



Superior vestibular neuritis: improved detection using FLAIR sequence with delayed enhancement (1 h)

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Received: 2 July 2019 / Accepted: 6 September 2019 / Published online: 17 September 2019
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Abstract

Introduction Vestibular neuritis is the second cause of vertigo and new imaging protocols using delayed FLAIR with double-dose of gadolinium are proposed for its diagnosis. Our aim is to demonstrate that a single dose of gadolinium is sufficient.

Methods Thirty-three patients with a unilateral vestibular neuritis are compared to a control group. All patients underwent a FLAIR sequence, 1 hour after intravenous injection of a single dose of gadolinium, on a 1.5 Tesla MRI. Two radiologists analyzed the enhancement intensity of the superior (sup VN) and inferior vestibular nerve (inf VN) and ratios to the signal of the cerebellum were calculated (supVN/C). The statistics were performed using Bayesian analysis.

Results A strong enhancement of the sup VN was observed on the pathological side in 85% of patients with vestibular neuritis. The average signal intensity of the pathological sup VN (139 units \pm 44) was more than two times the average intensity in the control group (58.5 units \pm 5). The average ratios supVN/C were significantly different between the pathological side in vestibular neuritis (2.43 units \pm 0.63) and the control group [1.16 \pm 0.14 (Pr(diff > 0) = 1)]. A delayed enhancement > 71.5 units had a sensitivity of 96% and a specificity of 100% for the diagnosis of superior vestibular neuritis.

Conclusion A delayed FLAIR sequence, acquired 1 hour after a single dose of gadolinium injection, is a useful method for the diagnosis of vestibular neuritis. An enhancement of the sup VN > 71.5 units was in favor of the diagnosis.

Keywords Neuritis · Vestibular neuronitis · Magnetic resonance imaging · Vestibulocochlear nerve · Vertigo

Abbreviations

MRI	Magnetic resonance imaging
FLAIR	Fluid attenuated inversion recuperation
BPPV	Benign positional paroxysmal vertigo
VHIT	Video head impulse test
Sup VN	Superior vestibular nerve
InfVN	Inferior vestibular nerve

SupVN/C	Ratio of the signal intensity of the superior vestibular nerve to the cerebellum
InfVN/C	Ratio of the signal intensity of the inferior vestibular nerve to the cerebellum

Key Points

Delayed contrast-enhanced FLAIR is useful for the diagnosis of vestibular neuritis.

An enhancement of the nerve > 71.5 units was in favor of neuritis.

Diagnosis of vestibular neuritis can be made with a single dose of gadolinium.

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Introduction

Vestibular neuritis is an acute dysfunction of the peripheral vestibular system, usually unilateral. The main symptom is the sudden occurrence of an intense rotatory vertigo, with a dizziness that usually lasts for a few days, associated with imbalance, nausea and vomiting, without any hearing loss or neurological symptoms. Vestibular neuritis is the second most frequent cause of vertigo after Benign Positional Paroxysmal Vertigo (BPPV) responsible of approximately 7% of peripheral vertigos [1]. Its etiology remains unknown although the most commonly accepted hypothesis is a viral origin, especially related to an infection by Herpes Simplex 1 in the vestibular ganglion or a reactivation of the virus [2, 3]. The diagnosis of vestibular neuritis is currently based on combination between clinical symptoms, unilateral caloric canal paresis and impairment of the video head impulse test (VHIT) corresponding to a vestibular nerve territory (superior, inferior or both). Until now, imaging is commonly used to exclude neurological differential diagnosis in atypical cases (such as cerebellar tumor, cerebellar infarct or auto-immune pathologies such as multiple sclerosis). The majority of authors tried to image vestibular neuritis using T1-weighted images after intravenous injection of Gd-chelates, but failed to obtain significant results [4, 5]. Karlberg et al. were able to describe a slight enhancement of the vestibular nerve on their T1-weighted gadolinium-enhanced images in two cases of vestibular neuritis, but they had to use three times the standard dose of gadolinium (Gd-DTPA, 0.3 mmol/kg) to do so [6]. In 2014, Park et al. suggested in a case-report that fluid attenuated inversion recovery (FLAIR) images after intravenous gadolinium injection could be useful for the positive diagnosis of vestibular neuritis, their images showing a high focal intensity of the vestibular nerve on the pathological side [7]. This finding was confirmed by Buyn et al. in 2018, but they used double-dose of gadolinium chelates for their delayed FLAIR images [8].

