VIA MEDICA

CASE REPORT
PRACA KAZUISTYCZNA

Child Neurology — Neurologia Dziecięca
2024, vol. 34, no. 62, 5–10

ISSN 1230-3690, e-ISSN 2451-1897

DOI: 10.5603/cn.100564

Damage to the nerve roots of the brachial plexus in the course of Lyme disease in a 15-year-old girl

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ABSTRACT

Lyme disease is a multi-organ infectious disease and the most common vector-borne disease in Europe and North America. An infection is caused by the spirochetes *Borrelia burgdorferi*, *B. afzelii*, and *B. garinii*, affecting all age groups, especially in temperate climates. We report a case of a 15-year-old female patient, who was hospitalised due to right upper limb paresis accompanied by a noticeable decline in both muscle strength and exteroceptive sensation. Extensive laboratory and imaging assessments were conducted to rule out injury complications, as well as vascular and neurological disorders of the head and cervical spine. Despite the absence of a history of tick bite or erythema migrans, *Borrelia*-specific IgM and IgG antibodies were detected in the patient's blood serum. Although initial cerebrospinal fluid analysis showed no antibodies, subsequent testing confirmed the presence of *Borrelia* antibodies, confirming the neuroborreliosis. The patient received a combination of antibiotic therapy, steroid therapy, and physiotherapy due to her clinical condition. Electroneurography revealed nerve damage in the weakened upper limb. Given the escalating incidence of borreliosis cases, particularly in paediatric populations, this case underscores the importance of heightened vigilance among specialists for such atypical presentations like radiculopathy, emphasising the need for timely diagnosis and intervention.

Keywords: Lyme disease, neuroborreliosis, upper limb paresis, radiculopathy

Neurol Dziec. 2024; 34; 62: 5–10

INTRODUCTION

Lyme disease is a complex infectious illness transmitted by ticks of the *Ixodes* genus, and it stands as one of the most frequently diagnosed zoonoses globally. Regrettably, its incidence and geographic spread among humans have shown a consistent upward trend year after year [1]. Within *the Borrelia burgdorferi* sensu lato complex, comprising 19 spirochete genospecies, several, including *B. Burgdorferi*, *B. garinii*, and *B. afzelii*, are considered to be associated with Lyme disease [2]. In Poland, the peak incidence of Lyme disease typically occurs during the summer months, notably in the northeastern regions, contrasting with fewer reported cases in the Greater Poland Voivodeship [3]. Research by Stańczak et al. [2] underscores *B. afzelii* as the predominant species in most areas of the country. The number of recorded cases of Lyme disease in Poland is systematically increasing. It doubled in the years 2008–2017, reaching 21,514 cases in 2017 [2, 4]. Epidemiological data resulting from the list of infectious diseases in Poland indicate that in 2023 the number of infections amounted to over 25,000, which means an increase of slightly over 30% in the number of cases compared to 2022 [5]. The disease manifests in 3 stages, although not all stages present in every patient. The clinical characteristics of each stage are outlined in Table 1 [6, 7]. Diagnosing Lyme disease can pose challenges for specialists, particularly in cases lacking a documented

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Received: 6.05.2024 Accepted: 31.07.2024 Published: 12.08.2024

Table 1. Clinical stadium of Lyme disease

Clinical stadium of Lyme disease
Early localised
1. EM
2. Borrelial lymphocytoma
3. Flu-like symptoms
Early disseminated
4. Multiple EM
5. Lymphadenopathy
6. Flu-like symptoms
7. Inflammation of the cranial nerves
8. Meningitis and encephalitis
9. Radiculitis
10. Polyarthritis
11. Myocarditis
12. Inflammation of the retina and choroid of the eye
Late
13. Single or multiple arthritis
14. ACA
15. Chronic meningitis and encephalitis
16. Peripheral neuropathy

ACA — Atrophic dermatitis; EM — Erythema migrans

tick bite history or the absence of erythema migrans characteristic for this disease. Thus, early detection, especially of the localised and early disseminated stages, is pivotal in clinical practice, given the potential for spirochete spread to various organs. Challenges in diagnosis may escalate in the chronic stage, marked by a spectrum of symptoms such as prolonged joint pain, neurological manifestations, muscle aches, psychiatric symptoms, or cardiac complications. A prompt and precise diagnosis offers the opportunity for early intervention, limiting disease progression and averting severe complications [8]. Our study focuses on the neurological manifestations of Lyme disease in paediatric patients, particularly radiculitis leading to upper limb weakness and paralysis, which is extremely rare in children [9].

