Auris Nasus Larynx xxx (xxxx) xxx

Contents lists available at ScienceDirect

Auris Nasus Larynx

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

Original Article

Vestibular and cochlear nerve enhancement on MRI and its correlation with vestibulocochlear functional deficits in patients with Ramsay Hunt syndrome

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ARTICLE INFO

Article history:
Received 21 February 2020
Accepted 27 August 2020
Available online xxx

Keywords:
Ramsay Hunt syndrome
Varicella-zoster virus
Vestibular nerve
Cochlear nerve

ABSTRACT

Objective: The correlation between enhancement of the vestibulocochlear nerves on gadolinium-enhanced magnetic resonance imaging (MRI) and vestibulocochlear functional deficits was examined in patients with Ramsay Hunt syndrome (RHS).

Methods: Nineteen patients with RHS who showed herpes zoster oticus, peripheral facial palsy, and vertigo were enrolled. Canal paresis (CP) in the caloric test, abnormal response to ocular and cervical vestibular myogenic potentials (oVEMP and cVEMP), and refractory sensorineural hearing loss were evaluated. MRI images perpendicular to the internal auditory canal were reconstructed to identify the superior (SVN) and inferior vestibular nerves (IVN) and the cochlear nerve (CV). The signal intensity increase (SIinc) of the four-nerve enhancement was calculated as an index.

Results: Among RHS patients, 79%, 53%, 17% and 26% showed CP in the caloric test, abnormal responses to oVEMP and cVEMP, and refractory sensorineural hearing loss, respectively. SIinc rates of the SVN were significantly increased in RHS patients with CP in the caloric test, and with abnormal responses to oVEMP and cVEMP. SIinc rates of the SVN tended to increase in RHS patients with refractory sensorineural hearing loss ($p = 0.052$). SIinc rates of the IVN were significantly increased in RHS patients with abnormal responses to oVEMP and cVEMP, and refractory sensorineural hearing loss, but not in those with CP in the caloric test. SIinc rates of the CN were significantly increased in RHS patients with CP in the caloric test, abnormal response to oVEMP and refractory sensorineural hearing loss, but not in those with abnormal response to cVEMP.

Conclusion: In patients with RHS, the origin of vertigo may be superior vestibular neuritis, which is affected by reactive varicella-zoster virus from the geniculate ganglion of the facial nerve through the faciovestibular anastomosis. The results also suggested that in some RHS patients, inferior vestibular neuritis contributes to the development of vertigo and that the origin of refractory sensorineural hearing loss is cochlear neuritis.

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https://doi.org/10.1016/j.anl.2020.08.027

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1. Introduction

Ramsay Hunt syndrome (RHS) is caused by the reactivation of varicella-zoster virus (VZV) that had latently infected in the geniculate ganglion of the seventh cranial nerve and is characterized by herpes zoster oticus, peripheral facial palsy, and eighth cranial nerve symptoms including vertigo and hearing loss [1]. Reactivated VZV in the geniculate ganglion induces inflammation and edema of the facial nerve, resulting in facial palsy [2]. Since gadolinium-DTPA (Gd) accumulates in the inflamed tissue due to a breakdown of the blood-nerve barrier, Gd-enhanced magnetic resonance imaging (MRI) can be used to visualize the inflammation of the cranial nerves. Gd-enhanced MRI studies reported a high frequency of enhancement in the regions of the internal auditory canal (IAC) and/or the intratemporal segments of the facial nerve in patients with RHS [3].

Because the eighth cranial nerve is close to the geniculate ganglion, reactivated VZV also induces inflammation with transneural infection, leading to vestibulocochlear symptoms in patients with RHS [4]. Our previous Gd-enhanced MRI study showed that the vestibulocochlear nerves are enhanced independently from the facial nerve, suggesting that vertigo and hearing loss in patients with RHS are induced by vestibular and cochlear neuritis, respectively [5].

In the present study, we investigated whether the enhancement of the vestibulocochlear nerves in the IAC on Gd-enhanced MRI is correlated with vestibulocochlear functional deficits in patients with RHS. MRI images perpendicular to the IAC were reconstructed to identify the superior (SVN) and inferior vestibular nerves (IVN) and cochlear nerve (CN) separately. The correlations between their enhancement and canal paresis (CP) in the caloric test, abnormal response on the cervical and ocular vestibular myogenic potentials (cVEMP and oVEMP), and refractory sensorineural hearing loss in the pure tone audiogram (PTA) were also examined.

