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Original Article

Audio-Vestibular Function in Patients Diagnosed with Lyme Neuroborreliosis

Elisa Skovgaard Jensen[1,2], Jonas Bruun Kjærsgaard[3,4], Per Cayé-Thomasen[5,6], Dan Dupont Hougaard[3,4], Nicolas Lyneborg[1], Christian Thomas Brandt[6,7,8], Malene Kirchmann[1]

[1]Department of Otorhinolaryngology, North Zealand Hospital, University of Copenhagen, Hillerød, Denmark
[2]Department of Otorhinolaryngology, Zealand Hospital, University of Copenhagen, Køge, Denmark
[3]Department of Otolaryngology, Head & Neck Surgery and Audiology, Aalborg University Hospital, Aalborg, Denmark
[4]Department of Clinical Medicine, Aalborg University, Aalborg, Denmark
[5]Department of Otorhinolaryngology Head & Neck Surgery and Audiology, Rigshospitalet, University of Copenhagen, Denmark
[6]Department of Clinical Medicine, University of Copenhagen Faculty of Health and Medical Sciences, Copenhagen, Denmark
[7]Department of Pulmonary and Infectious Diseases, North Zealand Hospital, University of Copenhagen, Hillerød, Denmark
[8]Department of Infectious Diseases, Zealand Hospital, University of Copenhagen, Roskilde, Denmark

ORCID IDs of the authors: E.S.J. 0000-0001-5705-1560, J.B.K. 0000-0001-7332-9024, P.C-T. 0000-0002-6837-694X, D.D.H. 0000-0002-4164-1758, N.L. 0009-0001-4367-7973, C.T.B. 0000-0003-0201-0440, M.K. 0000-0001-9193-1609.

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BACKGROUND: This study assessed the audio-vestibular function and symptoms in patients diagnosed with Lyme neuroborreliosis (LNB).

METHODS: Patients with LNB were included prospectively. Diagnosis was based on a cerebrospinal fluid leukocyte count $\geqq 10 \times 10^6$ cells/L and a positive blood antibody production or intrathecal Borrelia burgdorferi antibody titer together with symptoms of LNB. Vestibular assessments included video head impulse test and a caloric test. Hearing was assessed by pure-tone audiometry at discharge and 30 days after hospitalization and compared to a healthy age- and sex-matched control dataset. Symptoms were assessed by a structured interview and the Dizziness Handicap Inventory (DHI).

RESULTS: Nineteen patients were included in the study. Four (21%) patients had vestibular dysfunction; 2 were unilateral and 2 were bilateral. The lateral semicircular canal was dysfunctional in 3 patients, the anterior semicircular canal in 2 patients, and the posterior semicircular canal in 1 patient. Sensorineural hearing loss, defined by audiometry, was present in 9 (47%) patients according to the audiometry at discharge and 10 (59%) patients during the follow-up audiometry. Hearing did not differ significantly from the age- and sex-matched control dataset. Vestibular dysfunction was not significantly associated with the total DHI score or with the mean PTA (Pure tone audiometry). The most commonly reported symptoms were peripheral nerve palsy (47%), dizziness (32%), fatigue (32%) and headache (32%).

CONCLUSION: Audio-vestibular function was affected in patients with LNB but correlated poorly with the patients' self-reported audio-vestibular symptoms.

