Is there any relationship between unilateral vestibular neuritis and internal acoustic canal measurements?

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ABSTRACT

Objectives: In this study, we aimed to compare the internal acoustic canal (IAC) measurements of the affected and unaffected sides of patients with unilateral vestibular neuritis (VN) on magnetic resonance imaging (MRI).

Patients and Methods: January 2017 and December 2019, a total of 120 patients (65 males, 55 females; mean age 58.3±12.5 years; range, 21 to 83 years) who were diagnosed with VN according to clinical findings, video head impulse test (vHIT), and MRI findings were enrolled in this study. All measurements were performed on the axial constructive interference in steady state (CISS) sequences. The IAC inlet, mid-canal, and outlet widths were measured at the most distinctive cross-section of the facial nerve and superior vestibular nerve bifurcation. While calculating the length of IAC, the points were taken on the basis of where the inlet and outlet widths of the canal were measured.

Results: According to the vHIT results, a single semicircular canal (SCC) was affected in 102 (85%) patients, while more than one SCC was affected in 18 (15%) patients. The inlet, mid-canal and outlet values of the affected side were significantly lower than those of the unaffected side (p<0.05). Regarding the IAC length measurement, there was no significant differences between the affected and unaffected sides (p>0.05). All of these parameters were slightly higher in males; however, this difference was not statistically significant (p>0.05).

Conclusion: Our study results showed that the widths of the IAC were lower in the affected side of patients with unilateral VN. These findings may help to explain why acute VN cases occur only unilaterally, suggesting that a small sectional diameter of the IAC is a potential risk factor for VN.

Keywords: Internal acoustic canal, magnetic resonance imaging, vestibular neuritis, video head impulse test.

Vestibular neuritis (VN) can be defined as unilateral loss of function of the vestibular system, characterized by the acute onset of vertigo as well as nausea and vomiting. Auditory symptoms almost do not exist or complaints such as aural fullness, tinnitus, and hearing loss may occur rarely in certain patients, as the cochlear nerve is affected. Vestibular neuritis affects female and male equally and reaches its peak approximately at the age of 40 to 50 years. The clinical presentation appears as a single attack in the vast majority of cases; however, benign recurrent vertigo attacks may occur in less than 5% of patients. Vestibular neuritis is the second most common cause of peripheral vertigo following benign paroxysmal positional vertigo (BPPV).
Although causes such as reactivation of neurotropic viruses (i.e., HSV-1) in the vestibular ganglia (Scarpa), labyrinthitis, and vascular ischemia are thought to be responsible for development of VN, the evidence on this issue is uncertain and the exact etiological cause is not known. Diagnosis of VN can be defined as a diagnosis of exclusion based on clinical history, physical examination findings, vestibular tests and radiological imaging results. Four main diagnostic criteria have been described: (i) acute or subacute onset of symptoms; (ii) horizontal spontaneous nystagmus fast phase of which beating toward the unaffected ear; (iii) decrease of bithermal caloric test responses on the affected side (asymmetry more than 25% between both sides); and (iv) disruption in the vestibulo-ocular reflex. Contrast-enhanced magnetic resonance imaging (MRI) must be conducted to exclude possible retrocochlear pathologies. Although MRI is normal in the vast majority of patients with VN, contrast material involvement can be detected in the vestibular nerve or labyrinth in some cases.

Certain anatomical differences between the superior vestibular nerves (SVNs) on the affected and unaffected sides have been shown on MRI of patients with VN. In a study including patients with Bell’s palsy, a disease having similar pathophysiology as VN, the inlet and mid-canal diameters of the internal acoustic canal (IAC) on the affected side were found to be significantly narrower, compared to the unaffected side. To the best of our knowledge, there is no study comparing the measurements of IAC on MRI scans of patients with VN in the literature. In this study, therefore, we aimed to compare the IAC measurements of the affected and unaffected sides on MRI of patients with unilateral VN considering all these findings.

**PATIENTS AND METHODS**

This retrospective study was conducted in accordance with the principles of the Declaration of Helsinki.

