EPM: Equine Protozoal Myelitis

**Classic case:** Unilateral gluteal muscle atrophy but not lame
Weeks later: progressive hemiparesis, ataxia, Horner’s, unilateral masseter, temporalis atrophy

**Presentation:**
- History and signalment
  - **Two age groups** predominate
    - 1-5 years
    - Over 13 years
  - Risk factors
    - Heavy exercise, Warmer seasons
    - Breeding, Transportation
    - High stocking densities
    - Environmental change
    - Opossum feces in feed
- Clinical signs
  - **Unilateral gluteal muscle atrophy**
  - **Weeks later:** Broad spectrum CNS signs
    - Typically see multifocal, asymmetric cranial nerve involvement
    - Mild lameness, ataxia, head tilt
    - Progressive hemiparesis, Horner’s, unilateral masseter, temporalis atrophy,
    - Somnolence, seizures, recumbency
  - **Acute** or chronic. Progressive but may wax and wane
  - May be subclinical

**DDX:** Any equine neurologic disease that affects CNS
- EHV-1, cervical vertebral malformation, vertebral osteomyelitis, equine degenerative myeloencephalopathy, rabies, botulism, tetanus, Sorghum intoxication, lathyris, stringhalt, fibrotic myopathy, polyneuritis equi, peripheral nerve trauma, EEE, WEE, VEE, WNV, moldy corn poisoning, hepatonecephalopathy, head or vertebral trauma, verminous migration

**Test(s) of choice:** NO definitive antemortem test - rule out other diseases from differential list
- Radiography of skull, vertebral column, limbs - normal
- Bloodwork: CBC, serum chemistries - normal
- Serologic testing: Western blot: S neurona antibody presence in serum only indicates exposure, but if negative, EPM is highly unlikely
- Toxicity testing – negative
- Electromyography – helps to localize lesions but not diagnostic
- **CSF is usually normal**
  - Can show a nonsuppurative inflammatory response
  - Western blot: S neurona antibody presence in CSF is suggestive of EPM, but can be false positive because of blood/serum contamination
  - PCR for S neurona specific DNA is only positive in a small percentage of cases
Equine Protozoal Myelitis (EPM)

Rx of choice:
Antibiotics
- Folate-inhibitors
  - Keep horses on good quality, unprocessed green forage rich in folate
  - Monitor CBC
- Triazineones
- Nitrothiazoles
Anti-inflammatory drugs for up to 5 days in severe cases
- Phenylbutazone
- DMSO
- Dexamethasone

Prognosis:
- Fair to good (60%) for improvement
- Guarded to poor (10-20%) for cure

Prevention:
- Adjuvant-based vaccine made from killed *S. neurona* cultured merozoites
- Opossum control
  - Keep feed, fruit, and garbage well contained

Pearls:
- Mostly *Sarcocystis neurona*, however there are few reports of *Neospora* spp
- **Most common equine CNS inflammatory disease**
  - Many horses are infected but only few have the disease
  - Cannot be passed between horses
  - Horse infected from feed contaminated with opossum feces

Life cycle of *S. neurona* - key points
- **Definitive host – opossum**
  - Eats sarcocyst-infected tissue from an intermediate host
  - Sexual reproduction in host’s small intestine
  - Oocysts (containing two sporocysts) are passed in feces
  - Each sporocyst contains four sporozoites

- **Intermediate hosts**
  - Skunk, raccoon, armadillo, (otter, cat)
  - Ingests sporozoites
  - Sporozoites invade small intestine
  - Asexually produce merozoites which enter bloodstream
  - Form sarcocysts in skeletal muscle
**Equine Protozoal Myelitis (EPM)**

**Extended Version**

**Refs:**

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**Images courtesy, USDA**

A. **Cross section of spinal cord with focal areas of necrosis**

B and C. **Section of spinal cord of horse with EPM.**

The dots are merozoites.

*Images courtesy, USDA*
Sarcocystis neurona life cycle:
*Note* gluteal atrophy, masticatory muscle atrophy (yellow circle) Table courtesy, USDA

My Notes: