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One of the most prevalent and costly infectious viral diseases of horses is EHV-1. This virus can result in devastating consequences for horses when large scale outbreaks of either respiratory, abortion or myeloencephalopathy occur. The respiratory form of this disease destroys epithelium of the upper airway, while abortions caused by this virus result in infection and death of the fetus and finally the neurological form of the disease results from infection of the endothelial cells lining the nervous system leading to vasculitis and necrosis. EHV – 1 isolates recovered from several outbreaks contain a mutation of EHV-1 (A2254 → G mutation in genomic open reading frame # 30) virus that possesses enhanced neuropathogenicity. EHV-1, including the mutated strain appear to be entrenched in many horse populations. Work by Allen et al has shown that this Herpesvirus mutant has established a well-entrenched reservoir of latently infected horses. Molecular characterization from archived isolates of EHV – 1 from Kentucky abortions demonstrates a statistically significant increase in the proportion of isolates that contain the ORF30 A2254 → G mutation over the last 50 years.

This virus has the potential to increase its virulence and expand its cellular tropism. Outbreaks of EHM have been observed since 1966 but recently there appears to be an increase in the morbidity and mortality associated with the disease. Horses demonstrating neurological signs associated with outbreaks of EHV-1 were more sporadic prior to 2000. At this time there appears to be an emerging problem caused by a high-mortality EHV – 1 myeloencephalopathy in the U. S. where the potential to cause neurologic disease is significantly different between the wild-type EHV-1 virus and the mutant strain. In an experimental infection with the mutant strain, Allen et al used 12 old horses with no detectable cellular immunity against the Herpesvirus and resulted in a 67% neurologic attack rate and 75% neurologic mortality. Another experimental infection in 12 horses using wild-type abortion-storm isolate resulted in no neurologic disease.

The principal mode of transmission is horse to horse via the mucosal epithelium of the upper respiratory tract. Nasal shedding may last up to 14 days with clinical signs observed within 3 to 6 days after exposure. To prevent disease in naïve animal’s best practices should include vaccine maintenance, although existing vaccines may not provide sufficient protection. Vaccine ineffectiveness may be a result of exposure to a large quantity of virus or exposure to a particularly aggressive strain of virus or suboptimal performance of available vaccines.

How do we know it’s EHM? Typical clinical signs may be very helpful with identification of infection by this virus. Signs such as ascending neurological signs including poor tail tone, urinary incontinence and weakness and ataxia beginning in the pelvic limbs. In addition the finding of xanthochromic CSF, along with identification or isolation of EHV-1 from the respiratory system or the buffy coat is helpful. Identifying a four fold rise in complement fixing or virus neutralization antibodies in acute and convalescent serum titers collected 7 to 21 days apart is also very useful. PCR on nasal swabs or buffy coat samples, viral culture, and immunohistochemistry or immunofluorescence may be used. In some cases it is impossible to achieve a definitive diagnosis without benefit of a post-mortem.
The management of horses with suspected EHV1 myeloencephalopathy should be directed at achieving a safe environment and providing excellent nursing care. A horse with obvious bladder dysfunction should quickly and as frequently as possible have aseptic evacuation of the bladder. Currently available anti-viral agents do not appear to provide sufficient impact on the morbidity and mortality of horses stricken by this EHV – 1 infection, perhaps due to ineffective dosing or poor bioavailability. Recent information on the use of valacyclovir has been quite positive recommending doses of 27 mg/Kg tid for one week followed by 18mg/Kg bid for 14 days.\(^5\)

The principal means of prevention and control of EHV-1 diseases are maintenance of good vaccination of horses on the farm and careful attention to sound herd-management practices.

1. Allen, GP; Kydd J; Slater, JD; Smith KC. Equid Herpesviruses 1 and 4 infections. Infectious Diseases of Livestock, 2004; 2 (Ed2) :829-859.
5.Henninger, RW; Reed SM; Saville, WJA , Allen, GP; Hass, GF, Kohn, CW, Sofaly C. Outbreak of neurologic disease caused by equine Herpesvirus-1 at a university equestrian center. JVIM 2007; 21(1): 157-165