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CASE REPORT / ПРИКАЗ БОЛЕСНИКА

Suspected Lyme-associated peripheral facial palsy in an adolescent with pre-existing sensorineural hearing loss – a diagnostic challenge

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SUMMARY

Introduction Lyme neuroborreliosis (LNB) represents a neurological manifestation of the infection caused by *Borrelia burgdorferi sensu lato*. According to established European diagnostic criteria, confirmed LNB requires cerebrospinal fluid pleocytosis and evidence of intrathecal antibody synthesis; however, these criteria are not always fulfilled in clinical practice, which may lead to diagnostic uncertainty.

Case report A 15-year-old girl presented with acute right-sided peripheral facial palsy. Serological testing demonstrated positive IgM and IgG antibodies to *Borrelia burgdorferi sensu lato*. Cerebrospinal fluid analysis was not performed, precluding confirmation of neuroborreliosis. Brain magnetic resonance imaging (MRI) excluded central causes of facial palsy. The patient received empirical antibiotic therapy in combination with corticosteroids, followed by partial clinical recovery over time. Pre-existing sensorineural hearing loss and incidental MRI findings were considered unrelated to the acute presentation.

Conclusion This case highlights the limitations of attributing peripheral facial palsy to Lyme disease based solely on serological findings. In the absence of cerebrospinal fluid analysis, diagnostic uncertainty remains, emphasizing the need for cautious interpretation of laboratory results and thorough consideration of alternative etiologies.

Keywords: neuroborreliosis; Lyme disease; peripheral facial paralysis; adolescents; rehabilitation

INTRODUCTION

Lyme neuroborreliosis (LNB) represents a neurological manifestation of infection caused by *Borrelia burgdorferi sensu lato* and, according to established European Federation of Neurological Societies criteria, is defined by a compatible clinical presentation in combination with cerebrospinal fluid (CSF) pleocytosis, and evidence of intrathecal antibody synthesis [1]. These criteria are essential for confirming the diagnosis and distinguishing LNB from other neurological conditions.

Lyme borreliosis is the most common vector-borne disease in Europe, with a substantial and increasing public health burden. Recent epidemiological data indicate that an average 132,000 cases are reported annually across Europe, with neurological involvement occurring in a subset of patients [2, 3]. Among these, peripheral facial nerve palsy represents one of the most frequent clinical manifestations, particularly in pediatric populations [1, 4]. However, the presence of facial palsy alone is not sufficient to establish a diagnosis of LNB without appropriate CSF findings [1].

In clinical practice, the diagnostic evaluation of patients presenting with peripheral facial palsy may be challenging, particularly in regions with higher background seroprevalence of *Borrelia* exposure [5, 6]. Positive serological findings may reflect previous contact with the pathogen rather than active infection, which limits their specificity in the absence of confirmatory CSF analysis [1]. Consequently, attributing facial nerve palsy to Lyme disease based solely on serology may lead to overdiagnosis and misinterpretation of causality.

Although Lyme disease has been associated with a wide spectrum of neurological manifestations, isolated peripheral facial palsy remains a typical presentation, whereas involvement of additional cranial nerves or atypical features requires careful evaluation [1, 7]. Concurrent findings, such as sensorineural hearing loss, should be interpreted with caution, as clinical and laboratory findings in Lyme disease may be nonspecific and seropositivity does not necessarily indicate causality; therefore, such findings may represent pre-existing or unrelated conditions rather than manifestations of acute infection [3, 7].

This case report describes an adolescent presenting with peripheral facial palsy and positive *Borrelia* serology in a setting where CSF analysis was not performed. The aim of this report is not to demonstrate confirmed neuroborreliosis, but to highlight the diagnostic uncertainty and limitations associated with interpreting serological findings in the absence of CSF confirmation, as well as the importance of considering alternative etiologies.

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CASE REPORT

A 15-year-old previously healthy female from a suburban region of Belgrade was admitted to the pediatric cardiology department due to palpitations and intermittent chest discomfort. Initial evaluation, including Holter monitoring, revealed frequent ventricular extrasystoles without structural abnormalities or hemodynamic compromise. Further cardiological assessment, including ergospirometry, and repeat Holter electrocardiogram monitoring, showed no significant abnormalities. Routine laboratory analysis, including a metabolic panel and C-reactive protein, was within normal limits.

