Scoring System for Predicting No Hearing Recovery in Unilateral Idiopathic Sudden Sensorineural Hearing Loss

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ABSTRACT

***Background**: The varying degrees of hearing recovery in idiopathic sudden sensory neural hearing loss (ISSHL) patients indicate the need of model to predict no hearing recovery. We aimed to aid in the counseling of ISSHL patients about their recovery chances by developing a simple clinical scoring system to predict no hearing recovery using clinical information available at first visit.* **Methods:** *A retrospective cohort study, using medical records was conducted from January 2017-May 2019 in Cipto Mangunkusumo General Hospital and Proklamasi Ear, Nose, Throat, Head and Neck (ENT-HN) Surgery Specialized Hospital in Jakarta, Indonesia. The outcome measure is no hearing recovery and we built the prediction score developed based on multiple logistic regression analyses and tested for discriminative ability. There were 183 adults unilateral ISSHL patients included in the study.* **Results**: *The proportion of no hearing recovery was 56%. The independent predictors were older age 30-60 years and >60 years old (Odds Ratio 4.0; 95% CI 1.4-11.8; p=0.012 and OR 5.3; 95% CI 1.5-18.4; p=0.008, respectively) as compared with 18-<30 years old, later onset (onset 15-60 days and >60 days had OR 5.4; 95% CI 1.7-16.9; p=0.004 and OR 12.6; 95% CI 2.9-54.6; p=0.001, respectively, as compared with onset < 3 days), and presence of vertigo (OR 2.3; 95% CI 1.1-4.6; p=0.026). Prediction scores ranged from 3 to 12, with three categories for age, four for onset, and two for the presence of vertigo. The predictions showed adequate calibration and good discriminative ability (AUC 0.77).* **Conclusion**: *Using information of age, onset and presence of vertigo at first visit, ISSHL patient with increased risk of no hearing recovery can be identified with moderate accuracy. This prediction model could help clinician in predicting patients' prognosis.*

***Keywords**: idiopathic sudden sensory neural hearing loss, score system, no hearing recovery*

INTRODUCTION

Idiopathic sudden sensory neural hearing loss (ISSHL) or sudden deafness is one of the emergency conditions, defined as an abrupt loss of hearing with sensory neural type, hearing loss of more than 30 dB at a minimum of 3 (three) consecutive frequencies occurring in less than 72 hour.[1] During the two-year period, the incidence of SSNHL was estimated to be 27 per 100,000, with an average of 66,594 new cases per year among the insured population of the United States.[2] In Cipto Mangunkusumo Hospital (CMH), Jakarta, Indonesia, the number of patients with ISSHL was 149 cases between 2016-2019. In Proklamasi Ear Nose and Throat, Head and Neck (ENT-HN) Surgery Specialized Hospital, Jakarta, Indonesia, the number of cases between 2017-2019 was 342. The occurrence of ISSHL can potentially lead to persistent hearing loss that have impacts on communication, understanding, and sound lateralization, which will ultimately affect patient's quality of life.

Besides hearing impairment, another common complaint with ISSHL is tinnitus or ringing in the affected ear.[3] Tinnitus accompanies nearly 88% of sudden deafness cases and can last even after treatment.[4] This complaint can lead to depression, anxiety, and other psychological disturbances.[5] Until now, the cause and mechanism of ISSHL are still unclear. Nearly 90% of cases are idiopathic, but it has been indicated to be associated with inflammatory processes, vascular disorders, viruses, or other multiple etiologies.[1]

The aims of the treatment is to reduce inflammation and to improve vascular circulation and oxygenation in the inner ear. Corticosteroid, as a single or combined therapy, is frequently used as the initial treatment for ISSH and was administered in a various ways (i.e., systemic, intratympanic) and doses. Although none of meta-analyses or clinical trials identified one effective treatment as all existing therapies are still empirical treatment, [6–10] a literature review indicates that the effective treatment time for abrupt sensorineural hearing loss is two to four weeks. Therefore, it is prudent to begin treatment as soon as feasible.[11] Hearing improvement in patients with ISSHL varies greatly, ranging from complete recovery, partial recovery, slight recovery, to no improvement.[12] Based on the literature, ISSHL patients with severe loss of hearing can achieve slight improvement to complete recovery in about 20-40% cases, although some studies also reported spontaneous recovery ranging from 32% -70% of the cases.[13,14]

