Have you ever wished you could pull all the scientific recommendations on equine protozoal myeloencephalitis (EPM) diagnosis and treatment together in one place? Done! One practitioner summarized the available literature on EPM diagnosis and treatment for a large crowd of equine veterinarians.

“Sarcocystis neurona (a species of protozoa) infection is the most common cause of EPM,” began Amy L. Johnson, DVM, Dipl. ACVIM, of the University of Pennsylvania. “This neurologic disease presents a diagnostic challenge to practitioners because many horses are exposed to the protozoa and clinical signs can mimic many other conditions. Treatment is also challenging because several medications are available and response to treatment is not consistent among horses.”

Diagnosis
Diagnosing EPM in a live horse is challenging because no test is 100% accurate; the gold standard for diagnosis is finding the protozoan parasite in the spinal cord, which can only be sampled after the horse is euthanized. So for live horses, diagnostics include a complete neurologic exam, exclusion of other common neurologic conditions, and confirmation of exposure to S. neurona through analysis of serum (clear fluid portion of the blood) or cerebrospinal fluid (CSF, the fluid surrounding the brain and spinal cord).

“If clinical signs cannot be attributed to lesions in one or more regions of the central nervous system (CNS), EPM should not be considered,” Johnson advised. “However, EPM can mimic almost any neurologic disease and cause signs ranging from a single cranial or peripheral neuropathy to diffuse CNS dysfunction. Certain patterns, such as multifocal signs, mixed lower motor neuron and upper motor neuron signs (e.g., both muscle atrophy and spinal proprioceptive ataxia), and asymmetric signs are more commonly observed with EPM than other neurologic diseases.”

Tests for EPM in live horses include the Western blot, indirect fluorescent antibody test (IFAT), and surface antigen-1 ELISA test (SAG-1 ELISA). All detect the presence of antibodies in the horse’s serum or CSF, but keep in mind that a positive test on any of them does not necessarily mean the horse has clinical EPM. Antibodies just confirm that he has been exposed to S. neurona.

Treatment
“None of the available/approved treatments are obviously superior; all have shown about a 60% success rate in clinical trials,” reported Johnson.

She explained that sulfadiazine/pyrimethamine combination (Rebalance), ponazuril (Marquis), and nitazoxanide (Navigator) medications are the approved treatments that have been commercially available, while diclazuril is approved for EPM treatment, but is not yet marketed.

Navigator manufacture was discontinued in the spring of 2009, she noted.

Sulfadiazine/pyrimethamine interrupts folic acid metabolism, and this treatment is usually given for 90-270 days. It was successful at eradicating antibodies to the protozoa from CSF and/or improving neurologic signs by at least two grades in 61.5% of horses in one study. Side effects can include fever, anemia, anorexia, depression, worsening of ataxia (incoordination), and abortion.

Ponazuril inhibits energy metabolism in the protozoa, and it is given for 28 days. The published study on its use found 62% of horses had at least one grade of improvement of neurologic signs and/or no longer had antibodies to the protozoa in their CSF. Side effects are rare, but a few treated horses had blisters on the nose and mouth, skin rash or hives, loose stools, mild colic, and one horse had a seizure.

Nitazoxanide (no longer available) inhibits a step of anaerobic (without oxygen) energy metabolism in the parasite.
Improvement of at least one grade in neurologic signs was seen in 57% of horses in one study and 81% in a second study that was “less stringent” in selecting affected horses. Adverse reactions can potentially be more significant with this medication, including the possibility of fatal enterocolitis (inflammation of the small intestine and colon) at the recommended dose. Milder adverse effects included fever, anorexia, and lethargy/depression.

Diclazuril's mode of action is unknown, but it might be similar to that of the related medication ponazuril; it is also given for 28 days. In one study, 67% of affected horses improved by at least one neurologic grade and/or no longer had antibodies to protozoa in their CSF after treatment. Adverse effects observed might or might not have been due to diclazuril, and they included laminitis and deteriorating neurologic status.

