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Tick-Borne Encephalitis the situation in Poland 2024- concern that warrants attention?

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Abstract

Introduction

Tick-borne encephalitis (TBE) is a zoonotic infectious disease and the leading cause of viral meningitis in Poland. In recent years, there has been a marked increase in TBE cases, a trend possibly linked to global climate changes that are facilitating the spread of its primary vector, the Ixodes Ricinus tick species.

Aim of the Study

This paper aims to review the current understanding of tick-borne encephalitis and emphasize the importance of preventive measures.

Materials and Methods

This review compiles data from various sources, including recent epidemiological studies, clinical reports, and public health records. It examines the clinical progression of TBE, from the initial viremic phase to the possible development of severe neurological symptoms. Epidemiological data are analyzed to identify trends in TBE incidence, with a particular focus on how climate change affects the distribution and activity of tick populations.

Conclusions

The review underscores that TBE is an escalating public health issue in Poland, with incidence rates increasing alongside climate-induced changes in tick habitats. Effective prevention, particularly through vaccination, is essential for reducing the impact of TBE. However, despite the availability of effective vaccines, vaccination rates remain low in Poland. This highlights the need for improved public health efforts to promote vaccination and educate at-risk populations. Addressing the TBE threat requires a comprehensive strategy that includes better surveillance, preventive measures, and strong public health initiatives to protect against this increasingly common disease.

Key words: tick-borne encephalitis, tick-borne encephalitis virus, vaccination, epidemiology, Poland, global warming

Introduction and aim of study

Tick-borne encephalitis (TBE) is a zoonotic infectious disease which is the most common cause of viral meningitis in Poland, accounting for up to 40% of all reported cases[1]. In

recent years, there has been a significant increase in the incidence of this disease, which may be related to global climate changes, specifically the warming climate leading to the expansion of the primary vector of the disease, the tick species: Ixodes Ricinus species[12]. The aim of this paper is to review the current knowledge about this disease and to highlight the importance of prevention.

Description of the state of Knowledge

Etiology

Tick-borne encephalitis is a disease caused by the tick-borne encephalitis virus (TBEV), an RNA Flavivirus belonging to the Flaviviridae family [2,7]. There are three subtypes of the virus: European, Siberian, and Far Eastern[4]. The most common subtype in Poland is the European subtype, although each of the mentioned subtypes occurs in Eastern Europe[22]. The main vector of the TBE virus is the tick of the Ixodes Ricinus species, through which it is transmitted to hosts such as rodents, cattle, and sheep[4,23]. Rodents play the most important role in virus replication because they are the primary hosts of ticks, and the maintenance of an adequate tick population depends on their numbers. Also, in rodent bodies, the virus replicates asymptomatically, infecting subsequent ticks feeding on them.[4].TBEV is spread through the saliva of a tick that has been infected, typically within a few minutes following the tick bite[4,7], unlike other tick-borne disease, Lyme disease (which occurs within several hours). This is due to the presence of the virus in the tick's salivary glands rather than in the digestive tract, as is the case with the bacterium Borrelia[4]. This means that even early and proper tick removal does not protect against TBE virus infection[7]. Another way the TBE virus spreads is through the foodborne route. Thanks to its ability to survive in the acidic environment of the stomach, the virus can be transmitted through unpasteurized milk, mainly goat and sheep milk, less frequently cow milk. The pasteurization process leads to virus inactivation[5,6,23]. Individual cases of infection have also been described through blood transfusion and breastfeeding via mother's milk[7]. The virus initially reproduces at the injection site, primarily in the dendritic cells of the skin. These cells then transmit the virus to nearby lymph nodes, where lymphocytes become infected, leading to the systemic spread of the infection. After replicating in lymphoid tissues, the TBEV enters the bloodstream and crosses the blood-brain barrier to reach the brain[22]. Infection can affect any part of the central nervous system (CNS), but most commonly occurs within the brainstem, basal ganglia, and spinal cord[23,25]. The main cause of CNS damage during infection is the body's immune reaction mediated by

CD8+ T cells, leading to neuronal damage through inflammatory reactions[22]. Recovery from infection provides lasting immunity and leaves behind specific protective IgG antibodies[25].

