Western Equine Encephalitis

Revision Dates

Case Definition	January 2013
Reporting Requirements	January 2013
Remainder of the Guideline (i.e., Etiology to References sections inclusive)	December 2005

Case Definition

Confirmed Case

Clinical illness[1] with laboratory confirmation of infection:

- Isolation of western equine encephalitis (WEE) virus from an appropriate clinical specimen(e.g., serum, CSF)[2]

OR

- Demonstration of WEE virus antigen in tissue, blood or CSF

OR

- Detection of WEE viral nucleic acid (e.g., PCR) in an appropriate clinical specimen (e.g., plasma, CSF, serum) [2]

OR

- Seroconversion or significant difference between acute and convalescent phase WEE Hemagglutination Inhibition Assay (HAI) titers ideally taken at least two weeks apart from an appropriate clinical specimen (e.g., serum)[2] and confirmed by Plaque Reduction Neutralization Assay (PRNT)[3].

Probable Case

Clinical illness[1] and one of the following:

- Seroconversion or significant difference between acute and convalescent-phase WEE HAI titres ideally taken at least 2 weeks apart but not confirmed by PRNT[3]

OR

- Stable elevated serial HAI titres[4] to WEE that occur during a period when and where arboviral transmission is likely[5]

OR

- Single elevated HAI titre[4] to WEE that occurs during a period when and where arboviral transmission is likely.[5]

[1] Clinical illness is characterized by a febrile illness of variable severity associated with neurological symptoms ranging from headache to aseptic meningitis or encephalitis. Arboviral encephalitis cannot be distinguished clinically from other central nervous system (CNS) infections. Symptoms can include headache, confusion or other alteration in sensorium, nausea and vomiting. Signs may include fever, meningismus, cranial nerve palsies, paresis or paralysis, sensory deficits, altered reflexes, convulsions, abnormal movements and coma of varying degree.

[2]Refer to the National Microbiology Laboratory (NML) Guide to Services for current specimen collection and submission information.

[3] Seroconversion indicates recent infection with a flavivirus (e.g., dengue fever, California serogroup virus, West Nile virus or Yellow fever) but cannot pinpoint which one due to antibody cross-reactivity. These are considered probable cases. PRNT confirmatory testing may be requested at NML through the Provincial Laboratory for Public Health (ProvLab).

[4] A single elevated antibody titre of ≥ 1:40 by HAI suggests recent infection. It is recommended that a second specimen be collected. A second laboratory result with a stable (unchanged/static or ≤ 2-fold rise) elevated antibody titre is suggestive of recent infection.

[5] WEE exposure is most likely to occur from spring to early fall in Canada and western/central US, and year round in parts of South America.(1)

Reporting Requirements

1. **Physicians, Health Practitioners and others**
   Physicians, health practitioners and others listed in Sections 22(1) or 22(2) of the *Public Health Act* shall notify the Medical Officer of Health (MOH) (or designate) of all confirmed and probable cases in the prescribed form by mail, fax or electronic transfer within 48 hours (two days).

2. **Laboratories**
   Section 23(a)(i) of the *Public Health Act* requires that all laboratories, including regional laboratories and the ProvLab shall report all positive laboratory results by mail, fax or electronic transfer within 48 hours (two days) to the:
   - Chief Medical Officer of Health (CMOH) (or designate),
   - MOH (or designate) and
   - Attending/ordering physician.

3. **Alberta Health Services and First Nations and Inuit Health Branch**
   - The MOH (or designate) of the zone where the case currently resides shall forward the preliminary Notifiable Disease Report (NDR) of all confirmed and probable cases to the CMOH (or designate) within two weeks of notification and the final NDR (amendment) within four weeks of notification.
   - For out-of-zone reports, the MOH (or designate) first notified shall notify the MOH (or designate) of the zone where the client currently resides by mail, fax or electronic transfer and fax a copy of the positive laboratory report within 48 hours (two days).
   - For out-of-province and out-of-country reports, the following information should be forwarded to the CMOH (or designate) by phone, fax or electronic transfer within 48 hours (two days) including:
     - name,
     - date of birth,
     - out-of-province health care number,
     - out-of-province address and phone number,
     - attending physician (locally and out-of-province) and
     - positive laboratory report (faxed).

Etiology (2)

Western equine encephalitis (WEE) virus is a member of the *Alphavirus* genus. WEE cannot survive outside of the host and are sensitive to drying as well as moist and dry heat.

Clinical Presentation (2)

Most human cases of WEE are asymptomatic or may result in a nonspecific flu-like syndrome. The onset may be insidious or sudden with fever, headache, myalgia, malaise, and occasionally prostration. Studies have shown that invasion of the CNS generally follows initial viral replication in various peripheral sites and a period of viremia. The infection may lead to encephalitis with a fatal outcome or permanent neurologic sequelae, however, only a small proportion of infected persons develop encephalitis. Viral transfer through the olfactory tract has been suggested.

