



SCORE risk scale as a prognostic factor after sudden sensorineural hearing loss

Ana Sousa Menezes¹ · Daniela Ribeiro¹ · António Lima¹ · Daniel Miranda¹ · Joana Guimarães² · Luís Dias¹

Received: 29 November 2018 / Accepted: 13 June 2019 / Published online: 20 June 2019
© Springer-Verlag GmbH Germany, part of Springer Nature 2019

Abstract

Introduction Sudden sensorineural hearing loss (SSHL) is an otologic urgency whose treatment is still controversial. Its etiology remains largely unknown in most cases and predicting its prognosis is still a challenge. Cardiovascular risk factors (CVRF) have been implicated in the etiopathogenesis of this entity.

Objectives Application of the SCORE (Systematic Coronary Risk Evaluation) risk scale in patients with SSHL and evaluation of its potential prognostic value in recovery in patients with CVRF.

Materials and methods Prospective analysis of patients with SSHL admitted for protocol treatment including intravenous corticosteroid therapy associated to weekly intratympanic injection in the event of therapeutic failure or severe hearing loss at admission. Demographic, audiometric, clinical and imaging data were assessed. The SCORE risk scale was applied and the audiometric recovery was compared among different risk groups.

Results Our overall complete and partial recovery rates were 35.9% and 26%, respectively. More than a half of our patients had at least one CVRF. Of these, overweight/obesity, hyperlipidemia and hypertension were the most common. In our sample, patients with CVRF and higher SCORE risk presented higher PTA at admission and also worse hearing outcome, although these results were not statically significant.

Conclusion This preliminary study could not confirm the validity for SCORE scale for cardiovascular risk assessment in predicting audiometric recovery in patients with SSHL with multiple comorbidities. Further research with larger samples are needed to elucidate the etiology of SSHL and the exact role of cardiovascular risk factors in the pathophysiology of SSHL.

Level of evidence 4

Keywords Sudden sensorineural hearing loss · Cardiovascular risk factors · SCORE risk scale

Abbreviations

SSHL	Sudden sensorineural hearing loss
CVRF	Cardiovascular risk factors
SCORE	Systematic Coronary Risk Evaluation
PTA	Pure-tone average
BMI	Body mass index
MRI	Magnetic resonance imaging

Introduction

Sudden sensorineural hearing loss (SSHL) is an otologic urgency that affects 5–30 subjects per 100,000/year [1, 2]. It is defined as a hearing loss of at least 30 dB affecting at least three consecutive frequencies in standard pure-tone audiogram occurring over 72 h [3].

The etiology of SSHL remains unknown in 70–90% of cases, despite thorough evaluation [4]. The treatment of SSHL is still controversial as therapy decisions are usually made without knowing the etiology. Therefore, the prediction of its prognosis is still a challenge [3]. A maximum of 32–65% of cases of SSNHL may recover spontaneously [4, 5]. Various prognostic factors have been proposed including patient age, presence of vertigo at onset, degree of hearing loss, audiometric configuration, and time between onset of hearing loss and treatment [4, 6, 7]. Cardiovascular risk factors (CVRF) such as hypertension, diabetes and

✉ Ana Sousa Menezes
ana4644@gmail.com; ana.menezes@hospitaldebraga.pt

¹ Department Of Otorhinolaryngology-Head and Neck Surgery, Hospital De Braga, Sete Fontes, São Victor, 4710-243 Braga, Portugal

² Department of Otorhinolaryngology, Instituto Português de Oncologia Francisco Gentil Do Porto, Porto, Portugal

hyperlipidemia have been implicated in the etiopathogenesis of this clinical entity [8–10].