Course of Disease

Upon the entry of the virus into the body, the incubation period lasts 4-28 days following a tick bite and 3-4 days through the foodborne route[8]. Infection may manifest symptomatically or asymptomatically, with about one-third of cases being asymptomatic[5]. Infection may have a monophasic or more common biphasic course in 72–87% of patients[7] , with neurological symptoms characteristic of the European subtype virus predominant in Poland[4,11]. In the symptomatic biphasic course, the disease is divided into:

-The viremic phase, lasting 1-8 days, characterized by flu-like symptoms such as fever, nausea, vomiting, apathy, joint and muscle pain[7,9], with leukopenia and thrombocytopenia occurring in 70% of patients, along with increased liver aminotransferase activity[10,7].

-The neurological phase, associated with virus invasion of the central nervous system (CNS), most commonly presenting with fever, headache, insomnia, muscle pain, and neurological symptoms including altered consciousness, ataxia, limb weakness, and cranial nerve palsies. The summary of neurological symptoms is in table 3. Meningeal symptoms are observed in about 10-30% of patients[4,10]. This phase is characterized by Leukocytosis, elevated C-reactive protein (CRP) levels, and elevated OB[4,10]. Laboratory tests also show characteristic changes in cerebrospinal fluid, including moderate pleocytosis and elevated albumin levels in two-thirds of all patients[7]. The neurological phase most commonly presents as meningitis, less frequently as encephalitis, or meningoencephalitis, and rarely as myelitis[2].Up to 18% of individuals show abnormalities on MRI, commonly affecting areas such as the thalamus, cerebellum, brainstem, and nucleus caudatus. Abnormalities are also detected in 77% of cases through EEG analysis. However, neither MRI nor EEG abnormalities are not diagnostic [7].

The severity of TBE symptoms depends on age, with much more severe disease progression observed in elderly individuals and those with chronic diseases or undergoing oncological treatment[4,11]. In contrast, children often experience a milder course of the disease, although about one-third of them may develop a severe form leading to death. Serious complications occur in children about 10 times less frequently than in adults[4,11,12]. TBE can lead to numerous complications, significantly reducing patients' quality of life and causing lifestyle

changes[11,12], more common in patients with severe disease progression[4,12]. One of the most common complications is Post-Encephalitic Syndrome, affecting 40-50% of patients who have recovered from acute TBE[11]. Common symptoms of this condition include cognitive impairment, memory disturbances, irritability, sleep rhythm disturbances, visual and auditory impairments, balance and coordination disturbances, limb weakness or paralysis, and neuropsychological disorders such as depression, learning and work difficulties, concentration problems, memory and learning difficulties, and changes in eating habits[12].

Epidemiology

TBE is a seasonal disease that coincides with the activity period of the main vector, Ixodes Ricinus ticks, which peaks from April to October[12]. While endemic to Central-Eastern Europe, TBE's range now encompasses almost the entire continent[13]. According to the latest ECDC report in 2021, 3027 cases were reported across 25 European countries[13]. In recent years, there has been a significant increase in cases in Poland as well. In 2023, there were 659 confirmed cases of TBE, representing a 48% increase from 2022, when there were 445 confirmed cases. Detailed data regarding TBE cases in Poland from the Sanitary-Epidemiological Stations is presented in Figure 1[14].

