Ticks and Tick-borne Diseases 17 (2026) 102660

Contents lists available at ScienceDirect

Ticks and Tick-borne Diseases

journal homepage: www.elsevier.com/locate/ttbdis

Short communication

Two pediatric Lyme neuroborreliosis patients presenting with cerebrovascular events

Heidi Pöyhönen [a,*], Annukka Pietikäinen [b], Varpu Rinne [c], Aapo Sirén [d], Satu Ranne [e], Ville Peltola [e], Jukka Hytönen [b,c]

[a] Paediatric Neurology, Department of Paediatrics and Adolescent Medicine, Turku University Hospital and University of Turku, Turku, Finland
[b] TYKS Laboratories, Clinical Microbiology, Turku University Hospital, Turku, Finland
[c] Institute of Biomedicine, University of Turku, Turku, Finland
[d] Department of Radiology, University of Turku and Turku University Hospital, Turku, Finland
[e] Department of Paediatrics and Adolescent Medicine, Turku University Hospital and University of Turku, Turku, Finland

ARTICLE INFO

Keywords:
Lyme neuroborreliosis
Pediatric stroke
Cerebrovascular events
Cerebral vasculopathy
Posterior circulation
CXCL13

ABSTRACT

Background: Pediatric Lyme neuroborreliosis (LNB), caused by *Borrelia burgdorferi* sensu lato (Bbsl), may rarely present with cerebrovascular involvement, particularly affecting the posterior circulation and causing ischemic or hemorrhagic stroke. In endemic regions, LNB should be considered a potential cause of pediatric stroke.
Case presentation: Case 1: An 8-year-old girl presented with recurrent episodes of impaired awareness, transient right arm flaccidity, and constitutional symptoms. MRI showed leptomeningeal enhancement, and time-of-flight angiography revealed focal narrowing of both posterior cerebral arteries. Cerebrospinal fluid (CSF) demonstrated pleocytosis, elevated protein, and increased Bbsl-specific antibodies and CXCL13.
Case 2: A 13-year-old girl presented with acute dizziness, dysarthria, and left-sided weakness. Brain CT was normal, but CT angiography showed irregularities in the posterior circulation, and MRI performed 12 hours later confirmed an acute pontine infarct. CSF showed pleocytosis, elevated protein, increased Bbsl-specific antibodies and CXCL13, and positive Bbsl-PCR.
Intervention: Both patients received intravenous ceftriaxone, patient 1 for two, and patient 2 for three weeks. Case 1 was treated with aspirin, and Case 2 received high-dose methylprednisolone and anticoagulation, later transitioned to aspirin.
Results: Both patients improved rapidly. Case 1 became fully asymptomatic within one month, with near-complete radiologic resolution. Case 2 recovered functional independence and returned to school within weeks, with only mild residual neurological findings at follow-up.
Conclusion: These cases emphasize the varied neurological presentations of LNB and the importance of including Lyme diagnostics in the evaluation of pediatric ischemic events. It is important to recognize LNB as an etiology of pediatric stroke because effective antimicrobial therapy may dramatically change outcome.

1. Introduction

Lyme borreliosis (LB), caused by the bacterium *Borrelia burgdorferi* sensu lato (Bbsl), is the most common tick-borne disease in Europe (Steere et al., 2016). In Finland, 9000–10,000 LB cases are reported annually, according to national healthcare registers maintained by the Finnish Institute for Health and Welfare.

In European patients, Lyme neuroborreliosis (LNB) is the second most common manifestation of disseminated LB, after multiple erythema migrans. LNB may present as painful radiculitis, cranial nerve palsy, meningitis, or encephalitis (Mygland et al., 2010). In children, facial nerve palsy is the most common manifestation of LNB.

Though rare, LNB can lead to stroke or other cerebrovascular complications (Oschmann et al., 1998; Topakian et al., 2008) These complications are poorly documented, especially in pediatric populations (Topakian et al., 2008). LNB-related cerebrovascular events typically affect the posterior cerebral circulation and may cause subarachnoid or intracerebral hemorrhage, but most commonly result in ischemic stroke

* Corresponding author at: Department of Paediatrics and Adolescent Medicine, Turku University Hospital, P.O. Box 52, 20521 Turku, Finland.
E-mail address: hmlaut@utu.fi (H. Pöyhönen).

https://doi.org/10.1016/j.ttbdis.2026.102660
Received 24 March 2026; Received in revised form 10 May 2026; Accepted 15 May 2026
Available online 21 May 2026

due to cerebral vasculitis (Oschmann et al., 1998; Topakian et al., 2008).

Here, we report two distinct pediatric cases, 8- and 13-year-old girls of LNB presenting with cerebrovascular symptoms, one of which resulted in an ischemic stroke.

2. Case 1

2.1. Clinical presentation and initial evaluation

In February 2024, an 8-year-old girl with a history of surgically repaired cleft palate presented to the pediatric clinic at the Turku University Hospital due to reduced appetite, weight loss, and a sour oral taste. Basic laboratory tests revealed no explaining abnormalities (Table 1). Initial management included proton pump inhibitor therapy due to a clinical suspicion of gastroesophageal reflux. In July, the patient returned for evaluation due to new symptoms: persistent headaches, noticeable changes in personality, low mood, alopecia, tremor in hands, and recurrent episodes involving nausea, fever, impaired awareness, and transient flaccidity of the right arm.

