FOCAL POINT

Equine protozoal myeloencephalitis (EPM) can be easily misdiagnosed when physical and ancillary diagnostic findings are not carefully interpreted.

KEY FACTS

■ All horses being evaluated for EPM should undergo a complete physical and diagnostic evaluation for conditions that can mimic clinical signs before EPM-specific treatment is instituted. (p. 976)

■ Neurologic evaluation should not be based on one or two subtle abnormalities; corroborative evidence of neurologic deficits should be sought. (p. 976)

■ When interpreting laboratory tests for EPM, their limitations should be considered. (p. 978)

■ As a means of diagnosing EPM, response to treatment should be carefully interpreted; consideration should be given to the possibility that an animal has responded to some non-EPM-specific therapy or to test. (pp. 979–980)

■ An antemortem diagnosis of EPM is tentative. (p. 980)

Diagnosing Equine Protozoal Myeloencephalitis: Complicating Factors*

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ABSTRACT: Equine protozoal myeloencephalitis (EPM) may be one of the most difficult ante-mortem diagnoses for practitioners to make accurately. Much of this difficulty results from the perceived clinical variability in the presentation of EPM. Because antemortem laboratory testing for EPM is only one piece of the diagnostic puzzle, diagnostic accuracy relies on the completeness of physical, neurologic, and diagnostic evaluations.

Equine protozoal myeloencephalitis (EPM) is a disease of great concern to all members of the horse industry. Horses diagnosed with EPM may be of any breed or background. For the owners and trainers of these animals, a diagnosis of EPM carries a sentence of long-term treatment, uncertainty of response, frustrations associated with a myriad of opinions pertaining to the disease, and the possibility of relapse and/or lack of response to treatment.

Despite the existence of laboratory tests for EPM, no single test can be used as a definitive means of antemortem diagnosis. Indeed, to date, a definitive diagnosis is reached only by postmortem identification of characteristic spinal cord lesions and/or a causative parasite (e.g., *Sarcocystis neurona*, *Neospora species*). For this reason, the most accurate antemortem diagnosis is reached by careful interpretation of physical and neurologic examinations, ancillary diagnostics, and laboratory findings. Each of these components of the diagnostic process has its own inherent complicating factors.

PHYSICAL AND NEUROLOGIC EXAMINATION

Consideration of signalment, history, and disease progression often provide relevant clues in the initial evaluation of horses with neurologic disease. A general physical examination is important to provide a context within which neurologic examination findings are interpreted. The neurologic examination should identify and localize any demonstrable deficits of neurologic function. Ambiguous or nonspecific findings, such as lameness, weakness, loss of condition, uncharacterized muscle atrophy, and subtle gait changes, are not specifically diagnostic of neurologic deficits and are therefore even less definitively diagnostic of any specific neurologic disease, such as EPM.

Neurologic evaluation of horses relies heavily on assessment of gait. However, all horses with EPM do not necessarily manifest similar gait changes; gait aberrations vary greatly in severity and neuroanatomic location (Figure 1). In addition, numerous nonneurologic processes (e.g., musculoskeletal disease) can cause mild gait changes. Therefore, a change in gait should not be attributed to a neurologic abnormality based solely on one or two subtle maneuvers or stances. Corroborative neurologic abnormalities should be sought and carefully interpreted (Figure 2). A thorough gait evaluation must be performed with all clinically compatible neurologic and nonneurologic conditions in mind.

In one author's (B.G.B.) experience, cranial nerve (CN) deficits are often indiscriminately associated with EPM. Because of the long, superficial course of the facial nerve peripheral to the stylomastoid foramen, facial nerve deficits in horses without other neurologic abnormalities are often caused by direct trauma to this nerve (Figure 3). Facial nerve deficits also accompany vestibulopathy in horses with peripheral vestibular disease caused by otitis media/interna.[1] Clinical signs of ataxia and CN deficits are easily mistaken as specific signs of EPM when other neurologic disorders are not considered.

