JoS JOURNAL OF STROKE

Journal of Stroke 2022;24(2):303-306
https://doi.org/10.5853/jos.2022.00836

Letter to the Editor

Association of Symptomatic Hearing Loss with Functional and Cognitive Recovery 1 Year after Intracerebral Hemorrhage

Jessica R. Abramson,[a,b,c] Juan Pablo Castello,[a,b] Sophia Keins,[a,b,c] Christina Kourkoulis,[a,b,c] M. Edip Gurol,[b] Steven M. Greenberg,[b] Anand Viswanathan,[b] Christopher D. Anderson,[a,b,c,d] Jonathan Rosand,[a,b,c] Alessandro Biffi[a,b,c]

[a]Henry and Allison McCance Center for Brain Health, Massachusetts General Hospital, Boston, MA, USA
[b]Department of Neurology, Massachusetts General Hospital, Boston, MA, USA
[c]Center for Genomic Medicine, Massachusetts General Hospital, Boston, MA, USA
[d]Department of Neurology, Brigham and Women's Hospital, Boston, MA, USA

Dear Sir:

Survivors of intracerebral hemorrhage (ICH) are at high risk for poor functional and cognitive outcomes. At 12 months from the acute hemorrhage less than a third achieve functional independence, while over 25% are diagnosed with dementia and many more report milder cognitive deficits.[1-3] Hearing loss represents modifiable risk factor for functional decline cognitive dysfunction, yet it is often underdiagnosed and insufficiently addressed among individuals at risk.[4] We therefore sought to quantify the incidence of hearing loss among ICH survivors, identify associated risk factors, and determine whether it is associated with poor neurological recovery.

We analyzed data for consecutive patients admitted to Massachusetts General Hospital between January 1st 2006 and December 31st 2017 with a spontaneous ICH diagnosis.[5] Admission CT scans were analyzed to determine ICH location and hematoma volume.[2] We used validated ordinal scales to evaluate overall cerebral small vessel disease, cerebral amyloid angiopathy (CAA), and hypertensive arteriopathy burden on brain magnetic resonance imaging (MRI) scans obtained according to a previously validated protocol.[1] We initially screened for diagnosis of hearing loss by analyzing participants' electronic health records (EHR) using a natural language processing approach.[6] All hearing loss diagnoses were then confirmed by manual review of EHR. We captured information on functional performance status on the modified Rankin Scale (mRS) at discharge, 3 months, and 12 months after ICH.[7] We then subdivided participants in the following groups: (1) functional decline (i.e., higher mRS at 12 months vs. 3 months); (2) functional stability (i.e., same mRS at 12 months vs. 3 months); and (3) functional recovery (i.e., lower mRS at 12 months vs. 3 months).[8] We captured cognitive recovery by combining manual review of EHR with results from the modified Telephone Interview for Cognitive Status (TICS-m), administered at 3 and 12 months after ICH as previously described.[2] Among individuals with cognitive impairment at 3 months (major or minor neurocognitive disorder), we identified those who experienced cognitive recovery at 12 months based on either: (1) resolution of cognitive deficits (i.e., return to normal cognition) or (2) improvement from major to minor neurocognitive impairment. We performed univariable and multivariable analyses to identify risk factors for hearing loss diagnosis. We then performed multivariable analyses of likelihood to experience functional or cognitive recovery among study participants. For functional recovery, we created an ordinal logistic regression model quantifying likelihood of experiencing decline, stability, or improvement in functional performance. For cognitive recovery, we created a logistic regression model quantifying likelihood of experiencing improvement in cognitive performance (as previously defined). Additional information on study inclusion and exclusion criteria, enrollment procedures, data collection, and statistical methods are provided in the Supplementary Methods.

We initially screened a total of 1,339 consecutive ICH cases for inclusion in the present study. After application of exclusion and inclusion criteria (Supplementary Figure 1), we analyzed data for 737 ICH survivors. The majority of excluded participants (475/602, 78.9%) were excluded due to mortality within 1 year of the acute ICH. We identified 86 participants (11.7%) who received a diagnosis of hearing loss (Supplementary Table 1). In multivariable analyses, age, number of medical visits be-