2. Materials and methods

2.1. Participants

Nineteen patients with RHS who showed herpes zoster oticus, peripheral facial palsy, and vertigo (eight males and 11 females, 18–77 years old, mean age: 52.7 years) were enrolled in the present study. Five of these patients showed refractory sensorineural hearing loss. The House-Brackmann (HB) grade of their initial facial palsy ranged from IV to VI (IV: two, V: eight and VI: nine patients, Table 1). This retrospective study was approved by the Committee for Medical Ethics of Tokushima University Hospital. Within a week after onset, all patients were treated with systemic steroids (methylprednisolone: 250 mg × three days, 125 mg × three days, 80 mg × three days, intravenously) and antiviral agents (valaciclovir: 3000 mg × seven days, per os).

2.2. Caloric test

The caloric test was performed 35.1 ± 29.7 days after onset in 19 patients with RHS. Cold-water (20 °C, 5 mL) irrigation was used, and caloric nystagmus was recorded via electronystagmography. Its maximum slow-phase eye velocity was measured, and the caloric response was calculated by the addition or subtraction of the averaged slow-phase eye velocity of spontaneous nystagmus. A caloric response of less than 20°/s was determined to be CP [6].

Table 1
Patient's background and results of physical examination.

Patient No.	Age (years)	Sex	Affected side	HB grade	ENoG (%)	CP	oVEMP	cVEMP	PTA (dB)
1	68	F	L	V	67.1	+	normal	normal	44.2
2	22	F	L	VI	68.5	+	normal	normal	11.7
3	18	F	R	IV	58.0	-	normal	normal	3.3
4	50	F	L	VI	25.0	+	normal	normal	33.3
5	60	M	R	V	71.0	-	normal	normal	31.7
6	47	F	R	VI	28.6	-	normal	normal	18.3
7	39	M	L	V	0	+	absent	normal	19.2
8	67	M	L	VI	0	+	not detected	not detected	105*
9	73	F	L	V	0	+	absent	absent	43.8*
10	34	M	L	VI	23.6	+	absent	absent	70.8*
11	59	F	L	VI	33.5	+	absent	normal	28.3
12	77	M	L	VI	0	+	not detected	normal	94.2*
13	49	M	R	V	32.1	+	absent	normal	22.5
14	75	F	R	V	61.8	-	normal	normal	39.2
15	33	M	R	VI	0	+	absent	absent	12.5
16	61	F	R	VI	0	+	absent	normal	15.5
17	61	M	L	IV	50.0	+	absent	normal	53.8*
18	44	F	L	V	60.0	+	absent	normal	22.5
19	65	F	R	V	79.3	+	normal	normal	56.7

CP, canal paresis on the caloric test; cVEMP, cervical vestibular-evoked myogenic potential; ENoG, electroneurography; HB grade, House-Brackmann grade; oVEMP, ocular vestibular-evoked myogenic potential; PTA, the averaged hearing levels at 0.25, 0.5, 1 and 2 kHz on the pure tone audiogram; +, positive of canal paresis; -, negative of canal paresis; *, refractory hearing loss.

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2.3. Ocular vestibular-evoked myogenic potential

oVEMP was performed 36.6 ± 30.6 days after onset in 17 patients with RHS. The active electrodes were placed on the skin 1 cm below the center of each lower eyelid of patients in the supine position. The reference electrodes were placed 2 cm below the active electrodes with a ground electrode on the forehead. During testing, the patients were instructed to look upward at a small fixed target with a vertical visual angle of approximately 30° above the horizontal line. The electromyography signals were amplified and bandpass filtered between 1 and 1000 Hz using Neuropack X1 (Nihon Kohden, Tokyo, Japan). The stimulation rate was 5 Hz and the time of analysis was 50 ms. The stimulus using bone-conducted vibration consisted of 500 Hz tone-bursts (rise/fall time 1ms, plateau time 2 ms) by a vibrator (Minishaker 4810, Bruel and Kjaer Co, Naerum, Denmark) on the midline of the subject's hairline. We analyzed the latencies of the initial negative peak (nI) and the subsequent positive peak (pI), and the amplitude between nI and pI. The nI-pI amplitude between nI and pI was used as the parameter of interest. The amplitude and latency were calculated as an average of two trials. The asymmetry ratio (%) was calculated using the following formula: asymmetry ratio (%) = (Au-Aa)/(Aa+Au) × 100, where Au is the nI-pI amplitude on the healthy side and Aa is the nI-pI amplitude on the lesion side. An asymmetry ratio (%) of more than 33% indicates an abnormal response of oVEMP [7,8].

