International Journal of Medical and Pharmaceutical Research

Online ISSN-2958-3683 | Print ISSN-2958-3675
Frequency: Bi-Monthly
Available online on: https://ijmpr.in/

Research Article

Unilateral Sudden Sensorineural Hearing Loss: Effects Of Steroid On Vestibular Function

Dr Rahil Muzaffar[1], Dr Sonika Kotwal[2], Dr Abid Hussain[3]

[1]Associate professor, Department of ENT , Government Medical College Doda, Jammu and Kashmir, India
[2]Senior Resident, Department of ENT, Government Medical College Doda, Jammu and Kashmir, India
[3]Senior Resident , Department of ENT , Government Medical College Doda, Jammu and Kashmir, India

OPEN ACCESS

Corresponding Author:

Dr Sonika Kotwal
Senior Resident, Department of ENT, Government Medical College Doda, Jammu and Kashmir, India

Received: 21-09-2025
Accepted: 06-10-2025
Available online: 20-10-2025

ABSTRACT

Background: Unilateral sudden sensorineural hearing loss (SSNHL) may involve vestibular as well as auditory dysfunction. Vestibular assessment provides additional information on disease extent and treatment response. This study examined cervical vestibular evoked myogenic potentials (cVEMP) and video head impulse test (vHIT) outcomes before and after steroid therapy. **Methods:** Twenty-three patients with SSNHL underwent auditory and vestibular evaluation prior to and following treatment. cVEMP and vHIT results were compared between affected and unaffected ears, as well as between pre- and post-treatment stages. **Results:** In 26.08% of patients, cVEMP responses were absent in the affected ear at baseline; after therapy, responses were present in all cases. Vestibulo-ocular reflex (VOR) gain in the anterior and posterior semicircular canals of the affected ear improved significantly after treatment. Post-treatment VOR gain values were higher but not statistically different between ears. **Conclusion:** Vestibular dysfunction may accompany SSNHL. Steroid therapy can improve both hearing and vestibular function. Vestibular testing is a useful tool to determine the extent of inner ear involvement in SSNHL.

Keywords*: Sudden sensorineural hearing loss, cVEMP, saccule, semicircular canals, vHIT.*

INTRODUCTION

Sudden sensorineural hearing loss (SSNHL) was first described by De Kleyn in 1944 and is defined as a hearing loss of at least 30 dB over three contiguous audiometric frequencies and occurs within three days or less [1]. Its incidence is 5−20 cases per 100,000 people and affects men and women equally. SSNHL is more common in patients aged 50−60 years [2-4]. There are various reasons for SSNHL occurrence including infections, trauma, tumors, autoimmune diseases, ototoxic drugs, and metabolic/neurologic diseases [5]; however, theoretically, 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 often accompanied by tinnitus in 70−90% of cases and vertigo as 20−70% [7,8]. Vertigo is mostly found in patients with profound hearing loss and hearing improvement in patients with accompanying vertigo tends to be less than those without any vertigo [9]. The close proximity of vestibule to cochlea causes simultaneous vertigo and hearing loss (especially in high frequency). In this way, cochlear damage is transmitted to vestibular organs through ductus reunions [10]. There are different treatment options for SSNHL, including high dose oral steroids, antiviral drugs, steroids combined with antivirals 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 electro muscular response recorded from the 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). The video head impulse test (vHIT) is a relatively new objective and fast diagnostic method for the evaluation of semi-circular canal function 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 semi-circular canals in patients with SSNHL before and after steroid treatment.