Our aim is to demonstrate that a single dose of gadolinium is sufficient for the diagnosis of unilateral acute vestibular neuritis on a delayed FLAIR sequence—acquired 1 h after intravenous injection of Gd-chelates—compared to a control group.

Material and methods

Ethical considerations

The Ethics Committee of our institution approved the study (approval number FC/dossier 2017-29). The study has been registered on clinicaltrials.gov (NCT03452410). The patients gave their consent to participate to the study.

Study population

Consecutive patients who presented at our institution from January 2017 to January 2018 with a typical diagnosis of acute unilateral vestibular neuritis were included. All patients were evaluated by a clinical examination by an otorhinolaryngologist who performed an audiogram and a VHIT (Video Head Impulse Test). In the acute phase, the clinical examination revealed a unilateral, spontaneous horizontal nystagmus, unidirectional and normal-side-beating, associated with a postural deviation to the pathological side and an impaired vestibulo-ocular reflex of the superior and lateral semicircular canals on the pathological side. The patients did not have any other neurological symptoms. The VHIT was abnormal on the side of symptoms and the audiogram was normal. Exclusion criteria were: atypical diagnosis, bilateral vestibular neuritis, benign or malignant tumor of the cochlea-vestibular nerve and concomitant infections or inflammatory inner/middle ear pathologies (such as labyrinthitis). Thirty-three patients met the criteria and were included.

In addition, a control group of ten patients (20 ears) presenting only with acute sensorineural hearing loss, without any other symptom (no dizziness, imbalance or neurological symptom) was included.

MRI protocol

MRI was performed within the first month after the onset of symptoms. All participants underwent a standard CPA MRI with an additional axial FLAIR sequence, acquired 1 h after intravenous injection of a single dose of gadolinium, on a 1.5 Tesla MRI (Philips, Ingenia 1.5 T, Strasbourg, FRANCE) using an eight-channel head coil. This sequence was acquired 1 hour after intravenous injection of 0.1 mmol/kg (0.2 mL/kg) of gadoterate meglumine (Dotarem®, Guerbet, Roissy, France). The study box was placed parallel to the orbital roof. The acquisition parameters were: echo time (TE minfull): 546 ms, repetition time (TR): 7000 ms, IR time: 2250 ms, FOV: 230 × 198 × 30 mm, flip angle: 60°, number of excitations (Nex): 6, bandwidth: 189.9 kHz, voxel: 0.85 × 0.95 × 0.8 mm, slice thickness after reconstruction 0.4 mm. The acquisition time was 9 min 20 s.

MRI analysis

Visual analysis

A junior and a senior radiologist, specialized in head and neck imaging (5 and 35 years of experience), read the axial FLAIR sequence blinded to the clinical presentation. They visually analyzed the enhancement of the superior vestibular

nerve (utrículo-ampullar nerve) and adjacent ampullas (anterior, lateral, and posterior) on FLAIR-weighted images compared to the normal contralateral side and to both sides in the control group: Grade 0 was a normal nerve with no enhancement seen; Grade 1 was an intermediate enhancement; Grade 2 was a strong enhancement, very bright compared to adjacent normally enhancing structures.

Quantitative analysis

For the quantitative analysis, the signal intensity of the vestibular nerve and ampullas was measured using elliptical regions of interests (ROIs) based on a compartmental reading of the delayed FLAIR sequence. For each ear, a 0.5-mm² ROI was placed on an axial section on the superior vestibular nerve (utrículo-ampullar) and the inferior vestibular nerve (sacculo-ampullar). An additional 1.5-mm² ROI was placed on the grey matter of the ipsilateral cerebellum. The signal intensity ratios of the superior vestibular nerve and inferior vestibular nerve to that of the signal intensity of the cerebellum (supVN/C, infVN/C, respectively) were calculated to avoid bias due to patient-related artifacts. All measurements and visual analyses were carried out using the open-source Digital Imaging and Communication in Medicine (DICOM) OsiriX® software (available at: <https://www.osirix-viewer.com/>).

All structures are also assessed on both sides in the control group. All measures are carried out on the pathological side in vestibular neuritis patients and compared to the normal contralateral side.

Statistical analysis

Continuous variables were expressed as means with their standard deviation (SD) [range] and median (means ± SD [range], median). Categorical variables were expressed as frequencies and percentages for each modality. The interobserver agreement for the visual analysis was calculated using Cohen's κ . Between-group discrimination was estimated and described using ROC curves with its associated AUC [95% confidence interval], the optimal discriminating cutoff and the sensitivity and specificity for this cutoff.

Inferential statistical analyses were conducted using Bayesian methods [9, 10]. The data were repeated since each examination was read twice. The analysis used linear mixed models with a fixed group effect, fixed reader effect and a random subject effect. Because of the very high between-readers' reproducibility (intraclass correlation coefficients of about 0.98 for each measurements), results for groups are expressed as averaged over the two readers. Mean differences were estimated using normal lowly informative prior distribution ($N[\mu = 0, \sigma = 10]$) [11] in each case. The between-groups differences are presented as a between-groups mean

difference and its 95% credibility interval, with the probability that the mean difference is positive, i.e., $\text{Pr}(\text{diff} > 0)$. Larger values of this probability (near 1) indicate a higher likelihood of the difference being positive. A probability approaching 0.5 must be interpreted as the absence of difference. It should be noted that the Bayesian analysis does not use a (frequentist) p value and this probability must not be confused with a p value.

The Bayesian analyses were run with JAGS software (version 4.2.0.) and R software (version 3.2.0, available at <https://www.r-project.org/>) [12, 13]. After a burn-in of 5000 updates, 100,000 iterations were performed and convergence was checked using trace plots of the sample values for each iteration. Convergence was observed in each case.

Results

33 patients met the criteria and were included (18 men and 15 women). The clinical characteristics of the patients and control group are summarized in Table 1. All patients were analyzed and no patients were excluded.

Visual assessment

On the visual analysis, a strong delayed (grade 2) enhancement of the superior vestibular nerve (sup VN) was observed on the pathological side in 85% of the cases ($n = 28$) with a clinical vestibular neuritis (Fig. 1), three patients (9%) presented with a grade 1 enhancement and two patients (6%) with vestibular neuritis were considered normal (grade 0), with excellent interobserver agreement ($\kappa = 1.0$) (Table 2).

No pathological enhancement (grade 0) was seen in the control group (Fig. 2) or on the normal contralateral side of patients with vestibular neuritis.

Table 1 Clinical characteristics of patients with vestibular neuritis patients and control group

	Patients $n = 33$	Control group $n = 10$
Sex		
Men	18 (55%)	6 (60%)
Woman	15 (45%)	4 (40%)
Mean age (years ± SD [range])	48.9 ± 16.4 [19–85]	46 ± 17 [19–68]
Symptoms		
Vertigo	100% ($n = 33$)	0
Nausea/vomiting	50% ($n = 17$)	0
Tinnitus	12% ($n = 4$)	0
Acute sensorineural hearing loss	0	100%

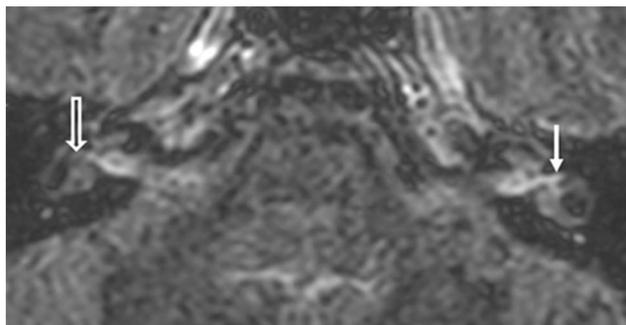


Fig. 1 Delayed contrast-enhanced FLAIR sequence, axial plane, acquired 1 h after intravenous gadolinium (single dose) in a patient with a left vestibular neuritis, showing an enhancement of the superior vestibular nerve (>71.5 units, arrow). There is no significant enhancement of the contralateral superior vestibular nerve (60 units, empty arrow)