KEYWORDS: Central nervous system infection, Lyme neuroborreliosis, otology, sensorineural hearing loss, vestibular diseases

INTRODUCTION

Lyme neuroborreliosis (LNB), caused by the tick-borne Borrelia burgdorferi spirochete, often poses a diagnostic challenge to physicians due to its ability to imitate other diseases. The spirochete can invade the central nervous system (CNS), resulting in LNB in up to 15% of the affected patients.[1-3] In Denmark, the incidence of neuroborreliosis is 4.7 per 100 000 inhabitants per year.[4] The most common manifestations of LNB in Europe are meningoradiculitis (Bannwarth syndrome) and lymphocytic meningitis.[2,4]

Corresponding author: Elisa Skovgaard, e-mail: elisaskovgaard@hotmail.com
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Although dizziness and vertigo are common complaints in LNB, studies on audio-vestibular disorders following LNB are lacking and only reported casuistically: clinical audio-vestibular manifestations include mild or moderate sensorineural hearing loss (SNHL) as well as vertigo due to vestibular nerve affection.[5-7]

Interestingly, audio-vestibular symptoms following other CNS infections are also common,[8-12] sensorineural hearing loss is the most common sequela following bacterial meningitis as the infection and subsequent inflammation in the CNS spreads to the cochlea through the cochlear aqueduct and the modiolus of the cochlea.[13] Hypothetically, such a spread could also occur in LNB, affecting cochlea and the closely related vestibular system.

Great progress has been made in the understanding of LNB. However, studies using systematic objective assessments of the vestibular end organ, such as the video head impulse test (vHIT) assessing the 6 semicircular canals (SCCs) and the caloric test, have never been described.

Vestibular dysfunction is associated with balance difficulties, decreased physical and social activities, as well as a reduced quality of life.[14] Early identification and rehabilitation of vestibular dysfunction are considered crucial to enable a fast and successful return to society.

The primary aim of this study was to determine the frequency and severity of vestibular dysfunction in patients with LNB.

METHODS

Study Design

The study was designed as a prospective observational study. The frequency and severity of vestibular dysfunction and SNHL was determined in patients with LNB. The study included patients referred over a 2-year period to Nordsjaelland University Hospital and Aalborg University Hospital in Denmark.

Audio-vestibular assessments were performed at the Departments of Otorhinolaryngology. Patients were scheduled for hearing assessment at discharge and for additional vestibular and hearing assessments 30 days after hospitalization. Sequelae and symptoms were assessed within 30 days after hospitalization at the Department of Infectious Diseases.

Inclusion and Exclusion Criteria

Inclusion criteria: patients 18 years old or older with neurological symptoms related to LNB and both of the following 2 inclusion criteria:

a. Cerebrospinal fluid (CSF) leukocyte count $\geq 10 \times 10^6$ cells/L.
b. Positive blood (probable LNB) antibody production or intrathecal *Borrelia burgdorferi* antibody production (definite LNB).

Exclusion criteria: history of: SNHL (except presbycusis), head trauma, stroke, CNS infection, drug abuse, inner ear surgery or disease, or intravenous administration of known ototoxic agents (e.g., gentamicin).

Vestibular Assessment

Vestibular assessments included objective testing with vHIT and caloric as well as subjective assessment of vertiginous symptoms by the Dizziness Handicap Inventory (DHI). Video head impulse test and caloric testing were conducted by experienced personnel, and test analysis was performed in collaboration with a senior consultant. Every test was preceded by a standard calibration according to the manufacturers' guidelines.

Video Head Impulse Test

Video head impulse test (vHIT) (ICS Impulse®, Natus Medical Incorporated©, Wisconsin, USA) was used to evaluate the function of all 6 semicircular canals by means of functional vestibulo-ocular reflex (VOR) testing. The function of the lateral SCC (lSCC), posterior SCC (pSCC), and anterior SCC (aSCC) was evaluated by means of both mean gain values and detection of pathological catch-up saccades.

Gain values of horizontal (lSCC) vHIT < 0.8 and vertical (pSCC or aSCC) vHIT < 0.7 were considered pathological based on normative data.[15] Saccades were systematically evaluated and defined as pathological based on a previously reported method.[16] A pathological vHIT was defined by pathological saccades and mean VOR gain values lower than the predefined cut-off values. Vestibular dysfunction was defined if a pathological test was found in at least 1 SCC.

