Controlling Urban Epidemics of West Nile Virus Infection

Robert W. Haley, MD

This summer, Dallas, Texas, has been in the center of its worst-ever regional epidemic of West Nile virus (WNV) infection, with one-quarter of the nation’s cases identified in Dallas County.1 With a record high number of trapped mosquitoes testing positive for WNV, from June 20 to August 21 WNV infections have been confirmed in 270 county residents; 160 have been hospitalized, 51 have required intensive care unit admission, and 11 have died.2 Given this epidemic in Dallas, which has spilled over into Fort Worth and the 14 surrounding counties, and similar WNV activity in Louisiana, Alabama, Oklahoma, and the Midwest,3 physicians and the public should be aware of key information about WNV infection2 to enable informed decision making in countering urban WNV epidemics.

West Nile Virus

West Nile virus is a single-stranded RNA virus, making it prone to mutation and adaptation. The virus is transmitted primarily by the bite of the Culex pipiens complex of mosquitoes, a medium-sized brown mosquito known as the common house mosquito. Culex pipiens subsp pipiens is found in the northern half of the United States and C pipiens subsp quinquefasciatus in the southern half. Culex lay eggs in man-made sources of stagnant water in urban environments, and upon hatching, the adults tend to remain nearby.4

West Nile virus survives in the environment through a transmission cycle in which mosquitoes develop infection from feeding on infected birds. The virus spreads to the mosquito’s salivary glands and is transmitted when the mosquito injects its infected saliva into other, uninfected birds. Infected birds develop WNV illness involving the circulation of large amounts of virus in the bloodstream for several days that readily infect the next biting mosquitoes. Infected mosquitoes occasionally bite mammals, including humans, and infect them with the virus. Most birds and mammals can be infected with mosquito-borne WNV, but only blue jays, crows, horses, and humans tend to die of the infection. The American robin contributes most to maintaining the transmission cycle because it has several broods per season and hatchlings are more susceptible to WNV infection than adult birds.5

Human infections with WNV typically begin in midsummer as the virus level in birds and mosquitoes reaches a critical threshold, then decline in September as nighttime temperatures drop, slowing mosquito activity. Significant WNV epidemics are usually presaged by the appearance of dead crows or blue jays.

Clinical Features

Infection with WNV followed by lifelong immunity occurs equally at all ages depending on mosquito exposure, but the risk of more severe disease increases with age and underlying medical conditions. The incubation period from the infecting mosquito bite to first symptoms is generally 2 to 14 days.

Infection with WNV causes a spectrum of disease. Approximately 80% of those infected remain asymptomatic (including virtually all previously well children and young adults), and 20% have only fever and headache (West Nile fever [WNF]).2 Approximately 1 in 150 infected individuals develop inflammation of the brain and nervous system, manifested by a wide variety of neurologic symptoms, most commonly disorientation, cognitive impairment, stiff neck, muscle weakness, Parkinson-like movement disorders, and, possibly, coma, collectively referred to as West Nile neuroinvasive disease (WNND), which typically affects people older than 50 years. Rarely, patients can develop a polio-like condition with flaccid paralysis from damage to anterior horn cell neurons. Among patients with WNND, the case-fatality rate is 4% to 18% (8% this summer in Dallas County5), with deaths generally confined to older patients with underlying medical conditions. Although many patients with WNND recover fully, a considerable number are left with long-term or permanent impairments, including depression, fatigue, cognitive deficits, movement disorders, or paralysis.

The diagnosis of WNF should be suspected in anyone with unexplained fever from late June through September, the season when other causes of fever are least common. Fever with disorientation, stiff neck, or neurologic deficits suggests WNND. The diagnosis of WNV infection is confirmed by testing blood and spinal fluid for IgG and IgM antibodies and for WNV antigen by polymerase chain reaction, available through local health departments.

Most patients with WNF can be treated symptomatically and do not require hospitalization. However, those with WNND should be hospitalized for lumbar puncture, ruling out other possible etiologies, observation for progression of neurologic deficits, and general supportive care. Progressive respiratory insufficiency from muscle weakness requires respiratory support. No medication prevents or improves the neurologic deficits or alters the likelihood of long-term disability or death. Information about WNV for patients can be found at http://www.jama.com.5

Author Affiliation: Division of Epidemiology, Department of Internal Medicine, University of Texas Southwestern Medical Center, Dallas.

