

Supplement

Management of Patients with West Nile Virus:

Guidelines for Health Care Providers



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**MANAGEMENT OF PATIENTS WITH
WEST NILE VIRUS:
GUIDELINES FOR HEALTH CARE PROVIDERS**

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Introduction

West Nile virus (WNV) was first identified in North America in 1999 after an outbreak of viral encephalitis in New York City had resulted in 62 confirmed human cases and seven deaths⁽¹⁾. In 2000, several Canadian provinces implemented enhanced passive and/or active surveillance for human cases of meningitis or encephalitis, but no cases were detected. In February 2000, Health Canada organized a National Steering Committee to develop a coordinated approach to surveillance, education, prevention, and response in anticipation of the arrival of WNV.

In 2001, the first WNV infected bird was detected in Ontario, and in 2002 the first confirmed human cases of illness due to WNV occurred in Quebec and Ontario. A total of 1,494 cases of illness due to WNV and 14 deaths were recorded in 2003, and 26 cases (two deaths) in 2004.

WNV is now established in the Canadian ecology. WNV activity has been recorded in dead birds only or in combination with mosquitoes and/or horses in Nova Scotia, New Brunswick, Quebec, Ontario, Manitoba, Saskatchewan, and Alberta. Human cases, unrelated to travel, have occurred in Quebec, Ontario, Manitoba, Saskatchewan, and Alberta. The aim of this document is to provide evidence-based guidelines (within the limits of present knowledge) for Canadian health care professionals who manage cases of WNV infection.

Transmission

WNV is a mosquito-borne flavivirus. The virus belongs taxonomically to the Japanese encephalitis serocomplex that includes the closely related St. Louis encephalitis virus. Transmission is primarily through infected mosquito bite. Less commonly,

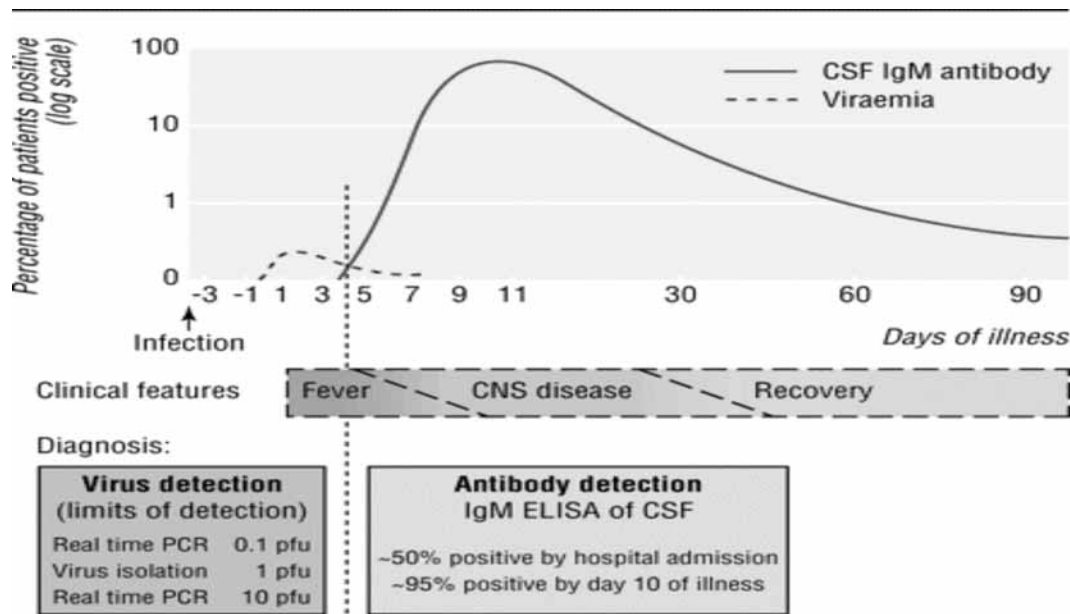
transmission has occurred through infected blood, tissues, and organs⁽²⁻⁴⁾. A small number of infections have been transmitted through needle stick or sharps injuries⁽⁵⁾. There is evidence that WNV may be transmitted in breast milk⁽⁶⁾. Transplacental transmission to the fetus has been recorded in a pregnant woman who had WNV infection in the second trimester⁽⁷⁾.