Caloric Test - Bithermal Air Caloric

Caloric test (ICS-AirCal®, Natus Medical Incorporated©, Wisconsin, USA) was used to further evaluate the function of the lSCC. Stimulation was delivered at 24 and 50 degrees Celsius and each stimulation lasted for 60 seconds. Four stimulations were delivered in a "warm right," "warm left", "cool right," "cool left" stimulus pattern. Eye movement recordings continued for 4 minutes after cessation of each stimulation with 7-minute intervals between individual stimulations. Vestibular dysfunction measured by caloric test was defined as unilateral weakness if the asymmetry of the peak slow-phase velocity (SPV) of nystagmus was above 25% and bilateral weakness if the total SPV in the right ear was below 12 degrees/sec and the total SPV in the left ear was below 12 degrees/sec.

Dizziness Handicap Inventory

During vestibular examination, all patients filled out a Danish translation of the Jacobsen DHI, which is a 25-item self-reported questionnaire used to assess disability due to vestibular symptoms. Total scores range between 0 and 100 points (maximum).[17]

Audiological Assessment

Patients underwent standard speech and pure-tone audiometry at the standard frequencies between 0.125 and 8 kHz. Pure-tone average (PTA-4) was calculated as the thresholds measured at 0.5, 1, 2, and 4 kHz. In ears with conductive hearing loss, bone-conduction

MAIN POINT

- Audio-vestibular function was assessed using the video head impulse test, caloric testing, and pure-tone audiometry. Hearing loss and vestibular dysfunction were common findings but showed poor correlation with patients' self-reported audio-vestibular symptoms.

thresholds were used instead. Hearing loss was classified for each ear according to the PTA-4 hearing level (HL): No hearing loss (≤20 dB HL), mild hearing loss (21-40 dB HL), moderate hearing loss (41-55 dB HL), moderately severe hearing loss (56-70 dB HL), severe hearing loss (71-90 dB HL), and profound hearing loss (>90 dB HL). The mean PTA-4 from the first audiometry in both ears was compared to the mean PTA-4 from an age- and sex-matched normative data set provided by (International Organization for Standardization) ISO 7029.[18]

Patient Data

Clinical data were obtained on admission and at follow-up. Data on brain imaging, biochemical, and microbiological data were obtained from patient files. Symptoms and sequelae were obtained from patient files as well as through a structured interview within 30 days after admission.

Onset of LNB was defined as the onset of neurological symptoms. Duration of neurological symptoms was defined as the number of days from the onset of neurological symptoms to the first hospital admission.

Serology

Borrelia-specific antibodies in serum: the IDEIA *B. burgdorferi* IgG and IgM (Immunoglobulin G and M) (Oxoid Hampshire, UK) were used. Borrelia-specific intrathecal antibody index (AI): the IDEIA flagella antigen-based enzyme-linked immunosorbent assay LNB test (Oxoid Hampshire, UK) was used to detect intrathecal synthesis of B. burgdorferi specific IgG and IgM antibodies. An AI > 0.3 was considered positive according to the manufacturer's instructions.

Statistical Analysis

The statistical evaluation was performed by R Studio version 1.4.1717 (Posit Software, PBC; Boston, MA, USA). The Mann-Whitney test was used to compare groups. Fisher's exact test was applied to calculate the correlation of categorical data. Multiple regression analysis was used to calculate the age- and sex-adjusted association between vestibular dysfunction and PTA-4. The significance level was set to $P < .05$.

Ethics

The study was approved by the Datatilsynet (The Danish Data Protection Agency); Copenhagen, Denmark (2012-58-0004, I-Suite 03637, January 4, 2015). Informed, written consent was obtained from all subjects before enrollment and the study was performed according to the ethical principles of the World Medical Association Declaration of Helsinki. The Committees on Biomedical Research Ethics for the Capital Region of Denmark (vek@regionh.dk) did not require ethical approval (January 8, 2017, No. H-1-2012-086) as the study did not affect treatment, and testing was safe and noninvasive. The study was reported to clinicaltrials.gov (identifier: NCT03715569).