Worsening symptoms led to investigations for neurological etiologies. Brain magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) revealed no abnormalities. Electroencephalogram (EEG) demonstrated slowing in the left cerebral hemisphere, while repeated EEG one month later showed a localized slowing pattern now involving the right hemisphere. The episodic symptoms, along with shifting cortical focal slowing observed in EEG, raised suspicion of hemiplegic migraine.

Over the following six months, the patient experienced five additional episodes consistent with the previously described clinical pattern. In February 2025, after experiencing a prolonged 12-hour episode with escalating symptoms, the patient sought emergency care at night. At admission, the patient was unable to use her right arm, stand without support, speak, or follow instructions.

Brain computed tomography (CT) was performed, as an MRI was not immediately available. The CT findings were nonremarkable. EEG demonstrated generalized bilateral slowing, suggestive of diffuse cerebral dysfunction.

The next day, a contrast-enhanced magnetic resonance imaging (MRI) of the brain and spine was obtained. Abnormal leptomeningeal enhancement was depicted in the spinal cord (Fig. 1a-b). The dental braces caused artefacts in the brain MRI, and brain MRI was repeated day after that as the braces were removed. MRI demonstrated striking leptomeningeal enhancement, particularly in the suprasellar and interpeduncular cisterns and cranial nerves VI, VII, VIII, and IX (Fig. 2a-b). No acute findings were seen in the brain parenchyma. Time-of-flight angiography (TOF) revealed focal narrowing in both posterior cerebral arteries (PCA) (Fig. 2c).

Table 1
Laboratory test results of patient 1.

Test	Result	Reference Range
B-Hb (g/L)	142	110–149
B-Leuk (× 10⁹/L)	5.4	4–10
B-Thrombocytes (× 10⁹/L)	262	150–360
B-Lactate (mmol/L)	5	<15
P-K (mmol/L)	4.1	3.4–4.4
P-Na (mmol/L)	138	139–146
P-Creatinine (μmol/L)	40	<63
P-Albumin (g/L)	40.4	39–47
P-Ionized Calcium (mmol/L)	1.24	1.16–1.30
fP-Phosphate (mmol/L)	1.19	1.2–1.8
P-Magnesium (mmol/L)	0.85	0.6–0.9
fP-Glucose (mmol/L)	5.1	4–6
P-Ferritin (μg/L)	102	8–79
P-CK (U/L)	55	56–254
P-ALAT (U/L)	16	<32
P-TSH (mU/L)	1.6	0.6–4.8
P-Free T4 (pmol/L)	20.0	12.5–21.5
Urine leukocytes / erythrocytes	negative	

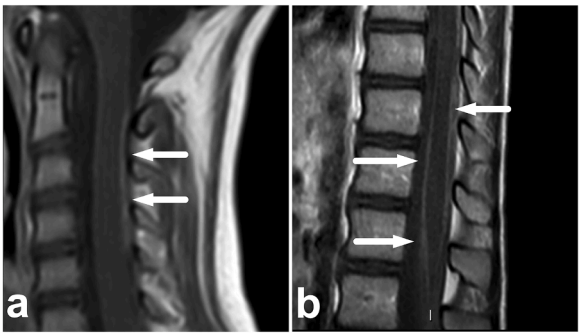

Fig. 1. a T1-weighted contrast-enhanced MRI of the cervical spine, sagittal plane. **b** T1 weighted contrast-enhanced MRI of the conus medullaris, sagittal plane (Case 1). Abnormal leptomeningeal enhancement is seen at the cervical spinal cord and conus medullaris (white arrows).

2.2. Further investigations

Cerebrospinal fluid sample (CSF) analysis revealed pleocytosis, high protein level, elevated total IgG index and oligoclonal banding consistent with central nervous system (CNS) infection or inflammation, as detailed in Table 2. Microbiological testing revealed elevated Bbsl-specific antibodies in both serum and cerebrospinal fluid, increased intrathecal Bbsl antibody index, and an increased C-X-C motif chemokine 13 (CXCL13) concentration in the CSF. However, CSF Bbsl-PCR was negative. Serum syphilis serology was negative (Alinity i TP assay, Abbott). Microbiological results are presented in Table 3.

2.3. Treatment

Intravenous antimicrobial therapy with ceftriaxone and acyclovir was initiated upon admission, due to signs of CNS infection in CSF sample. Acyclovir was discontinued after a few days when herpes simplex virus PCR returned negative while ceftriaxone was continued for a total of three weeks. Oral aspirin was initiated due to cerebral arteriopathy, with the aim of preventing ischemic stroke.

2.4. Follow-up and outcome

Following initiation of antibiotic therapy, the child began to recover rapidly. After six days of hospitalization, she was discharged. At that point, neurological assessment revealed only subtle balance difficulty when standing on the left leg. The next MRI was performed nine days after the admission. Leptomeningeal enhancement had decreased but not vanished (Fig. 2d). The irregularities in the PCAs remained (Fig. 2e-f). At a follow-up visit to the pediatric neurology outpatient clinic one month after discharge, the patient was completely asymptomatic, and neurological examination revealed no abnormalities. After 2 months, the leptomeningeal enhancement in brain MRI had resolved. Some slight irregularity in both PCAs could still be suspected, but compared to the initial MRA appearance, the findings were significantly diminished, and it was unclear whether the finding represented actual faint residual narrowing or was due to the technical limitations of the TOF with subtle changes in the vicinity of the arterial bifurcations (Fig. 3a-c, white arrowheads). All the MRI studies were performed with the same scanner and the same imaging parameters, which increases the ability to compare the studies.