METHODS

The study was performed at the Department of ENT Government medical college Doda. It was a comparative cross-sectional study conducted on 23 subjects with monaural sudden sensorineural hearing loss (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 ENT department from January 2023 to July 2023. The inclusion criteria were: age < 60 years; referral to clinic within one week after SSNHL onset; 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 included were lack of patient cooperation, neck pain during vHIT, and nausea or vomiting during vHIT. After obtaining written consent from subjects and evaluation of inclusion criteria, the 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 recorded by Bio-logic Navigator Pro (Germany). The electrode array comprised the non-inverting on the middle part of SCM, inverting on the upper part of sternum, and the ground on forehead. For contracting SCM, patients were positioned supine 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 gains, filtering between 10−1000 Hz, 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 was 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 semi-circular canal, the tester put her hands on the patient's 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 saccades. The timing and direction of impulses were different to avoid prediction [15]. After a rest, the stimulation of posterior and anterior semicircular canals was performed. For right posterior and left superior semi-circular canals, patient's 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 was performed by turning head 45 degrees to the left. Each stage was performed twice. Steroid therapy was given at 1mg/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 4mg/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 parameters 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 presents the distribution of SSNHL audiometric configurations before and after steroid therapy. Prior to treatment, a flat audiogram pattern was observed in 60.9% of patients with 43.5% showed severe SSNHL. Following therapy, the greatest recovery rate (13%) was seen in patients with an ascending audiogram configuration and those with mild SSNHL. Figure 1 illustrates the post-treatment changes in audiogram patterns. cVEMP responses were identified in 73.9% of affected ears compared with 100% of unaffected ears at baseline, with a significant interaural difference in response incidence ($p = 0.031$). After treatment, cVEMP responses were present in all ears. Quantitative cVEMP results are summarized in Table 2. Statistically significant differences were observed in mean p13 latency and p13–n23 amplitude between ears and across pre- and post-treatment stages ($p<0.05$). In addition, the cVEMP threshold in the affected ear, the interaural difference in mean p13–n23 amplitude before treatment, and the asymmetry ratio between pre- and posttreatment were all significantly different ($p < 0.05$). Table 3 outlines the mean VOR gain values for each semicircular canal. Significant differences were found in the posterior canal gain of the unaffected ear before and after treatment, as well as between anterior and posterior canal gains within each ear after therapy ($p < 0.05$). However, comparisons of VOR gain between ears after treatment revealed no significant difference ($p > 0.05$).

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.

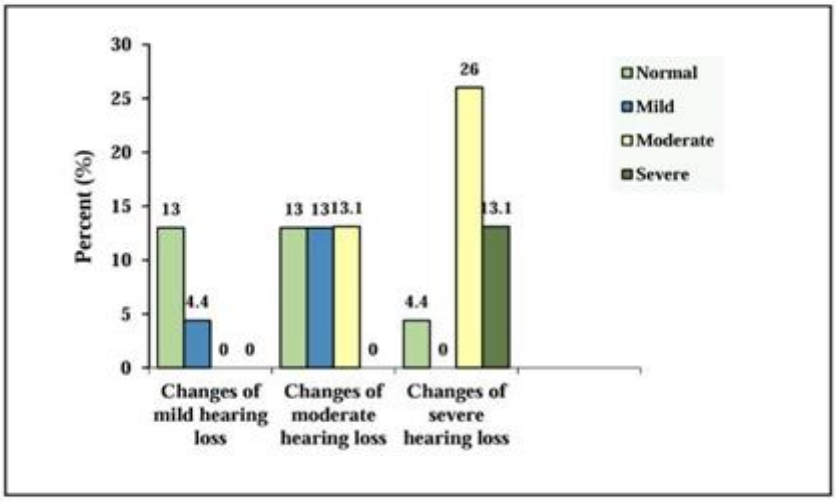

Fig. 1. Frequency distribution of changes in sudden sensorineural hearing loss after treatment.

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.

