

# Monthly and seasonal variations in vestibular neuritis

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## Abstract

Seasonal variations in vestibular neuritis (VN) could support the etiology of viral infection. However, several recent studies revealed no significant seasonal variations in VN. Further studies are necessary to determine the etiology of VN. We analyzed patients with VN to evaluate monthly and seasonal variations. Patients with VN who visited our otorhinolaryngology department or were referred to our department from the emergency department between March 2014 and February 2019 were included retrospectively in this study. Differences among the months and seasons of VN visits were analyzed. Patients were divided into 2 groups according to sex and age (65 years or older and younger than 65 years). Differences among the months and seasons of VN visits were analyzed between groups. There were no significant differences in monthly and seasonal distributions in 248 patients with VN. There were also no significant differences in monthly and seasonal distributions in male and female patients or in older and younger patients. There were no significant differences in monthly or seasonal distributions of patients with VN. Factors other than viruses, such as vascular ischemia, should also be considered in the incidence of VN, especially in older patients.

**Abbreviations:** BPPV = benign paroxysmal positional vertigo, cVEMP = cervical vestibular evoked myogenic potential, HSV = herpes simplex virus, SSNHL = sudden sensorineural hearing loss, VN = vestibular neuritis.

**Keywords:** incidence, month, season, seasonal variation, vestibular neuritis

## 1. Introduction

Vestibular neuritis (VN) is characterized by acute onset of vertigo without hearing loss, for which symptoms can last for days to weeks.<sup>[1-6]</sup> The annual incidence of VN has been estimated at 3.5 per 100,000.<sup>[3-10]</sup> VN is associated with sudden unilateral vestibular hypofunction.<sup>[6,7]</sup> Although the exact etiology of VN has not been established, viruses have been implicated because patients with VN experience a viral prodrome before onset, and the disease typically occurs in spring and early summer.<sup>[1,6-8]</sup> The histopathological findings of the atrophy of the vestibular nerve and the vestibular sensory epithelium in VN are similar to those in viral disorders of the inner ear such as measles and mumps.<sup>[1,7]</sup>

Several respiratory viral infections have been reported to exhibit seasonality in nationwide surveillance in Korea, with a peak from spring to early summer.<sup>[11]</sup> In addition, respiratory viral detection rate in patients with severe pneumonia was higher in winter than in other seasons in Korea.<sup>[12]</sup> Because the incidence of viral infections varies by season, seasonal variations in VN could support this viral hypothesis.<sup>[6]</sup>

However, several recent studies revealed no significant seasonal variations in VN. In a study with 52 patients with VN for 3 years in the United States, there were no significant annual or

seasonal variations.<sup>[6]</sup> In a prospective, population-based study with 79 patients with VN for 2 years in Croatia, there was also no seasonal variation.<sup>[8]</sup> In a study using a nationwide database with 6108 patients with VN for 3 years in Germany, no seasonal variation was demonstrated.<sup>[13]</sup>

Considering these recent controversial reports on seasonal variations in VN, further studies are necessary to determine the etiology of VN. Vascular ischemia and autoimmune reaction are additional possible causes of VN.<sup>[5-7,10,14]</sup> Thus, multiple etiologies could influence the incidence of VN. In the present study, we analyzed many patients with VN over a several-year period to evaluate monthly and seasonal variations.

## 2. Materials and methods

### 2.1. Patients

The Institutional Review Board of our institution approved this study (NHIMC 2019-07-041). Written informed consent was exempted by the institutional review board because this study was retrospective. Patients with VN who visited the otorhinolaryngology department of our secondary institution or were referred to the otorhinolaryngology department from the emergency department between March 2014 and

