SYSŁO, Oliwia, MARSZAŁ, Wiktoria, CZYZ , Sandra and SARACEN, Nina. Pediatric Neuroborreliosis: Current Challenges in Diagnosis and Treatment of Lyme Disease. ***Journal of Education, Health and Sport*****. 2025;86:65778. eISSN 2391-8306.**
https://doi.org/10.12775/JEHS.2025.86.65778
https://apcz.umk.pl/JEHS/article/view/65778

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 26.11.2025. Revised: 08.12.2025. Accepted: 08.12.2025. Published: 19.12.2025.

Pediatric Neuroborreliosis: Current Challenges in Diagnosis and Treatment of Lyme Disease

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ABSTRACT:

Introduction and Objective: Lyme disease is an infection caused by Gram-negative, microaerophilic spirochetes of the *Borrelia* genus, transmitted to humans through the bite of an infected tick, which serves as the vector of this disease. It is one of the most frequently diagnosed tick-borne illnesses worldwide, with a rising incidence rate. Notably, children represent an increasingly affected population, emphasizing the need for focused attention on pediatric manifestations and management.

Brief Overview of Current Knowledge - Lyme disease progresses through three clinical stages: early localized, early disseminated, and late stage. Each phase presents with characteristic clinical manifestations.

- The early localized stage typically features erythema migrans, most often appearing on the scalp, neck, shoulders, legs, or lower back.
- The early disseminated stage is marked by flu-like symptoms, lymphadenopathy, arthralgia, myalgia, cranial nerve palsies - most frequently involving the facial nerve (cranial nerve VII) - ocular involvement, and lymphocytic meningitis.
- The late stage is characterized predominantly by pauciarticular arthritis, affecting large joints - especially the knees. In some cases, cardiac manifestations may occur, such as conduction abnormalities, myocarditis, or pericarditis.

This progression underscores the multisystemic nature of Lyme disease and highlights the importance of early recognition and treatment to prevent long-term complications.

Summary: Neuroborreliosis, a complication of Lyme disease, is characterized in children by neurological symptoms such as facial nerve palsy, ophthalmic complications, and lymphocytic meningitis. Recognizing these manifestations is crucial, as they can guide diagnosis, indicate disease stage, and help assess severity. Accurate diagnosis relies on a combination of thorough medical history, serological testing, and cerebrospinal fluid analysis, while treatment primarily involves targeted antibiotic therapy. Although most patients recover completely, a subset may experience persistent neurological or cognitive symptoms, emphasizing the need for careful follow-up. This review aims to provide healthcare professionals with an evidence-based overview of neuroborreliosis in children, highlighting key diagnostic features, management strategies, and areas where further research is needed to improve patient outcomes.

Keywords: Borrelia; Children; Lyme disease; Lyme neuroborreliosis.

INTRODUCTION AND OBJECTIVE:

Lyme borreliosis (LB) is a multisystem infectious disease caused by spirochetes of the *Borrelia burgdorferi* sensu lato complex, transmitted by ticks of the genus *Ixodes* [1,2,3]. Since its discovery in 1975, it has become the most commonly reported vector-borne disease in both North America and Europe [4]. By 1989, Steere had described the etiology, vectors, animal reservoirs, clinical manifestations, pathogenesis, and treatment methods of Lyme borreliosis. Between 1988 and 1993, two animal models - murine and non-human primates - were developed, accurately reflecting the microbiological, clinical, immunological, and neuropathological aspects of the disease [5].

In Poland, Lyme borreliosis is the most frequently diagnosed zoonosis and represents a significant epidemiological challenge. Of the 19 genospecies currently identified within the *B. burgdorferi* sensu lato complex, nine have been recognized as pathogenic to humans: *Borrelia burgdorferi* sensu stricto, *Borrelia garinii*, *Borrelia afzelii*, *Borrelia bissettii*, *Borrelia spielmanii*, *Borrelia valaisiana*, *Borrelia lusitaniae*, *Borrelia bavariensis*, and *Borrelia kurtenbachii*[6].