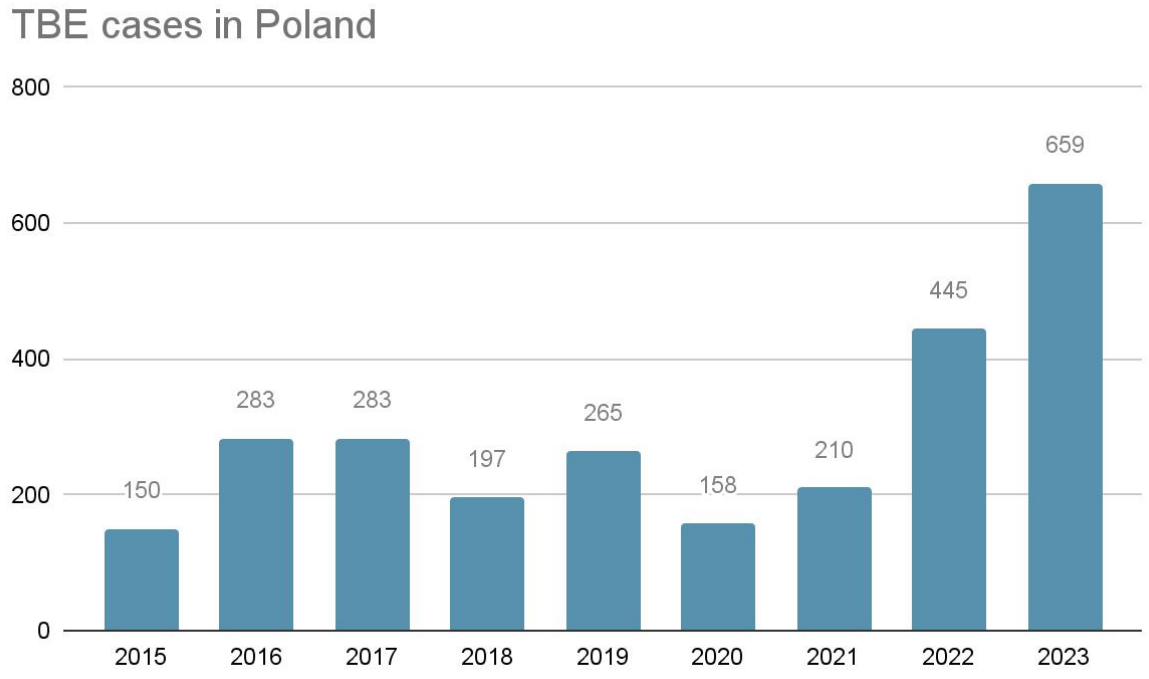

Figure 1. The number of reported TBE cases to epidemiological surveillance in Poland in the years 2015-2023. Source: NIPH-NIH.

The highest number of cases is detected in the Podlaskie and Warmian-Masurian Voivodeships, but other regions have also seen an increase in infections, which is in line with the overall European trend of rising infection rates and expanding tick habitats. Discrepancies in case numbers among different voivodeships, especially the low number of detected TBE infections in southwestern and southeastern regions, where more cases of unspecified etiology of CNS inflammation are reported. In 2023 36% of viral encephalitis cases had no confirmed etiology[14]. This may be due to diagnostic limitations compared to endemic regions in eastern Poland[12]. Figure 2 shows the average incidence of TBE per 100,000 inhabitants from 2015 to 2019 by voivodeship.

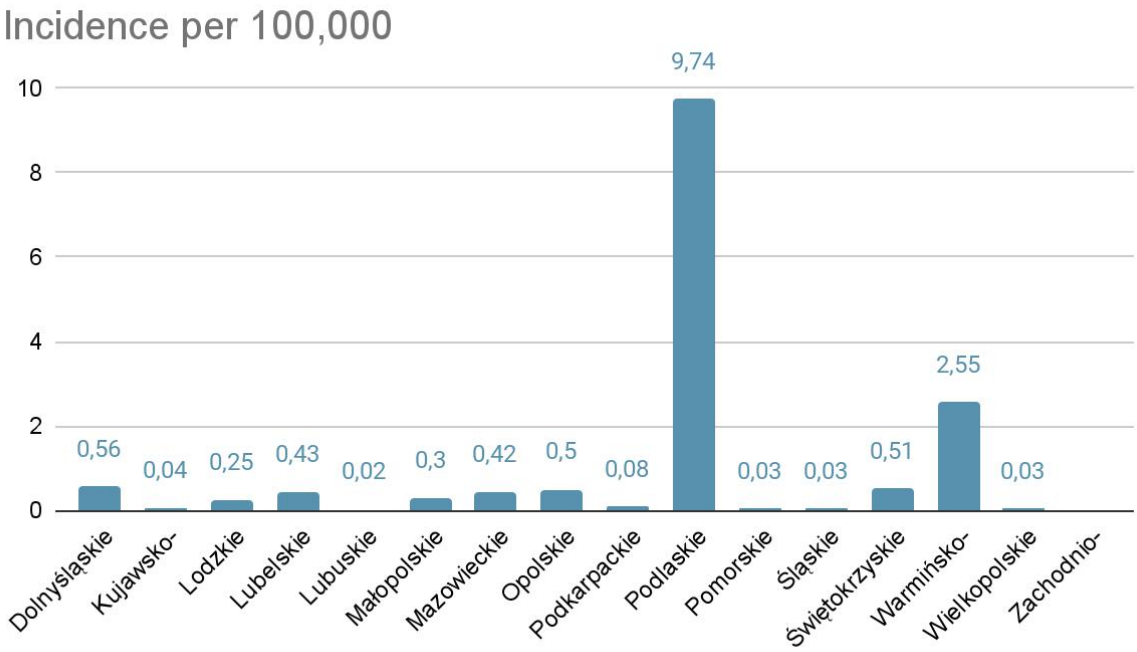

Figure 2. The average incidence of TBE per 100,000 inhabitants by voivodeships in the years 2015-2019. Source: NIPH-NIH

