DUCK HEPATITIS VIRUS TYPE I

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Infectious diseases prevent the intensive development of industrial duck breeding. Among them, viral hepatitis of type I ducklings has become widespread. The disease is registered in all countries of the world. This is due to the epizootology features, the stationarity of the foci due to the significant sustainability of the virus and its variability. The article presents data of domestic and foreign researchers on the epizootology of the disease, taxonomy and biological properties of the pathogen, pathogenesis and clinico-pathoanatomical manifestations of the disease.

Keywords: duck hepatitis virus, epizootology, biological properties, clinical signs, pathoanatomical changes

Epizootic features of the disease

Viral hepatitis of ducklings of type I (infectious hepatitis of ducks) is highly contagious, super acute among ducklings [3, 19] and latent among ducks disease [11, 23], with predominant liver damage and high mortality of young animals. Viral hepatitis of ducklings (DHV) causes considerable economic damage to duck farms, especially the industrial type, as it causes a massive loss of ducklings 1-30 – the daily age of 30-95% and a decrease in the productivity of ducks. Recovered ducklings are lagging behind in growth and development, which leads to a partial loss of meat production, a violation of breeding work. The damage from the DHV is exacerbated by the costs of restrictive measures that violate the economy of the economy, especially when the disease takes a stationary character [9, 13, 23].

In vivo, viral hepatitis can be affected by ducklings up to 40 days old, but more often, at 1-30 days old [19]. To the virus of hepatitis of ducklings, goose is also susceptible up to 10-12 days of age, both under natural conditions and when they are infected by artificial means [1, 29]. Rapid development of age-related immunity serves as a characteristic property of this infection, that is older ducklings and adult ducks are not clinically ill. Not susceptible to the causative agent of hepatitis ducklings also domestic, wild and laboratory animals, and other species of poultry. There is no documented disease in humans [37, 39].

The source of the causative agent of infection is a sick and sickly bird – virus carriers that release into the external environment of the pathogen with litter, nasal and conjunctival outflows. The duration of virus carrying after an illness varies from 60-75 to 300-650 days [26].

The causative agent of the disease is transmitted by ducklings and for incubation eggs delivered from disadvantaged farms at the DHV. Embryos from such eggs in 75-90% of cases die during incubation at various stages of embryonic development. Drift of the pathogen is possible with wild ducks and free-living birds [5, 21, 41].

Inside the farm, the virus is transmitted when a healthy and sick bird is together. The causative agent of the infection is also transmitted with infected food, water, litter, care items, transport and maintenance personnel. Infection occurs alimentary, but it is also possible aerogenic infection of ducklings. It is possible that the virus can enter the body of birds when injured, for example, when injecting various drugs, as well as by mechanical and transovarial routes [2, 5, 41].

There is a characteristic feature of the epizooticity of viral hepatitis, which is repeated almost in all cases: the death of ducklings is growing quite rapidly: the peak is 4-5 days, and the decrease by 7-8 days, by 10-12 days, there is a sharp decrease in the number of dead ducklings. In viral hepatitis, stationarity of the foci is noted, which is determined by the relatively high resistance of the pathogen in the external environment, the constant presence of ducks-virus carriers and susceptible livestock, especially in the year-round cultivation of ducklings [11, 27].

The reservoir of the virus can be rats [9]. One should also take into account the possibility of a "natural focality" of the pathogen. Wild ducks often settle on ponds near duck farms. They also get viral hepatitis and can spread the virus. There is no pronounced seasonality, but violations of conditions of maintenance, inadequate feeding of birds and fodder toxicosis contribute to the manifestation of the disease. It should be especially emphasized that a number of pathogens can complicate the course of the main infectious process, first of all, in ducklings, pathogens of salmonellosis, colibacillosis, aspergillosis and chlamydia, and

in ducks – hepadovirus infection. Such a bird should be classified as an increased risk [2, 5, 28, 35].

At the first appearance of the disease in a prosperous farm, the enzootic begins, as a rule, among ducklings 5-10 years old and affects a series of consecutive conclusions, quickly covering all susceptible livestock. The incidence of ducklings up to 3 weeks of age is 80-90%, the lethality with super-fast flow for the first 10 days of life reaches 100%, with acute flow – 70-80%. In permanently dysfunctional farms viral hepatitis is registere among ducklings 15-30 days old and older, the case in separate batches is 5-10%. If non-immune young animals re-enter this economy, the death rate among ducklings from party to party increases again and sometimes reaches 80-95% [19].

