smallpox, there is fever, depression, loss of appetite, purulent-ichorous discharge from the nose; a lot of saliva flows from the mouth with an unpleasant ichorous smell. Purulent inflammation of the subcutaneous tissue, extensive skin lesions are observed.

In the hemorrhagic form of smallpox (blackpox), as a result of hemorrhages in the skin and internal organs, vesicles and pustules acquire a dark red or black color, hematuria, bloody diarrhea, severe general condition of animals and high mortality (50-60%) are observed. The tips of the ears, large areas of skin, as well as areas of the lips and eyelids become necrotic and fall off.

In goats, smallpox occurs rarely and is accompanied by fever and depression, a characteristic smallpox lesion of the skin on the udder. Sometimes smallpox exanthema appears on the scalp, the inner surface of the thighs, the abdomen, on the mucous membranes of the external genitalia, oral and nasal cavities (Fig. 1). Abortions, complications with pneumonia, mastitis are possible.

Fig. 1. Exanthema on the scalp during the course of smallpox (https://homemasters.cx.ua/vispa-ovec-i-kiz-instrukcija-po-profilaktici-i.html)

Pathological anatomical changes. During the examination and autopsy of the corpses of animals killed by smallpox, characteristic exanthematous lesions of the skin in various parts of the body, hard nodules with cheesy decay under the pleura, inflammation of the mucous membranes of the respiratory tract and digestive tract, enlargement of the spleen, degenerative changes in the myocardium and liver, foci of hepatization in lungs Lymph nodes are swollen and enlarged. When dissecting the corpses of sheep in which the disease took place in a malignant form, in addition to the characteristic skin lesions, smallpox lesions are also found in the lungs, liver, kidneys, on the mucous membranes of the alimentary canal and respiratory organs. Multiple hemorrhages in the serous membranes, enlargement and hyperemia of lymph nodes, degenerative changes in the liver, kidneys, and heart are observed. Pulmonary pneumonia and gangrenous foci are often found in the lungs.

Laboratory diagnostics. Includes microscopic detection of elementary bodies in pathological material, infection of chicken embryos, and in doubtful cases - conducting a bioassay on sensitive animals.

Vesicular fluid collected in the capillaries of Pasteur pipettes and whole papules cut with scissors on the border with intact tissue are sent to the laboratory for examination, which are placed in a vial with a 50% glycerin solution. For viroscopy, thin smears and smears-imprints of smallpox skin lesions and pustules are prepared, which after drying are examined in their native state or stained according to the Morozov or Paschen method. During the microscopic examination of Morozov-stained smears on a light brown background of the drug, viral particles have the appearance of small rounded elementary bodies of black color, placed singly, in pairs, in short chains or clusters. In strokes colored according to Paschen, small rounded elementary bodies have a dark red color.

In case of an atypical form of smallpox in sheep, a biological sample is used for diagnosis. The studied pathological material in a volume of 0.1 ml is injected

intradermally into the hairless surface of the tail of a non-immune young sheep. In positive cases, a specific local smallpox process develops at the inoculation site within 10 days, which is confirmed by the presence of elementary bodies in smears.

Differential diagnosis. Presupposes the need to distinguish smallpox of sheep and goats from scabies. Scabies is diagnosed by the detection of microscopic fungi in the pathological material, scabies by the presence of specific mites in the crusts.

Immunity. In sheep infected with smallpox, permanent immunity is formed for 2-3 years. For specific prevention, hydroxydaluminum formol vaccine against sheep pox, dry culture virus vaccine against sheep pox and hydroxydaluminum formolglycerin vaccine against goat pox are used..

Hydroxidaluminum formol vaccine against sheep pox is used for preventive purposes in disadvantaged or threatened farms. Clinically healthy animals are vaccinated regardless of their physiological state and age. Lambs vaccinated before the age of 2 months are revaccinated after 2-3 months, then they are vaccinated twice a year. Immunity in sheep occurs on the 15th day after vaccination and persists for 6-8 months.

Dry cultured sheeppox vaccine virus is used for preventive immunization of clinically healthy sheep in epizootic centers and smallpox-threatening areas. Youngsters are vaccinated from the age of one month, revaccinated at the age of 6 months. Adult sheep are vaccinated once every 12 months. Immunity occurs after 4-5 days and lasts for 12 months.

Hydroxidaluminum formolglycerine vaccine against smallpox of goats is used for prophylactic purposes in all disadvantaged farms without any restrictions. The young are revaccinated after 3-4 months. Immunity lasts up to 6 months.

Treatment. Specific means of treatment of smallpox are not offered. Sick animals are isolated in dry, clean, well-ventilated rooms, provided with soft nutritious food. Symptomatic treatment is carried out. Mucous membranes are irrigated with antiseptic and astringent liquids, smallpox skin lesions are treated with various ointments (borane, propolis, zinc, salicylic), emulsions (streptocide, synthomycin) and antiseptic liquids. Broad-spectrum antibiotics are used to prevent and treat complications caused by bacterial microflora.

Measures for the prevention of sheep pox. In order to prevent the occurrence of smallpox and prevent its spread, heads of farms, as well as citizens - owners of sheep are obliged:

- prevent the introduction (importation) of sheep, as well as fodder and inventory from farms affected by sheep pox;
- keep all sheep newly brought to the farm in a 30-day preventive quarantine;
- constantly maintain pastures, watering places, livestock premises in proper veterinary and sanitary condition;
- to secure permanent service personnel for the flocks, as well as pasture areas, watering places and racing tracks;

- to provide systematic veterinary monitoring of the state of sheep.

Measures to eliminate sheep pox in a disadvantaged area

When the diagnosis is confirmed, the farm is declared to be in trouble due to sheep pox and a quarantine is established. Prohibited:

- introduction and importation into the unfavorable point, removal and removal from it of animals of all species, regrouping of animals within the farm, as well as grazing, drinking and maintenance of sick sheep together with healthy animals of all species;

- removal of fodder, which was encountered by sick sheep (in sheds, on pastures), from an unfavorable point. This fodder is fed on the spot to animals that are not susceptible to sheep pox, or to sick and infected sheep with small pox;

- use of sheep's milk and products obtained from it in an uncontaminated form. Milk obtained from sheep is subject to disinfection on the spot by pasteurization at a temperature of 85°C for 30 minutes. or boiling for 5 min. with further use of it in the economy;

- shearing of sheep affected by smallpox in the flock until the quarantine is lifted;

- trade in animals and livestock products, holding exhibitions, fairs, bazaars and other events related to the accumulation of animals, people, and transport in the quarantine area;

- traffic on the territory of the outbreak of sheep pox. Detour routes must be indicated for transport to its destination;

- access of people not related to the care of animals of disadvantaged groups to premises and other places where these animals are kept.

The corpses of sheep that died in the presence of clinical signs of smallpox are burned. It is forbidden to remove skins and use wool from corpses.

All clinically healthy sheep are vaccinated with smallpox vaccine. Vaccinated animals must be under the supervision of veterinary specialists for 14 days. When sick sheep are found among the vaccinated flock, they are transferred to the sick group and subjected to treatment.

One of the following disinfectants is used to disinfect premises, pens and other places where animals are kept: hot 2% solution of caustic sodium or potassium; hot 3% solution of sulfur-carbolic mixture; 20% solution of freshly slaked lime; clarified solution of perchloric lime or sodium hypochlorite, with a content of at least 2% active chlorine; 2% formaldehyde solution. Walls, fences and various wooden fences should be disinfected with a freshly prepared solution of quicklime. Manure is disinfected within 3 weeks by the biothermal method. Skins obtained during sheep slaughter are disinfected in a 3% solution of phenol or in a 2.5% creolin emulsion for 24 hours, after which they are dried. The export of sheepskin is allowed only after the quarantine is lifted.

Quarantine is lifted after the end of 20 days after the complete recovery, death or slaughter of the last sick sheep in this point. After the quarantine is lifted, sheep

newly introduced to the farm must be vaccinated against sheep pox during the period of preventive quarantine.

Control questions and tasks.

1. The causative agent of smallpox and its characteristics
2. Epizootological data of smallpox.
3. Stages of development of smallpox exanthema.
4. Forms of clinical manifestation of smallpox.
5. Clinical signs of smallpox.
6. Pathological-anatomical changes in smallpox.
7. Differential diagnosis of smallpox.
8. How are sick animals treated with smallpox?
9. What disinfectants are used to disinfect premises in case of smallpox?
10. Means of specific prevention of smallpox.
11. When quarantine is lifted in a dysfunctional farm?

Topic: Visna-maedi of sheep and goats

(diagnosis, prevention and control measures).

Visna-Maedi – slow viral infection of sheep and goats, manifested by signs of non-purulent meningoencephalitis or progressive interstitial pneumonia. Simultaneous damage to the nervous system and lungs is possible.

The name of the disease consists of two Icelandic words - "visna", which characterizes the state of paralysis, and "maedi" - a symptom of lung damage. Later, it was established that "Visna" and "Maedi" are two clinical forms of the same disease. It is registered in many countries of Western Europe - Iceland, Belgium, Denmark, France, England, Germany, Holland, as well as in the USA, on the African and Asian continents.

The causative agent of the disease. RNA-genomic virus from the family Retroviridae, genus Lentivirus. Virions are spherical in shape, 70-100 nm in diameter, covered with an outer lipoprotein shell. The core of the virion includes an icosahedral capsid and a helical nucleocapsid. The virus is cultivated in the primary culture of lamb kidney cells and sheep brain vascular plexus cells, where 9-12 days after infection, multinucleated giant cells - symplasts are detected. Laboratory animals are not sensitive to the causative agent of visna-mayedi. A significant resistance of the virus to the influence of various physico-chemical factors has been established: at 4-8°C, the virus is stored for several months, withstands repeated freezing and thawing, as well as UV irradiation. At 57°C, the virus is inactivated after 28 minutes.

It is believed that the hidden and long-term persistence of the virus can be caused by a close connection with the cells of the reticulo-histiocytic system. The pathogenetic

mechanism of visna-maedi is marked proliferation and hyperplasia of cells of lymphoid and epithelial tissues. The disease leads to a violation of immunological reactivity in the body of sheep. The virus that has entered the body enters the blood, accumulates in the cerebrospinal fluid, saliva, and then enters the central nervous system, where it causes disruption of the protein substance of the brain and causes large accumulations of cells in the meninges. Penetrating into the respiratory organs, it causes damage to the lungs and spleen. The pathogenetic process develops slowly. Long persistence of the virus in lymphocytes and macrophages is characteristic.

The diagnosis is made on the basis of the analysis of epizootological, clinical and patho-anatomical data, patho-histological changes and the results of laboratory tests.

Epizootological data. Sheep and goats older than 2 years are susceptible to the disease. The source of the causative agent of the disease is sick animals. Infection occurs when healthy and sick sheep are kept together in closed rooms. The main ways of infection are contact, airborne, alimentary. The virus is excreted from the body through milk, feces, and exhaled air.

From infected ewes, the virus is transmitted to lambs through colostrum, intraplacental infection is possible. A characteristic feature of the disease is the extremely slow development of enzootic disease and its long course. In the case of the manifestation of the disease in the form of maadi, the infection spreads very slowly, and in the first years after its introduction, the death of animals almost never happens. In the next 3-4 years, the disease progresses rapidly, especially under the influence of various stress factors (hypothermia, lack of feed, complications in females after birth of young). The clinical phase lasts from 3 to 8 months, the mortality rate during the year can reach 20-30%. The patient is characterized by lymphocytosis, which persists for years.

If the disease manifests itself in the form of visna, the course of the disease can last from several months to several years. The clinical phase lasts for one month. All sick animals die.

Clinical signs and course of the disease. The incubation period lasts from 6 months to two years. The clinical picture develops very slowly. Symptoms of the disease depend on the form of the infection.

Visna-maedi manifests itself in pulmonary and nervous forms. Early symptoms are slight trembling of the lips and an abnormal position of the head. Progression of the disease leads to paraplegia or general paralysis. The progression of the pulmonary form causes shortness of breath, then a dry cough and the appearance of mucous secretions from the nose. Exhaustion sets in. The disease always ends with the death of animals.

In visna form, signs of damage to the central nervous system dominate: movement coordination disorder, head tremors, twitching of the lips, neck curvature, paresis and paralysis of the limbs, impairment of the ability to extend the hind legs, due to which the animal cannot rise (Fig. 1).

Body temperature is normal. Clinical signs progress slowly. Changes in behavior are detected (depression, timidity, impaired coordination of movements). Sometimes an early sign is persistent twitching of the lips, head tilted to one side. Symptoms of the disease progress over 3-6, sometimes 12 months. The paralytic stage lasts from several weeks to 2 years. Paresis and paralysis of the pelvic limbs appear. General paralysis gradually develops, which leads to death.

Symptoms of lung damage are characteristic of the disease in maedi form: frequent, difficult breathing, lethargy, progressive weight loss (Fig. 2), despite a good appetite, dry cough. Pregnant females can abort. The animal lies more, breathes heavily. Body temperature is normal. The clinical stage lasts 3-6 months, sometimes several years. The end of the disease is always fatal. Sheep often die as a result of secondary infection.

Fig. 1. Paralysis of hind limbs

Fig. 2. Exhaustion in the case of visna-maedi (https://www.researchgate.net/figure/Clinical-symptioms-of-Louping-ill-disease-in-sheep-Depression-A-ataxia-of-the-hind_fig1_353041167)

Pathological anatomical changes. In visna, they are characterized by signs of diffuse encephalomyelitis with demyelination. Skeletal muscle atrophy and vascular congestion are also observed. It is characterized by a 2-4 times increase in the size of the lungs, which have a dense spongy consistency and a specific gray-yellow or gray-white color ("white" lungs).

Peribronchial, bronchial and mediastinal lymph nodes are enlarged and swollen. The bronchi are unchanged, sometimes small epithelial nodules with cubic or cylindrical epithelial cells are found inside the bronchi. During histological studies, progressive chronic interstitial pneumonia, proliferation and formation of lymph follicles in the lungs are revealed. In the parenchyma of the liver, interstitial tissue of the kidneys and udder, lymphohistiocytic cell proliferations are found, in the spleen and lymph nodes at the beginning of the disease, lymphoid tissue hyperplasia is detected.

Laboratory diagnostics. It involves the isolation of the causative agent of visna-mayedi from the pathological material of sick animals in cell cultures, the identification

of the isolated virus in the neutralization reaction, serological tests for the detection of specific antibodies in the blood. In case of suspicion of visna-maedi, pieces of the affected lungs, bronchial and mediastinal lymph nodes, brain are sent to the laboratory, which are fixed with a 10% formalin solution. Pathological material for research is selected from at least 5 animals. Isolation and identification of Visna-mayedi virus are associated with significant difficulties in growing it in cell cultures.

Differential diagnosis. It provides for the exclusion of scrapie, coenurosis, listeriosis, tuberculosis, Scottish encephalomyelitis. In this regard, it should be noted that laboratory animals do not get sick from visna disease. Viral, bacterial or invasive diseases can occur practically against the background of visna-maedi, introducing their specific nuances into the syndrome of the main disease.

Immunity. Does not form. Means of specific prevention have not been developed.

Treatment. No therapeutic drugs are offered.

Prevention and control measures. Aimed at preventing the introduction of the spring virus into healthy flocks. Restrictions are imposed on a dysfunctional economy. Prohibit sale, export, exhibitions, grazing on common pastures, exclude contact with animals of prosperous farms.

In unfavorable farms, serological tests are carried out at 6-month intervals, seropositive animals and sick animals are slaughtered. The lambs are isolated from the infected ewes immediately after the birth of the lambs and transferred to feeding with colostrum and cow's milk. The herd can be cured by culling seropositive animals if no more than 30% of the herd is infected.

The most effective method of eradicating the disease is the general slaughter of the herd in which visna-mayedi is found, cleaning and thorough disinfection.

2-4% solutions of caustic soda, 20% suspension of freshly slaked lime, clarified solution of chlorinated lime with an active chlorine content of at least 2% are used for disinfection of pens and care items.

Control questions and tasks.

1. The causative agent of visna-maedi and its characteristics.
2. Epizotological data of visna-maedi.
3. Forms of the clinical course and their main signs.
4. Characteristic patho-anatomical changes in the visna form.
5. Characteristic patho-anatomical changes at the maedi.
6. Differential diagnosis of visna-maedi.
7. How do you deal with sick animals in the visna-maedi?
8. What disinfectants are used to disinfect premises during visna-maedi?
9. At what intervals are serological tests carried out in disadvantaged farms?
10. At what level of infection can the herd be cured by culling seropositive animals?

Topic: Scrapie of sheep and goats

(diagnosis, prevention and control measures).

Scrapie is a prion infection of sheep and goats, which is characterized by a long incubation period, a slow course, itching, dystrophic-necrotic lesions of the central nervous system, which causes impaired coordination of movements (ataxia) and trembling of the whole body (tremor). The disease always ends fatally.

The disease is registered in many European countries (Belgium, Hungary, Bulgaria), Asia, North and Central Africa, South America, the USA and Japan.

The causative agent of the disease. An infectious prion protein (a specific sialoglycoprotein) that forms plaques of scrapie-associated fibrils in the brain. The nature of the causative agent of scrapie has not been fully elucidated. There are several hypotheses, including the one according to which the prion is a virus with a negative genome. "Protein reverse translation" is allowed, as a result of which DNA or RNA is first synthesized, and later the processes of biosynthesis of the virus-specific prion protein begin. They assume the existence of a gene in a normal mammalian cell that activates after infection and "starts" the process of prion protein biosynthesis.

The causative agent of the disease is isolated from the brain, spleen and lymph nodes of sick sheep. In goats, the causative agent is found in the highest concentration in the brain, adrenal glands, pancreas, and sometimes in the cerebrospinal fluid. It was not found in blood and urine. The prion is extremely resistant to physical and chemical factors: temperature rise, UV radiation, penetrating radiation, ultrasound, formalin, pepsin and trypsin. In the dried state, it remains viable at 8-12°C for 2 years. It is not destroyed by boiling tissue taken from sick sheep, even after 30 minutes. Not sensitive to ether, periodate, phenol, mixture of chloroform and methanol. Destroys at 120°C in just 30 minutes, as well as under the influence of 0.5% sodium hypochlorite solution.

The diagnosis is made by a complex method based on the analysis of epizootological data, the clinical picture of the disease and pathomorphological changes.

Epizootological data. Sheep and goats at the age of 15 months to 11 years, more often at the age of 2-4.5 years, get sick with scrapie. In ewes, the disease is detected during the period of lactation. More sensitive are goats, which are sometimes used for bio-testing. In the experiment, monkeys, minks, hamsters, and mice are easily infected by intracerebral, intraabdominal, intramuscular, intradermal, subcutaneous, and oral administration of infectious material. The disease is more often observed among improved breeds of sheep. The source of the pathogen is sick sheep or goats. Infection occurs by contact, as well as in pastures and in premises where cases of scrapie have previously been registered. The possibility of intrauterine infection is not excluded. Appears sporadically or in the form of minor enzootics. The gradual and constant

release of new sick animals is characteristic. The morbidity does not exceed 20%. The disease always ends fatally.

Clinical signs and course of the disease. The incubation period lasts from several months to a year. Infection occurs in utero or at an early age, signs of the disease appear much later. Early symptoms of the disease are itching, scratching of the skin, hair loss (Fig. 1).

Fig. 1. Formation of alopecia due to scratching
(https://fermer.ru/forum/veterinariya-ovets/66911)

Sick sheep rub against the surface of fences, trees, poles, scratch the skin of the head, neck, back with their hind limbs, damage it with their horns. In some animals, unusual behavior is noted - stomping on the spot, grinding teeth, restless, wary look, stupor (stupor-like state). Body temperature is normal. Over time, the animal loses its appetite, refuses feed and water, and loses weight quickly. The coordination of movements is disturbed, there is weakness of the limbs, more often the pelvic ones, falling while walking. The disease progresses slowly and, despite the absence of paralysis, the animal completely loses the ability to move, lies on its side with outstretched limbs, and dies in a state of stupor. In some animals, appetite and fatness remain throughout the disease.

Pathological anatomical changes. At autopsy, no characteristic pathological changes were found. Therefore, the results of histological studies are of decisive diagnostic value.

Pathomorphological changes. Localized mainly in the central nervous system. The medulla oblongata and midbrain, cerebellum are more often affected. Dystrophic and necrotic (non-inflammatory) lesions such as vacuolar dystrophy and nerve cell lysis, as well as vacuolization, hypertrophy, and proliferation of astrocytic glia are considered characteristic. Individual and numerous vacuoles of various sizes and shapes are observed in affected neurons and their processes, occupying almost the entire cell. As a result of the vacuolization of neurons and astrocytes, the gray matter acquires a characteristic spongy appearance, which is especially clearly expressed in sick goats.

The results of the pathomorphological examination of the brain are of decisive importance in making a diagnosis. The brains of animals immediately after removal from the cranial cavity are fixed in a specially prepared 10% saline solution of formalin and delivered to the laboratory. In the laboratory, fixation of the brain is continued for 14 days, replacing the fixing liquid after one week. Then, pieces 0.3-0.5 cm thick are cut from the medulla oblongata, varolius pons, and quadrituberous body, additionally fixed for 24 hours in the same saline solution of formalin, embedded in paraffin or celloidin, after which histological sections are prepared, which are stained with hematoxylin-eosin.

The diagnosis on the scraping is considered established when at least 15 vacuolated nerve cells are detected in one or at least 20 in two or three histopreparations, as well as neurons in a state of pyknosis, sclerosis and lysis, with significant vacuolization and thinning of the gray matter of the brain, hypertrophy and proliferation of astrocytes, in the absence of inflammatory phenomena.

Differential diagnosis. Presupposes the need to exclude Aujeski's disease, visna-maedi, rabies, the nervous form of listeriosis, and in the presence of itching - trichophytosis, scabies, streptotrichosis, demodecosis.

Aujeski's disease affects animals of all species, is characterized by high contagiousness, fever, acute course, rapid development of clinical symptoms. Biotesting on rabbits makes it possible to quickly and accurately establish the correct diagnosis. In visna, skeletal muscle paralysis, interstitial pneumonia, lymphocytic demyelinating meningoencephalitis are detected, there is no scratching, vacuolization and necrosis of neurons. The course of listeriosis is acute, sheep of all age groups get sick, especially lambs. Histological studies reveal purulent encephalitis, bacteriological - isolate the causative agent of listeriosis. Trchophytia, streptotrichosis, demodicosis, scabies are limited to damage only to the skin. Mycological studies ensure the isolation of the causative agent of the corresponding disease.

Treatment. Not conducted. Sick and suspected animal diseases are destroyed.

Immunity. Not studied. It is not possible to detect specific antibodies in sick animals with the help of existing serological reactions. A vaccine has not been developed.

Prevention and control measures. In scrapie-free states, precautionary measures are taken against the introduction of the pathogen from regions where this disease is registered. Such measures are carried out by specialists of the regional services of state veterinary control at the border and transport. In order to prevent the appearance of disease in healthy farms, it is necessary to first of all observe the zoo-veterinary rules for stocking farms with small cattle, as well as the rules for keeping, feeding, grazing, watering animals and caring for them. All newly arrived animals are quarantined for 30 days, and their clinical examination is carried out. Pastures and watering holes are kept in proper veterinary and sanitary condition. At the appearance of scrapie, all sheep (goats) of a dysfunctional flock (herd) are immediately slaughtered. Take the measures

prescribed by the relevant instructional documents to prevent the spread of infection. In permanently unfavorable farms with regard to scrapie, the most effective is the slaughter of all animals of the unfavorable farm and a complete replacement of livestock.

Control questions and tasks.

1. The causative agent of scrapie and its characteristics.
2. Epizootological features of scrapie.
3. Clinical signs and course of the disease.
4. Characteristic features of pathomorphological changes in scraping.
5. When is the diagnosis of the disease considered established?
6. From what diseases is it necessary to differentiate scrapie?
7. How are healthy and sick animals treated with scrapie in a dysfunctional household?
8. Prevention and control measures.

Topic: Malignant edema

(diagnosis, prevention, control measures).

Malignant edema (Oedema malignum, septicemia gangrenosa, wound gas edema, gas gangrene) is an acute non-contagious wound toxic infection of animals and humans, which is caused by a group of pathogenic clostridia and is characterized by inflammatory edema with the formation of gases, necrosis of affected tissues and intoxication of the body.

Malignant edema is registered in animals in the form of sporadic cases, sometimes - small outbreaks, it is found everywhere.

Pathogens of the disease. Malignant edema is a disease of polymicrobial etiology. The main role in the development of the infectious process is played by the following types of bacteria from the genus Clostridium: C. septicum, C. novyi, C. perfringens, C. chauvoei (important only in sheep), C. hystoliticum and C. sordellii. The disease is caused by each of these clostridia, as well as their associations. Other microorganisms do not cause disease by themselves, but provide more favorable conditions for the active reproduction of pathogens.

There are infectious and toxic stages of the disease. In the infectious stage, there is increased reproduction of microorganisms in the lesion (edema formation and gas infiltration of the affected tissues) and their rapid spread throughout the body with blood in an agony state. The toxic stage develops as a result of the action of toxins that spread in the tissues and cause the death of the body from toxemia. Lesions are more often observed in areas of the body rich in muscle tissue - croup, thigh, shoulder blade, back, neck, lower leg. Spores of the causative agents of the disease, having entered the damaged tissue, in the presence of anaerobic conditions, germinate, reproduce

intensively, releasing toxins. As a result of intoxication, the central nervous system, respiratory center are affected, cardiac activity is disturbed and death from intoxication occurs.

The diagnosis is made by a complex method based on the analysis of anamnestic data, clinical signs, patho-anatomical changes and the results of laboratory tests.

Epizootological data. Sheep, horses, mules, donkeys, cattle, pigs, deer are susceptible to malignant edema. Humans, carnivores, birds, dogs and cats rarely get sick. Cases of disease in other animal species have been registered. Horses and sheep are most susceptible.

The source of the causative agent of the infection is sick animals, which release the causative agent of the disease into the external environment with feces. The gates of infection are wounds and damage. The disease is registered in the form of sporadic cases after injuries, surgical interventions, castration, haircuts, trimming of tails, carried out without observing the rules of asepsis and antiseptics. The occurrence of the disease is facilitated by difficult births, delayed litter, prolapse of the uterus, introduction of infection during the provision of obstetric care.

Clinical signs and course of the disease. The duration of the incubation period depends on the causative agent and localization of the pathological process and ranges from 12 hours to several days. The course of the disease is acute, especially in sheep and horses. Malignant swelling lasts from 1 to 3-4, sometimes up to 7 days. The clinical manifestation and course of the disease depend on the type of animal, the type and toxicity of the pathogen or their association, the nature and localization of the lesions.

There are post-wound malignant edema, postpartum malignant edema, malignant edema of lamb's rennet, malignant edema of the head, etc.