Three months after discharge, EEG was normal. Five months after discharge, neuropsychological assessment revealed difficulties in verbal reasoning, consistent with a developmental disorder; no residual symptoms suggestively caused by LNB were observed. The final MRI, performed 9 months after the acute phase, showed complete resolution

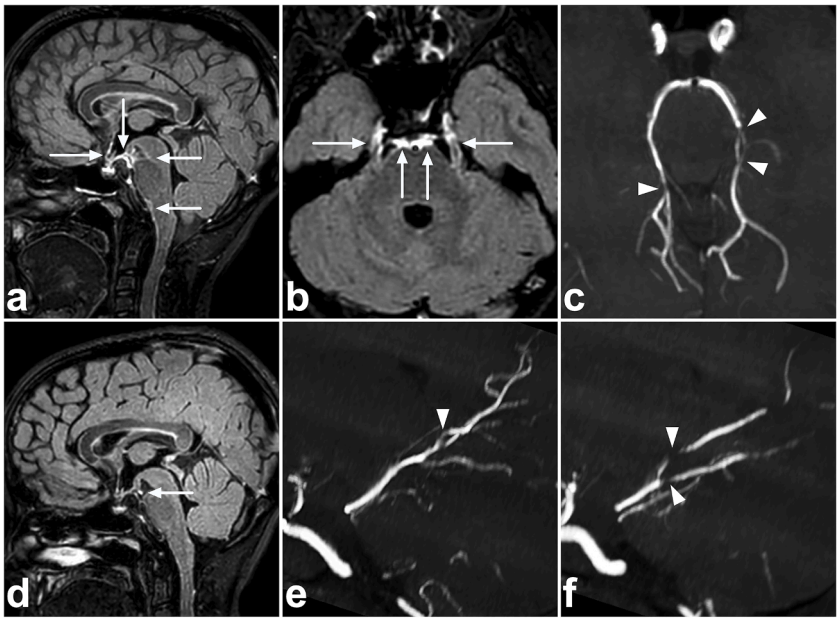

Fig. 2. **a** Contrast-enhanced FLAIR, sagittal plane. **b** Contrast-enhanced FLAIR, axial plane. Time-of-flight magnetic resonance angiography (TOF), maximum intensity projection, axial plane. **d** Contrast-enhanced FLAIR, axial plane. **e** TOF, maximum intensity projection, sagittal plane, right-sided off-midline view. **f** TOF, maximum intensity projection, sagittal plane, left-sided off midline view (Case 1). The first successful MRI demonstrated striking leptomeningeal enhancement (**a** and **b**, white arrows) and bilateral focal narrowing of PCAs (**c,** white arrowheads). After two weeks, partial resolution of the leptomeningeal enhancement was seen (**d**, white arrow), but the PCA abnormalities remained (**e** and **f**, white arrowheads).

Table 2
The CSF sample results of patient 1 and 2. *The leucocyte counts are reported from the third CSF tube.

CSF parameters	Patient 1	Patient 2	Reference Range
Erythrocytes	1×10^6/L	33×10^6/L	0–5×10^6/L
Leukocytes *	336×10^6/L	213×10^6/L	<5×10^6/L
Mononuclear cells	93 %	95 %	100 %
Polymorphonuclear cells	7 %	5 %	0 %
Total protein	>6000 mg/L	1291 mg/L	150–300 mg/L
IgG	1160 mg/L	310 mg/L	<40 mg/L
IgG Index	0.81	1.38	<0.7
Oligoclonal IgG bands (OCB)	Positive (13 CSF-restricted)	Positive (22; 2 matching serum)	Negative

of the vascular irregularities, and aspirin therapy was discontinued.

3. Case 2

3.1. Clinical presentation and initial evaluation

A girl with a history of cytomegalovirus hepatitis and iron deficiency anemia (both fully recovered), as well as previously diagnosed obsessive-compulsive disorder (OCD) and attention-deficit/hyperactivity disorder (ADHD), began experiencing episodes of headache accompanied by photophobia and nausea in November 2024. In January 2025, at the age of 13, she presented at the pediatric emergency department due to acute neurological symptoms, including dizziness, speech difficulty, and weakness affecting the left upper limb and facial muscles. These findings corresponded to a score of 4 on the Pediatric National Institutes of Health Stroke Scale (PedNIHSS) (Ichord et al., 2011), raising suspicion of stroke.

Upon admission, brain CT, CT angiography (CTA) of the head and neck, and brain perfusion CT were performed. No infarction, hemorrhage, or perfusion abnormalities were seen. CTA revealed non-occlusive irregularities of the arteries in the intracerebral posterior circulation, especially in the basilar artery (BA) (Fig. 4a).

Table 3
The microbiological results of Cases 1 and 2.