The study of Chang et al. assessed the role of Framingham Risk Score in predicting the prognosis of SSHL patients [11]. In this study, patients of the high-risk group based on Framingham Risk Score showed poor outcomes of hearing improvement compared to the low-risk group [11]. Framingham Risk Score is an instrument for predicting the 10-year risk of coronary heart disease or cardiovascular disease, based on American population-studies. The risk tool SCORE (Systematic Coronary Risk Evaluation) is a 10-year risk assessment system for fatal cardiovascular events in people 40–65 years of age [12, 13]. It is based on 12 European cohort studies and presents different national and regional risk calculation tables adapted to cardiovascular disease rate of each country. It classifies risk into different categories ranging from less than 1 to 15% or more and includes the variables gender, age, smoking, systolic blood pressure and total cholesterol. Patients with high risk (equal to or greater than 5%) are susceptible to more intensive prevention measures, including pharmacotherapy [12].

Therefore, we aimed to estimate the prevalence of CVRF in patients with SSHL and to determine the potential prognostic value of SCORE risk scale in the recovery after SSHL.

Materials and methods

Participants

The project was a prospective analysis of patients with SSHL admitted for protocol treatment in a tertiary university hospital, from January 2015 up to December 2017. This study received approval from the Ethics Committee of the institution. All patients experienced unilateral SSHL that developed within 72 h with ≥ 30 dB hearing loss at three consecutive frequencies. Patients were treated with the same protocol of intravenous dexamethasone (5–10 mg, twice daily, for 5 days) associated to intratympanic injection with dexamethasone in case of treatment failure or severe-profound hearing loss at admission. Intratympanic injection was administered weekly according to the response to treatment, at a maximum of 3 per patient. To prevent any gastric complication, pantoprazole 40 mg, was administered during therapy. Patients with any previous history of hearing loss or ear surgery on the affected side or HL developed more than 15 days before admission were excluded. Demographic, audiometric and clinical data were assessed.

Audiometric data

All patients were evaluated using the standard method for pure-tone average (PTA) threshold. The PTA threshold was calculated as the mean threshold at four frequencies (0.5, 1, 2, and 4 kHz). For thresholds that could not be tested due to the limit of the audiometric equipment, it was set at 120 dB in the present study.

Evaluation of hearing recovery

All patients underwent audiometry at admission, on day 3 and day 5 after the beginning of treatment and also on reevaluation on consultation (1–2 months after the admission).

Audiometric (dB HL) recovery was classified into complete, partial, and no recovery categories according to the following criteria [3]:

1. *Complete* PTA (dB HL) within 10 dB HL of initial HL or within 10 dB HL of the HL of the unaffected ear.
2. *Partial* PTA (dB HL) within 50% of initial HL or > 10 dB HL improvement of the HL.
3. *No recovery* < 10 dB HL improvement in HL relative to the initial HL.

Standard assessments

Standard assessments included routine audiometric testing and routine serological tests, including total cholesterol, high-density lipoprotein (HDL) cholesterol, blood pressure, weight and body mass index (BMI). It evaluated medical history of diabetes or hypertension, smoking, and any current medication for high blood pressure. It also assessed previous history of coronary heart disease and cerebrovascular disease. All patients underwent magnetic resonance imaging (MRI) of the internal auditory canal and cerebellopontine angle with gadolinium enhancement image during hospital stay. The SCORE risk scale was applied using the tables adapted to our country and patients were classified into the following risk categories: low risk ($< 1\%$), moderate risk ($\geq 1\%$ to $< 5\%$), high risk ($\geq 5\%$ to $< 10\%$) and very high risk ($\geq 10\%$). Audiometric recovery was compared among the different risk groups.

Statistical analyses

Statistical analysis was performed with the IBM SPSS Statistics program, 22nd version. Continuous variables are presented as mean \pm standard deviation and categorical variables as frequencies and percentages. Correlation between continuous variables was tested using Pearson or Spearman

correlation tests. Associations were tested for categorical variables using Chi square or Fisher's exact tests. Comparison between groups was tested, using independent *t* tests or ANOVA and Mann–Whitney or Kruskal Wallis tests for continuous variables. Post hoc tests were made using multiple pairwise comparisons with the Bonferroni correction for Kruskal–Wallis. Significance was settled for $p < 0.05$.

Results

Demographic characterization

There were 54 patients admitted for SSSL at our department during the study period.