To date, various studies have reported several predictor factors related to the recovery of patients with ISSHL. Younger age, presence of vestibular disorders, degree of hearing loss, and audiogram configuration are the most widely used predictors for improvements in patient's hearing threshold.[4,13,14] In addition, tinnitus is also identified as one of the predictors of patient's hearing threshold. The pitch of tinnitus is one of characteristics related to the location or tonotopy of the cochlear damage. The psychoacoustic testing can identify the tinnitus pitch within the area of impaired frequency or elsewhere.[3,15] In many cases, the tinnitus pitch measured using the pure-tone audiogram (audiogram edge) is located at the border of the area between normal and impaired frequency. This condition is supported by the theory explaining that the loss of neuronal inhibitions in the affected area would trigger neuronal excitations in adjacent areas that are not or only slightly disturbed.[15]

The varying degrees of hearing recovery in patients with ISSHL led to the need for clinicians and patients to have better understanding on factors that can predict hearing recovery. Therefore, this study aimed to determine the predictors and develop a simple clinical scoring system that can be used to predict no hearing recovery in patients with ISSHL.

METHODS

Source of Data

A retrospective cohort design was used in this study. We retrieved medical record data of patients with ISSHL who visited the ENT outpatient clinic in Cipto Mangunkusumo General Tertiary Hospital and Proklamasi ENT-HN Surgery Specialized Hospital, from January 2017 to May 2019. We observed the patients starting from the date of initiation of therapy until the last visit to the outpatient clinic with a completed final audiometry result.

Participants

The inclusion criteria were adults (age >18 years) diagnosed with unilateral ISSHL who had received corticosteroid therapy (i.e., systemic, intratympanic therapy, combination) as sudden deafness protocol, with or without hyperbaric therapy. We collected treatment information from patient medical record. We excluded patients with (1) history of tinnitus or previous sensory neural hearing loss (SNHL), (2) Meniere syndrome, (3) autoimmune disease, (4) pregnancy, (5) intracranial tumour, (6) history of stroke, and (7) chronic kidney disease with or without hemodialysis. We also excluded cases with incomplete medical records data. We did not obtain informed consent because the study was a retrospective review of patients' clinical records, and the information was anonymized and de-identified prior to analysis. This study was approved by the Ethics Committee of the Faculty Medicine Universitas Indonesia.

From the medical records, we also collected demographic and clinical data such as age, gender, clinical symptoms (e.g., tinnitus, vertigo), comorbidity, and initial audiometry examination results. The data for tinnitus, vertigo, and comorbidity is derived solely from patient histories. No objective measurements existed. We determined the hearing threshold by calculating the average threshold of four pure tones frequencies of 500 Hz, 1000 Hz, 2000 Hz, and 4000 Hz. We then determined the severity of hearing loss by categorizing the hearing threshold based on a modified ASHA classification into three groups: hearing threshold ≤40 dB, hearing threshold >40-70 dB, and >70 dB.[16]

We also classified the audiogram based on following patterns: (1) up-sloping (decline in low frequency), (2) down-sloping (decline at high frequency), (3) flat moderate to severe (decline in all frequencies involved), and (4) profound (flat audiogram with hearing threshold > 90 dB).

Outcome

The main outcome of this study is to determine the predictors for the development of a simple clinical scoring system that aims to predict no hearing recovery in ISSHL patients.

Predictors that were analyzed included patient's age, days since onset of symptoms and beginning of treatment, the presence of vertigo, audiometric patterns, and the severity of hearing loss. These predictors were measured without the knowledge of patient's outcome.

In order to assess the hearing improvement, we compared the last and initial audiometry result documented in the medical record and categorized it into no hearing recovery and hearing recovery. No hearing recovery was defined as no improvement of hearing threshold of at least 15 dB, or if the hearing threshold was 75 dB or more. Whilst hearing recovery was categorized into: complete recovery (improvement of hearing threshold > 25 dB); partial recovery (improvement of hearing threshold > 15 dB and the hearing threshold was between 25-45 dB); and slight recovery (improvement of the hearing threshold of 15 dB and the hearing threshold > 45 dB)

Ethical Approval

This study was approved by the Ethics Committee Faculty of Medicine Universitas Indonesia (KET-440/UN2.F1/ETIK/PPM.00.02/2019).

Sample Size

Minimum sample size was estimated using rule-of-thumb, which stated that for every predictor in the model there must be at least 10 subjects with an occurrence of the outcome of interest. Based on the incidence of hearing improvement post therapy of 40%, and a total set of 5 predictors of interest, we estimated that a cohort of 150 subjects would be sufficient.