The impact of climate changes on the prevalence of TBE

Climate change is one of the various factors contributing to the increase in TBE cases, as confirmed by several studies conducted in Europe [15]. Climate change has been linked to the spread of I. Ricinus ticks and the TBE virus to higher elevations in the Czech Republic[28] and Slovakia [26], as well as to more northern regions in Sweden[27]. This expansion is

evident at both the altitudinal and latitudinal limits of the virus's range. Climate change also indirectly affects tick habitats and human behavior, such as increased outdoor activities on warm days and the growth of vegetation providing shelter for ticks. Additionally, increased rainfall promotes fungal growth in forests, encouraging people to forage for mushrooms and exposing them to ticks bites. Environmental changes also contribute to an increase in rodent populations, the primary hosts of ticks and reservoirs of the TBE virus[15]. There are many models describing the future impact of climate change on the occurrence of tick-borne encephalitis (TBE) [15]. For example, models [29,30] project that the TBE virus will likely increase in higher altitudes and latitudes, consistent with empirical observations. However, they also indicate a potential decline of TBE virus in certain regions of its current main range, such as Switzerland and Hungary. This decline is attributed to climate change affecting the timing of different stages of the tick species Ixodes Ricinus, thus disrupting the transmission cycle among rodents, the primary hosts facilitating transmission between ticks through non-viremic means. Additionally, climate change may have further impacts beyond range shifts and incidence rate alterations. For instance, the prevalence and incidence of TBE virus display significant year-to-year fluctuations due to interactions among host population cycles, host immunity, and longevity. Paper [31] illustrated cyclical oscillations of TBE virus across large areas of Europe, including Sweden, Germany, the Czech Republic, Slovenia, Austria, and Italy. Moreover, climate change is shown to modify the duration of these cycles, with warmer conditions elongating the cycle frequency by extending host longevity, thereby slowing the variation in host immunity and influencing the level of TBE virus circulating within the host and tick populations[15].

Prevention and Treatment

Prevention of TBE can be divided into specific and nonspecific measures[4]. Nonspecific methods include avoiding areas where ticks are most commonly found, using insect repellents and insecticides, wearing appropriate clothing covering the body during outdoor activities, and conducting thorough body and clothing checks after activities to find ticks[4,16]. The most effective tick repellents contain DEET (N,N-Diethyl-meta-toluamide)[16]. Non-specific prevention against the transmission of the virus via foodborne route involves avoiding consumption of unpasteurized milk in endemic areas[2,4,16] The most effective method is specific prevention through vaccination[4,7]. In Poland currently (2024), there are two vaccine preparations available for children and adults: "Encepur" and "FSME-Immun". Both

require three primary doses within a year and booster doses every 3-5 years19]. According to the Polish Vaccination Program for 2024, vaccinations are particularly recommended for people in endemic areas, especially those who spend a lot of time outdoors, such as runners, dog owners, mushroom pickers, tourists, hunters, as well as professional groups such as forestry workers, military personnel, firefighters, border guards, and farmers [21]. The vaccine, when all doses are administered, demonstrates high efficacy confirmed by studies conducted in Europe, such as in Germany in endemic areas of Bavaria and Baden-Württemberg, where efficacy was 93.9%, and in Latvia, where efficacy was 98.6%[19]. Another example confirming the effectiveness of vaccinations is Austria, where the population's vaccination rate is estimated to be >80%, and the incidence of TBE decreased from 5.3/100,000 people per year before the introduction of vaccinations to 1-2 cases/100,000 people per year[20]. In Poland, however, the vaccination rate is relatively low; the latest data from 2022 reported 83,020 vaccinations. More detailed statistics regarding vaccinations in Poland are shown in Figure 3 [18].