Of these, 25 (46.3%) were male and 29 (53.7%) were female. The mean age of patients was 50.15 ± 14.59 years (minimum 18 years; maximum 82 years). The months of the year with higher admissions rates were April ($n = 11$) and August ($n = 10$). During the period of study, there was an increase in the hospital admissions due to SSSL and the mean hospital length of stay was 6.33 ± 1.49 days.

The mean time between the admission and the beginning of symptoms was 5.04 ± 3.98 days. Vertigo was present in 27.8% ($n = 15$) of patients at presentation. A summary of the clinical data collected is presented in Table 1.

Treatment protocol

Patients were administered dexamethasone in dosage of 10 mg/day ($n = 18$) or 20 mg/day ($n = 30$). Over 60% of patients ($n = 33$) underwent weekly intratympanic dexamethasone injection and the majority of these (70%) received three weekly injections.

Audiometric evaluation

The mean PTA of our sample was 77.84 ± 28.76 dB at admission, 71.74 ± 32.75 dB on day 3, 61.98 ± 35.75 dB on day 5 and 56.00 ± 33.90 dB on consultation. Considering all patients, complete recovery was observed in 35.9% ($n = 19$), partial recovery in 25.9% ($n = 14$) and no recovery in 38.9% ($n = 21$).

Cardiovascular risk factors assessment and SCORE calculation

The global prevalence of CVRF in our sample was 51.9% ($n = 28$). Nineteen patients presented hypertension, 23 patients had hyperlipidemia, 27 patients presented overweight or obesity, 5 patients had diabetes mellitus, 11 patients had smoking habits and 8 patients had history of previous cardiovascular event namely acute myocardial infarction and cerebrovascular accident. Remarkably, nine patients had inaugural diagnosis of hyperlipidemia (total cholesterol ≥ 200 mg/dL). PTA at admission was compared between the patients with and without CVRF. Patients with CVRF presented higher PTA at admission (83.79 ± 23.64 dB) than the patients without CVRF (71.43 ± 32.67 dB) although the differences were not statistically significant ($p > 0.05$). In addition, PTA at admission was analyzed considering each CVRF individually and despite the higher PTA at admission for patients with the CVRF, the differences were not significant ($p > 0.05$) (Table 2).

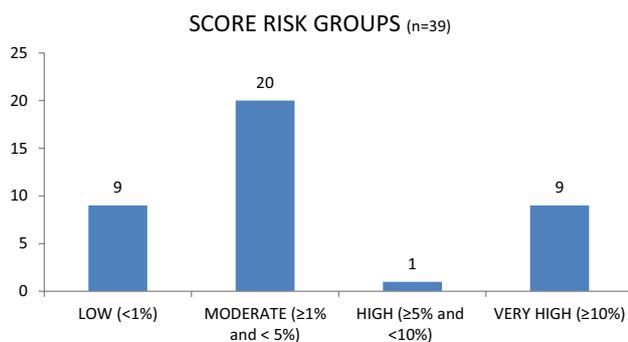
SCORE risk was calculated for 39 patients with ages between 40 and 65 years old: 9 patients presented low risk ($< 1\%$), 20 patients had moderate risk ($\geq 1\%$ and inferior to 5%), 1 patient had high risk ($\geq 5\%$ and inferior to 10%) and 9 patients had very high risk ($\leq 10\%$) (Fig. 1). There was a statistical significant hearing improvement in PTA level within each SCORE risk group from admission to the consultation

Table 1 Demographic features of patients admitted for SSSL, in 2015–2017 ($n = 54$)

Age (years)	50.15 ± 14.59		
Gender			
Female	53 (54.1%)		
Male	45 (45.9%)		
Hospital length of stay (days)	6.33 ± 1.49		
Time between the admission and the beginning of symptoms (days)	5.04 ± 3.98		
Year of hospital admission			
2015	13 (24.1%)		
2016	19 (35.2%)		
2017	22 (40.7%)		
Month, at the admission	January—2	May—1	September—4
	February—3	June—2	October—3
	March—7	July—5	November—5
	April—11	August—10	December—1

Table 2 Comparison of PTA at admission between groups with and without CVRF ($n=54$, unless stated otherwise)

