ORİJİNAL ARAŞTIRMA ORIGINAL RESEARCH

DOI: 10.24179/kbbbbc.2022-93010

Audiovestibular Findings in Superior Semicircular Canal Dehiscence Syndrome

Superior Semisirküler Kanal Dehissansı Sendromunda Odyovestibüler Bulgular

Berna Deniz AYDIN[a], Hatice Seyra ERBEK[b], Anı PARABAKAN POLAT[a], Evren HIZAL[a]

[a]Department of Audiology, University of Health Sciences Gülhane Faculty of Health Sciences, Ankara, Türkiye
[b]Department of Otorhinolaryngology, Head and Neck Surgery, Başkent University Faculty of Medicine, Ankara, Türkiye

ABSTRACT Objective: Superior semicircular canal dehiscence (SSCD) is a rare syndrome caused by a bone defect in the superior semicircular canal. The aim of this study was to evaluate the audiovestibular clinical findings in patients diagnosed with SSCD syndrome. **Material and Methods:** This study was designed as a clinical retrospective research. The archives of patients admitted to the otolaryngology clinic between June 2018 and June 2022 were examined. Records of ears diagnosed with SSCD syndrome have been reached. The audiovestibular symptoms, clinical findings, and high-resolution computed tomography (HRCT) scans of these ears were presented. **Results:** As a result of the retrospective evaluation, 5 ears diagnosed with SSCD syndrome were obtained. Audiovestibular symptoms and clinical findings such as pure tone audiometry, tympanometry, acoustic reflex, video head impulse test, videonystagmography, cervical and ocular vestibular evoked myogenic potentials (VEMP) test results were evaluated. HRCT scans of all ears were interpreted. **Conclusion:** Symptoms such as sound- and/or pressure-induced vertigo (Tullio phenomenon, Hennebert's sign), oscillopsia, hyperacusis, autophony, pulsatile tinnitus, aural fullness; findings such as elevated bone-conduction thresholds, increasing of VEMP wave amplitude, decreasing of VEMP response threshold, decreasing of semicircular canal gain suggestive of SSCD.

Keywords: Superior semicircular canal dehiscence syndrome; vestibular evoked myogenic potentials

ÖZET Amaç: Superior semisirküler kanal dehissansı [superior semicircular canal dehiscence (SSCD)], superior semisirküler kanaldaki kemik defektinin neden olduğu nadir görülen bir sendromdur. Bu çalışmanın amacı, SSCD sendromu tanısı alan hastalarda odyovestibüler klinik bulguları değerlendirmektir. **Gereç ve Yötemler:** Bu çalışma, klinik retrospektif araştırma olarak tasarlanmıştır. Haziran 2018-Haziran 2022 tarihleri arasında kulak-burun-boğaz kliniğine başvuran hastaların kayıtları incelenmiştir. SSCD sendromu tanısı alan hastaların verilerine ulaşılmıştır. Bu hastaların odyovestibüler semptomları, klinik bulguları ve yüksek çözünürlüklü bilgisayarlı tomografi [high-resolution computed tomography (HRCT)] taramaları sunulmuştur. **Bulgular:** Retrospektif değerlendirme sonucunda, SSCD sendromu tanısı alan 5 kulağa ulaşılmıştır. Bu 5 kulağın, odyovestibüler semptomları ve saf ses odyometri, timpanometri, akustik refleks, video head impulse test, videonistagmografi, servikal ve oküler vestibüler uyarılmış miyojenik potansiyeller [vestibular evoked myogenic potentials (VEMP)] test sonuçları gibi klinik bulguları değerlendirilmiştir. Tüm kulakların HRCT taramaları yorumlanmıştır. **Sonuç:** Yüksek şiddetli akustik uyaran (Tullio fenomeni) ve/veya basınçla (Hennebert belirtisi) ortaya çıkan vertigo ve/veya nistagmus, osilopsi, hiperakuzi, otofoni, kulakta dolgunluk hissi, pulsatil tinnitus gibi semptomlar; saf ses odyometri testinde kemik eşiklerinin düşmesi, vestibüler uyarılmış miyojenik potansiyeller testinde dalga amplitüdünün artması, VEMP eşiklerinin düşmesi, video head impulse testte semisirküler kanal kazançlarında azalma gibi bulgular SSCD sendromunu düşündürür.

Anahtar Kelimeler: Superior semisirküler kanal dehissansı sendromu; vestibüler uyarılmış miyojenik potansiyeller

Superior semicircular canal dehiscence (SSCD) syndrome is a bone defect in the superior semicircular canal (SCC) that leads to auditory and vestibular symptoms and was described by Minor et al.[1] The dehiscence in the otic capsule alters the biomechanics of the inner ear. Symptoms such as sound-induced vertigo, pressure-induced vertigo, hyperacusis, autophony, oscillopsia, aural fullness, pulsatile tinnitus,

Correspondence: Anı PARABAKAN POLAT
Department of Audiology, University of Health Sciences Gülhane Faculty of Health Sciences, Ankara, Türkiye
E-mail: aniparabakanbaskent@gmail.com

Peer review under responsibility of Journal of Ear Nose Throat and Head Neck Surgery.

Received: 22 Aug 2022 ***Received in revised form:*** 26 Sep 2022 ***Accepted:*** 26 Sep 2022 ***Available online:*** 05 Oct 2022

elevated bone-conduction thresholds may occur. SSCD is diagnosed with auditory and vestibular symptoms, pure tone audiometry, vestibular evoked myogenic potentials (VEMP) findings, high-resolution computed tomography (HRCT) scans, and magnetic resonance imaging (MRI).

The current study aimed to assess the audiovestibular clinical findings in patients diagnosed with SSCD syndrome.

