



Can Cochlear Nerve Size Assessment With Magnetic Resonance Enhance the Understanding of Idiopathic Sudden Sensorineural Hearing Loss?

Hande Arslan¹, Meltem Özdemir², Rasime Pelin Kavak²,
Kemal Keseroğlu³, Murad Mutlu³, and Mehmet Hakan Korkmaz⁴

¹Department of Otorhinolaryngology, University of Health Sciences, Samsun Training and Research Hospital, Ankara, Türkiye

²Department of Radiology, University of Health Sciences, Dışkapı Yıldırım Beyazıt Training and Research Hospital, Ankara, Türkiye

³Department of Otorhinolaryngology, University of Health Sciences, Dışkapı Yıldırım Beyazıt Training and Research Hospital, Ankara, Türkiye

⁴Department of Otorhinolaryngology, Ankara Yıldırım Beyazıt University, Ankara, Türkiye

Received May 9, 2023

Revised June 13, 2023

Accepted June 19, 2023

Address for correspondence

Hande Arslan, MD

Department of Otorhinolaryngology,

Samsun Training and

Research Hospital, Kışla, Barış Blv.

No: 199, 55090 İlkadim,

Samsun, Türkiye

Tel +90-533-430-95-28

Fax +90-(362)-277-88-65

E-mail handearslan5@yahoo.com

Background and Objectives: Idiopathic sudden sensorineural hearing loss (ISSHL) is a rapid loss of hearing, exceeding 30 dB in at least 3 consecutive frequencies within 3 days, without any identifiable cause despite thorough investigations. Currently, the etiology and pathogenesis of ISSHL have not been fully elucidated. This study aimed to assess the size of the cochlear nerve in patients with ISSHL and explore its relationship with pretreatment audiograms and treatment response. **Subjects and Methods:** A total of 125 patients (59 [47.2%] women; mean age 47.7 ± 13.8 years [minimum–maximum: 21–76]) and 60 healthy participants (27 [45%] women; mean age 45.7 ± 16.8 years [minimum–maximum: 20–76]) as a control group were included in this study. The size of the cochlear nerve was assessed on the affected side, compared to the control group, as well as on the unaffected side. Pretreatment and posttreatment audiological values were also analyzed. **Results:** The cross-sectional area (CSA), vertical diameter (VD), and horizontal diameter (HD) of the CN were found to be smaller on the affected side of ISSHL patients compared to the control group ($p < 0.01$; $p = 0.04$; $p = 0.02$, respectively). In the study group (affected side of ISSHL patients), there were no significant differences in VD, HD, and CSA values between pretreatment audiogram types ($p = 0.23$; $p = 0.53$; $p = 0.39$, respectively), and initial hearing levels ($p = 0.16$; $p = 0.22$; $p = 0.23$, respectively). Furthermore, there were no significant differences in VD, HD, and CSA values between the recovery groups according to Furuhashi criteria ($p = 0.18$; $p = 0.37$; $p = 0.27$, respectively). **Conclusions:** The size of the CN may be a risk factor for ISSHL, but it does not affect the type of audiogram curves and was not prognostic in terms of treatment response. **J Audiol Otol 2024;28(1):29-35**

Keywords: Sudden sensorineural hearing loss; Magnetic resonance imaging; Cochlear nerve; Audiogram curves; Treatment response.

Introduction

Sudden sensorineural hearing loss (SSHL) is a loss of hearing more than 30 dB in at least 3 consecutive frequencies within 3 days. SSHL for which no cause has been found despite ap-

propriate quests is called idiopathic sudden sensorineural hearing loss (ISSHL). Currently, etiology and pathogenesis of ISSHL have not been fully elucidated. For this purpose, many biochemical, clinical, and radiological studies have been conducted [1]. However, it is not yet understood who is more susceptible to ISSHL, unlike who will have the better prognosis. ISSHL was widely accepted as primarily caused by impaired hair cell function however in several studies [2,3], hair cell injury could not be shown in every ISSHL patient. Spiral gangli-

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<https://creativecommons.org/licenses/by-nc/4.0/>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

on neurons and cochlear nerve (CN) neuron dysfunction significantly contributes to ISSHL as described in the following.

