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MAIN ARTICLE

Evaluation of cardiovascular risks and recovery of idiopathic sudden sensorineural hearing loss in hospitalised patients: comparison between complete and partial sudden sensorineural hearing loss

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Abstract

Objective: To evaluate the presence of cardiovascular risk factors and recovery of idiopathic sudden sensorineural hearing loss in hospitalised patients.

Methods: A single-centre retrospective study of 80 patients hospitalised for idiopathic sudden sensorineural hearing loss was conducted over a 6-year period. Mean pure tone hearing thresholds were assessed by pure tone audiometry.

Results: Twenty-three of 80 patients (28.75 per cent) initially had no cardiovascular risk factors. Forty-five patients had hyperlipidaemia, 22 patients had hypertension, 7 patients had diabetes mellitus and 7 patients were obese. No statistically significant difference was observed between patients with complete versus partial sudden sensorineural hearing loss ($p = 0.0708$) concerning the cardiovascular risk factors. At long-term follow up, the hearing recovery rate was not significantly different between the two groups of patients ($p = 0.7541$).

Conclusion: The lack of a clear relationship between idiopathic sudden sensorineural hearing loss and cardiovascular risk factors suggests that sudden sensorineural hearing loss has a predominantly multifactorial disease profile regardless of hearing impairment severity.

Key words: Sensorineural Hearing Loss; Hypertension; Diabetes Mellitus; Hyperlipidemia

Introduction

Sudden hearing loss is defined as the rapid onset, within 72 hours, of a subjective sensation of hearing impairment in one or both ears. Sudden sensorineural hearing loss (SNHL) is a subset of sudden hearing loss, with sensorineural features and meeting certain audiometric criteria (i.e. hearing loss of 30 dB or more, affecting at least three consecutive frequencies). Idiopathic sudden SNHL is defined as sudden SNHL with no identifiable cause, despite adequate investigation.[1–6]

Sudden SNHL is a common clinical finding, with an estimated worldwide annual incidence of 5–20 cases per 100 000 inhabitants.[1–3] Sudden SNHL usually occurs in adults, and is essentially unilateral; bilateral sudden SNHL is also possible but is rare.[1–6]

Sudden SNHL is usually considered to be a medical emergency, requiring rapid treatment and often a short hospital stay.[1–6] However, some authors have shown that the delayed initiation of treatment does not appear to influence the final degree of hearing loss, suggesting that sudden SNHL should no longer be considered to be an otological emergency.[5] Nevertheless, the aetiology of sudden SNHL remains unknown.

The postulated pathophysiology concerns four theoretical pathways, including: labyrinthine viral infection, labyrinthine vascular compromise, intracochlear membrane rupture and immune-mediated inner-ear disease.[1–6] Notably, many studies have tried to identify a relationship between sudden SNHL and cardiovascular diseases which might have an impact on the severity of sudden hearing loss and its recovery, but sometimes with contradictory conclusions.[7–17]

The present study was designed to evaluate cardiovascular risk factors and hearing recovery in patients hospitalised for sudden SNHL, and particularly to compare total and partial SNHL. The primary objective was to confirm (or exclude) the vascular hypothesis, particularly for the development of sudden SNHL.

Materials and methods

This single-centre, retrospective study was conducted over a six-year period, following approval by the

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Amiens University Hospital's Institutional Review Board (France).

The study comprised 80 patients who were diagnosed with idiopathic sudden SNHL and hospitalised in the ENT and Head and Neck Surgery Department of the Amiens University Hospital, France, from 1 January 2007 to 31 December 2012. Patients with a clearly identified specific aetiology (i.e. head injury, vestibular schwannoma, Ménière's disease, infectious labyrinthitis and so on) were excluded from the study. Sudden SNHL was confirmed by pure tone audiometry and speech audiometry.

Every patient underwent an audiological evaluation in a sound-treated booth. The pure tone average (PTA) was calculated from the results of bone conduction at 0.5, 1, 2 and 4 kHz. The initial hearing threshold was calculated (for the idiopathic sudden SNHL ear and the contralateral 'control ear') using the mean of the four frequencies. The speech stimuli were based on the Fournier lists of spondaic words.

Patients were divided into two groups: patients with complete sudden SNHL (group A) and patients with partial sudden SNHL (group B). Complete sudden SNHL was defined when patients had an initial hearing threshold equal to or below 90 dB (profound deafness) and/or were unable to correctly repeat any words when performing speech audiometry, regardless of bone conduction.

