

An Overview of Powassan Virus Disease

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Powassan virus is a tick-borne flavivirus that circulates widely throughout parts of North America and eastern Russia.¹⁻³ Closely related to tick-borne encephalitis virus, Powassan virus was first identified in an encephalitic child from Powassan, Ontario, in 1958 (though was later retrospectively identified in ticks from northern Colorado from 1952).²⁻⁴ Powassan virus has 2 distinct genetic lineages that are clinically and serologically indistinguishable: lineage 1 (prototype Powassan virus) and lineage 2 (also known as deer tick virus).^{1,3} The primary vectors of Powassan virus are *Ixodes* species of ticks including *Ixodes cookei* (lineage 1), *Ixodes marxi* (lineage 1), and *Ixodes scapularis* or the black-legged/deer tick (lineage 2).¹⁻³ Small- to medium-sized forest rodents are thought to be amplifying hosts.^{1,3}

In the United States, there have been 125 human cases of Powassan virus disease reported from 2008 to 2017, mostly from the Northeast and Great Lakes regions.¹ Minnesota (n = 32), Wisconsin (n = 22), New York (n = 16), and Massachusetts (n = 16) reported the highest number of cases during this time period, possibly due to increased burden of disease and/or enhanced surveillance in these states.^{1,5} Most cases of Powassan virus disease occur from mid-spring through late fall (peaking in May and June), coinciding with when *Ixodes* species of ticks are most active.^{1,5} While all ages groups and both sexes can be affected, there appears to be a male predilection.^{1,5} Powassan virus disease has been historically underrecognized, though recognition may be improving with increased arboviral surveillance and education.⁵

Clinically, most Powassan virus infections are thought to be asymptomatic.¹ Among those who develop disease after an incubation period of 1 to 4 weeks, initial symptoms may include fever, headache, and nausea/vomiting.^{1,3,6} This may progress to meningitis and/or encephalitis with meningismus, confusion, decreased mental status, focal weakness, cranial nerve palsies, ataxia, and/or seizures.^{1,3-6} Cerebral edema and coma has been reported, and approximately 10% of those with Powassan virus disease die.^{1,3-6} Among survivors, neurologic sequelae are common including recurrent headaches,

cognitive problems, and/or focal neurologic deficits.^{1,3,4,6} However, the full clinical spectrum of Powassan virus disease is not known and is still being studied.

Diagnosis of Powassan virus infection is largely serologic by detection of viral-specific immunoglobulin M antibodies followed by confirmatory neutralization antibody tests on the serum and/or cerebrospinal fluid.¹ Rarely, detection of nucleic acid by reverse transcription polymerase chain reaction or antigen by immunohistochemistry may be useful.¹ Cerebrospinal fluid analysis may show a lymphocytic or early neutrophilic pleocytosis with normal glucose.^{1,6} Magnetic resonance imaging of the brain may show T2/fluid-attenuation inversion recovery lesions particularly in the deep gray matter, brain stem, and/or cerebellum without significant enhancement, though this may be variable.⁶ Pathologically, there may be focal perivascular and parenchymal inflammation consisting of lymphocytes and monocytes.³

Treatment of Powassan virus disease is largely supportive with emphasis on seizure prevention and management of any cerebral edema.^{1,3,6} No vaccine is currently available. Fortunately, infection is largely preventable through tick bite prevention: using insect repellent, wearing long-sleeved shirts and pants, avoiding brushy areas where ticks quest, and performing tick checks after being outdoors.^{1,3,5}

Neurologists should suspect Powassan virus disease in those with meningoencephalitis from spring through fall, particularly if they have had tick exposure in endemic areas.^{1,5} Suspected cases should be reported to state or local health

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departments who can often facilitate both testing and reporting.

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