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RESEARCH ARTICLE

Effect of steroid therapy on saccule and semicircular canals function in patients with unilateral sudden sensorineural hearing loss

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Abstract

Background and Aim: In unilateral sudden sensorineural hearing loss (SSNHL), the vestibular system may be involved in addition to the auditory system. Several hearing assessments have shown that the disease course and the patient's improvement, at least two step of vestibular assessments can help in better control of the patient's balance function. The aim of this study was to evaluate the results of cervical vestibular evoked myogenic potentials (cVEMP) and video head impulse test (vHIT) used for assessment of saccule and semicircular canals before and after steroid therapy.

Methods: Twenty three patients with SSNHL were evaluated for auditory and vestibular function before and after steroid therapy. The results of cVEMP and vHIT were compared between intact and impaired ears and between pretest/posttest stages before and after treatment.

Results: For 26.08% of patients, the cVEMP response was absent in the affected ear, but after treatment it was reported for all patients. There was a significant difference in vestibulo-ocular reflex (VOR) gain for both posterior and anterior semicircular canals of affected ear before and after treatment, but it was not significantly different after treatment as VOR gain increased.

Conclusion: Dysfunction of nervous and vestibular systems in SSNHL is possible. Steroid therapy can improve the vestibular function and hearing of these patients. Therefore, vestibular evaluation can be used to determine the extent of lesions in SSNHL.

Keywords: Unilateral sudden sensorineural hearing loss; vestibular evoked myogenic potentials; saccule; semicircular canals; video head impulse test

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Introduction

Sudden sensorineural hearing loss (SSNHL) was first described by De Kleyn in 1944 and was defined as hearing loss of at least 30 dB over three contiguous audiometric frequencies and occurs within three days or less [1]. Its

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incidence is 5−20 cases per 100000 people and is equal in men and women. SSNHL is more common in patients aged 50−60 years [2-4]. There are vaious reasons for SSNHL occurrence including infections, traumas, tumors, autoimmune diseases, ototoxic drugs, and metabolic/neurologic diseases [5]; however, theoritically, the main reasons are: viral infections, vascular blockage, cochlear membrane disruption and autoimmune diseases. In many cases, SSNHL is classified as "idiopathic" [6]. In SSNHL, hearing loss is accompanied by tinnitus as 70−90% and vertigo as 20−70% [7,8]. Vertigo is mostly found in patients with profound hearing loss; hearing improvement in patients with accompanying vertigo is less that patients without any vertigo [9]. The close proximity of vestibule to cochlea cause simultaneous vertigo and hearing loss (specially in high frequency). In this way, cochlear damage is transmitted to vestibular organs through ductus reuniens [10]. There are different treatments for SSNHL including high-dose oral steroids, antiviral drugs, steroids combined with antiviral or vasodilators, anticoagulants, vitamin B complex, benzodiazepine, magnesium, plasma expanders, carbogen inhalation, hyperbaric oxygen and intratympanic steroids. Steroid therapy is the most acceptable method among others [11]. Corticosteroids can lead to the improvement of inner ear inflammation; they are mostly given by mouth rather than by injection [4].

Cervical vestibular evoked myogenic potential (cVEMP) is an inhibitory electromascular response recorded from ipsilateral sternocleidomastoid (SCM) via sound, vibration and electrical stimulation. It is a saccular-originated response with a positive peak at a mean latency of 13 ms (p13) and a negative trough at 23 ms (n23). Video head impulse test (vHIT) is a relatively new objective and fast diagnostic method for the evaluation of semicircular canal evaluation through vestibulo-ocular reflex (VOR) [12,13].

By assuming the involvement of vestibular and nervous systems in SSNHL and considering the necessity of residual vestibular function evaluation (as a latent problem) in these patients before and after steroid therapy as the conventional treatment, the aim of the present study was to evaluate the results of cVEMP and vHIT used for assessment of superior and inferior vestibular nerves in saccule and semicircular canals of patients with SSNHL before and after steroid therapy.

Methods

The study was performed at Audiology Clinic of Tehran University of Medical Sciences (TUMS). This project is approved by the Research Ethics Committee of TUMS (Code: IR.TUMS.FNM.REC.1397.062). It is a comparative cross-sectional study conducted on 23 subjects with monaural SSNHL (9 females and 14 males; mean age, 38 ± 11.69 years). They were selected using convenience sampling technique from among patients referred to the Ear, Nose, and Throat Clinic of Amir Alam Hospital from Feb 2018 to July 2018. The inclusion criteria were: age < 60 years old; referral to clinic within one week after SSNHL; no other diseases that can cause vertigo and vestibular problems such as diabetes and vascular compression syndrome, regardless of having vertigo or not; cervical pain; limited cervical movement (according to the patients and test results), and no disease in middle ear. Exclusion criteria were patients' lack of cooperation, neck pain during vHIT, and nausea and vomiting during vHIT.