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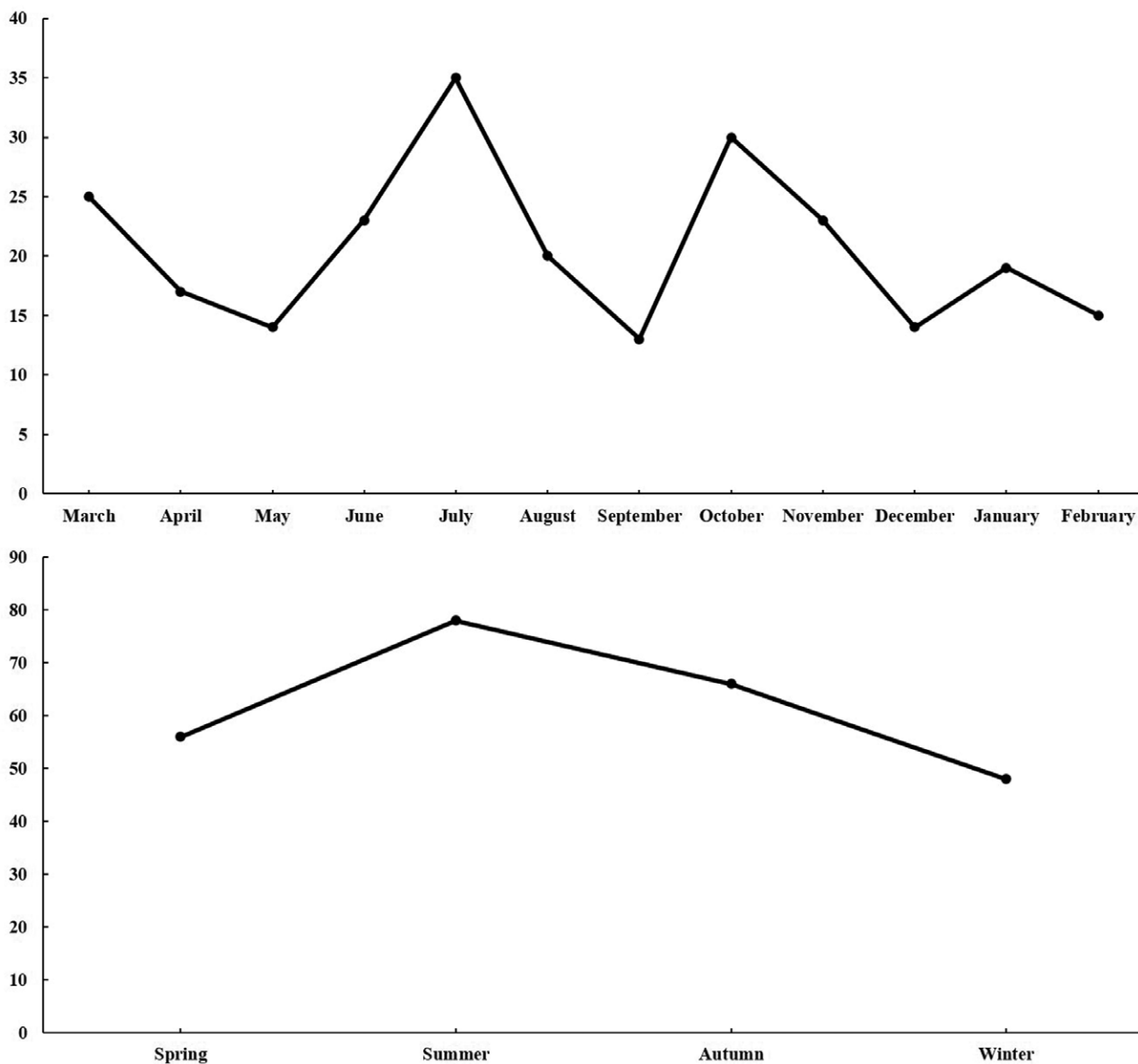
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February 2019 were included retrospectively in this study. The included patients had symptoms of acute severe vertigo for several days and presented with spontaneous nystagmus toward the nonaffected ear at their first visit. All included patients had no viral prodromal symptoms of upper respiratory infection within a few weeks of acute vertigo onset, which was confirmed through the medical chart. The patients had no symptoms of acute hearing loss, and vertigo improved gradually over weeks to months. Brain computed tomography or brain magnetic resonance imaging was performed for those with a possibility of central pathology, and these patients were excluded. Patients with diseases other than VN, such as benign paroxysmal positional vertigo (BPPV), labyrinthitis, Meniere disease, head trauma, sudden sensorineural hearing loss (SSNHL), or ear surgery, also were excluded. The diagnosis of VN and the involved site were confirmed with vestibular function tests such as bithermal caloric test, video head impulse test, or cervical vestibular evoked myogenic potential (cVEMP). Included patients showed 25% or greater canal paresis in the affected ear in a bithermal caloric test, and the video head impulse test showed catch-up

**Table 1**  
Demographic characteristics of patients with VN in this study.

	N
Sex	
Male	118 (47.6%)
Female	130 (52.4%)
Side	
Right	131 (52.8%)
Left	117 (47.2%)
Age	
≥ 65 years	57 (23.0%)
< 65 years	191 (77.0%)
Mean age (years)	53.9 ± 15.2 (range 11–89)
Total	248

saccade or gain decrease in at least 1 semicircular canal of the affected ear. Included patients revealed normal response, asymmetrical amplitude, or no response in the affected ear in cVEMP.