Most WEE infections are asymptomatic or present as a mild, non-specific illness. Persons with a clinically apparent illness generally experience sudden onset of fever, headache, nausea, vomiting, anorexia, and malaise followed by an altered mental status, weakness, and signs of meningeal irritation. Children, especially those under one year of age, are affected more severely than adults and may be left with permanent sequelae. This is seen in 5 – 30% of young patients. The overall mortality rate is about 3%.

Diagnosis

Diagnosis of WEE is made through rapid serologic assays such as IgM capture ELISA and IgG ELISA. In early infection IgM antibody is more specific, while later in infection, IgG antibody is more reactive. Viral isolation and identification have been useful in defining viral agents in serum, CSF, and mosquito vectors.

Epidemiology

Reservoir

The reservoir is most often blood-feeding mosquitoes. Humans and domestic animals can develop clinical illness but are usually dead-end hosts as they do not produce significant viremia and do not contribute to the transmission cycle.(3)

The true reservoir of WEE is unknown. It is thought that virus may overwinter in birds or other animals. Horses and humans are uncommon sources of mosquito infection.

Transmission

Infection is transmitted to humans through the bite of an infected mosquito. The mosquito becomes infected after feeding on a source infected with the virus and then spreading the infection in subsequent blood meals.

The enzootic cycle of WEE involves passerine birds and culicine mosquitoes. The mosquito is associated with irrigated agriculture and stream drainages. Virus is transmitted via the bite of a viremic mosquito. *C. tarsalis* is the primary vector. Other important mosquito vectors include *Aedes melanimon, Ae. dorsalis,* and *Ae. camperstris.*

Incubation Period

The incubation period of WEE is usually 5 – 15 days.

Period of Communicability
WEE is not directly transmitted from person to person. The virus is not demonstrable in blood or CSF after onset of disease. In birds, the viremia lasts two to five days. Mosquitoes are infected for life (1 – 4 months) and vertical transmission (female to egg) may exist. Viremia in horses is rarely present in high titres for long periods.

Host Susceptibility
Rural residents are at increased risk of exposure to WEE.

Occurrence
General
WEE was first isolated in California in 1930 from the brain of a horse. It remains an important cause of encephalitis in horses and humans in North America, in particular, the western parts of the US and Canada. Human cases are usually first seen in June or July. WEE occurs in western and central US, Canada and parts of South America (Argentina). Cases of WEE occur in temperate latitudes in summer and early fall and are limited to areas and years of high temperature and abundant mosquito populations.(4)

Three cases of WEE were reported in the early 1990s. No human cases of WEE have been reported since 1994 in the US. Epidemics of WEE recur at irregular intervals (generally every 10 – 11 years).(4)

Canada
WEE is not a nationally notifiable disease and the annual incidence is not known.

Alberta
There have been no cases of WEE reported in Alberta between 1994 and 2004.

Key Investigation
Single Case/Household Cluster
- Identify the potential source of the infection based on:
  - history of travel to endemic areas,
  - mode of transmission, and
  - incubation period.
- Assess for history of mosquito bites.

Control
Management of a Case
- Supportive care, including IV fluids and ventilation.
- Symptomatic treatment.

Treatment of a Case
- Antibiotics are not effective.
- No effective antiviral treatment has been identified.

Management of Contacts
- The infection is not passed from person to person.

Preventive Measures

- Educate the public about personal protective measures including:
  - reducing time outdoors at dusk,
  - wearing long pants and long sleeved shirts, and
  - applying mosquito repellent containing DEET.
- Spray of insecticides to kill larvae and adult mosquitoes.
- Immunize domestic animals (in endemic areas) and/or house them away from human living quarters.
  - Equine vaccine is available for WEE.(2)
- Support surveillance programs to allow for rapid identification of dangerous viruses in mosquito populations which can shorten public health response time and reduce the spread of infected vectors.
- Laboratory safety
  - Arbovirus requires biosafety level 3 practices.
  - Refer to current PHAC Laboratory Safety Guidelines at: http://www.phac-aspc.gc.ca/publicat/lbg-ldmbl-04/index.html

References

(1) Heymann, DL (Ed). Control of communicable diseases (19th ed). American Public Health Association: Washington, D.C; 2008.

(2) CDC. Information on Arboviral Encephalitides. Division of Vector-Borne Infectious Diseases. July 2001. http://www.cdc.gov/ncidod/dvbid/arbor/arbdet.htm

(3) CDC. Guidelines for arbovirus surveillance programs in the United States. Department of Health and Human Services. Colorado: April 1993. http://stacks.cdc.gov/view/cdc/6183/

(4) Centres for Disease Control and Prevention. Fact sheet: Arboviral Encephalitides. Division of Vector-Borne Infectious Diseases. July 2001. http://www.cdc.gov/ncidod/dvbid/arbor/arbofact.htm