Clinical signs: severe depression, refusal to feed, acceleration of the pulse, difficulty breathing, bluishness of the mucous membranes, body temperature is most often increased (but can be within the normal range) by 1-2°C, before death it decreases. In the absence of treatment, animals die within a few days. Most often, the emphysematous form of the disease is registered, which is characterized by the rapid development of gas infiltration of the affected tissues, the rapid increase in the area of the affected area (Fig. 1).

During palpation, crepitation is noted, when pressed, exudate with gas bubbles is released from the wound. In this form of the disease, the causative agent is more often S. perfringens. Edema-toxic form is characterized by the rapid development of edema of the affected tissues, processes in the muscles and gas formation are absent or weakly expressed. Signs of general intoxication of the body appear very quickly. S. novyi and S. septicum are isolated from the pathological material. The tissue-melting form is characterized by sharply expressed processes of tissue melting with bone exposure. Swelling and gas formation are weakly expressed. This form of malignant edema is very rare, it is caused by S. histolyticum. A mixed form is more common.

Pathological anatomical changes. Most of the corpses are bloated and decompose quickly. The connective tissue in the area of edema is soaked with a yellow and red liquid, sometimes hemolyzed, containing gas bubbles, with a rancid or putrid smell (Fig. 2).

The type of affected muscles and the amount of gas in the connective tissue depend on the type of pathogen. When animals die as a result of infection with S. novyi, gelatinous, colorless or cream-colored swelling is revealed. Lesions caused by S. histolyticum infection are quite characteristic: decay of all tissues from skin to bones, bloody exudate. The muscles are dark brown in color, easily torn, soft, juicy, difficult to cut. If the inflammatory process is caused by S. septicum, the muscles are red; if S. Perfringens - the muscles seem to be boiled, greenish in color, with a large number of gas bubbles; if S. novyi or S. Sordellii – the muscles are gelatinous, the swelling is gel-like. In case of mixed infection with the presence of putrefactive aerobes or anaerobes, the affected tissue has a gray, brown and bluish color. The chest and abdominal cavities contain bloody exudate. Changes in internal organs are atypical.

Fig. 1. Clinical signs of malignant edema

Fig. 2. Bubbles with gas in the connective tissue (https://www.fao.org/3/t0756e/T0756E06.htm)

Laboratory diagnostics. It involves microscopy of smears, bacteriological examination, bioassay on laboratory animals (guinea pigs) and neutralization reaction with specific sera to determine the type of pathogens.

Differential diagnosis. Presupposes the need to exclude the carbuncular form of anthrax and emphysematous carbuncle of cattle.

Treatment. Since the inflammatory process develops extremely quickly, the success of treatment mainly depends on how timely help is provided. The sick animal is isolated, provided with good food and abundant drinking water. Deep longitudinal incisions are made in the skin, subcutaneous tissue and affected muscles in the area of

edema, open the wound pockets and remove necrotized areas of tissue. After surgical treatment, the wound is abundantly irrigated with antiseptic solutions.

The basis of treatment is the use of antibiotics and sulfonamides locally and parenterally in large doses. In case of malignant edema of the uterus, along with antibiotic therapy, intrauterine administration of solutions of antibacterial and disinfectant substances is recommended. In some cases, antitoxic serum helps against anaerobic enterotoxemia and anaerobic dysentery of lambs (in the presence of the causative agent S. perfringens).

Immunity. Immunity in malignant edema is antitoxic. Due to the fact that malignant edema is a disease of polymicrobial etiology and occurs sporadically, means of its active prevention are not routinely used. However, polyanatoxin against sheep clostridiosis and polyvalent vaccine against brazot, enterotoxemia, malignant edema and dysentery of lambs can be successfully used.

Prevention and control measures. Preventive measures for malignant edema of animals are reduced mainly to strict asepsis during operations and timely treatment of wounds. For the prevention of obstetric malignant edema, it is necessary to observe sanitary and hygienic conditions during calving. Observance of sanitary and hygienic conditions during childbirth and the provision of obstetric care is of great importance. In hospital-affected areas, it is recommended to administer polyvalent antitoxic serum in combination with antibiotics to animals before mass processing of livestock, births associated with the possibility of injury.

When cases of malignant edema appear in the farm, restrictions are not imposed on the farm, but sick animals are isolated and treated. The premises in which the sick animals were kept are cleaned and disinfected with a 3% hot solution of sodium hydroxide, a 5% formalin solution, a 3-5% solution of a sulfur-carbolic mixture or a suspension of chlorinated lime with a content of at least 5% active chlorine. Infected manure is burned. The carcasses of the dead animals are sent to a waste plant or burned on the spot. Slaughter for the meat of sick and suspected animals and skinning are prohibited.

Control questions and tasks.

1. What types of bacteria from the genus Clostridium can be the causative agent of malignant edema?
2. Which distinguish stages of disease development and their characteristics.
3. Epizootological data of malignant edema.
4. Forms of clinical manifestation of malignant edema and their characteristics.
5. Pathological-anatomical changes in malignant edema.
6. Diagnosis of malignant edema.
7. Treatment of animals with malignant edema.
8. Prevention and control measures.

DISEASES OF PIGS

Topic: Classical swine fever

(diagnosis, prevention and control measures).

Classical swine fever (Pestis suum, European swine fever, CSF) is a highly contagious disease caused by a virus. In an acute course, it is characterized by hemorrhagic processes in parenchymal organs, serous and mucous membranes, and in a prolonged one, it is complicated by croupous and croupous-diphtheric inflammation of the lungs and large intestine.

The causative agent of the disease. RNA-genomic, pantropic virus from the family Togaviridae, genus Pestivirus, pathogenic only for pigs. Laboratory animals are immune to the virus. Adapts to the body of rabbits. It has a pantropic effect, i.e. it is able to accumulate in erythrocytes, spleen, lymph nodes, bone marrow, liver, endothelium of blood vessels.

The virulence of the swine fever virus outside the body is not constant and depends on the influence of various physical and chemical factors. In particular, in affected yards, rooms in dried substrates can be stored for a year. It is preserved for more than a year in blood preserved with a 0.5% phenol solution. The virus is stored for a long time at low temperatures: in chilled meat - 2-4 months; in frozen meat - several years; in smoked products - 1-1.5 months; in corned beef - 315 days; in salted and dried intestinal raw materials - 3-6 months.

The diagnosis of swine fever is established on the basis of a complex of: epizootological, clinical, hematological, patho-anatomical data.

Epizootological data. Only domestic and wild pigs are susceptible to the plague, regardless of breed and age. The source of the causative agent of the disease is sick pigs, which, starting from the incubation period and for 10 months after the illness, secrete the virus with all secretions and excreta, especially with urine, feces, conjunctival secretion, nasal mucus. The source of the pathogen can also be insufficiently immune pigs that have been vaccinated with inactivated vaccines and when in contact with the field, wild plague virus become chronically ill and virus carriers. Factors of transmission of the pathogen can be corpses of pigs killed by plague, meat of forcibly slaughtered animals, undamaged slaughterhouse and kitchen waste. The virus can spread through feed, water, care items, equipment, shoes and clothing of

people, vehicles that have been contaminated by secretions of sick pigs or virus carriers. Mechanical transmission of the virus by insects, birds, domestic animals, rodents, and humans is possible. Uncontrolled trade in pigs and their slaughter products on the markets, as well as the importation of virus-carrying breeding pigs from farms that are unfavorable in terms of plague, are often the cause of swine plague in healthy farms.

Infection occurs through direct contact of healthy pigs with sick ones, alimentary and aerogenous routes, through the mucous membrane of the nose, digestive tract, through the conjunctiva of the eyes and damaged skin. Epizootological features of classical swine fever are characterized by the lack of seasonality, the gradual spread of infection, the mass coverage of the disease in pigs of all age groups, and high mortality regardless of the age of the animals. A characteristic feature of classical swine fever in Europe is that, along with individual outbreaks, persistent infection with a different course is recorded, as well as the circulation of the virus among wild pigs.

Clinical signs and course of the disease. The incubation period lasts 2-9 days. The course of the disease is hyperacute, acute, subacute and chronic.

The hyperacute course is rarely registered and only among young children. It is characterized by rapid fluidity, high body temperature, sharp depression, complete lack of appetite, vomiting, severe cardiac and respiratory disorders, the appearance of bright red spots on the skin.

An acute course is recorded at the beginning of a plague outbreak. The disease begins with an increase in body temperature, which is maintained for 7-8 days, and before death it drops to 35-36°C. Appetite remains at first, worsens for 2-3 days, and then disappears completely. There is vomiting, constipation, which is later replaced by diarrhea, sometimes with an admixture of blood. Urinary excretion is difficult, in some animals the urine acquires a dark brown color. The conjunctiva turns red, the eyelids swell and stick together with mucous-purulent secretion. Sick pigs lie more, buried in litter, reluctant to get up, their gait becomes shaky. In some animals, convulsions, paresis or paralysis of the hind limbs are observed. Abortions occur in farrowing sows. On the 5-9th day of the disease, small hemorrhages appear in the skin, which later merge and form diffuse dark purple spots that do not disappear when pressed (unlike hysteria). Bluishness of the skin in the area of the ears, tail, heel, and belly is revealed. Some animals have nosebleedsAn early sign of the acute course of the plague is leukopenia, which reaches its maximum development on the 4th-6th day of the disease, when the number of leukocytes decreases to 1-3 thousand in 1 mm3. Sick animals die on the 7-12th day of illness.

The subacute course of the disease lasts up to 3 weeks. In sick animals, there is a periodic increase in body temperature, constipation that alternates with diarrhea, loss of appetite, exhaustion, and eczematous skin lesions. Secondary infections such as salmonellosis or pasteurellosis are often complications of the disease. In cases of complications of plague with salmonellosis, progressive exhaustion of the animal, foul-

smelling diarrhea with impurities of mucus and blood are revealed. The back of sick piglets is bent, the stomach is pulled up, the tail is contaminated with fecal masses, hangs down, the skin is dry and covered with eczematous crusts. When the plague is complicated by pasteurellosis, severe exhaustion, difficulty breathing, cough, mucous-purulent discharge from the nose, and pneumonia are detected. Sick animals try to lie on their chests, to facilitate breathing and reduce the pain of the pleura, they assume the posture of a sitting dog.

The chronic course of the disease lasts several weeks and even months. Decreased appetite, progressive weight loss, diarrhea, constipation, conjunctivitis, eczematous lesions, death and loss of tail and ear tips are observed. When complicated by salmonellosis or pasteurellosis, the symptom complex of the disease is supplemented by damage to the alimentary canal or lungs, respectively. Sick pigs die or are culled.

Pathological anatomical changes. They depend on the severity of the course of the disease and the nature and localization of complications.

The acute form of classical plague is characterized by a pronounced hemorrhagic diathesis. Upon external examination of the corpses in the area of the ears, neck, abdominal wall and inner surface of the thighs, dark red coloration of the skin, hemorrhages of various shapes and sizes, from single to multiple, covering the entire surface of the skin are observed. When dissecting corpses, hemorrhages of various sizes and shapes are found in all organs and tissues (Fig. 1).

Fig.1. Hemorrhages on the surface of the skin and internal organs.
(https://vetmarket.ltd/info/disease/kchs_klasichna_chuma_sviney/)

Lymph nodes in the area of the head, neck, mediastinum and mesentery are enlarged, flaccid, with a marble pattern on the cut surface. The spleen is not enlarged, grayish-steel color, with infarcts on its edges, which have the appearance of dense black-red elevations, wedge-shaped on section. The kidneys are anemic, covered with

small hemorrhages under the capsule, the cortical and medullary layers are smoothed. Hemorrhages are also found on the mucous membrane of the bladder. The liver is not enlarged, sometimes the phenomena of stagnant hyperemia and degeneration of the parenchyma are observed in it. The lungs are mottled, with isolated hemorrhages on the surface. The brain and spinal cord, as well as the meninges are swollen, hyperemic, covered with small hemorrhages. Acute catarrhal inflammation, hemorrhages, and hyperplasia of Peyer's patches are found in the stomach and intestines. Solitary follicles are swollen, clearly protrude above the surface of the mucous membrane of the colon.

The subacute and chronic course of the disease when complicated by pasteurellosis is accompanied by pneumonia, pleurisy, as well as splenic infarctions, hemorrhages in the intestines, swelling and ulcerative lesions of the follicles of the large intestine. In case of complications of salmonellosis, croupous-hemorrhagic enteritis is observed. With a chronic course in the cecum and colon, ulcerative-necrotic lesions are detected at the site of solitary follicles. The mucous membrane is covered with rounded, grayish-yellow diphtheritic ulcers with cheesy contents. In the large intestine, diffuse diphtheritic inflammation of the mucous membrane with bran-like grayish-yellow layers is quite often observed.

Laboratory diagnostics. Includes detection of viral antigen by immunofluorescence reaction, indirect hemagglutination reaction with erythrocyte diagnostics, diffuse precipitation reaction, virus isolation in primary cultures of kidneys and testicles of piglets or transplantable line RK-15, bioassay on 5 piglets 2-3 months old. Retrospective diagnosis of plague is carried out by detecting specific antibodies in the blood of sick pigs by the diffuse precipitation reaction, the indirect hemagglutination reaction, the immunofluorescence reaction (indirect method), the neutralization reaction and the ELISA method.

Blood samples, pieces of spleen, breast tissue, and lymph nodes are sent to the laboratory for research, which are taken in the first 2 hours after the death or slaughter of the animals. For a hematological examination, blood from the ear veins is taken into tubes with an anticoagulant (10% trilon solution at the rate of one drop per 1 ml of blood). The brain is sent for histological examination. Pathological material is delivered to the laboratory in a thermos with ice with accompanying documents.

Differential diagnosis. Prescribes the need to exclude African swine fever, pasteurellosis, erysipelas, salmonellosis, Aujesky's disease and swine flu.

In African swine fever, hemorrhagic diathesis symptoms are much more pronounced, the spleen is enlarged and softened, but it almost never has heart attacks. Kidneys and other parenchymal organs are filled with blood, lymph nodes, especially internal ones, have the appearance of blood clots, while in classic swine fever the external lymph nodes (submandibular, parotid, oropharyngeal) are primarily affected. Characteristic for the African plague is serous hepatitis with severe swelling of the gallbladder, serous-hemorrhagic pneumonia with sharp infiltration of the interlobular

connective tissue. With African plague, a significant accumulation of bloody fluid in the chest cavity, gelatinous edema.

Pasteurellosis never becomes as widespread as plague, occurs in the form of sporadic cases and minor enzootics, affects mainly adult pigs. With pasteurellosis, there are no hemorrhages on the skin, infarctions of the spleen, and marbling of the lymph nodes. In the area of the head, neck, chest, and submaxillary space, significant serous edema of the subcutaneous tissue is found.

Erysipelas is characterized by a very rapid development of the disease, high body temperature (up to 42°C), the appearance on the skin of the back and sides of crimson-red or dark-purple hyperemic spots of various sizes and shapes, which turn pale when pressed. The autopsy revealed an enlarged spleen, dystrophic changes in the parenchymal organs. During the chronic course of erysipelas, skin necrosis, arthritis, and endocarditis are observed.

Salmonellosis is registered more often in weaned piglets. The disease is characterized by exhausting diarrhea, an eczematous rash on the skin, and the formation of diphtheritic scaly, loose scabs in the colon. With salmonellosis, there are no hemorrhages, "marbling" of lymph nodes, or splenic infarctions.

Aujeski's disease mainly affects suckling piglets and piglets of weaning age, in adult pigs the course is mostly benign. In piglets, characteristic nervous phenomena and high mortality are observed; there are no hemorrhages in the skin. Infection of rabbits with pathological material from dead piglets causes typical clinical signs of scratching and itching only in Aujeski's disease.

Swine flu is benign. The disease is excluded by virological studies of pathological material.

Treatment. Not conducted. Sick pigs are slaughtered.

Immunity. Pigs that have been infected with the plague get permanent immunity for life. In the world, vaccines from various manufacturers are recommended for active immunization. Vaccines are administered intramuscularly in the thigh of pigs from the age of 3 months. Immunity occurs 5-7 days after vaccination and lasts for 1 year.

Prevention and control measures. In order to prevent the introduction of the plague, strict control over the equipment of the pig farm and the subsequent maintenance of pigs in accordance with veterinary and sanitary regulations are established. Farms are equipped with healthy animals only from prosperous farms. It is forbidden to have any contact with unfavorable and threatening points regarding swine fever; stocking pig farms with livestock from farms where food and slaughterhouse waste are used for feeding animals; use of non-contaminated food and slaughterhouse waste for feeding pigs; entering the territory of the pig farm by transport and visiting citizens not related to animal care. It is necessary to constantly carry out veterinary supervision of the population of wild pigs with timely detection of corpses and mandatory virological examination of them. It is necessary to systematically record and register foci of classical plague among wild pigs. In individual farms, pigs should

be kept in private yards and not use pastures intended for pig farms. Preventive vaccination of pigs against plague is carried out in farms in accordance with regional plans of anti-epizootic measures, regardless of the forms of ownership.

When the disease occurs, the farm (settlement) is declared unhealthy for classical swine fever, quarantine is immediately established, and the threatened territory is determined. According to the terms of the quarantine, it is prohibited to export, import and regroup pigs, export raw products of pig slaughter, departure of all types of transport from the quarantined territory, exit of service personnel in work clothes and shoes, holding of exhibitions, fairs, excursions, as well as trade within the quarantined and threatened territory pigs and raw slaughter products.

In the quarantined territory, security and quarantine posts with barriers, death barriers and paraformalin chambers are set up, people are on duty 24 hours a day, and they are provided with special clothes and special shoes. Signs "Quarantine. ", as well as signs informing about detours of the quarantined territory. At the entrance to livestock premises, tanks with disinfectant solution are installed, the work of sanitary passes is organized, and the daily processing of work clothes and special shoes in the paraformalin chamber is organized. All pig herds of dysfunctional farms are slaughtered at a sanitary slaughterhouse or in the slaughterhouses of a meat processing plant or on the farm at specially equipped slaughterhouses in compliance with the relevant veterinary and sanitary requirements. All pigs in safe premises, as well as pig herds in the threatened area, are vaccinated against plague, regardless of the dates of previous vaccination. Premises where sick and suspected cases of swine fever were located, as well as workshops of meat processing plants and slaughterhouses where infected animals are slaughtered, are subjected to mechanical cleaning and disinfection. Pig carcasses in a dysfunctional cell are destroyed by burning or subjected to technical disposal and processing into meat and bone meal. When the plague appears among wild pigs, they are shot.

Quarantine from a swine plague-prone point is lifted 30 days after the last case of disease, death or slaughter of sick pigs, provided that all prescribed veterinary and sanitary measures are carried out. In the future, the placement of pigs on the territory of former dysfunctional farms is carried out with the written permission of the chief state inspector of veterinary medicine of the region. Disinfection of the premises in a point affected by the plague is carried out every 5 days until the quarantine is lifted, and machines - after each case of isolation of sick or suspected pig disease. 2-3% hot solutions of caustic soda, 20% suspension of freshly slaked lime, clarified solution of chlorinated lime containing 2% active chlorine, 3% hot solution of sulfur-carbolic acid are used for disinfection of premises, equipment and inventory, walking yards, animal care items mixtures, 2% formaldehyde solution. Manure is disinfected by the biothermal method.

Control questions and tasks.

1. Describe the causative agent of classical swine fever.

2. Describe the epizootological features of classical swine fever.

3. What characteristic patho-anatomical changes are observed in classical swine fever?

4. From what diseases and on the basis of what data should classical swine fever be differentiated?

5. By what indicators is the diagnosis of classical swine fever considered established?

6. Name the general and specific measures for the prevention of classical swine fever in the farm.

7. What measures to eliminate classical swine fever are carried out in dysfunctional farms?

Topic: African swine fever

(diagnosis, prevention and control measures).

African swine fever (Pestis africana suum, ASF, Montgomery disease) is a highly contagious disease of pigs, characterized by fever, hemorrhagic diathesis, significant hemorrhages, dystrophic-necrotic changes in internal organs, extremely high mortality.

The causative agent of the disease. Cytoplasmic DNA virus, which was allocated to a separate family of Asfarviridae (african swine fever and related viruses), genus Asfivirus in 2000. The ASFV virus in pigs multiplies in monocytes, macrophages, neutrophils, endothelial cells, hepatocytes and epithelial cells of renal tubules. Also reproduces in ticks of the genus Ornithodorus for a long time (perhaps lifelong) and is transmitted sexually, transovarially and transstadially.

In the body of a sick animal, the virus accumulates in all organs. Cultivation of the virus is possible in the culture of bone marrow cells and leukocytes. When it is propagated in cultures of leukocytes or bone marrow cells of pigs, the phenomenon of erythrocyte adsorption on the surface of the affected cells is observed. The African swine fever virus is highly resistant: it can persist in the soil for up to 180 days; on wood and bricks - 120-180 days; in meat - 5-6 months; in the bone marrow - 6-7 months; in feces up to 160 days; in urine - 60 days; in carcasses from 17 days to 10 weeks, in premises after removal of sick pigs - at least 3 weeks. It has increased resistance to alkalis and formalin, but is sensitive to acids. It is advisable to use chlorine-containing preparations for disinfection.

The diagnosis of African swine fever is made by a complex method based on the analysis of epizootic and clinical data, patho-anatomical changes and the results of laboratory studies.

Epizootological data. Under natural conditions, wild African pigs (warthogs, river hogs, large forest hogs) and domestic pigs, regardless of breed and age, are sick.

In wild African pigs, the course of ASF is mostly latent, and their infection can only be established by bioassays on domestic pigs. The main reservoir and source of the pathogen of ASF in nature are wild virus-carrying pigs, contact with which leads to infection of domestic pigs. In stationary ASF-affected countries, the permanent reservoir and carrier of the virus are argas ticks of the genus Ornithodoros, in whose bodies the virus can persist for many years and even be transmitted to offspring transovarially.

The source of the pathogen can be domestic pigs that are sick with ASF. The ASF virus is released from the body of infected pigs with all secretions and excreta - saliva, urine, feces, semen, discharges from the eyes and nasal cavity. Factors of transmission of the pathogen can be various objects of the external environment - feed, water, manure, bedding, animal care items, clothes of service personnel, transport that were contaminated with the virus, as well as the corpses of pigs killed by plague. Especially dangerous are infected slaughter products, slaughterhouse and kitchen waste, which have repeatedly caused outbreaks of ASF in prosperous countries after the import of pigs and African livestock products (skins, meat, bristles). Mechanical carriers of the virus can be birds, rodents, insects, wild and domestic animals that are not sensitive to it, as well as people in an epizootic center. Infection occurs during direct contact of healthy pigs with infected ones, as well as through alimentary, aerogenous routes, through damaged skin and conjunctiva of the eyes. African swine fever occurs in the form of devastating epizootics, characterized by extremely high contagion, gradual, slow development of the epizootic process, almost 98-100% morbidity and mortality. In stationary unhealthy centers, the frequency of mass outbreaks of the disease is established every 2-4 or 5-6 years.

Clinical signs. The incubation period lasts 2-6 days. The disease has a hyperacute, acute, subacute, chronic and latent course, which is primarily due to the virulence of the virus and the presence of relative immunity in African pigs in natural foci of the disease.

During the *hyperacute course*, which is rare, sick animals die suddenly, without the manifestation of clinical signs characteristic of this disease.

During the *acute course*, which is registered most often, 4 periods are distinguished in the development of the disease: the first - incubation, the second - fibrillary (increased body temperature), the third - the development of the main symptoms of the disease, the fourth - coma, hypothermia, death. The incubation period lasts 5-9 days. In the next 3 days, a high body temperature of 41-42°C, sometimes increased excitability, serous conjunctivitis, swelling of the eyelids is noted. On the 4th day after an increase in body temperature, characteristic symptoms of the disease appear - cyanosis of the skin in the area of the submaxillary space, abdomen, chest, scrotum, on the ears, heel, limbs, hemorrhagic conjunctivitis, rhinitis with serous-

hemorrhagic discharge, anorexia, vomiting, constipation or diarrhea, sometimes with bleeding, nervous phenomena, unsteady gait, paresis and paralysis of the hind limbs. Most animals develop inflammation of the lungs, which is accompanied by heavy breathing and coughing. Pregnant sows abort.

In the *subacute course*, the incubation period is 5-9 days. The same symptoms of the disease as during the acute course are observed, but they are less pronounced and develop much more slowly. In many animals, exhaustion, complications with secondary bacterial microflora are observed. The duration of the disease is 15-25 days. It is registered in piglets, ends mostly fatally.

The *chronic course* is often a continuation of acute and subacute cases of the course of the disease. Symptoms of the disease are expressed vaguely and uncharacteristically. Intermittent shortness of breath, shortness of breath, cough, progressive weight loss, arthritis, skin ulcers are noted. Most patients die within 30-90 days.

The *latent course* is observed in wild African pigs, sometimes in domestic pigs at the end of an epizootic or in case of infection of animals immunized with attenuated strains of the virus. In these cases, there are no clinical signs of the disease, but the animal becomes a virus carrier and is a dangerous source of the pathogen for healthy pigs.

Pathological anatomical changes. They are characterized by the manifestation of hemorrhagic diathesis and damage to lymphoid organs. The autopsy revealed numerous hemorrhages on the mucous and serous membranes and in the organs of the abdominal and thoracic cavities. They are especially clearly expressed in adult pigs with a subacute and acute course of the disease. The skin in the area of the chest, the ventral part of the intestinal walls, the inner surface of the thighs is red or crimson-purple in color, blood or bloody fluid is released from the anus and nose.

Blood vessels of the subcutaneous tissue, organs of the abdominal cavity and mesentery are full of blood that does not clot in air, hemorrhages often occur along the course of the vessels. In the chest, abdominal and pericardial cavities, there is an accumulation of a significant amount of yellowish-red exudate, often with fibrin clots (Fig. 1).

The spleen is greatly enlarged, the pulp is flaccid, softened, dark red in color, full of blood, and gives large scrapings from the surface (Fig. 2). Swelling of the lungs, their gray-red color, as well as gelatinous swelling of the interlobular connective tissue and parenchyma, characteristic of serous-hemorrhagic pneumonia, are revealed. The kidneys are enlarged, full of blood, covered with numerous dotted hemorrhages.

The mucous membrane of the alimentary canal is hemorrhagically inflamed, with hemorrhages resembling hematomas. Gelatinous swelling of the submucosa of the cecum is determined. In the subacute course of the disease, serous-fibrinous pericarditis and numerous hemorrhages are often observed. In the chronic course, necrotic lesions of the skin, hepatitis are detected. They also note a sharp increase in

bronchial lymph nodes, lung damage. During the latent course of the disease, marbling of the portal and bronchial lymph nodes, focal lesions of the lungs are observed.

The gallbladder is filled with thick bile with blood impurities, its walls are significantly thickened due to swelling and expansion of blood vessels. Dotted or striped hemorrhages are observed under the epicardium and endocardium. Lymph nodes, especially of the stomach, liver, kidneys, and mesentery, are significantly enlarged, filled with blood, resembling blood clots or hematomas (Fig. 3).