Archive records of 145 patients diagnosed with unilateral VN based on clinical findings, video head impulse test (vHIT), and contrast-enhanced MRI results were screened. External auditory canals and eardrums of all patients were normal and there were no pathological findings in their neurological examinations. Patients with previous VN attack, labyrinthitis, sudden hearing loss, facial paralysis, chronic otitis media with or without cholesteatoma, hearing loss (conductive, sensorineural or mixed type), ear surgery, head trauma, neurological diseases, vertebrobasilar insufficiency, history of cancer and/or patients with congenital ear anomalies were excluded from the study. Patients having vestibular system disorders in their medical history such as BPPV, vestibular migraine, autoimmune inner ear disease, and Meniere’s disease were also excluded. Finally, a total of 120 patients (65 males, 55 females; mean age 58.3±12.5 years; range, 21 to 83 years) were included in the study.

**Measurement technique**

The 1.5 Tesla Siemens Sonata™ device (Siemens AG, Munich, Germany) and an eight-channel head coil were utilized for MRI. Routine temporal MRI images consists of T1- and T2-weighted fluid attenuated inversion recovery (FLAIR) and constructive interference in steady state (CISS) sequences in axial plan and T1-weighted sequences in post-contrast axial and coronal plan in our hospital. Gadolinium (0.2 mL/kg) was employed as the contrast agent. It was confirmed that there was no IAC or cerebellopontine angle pathology through a meticulous evaluation of all sections before proceeding to measurements. Axial plan images in the CISS sequence were utilized for all measurements.

The widest diameters of the inlet, mid-canal, and outlet of IAC were measured in the axial section where the facial nerve and SVN bifurcation were best distinguished. The length of IAC was calculated as the distance between two points where the inlet and outlet diameters of the canal were measured (Figure 1).
All measurements were recorded separately for the side affected by the disease (experimental group) and the healthy side (internal control group). All evaluations were conducted by two experienced Ear, Nose and Throat Diseases specialists simultaneously.

**Statistical analysis**

Statistical analysis was performed using the GraphPad Prism for Windows version 8.2.0 software (GraphPad Software, CA, USA). Descriptive data were expressed in mean ± standard deviation (SD), median (min-max) or number and frequency. Independent sample t-test was carried out to analyze significant differences between the groups. The Pearson and Spearman correlation analyses were performed to examine possible correlations between the categorical variables. A p value of <0.05 was considered statistically significant.

**RESULTS**

The chief complaint was sudden and acute onset of vertigo in all patients. Clinical and laboratory findings of VN were detected in 67 (55.8%) patients on the right side and in 53 (44.2%) patients on the left side. One single semicircular canal (SSC) was affected in 102 (85%) patients and more than one SSC was affected in 18 (15%) patients according to vHIT results. When the total number of affected SSC was analyzed, involvement of lateral SSC in 104 (75.4%) patients was detected, while posterior SSC involvement was detected in 28 (20.3%) patients and superior SSC involvement was detected in six patients (4.3%) (Table 1).

The IAC measurements of both sides (affected and unaffected) are summarized in Table 2. The inlet, mid-canal and outlet diameters of the IAC in affected side were significantly narrower, compared to the unaffected side (p<0.05). No significant difference was found between the groups in terms of the length of IAC (p>0.05). According to sex, the measurements in males were higher than the measurements in females; however, this difference was not statistically significant (p>0.05). No significant difference was detected in the correlation analysis between the asymmetry ratio (between the affected and unaffected sides) and the length and diameter of the inlet, mid-canal and outlet of the IAC on the affected side according to the vHIT results (p>0.05).
DISCUSSION

The vestibular nerve has three branches: the SVN (superior and lateral semicircular canal and utricle), the inferior vestibular nerve (IVN, saccule) and the singular nerve (posterior semicircular canal). The SVN is affected in nearly 85% of VN cases, irrespective of etiological factors, while the IVN is affected in remaining 15% of VN cases. We found that the SVN (104/120, 86.7%) was affected in the vast majority of the cases in our study which is consistent with the literature. Furthermore, both SVN and IVN involvements existed in 14 (11.7%) patients according to our vHIT results. In their study including VN patients, Taylor et al. used cervical/ocular vestibular evoked myogenic potentials (VEMP) and vHIT and reported that both SVN and IVN were affected in 55.8% of patients (24/43). Caloric test, vHIT, and VEMP results of 43 VN patients were analyzed by Lv et al. in another study. The authors reported that both vestibular nerves were affected in 51.2% of patients. The cervical VEMP evaluate the IVN, which carries the stimuli originating from the posterior SSC and saccule (more). The reason why the rate of IVN involvement was lower in our study compared to the literature may be associated with the use of only vHIT in our study and, accordingly, detection of IVN involvement less than it actually is.

