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NEUROLOGICAL SYNDROME DUE TO EQUINE HERPES VIRUS

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INTRODUCTION

Equine herpes virus is a worldwide known disease that affects horses of all breeds, sex or age. Its most common form is equine herpes virus-1 (HVE-1), which causes great economic impact as it is a major cause of abortions, perinatal mortality in foals, respiratory and neurological syndromes.^{2,9,8} Neurological disorders caused by equine herpes virus-1 (EHV-1) are called equine herpes virus-1-associated myeloencephalopathy (EHM) and are considered an emerging contagious syndrome.¹⁰ There are many challenges faced when dealing with the disease, with diagnosis, prevention and treatment being the main ones. Therefore, the objective of this work is to summarize the topic of myeloencephalopathy caused by VEH-1 in horses and highlight the main challenges encountered in relation to this disease.

METHODOLOGY

Scientific articles were used, including case reports, articles from indexed magazines and dissertations, all found on Google Scholar. Priority was given to reading the last 10 years due to the small number of articles published in the last 5 years on the topic.

THEME SUMMARY

The primary infection occurs in the respiratory epithelium and its dissemination results in infection of the respiratory ganglia within 24 to 48 hours after contamination, establishing viremia associated with leukocytes that carry it to other tissues.^{3,8,7} fig. 1. Viremia persists for at least 14 days, which is a prerequisite for EHM, allowing the transport of the virus to the CNS, as well as the risk factors that determine its occurrence, where infection of endothelial cells occurs, but the mechanism underlying CNS endothelial infection is unknown, as are the risk factors that determine its occurrence,

initiating an inflammatory cascade, vasculitis, microthrombosis and extravasation of mononuclear cells, resulting in perivascular cuffing and local hemorrhage.^{3,1}

Although clinical diagnosis is common in the field routine, it can be somewhat problematic due to the diffuse and multifocal distribution of lesions in the central nervous system, which can cause considerable variability in the clinical presentation of the disease 10. The neurological signs of MHE varied considerably in severity as described in several articles, which may appear 6 to 10 days after infection, reaching maximum severity within 48 hours⁴, ranging from quadriplegia to mild incoordination². The most common clinical signs are ataxia and flaccid paresis of the hind limbs.^{2,6,1}, in addition to hypotonia of the tail and anus, and urinary incontinence^{2,6,5}.

Treatment of the disease is predominantly supportive^{2,6}, and the use of corticosteroids is recommended, which can help control or prevent the cellular response adjacent to the infection of CNS endothelial cells, potentially reducing vasculitis, thrombosis and resulting neural injury³, dimethyl sulfoxide (DMSO) is also recommended as it will inhibit placental aggregation in addition to stabilizing free radicals^{2,6}. In addition, antibiotic therapy is used to prevent possible secondary infections.⁶ It is recommended that animals infected with the disease be isolated because the virus is contagious.^{4,6}, and in animals that are in lateral decubitus, the side of the decubitus must be changed every 2 or 4 hours to reduce musculoskeletal injuries⁴.

Herpes virus myeloencephalopathy is a disease with an unfavorable prognosis that commonly leads to death. In a study carried out by Van Galen (2015) in France from 2008 to 2011, a mortality rate due to MHE was found to be 46%. Despite the existence of a vaccine against herpes virus, several articles and case reports show that vaccination against

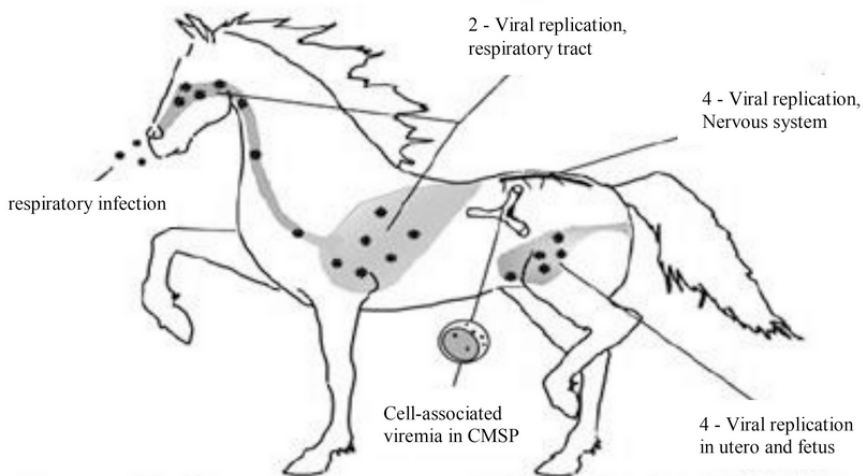


Figure 1: Schematic representation of the events that occur due to primary EVH-1 infection in a susceptible horse (source: Sáenz et al., 2008)

LVH can prevent miscarriage, however no current vaccine has been shown to prevent contamination by the virus or its neurological form.^{2,9,3,1} It was also found that there is a higher rate of isolated cases of the disease, with no relation to other cases¹⁰. In addition to the fact that infection generally occurs in the first weeks or months of the foal's life, and that vaccination, despite preventing abortion,^{2,9,3} does not prevent transmission of the virus from mother to foal⁹. As for the predisposition of EHV-1 to induce myeloencephalopathy, it may be associated with significant endotheliotropism⁶.

FINAL CONSIDERATIONS

The equine herpes virus can cause serious problems for equine farming, as it is a worldwide virus, present in almost the entire equine population. VEh manifests itself differently in each organism depending on the associated predisposing factor, the degree of infection and the viral strain. Despite the high rate of isolated cases, the virus is highly disseminated in herds and can remain in a latent period for several days, and even despite vaccination, infection can still occur. Studies have shown the low efficacy of the vaccine

against VEh, leading to the need for new research to develop a vaccine that is effective against the neurological form and that can reduce the spread of the disease. Research is also needed into the etiology of the disease so that we can develop a more effective treatment against MHE.

When it comes to diagnostic methods, there is still a certain difficulty in carrying out specific tests routinely in the field, meaning that many diagnoses are clinical without laboratory confirmation. The delay in receiving results, the lack of conditions in the field to properly send samples, as well as often the owner's refusal to send the tests are the biggest obstacles. The development of a rapid diagnostic test could solve these problems in addition to reducing underreporting of the disease.

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