Missing Data

In this study, we only calculated the cases with complete data. We considered to exclude any missing data from the analysis. Missing data were assessed using the missing value analysis to see whether the missing value may have an impact on the results since we only computed the instances with complete data in this study.

Statistical Analysis

We analyzed the data using the IBM SPSS Statistics 24.0 computer program. Patient characteristics or demographics was described in the baseline characteristic table. Numbers and percentages were used to represent categorical variables. The median (interquartile range/

IQR) of the numerical or continuous variables was shown. Bivariate and multivariate analysis were used. The multivariate analysis included all independent variables with a p-value of <0.25 in the bivariate analysis. The bivariate study employed a Chi-square test to evaluate the relationship between the independent factors and the outcome. In the multivariat logistic regression analysis, predictors with p-values <0.05 were considered as significant factors related to the clinical outcomes. A scoring system was developed based on multiple logistic regression analyses and tested for discriminative ability. The performance of the scoring system was assessed for its calibration ability (the agrrement between the obesrved outcomes and predictions) using the Hosmer-Lemeshow test and its discriminatory ability(the model's ability to discriminative between low and high risk patient) by estimating areas under receiving operating characteristic curves (AUC). To assure the robustness, model calibration was done. The bootstrapping method was used for internal validation. With replacement samples taken from the original sample, we performed the full modeling procedure, including variable selection, in 1000 samples.

RESULTS

Participants

Of 342 screened patients, 120 were excluded due to incomplete data (n=92), the existence of history of tinnitus (n=7), previous SNHL (n=5), Meniere disease (n=4), stroke (n=4), autoimmune disease (n=3), chronic kidney disease (n=3), and intracranial tumour (n=2). Of the remaining 222 patients that qualified for this study, 39 patients did not come to the hospital for follow up visits. This left 183 patients with ISSHL available for analysis (**Figure 1**). Median duration of follow up was 18 days (range 2-134 days).