**Figure 1.** Monthly and seasonal distributions of patients with VN. There was no significant difference in monthly or seasonal distribution.

**2.2. Monthly and seasonal variations in VN**

Age, sex, lesion side, and month of the first VN visit were evaluated. Differences among the months and seasons of VN visits were analyzed. The months of the calendar year were categorized according to season as follows: spring months comprised March, April, and May; summer was June, July, and August; autumn was September, October, and November; and winter comprised December, January, and February. For detailed investigation, patients were divided into 2 groups according to sex and age (65 years or older and younger than 65 years). Differences among the months and seasons of VN visits were analyzed between groups.

Differences among the months and seasons of VN visits were analyzed based on the Kruskal-Wallis test using IBM SPSS 23 (IBM, Armonk, NY).

**3. Results**

In total, 248 patients with VN were included over a period of 5 years. One hundred eighteen patients were male, and 130 were female. The average age was  $53.9 \pm 15.2$  years (range 11–89

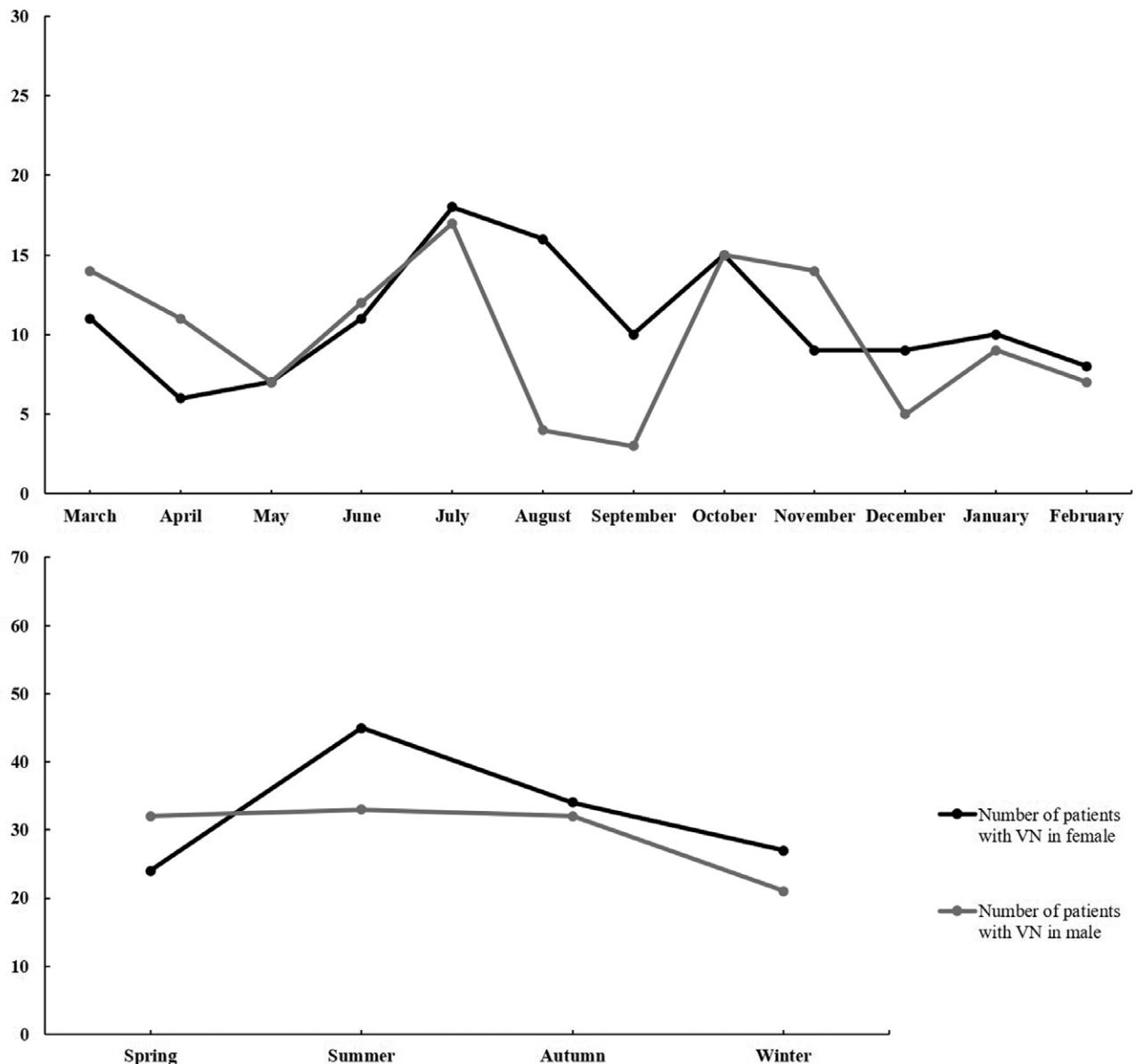
years), with 57 patients 65 years or older and 191 younger than 65 years. For lesion side, 131 cases involved the right, and 117 were in the left (Table 1).

