Early Signs of Equine Protozoal Myeloencephalitis

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KEY WORDS: Equine myeloencephalitis, *Sarcocystis neurona*, horses, central nervous system

ABSTRACT

The early signs of *Sarcocystis. neurona* encephalitis after experimental infection in 6 horses are described. Blinded examiners determined scores for ataxia, dysmetria, paresis, and spacitic y as part of a gait assessment score. Other signs of infection noted by blinded examiners were decreased tongue tone, facial paresis, increased or decreased skin sensation, muscle wasting, weakness on tail pull, and lameness. Signs other than ataxia were apparent to blinded examiners by 30 days after challenge and remained until the end of the observation period. Other signs of acute infection seen by non-blinded observers included behavior change, drooling, dropped feed, and transient cranial nerve deficits. A Grade 2 ataxia seen on 2 blinded examinations 30 days apart was used as a definition of equine protozoal myeloencephalitis (EPM) and was seen in 3 horses, and lameness was seen in 5 horses. Acute early signs of infection were present before ataxia in all horses that progressed to EPM or became lame. An observation period of 90 days was not long enough to observe EPM in one horse.

INTRODUCTION

Protozoa gained attention as an important cause of neurologic lameness in the horse in 1974 with the recognition of *Toxoplasma gondii*-like organisms in the central nervous systems (CNS) of diseased horses.[1–3] In 1991, protozoa from the spinal cord of a horse were isolated by continuous culture and named *Sarcocystis neurona*.[4-5] Since the identification of *S. neurona* as the etiologic agent of equine protozoal myeloencephalitis (EPM), the life cycle between natural host and several intermediate hosts has been achieved in the laboratory.[6–7] The premortem diagnosis of EPM includes lameness or neurologic disease that is due to another cause and presence of antibodies to *S. neurona* in the cerebral spinal fluid. Although the presence of antibody in the spinal fluid of horses with signs of neurologic abnormality measured by immunoblot is considered an aid for diagnosis of EPM, it is recommended that one consider the prevalence of disease in the area at a given time.

Probably, many more horses have marginal clinical signs of EPM that are never diagnosed but remain part of the large uncharacterized group of "poor performers." The early signs of infection by *S. neurona* are unrecognized because experimental reproduction of the disease has failed.[8] Experimental