European Centre for Disease Prevention and Control

Factsheet about tick-borne encephalitis (TBE)

factsheet

Tick-borne encephalitis (TBE) is a human viral infectious disease involving the central nervous system, and occurring in many parts of Europe and Asia. The virus is transmitted by the bite of infected ticks, found in woodland habitats.

TBE is most often manifested as a two-phased illness. The first phase is associated with symptoms like fever, fatigue, headache, muscular ache and nausea. The second phase involves the neurological system with symptoms of meningitis (inflammation of the membrane that surrounds the brain and spinal cord) and/or encephalitis (inflammation of the brain).

Like other tick-borne infectious diseases, the risk from TBE can be reduced by using insect repellents and protective clothing to prevent tick bites. A vaccine is available in some disease endemic areas.

1. Pathogen

Tickborne encephalitis (TBE) is a viral infectious disease that attacks the central nervous system and can result in long-term neurological symptoms, and even death. Tickborne encephalitis is caused by a virus (*Flavivirus* genus, family Flaviviridae) which includes three subtypes:

1. European subtype, transmitted by *Ixodes ricinus* ticks, endemic in rural and forested areas of central, eastern and northern Europe;
2. Far eastern subtype, transmitted mainly by *I. persulcatus*, endemic in far-eastern Russia and in forested regions of China and Japan; and
3. Siberian subtype, transmitted by *I. persulcatus*, endemic in Urals region, Siberia and far-eastern Russia, and also in some areas in north-eastern Europe.

Tickborne encephalitis has become a growing public health challenge in Europe and other parts of the world. The number of human cases of TBE in all endemic regions of Europe has increased by almost 400% in the last 30 years; the risk areas have spread and new foci have been discovered.

2. Clinical features and sequelae

The incubation period of TBE is seven days on average, but incubation of up to 28 days has been described. The incubation after foodborne infection is usually shorter, around four days.

Approximately two-thirds of human TBE virus infections are non-symptomatic. In clinical cases, TBE often has a biphasic course. The first viraemic phase lasts approximately five (range 2–10) days, and is associated with non-specific symptoms (fever, fatigue, headache, myalgia, nausea). This phase is followed by an asymptomatic interval lasting seven (range 1–33) days that precedes the second phase, when the central nervous system is involved (meningitis, meningoencephalitis, myelitis, paralysis, radiculitis).

The European subtype is associated with milder disease, with 20–30% of patients experiencing the second phase, mortality rates of 0.5–2%, and severe neurological sequelae in up to 10% of patients. In children, the second phase of illness is usually limited to meningitis, whereas adults older than 40 years are at increased risk of developing encephalitis, with higher mortality and long-lasting sequelae in those over the age of 60.

The far eastern subtype is associated with more severe disease: monophasic illness, with no asymptomatic interval preceding the onset of neurological disease, mortality rates of up to 35%, and higher rates of severe neurological sequelae.

The Siberian subtype is associated with a less severe disease (fatality rate of 1–3%), with a tendency for patients to develop chronic or extremely prolonged infections.

3. Transmission

Reservoir

Competent reservoir hosts of TBE virus are mainly small rodents (voles, mice), but also insectivores and carnivores. Indicator hosts supporting virus circulation indirectly by enabling tick multiplication include different species of wild and domestic mammals (e.g., foxes, bats, hares, deer, wild boar, sheep, cattle, goats, dogs). Humans are incidental and dead-end hosts.

Transmission mode

Tickborne encephalitis virus is transmitted by the bite of infected ticks. Humans may acquire infection by consumption of infected unpasteurised dairy products. Tickborne encephalitis virus is not directly transmitted from human to human, apart from the possibility of vertical transmission from an infected mother to the foetus. Laboratory accidents from needle-stick injuries or associated with aerosol infection have been reported.

Infected ticks can be found in woodland habitats—deciduous forests and transition zones between forests and grasslands. When infected, ticks can transmit the virus throughout their life (mainly nymphs and adults). Tick activity and life cycle depend on climatic factors (temperature, soil moisture and relative humidity). Wet summers and mild winters tend to increase tick population density. In central Europe, two peaks of activity of *I. ricinus* have been observed in April/May and in September/October. A single summer peak has been detected in colder regions of northern Europe and in mountain regions. Questing ticks are found mainly on low-lying vegetation. Sporadic cases during the cold season are also reported.

Risk groups

In endemic areas, people with recreational or occupational outdoor activities (e.g. hunting, fishing, camping, collecting mushrooms and berries, forestry, farming, military training) are potentially at risk of infection by contact with infected ticks.

4. Prevention measures

Tickborne encephalitis virus infection can be prevented by avoiding tick bites through the following methods:

1. vaccination against TBE (inactivated vaccine) is considered to be the most effective means of preventing TBE in endemic countries;
2. application of tick repellents;
3. wearing protective clothing, with long sleeves and long trousers tucked into socks treated with an appropriate insecticide
4. inspecting the body for ticks after outdoor activities and removing ticks with tweezers or forceps; and
5. avoiding consumption of unpasteurised dairy products in risk areas.

Diagnosis

The diagnosis of TBE is based on the detection of specific IgM antibodies in cerebrospinal fluid (intrathecal production) and/or serum, mainly by ELISA. Tickborne encephalitis antibodies appear 0–6 days after onset and are usually detected when neurological symptoms are present. Specific IgM antibodies can persist for up to 10 months in vaccinees or individuals who acquired the infection naturally; IgG antibody cross-reaction is possibly observed with other flaviviruses. Detection by PCR methods could be valuable for an early differential diagnosis of TBE.

5. Management and treatment

There is no specific antiviral therapy for TBE. Treatment relies on supportive management. Meningitis, encephalitis or meningomyelitis require hospitalisation and supportive care based on syndrome severity.

6. Key areas of uncertainty

There is a need for standardisation of case definitions, laboratory diagnosis, reporting and documentation of the disease. The reporting of TBE cases varies among countries making it difficult to compare surveillance data; TBE is not a notifiable disease at the EU level.

Note: The information contained in this factsheet is intended for the purpose of general information and should not be used as a substitute for the individual expertise and judgement of healthcare professionals.

7. References

CDC fact sheet available online: http://wwwnc.cdc.gov/travel/yellowbook/2010/chapter-5/tickborne- (http://wwwnc.cdc.gov/travel/yellowbook/2010/chapter-5/tickborne-) encephalitis.aspx (http://wwwnc.cdc.gov/travel/yellowbook/2010/chapter-5/tickborne-)

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