It has been shown in temporal bone dissection studies that SVN canal is longer and contain more bony spicules compared to the canals of the IVN and the singular nerve. Moreover, the arterial blood supply of SVN and its related neuroepithelial tissue have similar limitations. The SVN is more prone to edema and ischemia secondary to inflammation, as well as nerve damage, as a natural result of this anatomical difference. This can partly explain why the SVN is more frequently affected in VN cases, although it fails to explain why VN occurs unilaterally or why it specifically affects that side.

In their study, Fundakowski et al. compared SVN and IVN of 68 patients diagnosed with unilateral VN in parasagittal MRI sections in terms of height, width, and area parameters. The authors reported that the height and area parameters of the SVN on the affected side were significantly smaller compared to the unaffected side. No significant differences were observed in terms of any parameters between the IVNs of the affected and unaffected sides in this study. Diameters of the inlet, mid-canal, and outlet of the IAC and the length of the IAC were measured in the affected and unaffected sides in our study, since the resolution of the parasagittal MRI sections was not sufficient in some cases and SVN and IVN could not be clearly demonstrated in each case. We found that the diameters of the inlet, mid-canal, and outlet of the IAC on the affected side were significantly smaller, compared to the unaffected side. Our results are consistent with previous studies in terms of the sizes of IAC. Yilmaz et al. also reported that the inlet and mid-canal diameters of the IAC were significantly narrower in the affected side in patients with Bell’s palsy, which is similar to our results. Although viral reactivation or inflammatory processes affect IAC in both sides, neural structures in IAC, which are cross-sectionally larger, would have more space to expand. On the contrary, neural structures in IAC, cross-sectional diameter of which are narrower, would get stuck inside the canal and their blood flow would be impaired, in

<table>
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<th>Measurement</th>
<th>Affected side</th>
<th>Unaffected side</th>
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<tr>
<td></td>
<td>Mean±SD</td>
<td>Min-Max</td>
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<tr>
<td>IAC inlet</td>
<td>49.3±5.7</td>
<td>37-64</td>
<td>-3.21</td>
<td>0.001</td>
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<tr>
<td>IAC mid-canal</td>
<td>40.0±4.8</td>
<td>30-55</td>
<td>-2.93</td>
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<tr>
<td>IAC outlet</td>
<td>30.4±3.7</td>
<td>23-44</td>
<td>-3.97</td>
<td>0.000</td>
</tr>
<tr>
<td>IAC length</td>
<td>79.6±5.7</td>
<td>69-99</td>
<td>-2.29</td>
<td>0.226</td>
</tr>
</tbody>
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IAC: Internal acoustic canal; SD: Standard deviation; Min: Minimum; Max: Maximum; * Independent samples t-test.

**Table 2. IAC measurements of affected and unaffected sides**
the presence of edema. This hypothesis can help to explain why VN emerges unilaterally.

Nonetheless, this study has some limitations including its retrospective nature, lack of a control group consisting of healthy volunteers, and utilization of IAC measures in lieu of SVN or IVN measures. Further large-scale, randomized-controlled trials are required to reveal the relationship between VN and IAC and explain why the disease is unilateral.

In conclusion, our study results showed that the diameters of the inlet, mid-canal, and outlet of the IAC on the affected side in the unilateral VN cases were significantly smaller than the unaffected side in our study, although there was no significant difference in the length between the two sides. These findings may help to explain why acute VN cases occur only unilaterally. Based on these results, it can be speculated that a small sectional diameter of the IAC is a potential risk factor for VN.

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