MATERIAL AND METHODS

The present study was designed as a clinical retrospective research. The records of patients admitted to Başkent University Department of Otorhinolaryngology between June 2018 and June 2022 were examined. The data of patients diagnosed with SSCD syndrome were obtained. The audiological and vestibular symptoms, clinical findings such as pure tone audiometry, tympanometry, acoustic reflex, video head impulse test (vHIT), videonystagmography (VNG), cervical and ocular VEMP (cVEMP/oVEMP) test results were analyzed. HRCT scans of all ears were interpreted. This study was approved by Başkent University Medical and Health Sciences Research Council (Project no: KA22/338) and Non-interventional Clinical Research Ethics Committee (date: August 2, 2022, decision no: E-94603339-604.01.02-147771). Both verbal and written informed consents were obtained from all participants. This study was conducted in accordance with the Declaration of Helsinki principles.

RESULTS

As a result of the retrospective review, 5 ears, 4 cases diagnosed with SSCD syndrome were obtained. The mean age of four female patients was 35.35 years (range of 25-45 years).

Patients were admitted to the otolaryngology clinic with various complaints. Complaints of Case 1 were pulsatile tinnitus, hyperacusis and vertigo. Complaints of Case 2 were vertigo and hyperacusis. Complaints of Case 3 were left ear pain, hyperacusis and autophony. Complaints of Case 4 were vertigo, dizziness, aural fullness and hyperacusis. The otolaryngological examination was normal in all cases.

PURE TONE AUDIOMETRY AND SPEECH TESTS

Pure tone audiometry and speech tests were performed with the Interacoustics AC40 (Interacoustics, Denmark) clinical audiometer device in all patients. In Case 1, the average of air-conduction pure tone audiometry threshold was 1 dB bilaterally, the average of pure tone audiometry threshold of bone-conduction was 0 dB bilaterally. The speech reception threshold was 0 dB, the speech discrimination percentage was 100% bilaterally (Figure 1).

In Case 2, the average of air-conduction pure tone audiometry threshold was 5 dB in the right ear, 4 dB in the left ear. The bone-conduction threshold average was 1 dB in the right ear and 4 dB in the left ear (Figure 2).

In Case 3, the average of air-conduction pure tone audiometry threshold was 8 dB in the right ear, 10 dB in the left ear. The bone-conduction audiometry threshold average was 4 dB in the right ear, 0 dB in the left ear. Conductive hearing loss was observed in the left ear. The speech reception threshold was 5 dB in the right ear, 10 dB in the left ear. The speech discrimination percentage was 100% bilaterally. The uncomfortable loudness level was 105 dB in the right ear, 100 dB in the left ear (Figure 3).

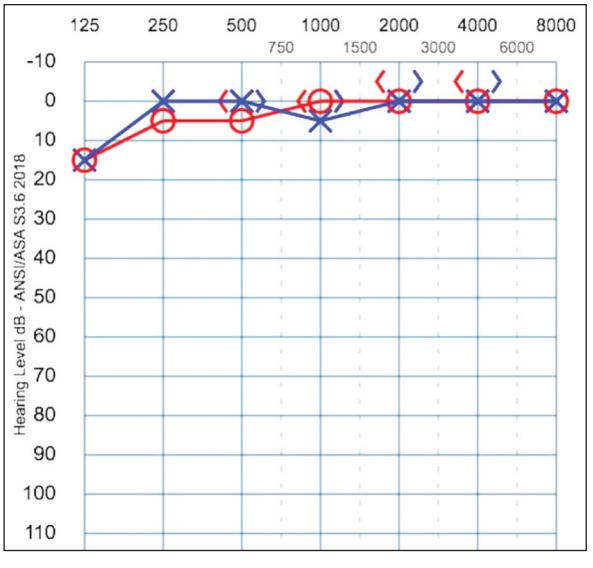

FIGURE 1: Pure tone audiometry thresholds of Case 1.
ANSI: American National Standards Institute; ASA: American Standards Association.

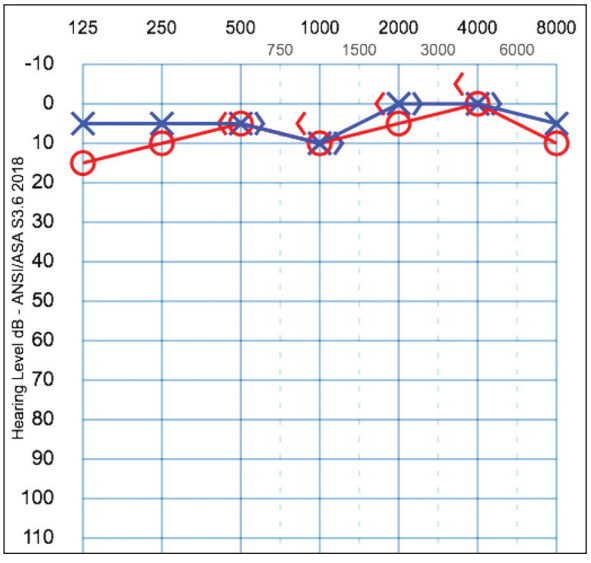

FIGURE 2: Pure tone audiometry thresholds of Case 2.
ANSI: American National Standards Institute; ASA: American Standards Association.