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

DISCUSSION

SSNHL frequently involves vestibular dysfunction due to the anatomical and vascular connections between cochlea and vestibule [10,16–19]. In this study, 13% of patients presented with vertigo, which declined to 8.7% after treatment. Reported rates vary widely; Yu and Li observed vertigo in 20–60% of SSNHL cases and emphasized that vestibular impairment can occur even in the absence of vertigo [8]. cVEMP findings demonstrated significant improvement after steroid therapy, with bilateral responses recorded in all patients. Amplitude asymmetry, initially present in 39.1%, decreased to 17.39% post-therapy, indicating partial but not complete recovery of saccular and inferior vestibular nerve function. These results are consistent with earlier reports showing cVEMP abnormalities in 0–77% of SSNHL cases [17,20] and improvement following treatment [21,22]. vHIT analysis showed that VOR gain in the anterior and posterior canals increased significantly after therapy. Before treatment, abnormal VOR gain (<0.7) was noted in 47.82% (posterior) and 30.43% (anterior) of patients; after therapy, this decreased to 8.7% and 13.04%, respectively. Some contralateral abnormalities were also identified, suggesting subclinical vestibular involvement. Our results parallel Pogson et al., who reported posterior canal dysfunction in 74% of SSNHL patients [24], and Yao et al., who showed that posterior canal deficits predominate in SSNHL, whereas vestibular neuritis more often affects all canals [25].

Steroid therapy remains the mainstay of SSNHL management, though its vestibular effects are less well established [26]. Improvements observed in cVEMP and vHIT in our cohort likely reflects recovery of reversible peripheral dysfunction through the anti-inflammatory and vasoactive properties of corticosteroids [26,27].

CONCLUSION

Vestibular dysfunction is common in sudden sensorineural hearing loss (SSNHL), affecting the saccule, semicircular canals, and associated pathways. Steroid therapy improved both auditory and vestibular outcomes, though residual abnormalities persisted in some patients. Comprehensive vestibular evaluation, combined with targeted rehabilitation, may enhance balance recovery in SSNHL.

Conflict of interest: None

REFERENCES

1. Schreiber BE, Agrup C, Haskard DO, Luxon LM. Sudden sensorineural hearing loss. The Lancet. 2010;375(9721):1203-11. Doi: 10.1016/S01406736(09)62071-7.
2. Kuhn M, Heman-Ackah SE, Shaikh JA, Roehm PC. Sudden sensorineural hearing loss: a review of diagnosis, treatment, and prognosis. Trends Amplif. 2011; 15(3):91-105.
3. Plontke SK. Diagnostics and therapy of sudden hearing loss. GMS Curr Top Otorhinolaryngol Head Neck Surg. 2018;16:Doc05.
4. Rauch SD, Halpin CF, Antonelli PJ, Babu S, Carey JP, Gantz BJ, et al. Oral vs intratympanic corticosteroid therapy for idiopathic sudden sensorineural hearing loss: a randomized trial. JAMA. 2011;305(20):2071-9.
5. Flint PW, Haughey BH, Robbins KT, Thomas JR, Niparko JK, Lund VJ, et al. Cummings otolaryngology – head and neck surgery e-book. 6th ed. Philadelphia: Sanders Elsevier; 2014.
6. Topuz E, Yigit O, Cinar U, Seven H. Should hyperbaric oxygen be added to treatment in idiopathic sudden sensorineural hearing loss? Eur Arch Otorhinolaryngol.
7. Michiba T, Kitahara T, Hikita-Watanabe N, Fukushima M, Ozono Y, Imai R, et al. Residual tinnitus after the medical treatment of sudden deafness. Auris Nasus Larynx. 2013;40(2):162-6.
8. Yu H, Li H. Vestibular dysfunctions in sudden sensorineural hearing loss: a systematic review and meta-analysis. Front 10.3389/fneur.2018.00045 Neurol. 2018;9:45.
9. Iwasaki S, Takai Y, Ozeki H, Ito K, Karino S, Murofushi T. Extent of lesions in idiopathic sudden hearing loss with vertigo: study using click and galvanic vestibular evoked myogenic potentials. Arch Otolaryngol Head Neck Surg. 2005;131(10):857-62.
10. Ben-David J, Luntz M, Podoshin L, Sabo E, Fradis M. Vertigo as a prognostic sign in sudden sensorineural hearing loss. Int Tinnitus J. 2002;8(2):127-8.