**3.1. Monthly and seasonal distributions of patients with VN**

The number of visits for VN was analyzed according to month and season. The number of visits was highest in July in the summer, but there were no significant differences in monthly ( $H [11] = 19.200, P = .058$ ) and seasonal ( $H [3] = 5.008, P = .171$ ) distributions (Fig. 1).

**3.2. Monthly and seasonal distributions of patients with VN in male and female patients**

The number of visits for VN was analyzed among months and seasons in male and female groups. Among the male patients, the number of visits was highest in July, but there were no significant differences in monthly ( $H [11] = 18.920, P = .063$ ) and seasonal ( $H [3] = 1.691, P = .639$ ) distributions. In female patients, the



**Figure 2.** Monthly and seasonal distributions in male and female patients with VN. There was no significant difference in monthly or seasonal distribution in male and female patients. VN = vestibular neuritis.

number of visits also was highest in July, but there were no significant differences in monthly ( $H [11] = 9.327, P = .592$ ) and seasonal ( $H [3] = 4.289, P = .232$ ) distributions (Fig. 2).

### 3.3. Monthly and seasonal distributions of patients with VN in older and younger patients

The number of visits for VN was analyzed among months and seasons in the 2 age groups. In patients who were younger than 65 years, the number of visits was highest in July in the summer, but there were no significant differences in monthly ( $H [11] = 16.647, P = .119$ ) and seasonal ( $H [3] = 5.466, P = .141$ ) distributions. In patients 65 years or older, the number of visits also was highest in July in the summer, but there were no significant differences in monthly ( $H [11] = 5.881, P = .881$ ) or seasonal ( $H [3] = 1.287, P = .732$ ) distributions (Fig. 3).

### 4. Discussion

Viral infection has been thought to be the cause of VN, but the evidence remains unclear.<sup>[3,5-7,9]</sup> Although Dix and Hallpike

suggested a possibility of infection of Scarpa ganglion or the vestibular nerve, it was not verified whether direct viral infection of the vestibular organ could induce VN.<sup>[4,6]</sup> An animal model of VN was developed by inoculating herpes simplex virus (HSV) type 1 into the auricle of mice, and HSV type 1 deoxy-nucleic acid was detected by polymerase chain reaction in about 60 percent of autopsied vestibular ganglia in human.<sup>[3,5-7,9]</sup> Thus, reactivation of latent viruses, such as HSV type 1, was thought to be associated with VN.<sup>[2,3,5-7,9,10]</sup>

A viral etiology had been supported previously by the following findings. VN has a viral prodrome, it can affect several members of the same family, it occurs in epidemics, and it more commonly occurs in spring and early summer.<sup>[1,8]</sup> However, several studies recently reported that there were no seasonal variations in VN.<sup>[6,8,13]</sup> In addition, 1 recent study reported that incidence of acute unilateral vestibulopathy due to viral etiology, which included VN, peaked in autumn, not in spring and early summer, among patients in the emergency department.<sup>[15]</sup> In general, there was no significant monthly and seasonal variations in number of visits for VN in the present study. Although there was an increasing trend in incidence of VN in