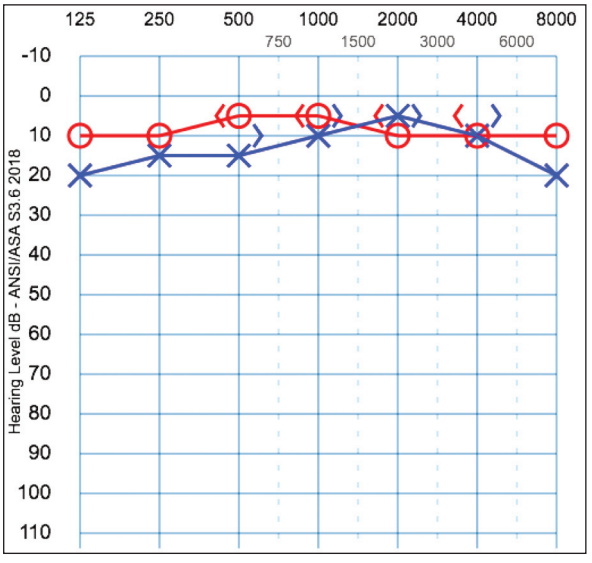

FIGURE 3: Pure tone audiometry thresholds of Case 3.
ANSI: American National Standards Institute; ASA: American Standards Association

In Case 4, the average air-conduction threshold was 21 dB in the right ear and 23 dB in the left ear. The bone-conduction threshold average was 18 dB bilaterally. The speech reception threshold was 25 dB in the right ear and 20 dB in the left ear. The speech discrimination percentage was 96% in the right ear and 84% in the left ear. The uncomfortable loudness level was 105 dB in the right ear and 110 dB in the left ear (Figure 4).

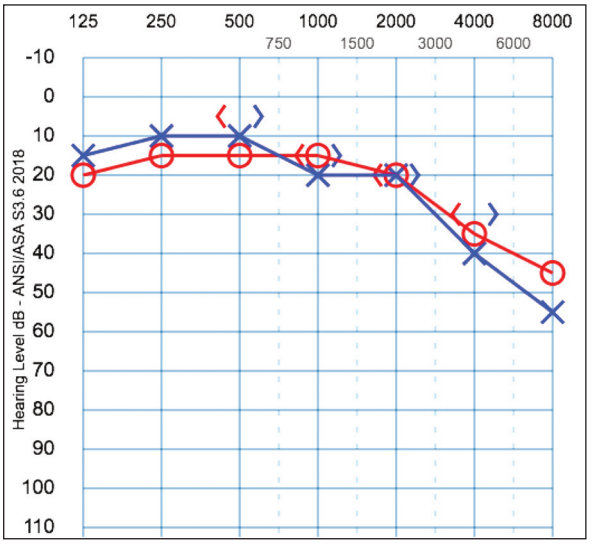

FIGURE 4: Pure tone audiometry thresholds of Case 4.
ANSI: American National Standards Institute; ASA: American Standards Association.

TYMPANOMETRY/ACOUSTIC REFLEX TESTS

Tympanometry/acoustic reflex tests were performed with GSI Tympstar Version 2 (Grason-Stadler, Inc., USA). In Case 1, A Type A_d tympanogram was elicited in the right ear, a Type A tympanogram was elicited in the left ear. Reflex response thresholds were 85 dB at 500 Hz, 85 dB at 1 kHz, 80 dB at 2 kHz in the right ear; 80 dB at 500 Hz, 90 dB at 1 kHz, 90 dB at 2 kHz in the left ear.

VEMP

cVEMP, oVEMP were performed with the Interacoustics Interacoustics Eclipse EP25 (Interacoustics, Denmark) device in all cases. In Case 1, in the cVEMP test with 500 Hz tone burst stimulus, P1 latency value was 14.00 ms, N1 latency value was 24.67 ms, P1-N1 amplitude value was 180.8 µV and the threshold was 70 dB in the right ear. P1 latency value was 16.33 ms, N1 latency value was 27.33 ms, P1-N1 amplitude value was 62.87 µV and the threshold was 80 dB in the left ear. The asymmetry ratio was 0.48. In the oVEMP test with 500 Hz tone burst stimulus, N1 latency value was 10.33 ms, P1 latency value was 16.67 ms, N1-P1 amplitude value was 7.028 µV and the threshold was 70 dB in the right ear. N1 latency value was 10.33 ms, P1 latency value was 14.67 ms,

N1-P1 amplitude value was 12.19 µV, the threshold was 90 dB in the left ear. The asymmetry ratio was 0.27. In the cVEMP test with 100 dB 500 Hz CHIRP stimulus, P1 latency value was 10.67 ms, N1 latency value was 19.67 ms, P1-N1 amplitude value was 158.2 µV and the threshold was 70 dB in the right ear. P1 latency value was 11.67 ms, N1 latency value was 20.67 ms, P1-N1 amplitude value was 58.34 µV and the threshold was 80 dB in the left ear. The asymmetry ratio was 0.46. In the oVEMP test with 100 dB 500 Hz CHIRP stimulus, P1 latency value was 12.33 ms, N1 latency value was 7.33 ms, N1-P1 amplitude value was 12.33 µV and the threshold was 90 dB in the right ear. P1 latency value was 13.00 ms, N1 latency value was 7.33 ms, N1-P1 amplitude value was 18.86 µV and the threshold was 80 dB in the left ear. The asymmetry ratio was 0.21. During the VEMP test, vertigo and nystagmus were observed with 100 dB acoustic stimulus (Figure 5, Figure 6).

In Case 2, in the cVEMP test with 500 Hz tone burst stimulus, P1 latency value was 18.33 ms, N1 latency value was 27.67 ms, P1-N1 amplitude value was 65.00 µV and the threshold was 85 dB in the right ear. P1 latency value was 22.67 ms, N1 latency value was 32.67 ms, P1-N1 amplitude value was 44.12 µV and the threshold was 95dB in the left ear. The asymmetry ratio was 0.19. In the oVEMP test with 500 Hz tone burst stimulus, N1 latency value was 12.00 ms, P1 latency value was 16.67 ms, N1-P1 amplitude value was 2.637 µV and the threshold was 90 dB in the right ear. N1 latency value was 12.00 ms, P1 latency value was 17.33 ms, N1-P1 amplitude value was 1.766 µV and the threshold was 95 dB in the left ear. The asymmetry ratio was 0.20 (Figure 7, Figure 8).