Test / Parameter	Method	Case 1	Case 2	Reference Range / Cut-off
Bbsl-PCR, CSF	GeneProof Borrelia PCR	Negative	Positive	Negative
Bbsl IgM antibody, CSF, RU/mL	Euroimmun Anti-Borrelia ELISA	7.9	8.0	<16
Bbsl IgG antibody, CSF, RU/mL	Euroimmun Anti-Borrelia plus VlsE ELISA	>200	>200	<16
Bbsl IgM quantitative, CSF, AU/mL	Diasorin, Liaison® Borrelia IgM Quant	19.3	9.5	<2.5
Bbsl IgG quantitative, CSF, AU/mL	Diasorin, Liaison® Borrelia IgG	164	187	<4.5
Intrathecal Bbsl IgM index	IDEIA™ Lyme Neuroborreliosis	0.7	5.6	<0.3
Intrathecal Bbsl IgG index	IDEIA™ Lyme Neuroborreliosis	6.3	11.8	<0.3
Bbsl IgM antibody, Serum, RU/mL	Euroimmun Anti-Borrelia ELISA	25.1	16.5	<16
Bbsl IgG antibody, Serum, RU/mL	Euroimmun Anti-Borrelia plus VlsE ELISA	190	>200	<16
Bbsl IgM quantitative, Serum, AU/ mL	Diasorin, Liaison® Borrelia IgM Quant	16	14	<18
Bbsl IgG quantitative, Serum, AU/mL	Diasorin, Liaison® Borrelia IgG	>240	>240	<10
CXCL13, CSF, pg/mL	Euroimmun CXCL13 ELISA	3600	780	<10

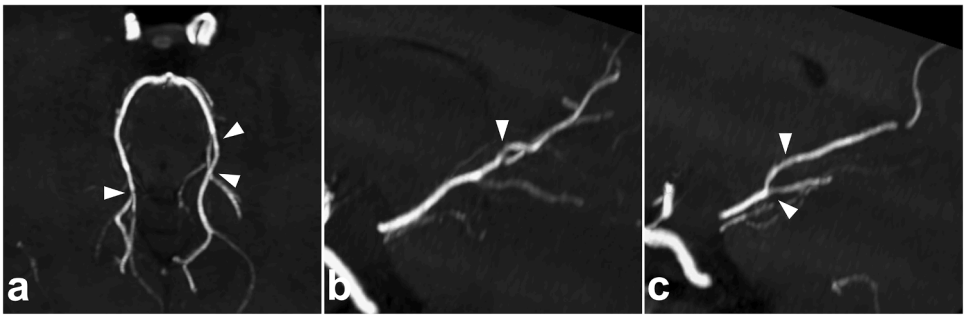

Fig. 3. **a** Time-of-flight magnetic resonance angiography (TOF), maximum intensity projection, axial plane. **b** TOF, maximum intensity projection, sagittal plane, right-sided off-midline view. **c** TOF, maximum intensity projection, sagittal plane, left-sided off-midline view (Case 1). Two months after the first MRI, bilateral PCA irregularities were mostly resolved (white arrowheads).

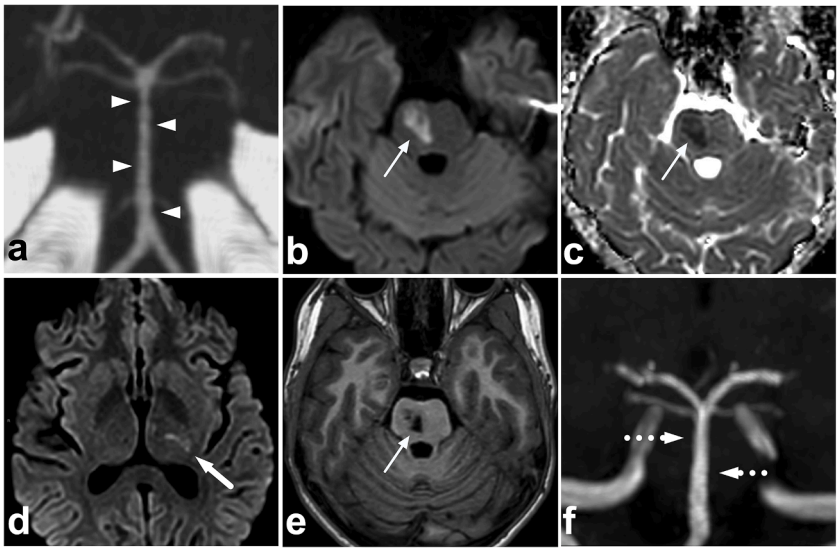

Fig. 4. **a** CTA, maximum intensity projection (MIP), coronal plane. **b** Diffusion weighted brain MRI, axial plane **c** Corresponding apparent diffusion coefficient map, axial plane **d** Diffusion weighted brain MRI, axial plane **e** T1-weighted gradient echo MRI, axial plane **f** Time-of-flight magnetic resonance angiography, maximum intensity projection, coronal plane (Case 2). Upon admission, CTA revealed diffuse narrowing of the basilar artery (**a**, white arrowheads). Twelve hours later, MRI demonstrated right-sided pontine infarction (thin white arrows). On the third day, a new infarction was seen in the left thalamus (**d**, white arrow). Two months later, a chronic infarction was seen at pons (**e**, thin white arrow), and the irregularity of the basilar artery had resolved (**f**, dashed white arrows).

CSF analysis showed signs of CNS infection, including pleocytosis, elevated protein concentration, high IgG and IgG index, and the presence of oligoclonal bands (Table 2). CSF polymerase chain reaction (PCR) testing was positive for Bbsl species (Table 3). Both serum and CSF Bbsl antibodies were positive and intrathecal Bbsl antibody index was increased. Additionally, CSF CXCL13 chemokine level was markedly elevated. Serum syphilis serology was negative (Alinity i TP assay, Abbott). These findings were consistent with active LNB. Nasopharyngeal swab testing was positive for adenovirus antigen.