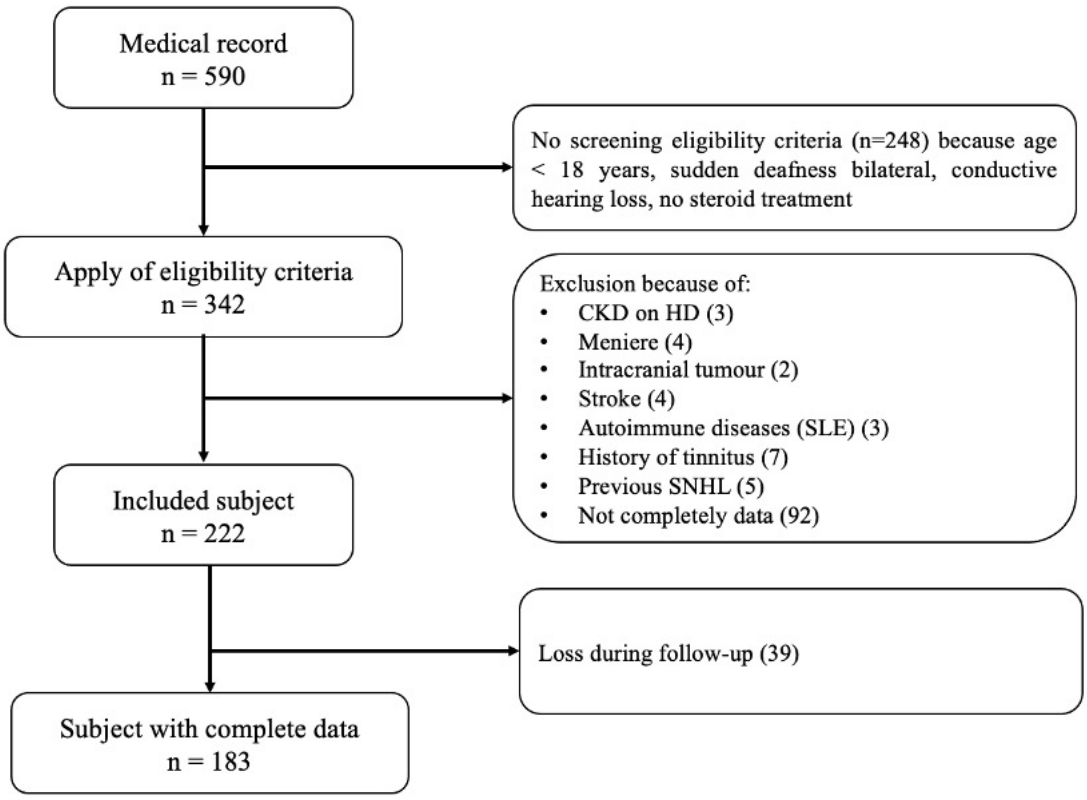

Figure 1. Flowchart of the Subject Recruitment

There were 103 males and 80 females with the mean age of 47.6 years with standard deviation (SD) of 14.4 years. The most affected ear was the left ear (55.2%) and most of the patients came to the hospital when they first had the symptoms of ISSHL in the preceding 3-14 days (45.9%). The median of initial hearing threshold was 70.61 dB with interquartile range (IQR) of 46.25 dB. Of all patients, 12.1% and 7.7% had diabetes mellitus and hypertension, respectively. Almost all patients had tinnitus (96.4%) and in 104 patients (56.8%) also experienced vertigo. (**Table 1**).

Table 1. Baseline Characteristics of the Subjects

Characteristic subject, (N=183)	**n**	**%***
Age (year), mean (SD)*	47.6	14.4*
Category age (year)		
18-<30 year	24	13.1
30-60 year	120	65.6
>60 year	39	21.3
Gender		
Male	103	56.3
Female	80	43.7
Affected ear		
Left ear	101	55.2
Right ear	82	44.8
Days since onset of symptoms		
< 3 days	25	13.7
3-14 days	84	45.9
15-60 days	45	24.6
>60 days	29	15.8
Vestibular Symptom		
Vertigo	104	56.8
No vertigo	79	43.2
Hearing threshold (dB), median (IQR)	70.61	46.25
Hearing threshold		
≤40 dB	30	16.4
>40-70 dB	62	33.9
>70 dB	91	49.7
Configuration of Audiogram		
Up Sloping	9	4.9
Down Sloping	17	9.3
Flat Moderate Severe	109	59.6
Profound	48	26.2
DM		
Yes	22	12.1
No	161	87.9
Hypertension (n=181)		
Yes	14	7.7
No	167	92.3
Tinnitus (n=169)		
Yes	163	96.4
No	6	3.6

The median hearing thresholds in the initial and last observation based on audiometry examination were 70.61 dB (IQR 46.25) and 55.86 dB (IQR 47.5), respectively. Most patients came with hearing thresholds > 70 dB (49.7%), followed by >40-70 dB (33.9%). The result of the bivariate analyses showed four predictors significant to no hearing recovery, which were age groups, days since onset of symptoms, presence of vertigo, and hearing thresholds categories (**Table 2**).

On the multivariate analyses, there were several predictor variables included for the model development, which were age (30-60 years and >60 years), days since onset of symptoms (15-60 days and >60 days), and vestibular symptom (vertigo) (**Table 3**).

Model Development and Performance

Table 4 shows the scoring system for prediction of no hearing recovery after ISSHL using information available at patients first visit. The scores were obtained by rounding up the regression coefficients of the predictors to the nearest integer.

After the calculation of score for no hearing

Table 2. Bivariate Analyses of the Predictor Variables of No Hearing Improvement

Predictor	Hearing status, n (%)		P value
	Good hearing recovery	No hearing recovery	
Age			0.050
18-<30 years	16 (66,7)	8 (33.3)	
30-60 years	49 (40.8)	71 (59.2)	
>60 years	15 (38.5)	24 (61.5)	
Days since onset of symptoms			<0.001
< 3 days	15 (60)	10 (40)	
3-14 days	49 (58.3)	35 (41.7)	
15-60 days	12 (26.7)	73.3 (25)	
>60 days	4 (13.8)	25 (86.2)	
Affected ear			0.394
Left ear	47(53.5)	54(46.5)	
Right ear	33(40.2)	49(59.8)	
Vestibular symptom			0.049
Vertigo	52 (50)	52 (50)	
No Vertigo	28 (35.4)	51(64.6)	
Hearing threshold category			0.005
≤ 40 dB	19 (63.3)	11 (36.7)	
>40-70 dB	18 (29.0)	44 (71.0)	
>70 dB	43 (47.3)	48 (52.7)	
Configuration			0.392
Up sloping	5 (55.6)	4 (44.4)	
Down sloping	5(27.8)	13(72.2)	
Flat moderate severe	51(46.8)	58(53.2)	
Profound	19(43.7)	28(56.3)	