Recent studies have demonstrated the clinical significance of morphometric changes detectable by magnetic resonance imaging (MRI) in the CN. The use of MR in patients recently affected with SSHL enabled the detection of morphometric changes in the CN. In principle, the primary goal of MRI in patients with SSHL is to rule out an acoustic neuroma, a necessity before labeling such a hearing loss (HL) as being ISSHL. Besides, an MRI of the CN has usually been performed in profoundly hearing-impaired individuals before cochlear implantation. For instance, reports were obtained in patients with long-standing HL [4,5]. CN imperfection has been demonstrated in congenital as well as acquired sensorineural hearing loss (SHL) patients [6]. Russo, et al. [5] and Kim, et al. [7] highlighted the change in the morphometric measurements of the CN in people with HL for a long time, in adults as well as children. Moreover, Islamoglu, et al. [8] measured CN size in long-lasting single-side deafness due to ISSHL and found no difference between healthy and diseased ears. However, in none of the studies conducted so far, CN size has not been measured and compared with both the healthy side of ISSHL patients and in healthy individuals. In addition, no comparison was made between the pretreatment audiogram type and treatment responses with the CN size.

The current research aimed to evaluate the size of the CN in patients with ISSHL. To this end, CN size was measured in ISSHL patients and compared with the healthy side and the control group, using three-dimensional constructive interference in a steady-state (3D-CISS) sequence on MRI. In addition, the relationship of CN size with pretreatment audiograms and treatment response was investigated.

Subjects and Methods

The present study was conducted in a tertiary referral center after the approval of the local ethics committee (No: 86/05: 20.04.2020). Written informed consent was taken from research participants. This research which involves human participants complies with the 1964 Helsinki declaration and its later amendments.

Patient selection

Patients who were diagnosed with ISSHL in 2015–2019 were included in this study. Unilateral 30 dBHL that developed in at least 3 consecutive frequencies in the last 3 days was considered SSHL. Records of the pretreatment and post-treatment audiograms, otologic examination, and contrast-enhanced MR images at the time of the first admission were

noted. Patients with bilateral SSHL, vestibular schwannoma, a history of acoustic trauma, otologic surgery, Meniere's disease, migraine, chronic otitis media, usage of ototoxic drugs, malignancy, and stroke were not included in the research. The control group was composed of age and sex-matched patients who went through temporal MRI studies during the same time frame. Facial palsy, and Meniere's disease were the exclusion criteria for the control group.

All participants involved in the study were assessed for vertical diameter (VD), horizontal diameter (HD), and cross-sectional area (CSA) of the CN, as demonstrated by the 3D-CISS sequence on MRI. Measurements were evaluated on both sides of all participants. Also, demographics, ear side with HL, concomitant audiovestibular symptoms such as vertigo and tinnitus, and comorbidities kind of diabetes mellitus and hypertension were analyzed. In the study group, 26 (21%) patients had tinnitus and the other 16 (13%) patients had vertigo with HL as initially. Only 10 (8%) patients had diabetes mellitus, and the other 9 (7%) patients had hypertension in the study group. However, these few numbers of cases were not sufficient for statistical evaluation.

MRI

The imaging examinations were performed on a 1.5-T unit MR scanner (Magnetom Aera, Siemens, Erlangen, Germany), with a 20-channel head coil within 48 hours of admission of patients with ISSHL. Morphometric analyses of the CNs were performed on the 3D-CISS images (repetition time/echo time: 1,200/271, field of view: 170 mm, flip angle: 150°, voxel size=0.3×0.3×0.3 mm).