Diagnosis hinges on a positive history of tick exposure, clinical symptomatology, and positive serological tests. In the diagnostic realm of *Borrelia burgdorferi* infection, blood assays stand as a primary modality, aiming to identify antibodies generated by the host's immune response. Laboratory tests focus on 2 key antibody types, which are Immunoglobulin M (IgM) antibodies responsible for emerging early during the infection phase and Immunoglobulin G (IgG) antibodies, which arise later in the infection's course and exhibit long-term persistence within the system. The presence of characteristic erythema migrans warrants treatment initiation without further testing. Antibiotic therapy remains the cornerstone in managing Lyme disease and its complications, tailored to the disease stage and affected organs, typically spanning 2–4 weeks [8, 10].

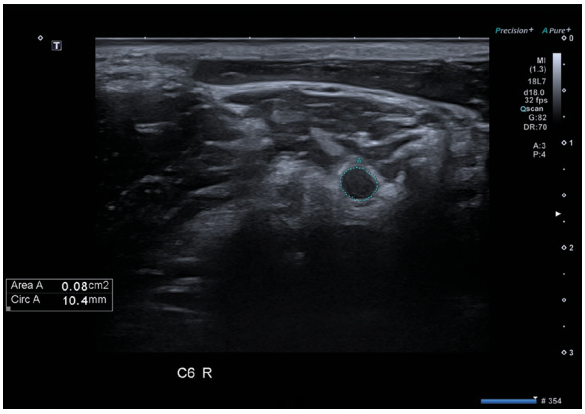

Figure 1. C6 root is of borderline size with a cross-sectional area of 0.08 cm^2

CASE REPORT

A 15-year-old girl was admitted to the Paediatric Neurology Unit due to right upper limb paresis and pain presenting for 2 days. The patient denied injuries. Neurological examination revealed lack of active movement of the right shoulder, elbow, and wrist joint and trace movement of the interphalangeal joints. Muscle strength and exteroceptive sensation markedly weakened in the right upper limb. In X-ray imaging no bone damage was presented. A cervical spine and head MRI examination was performed. The vertebral bodies showed normal height and signal morphology, and intervertebral discs without signs of protrusion into the spinal canal. Brain tissue exhibited a normal distribution of signal intensity, and the ventricular system was not dilated, displaced, or asymmetrical. There were no focal changes or signs of increased intracranial pressure. The signal of Willis circle arteries was normal. Laboratory tests for Lyme disease showed positive IgG antibodies (127.40 AU/mL) and IgM antibodies (55.18 AU/mL). On the third day of hospitalisation, an ultrasound of the right brachial plexus was performed. The examination revealed the roots of the brachial plexus in the range C5–C8 with preserved continuity. In the region of the intervertebral foramina exits, the roots exhibited decreased echogenicity, with blurred bundle structure; the C6 root was of borderline size with a cross-sectional area of 0.08 cm^2 (Fig. 1).

No other pathologies were noted in the examination, and the observation for Parsonage-Turner syndrome was suggested. Dexamethasone, omeprazole, potassium supplementation, calcium, and vitamin D3 and B supplementation were administered. In the electromyogram of the nerves of the right upper limb, damage to motor and sensory fibres was observed. The examination revealed electrophysiological features consistent with sensorimotor demyelinating