After obtaining written consent from subjects and evaluation of inclusion criteria, following tests were performed: otoscopy, tympanometry and pure tone audiometry air conduction (AC) and bone conduction (BC) at octave frequencies from 250 to 8000 Hz. Patients with mild to profound hearing loss were included in the study. The cVEMP was performed by Bio-logic Navigator Pro (Germany); the electrode array included non-inverting on the middle part of SCM, inverting on the upper part of sternum, and ground on forehead. For contracting SCM, patients were in supine position with head turned to the opposite side. Stimuli were delivered through inserting earphone to the ipsilateral ear. Stimulation had following characteristics: 500 Hz tone burst (2 ms rise/fall time and 0 ms plateau),

rarefaction polarity, 95 dB nHL stimulation level (5 per second), 5000 gain, 10−1000 Hz filtering, 50−100 ms time window, and 150 sweeps. To test the repeatability of the response, each intensity level of response was tested twice after a rest. To seek threshold, the test was started at 95 dB nHL and intensity was decreased in 5 dB steps. The lowest level in which a repeatable response was recorded, was defined as threshold. This process repeated for the second ear as well [14].

The vHIT was performed by ICS Head Impulse (GN Otometrics, Denmark). The goggle was put on the patients and the test process was explained to them. Patients were asked to look at the point on the wall from a 1 m distance. After calibration, for testing lateral semicircular canal, the tester put her hands on the patients' head and performed 10 fast impulse movements (at least 100 degrees per second) with 5−20 degrees to the sides. After each impulse, head remained in the last position to avoid masking any covert saccade. The timing and direction of impulses were different to avoid prediction [15]. After a rest, the stimulation of posterior and anterior semicircular canals were performed. For right posterior and left superior semicircular canals, patients' head was turned 45 degrees to the right with one hand on top of the head and the other beneath the chin. After a rest, stimulation of left posterior and right superior semicircular canals were performed by turning head 45 degrees to the left. Each stage was performed twice.

Steroid therapy included taking 1 mg/kg prednisolone every morning with an oral omeprazole every 12 hours for 10 days. If hearing was not improved after 10 days, intratympanic injection of 4 mg/mL dexamethasone twice a week for two weeks was prescribed (a total of four injections given in supine position with the head 45 degrees turned to the unaffected side). After one month, all procedures were performed again for all patients.

Kolmogorov-Smirnov test results showed that all collected data had normal distribution. Then, paired t-test was used for comparing quantitative parametrs of cVEMP and VOR gain in normal and abnormal ears before and after treatment and comparing the mean of these parameters before and after treatment between two ears. McNemar's test was used for comparing the cVEMP incidence ratio in each ear before and after treatment and between ears before and after treatment. All analyses were performed in SPSS 16 software.

Results

Table 1 summarizes the frequency of SSNHL configurations and rates in participants before and after steroid therapy. As can be seen, before treatment, flat audiogram was seen in 60.9% of cases and severe SSNHL was seen in 43.5% of cases. The highest rate of improvement following treatment (13%) was related to the ascending audiogram and for mild SSNHL. Fig. 1 shows changes in SSNHL after treatment. The cVEMP was seen in 73.9% of involved ears and 100% of normal ears before treatment. There was a significant difference in incidence ratio between two ears before treatment ($p = 0.031$). After treatment, cVEMP was recorded successfully in both ears.

Table 2 summarizes the quantitative results of cVEMP in each ear before and after treatment. There was a significant difference in the mean latency of p13 and the mean amplitude of p13-n23 between ears and between pretest/posttest stages before and after treatment ($p > 0.05$). The cVEMP threshold before and after treatment in the involved ear, the mean amplitude of p13-n23 before treatment between two ears, and the mean asymmetry ratio between pretest/posttest stages showed statistically significant differences ($p < 0.05$).

Table 3 summarizes the mean VOR gain for each semicircular canal. The mean VOR gain in posterior semicircular canal in normal ear before and after treatment and also between anterior and posterior canals in each ear after treatment was significantly different ($p < 0.05$). The mean VOR gain in each ear after treatment showed no significant difference ($p > 0.05$).