3.2. Further investigations

Twelve hours later, a brain MRI revealed an acute stroke in the right side of the pons (Figs. 4b-c, thin white arrows). No evidence of thrombus was found on lower limb venous ultrasound or echocardiography. EEG was normal. Routine blood tests, including complete blood count, metabolic screening, toxicology and drug screening, showed no abnormalities. Coagulation studies revealed elevated factor XIII level, which represents a risk factor for deep vein thrombosis. Phospholipid antibodies were also elevated, indicating an increased risk for both arterial and venous thrombosis. Protein C activity was decreased and was planned to be reassessed after the acute phase.

On the third day, a brain MRI was repeated, this time with Gadolinium-based contrast media, as CSF findings were suggestive of infection. A new, small infarction was seen in the left thalamus (Fig. 4d), and the arterial irregularities were still present. No abnormal meningeal enhancement, parenchymal lesions suggesting encephalitis, or other signs of infection were found.

3.3. Treatment

During the first day of hospitalization, the patient was admitted to the pediatric intensive care unit for closer monitoring due to worsening and fluctuating symptoms. Intravenous ceftriaxone and acyclovir were initiated following lumbar puncture. Acyclovir was discontinued after two days once herpes simplex virus testing returned negative, while ceftriaxone was continued for a total of two weeks. High-dose methylprednisolone therapy (1000 mg per day intravenously) for suspected vasculitis was initiated five days after admission and administered for three days. Subcutaneous injections of enoxaparin were administered for six weeks as prophylaxis against deep vein thrombosis and discontinued once follow-up coagulation studies were normal, including repeat testing of anti-phospholipid antibodies. Oral aspirin was started for secondary stroke prevention.

3.4. Follow-up and outcome

During hospitalization, the patient underwent rehabilitation according to the instructions of the physiotherapist, occupational therapist, speech therapist, and neuropsychologist. At discharge, after 17 days of hospitalization, she had weakness in the left lower limb and facial muscles, and slightly impaired fine motor skills in the left hand; however, she was able to walk independently. The third brain MRI was performed upon discharge. Arterial irregularities were no longer evident, nor were there further parenchymal infarctions.

The patient returned to school two weeks after discharge, and the performance and participation was like her pre-stroke level. Neuropsychological assessment performed one month after discharge revealed mild difficulties in perceptual functions, likely representing developmental challenges unrelated to the stroke. Follow-up visits in the pediatric neurology outpatient clinic at three and five months after discharge demonstrated slightly clumsier fine motor function in the left hand, hyperactive left-sided tendon reflexes, and very mild left facial paresis. Six months after the onset, last follow-up MRI was obtained, revealing only a chronic pontine infarction (Fig. 4e). The thalamic infarction had not left any visible damage. TOF findings were normal (Fig. 4f). Physiotherapy continued for six months after discharge, after which her functional abilities had improved sufficiently to discontinue therapy. Aspirin was recommended to use for two years to prevent new ischemic strokes.

4. Discussion

We present here two pediatric patients with LNB and cerebrovascular involvement, highlighting ischemic stroke as a rare but severe complication of LB. Neurological manifestations occur in a minority of LB cases (approximately 3 %), and the clinical presentation of LNB differs between children and adults, with facial nerve palsy being the most common manifestation in pediatric patients (Bingham et al., 1995; Stanek et al., 2012). Cerebrovascular complications are uncommon in both adults and children, with reported frequencies of 0.6–1.7 % (Mironova et al., 2021; Monteventi et al., 2018). However, as the incidence of LB continues to rise in many regions, the absolute number of rare complications, including LNB-associated cerebrovascular events, may also increase (Sykes, 2023)

LNB is classically divided into early and late forms, which differ in both timing and clinical presentation; however, both are accompanied by intrathecal Bbsl-specific antibody production. Early LNB typically develops within weeks to a few months after infection, and in children, most commonly manifests as lymphocytic meningitis or cranial neuropathy - particularly facial nerve palsy (Mygland et al., 2010; Stanek et al., 2011). Late LNB, by contrast, is defined by neurological symptoms persisting for more than six months and is rare in pediatric patients (Hildenbrand et al., 2009; Skogman et al., 2003). It may present with chronic or progressive deficits such as encephalomyelitis or peripheral neuropathy, or, in exceptional cases, vasculitic complications including cerebrovascular events, such as cerebral arteritis and ischemic stroke (Pachner and Steiner, 2007; Topakian et al., 2008).

The first case, an 8-year-old girl, fulfilled the definition of late LNB with a symptom duration exceeding six months before diagnosis, ultimately associated with cerebrovascular involvement. The second patient, a 13-year-old girl, represents early LNB in which stroke occurred during the initial disseminated phase of the infection - a rare but increasingly recognized manifestation in pediatric populations. (Philipps et al., 2022; Sendor et al., 2025).

Antimicrobial treatment of LNB depends on the clinical presentation and extent of CNS involvement. In children with meningitis or cranial neuritis, oral doxycycline is an effective option, and studies show outcomes comparable to intravenous beta-lactams (Kortela et al., 2021; Mygland et al., 2010). Doxycycline can be used also in pediatric patients (Pöyhönen et al., 2017; Todd et al., 2015; Volovitz et al., 2007). When CNS parenchyma is affected, such as in encephalitis or vasculitic complications, intravenous ceftriaxone is recommended because of the lack of robust data supporting the efficacy of doxycycline in these rare and severe manifestations (Kortela et al., 2021; Mygland et al., 2010). Treatment duration also varies by disease stage: 14 days for early LNB and 21 days for late or more severe manifestations. In our cases, intravenous ceftriaxone was chosen due to cerebrovascular involvement (Kortela et al., 2021; Mygland et al., 2010). The younger patient received a 21-day course, consistent with late LNB, as her symptoms had persisted for more than a year.