Table 3. Multivariate Analysis of the Predictor Variables of No Hearing Improvement

Predictor	OR	95% CI	P Value
Age			
18-<30 years	ref		
30-60 years	4.00	1.36-11.77	0.012
>60 years	5.33	1.54-18.43	0.008
Days since onset of symptoms			
< 3 days	ref		
3-14 days	1.25	0.46-3.37	0.662
15-60 days	5.37	1.71-16.87	0.004
> 60 days	12.61	2.91-54.59	0.001
Vestibular symptom			
No vertigo	ref		
Vertigo	2.25	1.10-4.59	0.026
Hearing threshold			
≤ 40 dB	ref		
>40-70 dB	2.58	0.89-7.41	0.078
>70 dB	1.36	0.51-3.62	0.537

Table 4. Scoring System for Prediction of No Hearing Improvement

Variables	B	SE	B/SE	B/SE Smallest score	Score	P value
Age						
18-<30 years	ref					
30-60 years	1.492	0.542	2.752	3.811	4	0.006
>60 years	1.776	0.626	2.837	3.940	4	0.005
Days since onset of symptoms						
<3 days	ref					
3-14 days	0.355	0.492	0.722	1	1	0.471
15-60 days	1.805	0.573	3.150	4.363	4	0.002
>60 days	2.755	0.727	3.789	5.248	5	<0.001
Vestibular symptom						
No vertigo	ref					
vertigo	0.886	0.353	2.509	3.475	3	0.012

B = Beta; SE = Standard Error

recovery are age >30 years (score 4), days since onset of symptoms 15-60 days (score 4) and >60 days (score 5), and vertigo (score 3), with the maximal score is 12. The AUC of the scoring system is shown on **Figure 2**, showing that the score was 0.77 (95% CI 0.70-0.85) indicating moderate accuracy as prediction model. Based on the internal validation with bootstrapping logistic regression, the result of Hosmer Lemeshow goodness of fit test was not significant (p=.284) indicating the data fit the model and were not diffferent than expectation based on the model.

From the sensitivity and specificity analysis (**Table 5**), the cut-off for the score was 7. From the calculation cross tabulation between cut-off score and clinical outcome, patients with score greater than 7 had probability for no hearing recovery by 81.3% while in patients with score 7 or lower the probability for no hearing recovery was 31.5%.

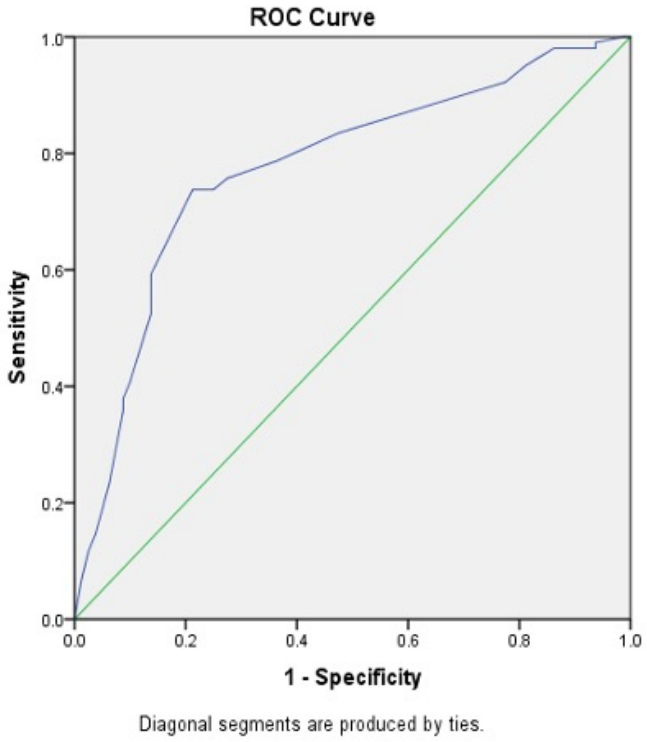

Figure 2. AUC of the Scoring System for Prediction of No Hearing Recovery

Table 5. Sensitivity and specificity score prediction no hearing improvement in ISSHL.