2.4. Cervical vestibular-evoked myogenic potential

cVEMP was performed 34.9 ± 30.5 days after onset in 18 patients with RHS. For recording cVEMP, the subjects were examined in the supine position. The active electrode was placed on the upper half of the sternocleidomastoid muscle and the reference electrode was placed on the medial end of the clavicle with a ground electrode on the forehead of the patients. During testing, the patients were instructed to lift their heads from the pillow during the stimuli in order to contract the sternocleidomastoid muscle. The stimulation consisted of an air-conducted short tone burst of 135 dB SPL at 500 Hz and was given through the headphone at a stimulation rate of 5 Hz. The analysis time was 50 ms. The first positive and second negative peaks are marked as p13 and n23, respectively. The latencies of p13-n23 and the amplitude p13-n23 were determined from the average of two trials. The asymmetry ratio (%) was calculated using the following formula: asymmetry ratio (%) = (Au-Aa)/(Aa+Au) × 100, where Au is the p13-n23 amplitude on the healthy side and Aa is the p13-n23 amplitude on the lesion side. An asymmetry ratio (%) of more than 33% indicates an abnormal response of cVEMP [9,10].

2.5. Pure tone audiogram

Hearing function was measured using a PTA. The initial PTA was performed 18.0 ± 23.9 days after onset in 19 patients with RHS. The mean hearing level was evaluated based on the four-tone average determined by (a + b + c + d) / 4. The variables a, b, c and d are hearing levels at 0.25, 0.5, 1 and 2 kHz, respectively. The patients with hearing levels of the affected ear that were still 40 dB or more a month after the onset were diagnosed as the refractory sensorineural hearing loss.

2.6. Contrast-enhanced magnetic resonance imaging

Pre- and post-contrast-enhanced three-dimensional T1-weighted fast field-echo (3D T1WI FFE) MR images of bilateral internal auditory canal (IAC) of each patient were obtained on a 1.5T MR scanner (Signa HDxt 1.5T, GE) 25.4 ± 15.8 days after onset. The imaging parameters were as follows: repetition time (TR) 10.5 ms, echo time (TE) 3.2 ms, flip angle (FA) 20°, slice thickness 1 mm, field of view (FOV) 200 mm, and a matrix of 256 × 224 (512 reconstruction). The contrast medium (Gd-DTPA) was administered intravenously at a dose of 0.1 mmol/kg. Patients' body and head was fixed to the MRI scanner and receiving head coil before and after intravenous administration to avoid misregistration of IACs. Multiplanar reconstruction (MPR) images perpendicular to both IACs were obtained using imaging workstation to identify the facial, SVN and IVN, and CN separately. The three-dimensional fast imaging employing steady-state acquisition (3D-FIESTA) sequence were obtained with same FOV and spatial resolution as 3D T1WI FFE sequences and reconstructed in a section perpendicular to the IAC to identify the facial nerve, SVN, IVN and CN, separately (Figs. 1AB and 2AB). The region of interest (ROI) of SVN, IVN and CN was placed on 3D- FIESTA images and the ROI was copy and pasted to 3D T1WI FFE images with/without Gd-enhancement (Figs. 1CD and 2CD). The signal intensity (SI) of each nerve was defined as the highest value in the ROI measured by two experienced radiologists who were blind to the clinical information. The degree of enhancement of the four nerves was measured for each nerve on both sides by calculating the value of SI increase (SIinc) were calculated using the following equation: SIinc (%) = {(SI post - SI pre) / SI pre} × 100, where SI post = SI measured on post-contrast T1-weighted images and SI pre = SI measured on pre-contrast T1-weighted images. SIinc rate indicates SIinc of the affected side/SIinc of the healthy side [11].

2.7. Statistical analysis

Mann–Whitney's U test and Kruskal–Wallis test were used for statistical analysis, and $p < 0.05$ was considered significant.

3. Results

The mean SIinc rate of the SVN (18.2 ± 24.3) was slightly, but not significantly increased, compared to those of the IVN (7.7 ± 10.0) and CN (9.5 ± 12.6) in patients with RHS.