Two national board-certified radiologists (M.Ö. and R.P.K.), who were blind to the group of participants evaluated the images separately and the average of these values was used in the analysis. Measurements were performed twice by Reader 1 with a 1-month interval to assess the intraobserver variability. To examine interobserver variability, measurements performed by Reader 2 were compared with the first set obtained by Reader 1. Measurements were then averaged for both readers. They performed the measurements on the picture archiving and communication system (ExtremePacs, Ankara, Türkiye). Initially, to make the measurements, the CN was identified on the axial image shown in Fig. 1. After that, a parasagittal oblique image of the internal acoustic canal (IAC) was formed by using the multiplanar reformation tools. The location of the CN closest to the fundus of the IAC where the CN could separately be identified, was fixed and measurements of VD and HD of the CN were performed, shown in Figs. 2 and 3. After obtaining the values of VD and HD, CSA of the nerve was calculated by using $CSA=\pi(VD/2)(HD/2)$ formula.

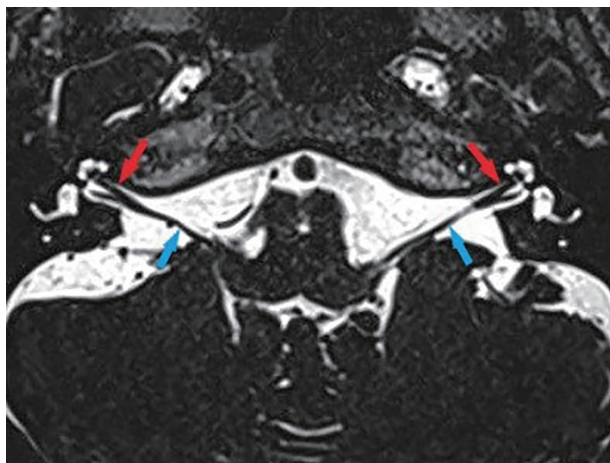


Fig. 1. Axial three-dimensional constructive interference in steady-state (3D-CISS) image of the ears of a 48-year-old man at the level of the internal acoustic canals. The courses of both vestibulocochlear nerves in the cerebellopontine angle cisterns (red arrows) and internal acoustic canals (blue arrows) are seen.

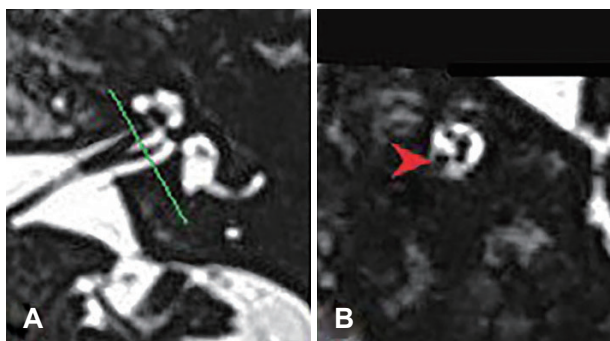


Fig. 2. Three-dimensional constructive interference in steady-state (3D-CISS) images of the left ear of the same patient as in Fig. 1. A: Axial section at the level of the internal acoustic canal. The green line which is drawn perpendicular to the course of the internal acoustic canal represents the plane in which the parasagittal oblique image is created. B: Parasagittal oblique section created with the use of multiplanar reformation tools. The arrowhead shows the cochlear nerve.

Audiological examination

After middle ear pathologies were excluded by otologic examination and tympanometry test (AZ 26 Clinical Audiometer; Interacoustics, Assens, Denmark), pure tone audiometry test between 250 Hz and 8,000 Hz frequencies was performed in a soundproof cabin (AC 33 Clinical Audiometer, Interacoustics). Pure tone average (PTA) was calculated by arithmetic mean for frequencies of 250–8,000 Hz. The speech discrimination scores (SDS) were detected at an easily detectable hearing level by 50 selected monosyllabic words, and calculated by the percentage of words correctly identified. Pretreatment and posttreatment hearing levels were analyzed.