Horses that display signs of vestibulopathy (CN VIII

Figure 1—Severe posterior ataxia. Lesions producing this should be localized and a complete nervous system evaluation performed before equine protozoal myeloencephalitis is diagnosed.

dysfunction) should be evaluated in order to determine whether the lesion is central or peripheral. Peripheral vestibular disease should always be considered in horses with vestibulopathy in the absence of other spinal cord neurologic deficits. Facial nerve deficits that are localized outside of the brain stem can be associated with peripheral vestibular disease. Diffuse CN involvement implies larger lesions of the brain stem. In addition to EPM, these findings can be associated with space-occupying lesions or other inflammatory diseases. Vestibular disease is not a specific finding of EPM. Despite a description by MacKay of a "brain-stem syndrome" involving CN VII and VIII,[2] there is currently neither a physiologic explanation nor pathologic evidence that *S. neurona* exhibits a specific affinity for any CN nucleus. Therefore, radiography, otoscopy, endoscopy, and/or scintigraphy of the skull for peripheral vestibular disease are indicated as components of the complete evaluation of horses exhibiting vestibulopathy.[1]

Prolonged illness unrelated to the central nervous system (CNS) can cause muscle wasting that can be mistaken for neurogenic muscle atrophy. Muscle wasting tends to occur more diffusely than does muscle atrophy induced by neurologic lesions. Muscle atrophy also occurs with disuse due to musculoskeletal pain. Severe muscle atrophy occurs when a lesion disrupts motor input to the muscle by destroying the lower motor neurons supplying the muscle. Despite the fact that any focal neurologic disorder can cause severe muscle atrophy, muscle atrophy has often been specifically associated with EPM. However, upper motor neuron lesions are probably more likely to occur with EPM than are lower motor neuron lesions, and severe muscle atrophy is not a common finding of upper motor neuron lesions. MacKay has noted that muscle atrophy is not usually obvious with spinal cord disease resulting from EPM.[2] Spasticity, loss of coordinated movement, and weakness are more frequently encountered with upper motor neuron lesions; these findings also are not specific for EPM.

Clinicians of equal competence can often reach different conclusions from a neurologic examination, leading to disagreements regarding the accepted definition of *neurologic deficit*. Indeed, the neurologic examination itself has its own "clinician-specific" sensitivity and specificity. Clinicians who tend to identify more subtle movements as neurologic deficits are likely to have a very high sensitivity in the clinical identification of EPM (i.e., they miss very few or no cases of EPM clinically). However, based on the generally accepted low prevalence of EPM, such clinicians will also inevitably misdiagnose many horses as having EPM (low specificity). Animals that are misdiagnosed with EPM will yield a preponderance of false-positive test results from cerebrospinal fluid (CSF) immunoblot analysis.

Before deciding to engage in what could be a lengthy diagnostic workup, it is advisable to solicit the opinion of clinicians who are experienced in the neurologic examination, diagnostic procedures, and lameness evaluations necessary for complete EPM evaluation. When such trained individuals are unavailable, multiple evaluations by several clinicians may provide a "consensus" on whether full diagnostics should be pursued; however, variability in the interpretation of findings can lead to many conflicting opinions with this approach.

LAMENESS EXAMINATION

A rigorous lameness examination is essential for animals that do not appear to exhibit neurologic deficits or those with questionable or mild deficits. It is important to recall that musculoskeletal disease (lameness) probably occurs much more commonly than does EPM. There is currently no evidence that EPM causes primary lameness. Indeed, rigorous evaluation, accurate recognition, and management of chronic musculoskeletal conditions may successfully address many problems attributed to EPM.

A decrease in performance level in the absence of other clinical signs is perceived to be a common clinical manifestation of EPM. Although this is possible, clinical experience in the field indicates that EPM is less likely to be a major cause of poor performance than are

Figure 2—Limb placement tests alone are not diagnostic of nervous system deficits. Corroborative abnormalities should be sought.

many other nonneurologic conditions. Practitioners who frequently diagnose EPM in mild and nonclinically progressive cases should review their diagnostic approach to the disease and consider more rigorous diagnostics for EPM, lameness, and other clinically compatible disorders. Lameness is a common cause of poor performance, but it can be very difficult to identify and localize. Subtle lameness evaluations may require further assistance from practitioners with extra training and skills in orthopedics, sports medicine, and diagnostic imaging. In one author's (B.G.B) experience, many horses suffer from age-related deterioration of performance and/or from a myriad of chronic musculoskeletal conditions. Intensive management of these conditions can be very helpful for such horses but requires detailed explanation to owners and trainers so that their expectations for their horses' response and recovery are realistic.