All patients were hospitalised for diagnostic investigation and therapeutic management. Audiometry and videonystagmography were performed on all patients on days 0, 2 and 5. Inner-ear and cerebellopontine angle magnetic resonance imaging was performed in all patients, sometimes during hospitalisation but often several weeks after discharge from hospital, essentially to identify a vestibular schwannoma. The following parameters were also recorded for all patients: body mass index, repeated blood pressure measurements, complete blood count, serum electrolyte levels, glycaemia, serum total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol and triglyceride levels.

All patients received intravenous methylprednisolone (1 mg/kg/day) and intravenous piracetam (9 g/day) over 5 days. After discharge from hospital, no medication was indicated.

Follow-up audiometry was performed on day 21, day 45, month 3, month 6 and up to 1 year in the case of partial recovery. The maximum recovery time was one year after the onset of sudden SNHL. Three parameters were considered to evaluate recovery after treatment, as follows. First, the absolute hearing gain on the deaf ear, defined as: decibel values from initial four-frequency PTA minus decibel values from the final four-frequency PTA on the last audiogram performed. Second, the percentage of hearing recovery, calculated as: decibel values from initial four-frequency PTA minus decibel values from final four-frequency PTA, divided by decibel values from the initial four-frequency PTA. Third, the global improved hearing gain, calculated as: (absolute hearing gain on the deaf ear divided by the initial four-frequency PTA of the contralateral ear minus the four-frequency PTA on the last audiogram) × 100 per cent.

Recovery was considered to be complete when the final four-frequency PTA was less than or equal to 10 dB compared to the initial PTA on a previous audiogram (before the onset of sudden SNHL) or compared to the contralateral ear (considered to be the control ear). Recovery was considered to be partial when the percentage recovery and/or global improved hearing gain was greater than or equal to 50 per cent. When the percentage recovery and/or global improved hearing gain was less than 50 per cent, patients were considered to show no recovery. As the hearing level of the contralateral ear does affect the global improved hearing gain, several patients may have obtained a global improved hearing gain greater than 100 per cent.

Based on the criteria of the European Guidelines on Cardiovascular Disease Prevention in Clinical Practice (version 2012), the following cardiovascular risk factors were taken into account in this study: hypertension, hyperlipidaemia, diabetes mellitus, obesity and smoking.[18] Patients were considered to have hypertension when they were already being treated for hypertension, or when at least two blood pressure readings were higher than 140/90 mmHg. Patients were considered to have hyperlipidaemia when they were already being treated for hyperlipidaemia, or when serum total cholesterol was higher than 200 mg/dl and/or low-density lipoprotein cholesterol was higher than 130 mg/dl and/or high-density lipoprotein cholesterol was less than 45 mg/dl and/or serum triglyceride levels were higher than 150 mg/dl. Patients were considered to have diabetes mellitus (type I or II) when they were already being treated for diabetes mellitus or when glycaemia was higher than 126 mg/dl. Obesity was defined by a body mass index greater than 30 kg/m^2.[18]

Between-group comparisons were performed with: a chi-square test (or Fisher's test) for categorical variables, and a student's *t*-test or Mann–Whitney U test for continuous variables. Statistical significance on univariate analysis was defined as $\alpha = 0.05$.

Results

Eighty patients (39 males and 41 females) were included in the study, with a mean age of 56 years (range, 22–80 years). Patient data and clinical data are presented in Table I.

No statistically significant differences in terms of patient and clinical data were observed between the two groups of patients (complete *vs* partial sudden SNHL), except for the presence of vertigo which was more frequent in group A (complete sudden SNHL patients) ($p = 0.0198$).

The cardiovascular risk factors identified in the patients are shown in Tables II and III.

