
CASE DEFINITION FOR WESTERN EQUINE ENCEPHALITIS

- 1. General disease/pathogen information:** Western equine encephalomyelitis (WEE) is a viral disease that mainly affects horses and infrequently humans; 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.
 - 1.1. Etiologic agent:** WEE is caused by the Western equine encephalomyelitis virus (WEEV), an *Alphavirus* of the family *Togaviridae*. It is closely related to the Eastern and Venezuelan equine encephalomyelitis viruses and Highlands J virus, all of which cause similar neurological dysfunction disorders in horses.
 - 1.2. Distribution:** WEEV is distributed throughout the Western Hemisphere. It is found in Western Canada, the Western United States, Mexico, and South America. The frequency of WEE in the United States has dramatically declined in recent years to almost no activity, and no significant U.S. outbreaks have occurred in more than 20 years.
 - 1.3. Clinical signs:** 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.
 - 1.4. Incubation period:** 5-15 days
 - 1.5. Differential diagnosis:** Diagnosis of WEE cannot be based on clinical signs alone because of its clinical similarity to other arboviral diseases (Eastern, Highlands J, and Venezuelan equine encephalomyelitis, West Nile encephalitis) and to other neurological disorders (rabies, botulism, hepatoencephalopathy, leukoencephalomalacia, equine herpesvirus myeloencephalopathy, bacterial meningitis, protozoal myeloencephalopathy, intoxications).
 - 1.6. Transmission and reservoir:** The WEE virus depends upon the cyclic transmission between birds and mosquitoes for its survival in nature and is heavily influenced by seasonal changes. Horses and other mammals serve as dead-end hosts. The primary



mosquito vector for WEEV is *Culex tarsalis*. This mosquito favors breeding in sunlit marshes and pools of irrigation waters on pastures. WEEV can also be transmitted by a tick vector, *Dermacentor andersoni*. Seasonal changes are crucial for WEEV transmission. Epizootics will typically occur following a pattern of heavy rainfall in the early spring that is followed with above normal summer temperatures. In regions with tropical or subtropical climates, WEEV infections can occur year-round. Transmission of WEEV is also influenced by the movement of infected insect vectors by wind currents and migration of infected bird hosts.

1.7. Epidemiology: Horses are the most susceptible domestic animal species to WEEV infection, with case-fatality rates of 20-50 percent. WEE is clinically similar to the disease caused by related EEE virus, yet WEE results in a subclinical or milder neurological disease and is less frequently encountered in the United States. Prevention of WEE outbreaks is possible through annual vaccination of horses.

2. Laboratory criteria: OIE Manual of Standards for Diagnostic Tests and Vaccines for Terrestrial Animals, 2008. Chapter 2.5.5.

2.1. Agent 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. Serological test: Tests include CF, IgM capture enzyme-linked immunosorbent assay (ELISA), or PRNT, or hemagglutination inhibition (HI).

3. Case definition:

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 Positive plaque reduction neutralization (PRNT); **OR**

3.2.1.2. ELISA detection of IgM antibody (acute infection)

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

4. Reporting criteria:

4.1. Suspect cases are reported according to individual State procedure, typically by notification of State arboviral coordinator or State animal health official.

4.2. Positive cases are to be reported to CDC's ArboNET by the State's chief animal health officer or appointed arboviral coordinators. Public distribution of this information is provided at:

- 4.2.1. USDA's National Animal Health Surveillance System (NAHSS) Western Equine Encephalitis virus information page
<http://www.aphis.usda.gov/vs/nahss/equine/ee/index.htm>
- 4.2.2. U.S. Department of the Interior's USGS Disease Maps information page
http://diseasemaps.usgs.gov/wee_us_veterinary.html
- 4.3. National Animal Health Reportable System (NAHRS) disease reporting of confirmed case occurrence. If a case meets the presumptive level of diagnostics only, animal health officials must use their discretion to decide if the case is valid. If considered valid, the case should be reported to NAHRS.

5. Control and surveillance procedures:

- 5.1. Annual vaccination of horses is recommended with increased vaccination protocols in areas of year-long active vector survival. Detailed vaccination guidelines are available from the American Association of Equine Practitioners (AAEP) at:
http://www.aaep.org/vaccination_guidelines.htm
- 5.2. Insect vector control – elimination of breeding sites, frequent manure removal, weed control, mosquito and tick protection of horses (insect repellants), and shelters (fans and screens)
- 5.3. Passive surveillance is conducted during routine activities by accredited veterinarians, State animal health officials, and others.

References

1. American Association of Equine Practitioners (AAEP) Vaccination Guidelines. Available online at http://www.aaep.org/vaccination_guidelines.htm.
2. Iowa State University: The Center for Food Security and Public Health: Eastern Equine Encephalomyelitis, Western Equine Encephalomyelitis, and Venezuelan Equine Encephalomyelitis Fact Sheet. 2008. Available online at http://www.cfsph.iastate.edu/Factsheets/pdfs/easter_wester_venezuelan_equine_encephalomyelitis.pdf
3. Merck Veterinary Manual. Equine Encephalomyelitis. Available online at: <http://www.merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/100900.htm&word=equine%2cencephalitis>
4. OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2008. Chapter 2.5.5. *Equine Encephalomyelitis (Eastern and Western)*. Available online at http://www.oie.int/eng/normes/mmanual/2008/pdf/2.05.05_EQUINE_ENCEPH.pdf
5. United States Department of the Interior, U.S. Geologic Survey (USGS) Disease Map 2008. <http://diseasemaps.usgs.gov/index.html> Accessed March 7, 2009.