



## Communicable Disease Case Reporting and Investigation Protocol **ARBOVIRAL INFECTION**

### **I. IDENTIFICATION AND DEFINITION OF CASES**

**A. Clinical Description:** Arboviral infection may be asymptomatic or result in a febrile illness of variable severity, sometimes associated with neurologic symptoms ranging from headache to aseptic meningitis and encephalitis. Arboviral encephalitis cannot be distinguished clinically from infection with other neurotropic viruses. Symptoms include fever, headache, confusion or other alterations in sensory, nausea, or vomiting. Signs may include evidence of elevated intracranial pressure, meningeal irritation, cranial nerve palsies, paresis or paralysis, altered reflexes, or convulsions. Less common neurological syndromes can include cranial and peripheral neuritis/neuropathies, including Guillain-Barré syndrome. Arboviruses causing encephalitis include the following: Mosquito-borne viruses occurring in the United States:

- West Nile virus (WNV)
- St. Louis encephalitis (SLEV)
- California encephalitis (California serogroup includes La Crosse [LACV], Jamestown Canyon [JCV], Snowshoe Hare [SSHV], and California [CEV])
- Eastern equine encephalitis (EEEV)
- Western equine encephalitis (WEEV)

Tickborne virus occurring in United States: Powassan encephalitis (POWV)

Mosquito-borne viruses associated with traveling to an endemic country:

- Dengue (DENV)
- Japanese encephalitis (JEV)
- Chikungunya (CHIKV)
- Other central nervous system infections transmitted by mosquitoes, ticks, or midges (Venezuelan equine encephalitis [VEEV], Cache valley encephalitis [CVV])

These viruses may also cause non-neuroinvasive syndromes, most commonly manifesting as febrile illnesses. These are non-localized, self-limited illnesses with headache, myalgias, and arthralgias, and sometimes accompanied by a skin rash or lymphadenopathy. Although rare, non-neuroinvasive syndromes caused by these viruses may also include myocarditis, pancreatitis, or hepatitis. Laboratory confirmation of arboviral illnesses lacking a documented fever does occur and overlap of the various clinical syndromes is common.

#### **Clinical Criteria for Diagnosis:**

- **Neuroinvasive disease** requires at least one of the following signs and symptoms, as documented by a physician, and in the absence of a more likely clinical explanation:
  - Acutely altered mental status (e.g., disorientation, confusion, memory deficit, stupor, coma), **OR**
  - Aseptic meningitis, encephalitis, **OR**
  - Acute flaccid paralysis (AFP); AFP may result from anterior “polio” myelitis, peripheral neuritis, or post-infection peripheral demyelinating neuropathy (i.e., Guillain-Barre syndrome), **OR**
  - Stiff neck, seizures, limb weakness, sensory deficits, abnormal reflexes, abnormal movements, cranial nerve palsies, **OR**
  - Pleocytosis (increased white blood cell count) in cerebrospinal fluid (CSF) or abnormal neuroimaging.
- **Non-neuroinvasive disease** requires the presence of documented fever (chills) as reported by the patient or clinician, the absence of neuroinvasive disease (above), and the absence of a more likely clinical explanation for the illness. Other signs and symptoms may include headache, stiff neck, myalgias, arthralgias, rash, lymphadenopathy, vertigo, or vomiting.

## B. Laboratory Criteria:

- Confirmatory laboratory evidence:
  - Isolation of virus from, or demonstration of specific viral antigen or nucleic acid in tissue, blood, cerebrospinal fluid (CSF), or other body fluid, **OR**
  - Fourfold or greater change in virus-specific quantitative antibody titers between acute (within two weeks of illness onset date) and convalescent sera (two to four weeks after illness onset date), **OR**
  - Virus-specific immunoglobulin M (IgM) antibodies in serum by antibody-capture enzyme immunoassay (Capture EIA/ELISA) AND confirmed by demonstration of virus-specific neutralizing antibodies (PRNT) in the same or later specimen, **OR**
  - Virus-specific IgM antibodies in CSF, with or without a reported increase in white blood cell count in CSF (pleocytosis), and a negative result for other IgM antibodies in CSF for arboviruses endemic to the region where exposure occurred.
- Supportive laboratory evidence:
  - Virus-specific IgM antibodies in CSF or serum detected by antibody-capture EIA/ELISA, but with no other testing in the same or later specimen.

**Note:** Positive results from a single serologic test can be misleading because serologic cross-reactivity often occurs between closely related arboviruses (especially between SLEV and WNV). It is, therefore, recommended that an arbovirus IgM panel (which includes testing for WNV, SLEV, LACV, JCV, EEEV, and POWV) be requested when there is clinical suspicion of arboviral disease, rather than requesting individual tests.

Arboviral transmission varies according to local climatic conditions, and in some patients, virus-specific IgM antibody can be detectable for more than a year following infection. Therefore, the importance of a recent travel history and thorough serologic testing cannot be overemphasized. IgG antibody can be detected throughout a person's lifetime after an infection. Thus, a positive IgG and a negative IgM may indicate previous infection at some point in time.