Upsloping (raising), downsloping (falling), U-shaped, and flat curves, and profound HL (hearing threshold shift higher than 90 dB in all frequencies) categories were used to classify

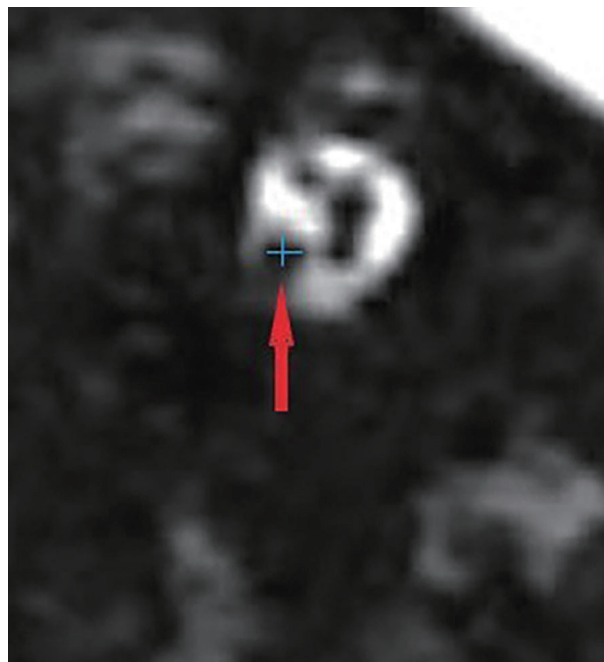


Fig. 3. Parasagittal oblique section through the point nearest to the fundus of the internal acoustic canal where the cochlear nerve (arrow) can be individually identified. The horizontal and vertical diameter measurements of the nerve are shown as blue lines.

the pretreatment audiograms.

The groups were also classified according to pretreatment PTA as follows: very severe (profound) (>90 dB), severe (71–90 dB), moderate to severe (56–70 dB), moderate (41–55 dB), mild (26–40 dB), and very mild (16–25 dB) HL.

Posttreatment recovery of PTA results was analyzed according to Furuhashi criteria (complete recovery: all 5 frequencies of final audiogram are 20 dB or less or improvement to the same degree of hearing as in the unaffected ear; marked improvement: PTA improvement >30 dB; slight improvement: 10 dB < PTA improvement < 30 dB; no change: PTA improvement < 10 dB) at the 6th month of treatment [9].

Treatment strategy

Methylprednisolone at a dose of 1 mg/kg/d was started orally and continued for about 10 days by reducing 16 mg every 3 days. If the patient was without complete recovery at the end of initial treatment, intratympanic steroid treatment (5 doses of 2 mg intratympanic dexamethasone once every 2 days) and hyperbaric oxygen therapy (2,5-atmosphere of 120 minutes for 20 consecutive days) were given respectively as salvage treatment.

Statistical analysis

IBM SPSS Statistics for Windows, Version 21.0 (IBM Corp., Armonk, NY, USA) was used for performing analysis of out-

puts. The Kolmogorov-Smirnov test was performed to test the normal distribution of data. The Mann-Whitney U test (for two groups) and the Kruskal-Wallis H or one-way analysis of variance tests (more than two groups) were used to compare groups. For categorical variables, chi-square test was performed. Statistical significance was defined as $p < 0.05$.

The intraclass correlation coefficient (ICC) and the lower and upper limits of the 95% confidence interval were determined for intraobserver and interobserver variability and interpreted as follows: 0.0–0.2, poor correlation; 0.21–0.4, fair correlation; 0.41–0.6, moderate correlation; 0.61–0.8, good correlation; 0.81–1, almost perfect correlation.

Results

Participants

After 29 patients were excluded due to the before-mentioned reasons, 125 patients (59 [47.2%] women; mean age 47.7 ± 13.8 years [minimum–maximum: 21–76]) were included in this study.

The control group consist of 60 healthy participants (27 [45%] women; mean age 45.7 ± 16.8 years [ranging from 20 to 76]). MRI examinations of 120 (right and left sides of 60 participants) sides were evaluated in the control group. There was no significant difference in gender and age between the study and the control group ($p = 0.76$ and $p = 0.77$, respectively).

Audiological test results

Pretreatment audiological test results (PTA and SDS) of control and study groups were shown in Table 1. Left ear was affected in 64 (51.2%) patients and the right ear was affected in 61 (48.8%) in the study group. Pretreatment audiogram types were flat in 36 (28.8%) patients, downsloping in 28 (22.4%) patients, upsloping in 26 (20.8%) patients, U-shaped in 18 (14.4%) patients, and profound in 17 (13.6%) patients. Pretreatment hearing levels were very mild in 8 (6.4%) patients, mild in 31 (24.8%) patients, moderate in 28 (22.4%) patients,

moderate-severe in 29 (23.2%) patients, severe in 15 (12%) patients, and very severe in 14 (11.2%) patients.

