

RABBIT HAEMORRHAGIC DISEASE IN CHINA

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ABSTRACT

Some advanced application research and techniques into RHD in China are reviewed in this paper, including clinical and postmortem manifestations, anatomical changes, vaccination and antibody, and other control and prevention.

Key words: RHD, China, review.

INTRODUCTION

Rabbit haemorrhagic disease (RHD) is an acute fatal disease of rabbits, which was first described in China in 1984 and has spread all over the world. It can cause high economic losses in rabbitries as well as high mortality in wild rabbits. Infection results in an acute disease, characterized by high morbidity and mortality. In general, rabbits will die within 48-72 hours post infection due to acute necrotizing hepatitis and haemorrhage, however, infected rabbits younger than two months of age usually survive. Some advanced application researches into RHD in China are reviewed in this paper.

CLINICAL AND POSTMORTEM MANIFESTATIONS

Clinically, three forms have been observed:

Per-acute form: The affected rabbits die suddenly at 10-12 hours after infection without showing distinctive symptoms. However, rabbits have been noted to jump several times in the cage before death, which occurred mostly in the early stage of an outbreak.

Acute-form: The body temperature rises to 41°C 4 to 40 hours after infection. The rabbits

show depression, increased thirst, and reluctance to move initially, but become excited before death, running in the cage, jerking and trembling, or squealing with pain prior to death. Some rabbits showed suffocation due to foamy, bloody fluid from nostrils. This form occurred usually in the mid stage of an outbreak.

The per-acute and acute forms in young and adult rabbits are generally accompanied by a flabby anus' sphincter . The surrounding skin and wool are tainted with yellowish mucus. The fecal balls show adhesion of yellowish gelatinous exudate on the surface.

Sub-acute forms: This form is usually observed in young rabbits under three months of age with a body weight of 1.0-1.5 kg, and occurred in the late stage of an outbreak. The affected rabbits show depression, decreased appetite and anorexia for one or two days, will appear emaciated with a rough hair coat with death in two days.

ANATOMICAL CHANGES

Rabbit haemorrhagic disease is an acute general infectious disease. Anatomical lesions may be observed in the various organs as follows.

Trachea: Severe hyperemia of mucous membrane, some with foamy blood.

Lung: Severely congested with edema, and millet or pea sized petechial hemorrhages.

Heart: Congested, some with petechial hemorrhages in edocardium.

Thymus: gelatinous edema, some with millet-sized petechial hemorrhages.

Liver: Enlarged, brittle, cut surface rough, with some petechial hemorrhages.

Gall bladder: Enlarged, thin bile.

Kidney: Partly enlarged, purplish in color, hyperemia, pin-point dark red petechial hemorrhages on cortex.

Spleen: Enlarged, congested, bluish- purple in color

Mesentery lymph nodes: gelatinous edema, petechial hemorrhages on cut surface.

Stomach and intestine: In some cases of longer course, pin-point to millet-sized petechial hemorrhages on the serosal membrane.

Uterus: In pregnant rabbits, congested mucous membranes with petechial hemorrhages, dead fetus.

Urinary bladder: Retention of urine in some cases.

VACCINATION AND ANTIBODY

Vaccination of all susceptible rabbits is important in epidemic areas or areas at risk of introduction. A tissue derived vaccine, inactivated with 0.4% formaldehyde, has been developed for the prophylaxis against RHDV. Both laboratory and field trials have established its safety, potency, and ease of application. Rabbits show protection at four to five days after vaccination and immunity can last as long as six months.

It was observed that only rabbits older than two months, particularly the adults, are the most susceptible to RHDV. It has been found that RHD can also infect rabbits younger than two months of age in some Chinese rabbit farms. When the rabbits aged one to two months were challenged with rabbit haemorrhagic disease virus, they can also become infected and die. However, the infection seen was different from that of typical rabbit haemorrhagic disease as seen in the adults. It shows the characteristics of a longer incubation period and disease course, no or very low haemagglutination (HA) titre and hyperplastic hepatitis (CHUANGYI J., *et al.* 1994).

It has been shown that the kits can acquire the maternal antibody from the placenta and colostrum. The antibody level of the suckling kits was higher than that of those kits before first milking. The maternal antibody would affect the immune response of suckling rabbits. But in the coming days, the maternal antibody decreased. At the age of 30-45 days, about 70-95% young rabbits could be easily infected by RHDV and subsequently die. It has been recommended for the young rabbits to be first vaccinated at the age of 30-45 days. But for those vaccinated rabbits, it seemed that they can't be protected from RHDV for a long time in practice. So another repeat vaccination was recommended at the age of 60 days (CHENGGANG T., QIANG W., GUOLIAN B. 2001).

Antibody could be detected five days later after the rabbits with negative maternal antibody were vaccinated. Seven days post vaccination the rabbits could be protected from RHDV absolutely. The antibody level increased at the 15th day post-vaccination, and peaked on the 21st day. Antibody decreases slowly afterwards. At the 180th day the HI titre was about 1:64, which still could protect the rabbits effectively. There was an obvious decrease in the antibody level of the rabbits with positive maternal antibody 3-7 days post vaccination. It was suggested that maternal antibody may prohibit the production of antibody, and it would be possible for those rabbits to be infected easily during that special

time (CHENGGANG T., QIANG W., GUOLIAN B. 2001).

OTHER CONTROL AND PREVENTION

No antibiotics are effective against RHDV. Inactivated tissue vaccine has proven to be effective and reliable in the prevention of the disease. At the same time better management including quarantine and sterilization should be emphasized.

Measures to prevent the introduction of RHDV into a rabbit farm or colony are similar to those used for any other infectious disease where direct contact is an important means of transmission. Restricted access and fumigation and disinfection of all equipment entering or leaving a site are desirable. It is essential that no rabbits are introduced from epidemic areas. If this cannot be avoided, it is recommended that all introduced rabbits are quarantined for at least two weeks and vaccinated at least one week before entry. Adequate disposal of diseased and dead rabbits in epidemic areas is essential. Disinfection with 1-2% sodium hydroxide or 10% formaldehyde solution is recommended for utensils, rabbit cages and other equipment.

Treatment with hyperimmune serum has been developed as an effective therapy in the beginning of the disease, and may be used as passive immunization in the epizootic region. Since it has been recommended that the effective dosage is 4-6 ml per rabbit, it is very difficult for a rabbit farm to get enough volume of hyperimmune serum, especially when there are large numbers of infected rabbits.

When RHD is introduced into a rabbit farm, it is recommended to immediately vaccinate all healthy and infected rabbits with inactivated tissue vaccine, at the dose of 4-5 ml per rabbit, which is different from the regular vaccination. It has been shown that RHD will be controlled, and there will be no more animals infected five days later. It is known that rabbits cannot develop enough antibody to protect themselves from RHDV until the 7th day post vaccination, but the interferon (IFN) could be induced quickly, which could be detected 6 hours post vaccination in some experiments. It was suggested that the interferon (IFN) has an effect on the control of RHD at the beginning of vaccination, while the special immune have no any obviously response at that time. The interferon (IFN) may induce a development of translation-inhibitory protein to protect the cell from the virus at the beginning (JINGNIAN L. 1995).

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