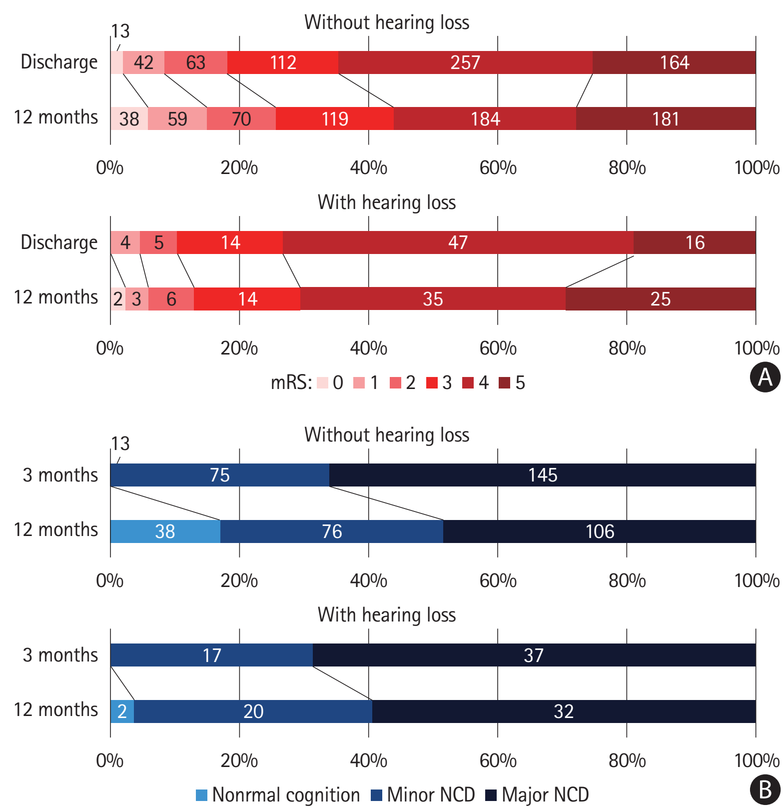


Figure 1. Functional and cognitive recovery following intracerebral hemorrhage (ICH) among survivors with and without hearing loss. (A) Comparison of distribution in modified Rankin Scale (mRS) scores at 3 months vs. 12 months among ICH survivors without (top bars) vs. with (bottom bars) hearing loss. Numbers in the bar section refer to count of individuals within each subgroup defined by mRS scores. (B) Comparison of distribution in cognitive status diagnoses at 3 months vs. 12 months among ICH survivors without (top bars) vs. with (bottom bars) hearing loss. Numbers in the bar section refer to count of individuals within each subgroup defined by diagnosis of major neurocognitive disorder (NCD), minor NCD, or normal cognition at each time point.

Table 1. Multivariable analyses of risk factors for hearing loss among ICH survivors

Variable	OR	95% CI	*P*
Age (/10 years)	1.08	1.05–1.11	<0.001
Race/Ethnicity (non-White)	0.68	0.31–1.50	0.341
Male sex	1.82	1.11–2.99	0.018
No. of medical visits (12 months before ICH)	1.03	1.01–1.05	0.001
No. of antihypertensive medications			
None	Reference	Reference	Reference
One	1.43	0.75–2.71	0.279
Two	1.00	0.47–2.10	0.990
Three or more	2.31	1.15–4.67	0.019
IVH volume (/10 cc)	0.74	0.50–1.11	0.147
CAA MRI score (for 1 point increase)	1.26	1.06–1.50	0.009

ICH, intracerebral hemorrhage; OR, odds ratio; CI, confidence interval; IVH, intraventricular hemorrhage; CAA, cerebral amyloid angiopathy; MRI, magnetic resonance imaging.

Table 2. Multivariable models for functional and cognitive recovery after ICH

Variable	Functional recovery (ordinal logistic regression)			Cognitive recovery (logistic regression)		
	OR	95% CI	*P*	OR	95% CI	*P*
Age (/yr)	1.00	0.99–1.02	0.732	1.00	0.97–1.02	0.955
Race/Ethnicity (non-White)	0.98	0.62–1.54	0.921	0.46	0.21–1.03	0.058
Male sex	1.45	1.02–2.05	0.038	1.26	0.69–2.29	0.453
Discharge mRS: 4 to 5	0.25	0.17–0.36	<0.001	-	-	-
ICH location: lobar	-	-	-	0.43	0.23–0.82	0.010
ICH volume (/10 cc)	-	-	-	0.88	0.78–1.00	0.050
GCS >8 at presentation	-	-	-	2.96	1.09–8.04	0.034
CSVD MRI score (/1 point increase)	0.34	0.29–0.39	<0.001	0.74	0.61–0.89	0.002
Hearing loss	0.43	0.25–0.76	0.004	0.32	0.13–0.79	0.014

ICH, intracerebral hemorrhage; OR, odds ratio; CI, confidence interval; mRS, modified Rankin Scale; GCS, Glasgow Coma Scale; CSVD, cerebral small vessel disease; MRI, magnetic resonance imaging.

fore ICH, number of anti-hypertensive medications used before ICH and CAA disease burden on MRI were independently associated with likelihood of receiving a diagnosis of hearing loss (Table 1). Among 737 participants, 60 (8.1%) experienced functional decline, 473 (64.2%) experienced functional stability, and 204 (27.7%) experienced functional recovery (Figure 1A). In multivariable analyses, hearing loss emerged as independently associated with functional recovery at 1 year from ICH (Table 2). At 3 months, 92 participants (12%) were diagnosed with minor neurocognitive disorder and 182 (25%) with major neurocognitive disorder, and were thus eligible for inclusion in subsequent analysis of cognitive recovery in the first year after ICH (Figure 1B). We found that 76/274 (28%) fulfilled criteria for cognitive recovery at 12 months from the initial hemorrhagic stroke event. In multivariable hearing loss was an independent risk factor for lower likelihood of cognitive recovery at 1 year from ICH (Table 2).