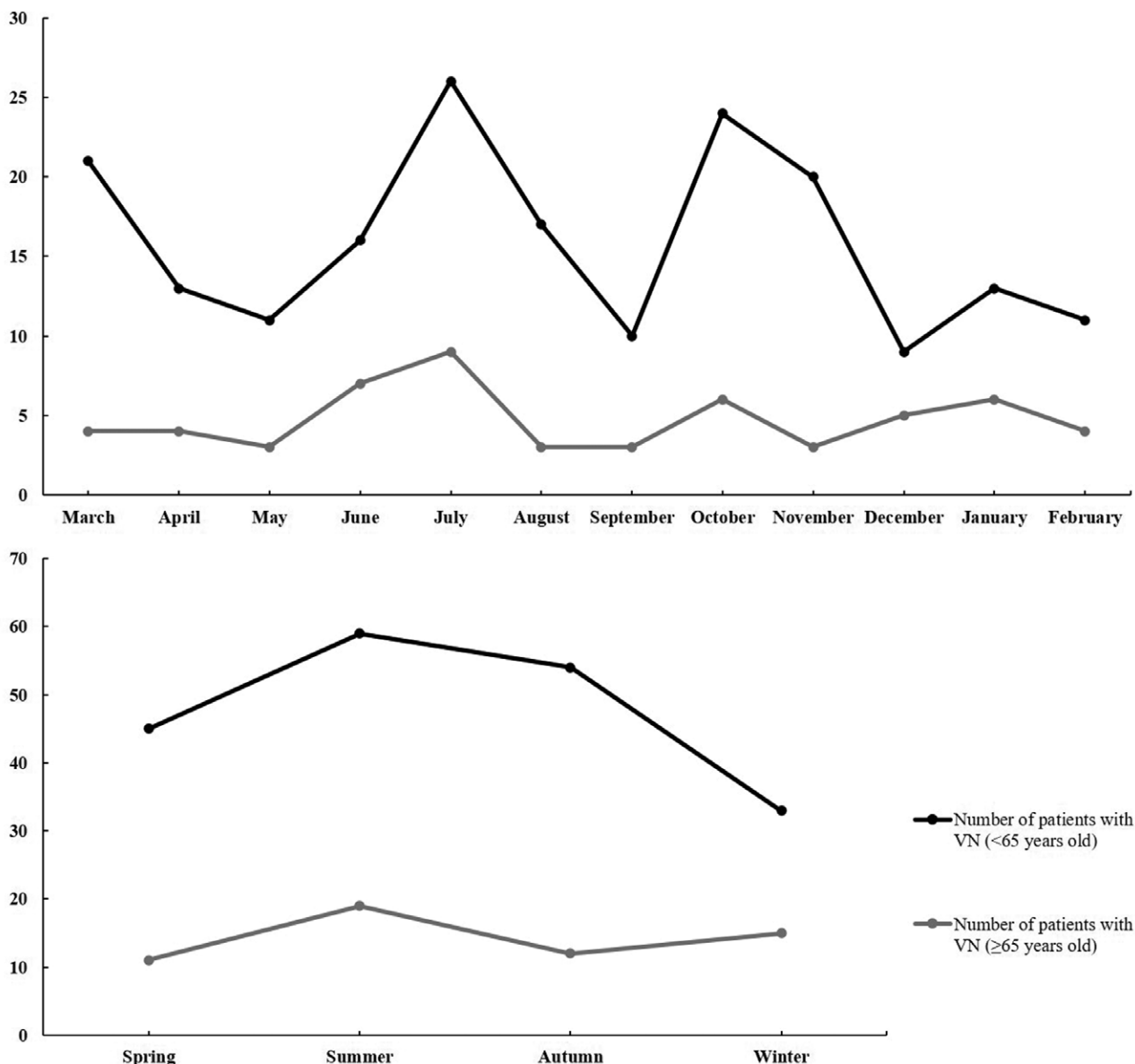


Figure 3. Monthly and seasonal distributions in older and younger patients with VN. There was no significant difference in monthly or seasonal distribution in patients 65 years or older vs younger than 65 years. VN = vestibular neuritis.

summer, it was not significant. In addition, in our analyses of subgroups according to sex and age, there were no significant monthly and seasonal variations in number of visits for VN.