Biological properties of the virus

To date, it is known that there are three types of viruses for the causative agents of ducklings: type 1 – "classical", widespread everywhere [44]; type 2, isolated in England [34], and type 3, isolated in the USA [40]. It was determined that viruses of type 1 and type 3 belong to the family of picornaviruses. Type 1 virus is reproduce in cell cultures and embryos of ducks, chickens and quails. Type 2 virus differs from type 1 in that it is well reproduced in embryos and in the body of ducks, in cultures of liver cells and in the kidneys of a duckling, but its reproduction in chicken and quail kidney culture is limited, and it is not reproduced in chick embryos. It is important to note that type 3 virus causes hepatitis among the ducklings immune to virus type 1, that is, these types of virus have antigenic differences. As for the type 2 virus, recent studies have shown that it belongs to astroviruses.

The type 1 causative agent is an RNA-containing virus belonging to the family Picornaviridae, the genus Avihepatovirus of type 1 hepatitis virus (Duck hepatitis virus type 1 DHV-1), which includes three serotypes of the DHAV-1 virus, DHAV-2, DHAV-3 (Resolution adopted by the world assembly of delegates of the oie in May, 2010) [42].

Electron microscopic studies have determined that these are specific viral particles of round or spherical shape, the size of the virus is from 20 to 40 – 60 nm. [8, 19]. The virus is resistant to ether and chloroform and various pH of the medium: 4.8; 7.8; 3.0 and from 6.8 to 7.4 [19]. The virus of hepatitis of ducklings is considerably resistant to the influence of the external environment: in the feeders survive more than 10 weeks, in the litter – 37 days, water – up to 74 and in soil – from 105 to 131 -157 days [8, 19]. The virus of hepatitis of ducklings kept pathogenicity while being on the surface of the walls of poultry houses from 20 to 40 days depending on the air temperature, in the litter – 15-20 days [22]. The vaccine strain of 3M duckweed hepatitis virus was viable in aerosol at a room temperature of 18-20 °C for 45 minutes [19]. The virus can withstand heating to 50 -56º C for 60 minutes or more. When stored in the refrigerator at minus 14 – 32 °C, the virus remained viable for several years [8].

Ultraviolet rays at a distance of a radiation source of 30 cm killed the virus in 3 minutes, at a distance of 60 cm in 10 minutes [6].

Disinfecting solutions of xylonaphtha, lysol, creolin and soda ash at ordinary concentrations are not effective. 1% chloramine solution has virucidal properties [31]. Influences the virus and the temperature of the disinfectant solution. Thus, 4% hot and 5% cold solutions of sodium hydroxide inactivated the virus of hepatitis of ducklings for the same time – 6 hours [22].

The virus of hepatitis ducklings replicates on 10-12 day old duck and 9-10 day old chick embryos by infecting them in the allantoic cavity [19, 36].

Embryos die in 2-6 days in 10-60% of cases, but some of them develop before withdrawal and hatch, which depends on the properties of the virus, the method of infection and the age of the embryo.

Passing the virus through embryos increases its virulence. The "peak" of virulence (up to 100%) falls on the 53th passage, and the concentration of the virus in the allantoic fluid after 48 (53-69) hours. The virus accumulates in the body of the embryo to 7.5 lg, in the chorioallantoic shell – 5.79 lg and in the allantoic fluid – 3.62 lg.

In dead embryos, the allantoic fluid and the contents of the yolk sac acquire a greenish tint. Hemorrhages and swelling of the thoracic and abdominal areas with hemorrhages are noted on the embryo body. Liver of loose consistency of gray-yellow color with foci of necrosis.

The virus is capable of replication in primary-trypsinized liver cells and kidneys of duck embryos [15, 26, 43], as well as on duck and chicken embryo fibroblasts with collagenase [16, 17].

After 3-7 days, the authors observed the formation of the symplasts, the appearance of granularity in the cytoplasm and the vacuolization of the affected cells, and then the destruction of the monolayer [25].

Pathogenesis of the disease

In natural conditions, infection of ducklings occurs mainly through the mucous membranes of the digestive and respiratory organs. The virus, infiltrated into the body, carries blood to many organs, primarily the liver and brain. The titer of the virus already in the first hours after infection in the blood is high, but gradually decreases to 48-72 hours. At the same time, the virus titer in the liver and brain is increased 48-72 hours after infection. Neutralizing barrier function of the liver decreases and toxic products are spread by blood throughout the body. The death of ducklings occurs as a result of irreversible changes in the liver and other organs. Bend, as a rule, ducklings with good fatness, with intoxication phenomena [7, 14, 19].

