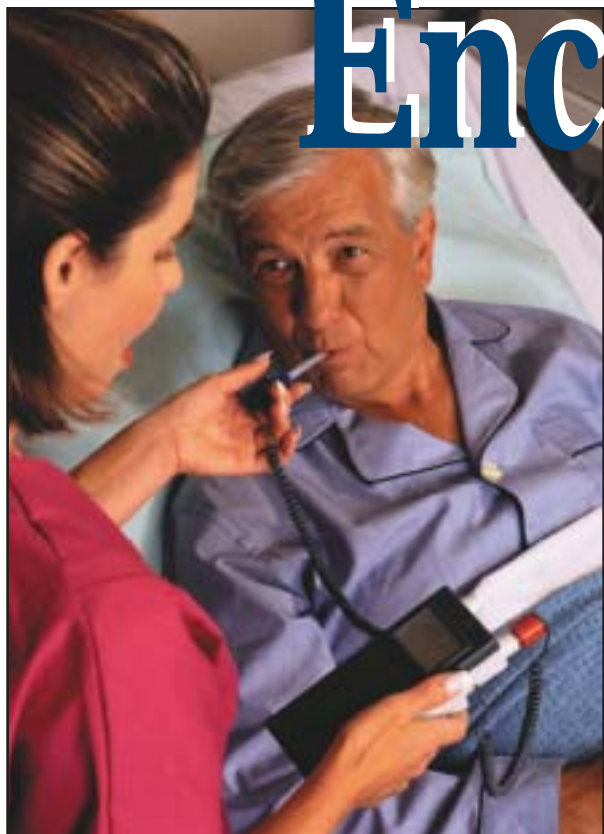

West Nile Encephalitis



“When you hear hoofbeats, don’t think of horses, but zebras.”

By Gary Victor, MB, FRCPC

In August 1999, an epidemic of West Nile virus (WNV) encephalitis and aseptic meningitis occurred in New York City (NYC).¹ There were 62 cases of laboratory-confirmed WNV in this outbreak: 59 people were hospitalized with severe neurologic illness and seven died.²

In 2001, a total of 57 human cases were reported in the USA, including four deaths. Also in 2001, WNV was confirmed in Ontario, Canada.

There were 128 positive cases found in dead birds in southwest and central east Ontario and seven WNV-positive mosquito pools, but no human cases were documented.³

WNV is a flavivirus, (family Flaviviridae)⁴ that was first isolated from the blood of a febrile woman in the West Nile district of Uganda in 1937.⁵

WNV has been known to occur in Africa, the Middle East, and in parts of Europe, Russia, India and Indonesia — resulting in sporadic epidemics. In areas of endemicity, illness is often asymptomatic. The introduction of this virus into areas without immunity can cause epidemics, which is what happened in NYC.¹

Transmission

Mosquitoes, largely bird-feeding species, are the principal vectors of WNV.⁴ The predominant genus *Culex* is the main vector and the virus has been isolated from 43

West Nile Encephalitis

mosquito species. Wild birds are the primary hosts of this virus, but other animals have been infected, including domestic fowl, large domestic animals and, more importantly, humans. High, long-term viremia has been observed, which can allow infection to occur *via* the vector mosquitoes. Although some transmission may occur by ticks, most transmissions are between mosquitoes and birds.^{2,5}

Epidemiology

In the Nile region of Egypt, where WNV is endemic, the seroprevalence among children ranges from 6% to 40% in young adults.⁶

In the NYC epidemic, there were 62 laboratory-positive cases, including 59 cases where patients were hospitalized with encephalitis and/or meningitis. Most of these cases involved residents of NYC, and its surrounding counties. One Canadian developed symptoms five days after visiting Queens, and subsequently died.

There were no common associations among the initial cluster of cases (*i.e.*, travel, social events, restaurants, medications, commercial foods or other contacts). There were minimal social contacts outside of the family and no other household members of the family were ill. All of these individuals spent time outdoors — especially in the evenings.

Clinical Findings

Mild illness includes three to five days of fever, headache, sore throat, backache, myalgia, arthralgia, fatigue, conjunctivitis and anorexia. A rash (maculopapular or roseolar) occurs in approximately half of the cases, spreading from the chest/abdominal areas to the extremities and head. Lymphadenopathy is not uncommon.^{2,4,7}

Dr. Victor is associate professor, University of Ottawa, Internal Medicine Program director, University of Ottawa/Ottawa Hospital, Ottawa, Ontario.

COPD
is *seldom* diagnosed
before the sixth decade.



But it
could be.

West Nile Encephalitis



More severe infections are marked by headache, high fever, neck stiffness, stupor, disorientation, coma, tremors, occasional convulsions and paralysis.

The incubation period in humans ranges from five to 15 days. Laboratory findings involve a slightly increased sedimentation rate and a mild leukocytosis; cerebrospinal fluid reveals elevated protein levels with moderate lymphocytic pleocytosis.

Magnetic resonance imaging (MRI) appears a better modality in detecting inflammation of the central nervous system (CNS) as compared to computed tomography (CT).⁸ Among the patients who underwent MRI evaluations, approximately a third showed findings consistent with encephalitis. Most fatal cases have been recorded in patients over the age of 50.