In Europe, the predominant etiological agents of Lyme borreliosis are *B. garinii* and *B. afzelii*, while *B. burgdorferi* sensu stricto is less frequently encountered [7]. The individual *Borrelia* species exhibit distinct tissue tropisms, which account for the variability in clinical manifestations. *B. garinii* displays neurotropism and is most commonly associated with neuroborreliosis. In contrast, *B. afzelii* shows dermatotropism and is more frequently responsible for skin manifestations such as erythema migrans and acrodermatitis chronica atrophicans [8].

Pathogenesis of Lyme Borreliosis

Borrelia burgdorferi is a Gram-negative bacterium transmitted by ticks of the genus *Ixodes*, which enters the human body during the feeding process of an infected vector - primarily through its saliva or regurgitated gut contents [9,10]. A key factor in the pathogenesis of Lyme borreliosis is the expression of variable outer surface proteins (Osps) by *Borrelia* spirochetes, particularly OspA, OspB, and OspC. OspA facilitates bacterial survival within the tick's midgut, while OspC is critical for the migration of the pathogen to the salivary glands, a prerequisite for successful transmission to the vertebrate host. Upon entry into the host organism, these proteins interact with Toll-like receptors (TLRs) on immune cells, predominantly macrophages, thereby initiating a complex immune response involving both innate and adaptive mechanisms. This activation leads to the recruitment of inflammatory cells, including monocytes, T and B lymphocytes, and macrophages, which contribute to the localized release of pro-inflammatory mediators such as interleukins (IL-1, IL-6), tumor necrosis factor-alpha (TNF-α), chemokines, extracellular matrix metalloproteinases, and adhesion molecules. The cumulative effect of these mediators results in tissue inflammation and localized damage at the site of infection [11-14]. In the early phase of infection, the bacteria remain at the inoculation site, where they proliferate and trigger a localized inflammatory response, clinically manifested as the characteristic erythema migrans. Within a few days to several weeks, the spirochetes disseminate hematogenously and via the lymphatic system to distant organs, such as the liver, spleen, myocardium, joints, retina, bone marrow, and the central nervous system - with potential involvement of cerebrospinal fluid. When the musculoskeletal system is affected, the arthritic form of Lyme borreliosis may develop, with pathogenesis primarily driven by immunological mechanisms. This includes fibroblast activation and synovial membrane hyperplasia, resulting in chronic inflammation, cartilage degradation, and damage to periarticular tissues. The clinical picture may resemble that of rheumatoid arthritis [9-14].

Epidemiology:

Epidemiological data from Poland indicate the significant scale of the problem. Between 2015 and 2019, a total of 94,715 cases of Lyme borreliosis were reported, corresponding to an average incidence rate of 49.3 per 100,000 inhabitants. In 2016, the number of cases reached 20,857, and in the following years remained at a comparable level. The highest incidence among the pediatric population was observed in children aged 5–15 years [15,16]. Data from 2022 show a renewed increase in reported cases and hospitalizations, particularly in eastern regions, which may suggest a return to previous epidemiological trends. The seasonality of infections correlates with tick activity - most cases are diagnosed in the third and fourth quarters of the year [17]

A cohort study conducted in Poland in 2020 found that neuroborreliosis and tick-borne encephalitis were the second most common causes of central nervous system (CNS) infections in children, accounting for 22% of all aseptic meningitis cases. The impact of Lyme borreliosis on the CNS during developmental age may lead to long-term complications - such as concentration disorders, chronic fatigue, mood disturbances, and cognitive impairment - even despite appropriate treatment [18].

Due to its diverse symptoms and clinical course, Lyme borreliosis continues to pose a challenge to the healthcare system. The aim of this article is to classify and provide an overview of the clinical features of neurological manifestations associated with neuroborreliosis, and to explore their relationship with disease severity.