Although back pain is a frequent clinical finding associated with EPM,[3] there is no evidence of or proposed cause for primary back pain due to EPM. Back pain associated with EPM is likely to be associated with gait changes that cause secondary pain in the epaxial musculature. Lameness of any type can also alter a horse's gait and lead to secondary back pain. Thus, back pain is also a nonspecific finding and is not necessarily indicative of EPM. In one author's (B.G.B.) experience, most horses exhibiting back pain are involved in regular performance and the presence of neurologic deficits is often difficult to substantiate. The clinical signs of neurologic deficits are often not easily distinguished from signs caused by or associated with musculoskeletal conditions.

LABORATORY AND ANCILLARY DIAGNOSTIC TESTING

Evaluation of horses displaying neurologic deficits often requires access to facilities, equipment, laboratories, and experienced people capable of supplying specialty services. Persistent frustration with variable performance levels caused by nonneurologic problems can easily lead to a diagnosis of EPM if physical, neurologic, and ancillary diagnostic evaluations are incomplete. Comprehen-

sive evaluation of horses should be aimed at ruling out the many possible nonneurologic causes of poor performance. Diagnostic evaluation should also address the other neurologic diseases that may cause clinical signs similar to those of EPM. CSF aspirates should be obtained only when a horse is determined to be neurologically abnormal and has undergone diagnostic evaluation to rule out other diseases. When this protocol is not followed, the proportion of false-positive immunoblot results will increase. Animals with clinical signs that can be explained by multifocal CNS lesions should have immunoblot analyses performed on blood and CSF.

The CSF albumin index and IgG quotient may be helpful in determining the integrity of the blood–brain barrier and intrathecal immunoglobulin production.[4,5] Although the albumin quotient is generally regarded as helpful in determining blood–brain barrier integrity, the IgG index has received less enthusiastic support as a useful diagnostic parameter. CSF cytology, protein content, and biochemical parameters can provide useful information for the evaluation of horses with neurologic deficits. Evaluation of CSF by polymerase chain reaction (PCR) testing for *S. neurona* can be performed but is apparently a low-yield test and may therefore be impractical for clinical use; this is presumably because neither the organism nor its DNA is normally found within CSF. It is currently unclear whether horses without clinical signs that are PCR-positive will necessarily become symptomatic. Treatment of horses that are PCR-positive regardless of clinical signs may be reasonable if PCR is assumed to exhibit high specificity.

The CSF immunoblot analysis can be a helpful diagnostic aid; it reportedly exhibits 89% sensitivity and specificity as defined against postmortem identification of EPM lesions.[8] Although the immunoblot is effective for detecting anti–*S. neurona* antibody, it is an indirect method of estimating the probability that an individual horse harbors *S. neurona*. Serum immunoblot analysis has been investigated and shown to only indicate exposure to the parasite[9-11]; thus the CSF immunoblot is be-

Figure 3—Left-sided peripheral facial nerve paralysis without other signs of nervous system deficit. In horses with left-sided facial nerve paralysis, equine protozoal myeloencephalitis should not be the primary differential diagnosis.

lieved to be a better indicator of the disease state.[12]

The experience of many clinicians, however, suggests that the rate of false-positive results of the CSF immunoblot may be underestimated. For this reason, it is important to consider other possible causes of a positive CSF immunoblot. The likelihood of a false-positive test result prevails when the disease prevalence is low.[6,13] EPM has been estimated to occur clinically in about 0.5% to 1.0% of the equine population[13]; based on the frequency of treatment of EPM in many practices, some clinicians may believe this estimate is low. However, most practitioners would probably agree that the incidence is less than 10%. Both of the above estimates suggest low disease prevalence, making the CSF immunoblot a poor screening test by yielding a preponderance of false-positive results.

Assuming that a group of neurologically abnormal horses will have a higher prevalence of EPM than will a neurologically normal group, the positive predictive value of the CSF immunoblot is maximized when it is performed only on neurologically abnormal horses.[6,8,13] Any neurologic condition may confound CSF immunoblot results when the blood–brain barrier is disrupted, allowing serum antibody leakage into the CSF. When this occurs, the albumin quotient can be used to help clarify the likelihood of antibody contamination of the CSF due to other neurologic disease.