COPD *The evidence*



at 40-50

at 50-55

at 55-60



Boehringer
Ingelheim

Committed to
respiratory care



West Nile Encephalitis

Less than 15% of cases of acute encephalitis, meningitis or encephalitis can be accompanied by hepatitis, anterior myelitis, pancreatitis and myocarditis.^{4,8}

To date, there have not been any controlled studies evaluating steroid therapy, osmotic agents or antiseizure therapy in patients with WNV encephalitis/meningitis.

Diagnostic methods

The most commonly used method to diagnose this illness is the IgM capture, enzyme-linked immuno-sorbent assay (ELISA), which measures IgM antibody.³ A single acute phase serum sample from an individual with encephalitis or meningitis having a high level of WNV — specific IgM antibody — is strongly suggestive of infection.

Definitive serologic diagnosis requires both acute and convalescent samples be compared, documenting a fourfold rise in antibody titre. Although viral isolation and detection can occur, a biosafety Level 3 facility is required.

Prevention and control

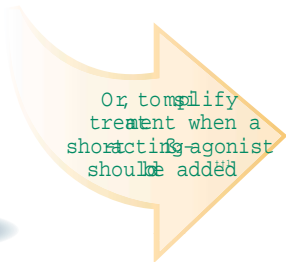
Avoidance of potentially infected mosquitoes is the most effective way to reduce the risk of infection due to WNV.

can be there *before 50.*

Diagnose ~~early~~ with ~~anticholinergic~~ ~~inhalant~~ ~~ipratropium~~



at 60-70



Atrivent inhalant is indicated for the maintenance therapy of reversible airways obstruction, such as chronic bronchitis and asthma. Combivent inhalant is indicated for the treatment of bronchospasm associated with chronic obstructive pulmonary disease (COPD). The most common side effects of Atrivent were dry mouth (9.4%), headache (7.9%), bad taste (3.8%) and palpitation (2.1%) (N=605). The most common side effects of Combivent were headache (1%), bronchitis (1%) and cough (1.4%) (N=358).

^{††} Ensure patients well controlled on separate and titrates are equalent
 1. Guidelines for the Treatment of Chronic Obstructive Pulmonary Disease (COPD) 1st Edition 1998, Canadian Respiratory Review Panel
 2. Chapman KR. Am J Med 1996; 100 (suppl 1A): 1A-55A-9S
 © Registered trademark of Boehringer Ingelheim Canada Ltd. PAAB*

West Nile Encephalitis

As discussed by Marfin,² personal protection behaviours should include:

- Avoiding activity in areas when potentially infected mosquitoes are present;
- Maintaining door and window screens;
- Wearing long-sleeved shirts and long pants when outdoors;
- Using insect repellents containing DEET or permethen on clothes; and
- Applying DEET-containing repellants to exposed skin.

DEET should not be used on children less than two years of age or on the hands of older children who may rub their eyes or mouth.



Summary

WNV can cause sporadic human disease outbreaks in areas where this disease has never occurred before. It emerged in North America in 1999, has persisted in the Eastern United States and can spread into new geographic areas.

Physicians should consider WNV infection in the evaluation of patients with febrile illnesses in the summer, especially those with aseptic meningitis, encephalitis and motor abnormalities. Health authorities must be vigilant in the surveillance of birds and mosquitoes. Other public health partners (*e.g.*, veterinarians, ornithologists) also play an important role in monitoring disease in animals which may be harbingers of human disease outbreaks. **Dx**

References

1. Asnis DS, Conetta R, Teixeira AA, et al: The West Nile virus outbreak of 1999 in New York. The Flushing Hospital Experience. *Clinical Infectious Dis*. 2000;30:413-8.
2. Marfin AA, Gubler DJ: West Nile encephalitis: An emerging disease in the United States. *Clinical Infectious Dis* 2001;31:1713-9.
3. D'Cuba CO: Human Surveillance of West Nile virus encephalitis through autumn 2002 (Letter). Ministry of Health and Long-Term Care. April 2002.
4. Hubalek Z, Halouzka J: West Nile fever, a re-emerging mosquito-borne viral disease in Europe. *Emerging Infectious Diseases* 1999;5:643-650.
5. Smithburn KC, Hughes TP, Burke AW et al: A neurotropic virus isolated from the blood of a native of Uganda. *Am J Trop Med* 1940;20:471-92.
6. Rappole JH, Derrickson SR, Hubalek K: Migratory birds and spread of West Nile virus in the western hemisphere. *Emerging Infectious Diseases* 2000;6:319-28.
7. Corwin A, Habib M, et al: Community-based prevalence profile of arboviral, rickettsial and Hantoon like viral antibody in the Nile River Delta of Egypt. *Am J Trop Med* 1993; 48:776-83.
8. Peiris JS, Amerasinghe FP: West Nile Fever. In: Beron GW, Steele JH editors. *Handbook of zoonoses section B: viral* 2nd edition Boca Raton FL CR C Press; 1994: p139-48.
9. Nash D, Mostashari F, Fine A, et al: Outbreak of West Nile Virus infection, New York City area. *N Engl J Med* 2001; 344:1807-14.