11. Filipo R, Covelli E, Balsamo G, Attanasio G. Intratympanic prednisolone therapy for sudden sensorineural hearing loss: A new protocol. Acta Otolaryngol. 2010; 130(11):1209-13.
12. Maheu M, Houde MS, Landry SP, Champoux F. The effects of aging on clinical vestibular evaluations. Front Neurol. 2015;6:205.
13. Duchoud L, Maire R. [The video Head Impulse Test in the vertiginous patient]. Rev Med Suisse. 2017; 13(577):1694-7.
14. McCaslin DL, Jacobson JP. Vestibular-Evoked Myogenic Potentials (VEMPs), in Jacobson JP, Shepard NT, editors. Balance function assessment and management. 2 nd ed. San Diego: Plural Publishing; 2016. P. 533-80.
15. Hougaard DD, Abrahamsen AR. Functional testing of all six semicircular canals with video head impulse test systems. J Vis Exp. 2019;(146).
16. Xiao–tong Z, Bin S, Wen–Juan Z, Min X, Jun–rong W. Concurrent symptoms and disease conditions in sudden deafness. J Otol. 2010;5(1):20-3.
17. Fujimoto C, Egami N, Kinoshita M, Sugasawa K, Yamasoba T, Iwasaki S. Involvement of vestibular organs in idiopathic sudden hearing loss with vertigo: an analysis using oVEMP and cVEMP testing. Clin Neurophysiol. 2015;126(5):1033-8.
18. Stamatiou G, Gkoritsa E, Xenellis J, Riga M, Korres S. Semicircular canal versus otolithic involvement in idiopathic sudden hearing loss. J Laryngol Otol. 2009; 123(12):1325-30.
19. Hong SM, Byun JY, Park CH, Lee JH, Park MS, Cha CI. Saccular damage in patients with idiopathic sudden sensorineural hearing loss without vertigo. Otolaryngol Head Neck Surg. 2008;139(4):541-5.
20. Korres S, Stamatiou GA, Gkoritsa E, Riga M, Xenelis J. Prognosis of patients with idiopathic sudden hearing loss: role of vestibular assessment. J Laryngol Otol. 2011;125(3):251-7.
21. Peng L, Chen R, Yuan H, Liang J. [The value of otolith function test in the prognosis of sudden sensorineural hearing]. Lin Chung Er Bi Yan Hou Tou Jing Wai Ke Za Zhi. 2016;30(4):272-6.
22. Chen CN, Young YH. Differentiating the cause of acute sensorineural hearing loss between Ménière's disease and sudden deafness. Acta Otolaryngol. 2006;126(1):2531.
23. Bansal S, Sinha SK. Assessment of VOR gain function and its test-retest reliability in normal hearing individuals. Eur Arch Otorhinolaryngol. 2016;273(10): 3167-73.
24. Pogson JM, Taylor RL, Young AS, McGarvie LA, Flanagan S, Halmagyi GM, et al. Vertigo with sudden hearing loss: audio-vestibular characteristics. J Neurol. 2016;263(10):2086-96.
25. Yao Q, Xu C, Wang H, Shi H, Yu D. Video head impulse test results suggest that different pathomechanisms underlie sudden sensorineural hearing loss with vertigoand vestibular neuritis: Our experience in fiftytwo patients. Clin Otolaryngol. 2018;43(6):1621-4.
26. Kitahara T, Kondoh K, Morihana T, Okumura S, Horii A, Takeda N, et al. Steroid effects on vestibular compensation in human. Neurol Res. 2003;25(3):287-9.
27. Moskowitz D, Lee K, Smith HW. Steroid use in idiopathic sudden sensorineural hearing loss. The Laryngoscope. 1984;94(5):664-6.