Magnetic resonance image analysis results

The intraobserver and interobserver variability were small with the ICCs being 0.968 (0.907–0.989) and 0.959 (0.832–0.990), respectively. Measurements were then averaged for both readers. Magnetic resonance image analysis results in terms of HD, VD, and CSA of the CN were shown in Table 2.

The differences in CSA ($0.10 \pm 0.17 \text{ mm}^2$) and HD ($0.05 \pm 0.15 \text{ mm}$) values between affected and unaffected sides in the study group were higher than the differences (CSA, $0.042 \pm 0.15 \text{ mm}^2$; HD, $0.02 \pm 0.06 \text{ mm}$) between the affected side of the study group and control group ($p = 0.02$ and $p < 0.012$, respectively). Table 3 shows the age- and sex-adjusted general linear model analysis. According to this model, age and sex did not affect CN sizes. The values of VD, HD, and CSA did not differ between pretreatment audiograms types ($p = 0.23$; $p = 0.53$; $p = 0.39$, respectively) and initial hearing levels ($p = 0.16$; $p = 0.22$; $p = 0.23$, respectively).

Table 2. Results of magnetic resonance image analysis of the cochlear nerve of the study group and both sides of the control group

	Control group (n=120)	Study group		p-value
		Unaffected side (n=125)	Affected side (n=125)	
HD (mm)	1 (0.7–1.5)	1 (0.5–1.2)	0.9 (0.4–1.2)	0.47* 0.02† 0.07‡
VD (mm)	1.2 (0.9–1.7)	1.2 (0.7–1.6)	1.2 (0.5–1.6)	0.76* 0.04† 0.18‡
CSA (mm ²)	1 (0.3–1.8)	0.9 (0.3–1.5)	0.8 (0.2–1.4)	0.51* <0.01† 0.02‡

Data are presented as median (min–max). *control group vs. unaffected side of the study group; †control group vs. affected side of the study group; ‡unaffected side of the study group vs. affected side of the study group. HD, horizontal diameter; VD, vertical diameter; CSA, cross-sectional area

Table 1. PTA and SDSs of the control and the study groups

	Control group (n=60)	Study group		p-value
		Unaffected side (n=125)	Affected side (n=125)	
PTA (dB)	20.8 ± 6.9	20.7 ± 6.7	57.6 ± 27.3	0.94* <0.01†
SDS (%)	96.6 ± 3.8	96.8 ± 3.6	62.3 ± 35.9	0.93* <0.01†

Data are presented as mean \pm standard deviation. *control group vs. unaffected side of the study group; †control group vs. affected side of the study group. PTA, pure tone average; SDS, speech discrimination score

Table 3. The age- and sex-adjusted comparison of cochlear nerve measurements

	General linear model		
	F	Partial eta squared	p-value
CSA (mm ²)	1.799	0.010	0.17
HD (mm)	0.942	0.005	0.39
VD (mm)	0.829	0.005	0.44

HD, horizontal diameter; VD, vertical diameter; CSA, cross-sectional area

Table 4. Treatment responses according to Furuhashi criteria in different audiogram curves

	Downsloping (n=28)	Flat (n=36)	Upsloping (n=26)	U shaped (n=18)	Profound (n=17)	p-value
Complete recovery	13 (46)	16 (44)	20 (77)	13 (72)	1 (6)	<0.001
Marked improvement	1 (4)	5 (14)	0 (0)	3 (16)	9 (53)	
Slight improvement	6 (21)	7 (19)	2 (8)	0 (0)	1 (6)	
No change	8 (29)	8 (22)	4 (15)	2 (11)	6 (35)	

Data are presented as n (%). Complete recovery: all 5 frequencies of final audiogram are 20 dB or less or improvement to the same degree of hearing as in the unaffected ear; Marked improvement: PTA improvement >30 dB; Slight improvement: 10 dB < PTA improvement <30 dB; No change: PTA improvement <10 dB. PTA, pure tone average