RESULTS

Of 35 identified cases with LNB, 19 patients were eligible for inclusion. Four patients were excluded due to possible ototoxic-related hearing loss and 1 patient was excluded because of severe cognitive impairment. Eight patients were not available for inclusion because of short stays at the hospital and 3 patients did not show up for their appointments. The median age was 53 years, interquartile range (IQR), 49-59 years. Fourteen (74%) patients were male. The duration of symptoms upon admission was 14 days, IQR, 11-59 days. Eight patients underwent brain imaging with computed tomography or magnetic resonance imaging with meningeal enhancement (n = 2), parenchymal enhancement (n = 1), and facial nerve enhancement (n = 1). A median CSF leukocyte concentration of 139 × 10^6 cells/L (IQR, 44-227), a median CSF protein concentration of 1.6 g/L (IQR, 0.9-1.9), and a median CSF lactate concentration of 2.3 mmol/L (IQR, 2.0-3.3) was observed.

The median duration of hospitalization was 3 days (IQR, 2-6 days). The most common symptoms during the first 30 days after hospitalization were peripheral nerve palsy (47%), dizziness (32%), fatigue (32%) and headache (32%) (Table 1). Nine patients (47%) presented with a facial nerve palsy; all but 1 was unilateral. No patients had any known hearing loss prior to hospitalization.

Vestibular Assessment

All patients underwent vestibular assessment after a median of 44 days from admission (IQR 28-60, range 13-98). All patients were assessed by vHIT (lSCC n = 19; aSCC n = 17; pSCC n = 17). Nine patients underwent additional caloric testing. However, 2 caloric tests were excluded because of technical errors. For the remaining 10 patients, caloric testing was not performed due to either difficulty in cooperation or technical failure.

Four patients (21%) had vestibular dysfunction; 2 were unilateral and 2 were bilateral (Table 2 and supplemental material case 1-4). Among these 4 patients, vHIT was pathological in 3 patients, and the caloric test was pathological in 1 patient. The lSCC was pathological in 3 patients, the pSCC in 1 patient, and the aSCC in 2 patients.

Additionally, 1 patient displayed pathological gain values in the aSCC bilaterally and pSCC unilaterally but in the absence of saccades.

All patients filled out the DHI questionnaire. In 14 patients, the total DHI score was 0 and in 5 patients, the score was above 0 (DHI scores 12, 12, 14, 26, and 42 respectively). The 4 patients with vestibular dysfunction had a total DHI score of 0.

Table 1. Symptoms in Patients with Lyme Neuroborreliosis Within 30 Days After Admission

Characteristics	Symptoms n = 19 [n (%)]
Peripheral nerve palsy*	11 (58)
Dizziness	6 (32)
Fatigue	6 (32)
Headache	6 (32)
Hyperacusis	2 (11)
Difficulty concentrating	2 (11)
Radicular pain	2 (11)
Sleep disturbances	2 (11)
Memory impairment	1 (5)
Subjective hearing loss	1 (5)
Tinnitus	1 (5)

*Including palsy of the facial nerve, bladder, m. rectus abdominis, left leg.

Table 2. Audio-Vestibular Test Results in 4 Patients with Vestibular Dysfunction

			Video Head Impulse Test												Caloric Test		Audiometry				DHI
			Anterior Semicircular Canal				Lateral Semicircular Canal				Posterior Semicircular Canal										
			Gain		Saccade		Gain		Saccade		Gain		Saccade				PTA-4 (dB) first		PTA-4 (dB) second		
Case	Age	Sex	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	UW%	Side	Right	Left	Right	Left	
1	45	M	**<0.7****	>0.7*	**S**	A	>0.8*	>0.8*	A	A	>0.7*	>0.7*	A	A	23	Right	3	2	5	3	0
2	78	M	**0.65**	0.75	A	A	**0.64**	**0.58**	**S**	**S**	0.97	**0.54**	A	**S**	na	na	**35**	**40**	**36**	**40**	0
3	56	M	na	na	na	na	**0.72**	**0.70**	**S**	**S**	na	na	na	na	na	na	**41**	**47**	**38**	**46**	0
4	58	M	>0.7*	>0.7*	A	A	>0.8*	>0.8*	A	A	>0.7*	>0.7*	A	A	**26**	Left	**35**	**33**	**30**	**31**	0