Childhood acute ischemic stroke is rare with an incidence of approximately 1–8 per 100 000 children per year, and an in-hospital mortality of about 3–6 % (Panagopoulos et al., 2021; Rawanduzy et al., 2022). In school-aged children, including the age range of our patients (8 and 13 years), the incidence is closer to 1 per 100,000 per year (Ferriero et al., 2019). Cerebral arteriopathy is the single most common cause of childhood stroke, reported in one-third to one-half of cases (21-53 %) and, also the strongest predictor of recurrent stroke (Rafay et al., 2020; Sun and Lynch, 2023; Wintermark et al., 2014). Several forms of arteriopathy, including focal cerebral arteriopathy of inflammatory type (FCA-i), have been linked to preceding or ongoing infections, and CNS infections such as LNB, varicella zoster virus infection, and other inflammatory or post-infectious processes may precipitate vascular inflammation and focal stenosis (Ferriero et al., 2019; Monteventi et al., 2018).

In this context, CSF analysis is an important component of the diagnostic work-up in childhood acute ischemic stroke and associated arteriopathies, as it may identify CNS infection or inflammatory processes not evident on neuroimaging or blood studies (Ferriero et al., 2019). In our patients, CSF analysis was performed immediately upon hospital admission, directly after neuroimaging, which enabled early identification of CNS inflammation and prompt initiation of targeted therapy. Early diagnosis and treatment are important in LNB, as timely initiation of appropriate antimicrobial therapy is recommended in current guidelines (Mygland et al., 2010).

Although adenovirus was detected in Case 2, its clinical relevance appears minimal. Upper-respiratory adenovirus carriage is common in children and does not indicate systemic or CNS infection. The patient showed no signs of adenovirus-associated disease, and the CSF profile (pleocytosis, elevated intrathecal Bbsl-specific antibodies, and markedly increased CXCL13) was fully consistent with active LNB. Thus, adenovirus is unlikely to explain the neurological presentation.

In children with suspected stroke, MRI is the preferred imaging modality. Diffusion-weighted MRI detects ischemic changes within minutes of symptom onset and is clearly more sensitive than CT for early infarction, while also allowing assessment of stroke mimics and associated arteriopathy with MRA (Ferriero et al., 2019; Roach et al., 2008). In contrast, non-contrast CT is primarily useful to exclude intracranial hemorrhage but may appear normal in the hyperacute phase of ischemic stroke, especially in posterior circulation events (Sun and Lynch, 2023). In our case 2, the initial non-contrast CT scan of the head was normal, and a pontine infarct was only identified on subsequent MRI, illustrating the limited sensitivity of CT for early brainstem ischemia in children and underscoring the importance of MRI-based imaging protocols in pediatric stroke evaluation (Ferriero et al., 2019; Roach et al., 2008).

Both of our patients had unequivocal luminal narrowing in the posterior circulation on CTA and/or TOF angiography. Unfortunately, MR vessel wall imaging was not performed in the acute setting. Therefore, we were unable to determine whether the abnormalities represented LNB-related vasculitis or reactive, parainfectious arteriopathy (Cox et al., 2005; Topakian et al., 2008). Complete resolution of the arterial abnormalities, however, might suggest a parainfectious process rather than vessel wall inflammation. Our cases underline the importance of cerebrovascular imaging in patients with suspected or confirmed meningitis or encephalitis. CT angiography is the gold standard for arterial imaging of patients with acute stroke, offering high

spatial resolution and diagnostic accuracy. Especially when a detailed assessment of the brain parenchyma or follow-up imaging is needed, MR angiography should be considered as a radiation-free alternative. Digital subtraction angiography is an invasive procedure that exposes the child to a significant dose of ionizing radiation; however, in small vessel arteriopathy, it may be the only imaging modality that demonstrates the abnormalities. The threshold for performing MR vessel wall imaging should be kept low, as patients with vasculitis's have an increased risk of chronic cerebrovascular pathologies, such as aneurysms (Kortela et al., 2017).

Recognition of arteriopathy is clinically critical because it is the strongest predictor of recurrent stroke in children. Moreover, precise diagnosis of arteriopathy subtype (e.g., FCA, dissection, moyamoya, vasculitis) helps to guide management decisions, including choices about antithrombotic therapy, immunomodulation, need for long-term vascular imaging follow-up, and possible surgical interventions.

In selected pediatric inflammatory cerebral arteriopathies, high-dose intravenous methylprednisolone is commonly used to suppress vascular inflammation and limit disease progression (Benseler, 2006; Elbers and Benseler, 2008). In LNB-associated cerebrovascular complications, however, evidence supporting corticosteroid therapy is limited to case reports and small case series (Mironova et al., 2021; Monteventi et al., 2018). Adjunctive corticosteroids have nevertheless been administered in individual pediatric cases with suspected vasculitic involvement, in addition to appropriate antimicrobial therapy, with the aim of controlling vessel wall inflammation (Monteventi et al., 2018). Given the rarity of LNB-associated cerebral vasculitis and the lack of controlled studies, the role of methylprednisolone remains uncertain, but it may be considered on a case-by-case basis, particularly when imaging or clinical features raise concern for active inflammatory arteriopathy (Elbers and Benseler, 2008; Mironova et al., 2021).