Treatment response results

There were no differences in VD, HD, and CSA values between recovery groups according to Furuhashi criteria ($p=0.18$; $p=0.37$; $p=0.27$, respectively). The treatment responses differ in groups of pretreatment audiogram types that were shown in Table 4. The treatment responses were also different according to initial hearing levels. Eight (100%) patients with very mild HL, 27 (87.1%) patients with mild HL, 14 (50%) patients with moderate HL, 9 (31%) patients with moderate-severe HL, 4 (3.2%) patients with severe HL, and 1 (7.1%) patient with very severe HL had complete recovery ($p<0.001$).

Discussion

Data from the current study show that patient with ISSHL had a smaller CSA of the CN, on the affected side, compared to the control group as well as on the unaffected side. Also, patient with ISSHL had a smaller VD and HD of the CN than control group. For the first time in the literature, the relationship between morphologic measurements of CN and the type of pretreatment audiograms was examined in the current study and it was found that the morphologic measurements of the CN did not have any relation with the type of pretreatment audiograms. Also, the morphologic measurements of the CN did not affect the treatment response; and the treatment responses of patients were only related to the type of pretreatment audiograms and the severity of pretreatment HL.

The 3D-CISS is a banding artifact-free type of gradient-echo MRI sequence. In this modality, image contrast is determined by the ratio of T2/T1. When structures are surrounded by cerebrospinal fluid, a perfect contrast between the cerebrospinal fluid and other structures is realized by 3D-CISS. Hence it is preferred in the imaging of the structures within the cerebellopontine angle and IAC. Another advantage is the applicability of this imaging method in MRI devices with 1.5-T as well as 3-T field strengths [10]. Nowadays, there are limited studies on CN morphologic measurements using 3D-CISS.

In a recent study, 53 patients with postlingual sudden HL for 5–20 years were evaluated by comparing the CN sizes with

the healthy side ears. No difference was found between the CN sizes in the healthy and deaf sides of these patients. Mean values of HD and area of CN were determined as a mean of 0.82 mm and 1.16 mm in the healthy ear, 0.75 mm and 1.14 mm in the deaf side, respectively without statistically significant difference. This may be owing to small sample size of study and the absence of a control group [8].

Measurements of CN sizes by MRI technique have clinical significance in cochlear implant patients. In the study in which 68 postlingually sensorineural HL patients underwent cochlear implantation (CI) were included, there was a negative correlation between the prolongation of sensorineural HL and CSA. Although it was not statistically significant, a negative correlation was detected between the severity of HL and CSA. Also in this study, it was found that there was a positive correlation between CSA and hearing success after CI. The authors suggested that MRI could be used to foresee residual hearing and hearing success after CI in patients with postlingual sensorineural HL. Although this study provides important information between deafness and CN size, it does not fully reflect the CN size in ISSHL patients due to the heterogeneous group of the patients included in the study [7]. CN diameter was used in another study aiming to predict the results in cochlear implant patients [11].

In studies conducted on children with unilateral SSSL, CN size on the affected side was found to be smaller than that on the healthy side. This was found to be more pronounced, especially in patients with genetic disorders [12,13]. Nowadays, there are articles about the possibility of genetic disorders with ISSHL in adults, so there may be a high probability of differences between CN diameters in adults as shown in our study [14,15]. When the current literature is reviewed, it is considered that this condition is frequently related to late-onset genetic diseases in patients with bilateral profound sensorineural HL [16]. However, there is a need for further genetic studies showing that genetic predisposition also has an effect on unilateral SSNHL disease.

Apart from these studies using imaging methods, there are also morphological studies on the human temporal bone. In

these cadaver studies, diameters of CNs were smaller in deaf subjects than those in normal controls [4]. In another cadaver study, a strongly positive correlation was found between the diameters of vestibulocochlear nerve (and also one by one cochlear and vestibular nerves), and residuary spiral ganglion cell count in patients with sensorineural HL [17]. These results may explain the fewer spiral ganglion cells found in patients with CNs of smaller diameters. In case of ischemia or inflammation, a scant amount of spiral ganglion cells present in ISSHL patients who have smaller CNs may be prone to loss of all these cells easily, whereas those patients with a sufficient amount of spiral ganglion cells may not lose all of these cells and be protected from ISSHL. This hypothesis may explain our findings in this study.