Fig. 1. Hydropericardium and multiple epicardial hemorrhages.

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Fig. 2. Splenomegaly (above) is a pathognomonic sign of ASF (below, for comparison, the spleen in CSF)

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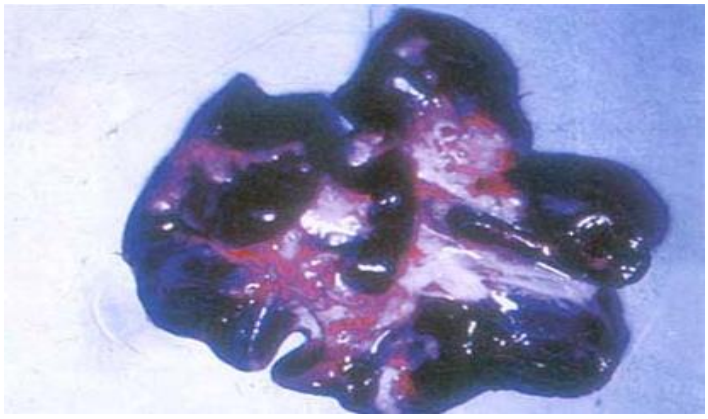

Fig. 3. Enlarged and hemorrhagic mesenteric lymph nodes.

Laboratory diagnostics. Laboratory diagnosis of ASF is carried out by specialized laboratories of veterinary medicine for particularly dangerous infectious diseases of animals or research institutions that have accreditation for work with pathogens of particularly dangerous infections.

Differential diagnosis. Presupposes the need to exclude classical swine fever, erysipelas, pasteurellosis, salmonellosis.

With classic swine fever, the entire symptom complex of the disease develops simultaneously with an increase in body temperature, with African swine fever, fever appears more often at the end of the disease. In the first case, the external lymph nodes are affected, in the second, the lymph nodes of the internal organs. With classical swine fever, marbling of the lymph nodes (without their sharp increase), infarctions on the edges of the spleen, anemia of the kidneys with small hemorrhages in the cortical layer, diphtheritic inflammation of the intestines is detected. In the case of African swine fever, the phenomena of hemorrhagic diathesis, significant expansion of blood vessels, hematomas, especially in the groin, scapular area and muscles, hemorrhages of various shapes and sizes in organs and serous membranes are characteristic. Lymph nodes, especially of the stomach, liver and mesentery, are so hemorrhagic that they resemble clots of blood that have coagulated.

With erysipelas, as a rule, pigs older than 3 months are sick, their morbidity does not exceed 20-30%, and the mortality rate is 55-80%. Characteristic erythema, urticaria, serous or hemorrhagic lymphadenitis, glomerulo-nephritis, arthritis.

Pasteurellosis occurs sporadically, accompanied by swelling of the subcutaneous tissue in the area of the chest and neck, bilateral pleuropneumonia, serous lymphadenitis. Salmonellosis occurs in the form of enzootic disease, suckling piglets and weanling piglets are affected. The main pathological changes are localized in the intestines, lungs, and spleen.

Treatment. It is forbidden. Sick pigs are slaughtered by the bloodless method and burned.

Immunity. Means of specific prevention of the disease have not been developed.

Prevention and control measures. Preventive measures against the introduction of ASF into prosperous countries and zones are based on the clear implementation of general preventive measures and veterinary and sanitary rules. It is prohibited to import pigs, including wild ones, and their slaughter products from countries that are disadvantaged and at risk of ASF into the territory of Ukraine.

In case of an immediate threat of introduction of the African swine fever virus from the countries bordering Ukraine, the authorities are obliged to create a special commission to organize and monitor the implementation of measures for the prevention and elimination of the disease, to inform the population about the emergence of a danger to pig farming and measures to prevent introduction of the virus. If ASF is suspected, it should be immediately reported to the higher veterinary

authorities, as well as to the local authorities to take urgent measures. Patients and suspects of animal diseases are isolated directly in the farm in the premises where they were located, people, except for service personnel and veterinary medicine specialists, are prohibited from accessing them, measures are taken to clarify the diagnosis. It is not allowed to leave the territory of the dysfunctional economy of people; the removal of animals of all types, including poultry, from a dysfunctional farm, as well as the procurement of all types of raw products and raw materials of animal origin, the removal of fodder; trade in animals and products of animal origin in markets is prohibited; it is not allowed to hold exhibitions and other events related to the gathering of animals.

A specially created state commission conducts a thorough epizootological examination of the affected point, clinical examination of animals, autopsy of dead pigs, selection of pathological material and sending it with a courier to a specialized veterinary laboratory to confirm the diagnosis. At the same time, comprehensive measures are being organized to prevent the spread and elimination of the disease. After the diagnosis is confirmed, the relevant authorities issue a decision to declare the farm, district, or settlement unhealthy and establish a quarantine. Determine the boundaries of the epizootic center, the boundaries of the first and second dangerous zones, organize the implementation of appropriate measures for the prevention and elimination of the disease in them. For the entire period of the quarantine, the import and export of animals of all kinds, the procurement and export of raw materials and products of animal origin, plant products, the entry into the unfavorable territory of outsiders, transport, regrouping in the pig farm, trade are prohibited in the quarantined administrative areas animals and products of animal origin in markets and other places, holding fairs, exhibitions.

In the epizootic center, where there are animals sick with African plague, all pigs are destroyed by a bloodless method, the corpses of slaughtered and dead pigs, manure, feed residues, low-value equipment, old premises, wooden floors, feeders, fences are burned on the spot. In the absence of possibilities to burn corpses, they are buried in a trench filled in advance with chlorinated lime, to a depth of at least 2 m. The premises, pens and other places where pigs are kept are disinfected three times: the first time immediately after the destruction of the animals, the second time - after the removal of the wooden floor, partitions, feeders and mechanical cleaning, the third time - before the lifting of the quarantine.

In the first threatened zone, which covers the territory immediately adjacent to the epizootic center of ASF and 5-10 km deep from its borders, the purchase of pigs from the population and their slaughter are organized, and meat processing enterprises for slaughtering and processing pigs are identified, as well as sources financing.

In the second threatened zone, which covers the territory that will limit the first threatened zone in depth 100-150 km from the epizootic center, trade in pigs and pig products is prohibited in the markets, the entire pig herd is recalculated, grazing of pigs

is prohibited, veterinary health supervision is strengthened pigs in farms of all categories. They carry out the entire set of organizational and preventive measures provided for in the instructions for the second dangerous zone.

Quarantine from an ASF-unfavorable point is lifted 30 days after the destruction of all pigs in the epizootic center and slaughter in the first threatened zone, the implementation of all other instructional measures, as well as the submission of the commission's conclusions on the completeness of all measures. After the quarantine is lifted, restrictions are set for a period of 6 months.

Control questions and tasks.

1. What is the nosoarea of this disease?
2. Describe the epizootological features of African swine fever.
3. What is the differential diagnosis of classical and African swine fever based on?
4. How to diagnose ASF?
5. What measures must be taken to prevent the introduction of ASF from abroad?
6. Describe the general veterinary and sanitary, quarantine and special measures carried out in the epizootic outbreak, the first and second dangerous zones.

Topic: Erysipelas in pigs

(diagnosis, prevention and control measures).

Erysipelas in pigs (Erysipelas suum) is an infectious disease of pigs 3-12 months old, characterized in the acute course by septicemia and inflammatory erythema of the skin, in the chronic course by endocarditis, polyarthritis and necrotic skin lesions. People can get sick with erysipelas.

Erysipelas is an extremely common disease and occurs almost everywhere where pigs are raised. The economic losses caused by swine erysipelas were significant in the past and were mainly due to high mortality and forced slaughter of sick pigs. The widespread introduction into practice of effective vaccines and planned preventive vaccinations dramatically reduced the morbidity and mortality of pigs from erysipelas.

The causative agent of the disease. The bacterium Erysipelothrix insidiosae is a thin, straight or slightly bent rod. The biology of the pathogen is characterized by a significant range of variability in morphological, cultural and antigenic properties. It is placed singly, in pairs, in the form of small clusters. In old broth cultures, as well as in smears from the endocardium and synovial fluid of the joints, during the chronic course of the disease, it appears in the form of long intertwined threads. The bacteria are immobile, do not form spores and capsules, are gram-positive, and stain well with all aniline dyes. Cultivated in aerobic and anaerobic conditions on conventional nutrient media. Growth in meat-peptone broth is accompanied by slight turbidity followed by the formation of a precipitate, which, when shaken, rises in the form of clouds, so-

called "moiré waves". After 24-48 hours, small dew-like colonies of S-form bacteria appear on the agar, which are isolated during the septic course, and R-form - during the chronic course of the disease and in old broth cultures. Erysipelas bacteria are very common among many species of birds, insects, rodents, and arthropods. They are found in food products, rotting corpses, river silt, stagnant water bodies, soil rich in organic matter, where under favorable conditions they can even reproduce. They are found in the tonsils, intestines, and gall bladder of healthy pigs. White mice and pigeons are sensitive to the erysipelas bacteria among laboratory animals, which die 3-4 days after infection.

The causative agent of erysipelas, due to the increased content of wax-like substances in its shell, is very stable in the external environment. Retains viability in river water at 4°C - 72-86 days, in tap water - 100-108 days, in smoked products - up to 3 months, in corned beef - up to 6 months, in pig urine - 113-145 days, in soil - 32-128 days, in feces - 38-78 days, in corpses buried in the ground - up to 280 days, in rotting corpses - up to 9 months. When cooking meat in pieces 8-10 cm thick, bacteria die after 150 minutes. The bacilli is exceptionally resistant to sunlight, which inactivates it after only 12 days, i.e., longer than spore bacilli. When dried, it dies only after 1 month. At the same time, the pathogen is quickly destroyed under the influence of high temperatures (at 70°C - after 2-5 minutes, at 100°C - after a few seconds), as well as various disinfectants (2% solutions of caustic soda or formaldehyde, 10-20 % solutions of chlorine and freshly slaked lime, 3% phenol solution, etc.).

The diagnosis of erysipelas is made by a complex method on the basis of clinical and epizootological, patho-anatomical data and the results of laboratory studies.

Epizootological data. Under normal conditions, pigs 3-12 months old, sometimes lambs up to 4-8 months old and, as an exception, chickens, turkeys, pheasants, ducks, geese are sick. Sporadic cases of erysipelas in horses, cattle, dogs, reindeer, various wild animals in zoos, rodents and other animal species are described. The main source of the pathogen is sick pigs, which during the acute course of the disease emit a significant amount of virulent bacteria with urine and feces. Clinically healthy bacteriorion-bearing pigs in case of exacerbation of latent infection due to adverse conditions also become a source of the causative agent of the disease. Factors of transmission of the causative agent can be all objects of the external environment, contaminated with secretions of sick animals carrying bacteria (feed, water, equipment and inventory of walking yards, summer camps, livestock premises), undamaged products from the slaughter of sick pigs, slaughterhouse and kitchen waste, as well as carcasses of animals that died from erysipelas. Rodents, birds, flies and insectivores can play a role, primarily as mechanical carriers of the causative agent of the disease. Infection occurs through the digestive tract, less often through the skin. Erysipelas in pigs occurs as enzootic or sporadic cases, but never affects all pigs. The disease is most often observed among repair and fattening young animals, since suckling piglets have passive immunity, and adult pigs are not susceptible due to immunization. Seasonality

and stationarity are characteristic of erysipelas in pigs, which must be taken into account when drawing up plans for anti-epizootic measures and control with vaccinations.

Pathogenesis. Depending on the ways of penetration into the body, erysipelas bacteria are localized and multiply in the tonsils and solitary follicles of the intestines (in the case of oral infection) or in the lymphatic vessels of the skin and regional lymph nodes. When penetrating through the skin, they form a strong toxin that causes sensitization of the body. Over time, the pathogen destroys the protective barriers of the body, penetrates into the lymph, blood, and then into the parenchymal organs. The generalization of the infection and the accumulation of bacterial products cause a severe general condition, the development of a septic process, deep dystrophic changes in organs and tissues, degenerative processes in the heart muscles, blood vessels and capillaries, which causes significant stagnation in the skin and parenchymal organs. Heart failure, pulmonary edema and the death of the animal develop rapidly. Sick pigs and animals with latent infection develop an allergic condition.

Clinical signs and course of the disease. The incubation period is 2-5 days. There are fulminant, acute, subacute and chronic course of swine distemper, as well as white, septic, skin (urticaria) and latent forms of the disease.

Lightning (white form) course is rarely registered and only among piglets 7-10 months old, placed in cramped, hot, poorly ventilated rooms, as well as in case of severe overheating or hypothermia during transportation. Due to the absence of red spots on the skin, this form of the disease received the name "white erysipelas".

The acute course (septic form) is characterized by general septic phenomena and typical skin changes (erysipelas erythema) (Fig. 1). The disease begins with a sudden rise in body temperature to 42-42.5°C, refusal of food, severe depression, constipation, which is replaced by diarrhea, sometimes with blood, shaky gait. General weakness of the back develops, lacrimation, mucous and muco-purulent discharge from the eyes appear. Characteristic dark red spots with a bluish tint appear on the skin of the neck, which disappear when pressed. Breathing becomes hoarse, pulmonary edema develops, and the animal dies after 2-4 days.

The subacute course (cutaneous form, urticaria) lasts 10-12 days. There is an increase in body temperature up to 41°C and above, depression, weakness. After 1-2 days, on the sides, less often on other parts of the body, peculiar limited swellings of dark red color, of different sizes and shapes, dense to the touch, as well as erythematous spots like urticaria appear (Fig. 2). With urticaria, there is no bacteremia, the pathogen is localized only in the affected areas of the skin. With the appearance of edema, the body temperature decreases, the general condition improves and recovery occurs.

The chronic course develops as a continuation of the acute and subacute course and is manifested by damage to the endocardium (verrucous endocarditis), skin necrosis on the ears, tail, back, and possible damage to the joints. With a chronic course, the disease can develop for months and end with recovery or death of animals.

Pathological anatomical changes. Not always characteristic. In pigs that died as a result of the disease with an acute or subacute course, large diffuse skin lesions of dark purple color are observed in the area of the chest, neck, ears, limbs, and abdominal wall. An autopsy revealed blood filling and stagnant hyperemia in all internal organs, acute catarrhal inflammation of the small intestine, hemorrhagic lymphadenitis and glomerulonephritis. With a chronic course, warty growths on the heart valves (verrucous endocarditis), polyarthritis, and less often - skin necrosis are detected.

Fig.1. Erysipelas erythema.
(https://www.biotestlab.ua/ru/articles/vibir-vaktcini-proti-beshikhi-svinei/)

Fig.2. Urticaria. Cutaneous form of swine erysipelas.
(https://lifehacker.org.ua/roja-svinei-prichini-i-oznaki-simptomi-i-metodi-likyvannia/5/)

Laboratory diagnostics. Includes microscopic examination of smears from pathological material, cultures on nutrient media, and, if necessary, infection of laboratory animals. For research, a whole animal carcass or heart, liver, spleen, kidney and tubular bone are sent to the laboratory. In the case of suspicion of a chronic course of the disease, the heart must be sent with the vessels ligated at the base. For microscopic examination, smears-imprints are prepared from the organs and stained according to Gram. At the same time, swabs are prepared for examination by the immunofluorescence method. In the chronic course of the disease, smears are also prepared from the affected heart valves. With positive results, Gram-stained smears reveal gram-positive rods placed singly, in pairs, or in clusters. For bacteriological research, cultures are carried out from the blood of the heart, affected heart valves, kidneys, spleen, liver, bone marrow on meat peptone agar, meat peptone broth or

Hottinger broth. The crops are incubated at 37°C for 24-48 hours, and in the absence of growth - for another 24 hours. The isolated culture is identified by morphological, cultural and biochemical properties, as well as by means of an agglutination reaction with positive serum.

The bioassay is placed on two white mice, which are injected subcutaneously with a 10% suspension of organs or a 2-day agar culture of the isolated erysipelas pathogen in a dose of 0.1-0.2 ml. Infected animals are observed for 6 days. White mice die of sepsis after 2-4 days. The organs of dead mice are cultured on nutrient media for the reisolation of the erysipelas bacillus. A laboratory diagnosis of erysipelas in pigs is considered established when one of the following indicators is obtained: detection of the causative agent of erysipelas in the original pathological material (or in a mixed culture) by the method of fluorescent antibodies (without isolation of a pure culture); isolation from the pathological material of a culture with properties characteristic of the causative agent of erysipelas, if even in cultures from the original pathological material of the culture, the causative agent is not isolated. The research period is up to 7 days.

Differential diagnosis. Erysipelas in pigs must be differentiated from plague, pasteurellosis, salmonellosis, anthrax and listeriosis.

Pigs of all age groups and at any time of the year get plague. A longer and less acute course of the disease is characteristic. When the corpse is dissected, the phenomena of hemorrhagic diathesis, splenic infarcts, "marbling" of the lymph nodes, lesions of the large intestine ("plague buds") are observed. An indirect indicator can be the effectiveness of specific sero- and antibiotic therapy for erysipelas in pigs.

Pasteurellosis is accompanied by croupous pneumonia, fibrinous pleurisy, pericarditis, and hemorrhagic diathesis of the chest cavity. In the acute course of salmonellosis, hemorrhagic diathesis, spleen hyperplasia, and liver necrosis are detected. Anthrax is accompanied by angina, inflammatory swelling in the submaxillary space, listeriosis - damage to the central nervous system. In all cases, the final diagnosis is based on the results of bacteriological studies.

Treatment. They are treated with hyperimmune serum against swine erysipelas and antibiotics (penicillin, ekmonovocillin, erythromycin, oxytetracycline). Medicines are administered intramuscularly, together or separately, at the rate of serum - 1.5 ml per 1 kg of the animal's body weight, antibiotics (with the exception of drugs with prolonged action) - 2-3 thousand. Prolonged antibiotics are administered three times, with an interval of 24 hours , in a dose of 5-10 thousand units per 1 kg of body weight. Symptomatic drugs are used at the same time.

Immunity. After getting sick with erysipelas, it is long and intense. Live and inactivated vaccines are used for active immunization against swine distemper.

Prevention and control measures. For the prevention of erysipelas in pigs, the veterinary and sanitary rules and technological requirements for the collection, transportation, placement, care, feeding and veterinary care of pigs should be followed.

Equip pig farms only with healthy animals from farms safe from infectious diseases. All pig herds should be vaccinated against erysipelas in pigs starting at 2 months of age in the doses prescribed by the instructions for the use of the corresponding vaccine. Only healthy, distemper-vaccinated pigs must be imported to reproductive farms and feedlots, which must be kept in quarantine for at least 30 days. All young pigs are vaccinated and revaccinated in the appropriate terms. Mechanical cleaning, disinfection, deratization, and disinsection are regularly carried out in livestock premises. It is not allowed to feed pigs with collected food and slaughterhouse waste in an uncontaminated form. Slaughter of pigs should be carried out only at meat plants, slaughterhouses and special slaughterhouses.

When erysipelas occurs in the farm, quarantine restrictions are imposed on the export, import and regrouping of pigs, export of fodder, meat products and by-products, skins. A clinical examination and thermometry of the entire livestock are carried out. Sick and suspected pigs are isolated and treated. Clinically healthy pigs are vaccinated and veterinary supervision is established for 10 days. Machines are regularly disinfected, and the entire piggery is disinfected every 10 days. Manure is disinfected by a biothermal method. Restrictions on a dysfunctional farm are lifted 14 days after the last case of recovery of a sick animal, vaccination of the entire pig herd against distemper, thorough cleaning and final disinfection of premises, walking yards, as well as care items.

For disinfection, use: solutions of chlorinated lime with a content of active chlorine of 3% for exposure of 2 hours; 2% hot solution of caustic soda after exposure for 1 hour; 20% suspension of freshly slaked lime after exposure for 1 hour; 0.5% formaldehyde solution for exposure for 1 hour; 5% hot solution of soda ash after exposure for 3 hours; 5% solution of iodine chloride (at the rate of 0.5 l per 1 m2 of area) for exposure for 3 hours. Disinfection of the premises is also carried out by the aerosol method: with a 20% aqueous solution of formaldehyde at the rate of 15 ml per 1 m3 of the room for 3 hours of exposure or with a formalin-creolin (xylon-naphtha) mixture, which consists of three parts of formalin and one part of disinfecting creolin or xylon-naphtha at the rate of 10 ml per 1 m3 of the room for exposure of 6 hours. After the final disinfection, the premises are whitewashed with freshly slaked lime.

Erysipelas (erysipeloid) in humans. Workers of meat and fish processing plants, slaughterhouses, veterinary medicine specialists, butchers, housewives, as well as people who serve patients with swine erysipelas are sick of a professional nature. Infection occurs after minor injuries (injections) to the skin. The incubation period lasts 1-2 days. Initially, a limited red spot appears at the site of skin injuries, more often on the fingers, an increase in regional lymph nodes and a slight fever are noted. Over time, the spot increases in size, pales in the center, and becomes bluish-red on the periphery. After 2-3 weeks, the stain disappears without a trace. Sometimes the course of the disease can be chronic with damage to the joints (swelling, thickening, pain in the area of the phalanx joints, deformative changes).

Control questions and tasks.

1. Who is the source and reservoir of the causative agent of erysipelas in pigs?
2. Can the disease occur in a prosperous household without introducing the pathogen from the outside and under what conditions?
3. Describe the forms of clinical manifestation of erysipelas in different courses of the disease. 4. What data can be used to distinguish erysipelas from classical swine fever?
5. Name the general and specific methods and means of prevention of erysipelas of pigs in animals and humans.

Topic: Reproductive and respiratory syndrome of pigs

(diagnosis, prevention and control measures).

Reproductive and respiratory syndrome of pigs (synonyms - "blue ear", epizootic abortion of pigs) is a contagious disease characterized by mass abortions in sows at the last stage of pregnancy, premature or late farrowing, the birth of non-viable offspring, and is also accompanied by signs of damage to the respiratory organs of piglets.

The causative agent of the disease. RNA-genomic virus, family Arterivirus, family Arteriviridae. A marker of arterivirus is the ability to reproduce in epithelial cell cultures. There are two genotypes of the virus: American (A) and European (B), which have cross-serological relationships and differ in biological properties. The circulation of highly, low-pathogenic and apathogenic strains of the reproductive respiratory syndrome virus in pigs was confirmed, which determines the course of the disease. The virus is inactivated by heating at 55°C for 45 minutes, at 37°C after 48 hours. It is sensitive to the action of light and UV rays, as well as to changes in the pH of the medium. In rooms that cannot be disinfected, the virus remains virulent for 3 weeks.

The diagnosis of reproductive-respiratory syndrome of pigs is made by a complex method on the basis of anamnestic, epizootic, clinical, patho-anatomical data with mandatory examination of blood serum.

Epizootological data. Pigs of all age categories and breeds are susceptible to reproductive and respiratory syndrome. However, reproductive function disorders are observed only in pregnant pigs, and respiratory disease is observed in all age groups. The source of the causative agent of infection is sick and sick pigs, which secrete the virus with nasal mucus, feces, urine and semen. The main ways of transmission of the pathogen are: direct contact of intact pigs with infected ones; feeding of disinfected products and raw materials obtained from sick animals; infected animal care items, fodder, vehicles; rodents that live on the farm. The virus can be transmitted through the sperm of breeding boars during mating or during artificial insemination. The possibility of an aerogenic way of transmitting the pathogen over considerable distances has been established. Most often, the reproductive respiratory syndrome of pigs occurs after the

purchase of clinically healthy repair herds of pigs, which may carry the virus or have an asymptomatic course of the disease in a latent form. Exacerbating the reproductive respiratory syndrome of pigs and turning the latent stage of the disease into an acute one, various stressful events associated with changes in feeding, care, transportation and regrouping, veterinary preventive manipulations. Intoxication occurs in the form of epizootics at different times of the year, with the most pronounced manifestation during the farrowing period.

Clinical signs and course of the disease. The incubation period lasts from 4-7 to 35 days. The disease occurs in acute, chronic, subclinical and latent forms. In the acute form, depression, refusal of feed, a short-term increase in body temperature up to 40.5-42˚C, mass abortions in sows at the last stage of pregnancy (90-110 days), premature or late farrowing, births of dead, sometimes mummified, are registered in pigs fetuses, mass birth of non-viable piglets. There is a transient cyanosis of the ears, heel, tail, skin of the mammary glands, vulva, which lasts from several hours to several days. This sign gave the name "blue abortion", or "blue ear". The duration of farrowing in sick sows is significantly longer, while the intensity of pregnancy activity decreases. These signs are characteristic of 85-100% of sick sows. Underdevelopment of the lower jaw, dome-shaped head, and other developmental defects of the body are noted in some newborn piglets. In one nest from sick sows there can be both dead and mummified fetuses and live, normally developed piglets, most of which still die within the first week of life. In piglets born from sick sows, conjunctivitis, edema and inflammation of the eyelids are noted. Also, in piglets that are in farms that are unfavorable in relation to the reproductive respiratory syndrome of pigs, a severe course of respiratory diseases of various etiologies is noted. Respiratory syndrome is characterized by frequent breathing and coughing. In adult animals, it passes quickly. In piglets of different ages, signs of damage to the respiratory organs continue to progress, which is associated with secondary infection. In piglets, the most frequent clinical signs of infection are dyspnea of a mixed type, rapid and difficult breathing, cough, vomiting, and skin hemorrhages. The chronic form of the disease is accompanied by loss of appetite, conjunctivitis, inflammation of the eyelids, which often leads to keratitis and vision loss. Also, in the chronic form, damage to the respiratory organs by a polyetiological complex of pathogens is noted in animals. In boars, the disease occurs with signs of depression, impotence, significant deterioration of sperm quality. Usually, the disease occurs in the form of epizootics at different times of the year, especially during the farrowing period. The disease may not be clinically manifested in all animals, but mass barrenness is often noted in sows that have fallen ill before.

Pathological anatomical changes. They are not pathognomonic (purely characteristic of this disease). No characteristic patho-anatomical changes were found in breeding boars. Sows develop vaginitis and endometritis after farrowing or abortion, which can be observed under the influence of other etiological factors. The most characteristic macro- and microscopic changes are noted in aborted and newborn

piglets. An external examination reveals a bluish coloration of the ears caused by subcutaneous hemorrhages. Anomalies of eye development were characterized by microphthalmia, hypertrophy of the eyeballs with exophthalmos, inversion of the eyelids, and leakage of the eyeball. Disturbances in the formation of the skull are manifested in a dome-shaped head, the development of cerebral hernias and cerebral edema, non-union of the upper palate, and underdevelopment of the lower jaw. Macroscopic pathological anatomical changes of the bone base of the skeleton are manifested in the absence of ossification of the pelvic part of the spine, underdevelopment and clubfoot of the pelvic limbs, curvature of the spine. All stillborn and piglets that died in the first three days have accumulation of transudate in natural cavities, infiltration of subcutaneous tissue. There are dystrophic changes in the liver, myocardium, hemorrhagic diathesis in the kidneys, inflammatory-necrotic foci in the adrenal glands, as well as hyperplasia and hyperemia of the spleen and lymph nodes (Fig. 1).