A, absent; DHI, Dizziness Handicap Inventory score; M, male; na, not assessed; PTA-4, pure-tone-average-4 (air-and bone conduction were the same); S, saccades; UW%, unilateral weakness in percentage.
Bolded values represent pathological findings as defined by the predetermined cut-off values.
*Normal, the precise value is lost due to technical error.
**Pathological low gain value documented in the patient's medical charts, the precise value is lost.

Pure-Tone Audiometry

Nineteen patients underwent standard pure-tone and speech audiometry. The initial audiometry was performed after a median of 5 days following admission (IQR 3-12, range 1-78 days). Seventeen patients attended the second audiometry after a median of 70 days following admission (IQR 38-90, range 24-140 days). The 2 patients not attending the second audiometry had normal hearing thresholds at the initial audiometry.

Sensorineural hearing loss was present in 9 (47%) patients according to the initial audiometry and 10 (59%) patients according to the second audiometry (see Table 3). Hearing loss among patients with SNHL was classified as mild or moderate. Hearing loss was predominantly detected in the high frequency range (4-8 kHz) and bilateral in all cases except for 2 patients (Figure 1)

The hearing threshold levels in the 17 patients attending the second audiometry remained permanent and unchanged in 13 (76%) patients, improved unilaterally in 3 (18%), and deteriorated unilaterally in 1 (6%) as a late onset of SNHL.

Though the mean PTA-4 in the cohort was 8.14 dB higher than in the healthy age- and sex-matched control dataset, the difference in PTA-4 was not significant ($P = .075$).

Five (56%) out of 10 patients with hearing loss also presented with concomitant facial nerve palsy.

Correlations Between Vestibular Function, Total Dizziness Handicap Inventory Scores, and Pure-Tone Average

Vestibular dysfunction was not associated with the total DHI scores. Patients with vestibular dysfunction had a mean PTA-4 that was 13 dB higher than patients with normal vestibular function (28 dB vs. 15 dB). However, vestibular dysfunction was not significantly associated with mean PTA-4, neither with ($P = .10$), 95% CI: (−2;21) nor without age- and sex-adjustments.

DISCUSSION

To the authors' knowledge, this is the first study to assess vestibular function and hearing loss in a clinical prospective trial with a cohort of patients diagnosed with LNB.

Vestibular Impairment Among Patients with Lyme Neuroborreliosis

Vestibular dysfunction was present in 4 (21%) of 19 patients with LNB. During vestibular evaluation, 5 patients (26%) had self-reported

Table 3. Classification of Hearing Loss in Patients with Lyme Neuroborreliosis. Classification of Hearing Loss is Based on Pure-Tone-Average in the Worse Hearing Ear

Hearing Loss (PTA-4)	First Audiometry n = 19 [n (%)]	Second Audiometry n = 17* [n (%)]
No (≤20 dB)	10 (53)	7 (41)
Mild (21-40 dB)	8 (42)	10 (59)
Moderate (41-55 dB)	1 (5)	0 (0)
Moderate-profound (>56 dB)	0 (0)	0 (0)

PTA-4, pure-tone-average-4.
*2 patients with normal hearing at the first audiometry did not show up.