In the liver of infected embryos and diseased ducklings, hepatosis and hepatitis develop, which are naturally accompanied by necrobiosis and necrosis of cell elements, as well as a decrease in the level of total protein and albumin in the blood serum, a decrease in the protective properties of serum colloids and alkaline phosphatase, glutamate-pyruvate transaminase, bilirubin and creatine. In chronic course of viral hepatitis, changes in organs are of the same nature, but foci of necrosis in the liver of ducklings are more extensive.

Clinical and pathoanatomical picture

Viral hepatitis of ducklings proceeds superfast; describes the chronic course and atypical form of the disease. The incubation period with natural infection is 1-5 days, and with artificial infection – 1-8 days. A shorter incubation period with oral, intranasal and aerogenic infection compared with the parenteral method [18, 33, 38].

In conditions of unfavorable farms in the acute course of the disease, the incubation period is 1-7 (less often 12-13) days, and the duration of the disease is 1-3 hours, less often 4-5 hours. The disease with visible clinical signs proceeds rapidly, and often the period of the disease precursors with the first clinical signs of hepatitis remains unnoticed. Often in clinically healthy from the evening of the herds in the morning are found many dead ducklings. At the same time, it was noted that most ducklings ran from the room to the paddock, and some ducklings were sitting, swollen or falling and died immediately with seizures up to 95% [10, 12, 38].

At viral hepatitis ducklings develop the following signs of the disease: refusal of feed, drowsiness, lack of mobility, ducklings sit for a long time, movement disturbs coordination of movements, sometimes diarrhea, rhinitis, conjunctivitis. After 1-2 hours, less often after 5-6 hours, from the moment of the appearance of the nerve signs of the disease, convulsions appear, while the limbs are stretched along the trunk, the ducklings lie on the back or on the side with the head thrown back (opisthotonus), make swimming movements. After several seizures, death occurs. Ducklings recover completely rarely, sometimes the disease takes a chronic course, while the bird lags behind in growth and development [11, 12, 18, 20, 32].

The chronic course of the disease is observed in 3-4-week-old ducklings. The disease lasts 10 to 20 days, sometimes more, accompanied by diarrhea. Ducklings become inactive, some swollen joints of the limbs. There is a penguin-like gait – the ducklings move, keeping the vertical position of the body [8, 11].

Clinical signs do not always accompany disease of the ducklings. The disease in such ducklings is asymptomatic, or subclinically [23]. Not all ducklings that showed the first signs of the disease die, some of them recover, and they cannot be distinguished from healthy ones. From these ducklings, it is possible to isolate the virus, as well as to detect virus-neutralizing antibodies in serum.

There is an associated course of viral hepatitis with salmonellosis, influenza, mycoplasmosis, colibacillosis and aspergillosis. Viral hepatitis remains the leading [20].

When autopsies of fallen ducklings in an acute course, characteristic changes are found in the liver, which appears to be markedly enlarged in size, ocher-yellow, the consistency of its parenchyma is flabby, easily destroyed under pressure, in most cases its surface is strewn with hemorrhages, from point to spotted, hemorrhages without clear boundaries. The gallbladder, as a rule, is full of bile. The kidneys are swollen, blood-filled. Changes in the spleen are not the same type and are not characteristic. It is pale – or dark red, of normal size or enlarged, sometimes bumpy, mottled. The cardiac muscle in the state of granular dystrophy has the form of boiled meat, coronary vessels are blood-filled, in the pericardial cavity, and often an increased amount of serous fluid is noted. The brain vessels are full-blooded. Many ducklings find catarrhal inflammation of the intestinal mucosa, which, corresponds to complication of hepatitis with bacteriosis, primarily salmonella.

In the chronic course of the disease at the dissection, the liver is usually enlarged 1.5 times, spotted, granulomas similar to leukemias are found, and the spleen is blood-filled.

Pathoanatomical studies show the development of the liver and spleen of necrobiotic processes, perivascular lymphocytic-plasma infiltrations. Changes in the brain indicate serous encephalitis [4, 24, 30].

Conclusion

In recent years, against the background of relatively stable well-being for viral hepatitis, ducklings are constantly occurring sporadic cases of the disease.

Essential factors contributing to the emergence of cases of the disease are favorable conditions for the passivation of opportunistic microorganisms. High – density of planting of livestock, heterogeneity of its immunological status, optimal habitat conditions for pathogens, as well as untimely implementation of antiepizootic measures without biology of the pathogen as its ability to prolonged asymptomatic persistence in the body of a sick bird.

Given these circumstances and taking into account the flow of information on viral hepatitis ducklings over the past two to three decades, we were convinced of the relevance and timeliness of summarizing the data in the review article.

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