Quantitative analysis

Superior vestibular nerve

The average enhancement of the sup VN on the pathological side in vestibular neuritis was $139 \text{ units} \pm 44$ [55–273 units], 131 compared to 78.5 ± 18 [50–110], 78 on the normal contralateral side, $78.6 [+70.3, +105.4]$ $\text{Pr}(\text{diff} > 0) > 0.999$ and to $58.5 \text{ units} \pm 5$ [50–70], 60 in the control group, $87.9 [+70.3, +105.4]$ $\text{Pr}(\text{diff} > 0) > 0.999$ (Fig. 3). The optimal cutoff point to differentiate between a normal and a pathological vestibular nerve on delayed FLAIR sequence was 71.5 units [67.5; 82.5], with an $\text{AUC} = 0.9824$ [0.9575; 1] (Fig. 4). With this cutoff, the delayed FLAIR correctly diagnosed superior vestibular neuritis with a sensitivity of 96% and a specificity of 100%.

The average ratio of the signal intensity of the superior vestibular nerve on the signal of the cerebellum (supVN/C) was also higher on the pathological side in vestibular neuritis ($2.43 \text{ units} \pm 0.63$ [1.04–4.5 units], 2.40) significantly different if compared to the control group (1.16 ± 0.14 [0.94–1.44 units], 1.14), $1.43 [+1.18, +1.67]$ $\text{Pr}(\text{diff} > 0) > 0.999$ and to the normal contralateral side (1.40 ± 0.33 [0.80–2.44 units], 1.4), $1.37 [+1.24, +1.51]$ $\text{Pr}(\text{diff} > 0) > 0.999$ (Fig. 5). The difference between the control group and the normal contralateral side for supVN/C was low or negligible $0.06 [-0.192, +0.30]$.

Inferior vestibular nerve

The difference regarding the signal intensity of the inferior vestibular nerve (inf VN) on the pathological side in case of neuritis and the control group ($\text{Pr}(\text{diff} > 0)$ 10.16 [0.76 + 19.42]) and regarding the normal contralateral side (3.22 [1.50 + 4.97]) was also low or negligible, with

an average enhancement of the inferior vestibular nerve on the pathological side in vestibular neuritis ($80 \text{ units} \pm 15$ [55–125 units], 80) compared to the control group ($56.2 \text{ units} \pm 6$ [44–64], 57) and ($78.5 \text{ units} \pm 17.6$ [50–110 units], 78) on the normal contralateral side.

Same observations were made for the average ratio of the signal intensity of the inferior vestibular nerve on the cerebellum (infVN/C) between the pathological side in vestibular neuritis and the control group ($0.06 [-0.09 + 0.22]$) and regarding the normal contralateral side (0.06 [0.03 + 0.10]), with an average ratio infVN/C of $1.43 \text{ units} \pm 0.22$ [0.78–2.08 units], 1.45 in patients with vestibular neuritis, compared to $1.11 \text{ units} \pm 0.15$ [0.85–1.45 units], 1.12 in the control group and the normal contralateral side ($1.26 \text{ units} \pm 0.33$ [0.71–2.04 units], 1.28 (Fig. 6).

Discussion

Synopsis of key/new findings and strengths of the study

If contrast-enhanced FLAIR imaging has been used for a few years to image facial neuritis (for its diagnosis and differential diagnosis), no consensus appears regarding the imaging protocol in acute vestibular neuritis. In our study, we have been able to diagnose unilateral superior vestibular neuritis in 85% of the cases using a delayed FLAIR sequence acquired 1 hour after an intravenous injection of a single dose of gadolinium (0.1 mmol/kg of Gd-DOTA, Dotarem, Guerbet, Paris, FRANCE), while at the same time being able to exclude other potential differential diagnosis. An enhancement > 70 units of the superior vestibular nerve was in favor of an acute vestibular neuritis.

Comparisons with other studies

This confirms the findings of Buyn et al., who diagnosed vestibular neuritis in 69% of their cases, using a double-dose of gadolinium chelates (0.2 mmol/kg) Gd-diethylenetriamine penta-acetic acid (DTPA, Bono-I; CMS, Korea) for their delayed FLAIR images [8]. Other authors who used T1-weighted images, such as Karlberg et al., had to use three times the usual dose of Gadolinium (triple dose of Gd-DTPA, 0.3 mmol/kg) to obtain such results [6]. With our delayed-FLAIR technique, we used the standard single dose of gadolinium (GD-Dota 0.1 mmol/kg).

