Effect of early high-dose steroid treatment in patients with acute vestibular neuritis: a retrospective case-control study

Jung-Yup Lee*, Hyun-Seok Kang*, Sang-Hyun Kim, Min-Beom Kim

Department of Otorhinolaryngology-Head and Neck Surgery, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, Korea

Objectives: This study is performed to evaluate the effect of early steroid treatment within 24 hours of onset in acute vestibular neuritis (AVN).

Methods: We performed a retrospective case-control study with 46 patients with AVN. Video head impulse test paradigm (HIMP) and suppression HIMP were performed, and Dizziness Handicap Inventory (DHI) was determined at initial; all tests were repeated at 1 month. Patients were divided into two groups depending on whether they were treated with steroids (group S, n=21) or not (group n-S, n=25).

Results: There was no significant difference in age, sex, and side between the two groups. In HIMP, group S showed a significantly lower occurrence of overt corrective saccade (CS) ($p=0.034$) and lower peak velocity of overt CS ($p=0.020$) than group n-S at 1 month. In addition, the DHI score at 1 month was significantly lower in group S than in group n-S ($p=0.040$). In correlation analysis between subjective symptom and objective parameters, the DHI score showed a significant correlation with the occurrence of overt CS ($p=0.028$) and Perez-Rey score (coefficient of variance; $p=0.006$) at 1 month.

Conclusions: Early steroid treatment in AVN would be helpful for relieving symptoms and the improvement of vestibular ocular reflex function in the recovery phase.

Keywords: Vestibular neuronitis; Vestibular function test; Steroids; Head impulse test

INTRODUCTION

Acute vestibular neuritis (AVN) is a condition that arises from the abrupt unilateral impairment of peripheral vestibular function. The characteristic feature of AVN is acute spontaneous vertigo, which is often accompanied by symptoms such as nausea, vomiting, and balance problems. In the majority of cases, AVN patients improve spontaneously and return to their daily lives, but some patients experience residual symptoms with chronic course [1]. Patients with AVN usually receive conservative therapy including sedatives or antiemetics as the underlying pathophysiology is not exactly known. In recent, viral etiology such as herpes simplex virus 1 inflammation has become increasingly recognized as a possible contributing factor rather than labyrinthine ischemia [2].

The treatment policy for peripheral recovery of function after AVN consists of corticosteroids, antiviral therapy, and vestibular exercises. Based on the theory of viral etiology in AVN, a combination of corticosteroids and an
antiviral agent have been tried. One recent meta-analysis supports the effectiveness of steroids in vestibular neuritis [3], while another suggests no benefit [4]. In other words, the efficacy of corticosteroids in previous studies is still conflicting.

The caloric test has been regarded as the gold standard for testing lateral semicircular canal function and diagnosis of AVN. So, the results of most studies on the recovery of AVN were based on the caloric test [5]. However, the reduced vestibular response from the caloric test could not demonstrate predictive capability for chronic symptoms in AVN [6]. The video head impulse test (vHIT) is utilized as a novel tool for diagnosis and follow-up of AVN. We previously demonstrated that new saccadic behaviors of conventional head impulse test paradigm (HIMP) and suppression HIMP (SHIMP) reflecting dynamic vestibular deficits can predict recovery of vestibular symptoms in patients with AVN [1].

The aim of this study was to evaluate the efficacy of early steroid treatment for AVN in objective and subjective methods. We conducted a retrospective case-control study according to whether the patients were treated with early steroids.

**METHODS**

**Ethics Statement**
The study meets the standards of the Helsinki Declaration and was approved by the Ethics Committee of the Kangbuk Samsung Hospital (No. 2023-03-028-001). Informed consent was waived due to the study’s retrospective nature.

**Selection of Participants**
We retrospectively analyzed patients with AVN who were admitted to our hospital through the Department of Emergency Medicine by medical chart review from February 2018 to August 2022. Inclusion criteria were: (1) a history of acute onset of severe and prolonged vertigo, (2) spontaneous horizontal-torsional nystagmus (SN), (3) ipsilateral deficit of the horizontal semicircular canal on the head impulse test, (4) a canal paresis (CP) of 25% or more on bithermal caloric test. Exclusion criteria were: (1) acute hearing loss, (2) neurologic signs or symptoms that imply a central lesion, (3) prior medical history of neuro-otologic disorders, (4) treatment started after 24 hours, and (5) inability to receive steroid treatment due to underlying disease such as severe hypertension (blood pressure, >180 mmHg systolic or >110 mmHg diastolic), severe diabetes mellitus (DM; fasting blood glucose level >180 mg/dL), glaucoma, peptic ulcer disease, psychiatric disorders, or pregnancy [7].