CVRF	PTA at admission (dB)	<i>p</i> value
Hypertension		
Yes ($n=19$)	80.00 ± 25.39	> 0.05
No ($n=35$)	76.67 ± 30.72	
Hyperlipidemia		
Yes ($n=23$)	85.76 ± 24.10	> 0.05
No ($n=31$)	71.97 ± 30.85	
Diabetes mellitus		
Yes ($n=5$)	82.00 ± 23.86	> 0.05
No ($n=49$)	77.29 ± 29.69	
Overweight/obesity ($n=41$)		
Yes ($n=27$)	82.36 ± 30.16	> 0.05
No ($n=14$)	76.60 ± 31.78	
Smoking habits ($n=47$)		
Yes ($n=11$)	81.93 ± 28.29	> 0.05
No ($n=36$)	78.47 ± 29.97	

**Fig. 1** SCORE risk groups of patients admitted for SSHL, in 2015–2017

evaluation ($p < 0.05$) (Fig. 2). Patients with moderate and very high SCORE risk presented higher initial mean PTA thresholds at admission and also presented worse audiometric outcome after protocol treatment. However, these differences were not statistically significant ($p > 0.05$). In fact, patients with moderate risk presented higher PTA at admission than the very high-risk group (Table 3).

Imaging

All patients underwent MRI of the internal auditory canal and cerebellopontine angle with gadolinium enhancement image during hospital stay. Almost 50% ($n=27$) of our sample presented an abnormal MRI. The most common lesions were chronic cerebral microvascular ischemic changes ($n=15$), acoustic neuroma ($n=3$) and abnormal labyrinthine enhancement suggesting labyrinthitis ($n=4$). We

have analyzed the differences of the SCORE risk among the types of MRI changes (vascular, inflammatory or structural). In spite of the patients with evidence of chronic cerebral microvascular ischemic changes in the MRI having a trend to present a higher risk SCORE, there were no significant differences among groups (Table 4).

We have evaluated SCORE risk scale in our sample, excluding the patients with acoustic neuroma ($n=3$) and labyrinthitis ($n=4$) diagnosed with RMI: 7 patients presented low risk (< 1%), 17 patients had moderate risk (≥ 1% and inferior to 5%), 1 patient had high risk (≥ 5% and inferior to 10%) and 7 patients had very high risk (≥ 10%). Once again, we compared initial mean PTA thresholds at admission and audiometric outcome after protocol treatment between SCORE risk scale categories and there were no differences between groups ($p > 0.05$).

In addition, we have compared the audiometric recovery among the three groups of MRI changes (vascular, inflammatory or structural). Considering the 15 patients with vascular lesions in the MRI, only 4 of these presented a complete audiometric recovery, although these differences were not statistically significant ($p > 0.05$) (Table 4).

Discussion

We have analyzed the efficacy of our protocol, including systemic and intratympanic corticosteroids, in the hearing improvement after SSHL. Our overall complete recovery rate with SSHL was 35.9%. Around 26% of our patients experienced partial recovery. However, 38.9% of our patients had no recovery after treatment. Our results are in agreement with the literature that reports approximately one-third to two-thirds of patients with SSHL having potential of recovery some percentage of their hearing [5]. Furthermore, we have studied the potential effect of CVRF in the presentation and outcome of patients with SSHL. We have observed a high incidence of CVRF in our sample. Indeed, more than a half of our patients admitted for SSHL had at least one CVRF. Of these, overweight/obesity, hyperlipidemia and hypertension were the most common. Remarkably, nine patients had inaugural diagnosis of hyperlipidemia. This high incidence of incidental diagnosis has already been reported in the study of Haremza et al., which found hyperlipidemia in 45 patients (56.3%) with only 10 patients (12.5%) had been previously treated for this disorder [14]. We have applied the cardiovascular risk tool, SCORE, which is adapted to the European population and recommended for cardiovascular risk assessment by the European Society of Cardiology. The majority of the patients admitted for SSHL presented a moderate SCORE risk.