Positive if Greater Than or Equal To[a]	Sensitivity	Specificity
-1.0	1.000	0.000
0.5	1.000	0.013
2.0	0.990	0.063
3.5	0.981	0.063
4.5	0.922	0.225
6.0	0.767	0.638
7.5	0.718	0.788
8.5	0.359	0.913
10.0	0.223	0.950
11.5	0.068	0.988
13.0	0.000	1.000

DISCUSSION

The proportion of no hearing recovery in this study was 0.56. This number is comparable to the findings of Xie et al [17] which show a proportion of 0.57 for no improvement based on an audiometric examination during 50 days follow up after initial treatment. The model prediction score of no hearing recovery in ISSHL in this study consists of several predictors, including age, onset of symptom, and the presence of vestibular symptom (vertigo).

In this study, age more than 30 years had a score of 4 from the model, however, there is still unclear explanation why this age affected the clinical outcome. A study in United States about the prevalence of hearing loss in adults aged 20-69 years from 2011 until 2012 showed that the prevalence of hearing loss rises sharply above ages 30 to 39 years and on average 3-fold increased risk of hearing loss per decade.[18] This condition indicates an age-related condition which may affect the clinical outcome of ISSHL

patients. Two studies assessing prognostic factors of ISSHL also consistently showed age as one of the most important prognostic factors for the hearing outcome.[17,19]

Onset of symptom until first initial treatment in this model consists of 15-60 days (score 4) and more than 60 days (score 5). Several studies have concluded that the earlier the patients receive the treatment, the better the outcome. A study from Cvorovic et al[20] showed that patients who had the treatment within 7 days following the first symptom had hearing improvement rate of 60% and this number decreased in patients receiving the treatment after 7 days. Another study from Anyah et al[21] reported that patients with the onset of more than 90 days showed no hearing improvement. The variation of association between interval time and clinical outcome, could be affected with spontaneous recovery which has been seen in 32%-81% of ISSHL patients.[22]

Cochlea condition can also be related to the duration of symptom and initial treatment. Cochlea is an organ with highly dependent on blood and oxygen supply to maintain its function. The cochlea becomes hypoxic or ischemic within one minute after occlusion of the labyrinthine arter.[23]An experimental study on guinea pigs' cochleas showed that a 60-minute anoxia induced by pressing the labyrinthine artery resulted in irreversible lesions in the cochlea.[24] The result was in line with ISSHL patients, showing that there were impaired blood flow and oxygen delivery to the cochlea, giving a negative impact to the clinical outcome.

In this study, vertigo was the most common symptom found in ISSHL patients (56%), and among patients with vertigo, 65% had no hearing recovery. The percentage of patients complaining simultaneous vertigo was nearly 20-60% in several studies.[11,25] Both cochlea and vestibular organs has close relation in anatomy, therefore the presence of vertigo may indicate a widespread involvement of the disease in the inner ear that could explain why there was a reduced probability of recovery in ISSHL patients. A systematic review [26] identified patterns of vestibulocochlear lesion in relation to ISSHL, showed that utriculus and superior vestibular nerves were most prone to damage in ISSHL. It then was followed by the lateral semi-circular canal and superior vestibular nerve, saccule and inferior vestibular nerve. The superior vestibular nerve has long and narrower bony canal, thus leading to a higher susceptibility to ischemic labyrinthine changes compared to the others.[26]

Vertigo was also found in ISSHL patient with a poorer hearing threshold in high frequency compared to patient without vertigo and it is significantly related with poor hearing recovery.[25,27,28] Poorer hearing threshold in high frequency was commonly found in ISSHL patients. The higher frequency level close proximity to basal turn of the cochlea to the vestibuli related to otolithic pathology and also blood supply to both organs.[25]

This study developed prediction model for no hearing recovery in ISSHL patients which can be applied during the first visit. Although, there were 92 missing data in this study, we found the same characteristic subject within those groups, thus we concluded that the missing was at random and did not affect the analysis. The model performs well and helps to distinguish patients with high risk of no hearing recovery from those with low risk. This study did not consider the comorbidities which might influence patient's prognosis, such as hypertension, diabetes mellitus and dyslipidemia. However, Menezes et al[29]showed that the presence of comorbidities, such as hypertension, diabetes mellitus, dyslipidemia, overweight or obesity, smoking and previous cardiovascular event were not associated in hearing improvement. Another study from Wang et al[30] showed hypertension and diabetes mellitus were not correlated with hearing improvement in ISSHL patient.

