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Case Study

INCIDENCE OF RABBIT HEMORRHAGIC DISEASE (RHD) IN MEAT TYPE RABBITS

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ABSTRACT
An outbreak of Rabbit Haemorrhagic Disease among meat type rabbits at the Institutional demonstration rabbit farm has been described. The clinical signs, post mortem lesions and treatment / control procedure undertaken were discussed.

KEYWORDS: hemorrhagic disease, meat type, Rabbit, post mortem lesion.

INTRODUCTION

In recent years there has been rising awareness on increasing meat requirements due to rapidly increasing human population in developing countries like India. The availability of meat in India is 5.2 Kg per person per year against the requirement of 16.0 Kg per person per year (FAO, 2004). Amongst meat animals, rabbit is one of the important livestock species which supplies meat protein to human consumption. In backyard rearing, rabbit can meet the animal protein needs of an average family and is a suitable and cheaper alternative to poultry. Rabbit is a herbivorous animal and strives mostly on natural vegetation. It can consume large quantities of forages that are not used by human beings. Rabbit farming shall be advantageous in view of the low capital investment, less space requirements, short generation interval, better growth rate, high reproductive potential and ability to utilize the abundant forages and fibrous agricultural by-products (Cheeke, 1986 and Joseph *et al*., 1997). The diseases that are encountered by rabbits are the limiting factors for the successful multiplication and on the whole the growth of the rabbit industry. Though rabbits are susceptible to many bacterial, viral and protozoan diseases, some infections are very acute and make a clean sweep of the rabbit population. Rabbit haemorrhagic disease (RHD) is a highly infectious disease epidemic among meat type rabbits causing 100% mortality. An epidemic of RHD has been described.

MATERIALS AND METHODS

The outbreak of an epidemic was observed at the rabbit unit of the Livestock Farm, Veterinary College and Research Institute, Namakkal, Tamil Nadu, India. Initially, it was observed that adult animals were dull, went off feed and did not drink water. Subsequently, a febrile condition with temperature of 49^0 C along with lacrimation and nasal discharge followed by dyspnoea was observed. Affected animals died within 24 to 48 hours after onset of symptoms. The dead animals showed bleeding from the nostrils and eyes (Figure.1). Typical symptoms of head pressing over the floor (Figure.2) and lateral recumbency were observed in most of the affected rabbits. The outbreak lasted about 20 days, during which 60% mortality was observed in adult animals. Although all the animals were housed in the same rabbitry, those younger than 12 weeks of age remained healthy and did not show any symptoms. The dead animals were subjected to detailed post mortem examination.

FIGURE 1. Haemorrhages in the eye

FIGURE 2: Head pressing over the floor

Past history of RHD

RHD caused by and RNA virus of the Calicivirus genus. It was a notifiable disease in the UK (the only one for rabbits). It first occurred in China in 1984 and then spread through many countries and most of those in Europe by 1992, when it reached England (Sandford, 1996). No report of the incidence could be traced in Indian rabbit farming experience. But, Patton (1989) reported similar type of signs and lesions in rabbits outside India as occurring in China, Mexico and few European countries. Later in India, Sundaram *et al* (1991) reported a note on the incidence of haemorrhagic disease with similar clinical signs and post mortem lesions. After this, no literature could be detected on this issue.

Post mortem and laboratory examination

Post mortem examination revealed profuse hemorrhage in the lungs (Fig.3), inflammation with typical reticular pattern in the liver (Fig.4) and haemorrhage in the small intestine. On suspicion for a viral infection, the materials were subjected to laboratory testing. For want of a specific antigen the specific tests were not possible. No haemagglutinating agents could be detected in HA test. The trachea showed diffuse severe congestion. The liver showed multifocal necrotic hepatitis. The histopathological examination revealed multifocal moderate haemorrhage in lungs and diffuse vacuolar changes of transitional epithelial cells in the urinary bladder. Viral antigens could not been detected through ELISA. The electron microscopic study also failed to reveal the virus. Though all the tests carried out failed to prove the infection of calici virus, the clinical signs and post mortem lesions were very close the calici virus infection.

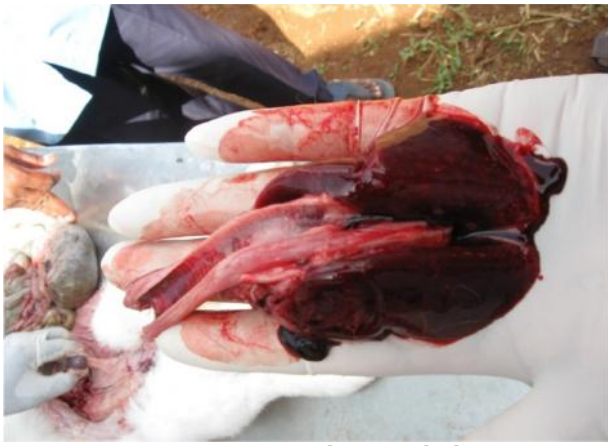

FIGURE 3: Haemorrhages in the lung

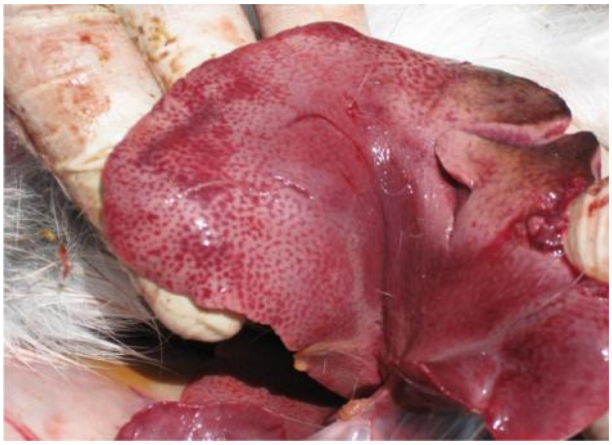

FIGURE 4: Reticular pattern of the liver

TREATMENT AND CONTROL

The rabbits failed to respond for the treatment with broad spectrum antibiotics. As a control measure, liver and spleen samples were collected from dead rabbits and brought to the laboratory on ice. Pooled liver and spleen samples were triturated and 20% suspension was made with phosphate buffer saline and clarified at 3000 rpm for 15 minutes. The clear supernatant was collected, inactivated with 1% formalin for overnight and sterility test was carried out on the next day according to the standard protocol. Both affected and apparently healthy rabbits were vaccinated at the rate of 1 ml per rabbit intramuscularly and booster vaccination was given after two weeks. The vaccinated rabbits were observed for the efficacy of the autovaccine. Meanwhile the shed was sprayed with disinfectant such as 1% formalin and 10% house hold bleach.After five days of vaccination, mortality has come down gradually and it was almost completely stopped after 14 days. Based on the clinical signs, lesions and responding to autovaccine, it was concluded that the rabbit might be infected with rabbit haemorrhagic disease virus.

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