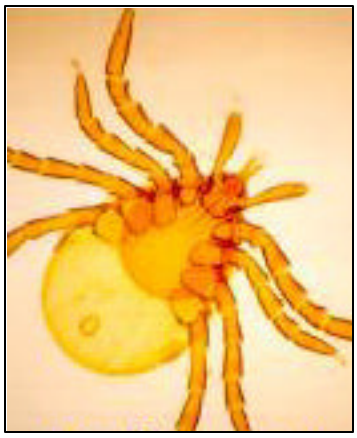
Tick-borne Encephalitis

Fact Sheet

What is tick-borne encephalitis?

Tick-borne encephalitis, or TBE, is a human viral infectious disease involving the central nervous system. The disease is most often manifest as meningitis (inflammation of the membrane that surrounds the brain and spinal cord), encephalitis (inflammation of the brain), or meningoencephalitis (inflammation of both the brain and meninges). Although TBE is most commonly recognized as a neurologic disease, mild febrile illnesses can also occur. Long-lasting or permanent neuropsychiatric sequelae are observed in 10-20% of infected patients.



Tick image of Ixodes species.

What causes tick-borne encephalitis?

TBE is caused by tick-borne encephalitis virus (TBEV), a member of the family Flaviviridae, that was initially isolated in 1937. A closely related virus in Far Eastern Eurasia, Russian spring-summer encephalitis virus (RSSEV), is responsible for a similar disease with a more severe clinical course.

How is TBEV spread, and how do humans become infected?

Ticks act as both the vector and reservoir for TBEV. The main hosts are small rodents, with humans being accidental hosts. Large animals are feeding hosts for the ticks, but do not play a role in maintenance of the virus. The virus can chronically infect ticks and is transmitted both transtadially (from larva to nymph to adult ticks) and transovarially (from adult female tick through eggs). TBE cases occur during the highest period of tick activity (between April and November), when humans are infected in rural areas through tick bites. Infection also may follow consumption of raw milk from goats, sheep, or cows. Laboratory infections were common before the use of vaccines and availability of biosafety precautions to prevent exposure to infectious aerosols. Person-to-person transmission has not been reported. Vertical transmission from an infected mother to fetus has occurred.

Where is the disease found?

TBE is an important infectious disease of in many parts of Europe, the former Soviet Union, and Asia, corresponding to the distribution of the ixodid tick reservoir. The annual number of cases (incidence) varies from year to year, but several thousand are reported annually, despite historical under-reporting of this disease.

What are the symptoms of TBE?

The incubation period of TBE is usually between 7 and 14 days and is asymptomatic. Shorter incubation times have been reported after milk-borne exposure. A characteristic biphasic febrile illness follows, with an initial phase that lasts 2 to 4 days and corresponds to the viremic phase. It is non-specific with symptoms that may include fever, malaise, anorexia, muscle aches, headache, nausea, and/or vomiting. After about 8 days of remission, the second phase of the disease occurs in 20 to 30% of patients and involves the central nervous system with symptoms of meningitis (e.g., fever, headache, and a stiff neck) or encephalitis (e.g., drowsiness, confusion, sensory disturbances, and/or motor abnormalities such as paralysis) or meningoencephalitis. In contrast to RSSE, TBE is more severe in adults than in children.

During the first phase of the disease, the most common laboratory abnormalities are a low white blood cell count (leukopenia) and a low platelet count (thrombocytopenia). Liver enzymes in the serum may also be mildly elevated. After the onset of neurologic disease during the second phase, an increase in the number of white blood cells in the blood and the cerebrospinal fluid (CSF) is usually found. Virus can be isolated from the blood during the first phase of the disease. Specific diagnosis usually depends on detection of specific IgM in either blood or CSF, usually appearing later, during the second phase of the disease.

Are there any complications after recovery?
In approximately two-thirds of patients infected with the TBE virus, only the early (viremic) phase is seen. In the remaining third, patients experience either the typical biphasic course of the disease or a clinical illness that begins with the second (neurologic) phase. The convalescent period can be long and the incidence of sequelae may vary between 30 and 60%, with long-term or even permanent neurologic symptoms. Neuropsychiatric sequelae have been report in 10-20% of patients.

Is the disease ever fatal?
Yes, but only rarely. In general, mortality is 1% to 2%, with deaths occurring 5 to 7 days after the onset of neurologic signs.

How is TBE treated?
There is no specific drug therapy for TBE. Meningitis, encephalitis, or meningoencephalitis require hospitalization and supportive care based on syndrome severity. Anti-inflammatory drugs, such as corticosteroids, may be considered under specific circumstances for symptomatic relief. Intubation and ventilatory support may be necessary.

Who is at risk for TBEV infection?
In disease endemic areas, people with recreational or occupational exposure to rural or outdoor settings (e.g., hunters, campers, forest workers, farmers) are potentially at risk for infection by contact with the infected ticks. Furthermore, as tourism expands, travel to areas of endemicity broadens the definition of who is at risk for TBE infection.

How can TBEV infections be prevented?
Like other tick-borne infectious diseases, TBEV infection can be prevented by using insect repellents and protective clothing to prevent tick bites. A vaccine is available in some disease endemic areas (though not currently in the United States); however, adverse vaccine-reactions in children limit the utility of the product.

Other related viruses.
The family Flaviviridae includes other tick-borne viruses affecting humans and these viruses are closely related to TBEV and RSSEV, such as Omsk hemorrhagic fever virus in Siberia and Kyasanur Forest disease virus in India. Louping ill virus (United Kingdom) is a member of this family; it cases disease primarily in sheep, and has been reported as a cause of a TBE-like illness in laboratory workers and persons at risk for contact with sick sheep (e.g., veterinarians, butchers).

Suggested Reading

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