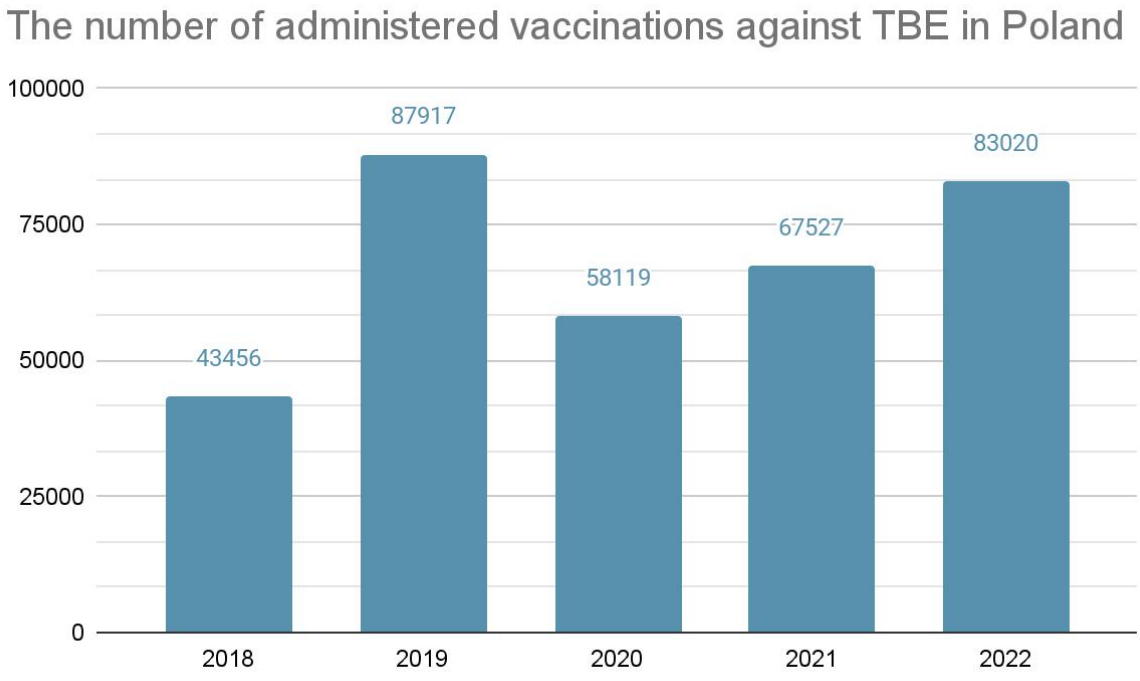

Figure 3. The number of administered vaccinations against TBE in Poland Source:NIPH-NIH

Treatment

There are no registered antiviral drugs for the treatment of TBE[4,32]. When infection occurs, treatment for TBE is symptomatic. In milder cases, it involves using antipyretic, analgesic, antiemetic, anti-edematous medications, and correcting acid-base and water-electrolyte imbalances[12,32].Cases with severe symptoms require treatment within intensive care units. In a prospective study conducted in Germany, 12% of patients required treatment in an intensive care unit, and 5% needed assisted ventilation[33]. Cerebral edema, a complication of acute viral encephalitis, can worsen the prognosis of patients. Treatment often involves intravenous mannitol or steroids to reduce intracranial pressure. Mannitol shifts fluid from the brain to the bloodstream, increasing circulation and reducing intracranial pressure. Dexamethasone has shown potential in reducing brain edema in viral encephalitis. In the Baltics and Eastern Europe, corticosteroids are commonly used for TBE, based on perceived clinical improvement[32]. However, there are also studies showing the adverse effects of steroids, which prolong the hospital stay of patients. Among Polish patients diagnosed with tick-borne encephalitis (TBE), dexamethasone was administered to 54.8% of those with meningitis, 69.6% with meningoencephalitis, and 78.3% with meningoencephalomyelitis. Notably, in this investigation, prolonged hospital stays were observed solely in individuals who received dexamethasone for more than 10 days[34]. Possibly, corticosteroid therapy demonstrates efficacy in specific cases, but until randomized studies provide more data, corticosteroids cannot be recommended as standard treatment[32,34].

Summary

Tick-borne encephalitis (TBE) presents a significant public health challenge in Poland, with a notable increase in reported cases in recent years. This rise aligns with broader European trends, likely influenced by various factors, including climate change. The expansion of the tick vector's habitat due to warming temperatures has facilitated the spread of TBE to new regions, contributing to the surge in infections. Additionally, environmental changes have led to shifts in human behavior, increasing the risk of exposure to infected ticks.Understanding the etiology, course of disease, and epidemiology of TBE is crucial for effective prevention and management. Prevention remains the cornerstone of TBE control. Non-specific measures such as avoiding tick habitats and using repellents can mitigate exposure, while specific prevention through vaccination offers the most effective protection. Vaccination programs targeting high-risk populations, such as outdoor enthusiasts and certain occupational groups,

are essential for reducing TBE incidence. However, vaccination rates in Poland remain suboptimal, highlighting the need for improved public awareness and access to vaccines.

To sum up, addressing the growing threat of TBE requires a multifaceted approach encompassing surveillance, prevention, and treatment strategies. Collaborative efforts between healthcare providers, policymakers, and the public are essential to mitigate the impact of TBE and safeguard community health in Poland and beyond.

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