Contrast-enhanced FLAIR images have been used for years to image acute facial neuritis, by multiple authors such as Nakata et al., Lim et al. and Chung et al. [14–16]. Lim et al., in their study on 36 patients suffering from facial neuritis, also the same contrast-agent at the same concentration as in our study (0.1 mmol/kg—single dose—of Gd-DOTA,

Table 2 Clinical characteristics of patients with vestibular neuritis

Patient number	Age (years)	Gender	Rotatory vertigo	Nystagmus	Romberg	Hearing loss	VHIT	Grade of enhancement on analysis (0–1 or 2)	Enhancement of the superior VN > 71.5
1	37	M	+	+	+	–	Superior vestibular neuritis	1	Yes
2	25	M	+	+	+	–	Superior vestibular neuritis	0	Yes
3	57	M	+	+	+	–	Superior vestibular neuritis	2	Yes
4	58	M	+	+	+	–	Superior vestibular neuritis	2	Yes
5	25	M	+	+	+	–	Superior vestibular neuritis	2	Yes
6	35	F	+	+	+	–	Superior vestibular neuritis	2	Yes
7	45	M	+	+	+	–	Superior vestibular neuritis	2	Yes
8	65	F	+	+	+	–	Superior vestibular neuritis	2	Yes
9	30	F	+	+	+	–	Superior vestibular neuritis	2	Yes
10	78	M	+	+	+	–	Superior vestibular neuritis	2	Yes
11	85	M	+	+	+	–	<i>Missing data</i>	0	No
12	52	M	+	+	+	–	Lateral canal areflexia	1	Yes
13	58	M	+	+	+	–	Superior vestibular neuritis	2	Yes
14	76	M	+	+	+	–	Superior vestibular neuritis	2	Yes
15	56	M	+	+	+	–	Superior vestibular neuritis	2	Yes
16	54	M	+	+	+	–	Superior vestibular neuritis	2	Yes
17	37	F	+	+	+	–	Superior vestibular neuritis	2	Yes
18	54	F	+	+	+	–	Superior vestibular neuritis	2	Yes
19	27	F	+	+	+	–	Superior vestibular neuritis	2	Yes
20	19	F	+	+	+	–	Superior vestibular neuritis	2	Yes
21	43	F	+	+	+	–	Superior vestibular neuritis	2	Yes
22	60	M	+	+	+	–	Superior vestibular neuritis	2	Yes
23	60	M	+	+	+	–	Superior vestibular neuritis	2	Yes
24	46	M	+	+	+	–	Superior vestibular neuritis	2	Yes
25	49	F	+	+	+	–	Superior vestibular neuritis	2	Yes
26	47	M	+	+	+	–	Superior vestibular neuritis	2	Yes
27	50	M	+	+	+	–	Superior vestibular neuritis	2	Yes

Table 2 (continued)

Patient number	Age (years)	Gender	Rotatory vertigo	Nystagmus	Romberg	Hearing loss	VHIT	Grade of enhancement on analysis (0–1 or 2)	Enhancement of the superior VN > 71.5
28	52	F	+	+	+	–	Superior vestibular neuritis	2	Yes
29	66	F	+	+	+	–	Missing data	0	No
30	53	F	+	+	+	–	Missing data	0	Yes
31	32	F	+	+	+	–	Missing data	1	Yes
32	61	F	+	+	+	–	Missing data	0	Yes
33	22	F	+	+	+	–	Missing data	0	Yes

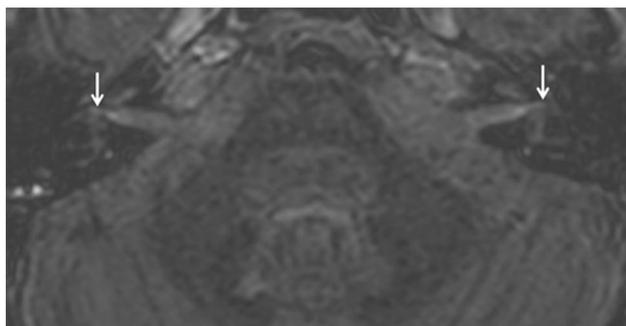


Fig. 2 Delayed contrast-enhanced FLAIR sequence, axial plane, acquired 1 h after intravenous gadolinium sequence in a normal patient showing no pathological enhancement of the superior vestibular nerves on the right and left side (50 units, arrows)