Clinical Manifestations of Lyme Borreliosis in Children

The clinical symptoms of Lyme borreliosis in children can be diverse and may involve multiple organ systems. One of the most characteristic manifestations of the early phase of Lyme disease is **erythema migrans**, which typically appears within 3 to 30 days following infection. It presents as a single (less commonly multiple), non-itchy, and painless skin lesion - initially as a red macule or papule that rapidly expands (to >5 cm in diameter), acquiring a characteristic ring-like appearance with central clearing. In a somewhat later phase of the disease - several months after infection, including during the winter season when Lyme disease is less frequently considered in differential diagnosis - **neuroborreliosis** may develop [15,16,19,47].

Neuroborreliosis may involve various structures of both the central and peripheral nervous systems. The clinical presentation of pediatric neuroborreliosis can be classified based on the time elapsed since infection.

In the **early disseminated stage** (within 6 months of infection), the most frequently observed manifestations include:

- Cranial nerve palsies, especially of the facial nerve (cranial nerve VII),
- Lymphocytic meningitis,
- Acute painful radiculopathies,
- Acute encephalitis or encephalomyelitis.

In contrast, the late stage (more than 6 months post-infection) is characterized by chronic changes, such as:

- Encephalopathy,
- Chronic polyradiculopathies,
- Chronic meningitis,

- Encephalomyelitis,
- Chronic cerebral vasculitis

In addition to neurological symptoms, patients may experience nonspecific complaints, such as:

- Chronic fatigue,
- Loss of appetite,
- Mood fluctuations.

During the acute disseminated phase of Lyme borreliosis, ophthalmologic complications may also occur, including visual disturbances [2,20-24]

Many researchers indicate that the most common clinical forms of neuroborreliosis in children include:

• Subacute lymphocytic meningitis
• Facial nerve palsy
• Bannwarth syndrome

There are also rare neurological presentations that do not fit clearly into the above classification. These may encompass a broad spectrum of symptoms and pose a diagnostic challenge. This section outlines the characteristics of pediatric neuroborreliosis, taking into account the frequency, duration of symptoms, and the clinical dynamics of disease progression [46,47].

1. Facial Nerve Palsy

Facial nerve (cranial nerve VII) palsy is the most common manifestation of neuroborreliosis in children. It typically occurs during the second stage of the disease, when Borrelia spirochetes disseminate to the nervous system. The clinical picture includes weakness of facial muscles, flattening of the nasolabial fold, drooping of the mouth corner, inability to close the eyelid (Bell's phenomenon), difficulty wrinkling the forehead, and facial asymmetry [25-28]. Associated symptoms may include paresthesia's, altered taste sensation in the anterior two-thirds of the tongue, hyperacusis, and either excessive tearing or dryness of the eye. Bilateral facial nerve palsy - rare in other conditions - may serve as an important diagnostic clue in pediatric neuroborreliosis. Symptoms can occur with a delay of several weeks after tick exposure and may develop in the absence of erythema migrans [14, 45]. Diagnosis is based on patient history, neurological examination, and confirmation of *Borrelia* spp. infection via serological testing and/or cerebrospinal fluid (CSF) analysis. Treatment involves antibiotic therapy, typically with ceftriaxone or doxycycline, along with supportive rehabilitation to aid nerve recovery [27-29].

2. Lymphocytic Meningitis (LM)

Neuroborreliosis may also present as aseptic meningitis, characterized by lymphocytic pleocytosis in CSF analysis. The disease often has a slow and non-specific onset, contributing to diagnostic difficulty. Common symptoms include fatigue, headache, photophobia, phonophobia, and neck stiffness, which is often less pronounced than in bacterial meningitis [30,31,32].