In Canada, enzootic transmission occurs during the period of April or May through to the first hard frosts, normally in October. Most human cases are recorded during late July, August, and September. Regional variations could occur as a result of a variety of factors, including climate, daylight hours, and vector species. WNV has spread to southern U.S. states, Central and South America, and the Caribbean, creating the potential for year-round transmission due to the temperate and subtropical environments⁽⁸⁾. Therefore, WNV infection should be considered in any person with unexplained encephalitis or meningitis who has travelled to any of these areas within the 2 to 15 days' incubation period of the disease. Immunocompromised individuals may have prolonged incubation periods⁽⁹⁾.

Incubation period and period of viremia

In cases of clinical illness, the incubation period ranges from 2 to 15 days, as shown in Figure 1. However, prolonged periods of up to 21 days have been observed in patients following organ transplantation⁽⁴⁾. The period of viremia begins several days (up to 6-7 days) before the onset of clinical illness and ends shortly after symptom onset⁽⁹⁾.

Figure 1. Clinical course of West Nile virus encephalitis: viremia, development of antibody, implications for diagnosis*



*Solomon T, Ooi M, Beasley D et al. BMJ 2003;326:865-9 (reproduced with permission from the *British Medical Association Journal*).

Clinical diagnosis

Diagnosis of WNV infection is currently based on clinical suspicion, laboratory confirmation, and one of the following:

- **History of exposure** when and where WNV transmission is present, or could be present, or history of travel to an area with confirmed WNV activity in birds, horses, other mammals, sentinel chickens, mosquitoes, or humans. Information on recent WNV activity in Canada is readily available at the following website: <www.westnilevirus.gc.ca>.
- **History of exposure to an alternative mode of transmission**, identified to date, including laboratory-acquired; *in utero*; receipt of blood components; organ/tissue transplant; and, possibly, breast milk.
- **Laboratory evidence of arboviral or WNV infection in the individual.** The current front-line test identifies the presence of flaviviruses, which include, but may not distinguish, WNV. Both false-positive and false-negative results have been recorded for these tests,

therefore results should not be relied on as definitive without further evidence. Confirmation tests are available at the National Microbiology Laboratory, Public Health Agency of Canada. (See later for further laboratory and diagnostic information.)

- **Unexplained encephalitis, meningitis, motor neuropathy, or recent onset of movement disorders with parkinsonian features** in the spring, summer, or early fall, particularly in adults aged ≥ 50 years or in travellers to the southern U.S., the Caribbean, or Central or South America at other times of the year.

Clinical case definitions

WNV infection in humans may be asymptomatic (80% of infected individuals), or it may manifest as West Nile non-neurological syndrome (WN Non-NS, previously referred to as West Nile fever) (20% of infected individuals) or as West Nile virus neurological syndrome (WNNS) (< 1% of infected individuals)⁽¹⁰⁾. The full spectrum of clinical illness associated with WNV infection continues to be defined. The informa-

tion that follows is based on illness reported in Canada and the U.S. by August 2004.

WNNS

Less than 1% (about 1 in 150) of WNV infections result in severe neurological disease. Although severe manifestations can occur in all age groups, there is an increased risk and incidence of severe neurological illness (e.g. encephalitis) with increasing age. WNNS is defined below.

Clinical criteria

History of exposure in an area where WNV activity is occurring

OR

history of exposure to an alternative mode of transmission

AND

onset of fever

AND RECENT ONSET OF AT LEAST ONE of the following:

- encephalitis (acute signs of central or peripheral neurologic dysfunction), or
- viral meningitis (pleocytosis and signs of infection, e.g. headache, nuchal rigidity), or
- acute flaccid paralysis (e.g. poliomyelitis-like syndrome or Guillain-Barré-like syndrome), or
- movement disorders (e.g. tremor, myoclonus), or
- Parkinsonism or Parkinsonian-like conditions (e.g. cogwheel rigidity, bradykinesia, postural instability), or
- other neurological syndromes as defined below.

Encephalitis/meningoencephalitis is the most common neurological presentation, although symptomatic young individuals are more likely to exhibit meningitis than encephalitis (Table 1)⁽¹¹⁻¹³⁾. Immunosuppression, particularly after solid organ or bone marrow transplantation, also appears to increase morbidity.

