Equine Herpes Myeloencephalopathy
A Practical Approach to Diagnosis, Treatment and Quarantine

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Equine herpesvirus – 1 is a ubiquitous virus among the equine population. It has been estimated that up to 80% of horses are latently infected regardless of their vaccination status.¹ There are two variants of the virus due to a single nucleotide mutation in the DNA polymerase gene. The mutation leads to an amino acid residue change at position 752.² The variant D752 is the neuropathogenic form that is estimated to be responsible for 74-85% of the cases of equine herpes myeloencephalopathy (EHM) and is associated with high levels of viremia. The nonneuropathogenic form of the virus, N752, is responsible for 15-26% of EHM cases and 81-98% of abortions.³

Initial infection occurs at the time of weaning through the respiratory route regardless of vaccination status.¹,⁴ Nasal shedding and fever begin on day 1. On days 2-4, the virus infects lymphoid tissue and local lymph nodes. A cell associated viremia occurs 4-10 days later. The viremia causes a second fever and disperses virus throughout the body where it contacts endothelial cells and results in a vasculitis. Nasal shedding usually continues for 14 days but it has been documented for up to 3 weeks. In an EHM outbreak 10-40% of horses will exhibit neurologic signs that occur during the viremia or second fever wave.³ Latency occurs several weeks after the primary infection in the lymphoreticular system and trigeminal ganglion. Reactivation due to stress occurs from these sites often with no clinical signs.¹

If confronted with a possible case of equine herpes myeloencephalopathy (EHM) the veterinarian must think of other possible causes of neurologic signs. Ask about rabies vaccinations status. Even though rabies vaccinations are thought to be 100% protective, due to the zoonotic potential and fatal outcome, any horse with unexplained neurologic disease that dies should always be tested for rabies. A list of all people who have had contact with the horse over the last 10-14 days should be compiled by the owner or barn manager. Wear gloves to protect yourself and minimize spread of disease. The diagnostic rule out list for neurologic disease in the horse is long. Clinical signs most consistent with EHM are fever, ascending paralysis, urinary incontinence and cranial nerve signs. Severely affected horses may be recumbent or in a dog sitting position. Possible rule outs depending on clinical signs at presentation are equine protozoal myelitis, trauma or tumor of the lumbar spine, West Nile virus, Lyme’s disease, and botulism. Although encephalopathy rarely occurs with EHM, if it is present, hepatoencephalopathy and eastern equine encephalitis should be considered.

Real time or nested PCR performed on the buffy coat of whole blood and on nasal swabs is the diagnostic test of choice for EHM in a clinical setting. Know what lab you will use in advance. The Virginia state diagnostic lab and many university and commercial labs provide this service. Request typing of the virus if the sample is positive for viral DNA. If the animal is to be euthanized, collect blood and the nasal swab before euthanasia. Testing for rabies will delay any post mortem results. Having the results of the bloodwork and nasal swab will help determine if a continued quarantine is necessary as soon as possible. The veterinarian may want to consider other diagnostic tests depending on clinical signs. Cerebrospinal fluid collected from a lumbosacral tap and serum can be sent for sage 2/3/4 serum:csf titer ratio for equine protozoal myelitis and serum:csf titer ratio for multiplex Lyme’s testing. Cytology and fluid analysis of the spinal fluid may reveal xanthochromia, an increased protein concentration and an increased albumin quotient in the case of EHM. If the horse is vaccinated for West Nile Virus, blood in a red top tube should be submitted for an IgM. Serum chemistry and blood ammonia should be run if hepatoencephalopathy or hyperammonemia are suspected. Trauma of the lumbosacral spine can be difficult to document with radiographs or ultrasound. A bone scan may be required depending on location.
If you suspect EHM, please call the State Veterinarian’s office immediately. In order to prevent further spread of the disease, impose a voluntary quarantine on the farm. No horses should enter or leave the premises. Isolate all horses with clinical signs including a fever. Separate exposed horses by at least 30 feet from non-exposed horses. Advise the barn manager to take the temperature of all horses twice a day. No people including the farrier should have unnecessary contact with any of the exposed horses. Instruct the barn staff on biosecurity protocol.

There is no specific treatment for EHM. Therapy should be directed at reducing inflammation, supportive nursing care and providing adequate nutrition. Horses showing neurologic signs should be placed in a padded stall or small paddock that has good footing. Slings should be used for horses that require assistance to stand but once standing can support their own weight. If a horse is recumbent, it should be maintained in sternal and rolled every 2-4 hours. Special attention should be paid to keeping the bedding clean to avoid decubital ulcers. Current recommendations are to treat febrile only horses with antivirals and anti-inflammatories and to treat neurologic horses with dexamethasone, antivirals and anti-inflammatories. Flunixin meglamine is the most commonly used non-steroidal anti-inflammatory. Dexamethasone may help decrease CNS inflammation if used for 2-3 days (0.05-0.25 mg/kg IV, IM q 24 hr). Valacyclovir (30 mg/kg per OS q 8 hours for 6 treatments followed by 20 mg/kg q 12 hours for a total of 10 days) may help prophylactically and for early treatment. Gancyclovir (2.5 mg/kg q 8 hour IV for 1 day then 2.5 mg/kg q 12 hours IV for one week) may be more useful later in the disease course. Pentoxifylline to decrease thromboembolic events and α-tocopherol, an anti-oxidant may also be used. Manual evacuation of the bladder or bladder catheterization may be necessary. In these cases, broad spectrum antibiotics such as trimethoprim-sulfa are indicated to prevent cystitis.

Vaccination as a control strategy in the face of an outbreak is controversial. Vaccination will not block infection, prevent viremia or prevent latency. One study identifies vaccination within 5 weeks of an outbreak as a risk factor for the development of EHM. However, nasal viral shedding is reduced in horses vaccinated with single component inactivated vaccines. These vaccines induce high serum titers of virus neutralizing (VN) antibody. If vaccination is chosen during an outbreak, only afebrile and asymptomatic horses should be vaccinated. Pneumabort-K® (Zoetis) and Prodigy® (Merck) are currently the vaccines of choice.

References