We have compared the PTA at admission and PTA recovery after treatment between patients with and without CVRF

Fig. 2 Comparison of audiometric recovery from the admission to the consultation within and between SCORE risk groups of patients admitted for SSHL, in 2015–2017

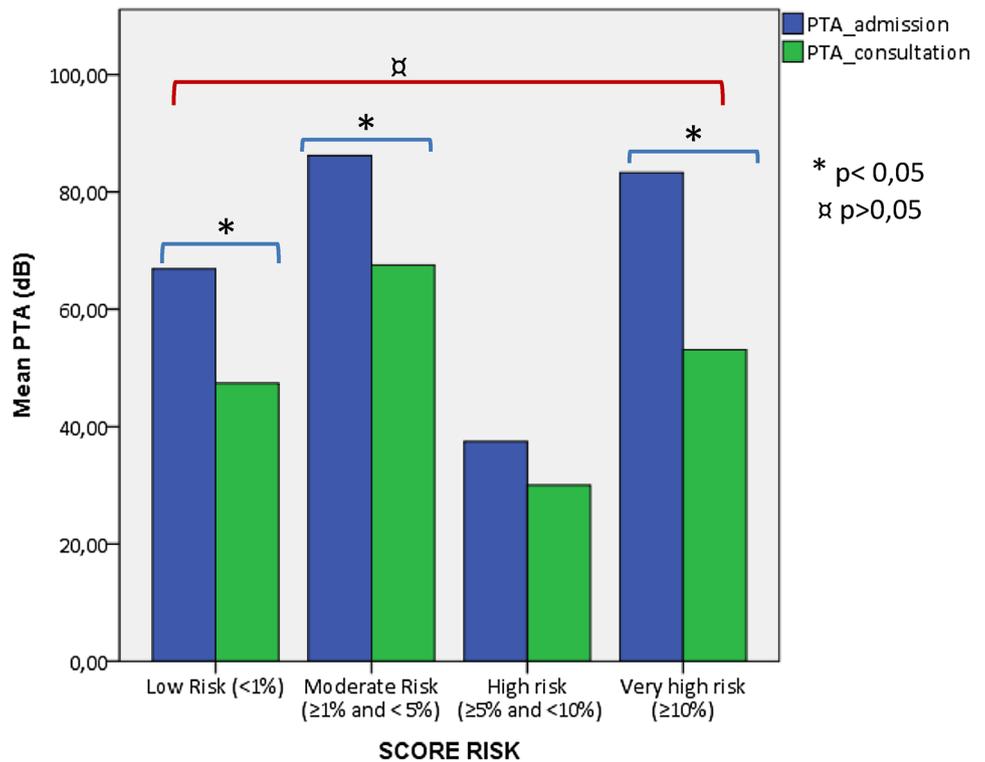


Table 3 Comparison between SCORE risk groups for patients admitted for SSHL, from 2015 to 2017 (n=39)

SCORE risk groups	Low risk (<1%) (n=9)	Moderate risk (≥1% and <5%) (n=20)	High risk (≥5% and <10%) (n=1)	Very high risk (≥10%) (n=9)	p value
Age (years)	48.33 ± 5.14	57.70 ± 9.95	66.00	62.44 ± 10.93	0.016
PTA at admission (dB)	68.89 ± 27.64	85.62 ± 27.04	37.5	85.62 ± 23.98	> 0.05
PTA at D3	63.06 ± 29.98	77.43 ± 33.36	37.50	82.50 ± 23.40	> 0.05
PTA at D5	59.06 ± 33.60	68.52 ± 36.40	37.50	78.47 ± 25.06	> 0.05
PTA at consultation	47.34 ± 37.06	67.50 ± 33.46	30.00	53.06 ± 21.24	> 0.05
Hearing recovery	No recovery—4 Partial—1 Complete—4	No recovery—9 Partial—6 Complete—5	Partial—1	No recovery—3 Partial—4 Complete—2	> 0.05

and also between patients of different SCORE categories. In spite of our patients with CVRF and higher SCORE risk categories presenting higher PTA at admission and also worse hearing outcome, SCORE system stratification of patients did not show any predictive or prognostic role in this study population. Nevertheless, there was a significant improvement of hearing within patients of the same risk category from admission to the consultation reevaluation. The clinical implication of this present study is undoubtedly limited by our small sample size of patients with CVRF who were eligible for SCORE calculation.