Among RHS patients who underwent the caloric test (n = 19), 15 patients (79%) showed CP. Of RHS patients

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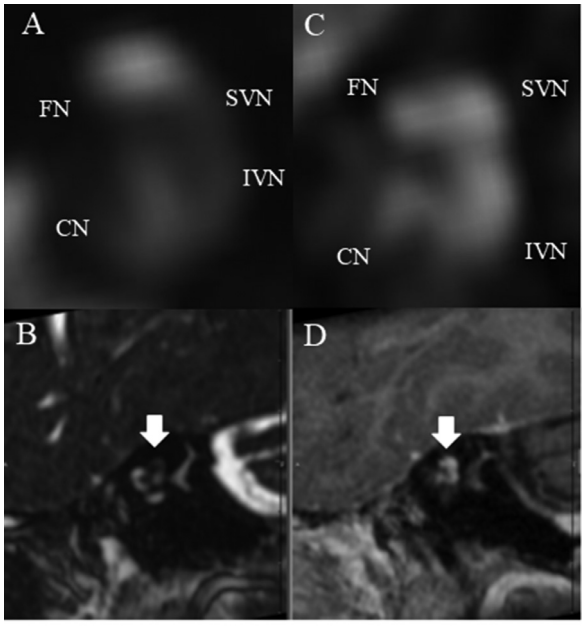

Fig. 1. Representative MR images of the affected side for measurement of SIinc rates of SVN, IVN and CN (Case 10, see Table 1). A, B: 3D-FIESTA images of the section perpendicular to the IAC to identify the anatomical location of four nerves. A: a magnified image of the area indicated by a white arrow in B. C, D: 3D T1WI FFE MR images of the section perpendicular to the same area of the IAC. C: a magnified image of the area indicated by a white arrow in D. The region of interest was placed on T1-weighted images with Gd-enhancement (C) in alignment with 3D-FIESTA image of four nerves (A). Calculated SIinc rates of SVN, IVN and CN were 35.4, 22.7 and 38.4, respectively. FN: facial nerve, SVN: superior vestibular nerve, IVN: inferior vestibular nerve, CN: cochlear nerve, IAC: internal auditory canal, 3D-FIESTA: three-dimensional fast imaging employing steady-state acquisition, 3D T1W1 FFE: three-dimensional T1-weighted fast field-echo.

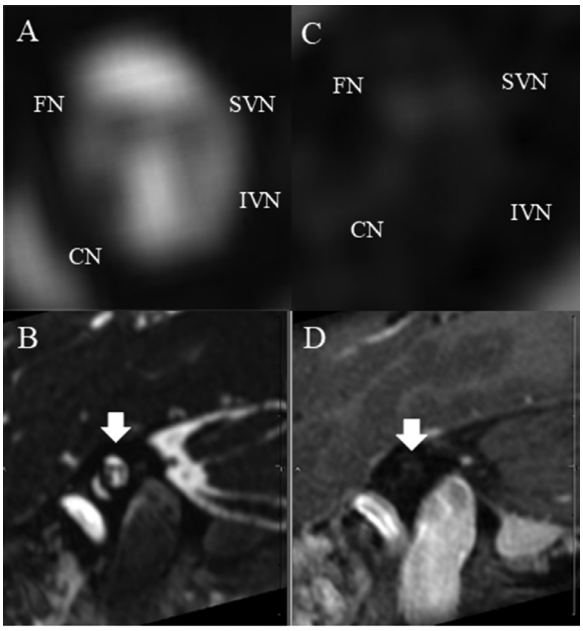

Fig. 2. Representative MR images of the healthy side for measurement of SIinc rates of SVN, IVN and CN (Case 10, see Table 1). A, B: 3D-FIESTA images of the section perpendicular to the IAC to identify the anatomical location of four nerves. A: a magnified image of the area indicated by a white arrow in B. C, D: 3D T1WI FFE MR images of the section perpendicular to the same area of the IAC. C: a magnified image of the area indicated by a white arrow in D. The region of interest was placed on T1-weighted images with Gd-enhancement (C) in alignment with 3D-FIESTA image of four nerves (A). FN: facial nerve, SVN: superior vestibular nerve, IVN: inferior vestibular nerve, CN: cochlear nerve, IAC: internal auditory canal, 3D-FIESTA: three-dimensional fast imaging employing steady-state acquisition, 3D T1W1 FFE: three-dimensional T1-weighted fast field-echo.

who were subjected to oVEMP ($n = 17$), nine patients (53%) showed an abnormal response. Among RHS patients who were subjected to cVEMP ($n = 18$), three patients (17%) showed an abnormal response. Five RHS patients (26%) who were subjected to PTA ($n = 19$) showed refractory hearing loss ranging from 43.8dB to scale out (Table 1).

