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Sensorineural hearing loss in stroke: an age-matched retrospective study

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Abstract

Objective. To compare characteristics between stroke populations with and without sensorineural hearing loss (SNHL) and assess the impact of SNHL on stroke outcome.
Methods. A retrospective study of patients admitted with stroke was carried out. Patients were divided into two groups, where group A were diagnosed with SNHL and group B were without SNHL. Baseline age, gender, vascular risk factors and disability were compared. Logistic regression analyses were performed with three-month mortality and SNHL as dependent variables.
Results. A total of 631 admissions were reviewed, with mean age 79.2 years, including 305 patients with SNHL and 326 without. More severe disability was more prevalent in patients with SNHL. Sensorineural hearing loss was not associated with increased mortality (odds ratio = 1.1, 95 per cent confidence interval = 0.7–2.0, $p = 0.668$). Hypertension was present in 210 (68.9 per cent) with SNHL versus 189 (58 per cent) without SNHL ($p = 0.005$). Small-vessel disease aetiology was more prevalent in SNHL 51 (16.7 per cent) versus 30 (9.2 per cent) without SNHL ($p = 0.005$).
Conclusion. Sensorineural hearing loss appears to have an association with stroke of small-vessel disease aetiology and hypertension. Sensorineural hearing loss does not affect three-month mortality but is associated with increased disability.

Introduction

Hearing loss is a common problem worldwide, with increasing prevalence in the aging population.[1] Sensorineural hearing loss (SNHL) has multiple causes, but one potentially important cause is ischaemia. Sudden SNHL can be an important sign in anterior inferior cerebellar artery infarction confirmed by middle cerebellar peduncle infarct on magnetic resonance imaging.[2]

A large meta-analysis suggested people with SNHL face increased risk of stroke,[3] but there are suspected differences between sudden-onset SNHL and gradual development of hearing loss in terms of ischaemic aetiology.[4,5] The exact mechanisms connecting stroke and SNHL are unknown, but such knowledge may be of relevance for stroke management.

Hearing loss has been associated with increased frailty and falls in older people,[6] has a negative impact on quality of life and could affect stroke rehabilitation. People with disabilities have been underrepresented in clinical trials and this may include studies of stroke assessment grading systems. There is ongoing prejudice and negativity surrounding disability that may impact clinical outcomes,[7] specifically relating to implementation of ceilings of care or intensity of rehabilitation therapy and treatment goals.

Given the current gaps in knowledge of the potential significance of known SNHL in patients with stroke, this study aimed to determine whether the presence of SNHL was linked to stroke outcomes using retrospective, age-matched settings.

Material and methods

Study settings and patient selection

A retrospective analysis was carried out in a tertiary referral university centre with a catchment of 1.2 million. The study was approved by the Research Committee Board and Caldicott's guardian approval was obtained.

We reviewed all admissions who had a diagnosis of acute stroke between 1 January 2017 and 31 December 2017. The patients were organised into two groups: one group included all patients with acute ischaemic stroke and known SNHL diagnosed by the Otolaryngology and Audiology Service within the previous eight years, and the other group was derived from the cohort who had acute ischaemic stroke without known hearing impairment. This second, control cohort were age-matched using SPSS version 27 allowing a tolerance of one year each way to minimise this clear potential confounder. Centralisation of the otolaryngology service ensured capture of all patients with SNHL treated within NHS Greater Glasgow and Clyde Health Board in the eight-year period before acute ischaemic stroke.

Included data

We recorded demographics, age, sex and risk factors for stroke, namely hypertension, ischaemic heart disease, smoking history, diabetes mellitus, atrial fibrillation,

Main Article

Sam Neilson takes responsibility for the integrity of the content of the paper

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hyperlipidaemia, peripheral vascular disease and previous stroke. The National Institutes of Health Stroke Scale, designed to measure stroke severity in acute stroke,[8] was recorded as well as modified Rankin scale pre-morbid disability estimation.[9,10] This was based on admission documentation and initial specialist physiotherapy and occupational therapy assessments.