Therefore, there might be VN etiologies other than systemic viral infection. Vascular ischemia could be another factor in VN. In the present study, for patients 65 years or older, monthly and seasonal distributions of visits for VN were flatter than for patients younger than 65 years. Older patients with VN could have more possible etiologies of vascular ischemia due to hypertension, diabetes, dyslipidemia, and previous cerebrovascular attack than younger patients. Accordingly, viral infection could have a smaller effect on the incidence of VN in older patients than in younger patients, which resulted in flatter monthly and seasonal distributions of VN. Cardiovascular risk factors were reported to be associated with VN, suggesting that vascular occlusion and labyrinthine ischemia could be attributed to the association.<sup>[14]</sup>

Bell palsy and SSNHL are thought to have an etiology of viral infection.<sup>[16–21]</sup> The lesion is in the vestibular nerve in VN, and VN is the vestibular nerve equivalent of SSNHL.<sup>[7]</sup> Thus, seasonal variations of Bell palsy and SSNHL could be considered to verify seasonal variations of VN. There have been several studies on the seasonality of Bell palsy and SSNHL. Studies have revealed that Bell palsy was significantly higher during the cold season,<sup>[17,21,22]</sup> although other studies have shown unclear association between Bell palsy and seasons.<sup>[16,23]</sup> One study revealed that the incidence of SSNHL peaked in autumn,<sup>[18]</sup> but most other studies showed nonsignificant distributions for seasons.<sup>[19,20,24]</sup> Reactivation of latent herpes virus infection could be aggravated by upper respiratory infections, which are more common in cold season,<sup>[17,21]</sup> but there is no evident association.<sup>[17]</sup>

Proposed etiologies of VN are latent viral infection, vascular ischemia, and autoimmune reaction.<sup>[5,7,14]</sup> The anatomic differences of the superior vestibular nerve channel, which is a longer and narrower channel with more interspersed bony spicules, make the superior vestibular nerve more susceptible to entrapment and ischemia due to viral inflammatory processes than the inferior vestibular nerve or singular nerve.<sup>[2,3,5,7,9,14]</sup> Proinflammatory activation and thrombosis could lead to reduced microvascular perfusion of vestibular organs.<sup>[7]</sup> It is possible that an inflammatory process causes VN rather than a virus itself.<sup>[10]</sup> Thus, multiple etiologies such as reactivation of latent viral infection and vascular ischemia should be considered when analyzing incidence of VN.

Seasonal variations in incidence of BPPV, which is the most common vestibular disease, have been reported in several studies. Seasonal variation is attributed to association of BPPV with vitamin D, which shows characteristic seasonal variation. However, we previously reported that there were no significant monthly or seasonal variations in patients with BPPV. We assumed that not only vitamin D, but also factors such as viral infection, ischemia, and physical activity might be associated with BPPV.<sup>[25]</sup> Likewise, there could be multiple contributing factors such as viral infection, vascular ischemia, and autoimmune for VN.

There are several limitations in this study. First, this study included patients with VN from a single secondary institution, and it was conducted retrospectively. Second, the patients with VN were not divided according to involvement of the superior or inferior vestibular nerve because of lack of early availability of tests to evaluate the function of inferior vestibular nerves such as cVEMP and video head impulse test. Third, this study included only patients with VN who had visited our otorhinolaryngology department or had been referred to our department from the emergency department. However, patients suspected to have VN usually are referred to our department. Fourth, audiometry to evaluate symmetric or asymmetric hearing loss was not performed because the included patients had no symptoms of acute hearing loss and routine audiometry was not possible considering the cost of the test. Despite these limitations, we analyzed many patients with VN over a long period of time, and we

evaluated monthly and seasonal variations in number of visits for VN. Further international multi-center large-scale studies are required to verify the seasonal variation and etiology of VN.

## 5. Conclusions

There were no significant differences in monthly or seasonal distributions of patients with VN. Based on the results of this study, VN might have multiple etiologies, and factors other than viruses, such as vascular ischemia, also should be considered in the incidence of VN, especially in older patients.

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