## C. Wisconsin Surveillance Case Definition:

- **Confirmed:** A case that meets one of the above clinical criteria for diagnosis, **AND** at least one of the confirmed laboratory criteria, **AND** occurred when and where there is a high likelihood of vector activity.
- **Probable:** A case that meets one of the above clinical criteria for diagnosis, **AND** at least one of the supportive laboratory criteria, **AND** occurred when and where there is a high likelihood of vector activity.
- **Suspect:** A case that meets the supportive laboratory criteria, **OR** meets one of the above clinical criteria for diagnosis.

## II. REPORTING

- A. **Wisconsin Disease Surveillance Category II – Methods for Reporting:** This disease shall be reported to the patient's local health officer or to the local health officer's designee within 72 hours of recognition of a case or suspected case, per Wis. Admin. Code § [DHS 145.04 \(3\) \(b\)](#). Report electronically through the Wisconsin Electronic Disease Surveillance System (WEDSS), or mail or fax a completed Acute and Communicable Disease Case Report ([F-44151](#)) to the address on the form.
- B. **Responsibility for Reporting:** According to Wis. Admin. Code § [DHS 145.04\(1\)](#), persons licensed under Wis. Stat. ch. [441](#) or [448](#), laboratories, health care facilities, teachers, principals, or nurses serving a school or day care center, and any person who knows or suspects that a person has a communicable disease identified in [Appendix A](#).
- C. **Clinical Criteria for Reporting:** Clinically compatible illness for either neuroinvasive or non-neuroinvasive disease.
- D. **Laboratory Criteria for Reporting:** Laboratory evidence of infection by detection of virus-specific IgM, virus-specific immunoglobulin G (IgG), virus-specific ribonucleic acid sequence by polymerase chain reaction (PCR) in clinical specimens, or detection of specific viral antigen by immunohistochemistry.

### III. CASE INVESTIGATION

- A. **Responsibility for case investigation:** It is the responsibility of the local health department (LHD) to investigate or arrange for investigation of suspected or confirmed cases as soon as is reasonably possible. A case investigation may include information collected by phone, in person, in writing, or through review of medical records or communicable disease report forms, as necessary and appropriate.
- B. **Required Documentation:**
  - 1. Complete the WEDSS disease incident investigation report, including appropriate, disease-specific tabs. This may be facilitated by completing an Arbovirus Infection Follow-up Form.
  - 2. Upon completion of investigation, set WEDSS disease incident process status to “Sent to State.”
- C. **Additional Investigation Responsibilities**
  - 1. Obtain detailed travel history and vaccination history against related viruses (e.g., yellow fever or Japanese encephalitis vaccines).
  - 2. Determine if the patient has had a previous arboviral illness diagnosis and, if so, the month and year when this illness occurred.
  - 3. For Powassan virus infections, identify if the patient was exposed to ticks, places of exposure, and travel history approximately 14 days before illness onset.

### IV. PUBLIC HEALTH INTERVENTIONS AND PREVENTION MEASURES

- A. In accordance with Wis. Admin. Code § [DHS 145.05](#), local public health agencies should follow the methods of control recommended in the current editions of *Control of Communicable Diseases Manual*, edited by David L. Heymann, published by the American Public Health Association, and the American Academy of Pediatrics’ *Red Book: Report of the Committee on Infectious Diseases*, unless otherwise specified by the state epidemiologist.
- B. Source investigation by LHD to identify mosquito breeding sites or tick habitat near the probable location of the exposure.
- C. Educate patients on preventing mosquito or tick bites and eliminating mosquito breeding sites or tick habitat.

### V. CONTACTS FOR CONSULTATION

- A. Local health departments and tribal health agencies:  
<https://www.dhs.wisconsin.gov/lh-depts/index.htm>
- B. Bureau of Communicable Diseases, Communicable Diseases Epidemiology Section: 608-267-9003
- C. Wisconsin State Laboratory of Hygiene: 1-800-862-1013

### VI. RELATED REFERENCES

- A. Heymann DL, ed. Arthropod-Borne Viral Diseases. In: *Control of Communicable Diseases Manual*. 20th ed. Washington, DC: American Public Health Association, 2015: 26-42.
- B. Pickering LK, ed. Arbovirus Infections. In: *Red Book: 2015 Report of the Committee on Infectious Diseases*. 30th ed. Elk Grove Village, IL: American Academy of Pediatrics, 2015: 240-246
- C. Centers for Disease Control and Prevention vector-borne diseases website:  
<https://www.cdc.gov/ncezid/dvbd/index.html>
- D. Hoang Johnson DK, Staples JE, Sotir MJ, *et al.* Tickborne Powassan virus infections among Wisconsin residents. *Wis Med J.* 2010; 109:91-97.