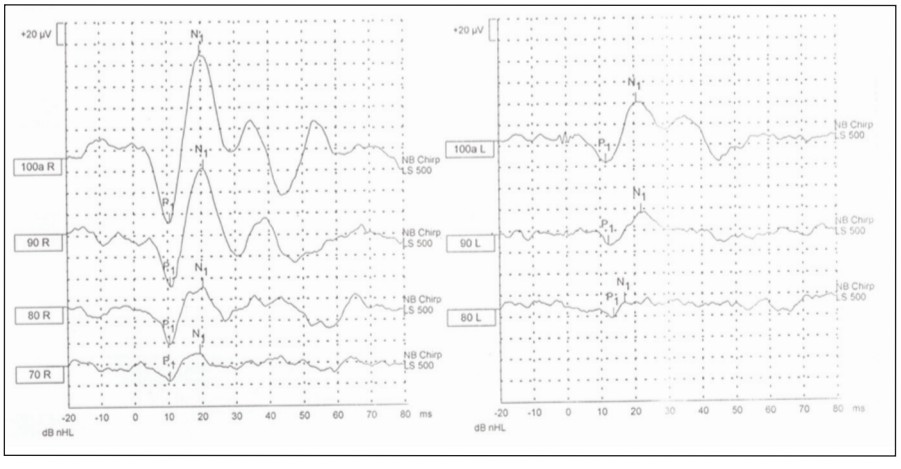

FIGURE 5: Cervical vestibular evoked myogenic potentials test result of Case 1.

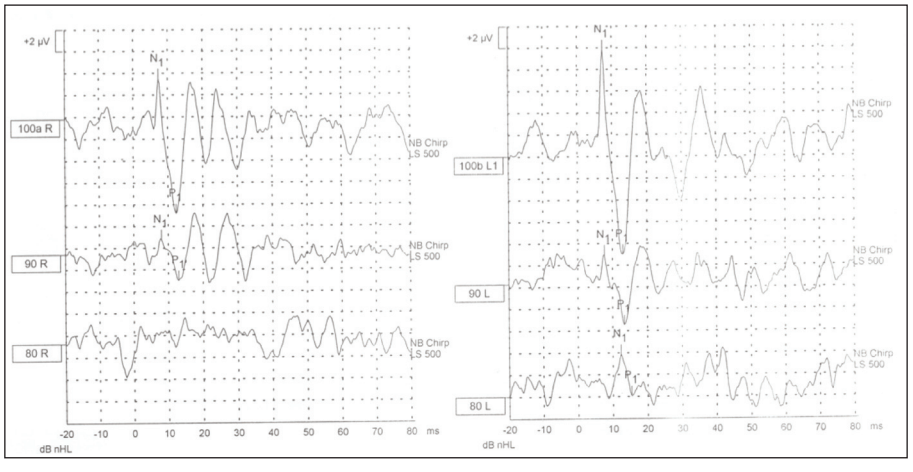

FIGURE 6: Ocular vestibular evoked myogenic potentials test result of Case 1.

In Case 3, in the cVEMP test with 500 Hz tone burst stimulus, P1 latency value was 14 ms, N1 latency value was 24.67 ms and P1-N1 amplitude value was 266.1 µV in the right ear. P1 latency value was 13.33 ms, N1 latency value was 28.33 ms and P1-N1 amplitude value was 198.5 µV in the left ear. The asymmetry ratio 0.15. In the oVEMP test with 500 Hz tone burst stimulus, N1 latency value was 9.33 ms, P1 latency value was 15.33 ms and N1-P1 amplitude value was 17.92 µV in the right ear. N1 latency value was 9.00 ms, P1 latency value was 16.00 ms and N1-P1 amplitude value was 52.07 µV in the left ear. The asymmetry ratio was 0.49 (Figure 9, Figure 10).

In Case 4, in the cVEMP test with 500 Hz tone burst stimulus, P1 latency value was 14.00 ms N1 latency value was 23.33 ms, P1-N1 amplitude value was 263.8 µV and the threshold was 75 dB in the right ear. P1 latency value was 14.67 ms, N1 latency value was 25.67 ms, P1-N1 amplitude value was 138.1 µV and the threshold value was 80 dB in the left ear. In the oVEMP test with 500 Hz tone burst stimulus, N1 latency value was 10.33 ms, P1 latency value was 15.67 ms and N1-P1 amplitude value was 10.02 µV in the right ear. N1 latency value was 10.33 ms, P1 latency value was 14.67 ms and N1-P1 amplitude value was 12.60 µV in the left ear (Figure 11, Figure 12).

vHIT

vHIT was performed with the Interacoustics Eye-SeeCam vHIT (Interacoustics, Denmark) device. In Case 1, The mean gain values were 0.95 in the right lateral SCC, 1.04 in the left lateral SCC, 0.78 in the right anterior SCC, 1.05 in the left anterior SCC, 0.94 in the

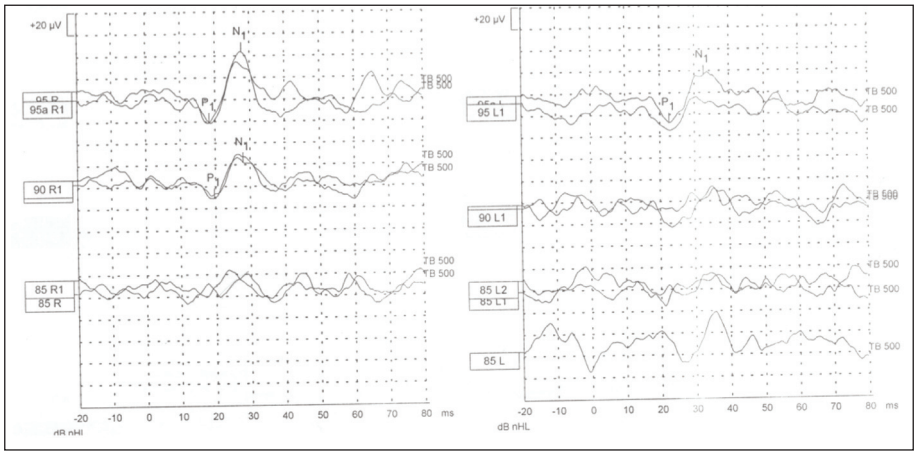

FIGURE 7: Cervical vestibular evoked myogenic potentials test result of Case 2.