polyneuropathy — most pronounced in the median, ulnar, and radial nerves. The immunoblot test confirmed the presence of IgM and IgG antibodies against Lyme disease. Therapy with ceftriaxone was initiated. On the fifth day of hospitalisation, cerebrospinal fluid was collected during a lumbar puncture. No IgM or IgG antibodies against antigens characteristic of Lyme disease were detected. Pleocytosis was not observed, and the protein level was normal (17.1 mg/dL). Encephalitis and meningitis were ruled out. The results of the cerebrospinal fluid culture for aerobic bacteria were negative. A rehabilitation consultation additionally revealed weakness in the right hip flexor muscles and weakness in the right foot dorsal flexors. Physiotherapy treatments were prescribed. Tick-borne encephalitis was ruled out in subsequent examinations. Follow-up tests showed elevated leukocytosis, probably due to steroid therapy. On the 17th day of hospitalisation, magnetic resonance imaging of the lumbosacral spine revealed signs of dehydration of the L5–S1 intervertebral disc and protrusion of the disc (approximately 4 mm in the AP dimension) with a herniated nucleus pulposus. There were no evident signs of nerve compression. Over the next few days, significant improvement in the strength of the right upper limb muscles during exercises was noted, but there was also observed scapular winging during limb elevation. The patient reported a lowered mood and perceived no improvement in health status. Therefore, a psychological consultation was requested, revealing a mood decrease with a tendency towards dysphoria, marked by lability. Hence, the psychologist recommended periodic psychological support. On the same day, an electrocardiogram (ECG) showed features of accelerated atrioventricular conduction (PQ 0.1 s) and nonspecific ST-T changes (horizontal ST depression of 1 mm in I, II, III, V4–V6). On the 19th day of hospitalisation, the patient reported discomfort in the left knee patella area. Medical history included orthopedically treated due to patellofemoral conflict. Examination revealed no patellar ballottement, range of motion limitations, or superficial sensation disorders. Subsequently, the girl complained of increased knee pain upon loading. Therefore, an orthopaedic examination was conducted, followed by a referral to an orthopaedic clinic for knee joint injections. Physiotherapy treatments and antibiotic therapy were continued. The intravenous dexamethasone dosage was gradually reduced. At the end of hospitalisation, a lumbar puncture was performed again, and an immunoblot test was conducted, which showed the presence of IgG class antibodies (BB_p38+, BB_A34+, OspC+, BmpA+, Flagellin+). However, IgM class antibodies were absent. On the day of discharge, the patient did not report any sensory disturbances. Their deep reflexes were symmetrical, and meningeal signs were negative. Additionally, minimal weakness was noted in shoulder abduction and elbow flexion. The patient, in good condition, was discharged home with recommendations for continuing antibiotic therapy and gradually tapering off steroid therapy.

DISCUSSION

Lyme disease presents with distinctive clinical symptoms categorised into 3 stages, as presented in Table 1. The initial stage is marked by skin changes, notably erythema migrans, a ring-shaped lesion with central clearing typically appearing within 2 weeks of tick bite. It typically appears in lower body parts for adults and upper body parts for children. In the United States, it is generally recognised as erythema migrans when its size reaches a minimum of 5 cm [11]. The appearance of it can be associated with pain and warming of the skin. Its presence, coupled with confirmation of prior tick exposure, serves as a key indicator for disease diagnosis and prompts the initiation of antibiotic therapy. While erythema migrans is considered the hallmark of Lyme disease, its absence in up to 50–80% of cases requires in the diagnosis a comprehensive approach that integrates clinical assessment, laboratory testing, and consideration of the patient's history and symptoms. The laboratory diagnosis of Lyme disease involves several key methods, such as serological tests, the approach to which is to detect antibodies (IgM and IgG) produced by the immune system in response to *Borrelia burgdorferi* infection. Cerebrospinal fluid examination is crucial in the diagnosis of neuroborreliosis. Intrathecal synthesis of IgM antibodies occurs in nearly 100% of cases, while IgG synthesis is detected in about 60% of patients [12]. Polymerase chain reaction (PCR) testing can detect the genetic material of *Borrelia burgdorferi* in biological samples such as skin lesions, cerebrospinal fluid, or synovial fluid, in the early stages of infection when antibody levels may be low or do not occur. However, PCR's sensitivity is low, and there is a lack of standardisation in Lyme disease diagnostics. It is essential to note that a positive serological test without additional clinical symptoms is not enough for diagnosis or treatment initiation [7, 8, 10, 13]. Less commonly encountered skin changes include lymphocytoma, which is more prevalent among paediatric patients and may manifest, for instance, on the earlobe or nipple. These manifestations typically resolve spontaneously within a few weeks or may not manifest throughout the entire disease course. In the second stage of borreliosis, characterised by systemic dissemination of the infection, various organs including the heart, joints, and nervous system are often affected. Clinical manifestations during this phase may resemble flu-like symptoms, and in some instances, cardiac arrhythmias may manifest. Neuroborreliosis, frequently observed during this stage, may present with cranial nerve paralysis, inflammation of the brain and spinal cord membranes, or

Table 2. Diagnostic criteria for Lyme neuroborreliosis [36]

