Equine Encephalomyelitis… Eastern Equine Encephalomyelitis… Western Equine Encephalomyelitis and Venezuelan Equine Encephalomyelitis

Eastern, Western, and Venezuelan equine encephalomyelitis are arthropod borne, viral infections that can cause severe encephalitis in horses and humans.

Etiology

Eastern, Western and Venezuelan equine encephalomyelitis result from infection by the respectively named viruses in the genus *Alphavirus* (family Togaviridae).

Epidemiology…..

1-These encephalitis viruses cause disease in horses, humans, pigs, and birds

2- Humans, horses, cattle, pigs, dogs, and ratites are accidental hosts of the virus.

3-The disease transmitted mostly by mosquitoes *Culex tarsalis*

4- Recovered . horses are resistant to infection for at least 2 years

5- Mortality is higher, in unvaccinated horses than in vaccinated horses. Moreover mortality in young foals from non-immune mares, that are infected with the disease , is always high, often as high as 100 % .

6-Housed animals are more susceptible than grazing animals

7- Morbidity varies widely depending upon seasonal conditions and the prevalence of insect vectors; cases may occur sporadically or in the form of severe outbreaks affecting 20% or more of a group.

8- The case fatality rate differs with the strain of the virus; in infection with the WEE virus it is usually 20-30% and with the EEE it is usually between 40 and 80% and may be as high as 90% .

9-The susceptibility of humans to the causative virus gives the disease great public health importance. Humans can become infected with the EEE virus and the WEE virus.

10-Person-to-person transmission has not been reported

Pathogenesis….

1-Inapparent infection is the mildest form of the disease and may be characterized by only a transient fever

2-A transitory viremia occurs at the height of the fever.

3-Pnetration of the virus into the brain does not occur in all cases and the infection does not produce signs, other than fever, unless involvement of the central nervous system occurs.

4-The lesions produced in nervous tissue are typical of a viral infection and are localized particularly in the gray matter .of the cerebral cortex, thalamus and hypothalamus, with minor involvement of the medulla and spinal cord.

5-The distribution of lesions that is responsible for the characteristic signs of mental derangement, followed at a later stage by paralysis. The early apparent blindness and failure to eat or drink appear to be cortical in origin. True blindness and pharyngeal paralysis occur only in the late Stages.

Clinical findings….

1-The diseases associated with EEE and WEE viruses are clinically indistinguishable. The incubation period for EEE is 1-3 days and 2-9 days for WEE. Uncomplicated disease usually lasts about 1 week.

2-In the initial viremic stage there is fever, which may be accompanied by anorexia and depression, but the reaction is usually so mild that it goes unobserved. In the experimental disease, the temperature may reach 41 C persisting for only 24-48 hours, with signs of neurologic dysfunction appearing at the peak of the fever. Animals that have signs of neurologic disease for more than 24 hours are often not pyrexic.

3-Initial signs of neurologic disease include hypersensitivity to sound and touch, and in some cases transient periods of excitement and restlessness, with apparent blindness. Horses can have a period of anorexia and colic before onset of signs of neurologic disease.

4-Affected horses may walk blindly into objects or walk in circles

5-Involuntary muscle movements occur, especially tremor of shoulder and facial muscles and erection of the penis and A stage of severely depressed will follows.

6-Affected horses stand with the head hung low; they appear to be asleep and may have a half-chewed mouthful of feed hanging from the lips. At this stage the horse may eat and drink if food is placed in its mouth. The pupillary light reflex is still present. The animal can be aroused,but soon relapses into a state of somnolence.

7-A stage of paralysis follows. There is inability to hold up the head, and it is often rested on a solid support. The lower lip is pendulous and the tongue protrude from the mouth. Unnatural postures are adopted, the horse often standing with the weight balanced on the forelegs or with the legs crossed.

8-On walking, there is obvious incoordination, particularly in the hindlegs, and circling is common. Defecation and urination are suppressed and the horse is unable to swallow.

9-Complete paralysis is the terminal stage. The horse goes down, is unable to rise and usually dies within 2-4 days from the first signs of illness. A proportion of affected horses do not develop paralysis and survive, but have persistent neurological deficits.

Clinical sings in human …characterized by

1-incubation period is 5-10 days and depend on the type

2-The disease start with fever, chills, myalgia and arthralgia. This prodrome is often but not always followed within a few days by neurologic signs.
2- The symptoms of encephalitis may include headache, irritability, focal neurologic deficits, neck stiffness, confusion, somnolence or stupor, disorientation, tremors, seizures and paralysis. Some patients enter a coma. Abdominal pain, vomiting and diarrhea may also be seen.
3- Children sometimes develop generalized edema, facial edema or periorbital edema, together with paralysis.
4- A biphasic illness, with apparent recovery from the prodromal illness before the onset of encephalitis, may also be seen in young patients.
5- In infants, central nervous system disease can occur suddenly, without prodromal signs

Clinical pathology….

1- There are no characteristic hematological or biochemical abnormalities.
2- Diagnostic confirmation is achieved by one or more of several means:
a- Isolation of virus from an affected animal
3-Detection of viral antigen or nucleic acid in an animal with appropriate clinical signs
4- Seroconversion or an increase in serum titer of sick or recovered animal. serological tests included

a- Neutralization test (plaque reduction neutralization or PRN test)
b- Hemagglutination inhibition,
c-ELISA and complement fixation.

DD….

1- West Nile encephalitis
predominantly a myelitis with later development of signs of neurologic disease whereas EEE and WEE have predominant signs of encephalopathy.
2- Rabies
3- Borna disease
4- Japanese encephalitis
5- Hepatic encephalopathy
6- Botulism

Treatment….

There is no definitive or specific treatment. Supportive treatment may be given with the intention to prevent self-injury, and maintain hydration and nutritional status.

Control….

Control of viral encephalomyelitis of horses is based on
1-Accurate clinical and laboratory diagnosis of the disease in horses
2-Quarantine of infected horses to stop movement of virus donors

3-Control of insect
4- Vaccination of all horses….
Formalin-inactivated EEE and WEE virus vaccines are available and
Annual revaccination is currently recommended