The TOAST classification of stroke aetiology derived from the Trial of Org 10172 in Acute Stroke Treatment[11] was recorded and includes different categories. Large-vessel stroke includes those caused by carotid artery stenosis of greater than 50 per cent, and cardioembolic aetiology includes atrial fibrillation, small-vessel disease caused by occlusion of perforator branch arteries, other category and undetermined or dual pathologies. This was ascertained by reviewing clinical investigations, including cardiac and imaging reports and discharge letters completed by the responsible stroke physicians. Death at three months was recorded as an outcome.

Statistical analysis

Group comparisons were performed using student's *t*-test for normally distributed continuous variables and the chi-square test or Fisher's exact test for categorical variables. We performed binary logistic regression analysis using patient demographics and identified risk factors and stroke characteristics that differed between the two groups to establish which were independently associated with SNHL. We performed binary logistic regression analysis with three-month mortality as a dependent variable using known risks for stroke mortality[12] and factors differing between the two groups as covariates.

Results and analysis

The cohort of people with SNHL and acute ischaemic stroke consisted of 305 patients while the control cohort had 326 patients with acute ischaemic stroke. The mean age of the entire cohort of 631 participants was 79.2 years (standard deviation = 10.4 years). Baseline characteristics are presented in Table 1.

Differing distributions of modified Rankin scale subgroups are shown in Figure 1. The distribution of stroke aetiology by TOAST criteria differed between groups. The vascular risk factors hypertension ($p = 0.005$) and previous stroke ($p = 0.017$) were more common in the SNHL group than in the control group.

Using binary logistic regression, adjusted for age, smoking, hyperlipidaemia, previous stroke and disability score, and TOAST classification, hypertension was significantly associated with SNHL (odds ratio = 1.5, 95 per cent confidence interval (CI) = 1.0–2.1, $p = 0.034$). Small-vessel disease stroke aetiology was also associated with SNHL (odds ratio = 2.4, 95 per cent CI = 1.3–4.2, $p = 0.003$).

Three-month mortality was similar for the SNHL group (22 per cent) and the control group (18.7 per cent) ($p = 0.310$). The binary logistic regression analysis adjusting for compounding factors revealed that hearing impairment had no impact on mortality at three months (odds ratio = 1.1, 95 per cent CI = 0.7–2.0, $p = 0.668$) (Table 2).

Discussion

Main findings

We performed this retrospective study to analyse differences between two cohorts of people who had an ischaemic stroke with and without SNHL. We used an age-matching sampling technique to minimise this clear confounder. The main finding from the study was that known SNHL is not independently associated with increased or decreased mortality at three months after acute ischaemic stroke (Table 1). Interestingly, however, baseline disability measured by the modified Rankin scale was distributed towards higher disability in patients with SNHL. The baseline risk factors more prevalent in the SNHL group were hypertension and previous stroke. It is not clear whether these factors alone would explain the difference in baseline disability. Importantly, the stroke aetiology more commonly associated with SNHL was small-vessel disease, which is consistent with an underlying vascular aetiology as a relevant cause.

Clinical significance of current findings

Sensorineural hearing loss has been observed to represent increased risk of subsequent stroke.[2] Other studies have suggested an ischaemic component to hearing loss in certain cases,[13] with sudden hearing loss considered a sign of, and risk factor for, stroke.[4] Stroke presenting with acute hearing loss is often associated with vestibular disturbance caused by small perforators originating from the anterior inferior cerebellar artery.[14] The blood supply of the auditory regulation system originates from the vertebrobasilar system, and the distal small vessels involved are potentially susceptible to small-vessel disease ischaemia because of their small size and the absence of collateral circulation.