Diagnostic criteria for Lyme neuroborreliosis
Possible neuroborreliosis • typical clinical picture (cranial nerve deficits, meningitis/meningoradiculitis, focal neurological deficits) • Borrelia-specific IgG and/or IgM antibodies in serum • Differentiation from other causes
Probable neuroborreliosis • possible Lyme neuroborreliosis, additionally • CSF with lymphocytic pleocytosis, blood-CSF barrier dysfunction, and intrathecal immunoglobulin synthesis
Definite neuroborreliosis • probable neuroborreliosis, additionally • Borrelia-specific antibodies (positive IgG and/or IgM antibody index) in CSF • positive culture or nucleic acid detection (PCR) in CSF

CSF — cerebrospinal fluid; IgG — immunoglobulin G; IgM — immunoglobulin M; PCR — polymerase chain reaction

encephalitis. In Europe, neuroborreliosis predominantly manifests in the paediatric population, with lymphocytic meningitis accounting for 35% and facial nerve palsy for 55% of cases. Common symptoms among affected children with nervous system involvement include headache (61%), fatigue (60%), cranial nerve palsy (59%), neck pain (36%), and fever (30%). Additionally, subtle neurological manifestations such as loss of appetite or mood changes may be observed, as noted in the described patient. Although radicular symptoms in children are exceedingly rare, isolated case reports have documented hemiparesis or ataxia in the context of paediatric neuroborreliosis [14–18]. Symptoms of radiculitis typically appear within 4–6 weeks after the tick bite. The initial complaints reported by patients are usually persistent pain, which intensifies at night. In most patients, neurological deficits with weakness and sensory disturbances develop within 1–4 weeks [19]. Diagnosis of neuroborreliosis is based on the criteria presented in Table 2. Joint inflammation, also observed in the second phase of infection, is characterised by fluid accumulation in joints such as the ankle, shoulder, or knee. These changes are accompanied by warmth, swelling, and pain. The third stage is associated with permanent damage to specific organs and serious consequences for their functioning [2].

In the case of our paediatric patient, no erythema migrans was visible, and the medical history did not indicate the possibility of a tick bite. However, symptoms related to the nervous system and positive immunological tests in blood serum led clinicians to the possibility of diagnosing neuroborreliosis. Due to the potential involvement of the nervous system, a lumbar puncture was performed to examine the cerebrospinal fluid for neuroborreliosis. Interestingly, the first cerebrospinal fluid [CSF] test results did not confirm the diagnosis. However, the progressive paresis and pain of the upper limb, the appearance of knee joint pain, and changes in the electrocardiography recording prompted doctors to start treatment with antibiotics and glucocorticosteroids. In the last days of hospitalisation, a lumbar puncture was performed again. It showed the presence of IgG antibodies against *B. burgdorferi* in the CSF. The presence of IgG antibodies in the blood serum indicated the presence of spirochete infection. After the treatment, the symptoms disappeared and the patient's health improved. In this case, it turned out to be extremely important to initiate treatment based on the patient's clinical picture, despite the lack of antibodies in the CSF in the first examination. There are reports that the result of CSF examination in the course of neuroborreliosis without involvement of the meninges may be normal. Thus, the presence of antibodies in the blood and the manifestation of neurological symptoms alone should prompt treatment initiation [21]. In a study conducted by K. Ogrinc et al. [22], who examined the presence of anti-*Borrelia* antibodies in the cerebrospinal fluid of patients with early neuroborreliosis (Bannwarth syndrome), the presence of antibodies against *B. burgdorferi* was tested. IgM antibodies were present in 63% of patients, and IgG antibodies in 88.7% of patients tested. In the differential diagnosis, it is also worth considering Bennworth's syndrome, which is characterised by the classic triad of painful radiculopathy, cranial nerve inflammation, and lymphocytic pleocytosis in the cerebrospinal fluid [23]. However, Bennworth's syndrome primarily affects adults with early neuroborreliosis (36%), and it is the second most common manifestation of acute Lyme borreliosis in adults in Europe, following erythema migrans [24]. In children, it represents only 10.5% as a manifestation of Lyme disease [25]. Most often, neuroborreliosis results in facial nerve paralysis and/or meningitis [26]. However, paresis and pain of the upper limb accompanied by decreased sensation is not a very common manifestation of Lyme disease in children with involvement of the nervous system, which is associated with numerous diagnostic difficulties. In the literature, there are references to paralysis of the nerves of the upper limbs in the course of Lyme disease. Monteiro et al. [27] described the case of a 63-year-old woman with bilateral paralysis of the upper limbs and diaphragm accompanied by pain, and CSF examination confirmed the presence of IgM and IgG antibodies indicating *Borrelia* infection. Just like in our case, quick initiation of antibiotic therapy resulted in the described patient's symptoms disappearing. Non-specific symptoms affecting the nervous system should raise the suspicion of neuroborreliosis and prompt clinicians to examine the cerebrospinal fluid. The patient under consideration exhibited a solitary characteristic symptom of Bannwarth syndrome, namely, painful radiculopathy. Nevertheless, our patient met the diagnostic criteria for

