Equine encephalomyelitis (Western)

Case Definition

(Notifiable)

1. Clinical Signs
   1.1 Clinical Signs: Western equine encephalomyelitis (WEE) is a viral disease that mainly affects equids and infrequently humans caused by the Western equine encephalomyelitis virus (WEEV), an Alphavirus of the family Togaviridae. It is primarily transmitted by mosquitoes and less commonly by ticks. Like Eastern equine encephalomyelitis (EEE), WEE is called sleeping sickness due to the characteristic somnolence exhibited by affected horses. The causal virus is maintained in nature in an alternating infection cycle between mosquitoes and birds. Humans and horses serve as dead-end hosts. Although horses and humans are most often clinically affected by the virus, birds may exhibit clinical signs, and infection of other livestock species, deer, dogs, and a variety of small mammals, reptiles, and amphibians has also been reported. WEE is characterized by central nervous system dysfunction. In contrast to EEE, which is often fatal in horses, WEE results in a milder disease with lower case-fatality rate but potentially persistent neurologic deficits.

   Horses infected with WEEV develop subclinical or milder disease than horses with EEE virus infection; however, the clinical signs are similar, if not indistinguishable. Initially horses develop fever, lethargy, and anorexia. A characteristic somnolence may be observed, thus the name sleeping sickness. Neurological signs usually develop approximately 1 week after infection and include altered mentation, impaired vision, circling, head pressing, wandering, and difficulty swallowing. In the more severe form, the disease can progress with hyperexcitability, ataxia, convulsions, and death. Most deaths occur 2-3 days after onset of neurologic signs. Most horses will recover from WEEV infection; however, they may experience residual neurologic deficits. The incubation period is 5-15 days.

2. Laboratory criteria:
   2.1 Agent isolation and identification: Reverse transcriptase polymerase chain reaction (RT-PCR) may be performed on unfixed brain tissue. Virus isolation is performed on brain or other tissue, with virus identification by complement fixation (CF), direct immunofluorescence staining, plaque reduction neutralization (PRNT), or RT-PCR.

   2.2 Serology: Tests include CF, IgM capture enzyme-linked immunosorbent assay (ELISA), or plaque reduction neutralization test (PRNT), or hemagglutination inhibition (HI). Vaccination for WEE can produce positive PRNT, HI, CF, and possibly IgM test results.

3. Case definition and Reporting Criteria
   3.1 Suspect case: A susceptible equid with clinical signs consistent with WEE and is located in or has recently visited an area with appropriate climate and active hematophagous insects.

   3.2 Presumptive positive case: A suspect case that:
3.2.1 Has antibody against WEE virus (and is not vaccinated)

3.2.1.1 Non-negative PRNT, CF, or HI test result in the absence of vaccination

3.3 **Confirmed positive case:** Compatible clinical signs and may meet presumptive level of certainty; **AND**

3.3.1 Isolation of WEEV (seldom isolated); **OR**

3.3.2 RT-PCR detection of specific viral antigen or genomic sequences; **OR**

3.3.3 Serological confirmation of WEEV infection with a fourfold or greater change in antibody titer between paired serum samples collected 10-14 days apart (nonvaccinated or known vaccine history); **OR**

3.3.4 Positive immunohistochemistry for WEEV antigen in tissue; **OR**

3.3.5 ELISA detection of IgM antibody (acute infection)