In summary, we leveraged data from a single-center study of ICH survivors to investigate the prevalence of hearing loss in this patient group at high risk for poor functional and cognitive outcomes. We found that over 10% of them displayed evidence of symptomatic hearing loss, with multiple factors contributing to individuals' likelihood of receiving this diagnosis. We specifically identified a novel association with CAA disease severity, as quantified via a validated MRI scoring system. Furthermore, we found that hearing loss was associated with lower likelihood of good functional and cognitive outcomes at 1 year after ICH. Overall, our results indicate hearing loss might serve as a key, underappreciated barrier preventing survivors of primary ICH from achieving their maximum recovery potential. Additional studies will be required to investigate mechanisms accounting for this association, including determining: (1) impacts engagement with post-stroke rehabilitation effort; (2) serves as a surrogate marker of underlying brain health and its expected impact on recovery; and (3) directly contributes to decreased neuronal plasticity.[9]

Our study has several limitations. Our approach likely resulted in imprecise capture of hearing performance—including potential for incorrect hearing loss diagnoses and limited ability to quantify etiology, laterality, severity, and treatment course of hearing impairment. We also utilized phone-based cognitive testing, which could potentially introduce bias towards worse cognitive performance among participants with hearing loss. To address this possibility, we conducted a validated screening of hearing performance prior to phone-based cognitive testing, as in prior studies.[2] We also conducted a parallel analysis using EHR-derived cognitive performance data to bolster our findings. Finally, we leveraged data from a single, tertiary care center with dedicated expertise in ICH management. This may have therefore introduced referral and severity bias, potentially limiting generalizability to ICH survivors at large.

Supplementary materials

Supplementary materials related to this article can be found online at https://doi.org/10.5853/jos.2022.00836.

References

1. Pasi M, Sugita L, Xiong L, Charidimou A, Boulouis G, Pongpitakmetha T, et al. Association of cerebral small vessel disease and cognitive decline after intracerebral hemorrhage. *Neurology* 2021;96:e182-e192.
2. Biffi A, Bailey D, Anderson CD, Ayres AM, Gurol EM, Greenberg SM, et al. Risk factors associated with early vs delayed dementia after intracerebral hemorrhage. *JAMA Neurol*

2016;73:969-976.
3. Benedictus MR, Hochart A, Rossi C, Boulouis G, Hénon H, van der Flier WM, et al. Prognostic factors for cognitive decline after intracerebral hemorrhage. *Stroke* 2015;46:2773-2778.
4. Vaisbuch Y, Santa Maria PL. Age-related hearing loss: innovations in hearing augmentation. *Otolaryngol Clin North Am* 2018;51:705-723.
5. Biffi A, Anderson CD, Battey TW, Ayres AM, Greenberg SM, Viswanathan A, et al. Association between blood pressure control and risk of recurrent intracerebral hemorrhage. *JAMA* 2015;314:904-912.
6. Biffi A, Murphy MP, Kubiszewski P, Kourkoulis C, Schwab K, Gurol ME, et al. APOE genotype, hypertension severity and outcomes after intracerebral haemorrhage. *Brain Commun* 2019;1:fcz018.
7. Janssen PM, Visser NA, Dorhout Mees SM, Klijn CJ, Algra A, Rinkel GJ. Comparison of telephone and face-to-face assessment of the modified Rankin Scale. *Cerebrovasc Dis* 2010;29:137-139.
8. Murphy MP, Kuramatsu JB, Leasure A, Falcone GJ, Kamel H, Sansing LH, et al. Cardioembolic stroke risk and recovery after anticoagulation-related intracerebral hemorrhage. *Stroke* 2018;49:2652-2658.
9. Johnson JC, Marshall CR, Weil RS, Bamiou DE, Hardy CJ, Warren JD. Hearing and dementia: from ears to brain. *Brain* 2021;144:391-401.

