Viral Haemorrhagic Disease of Rabbits and the European Brown Hare Syndrome

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Source: Journal of Wildlife Diseases, 29(4) : 623-624

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-29.4.623

Journal of Wildlife Diseases, 29(4), 1993, pp. 623–624

BOOK REVIEW . . .

Viral Haemorrhagic Disease of Rabbits and the European Brown Hare Syndrome, J. P. Morisse (ed.). Scientific and Technical Review, Office International des Epizooties. 12, Rue de Prony, 75017, Paris, France. June 1991, Vol. 10, No 2; pp. 263–525 ($32.00 U.S.).

Fifteen papers on two emerging diseases of Leporidae, viral hemorrhagic disease (VHD) of rabbits and the European brown hare syndrome (EBHS), were presented at an international symposium sponsored by the Office International des Epizooties (OIE) held in Paris in 1991. Dr. J. Blancou, Director General, OIE, reviewed the history of the diseases and noted that VHD was first described in China in 1984. He described the meeting as an opportunity to bring together researchers to focus on the origin and collaborate on these emerging diseases.

The first four and fourteenth papers centered on virological aspects of VHD. Morisse et al. related certain pathological, clinical, and epidemiological similarities of viral hepatitis in humans with VHD and EBHS. The authors described the fecal-oral route of transmission of VHD as similar to that of human hepatitis A and E. The characterization of the causative agent of VHD remains ambiguous as workers have described it as a calicivirus, a parvovirus, and a picornavirus. Ohlinger and Thiel described physico-chemical, biochemical, and immunological properties of viral particles extracted from the liver of VHD infected rabbits characteristic of a calicivirus with one major structural protein and an RNA genome. Based on immunological studies, EBHS appears serologically distinct from VHD virus (VHDV). Du indicated the nucleic acid of liver origin VHD was DNA and that the virus contained four structural proteins. He noted that there were few common restriction sites comparable to parvoviruses but that there was homology on Southern blotting with selected parvoviruses. Ji, Du, and Xu described, for the first time, the adaptation of VHD to a rabbit kidney cell line (DJRK). The cell culture virus caused VHD in rabbits, and a cell culture-derived vaccine protected against challenge inoculation with VHD. They found the virus to contain single-stranded DNA and portrayed VHDV to be a parvovirus-like virus. Also relating to the virology of VHDV, in the fourteenth paper, Gong and Ji described the morphogenesis of VHDV in infected cell cultures and rabbits. Viral replication was in the nucleus with release of virus to the cytoplasm.

The diagnosis and clinicopathological features of VHD and EBHS were described in two papers. Capucci et al. described the use of hemagglutination, hemagglutination inhibition, immune electron microscopy, and ELISA for the diagnosis of VHD and EBHS. They differentiated VHDV from EBHS virus (EBHSV) with a monoclonal antibody. Based upon virological, serological, and epidemiological evidence, they suggested that VHD and EBHS are "two distinct diseases, each caused by its own aetiological agent." In the next paper, Marcato et al. noted the similarity between the clinical and pathological changes caused by VHDV and EBHSV. Animals under 2 mo of age are quite resistant to VHD. Both diseases have an acute phase which may manifest epistaxis, sometimes nervous signs, and sudden death caused by severe hepatic damage with multifocal hemorrhages leading to fatal shock. Direct damage to hepatocytes and endothelial cells with a defect of coagulation appear responsible for the hemorrhagic syndrome. Lung edema and hemorrhage are common.

The epidemiology of VHD and EBHS was evaluated in five papers. Xu noted that in the People's Republic of China (PRC), only adult rabbits, not other livestock, are affected and that VHD occurs throughout the year. The disease has been controlled effectively in China with inactivated vaccines, now of cell culture origin. Cancellotti and Renzi stated that a new disease spread through Italy in the mid-1980's and is currently having a severe impact on domestic rabbit production. In 1989 EBHSV was identified in hares in northern Italy. Loliger and Eskens stated that VHD is widespread and EBHS has been reported in all of the Federal states of Germany. It appears that infection of rabbits and hares is possible with either of the agents. Gregg et al. described the eradication of VHD from Mexico by "stamping out." Eastern cottontail rabbits (*Sylvilagus floridanus*) and black-tailed jackrabbits (*Lepus californicus*) were reported resistant to VHD. Reference was made to the resistance of volcano rabbits (*Romerolagus diazzi*) to VHD determined in another study. Gavier-Widen and Morner reviewed the epidemiology of EBHS in Scandinavia. The disease has been seasonal in Denmark and southern Sweden since the early 1980's, with the highest incidence in the fall. The EBHS has not been reported in Norway or Finland.

Vaccination and control of VHD was discussed. Arguello Villares described large-scale vaccination in Spain using a commercial beta-propiolactone inactivated tissue-origin vaccine. Vaccinated animals responded serologically and were protected in the face of the disease within

4 to 5 days after vaccination. One dose of vaccine protected rabbits during their productive life (about 1 yr). Huang described the use of tissue-origin and cell culture-origin vaccines to effectively control VHD in the PRC. Passive protection with hyperimmune serum provided short-term protection for exposed rabbits. Rodak et al. reviewed the effective diagnosis of VHD in the Czech and Slovak Federal Republics using a wide array of diagnostic tests including antibody detection by ELISA and Western blotting. Aluminum hydroxide and oil-adjuvanted inactivated tissue-origin vaccines induced protective immunity.

The OIE provided a significant service by sponsoring a symposium to focus on VHD and EBHS. The resulting publication is of great value to those interested in gaining an in-depth perspective on these emerging diseases.

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