During hospitalization, on the first day after admission, the patient developed acute right-sided facial weakness, characterized by progressive inability to fully close the right eye and facial asymmetry (House–Brackmann grade IV) [8]. She denied fever, headache, recent infection, head trauma, rash, otalgia, vertigo, or known tick exposure. Past medical history was notable only for febrile seizures in early childhood, with no recurrence after the age of five. Immunizations were up to date, and family history was negative for neurological or autoimmune diseases.

Neurological examination revealed a peripheral right facial nerve palsy, including loss of forehead wrinkling, lagophthalmos, flattening of the nasolabial fold, and drooping of the mouth angle. Motor strength, sensory examination, cerebellar function, gait, and deep tendon reflexes were normal. Otoscopic examination showed no signs of middle ear pathology.

Audiological evaluation was performed as part of the assessment of cranial nerve function. Pure-tone audiometry demonstrated bilateral moderate-to-severe high-frequency sensorineural hearing loss (right ear: 2 kHz = 60 dB, 4 kHz = 80 dB; left ear: 2 kHz = 60 dB, 4 kHz = 65 dB), consistent with previously documented congenital hearing impairment. Tympanometry was type A bilaterally. Acoustic reflexes were reduced at 500 Hz on the right and absent at higher frequencies bilaterally.

Ophthalmological examination revealed conjunctival hyperemia and early signs of exposure keratopathy, and treatment with artificial tears and lubricating gel was initiated.

Laboratory and diagnostic evaluation for infectious and autoimmune causes included complete blood count, inflammatory markers, and serological testing. Serological analysis for *Borrelia burgdorferi sensu lato* was performed using a line blot assay and demonstrated the presence of specific IgM and IgG antibodies. CSF analysis was not performed.

Brain magnetic resonance imaging and magnetic resonance angiography showed no evidence of central nervous system pathology. An incidental choroidal fissure cyst without clinical significance was identified (Figure 1).

The patient was treated with intravenous ceftriaxone, followed by oral doxycycline (100 mg twice daily for seven days), in combination with corticosteroid therapy. Supportive management included artificial tears and physical therapy.

Clinical improvement was observed during hospitalization, with partial recovery of facial movements and improved eyelid closure. No systemic complications were noted.

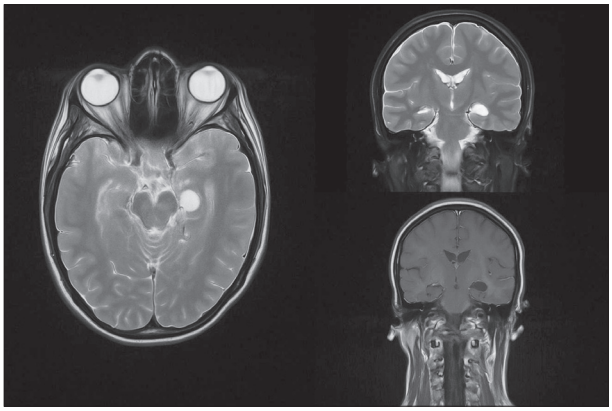

Figure 1. Magnetic resonance imaging diagnostics for exclusion of central facial palsy – cyst of the choroidal fissure; the patient was admitted to a pediatric day hospital and received intravenous ceftriaxone, corticosteroids with gradual tapering, gastroprotection, structured facial physiotherapy including electrostimulation of the right facial nerve and facial exercises, and continuous ocular lubrication; coagulation analysis, including von Willebrand factor antigen and factor XIII activity, were within normal limits; cardiac evaluation showed persistent but hemodynamically insignificant ventricular extrasystoles

After 20 days of hospitalization, the patient was discharged in stable condition with mild residual facial asymmetry. Outpatient management included continuation of doxycycline therapy, physiotherapy, and scheduled follow-up with neurology, otorhinolaryngology, ophthalmology, and cardiology.

DISCUSSION

Peripheral facial palsy represents a common clinical presentation in pediatric patients and may be associated with Lyme disease; however, it is not specific for LNB. According to established European criteria, confirmed neuroborreliosis requires CSF pleocytosis and evidence of intrathecal antibody synthesis [1, 9, 10]. In the absence of CSF analysis, the present case can only be classified as suspected Lyme-associated facial palsy rather than confirmed neuroborreliosis. This distinction is essential to avoid overinterpretation of clinical and laboratory findings.