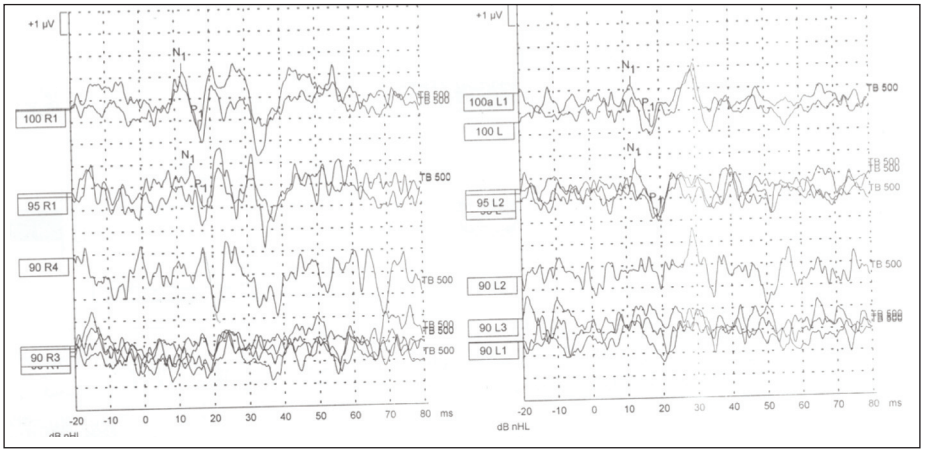

FIGURE 8: Ocular vestibular evoked myogenic potentials test result of Case 2.

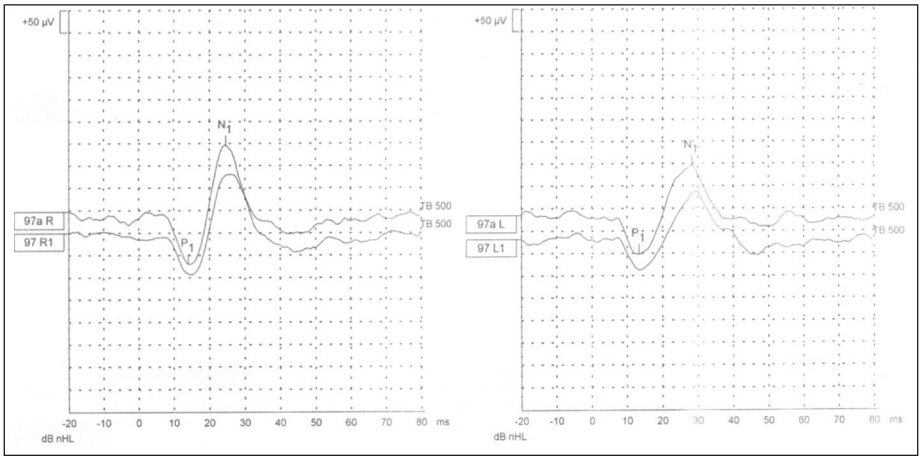

FIGURE 9: Cervical vestibular evoked myogenic potentials test result of Case 3.

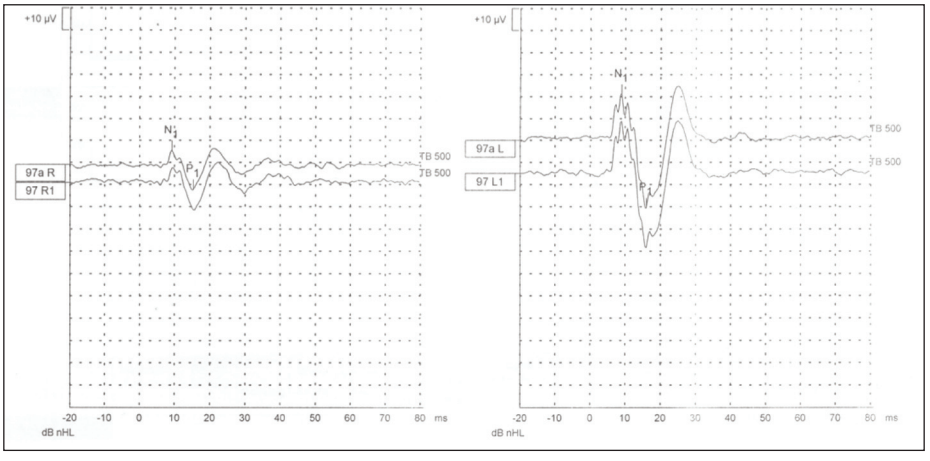

FIGURE 10: Ocular vestibular evoked myogenic potentials test result of Case 3

right posterior SCC and 0.87 in the left posterior SCC.

In Case 2, the mean gain values were 1.18 in the right lateral SCC, 1.04 in the left lateral SCC, 0.8 right posterior SCC, 0.8 in the left posterior SCC, 0.96 in the right anterior SCC and 0.97 in the left posterior SCC.

In Case 4, the mean gain values were 1.01 in the right lateral SCC, 0.78 in the left lateral SCC, 0.7 in the right anterior SCC, 0.86 in the left anterior SCC, 0.87 in the right posterior SCC and 0.87 in the left posterior SCC.

VNG

Oculomotor tests (gaze, saccade, smooth pursuit, optokinetic), spontaneous nystagmus, post-head-shaking nystagmus, and positional tests (Dix-Hallpike, Roll) were assessed with the Micromedical Spectrum VNG (Interacoustics, USA) device. In Case 1, oculomotor test results were normal. Left beating horizontal nystagmus was observed in the left Dix-Hallpike, right Dix-Hallpike and, Roll tests. In Case 2, gaze test results were normal. Left beating horizontal nystagmus was observed in post-head-shaking test. In Case 4, right beating nystagmus was observed during post head shaking test, and head roll test.