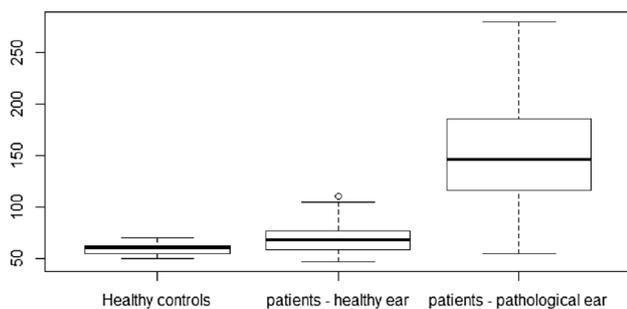


Fig. 3 Comparison between the average enhancement of the sup VN on the pathological side in vestibular neuritis (139 units \pm 44 [55–273 units], 131) compared to 58.5 units \pm 5 [50–70], 60) in the control group: the enhancement of the supVN is more than two times higher in vestibular neuritis (78.6 [+70.3, +86.9] Pr(diff>0) > 0.999)

Dotarem, Guerbet, Paris, FRANCE) and found an enhancement of segments of the facial nerve in 100% of their cases [15]. They also concluded that FLAIR sequences were superior to contrast-enhanced T1-weighted sequences injection for the diagnosis of acute facial neuritis. Normal moderate neural enhancement may be observed around nerves (either facial or vestibular) and are related to the presence of flux in the circumneural arteriovenous plexus, but a marked

enhancement of the nerve is pathological and occurs in neuritis [17, 18].

On both our visual analysis and the quantitative evaluation using ROIs, a strong pathological enhancement of the superior vestibular nerve (utrículo-ampullar nerve) was observed in 85% of the cases on the side of the clinical symptoms. According to the literature, vestibular neuritis affects both superior and inferior vestibular nerves, but the involvement of the superior vestibular nerve appears to be more frequent, as described by Taylor et al. [19]. We found matching results in our study, we observed a strong enhancement on delayed FLAIR sequences of the superior portion of the vestibular nerve in 85% of the cases. VHIT analysis confirmed our results and showed an involvement of the superior portion of the vestibular nerve (no patient presented with a total involvement of the nerve on VHIT in our study). The absence of significant enhancement of the inferior portion of the vestibular nerve (sacculo-ampullar nerve) may be related to the absence of involvement of the inferior vestibular nerve on VHIT in all 33 patients. Furthermore, the small size of the inferior portion of the nerve may have rendered the measurement difficult, and hazardous to reproduce. The superior vestibular nerve was easier to analyze, with excellent interobserver agreement ($\kappa=1$). This preliminary study on 33 patients with unilateral acute vestibular neuritis, shall be completed by a study including an increased number of patients to confirm our findings.

Clinical applicability of the study

The two major interests of an imaging protocol in vestibular neuritis (quite similar to those already part of the clinical routine for facial neuritis) are (1) confirm the diagnosis and reassure the patient, (2) eliminate potential differential diagnosis. Another interest here is the limited use of contrast-agent (single dose) required for this technique. Imaging not necessary for patients' follow-up, similarly as in facial neuritis.

In conclusion, MRI with a delayed FLAIR sequence (1 h) acquired after an intravenous injection of a standard dose of

Fig. 4 The optimal cutoff point to differentiate between a normal and a pathological vestibular nerve on delayed FLAIR sequence was 71.5 units [67.5; 82.5], with an AUC=0.9824 [0.9575; 1]. With this cutoff, the delayed FLAIR correctly diagnosed superior vestibular neuritis with a sensitivity of 96% and a specificity of 100%