In our study, the main finding is the lower CN diameters in ISSHL patients. This may be a profound finding due to the fewer number of spiral ganglion cells in these patients as a result of several causes which can be congenital or acquired. As we have performed MRI imaging within 2 days of admission, we do not expect CN atrophy in such a short term, but an acute injury to spiral ganglion cells which are proposed to be profoundly lower in ISSHL by lower CN diameters is thought to be the main cause of SHL in our study population.

The definitive prognostic factors of ISSHL include advanced age, the severity of initial HL, existence of vertigo, concomitant comorbidities, and delay between starting therapy and onset of symptoms [18-21]. In our study, in line with the literature, a relationship was found between curve type and recovery. It has been observed that patients with a U-shaped audiogram had the best prognosis. With regard to degree of HL, patients with severe HL had the worst prognosis. However, in our study, no relation was found between CN size and curve type, degree of HL, and response to treatment. This may support that the curve type and response to treatment were related to the affected area in the cochlea rather than the CN diameter.

Limitations of the study

The number of patients was relatively small to categorize according to the curve types, degrees of HL, and treatment response. This may be another reason why there is no relationship between audiogram curve types, pretreatment degree of HL, response to treatment, and CN diameter. Besides, prognostic factors of ISSHL could not be homogenized in terms of evaluation of response to treatment.

Conclusion

In our study, it has been shown that CN size may be a risk factor for ISSHL to occur. But CN size was not prognostic in

terms of treatment response. These findings may explain why ISSHL disease is often unilateral; smaller diameter CN may indicate fewer spiral ganglion cells, indicating that the cochlea is more susceptible to ischemia or inflammation. Cohort studies involving larger numbers of participants are needed to support this hypothesis.

Acknowledgments

None

Conflicts of Interest

The authors have no financial conflicts of interest.

Author Contributions

Conceptualization: Hande Arslan. Data curation: Meltem Özdemir, Rasime Pelin Kavak, Kemal Keseroğlu. Formal analysis: Hande Arslan. Investigation: Hande Arslan, Meltem Özdemir. Methodology: Hande Arslan. Project administration: Hande Arslan. Software: Kemal Keseroğlu. Supervision: Murad Mutlu, Mehmet Hakan Korkmaz. Validation: Kemal Keseroğlu, Rasime Pelin Kavak. Visualization: Murad Mutlu, Mehmet Hakan Korkmaz. Writing—original draft: Hande Arslan. Writing—review & editing: Hande Arslan, Kemal Keseroğlu, Mehmet Hakan Korkmaz. Approval of final manuscript: all authors.

ORCID iDs

Hande Arslan	https://orcid.org/0000-0003-0344-2712
Meltem Özdemir	https://orcid.org/0000-0002-7388-2871
Rasime Pelin Kavak	https://orcid.org/0000-0001-9782-0029
Kemal Keseroğlu	https://orcid.org/0000-0002-6589-1663
Murad Mutlu	https://orcid.org/0000-0003-0325-5511
Mehmet Hakan Korkmaz	https://orcid.org/0000-0001-8732-3061