Here, we have reported two pediatric patients with cerebrovascular manifestations of LNB. Several reports and a systematic review support a causal link between LNB and inflammatory cerebral arteriopathy or vasculitis leading to ischemic stroke, also in children (Cox et al., 2005; Kortela et al., 2017; Monteventi et al., 2018; Topakian et al., 2008). It is important to recognize infectious etiologies, particularly LNB, in pediatric stroke because effective antimicrobial therapy is available and may dramatically change outcome. Early identification permits initiation of targeted therapy, typically with intravenous antibiotics (e.g., ceftriaxone) as recommended in current LNB guidelines (Akkurt et al., 2023).

Funding

This work was supported by the Finnish Child Neurology Association's Märta Donner Grant.

Ethics approval and consent to participate

According to national regulations, case reports are not evaluated by ethics committees. Verbal informed consent for publication was obtained from the patients' parents.

CRediT authorship contribution statement

Heidi Pöyhönen: Writing – original draft, Visualization, Project administration, Funding acquisition, Data curation. **Annukka Pietikäinen:** Writing – original draft, Visualization, Validation, Methodology. **Varpu Rinne:** Writing – original draft. **Aapo Sirén:** Writing – original draft, Visualization, Validation, Methodology. **Satu Ranne:** Writing – review & editing, Visualization. **Ville Peltola:** Writing – review & editing, Validation, Supervision, Conceptualization. **Jukka Hytönen:** Writing – review & editing, Visualization, Validation, Supervision, Investigation, Conceptualization.

Declaration of competing interest

The authors declare no conflicts of interest.

Data availability

Data will be made available on request.

References

Akkurt, B.H., Kraehling, H., Nacul, N.G., Elsharkawy, M., Schmidt-Pogoda, A., Minnerup, J., Stracke, C.P., Schwindt, W., 2023. Vasculitis and ischemic stroke in Lyme neuroborreliosis – interventional management approach and literature review. Brain Sci. 13, 1388. https://doi.org/10.3390/brainsci13101388.

Benseler, S.M., 2006. Central nervous system vasculitis in children. Curr. Rheumatol. Rep. 8, 442–449. https://doi.org/10.1007/s11926-006-0040-4.

Bingham, P.M., Galetta, S.L., Athreya, B., Sladky, J., 1995. Neurologic manifestations in children with Lyme disease. Pediatrics 96, 1053–1056.

Cox, M., Wolfs, T., Lo, T., Kappelle, L., Braun, K., 2005. Neuroborreliosis causing focal cerebral arteriopathy in a child. Neuropediatrics 36, 104–107. https://doi.org/10.1055/s-2005-837573.

Elbers, J., Benseler, S.M., 2008. Central nervous system vasculitis in children. Curr. Opin. Rheumatol. 20, 47–54. https://doi.org/10.1097/BOR.0b013e3282f3177a.

Ferriero, D.M., Fullerton, H.J., Bernard, T.J., Billinghurst, L., Daniels, S.R., DeBaun, M. R., deVeber, G., Ichord, R.N., Jordan, L.C., Massicotte, P., Meldau, J., Roach, E.S., Smith, E.R., 2019. Management of stroke in neonates and children: a scientific statement from the American heart association/American stroke association. Stroke 50, e1–e100. https://doi.org/10.1161/STR.0000000000000183.

Hildenbrand, P., Craven, D.E., Jones, R., Nemeskal, P., 2009. Lyme neuroborreliosis: manifestations of a rapidly emerging zoonosis. Am. J. Neuroradiol. 30, 1079–1087. https://doi.org/10.3174/ajnr.A1579.

Ichord, R.N., Bastian, R., Abraham, L., Askalan, R., Benedict, S., Bernard, T.J., et al., 2011. Interrater reliability of the pediatric national institutes of health stroke scale (PedNIHSS) in a multicenter study. Stroke 42, 613–617. https://doi.org/10.1161/STROKEAHA.110.607192.

Kortela, E., Hytönen, J., Numminen, J., Overmyer, M., Saxen, H., Oksi, J., 2017. Cerebral vasculitis and intracranial multiple aneurysms in a child with Lyme neuroborreliosis. JMM Case Rep. 4, 5090. https://doi.org/10.1099/jmmcr.0.005090.

Kortela, E., Kanerva, M.J., Puustinen, J., Hurme, S., Airas, L., Lauhio, A., Oksi, J., 2021. Oral doxycycline compared to intravenous ceftriaxone in the treatment of Lyme neuroborreliosis: a multicenter, equivalence, randomized, open-label trial. Clin. Infect. Dis. 72, 1323–1331. https://doi.org/10.1093/cid/ciaa217.

Mironova, M., Kortela, E., Kurkela, S., Kanerva, M., Curtze, S., 2021. Lyme neuroborreliosis-associated cerebrovascular events in the Finnish endemic area. J. Neurol. Sci. 427, 117544. https://doi.org/10.1016/j.jns.2021.117544.

Monteventi, O., Steinlin, M., Regényi, M., Roulet-Perez, E., Weber, P., Fluss, J., 2018. Pediatric stroke related to Lyme neuroborreliosis: data from the Swiss NeuroPaediatric Stroke Registry and literature review. Eur. J. Paediatr. Neurol. 22, 113–121. https://doi.org/10.1016/j.ejpn.2017.10.010.

Mygland, Å., Ljøstad, U., Fingerle, V., Rupprecht, T., Schmutzhard, E., Steiner, I., 2010. EFNS guidelines on the diagnosis and management of European Lyme neuroborreliosis. Eur. J. Neurol. 17, 8. https://doi.org/10.1111/j.1468-1331.2009.02862.x.

