Neurologic Conditions Affecting the Equine Athlete

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KEYWORDS

- Equine herpes virus (EHV-1) myeloencephalopathy (EHM)
- Equine protozoal myeloencephalitis (EPM)
- Equine degenerative myeloencephalopathy (EDM)
- Cervical vertebral stenotic myelopathy (CVSM)

KEY POINTS

- Equine protozoal myeloencephalitis, cervical vertebral stenotic myelopathy, and equine degenerative myeloencephalopathy are 3 of the most common neurologic diseases in US horses, with the latter 2 conditions being most prevalent in young horses. Furthermore, horses competing at shows and performance events are at greater risk of exposure to highly contagious, neurologic equine herpes virus-1 outbreaks.
- Horses with mild or early clinical signs of neurologic gait deficits often present for performance-related concerns that can be difficult to discern from a lameness condition. Horses with unspecific gait changes should therefore undergo a complete neurologic examination.
- A diagnosis of neurologic disease should always start with a detailed clinical examination and not be based on diagnostic imaging or serologic testing of prevalent disease conditions alone.

INTRODUCTION

Neurologic disease can often mimic or be mistaken for an orthopedic condition when horses present for performance-related concerns. A careful history, clinical examination, appropriate diagnostic testing, and interpretation are thus essential for an accurate diagnosis. A thorough history includes the duration and extent of the problem, a change or progression of clinical abnormalities over time, response to analgesia, events of tripping or falling, and exacerbation of clinical signs during specific activities or movements.

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In general, lame horses are more commonly reluctant to work or to continue to work than ataxic horses. However, both conditions may lead to premature exhaustion or exercise intolerance due to inefficiency of locomotion, especially if ataxia is associated with weakness. Most orthopedic conditions are affected by the level and duration of exercise; whereas osteoarthritis may improve after the horse has warmed up, lameness associated with acute arthritis will likely become more severe during increasing exercise. These effects are not commonly noted in ataxic horses.[1] Similarly, most causes of neurologic disease do not induce pain, with the notable exception of some forms of cervical vertebral stenotic myelopathy (CVSM) or other vertebral arthropathies. Perineural and/or intrasynovial (local) anesthesia may thus help in the differentiation of lameness and ataxia. Systemic analgesia (a single intravenous (IV) dose of phenylbutazone) may lead to a more obvious response in lameness reduction for patients with acute single-limb lameness, than in the typical clinical patient with chronic multilimb lameness. The latter more consistently show a general increase in willingness to work with nonsteroidal anti-inflammatory drugs (NSAIDs), whereas no such change is usually expected in pain-free ataxic horses. In contrast, ataxia is usually exacerbated after sedation (eg, for obtaining radiographs of the cervical spine).[1]

It is important that the diagnosis of neurologic disease always starts with a detailed clinical examination and is not based on diagnostic imaging or serologic testing of prevalent disease conditions alone. There is generally little disagreement between clinicians when assessing the presence or absence of neurologic signs in moderately to severely affected horses. However, considerable interobserver variability exists in both the recognition and grading of neurologic abnormalities, especially when the clinical signs in the horse are subtle.[2,3] Athletes, such as hunters, jumpers, and dressage horses, with mild neurologic disease can often meet performance expectations to a certain point, or complete their existing job quite well (until their disease progresses or confounding conditions, such as lameness, develop). The true onset of their neurologic signs can thus be difficult to ascertain.

The current article focuses on the clinical recognition, diagnosis, and management of the 3 most commonly reported neurologic conditions in US horses (equine protozoal myeloencephalitis [EPM], CVSM, and equine degenerative myeloencephalopathy [EDM]); in addition to equine herpes virus-1 (EHV-1) myeloencephalopathy as a highly contagious infectious disease of increasing importance at performance venues and large equestrian farms.[4] Many additional neurologic disorders exist that may result in gait deficits or performance problems, but are beyond the scope of this review.

CERVICAL VERTEBRAL STENOTIC MYELOPATHY

CVSM is almost certainly one of the most common causes of ataxia in sport horses. Its etiology is complicated, and CVSM is widely considered to be a developmental abnormality modulated by genetic predispositions and environmental influences, including diet, rate of growth, workload, and trauma. The pathophysiology of the disease involves spinal cord compression due to malformation, malarticulation, instability, and soft tissue or bony changes of the cervical vertebrae, their articulations, and associated soft tissue structures. Various investigators have attempted to categorize CVSM based on the structural abnormalities. Rooney[5] described 3 types: type I, a fixed flexural deformity of the neck that usually occurs at C2-C3 and is present at birth; type II, symmetric overgrowth of the articular processes causing spinal cord compression during neck flexion, usually occurring in the mid-cervical region of foals and weanlings; and type III, asymmetrical overgrowth of 1 articular process that compresses the spinal cord directly by bony proliferation or indirectly by associated soft