Equine Protozoal Myeloencephalitis

STACEY OKE, DVM, MSC

A well-known cause of neurologic disease in horses, EPM is considered a diagnosis of exclusion

Equine protozoal myeloencephalitis (EPM) was first recognized in the mid- to late 1960s as a “segmental myelitis.” It wasn’t until 1974 that scientists identified a single-celled parasite that prompted the name change to equine protozoal myeloencephalitis. We now know two parasites, Sarcocystis neurona or Neospora hughesi, can cause EPM, but most cases are caused by S. neurona.

How S. Neurona Causes Disease

Opossums are the definitive hosts for S. neurona. They become infected by scavenging on intermediate hosts (skunks, cats, raccoons, armadillos) with S. neurona sarcocysts—walled-off “pockets” containing the infectious form of the parasite, which has reproduced here asexually—in their muscles. Once ingested, mature sarcocysts release bradyzoites, which transform and reproduce sexually in the small intestine, producing oocytes. The opossum sheds fully sporulated oocysts or sporocysts in its feces. They contaminate the environment and infect intermediate hosts when consumed.

Horses are incidental hosts that become infected by consuming the sporocysts in the environment. Unlike intermediate hosts, horses— as incidental or aberrant intermediate hosts—do not serve as reservoirs for infecting opossums (i.e., sarcocysts do not form that opossums later consume) and do not spread S. neurona directly to other horses.

Once ingested, what happens to those sporocysts remains unclear. Researchers know they release sporozoites, which travel to the spinal cord but not how and when. They also don’t know why the sporozoites migrate to the spinal cord in some horses but not others.1,2

Picking and Choosing its Victims

Exposure to S. neurona is common, but infection is rare. For example, anywhere from 15-89% of horses have antibodies to S. neurona in their bloodstream, indicating they’ve been exposed to the parasite; however, the annual incidence of disease is less than 1%. In other words, infection does not equate to disease.1,3

Risk Factors

Horses diagnosed with EPM are often younger than 4 or older than 13. The highest number of cases occur in the fall and the fewest in winter. When caretakers prevent wildlife access to horse feed, the likelihood of disease drops by one-third.

Stress might increase a horse’s chances of developing EPM. Specifically, immune compromise, heavy exercise, transport, injury, surgery, or foaling increase a horse’s chances of infection resulting in clinical disease.1,3

The Shadowy Face of EPM

When the parasite spreads throughout the horse’s body, it can “land” anywhere in the brain, brainstem, or spinal cord. Thus, every case is unique (i.e., EPM has no “classic” presentation).

Horses typically exhibit signs consistent with spinal cord injury, such as ataxia (incoordination), gait abnormalities that can mimic lameness, and muscle weakness and/or atrophy.1,5 Less commonly, horses have encephalopathic (brain) manifestations such as cranial nerve deficits manifesting as dysphagia (difficulty swallowing, abnormal airway function) or abnormal behavior and state of consciousness or even seizures.1,3

The parasite can infect both white and gray matter anywhere in the brain or along the spinal cord.1,6 The parasites can be found in multiple locations with no apparent pattern.

One relatively consistent finding, however, is asymmetric neurologic deficits and focal muscle atrophy in affected horses. This is because the parasites do not affect both sides of the brain or spinal cord evenly. Further, the disease can begin acutely or have an insidious onset. In either case it is progressively debilitating.1,3

A Diagnostic Challenge: Exclusion Is Key

Albeit an uncommon disease, EPM is still the most common infectious neurologic disease diagnosed in horses. Vets must always consider EPM in horses displaying signs consistent with central nervous system (CNS) disease and distinguish EPM from lameness, which is not always easy.2,4

The long list of neurologic conditions vets must distinguish EPM from includes cervical vertebral stenotic myelopathy, equine herpesvirus-1 myeloencephalopathy (EHM), rabies, toxins, trauma, Lyme neuroborreliosis, equine degenerative myelopathy, and more.2 Recently, scientists identified Toxoplasma gondii as another parasite capable of causing EPM-like disease in horses.6

Currently, the only way to definitively diagnose EPM is to identify parasites in the brain or spinal cord post-mortem. Even then, parasites are identified in fewer than 50% of cases with other characteristic microscopic changes (e.g., inflammatory changes in the brain and spinal cord). This approach is not helpful in a clinical setting, and laboratory testing must be performed in conjunction with a comprehensive clinical examination.1,2

Simply detecting antibodies against S. neurona or N. hughesi in the horse’s bloodstream has minimal diagnostic value. Only if a horse is negative can you rule out EPM (although there are exceptions to this rule in very acute cases). Even the presence of antibodies in the cerebrospinal fluid (CSF) bathing the brain and spinal cord does not definitively diagnose EPM, because antibodies can passively diffuse from the bloodstream into the CSF.

Therefore, the serum:CSF antibody titer ratio is
When it comes to EPM, time matters.

EARLIER TREATMENT CAN LEAD TO BETTER OUTCOMES.

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