Laboratory diagnostics. For laboratory tests, samples of blood or internal organs (lungs, mediastinal lymph nodes, etc.), chest cavity exudate from several aborted fetuses or forcibly killed non-viable newborn piglets (1-3 days old) are taken. Samples of biological material (weighing 10-15 g) are placed in sterile vials, hermetically closed with rubber caps, placed in a plastic bag, in a thermos with ice and sealed.

Blood sera from several animals (2-5 ml) are delivered to the laboratory of veterinary medicine to detect antibodies to the reproductive and respiratory syndrome of pigs. In order to carry out a comprehensive serological screening of the epizootic state regarding this disease, it is necessary to conduct a study of paired blood sera from as many technological groups of pigs as possible (pregnant and barren sows, boars, piglets of various ages). The accompanying note describes the pathology, epizootic indicators and other supporting information in detail. Samples of biological material for laboratory research are sent to state and authorized laboratories of veterinary medicine. Currently, serological reactions are used to detect antibodies to the reproductive and respiratory syndrome of pigs virus: enzyme immunoassay, neutralization and indirect immunofluorescence reactions, immunoperoxidase test, which have different sensitivity and specificity.

The presence of antibodies in unvaccinated animals indicates the circulation of the causative agent of the disease among the pig herd, and obtaining positive results by the polymerase chain reaction method confirms this.

Anomalies of fetuses.

Cyanosis of the ears ("blue ear").

Acute catarrhal bronchopneumonia.

Fig. 1. Pathological changes in reproductive and respiratory syndrome of pigs.

Differential diagnosis. In the differential diagnosis, it is necessary to exclude viral and bacterial diseases that occur with signs of damage to the reproductive and respiratory organs, as well as poisoning. First of all, exclude African swine fever, classical swine fever, swine flu, parvovirus disease, leptospirosis and chlamydia.

Treatment. There is no specific treatment. Given that the causative agent causes an immunodeficient state in the body, sick animals are treated symptomatically to prevent complications and secondary infection.

Immunity. As means of specific prevention of reproductive and respiratory syndrome of pigs, live and inactivated vaccines have been developed: emulsion monovaccine for reproductive livestock; associated - against reproductive and respiratory syndrome of pigs and parvovirus infection; Aujeski's disease, reproductive and respiratory syndrome of pigs, parvovirus infection and leptospirosis. The use of live vaccine is recommended only in farms that are disadvantaged by reproductive and respiratory syndrome of pigs.

Prevention and control measures. For the prevention of reproductive and respiratory syndrome of pigs, heads and specialists of farms, regardless of the form of ownership, are obliged to strictly implement the measures provided for by the veterinary and sanitary rules for pig farms. It is necessary: to observe the current technological and veterinary-sanitary rules for keeping animals; take measures to protect the economy from the introduction of the causative agent of the disease; equip farms with healthy animals from farms that are safe in terms of reproductive and respiratory syndrome of pigs; do not allow food and slaughterhouse waste to be fed to pigs without prior heat treatment.

Upon confirmation of the diagnosis and establishment of reproductive and respiratory syndrome of pigs, the holding is declared unhealthy in accordance with the established procedure and quarantine restrictions are imposed, under which it is prohibited: transportation pigs from unfavorable premises within the farm; slaughter and rearrangement of pigs in the farm without the permission of veterinary medicine specialists; removal of boar sperm outside the unfavorable point; sale of raw meat is prohibited.

Premises, care items, vehicles are disinfected with 5% chloramine solution, 3% hot caustic sodium solution daily and after vacating the premises.

Restrictions on farms affected by reproductive and respiratory syndrome of pigs are removed 60 days after the last selection of sick animals and the completion of all veterinary and sanitary measures, final disinfection, deratization.

Control questions and tasks.

1. What are the main epizootological features of reproductive and respiratory syndrome of pigs?
2. What are the main clinical and pathological forms of the disease?
3. What material is sent to the laboratory and when is the diagnosis of reproductive and respiratory syndrome of pigs considered established?
4. Name the main measures for the prevention and elimination of the disease.

Topic: Swine flu

(diagnosis, prevention and control measures).

Swine flu (Grippus suum, swine influenza, enzootic bronchopneumonia) is an acute highly contagious disease of pigs, which is characterized by fever, general weakness and damage to the respiratory organs.

The causative agent of the disease. RNA-genomic virus from the family Orthomyxoviridae, genus Influenzavirus type A. It is antigenically related to human influenza virus type A and bird influenza virus. Influenza virus is cultivated in 9-12-day-old chicken embryos, as well as in primary cultures of piglet kidney and lung cells. From laboratory animals, white mice, rats, gophers, and ferrets are susceptible, which die 24-48 hours after infection.

The influenza virus is unstable and quickly destroys when dried and exposed to direct or diffused sunlight. At 60°C, the virus is inactivated after 20 minutes, at 18-22°C – after 6 days, at 2-4°C – after 20 days. Solutions of chloramine, perchloric lime, carbolic acid, formalin in the concentrations accepted for disinfection inactivate the virus after 5-10 minutes.

The diagnosis is made by a complex method on the basis of epizootological data, clinical manifestations of the disease, patho-anatomical changes, as well as the results of laboratory studies.

Epizootological data. Pigs of various breeds and ages are susceptible to the disease, but young animals aged 2 to 8 weeks are more often affected, and weaned piglets are less often affected. The source of the causative agent of the infection is virus-carrying pigs that are sick with influenza, which release the virus during coughing, sneezing, and with nasal mucus. Infection occurs by air, as well as through feed, water, bedding, manure, contaminated with secretions from the respiratory tract of sick animals. The disease occurs mainly in the cold, rainy season against the background of a sharp decrease in the resistance of the piglets' body under the influence of colds and poor feeding. Occurs in the form of enzootic outbreaks, lasting 7-10 days. With a large crowd of animals and high relative humidity, which contribute to the transmission of the pathogen by airborne droplets, the flu can spread significantly. Sucklings and weaned piglets are seriously ill.

Clinical signs and course of the disease. The incubation period lasts 1-7 days. The course of the disease is acute and subacute. In adult pigs, during the acute and subacute course, a sharp rise in body temperature up to 41-42°C, depression, lack of appetite. On the 2nd or 3rd day, conjunctivitis, discharge from the nose and eyes, sneezing, wheezing, cough appear (Fig. 1). Some animals develop lung inflammation. Breathing becomes difficult, abdominal type. Sick pigs lie buried in the litter, often assume the "sitting dog" position to facilitate breathing. The disease lasts 7-10 days and mostly ends with recovery. When complicated by a bacterial infection, the duration of the disease is extended to 2-3 weeks. Bronchopneumonia, pleurisy, pericarditis develop, muscle and joint damage (arthritis) is observed.

Fig.1. Serous rhinitis.
(https://www.vetfactor.com/ua/news/gripu-svinei-neobkhidno-pridilyati-bilshe-uvagi/)

In suckling piglets and weaned piglets, the course of the disease is more malignant and often becomes protracted. In addition to inflammation of the lungs, they have pleurisy, pericarditis, and skin lesions. Wheezing, shortness of breath, convulsive cough, refusal to feed appear. Recovery is very slow. Piglets lag behind in development and gradually become exhausted. Skin damage is accompanied by the formation of blackish-brown crusts and scabs.

Pathological changes. Autopsies reveal hyperemia and swelling of the mucous membranes of the nasal cavity, trachea, bronchi, catarrhal inflammation of the lungs, and the accumulation of bloody foamy fluid in them. On the serous and mucous membranes and in the parenchymal organs, there are numerous dotted and striped hemorrhages. In chronic cases, bronchopneumonic foci and atelectasis with foci of connective tissue, as well as pleurisy, pericarditis, and purulent foci in the lungs (Fig. 2).

Fig. 2. Atelectasis of the lungs.
(https://vetmarket.ltd/info/disease/grip_sviney/)

Laboratory diagnostics. It involves the identification of a viral antigen in pathological material (with erythrocytes of chickens or guinea pigs); detection of cytoplasmic basophilic inclusion bodies; isolation of the virus on chicken embryos and in primary cell cultures; identification of the isolated virus by hemagglutination delay reaction, neutralization reaction and immunoenzymatic analysis; conducting a bioassay on white mice; serological studies. Nasal washes, scrapings or smears-prints and secretions from the nasal cavity are sent to the laboratory; pieces of the mucous membrane of the nose and affected lungs are taken from the corpses. For the purpose of retrospective diagnosis, paired blood sera collected from 10-20 pigs with an interval of 10-14-28 days are sent. To isolate the virus, 9-12-day-old chicken embryos are infected with pathological material in the allantoic or amniotic cavity and primary cultures of kidney cells of piglets. Swine influenza virus is identified by the delayed hemagglutination reaction and neutralization reaction with specific serum for swine influenza virus or human influenza virus. The bioassay is carried out on white mice, which are injected with a suspension of pathological material intranasally. Clinical signs of experimental infection appear in them after 1-2 days, death occurs after 4-7 days.

Retrospective diagnosis is carried out by delayed hemagglutination reaction, indirect hemagglutination reaction and neutralization reaction with paired blood sera of sick piglets. A serological diagnosis of influenza is considered positive if a 2-4-fold increase in the titers of specific antibodies is detected in blood sera collected from pigs at intervals of 10-14 days.

Differential diagnosis. Presupposes the need to exclude plague, pasteurellosis and mycoplasmosis. Swine fever occurs at any time of the year, its course is more malignant, often with nervous symptoms. At the autopsy, multiple hemorrhages on the mucous and serous membranes, "marbling" of the lymph nodes, the formation of "plague buds" on the mucous membrane of the intestines, infarcts of the spleen. Pasteurellosis is manifested by septicemia, signs of croup pneumonia. Pasteurella culture is isolated during bacteriological examination. Weaned piglets suffer from mycoplasmal pneumonia, the course of the disease is chronic. During the bacteriological examination, mycoplasmas are isolated.

Treatment. There are no specific treatments for swine flu. Optimal housing conditions are created for sick animals, and they provide complete dietary nutrition. In order to prevent bacterial complications, broad-spectrum antibiotics and sulfonamide drugs are used. Symptomatic treatment is carried out.

Immunity. After pigs become sick with influenza, it lasts only 3-4 months. Specific drugs for the prevention of the disease have not been developed.

Prevention and control measures. They should be aimed at creating proper zoohygienic conditions for keeping pigs, providing them with full-fledged rations. In the summer, all pigs should be kept in camps, and in the winter - in dry, well-ventilated rooms, to prevent moisture and cold. Precautionary veterinary and sanitary measures are carried out against the introduction of the causative agent of the disease into healthy farms. All pigs arriving from outside should be kept in quarantine units for 30 days and examined for the absence of virus-carrying. When the disease appears, quarantine restrictions are introduced, a clinical examination and thermometry of all animals in a dysfunctional piggery are carried out. Patients and suspects of swine flu are immediately isolated, and appropriate care and treatment is arranged for them.

Dry lime is used to disinfect pig houses and prevent the accumulation of excess moisture. 2% hot caustic soda solution, 20% slaked lime suspension, chlorinated lime solution containing 3% active chlorine are also recommended. Manure is disinfected biothermally.

Control questions and tasks.

1. Describe the clinical and epizootological features of swine flu.
2. How is the diagnosis made and from which diseases should swine flu be differentiated?
3. Measures to prevent and eliminate swine flu.

Topic: Infectious atrophic rhinitis of pigs

(diagnosis, prevention and control measures).

Infectious atrophic rhinitis of pigs (Rhinitis atrophica infectiosa, IAR, porcine bordetellosis) is a chronic respiratory disease characterized by serous-purulent rhinitis, atrophy of the nasal concha, and deformation of the skull bones. The vast majority of piglets have catarrhal pneumonia and growth retardation.

The causative agent of the disease. Bordetella bronchiseptica. A non-motile, gram-negative bacillus, does not form spores and capsules, a strict aerobe. Grows well on simple nutrient media. It forms dermonecrotic exo- and endotoxins and a lipopolysaccharide complex that has adhesiveness to the cells of the mucous membrane of the respiratory tract. In livestock premises, the causative agent remains viable for at least 16 days. Freezing preserves it for up to 4 months or more. Solutions of caustic soda (3%), formaldehyde (1%) and a suspension of freshly slaked lime (20%) inactivate the pathogen within three hours.

The diagnosis of infectious atrophic rhinitis is made by a complex method on the basis of epizootological, clinical, patho-anatomical data. For intravital diagnosis and timely detection of latently infected animals, radiography of the frontal part of the head and laboratory examination of nasal mucus for the presence of Bordetella bronchiseptica are recommended.

Epizootological data. In natural conditions, infectious atrophic rhinitis is most pronounced in pigs, especially in piglets. Adult pigs are relatively resistant. Bordetella bronchiseptica, having a pronounced tropism to respiratory organs, can cause atrophic rhinitis and pneumonia in rabbits, rats, mice, guinea pigs and other rodents under natural conditions. Sporadic cases of the disease are registered in dogs, horses and sheep. The source of the causative agent of the infection is pigs clearly suffering from rhinitis, which release the causative agent into the environment during sneezing, coughing and with nasal discharge. Often, adult animals, especially sows, get sick without symptoms and are the main danger in the spread of the disease among newborn piglets. In the household, the causative agent of the disease is transmitted mainly by airborne droplets, because infectious atrophic rhinitis is a typical respiratory infection; between farms, the pathogen spreads with newly imported animals from disadvantaged farms. The spread of the causative agent of the disease with breeding pigs - bordethelon carriers, which among disadvantaged herds is 25-50%, as well as with animals of other species (reservoir of the causative agent) is especially dangerous.

The epizootic process during infectious atrophic rhinitis develops slowly and reaches its maximum development 2-4 years after the introduction of Bordetel carrier pigs into the farm. The economy, as a rule, becomes permanently dysfunctional. First, the disease of piglets is noted in the nests of individual sows, then in most nests. The maximum rise of epizootics occurs during the period of receiving and weaning piglets. The emergence and spread of the disease is often determined by violations of the established balance between the pathogen, the host (pigs) and the environment. Massive and multiple doses of the pathogen during natural infection, low level of general resistance and inflammation of the mucous membranes of the upper respiratory

tract, poor conditions of feeding and keeping, along with the virulence of the pathogen, determine the breadth of distribution and intensity of the disease. Untimely and unsystematic health measures lead to the manifestation of secondary infections and a severe course of the disease. If there is no fight against the disease, unsystematic health measures, the economy may remain dysfunctional for several years. Morbidity among piglets can reach 50-80%.

In farms, with the elimination of unfavorable factors, especially during the organization of camp keeping of pigs, providing animals with complete feed and creating a normal microclimate, the epizootic process subsides and infectious atrophic rhinitis manifests itself without pronounced clinical signs, with weak shell atrophy and even without symptoms.

Clinical signs and course of the disease. The incubation period lasts 3-15 days. The disease has a chronic course. Clinical signs before the appearance of curvature of the facial bones of the skull are uncharacteristic.

As a rule, suckling piglets are infected in the first days of life. The first signs of the disease appear after 7-12 days in the form of frequent sneezing and serous discharge from the nose (catarrhal rhinitis). Due to severe damage, nosebleeds are possible. Then the secretions become mucous-purulent, swelling of the lower eyelids, lacrimation with the formation of dark spots in the inner corners of the eyes appear. During this period, 10-90% of piglets may develop clinical signs of bronchopneumonia and diarrhea, the appearance of which is facilitated by poor feeding conditions and keeping of sows and piglets. Some of the piglets with complications die, and others turn into dead animals. In some piglets, after the disappearance of signs of acute catarrhal rhinitis, which lasts no more than 2-3 weeks, the disease takes a latent course. In such cases, shell atrophy either does not develop or is weak. The facial bones of the skull do not change, and only in some animals it is possible to detect an abnormal bite. In the rest of the animals, atrophy of the nasal conchas and facial bones of the skull gradually develops, the upper cleft becomes shorter and a discrepancy between the incisors between the jaws appears by 0.5-1 cm at 1-2 months of age, and by 1-3 cm at 3-6 months of age. In most sick piglets, a fold of skin forms on the nose behind the heel, the lower lip protrudes forward, and food intake is sharply impaired. With a bilateral lesion of the nasal cavities, animals develop a pug-like appearance, and with a unilateral lesion of the upper jaw to the right or left (curvature). This condition can occur in 50% of sick animals at the age of 3-4 months (Fig. 1). Patients with characteristic clinical signs of infectious atrophic rhinitis constantly notice purulent discharge from the nose, wheezing, sneezing and coughing. If the ethmoid bone and meninges are involved in the inflammatory process, then signs of damage to the nervous system will appear, which are clinically similar to the signs of Aujeski's disease. Quite often, sick animals have itching in the area of the heel and severe bleeding from the nose. The middle ear is inflamed, which is manifested by an unnatural position of the head, circular movements, tense gait, squint and convulsions. As a rule, the growth of clearly sick

piglets slows down significantly, they differ noticeably in terms of body weight in large fattening groups from healthy animals. Some animals develop otitis, nervous excitement (animals dig the ground, bite their tails, etc.).

Fig.1. Atrophy of nasal conchas with malocclusion.
(https://pigua.info/uk/post/nekrozi-svinej-uk)

Pathological changes. Longitudinal (sagittal) and transverse (at the level of the other premolars) dissection of the facial bones of the skull is performed to detect pathological changes. In the initial stage of the disease, hyperemia of the mucous membrane of the nasal cavity and accumulation of thick mucus are detected. At later stages, in subclinical and latent forms of the disease, different degrees of atrophy of the nasal conchas are revealed, shortening of the upper jaw is possible. In severe cases of the disease, the nasal turbinates are completely destroyed, the mucous membranes are covered with pus and thinned, asymmetrical and deformed. Abscesses are found in the regional lymph nodes. Sometimes foci of catarrhal or catarrhal-purulent pneumonia and hyperemia of the meninges are detected.

Differential diagnosis. It is necessary to exclude the flu, fibrous osteodystrophy (necrotic rhinitis). Influenza outbreaks are acute, the disease spreads quickly in the herd. With necrotic rhinitis, soft tissues, cartilage and bones of the nose disintegrate with the formation of ulcers. In all doubtful cases, additional pathological, histological, bacteriological, virological and serological studies are carried out.

Treatment. It should be carried out in the initial stage of the disease. There are no specific remedies. Individual irrigation of the nasal cavity with solutions of antibiotics and sulfonamide drugs (penicillin, biomycin, chloramphenicol) in combination with intramuscular injections of vitamin B is recommended. Bordetellas are particularly sensitive to sulfamethazine and sulfathiazole. These and other drugs (biovit-40-80-120, teramycin, biomycin) should be fed with feed according to the defined schemes. Treatment prevents the development of atrophy of the nasal concha.

However, animals that have recovered can remain carriers of the pathogen, they cannot be used in breeding work. Such animals are fattened and handed over for slaughter.

Immunity. For the specific prevention of infectious atrophic rhinitis in pigs, live and inactivated vaccines made from field and attenuated strains of Bordetella bronchiseptica have been developed. Such vaccines in a complex with general veterinary-sanitary and medical-prophylactic treatments contribute to the quick and reliable sanitation of the herd from carrying bordetel.

Prevention and control measures. In the prevention of infectious atrophic rhinitis, the correct genetic selection and full feeding of sows, compliance with veterinary sanitary and zootechnical rules for reproduction, breeding and fattening of pigs are of particular importance. Breeding animals that are purchased from other farms are subjected to a thorough clinical examination, quarantined and, if necessary, patho-anatomical and laboratory tests are carried out. Clinical examinations of animals and disinfection of pigsties are carried out regularly. Considerable attention should be paid to monitoring the reproduction of piglets and their health. Timely and constant feeding of piglets is organized, protein, vitamin and mineral supplements are introduced into the rations, as well as medicinal preparations in the form of premixes. When a diagnosis of infectious atrophic rhinitis of pigs is established, the farm is declared unhealthy. All livestock are carefully clinically examined and the animals are divided into 3 groups:

1) a group of sick pigs, which have characteristic clinical signs of the disease, are isolated, fattened and slaughtered;

2) the group of conditionally healthy pigs, from which the sick ones were selected, is clinically examined every 5-6 days; the sick are isolated and put on fattening; if at least one piglet with rhinitis is found in the nest, all piglets together with the sow are isolated, fattened and slaughtered;

3) a group of healthy pigs, among which no rhinitis patients were found, are kept under special observation, zootechnical records are established and measures are taken to protect against infection.

If the disease becomes widespread (more than 50% of the herd is infected), the reproduction of the herd is stopped, all pigs are put to fattening. Older sows are kept for breeding purposes. A system of step-by-step isolated rearing of young animals is being introduced to reproduce the herd, which is based on biological testing of sows for the quality of the offspring. It is advisable to send sows and boars, in whose offspring patients with rhinitis are registered, to slaughter.

A group method of using fodder antibiotics and sulfonamide preparations in the form of premixes is economically justified for a disadvantaged farm with preventive and curative purposes. It is forbidden to take pigs from a dysfunctional farm to other farms to reproduce the herd. Pig pens and the territory of farms are systematically cleaned and disinfected with solutions of caustic soda, formaldehyde, freshly slaked lime.

Restrictions from farms that are not prosperous due to infectious atrophic rhinitis are removed a year after the end of the allocation of patients and on the condition that there is no infectious atrophic rhinitis among piglets of the last two farrowings from the main sows of a conditionally prosperous herd.

Control questions and tasks.

1. What complex of etiological factors causes the appearance of the disease?
2. Describe the clinical and epizootological features of the manifestation of infectious atrophic rhinitis in pigs of different ages.
3. How are animals secretly sick with infectious atrophic rhinitis detected?
4. Are there methods for determining the extent of microbiosis in infectious atrophic rhinitis?
5. How to improve the health of farms affected by infectious atrophic rhinitis?

Topic: Vesicular disease of pigs

(diagnosis, prevention and control measures).

Vesicular disease of pigs (Morbus vesicularis suum) is an acute highly contagious disease of pigs, which is characterized by fever, vesicular rash on the skin of the heel, udder, areola, integumentary gap, soft tissue and mucous membrane of the oral cavity. People also can get sick.

The causative agent of the disease. RNA-genomic virus from the Picornaviridae family. Spherical virions have no antigenic affinity to any of the known epitheliotropic viruses of pigs. Without adaptation, it reproduces in primary cultures of kidney cells of piglets and transplanted culture RK-15, causes CPD, forming clusters in the form of clusters. Hemagglutinating and hemadsorbing properties of the virus have not been established. The virus in high concentrations is detected in the epithelium of affected areas of the skin, lymph nodes and bone marrow. Experimentally, one-day-old mice are quite easily infected, in which, 24-30 hours after the introduction of the infected material, clear signs of damage to the central nervous system (tremor, impaired coordination of movements, paralysis and death on the 7th day) are observed. The disease is easily reproduced by oral, subcutaneous and intradermal infection in pigs of any age. After 36 hours, vesicles are formed at the place of introduction of the virus into the piglet, which later turn into erosions. Generalization of the process is accompanied by damage to the skin in the area of the corolla, interradicular gap, and soft tissues. The virus is stable in the external environment - it can be stored in feces and urine for 3 months, in manure for 8 weeks, and for at least 20 months on contaminated surfaces at sub-zero temperatures. In infected carcasses at minus 20°C, it remains viable for 11 months, in frozen pork - more than a year, in meat waste from slaughtered sick pigs at a temperature not higher than 0°C - 100 days. Does not break

down under the action of lactic acid during meat maturation, in smoked sausages and hams - 400 and 780 days, respectively. Resistant to ether and environmental reactions in the pH range from 2.5 to 10.5, as well as to such disinfectants as formalin and caustic sodium. When heated to 60°C, it is inactivated after 30 minutes.

The diagnosis is made by a complex method on the basis of characteristic clinical signs of the disease, taking into account epizootological and patho-anatomical data. Virological studies are of decisive importance.

Epizootological data. In natural conditions, only domestic and wild pigs of all age groups get sick. The source of the causative agent of infection is sick pigs, from whose bodies the virus is released in large quantities into the external environment with saliva, nasal mucus, feces and urine. Asymptomatic sick pigs, which become virus carriers within 4-6 months, pose a special threat in maintaining the epizootic process. Factors of virus transmission can be feed, water, infected products from the slaughter of sick pigs, uninfected animal waste, as well as premises, care items, vehicles. Infection occurs through direct contact with sick animals, as well as orally when feeding uninfected slaughterhouse and kitchen waste. Sometimes the virus enters the body through microtraumas of the skin and various damaged parts of the limbs. The disease proceeds benignly, in the form of separate outbreaks or limited epizootics. Morbidity – 60%, mortality – 10%.

Clinical signs and course of the disease. The incubation period lasts 2-6 days. The course of the disease is acute, subacute and chronic.

During an acute course, morbidity can reach 100% of the pig herd. Most animals have fever (40.5-41.5°C), depression, and decreased appetite. At the same time, vesicles appear on the skin in the area of the corolla, the interspinous cleft, and on the pulp of the scrotum, which later burst, revealing painful erosions and ulcers (Fig. 1). They note severe lameness, difficulty in moving, in some animals - the fall of the horn shoe. In approximately 10% of sick animals, vesicles are also found on the mucous membrane of the oral cavity, lips, and tongue. With a mild form of the disease, pigs fully recover in 1-3 weeks. In a severe form of the disease, there are signs of damage to the central nervous system (excitement, impaired coordination of movements, paralysis), often with a fatal outcome. Abortions, vesicular lesions of the udder skin may be observed in sows.

Fig. 1. Erosions.

During the subacute course of the disease, clinical signs are less pronounced. Only some animals are sick, in which single vesicles on the corolla, lethargy, weight loss, sometimes swelling of the joints, lameness, diarrhea are found. Sick pigs usually recover. In the case of secondary microflora complications, enterocolitis, pneumonia and significant death among suckling piglets are possible.

The chronic course of the disease is detected only by the presence of specific antibodies in high titers in blood serum.

Pathological changes. Very similar to those observed in pigs with foot-and-mouth disease. A vesicular lesion of the skin on the limbs in the area of the corolla, integumentary cleft, soft tissues of the udder is revealed, much less often – lesions of the mucous membrane of the oral cavity and udder. At the site of the burst vesicles, erosions and ulcers are noted. Diseased animals have long-term traces of the disease in the form of scars and deposits of horny substance in the areas of the legs.

Laboratory diagnostics. It involves the detection and identification of the viral antigen in the pathological material, the isolation of the virus in cell culture, conducting a bioassay, and the detection of specific antibodies in the blood sera of sick pigs. At least 2 ml of vesicular fluid from 2-5 sick animals, the walls of unopened vesicles from the affected areas of the skin of the heel, crown, hooves, and udder are sent to the laboratory for research. Blood sera from 5-10 ill pigs are sent for retrospective diagnosis.