Discussion

Subjects with SSNHL might suffer from vestibular dysfunction as well [16-18]. This occurs

Table 1. Pattern and degree of hearing loss and degree of hearing recovery before and after treatment in patients with unilateral sudden sensorineaural hearing loss

Hearing loss		Pre-treatment (%)	Post-treatment (%)		
			Full recovery	Partial recovery	No recovery
Pattern					
	Sloping	21.7	8.7	0	13
	Rising	17.4	13	4.4	0
	Flat	60.9	8.7	39.1	13.1
Degree					
	Mild	17.4	13	0	4.4
	Moderate	39.1	13	13	13.1
	Severe	43.5	4.4	26	13.1

Full recovery: recovery of SRT and PTA thresholds in range of 10dB of those thresholds before hearing loss; Partial recovery: recovery of PTA and SRT thresholds in range of 50% of hearing thresholds before hearing loss; No recovery: recovery in range of less than 50% of hearing level before hearing loss.

due to the close proximity of vestibule to cochlea or deficit in common blood supply of these two organs [10,19]. In the present study, 13% of patients had vertigo as well which decreased to 8.7% after treatment. Studies have reported different incidence rates for vertigo. Yu and Li reported that 20−60% of patients with SSNHL had accompanying vertigo, and stated that vertigo is not a determining factor since there are some cases suffering from vestibular dysfunction without any vertigo [8]. Biochemical changes in the inner ear fluid might be another

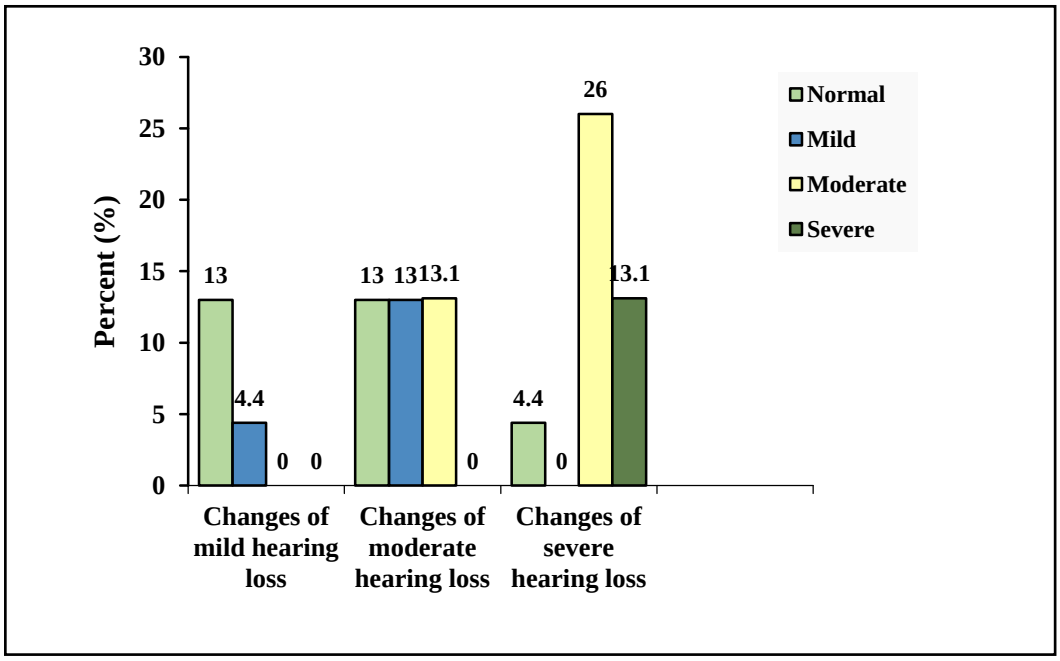

Fig. 1. Frequency distribution of changes in sudden sensorineural hearing loss after treatment.

Table 2. Comparison of mean and standard deviation of cervical evoked myogenic potential parameters in patients with unilateral sudden sensorineural hearing loss between ears and between pre- and post-treatment stages

Parameter	Mean (SD)- pre-treatment			Mean (SD)- post-treatment			p	
	Normal ear (n = 23)	Involved ear (n = 17)	Pre-treatment	Normal ear (n = 23)	Involved ear (n = 23)	Post-treatment	Pre- and post-treatment/ normal	Pre- and post-treatment/ involved
Latency (ms)	15.47 (1.97)	16.40 (2.61)	0.1	15.68 (2.03)	14.73 (3.26)	0.22	0.10	0.33
Threshold (dB nHL)	81.30 (4.32)	82.06 (5.6)	0.65	81.09 (4.25)	80.7 (5.57)	0.84	0.74	0.046
Amplitude (Mv)	150.64 (220.38)	146.09 (100.90)	0.006	210.22 (157.90)	177.49 (128.79)	0.30	0.67	0.26
Amplitude asemmetry ratio	0.46 (0.35)		0.032	0.26 (0.18)				

In 6 patients cVEMP response was absent before treatment.

reason for transmission of dysfunction from cochlea to vestibule [19].