Borrelial meningitis is diagnosed in approximately 1% of children infected with *Borrelia* spp. [31]. CSF findings include elevated protein levels, normal or decreased glucose concentration, and the presence of plasma cells - suggestive of a local humoral immune response. A key diagnostic element is the detection of *Borrelia burgdorferi*-specific antibodies in CSF and evidence of intrathecal antibody synthesis, in accordance with EFNS criteria [3,32]. The disease typically follows a benign course, and most cases respond well to antibiotic therapy.

3. Bannwarth Syndrome

Bannwarth syndrome (also known as Garin-Bujadoux-Bannwarth syndrome) is one of the classic forms of early neuroborreliosis in Europe. It is characterized by a triad of symptoms: lymphocytic meningitis, cranial neuropathies, and painful, asymmetric polyradiculopathy [20,34,35]. Additional symptoms may include headaches, chronic fatigue, concentration difficulties, drowsiness, and both psychiatric and cognitive disturbances [20,33,41].

Neurological deficits develop in approximately 75% of patients within 1 to 4 weeks - most commonly manifesting as flaccid paresis and segmental sensory disturbances [33]. Differential diagnosis should include other conditions causing meningitis or root compression syndromes, such as herniated intervertebral discs [36,37].

Diagnosis, as with other forms of neuroborreliosis, relies on clinical evaluation, CSF analysis, and serological testing. Despite effective antibiotic therapy, some children may experience persistent, nonspecific symptoms such as fatigue, sleep disturbances, or concentration difficulties - collectively referred to as **post-treatment Lyme disease syndrome (PTLDS)**[24,46]. Accurate diagnosis of neuroborreliosis requires a thorough patient history - gathered both from caregivers and, when possible, from the child. Particular attention should be paid to reports of tick bites, the appearance of erythema migrans, and systemic symptoms such as fever, fatigue, or musculoskeletal pain. Neurological manifestations of Lyme borreliosis in children usually follow a subacute course and are frequently misdiagnosed as other conditions, such as viral meningitis (most often caused by enteroviruses), idiopathic facial nerve palsy, or migraine. While *Borrelia burgdorferi* infection occurs in both children and adults, the clinical course of the disease may vary with age.

In the diagnostic evaluation of Lyme neuroborreliosis, in addition to a thorough medical history and physical examination, laboratory investigations - particularly cerebrospinal fluid (CSF) analysis - are of critical importance. In suspected cases of central nervous system (CNS) involvement, measurement of inflammatory markers in the CSF is recommended. Among these, the chemokine CXCL13 (CSF-CXCL13) has emerged as a highly sensitive biomarker indicative of active CNS inflammation caused by *Borrelia* spp. infection [49].

The diagnosis of Lyme borreliosis typically adheres to the standard two-tiered testing (STTT) algorithm, which is endorsed in both the United States and several European countries. The initial step involves serological screening, most commonly by enzyme-linked immunosorbent assay (ELISA) or an equivalent immunoassay. If the screening result is positive or equivocal, a confirmatory immunoblot (Western blot) is performed to detect IgM and IgG antibodies against *Borrelia* antigens. Interpretation of the results follows established diagnostic guidelines and criteria [50].

It is important to emphasize that inappropriate use of serologic testing, particularly in children, may result in false-positive results. This can lead to unnecessary antibiotic treatment, which is especially problematic in the absence of clear clinical signs suggestive of infection. Therefore, Lyme disease diagnostics should always be preceded by a thorough clinical evaluation and an assessment of tick exposure risk factors [30,38,39,49,50].

According to the guidelines of the European Federation of Neurological Societies (EFNS), a confirmed diagnosis of neuroborreliosis requires fulfillment of all three of the following criteria:

1. Presence of neurological symptoms,
2. CSF pleocytosis,
3. Detection of *Borrelia burgdorferi*-specific intrathecally produced antibodies.

Meeting two out of the three criteria supports a possible diagnosis of neuroborreliosis [2,14,40]. Awareness of the nonspecific symptoms of neuroborreliosis in children is essential for timely and accurate diagnosis, allowing for early initiation of appropriate treatment.