A significant feature of WNV neurological illness may be marked muscle weakness, which is more frequently

unilateral but can be bilateral. WNV should be considered in the differential diagnosis of all suspected cases of acute flaccid paralysis with or without sensory deficit. WNV-associated weakness typically affects one or more limbs. Muscle weakness may be the sole presenting feature of WNV illness (in the absence of other neurologic features) or may develop in the setting of fever, altered reflexes, meningitis, or encephalitis. Weakness typically develops early in the course of clinical infection. Patients should be carefully monitored for evolving weakness and, in particular, for acute neuromuscular respiratory failure, which is a severe manifestation associated with high morbidity and mortality. Electromyography and lumbar puncture should be performed to differentiate WNV-associated paralysis from acute demyelinating polyneuropathy (e.g. Guillain-Barré syndrome). Lymphocytic pleocytosis (an increase in white blood cells with a predominance of lymphocytes in the cerebrospinal fluid [CSF]) is commonly seen in acute flaccid paralysis due to WNV, whereas pleocytosis is not a feature of Guillain-Barré syndrome (Table 2)^(11,12).

Other emerging clinical syndromes, identified in recent years, include but are not limited to the following: myelopathy, rhabdomyolysis (acute destruction of skeletal muscle cells), peripheral neuropathy, polyradiculoneuropathy, optic neuritis, and acute demyelinating encephalomyelitis⁽¹¹⁻¹³⁾. Ophthalmologic conditions, including chorioretinitis and vitritis, and facial weakness have also been also reported, as have gastrointestinal (GI) symptoms (nausea, vomiting, and diarrhea)⁽¹⁴⁾. Myocarditis, pancreatitis, and fulminant hepatitis have not been identified in North America but have been reported in outbreaks of WNV in South Africa. “Aseptic” meningitis without encephalitis or acute flaccid paralysis occurring in August and September when WNV is circulating may be due to non-polio enteroviruses circulating at the same time. These should be considered in the differential diagnosis.

WN Non-NS

Generally, WNV infection is characterized by a mild febrile illness with sudden onset and usually resolves in 3 to 6 days. Conventional terminology used in past

Table 1. Diagnostic findings for West Nile virus neurological syndromes

Diagnosis	Clinical presentation	CSF findings	Neurological imaging
West Nile meningitis	Signs of meningeal inflammation: nuchal rigidity; jolt accentuation of headache; Kernig or Brudzinski sign; or photophobia, without evidence of neuraxis involvement* Fever ($\geq 380^{\circ}$ C) or hypothermia ($< 350^{\circ}$ C)	Pleocytosis (≥ 5 leukocytes/mm ³)	Evidence of acute meningeal inflammation, however may present with normal findings. Neuroimaging is useful to rule out other pathology.
West Nile encephalitis	Signs of encephalopathy: depressed or altered level of consciousness; lethargy; or personality change lasting > 24 hours** Fever ($\geq 380^{\circ}$ C) or hypothermia ($< 350^{\circ}$ C); seizures, either new onset or exacerbation of previously controlled symptoms; focal neurological deficit; meningismus	Pleocytosis (≥ 5 leukocytes/mm ³)	Consistent with acute inflammation (with or without meninges involvement) or acute demyelination; electroencephalograph findings consistent with encephalitis
Acute flaccid paralysis	Acute onset of limb weakness with marked progression over 48-hour period*** Asymmetrical weakness; areflexia of affected limb(s); absence of pain, paresthesia, or numbness in affected limb(s)	Pleocytosis (≥ 5 leukocytes/mm ³) and elevated protein levels (≥ 0.45 g/dL)	Electrodiagnostic studies (EMG, nerve conduction) consistent with an anterior horn cell process Spinal cord magnetic resonance imaging documenting abnormal increased signal in gray matter

Source: Sejvar JJ, Haddad M, Tierney B et al. JAMA 2003;290:511-15
 *AND additional evidence of acute infection including 1 or more of the remaining findings (including CSF and neurological)
 **AND additional evidence of central nervous system inflammation, including 2 or more remaining findings (including CSF and neurological)
 ***AND at least 2 of the remaining findings (including CSF and neurological)

years to describe this mild febrile illness has been West Nile fever (WNF). In Canada, during the 2003 season, a noticeable percentage of “West Nile fever” cases were noted not to have a documented fever as a sign/symptom. In Alberta, 33% (73/221) of WNF did not have a documented fever⁽¹⁴⁾. Therefore, in 2005 the terminology for this category changed to West Nile non-neurological syndrome (WN Non-NS), more accurately reflecting these evolving clinical findings. WN Non-NS is defined below.