HRCT

A HRCT scan of temporal bone revealed bilateral SSCD in Case 1, right SSCD in Case 2, left SSCD in Case 3 and left SSCD in Case 4 (Figure 13, Figure 14, Figure 15, Figure 16 and Figure 17).

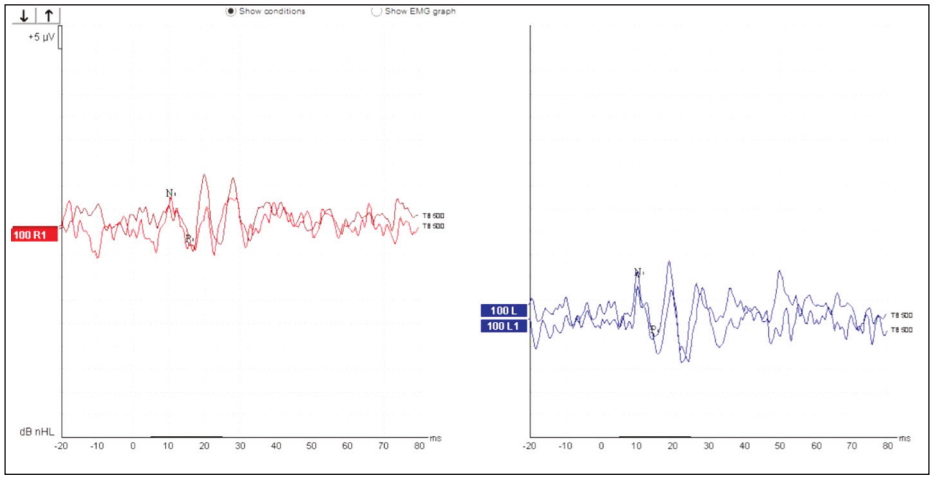

FIGURE 11: Ocular vestibular evoked myogenic potentials test result of Case 4.

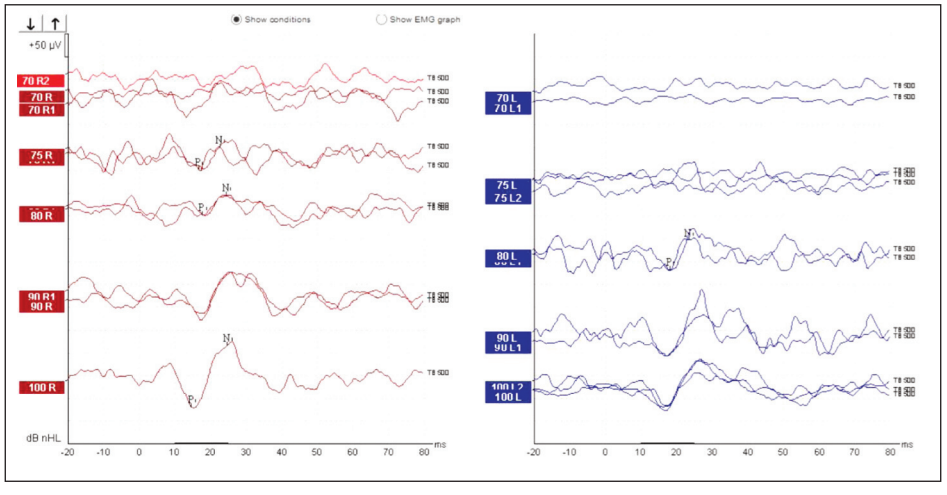

FIGURE 12: Cervical vestibular evoked myogenic potentials test result of Case 4.

DISCUSSION

Minor et al. published SSCD case series consisting of 8 patients with symptoms of vertigo and oscillopsia induced by sound and pressure.[1] The dehiscence in the otic capsule alters the biomechanics of the inner ear. SSCD creates a third window effect which creates a low-impedance pathway that ensures the transmission of pressure and acoustic energy to the vestibule. Due to this, the hyperactivity of the vestibular end organs to acoustic stimuli and/or pressure changes increases. Therefore, symptoms such as sound-induced vertigo, pressure-induced vertigo, hyperacusis, autophony, pulsatile tinnitus, oscillopsia, aural fullness, elevated bone-conduction thresholds arises. Sound-induced vertigo (Tullio phenomenon) and pressure-induced nystagmus (Hennebert sign) are highly suggestive of SSCD. In the current study, vertigo and nystagmus were observed with 100 dB acoustic stimulus in Case 1.

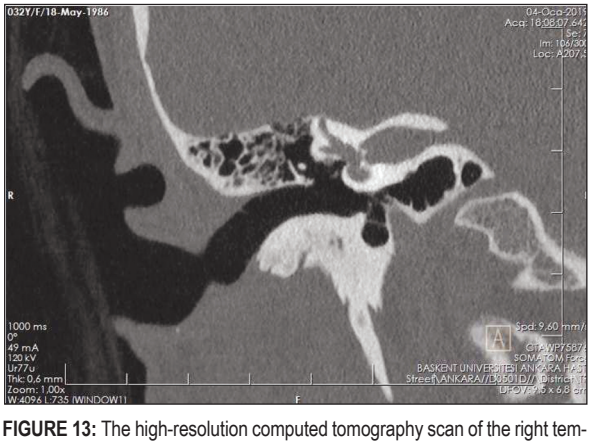

FIGURE 13: The high-resolution computed tomography scan of the right temporal bone of Case 1.

FIGURE 14: The high-resolution computed tomography scan of the left temporal bone of Case 1.