Corresponding Author: Robert W. Haley, MD, Division of Epidemiology, Department of Internal Medicine, University of Texas Southwestern Medical Center, 5323 Harry Hines Blvd, Dallas, TX 75390 (Robert.Haley@UTSouthwestern.edu).

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Prevention of WNV

There is no WNV vaccine to protect humans; therefore, prevention through public health action, based on knowledge of how infection is acquired, is crucial. When WNV infections appear, prevention of further cases relies on 2 approaches. The first is for individuals, particularly those older than 50 years and those with chronic medical conditions, to use personal protective practices including eliminating stagnant water sources around the home; ensuring well-fitting screens on doors and windows; staying inside between dusk and dawn; and wearing long pants, long sleeves, and DEET insect repellent outdoors. Transmission between humans or from animals does not occur. Transmission has been described from transfusions, tissue transplants, laboratory mishaps, pregnancy, and breastfeeding but appears rare.3

The second approach involves disease prevention by local health departments supported by their state health departments and the Centers for Disease Control and Prevention.6 Local health departments must conduct active surveillance to detect and monitor infection rates in the community. They should maintain specialized knowledge of WNV epidemiology and disease trends to develop control measures. Health departments should inform policy makers and educate the public. In urban areas, surveillance includes setting mosquito traps at strategic locations and periodically testing trapped mosquitoes for WNV infection. Increasing rates of mosquito infection predict increasing rates of human infection.

When surveillance detects an unusual increase in rates of WNV-infected trapped mosquitoes and human WNV infections, local health departments should implement a graduated series of control measures to abort an impending epidemic. These include warning and educating the public about the risk and the need to exercise the personal preventive measures. Where surveillance identifies hot spots of highly infected mosquito populations, pesticide application on the ground by trucks and by workers on foot is warranted. When infection transmission is too widespread for ground spraying, aerial spraying of the entire epidemic area is necessary. These measures require action by well-informed local health officials, support by the medical community, receptivity by political decision makers (educated in advance), proactive leadership, and public education.

Aerial spraying has been used extensively throughout the world for decades to prevent or control epidemics of WNV and other mosquito-borne diseases.7 Ground and aerial spraying use the same pesticides. Aerial spraying uses much lower doses per acre compared with ground application, covers large urban areas more uniformly and more quickly, and reaches the tops of trees where the Culex mosquitoes mostly dwell.4 In several WNV epidemics, aerial spraying was effective in abruptly stopping the occurrence of new cases. Most important, aerial spraying is safe for humans and non-targeted wildlife.7

Current urban spraying uses the class of pesticides called pyrethroids, which are synthetic forms of pyrethrins that kill only adult mosquitoes.8 The pesticide is delivered by ultra low-volume (ULV) aerial spraying, in which a light plane flying at 300 ft delivers a mist of extremely fine droplets at a rate of around 1 oz per acre—far less than by ground spraying. Spraying at night minimizes contact with most insects and wildlife that are not active at night. Timely publicity of spraying schedules allows residents to stay inside, keep windows closed, and avoid being exposed to the pesticide. The extremely low ambient concentrations delivered by ULV aerial spraying result in human exposure levels one-tenth that from ground spraying and far below the level of concern for adverse effects to children or adults set by the US Environmental Protection Agency.9 The benefits of interrupting the WNV transmission cycle and preventing potential neurologic impairment and deaths that can occur with a WNV epidemic outweigh the theoretical risks of ULV aerial spraying.

Conclusion

The ability of WNV to mutate and adapt to the environment and the continuing occurrence of large urban epidemics, as seen in Dallas this year, indicate that WNV is likely to remain a serious threat into the foreseeable future. The explosive pattern of WNV epidemics that could affect any US city coupled with the morbidity and mortality attributable to this disease require preparedness and early decisive action. Given the effectiveness and safety of available control measures, this is a good time for all local governments to reevaluate and establish policy for response before it is their turn to be visited by WNV.

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REFERENCES


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