Clinical criteria

History of exposure in an area where WNV activity is occurring

OR

history of exposure to an alternative mode of transmission

AND AT LEAST TWO of the following:

- fever,
- myalgia,
- arthralgia,
- headache,
- fatigue,
- lymphadenopathy,
- maculopapular rash.

It is possible that other clinical signs and symptoms could be identified that have not been listed and that may accompany the diagnostic test criteria for probable or confirmed cases. For example, GI symptoms were seen in many WNV patients in Canada⁽¹⁴⁾ and the U.S. in 2003 and 2004.

Table 2. Differential diagnosis: West Nile virus poliomyelitis-like syndrome and Guillain-Barré syndrome*

	WNV poliomyelitis-like syndrome	Guillain-Barré syndrome
Fever, elevated white blood cell (WBC) count	Present	Absent
Weakness	Asymmetric, +/- monoplegia	Symmetric, proximal and distal
Sensory symptoms	Usually absent or minimal. Some will have sensory symptoms	Painful distal dysesthesias, sensory loss
Bowel, bladder involvement	Often involved	Rarely involved
Encephalopathy	Often present	Absent
Cerebral spinal fluid (CSF)	Elevated WBC and protein	Albuminocytological dissociation (elevated protein, normal cell count)
Electrodiagnostics	Typically, denervation (anterior horn cell/ motor axon) Occasionally, demyelination	Demyelination
Serology	WNV IgM (CSF, serum)	IgM antibodies to nerve components: GM1, GM2, GD1a
Note: these features are variable, and in some cases there may be overlap. *Adapted with permission from Dr. K. McClean, MD, FRCPC, University of Saskatchewan.		

Diagnostic testing

Laboratory tests for WNV are available through provincial public health laboratories (supplementary testing is available at the National Microbiology Laboratory upon referral from provincial laboratories). Specific clinical and laboratory tests and their interpretation are shown in Tables 3 and 4.

Front-line serologic tests use an IgM antibody capture, enzyme-linked immunosorbent assay (MAC-ELISA) to detect the presence of IgM antibodies to WNV. Specimens should be collected for testing within 8 days of the onset of illness. Because of serologic cross-reactivity among flaviviruses, false-positive results may occur with serum specimens collected from individuals exposed to related viruses, such as St. Louis encephalitis and dengue viruses. IgM ELISA false-positive results may occur in individuals vaccinated against yellow fever or Japanese encephalitis, but cross-reactivity is relatively uncommon. Some patients with WNF may have false-negative results early in the course of their illness, and therefore a follow-up serum sample is necessary to document a WNV seroconversion. Results are normally available within 48 hours.

Confirmatory testing is currently available in Ontario and Quebec, and at the National Microbiology Laboratory, Winnipeg. These tests detect an increase in WNV-specific neutralizing antibody titre between serum specimens obtained in the acute and convalescent stages of disease. Results take 2 to 3 weeks.

WNV can be isolated from, or viral antigen or nucleic acid can be detected in, CSF, serum, other body fluids, and tissue; however, test sensitivity is lower than serologically based methods because of the low concentration of virus in the specimens and the transient nature of WNV viremia. Virus isolation or immunohistochemistry is laborious and may take weeks to perform; polymerase chain reaction (PCR) procedures are rapid to carry out (1-2 days), and the sensitivity of nucleic acid testing varies from 10% to 50%. It should be noted that a minority of individuals may have PCR positive and IgM ELISA negative results when samples from the acute stage are tested, therefore both test methods may be warranted.