Oschmann, P., Dorndorf, W., Hornig, C., Schäfer, C., Wellensiek, H.J., Pflughaupt, K.W., 1998. Stages and syndromes of neuroborreliosis. J. Neurol. 245, 262–272. https://doi.org/10.1007/s004150050216.

Pachner, A.R., Steiner, I., 2007. Lyme neuroborreliosis: infection, immunity, and inflammation. Lancet Neurol. 6, 544–552. https://doi.org/10.1016/S1474-4422(07)70128-X.

Panagopoulos, D., Karydakis, P., Markogiannakis, G., Themistocleous, M., 2021. Pediatric arterial ischemic stroke: overview of the literature and of the most cited relevant articles. Interdiscip. Neurosurg. 23, 100924. https://doi.org/10.1016/j.inat.2020.100924.

Philipps, J., Erdlenbruch, B., Kuschnerow, M., Jagoda, S., Salihaj, B., Glahn, J., Schellinger, P.D., 2022. Hyperacute treatment of childhood stroke in Lyme neuroborreliosis: report of two cases and systematic review. Ther. Adv. Neurol. Disord. 15, 1–12. https://doi.org/10.1177/17562864221102842.

Pöyhönen, H., Nurmi, M., Peltola, V., Alaluusua, S., Ruuskanen, O., Lähdesmäki, T., 2017. Dental staining after doxycycline use in children. J. Antimicrob. Chemother. 72, 2887–2890. https://doi.org/10.1093/jac/dkx245.

Rafay, M.F., Shapiro, K.A., Surmava, A.-M., deVeber, G.A., Kirton, A., Fullerton, H.J., Bernard, T.J., 2020. Spectrum of cerebral arteriopathies in children with arterial ischemic stroke. Neurology 94, e1–e12. https://doi.org/10.1212/WNL.0000000000009557.

Rawanduzy, C.A., Earl, E., Mayer, G., Lucke-Wold, B., 2022. Pediatric stroke: a review of common etiologies and management strategies. Biomedicines 11, 2. https://doi.org/10.3390/biomedicines11010002.

Roach, E.S., Golomb, M.R., Adams, R., Biller, J., Daniels, S., deVeber, G., Smith, E.R., 2008. Management of stroke in infants and children. Stroke 39, 2644–2691. https://doi.org/10.1161/STROKEAHA.108.189696.

Sendor, A., Wentrys, L., Stopyra, L., 2025. Neuroborreliosis as a cause of acute ischemic stroke in a 13-year-old patient. Cureus 17, e91374. https://doi.org/10.7759/cureus.91374.

Skogman, B.H., Croner, S., Ödkvist, L., 2003. Acute facial palsy in children – a 2-year follow-up study with focus on Lyme neuroborreliosis. Int. J. Pediatr. Otorhinolaryngol. 67, 597–602. https://doi.org/10.1016/S0165-5876(03)00061-2.

Stanek, G., Fingerle, V., Hunfeld, K.-P., Jaulhac, B., Kaiser, R., Krause, A., et al., 2011. Lyme borreliosis: clinical case definitions for diagnosis and management in Europe. Clin. Microbiol. Infect. 17, 69–79. https://doi.org/10.1111/j.1469-0691.2010.03175.x.

Stanek, G., Wormser, G.P., Gray, J., Strle, F., 2012. Lyme borreliosis. Lancet 379, 461–473. https://doi.org/10.1016/S0140-6736(11)60103-7.

Steere, A.C., Strle, F., Wormser, G.P., Hu, L.T., Branda, J.A., Hovius, J.W.R., Li, X., Mead, P.S., 2016. Lyme borreliosis. Nat. Rev. Dis. Primers 2, 16090. https://doi.org/10.1038/nrdp.2016.90.

Sun, L.R., Lynch, J.K., 2023. Advances in the diagnosis and treatment of pediatric arterial ischemic stroke. Neurotherapeutics 20, 633–654. https://doi.org/10.1007/s13311-023-01373-5.

Sykes, J.E., 2023. Tick-borne diseases. Vet. Clin. North Am. Small Anim. Pract. 53, 141–154. https://doi.org/10.1016/j.cvsm.2022.07.011.

Todd, S.R., Dahlgren, F.S., Traeger, M.S., Beltrán-Aguilar, E.D., Marianos, D.W., Hamilton, C., McQuiston, J.H., Regan, J.J., 2015. No visible dental staining in children treated with doxycycline for suspected Rocky Mountain spotted fever. J. Pediatr. 166, 1246–1251. https://doi.org/10.1016/j.jpeds.2015.02.015.

Topakian, R., Stieglbauer, K., Nussbaumer, K., Aichner, F.T., 2008. Cerebral vasculitis and stroke in Lyme neuroborreliosis. Cerebrovasc. Dis. 26, 455–461. https://doi.org/10.1159/000155982.

Volovitz, B., Shkap, R., Amir, J., Calderon, S., Varsano, I., Nussinovitch, M., 2007. Absence of tooth staining with doxycycline treatment in young children. Clin. Pediatr. 46, 121–126. https://doi.org/10.1177/0009922806290026.

Wintermark, M., Hills, N.K., deVeber, G.A., Barkovich, A.J., Elkind, M.S.V., Sear, K., Forsyth, R., 2014. Arteriopathy diagnosis in childhood arterial ischemic stroke. Stroke 45, 3597–3605. https://doi.org/10.1161/STROKEAHA.114.007404.