In the laboratory, the viral antigen in the pathological material is detected by the complement binding reaction, the immunofluorescence reaction, and the diffuse precipitation reaction. Identification of isolated cultured virus is carried out by neutralization reaction, complement binding reaction and immunofluorescence reaction. The bioassay is carried out on one-day-old white mice, which are infected intracerebrally. If the virus is present in the pathological material, the mice die within 3-10 days with the characteristic phenomena of paralysis. To determine the specificity of the viral antigen, the suspension of their organs is examined by the complement binding reaction. For the retrospective diagnosis of vesicular disease, paired or single-selected sera from infected pigs are examined by complement binding reaction, neutralization reaction and diffuse precipitation reaction. Serological diagnosis is considered positive with at least a two-fold increase in the titer of specific antibodies in the second paired blood serum.

Differential diagnosis. Provides for the exclusion of foot-and-mouth disease, vesicular exanthema and vesicular stomatitis of pigs. In case of foot-and-mouth disease, in contrast to vesicular disease, in addition to pigs, other types of animals are also affected. In pigs, the limbs and piglets are affected, and the mucous membrane is affected only as an exception. Conducting a bioassay on guinea pigs that are not susceptible to the vesicular disease virus, as well as the results of the complement

binding reaction with specific antigens and foot-and-mouth disease antisera, make it possible to reliably differentiate these diseases. Vesicular stomatitis affects not only pigs, but also cattle and horses. Vesicular exanthema has a more malignant course than vesicular disease, spreads much more slowly.

Treatment. It is carried out with antiseptic, weak disinfectant and astringent agents (potassium permanganate solution, copper sulfate). Antibiotics and various ointments are also used.

Immunity. Pigs infected with vesicular disease develop persistent immunity for up to 2 years. Piglets acquire passive immunity for 2-3 weeks through the colostrum of immune sows. For specific prevention, an inactivated vaccine is proposed, after the use of which immunity is formed in piglets lasting up to 3 months, in adult animals - up to 9 months.

Prevention and control measures. There are ways to prevent the introduction of the causative agent of swine vesicular disease into the territory of Ukraine. For this purpose, it is forbidden to import pigs, meat products and leather from disadvantaged countries and regions, and to use kitchen waste collected in airports, planes, trains and steamships serving international lines for feeding pigs. All pigs arriving from safe countries must be quarantined and selectively tested for vesicular disease by a serological method. In the case of the occurrence of vesicular disease of pigs, the unfavorable point is quarantined, and restrictions are introduced in farms and meat processing enterprises with which inter-farm relations were maintained 10 days before the appearance of the disease. Strict measures are taken to isolate the epizootic center and destroy the causative agent of the disease in the external environment. In the unfavorable point, the meat of the entire unfavorable group of pigs is slaughtered at a specially equipped site or at the sanitary slaughterhouse of the nearest meat processing plant. Slaughtered pig carcasses and offal are used for the production of boiled and boiled-smoked products. Disadvantaged pigsties, where infected pigs temporarily stayed, as well as walking yards, inventory, machines, equipment, transport are subjected to thorough mechanical cleaning and disinfection with a hot 4% solution of caustic sodium or a 2% solution of formaldehyde twice with an interval of 5 days or a 3% solution of naphthalizol. Manure is neutralized by the biothermal method. Pig carcasses are burned.

Control questions and tasks.

1. Describe the epizootological features and clinical signs of swine vesicular disease.
2. Reveal the method of taking and sending pathological material.
3. On the basis of what data, it is possible to distinguish vesicular disease of pigs from foot-and-mouth disease, vesicular exanthema of pigs and vesicular stomatitis?
4. What is the basis of prevention and health measures for vesicular disease of pigs?

Topic: Vesicular exanthema of pigs

(diagnosis, prevention and control measures).

Vesicular exanthema (Exanthema vesicularis) is an acute highly contagious disease of pigs, which is characterized by fever, vesicular lesions of the mucous membrane of the oral cavity and skin in the area of the crown, interstitial cleft and soft tissue.

The causative agent of the disease. RNA-genomic virus from the Picornaviridae family. The virus is epitheliotropic, has 16 antigenically different serotypes. It is found during the period of fever in the blood and internal organs of sick pigs, and later in the epithelial cells, walls and contents of vesicles. The virus is cultivated in primary cell cultures of piglet kidneys and pig embryo kidneys. Laboratory animals are immune to the vesicular exanthema virus.

The virus is stable in the external environment and against various physical and chemical factors. Retains infectious activity at room temperature for 6 weeks, at 37°C – 24 hours. It remains viable for years in the infected walls of vesicles preserved in a 5% aqueous solution of glycerol at 4°C. It can be stored in pork at 7°C for up to 30 days, at minus temperature - up to 4 months, in slaughter waste at 7°C - for 4-5 weeks. Resistant to the action of ether, chloroform, sodium deoxycholate and to the reaction of the environment in the range of pH = 5.0-10.0. Quickly inactivated under the influence of 2% sodium hydroxide solution, 2-3% formalin solution and at temperatures above 50°C.

The diagnosis is made by a complex method on the basis of clinical signs of the disease, taking into account epizootological and patho-anatomical data. Laboratory studies are of decisive importance.

Epizootological data. Domestic pigs are sick regardless of breed and age. Experimentally, it is possible to infect horses, from laboratory animals - gophers. The source of the causative agent of the infection is sick pigs, which begin to secrete the virus already in the incubation period, as well as virus carriers within 3-4 months after becoming ill. The virus is released from the body of infected pigs along with the walls and contents of the vesicles, urine, and feces. Infection occurs through direct contact with infected animals, as well as through virus-contaminated care items, feed, water, premises and pens where sick pigs are kept. Uninfected slaughterhouse and kitchen waste is a particular danger, the use of which for feed can lead to an outbreak of infection and mass disease of animals. Lethality is less than 5%.

Clinical signs and course of the disease. The incubation period lasts 1-12 days. The course of the disease is acute, although cases of inapparent course with virus-carrying have also been registered. At the beginning of the disease, pigs show fever, lethargy, decreased appetite, and drooling. At the same time, primary vesicles with serous exudate appear on the nipple and the mucous membrane of the oral cavity, which

quickly burst, and painful erosions and ulcers form in their place (Figs. 1, 2). Over time, their surface is covered with fibrinous films, the affected areas heal quickly. The temperature decreases, but the general condition of the sick pigs does not improve. Secondary vesicles are formed on the skin of the limbs in the area of the corolla, wrist, soft tissue, as well as the interradicular gap. Sick animals are depressed, exhausted, lie down more, get up with great difficulty, and limp severely. At the end of the first week, vesicular skin lesions disappear, and in the next 7-10 days the animals recover. In the case of secondary microflora joining the disease, various complications develop (panaritium, loss of the horn shoe), the mortality rate among piglets increases significantly (up to 10%). In sows during this disease, abortions, vesicular lesions of the udder, decreased milk secretion can be observed.

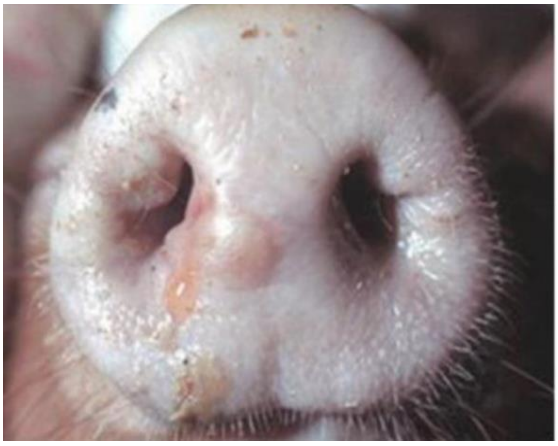

Fig. 1. Serous exudate from the nose.

Fig. 2. Erosions.

(https://studfile.net/preview/5710154/)

Pathological anatomical changes. Appear on the skin of the limbs and the mucous membrane of the oral cavity in the form of vesicular lesions, erosions and ulcers caused by the reproduction of the virus in the Malpighian layer of the epidermis.

Laboratory diagnostics. It is based on the isolation of the virus from the pathological material, its identification and determination of the type of the isolated virus by complement binding reaction. For research, the walls and contents of the vesicles, selected on the first day of their formation, are sent to the laboratory. In order to isolate the virus, the primary kidney cell cultures of piglets are infected with pathological material.

In the presence of the vesicular exanthema virus in infected cell cultures, a cytopathogenic effect appears after 8-10 hours in the form of large light and small dark plaques along the entire monolayer, degeneration of the cytoplasm and cell nucleus, and the formation of cluster-like clusters of virions in the cytoplasm. Identification of the isolated virus is carried out using a neutralization reaction with a specific serum. To type the virus, studies of vesicular fluid and suspension from vesicular walls are carried out according to the complement binding reaction with type-specific sera.

Differential diagnosis. Presupposes the need to exclude foot-and-mouth disease, vesicular stomatitis and vesicular disease of pigs. In case of foot-and-mouth disease in pigs, the piglet, limbs, udder nipples are affected, rarely the oral cavity; positive bioassay on guinea pigs. All domestic animals suffer from vesicular stomatitis, there is no simultaneous formation of vesicles in the oral cavity and on the limbs; positive bioassay on mice. Vesicular disease affects only pigs, other types of animals do not get sick. Positive bioassay on one-day-old mice. In all cases, the final diagnosis is made on the basis of the results of the examination of pathological material using the complement binding reaction and the neutralization reaction.

Treatment. Specific means of treatment are not offered. Various disinfectant and astringent solutions are used, the affected skin areas are lubricated with ointments and antiseptics. Antibiotics are used.

Immunity. After an illness, pigs develop short-term immunity. Vaccination of pigs is not promising due to the large number of types of vesicular exanthema virus.

Prevention and control measures. It is necessary to strictly observe the requirements of veterinary and sanitary supervision during the selection and import of pigs from abroad. When the disease appears, it is advisable to carry out a general slaughter of pigs of the disadvantaged group, followed by thorough cleaning of the places of temporary stay of animals and disinfection. Carcasses of sick pigs and slaughter waste are burned. After the final disinfection, the premises are left pig-free in the summer for 2 months, in the winter – throughout the winter.

Control questions and tasks.

1. Describe the clinical signs and epizootological features of vesicular exanthema of pigs.
2. Reveal the method of taking and sending pathological material to the laboratory.
3. What methods are used for diagnosis?
4. From what diseases and on the basis of what data is it necessary to differentiate vesicular exanthema?
5. How to heal dysfunctional farms from vesicular exanthema?

Topic: Enzootic encephalomyelitis of pigs (Techen's disease)

(diagnosis, prevention and control measures).

Teschen disease (Encephalomyelitis enzootica suum, enzootic encephalomyelitis of pigs) is an acute contagious disease of young pigs, which is characterized by signs of damage to the central nervous system (non-purulent encephalomyelitis and paralysis).

The causative agent of the disease. The RNA-genomic virus, which belongs to the Picornaviridae family, has a spherical shape. The virus is neurotropic, pathogenic for laboratory animals and chicken embryos. At the beginning of the manifestation of the clinical picture, the virus in maximum titers is detected in the cervical and thoracic parts of the spinal cord. After the appearance of paralysis, its amount in the body constantly decreases, and at the time of the animal's death, it may not be present at all. It appears in the tissues of the alimentary canal 24-72 hours after infection and is detected within 5-7 weeks. The virus stays in the blood and internal organs for a short time, between 4-6 days after infection, and in a small amount. During the incubation period and in the first two days of the disease, it is released into the environment with feces, urine, saliva, and nasal mucus. It is reproduced in primary cultures of kidney cells of piglets or pig embryos. The virus is resistant to the action of various physical and chemical factors and persists for a long time in the external environment. Withstands heating up to 56°C for one hour, at 37°C it is stored for up to 17 days, in salted and smoked meat products - for more than 3 weeks; in pus and infected premises, on the floor, walls - 6-8 weeks. The virus can withstand drying in the sun for up to 3 weeks; does not change in a wide pH range (2.8 - 9.5); destroys under the action of 0.5% phenol solution for 18 hours, 2% caustic soda solution for 7 hours, 2% formalin solution for 1 hour, 5% chloroform solution for 3 hours. It is quickly inactivated during the development of putrefactive processes, as well as under the influence of high temperature; when boiled, it is destroyed after a few seconds.

The diagnosis is made by a complex method on the basis of epizootological data, clinical features of the disease, patho-anatomical and histological changes, as well as the results of virological studies, and, if necessary, bio-samples.

Epizootological data. Only pigs, including wild ones, are susceptible to Techen disease. Piglets and piglets aged 1.5-4 months are more sensitive. Newborn piglets do not get sick up to 15 days of age. The source of the pathogen is clinically and latently sick pigs, as well as sick animals that can be carriers of the virus for up to one year. Most often, Teshen's disease appears in the farm after animals have been imported from unfavorable locations. Infection occurs by contact when healthy pigs are kept together with sick ones, as well as through virus-contaminated feed, water, air, care items, slaughterhouse and kitchen waste. The causative agent enters the body through the mucous membranes of the nose and digestive tract. Various rodents, birds, and even humans can be mechanical carriers of the virus. In disadvantaged areas, the spread of the disease is facilitated by the movement of pigs.

Techen's disease occurs in the form of sporadic cases or enzootics. In some cells, a simultaneous mass release of sick animals is observed, in others - with an interval of different duration. The disease can occur at any time of the year, but the course of the disease is more severe in the autumn-winter and winter-spring periods. During enzootic Teshen disease, from 20 to 90% of animals become ill, mortality can reach 90%.

Clinical signs and course of the disease. The incubation period lasts 1-4 weeks. The course of the disease is acute, subacute and chronic. In piglets aged up to 2 months. cases of hyperacute course can be observed, when the death of animals occurs after 24-48 hours against the background of general paralysis. The acute course is most common among piglets aged 2 to 10 months. A short-term (1-2 days) increase in body temperature up to 41.5°C, weakness, bad appetite, eating litter, various objects, depression, vomiting, constipation, sometimes disorder of coordination of movements, skin hyperesthesia is observed. On the 2nd or 3rd day, symptoms of spinal cord damage appear - a shaky, unsteady gait, paralysis of the back and then the front limbs (Fig. 1). In severe cases, paralysis of the muscles of the neck and head is noted. Animals lose the ability to stay on their feet, lying on their sides, make continuous swimming movements, sometimes lie on their stomachs and try to move forward, grind their teeth, sometimes scream loudly. This is followed by complete paralysis, which is a characteristic feature of this disease. A paralyzed animal does not refuse food and water. Sick piglets die 1-3 days after the onset of paralysis.

Fig.1. Paralysis of pelvic limbs.
(https://selo-exp.com/svinii/bolezn-teshena-svinej.html)

The subacute course of the disease is observed in stationary dysfunctional farms. It is characterized by the absence of fever and excitement. Incomplete, less often complete, paralysis is noted. The animals mostly lie down, sometimes they assume a "sitting dog" pose. The duration of the disease is 6-8 days, the mortality rate is 30-50%. In the subacute course, death occurs as a result of respiratory paralysis.

The chronic course is registered mainly in adult pigs, accompanied by paralysis of the hind and sometimes front limbs, exhaustion. Animals lie for a long time, move with effort, often fall. The duration of the disease is from several weeks to months. Lethality - up to 20%. Complete recovery is rare, most sick animals show lameness, muscle atrophy, especially in the hind limbs, tendon contracture.

Pathological changes. Autopsies reveal hyperemia and swelling of the medulla and gray matter of the brain. Hemorrhages in the spinal cord. Hemorrhagic inflammation of the mucous membrane of the nose and sinuses, which have a bluish

color, is almost always detected. During a histological examination, infiltration changes characteristic of Teshen's disease are determined in the gray part of the spinal cord, in the brain and on the meningeal membranes (Fig. 2). Lymphocytic foci around blood vessels in the ventral horns of the spinal cord are also detected, in which degeneration of neuroglia cells is observed. In addition, diffuse and focal round cell infiltration is detected in the brain and meninges, leptomeningitis in the cerebellum.

Laboratory diagnostics. Includes the detection of a specific viral antigen in swabs-imprints from pathological material; virus isolation in cell cultures and its identification; retrospective diagnosis, and if necessary, a bioassay on piglets.

Feces and washings from the rectum are sent to the laboratory for research during life, and for postmortem diagnosis - in a thermos with ice or preserved in a 30% solution of glycerol, pieces of the cerebellum, medulla oblongata and spinal cord of dead or forcibly slaughtered sick animals at the stage of paralysis. To isolate the virus, primary cultures of kidney cells of piglets or pig embryos are infected. Identification of the isolated virus is carried out by neutralization reaction, immunofluorescence reaction, complement binding reaction, diffuse precipitation reaction and enzyme immunoassay with appropriate diagnostic sera. The bioassay is carried out on 4 healthy piglets 2-4 months old from prosperous farms. The bioassay is considered positive when the clinical picture of Teschen disease develops in infected piglets and there are no signs of the disease in control animals.

Differential diagnosis. Teschen's disease must be differentiated from rabies, Aujeski's disease, swine fever, listeriosis and feed poisoning.

All types of domestic and wild animals, sensitive laboratory animals are susceptible to rabies. Sick pigs are aggressive towards other animals and humans. During microscopic examination, Babesh-Negri bodies, intensive infiltration of spinal and sympathetic ganglia with inflammatory cells, enlargement of nuclei in nerve cells, karyolysis, vacuolization, granular-fatty degeneration are revealed in histological preparations from the horn of Ammonia and salivary glands. Piglets under the age of 10 days are seriously ill with Aujeski's disease, with high mortality. Dogs, cats, and rodents on the farm are susceptible. A positive bioassay on rabbits is characteristic (excitement, itching at the site of introduction of pathological material). In Aujeski's disease, the cerebellum is most affected. Pigs of different ages are affected by plague. High fever of a constant type, hemorrhagic diathesis phenomena, splenic infarctions, inflammation of Peyer's patches are observed. Sick animals never experience complete paralysis. Microscopically, the largest lesions in swine fever are found in the middle and back part of the brain, the spinal cord and gray matter are rarely affected. Listeriosis in pigs occurs in septic and nervous forms, but there is no paralysis. Sows have abortions and fattening. The causative agent of the disease is isolated in the sows on bacteriological media. Poisoning is ruled out on the basis of anamnesis and the results of toxicological studies.

Treatment. Not developed.

Immunity. Sickness in pigs is accompanied by the development of long-term and intense immunity, which is transmitted with colostrum to newborn piglets. The use of hyperimmune sera or sera of convalescents in Techen disease is ineffective.

For specific prevention, a cultural inactivated emulsified vaccine is used, which is injected into the neck area intramuscularly, once, to piglets aged from 1 to 60 days - 1 ml each, to pigs aged 60 days and older - 2 ml each. Immunity occurs after 2 weeks and lasts for at least 11 months. Sows are vaccinated regardless of the period of gestation, but no later than 3 weeks before farrowing. Piglets born from vaccinated sows are vaccinated 3-4 weeks after birth.

Prevention and control measures. They should be directed primarily to the protection of farms from the introduction of the causative agent of Teshen's disease and the elimination of infection in disadvantaged areas. In disadvantaged areas, it is prohibited to trade pigs in markets, sell pigs to other farms, as well as use uncontaminated waste from slaughterhouses and canteens for feed. Pigs must be brought in only from safe areas for stocking pig farms, planned preventive measures should be systematically carried out, including disinfection and deratization works. In unfavorable and dangerous areas, the entire pig herd is vaccinated with vaccines. When Teshen's disease occurs, quarantine restrictions are introduced in the farm. When a disease appears in a previously prosperous farm, as well as in feedlots and auxiliary departments, it is advisable to kill all animals, and replace the herd with a new herd of pigs. In these cases, it is allowed to bring in a new healthy pig herd only after appropriate veterinary and sanitary measures and final disinfection have been carried out on the farm. Imported pigs are placed in quarantine facilities for 30 days, and during this time the necessary diagnostic and preventive measures are carried out. All pigs must be vaccinated against Techen disease and undergo veterinary observation for 6 months. In farms where it is impractical to slaughter the entire herd, only sick and suspected animals are slaughtered, and the rest of the pigs are vaccinated. Vaccination of all pigs, without exception, is carried out in those disadvantaged farms where the disease of newborn piglets is observed. Sows are vaccinated regardless of the period of gestation.

Pigs are vaccinated from the age of 2-3 months in farms that are disadvantaged and at risk of Teshen's disease. Veterinary surveillance is established for vaccinated animals, thorough mechanical cleaning, washing and disinfection of premises, adjacent territories and vehicles located in them are carried out. Disinfection of premises and equipment is carried out every 5 days until the final disinfection. For this, a 3% hot solution of caustic soda is used; 2% formaldehyde solution; clarified solution of perchloric lime, containing at least 3% active chlorine, at the rate of 1 liter per 1 m3 of the room for 3-4 hours of exposure. Quarantine from a dysfunctional farm is lifted 40 days after the last death or forced slaughter of sick animals and the completion of all prescribed veterinary and sanitary measures.

Control questions and tasks.

1. Describe the epizootological features of enzootic encephalomyelitis of pigs.
2. Carry out a differential diagnosis of enzootic encephalomyelitis from other diseases with signs of damage to the central nervous system.
3. When is the diagnosis of enzootic encephalomyelitis considered established?
4. What vaccines are used against enzootic encephalomyelitis?
5. Describe the measures for the prevention and elimination of the disease.

Topic: Transmissible gastroenteritis of pigs

(diagnosis, prevention and control measures).

Transmissible gastroenteritis of pigs (Transmissible gastroenteritis suum, viral gastroenteritis, Doyle and Hutchings disease, TGS) is an acute highly contagious disease of pigs, which is characterized by signs of catarrhal and hemorrhagic gastroenteritis (exhausting diarrhea, vomiting, dehydration of the body) and high mortality of piglets in the first 10 days of life.

The causative agent of the disease. RNA-genomic virus from the Coronaviridae family. Virions are spherical in shape, covered with a lipoprotein shell with club-like outgrowths that give them the appearance of a solar corona. It reproduces in the cytoplasm of infected cells of the epithelium of the small intestine, especially in the duodenum and jejunum. Antigenically, the virus is homogeneous and close to intestinal coronavirus of dogs, respiratory coronavirus of humans and infectious peritonitis virus of cats. In the body of sick piglets, the virus accumulates in the epithelium of the small intestine, in the contents of the alimentary canal, and in the lungs. During the period of viremia, it is detected in parenchymal organs, as well as in the mucous membrane of the nose, trachea, tonsils, and in low titers - in the blood. In the internal organs and lymph nodes of infected animals, the virus persists for many months and years. The virus is cultivated in primary cultures of cells of the thyroid gland, kidneys and testicles of piglets, kidneys of pig embryos, and epithelial cells of the lungs. The virus is quite stable in the external environment: it remains viable at 4°C for 3 months, at room temperature for 45 days, in a lyophilized state and at 4°C for 30 days. The virus is stored for 10 days in the contents of the alimentary canal, during rotting and drying, for several weeks in frozen material. Sensitive to the action of light - at room temperature in the light, the infectivity of the virus decreases for 3 days by 90-95%, remaining unchanged in the dark under the same conditions. Under the influence of direct sunlight, the virus is destroyed in 1-2 days, in liquid manure in the sun - in 6 hours, in the shade - in 3 days, at 80-100°C - in 3-5 minutes. Unlike enteroviruses, it is

very sensitive to chloroform and ether, resistant to trypsin. It is quickly inactivated under the influence of ultraviolet radiation and various disinfectants.

The diagnosis of transmissible gastroenteritis is made by a complex method on the basis of epizootological, clinical, pathomorphological data and the results of laboratory studies.

Epizootological data. Only pigs get sick, especially sensitive piglets in the first week after birth. The source of the causative agent of infection is sick and sick pigs, which, starting from the incubation period and during the next 2-3 months, release the virus with feces and urine. In the first 6-7 days after farrowing, sows secrete the virus also with milk. Factors of transmission of the pathogen are fodder, water, equipment, bedding, slaughter products, food waste, vehicles and other objects contaminated with secretions of sick animals or virus carriers. Infection of piglets occurs through food and water, in direct contact with sick animals; airborne transmission of the pathogen cannot be ruled out. The disease is registered at any time of the year, but most often - at the beginning of spring, during mass farrowing and significant accumulation of the virus in the external environment due to its high resistance in the cold period of the year. An epizootological feature of transmissible gastroenteritis is a tendency to significant spread and high mortality among newborn piglets, with a benign course - among weaned piglets, piglets and adult pigs.

At the initial outbreak, the disease can spread quickly among pigs of all age groups, with almost 100% mortality in 1-10-day-old suckling piglets and 2-4% in weaned piglets. Outbreaks of the disease in epizootic centers disappear after 4-6 weeks after their occurrence. With further circulation of the virus in the farm and the presence of lactogenic immunity among suckling pigs, the disease is observed mainly among suckling pigs older than 1 month and weaned piglets. With the unsystematic implementation of health measures, transmissible gastroenteritis acquires a stationary character and systematically occurs among non-immune piglets. Viral gastroenteritis can be complicated by salmonellosis, colibacteriosis, dysentery and other infections.

Clinical signs and course of the disease. The incubation period for 1-5 day old piglets is 12-18 hours. In sick piglets, there is a decrease in body temperature to 36.8°C, depression, weakness, thirst, vomiting, profuse diarrhea, liquid, watery, foamy, yellow-green excrement with clots of colostrum (Fig. 1).

The color of the skin quickly changes from pink and shiny to brownish-gray, dull, the bristles become disheveled and dirty. Piglets die after 3-4 days. With age, the incubation period increases, mortality decreases: in 6-10-day-old piglets, the incubation period lasts 18-36 hours, mortality is 67%, in older animals - 7 days, mortality - 3.5%.

In the clinical course of the disease in piglets 6-10 days old, three stages are distinguished - preclinical, clinical and final. In the preclinical stage, there is a decrease in appetite, drowsiness, increased thirst, vomiting, and sometimes an elevated temperature up to 41-41.5°C. In the clinical stage, diarrhea and dehydration of the body

are strongly expressed. Feces are gray-red or yellow-green, contain gas bubbles. Sick piglets feel very thirsty, urinate milk, which in an undigested form appears in the feces. In the final stage, the disease ends with the recovery or death of sick piglets. Before death, a coma is often observed, which occurs on the 3rd or 4th day of the illness. When the piglets recover, diarrhea stops after 3-4 days, and villus regeneration begins. In the case of disease in piglets at the age of 1-2 weeks, the clinical signs are less pronounced, the course of the disease is much easier and most of the sick animals recover. Ill piglets do not eat and digest feed well, they lag sharply behind in growth.

Fig.1. Diarrhea.
(https://pigua.info/uk/post/comu-navcila-eds-uk)

In sows and boars, the course of infection is asymptomatic, although piglets born to them die within 2-5 days. In some sows, on the 3-5th day of lactation, agalactia is noted, sometimes mastitis, loss of appetite, depression, recovery occurs after 7-10 days.