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TABLE I
POPULATION AND CLINICAL DATA

Parameter	Group A (complete sudden SNHL)	Group B (partial sudden SNHL)	*p*
Patients (*n*)	28	52	0.4391
Age (mean ± SD; years)	53.2 ± 15	53.3 ± 15.1	0.9775
Time to management (mean ± SD; days)	4.9 ± 7.5	5.7 ± 9.9	0.1253
Hospital stay (mean ± SD; days)	5.6 ± 1	5.3 ± 1.4	0.3494
Tinnitus (*n* (%))	18 (64.29)	37 (71.15)	0.5225
Vertigo (*n* (%))	12 (42.86)	9 (17.31)	0.0198*

*Indicates statistical significance. SNHL = sensorineural hearing loss; SD = standard deviation

TABLE II
CARDIOVASCULAR RISK FACTORS

Cardiovascular risk factor	Group A (complete sudden SNHL)*	Group B (partial sudden SNHL)†	*p*‡
Diabetes mellitus	2 (7.14)	5 (9.6)	0.3276
Hyperlipidaemia	13 (46.4)	32 (61.5)	0.3309
Hypertension	7 (25)	15 (28.8)	0.7133
Obesity	0	7 (13.5)	0.0875

Data represent numbers (and percentages) of patients with cardiovascular risk factors, unless indicated otherwise. *$n = 28$; †$n = 52$. ‡None of the *p* values were statistically significant (all $p > 0.05$). SNHL = sensorineural hearing loss

TABLE III
NUMBER OF CARDIOVASCULAR RISK FACTORS PER PATIENT BY GROUP

Cardiovascular risk factors per patient (*n*)	Group A (complete sudden SNHL)*	Group B (partial sudden SNHL)†
0	10 (35.7)	13 (25)
1	12 (42.8)	21 (40.3)
2	6 (21.4)	10 (19.2)
3	0 (0)	6 (11.5)
4	0 (0)	2 (3.8)

Data represent numbers (and percentages) of patients. *$n = 28$; †$n = 52$. SNHL = sensorineural hearing loss

There were no statistically significant differences in cardiovascular risk factors between the two groups; however, there was a statistical tendency in favour of cardiovascular risk factors in group A patients, with a *p* value of 0.0708 (odds ratio = 0.620; 95 per cent confidence interval = 0.369–1.041).

During hospitalisation for sudden SNHL, hyperlipidaemia was diagnosed in 45 patients (56.3 per cent), and hypertension was diagnosed in 2 patients (2.5 per cent) in whom this diagnosis was previously unknown (anti-hypertensive treatment was prescribed after discharge from the hospital).

In group A, complete recovery was observed in three patients (11 per cent) and partial recovery was observed in seven patients (25 per cent). In group B, complete recovery was observed in 12 patients (23 per cent) and partial recovery was observed in 11 patients (21 per cent). Overall, at long-term follow up (up to one year after idiopathic sudden SNHL), no statistically significant difference was observed between the two groups of patients in terms of complete or partial recovery (Table IV).

Discussion

Numerous heterogeneous studies on sudden SNHL and general cardiovascular diseases have been published in the medical literature, and their conclusions are variable and sometimes controversial.[7–17]

In a prospective series of 35 patients, Ullrich *et al.* found that the frequency of cardiovascular risk factors (male gender, smoking, obesity and hyperlipidaemia) was identical in the general population and in patients with sudden SNHL.[7] Similarly, Ballesteros *et al.*, in a study of 99 patients, failed to demonstrate any clear relationships between sudden SNHL and cardiovascular risk factors.[8]

In contrast, many studies suggest that sudden SNHL is related to cardiovascular diseases. Rudack *et al.*, in a case–control study of 142 sudden SNHL patients versus 84 control participants, showed that hypercholesterolaemia and hypoalphalipoproteinaemia (reduced high-density lipoprotein cholesterol levels) did not appear to be major risk factors for sudden SNHL, whereas GPIa C807 T polymorphism, elevated fibrinogen levels and smoking were associated with an increased risk for sudden SNHL.[9] Aimoni *et al.*, in a case–control study of 141 sudden SNHL patients and 271 control patients, suggested that diabetes mellitus, hypercholesterolaemia and a high burden of cardiovascular risk factors were associated with an increased risk of sudden SNHL.[10] Ciccone *et al.*, in a study comparing 29 sudden SNHL patients and 29 healthy controls, showed that sudden SNHL seemed to be associated with vascular endothelial dysfunction and an increased cardiovascular risk, which supports the hypothesis of a vascular aetiology for this disease.[11]

Teranishi *et al.* reported the results of nationwide epidemiological surveys on sudden deafness in Japan, which showed that patients with sudden SNHL more frequently had hypertension and diabetes mellitus (in the fourth survey (2001) compared to other surveys, notably the first survey (1972)), particularly among elderly patients.[12] A large-scale study by Lin *et al.* (involving 1423 sudden SNHL patients and 5692