REFERENCES

- 1) Stachler RJ, Chandrasekhar SS, Archer SM, Rosenfeld RM, Schwartz SR, Barrs DM, et al. Clinical practice guideline: sudden hearing loss. *Otolaryngol Head Neck Surg* 2012;146(3 Suppl):S1-35.
- 2) Vasama JP, Linthicum FH Jr. Idiopathic sudden sensorineural hearing loss: temporal bone histopathologic study. *Ann Otol Rhinol Laryngol* 2000;109:527-32.
- 3) Yoon TH, Paparella MM, Schachern PA, Allegra M. Histopathology of sudden hearing loss. *Laryngoscope* 1990;100:707-15.
- 4) Nadol JB Jr, Xu WZ. Diameter of the cochlear nerve in deaf humans: implications for cochlear implantation. *Ann Otol Rhinol Laryngol* 1992;101:988-93.
- 5) Russo EE, Manolidis S, Morriss MC. Cochlear nerve size evaluation in children with sensorineural hearing loss by high-resolution magnetic resonance imaging. *Am J Otolaryngol* 2006;27:166-72.
- 6) Glastonbury CM, Davidson HC, Harnsberger HR, Butler J, Kertesz TR, Shelton C. Imaging findings of cochlear nerve deficiency. *AJNR Am J Neuroradiol* 2002;23:635-43.
- 7) Kim BG, Chung HJ, Park JJ, Park S, Kim SH, Choi JY. Correlation of cochlear nerve size and auditory performance after cochlear implantation in postlingually deaf patients. *JAMA Otolaryngol Head Neck Surg* 2013;139:604-9.
- 8) Islamoglu Y, Kesici GG, Ercan K, Babademez MA. Single-sided deafness after sudden hearing loss: late effect on cochlear nerve size. *Eur Arch Otorhinolaryngol* 2020;277:2423-6.
- 9) Furuhashi A, Matsuda K, Asahi K, Nakashima T. Sudden deafness: long-term follow-up and recurrence. *Clin Otolaryngol Allied Sci* 2002;

- 27:458-63.
- 10) Hingwala D, Chatterjee S, Kesavadas C, Thomas B, Kapilamoorthy TR. Applications of 3D CISS sequence for problem solving in neuroimaging. *Indian J Radiol Imaging* 2011;21:90-7.
 - 11) Han JJ, Suh MW, Park MK, Koo JW, Lee JH, Oh SH. A predictive model for cochlear implant outcome in children with cochlear nerve deficiency. *Sci Rep* 2019;9:1154.
 - 12) Nakano A, Arimoto Y, Matsunaga T. Cochlear nerve deficiency and associated clinical features in patients with bilateral and unilateral hearing loss. *Otol Neurotol* 2013;34:554-8.
 - 13) Furuta S, Ogura M, Higano S, Takahashi S, Kawase T. Reduced size of the cochlear branch of the vestibulocochlear nerve in a child with sensorineural hearing loss. *AJNR Am J Neuroradiol* 2000;21:328-30.
 - 14) Gäckler A, Eickelmann AK, Brors D, Dazert S, Epplen JT, Kunstmann E. Positive family history of idiopathic sudden sensorineural hearing loss. *Eur Arch Otorhinolaryngol* 2010;267:1843-8.
 - 15) Binnetoğlu A, Yumuşakhuylu AC, Demir B, Bağlam T, Derinsu U, Sarı M. Association between family history and idiopathic sudden sensorineural hearing loss. *J Int Adv Otol* 2015;11:30-2.
 - 16) Uehara N, Fujita T, Yamashita D, Yokoi J, Katsunuma S, Kakigi A, et al. Genetic background in late-onset sensorineural hearing loss patients. *J Hum Genet* 2022;67:223-30.
 - 17) Nadol JB Jr. Patterns of neural degeneration in the human cochlea and auditory nerve: implications for cochlear implantation. *Otolaryngol Head Neck Surg* 1997;117(3 Pt 1):220-8.
 - 18) Chang NC, Ho KY, Kuo WR. Audiometric patterns and prognosis in sudden sensorineural hearing loss in southern Taiwan. *Otolaryngol Head Neck Surg* 2005;133:916-22.
 - 19) Attanasio G, Russo FY, Di Porto E, Cagnoni L, Masci E, Ralli M, et al. Prediction of hearing recovery in sudden deafness treated with intratympanic steroids. *Acta Otorhinolaryngol Ital* 2018;38:453-9.
 - 20) Belhassen S, Saliba I. Intratympanic steroid injection as a salvage treatment for sudden sensorineural hearing loss. *J Laryngol Otol* 2014; 128:1044-9.
 - 21) Atay G, Kayahan B, Çınar BÇ, Saraç S, Sennaroğlu L. Prognostic factors in sudden sensorineural hearing loss. *Balkan Med J* 2016;33:87-93.