The patients in the study did not receive any specific guidance or instruction regarding personalized vestibular rehabilitation. Instead, they were advised to restart their daily activities as soon as possible. All included patients were hospitalized until acute symptoms subsided and patients had an outpatient follow-up visit 1 month after the onset of symptoms. Initially, we used video-nystagmography and for evaluating the patients. These tests were also performed at the scheduled outpatient visit. The degree of SN and several vHIT parameters were quantitatively assessed in our study. This study protocol was approved by the institutional review board of our institute.

**Vestibular Function Tests**
Eye movements were recorded with a commercial binocular video-nystagmography system (SLMED) to measure SN without visual fixation. The bithermal caloric test was conducted with simultaneous eye movement tracking using a video-based system. Each ear was stimulated with a constant flow of water (50 mL) at alternate temperatures of 30 °C for the cold test and 44 °C exceeding for the warm one for a duration of 40 seconds. Each irrigation procedure was conducted at a 5-minute interval. Subsequently, the maximum slow-phase velocity of nystagmus was assessed, and CP was determined using Jongkees’ formula. An abnormal CP was defined as 25% or higher.

In this study, HIMP and SHIMP were performed using the vHIT device (ICS Impulse, Otometrics). In HIMP tests, individuals were instructed to maintain their gaze at an earth-fixed dot on a wall about 100 cm away from the subject. The examiner tilted the patient’s head 30° below horizontal to bring the horizontal semicircular canal into the plane of head rotation. The examiner manually administered approximately 20 horizontal head impulses to each side, with unpredictable timing and direction. The target amplitude of the head rotation range was 15° to 20°, and the peak head velocity of the impulses was about 150°/sec to 250°/sec. The
SHIMP followed the same procedure as the HIMP, with the only variation being that patients were instructed to focus on a target that moved along with their head. The moving target was a laser dot projected by a small laser pointer mounted on the goggles.

In HIMP, we assessed the vestibulo-ocular reflex (VOR) gain in the horizontal semicircular canal plane. We categorized corrective saccades (CS) as covert if they occurred prior to the completion of head movement, or as overt if they started after the completion of head movement [8]. To be considered a CS, the amplitude of the saccade needed to exceed 100°/sec, and their peak velocity was recorded. We also measured the occurrence and peak velocity of both covert and overt CS. And if individual head impulse had the combination of covert and overt saccade, we counted each overt and covert saccade on data. For the calculation of VOR gain, we calculated the area under the curves from head impulse onset to the backcrossing of zero velocity. We defined VOR gain as the ratio of the area under the eye velocity to the area under the head velocity (Fig. 1).

The occurrence of CS was demonstrated by calculating the percentage of impulses with saccadic responses in each vHIT. Similarly, in SHIMP, we assessed the VOR gain, occurrence, and peak velocity of anti-CS. The occurrence and peak velocity of anti-CS in HIMP were defined in the same way.

To assess the organization pattern of the saccadic response appearances in the time domain, we used the Perez-Rey score (PR score, coefficient of variance) and measured it with a software called HitCal developed and tested by the manufacturer (Jorge Rey-Martinez). To calculate the PR, the software uses an algorithm that has been previously published. The algorithm of the software computes the PR score for each group. This value is derived from the variability of the time it takes for reflexion saccades to occur in all instances of the same test. In this measure, a PR value of 0 indicates the highest concentration of responses, while a PR value of 100 indicates the most scattered responses (Fig. 2) [9]. In other words, a low PR score means that the reflexion saccades occur almost similarly time-locked to the initiation of the head impulse, and a high PR score means that the reflexion saccades occur at different periods.

### Questionnaires

The severity of symptoms was evaluated using the Dizziness Handicap Inventory (DHI), a validated questionnaire consisting of 25 items. This questionnaire assesses both physical and emotional symptoms, as well as functional impairment caused by dizziness [10]. DHI score was evaluated at the first visit and 1-month follow-up visit.

### Group Evaluation

The patients who visited our hospital from February 2018 to June 2020 only received symptom-relieving therapy including vestibular sedatives and antiemetics (group n-S). Otherwise, the patients who visited our hospital from July 2020 to August 2022 received both symptom-relieving medications and oral steroids (methylprednisolone 48 mg for 7 days, followed by 3 days of dose tapering; group S).

