

E X T E N S I O N

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# Equine

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## WEST NILE VIRUS ENCEPHALOMYELITIS IN HORSES: 46 CASES (2001)

**Objective**—To determine signalment, clinical findings, results of diagnostic testing, outcome, and post-mortem findings in horses with West Nile virus (WNV) encephalomyelitis. Design—Retrospective study.

**Animals**—46 horses with WNV encephalomyelitis.

**Procedure**—Clinical data were extracted from medical records of affected horses.

**Results**—On the basis of clinical signs and results of serologic testing, WNV encephalomyelitis was diagnosed in 46 of 56 horses with CNS signs. Significantly more males than females were affected. Increased rectal temperature, weakness or ataxia, and muscle fasciculations were the most common clinical signs. Paresis was more common than ataxia, although both could be asymmetrical and multifocal. Supportive treatment included anti-inflammatory medications, fluids, antimicrobials, and slinging of recumbent horses. Results of the IgM capture ELISA and the plaque reduction neutralization test provided a diagnosis in 43 horses, and only results of the plaque reduction neutralization test were positive in three horses. Mortality rate was 30% and 71% of recumbent horses were euthanized. One horse that had received two vaccinations for WNV developed the disease and was euthanized. Follow-up communications with 19 owners revealed that most horses had residual deficits at 1 month after release from the hospital; abnormalities were resolved in all but two horses by 12 months after release.

**Conclusions and Clinical Relevance**—Our findings were similar to those of previous WNV outbreaks in horses but provided additional clinical details from monitored hospitalized horses. Diagnostic testing is essential to diagnosis, treatment is supportive, and recovery rate of discharged ambulatory horses is 100%.

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## WEST NILE VIRUS VACCINATION IN MARES AND FOALS

W. David Wilson, MS, BVMS, MRCVS, of the Department of Medicine and Epidemiology in the School of Veterinary Medicine at the University of California, Davis, hopes to determine the best WNV vaccination protocol for foals in a research study he has submitted to the Grayson-Jockey Club Research Foundation for review.

“We do not yet have any concrete information on which to base recommendations for vaccination of foals against WNV. However, it seems reasonable to use information gained from studies with other inactivated vaccines in foals as a basis for extrapolation. We’ve been recommending, based on extrapolation of information from studies with other vaccines, that if the mares are not vaccinated against WNV or they haven’t been exposed—which is now the situation for only horses in the far western states—that foals can be vaccinated starting at two to three months of age,” Wilson explained. “But I would have serious concerns about vaccinating foals at such a young age if their dams were vaccinated or had been previously exposed to WNV. Studies with influenza, EEE, WEE, tetanus, rabies, and EHV have shown that maternal antibody interference extends up to to six months and beyond.” Therefore, many foals vaccinated at less than six months of age fail to mount a protective immune response to the standard two-dose primary vaccination series.

Maternal antibody interference occurs when antibodies absorbed by the foal from colostrum are still present in the foal’s circulation at a high enough concentration to neutralize the vaccine.

“Essentially, you give the antigen in the vaccine, and it gets mopped up by the (maternal) antibodies and is no longer present to immunize the foal,” explained Wilson. When the maternal antibodies start to wane, the foal has to develop antibodies on his own to protect against that disease. His immune system won’t generate an antibody response until the maternal antibodies wane to the point that they can’t handle the threat.

To avoid this problem, Wilson has recommended that veterinarians delay vaccination of foals from mares which were vaccinated or exposed to WNV until the foal is about six months of age. Wilson recommends the following series:

- First vaccination at six months or older;
- Second vaccination three to four weeks later;
- Third vaccination six to eight weeks after the second dose of vaccine.

“What we’ve found with other vaccines is that many (foals) don’t respond optimally after two doses of vaccine even when vaccination is started after maternal antibodies have waned. A third dose gives a little more assurance that the ones that haven’t responded to two doses will respond to the third dose,” he explained.

Wilson said that the research group should have some preliminary information by next spring—hopefully in time to make more definitive recommendations for the 2003 foal crop. For the current research, Wilson said they have started blood sampling and vaccinating foals from mares which haven’t been vaccinated or exposed to WNV, as well as mares which have been vaccinated. Some of the foals are being vaccinated at three months of age, some at six months, and some at nine months. The full results will take more than a year to obtain. At that time, definitive recommendations based on scientific evidence will be formulated.