The limitation of this study was data solely collected based on history taking and lack of objective measurements. This included the information on vertigo, in which we were unable to obtain information regarding vestibular tests from the medical record. Nonetheless, because vertigo is a common symptom of ISSHL, this historical information is still reliable. Another limitation in this study is unavailability of the pitch of tinnitus data. Since the psychoacoustic tinnitus measurement for identifying the

specific frequency of tinnitus was not a routine audiological examination for ISSHL patients in our hospitals, we did not collect and analyze any further due to potential incomplete data. Regarding the applicability of this model, since in this study only included the subject with normal hearing and no tinnitus prior ISSHL, this model would not apply to patient with some degree of pre-existing hearing loss or tinnitus.

To the best of our knowledge, this is the first study which developed the scoring system to predict no hearing recovery in ISSHL patients. In Indonesia, ISSHL patients came with various onset, and this scoring system will help clinician to determine the management strategy of ISSHL patient.

CONCLUSION

From this study, we can conclude that the prediction model for no hearing recovery ISSHL could be applied in clinical setting, especially at first visit concerning their eventual recovery This model consisted of simple variable that we can identify from the history taking. The model performs well and helps to distinguish patients with high risk of no hearing recovery from those with low risk. However, we must also consider about various clinical variation of ISSHL patients and the wider of range possibility of spontaneous recovery in ISSHL patient. It is important to continue this study to validate this model to the external population.

AUTHORSHIP

EDS: Conceptualization, data curation, formal analysis, funding acquisition, investigation, methodology, supervision project administration, writing original draft preparation, review and editing. KH: Conceptualization, formal analysis, methodology, validation, writing original draft preparation. RR: Conceptualization, formal analysis, validation, writing original draft preparation, review and editing. JB: Conceptualization, methodology, investigation, review and editing. WA: Conceptualization, methodology, investigation, review and editing. AS: Formal analysis, methodology, writing original draft preparation, review and editing.

CONFLICT OF INTEREST AND SOURCE OF FUNDING

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REFERENCES

1. Stachler RJ, Chandrasekhar SS, Archer SM, et al. Clinical practice guideline: Sudden hearing loss. Otolaryngol Neck Surg [Internet]. 2012 [cited 2018 Dec 9];146(1S):1–35. Available from: http://otojournal.org
2. Alexander TH, Harris JP. Incidence of sudden sensorineural hearing loss. Otol Neurotol. 2013;34(9):1586–9.
3. Ding X, Zhang X, Huang Z, plasticity XF-N, 2018 undefined. The characteristic and short-term prognosis of tinnitus associated with sudden sensorineural hearing loss. HindawiCom [Internet]. 2018;2018. Available from: https://www.hindawi.com/journals/np/2018/6059697/abs/
4. Hikita-Watanabe N, Kitahara T, Horii A, Kawashima T, Doi K, Okumura SI. Tinnitus as a prognostic factor of sudden deafness. Acta Otolaryngol. 2010;130(1):79–83.
5. Michiba T, Kitahara T, Hikita-Watanabe N, et al. Residual tinnitus after the medical treatment of sudden deafness. Auris Nasus Larynx [Internet]. 2013;40(2):162–6. Available from: http://dx.doi.org/10.1016/j.anl.2012.06.007