In the study of Chang et al., which assessed cardiovascular risk through the application of Framingham risk scale, high-risk group showed poor outcomes of hearing

improvement compared to the low-risk group in terms of mean PTA threshold [11]. All patients admitted for SSHL underwent MRI study. Notably, the most common finding was evidence of chronic cerebral microvascular ischemic changes. Furthermore, patients with evidence of microvascular ischemic changes in the MRI presented higher risk SCORE, although the differences were not significant. In addition, a smaller proportion of patients with vascular lesions in the MRI presented a complete audiometric recovery, although these differences were not statistically significant between the groups. Similarly, SSHL co-occurring with diabetes, hypertension, and hyperlipidemia in older patients has been shown to be associated with MRI

Table 4 Analysis of SCORE risk and audiometric recovery in patients admitted for SSHL with MRI abnormal findings

MRI findings	Vascular	Inflam- matory	Structural	<i>p</i> value
SCORE risk groups				
Low risk (<1%)	3	1	1	>0.05
Moderate risk (≥1% and <5%)	6	2	1	>0.05
Very high risk (≥10%)	6	0	2	>0.05
Total	15	3	4	
Audiometric recovery				
No recovery	6	3	3	>0.05
Partial	6	0	1	>0.05
Complete	4	1	2	>0.05
Total	16	4	6	

evidence of cerebral microangiopathy and prognosis, but the association's clinical significance is unclear [3].

It has been previously reported that risk factors for ischemic vascular disease such as cigarette smoking, hypertension, hyperlipidemia, and diabetes are risk factors for the development of SSHL [8, 15]. However, there is a lack of a clear relationship between SSHL and CVRF, with many heterogeneous and controversial studies published in the medical literature. Ullrich et al. in a prospective study of 35 patients found that the frequency of CVRF (male gender, smoking, obesity and hyperlipidemia) was identical in the general population and in patients with sudden SSHL [16]. Similarly, Ballesteros et al. in a study of 99 patients, failed to demonstrate any clear relationships between SSHL and CVRF [17]. Furthermore, a recent systematic review failed to show any association between serum lipids and SSHL [18]. The study of Haremza et al. evaluated the differences between patients with complete (defined as profound deafness, and/or inability to correctly repeat any words when performing speech audiometry, regardless of bone conduction) versus partial sudden sensorineural hearing loss [14]. No statistically significant difference was observed between patients with complete versus partial sudden sensorineural hearing loss ($p = 0.0708$) concerning the cardiovascular risk factors. However, the same study, reported a statistical tendency in favor of CVRF in the group with complete hearing loss [14].

All studies previously mentioned evaluating CVRF and SSHL are particularly limited by sample size and their observational nature. The lack of a clear relationship between sudden SSHL and CVRF might suggest a predominantly multifactorial disease profile, regardless of hearing impairment severity [14]. Despite all this, we could report a high incidence of CVRF in our sample. In addition, the most common finding in the MRI study was evidence of chronic cerebral microvascular ischemic changes. Indeed,

the observations made in this preliminary study might shed light on possible etiological factors for SSHL patients with multiple comorbidities, although, we could not confirm the prognostic role of SCORE risk scale.

Conclusion

This preliminary study could not confirm the validity for SCORE scale for cardiovascular risk assessment in predicting audiometric recovery in patients with SSHL with multiple comorbidities. Further research with larger samples are needed to elucidate the etiology of SSHL and the exact role of cardiovascular risk factors in the pathophysiology of SSHL.

Author contributions ASM takes full responsibility for the integrity of the data presented. AL contributed to the collection of data. All the other authors contributed to the acquisition, analysis and interpretation of the data for the work. DR and DM had full access to all data and made substantial contributions to the analysis and interpretation of data. JG and LD were also responsible for the study supervision. ASM was responsible for the drafting of the manuscript and all the authors for revising it for important intellectual content.

