



## AAEP VENEZUELAN EQUINE ENCEPHALITIS (VEE) GUIDELINES

### Summary

Venezuelan Equine Encephalitis (VEE) is caused by the arbovirus Venezuelan Equine Encephalitis Virus (arthropod-borne virus). VEEV, along with EEEV and WEEV, are alphaviruses in the family *Togaviridae*.

In the United States, epizootic VEE is a foreign animal disease that was last reported in Texas in 1971. The geographic distribution of the disease is largely restricted to northern South America, Central America, and southern Mexico. However, there is continuing risk of introduction of VEE into the US. Many mosquito species located throughout the US can serve as possible vectors for VEEV, including *Aedes* spp., *Anopheles* spp., and *Culex* spp.

*Note: VEE is a reportable disease (all alphaviruses [EEE, WEE] are reportable); consult your state and federal Animal Health Officials when disease is suspected.*

### Clinical Signs

The clinical signs of VEE are highly variable and similar to the signs associated with EEE. There are no pathognomonic clinical signs. Clinical signs consistent with VEE are:

- Moderate to high fever (>102.5°F; >39°C)
- Dull or obtunded mentation
- Lethargy
- Inappetence
- Diarrhea (VEE only)
- Signs of encephalitis may include:
  - Dysphagia
  - Head pressing
  - Tremors
  - Weakness
  - Ataxia
  - Circling
  - Blindness
  - Altered behavior such as agitation, aggression, somnolence, hyperexcitability, mania, self-mutilation
  - Seizures
  - Cranial neuropathy: nystagmus, facial nerve paralysis, and weakness of the tongue and pharynx
  - Coma
- Death from recumbency, seizures, or severe neurologic compromise. Some may be found dead without obvious cause.
- *Note:* Enzootic VEEV strains can cause subclinical disease in some horses



Differential diagnoses include any diseases capable of causing central nervous system dysfunction, including EPM, EHM, arboviral encephalitides, rabies, metabolic derangements, myopathies, and other intoxications. VEE is associated with a broader range of mortality (40–90%) compared to EEE (75–90%). Horses that survive VEE may have permanent neurologic deficits including abnormal mentation and/or residual ataxia.

### **Incubation Period**

12 hours to 5 days; most commonly about 3 days

### **Risk Factors**

- Horses residing in or traveling to VEE endemic regions
- Horses residing in regions adjacent to the US southern border

### **Transmission**

Indirect transmission to horses occurs through the bite of infected mosquitos (generally *Culex [Melanoconion] spp.*). Mosquitoes become infected after feeding on viremic rodents, which serve as natural reservoirs for the virus. Horses develop sufficient viremia to serve as amplification hosts (in contrast to EEE and WEE, where horses are dead-end hosts). Efficient amplification of the virus by horses is the hallmark of epizootic VEE, and horses therefore play an important role in the epidemiology of the disease in both horses and humans. Many other terrestrial species are susceptible to infection and can develop viremia (human, dogs, domestic rabbits, small ruminants).

### **Diagnostic Sampling, Testing and Handling**

Antemortem testing for VEE in the US utilizes the IgM capture ELISA performed on serum by the USDA-APHIS National Veterinary Services Laboratories (NVSL) in Ames, Iowa. Brain and spinal cord tissue may also be tested via PCR and VI. These assays are also performed at NVSL. If VEE is suspected, contact your state and federal Animal Health Officials for guidance on testing.

*Note: Rabies is a differential diagnosis for horses with acute neurologic disease, and all VEE suspect cases should be handled with appropriate personal protective equipment (PPE).*

### **Treatment**

There is no specific treatment for VEE. Supportive care, including NSAID anti-inflammatories and fluid therapy, may be helpful in some cases.

### **Postmortem Findings**

There are no consistent gross lesions, except that meninges are often congested (similar to EEE). Histopathologic features of VEE are similar to those seen with EEE, and include acute to subacute, multifocal to diffuse meningoencephalomyelitis, predominantly affecting the gray matter.



- Fix at least one-half of the brain for histopathology. Fresh brain should be submitted for concomitant PCR, virus isolation, immunochemistry, and rabies testing.
- Note: A rabies prevention protocol should be followed for ALL horses demonstrating signs of encephalitis that undergo postmortem examination.
- For some neurologic cases, submission of the entire carcass to the diagnostic laboratory for postmortem examination is recommended due to the time and labor required to collect samples from the equine CNS.

### **Shedding of Virus Following Resolution of Clinical Signs**

Horses are amplification hosts and can therefore be important sources of virus through bites of mosquitoes. The World Organization for Animal Health (WOAH, formerly OIE) defines the infective period as 14 days.

### **Environmental Persistence**

Alphaviruses do not persist outside of the host and are susceptible to drying, ultraviolet light, and detergents. However, VEE virus can survive in blood and exudates under the right conditions.

### **Specific Control Measures**

#### Vaccination:

Unlike EEE and WEE, VEE is not currently considered a core vaccine in the US. While vaccination has been proven highly effective as a control measure in VEE outbreaks in South America, vaccines are not currently widely used in North America because they compromise the international movement of horses for competition and breeding. Vaccination may be recommended in high-risk horses, such as those residing in US southern border states and/or horses traveling to VEE-endemic countries.

#### Vector Control:

- Reduce mosquito populations through removal of standing water in feed tubs, flowerpots, outdoor equipment, and other areas of potential water collection.
- Implement mosquito mitigation measures for stock tanks. These can include *Bacillus thuringiensis* (Bti/mosquito dunks), mosquitofish/goldfish, a few tablespoons of mineral oil (enough to coat the surface), or a barley straw bundle.
- Regularly apply insect repellants to the horse.
- Avoid turning out horses at dusk and dawn.
- Use fans to promote air circulation in stables.
- Use physical mosquito barriers (fly sheets, face masks, and fly bandages).

#### Regulatory Actions:

- VEE is a foreign animal disease in the US.
- Suspect and confirmed cases will be quarantined by state Animal Health Officials.
- Epidemiological investigation and response measures will be conducted by state and federal animal health authorities.



### **Biosecurity Considerations**

While VEE does not transmit via direct contact, fomites, or environmental contamination, infected horses can serve as sources of virus for infecting other horses and humans.

### **Release of Animals from Quarantine**

Release of quarantines on suspect or confirmed VEE cases will be directed by state/federal Animal Health Officials.

### **Zoonotic Potential**

Horses are amplification hosts for VEE and are an important reservoir of virus for humans and other horses. VEE has high zoonotic potential, and humans in physical proximity to VEE-infected horses are at risk of contracting VEE via arthropod transmission. Therefore, some additional public health measures may be directed by state health officials. Appropriate PPE is indicated when performing necropsy examinations on neurologic horses of unknown etiology.

### **Resource Information**

Long MT, Gibbs EPJ. Equine alphaviruses. In Sellon D, Long M. Equine Infectious Diseases, 2nd ed. Philadelphia, PA: Saunders Elsevier 2014.

Luethy D. Eastern, Western, and Venezuelan Equine Encephalitis and West Nile Viruses: Clinical and Public Health Considerations. Vet Clin North Am Equine Pract. 2023. 39(1);99-113.

**Author:** Daniela Luethy, DVM, MPH, DACVIM (LAIM)

**Supported and reviewed by:** AAEP Infectious Disease Committee