In the present study, vestibular function was evaluated in patients with SSNHL before and after treatment by using cVEMP and vHIT. The cVEMP response in affected ear changed significantly after treatment such that cVEMP was recorded in both ears. In 39.1% of patients, the amplitude asymmetry was present and decreased significantly after treatment; however, amplitude asymmetry was still present in 17.39% of patients.

The cVEMP results reported normal function of saccule, inferior vestibular nerve, lateral vestibular nucleus, vestibulo-spinal tract and SCM. The deficit in each of the mentioned components can remove cVEMP [20]. Based on the results of the present study, there is possibility of dysfunction in saccule, inferior vestibular nerve and related pathways in patients with SSNHL. Previous studies reported 0−77% chance of cVEMP abnormalities in patients with SSNHL and this was attributed to vestibular hair cell loss [17,20]. Our result is consistent with the results of Peng et al. who showed that results of cVEMP and ocular vestibular evoked myogenic potential (oVEMP) improved significantly after SSNHL treatment [21]. Chen and Young conducted a study to achieve a differential diagnosis of Meniere's disease and SSNHL. They showed that 3 out of 14 patients with SSNHL had abnormal cVEMP. After treatment, one of them showed normal cVEMP [22].

In the present study, vHIT was performed and VOR gain was examined. It was shown that VOR gain in posterior and anterior semicircular

Table 3. Comparison of mean (standard deviation) of semicircular canal vestibule-ocular gain in normal and involved ear in patients with unilateral sudden sensorineural hearing loss pre- and post-treatment

Semicircular canal gain	Mean (SD)- pre-treatment			Mean (SD)- post-treatment			p	
	Normal ear (n = 23)	Involved ear (n = 23)	Pre-treatment	Normal ear (n = 23)	Involved ear (n = 23)	Post-treatment	Pre- and post-treatment/ normal	Pre- and post-treatment/ involved
Posterior	0.80 (0.15)	0.69 (0.19)	0.017	0.89 (0.10)	0.88 (0.13)	0.816	0.008	0.000
Anterior	0.76 (0.16)	0.76 (0.13)	0.891	0.89 (0.19)	0.83 (0.15)	0.377	0.009	0.076
Horizontal	1.03 (0.16)	1.03 (0.22)	0.922	1.00 (0.1)	1.01 (0.15)	0.672	0.407	0.721

canals was significantly diferent before and after treatment in both ears. VOR gain significantly increased after treatment. Before treatment, VOR gain for posterior and anterior semicircular canals was less than 0.7 in 47.82 and 30.43% of patients, respectively. After treatment, it decreased to 8.7 and 13.04% respectively. There was also patients with abnormality in their non-affected ear which might be due to unclear problems. VOR is for fixating eyes on a target with making a head movement. This works by moving eyes at the same speed as head movement but in the opposite direction. Main components of VOR are semicircular canals, vestibular nuclei, ocular movement nuclei and extraocular muscles. The vHIT provides information about VOR gain and helps in diagnosis of vestibular dysfunction [23].

Pogson et al. in a retrospective study, used vHIT to evaluate semicircular canals in 27 subjects with SSNHL and found out that 74% of patients had posterior canal dysfunction, 41% superior canal dysfunction and 30% anterior canal dysfunction (reduced VOR gain). Posterior canal had the highest rate of dysfunction due to labyrinthine artery infarction [24]. Yao et al. studied 22 patients with vestibular neuritis and 30 patients with SSNHL by using vHIT and showed that posterior canal dysfunction was more prevalent in SSNHL patients, while dysfunction in all canals was more probable in patients with vestibular neuritis. They also stated that internal auditory artery dysfunction is the predominant cause of SSNHL but viral infection is main cause of vestibular neuritis [25].

In our study, patients received steroid therapy as the most common method for SSNHL treatment; however, its effect on vestibular labyrinth and efferents are not clear yet [26]. Steroids may improve prepheral system functions by their anti-inflammatory, anti-alergic, anti-edema and vaso-active properties [26]. They are effective when the damage is mild and reversible [27] and also is in the acute phase [26]. Therefore, the improvement of cVEMP and VOR gain under vHIT after steroid therapy in our study may be because that there was a mild damage limited to peripheral vestibular system.

Conclusion

Evaluation of vestibular function in addition to hearing assessment can estimate the degree of damage to peripheral system related to sudden sensorineural hearing loss (SSNHL). Based on the results, it can be concluded that saccule, semicircular canals and related pathways are involved in SSNHL and steroid therapy not only can improve auditory function but also vestibular function. It is suggested that vestibular rehabilitation should be used for the balance improvement of SSNHL patients.

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Conflict of interest

The authors declared no conflicts of interest.

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