WNV IgM antibodies may persist for more than a year, and the demonstration of IgM antibodies in a patient's serum, particularly in residents of endemic areas, may not be diagnostic of an acute WNV infec-

Table 3. West Nile virus-specific diagnostic findings

Test	Observation
Cerebrospinal fluid	Bacterial culture negative Pleocytosis with leukocyte counts > 5 leukocytes/mm ³ , usually with lymphocytes predominating Elevated protein (≥ 45 mg/dL) Glucose is normal
Sodium	Hyponatraemia sometimes present, particularly in patients with encephalitis
Computed tomographic brain scan	Usually normal in WNV disease. This is useful to rule out other intracranial diseases/processes that have similar presentations.
Magnetic resonance imaging	Normal in most patients or nonspecific (enhancement of the leptomeninges and/or periventricular areas). Occasionally show focal lesions in the pons, basal ganglia, and thalamus. Fluid-attenuated inversion recovery (FLAIR) sequence abnormalities late in the course of unusually severe disease. Increased signal in diffusion-weighted images may precede FLAIR abnormalities.
Electromyogram/nerve conduction studies	(In acute flaccid paralysis) Typical features of denervation (decreased motor amplitude with normal conduction velocity interpreted as motor neuronopathy) Occasionally, features of demyelination (decreased conduction velocity interpreted as motor axonopathy)

tion⁽¹⁵⁾. Seroconversion (by hemagglutination inhibition, IgG ELISA, or the plaque reduction neutralization assay) demonstrates a current WNV infection. Therefore, the collection of sera during the acute and convalescent phases for serologic analysis is particularly important to rule out diagnostic misinterpretation early in the WNV season (e.g. May, June) and to identify initial cases in a specific jurisdiction. However, it should be noted that seroconversions may not always be documented because of the timing of acute sample collection (i.e. titres may have already peaked). If static titres are observed in sera paired from the acute and convalescent stage, it is still possible the case may represent a recent infection. To help resolve this question, the use of IgG avidity testing may be considered to distinguish between current and past infection. The presence of both IgM antibodies and low avidity IgG in a patient's serum sample collected during the convalescent stage is consistent with a current case of viral illness. Test results that show the presence of IgM and high avidity IgG are indicative of exposures that have occurred in the previous season (P.N. Levett: personal communication, 2005).

Early in infection the immune system generates antibodies that bind relatively weakly to viral antigen (low avidity). As the infection proceeds, an increasing percentage of newly generated IgG antibodies display higher binding affinity to virus antigen and thus avidity also rises (Note: avidity is usually measured on the basis of the ability of IgG to dissociate from antigen preparations after incubation with a solution of urea). As long as high avidity IgG has not yet been detected in the serum it can be assumed that the individual was exposed to the viral agent recently. With respect to WNV infection, it has not been precisely determined when (i.e. how long after exposure) high avidity antibodies reach levels in serum that can be accurately detected by serologic assays (there may be significant variation depending on the individual). However, it has been shown that > 95% of sera collected from individuals exposed to WNV 6-8 months previously will have IgG antibodies that bind strongly to viral antigen and will give high avidity scores using both indirect fluorescent antibody and ELISA testing formats. **Note: Avidity testing will not replace confirmatory neutralization testing; non-WNV flavivirus IgG antibodies (e.g. dengue, St. Louis encephalitis) may bind to the antigen preparations used in avidity assays⁽¹⁶⁾.**