SIinc rates of the SVN were significantly increased in RHS patients with CP in the caloric test, abnormal response to oVEMP, and abnormal response to cVEMP. SIinc rates of the SVN tended to increase in RHS patients with refractory sensorineural hearing loss ($p = 0.052$, Fig. 3). SIinc rates of the IVN were significantly increased in RHS patients with abnormal response to oVEMP, abnormal response to cVEMP and refractory sensorineural hearing loss but not in patients with CP in the caloric test (Fig. 4). SIinc rates of the CN were significantly increased in RHS patients with CP in the caloric test, abnormal response to oVEMP and refractory sensorineural hearing loss but not in patients with abnormal response to cVEMP (Fig. 5). The mean values of SIinc rates of the SVN, IVN and CN in all patients were 18.2 ± 24.3, 7.7 ± 10.0 and 9.5 ± 12.6, respectively.

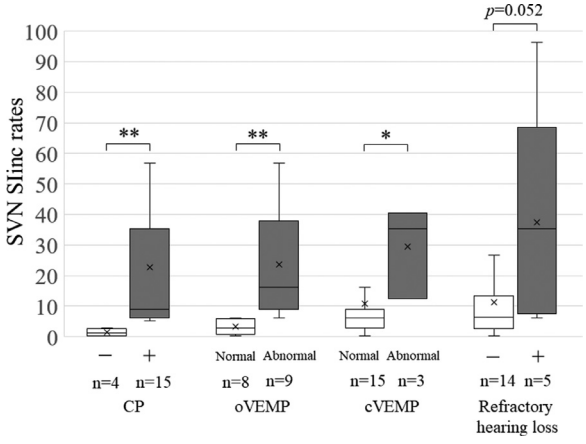

Fig. 3. SIinc rates of the superior vestibular nerve in Ramsay Hunt patients with/without CP in the caloric test, abnormal responses to oVEMP and cVEMP, and refractory sensorineural hearing loss. * $p < 0.05$, ** $p < 0.01$.

4. Discussion

In the present study, the SVN in the IAC on MRI was enhanced in RHS patients with CP in the caloric test or abnormal response to oVEMP. The SVN contains afferents from the lateral semicircular canal (SCC) and anterior SCC, and

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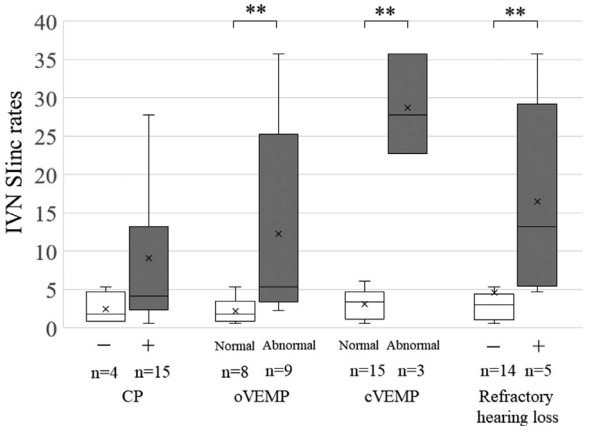

Fig. 4. SIinc rates of the inferior vestibular nerve in Ramsay Hunt patients with/without CP in the caloric test, abnormal responses to oVEMP and cVEMP, and refractory sensorineural hearing loss. ** $p < 0.01$.

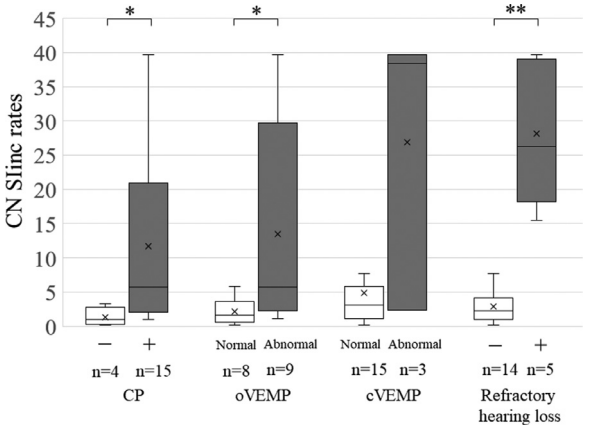

Fig. 5. SIinc rates of the cochlear nerve in Ramsay Hunt patients with/without CP in the caloric test, abnormal responses to oVEMP and cVEMP, and refractory sensorineural hearing loss. * $p < 0.05$, ** $p < 0.01$.