neuroborreliosis, wherein the presence of IgM and IgG antibodies in the cerebrospinal fluid, coupled with neurological symptoms, enabled the clinician to establish a diagnosis. The first-line drugs for Lyme disease include doxycycline (for those over 9 years old), amoxicillin, cefuroxime, ceftriaxone, and cefotaxime. After antibiotic therapy, most patients fully recover, but some report persistent symptoms known as post-treatment Lyme disease syndrome (PTLDS). This syndrome is more common in individuals with chronic Lyme disease (CLD). Unfortunately, antibiotics may not always be effective for this form of the disease, and the cause of persistent symptoms accompanying PTLDS is not yet fully understood. Therefore, maintaining diagnostic vigilance regarding nonspecific symptoms in Lyme disease is crucial, because untreated Lyme disease can lead to the development of many other symptoms, including chronic, recurrent arthritis, especially in children, and neurological complications [30, 31]. A study led by R. Dersch et al. [32] evaluated the effectiveness of various antibiotics in treating neuroborreliosis and found no statistically significant differences in the resolution of neurological symptoms during or after treatment. Additionally, the occurrence of adverse effects showed no statistically significant differences. In studies conducted so far, most patients (90.6%) do not exhibit any neurological symptoms 3 months after completing antibiotic therapy [33]. Residual symptoms were observed in 687 patients who had experienced neuroborreliosis included sensory disturbances (5.24%), facial nerve palsy (3.6%), limb weakness (2.33%), pain (2.77%), and unsteady gait/dizziness/ataxia (2.62%). Only 15 patients presented with symptoms of late-stage neuroborreliosis (0.02%) [34]. Insufficient research exists to determine recommended antibiotics for treating neuroborreliosis in children. However, incorporating non-pharmacological therapy such as physiotherapy, occupational therapy, speech therapy, neuropsychological training, psychosocial interventions, and the administration of analgesic medications is advised if symptoms necessitate it [35]. In children, a 14-day course of antibiotic therapy is recommended, with dosing based on weight in kilograms per day. Treatment regimens include doxycycline (for children above 9 years old) at a dose of 4 mg/kg orally, ceftriaxone 50 mg intravenously, cefotaxime 100 mg intravenously, or penicillin G at a dose of 200–500,000 IU intravenously. The effectiveness of treating neuroborreliosis in children is high. Early recognition of the disease and the implementation of appropriate antibiotic therapy offer a chance for rapid improvement in the patients' condition [36].

CONCLUSIONS

Due to the increase in recorded cases of *Borrelia* infections, it is recommended that specialists, especially in the case of non-specific symptoms such as in our patient, be more vigilant and include Lyme disease in the differential diagnosis. In such cases, the patient's interview should be carefully read, during which special attention should be paid to the history of tick bites or the appearance of characteristic skin lesions. Appropriate diagnosis and the fastest possible implementation of antibiotic therapy in accordance with the recommendations are the basis of treatment and are crucial in the fight against this disease.

ARTICLE INFORMATION AND DECLATARIONS

Ethics statement

The work has not been published before. It has not been submitted for publication in another journal. All persons listed as authors of the work have read it and approved it for publication. The head of the institution where the work was created consents to sending the work.

Author contributions

Conceptualization: WS, NW, EP, KW, MO, MP, MCK; **validation:** WS, NW, EP, KW, MO, MP, MCK; **resources:** MO; **data curation:** WS, NW, EP, KW, MO, MP, MCK; **writing – original:** WS, NW, EP, KW, MO, MP, MCK; **draft preparation:** WS, NW, EP, KW, MO, MP, MCK; **writing — review and editing:** MP, MCK; **supervision:** WS, NW; **project administration:** WS, NW, EP, KW.

Funding

This research received no external funding.

Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or

financial relationships that could be construed as a potential conflict of interest.

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