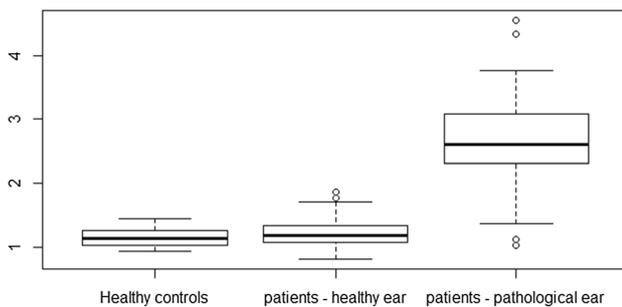
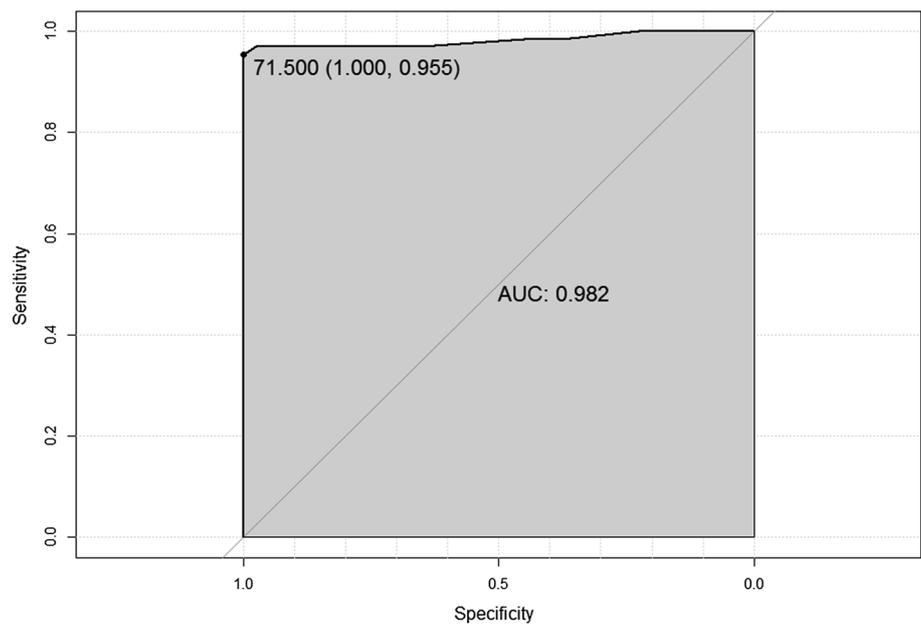


Fig. 5 The average ratio of the signal intensity of the superior vestibular nerve on the signal of the cerebellum (supVN/C) was also two times higher on the pathological side in vestibular neuritis ($2.43 \text{ units} \pm 0.63$) than in the control group (1.16 ± 0.14), $\text{Pr}(\text{diff} > 0) > 0.999$ and the normal contralateral side (1.40 ± 0.33 , $\text{Pr}(\text{diff} > 0) > 0.999$)

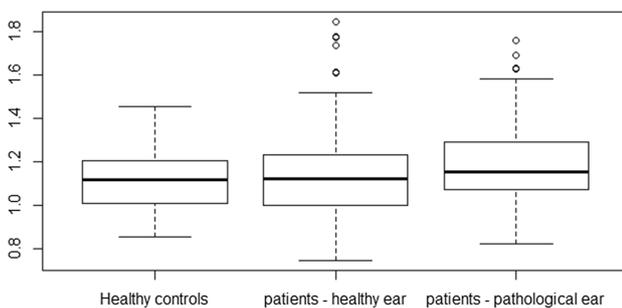


Fig. 6 There was no significant difference regarding the average ratios infVN/C between the pathological side in case of neuritis ($1.20 \text{ units} \pm 0.22$), the control group ($1.11 \text{ units} \pm 0.14$) and the normal contralateral side (healthy ear of patients ($1.43 \text{ units} \pm 0.29$))

gadolinium (0.1 mmol/kg) is a useful and fast method for the diagnosis of acute superior vestibular neuritis, as a strong enhancement of the superior vestibular nerve was observed on the pathological side in 85% of patients with vestibular neuritis (more than two times higher compared to the control group). An enhancement of the nerve > 71.5 units was in favor of acute superior vestibular neuritis.

Funding No funding was received.

Compliance with ethical standards

Conflict of interest The authors declare they have no conflict of interest.

