Equine Protozoal Myeloencephalitis….

Equine Protozoal Myeloencephalitis is a protozoal disease of horse that affects the central nervous system. This diseases was originally identified in the 1960's by Dr. Jim Rooney, although protozoa was first identified in the lesions in 1974. This disease has been considered to be sporadic and rare, but recently, researchers have begun to learn the extent of distribution of this disease.

Etiology …

The disease caused by ***Sarcocystis neurona.***

Epidemiology…

1-The disease infected horses and ponies and has not been reported in donkeys and mules

2- The disease usually occurs sporadically in endemic areas, although epidemics on individual farms are reported.

3- The case fatality rate is approximately 7- 14% of horses which might depend on season of year ,type of feeding and the opossums feces حيوان الابسومب (الفأر الكيسي)

4-The disease occurs in horses from 2 months to 19 years of age. Horses <1 year of age are at lower risk of developing disease than are horse 1-4 years of age.

5- S. neurona is believed to have the two host life cycle as in toxoplasmosis The definitive host is the opossum and intermediate hosts include raccoons,cats, skunks, sea otter كلب البحر, and armadillo حيوان المدرعwhich ingestion of sporocysts and develop sarcocysts in muscle which when fed to opossums induce shedding of sporocysts.

The definitive host is infected by ingestion of sarcocysts of S. neurona encysted in muscle of the intermediate host. The intermediate host is infected by ingestion of sporocysts derived from rupture oocysts passed in the feces of the definitive host.

6- There is no evidence of transplacental infection of foals.

Pathogenesis ….

1-Details of the pathogenesis of EPM are unknown. It is assumed that after infection, probably by ingestion, sporocysts and release sporozoites which penetrate the gastrointestinal tract and enter endothelial cells. Subsequently, schizonts develop which on maturation rupture and release merozoite.

2- Schizonts are present in cells of the central nervous system, including neurons,glial cells, and intrathecal macrophages. Schizonts multiply in the infected cells

3- Infection induces a non-suppurative inflammation, characterized by accumulations of lymphocytes, neutrophils,eosinophils, and gitter cells. Infection of neurons, and the associated inflammatory reaction, disrupt

normal nervous function and contribute to the clinical signs of weakness, muscle atrophy,

Clinical findings ….

1- The incubation period after experimental infection of young horses ranges between 28 and 42 days, but is not known for the spontaneous disease. The clinical findings of EPM in horses are protean, متقلب and in endemic areas EPM should be considered as a diagnosis in any horse with clinical signs referable to the nervous system.

2- Clinical signs of EPM range from changes in gait or behavior to recumbency, muscle atrophy,or seizures. The onset of signs may be gradual, or acute and rapidly progressive. Affected horses do not have increased temperature or heart rate, unless complications of the nervous disease occur.

3-Spinal ataxia, evident as weakness,hypometria or hypermetria, while some times it appear in form of swaying , toe dragging , and circumduction of feet, to spontaneous falling and recumbency.

4- Lesions in the sacral cord cause signs of cauda equina syndrome, including tail paresis and urinary and fecal incontinence

5- Lesions affecting spinal cord gray matter cause focal, asymmetric muscle atrophy,absent reflexes, or focal areas of sweating.

6- Cranial nerve disease is a common manifestation of EPM and Common syndromes include:

a- Vestibular disease evident as circling, nystagmus, head tilt, and falling toward the affected side.

b- Unilateral facial nerve paralysis evident as ear droop, lack of palpebral or corneal reflex and menace on the affected side, and displacement of the upper lip and nares away from the side of the lesion.

c- Dysphagia and persistent dorsal displacement of the soft palate

d- Tongue paralysis

e- Masseter atrophy and weakness

f- Hypalgesia (Lack of sensation) of the nostrils and skin of the face

PM.changes …

Its limited to the spinal cord and brain, with the exception of neurogenic muscle atrophy. Gross lesions of hemorrhage and malacia may be visible in the central nervous system tissue. The lesions are asymmetrical, but may be more frequently encountered in the cervical parts

Clinical pathology….

1-here are no characteristic changes in the hemogram or serum biochemical variables. Diagnosis has focused on the demonstration of antibodies to S. neurona in serum or CSF by western blot, indirect fluorescence testing, or IgM capture ELISA.

2-Histopathological diagnosis .

Treatment….
Specific treatment of EPM involves the administration of antiprotozoal drugs including
1-**Ponazuril**, or **diclazuril,**
Ponazuril, is usually administered at a dosage of 5-10 mg/kg body weight orally once daily for 28 days. administration of the drug results in resolution of clinical signs in approxinlately 60% of horses with EPM
2-Nitazoxanide is administered at 25 mg/kg bodyweight orally for the first 5 days of treatment and then at 50 mg/kg orally for days 6-28 of treatment
3- Administration of the combination of sulfadiazine (20 mg/kg,PO) and pyrimethamine (1-2 mg/kg, PO)every 24 hours given 1 hour before feeding is effective in approximately 60-70 % of cases. This treatment is continued for at least 90 days
4- Supportive treatment of affected horses includes anti-inflammatory drugs (flunixin 1 mg/kg IV, every 8-12 h;or dimethyl sulfoxide, 1 g/kg as a 10% solution in isotonic saline IV, every 24 h for 3 days)
5- Supplementation with vitamin E, an antioxidant, is often recommended to aid healing of nervous tissue
Control ….
1- There is currently a killed vaccine to immunize against *Sarcosystis neurona*, however, the efficacy is unknown at this time.
2-

Keep feed rooms and containers closed and sealed.

- Use feeders, which minimize spillage and are difficult for wild animals to access.
- Clean up any dropped grain immediately to discourage scavengers.
- Feed heat-treated cereal grains and extruded feeds since these processes seem to kill the infective sporocysts.
- Keep water tanks clean and filled with clean fresh water