Equine Protozoal Myeloencephalitis

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KEYWORDS

• *Sarcocystis* • *Neospora* • Opossum • Protozoa • EPM • Central nervous system

KEY POINTS

- Equine protozoal myeloencephalitis is an infectious neurologic disease of horses in North, Central, and South America. The disease is caused by the coccidian parasite *Sarcocystis neurona* and less frequently by the related pathogen *Neospora hughesi*.
- Horses are infected with *Sarcocystis neurona* by ingesting food or water that has been contaminated with feces from an infected opossum. The mode of transmission remains uncertain for *Neospora hughesi*.
- In many geographic areas of the Americas, infection is common, as evidenced by the proportion of horses exhibiting antibodies against the parasites. However, clinical disease is uncommon (<1% of seropositive horses).
- Anticoccidial drugs will halt infection, but early diagnosis and treatment are critical to minimize immune-mediated damage in the central nervous system.

A video of the horse with EPM, acute ataxia caused by equine protozoal myeloencephalitis accompanies this article.

INTRODUCTION

An unusual neurologic condition of horses termed *segmental myelitis* was first observed by Rooney in Kentucky in 1964.[1] Rooney renamed the syndrome *focal encephalitis-myelitis* because of brain involvement, and Prickett, Rooney and others reported on 44 cases at the annual meeting of American Association of Equine Practitioners in 1968[2] and on 52 cases in 1970.[1] In 1974, protozoa were first seen in association with characteristic lesions,[3] and the disease was given its current name, equine protozoal myeloencephalitis (EPM) by Mayhew and colleagues,[4] who reported on 45 cases at the American Association of Equine Practitioners meeting in 1976. Over the

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years, a better understanding of EPM etiology and epidemiology has been obtained. However, EPM pathogenesis remains uncertain.

ETIOLOGIC AGENTS

Sarcocystis neurona[5] and *Neospora hughesi*[6–10] are the 2 known causative agents of EPM, although most cases are caused by infection with *S neurona*. Both are protozoan parasites in the phylum, Apicomplexa, which is a broad and important group of obligate intracellular pathogens that cause significant disease in humans and animals.

All species of *Sarcocystis* have a 2-host life cycle that alternates between definitive and intermediate hosts. The opossum (*Didelphis virginiana*) is the definitive host for *S neurona* in North America.[11] As well, South American opossums can act as definitive hosts for *S neurona*.[12] The parasite undergoes sexual reproduction in the intestinal epithelium of the infected opossum, resulting in the production of sporozoite-containing sporocysts that are passed in the feces and are infectious for the intermediate hosts. Skunks,[13] raccoons,[14] armadillos,[15] and cats[16] have been identified as intermediate hosts for *S neurona*. In the natural intermediate hosts, *S neurona* forms latent sarcocysts in the muscle tissue, which is the source of infection for the opossum definitive hosts. Opossums are commonly infected with *S neurona*,[17] so there can be significant contamination of the environment in locations in which opossums are frequently observed.

Horses become infected with *S neurona* when they ingest food or water contaminated with feces from an infected opossum. Horses are considered incidental/dead-end hosts that do not contribute to the parasite's life cycle, because *S neurona* sarcocysts are not found routinely in these animals. However, *S neurona* sarcocysts were described in 1 case of a 4-month-old foal with clinical signs of EPM.[18] Although it remains unlikely that horses play a major role in the life cycle of *S neurona*, this finding suggests that the parasite has the capacity to establish long-term latent infection in these animals. It is important to note that *S neurona* cannot be transmitted horizontally between horses nor can it be transmitted to horses from the intermediate hosts.

The complete lifecycle of *N hughesi* is unknown, so the mode(s) of transmission of this parasite to horses remains uncertain. Canids are known to be a definitive host for the related species *Neospora caninum*,[19] but it has not been established that *N hughesi* use dogs as a definitive host. Vertical transmission of *N caninum* is efficient in cattle, and several studies now suggest that transplacental passage of *N hughesi* can occur in horses.[20,21]

EPIDEMIOLOGY

The first national epidemiologic survey of EPM used postmortem data gathered retrospectively from 10 diagnostic centers throughout the United States and Canada.[22] Most horses (61.8%) were 4 years of age or less and, 19.8% were 8 years of age or older. Although Thoroughbreds, Standardbreds, and Quarter Horses were most commonly affected, no breed, gender, or seasonal bias was established. In a smaller retrospective study, 82 horses with histologic lesions compatible with EPM were reviewed.[23] Disease risk was highest among male Standardbred horses compared with the gender and breed distributions of the attendant hospital population. The mean age of affected horses was 3.6 ± 2.8 years, similar to the findings of Fayer and colleagues.[22]

The prevalence of *S neurona*–specific serum antibodies in horses from the United States has varied widely, ranging from as low as 15% to a high of 89%, depending on geographic location.[24–28] Seroprevalences of 35.6% and 35.5% were reported in horses in Brazil and Argentina, respectively,[29,30] thus, showing that horses in South America are commonly exposed to the parasite.