The initial diagnostic consideration in this case included the possibility of broader cranial nerve involvement due to the coexistence of facial palsy and sensorineural hearing loss [3, 7]. However, audiological findings were consistent with previously documented congenital impairment, and no additional neurological deficits were identified. Therefore, the clinical presentation was ultimately limited to isolated peripheral facial palsy. This highlights the importance of careful interpretation of concurrent findings, particularly when pre-existing conditions may confound the clinical picture.

The interpretation of *Borrelia* serology requires particular caution. Positive IgM and IgG antibodies may reflect previous exposure rather than active infection, especially in regions with higher background seroprevalence. Importantly, seropositivity alone is not sufficient to

establish a diagnosis of active Lyme disease in any clinical form [7, 11, 12]. In the absence of CSF analysis, neither neuroborreliosis nor Lyme-associated cranial neuritis can be confirmed. While lumbar puncture may be deferred in selected pediatric cases, its role remains central when neuroborreliosis is considered. Furthermore, alternative and more common causes of peripheral facial palsy, such as idiopathic (Bell's palsy) or viral etiologies, should be carefully considered, particularly in cases with inconclusive diagnostic findings [5, 13].

Current guidelines recommend oral doxycycline or intravenous ceftriaxone for the treatment of Lyme-associated peripheral facial palsy, typically administered for 14–21 days [7, 9, 14]. In the present case, a sequential regimen of intravenous ceftriaxone followed by oral doxycycline, in combination with corticosteroids, was used. This approach does not represent a standard treatment strategy and likely reflects the diagnostic uncertainty at the time of clinical decision-making. The use of corticosteroids in suspected Lyme-associated facial palsy remains controversial, with limited and conflicting evidence regarding their benefit, particularly in the absence of confirmed neuroborreliosis [15].

Supportive management, including eye protection and physical therapy, was implemented during hospitalization [16]. However, given the limited detail regarding the type and duration of rehabilitation, its contribution to clinical recovery cannot be clearly established [17].

This case also underscores the importance of aligning reported findings with documented clinical data. Follow-up neurological and audiological outcomes should be interpreted cautiously and, where relevant, clearly presented within the case description to support subsequent discussion [1, 7].

In conclusion, this case illustrates the diagnostic challenges in evaluating peripheral facial palsy in the context of positive *Borrelia* serology. The absence of CSF analysis precludes confirmation of neuroborreliosis, emphasizing the limitations of serological testing alone. Careful adherence to established diagnostic criteria, critical interpretation of laboratory findings, and consideration of alternative etiologies are essential to avoid overstated causal associations.

Ethics: Written informed consent was obtained from the patient's parents for treatment and publication of this case report. No identifiable patient information is included in this case report, and all data and images have been fully anonymized.

Conflict of interest: None declared.

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Суспектна периферна фацијална парализа удружена са лајмском болешћу код адолесцента са претходно дијагностикованим сензоринеуралним губитком слуха – дијагностички изазов

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САЖЕТАК

Увод Лајмска неуроборелиоза представља неуролошку манифестацију инфекције изазване бактеријом *Borrelia burgdorferi sensu lato*. Према утврђеним европским дијагностичким критеријумима, потврђена лајмска неуроборелиоза захтева плеоцитозу у цереброспиналној течности и доказе о интратекалној синтези антитела; међутим, ови критеријуми се не испуњавају увек у клиничкој пракси, што може довести до дијагностичке несигурности.

Приказ болесника Девојчица од 15 година примљена је због акутне периферне парализе десне стране лица. Серолошко тестирање је показало присуство позитивних *IgM* и *IgG* антитела на *Borrelia burgdorferi sensu lato*. Анализа цереброспиналне течности није извршена, чиме је потврда неуроборелиозе онемогућена. Магнетна резонанца мозга искључила је централне узроке парализе лица. Болесница је примила емпиријску антибиотску терапију у комбинацији са кортикостероидима, након чега је временом постигнуто делимично клиничко побољшање. Претходно дијагностикован сензоринеурални губитак слуха и инцидентални налази магнетне резонанце сматрани су неповезаним са акутном презентацијом.

Закључак Овај случај истиче ограничења приписивања периферне парализе лица лајмској болести на основу само серолошких налаза. У одсуству анализе цереброспиналне течности, дијагностичка несигурност остаје, наглашавајући потребу за опрезном интерпретацијом лабораторијских резултата и детаљним разматрањем алтернативних етиологија.

Кључне речи: неуроборелиоза; лајмска болест; периферна парализа фацијалиса; адолесцент; рехабилитација