Because of an apparent high yield of positive results and the ambiguity of interpretation, some clinicians no longer perform the CSF immunoblot. However, because negative immunoblot results are uncommon in horses with EPM, we advocate the CSF immunoblot as part of the routine EPM evaluation. When a negative CSF immunoblot result occurs, time and money are saved by minimizing the diagnostic pursuit and treatment of the wrong disease. If clinical signs are acute in onset, it may be worthwhile to perform a repeat CSF immunoblot in a few weeks to allow time for intrathecal antibody production. At this time, a negative result on a CSF immunoblot performed and interpreted with

consideration of all of the diagnostic tests' limitations precludes the diagnosis of EPM.

As mentioned, neurologic conditions other than EPM must be considered in the diagnosis of horses exhibiting compatible neurologic deficits. These conditions should be ruled out to maximize the likelihood that a positive CSF tap identifies a clinical case of EPM. A major diagnostic differential for EPM is cervical compressive myelopathy (CCM). Our clinical experience has indicated that many horses diagnosed with CCM have positive CSF immunoblot results. Seilman and coworkers found that a large proportion of horses with an antemortem diagnosis of EPM had spinal cord lesions caused by CCM without evidence of EPM lesions.[14] Therefore, high-quality survey cervical radiographs; measurements of minimal sagittal diameter; and, when indicated, myelography are important in the evaluation of horses for EPM.

Practitioners should compare the cost of a complete workup with that of prolonged treatment. Complete evaluation in a referral setting often compares favorably to the projected cost of treatment. Although a diagnosis of EPM can be reached in a number of ways, an accurate diagnosis is the product of complete physical, neurologic, ancillary diagnostic, and laboratory evaluation.

ANTIBODY AND CEREBROSPINAL FLUID

An assumption currently made regarding the interpretation of CSF immunoblot analysis and the pathogenesis of EPM is that a positive immunoblot result indicates the presence of *S. neurona* in the CNS, which invariably means that a horse is diseased. Although this assumption simplifies the diagnostic process, it is as yet unsubstantiated. The presence of anti–*S. neurona* antibody could be found within CSF (i.e., positive immunoblot result) of horses without clinical signs of EPM because of (1) blood contamination of CSF aspirate, (2) breakdown of the blood–brain barrier resulting from disease/inflammation unrelated to EPM, (3) normal filtration of antibody from blood containing high levels of antibody into the CSF at levels detectable by immunoblot analysis, (4) persistence of antibody in the CSF for long periods after resolution of clinical signs, and (5) the presence of *S. neurona* within the spinal cord without clinical signs of EPM. To date, it is unknown whether all horses that actually harbor *S. neurona* (either in the CNS or other undefined location[s]) have clinical signs of EPM. However, it is likely that these horses will have positive serum and possibly positive CSF immunoblot results.

Tourtellotte indicated that, in humans, antibody is usually filtered in low levels through the normal blood-CSF barrier.[15] In addition, persistence of intrathecal anti–*Borrelia burgdorferi* antibody has been recorded in humans years after clinical resolution of neuroborreliosis.[16] Lappin and coworkers showed that *Toxoplasma gondii*–naïve cats inoculated with soluble tachyzoite antigen plus adjuvant and cats previously orally infected with *T. gondii* tissue cysts that received adjuvant developed ocular and CSF *T. gondii*–specific IgG Goldman-Witmer coefficient (C) values greater than 1.[17] These authors concluded that *T. gondii*–specific IgG C values greater than 1 did not prove ocular or CNS infection in all cats.[17]

The C value assumes that the ratio of the amount of a specific IgG within the CSF to that in plasma is proportional with the ratio of total IgG found within the CSF to total IgG in plasma. To calculate the C value, the specific IgG ratio is multiplied by the reciprocal of the total IgG ratio; this product should normally equal 1. With intrathecal production of IgG resulting from a pathogen, the specific IgG ratio is expected to be greater than the reciprocal of the value for the total IgG; this results in a calculated C value of greater than one.