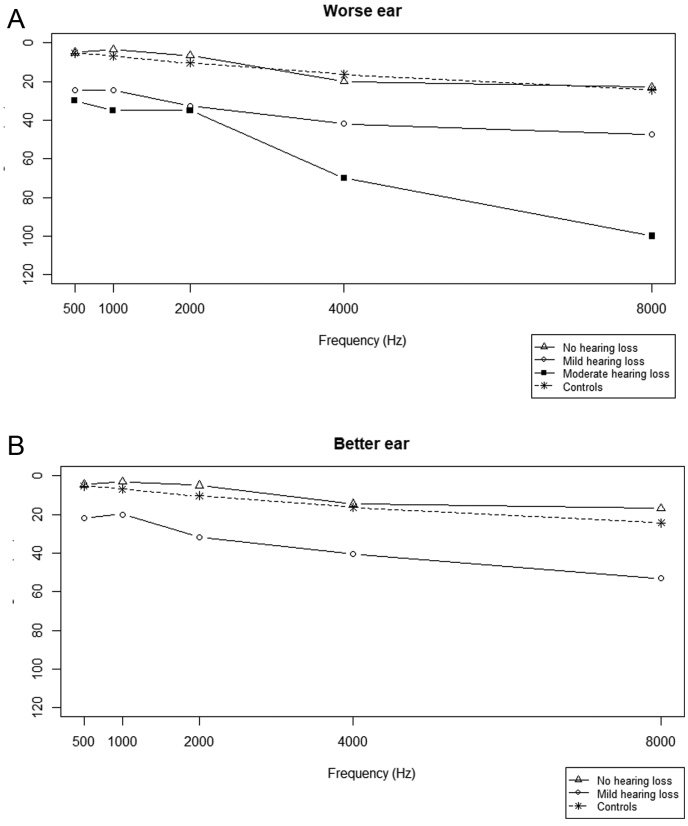

Figure 1. Mean pure-tone thresholds in patients with Lyme neuroborreliosis according to the first audiometry. A. Data obtained from the worse hearing ear. B. Data obtained from the better hearing ear. Data represent patients with no (PTA-4 ≤ 20 dB), mild (PTA-4, 21-40 dB), or moderate hearing loss (PTA-4, 41-55 dB). PTA, pure-tone average.

vestibular complaints according to the DHI questionnaire. However, none of the patients with a pathological vestibular test had a total DHI score above zero. The low correspondence between objective vestibular dysfunction and self-reported dizziness is well known,[11,19] and may be caused by central compensation. In the present study, only 1 patient with objective vestibular dysfunction attended an additional vestibular follow-up (case 3, Table 2), and even though vestibular dysfunction persisted in this case, no assumptions can be made about the persistency of vestibular impairment in LNB.

Being the first study to assess vestibular function in patients with LNB, no comparable studies exist. However, the incidence of Lyme disease, defined by positive serum antibodies against B. burgdorferi, in a cohort of vertigo patients, was 2%-14% with manifestations mimicking benign paroxysmal positional vertigo and vestibular neuritis.[20-23] Further, Selmani et al[21] detected positive antibodies in the blood against B. burgdorferi in 11 out of 102 patients presenting with vertigo. In these patients, clinical manifestations with Meniere-like symptoms were present in 54%.

A comparison of previous studies on Lyme disease poses difficulties because the diagnostic criteria vary. Further, the use of serology for diagnosing LNB is associated with several challenges: individuals with an early infection may test negative as antibodies take weeks to develop, and serological tests are unable to distinguish between prior exposure and an active infection.[24]

Hearing Loss Among Patients with Lyme Neuroborreliosis

Sensorineural hearing loss was present in 10 (53%) patients with LNB, which is slightly higher than the rate of 15%-44% previously been reported.[25,26] Subjective hearing loss, reported by only 5 (1%), corresponds poorly with the objective findings of SNHL. The low self-reported prevalence of hearing loss is consistent with the findings of other studies.[27,28] This may be explained by the fact that the SNHL is mild and predominantly involves the high frequencies. Sensorineural hearing loss may have occurred gradually before admission, which is slightly supported by the borderline insignificant difference with the age- and sex-matched control data. Furthermore, the patients may have paid more attention to the more severe symptoms of LNB than the accompanying hearing loss.