The following data were compared between group n-S and group S: age, sex, hypertension, and DM; degree of SN and initial CP in video-nystagmography; VOR gain, occurrence and peak velocity of both covert and overt CS, and PR score of HIMP both at initial and 1 month; VOR gain, occurrence and peak velocity of anti-CS, and PR score of SHIMP both at initial and 1 month.

---

**Fig. 1.** The vestibulo-ocular reflex (VOR) gain was defined by the ratio of the area under the eye velocity (green line) to the area under the head velocity (blue line). The patient with deficit of VOR gain shows corrective saccades (red line) before (covert saccade) or after (overt saccade) crossing zero of the head velocity.
Data Analysis

The statistical analyses were conducted using IBM SPSS Statistics ver. 24.0 (IBM Corp.). We utilized the Mann-Whitney test to compare various clinical parameters between group n-S and group S. In addition, the Spearman correlation test was used to identify a relationship between objective parameters which was statistically significant in the Mann-Whitney test and subjective symptoms at 1 month.

RESULTS

A total of 46 patients (34 male and 12 female patients; mean age, 58.44±11.38 years; age range, 38–84 years) with AVN were included in the present study. Among them, 21 patients were assigned to group S, whereas 25 patients were assigned to group n-S. There was no significant difference between the two groups in age, sex, and the presence of hypertension or DM (Table 1). Various parameters of vestibular function tests (VFTs) showed no significant results between the two groups at initial. The results of DHI scores at the onset of the symptoms also did not differ between the two groups (Table 2).

All parameters of VFTs showed improvement after 1 month of symptom onset in all patients. Among these changes, group S showed a significantly lower occurrence (p=0.034) and peak velocity (p=0.020) of overt CS than group n-S. PR score was also significantly lower in group S than in group n-S (p=0.048). Finally, the DHI score was lower in group S than group n-S (p=0.040) at 1 month (Table 3).

In correlation analysis between objective parameters and subjective symptoms at 1 month, there was a significant correlation between PR score and DHI score (r²=0.398, p=0.006) (Fig. 3A). The occurrence of overt CS also showed

![Fig. 2. Two video head impulse test examples according to Perez-Rey (PR) scores. A low coefficient of variance, PR value indicates gathered responses, while a high PR value indicates scattered responses. This scores were measured with a software called Hit-Cal developed and tested by the manufacturer (Jorge Rey-Martinez).](https://doi.org/10.21790/rvs.2024.007)
Table 2. The results of vestibular function tests and DHI score at initial

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group S (n=21)</th>
<th>Group n-S (n=25)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Video-nystagmography</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spontaneous nystagmus</td>
<td>15.48±6.64</td>
<td>18.20±11.18</td>
<td>0.490</td>
</tr>
<tr>
<td>Canal paresis</td>
<td>96.12±18.32</td>
<td>97.92±15.11</td>
<td>0.640</td>
</tr>
<tr>
<td>HIMP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VOR gain</td>
<td>0.51±0.19</td>
<td>0.47±0.16</td>
<td>0.622</td>
</tr>
<tr>
<td>Occurrence of covert saccade</td>
<td>31.33±29.05</td>
<td>26.36±28.35</td>
<td>0.456</td>
</tr>
<tr>
<td>Peak velocity of covert saccade</td>
<td>171.22±98.54</td>
<td>156.20±120.40</td>
<td>0.710</td>
</tr>
<tr>
<td>Occurrence of overt saccade</td>
<td>80.44±24.70</td>
<td>81.60±23.67</td>
<td>0.661</td>
</tr>
<tr>
<td>Peak velocity of overt saccade</td>
<td>215.78±48.91</td>
<td>236.04±65.39</td>
<td>0.323</td>
</tr>
<tr>
<td>PR score</td>
<td>68.00±27.84</td>
<td>71.16±26.56</td>
<td>0.368</td>
</tr>
<tr>
<td>Combination of overt and covert saccade</td>
<td>14 (66.7)</td>
<td>15 (60.0)</td>
<td>0.644</td>
</tr>
<tr>
<td>SHIMP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VOR gain</td>
<td>0.41±0.02</td>
<td>0.37±0.17</td>
<td>0.767</td>
</tr>
<tr>
<td>Peak velocity of antisaccade</td>
<td>130.83±115.98</td>
<td>122.40±91.92</td>
<td>0.340</td>
</tr>
<tr>
<td>DHI score</td>
<td>78.16±18.98</td>
<td>76.89±12.80</td>
<td>0.236</td>
</tr>
</tbody>
</table>

Values are presented mean±standard deviation or number (%).