December 2002

## DIFFERENTIAL DIAGNOSIS FOR NEUROLOGIC DISEASE IN THE HORSE

The following is a list of differential diagnoses for neurologic disease in the horse: West Nile Virus, Rabies, Equine Protozoal Myeloencephalitis (EPM), Cervical Vertebral Myelopathy (CV, Wobbler), Equine Degenerative Myelopathy (EDM), Equine Herpes Virus1, other viral encephalitides (EEE, WEE, VEE), other CNS diseases, and other diseases that may mimic CNS diseases.

### West Nile Virus

#### **Clinical Signs:**

All cases examined in an outbreak of the disease in Italy exhibited varying degrees of ataxia and weakness in the pelvic limbs. Asymmetric weakness was detected in the rear limbs of some horses. Some cases also had involvement of one or both thoracic

limbs. In six cases, there was progression of clinical signs with ascending paresis leading to tetraplegia and recumbency within 9 days. Depressed mental state and tremors were noted in a few cases, however, there were no behavioral or head posture abnormalities or signs of cranial nerve involvement. In the US cases, death or euthanasia usually occurred within 3 to 5 days of onset of clinical signs, except in one case that lived for 14 days (Dr. Sue Trock, Extension Veterinarian, Cornell University; Personal Communication).

#### **Diagnosis:**

This disease is considered a Foreign Animal Disease by the USDA. Due to the zoonotic potential of West Nile Virus, horses who develop neurologic signs from August to October should be considered WNV suspects. To test for WNV, serum samples should be submitted to the Utah Veterinary Diagnostic Laboratory. Serum is tested for IgM levels, which indicates exposure to WNV. (An IgG response is present following West Nile vaccination.) Complete histories should be submitted with the serum samples. If the horse dies or is euthanized, samples from the spleen, liver, kidneys, and lungs should be submitted. If the horse is demonstrating neurologic signs prior to death, the brain and cervical spinal cord should also be submitted for WNV testing. If a post-mortem is performed in the field, precautions should be taken to minimize exposure to potential pathogens. Other diagnostics include PCR of CNS tissues and immunohistochemistry.

### Rabies

#### **Clinical Signs:**

Rabid horses may present symptoms in a number of ways, and can have any of a wide variety of clinical signs. Like several neurologic diseases in horses they can be difficult to diagnose. The spinal cord form and the dumb form are the most common. The spinal cord form is characterized by ataxia, fever and hyperesthesia followed by ascending paralysis, recumbency and loss of anal sphincter tone. The dumb form is characterized by drooling, pharyngeal paralysis, depression and anorexia. Death is usually by cardiorespiratory failure. After the onset of signs, survival ranges from 2 to 5 days with rare horses surviving for 2 weeks. (Sheryl L. Green; Vet Clinics North America, Equine Practice, April, 1997.)

#### **Diagnosis:**

The rabies virus-specific fluorescent antibody test is the most widely used method for confirming rabies infection. Due to the zoonotic potential of this virus, specialized laboratories are set up for diagnosing this disease. Cases where you suspect rabies should be sent to the Utah Veterinary Diagnostic Lab for diagnostic testing.

## **Equine Protozoal Myeloencephalitis (EPM)**

### **Clinical Signs:**

The disease is caused by a protozoan parasite called *Sarcocystis neurona*. It can affect any age, breed, or gender, but the highest risk is in young horses and older horses. The classic clinical signs are an asymmetric ataxia, weakness and spasticity with associated muscle atrophy. This parasite can affect any component of the central nervous system, however, the most commonly affected areas are the brainstem and spinal cord.