References

1. Strupp M, Zingler VC, Arbusow V et al (2004) Methylprednisolone, valacyclovir, or the combination for vestibular neuritis. *N Engl J Med* 351:354–356. <https://doi.org/10.1056/NEJMoa033280>
2. Gacek RR, Gacek MR (2002) The three faces of vestibular ganglionitis. *Ann Otol Rhinol Laryngol* 111:103–114. <https://doi.org/10.1177/000348940211100201>
3. Gacek RR, Gacek MR (2002) Vestibular neuronitis: a viral neuropathy. *Adv Otorhinolaryngol* 60:54–66
4. Hasuike K, Sekitani T, Imate Y (1995) Enhanced MRI in patients with vestibular neuronitis. *Acta Otolaryngol*. <https://doi.org/10.3109/00016489509121922>
5. Strupp M, Jäger L, Müller-Lisse U, Arbusow V, Reiser M, Brandt T (1998) High resolution Gd-DTPA MR imaging of the inner ear in 60 patients with idiopathic vestibular neuritis: no evidence for

- contrast enhancement of the labyrinth or vestibular nerve. *J Vestib Res* 8:427–433. <https://doi.org/10.3233/VES-1998-8603>
6. Karlberg M, Annertz M, Magnusson M (2004) Acute vestibular neuritis visualized by 3-T magnetic resonance imaging with high-dose Gadolinium. *Arch Otolaryngol Head Neck Surg* 130:229–232. <https://doi.org/10.1001/archotol.130.2.229>
 7. Park KM, Shin KJ, Ha SY, Park JS, Kim S (2014) A case of acute vestibular neuritis visualized by three-dimensional FLAIR-VISTA magnetic resonance imaging. *Neuro-Ophthalmology* 38:60–61. <https://doi.org/10.3109/01658107.2013.874454>
 8. Byun H, Chung JH, Lee SH, Park CW, Park DW, Kim TY (2018) Clinical value of 4-h delayed gadolinium-enhanced 3D FLAIR MR images in acute vestibular neuritis. *Laryngoscope* 128:1946–1951
 9. Freedman L (1996) Bayesian statistical methods. *BMJ* 313:569–570
 10. Ntzoufras I (2009) Bayesian modeling using WinBUGS. Wiley, New York
 11. Hoff PD (2009) A first course in Bayesian statistical methods, Springer texts in statistics. Springer, New York
 12. R Core Team (2016) A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna
 13. Plummer M (2003) JAGS: a program for analysis of bayesian graphical models using gibbs sampling. In: Proceedings of the 3rd international workshop on distributed statistical computing (DSC 2003), Vienna, Austria
 14. Nakata S, Mizuno T, Naganawa S et al (2010) 3D-FLAIR MRI in facial nerve paralysis with and without audio-vestibular disorder. *Acta Otolaryngol* 130:632–636. <https://doi.org/10.3109/00016480903338123>
 15. Lim HK, Lee JH, Hyun D et al (2012) MR diagnosis of facial neuritis: diagnostic performance of contrast-enhanced 3D-FLAIR technique compared with contrast-enhanced 3D-T1-fast-field echo with fat suppression. *AJNR Am J Neuroradiol* 33:779–783. <https://doi.org/10.3174/ajnr.A2851>
 16. Chung MI, Lee JH, Kim DY et al (2015) The clinical significance of findings obtained on 3D-FLAIR MR imaging in patients with Ramsay–Hunt syndrome. *Laryngoscope* 125:950–955. <https://doi.org/10.1002/lary.24973>
 17. Büki B, Hanschek M, Jünger H (2017) Vestibular neuritis: involvement and long-term recovery of individual semicircular canals. *Auris Nasus Larynx* 44(3):288–293. <https://doi.org/10.1016/j.anl.2016.07.020> (**Epub 2016 Aug 18**)
 18. Kum RO, Kum YN, Ozcan M et al (2014) Elevated neutrophil-to-lymphocyte ratio in Bell’s palsy and its correlation with facial nerve enhancement on MRI. *Otolaryngol Head Neck Surg* 152:130–135. <https://doi.org/10.1177/0194599814555841>
 19. Taylor RL, McGarvie LA, Reid N, Young AS, Halmagyi GM, Welgampola MS (2016) Vestibular neuritis affects both superior and inferior vestibular nerves. *Neurology* 87(16):1704–1712 (**Epub 2016 Sep 30**)

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