

What FPs need to know about West Nile virus disease

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The Centers for Disease Control and Prevention (CDC) reports that West Nile virus infection in humans or animals has occurred in most states, and mosquito bite is now known to be just 1 of several means of virus transmission. Since many infected persons are asymptomatic, the challenge of controlling the spread is made even more difficult. At the time this issue went to press, August 19, a total of 599 human cases and 11 deaths had been reported to the CDC by state and local health authorities. Last year, a total of 638 cases and 31 deaths had been reported by August 30, and for the entire year, 4156 lab-positive human cases and 284 deaths.

This article describes transmission, diagnosis, treatment, and prevention of West Nile virus infection.

■ HOW THE VIRUS SPREADS

The virus, an RNA virus from the Flaviviridae family, is maintained in a bird-mosquito-bird cycle that begins in the spring when mosquitoes emerge and ends in early fall when they become dormant. By mid to late summer, the virus popula-

“Advice from your doctor: How to prevent West Nile virus infection,” page 712, may be photocopied for distribution to patients.

tion has sufficiently amplified in both these hosts. At this point, other mosquitoes act as "bridge vectors" that bite both humans and birds, thus initiating West Nile virus infection in humans.

Avian mortality is documented in 162 North American species. It approaches 100% in laboratory-infected crows (making crows an important marker for the spread of West Nile virus in a specific community). House sparrows may develop high-level viremia for several days without dying, making them important amplifying hosts. Viremia in humans is low-grade and short-lived.

■ TRANSMISSION TO HUMANS: NEW ROUTES DISCOVERED

Most cases of West Nile virus infections in humans result from mosquito bites, but other mechanisms have been discovered: needle sticks in lab workers, possibly breast milk (1 case), and possible transplacental transmission to the fetus (1 case).

More importantly, there is evidence of transmission through contaminated blood. Since many

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ADVICE FROM YOUR DOCTOR

What you can do to prevent West Nile virus infection

Here are practical steps you can take to avoid being infected with West Nile virus, and to help stop the spread of disease.

To prevent mosquito bites

- **Use an insect repellent** that contains DEET at concentrations less than 50% for adults and less than 10% for children.
- **Wear long sleeves and socks** when outdoors.
- **Be particularly careful during evening and early morning hours**, when mosquitoes are most active.

Mosquito-proof your home

- Drain standing water regularly to minimize mosquito breeding grounds.
- Install or repair screens on windows and doors.

Report dead birds to local health authorities so they can better monitor the activity of West Nile virus transmission.

Asymptomatic infection. Infection is not clinically apparent in most people.

Mild infection. About 20% of persons infected exhibit West Nile fever, a mild illness that follows an incubation period of 3 to 14 days.

The syndrome lasts 3 to 6 days, and is characterized by a sudden febrile illness often with an array of nonspecific signs and symptoms such as malaise, anorexia, nausea and vomiting, eye pain, headache, myalgia, rash, and lymphadenopathy.

Severe infection. About 1 in 150 infected persons develops severe neurological disease (encephalitis or meningitis are most common).

Being older than 50 years is the most significant risk factor for neurologic disease. In hospitalized patients, the most common symptoms accompanying neurologic disease are fever, weakness, gastrointestinal symptoms, and mental status changes. A smaller number of patients have a maculopapular or morbilliform rash of the trunk or extremities.

Outcomes. Advanced age is the most important risk factor for death, with mortality reaching 20% among patients older than 70 years. Unfortunately, there appears to be substantial neurologic morbidity for those surviving hospitalization.

The US case-fatality rate in 2002 was 9% in patients with meningoencephalitis.

■ DIAGNOSIS

Diagnosis of symptomatic West Nile virus disease is based on clinical findings, a suggestive epidemiologic context, and specific laboratory test results.

Clinical and epidemiologic clues. Unexplained encephalitis or meningitis, particularly in a patient older than 50 years, during the summer or early fall, should trigger suspicion. Evidence of locally active disease in birds or humans, or recent travel to an area of known active disease, increases suspicion.

MAC-ELISA test and PanBio assay. The best test for diagnosis is detection of immunoglobulin M (IgM) antibody to West Nile virus in serum or cerebrospinal fluid within



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people infected with West Nile virus are asymptomatic, screening donors by clinical history is inadequate. In June 2003, blood-testing centers began screening the blood supply for West Nile virus using an experimental kit approved by the FDA.

■ CLINICAL COURSE

Like many other viral infections, West Nile virus infection manifests in several ways.

8 days of illness onset (MAC-ELISA test, available through local and state health departments).

The FDA recently approved a commercial product, the PanBio West Nile virus IgM assay, which correctly identified the antibody in 90%–99% of cases. IgM antibody does not cross the blood-brain barrier, so its detection in cerebrospinal fluid is presumptive evidence of central nervous system infection.

Other laboratory findings include:

- normal or increased white blood cell counts, sometimes with lymphopenia or anemia
- occasional hyponatremia, especially in patients with encephalitis
- cerebrospinal fluid pleocytosis (usually lymphocytic), increased protein and normal glucose
- normal computed tomography brain scans
- abnormal magnetic resonance images in one third of patients.

■ TREATMENT IS SUPPORTIVE

Treatment of severe disease is supportive. No evidence indicates efficacy of ribavirin, interferon, steroids, or other agents.

■ HOW TO USE

PUBLIC HEALTH RESOURCES

Prevention of West Nile virus disease will require both clinical and public health efforts. A good surveillance system is vital, providing clinicians and the community with knowledge about disease activity in birds and humans.

- Local or state health departments must coordinate, investigate, and track reports of dead birds by community members.
- Clinicians must notify the health department about suspected infections in humans.
- By publicizing the results of an active surveillance program, the health department assists clinicians in identifying cases more quickly and helps motivate the community to take appropriate preventive measures.

A short history of West Nile virus disease

In late August 1999, an infectious disease specialist reported 2 patients with encephalitis at 1 hospital in Queens to the New York City Department of Health. An ensuing investigation revealed 6 additional cases at nearby hospitals. The illnesses were characterized by fever, severe muscle weakness (7 of 8 persons), and flaccid paralysis (4 of 8). Cerebrospinal fluid test results suggested viral infection.

So began the saga of human West Nile virus in the United States.

The virus was first isolated from a patient in Uganda, and is now distributed throughout Africa, the Middle East, parts of Europe, southwestern Asia, and Australia. Disease outbreaks in other parts of the world were infrequent until 1996.

West Nile virus is thought to have come to North America from Israel, but it is not clear how. Since 1999, the virus has spread rapidly throughout the US. Interestingly, the number of human cases reported annually was low (20–60) until 2002, when more than 4000 cases were reported. Only 9 continental states had avoided human cases of West Nile virus, and only 4 had reported no human or animal cases.

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