The present study found that LNB patients with vestibular dysfunction had a higher degree of hearing loss compared to LNB patients with normal vestibular function. However, this tendency was not significant.

Pathophysiology

Borrelia spirochetes invade the CNS by spreading along structures such as the peripheral nerves or the bloodstream, where they can induce direct cytotoxicity, neurotoxic mediators, or autoimmune reactions.[29] The pathophysiology has not yet been clarified; peripheral nerve involvement has been suggested to be caused by multifocal axonal loss.[30,31] Other studies have found spirochetes in the dorsal root ganglia but not in the peripheral nerves.[32]

The limited studies on Lyme disease do not allow one to draw conclusions on the pathophysiology behind an associated vestibular impairment inasmuch as the mechanisms may differ between those with and without LNB. In the present study, all 6 SCCs by vHIT were tested, thereby assessing the function of both the inferior and superior vestibular nerves. In LNB patients with vestibular dysfunction, the 3 SCCs were occasionally affected, yet no distinct pattern was observed. Therefore, the dysfunction may lie in the vestibular nerve (meningoradiculitis) as well as within the individual end organ. The lSCC was the most affected canal, and in 50% of these cases, isolated vestibular damage in the lSCC with hearing loss was observed, which points in the direction of structural damage rather than damage to the vestibulocochlear nerve. In CNS infections, the inflammation may spread to all compartments of the inner ear, and this is supported by the fact that the LNB patients with vestibular dysfunction had a higher degree of accompanying hearing loss. Damage to the vestibulocochlear nerve would also induce hearing loss; however, all ipsilateral SCCs were expected to be affected at the same time.

Surprisingly, vestibular dysfunction was registered bilaterally in 2 (50%) and hearing loss in 8 (80%) patients, suggesting a hematogenic spread or otherwise a more unlikely direct spread through the cochlear aqueduct bilaterally. Further studies would be needed to clarify these yet largely unknown pathophysiological processes.

Study Limitations

The study population was relatively small, making it difficult to draw any significant conclusions. The strength of the study was also weakened by the fact that not all patients underwent caloric testing. Furthermore, performing and analyzing vHIT and caloric tests

is dependent on staff expertise as well as a high degree of patient cooperation, making the results susceptible to additional bias. The frequency of self-reported symptoms of vestibular deficits was much higher than the frequency detected by the vestibular test battery. The function of the saccule and utricle was not assessed in this study and could therefore be an undetected site of lesion. Adding evaluation with vestibular evoked myogenic potential (VEMP) testing would have given a complete overview of the vestibular end organ function. Vestibular evoked myogenic potential, on the other hand, may face other difficulties as cVEMP (cervical Vestibular Evoked Myogenic Potential) is known to be influenced by conductive hearing loss, which could lead to a false detection of sacculus hypofunction.[33] Furthermore, the patients may have had vestibular dysfunction prior to the enrollment in this study, however, age does not seem to affect vestibular function,[34] as well as applied conservative diagnostic and inclusion criteria to rule out prior vestibular deficits.

CONCLUSION

The high frequency of SNHL among patients with LNB suggests that routine audiological evaluation may be warranted. The frequency of vestibular dysfunction was low, and no unambiguous conclusion on this matter can be drawn. Therefore, larger studies on this topic are needed. Furthermore, self-reported audio-vestibular symptoms were not a reliable indicator of audio-vestibular impairment.

Data Availability Statement: The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Ethics Committee Approval: N/A.