Group S, group that received steroid treatment; group n-S, group that did not receive steroid treatment.

HIMP, head impulse test paradigm; VOR, vestibulo-ocular reflex; PR score, Perez-Rey score; SHIMP, suppression HIMP; DHI, dizziness handicap inventory.

Table 3. The results of vestibular function tests and DHI score at 1 month after symptoms onset

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group S (n=21)</th>
<th>Group n-S (n=25)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Video-nystagmography</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spontaneous nystagmus</td>
<td>1.76±2.13</td>
<td>1.83±1.65</td>
<td>0.582</td>
</tr>
<tr>
<td>HIMP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VOR gain</td>
<td>0.64±0.24</td>
<td>0.76±0.28</td>
<td>0.252</td>
</tr>
<tr>
<td>Occurrence of covert saccade</td>
<td>36.72±37.88</td>
<td>18.89±29.20</td>
<td>0.151</td>
</tr>
<tr>
<td>Peak velocity of covert saccade</td>
<td>114.06±124.09</td>
<td>168.52±123.83</td>
<td>0.155</td>
</tr>
<tr>
<td>Occurrence of overt saccade</td>
<td>44.78±40.85</td>
<td>73.80±34.39</td>
<td>0.034*</td>
</tr>
<tr>
<td>Peak velocity of overt saccade</td>
<td>119.83±93.92</td>
<td>189.44±63.20</td>
<td>0.020*</td>
</tr>
<tr>
<td>PR score</td>
<td>32.06±32.65</td>
<td>48.04±29.79</td>
<td>0.048*</td>
</tr>
<tr>
<td>Combination of overt and covert saccade</td>
<td>9 (42.9)</td>
<td>10 (40.0)</td>
<td>0.739</td>
</tr>
<tr>
<td>SHIMP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VOR gain</td>
<td>0.66±0.30</td>
<td>0.58±0.23</td>
<td>0.391</td>
</tr>
<tr>
<td>Peak velocity of antisaccade</td>
<td>230.93±101.83</td>
<td>265.32±90.13</td>
<td>0.233</td>
</tr>
<tr>
<td>DHI score</td>
<td>6.00±5.86</td>
<td>19.52±22.49</td>
<td>0.040*</td>
</tr>
</tbody>
</table>

Values are presented mean±standard deviation or number (%).

Group S, group that received steroid treatment; group n-S, group that did not receive steroid treatment.

HIMP, head impulse test paradigm; VOR, vestibulo-ocular reflex; PR score, Perez-Rey score; SHIMP, suppression HIMP; DHI, dizziness handicap inventory.

* p<0.05.

a significant correlation with the DHI score ($r^2=0.325$, $p=0.028$) (Fig. 3B). Otherwise, the peak velocity of overt CS was not correlated with the DHI score ($r^2=0.023$, $p=0.878$).

**DISCUSSION**

In this study, we found that steroid treatment within 24 hours in AVN is helpful for reducing vestibular symptoms at 1 month. In addition, it appears that peripheral vestibular loss can be recovered more quickly by early steroid treatment. The results of the present study correspond with those of earlier studies which reported that steroid treatment is helpful for recovery of AVN.

Previous studies have shown that using steroids as a treatment can effectively improve clinical parameters in patients with AVN during long-term monitoring [5,11].
They mainly assessed the effect of steroid treatment by caloric recovery and questionnaire. However, the caloric test can probe the VOR at a very low frequency (0.002–0.004 Hz) which is far below the physiologic range (0.5–8 Hz). However, there are few reports regarding the results of vHIT after steroid treatment in AVN. In the present study, we investigated VOR function with vHIT of which frequency is similar to natural stimuli experienced in everyday life.

Similar to AVN, the precise etiology of idiopathic sudden hearing loss, optic neuritis, and Bell’s palsy is unknown. The most established theory in the pathophysiology of the above diseases is the inflammation of the nerve which is the same as for AVN. In the treatment of Bell’s palsy, combination therapy with steroid treatment and an antiviral agent such as acyclovir was shown to be effective. However, the results of previous studies which evaluated the efficacy of steroid treatment in AVN are still controversial. The vestibular nerve is susceptible to entrapment caused by inflammatory swelling of the nerve because it is surrounded by a long and narrow bony canal. One of the effects of glucocorticoid in AVN may be a reduction in nerve inflammation, as well as nerve edema and swelling, thus reducing entrapment and nerve damage [12].