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TABLE IV
AUDIOLOGICAL DATA

Parameter	Group A (complete sudden SNHL)*	Group B (partial sudden SNHL)†	*p*
Initial Δ4PTA (mean ± SD; dB)			
– Idiopathic sudden SNHL ear	97.9 ± 15.6	58.5 ± 12.3	
– Contralateral ear	24.2 ± 20.5	28.4 ± 25.4	
Δ4PTA after treatment (mean ± SD; dB)			
– Idiopathic sudden SNHL ear	92 ± 21.9	41.5 ± 17.7	
– Contralateral ear	22.6 ± 19.4	27.5 ± 24.9	
Hearing recovery after treatment			<0.0001‡
– Percentage (± SD) of hearing recovery	5.1 ± 22.5	29.2 ± 26.1	
– Number of patients with >50% recovery	2/28	13/52	
Global improved hearing gain after treatment			0.0003‡
– Percentage (± SD) of improved gain	0.78 ± 35.9	43.1 ± 64.2	
– Number of patients with >50% improved gain	2/28	24/52	
Final Δ4PTA at long-term follow up (mean ± SD; dB)			
– Idiopathic sudden SNHL ear	70.3 ± 35.1	41.7 ± 22.5	
– Contralateral ear	26 ± 22.1	30.1 ± 26.9	
Hearing recovery at long-term follow up			0.7925
– Percentage (± SD) of hearing recovery	31.4 ± 17.9	29.1 ± 18.4	
– Number of patients with >50% recovery	9/24	13/43	
Global improved hearing gain at long-term follow up			0.7541
– Percentage (± SD) of improved gain	40.2 ± 20.6	45.3 ± 23.1	
– Number of patients with >50% improved gain	10/24	23/43	

The 'after treatment' values were collected on days 5–21. *Of the 28 patients in this group, 4 were lost to follow up, and there are no long-term follow-up data for these patients. †Of the 52 patients in this group, 9 were lost to follow up, and there are no long-term follow-up data for these patients. ‡Indicates statistical significance. SNHL = sensorineural hearing loss; Δ4PTA = four-frequency pure tone average threshold; SD = standard deviation

'control' patients hospitalised for appendectomy) suggested that sudden SNHL could be an early warning sign of impending stroke, and indicated that sudden SNHL should be considered a vascular disease.[13]

The majority of published studies concern diabetes mellitus (type I or II) and sudden SNHL.[10,14–17] Nishio *et al.*, in a case–control study (involving 72 sudden SNHL patients and 2161 control patients), concluded that CFH Y402H polymorphism and sudden SNHL risk were significantly related, and that diabetic CFH Y402H minor allele carriers may be more susceptible to sudden SNHL.[14] This study also showed that diabetes mellitus alone was related to sudden SNHL, but not hyperlipidaemia or hypertension.[14]

Rust *et al.*, in an animal study (involving congenic spontaneous hypertensive/National Institutes of Health (Bethesda, Maryland, USA)-corpulent (SHR/N-cp) rats, which is a model for non-insulin-dependent diabetes mellitus) showed a relationship between non-insulin-dependent diabetes mellitus and inner-ear damage.[15] The authors suggested that outer hair cell loss could be related to hyperglycaemia and a genetic predisposition for glucose intolerance.[15]

Aimoni *et al.* showed that diabetes mellitus was more frequent in the sudden SNHL group (141 patients, 15.6 per cent) than in the control group (271 patients, 8.5 per cent) ($p = 0.03$), which is consistent, as diabetes mellitus plays a major role in the development of microangiopathy.[10]

In a retrospective study of 67 diabetes mellitus patients with sudden SNHL, Weng *et al.* suggested that these patients commonly had hearing loss in the contralateral ear and profound hearing loss in the affected ear.[16] Age and postprandial plasma glucose level were significantly correlated with contralateral hearing loss. The poor prognosis of sudden SNHL in diabetes mellitus patients might be related to pre-existing microvascular lesions in the inner ear, and postprandial plasma glucose level could be a risk factor indicator for cochlear dysfunction in diabetic patients.[16]

Finally, in a retrospective study of 148 sudden SNHL patients, Fukui *et al.* showed that 24 patients with sudden SNHL (16 per cent) had type 2 diabetes mellitus, and this subgroup was associated with more severe hearing loss.[17]

In our study, only 4 patients had type I diabetes mellitus and only 3 patients had type II diabetes mellitus out of a total of 80 patients (i.e. 8.8 per cent of cases), which is a relatively low percentage. This is possibly the result of a recruitment bias, as this study exclusively concerned hospitalised patients.