Pathological changes. The carcasses of suckling piglets are skiny, the skin is gray, and the anus area is contaminated with yellow-green feces. Piglet, mucous membranes of the oral and nasal cavities are cyanotic. Subcutaneous tissue, skeletal muscles, peritoneum and pleura are dehydrated and dry. When dissecting corpses, patho-anatomical changes are noted mainly in the stomach and intestines. Clots of undigested colostrum, catarrhal or catarrhal-hemorrhagic inflammation of the mucous membrane, hemorrhages and necrotic cells at the bottom of the stomach are found in the stomach. Focal catarrhal and hemorrhagic inflammation of the mucous membrane, spotty hemorrhages are observed in the small intestine; the walls of the intestines are thin, transparent. The mucous membrane of the large intestine is full of blood or in a state of catarrhal and hemorrhagic inflammation. In the caecum and colon, surface necrosis in the form of bran-like plaque is detected. Mesenteric lymph nodes are

enlarged, hyperemic. Congestion in the spleen, hemorrhages. Small speckled hemorrhages under the kidney capsule, hyperemia, edema, hemorrhages in the brain.

In adult animals, catarrhal, rarely hemorrhagic gastroenteritis, degenerative changes in the kidneys are determined. During the histological examination, significant atrophy of the villi of the jejunum and ileum, as well as changes in the morphology of the epithelial cells, which acquire a cubic or rounded shape and contain numerous cytoplasmic vacuoles, are revealed. Pyknosis and lysis of nuclei, necrosis of individual epithelial cells is observed. In the brain, perivascular lymphocytic "couplings", foci of glial proliferation are detected.

Laboratory diagnostics. The laboratory diagnosis of transmissible gastroenteritis of pigs is based on the indication of the virus in the pathological material using immunofluorescence and bioassay, the isolation of the virus from the pathological material in cell cultures and its identification using the neutralization reaction, the detection of specific antibodies in the blood serum of sick animals (retrospective diagnosis).

For research, pieces of lungs, liver, spleen, kidneys, brain, and affected areas of small intestines, primarily duodenum, jejunum, and ileum, as well as mesenteric lymph nodes, taken from sick piglets slaughtered for diagnostic purposes, are sent to the laboratory, no later than 2 hours after moment of slaughter Pathological material is transported in tightly closed vials made of dark glass, in a thermos with ice or with liquid nitrogen. For retrospective diagnosis of the disease, paired blood sera of sick and sick animals are sent. The indication and identification of the viral antigen is carried out by the immunofluorescence reaction, the indirect hemagglutination reaction and the ELISA method. Most often, smears-prints and histological sections are prepared from pathological organs and tissues, as well as from infected cell cultures, which are stained with fluorescent specific serum. Immunofluorescence is considered positive if there is a bright emerald-green glow in the preparations of virus-affected cells or cytoplasm with a bright green glow of granules of different sizes in the absence of fluorescence in control (without viral antigen) preparations. Due to the fact that viral transmissible gastroenteritis is very often caused by virus strains that do not cause cytopathogenic effects in infected cell cultures, the most reliable method of diagnosing the disease is considered to be a bioassay conducted on 4-6 piglets 2-3 days old, which are infected orally or intranasally. The bioassay is considered positive if, 1-3 days after infection, piglets become ill with characteristic clinical signs (profuse diarrhea, vomiting, dehydration), followed by death in 3-5 days. An autopsy reveals catarrhal or catarrhal-hemorrhagic inflammation of the small intestine, and a histological examination reveals atrophy of intestinal villi. If necessary, isolate and identify the virus from pathological material from experimental piglets.

Differential diagnosis. Presupposes the need to rule out enterovirus gastroenteritis, colibacteriosis, dysentery, swine fever.

Enterovirus gastroenteritis mainly affects weaned piglets, is characterized by less contagiousness, anorexia, and nervous symptoms. With colibacteriosis, clinical signs of the disease are observed only in newborns and weaned piglets, while with transmissible gastroenteritis of pigs, sows can also get sick. During the bacteriological examination, seropathogenic strains of Escherichia coli are isolated. Effective treatment with antibiotics. Dysentery is clinically manifested in pigs of different age groups, is characterized by the presence of blood in feces, and high mortality. An autopsy revealed characteristic lesions of the large intestine in the form of ulcers and mucosal necrosis. Borrelia is isolated during bacteriological studies. Swine fever affects animals of all ages and is characterized by high mortality. The disease is accompanied by septicemia, high fever of a constant type, nervous phenomena, hemorrhagic diathesis, specific lesions of the large intestine ("buds"), spleen (infarcts) and lymph nodes ("marbling").

Treatment. Specific means of therapy are not offered. Symptomatic treatment is carried out, but it is ineffective in suckling piglets.

Immunity. Sick pigs develop immunity to re-infection for 2 years. In piglets, due to the extremely acute course of the disease, only passive lactogenic immunity is possible, which is ensured by the constant supply of secretory immunoglobulins of the IgA class with the colostrum of an immune sow. For vaccination of pregnant sows, live and inactivated vaccines against transmissible gastroenteritis are proposed, the use of which provides passive protection of offspring.

Prevention and control measures. Measures to prevent the disease of pigs from TGS are based on the protection of the farm from the introduction of the causative agent of the disease, the organization of separate farrowings of main and repair sows, compliance with the "free-busy" principle during farrowing, thorough mechanical cleaning and disinfection of farrowing premises during the preventive break, and as well as inventory and equipment in them. In order to control the epizootic condition in breeding farms, blood sera of 5% of the herd are selectively examined twice a year using an indirect hemagglutination reaction with erythrocyte antigen. The procedure for stocking the pig farm is established depending on the epizootic well-being of pigs of buyers and suppliers with regard to transmissible gastroenteritis. Pig farms free from transmissible gastroenteritis should be filled with pigs only from farms with the same epizootic condition, with mandatory serological tests for TGS during the 30-day preventive quarantine of imported livestock. In the case of detection of positively reacting animals, the entire imported group of pigs is not used to complete a TBS-free farm, but is slaughtered or transferred to a farm with a similar epizootic condition. In disadvantaged farms and farms with animals positively reacting to transmissible gastroenteritis of pigs, all breeding stock is vaccinated. After each farrowing cycle, the premises are completely cleared of animals and during the preventive break they are thoroughly cleaned and disinfected.

When an animal disease is detected on TGS, the farm is declared unhealthy for this disease and quarantine restrictions are introduced in it. It is prohibited to bring in and take out pigs, regroup the pig herd, and visit a dysfunctional farm by persons not related to animal care. Immediate measures are taken to eliminate those violations in the technology of keeping pigs that led to the occurrence of the disease in the farm. It is strictly forbidden to fill the brood stock with fattening sows, as well as continuous farrowing in the same premises, it is not allowed to violate the terms of preventive breaks and the deadlines for the rehabilitation of sow pens or isolated sections for farrowing. The corpses of dead animals are subjected to heat treatment or burned. All pregnant sows are vaccinated with a vaccine against transmissible gastroenteritis. If the disease occurs only among animals located in one isolated section, in order to prevent the further spread of infection, all animals of this section are sent for slaughter to a sanitary slaughterhouse or meat processing plant. In the event that it is impossible to slaughter all sick and suspected animal diseases in the premises where they are kept, no new animals are introduced for farrowing, the sick are treated symptomatically. In the future, the mother pig herd remaining after the outbreak of infection is sent for fattening and is not used for reproduction. The premises, after being completely freed from pigs, are thoroughly cleaned and disinfected.

The farm is declared free of transmissible gastroenteritis of pigs 3 months after the last case of death or recovery of sick animals and the final disinfection. During the next 3 months, it is prohibited to take pigs to other farms for fattening and during 12 months - for reproduction. For disinfection of livestock premises, use 4% solutions of caustic sodium or potassium, 20% suspension of freshly slaked lime, clarified solution of chlorinated lime containing 2% active chlorine, 3% hot solution of sulfur-carbolic mixture, 2% solution of formaldehyde after exposure for 3 hours. Manure is disinfected by the biothermal method.

Control questions and tasks.

1. What are the factors of transmission and the main ways of spread of the causative agent of transmissible gastroenteritis of pigs?
2. What are the differences in the clinical and epizootological manifestations of transmissible gastroenteritis in weanling piglets, weaned piglets and adult pigs during an acute outbreak of the disease and inpatient distress?
3. On the basis of which data is the final diagnosis of the disease considered established?
4. Describe the main principles of prevention and elimination of transmissible gastroenteritis in the economy.

Topic: Colienterotoxemia of piglets

(edematous disease)

(diagnosis, prevention and control measures).

Edema disease (Morbus oedematosus, colienterotoxemia) is an acute infectious disease of piglets, mainly of weaning age, which is characterized by a violation of the function of the central nervous system, enterotoxemia, the formation of edema in various organs and tissues.

The causative agent of the disease. Enteropathogenic β-hemolytic serovars of Escherichia coli, which belong to the Enterobacteriaceae family and are polymorphic, thick, motile, gram-negative rods with rounded ends, are placed in smears singly or in pairs. In some escherichia, a capsule is found, these bacteria do not form spores. Hemolytic strains of Escherichia coli produce hemolysin, necrotoxin, coagulase, synthesize colicin, as well as the enzyme histidine decarboxylase, which catalyzes the formation of histamine. Escherichia coli is cultured on conventional nutrient media, both in aerobic and anaerobic conditions. On meat-peptone agar after 16-24 hours, escherichia form colonies of grayish-blue or grayish-white color, with smooth edges and a convex, shiny surface. Escherichia growth in the broth is accompanied by uniform turbidity of the medium and the formation of a whitish amorphous sediment, which crumbles when shaken. Sometimes a very thin film forms on the surface of the broth. Escherichia are characterized by well-expressed enzymatic activity, break down various carbohydrates, and form indole. On the elective medium, Endo forms specific colonies of dark cherry or crimson-red color with a metallic sheen. Escherichia coli colonies are red on Zh (Ploskyrev) bactoagar, and blue or black on Levin's medium. 157 somatic O-antigens, 94 surface K-antigens and 50 flagellar H-antigens have been identified in E. coli, various combinations of which determine the specificity of individual strains of E. coli, their biological properties and belonging to certain serological groups or types. Determination of serotype specificity of escherichia is carried out using an agglutination reaction with O-antigens, which are characteristic for each serological group or type. Within the type, individual variants are distinguished by K- and H-antigens. Differentiation of Escherichia is extremely important because only certain serotypes are capable of causing massive gastrointestinal and septic diseases. It was established that enteropathogenic β-hemolytic strains of Escherichia coli of four serotypes - O 138, O 139, O 141 and O 26, which have pil antigens, are the causative agents of swine edema disease in Ukraine.

Escherichia are stable in the external environment, can be stored in soil, water, manure and livestock premises for 1-2 months. When milk is heated to 74-76°C, escherichia are destroyed after 15-20 seconds. Under the action of such disinfectants as a 4% hot solution of caustic soda or a clarified solution of chlorinated lime with a content of 3% active chlorine, inactivation of escherichia occurs in a few minutes.

The diagnosis is made by a complex method on the basis of epizootological data, characteristic clinical signs of the disease, patho-anatomical changes and the results of laboratory studies.

Epizootological data. Pigs of all age groups are susceptible to edema disease, but the most sensitive are piglets 8-10 weeks old, 3-10 days after weaning from the sow. The disease occurs unexpectedly in the household. The beginning of an outbreak is usually accompanied by the death of several piglets without the manifestation of clinical signs. In a short time (7-10 days), the disease spreads to a significant part of the livestock. First of all, well-fed piglets get sick. The source of the causative agent of the disease is sick and diseased bacteria-carrying animals. During enzootic disease outbreaks, hemolytic coliform carriers in clinically healthy sows reach 43.6%, in weaned piglets - 82.8%, suckling piglets - 25%. Clinically healthy bacteria-carrying pigs can be the reason for the spread of hemolytic strains of Escherichia coli within one farm and their introduction into healthy farms. Ways of transmission of the causative agent of infection have not been sufficiently studied. In the case of primary infection in the farm, weaned piglets first become ill, then young animals of other age groups (suckling piglets, gilts 4-6 months old). In inpatient facilities, 1-month-old suckling piglets, piglets, fattening pigs and sows are the first to get sick. Several piglets in one or more farms can get sick at the same time, but the spread of infection to the entire litter from one sow is rarely observed. Sometimes an outbreak is limited to piglets from only one nest or pen, in other cases the infection spreads to several pens, and not necessarily adjacent ones. The duration of an outbreak is from 1-2 weeks to 5-8 months. The seasonality of edematous disease is not pronounced, its outbreaks are most often associated with the farrowing period, several outbreaks of the disease can be observed in one and the same farm during one year. With edema, piglets rarely recover, most of them die within the first two days. Older pigs survive more often, their recovery is slow. The fatality rate ranges from 25.7 to 54.2%. It is believed that the occurrence of the disease is caused by sharp disturbances in the feeding and maintenance of piglets after weaning from the sow (change in feed ration, keeping in wet rooms, transportation, etc.).

Clinical signs and course of the disease. The incubation period lasts 6-10 hours. The course of the disease is hyperacute (lightning), acute and subacute. There are four forms of disease manifestation - intestinal, edematous, nervous, mixed.

The hyperacute course is observed in well-fed piglets, the animals suddenly die within 0.5-1 hour without showing characteristic symptoms of the disease. It is more common at the beginning of an enzootic outbreak of infection.

The acute course of edematous disease is the most typical. The disease lasts from several hours to 2-3 days. Typical clinical manifestations of one of these forms of the disease are noted. The intestinal form is found in piglets of average and below average fatness and is characterized by symptoms of digestive tract dysfunction. In sick animals, depression, a slight increase in body temperature, diarrhea, which often turns

into constipation, sometimes vomiting, abdominal distension are noted. A sharp depression of the sick animal, bluishness of the mucous membranes and skin in the area of the heel, ears, lower part of the abdominal cavity and the inner surface of the limbs occurs quite quickly. The duration of the disease is 2-3 days. The edematous form is found in well-fed piglets and is characterized by general weakness, conjunctivitis, swelling of the eyelids, subcutaneous tissue in the area of the head, chest, and abdominal wall (Fig. 1). Duration - 1-3 days. Almost all sick piglets die.

Fig.1. Conjunctivitis with swelling of the eyelids.
(https://lifehacker.org.ua/nabriakova-hvoroba-porosiat-simptomi-i-likyvannia-profilaktika/6/)

The nervous form is characterized by symptoms of damage to the central nervous system. In animals, hyperesthesia of the skin, depression, which is replaced by sudden excitement, tense shaky gait, circular movements, efforts to move forward are detected. Some of the sick animals lie in a state of numbness and react to even a slight touch of the skin with a sharp shudder of the whole body. Subsequently, epileptic seizures, muscle tremors, paresis and paralysis of the limbs appear, convulsive contractions of the muscles of mastication, muscles of the limbs, the head is often thrown back. Epileptic seizures do not last long, but very often repeat themselves until death. At the same time as paralysis, symptoms of asphyxia develop, the temperature gradually drops and the death of the animal occurs. In the case of a mixed form, symptoms of damage to the digestive tract, cardiovascular system and swelling of the eyelids are observed. Most sick piglets die after 6-8 hours.

The subacute course is more often registered in pigs of older age groups and in stable farms that are not healthy. The most characteristic manifestation of the disease is swelling of the eyelids (not in all animals) and conjunctivitis. Sometimes swelling is also found in the area of the nose, heel and pharynx. The duration of the disease is 5-7 days. In the subacute course of the disease, self-recovery is possible.

Pathological changes. They are characterized by the appearance of gelatinous swellings in various organs and tissues, mainly in the subcutaneous tissue of the head, meninges and brain substance, under the mucous membrane of the stomach, in the mesentery, and in the large intestine. Accumulation of a significant amount of pink or yellowish-red serous-fibrinous transudate in the thoracic, abdominal, and especially pericardial cavities. Some animals, mainly with the intestinal form of the disease, show

signs of catarrhal hemorrhagic gastroenteritis. When dissection of the stomach along the greater curvature, swelling of the submucosa with accumulation of gelatinous fluid is often observed, which causes a significant thickening of the stomach wall. In the area of edema, the mucous membrane of the stomach loses its wrinkling, is in a state of acute catarrh, in the area of the bottom of the stomach there is hemorrhagic inflammation. A serous swelling of the mesentery between the rings of the colon, which can cover the subserosal and muscular layer of the intestine, is particularly characteristic of swine edema disease (Fig. 2).

Fig.2. Inflating corpses.
(https://agroexpert.ua/ak-mozna-zahistiti-porosat-vid-vodanki-0/)

Laboratory diagnostics. It involves the selection of pure cultures of Escherichia coli, their serological typing, determination of hemolytic and pathogenic properties. Feces of diseased animals from the rectum or freshly excreted feces are sent to the laboratory for ex vivo diagnostics, for postmortem diagnostics – a fresh corpse of an animal (no later than 6 hours after death) or a thin section of intestines with contents, blood from the heart, mesenteric lymph nodes, parenchymal organs.

For intravital bacteriological research, cultures of feces suspension are carried out on Endo agar in Petri dishes, and after 18-24 hours - screening of typical colonies on myso-peptone agar and myso-peptone broth, followed by the study of mormological, tinctorial and cultural-biochemical properties of the selected cultures. For post-mortem bacteriological diagnosis, cultures from the intestinal contents and the thickness of the mucous membrane of the small intestines are carried out on Endo agar, and from mesenteric lymph nodes, blood and parenchymal organs - on myso-peptone broth and myso-peptone agar with rabbit blood to obtain pure hemolytic cultures of Escherichia coli.

Serological identification of selected Escherichia cultures is carried out with type-specific mono- and polyvalent agglutinating coli sera of the corresponding O- or OB-groups using dropwise (on glass) and linear (in test tubes) agglutination reactions.

A bacteriological diagnosis of edematous disease is considered to be established when β-hemolytic escherichia are isolated and characteristic patho-anatomical changes are present in dead pigs without determination of pathogenicity and serological

affiliation; in case of isolation of β-hemolytic escherichia, causing the death of at least two infected white mice, or belonging to O-serogroups recognized as pathogenic for animals.

Differential diagnosis. Presupposes the need to exclude table salt poisoning, as well as diseases such as listeriosis, Aujesky's disease, swine fever, Teshen's disease, other infectious diseases and vitamin B group.

Salt poisoning occurs mainly in weaned, exhausted piglets and piglets, and is accompanied by a malfunction of the nervous system (convulsions, paralysis, uncontrolled movements of the limbs) and the digestive tract (salivation, thirst, vomiting, diarrhea often with blood). Anamnestic data and the results of a chemical study of feed and the contents of the alimentary canal are of decisive importance for diagnosis. Listeriosis is characterized by contagiousness, an increase in body temperature up to 41-42°C, the appearance of smallpox-like lesions on the skin, nervous phenomena. Isolation of the culture of the causative agent of listeriosis during bacteriological studies of the internal organs and brain ensures the reliability of the diagnosis. Pigs of all age groups are affected by Aujeski's disease, but suckling piglets are the most susceptible. The disease is easily diagnosed with the help of a characteristic bioassay on rabbits. Swine fever is a highly contagious disease of animals of all age groups, during which a high mortality rate is observed among suckling piglets. Pathological changes such as hemorrhagic diathesis, splenic infarcts, croupous inflammation of the lungs, formation of ulcers with button-like scabs-buds in the colon at the place of solitary follicles are very characteristic of this disease. Teshen's disease occurs more often in the cold and wet season, is accompanied by high temperature, impaired coordination of movements; there are no diffuse swellings. Characteristic inflammatory phenomena in the brain, typical histological changes - dystrophy of ganglion cells in the gray matter of the brain and lesions of the spinal cord. The disease is easily reproduced in piglets weighing 8-10 kg with intracerebral injection of brain or spinal cord emulsion of diseased animals. During the differential diagnosis of edematous disease from dysentery, salmonellosis, pasteurellosis and leptospirosis, differences in the age-related susceptibility of pigs and the results of the selection of the causative agents of the respective diseases from the pathological material are taken into account. Avitaminoses are observed in winter and early spring, last for months, are accompanied by growth retardation of young, skin lesions, low mortality.

Treatment. No specific methods of therapy for porcine edema have been developed. It is believed that treatment can be effective only at the beginning of the disease, before the development of a nervous symptom complex. Various antibiotics (levomycetin, biomycin, monomycin, kanamycin, neomycin), sulfonamide drugs (sulfadimezin, norsulfasol, furazolidone) are recommended, which are more appropriate to use simultaneously, as well as in combination with polyvalent serum against colibacteriosis, B vitamins and various anti-stress drugs (aminazin, diphenhydramine, prednisolone). In the case of edematous disease, it is also

recommended to carry out complex preventive therapy. Sick animals are removed to a cozy room, kept on a starvation diet for 15-20 hours, then they are given Glauber's salt or magnesium sulfate in maximum therapeutic doses once. Feeding animals is not limited. In the first 5-7 days, reduce the rate of concentrates by 30-50%, which are replaced by juicy feed and lactic acid products, increase mineral and vitamin feeding. Internally, antibiotics neomycin, monomycin, colimycin, chloramphenicol in a dose of 0.015-0.020 g per 1 kg of weight 2-3 times a day for 3 days. Sulfadimezin in a dose of 1 g and furazolidone 0.25 g orally 2-3 times a day, 10% calcium chloride solution 20 ml twice a day or intramuscularly with a 3-4% solution of urotropin 5-10 ml are also used. It is good to introduce a 10% solution of calcium gluconate with a 1% solution of novocaine in a dose of 15-20 ml intramuscularly or intraperitoneally. After stopping the administration of antibiotics, acidophilic drugs are prescribed.

Immunity. A hydroxydaluminum formolthiomersal vaccine is proposed, which includes 9 strains of different serogroups of Escherichia.

Prevention and control measures. They should be aimed primarily at the elimination of unfavorable factors that lead to the occurrence of the disease. For this purpose, it is necessary to provide complete fodder rations for sows and piglets, paying special attention to the presence of vitamins and minerals, especially calcium. Do not allow piglets to be weaned before the age of 2 months, carry it out gradually, wean sows from piglets, and not the other way around, so that the piglets do not undergo sudden changes in the external environment. In dysfunctional farms, all weaned piglets are transferred to temporary full or partial starvation with a gradual transition to normal feeding rates, in no case allowing overfeeding with protein feed. Animals should be given laxative drugs (mainly salts), which is necessary not only to prevent edema, but also to accelerate the evacuation of the contents of the digestive tract and reduce the absorption of toxins. In the first days after fasting, it is recommended to give bran, pea, flax and other decoctions that envelop the mucous membrane of the intestines.

Quarantine restrictions are introduced in farms affected by edema disease, the movement of pigs within the farm and their export to other farms are prohibited. Sick piglets are isolated and comprehensive treatment is carried out. Special attention is paid to regular cleaning and disinfection of machines where sick piglets are kept. For disinfection, use 4% hot (70°C) sodium hydroxide solution, 4% formalin solution, 20% suspension of freshly slaked lime. Quarantine restrictions on farms affected by edematous disease are removed 1 month after the elimination of the disease, completion of all prescribed measures and final disinfection.

Control questions and tasks.

1. In piglets of what age and under what conditions of feeding and maintenance does this disease occur?
2. What clinical signs and patho-anatomical changes have diagnostic value?

3. From what diseases and according to what data is it necessary to differentiate the edematous disease of piglets?
4. Describe the methods and means of treating sick animals.
5. What set of measures should be taken to prevent the occurrence of the disease?

Topic: Swine dysentery

(diagnosis, prevention and control measures).

Swine dysentery (Dysenteria bacterialis suum) is a highly contagious disease of pigs, which is characterized by symptoms of acute catarrhal and hemorrhagic colitis and is manifested by profuse diarrhea with impurities of blood and mucus in the feces.

The causative agent of the disease. Borrelia hyodysenteria, which belongs to the family Treponemataceae. Borrelia are permanent residents of the alimentary canal of healthy piglets and show their pathogenicity only in case of a sharp decrease in the body's resistance. Borrelia are snake-like, anaerobic spirochetes with smooth, well-placed coils and sharp ends. Gram-negative, stain well with all aniline dyes, especially gentian violet. Borrelia are detected under a microscope in the dark or phase-contrast field of smears made from scrapings of the affected part of the mucous membrane of the large intestines, much less often - from the feces of sick pigs. They are cultivated in strictly anaerobic conditions, at a temperature of 42°C, on special nutrient media - tryptic-soy agar and broth with blood or blood serum of fetal cattle and spectomycin. Culture growth on tryptic-soy blood agar is observed 5-8 days after seeding in the form of a gentle diffuse plaque and is accompanied by clear hemolysis of erythrocytes; Borrelia colonies do not form. On tryptic-soy blood broth, the growth of the pathogen is detected after 2 days in the form of a grayish mucous sediment at the bottom of the test tube, which rises up in the form of a pigtail when shaken. After 3-4 days, the environment acquires a dark brown, almost black color.

The causative agent is unstable in the external environment. In the liquid feces of sick pigs, it remains viable at temperatures from 0 to +10°C – 48 days, at +5°C – 42 days, at +10°C – 36 days, at +25°C – up to one week. It remains viable in the manure for a long time. Laboratory animals are not susceptible to the causative agent of dysentery.

The diagnosis is made by a complex method based on the analysis of the results of an epizootological examination of a dysfunctional farm, the characteristic clinical picture of the disease, patho-anatomical changes and laboratory test data.

Epizootological data. Pigs of different ages are sick, especially young animals 1-6 months old. During enzootic outbreaks of dysentery, bloody diarrhea is also observed among suckling piglets. The appearance of the disease is often caused by sharp disturbances in the feeding and maintenance of pigs (change of feed, feed poisoning, overcrowding, drafts). The role of these factors is especially evident in

permanently dysfunctional farms. The source of the causative agent of infection is clinically and latently sick pigs and convalescents, which emit borrelia with feces up to 5 months after becoming ill. Infection of healthy pigs occurs alimentary, through feed, water, litter, animal care items contaminated with the pathogen. Dysentery is characterized by a very rapid spread of the disease, which takes the form of enzootic, sometimes epizootic. The disease is registered throughout the year, but most often in autumn, winter and spring. The appearance of the disease in previously prosperous farms is mainly associated with the importation of repair pigs and piglets for stocking. In these cases, the disease takes on the character of an epizootic with the coverage of pigs of all age groups within 1-3 days. Morbidity and mortality among suckling piglets can reach 80-100%, in sows and adult pigs the disease is more benign, with lower mortality. Later, in these farms, dysentery becomes a stationary infection, accompanied by the presence of a large number of microbe-carrying pigs, and regularly appears among young piglets 1-6 months old in the form of enzootic outbreaks. Morbidity among these age groups is 80-90%, mortality - 10-60%.

Clinical picture and course of the disease. The incubation period lasts 2-30 days. The course of the disease is acute, subacute and chronic. Cases of hyperacute course are possible, when animals die 12-20 hours after the appearance of bloody diarrhea.