Hearing loss has been associated with an increased risk of dementia through longitudinal study[15] and there is apparently overlap between hypertension, white matter hyperintensity burden and hearing loss.[16] The increased prevalence of small-vessel disease aetiology in our group with SNHL suggests a possible vascular origin in a proportion of this group, accepting that hypertension was more prevalent in these patients. Observational studies have associated hearing loss with increased frailty and decreased physical function, including increased risk of falls.[6,17]

Hearing impairment could have an impact at multiple stages on the stroke journey. The stroke severity assessment scores include multiple domains where hearing impairment could impact assessment without adequate appreciation of this potential limitation.[8,9] The assistance of family or friends in communication could be interpreted as a reason to allocate a higher disability score to this group of patients than they merit. Communication between healthcare providers and people with stroke is essential to maximise benefit from rehabilitation therapy. Patients with significant hearing impairment and communication barriers may be deprived of an adequate length of time for rehabilitation because of the time constraint and slow progress in a resource-limited health setting.

Hearing impairment is a potential barrier to effective communication but is common after stroke.[18] Guideline recommendations include audiology review, use of amplification aids and minimising background noise.[19] None of these, if considered, are practicable in an emergency assessment environment. The fact that our study showed that increased disability in the cohort with hearing impairment did not correlate with three-month mortality supports the possibility that disability is overestimated in this group.

A previous study found an association between hearing impairment and lower self-reported performance of activities of daily living.[20] It remains possible that the standardised scoring systems available for stroke and disability are not well

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Table 1. Comparison between the groups of patients with and without SNHL

Category	SNHL ($n = 305$)	No SNHL ($n = 326$)	p value
Age (mean ± SD; years)	79.9 ± 10.4	78.6 ± 10.3	0.113
Sex (male) (n, %)	126 (41.3)	149 (45.7)	0.266
NIHSS (median (IQR))	5 (2–12)	5 (2–13)	0.389
Modified Rankin scale (median (IQR))	1 (0–3)	1 (0–2)	**<0.001**
– 0 (n (%))	96 (31.9)	155 (47.5)	
– 1 (n (%))	75 (24.9)	63 (19.3)	
– 2 (n (%))	36 (12.0)	37 (11.3)	
– 3 (n (%))	58 (19.3)	51 (15.6)	
– 4 (n (%))	35 (11.6)	16 (4.9)	
– 5 (n (%))	1 (0.3)	4 (1.2)	
Missing data (n (%))	0 (0)	4 (1.3)	
TOAST criteria (n (%))			
– Large vessel	46 (15.1)	51 (15.6)	0.845
– Cardioembolic	91 (29.8)	103 (31.6)	0.692
– Small vessel	51 (16.7)	30 (9.2)	**0.005**
– Dual pathology	35 (11.5)	24 (7.4)	0.076
– Undetermined	80 (26.2)	117 (35.9)	**0.009**
– Other	2 (0.7)	1 (0.3)	0.475
Hypertension (n (%))	210 (68.9)	189 (58)	**0.005**
Ischaemic heart disease (n (%))	88 (28.9)	80 (24.5)	0.211
Diabetes (n (%))	76 (24.9)	68 (20.9)	0.225
Atrial fibrillation (n (%))	85 (27.9)	94 (28.8)	0.788
Smoking (n (%))	43 (14.3)	62 (19.1)	0.109
Peripheral vascular disease (n (%))	20 (6.6)	17 (5.2)	0.473
Previous stroke (n (%))	103 (33.8)	82 (25.2)	**0.017**
Hyperlipidaemia (n (%))	62 (20.3)	52 (16)	0.153
Death at three months (n (%))	67 (22)	61 (18.7)	0.310

p values < 0.05 in bold. SNHL = sensorineural hearing loss; SD = standard deviation; NIHSS = National Institutes of Health Stroke Scale; IQR = interquartile range

validated in a population with significant hearing impairment or that interpretation of the responses in a busy hospital environment is leading to overestimation of physical disability due to the hearing impairment itself.