In addition to recovering peripheral vestibular function, steroid treatment might be also helpful in restoring central vestibular compensation. Olabi et al. [13] reported that treatment with glucocorticoid speeds up central vestibular compensation after unilateral vestibular loss in animal study. The vestibular commissural inhibitory system links the vestibular nuclei of two sides in the brainstem, both in causing the initial severe oculomotor and postural symptoms of vestibular deafferentation, and in the subsequent recovery that takes place in the early stages of compensation. The author suggested that GABAergic neurotransmission within this commissural system might be modulated by glucocorticoid. Kitahara et al. [14] also proposed that glucocorticoid would induce central vestibular plasticity more quickly even in the phase when peripheral vestibular loss remains. Therefore, early peripheral vestibular recovery by reducing the nerve inflammation and central vestibular compensation through the vestibular commissural system are postulated as the mechanism of steroid treatment in AVN recovery.

In our analysis, we excluded the patients who received steroid treatment after 24 hours of symptom onset to highlight the timing of steroid treatment. According to a previous study, patients with AVN treated with steroid treatment within 24 hours after symptom onset had a
better compensation of vestibular function compared with the patients who received steroid treatment after 25 to 72 hours [15]. Karlberg et al. [11] also suggested that early steroid treatment administration after onset of AVN improves long-time recovery of vestibular function and decreases length of hospital stay. Our results are different from those of Yoo et al. [16] who studied the efficacy of steroid therapy in AVN conducted as a prospective randomized controlled trial. The steroid treatment showed no difference in the improvement of AVN in the study. However, they did not consider the onset timing of AVN in inclusion criteria. If information on the timing of steroid administration was included in the criteria, the possibility that the results would be different cannot be excluded from the study.

We previously reported the importance of saccadic pattern change in the recovery phase of AVN [1]. According to the research, not only VOR gain but also the occurrence of CS or PR score were valuable parameters in vHIT. Furthermore, those parameters were well correlated with subjective symptoms in the chronic phase of AVN. In the present study, we used only DHI as subjective indicator because all patients discharged if acute symptoms subsided and they felt they can resume daily activities, and period of hospitalization was almost similar. We could identify the correlation between some parameters of vHIT and DHI score. It is presumed that the reorganization of the saccadic pattern has a role in vestibular compensation with unilateral vestibular loss [17]. Even though the precise mechanism by which patients get an organized eye response after unilateral VOR deficit remains unclear, the results obtained in our study indicate that the PR score is a significant factor in the recovery phase of AVN.

This study has important limitation, however, mostly stemming from its small sample size and retrospective case-control design. Our study was based on a single institution who are admitted through the emergency department of our hospital. Inclusion criteria were also restrictive because patients who could not use steroid treatment due to underlying diseases were excluded. Also, some people did not return to the hospital, maybe due to improvement of symptoms.

However, the results do suggest that the early steroid treatment group had better improvements in several vestibular tests and DHI.

In addition, patients were only recommended to continue daily activities as soon as they could totally confirm the effect of steroid treatment even though customized vestibular exercise is necessary in general. Therefore, large-scale, adequately powered, randomized controlled trials would be needed to verify the effectiveness of steroid treatment in the future.

The major findings in this study suggest that early steroid treatment in patients with AVN might help the recovery of both objective parameters and subjective symptoms at 1 month. The timing of treatment, within 24 hours of symptoms onset, seems to be very important. Finally, the reorganization of the saccadic pattern in vHIT should be noted when we follow up on a patient with AVN.

**ARTICLE INFORMATION**

**Funding/Support**

None.

**Conflicts of Interest**

No potential conflict of interest relevant to this article was reported.

**Availability of Data and Materials**

All data generated or analyzed during this study are included in this published article. For other data, these may be requested through the corresponding author.

**Authors’ Contributions**

Conceptualization: MBK, JYL; Data curation, Software, Visualization: SHK; Formal analysis, Methodology, Project administration, Supervision: MBK; Investigation, Resources: HSK, SHK; Validation: JYL; Writing–original draft: JYL; Writing–review & editing: MBK, HSK.

All authors read and approved the final manuscript.

**ORCID**

Jung-Yup Lee, https://orcid.org/0000-0002-0823-4245
Hyun-Seok Kang, https://orcid.org/0000-0001-5443-0776
Sang-Hyun Kim, https://orcid.org/0000-0002-2517-5523
Min-Beom Kim, https://orcid.org/0000-0001-8849-5148

https://doi.org/10.21790/rvs.2024.007
REFERENCES