

Equine Protozoal Myeloencephalitis: Less Common Than We Thought?

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"Equine protozoal myeloencephalitis (EPM) is a common cause of neurological disease of horses in North and South America, and results from a protozoal infection with *Sarcocystis neurona* or *Neospora hughesi* (less commonly)," stated Steve Reed, DVM, Dipl. ACVIM, of The Ohio State University (OSU), at the Western Veterinary Conference held Feb. 20-24 in Las Vegas, Nev. However, it might not be as common as was previously thought.

"Why bother EPM testing?" Reed asked the audience. While studies have shown the exposure rate of horses to *S. neurona* to be only about 52%, he said, many practitioners assume that nearly all horses have been exposed and elect to begin treating suspect cases rather than spending time and money on a test they think will be positive anyway.

"But what if that horse is seronegative (indicating no exposure to the parasite)?" he asked. "Do we spend hundreds if not thousands of dollars of the client's money to treat a negative horse on a therapeutic trial?" he went on. "*S. neurona* Western blot test (WBT) on (blood) serum is cheap and easy, with very few false negatives. With a very acute onset you might get a false negative, but there are very few of those."

Although complete results are not yet available, Reed discussed an ongoing study of EPM exposure being conducted by OSU and Equine Biodiagnostics/IDEXX. The researchers evaluated serum WBT results in neurological patients presented for diagnosis from 2000-2003.

They found that the incidence of seropositive (exposed) horses was as follows: California 35%, Oklahoma 80%, Texas 65%, Kentucky 66%, Florida 56%, New York 57%. In most cases, these numbers are far from supporting any assumption that nearly all horses are exposed to EPM.

In addition, "The actual seropositives in the general population may be much lower," Reed commented. In other words, since most of the study horses were tested because they already exhibited neurologic signs, they don't represent a random sample and might be expected to have a much higher incidence of any pathogens causing neurologic signs than the general horse population. "Seropositive rates of horses with CNS disease are about twice as high as the general horse population," he added.

Jennifer Morrow, PhD, a scientist and equine consultant with Equine Biodiagnostics/IDEXX, who is working on this study, notes that several published numbers on EPM seroprevalence are geographically narrow. "They usually only studied several hundred horses in a fairly limited geographic area," she said. In contrast, the current study looks at thousands of samples from horses living in many widely scattered states.

In consulting with IDEXX's sales representatives, who talk to veterinarians in many regions, she commented, "It has become clear to us that there is a misconception that all samples test positive, so many practitioners don't test suspected cases any more. There is a lot of value to a negative result, probably more so than a positive result. It's a small thing to help make a better diagnosis.

"There are some really big differences by region--it's really kind of amazing," she added. "So far year to year, (seroprevalence rates) are pretty consistent." More data has yet to be analyzed; the complete results will be released later.

What's Next?

It is certain that new tests need to be developed, specifically ones that will provide clinically

relevant information, such as possible predispositions of horses to develop clinical EPM.

"We are working on an assay at Ohio State that we believe might give an indication of how recently a horse was exposed and/or infected," Reed said.

"It should be emphasized, however, that any new test should be based on scientifically accepted principles and be properly and adequately validated prior to actual use," Morrow cautioned.

What We Do Know

Reed also discussed some of the basics of what is known about this sometimes elusive disease. "Some of the controversies that we first started recognizing about this disease have remained for so long afterward," he said. "The classic asymmetric dramatic muscle wasting (clinical sign), if consistent, would help us a lot, but it's not always that way. We say it's asymmetric, but often it's symmetric. We say the disease only causes lower motor neuron signs (such as weakness with muscle wasting), but it often causes upper motor neuron signs (such as spasticity and circumduction--moving a body part in a circle--as well as weakness). It's still difficult to evaluate and test for. And I can't tell you a lot about vaccination because we're still doing studies with the company."

He went on to show videos of several confirmed EPM cases to illustrate the clinical signs peculiar to the disease, and described OSU's previous work with the disease. "Our research team on EPM began with clinical cases, then focused on risk factors, the life cycle of *S. neurona* and a model of this disease, clinical trials to evaluate new treatments, and disease prevention. This work requires many people with broad knowledge, interests, and talents."

He reported that a retrospective case control study investigating EPM risk factors found the following factors to increase a horse's risk of getting EPM:

- Season (spring/summer worse);
- Previous cases diagnosed on the premises;
- Opossums on the premises;
- Stress prior to admission; and
- Racehorse and show horse occupations.

The OSU team found that feed security (from vermin) and having a creek or river on the premises decreased a horse's risk of getting EPM. "So if the definitive host (the opossum) can't get in and contaminate feed, this is good. And if there was another water source for opossums (decreasing their need to raid horses' water sources and thus their contact with the horses' environment), that helped a lot too," Reed commented.

The probability of a horse having EPM increases with neurological signs, positive WBT result on a blood sample, positive WBT result on cerebrospinal fluid (CSF), and a positive PCR (polymerase chain reaction) result on CSF. Characteristic lesions at necropsy or finding the organism in tissues at necropsy are the gold standards of diagnosis.

He also noted that treatment with targeted medications increased the likelihood that an affected horse's condition would improve, while a decreased likelihood of improvement was seen with breeding and pleasure horse occupations.

With *S. neurona*, the opossum is the definitive host, the horse is an aberrant host, and raccoons, cats, armadillos, and skunks are intermediate hosts. "If you find these animals dead on your property, get rid of them," he urged. "Burn, bury, or otherwise dispose of them. The parasite tends to be in tongue muscles, and there can be up to a billion sporocysts (the stage of *S. neurona*'s life cycle that can infect horses) in a gram of (opossum) feces. A horse can easily ingest these."