Informed Consent: Written informed consent was obtained from the subjects who agreed to take part in the study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – C.T.B., P.C.T., M.K.; Design – C.T.B., P.C.T., M.K.; Supervision – C.T.B., P.C.T., M.K.; Resources – C.T.B., D.D.H., J.B.K., M.K.; Materials – E.S.J.; Data Collection and/or Processing – E.S.J., M.K., C.T.B., N.L., J.B.K., D.D.H.; Analysis and/or Interpretation – E.S.J., M.K., C.T.B., N.L., J.B.K., P.C.T., D.D.H.; Literature Search – E.S.J.; Writing Manuscript – E.S.J., M.K.; Critical Review – M.K., C.T.B., N.L., J.B.K., D.D.H., P.C.T.

Declaration of Interests: The authors have no conflicts of interest to declare.

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SUPPLEMENTAL MATERIAL, CASE DESCRIPTIONS

Case 1:

A 44-year-old man was diagnosed with Lyme neuroborreliosis (LNB) and left sided peripheral facial nerve palsy after a tick bite seven months prior to admission. He had a positive blood IgG against B. Burgdorferi and a CSF leucocyte count of 31 x 10^6 cells/L. He was treated with peroral doxycycline for 14 days in combination with prednisone.

Through the course of disease, the patient had no vestibular complaints and the total DHI score was zero. However, during the vestibular assessment follow-up, one month after admission, unilateral vestibular dysfunction was detected: vHIT showed a pathological low mean gain value as well as catch-up saccades in the right anterior SCC. The rest of the tests were normal. The patient did not show up for any further follow-ups and the facial nerve palsy fully remitted.

Case 2:

A 78-year-old man was diagnosed with LNB after exposure to tick bites and 14 days with radiating pain in his left arm. He had a positive intrathecal IgM against B. burgdorferi and a CSF leucocyte count of 10 x 10^6 cells/L. He was treated with peroral doxycycline for 14 days.

Even though the patient did not present with any vestibular symptoms and the total DHI score was 0, the vestibular tests, one months after admission detected bilateral vestibular dysfunction in the ISCCs as well as a left sided vestibular dysfunction in pSCC. Furthermore, the right aSCC displayed lowered gain value but both without catch-up saccades. He was classified with a persistent bilateral mild SNHL.

The patient did not show up for any further follow-ups.

Case 3:

A 56-year-old man was diagnosed with LNB and a left sided peripheral facial nerve palsy after 11 days of pain in his left arm and one day of facial weakness. A positive blood IgM and IgG against B. Burgdorferi and a CSF leucocyte count of 131 x 10^6 cells/L was found. He was treated with i.v. ceftriaxone for five days and then switched to a combination of valaciclovir and peroral doxycycline for 14 days.

During the first vestibular follow up, 2 months after admission, the patient complained of intermittent left sided lateralization during walks in the dark and bilateral hearing loss. vHIT revealed bilateral covert saccades and reduced mean gain values in the lateral SCCs. The vertical SCCs as well as the caloric test was not performed on the patients request.

Upon the second vestibular examination, 5 months later, vHIT showed bilateral reduced gain values in the lateral SCC as well as covert saccades in the left lateral SCC.

Hearing was classified as a moderate sensorineural hearing loss, that persisted. The patient was assigned bilateral hearing aids. The facial palsy fully recovered. No radiology was performed.

Case 4

A 58-year-old man was diagnosed with LNB with a positive IgM and IgG intrathecal antibody against *b. Burdorferi* and a CSF leucocyte count of 178 x 10^6 cells/L. He was treated with iv. ceftriaxone and acyclovir for 6 days until serology confirmed the diagnosis. He was then switched to peroral doxycycline for an additional 8 days.

He had no vestibular complains and the DHI score was 0.

Vestibular follow-up revealed borderline vestibular dysfunction as the caloric test detected unilateral left sided weakness of 26%. The vHIT was normal in all SCCs.

The patient was classified with a persistent mild bilateral SNHL. CT angiography and MRC was normal. The patient did not attend any further follow-ups.