Funding The authors did not receive any specific financial or material support from agencies from the public sector, commercial sector or non-profit entities.

Compliance with ethical standards

Conflict of interest All the authors declare that they have read and understood the policy on declaration of interests of this magazine and have no competing interests.

References

1. Wu CS, Lin HC, Chao PZ (2006) Sudden sensorineural hearing loss: evidence from Taiwan. *Audiol Neurootol* 11(3):151–156
2. Nosrati-Zarenoe R, Arlinger S, Hulcrantz E (2007) Idiopathic sudden sensorineural hearing loss: results drawn from the Swedish national database. *Acta Otolaryngol* 127(11):1168–1175
3. Stachler RJ et al (2012) Clinical practice guideline: sudden hearing loss. *Otolaryngol Head Neck Surg* 146(1S):S1–S35
4. Conlin AE, Parnes LS (2007) Treatment of sudden sensorineural hearing loss, I: a systematic review. *Arch Otolaryngol Head Neck Surg* 133(6):573–581
5. Mattox DE, Simmons FB (1977) Natural history of sudden sensorineural hearing loss. *Ann Otol Rhinol Laryngol* 86(4 Pt 1):463–480
6. Fetterman BL, Saunders JE, Luxford WM (1996) Prognosis and treatment of sudden sensorineural hearing loss. *Am J Otol* 17(4):529–536
7. Haynes DS, O'Malley M, Cohen S, Watford K, Labadie RF (2007) Intratympanic dexamethasone for sudden sensorineural hearing loss after failure of systemic therapy. *Laryngoscope* 117(1):3–15

8. Capaccio P, Ottaviani F, Cuccarini V et al (2007) Genetic and acquired prothrombotic risk factors and sudden hearing loss. *Laryngoscope* 117(3):547–551
9. Aimoni C, Bianchini C, Borin M et al (2010) Diabetes, cardiovascular risk factors and idiopathic sudden sensorineural hearing loss: a case–control study. *Audiol Neurootol* 15(2):111–115
10. Arjun D, Neha G, Surinder KS, Ravi K (2015) Sudden sensorineural hearing loss; prognostic factors. *Iran J Otorhinolaryngol* 27(82):355–359
11. Chang YS et al (2017) Framingham risk score as a prognostic predictor of sudden sensorineural hearing loss: a preliminary study. *Ann Otol Rhinol Laryngol* 126:382–387
12. Conroy RM, Pyörälä K, Fitzgerald AP, Sans S, Menotti A, De Backer G et al (2003) Estimation of ten-year risk of fatal cardiovascular disease in Europe: the SCORE project. *Eur Heart J* 24(11):987–1003
13. Perk J, De Backer G, Gohlke H et al (2012) European Guidelines on cardiovascular disease prevention in clinical practice (version 2012): the Fifth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice. *Eur Heart J* 33:1635–1701
14. Haremza C, Klopp-Dutote N, Strunski V, Page C (2017) Evaluation of cardiovascular risks and recovery of idiopathic sudden sensorineural hearing loss in hospitalised patients: comparison between complete and partial sudden sensorineural hearing loss. *J Laryngol Otol* 131(10):919–924
15. Chau JK, Lin JR, Atashband S, Irvine RA, Westerberg BD (2010) Systematic review of the evidence for the etiology of adult sudden sensorineural hearing loss. *Laryngoscope* 120(5):1011–1021
16. Ullrich D, Aurbach G, Drobik C (1992) A prospective study of hyperlipidemia as a pathogenic factor in sudden hearing loss. *Eur Arch Otorhinolaryngol* 249:273–276
17. Ballesteros F, Alobid I, Tassies D, Reverter JC, Scharf RE, Guilemany JM et al (2009) Is there an overlap between sudden neurosensorial hearing loss and cardiovascular risk factors? *Audiol Neurootol* 14:139–145
18. Chang IJ, Kang CJ, Yueh CY, Fang KH, Yeh RM, Tsai YT (2015) The relationship between serum lipids and sudden sensorineural hearing loss: a systematic review and meta-analysis. *PLoS One* 10(4):e0121025

Publisher's Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.