Lyme Disease and Pregnancy

According to current knowledge, no studies have definitively demonstrated a link between the presence of Lyme disease during pregnancy and adverse neuropsychological outcomes in childhood or adolescence. Vertical transmission of *Borrelia burgdorferi* from mother to fetus is possible, with the most likely mechanism being hematogenous infection leading to placental involvement. This mechanism is analogous to that observed in congenital syphilis, caused by *Treponema pallidum*. Although a single case of *Borrelia* presence in breast milk has been reported, no significant risk of lactational transmission has been established. Therefore, breastfeeding remains recommended [42-44].

SUMMARY:

Neuroborreliosis, a well-recognized complication of Lyme disease, encompasses a broad spectrum of neurological manifestations, including cranial neuropathies such as facial nerve palsy, radiculitis, meningitis, and, in severe cases, encephalopathy. The clinical presentation may vary depending on patient age, immune status, and stage of infection, which often complicates prompt diagnosis. Accurate identification of neuroborreliosis requires a comprehensive approach, combining detailed medical history, serological testing for Borrelia-specific antibodies, and cerebrospinal fluid analysis to detect intrathecal antibody production or inflammatory markers. Neuroimaging, particularly MRI, may provide additional diagnostic support in atypical or complicated presentations. Management of neuroborreliosis relies on appropriately selected antibiotic therapy, guided by the type and severity of neurological involvement. Most patients experience complete recovery when treated promptly; however, a subset may develop persistent symptoms, including fatigue, cognitive difficulties, or neuropathic pain, underscoring the importance of long-term follow-up and supportive care strategies. This review highlights the significance of neurological manifestations not only as diagnostic indicators but also as potential markers of disease severity and prognosis. Enhancing clinician awareness of the clinical spectrum of neuroborreliosis can facilitate earlier recognition and more effective management of affected patients.

Despite advancements in diagnostics and therapeutics, critical gaps remain in understanding the pathophysiology, optimizing treatment regimens, and predicting long-term outcomes. Continued research is therefore essential to refine diagnostic criteria, improve therapeutic strategies, and mitigate the burden of persistent symptoms. Overall, this review aims to provide healthcare professionals, particularly those in neurology, pediatrics, and primary care, with an evidence-based synthesis of current knowledge on the recognition, diagnosis, and management of neuroborreliosis, ultimately supporting improved patient care and clinical outcomes.

Disclosure:
The authors declare that they have no financial or non-financial conflicts of interest that could be perceived as influencing the interpretation of the research findings or the content of this manuscript. This work was conducted indepedently without any external funding or support.

Author's contribution

Conceptualization: Oliwia Sysło; **Methodology:** Oliwia Sysło; **Software:** Nina Saracen, Sandra Czyż; **Validation:**Wiktoria Marszał; **Formal analysis:** Nina Saracen, Wiktoria Marszał; **Investigation:** Sandra Czyż; **Resources:** Oliwia Sysło, Wiktoria Marszał; **Data curation:** Nina Saracen; **Writing – original draft:** Wiktoria Marszał; **Writing – review & editing:** Oliwia Sysło, Sandra Czyż; **Visualization:** Wiktoria Marszał; **Supervision:** Nina Saracen; **Project administration:** Oliwia Sysło.

All authors have read and agreed with the published version of the manuscript.

Funding Statement
The authors declare that there are no conflicts of interest or financial disclosures associated with this scientific work.

Institutional Review Board Statement
Not applicable

Informed Consent Statement
Our work did not involve direct human subject research or obtaining their consent for participation in the study.

Data Availability Statement
As a review paper, our work does not present new data or analyses. Therefore, there are no specific databases or data availability to report. The information and findings presented in this review are based on previously published studies, which can be accessed through their resective sources as cited in the reference section.

Conflicts of Interest Statement
The authors declare that there are no significant conflicts of interest associated with this research work.

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