Few studies on sudden SNHL and hyperlipidaemia have been published in the literature. Only Ciccone *et al.* showed that plasma total cholesterol and low-density lipoprotein cholesterol were significantly higher in sudden SNHL patients than in control patients (188 ± 33 mg/dl *vs* 171 ± 29 mg/dl, $p < 0.05$).[11] However, there was no statistically significant difference between the two groups of patients concerning other cardiovascular risk factors.[11] In contrast, Ullrich *et al.* showed no association between hyperlipidaemia and sudden SNHL.[7]

Hyperlipidaemia was diagnosed in 45 patients in our series (56.3 per cent), and only 10 patients (12.5 per cent) had been previously treated for this disorder. This finding should probably be taken into account,

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but more studies are needed to demonstrate a significant correlation between hyperlipidaemia and sudden SNHL.

No study in the literature has clearly demonstrated a relationship between hypertension and sudden SNHL. Only Lionello *et al*., who recently reported a series of 117 sudden SNHL patients, showed, on univariate analysis, that hypertension was significantly related to hearing outcome ($p = 0.015$).[2] In our series, 22 patients (27.5 per cent) had hypertension, and 2 cases of hypertension were discovered during hospitalisation for sudden SNHL in patients in whom this diagnosis was previously unknown.

Concerning the prognosis of hearing recovery, 32–65 per cent of sudden SNHL patients may recover spontaneously, but SNHL classically has a poor prognosis.[3–5] Early recovery, particularly during or immediately after treatment, is a good indicator for final recovery, regardless of hearing loss severity.[3–5] Furthermore, patients with no recovery during the first two to three weeks have a poor prognosis of recovery.[3–5] However, the concept of recovery must be clearly defined. A definition of recovery based on absolute values may not be entirely appropriate, as it depends on the initial hearing impairment (i.e. a 30 dB hearing gain does not have the same significance for an initial hearing loss of 30 dB or 80 dB), which is why we did not use this method to rate hearing recovery in our patients except in cases of complete recovery. Our data concerning sensorineural hearing loss are consistent with data in the literature, as partial or complete hearing recovery rates were initially poorer than in cases of partial sudden SNHL, but not at long-term follow up.

- **Numerous heterogeneous studies on sudden sensorineural hearing loss (SNHL) and cardiovascular diseases have been published**
- **The relationship between sudden SNHL and cardiovascular risk factors remains unclear**
- **Sudden SNHL might reveal previously unknown hyperlipidaemia or hypertension**

The primary objective of this study was to confirm (or exclude) a vascular cause for sudden SNHL, particularly complete sudden SNHL, based on the hypothesis that sudden SNHL may occur more frequently in 'vascular patients', and these patients may have a poorer prognosis of recovery because of the severity of sensory impairment and the presumed presence of cardiovascular risk factors. Although our study failed to support these hypotheses statistically (possibly because of the relatively small sample size), a statistical trend was observed in favour of the presence of more cardiovascular risk factors in patients with sudden SNHL. The episodes of sudden SNHL also revealed previously unknown hyperlipidaemia in 24 patients, which, in our opinion, may constitute a valuable finding. However, at long-term follow up (up to one year after sudden SNHL), the prognosis of hearing recovery was identical for patients with complete and for those with partial sudden SNHL.

The principal bias of our study was that it exclusively concerned hospitalised patients, and not all patients managed in our department between 2007 and 2012 for sudden SNHL. A study conducted by Huy and Sauvaget in France (published in 2005) suggested that sudden SNHL was no longer an emergency; the article stated that many ENT departments or physicians no longer systematically hospitalise patients for sudden SNHL, especially in the presence of mild or moderate hearing impairment.[6] The number of patients not included in this hospital population and the possible impact on statistical analysis are unknown.

Conclusion

The lack of a clear relationship between sudden SNHL and cardiovascular risk factors suggests a predominantly multifactorial disease profile, regardless of hearing impairment severity. However, as idiopathic sudden SNHL can be considered an early 'vascular symptom', a vascular disease investigation should be performed in all sudden SNHL patients. Sudden SNHL may reveal previously unknown cardiovascular risk factors, which could have important implications for therapeutic and preventive strategies. Further larger studies are necessary to determine the exact role of cardiovascular risk factors in the pathophysiology of sudden SNHL.

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