Table 4. West Nile virus laboratory diagnostic tests

Name and type of test	Time requested to complete test and obtain result	Interpretation
<p>Hemagglutination inhibition (HI) Tests for the presence of IgM and IgG flavivirus antibodies. Two serum samples are requested from each patient: a sample from the acute phase and a convalescent sample taken 2 to 3 weeks later. HI test determines the presence of antibodies and documents seroconversions.</p>	<p>This test takes approximately 48 hours optimum.</p>	<p>Positive result indicates flavivirus infection. This test is not specific for WNV and will cross-react with antibodies against other members of the flavivirus family.</p>
<p>WNV IgM ELISA Detects the presence of WNV antibodies in serum and CSF.</p>	<p>Different formats exist that differ in the time required to perform the assay. Commercial kits (e.g. Focus, PanBio IgM ELISA) can be completed in 4-5 hours. The CDC IgM ELISA takes 48 hours to obtain results.</p>	<p>Positive results generally suggest recent WNV infection (persistence of IgM antibodies can be a factor in test interpretation). IgM antibody in CSF is strongly suggestive of CNS infection.</p>
<p>Flavivirus IgG ELISA Tests for the presence of IgG flavivirus antibodies. Two serum samples are required: a sample from the acute phase and a convalescent sample taken 2 to 3 weeks later. IgG ELISA determines the presence of antibodies and can document seroconversions.</p>	<p>Paired (acute and convalescent) serum samples taken 2 to 3 weeks apart. The test takes approximately 1 day.</p>	<p>Positive result indicates flavivirus infection. This test is not specific for WNV and is extremely cross-reactive with other flavivirus antibodies.</p>
<p>Plaque reduction neutralization test Tests for the presence of WNV neutralizing antibodies and is a confirmatory serologic test.</p>	<p>This test takes at least 7 days.</p>	<p>Confirms current WNV infection by detecting the presence of specific WNV neutralizing antibodies in convalescent sera.</p>
<p>Polymerase chain reaction test A less frequently requested test to demonstrate the presence of WN viral genome in patient samples such as cerebrospinal fluid, blood, and brain tissue.</p>	<p>This test takes approximately 24 hours.</p>	<p>Confirms WNV infection</p>

Immune compromised individuals may not be able to mount an immune response necessary for a serologic diagnosis. WNV diagnostic test criteria for these individuals should be discussed with a medical microbiologist.

Reporting

WNV infection became a nationally reportable disease in Canada on 1 June, 2003. Cases should be reported as soon as possible to provincial/territorial public health authorities by the usual mechanism for each jurisdiction. The reporting categories are defined as follows:

Suspect case meets the clinical criteria (see WNNS or WN Non-NS described earlier) **IN THE ABSENCE OF OR PENDING** diagnostic tests **AND IN THE ABSENCE** of any other obvious cause.

Probable case meets the clinical criteria (see WNNS or WN Non-NS) **AND AT LEAST ONE** of the probable diagnostic test criteria (see Table 5).

Confirmed case meets the clinical criteria (see WNNS or WN Non-NS) **AND AT LEAST ONE** of the confirmed case diagnostic test criteria (see Table 5).

Table 5. West Nile virus laboratory diagnostic criteria

Classification	Laboratory
Probable case	Detection of flavivirus antibodies in a single serum or CSF sample using a WN virus IgM ELISA without confirmatory neutralization serology (e.g. plaque reduction neutralization test [PRNT]) OR
	A four-fold or greater change in flavivirus HI titres in paired acute and convalescent sera or demonstration of a seroconversion using a WN virus IgG ELISA* OR
	A titre of $\geq 1:320$ in a single WN virus HI test, or an elevated titre in a WN virus IgG ELISA, with a confirmatory PRNT result OR
	(Note: A confirmatory PRNT or other kind of neutralization assay is not required in a health jurisdiction/authority where cases have already been confirmed in the current year.)
	Demonstration of Japanese encephalitis (JE) serocomplex-specific genomic sequences in blood by nucleic acid screening of donor blood, by blood operators in Canada.
Confirmed case	A four-fold or greater change in WN virus neutralizing antibody titres (using a PRNT or other kind of neutralization assay) in paired acute and convalescent sera, or CSF OR
	Isolation of WN virus from, or demonstration of WN virus antigen or WN virus-specific genomic sequences in tissue, blood, CSF or other body fluids OR
	Demonstration of flavivirus antibodies in a single serum or CSF sample using a WN virus IgM ELISA* confirmed by the detection of WN virus-specific antibodies using a PRNT (acute or convalescent specimen) OR
	A four-fold or greater change in flavivirus HI titres in paired acute and convalescent sera or demonstration of a seroconversion using a WN virus IgG ELISA* AND the detection of WN virus-specific antibodies using a PRNT (acute or convalescent serum sample).
*Both CDC and commercial IgM/IgG ELISAs are now available for front-line serologic testing. Refer to appropriate assay procedures and kit inserts for the interpretation of test results.	