Recent research has investigated one more option, Johnson reported—that of toltrazuril sulfone or ponazuril given at a loading dose along with dimethyl sulfoxide (DMSO) to improve uptake of the medications. She noted that with this strategy, high levels of the medications show up in the CSF in about a day, possibly resulting in quicker action of the medications and a shorter duration of treatment/less risk of adverse effects.

"Prospective, randomized, blinded clinical trials would aid in assessing if one drug shows superior efficacy," Johnson concluded. “Based on reported side effects, ponazuril and diclazuril seem to have the fewest reported adverse effects.”

West Nile Virus: A Different Genetic Lineage Evolving?

It's a variant of Murphy's Law: Anytime you think you have a handle on something, the unexpected happens and you're off balance again. The equine health world might be in this boat now concerning West Nile virus infection in horses. With several different types of vaccines available and in widespread use in the United States over the last several years, West Nile virus had gone from terrifying to just another disease to watch out for.

Enter a new variety of West Nile virus, known as West Nile virus genetic lineage 2. This form was previously only seen in horses in sub-Saharan Africa and Madagascar, but it is now showing up in Europe, reported Orsolya Kutasi, DVM, of the Szent Istvan University, in Hungary. She discussed the first known outbreak of West Nile virus genetic lineage 2 infection in European horses, which affected 18 animals in Hungary in 2008.

Previously, the lack of significant outbreaks with this form had led to the belief that it was not very virulent—that it was not capable of causing severe disease like the lineage 1 form. However, this case series suggests the virus' virulence might have increased, as it showed morbidity (illness) and mortality (death) rates in this outbreak that are similar to published lineage 1 rates.

Kutasi explained the 18 cases of lineage 2 infection showed clinical signs quite similar to those of lineage 1 infection—weakness, ataxia (incoordination), muscle twitching, recumbency (lying down), fever, and hyperesthesia (hypersensitivity). Thirteen of the horses survived, and 10 recovered fully.

"The occurrence of the disease was not limited to a certain wetland area as it had been typical for European outbreaks, but we experienced significant northwestern spread of the pathogen," noted Kutasi.

Perhaps the largest cause of concern with this form is that the West Nile virus vaccines currently on the market were not tested against lineage 2 strains, according to Kutasi—thus, the vaccines might or might not cross-protect against lineage 2. Researchers hope they will soon be able to answer this question.

New EHV Treatment Option Studied

The typical veterinarian's arsenal against equine herpesvirus includes vaccination to prevent the disease and supportive care/antiviral medication when the disease strikes. However, one more weapon might eventually be added to that list, somewhere between vaccination and treatment: siRNA administration during outbreak conditions.

Small interfering RNA, or siRNA for short, is a Nobel Prize-winning technology that Cornell University researchers have been studying for equine use. A study on the efficacy of siRNA treatment against equine herpesvirus-1 (EHV-1) infection was presented by Margaret M. Brosnahan, DVM, MS, Dipl. ACVIM, of Cornell.

"Complete prevention of EHV-1 infection is ideal, but unrealistic," she commented, noting that the virus can become dormant in the body (latent infection) and reactivate later. Also, immunity from vaccination is very short-lived, making it very hard to completely prevent disease. “So our goals are to reduce viral shedding, minimize disease transmission, reduce viremia (levels of virus in the bloodstream), and reduce the severity of clinical signs.”

The treatment strategy used siRNAs targeted against two different EHV-1 genes involved in viral entry and replication. Equine herpesvirus can replicate (reproduce) very quickly, generating large amounts of virus to sicken the horse and spread to others. Thus, inhibiting its replication would logically reduce viral shedding into the environment (and, thereby, disease transmission), viremia, and the severity of clinical signs of disease.

For the study, 10 horses 3-18 years of age were given siRNA targeted against EHV-1 intranasally (into the nose) 12 hours before and after the horses received a “dose” of EHV-1 intranasally. Four horses received siRNA directed against a firefly gene as controls.

Unexpectedly, Brosnahan reported, “There was no significant difference in viral shedding, viremia, or initial fever between the two groups.” However, clinical signs of disease developed in fewer treated
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