### **Diagnosis:**

Immunoblot analysis of serum and cerebrospinal fluid (CSF) provides antemortem information about exposure to *S. neurona*. The test utilizes cultured merozoites to detect antibodies directed against proteins that are unique to *S. neurona*. Antibodies produced to other organisms can be differentiated. Immunoblot testing of CSF has demonstrated 89% specificity and sensitivity among approximately 300 neurologic cases that received post mortem examination. Positive CSF indicates that parasites have penetrated the blood-brain barrier and stimulated a local immune response. If the integrity of the blood-brain barrier is compromised, circulating antibodies may leak across and produce a false positive test result. False negative results have been rare, but may occur. Some horses may simply fail to respond to the *S. neurona*-specific proteins identified. The possible causes of false negative responses are important to consider so that affected horses are not misdiagnosed. Horses that initially tested positive have become negative after several weeks of treatment and are apparently recovered. Chronically affected horses may test negative and still be infected or the horse may still exhibit neurologic signs. We speculate that this may be due to permanent CNS damage and that parasites are either no longer present, or antibody production is below test sensitivity. The use of PCR testing aids in parasite detection when antibody level is low. Acute cases that test negative should be re-tested in two to three weeks. However, the incubation period appears to be sufficiently long to allow production of detectable amounts of IgG before the onset of clinical signs in most cases.

## **Cervical Vertebral Myelopathy (CVM, Wobbler)**

### **Clinical Signs:**

This disease also primarily affects young horses 1 to 3 years of age, although caudal cervical osteoarthropathy is important in horses >5 yrs of age. The clinical signs are bilaterally symmetric with the rear limbs more severely affected than the forelimbs. Horses with CVM usually exhibit ataxia, weakness and spasticity in all four limbs.

### **Diagnosis:**

Standing lateral radiographs of the cervical vertebral column will give some indication whether spinal cord compression is occurring. This is enhanced by measuring the sagittal ratio by comparing the width of the vertebral canal and comparing it to the width of the vertebral body. If the ratio is less than 52%, the probability of compression is 85%. Confirmation of the compression can be accomplished by performing a myelogram.

## **Equine Degenerative Myelopathy (EDM)**

### **Clinical Signs:**

This disease also affects primarily young horses, often <1 year of age. Clinical signs include symmetric ataxia, weakness and spasticity in all four limbs, usually worse in the rear limbs. Hyporeflexia over the neck and trunk may be present, particularly in severe or longstanding cases.

### **Diagnosis:**

Low serum vitamin E levels may support a diagnosis. However, antemortem diagnosis is a diagnosis by exclusion. Therefore, appropriate ruleouts are essential. The only definitive diagnosis is at post mortem.

## **Equine Herpes Virus 1**

### **Clinical Signs:**

May occur as sporadic individual cases or in outbreaks where many are affected. It can affect any age, breed or gender. Cases may follow a previous outbreak of the abortion or respiratory forms. Has also occurred where there have been no additions to the farm and no previous outbreaks of any form. Most commonly the clinical signs are ataxia and weakness in the limbs, sometimes with urinary incontinence and fecal retention. Often signs start in the rear limbs and progress to the fore limbs. Many horses progress to recumbency.

### **Diagnosis:**

Historical findings and ruleouts of other neurologic diseases. CSF is often xanthochromic with few to no cells present. Antemortem diagnosis is difficult as the results are not consistent. However, acute and convalescent serum testing as well as antibody detection in CSF may be helpful in supporting the diagnosis.

## **Other Viral Encephalitides (EEE, WEE, VEE):**

### **Clinical Signs:**

About 5 days post-infection, horses begin to show fever and neurologic signs. These signs include extreme depression or drowsiness, paralysis, circling, impaired vision, irregular gait, dysphagia, and photosensitivity. Horses often hang their heads or grind their teeth. Mildly effected horses may recover over a few weeks but may be left with permanent brain damage.

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**Diagnosis:**

Serologic diagnosis can be made by virus neutralization ELISA, hemagglutination inhibition, or complement fixation. If a horse has died rapidly and paired serum samples are unavailable, virus isolation can be done from brain tissue. Although histopathology on brain tissue is useful, findings are not pathognomonic.

Virus isolated from fresh brain tissue (unfixed) is best when seeking a definitive diagnosis.

Other CNS diseases that should be considered are: trauma, polyneuritis equi, visceral larval migraines, and moldy corn poisoning.

Other diseases that may mimic CNS diseases: laminitis, rhabdomyolysis (tying up), hypocalcemia, and colic.

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