

West Nile syndrome

West Nile virus is a *flavivirus* that belongs to the serocomplex of the Japanese encephalitis^[1] virus. It is therefore a single-stranded **RNA virus**.

Transmission

Birds are the reservoir of this disease. The transmission occurs by a **bite of the Culex mosquito** (transmission by Aedes and Anopheles mosquitoes has also been recorded). After biting an infected bird, it can spread the virus among birds or transmit it to humans and other mammals after about a week. However, mammals are “dead end” hosts and the virus can no longer spread from their blood. Less common routes of transmission are blood transfusions, organ transplants, and mother-to-child transmission (transplacentally, during childbirth and breastfeeding).^[2]

Epidemiology

The disease was first described in Uganda (West Nile Province) in 1937. However, it soon spread further into the world, and after World War II, it was reported in Asia. Eventually, it also appeared in southern and eastern Europe and Australia. And finally, in 1999, it expanded from Israel to North America.^[3] The incubation period is 3–14 days.^[4]

Symptoms

In 80% of the infected, the disease is asymptomatic, in 20%, the disease manifests as a **fever with headache and joint pain, vomiting, diarrhea or rash**. Only 1% of those infected develop severe encephalitis or meningitis (headache, fever, stiff neck, disorientation, coma, convulsions or paralysis). **Patients over the age of 50** with cancer, diabetes, hypertension or kidney failure are **at risk** of complications. About 10% of patients with neurological manifestations die of the disease.^[5]

Prevention, treatment and diagnosis

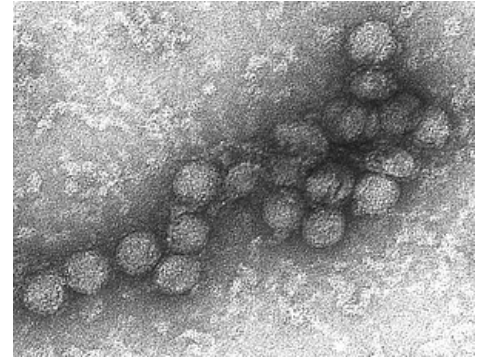
There is no vaccine or specific treatment. Treatment is only symptomatic and consists of pain treatment and rehydration.^[6] In cerebrospinal fluid, there is a serous finding with a cellularization of 150–200/mm³ and a protein level of up to 1 g/l. Magnetic resonance imaging is also used in diagnostics, with 50% chance of NMR showing an uncharacteristic finding in the white matter of the hemispheres, thalamus, basal ganglia and upper trunk. Half of the patients with encephalitis have basal ganglia disorders (tremor, myoclonus, Parkinson's syndrome).^[7] The virus can be diagnosed serologically (IgM and IgG by ELISA) or by PCR.^[8] IgM antibodies remain in the body in 20% of cases for 18 months or more. IgG antibodies persist in the body for years.

Links

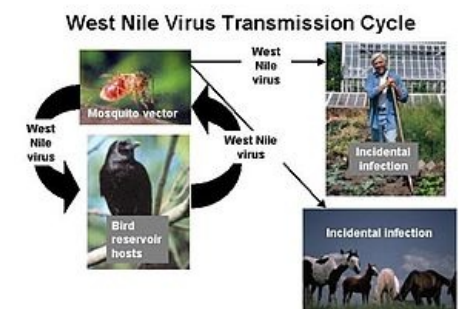
Reference

1. <https://web.archive.org/web/20160331222721/http://zdravi.e15.cz/clanek/priloha-lekarske-listy/aktuality-v-oblasti-neuroinfekci-451459>
2. <http://www.cdc.gov/westnile/transmission/>
3. <http://www.cdc.gov/westnile/transmission/>
4. <http://www.who.int/mediacentre/factsheets/fs354/en/>
5. <http://www.who.int/mediacentre/factsheets/fs354/en/>
6. <http://www.cdc.gov/westnile/symptoms/>
7. <https://web.archive.org/web/20160331222721/http://zdravi.e15.cz/clanek/priloha-lekarske-listy/aktuality-v-oblasti-neuroinfekci-451459>
8. <http://www.who.int/mediacentre/factsheets/fs354/en/>

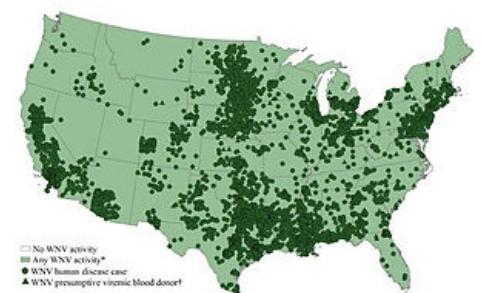
References



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West Nile virus cycle



West Nile virus in USA in 2012

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- GÖPFERTOVÁ, Dana – PAZDIORA, Petr. *Epidemiologie : (obecná a speciální epidemiologie infekčních nemocí)*. 1. edition. Karolinum, 2006. ISBN 80-246-1232-1.

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