A California study compared antemortem *S. neurona* immunoblot results with postmortem findings in approximately 150 neurologically normal and abnormal horses.[18] The seroprevalence in this population was found to be 35%, and positive results on the CSF immunoblot occurred in 20% of the horses. Of 122 neurologically normal horses, 25 had positive CSF immunoblot results. Only 1 CSF immunoblot–positive horse was classified as EPM-positive by postmortem examination; 2 horses were classified as EPM-suspect. In the same study, 3 horses with negative CSF western blot results were classified as EPM-suspect, and 3 additional horses had slight CNS inflammation in which an unidentified protozoa was detected.[18] In Kentucky, Bernard found that the prevalence of positive CSF immunoblot results was 31.1% in a group of neurologically normal young horses and 29.7% in a neurologically abnormal population.[19]

EVALUATION OF RESPONSE TO TREATMENT

In many situations, evaluation of response to treatment plays the main role in the diagnosis of EPM. Although this is often a necessary approach in ambiguous disease processes, it can frequently lead to inaccurate conclusions. Many clinical signs attributed to EPM may nonspecifically improve with the administration of any number of treatments commonly prescribed for EPM or with rest alone. Common EPM treatments include broad-spectrum antimicrobial agents (a sulfa drug and/or oxytetracycline); rest during treatment; NSAIDs; steroids; dimethyl-sulfoxide; vitamin E; folic acid; and

many other compounds, agents, magnetic devices, herbs, or supplements as well as acupuncture. There are many diseases or processes that could respond to some combination of these treatments. Rest and the placebo effect may account for many instances of perceived improvement in ambiguous cases.

Horses in which a history of poor performance is the sole clinical abnormality are commonly diagnosed with EPM via immunoblot and the presumption that a neurologic deficit is manifest as poor performance, and treatment is often initiated. Improvement in performance over time may occur because of waxing and waning of chronic problems, with aging, or with intermittent administration of non–EPM-specific therapies (e.g., NSAIDs, rest). Such improvement is often interpreted as a positive response to EPM treatment and thus as confirmation of the diagnosis. Although incorrect diagnosis and treatment may be a major component of lack of treatment response or perceived relapse in these cases, a subsequent decline in performance is often interpreted as relapse. Alternative therapies are often sought out of frustration. Although EPM is unlikely to be the cause of poor performance in these cases, those involved often have difficulty overcoming the stigma of a positive immunoblot result.

Because of the difficulties encountered in the diagnostic process, it is usually advisable to evaluate all potential EPM patients as completely as possible. However, the process is often restricted to the evaluation of treatment response. When treatments fail or the client repeatedly reports relapses, the horse should be reevaluated for neurologic deficits, including their severity and any changes that have occurred. It is good practice to reconsider the diagnosis regardless of whether the animal displays subtle or severe neurologic deficits. A lack of response or the report of a relapse may also indicate that the clinical signs are caused by another neurologic or nonneurologic disorder.

SUMMARY

The antemortem diagnosis of EPM is tentative and fraught with numerous complicating issues regarding clinical examination and laboratory and ancillary diagnostic findings. It is therefore necessary to carefully evaluate and interpret all information obtained from the diagnostic process, keeping in mind the limitations and complicating factors associated with each procedure.

For the many horses that are not overtly neurologically abnormal, clients and their veterinarians should evaluate the horse as completely as possible. Diagnostics should be performed with consideration of all neurologic and nonneurologic conditions that could explain the horse's clinical signs and the owner's complaints. In ambiguous cases, special attention should be given to the evaluation of lameness and other neurologic and nonneurologic conditions. Response to treatment as a means of diagnosing EPM should be carefully interpreted; the possibility that an animal has responded to some non–EPM-specific therapy or to rest should be considered. Subtle changes in performance may be attributed to EPM when rigorous diagnostics and evaluations have ruled out other explanations. In these cases, response to therapy will need to be carefully interpreted to support a tentative diagnosis. If response to treatment does not occur or if relapses appear to be frequent, the diagnosis should be reevaluated.
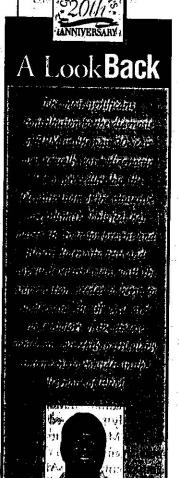
Until a definitive antemortem testing strategy is available, patients will continue to be misdiagnosed with EPM. Misdiagnosis and frustration will be minimized by a conscientious and informed approach to the diagnosis and treatment of EPM.

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