During the acute course, a short-term increase in body temperature to 40.5-41°C, severe depression, lack of appetite, and thirst are noted. 48-82 hours after infection, severe diarrhea appears. Feces are initially dirty-gray in color, then become dark brown or earthy-black with admixtures of mucus and blood, which is the most characteristic diagnostic sign of dysentery (Fig. 1). Body temperature drops quickly, shaky gait, shortness of breath appear; the back is hunched, the stomach is pulled. On the 5-6th day of the disease, complete exhaustion and death of the animal occurs. Piglets aged 3-6 weeks are more seriously ill than piglets, but diarrhea with blood is more common in older animals. Many pigs, especially young ones, often have relapses of the disease after recovery.

During the subacute course, the body temperature is within the normal range or is reduced. Exhausting bloody diarrhea with impurities of mucus, strong thirst, lack of appetite, exhaustion, unsteady gait are observed. The duration of the disease is 15-17 days. Most animals die in 12-15 days.

During the chronic course, diarrhea and constipation, exhaustion, lethargy, possible eczematous skin lesions. Blood in feces is rarely found and only in individual pigs. The duration of the illness is 14 days. Cases of complications of dysentery with salmonellosis, pasteurellosis and other secondary infections are registered.

Fig.1. Fecal masses in dysentery.
(https://uvt.com.ua/svini/kategorii-bolezney-sviney/zabolevaniya-organov-pishchevareniya/dizenteriya/diagnostika-dizenterii-sviney/)

Pathological changes. During the examination of the corpse, general exhaustion, bluish skin in the area of the ears and the lower part of the body are revealed. At the autopsy of the corpse, the main patho-anatomical changes are revealed in the large intestine (Fig. 2). In the acute course of the disease, thickening, hemorrhagic inflammation and folding of the mucous membrane of the colon and cecum, later - diphtheritic bran-like layering and foci of necrosis; hemorrhages in lymph nodes and some organs. In the chronic course of the disease, cheesy plaques of black and white, yellowish or red color, hemorrhages, as well as degenerative changes in the liver and kidneys are noted in the colon. The contents of the intestines have a reddish, sometimes coffee color due to blood impurities.

Fig 2. Diphtheritic layering in the large intestine.
(https://agrotimes.ua/article/likuyemo-dyzenteriyu-bez-antybiotykiv/)

Laboratory diagnostics. Includes detection of Borrelia during microscopic examination of pathological material using phase contrast or dark field condenser. For lifelong diagnosis, feces are taken from the rectum of sick animals, for postmortem – the mucous membrane of the large colon and cecum of deceased, or better, sick pigs slaughtered for diagnostic purposes.

From the mucous membrane, washed with running water, scrapings are made, and then a 10% suspension in physiological solution, which is immediately examined

under a "dark field" microscope in its native form or in a light microscope after Gram, Romanowski-Giemsa or Pfeiffer fuchsin staining. In the case of positive results in native preparations, 5-10 or more Borrelia are found in the crushed drop in the form of long winding threads with evenly spaced curls and sharp ends, which have translational movement. Gram-negative microorganisms with a morphology characteristic of Borrelia are detected in the fixed preparations.

Differential diagnosis. Dysentery must be differentiated from plague, salmonellosis, viral transmissible gastroenteritis, colienteritis, and also food poisoning.

With plague, pigs of all age groups get sick and die, constant fever, hemorrhagic diathesis phenomena, massive hemorrhages in all organs and tissues, spleen infarctions, "marbling" of lymph nodes, presence of "buds" in the mucous membrane of the large intestine are observed. Salmonellosis mainly affects newly weaned piglets, sows may have abortions. High body temperature, blueness of the skin of the ears, chest, and perineum are observed. In the chronic course of the disease, necrosis of solitary follicles of the mucous membrane of the colon with the formation of brittle scabs and ulcers is revealed. Bacteriological studies establish the causative agent of the disease.

With viral transmissible gastroenteritis, only suckling piglets under 15 days of age get sick and die. There is no blood in the fecal masses, no diphtheritic and necrotic lesions of the large intestine are found at autopsy. Piglets under the age of 15 are mainly affected by colienteritis. Fecal masses are liquid, without blood and mucus. During the bacteriological examination, a pathogenic Escherichia coli is isolated. Gastroenteritis of non-infectious origin occurs after feeding poor-quality feed, affects all pigs of the disadvantaged group at the same time, is accompanied by diarrhea without blood impurities, stops after the elimination of poor-quality feed from the diet.

Treatment. Osarsol, vetdipasfen, nifulin, emgal, tilan, pharmazine-200, trichopol, various antibiotics (biomycin, chloramphenicol) and sulfonamide drugs (furazolidone) are successfully used for therapeutic and preventive purposes. Osarsol is considered the best, which, if used in a timely manner, prevents the death of pigs. It is used twice a day for 3 days in a row with feed in doses: for piglets 30-60 days old for treatment - 0.1-0.2 g, for preventive purposes - 0.1 g; 3-4 months old 0.2-0.3 and 0.1; 4-8 months old – 0.3-0.4 and 0.25; 8-12 months –0.4-0.5 and 0.3; older than 12 months 0.5-0.7 and 0.4. In case of incomplete healing, Osarsol is given again after 5-6 days.

Nifulin is added to feed for therapeutic purposes in a dose of 5 kg per 1 ton of compound feed, for preventive purposes - 2 kg per 1 ton; apply twice a day for 7 days in a row. Severely ill animals are given individually, in a dose of 100-120 mg per 1 kg of weight once a day, for up to 5 days in a row. After 21 days, the administration of the drug is repeated in a half dose. Tylan is used with feed for the treatment of adult animals, for suckling piglets - individually with water twice a day, 3 days in a row, in doses of 1.25-2.5 mg per 1 kg of weight. Administration is repeated after 3 days. After

treatment, animals cannot be slaughtered for meat earlier than 21 days later. Farmazine is used for suckling piglets individually for therapeutic and preventive purposes 3-5 days in a row, diluted in doses of 0.25-0.5 g in 1 liter of boiled water. Emgal is fed to clinically sick pigs at a dose of 4-5 kg per 1 ton of feed for 10 days in a row. For preventive purposes, emgal is added in a dose of 2 kg per 1 ton of feed. Trichopolum is used for prophylactic and therapeutic purposes in adult pigs - orally and intramuscularly. It is given orally with food for 3 days in a row, twice a day in doses of 0.25-0.5 g. If necessary, the course of treatment is repeated after 7-10 days. Intramuscular use is possible. Biomycin chloride is administered orally at the rate of 20 mg per 1 kg of the animal's body weight twice a day; Levomycetin sodium succinate is used at the rate of 20 mg per 1 kg of body weight in the morning and in the evening for 3 days in a row intramuscularly. Furazolidone is prescribed immediately after treatment with osarsol at a dose of 3-5 mg per 1 kg of weight, twice a day, for 3-5 days in a row to prevent relapses of the disease.

Immunity. Means of specific prevention have not been developed.

Prevention and control measures. Due to the lack of specific drugs, the prevention of dysentery is based exclusively on the strict observance of veterinary and sanitary rules for stocking and breeding animals. All efforts should be aimed primarily at preventing the introduction of pigs from dysentery-prone farms to the pig farm. During the 30-day quarantine period, it is advisable to conduct a bioassay by adding 5-10 healthy piglets from your farm to the imported group and organizing veterinary monitoring of them. If there are no cases of swine disease during the quarantine period, as well as negative results of laboratory examination, the imported pigs are considered uninfected. During the period of growing piglets, it is necessary to monitor compliance with the normative indicators of the microclimate of the premises, complete feeding of pigs, systematic preventive disinfection, disinsection and deratization. Purchased breeding stock should be subjected to preventive treatment with trichopol, pharmazine, dynamitulin with simultaneous disinfection of the skin with a solution of alkaline formalin. When dysentery occurs, all sick pigs with bloody diarrhea are immediately isolated and sent for slaughter. In the machines where the sick pigs were, thorough mechanical cleaning and disinfection are carried out, and urgent measures are taken to clarify the diagnosis. After receiving the final diagnosis from the laboratory, the farm is declared unhealthy for dysentery, quarantine restrictions are introduced in it, which prohibit regrouping, import and export of pigs, and the use of sick pigs for reproduction. All pigs in a poor facility are treated prophylactically with one of the anti-dysenteric drugs. The next day, all treated pigs are transferred to clean, disinfected premises or taken to summer camps. After 10-14 days, the treatment of animals and disinfection of their skin is repeated. After the end of the summer camp period, the pigs are again treated with anti-dysentery drugs, washed in an alkaline solution of formalin and placed in renovated and disinfected premises. 7 days before farrowing, sows are given anti-dysenteric drugs, bathed in an alkaline formalin solution.

The farm is declared safe and restrictions are removed from it 3 months after the last case of swine dysentery was detected. Special attention should be paid to the rehabilitation of breeding and breeding farms that are not affected by dysentery. The most expedient is the complete replacement of the mother herd of pigs by importing breeding young from previously prosperous farms and their isolated cultivation. If it is not possible to replace, the gradual formation of a new, infection-free breeding nucleus is carried out due to the selection and isolated maintenance of dysentery-free sows over the age of 1.5-2 years, the establishment of permanent laboratory control and monitoring of the condition of the young obtained from them.

In case of dysentery, a 4% hot solution of caustic soda is used to disinfect the premises; chlorinated lime solution containing 3% active chlorine; 10% disinfectant creolin emulsion; 20% suspension of freshly slaked lime; 2% formaldehyde solution; soap-carbol mixture. Disinfection can be carried out by the aerosol method. For aerosol disinfection, formaldehyde is used at the rate of 15 ml per 1 m3 for exposure of 6 hours, followed by bleaching of cages with a 20% suspension of freshly slaked lime. Manure from sick pigs is disinfected with chlorinated lime.

Control questions and tasks.

1. Give a description of the causative agent and the conditions for the occurrence of the disease in a previously prosperous household.
2. What are the clinical and epizootological features of swine dysentery?
3. How is the diagnosis and differential diagnosis made for this disease?
4. How to rehabilitate a pig farm suffering from dysentery?

Topic: Enzootic pneumonia of pigs

(diagnosis, prevention and control measures).

Enzootic pneumonia of pigs (mycoplasma pneumonia, respiratory mycoplasmosis, mycoplasmosis of pigs (pneumonia enzootica suum)) is a chronic infectious disease characterized by inflammation of the lungs, serous membranes and impaired reproductive function in sows.

The causative agent of the disease. Mycoplasma hyopneumoniae from the Mycoplasmataceae family. It is characterized by pronounced polymorphism. Spherical, star-shaped, thread-like and other forms are found in smears from the culture. Mycoplasmas are gram-positive, they stain well according to Romanovsky-Giemza and Dins. In smears-imprints from the affected lungs, the pathogen is represented in the form of small cocci, ring-shaped and spherical formations. The causative agent is cultivated on special nutrient media enriched with serum. Primary isolation of it from pathological material poses significant difficulties; growth is slow (7–10 days). The pathogen is cultivated in anaerobic conditions. On agar, Mycoplasma

hyopneumoniae forms small colonies in which, unlike other types of mycoplasmas, there is no thickening of the central part. The causative agent ferments glucose with the formation of acid.

Mycoplasma hyopneumoniae is very sensitive to the influence of external factors. Antibiotics of the tylosin group and tetracycline drugs inhibit the growth of mycoplasma, but it is resistant to penicillin, streptomycin, polymyxin, neomycin, and thallium acetate. The survival time of the pathogen in the environment depends on the temperature, humidity and intensity of ultraviolet rays. At a temperature of 5-10 C and a humidity of 75-80%, it remains viable for 28 days, in litter - 1-5 days, at minus 20 C it is stored for months. Heating to a temperature of 50 C or more kills the pathogen instantly.

The diagnosis of enzootic pneumonia of pigs is made by a complex method on the basis of epizootic, clinical, patho-anatomical data. Laboratory studies (bioassay on piglets, bacteriological and serological research) are of decisive importance.

Epizootological data. Under natural conditions, suckling piglets, weaned piglets and piglets up to 6-7 months of age are affected by enzootic pneumonia. Adult pigs are resistant to the pathogen, can get sick infrequently and relatively easily. The source of Mycoplasma hyopneumoniae is sick, terminally ill and latently infected pigs, in whose body mycoplasmas can persist for up to 13-15 months, which secrete the pathogen during sneezing, coughing, as well as with milk and mucus from the vagina. The main route of infection is aerogenous. Piglets are infected in the first days of life from their mothers and when they are kept together with sick animals. Especially dangerous are sick animals in which manifest forms of the course of the disease (typical clinical signs) are not manifested, carriers in which are mostly lifelong. In farms, mycoplasma is isolated from both sick and clinically healthy animals. In dysfunctional farms, the disease covers from 30 to 80% of the pig herd, the death of piglets ranges from 3-5 to 15-30%. In sick piglets, growth decreases by 16%, and feed consumption increases by 22%.

Enzootic swine pneumonia is a typical respiratory infection. Inside the farm, the pathogen is mainly spread by airborne droplets. In the spread of enzootic pneumonia, the regrouping and introduction into the herd of mycoplasma-carrying pigs purchased from dysfunctional farms are of particular importance. Such situations often occur in fattening farms, and therefore the disease takes on the character of an epizootic outbreak.

In modern pig farming, epizootics of enzootic pneumonia do not have a pronounced seasonality - the disease occurs at any time of the year. The epizootic process of enzootic swine pneumonia is characterized by the stationarity of epizootic foci and variation in its intensity from sporadic to widespread disease. In dysfunctional breeding farms, the greatest morbidity occurs during the technological periods of mass farrowing and weaning of young animals, and in fattening farms (in the absence of a full production cycle) - during the first 2-3 months. after the formation of fattening

groups of animals. The speed of spread and the severity of the course of the disease depends on many factors: the young age of the animals; keeping in poorly ventilated and cold rooms on cement floors; poor nutrition; placement of piglets after weaning in significant heterogeneous groups. Under unsatisfactory housing and feeding conditions, enzootic pneumonia manifests itself as an infection complicated by various secondary microflora (Pasterel, Bordetella, streptococci, staphylococci, etc.) with the development of severe forms of pneumonia, in this case the mortality rate can increase to 80-90%.

Clinical signs and course of the disease. The incubation period for suckling piglets lasts from 8 to 56 days or more. The course of the disease is mostly subacute and chronic. The first signs of the disease appear gradually, between the third and tenth weeks of the piglets' life in the form of depression, decreased appetite, slight hyperthermia, sneezing and rare dry (paroxysmal unproductive) cough (subacute course). Up to two weeks, piglets still eat feed well, the general condition is satisfactory. In the future, the course of the disease becomes more severe, signs of lung damage begin to prevail. The cough becomes wet.

The chronic course lasts several weeks and even months. The main symptom is a cough, strong attacks of which are observed every morning during animal feeding and walks. Breathing becomes heavy and accelerated, the animals stand with their legs wide apart. In sick piglets, depression, remititive type of fever, abdominal type of breathing, suffocation are observed, they eat feed poorly, lose weight and lag behind in growth. Such animals often develop mucopurulent conjunctivitis, eczema, and the formation of scabs. In the case of a bacterial complication, the symptoms of pneumonia progress and the course of the disease may worsen. They are especially pronounced during a mixed infection of Mycoplasma hyopneumoniae with Pasteurella, Bordetella and Corynebacteria. Such mixed infections most often develop among fattening pigs and cause significant economic losses. Sometimes such piglets develop digestive disorders in the form of diarrhea and constipation.

Pathological changes. They depend on the stage and duration of the disease, the severity of clinical signs and the presence of a mixed infection. In the initial stage, lobular or lobar serous-catarrhal pneumonia is detected with the predominant localization of inflammation in the cardiac and apical lobes, the front edges of the diaphragmatic lobes (Fig. 1). The affected areas of the lungs are compacted, grayish-pink in color, with a bluish tint and a significant accumulation of turbid-foamy liquid.

Fig. 1. Lobar serous catarrhal pneumonia.
(https://eurovet.com.ua/novini/silsko-gospodarski-tvarini/enzootichna-pnevmonija-svinej/)

Respiratory tract without visible changes. Only moderate focal hyperemia of the mucous membrane of the trachea and large bronchi is revealed. Their lumens contain a relatively small amount of viscous, grayish, rarely bloody liquid. Kidney, spleen, liver and brain without visible changes. During the histological examination, clear lymphoid-monocytic proliferation is revealed. In the case of complications of the primary process, signs of serous inflammation of the cervical lymph nodes, catarrhal-purulent lobar pneumonia, infrequently adhesive pleurisy and pericarditis dominate. Bronchial lymph nodes are greatly enlarged. The corpse is exhausted, anemic, parenchymatous organs are regenerated. Histological examination of parenchymal organs shows significant infiltration of them by macrophages, including epithelioid and giant multinucleated cells, as well as lymphocytes. In chronic cases, when the inflammatory process is complicated by bacterial microflora, peritonitis, pleurisy and purulent-necrotic processes in the lungs develop.

Laboratory diagnostics. From the material delivered to the veterinary medicine laboratory (lungs), a 10% suspension is prepared (from the affected areas) in a phosphate buffer solution (pH 7.2), to which 500 units/cm3 of penicillin and 100 units/cm3 of streptomycin are added. After 12-18 hours of exposure in a plus refrigerator (2-4 C), control cultures are carried out on myso-peptone broth, myso-peptone agar and Keith-Tarozzi medium for the purpose of sanitation of foreign microflora and on a special medium for the isolation of mycoplasmas. Final identification of selected strains is carried out using serological reactions: mycoplasma growth inhibition reaction, complement binding reaction, etc. Indication of mycoplasmas in pathological material is also carried out using the immunodiffusion reaction, enzyme immunoassay, DNA probes and polymerase chain reaction.

Differential diagnosis. It is necessary to exclude influenza, Aujeski's disease, pasteurellosis, salmonellosis, infectious atrophic rhinitis, actinobacillus pleuropneumonia of pigs, hemophilic polyserositis, ascariasis and pneumonia of non-

infectious etiology. When differentiating enzootic pneumonia from swine flu, it is taken into account that pigs of all age groups are affected by the latter (although 2-8 week old animals are most sensitive). Chicken embryos, cell culture, laboratory animals (white mice, hamsters) can be used to isolate the influenza virus. Indication of the influenza virus is carried out with the use of enzyme-linked immunosorbent assay. All age groups of pigs can be affected by Aujeski's disease. The disease is most malignant in suckling (up to 10 days old) piglets, in groups of which morbidity and mortality can be up to 100%. In sick animals, a constant type of fever prevails. Along with the respiratory form of the course, symptoms of damage to the central nervous system are observed (stupor-like and epileptic forms of the course). Other types of animals can get sick. Finally, the virus can be isolated on cell culture, chicken embryos, and bioassayed on rabbits or kittens. Indication of Aujeski's disease virus directly from pathological material can be carried out using enzyme immunoassay. Infectious atrophic rhinitis is characterized by specific clinical signs - pug-like and crooked nose. In the subacute and chronic course of infectious atrophic rhinitis, signs of inflammation of the middle or inner ear are noted, in this case the animal hangs its head to the side, the ear hangs down. As a result of the damage to the central nervous system, the coordination of movements is disturbed, paralysis, mange movements are manifested. In hot weather there are nosebleeds. Finally, a complete bacteriological examination is carried out. Pigs die in 1-2 days during an acute course of swine pasteurellosis. The body temperature of sick animals rises to 42°C, the pulse and breathing accelerate. The appearance of hot, painful swellings in the neck area is possible. During the development of edema, breathing becomes difficult, accompanied by wheezing. Sick animals assume the posture of a sitting dog. At autopsy, hemorrhages under serous coverings. The lungs are also hemorrhagic, swollen. Croupous pneumonia occurs during the subacute course. During the laboratory study, it is taken into account that white mice, rabbits, guinea pigs, and pigeons are sensitive to the causative agent of pasteurellosis. The acute course of salmonellosis in pigs is characterized by high fever, loss of appetite, inflammation of the mucous membrane of the eyes, depression and diarrhea. The chronic course is characterized by the development of rhinitis and lung damage. Pay attention to the bluish-red color of the bottom of the belly, heel, limbs, ears. An autopsy revealed foci of necrosis in the mesenteric lymph nodes and liver. In the lungs, the affected areas are located in the front and heart lobes, they are reddened, compacted, and gray-yellow cheesy foci are visible on their section. The development of fibrinous pleurisy and pericarditis is possible. The causative agent of salmonellosis can easily be isolated on the selective media of Endo, Levin, and Ploskirev. During the laboratory study, it is taken into account that white mice, rabbits, and guinea pigs are sensitive to the causative agent of salmonellosis. When differentiating enzootic pneumonia from actinobacillary pleuropneumonia, it is taken into account that during the latter, at autopsy, the accumulation of bloody fluid in the chest cavity (hemorrhagic component), in the lungs hemorrhagic cells the size of a chicken egg, mainly in the

diaphragmatic lobes (mostly in the right), in the bronchi and trachea - blood clots, in the lungs - blood saturated with air bubbles. With hemophilic polyserositis in piglets, during the acute course, low fatness is detected, in the heart sac, chest and abdominal cavities, a large amount of cloudy or straw-colored liquid with fibrin flakes. During the subacute and chronic course of the disease, there is little fluid in the cavities, but the deposition of fibrin films on the heart, pleura, and intestines attracts attention. The intestinal loops are connected by fibrinous membranes, and the pericardial sac fuses with the heart. Catarrhal bronchopneumonia and joint damage are found in many piglets. The leading patho-anatomical signs of hemophilic polyserositis are: absence of sedation; the presence of a significant amount of exudate in the chest and abdominal cavities with the accumulation of fibrin and its deposition on serous covers and organs; adhesive inflammation of the pleura and pericardium, pulmonary and costal pleura, intestinal loops.

Treatment. Despite the high sensitivity of M. hyopneumoniae to broad-spectrum antibiotics (oxytetracycline, tylan tartrate and tylosin phosphate, tiamutin, lincomycin, spiramycin, chloramphenicol, tetracycline), sulfonamide drugs (ethazol, norsulfazole, sulfamethazine, etc.), as well as aluminum iodide and chloramine B, in practical conditions they only prevent the development of clinical signs of the disease, but do not prevent infection and do not rid the animal's body of the pathogen. To increase the effectiveness of the treatment, symptomatic therapy is carried out, as well as means against bacterial secondary infections are used. Severely ill animals are culled.

Immunity. The presence of resistance in pigs infected with enzootic pneumonia to re-infection with the causative agent, the connection of immunity with a high titer of complement-binding antibodies, the direct relationship between the release of the pig's body from the causative agent and the intensity of the agglutination reaction open the possibility of herd recovery using specific immunoprophylaxis. The use of vaccination in modern pig farming is the most economically and epizootically justified way of controlling enzootic pneumonia in pigs. Practice has shown the high effectiveness of inactivated vaccines against enzootic pneumonia, the use of which significantly reduces lung damage in pigs. For the purpose of prevention, the company Bioveta a.s. (Czech Republic) offers the vaccine Biosuis M. Hyo. One dose contains 2 cm3 of inactivated Mycoplasma hyopneumoniae strain. Vaccination of fattening pigs, starting from the age of 10 days, in a single dose, by intramuscular injections in the neck area behind the auricle. In vaccinated animals, permanent immunity is formed 14 days after vaccination and lasts for 6 months. To maintain immunity, revaccination with a single vaccination every six months is recommended. In some cases, with a complex epizootic situation, it is possible to vaccinate animals starting from the age of 7 days in a dose of 2 ml twice with an interval of 3 weeks.

Prevention and control measures. All preventive and health measures for enzootic swine pneumonia are aimed at breaking the epizootic chain, remediating the environment with the aim of reducing the level of infection and increasing the natural

resistance of animals. In the case of preventive quarantine, it is necessary to carry out a thorough investigation in order to identify possible mycoplasma carriers. It is also necessary to strictly approach the selection of farms-suppliers and not to import young animals for fattening from farms where respiratory diseases are registered. The prevention system should include periodic clinical examinations of animals and timely isolation of sick and suspected animals, carrying out differential diagnostics and providing animals with complete feed and satisfactory housing conditions. Particular attention is paid to the microclimate, compliance with the temperature regime and the condition of the floors. The method of separate keeping of pigs by age and production groups should be strictly followed, and overcrowding should not be allowed. The fight against enzootic pneumonia should be carried out comprehensively. In addition to vaccine prophylaxis and antibiotic treatment, it is necessary to constantly monitor compliance with sanitary and hygienic norms of the microclimate in the premises.

When enzootic swine pneumonia appears, the farm is declared unhealthy and restrictions are imposed on the export of animals to other farms for reproduction and fattening. A thorough clinical examination of the entire pig herd is carried out. Sick animals with severe lung damage are slaughtered, those suspected of having the disease are treated, fattened in isolated conditions and sent to slaughter. Those suspected of infection are vaccinated with inactivated vaccines. Premixes are used for preventive purposes. If signs of enzootic pneumonia appear among piglets in any nest, the sow with all piglets is transferred to a fattening farm. In order to timely identify problems in the farm, it is sometimes recommended to carry out a diagnostic slaughter of piglets 2-3 months old. A breeding farm is considered healthy if piglets that are healthy in terms of respiratory diseases are obtained after the first and second farrowings.

Improvement of the economy can also be carried out by simultaneously replacing the entire livestock with pigs from prosperous farms. This method also requires a careful approach, taking into account the specific conditions and capabilities of each farm. The introduction of healthy animals is allowed only after the elimination of the unhealthy herd and thorough sanitation of the premises and territory of the farm from the pathogen. For disinfection, solutions of caustic soda (2%), formaldehyde (2%) and chlorinated lime containing 2% active chlorine are used. The farm is declared healthy after 2 months. after the importation of healthy pigs and their lack of signs of respiratory diseases.

Control questions and tasks.

1. Describe the pathogenicity factors of mycoplasmas and their impact on the animal body.
2. Describe the etiology, epizootological features, differential diagnosis, and treatment of enzootic swine pneumonia.
3. Give a comparative assessment of the methods of diagnosis of enzootic pneumonia of pigs?

4. What is the system of preventive, therapeutic and health measures for enzootic pneumonia of pigs?

Topic: Haemophilic polyserositis of pigs (Glesser's disease)

(diagnosis, prevention and control measures).

Hemophilic polyserositis (Poliserositis haemophilosis) is an acute septic disease of weaned piglets, characterized by serous-fibrinous inflammation of serous membranes (peritoneum, pleura, pericardium), joints and non-purulent meningoencephalitis.

The causative agent of the disease. Haemophilus parasuis - belongs to the family Brucellaceae, genus Haemophilus, is a permanent inhabitant of the upper respiratory tract of clinically healthy pigs, has a pronounced tropism to serous membranes, peritoneum, pleura and pericardium. The hemophilic bacterium is a stationary gram-negative aerobic bacillus, which is surrounded by a capsule and does not form spores. 5 capsular serogroups of the causative agent have been established - A, B, C, D, E, while strains of serogroups A and D were isolated from the affected lungs, and strains of group B - from septic manifestations of the disease. The degree of virulence of the strain was not dependent on serogroup affiliation. Among laboratory animals, guinea pigs are sensitive to Haemophilus bacillus, especially in case of intranasal infection.