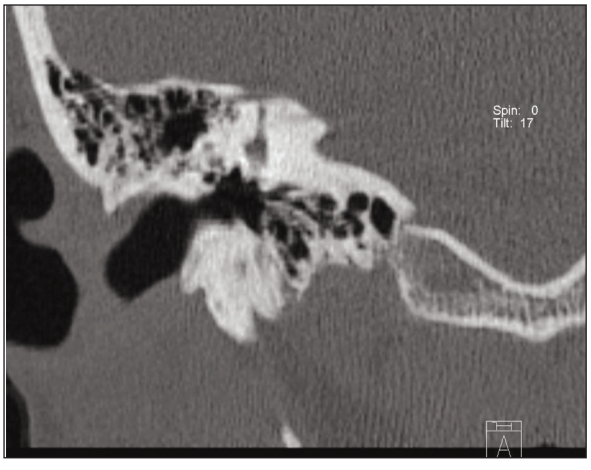

FIGURE 15: The high-resolution computed tomography scan of the temporal bone of Case 2.

Pulsatile tinnitus may occur as a result of pressure changes due to intracranial pulsation being transmitted to the perilymph and cochlea through the moving window created by dehiscence.[2] In the current study, Case 1 has been complaining of pulsatile tinnitus for nine months.

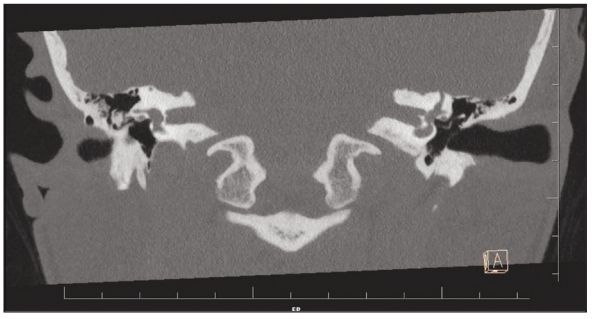

FIGURE 16: The high-resolution computed tomography scan of the temporal bone of Case 3.

The third window may increase the pressure difference between oval and round windows, which causes bony hyperconductivity, resulting in autophony or hyperacusis.[3] Minor et al. reported hyperacusia in 11 of 28 cases.[4] In another publication, Minor reported 36 of the 60 cases had autophony and 31 of the 60 cases had hyperacusis.[5] In this study, hyperacusis and/or autophony complaints are present in all cases. However, no clinical findings were observed.

Cases with symptoms suggestive of SSCD are evaluated by HRCT and MRI imaging. MRI excludes vestibular schwannoma and other tumors of brainstem, posterior fossa, and temporal bone; assess arterial dissection, brainstem infarct, and demyelinating disease. MRI FIESTA scans have recently been used to image SSCD. HRCT assess the middle ear for diseases, rules out otosclerosis, evaluate the integrity of

FIGURE 17: The high-resolution computed tomography scan of the temporal bone of Case 4.

the SCCs.[6] Temporal bone HRCT scans are useful to confirm the diagnosis of SSCD due to its high sensitivity. Evaluation of standard planes (axial, coronal, sagittal) in routine temporal HRCT scans may not distinguish thin bone overlying superior SCC from dehiscence in some cases. HRCT scans reformatted in the planes of Stenver and Poschl may differentiate thin and dehiscent superior SCC in these cases.[7]

In addition to radiological evaluation, VEMP findings are beneficial in the diagnosis of SSCD.[8] VEMPs are short latency muscle reflex responses triggered by stimulation of peripheral otolith organs by sound, vibration, or electrical stimulation. In healthy individuals, the normal wave amplitude values are 50-160 µV for cVEMP, 5-12 µV for oVEMP. In the SSCD cases, the hyperactivity of the vestibular end organs to acoustic stimulus increases VEMP wave amplitudes and decreases VEMP response thresholds.

Zuniga et al. reported that the oVEMP test is an excellent screening test without the risk of radiation exposure, as in HRCT.[9] The VEMP test is important in determining whether the dehiscence displayed on HRCT causes a pathological pressure transition between the otic capsule and the intracranial spaces.[10] VEMP test reliably distinguishes SCC dehiscence from patients with thin or normal bone covering the SCC.[11] A limited number of studies reporting VEMP response thresholds of SSCD cases are available in the literature.

The average cVEMP response threshold was 81 dB in 51 cases, 72 dB in 8 cases, 96 dB sound pressure level (SPL) in 21 cases, 65 dB hearing level (HL) in 37 cases and 94 dB SPL in 13 cases.[5,8,12-14] The average oVEMP threshold was 96 dB SPL in 13 cases and 82.5 dB nHL in 9 cases.[11,14] In the current study, the average cVEMP threshold was 78.8 dB, and the average oVEMP threshold was 83.3 dB HL in 5 ears.

A limited number of studies reporting VEMP wave amplitude are available in the literature. Pereira et al. reported that the cVEMP amplitude with 100 dB stimulus was 115.8 µV in the right ear and 213.2 µV in the left ear of a patient with bilateral SSCD.[15] Govender et al. reported that the average oVEMP amplitude was 35.6 µV in 13 cases.[14] They did not find a significant difference in amplitude between the SSCD cases and the control group. In the present study, the average cVEMP amplitude was 143.1 µV, and the average oVEMP amplitude was 19.70 µV in 5 ears.

Zuniga et al. stated that oVEMP amplitude values are superior to cVEMP thresholds in diagnosing SSCD.[9] In this study, the oVEMP response amplitude of the right ear was higher than the oVEMP response amplitude of the contralateral ear in Case 2. The oVEMP response amplitude of the left ear was higher than the oVEMP response amplitude of the contralateral ear in Case 3.

Beside VEMP findings, the vHIT gains of SCCs can also be affected by SSCD. In the vHIT test, a decrease in dehiscence canal gain can be observed as a result of compression of the membranous canal by the temporal lobe and dura.[5] Mukherjee et al. reported that superior SCC hypofunction was observed in the vHIT test in 9 of 11 ears.[16] In the current study, gains of right superior SSCs which are dehiscent in Case 1 and Case 2 were lower than the gains of the other canals.

