West Nile Virus in Big Sky Country

West Nile virus (WNV) is an arthropod-borne flavivirus (i.e., arbovirus) transmitted primarily through the bite of infected mosquitoes. WNV causes seasonal epidemics and epizootics during the late summer and early fall months. There is substantial variability in the intensity and geographic spread of WNV year-by-year, making future seasons difficult to predict. Since emerging in the United States in 1999, WNV has become the leading cause of neuroinvasive arboviral disease in humans and resulted in over 25,000 cases of WNV neuroinvasive disease in U.S. horses. WNV is maintained in an enzootic cycle involving birds and ornithophilic mosquito species. Humans, horses, and other mammals are incidental dead-end hosts. Almost 97% of all non-human mammalian WNV cases occur among horses. In both horses and humans, the majority of those infected will be asymptomatic. However, horses and humans developing WNV neuroinvasive disease have high case-fatality rates of 33% and approximately 10%, respectively.

In this issue of Montana One Health, we review key clinical and epidemiologic features of both equine and human WNV infections, and offer recommendations for preventing the transmission of WNV to both horses and humans.

Equine West Nile Virus

In Montana, WNV was first detected among horses in 2002 (Figure 1). By the end of 2003, 328 equine WNV cases had been reported in Montana. In 2012, equine WNV cases were reported in Carbon (1 case), Cascade (1), Powder River (2), Richland (1), and Yellowstone (1) counties (Figure 2).

The incubation time for WNV in horses is 3–15 days. The majority of WNV infections in horses are asymptomatic. Clinical disease associated with WNV infection, encephalomyelitis, is characterized by ataxia, abnormal gait, muscle fasciculation, depression, limb paralysis, inability to stand, and death. Ten to twenty percent of horses that survive have residual effects, including gate and behavioral abnormalities.

Plaque reduction neutralization testing (PRNT) can be used to diagnose acute infection through detection of virus-specific neutralizing antibodies in serum. Recent WNV exposure and vaccination both result in elevated antibody levels on PRNT; therefore, vaccination history is important when interpreting PRNT results. Testing of serum and cerebrospinal fluid (CSF) using enzyme-linked immunosorbent assay (ELISA) to detect increases in virus-specific IgM and IgG is also available. Post-mortem testing for WNV is accomplished by testing nervous system tissue with PCR, immunohistochemistry, and virus isolation.

Vaccination can significantly reduce the risk of WNV infection and is recommended for all North American horses in areas where WNV activity occurs. Since 2005, none of Montana’s 73 confirmed equine WNV cases have been up-to-date on their WNV vaccinations. Four USDA-licensed vaccines are currently available. Vaccination should occur each spring before mosquito activity increases. For more information about vaccination, see http://www.aaep.org/wnv.htm.

No specific treatment exists for WNV infection. Treatment of WNV infection relies on supportive care measures.
Human West Nile Virus
WNV first emerged in Montana in 2002 (Figure 1) and has since caused 9 deaths. The number of reported WNV cases per year ranged from 0 in 2010 to 222 in 2003. During 2012, WNV infections were reported among residents of Chouteau (2 cases), Custer (2), Prairie (1), and Richland (1) counties (Figure 2).

Clinical Syndromes
The incubation period ranges from 2–14 days. About 80% of WNV infections are asymptomatic. Almost 20% of infections result in West Nile fever (WNF), an acute systemic febrile illness often characterized by headache, myalgias, arthralgias, and rash. WNF can result in persistent fatigue, muscle pain, muscle weakness, and headache. Less than 1% of infections result in neuroinvasive disease. WNV neuroinvasive disease is characterized by acute onset of fever with stiff neck, limb weakness, altered mental status, seizures, CSF pleocytosis, and abnormal neuroimaging. The incidence of neuroinvasive disease increases with increasing age; the highest incidence occurs in those aged ≥70 years. The case-fatality rate for neuroinvasive disease varies by age and ranges from 0.8% among patients aged <40 years to 17% for patients aged >60 years. Neurological sequelae can persist in survivors months to years after acute illness.

Diagnosis
The diagnosis of WNV typically relies on a combination of compatible clinical signs and symptoms, and detection of virus-specific IgM by ELISA in serum or CSF. Confirmation of WNV disease occurs by demonstration of a 4-fold rise in IgM (or virus-specific neutralizing antibodies on PRNT) between acute and convalescent serum specimens collected at least 2 weeks apart. Diagnosis can also occur by isolation of virus, or detection of specific viral antigen in nucleic acid, tissue, blood, CSF, or other body fluid.

Treatment
No specific treatment for WNV infection currently exists. Treatment consists of supportive measures.

References

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