Equine Protozoal Myeloencephalitis (EPM)

A PowerPage Presented By

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EPM is a common neurologic disease in the horse that has no breed predisposition. Any age can be affected, with some reports documenting an increased incidence in horses less than 4 years of age while others stating an increased incidence in horses greater than 13 years of age. The causative organism is *Sarcocystis neurona*; the life cycle of this organism involves numerous intermediate hosts.

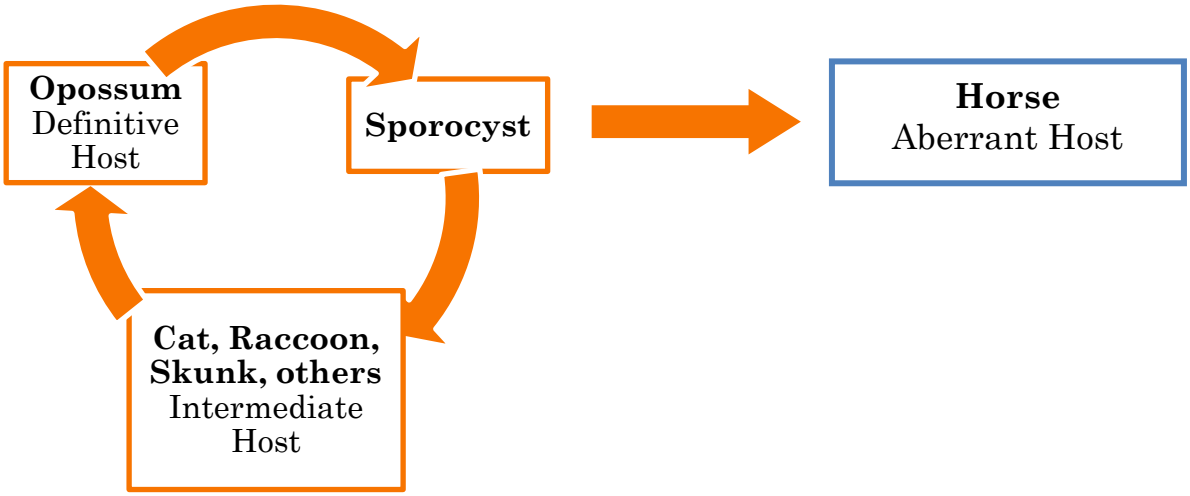
Key Points

- Caused by *Sarcocystis neurona*
- Opossum is definitive host
- Horse is an aberrant (dead-end) host

EPM Life Cycle

- Only a general knowledge of the intermediate and definitive host is needed:
  - The definitive (predator) host is the opossum which releases infective sporocysts into the food and water of intermediate (prey) hosts
  - Upon ingestion by the intermediate host, the sporocysts excyst and eventually enter skeletal muscle cells where they develop into sarcocysts
  - Life cycle is completed when the definitive host ingests the sarcocysts within the muscle of the intermediate host
- The **horse is an aberrant (and dead-end) host that ingests sporocysts**
- Large number of horses are exposed to the organism; however, only a minor population of all horses develop neurologic signs when the parasite is able to establish itself in the CNS of the horse

Abbreviated Life Cycle of *Sarcocystis neurona*



Clinical Signs

- EPM can cause a variety of clinical signs as the organism causes multifocal areas of necrosis in random areas of the spinal cord and brain
- The affected horse is usually bright and alert but demonstrates clinical signs including **asymmetric ataxia**, weakness and muscle atrophy
- Incoordination of one or more limbs may be perceived as lameness
- Other less common but possible clinical signs include head tilt, facial paralysis, and masseter muscle atrophy in horses with brainstem involvement
- In general terms, the **asymmetry associated with EPM is a distinguishing feature** when compared to cervical vertebral malformation

Diagnosis

- Definitive diagnosis can be, at times, difficult
- Suspect EPM based on clinical signs (asymmetric ataxia, weakness, muscle atrophy)
- Supportive diagnostics such as positive western blot analysis of CSF for *S. neurona* is available as well as immunofluorescent antibody testing. However, some of these tests are difficult to interpret, especially if the CSF sample is contaminated by peripheral blood
- Because of the peculiarities of some of these ancillary diagnostic tests for EPM, it is unlikely that there will be questions on the diagnostic tests of EPM

Treatment

Three primary treatments have been used:

1. Trimethoprim-sulfonamide & pyrimethamine: causes blockade of folate metabolism in protozoa
2. Ponazuril (Marquis®): anti-protozoal drug believed to target protozoal organelle (plastid)
3. Nitazoxinade (Navigator®): thought to inhibit electron transfer reactions essential for energy metabolism

Prognosis

Variable. Some horses can completely recover from EPM with appropriate therapy and have gone on to perform normally. Others may have little to no improvement or have residual neurologic deficits.