CONCLUSION

Symptoms such as sound- and/or pressure-induced vertigo, autophony, hyperacusis, oscillopsia, aural fullness, pulsatile tinnitus; findings such as elevated bone-conduction thresholds, increasing of VEMP wave amplitude, decreasing of VEMP response threshold, decreasing of SCC gain suggestive of SSCD.

Source of Finance

During this study, no financial or spiritual support was received neither from any pharmaceutical company that has a direct connection with the research subject, nor from a company that provides or produces medical instruments and materials which may negatively affect the evaluation process of this study.

Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

Idea/Concept: *Berna Deniz Aydın, Hatice Seyra Erbek;* ***Design:*** *Berna Deniz Aydın, Anı Parabakan Polat;* ***Control/Supervision:*** *Hatice Seyra Erbek, Evren Hızal;* ***Data Collection and/or Processing:*** *Berna Deniz Aydın, Evren Hızal;* ***Analysis and/or Interpretation:*** *Berna Deniz Aydın, Anı Parabakan Polat;* ***Literature Review:*** *Berna Deniz Aydın, Anı Parabakan Polat;* ***Writing the Article:*** *Berna Deniz Aydın;* ***Critical Review:*** *Hatice Seyra Erbek, Evren Hızal, Berna Deniz Aydın, Anı Parabakan Polat;* ***References and Fundings:*** *Berna Deniz Aydın, Hatice Seyra Erbek;* ***Materials:*** *Berna Deniz Aydın, Evren Hızal.*

REFERENCES

1. Minor LB, Solomon D, Zinreich JS, Zee DS. Sound- and/or pressure-induced vertigo due to bone dehiscence of the superior semicircular canal. Arch Otolaryngol Head Neck Surg. 1998;124(3):249-58. [Crossref] [PubMed]
2. Bi WL, Brewster R, Poe D, Vernick D, Lee DJ, Eduardo Corrales C, et al. Superior semicircular canal dehiscence syndrome. J Neurosurg. 2017;127(6):1268-76. [Crossref] [PubMed]
3. Nikkar-Esfahani A, Whelan D, Banerjee A. Occlusion of the round window: a novel way to treat hyperacusis symptoms in superior semicircular canal dehiscence syndrome. J Laryngol Otol. 2013;127(7):705-7. [Crossref] [PubMed]
4. Minor LB, Cremer PD, Carey JP, Della Santina CC, Streubel SO, Weg N. Symptoms and signs in superior canal dehiscence syndrome. Ann N Y Acad Sci. 2001;942:259-73. [Crossref] [PubMed]
5. Minor LB. Clinical manifestations of superior semicircular canal dehiscence. Laryngoscope. 2005;115(10):1717-27. [Crossref] [PubMed]
6. Browaeys P, Larson TL, Wong ML, Patel U. Can MRI replace CT in evaluating semicircular canal dehiscence? AJNR Am J Neuroradiol. 2013;34(7):1421-7. [Crossref] [PubMed] [PMC]
7. Mau C, Kamal N, Badeti S, Reddy R, Ying YM, Jyung RW, et al. Superior semicircular canal dehiscence: Diagnosis and management. J Clin Neurosci. 2018;48:58-65. [Crossref] [PubMed]
8. Streubel SO, Cremer PD, Carey JP, Weg N, Minor LB. Vestibular-evoked myogenic potentials in the diagnosis of superior canal dehiscence syndrome. Acta Otolaryngol Suppl. 2001;545:41-9. [Crossref] [PubMed]
9. Zuniga MG, Janky KL, Nguyen KD, Welgampola MS, Carey JP. Ocular versus cervical VEMPs in the diagnosis of superior semicircular canal dehiscence syndrome. Otol Neurotol. 2013;34(1):121-6. [Crossref] [PubMed] [PMC]
10. Janky KL, Nguyen KD, Welgampola M, Zuniga MG, Carey JP. Air-conducted oVEMPs provide the best separation between intact and superior canal dehiscent labyrinths. Otol Neurotol. 2013;34(1):127-34. [Crossref] [PubMed] [PMC]
11. Thabet EM, Abdelkhalek A, Zaghloul H. Superior semicircular canal dehiscence syndrome as assessed by oVEMP and temporal bone computed tomography imaging. Eur Arch Otorhinolaryngol. 2012;269(5):1545-9. [Crossref] [PubMed]
12. Niesten ME, McKenna MJ, Herrmann BS, Grolman W, Lee DJ. Utility of cVEMPs in bilateral superior canal dehiscence syndrome. Laryngoscope. 2013;123(1):226-32. [Crossref] [PubMed]
13. Hunter JB, Patel NS, O'Connell BP, Carlson ML, Shepard NT, McCaslin DL, et al. Cervical and ocular VEMP testing in diagnosing superior semicircular canal dehiscence. Otolaryngol Head Neck Surg. 2017;156(5):917-23. [Crossref] [PubMed]
14. Govender S, Fernando T, Dennis DL, Welgampola MS, Colebatch JG. Properties of 500Hz air- and bone-conducted vestibular evoked myogenic potentials (VEMPs) in superior canal dehiscence. Clin Neurophysiol. 2016;127(6):2522-31. [Crossref] [PubMed]
15. Pereira D, Leonardo A, Duarte D, Oliveira N. Bilateral superior semicircular canal dehiscence: bilateral conductive hearing loss with subtle vestibular symptoms. BMJ Case Rep. 2020;13(3):e233042. [Crossref] [PubMed] [PMC]
16. Mukherjee P, Chiarovano E, Cheng K, Manzari L, McGarvie LA, MacDougall HG. Video-head impulse test in superior canal dehiscence. Acta Otolaryngol. 2021;141(5):471-5. [Crossref] [PubMed]