the utricle. The caloric test is a clinical test for function of the lateral SCC and the superior vestibular nerve, while the oVEMP is a clinical test of the function of the utricle and the SVN [7,8]. Therefore, the present findings suggest that the SVN was affected by the reactivated VZV, resulting in CP in the caloric test and abnormal response to oVEMP. Indeed, among 17 patients with RHS who underwent both the caloric test and oVEMP, nine patients with CP showed an abnormal oVEMP response. Abnormal responses in both the caloric test and oVEMP were also reported in patients with RHS [12]. Furthermore, the mean values of SIinc rates of the SVN was the highest out of the three nerves. Since Gd accumulates in inflamed cranial nerves due to a breakdown of the blood-nerve barrier [3], it is assumed that the origin of vertigo is superior vestibular neuritis in patients with RHS. The facial nerve is connected to the SVN by the faciovestibular anastomosis [13], suggesting that reactivated VZV spreads from the geniculate ganglion of the facial nerve to the SVN through the faciovestibular anastomosis to trigger superior vestibular neuritis.

The SVN was also enhanced in RHS patients with abnormal response to cVEMP and tended to be enhanced in patients with refractory sensorineural hearing loss. The IVN contains afferents from the posterior SCC and the saccule and cVEMP is a clinical test of saccule and the IVN function [9,10]. It was reported previously that RHS patients with retrocochlear hearing loss show poor hearing recovery [14]. Therefore, these findings suggest that the reactivated VZV may further spread from the SVN to the IVN and then the CN.

The IVN in the IAC on MRI was enhanced in RHS patients with an abnormal response to cVEMP. Since cVEMP is a clinical test of saccule and IVN function [9,10], the findings suggest that the IVN was affected by the reactivated VZV, resulting in an abnormal response to cVEMP in some patients. An abnormal response to cVEMP has also been reported in patients with RHS [15,16]. Although there is no anastomosis between the SVN and IVN, the proximal ends of both nerves merge in the vestibular ganglion stem at the stem end of the internal auditory canal [17]. Therefore, the reactivated VZV may spread further from the SVN to the IVN, and the resultant inferior vestibular neuritis may have contributed to the development of vertigo in some patients with RHS. Indeed, among 17 RHS patients who underwent both oVEMP and cVEMP tests, only three patients showed abnormal responses to oVEMP and cVEMP, and all showed CP in the caloric test. The IVN was also enhanced in RHS patients with refractory sensorineural hearing loss. Since the IVN is connected to the CN by Oort's anastomosis [18], both the IVN and CN may be affected by the reactivated VZV in some patients with RHS.

The CN in the IAC on MRI was enhanced in RHS patients with refractory sensorineural hearing loss. This finding suggests that the CN is affected by reactivated VZV, resulting in refractory sensorineural hearing loss in some patients, and that the origin of refractory sensorineural hearing loss is cochlear neuritis in patients with RHS. Indeed, it was shown previously that RHS patients with retrocochlear hearing loss show poor hearing recovery [14]. The CN was also enhanced in RHS patients with CP in the caloric test or abnormal responses to oVEMP. The reactivated VZV may further spreads from the SVN to the CN in some patients, although an anastomosis between the SVN to the CN has not been reported. Indeed, among five RHS patients with refractory sensorineural hearing loss, all showed CP and three patients showed an abnormal oVEMP response.

5. Conclusion

The present study suggests that vertigo in patients with RHS is mostly induced by superior vestibular neuritis triggered by reactivated VZV from the geniculate ganglion of the facial nerve through the faciovestibular anastomosis, which causes CP in the caloric test and abnormal response to oVEMP. In some RHS patients, inferior vestibular neuritis triggered by reactivated VZV spread from the SVN may also contribute to the development of vertigo that is associated with an abnormal response to cVEMP. Refractory sensorineural hearing loss in some RHS patients may also be due to cochlear neuritis triggered by reactivated VZV from

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Oort's anastomosis. Thus, reactivated VZV may spread from the geniculate ganglion to the SVN and IVN and then CN in patients with RHS. Indeed, 79%, 53%, 17% and 26% of RHS patients showed CP in caloric test, abnormal responses to oVEMP and cVEMP, and refractory hearing loss, respectively.

Declaration of Competing Interest

The authors declare that they have no conflict of interest.

Acknowledgements

This study was financially supported in part by Grant-in-Aid from the Japan Society for the Promotion of Science.

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