6. Wei BPC, Stathopoulos D, O'Leary S. Steroids for idiopathic sudden sensorineural hearing loss. Cochrane Database Syst Rev. 2013;2013(7).
7. Filipo R, Attanasio G, Russo FY, Viccaro M, Mancini P, Covelli E. Intratympanic steroid therapy in moderate sudden hearing loss: A randomized, triple-blind, placebo-controlled trial. Laryngoscope. 2013;123(3):774–8.
8. Crane RA, Camilon M, Nguyen S, Meyer TA. Steroids for treatment of sudden sensorineural hearing loss: A meta-Analysis of randomized controlled trials. Laryngoscope. 2015;125(1):209–17.
9. Eftekharian A, Amizadeh M. Pulse steroid therapy in idiopathic sudden sensorineural hearing loss: A randomized controlled clinical trial. Laryngoscope. 2016;126(1):150–5.
10. Han X, Yin X, Du X, Sun C. Combined intratympanic and systemic use of steroids as a first-line treatment for sudden sensorineural hearing loss: A meta-analysis of randomized, controlled trials. Otol Neurotol. 2017;38(4):487–95.
11. Rauch S. Clinical practice. Idiopathic sudden sensorineural hearing loss. N Engl J Med. 2008;359:833–40.
12. Ryu IY, Park SH, Park EB, Kim HJ, Kim SH, Yeo SG. Factors prognostic of season-associated sudden sensorineural hearing loss: A retrospective observational study. J Audiol Otol. 2017;21(1):44–8.
13. Yu H, Li H. Association of vertigo with hearing outcomes in patients with sudden sensorineural hearing loss a systematic review and meta-analysis. JAMA Otolaryngol - Head Neck Surg. 2018;144(8):677–83.
14. Lee HY, Kim DK, Park YH, Cha WW, Kim GJ, Lee SH. Prognostic factors for profound sudden idiopathic sensorineural hearing loss: a multicenter retrospective study. Eur Arch Oto-Rhino-Laryngology. 2017;274(1):143–9.
15. Brian C. J. Moore. The Psychophysics of Tinnitus. In: Jos J. Eggermont F-GZ, Arthur N. Popper RRF, editors. New York: Tinnitus, Springer Science, Business Media New York; 2012. p. 189–211.
16. American Speech-Language-Hearing Association (n.d.). Hearing loss in adults (Practice Portal) [Internet]. /Practice-Portal/Clinical-Topics/Hearing-Loss/. [cited 2021 Dec 10]. Available from: https://www.asha.org/practice-portal/clinical-topics/hearing-loss/#collapse_9
17. Xie Y, Orabi NA, Basura GJ, Zwolan TA. Outcomes of unilateral idiopathic sudden sensorineural hearing loss : Two decades of experience. 2019;693–702.
18. Hoffman HJ, Dobie RA, Losonczy KG, Themann CL, Flamme GA. Declining prevalence of hearing loss in US adults aged 20 to 69 years. JAMA Otolaryngol - Head Neck Surg. 2017;143(3):274–85.
19. Chen C, Shi G, He M, et al. Characteristics and prognosis of idiopathic sudden sensorineural hearing loss in aged people: a retrospective study. Acta Otolaryngol [Internet]. 2019;139(11):959–65. Available from: https://doi.org/10.1080/00016489.2019.1657589
20. Čvorović L, Eric D, Probst R, Hegemann S. Prognostic model for predicting hearing recovery in idiopathic sudden sensorineural hearing loss. Otol Neurotol. 2008;29(4):464–9.
21. Anyah A, Mistry D, Kevern E, Markiewicz K. Idiopathic sudden sensorineural hearing loss: average time elapsed before presentation to the otolaryngologist and effectiveness of oral and/or intratympanic steroids in late presentations. Cureus. 2017;9(12).
22. Nosrati-Zarenoe R. Idiopathic sudden sensorineural hearing loss: corticosteroid treatment, the diagnostic protocol and outcome. Medicine. 2011.
23. Tabuchi K, Nishimura B, Tanaka S, Hayashi K, Hirose Y, Hara A. Ischemia-reperfusion injury of the cochlea: pharmacological strategies for cochlear protection and implications of glutamate and reactive oxygen species. Current Neuropharmacology. 2010;8.
24. Tabuchi K, Kusakari J, Ito Z, Takahashi K, Wada T, Hara A. Effect of nitric oxide synthase inhibitor on cochlear dysfunction induced by transient local anoxia. Acta Otolaryngol. 1999;119(2):179–84.
25. Park HM, Jung SW, Rhee CK. Vestibular diagnosis as prognostic indicator in sudden hearing loss with vertigo. Acta Oto-Laryngologica. 2001;Suppl;80–3.
26. Murofushi T, Barbara M, Li H, Yu H. Vestibular dysfunctions in sudden sensorineural hearing loss: A systematic review and meta-analysis. 2018 [cited 2020 Jan 6];9:45. Available from: www.frontiersin.org
27. Niu X, Zhang Y, Zhang Q, et al. The relationship between hearing loss and vestibular dysfunction in patients with sudden sensorineural hearing loss. Acta Otolaryngol. 2016;136(3):225–31.
28. Korres S, Stamatiou GA, Riga M, Xenelis J. Prognosis of patients with idiopathic sudden hearing loss: Role of vestibular assessment. J Laryngol Otol. 2011;125(3):251–7.
29. Menezes AS, Ribeiro D, Lima A, Miranda D, Guimarães J, Dias L. SCORE risk scale as a prognostic factor after sudden sensorineural hearing loss. Eur Arch Oto-Rhino-Laryngology [Internet]. 2019;276(10):2739–45. Available from: https://doi.org/10.1007/s00405-019-05518-1
30. Wang C Te, Huang TW, Kuo SW, Cheng PW. Correlation between audiovestibular function tests and hearing outcomes in severe to profound sudden sensorineural hearing loss. Ear Hear. 2009;30(1):110–4.