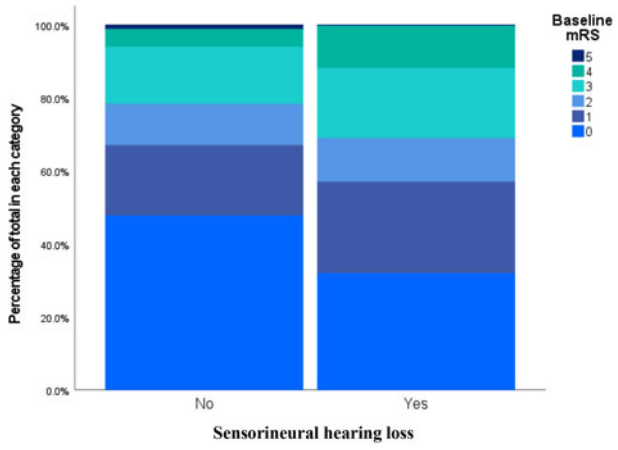

Figure 1. Distribution of admission modified Rankin score in each group. mRS = modified Rankin scale.

The findings from this study suggest an ischaemic aetiology for a proportion of people with SNHL, potentially associated

Table 2. Results of binary logistic regression with three-month mortality as dependent variable after correcting for clinical features associated with mortality and differing between the two groups

Factor	Odds ratio	95% CI	p value
Sensorineural hearing loss	1.1	0.7–2.0	0.668
Age	1.1	1.0–1.1	**0.001**
Admission NIHSS	1.2	1.1–1.2	**<0.001**
Admission modified Rankin scale	1.4	1.1–1.7	**0.001**
HTN	1.1	0.6–1.9	0.748
Diabetes	0.7	0.4–1.5	0.386
IHD	1.1	0.6–1.9	0.789
Previous stroke and/or transient ischaemic attack	1.1	0.6–1.9	0.707
TOAST aetiology small-vessel disease	1.9	0.5–6.5	0.322

$p < 0.05$ in bold. CI = confidence interval; NIHSS = National Institutes of Health Stroke Scale; HTN = hypertension; IHD = ischaemic heart disease

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with small-vessel disease. We found an increased estimation of disability in the cohort with SNHL that did not result in increased mortality three months after stroke.

Study limitations and strengths

The limitations of this study include its retrospective observational design, despite the number of people in each group. This makes it difficult to eliminate confounding factors completely. We used age-matching to eliminate this important confounder, and most baseline factors were well matched between groups. However, there were still important differences, such as the proportion with hypertension and previous stroke, that limit the confidence in interpretation of specific predictors.

Patients who have had strokes can also develop hearing impairment afterwards. Unfortunately, this information is not consistently available, as not all patients with stroke have had a hearing assessment. This would limit the interpretation of our study findings.

Our study is one of the larger studies to specifically investigate SNHL in patients with ischaemic stroke, shedding light on an important and common factor in stroke care.

- Sensorineural hearing loss (SNHL) is common in the older population
- Ischaemia may be one aetiology of SNHL in older people
- In this study SNHL was associated with hypertension and stroke of small-vessel aetiology
- The cohort with SNHL had a higher disability score distribution
- Sensorineural hearing loss had no impact on three-month mortality
- These findings are consistent with an ischaemic aetiology to a proportion of sensorineural hearing loss

It would be beneficial to specifically assess the impact of hearing impairment on disability outcome measures and to further investigate standardised tools for the assessment of stroke severity and disability in people with hearing impairment to enhance their validity in this important patient group, as well as any potential impact this might have on rehabilitation outcomes.

Conclusions

Our age-matched observational study shows that SNHL is associated with increased baseline disability but does not have a significant effect on the three-month mortality outcome. Patients with SNHL have a higher prevalence of underlying hypertension and small-vessel disease, which is more often the underlying stroke aetiology. These key findings contribute to the current literature indicating a potentially ischaemic aetiology of SNHL.

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