Treatment

Treatment of WNV infection is supportive. In patients with severe disease, this may include hospitalization, intravenous fluids, respiratory support, and prevention of secondary infection. Empirical treatment of other treatable conditions with similar severe presentations should not be delayed pending WNV virologic results. Currently, there is no conclusive evidence that ribavirin, interferon (alpha-2b), steroids, anti-seizure drugs, osmotic agents, or immune globulin is effective in the management of WNV infection⁽¹⁷⁾.

Clinical outcome

Most patients with WNV illness make a full recovery. While patients with WNNS can make a full recovery, many experience severe long-term sequelae, including physical, cognitive, and functional deficits. WNNS cohort studies have found persistent symp-

toms, including fatigue, difficulty in walking, muscle weakness, memory loss, and depression⁽¹⁸⁾. Long-term follow-up is recommended, especially for patients with WNNS.

Case fatality rates among hospitalized patients in North America are approximately 12%⁽⁹⁾. Mortality risk factors include advancing age (especially > 70 years); clinical manifestations that include encephalitis with severe muscle weakness or altered level of consciousness; and possibly (limited data) pre-existing conditions such as diabetes mellitus and immunosuppression⁽⁹⁾.

Prevention

Current federal/provincial/territorial and local prevention activities include raising public health awareness about the need to avoid mosquito bites and implementing measures to protect blood and tissue donations. Currently, human vaccines are undergo-

ing development and clinical trials in the U.S. If successful, they may become available there by 2006-07. The National Advisory Committee on Immunization (NACI) will be developing vaccine use strategies for Canada.

For persons whose occupation may place them at risk of infection through mosquito bites (work outdoors) or handling of dead birds or animals (pick-ups, necropsy suites, slaughter houses, poultry plants), an occupational health advisory developed in 2000 (and revised 24 June, 2003) by the Health Canada Workplace Health and Public Safety Programme is available at <http://www.phac-aspc.gc.ca/wnv-vwn/pdf/wnv_occhealth2003_e.pdf>.

Precautions for health care providers

Currently, there is no evidence that WNV can be transmitted directly from person to person (e.g. through touch, aerosols, respiratory droplets, fecal-oral or sexual contact). There is evidence that blood and CSF contain live virus during the viremia stage of infection, beginning several days before but rapidly disappearing after symptom onset^(1,2). Therefore it is recommended that standard universal precautions be taken to prevent infection when handling body fluids. While the risk of acquiring WNV infection from needle-stick or sharps injuries is low as a result of the low grade/transient viremia in human hosts, health care workers who sustain these injuries should be reported to a physician, infection control program, and/or occupational health and safety services. The only cases reported to date from sharps injuries have occurred in the laboratory setting⁽⁵⁾. Currently, there are no specific guidelines regarding WNV infection and needle-stick or sharps injuries. Individual institutions that choose to develop such protocols should refer to the following documents, available at <<http://www.phac-aspc.gc.ca/publicat/ccdr-rmtc/index.html>>.

- An Integrated Protocol to Manage Health Care Workers Exposed to Bloodborne Pathogens. CCDR 1997;23S2.

- Preventing the Transmission of Bloodborne Pathogens in Health Care and Public Service Settings. CCDR 1997;23S3.
- Prevention and Control of Occupational Infections in Health Care. CCDR 2002;28S1.

Blood, tissues and organs

Timely identification of viremic patients or those with acute WNV infection has important implications for the blood supply in Canada. All probable and confirmed cases involving blood donors or recipients (especially those who have donated or received blood 56 days before symptom onset) should be reported immediately to the Canadian Blood Services (your local Blood Centre or to 1-888-236-6283: select the "Nursing" option) or Héma Québec (1-888-646-2237, in the province of Quebec only). Cases involving donors or recipients (within 56 days of transplantation) of human cells, tissue, or organs should be reported to the local/regional cell/tissue/organ bank and federal, provincial, territorial or local public health authorities as required by law.

Further information

Further information concerning the epidemiology, prevention, and control of WNV as well as public education about WNV is available at <www.westnilevirus.gc.ca>.

As more information about the clinical presentation and management of WNV becomes available, the guidance provided in this document may be revised. Clinicians are advised to consult the Public Health Agency of Canada or provincial/territorial WNV websites regularly for revisions. (See Public Health Agency of Canada's WNV web site at <www.westnilevirus.gc.ca>.)

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