For the cultivation of the causative agent of the disease, selective nutrient media are used - chocolate agar, broth and Liventhal agar, as well as blood or serum meat-peptone agar. On blood and serum agar, the pathogen grows in the form of small dew-like colonies without causing hemolysis of erythrocytes. On chocolate agar, within 24-48 hours, it forms large and small grayish-white rounded mucous colonies with smooth edges and a smooth convex surface. On Levinthal agar, after 24 hours, it forms transparent, shiny, fluorescent M-form and S-form colonies, as well as non-fluorescent R-forms. In smears from agar culture, bacteria are found in the form of filaments of various sizes and small gram-negative rods. In serum broth and Levinthal broth, hemophilic bacteria after 24 h of growth cause uniform turbidity of the media followed by clarification. Hemophilic bacilli are quite stable in the external environment, especially at low temperatures. Disinfectants inactivate them within 1-3 hours.

The diagnosis is made by a complex method on the basis of epizootological and clinical data, patho-anatomical changes, results of laboratory studies.

Epizootological data. Hemophilic polyserositis affects piglets up to 3 months of age, more often 8-15 days after weaning from the sow. The main source of the pathogen is piglets suffering from hemophilia, as well as sows and repair pigs, 40-70% of which are found to be carriers of the hemophilus bacillus. An outbreak of the disease is often

observed when new piglets are introduced into groups of newly weaned piglets. Spontaneous infection occurs aerogenously, but oral infection of piglets through feed and water contaminated with the pathogen is possible. The disease is registered at different times of the year, mainly during severe cold weather. An important factor that contributes to the appearance of the disease is a decrease in the body's resistance due to various violations in the maintenance of piglets - long transportation, sudden changes in temperature, significant deviations from the normative indicators of the microclimate. Hemophilosis is registered as sporadic cases or minor enzootics. At the same time, individual weak piglets first get sick, and later, with an increase in the virulence of the pathogen due to passage, the number of sick piglets increases rapidly and can reach 70%, and the mortality rate is 50%. A characteristic epizootic feature of hemophilic polyserositis is a wide and long-term bacteremia and the possibility of a mixed course with other infectious diseases.

Clinical signs and course of the disease. The incubation period is 5-7 days. Acute and subacute courses of the disease are distinguished.

The acute course is characterized by an increase in body temperature up to 40.5-41.5°C, depression, refusal to feed, accelerated heavy breathing, coughing, sneezing, sometimes vomiting. As the transudate accumulates in the abdominal and thoracic cavities, the pain of their walls increases, peritonitis, pleurisy develop, and cardiac impulses appear with significant difficulties. Piglets acquire a characteristic "sitting dog" posture, bring their hind limbs under themselves, move with great care. Swelling appears in various areas of the ears, muzzle, limbs and abdominal wall. Most of the sick piglets die within the first day.

The subacute course is manifested by fever, arthritis, severe limping, sometimes complete loss of the ability to move, exhaustion, signs of damage to the central nervous system. Some piglets die within 4-8 days, most recover. However, later piglets develop intestinal adhesions, pericardium and epicardium, they lag behind in growth and are culled.

Pathological changes. When dissecting the corpses of piglets that died in the first days of the disease, in the abdominal and pleural cavities, pericardial sac, accumulation of a significant amount of cloudy liquid with fibrin flakes, serous-fibrinous inflammation of the pleura, peritoneum, and pericardium is revealed (Fig. 1). In the subacute course, adhesive inflammation of the serous membranes and adjacent organs is observed, catarrhal pneumonia, damage to the joints, most often the hock, sometimes meningitis are observed.

Laboratory diagnostics. Includes microscopy of swabs from pathological material, selection and identification of the culture of the pathogen, determination of its virulence for laboratory animals. 2-3 corpses of piglets or samples of exudate from the abdominal, pleural and pericardial cavities, synovial fluid from the affected joints, scrapings from the surface of the affected serous membranes (pleura, pericardium,

pericardium) are sent to the laboratory in a thermos with ice, which are taken no later than 4 - 6 hours after the death of the animal.

Pleural effusion. Catarrhal pneumonia.

Fig.1. Pathological-anatomical changes in hemophilic polyserositis.
(https://uvt.com.ua/svini/kategorii-bolezney-sviney/infektsionnye-zabolevaniyas/gemofileznyy-poliserozit/diagnostika-gemofileznogo-poliserozita-sviney/)

In the presence of a clinical picture of damage to the central nervous system, the brain and contents of the cerebral ventricles are taken. During the microscopic examination of smears made from pathological material, the causative agent of hemophilic polyserositis is found in the form of small polymorphic gram-negative rods, diplobacteria, and short chains. The growth of hemophilic bacteria on blood agar is observed after 24 hours of culture incubation in the form of small round colonies with a convex smooth surface.

The virulence of the isolated hemophilic culture is determined by intraperitoneal infection of three guinea pigs. A culture is considered virulent if one or more guinea pigs die within 5 days. A laboratory diagnosis of hemophilic serositis is considered established when a culture of the pathogen virulent for guinea pigs is isolated from the pathological material.

Differential diagnosis. Hemophilic polyserositis needs to be differentiated from polyserositis of other etiology.

Mycoplasma polyserositis covers a certain part of the livestock (5-6%), their course is not so acute, without an increase in body temperature. During the subacute and chronic course of streptococcosis, not only serositis and arthritis are observed, but also periodic diarrhea, ulcers on joint surfaces. In all cases, the final diagnosis is made on the basis of the results of bacteriological studies.

Treatment. Clinically expressed forms of hemophilic serositis are economically unprofitable, as sick piglets lag sharply behind in growth and development, and over time most of them die. In the early stages of the disease, various antibiotics (penicillin,

tetracycline, erythromycin, chloramphenicol) and sulfonamide drugs are used to treat piglets.

Immunity. There is no vaccine against hemophilic polyserositis.

Prevention and control measures. To prevent the occurrence of hemophilic polyserositis in piglets, veterinary and sanitary rules and animal hygiene standards for keeping sows and piglets should be followed in all farms. It is necessary to systematically monitor the health of the entire pig herd, especially piglets of weaning age. In dysfunctional farms, sows whose litters contain sick piglets should be culled, daily clinical examination and thermometry of susceptible piglets should be carried out, sick animals should be isolated and treated as soon as possible, and disinfection should be carried out regularly.

For disinfection of premises, walking yards, inventory, a suspension of chlorinated lime containing 2% active chlorine, a 20% suspension of freshly slaked lime with two applications at an interval of 1 hour, a 2% solution of formaldehyde for exposure of 1 hour, a 5% emulsion of xylon at room temperature for exposure for 2 hours, 5% emulsion of naphthalizol for exposure for 3 hours, 5% hot solution of soda ash for exposure for 3 hours, 5% solution of iodine chloride (0.5 l per 2 m2 area) for exposure for 3 hours. Aerosol disinfection is carried out with a 20% aqueous solution of formaldehyde, at the rate of 15 ml per 1 m3 of the room after exposure for 3 hours, or with a formalin-creolin mixture consisting of three parts of formalin and one part of disinfecting creolin at the rate of 10 ml per 1 m3 of the room after exposure for 6 hours . Manure is disinfected by the biothermal method.

Control questions and tasks.

1. Etiology and epizootological features of hemophilic polyserositis.
2. Describe the course and clinical manifestation of the disease.
3. What pathological material should be sent to the laboratory and when is the diagnosis considered established?
4. What means are recommended for treatment and what is their effectiveness?
5. What are prevention and control measures?

Topic: Hemophilic pleuropneumonia of pigs

(diagnosis, prevention and control measures).

Hemophilic pleuropneumonia of pigs (Pleuropneumonia haemophilosis suum) is a septic contagious disease, characterized in the acute course by hemorrhagic

inflammation of the lungs and fibrinous pleurisy, in the subacute and chronic course by focal purulent necrotizing pneumonia and fibrinous pleurisy.

The causative agent of the disease. Haemophilus pleuropneumoniae (syn. H. parahaemolyticus) is immobile, gram-negative coccobacilli and rods, which are characterized by pronounced polymorphism. Spores do not form. Virulent strains have a capsule, well-defined tropism to lung tissue. For the cultivation of hemophilic bacteria, chocolate (with blood) agar, serum-yeast agar, and Levinthal agar are used, on which the pathogen retains its typical morphology and stability. Haemophilic bacteria are not resistant to the influence of various environmental factors.

The diagnosis is made by a complex method on the basis of epizootological examination indicators, clinical and patho-anatomical data and the results of bacteriological examination of pathological material.

Epizootological data. Pigs of all age groups are susceptible to the disease, but the most severe course of the disease occurs in weaned piglets and piglets. The source of the causative agent of infection is sick pigs, in the tonsils of which hemophilic bacteria are stored for up to 4 months. The causative agent of the disease is released from the animal body during coughing and sneezing. Natural infection occurs by air. The possibility of infection through feed and water contaminated with secretions from the respiratory tract of sick animals is not excluded. Outbreaks of the disease are registered throughout the year, but the highest incidence occurs during its cold period. Hemophilic pleuropneumonia is observed in the form of enzootia, it spreads especially quickly among pigs, which are kept crowded, in cold, damp, insufficiently ventilated rooms. Lethality can reach 100%. Diseases almost never occur in the conditions of free-range keeping.

Clinical signs and course of the disease. The incubation period is 4-24 hours. The course of the disease is hyperacute, acute and chronic.

In the hyperacute course of the disease, which occurs more often in 35-120-day-old piglets in the case of the initial occurrence of the disease in the farm, there is an increase in body temperature to 41-42°C, depression, lack of appetite, rapid breathing, shortness of breath, bluish skin in the area of the ears, of the stomach, abdominal and chest walls, discharge from the nose of a frothy bloody liquid, sometimes blood. During the first 6-12 hours, sick piglets die.

During the acute course, the signs of septicemia are weakly expressed, the constant fever and symptoms of pneumonia prevail - shortness of breath, wheezing, heavy cough, discharge from the nose, sometimes bloody. The death of the animal occurs on the 2-5th day of the disease.

The chronic course is accompanied by a periodic increase in temperature, exhaustion, cough, and developmental delay. Sick piglets are forcibly slaughtered.

Pathological changes. With a hyperacute course, the autopsy reveals signs of one- or two-sided hemorrhagic inflammation of the lungs and their swelling (Fig. 1). The parenchyma of the lungs is cherry-red with gray cells, compacted, easily torn when

pressed. In the central part of the affected part of the lung, 1-2 primary cells are noted, in the zone of which growth of the pulmonary and costal pleura occurs (fibrinous pleurisy). From 50 to 400 ml of bloody fluid is detected in the chest cavity.

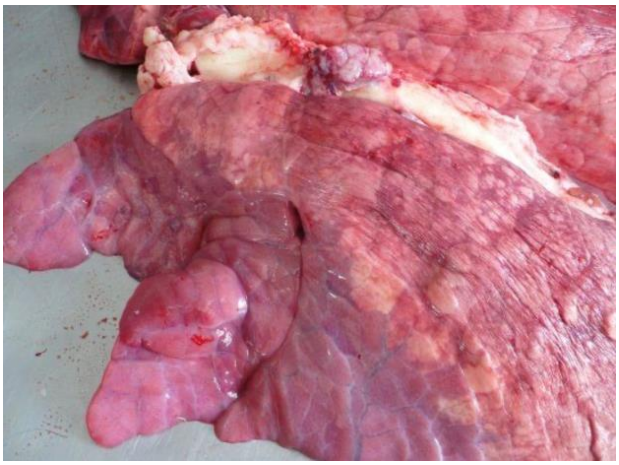

Fig. 1. Hemorrhagic pneumonia.
(https://vetmarket.ltd/info/disease/gemofiloz/)

Bronchial, mediastinal and superficial cervical lymph nodes are enlarged, hyperemic, wet on section. In the acute course of the disease, a focal lesion of one of the lobes of the lungs is determined, in the zone of the centers - fibrinous pleurisy. On the dark red surface of the lungs, gelatinous-edematous strands of gray-yellow or gray connective tissue are visible. Bone and lung pleura are inflamed, covered with fibrin films. Up to 200 ml of bloody fluid with fibrin flakes is detected in the chest cavity. With a chronic course, there are encapsulated cells 1×4 cm in size with necrotized lung tissue in the lungs, and fibrinous pleurisy in the area of the primary cell.

Laboratory diagnostics. Pieces of affected lungs, mediastinal and bronchial lymph nodes, which are taken from 2-3 pigs at the border of affected and healthy tissues, are sent for laboratory research in a thermos with ice. In the laboratory, swabs-imprints are examined, cultures are carried out on blood agar, meat peptone agar and meat peptone broth without growth factor. The causative agent of hemophilic pleuropneumonia does not grow on ordinary meat peptone agar and meat peptone broth. The pathogenicity of the selected culture of Haemophilus bacillus is determined by intraperitoneal infection of white mice.

Differential diagnosis. It provides for the exclusion of pig disease for pasteurellosis, salmonellosis and mycoplasmosis based on the results of bacteriological studies.

Treatment. Sick animals are treated with antibiotics, sulfonamide and nitrofuran drugs. Symptomatic therapy is used.

Immunity. After a relapse of hemophilic pleuropneumonia, it is not stable and not long-lasting. For vaccination, a vaccine is proposed, which is used to prevent the disease in sows and piglets.

Prevention and control measures. Preventive measures should be aimed at keeping animals in proper zoohygienic conditions and providing them with complete diets. Pig farms should be stocked only with healthy animals from farms free of infectious diseases. Newly arrived pigs must be kept in preventive quarantine for 30 days. In the case of the primary occurrence of hemophilic pleuropneumonia in a previously prosperous farm, it is considered appropriate to completely replace the entire pig herd. Quarantine restrictions are immediately introduced in the farm, the regrouping of pigs, the export and import of new animals are prohibited, and the conditions of keeping and feeding are improved. Sick and suspected piglets are isolated and treated. Healthy pigs are vaccinated. The premises are thoroughly cleaned, sanitary repaired and disinfected. Dry lime, 20% hot caustic soda solution are used for disinfection. Manure is disinfected by the biothermal method.

Control questions and tasks.

1. In which infectious diseases of pigs, lung damage is considered the leading clinical and patho-anatomical sign?
2. How is hemophilic pleuropneumonia of pigs diagnosed?
3. What means are used for treatment and specific prevention?
4. Describe the general veterinary sanitary and specific measures to eliminate the disease.

Topic: Chlamydiosis of pigs

(diagnosis, prevention and control measures).

Chlamydiosis of pigs (Chlamydiosis suum) is a chronic disease of pigs of all age groups, which is characterized by abortions in sows, the birth of dead or non-viable offspring, in boars by orchitis and balanoposthitis, in piglets by pneumoenteritis, encephalomyelitis, arthritis.

The causative agent of the disease. Chlamydiae psittacci var. Suis - belongs to the genus Chlamydia. Chlamydia are mostly spherical in shape, surrounded by two three-layer envelopes (an outer cell wall and an inner cytoplasmic membrane), pass through bacterial filters, and contain both RNA and DNA (unlike virions). They are reproduced in the cytoplasm of sensitive cells by binary fission of initial particles, which are transformed into initial elementary bodies. Chlamydia are cultivated in the yolk sac of 6-7 day old chicken embryos, as well as in the body of white mice in case of their intracerebral or intraperitoneal infection. Abortions are induced in pregnant guinea pigs.

Chlamydia are stable in the external environment. In a lyophilized state, they remain viable for more than 3 years, in a frozen state to -42°C - up to one year, at room temperature - up to 10 days, in water - up to 17 days, in livestock premises in dried

excrement - up to 6 months. Chlamydia are inactivated after 30 minutes at 80°C, after 45 minutes at 70°C, after 30 seconds under the influence of ultraviolet radiation. They quickly die under the influence of a 2% solution of caustic soda, a 1% solution of hydrochloric acid, and 75° ethyl alcohol. Sensitive to tetracycline antibiotics.

The diagnosis is made by a complex method on the basis of a clinical and epizootological examination of livestock, pathomorphological data and the results of laboratory studies.

Epizootological data. Chlamydia affects pigs of all age groups. Despite the significant distribution of chlamydia among various species of domestic and wild animals and birds, the issue of interspecies transmission of these microorganisms has not been definitively clarified. The main source of the causative agent of infection in pigs is infected breeding boars and sows, in whose bodies chlamydia persists throughout life. The causative agent is released from the body of infected pigs with feces, semen, bronchial mucus, amniotic fluid, and aborted fetuses. Infection occurs through alimentary and aerogenous routes, as well as through infected sperm during mating. Excretion of sick animals, bedding, care items, milk of sick cows and pigs, as well as ticks, mealybugs, and flies can be factors of pathogen transmission. In pigs, chlamydia manifests itself seasonally, the largest number of abortions and stillborn piglets occurs in the winter-spring period.

Clinical signs and course of the disease. At the initial occurrence of infection in the farm, mass abortions are observed in single sows, which occur at the end of puberty and can reach 80-100% of cases. Abortions and the birth of dead and non-viable piglets are also noted in some of the main sows (Fig. 1). Abortions are registered only in one-time sows in permanently dysfunctional farms. At the same time, there are usually no deviations in the state of health of aborted animals, with the exception of a slight increase in body temperature and a decrease in appetite, sometimes - agalactia, metritis.

Fig.1. Chlamydial abortion.

(https://vetmarket.ltd/info/disease/khlamidioz/)

In adult boars, the disease is chronic, without pronounced clinical signs, orchitis, arthritis, and decreased sexual activity are sometimes recorded. However, in young boars that have entered the center of infection for the first time, chlamydia is acute and is accompanied by damage to the respiratory, digestive, and reproductive systems. They experience depression, refusal to feed, an increase in body temperature to 40.5-41.0°C, a deep and frequent cough, diarrhea, constipation, vomiting, depression, and sometimes death. As a result of damage to the joints, some boars limp. After 8-10 days, the clinical signs of the disease disappear, the animals begin to recover slowly. Swelling in the foreskin, enlarged testicles, accelerated urination are noted in sick boars.

Newborn piglets are lethargic, with a weak sucking reflex, the skin is hyperemic, with a bluish tint, the mucous membranes are pale and dry. Such piglets die on the 5-7th day of life. When piglets 3-4 days old are sick, there is an increase in body temperature up to 41-42°C, cyanosis of the mucous membranes, catarrhal rhinitis, serous in 3-5% of sick piglets, later - purulent conjunctivitis, short-term diarrhea. Rapid exhaustion of sick piglets occurs, the skin acquires a yellow-brown color, dark brown crusts form on it. Some piglets show signs of damage to the central nervous system. Up to 20-60% of piglets may die during 2-3 weeks of enzootic disease. When piglets older than 2 months are affected, damage to the respiratory organs, conjunctivitis, short-term diarrhea, and weight loss are noted. In most animals, limited necrotic lesions of the skin in the areas of the ears, trunk, and tail are detected, in some piglets - polyarthritis.

During the chronic course of chlamydia, arthritis, enteritis, conjunctivitis, and pneumonia are found in all age groups. In boars, the chronic course of the disease is most often registered, without clearly expressed clinical signs. Weight loss, decreased sexual activity, orchitis and arthritis are observed. However, young, non-infected boars brought into a dysfunctional farm may develop an acute course of the disease. After 3-4 weeks, they show depression, refusal to feed, an increase in body temperature to 40.5-41°C, cough, vomiting, and short-term diarrhea. On the 8th-10th day, these phenomena disappear, the boars begin to recover slowly, but some of them have enlarged testicles, swelling of the foreskin, accelerated urination.

Pathological changes. Endometritis, sometimes focal necrosis of the uterine mucosa, as well as edema and infiltration of the placenta are found in sows that have aborted. In dead boars, a 1.5-2 times increase in the size of the epididymis, hemorrhagic inflammation of the vas deferens, balanoposthitis, orchitis are found. In aborted fetuses and piglets that died in the first days of life, edema of the subcutaneous connective tissue in the head, chest, shoulder blades, diffuse hemorrhages in the parietal part of the head and on the limbs, accumulation of exudate in the chest and abdominal cavities, pericardial sac, subpleural spaces, blood filling and hemorrhages in the liver, foci of

inflammation in the lungs. When joints are affected in piglets, an increase in the amount of synovial fluid, roughness and redness of the capsule of the inner surface of the joints are noted. During the pathohistological examination, significant lymphoid-histiocytic and neutrophilic infiltration of the uterine mucosa, perivascular lymphoid-cellular infiltrates, foci of necrosis and necrobiosis in the liver of aborted fetuses, significant blood filling and necrotic foci in the center of individual seminiferous tubules are revealed.

Laboratory diagnostics. It provides for the microscopy of smears-imprints from the affected organs of slaughtered sick sows, as well as aborted fetuses; isolation of the pathogen on chicken embryos and its identification; biological test on white mice; detection of anti-chlamydial antibodies in blood serum. The following are urgently sent to the laboratory in a thermos with ice: rennet, pieces of placenta, whole aborted fetuses, vaginal mucus taken from slaughtered sows that have aborted. Pieces of the liver, lungs, spleen, lymph nodes, bladder, and synovial fluid are collected and sent from dead or aborted piglets. In case of suspicion of a disease of the spermatozoa - fresh or frozen sperm, and in case of slaughter - testicles, parts of parenchymal organs, lymph nodes. Blood serum of suspected animal diseases, as well as those sows that have aborted, are sent for serological tests. For microscopic examination, smears and smear-imprints are prepared from pathological material, which are fixed with methanol, stained according to Romanovsky-Giemza and Macchiavello, Stamp, as well as direct and indirect methods of immunofluorescence. With the help of immersion microscopy, colored elementary bodies and cytoplasmic inclusions are detected, and under a fluorescent microscope - their specific bright green glow. In order to isolate chlamydia, infection with pathological material is carried out in the yolk sac of 6-7 day old chicken embryos. A positive bioassay results in the death of infected chicken embryos. Microscopy of smears from yolk membranes, sometimes from the chorionallantoic membrane reveals elementary bodies of chlamydia. To conduct a bioassay, the pathological material in a volume of 0.5 ml is injected into three white mice intranasally, intracerebrally or into the abdominal cavity. With positive results, infected mice die in 3-5 days or 1-3 weeks. At their autopsy, a significant increase in the spleen is revealed, and at the microscopy of smears-imprints, elementary bodies of chlamydia.

Serological studies involve the use of diagnostic group or species-specific chlamydial antigen. Serum titers in a dilution of 1:8-1:16 indicate a latent infection or the beginning of the disease, 1:32 and above - a transferred disease.

Differential diagnosis. When diagnosing chlamydia, brucellosis, leptospirosis, and salmonellosis must be excluded. The results of the isolation of the causative agent of the corresponding disease from the pathological material and the detection of specific antibodies in the blood sera are of decisive importance for this.

Treatment. Not conducted. Animals that are sick or suspected of having chlamydia are slaughtered at a sanitary slaughterhouse.

Immunity. For specific prevention, an inactivated emulsin vaccine against chlamydial abortion of cattle, sheep, goats and pigs is proposed. The vaccine is intended for use in disadvantaged and chlamydia-threatening farms. Animals are vaccinated subcutaneously once, once a year in doses: pigs aged 1 to 6 months - 1 ml, older than 6 months - 2 ml. Immunity is formed 20-25 days after vaccination and lasts for 12 months.

Prevention and control measures. They include strict observance of veterinary sanitary and zoohygiene rules during the collection and breeding of pigs. Special attention should be paid to the protection of the farm against the introduction of the causative agent of the disease from the outside. In this regard, breeding farms that are chronically unfavorable for chlamydia are especially dangerous. Therefore, it is necessary to carry out serological tests for chlamydia in advance in supplier farms, for which blood is selectively taken from sows and boars and examined with the help of the complement binding reaction. During the 30-day quarantine, 10% of imported pigs must be examined for chlamydia by serological method. At the same time, even a low titer (1:4) of indicators of the complement binding reaction indicates the presence of chlamydia and the infection of livestock. At the same time, a thorough clinical examination of the pig herd and all suspected animals with manifestations of conjunctivitis, orchitis, rhinitis, local skin necrosis is carried out, and chlamydia is checked serologically. For preventive purposes, at stations and points of artificial insemination, all breeding boars are examined for chlamydia serologically once every 6 months, and when a new herd of boars is introduced, they are examined during their quarantine period. When pigs are transferred to the main farm, their skin and limbs are treated with 1% formaldehyde solution or 0.5% sodium hydroxide solution. General preventive measures are carried out on the farm. For the purpose of timely detection of chlamydia disease in case of abortions, birth of dead, non-viable offspring, death of piglets in the first days of life, as well as other clinical signs characteristic of chlamydia, complex laboratory tests are immediately carried out. When chlamydia is detected, the farm is declared unhealthy, appropriate restrictions are introduced in it, first of all, a ban on the export and import of pigs, their regrouping, and health measures are carried out. Aborted fruits, fruit membranes, corpses of dead animals are collected in moisture-proof containers and disposed of. Machines and premises in which sick animals were located are thoroughly cleaned and disinfected. All premises, as well as equipment, animal care items are disinfected every 7-10 days. All sick pigs with clinical signs of chlamydia and suspected animal diseases are slaughtered at a sanitary slaughterhouse. Healthy pigs that have had contact with sick pigs are put on fattening and then sent for slaughter. They stop pairing pigs, getting sperm from boars. All boars are serologically tested for chlamydia. Boars that have a positive complement-fixation reaction or chlamydia in their semen are slaughtered. Sperm obtained from them earlier are destroyed by boiling for 10-15 minutes. Boars that gave negative results during the chlamydia test, but were in contact with patients, as well as sows and repair pigs are

treated with oxytetracycline or dibiomycin. Oxytetracycline is administered intramuscularly twice a day for 5 days at 5,000 units/kg; dibiomycin is used in the form of an oil mixture intramuscularly, twice, with an interval of 10-12 days, 10-15 thousand units per 1 kg of weight. The treatment course is considered completed 7 days after the last administration of the drug. Sows are inseminated only artificially, for which they use sperm from boars from prosperous farms.

2-3% hot caustic soda solution, 2% formalin solution, clarified chlorinated lime solution containing 3% active chlorine, 5% hot soda ash solution, neutral calcium hypochlorite solution, two-tertiary calcium hypochlorite salt containing active chlorine 3%, phenosmolin 4-5% concentration during exposure for at least 3 hours. Manure is disinfected by the biothermal method. Gradually, the entire pig population of the farm is replaced by healthy animals raised on an isolated farm. The farm is recognized as cured of pig chlamydia if no clinically sick pigs with negative serological test results have been registered for three years.

Control questions and tasks.

1. By what cultural-morphological, pathogenic, toxigenic and antigenic properties do chlamydia differ from rickettsiae and mycoplasmas?
2. Reveal the manifestation of the symptom complex of chlamydia in pigs.
3. List the means and methods of specific diagnosis and immunoprophylaxis of pig chlamydia.
4. How to prevent chlamydia in pigs?
5. What means are recommended for etiotropic and symptomatic treatment of chlamydia in pigs?
6. Name the main measures to eliminate pig chlamydia.

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