Correspondence: Alessandro Biffi
Divisions of Stroke, Behavioral Neurology and Memory Disorders, Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Room 2064, 100 Cambridge Street, Boston, MA 02144, USA
Tel: +1-617-726-5358
Fax: +1-617-643-3293
E-mail: abiffi@mgh.harvard.edu
https://orcid.org/0000-0001-7063-455X

Received: March 2, 2022
Revised: March 7, 2022
Accepted: March 7, 2022

The authors' work on this study was supported by funding from the US National Institute of Health (K23NS100816, R01NS093870, R01NS103924, and R01AG26484). The funding entities had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; and preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

All statistical analyses performed by Jessica R. Abramson, BA and Alessandro Biffi, MD (Massachusetts General Hospital).

Ms. Abramson and Dr. Biffi had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

Ms. Abramson, Dr. Castello, Ms. Keins, Ms. Kourkoulis, Dr. Gurol and Dr. Viswanathan report no disclosures. Dr. Greenberg is supported by NIH R01AG26484. Dr. Christopher D. Anderson is supported by NIH R01NS103924, U01NS069763, the American Heart Association (AHA) (18SFRN34250007), the AHA-Bugher Foundation, receives sponsored research support from Massachusetts General Hospital and Bayer AG, and consulting for ApoPharma and Invitae. Dr. Rosand is supported by the AHA-Bugher Foundation, by NIH R01NS036695, UM1HG008895, R01NS093870, R24NS092983, and has consulted for Boehringer Ingelheim. Dr. Biffi is supported by Massachusetts General Hospital, the AHA-Bugher Foundation, and by NIH K23NS100816.

Supplementary Methods

Study inclusion and exclusion criteria

We performed a retrospective analysis of prospectively collected data drawn from the ongoing longitudinal intracerebral hemorrhage (ICH) study conducted at Massachusetts General Hospital (Boston, MA, USA).[1] Study subjects were consecutive patients admitted to Massachusetts General Hospital between January 1st 2006 and December 31st 2017 with a diagnosis of spontaneous ICH. All participants were aged 18 years or older at time of acute primary (i.e., spontaneous) ICH. The initial ICH diagnosis was formulated by the attending stroke neurologist and confirmed via computed tomography (CT) scan obtained within 24 hours of symptoms' onset. Patients with intracranial hemorrhage due to trauma, conversion of an ischemic infarct, rupture of a vascular malformation or aneurysm, or brain tumor were not considered eligible. Because we sought to study the association between hearing loss and neurological recovery during the first year after ICH, we excluded patients who died before 12 months from the acute hemorrhage.

Enrollment, baseline data collection, and longitudinal follow-up

Following screening and enrollment, participants or reliable informants were interviewed in-person by dedicated study staff.[2] Demographic, social, and medical history information were collected, including self-reported race and ethnicity. Study staff conducted follow-up phone interviews blinded to baseline and neuroimaging information at 3, 6, and 12 months after ICH. The Enterprise Data Warehouse (EDW), a secure data storage and retrieval platform for institutions in our healthcare delivery network, was used to augment patient-provided information with Electronic Health Records (EHR). The EDW was used to obtain additional information on medical visits and social determinants of health. Manual review of EHR was used to attempt to fill in missing data. Data remaining missing after manual review were imputed using random sampling of existing values. Median incomes were captured at the zip code level using a publicly available database (https://www.psc.isr.umich.edu/dis/census, accessed on March 1st, 2021).

CT and magnetic resonance imaging data capture and analyses

Admission CT scans were analyzed to determine ICH location, hematoma volume, and volume of intraventricular blood according to a previously validated methodology.[3] Magnetic resonance imaging (MRI) Images were obtained using a 1.5 or 3.0 Tesla magnetic resonance scanner, according to a previously validated protocol.[4] Neuroimaging markers of cerebral small vessel disease (CSVD) severity (white matter hyperintensities, cerebral microbleeds, expanded perivascular spaces, lacunes, and cortical superficial siderosis) were rated according to STandards for ReportIng Vascular changes on nEuroimaging (STRIVE) consensus criteria, as previously described.[5] Based on a recently described and validated total CSVD score, we rated microvascular disease burden on an ordinal scale from 0 to 6.[4,6] We also evaluated total cerebral amyloid angiopathy burden on MRI using a validated ordinal score that ranges from 0 to 6,[7] and total hypertensive arteriopathy burden on MRI using a validated ordinal scale ranging from 0 to 4.[8]

Hearing loss diagnosis

We initially screened for diagnosis of hearing loss utilizing encounter information available for each inpatient and outpatient visit. We then utilized a two-stage process (natural language processing analysis followed by manual review) to extract from EHR (within 6 months before or after MRI) confirmation of diagnosis of hearing loss in the form of: (1) active medical problem; (2) physical exam findings; (3) mention by healthcare provide in review of systems (within 6 months before or after MRI); and (4) audiogram with consistent findings. The initial natural language processing analysis utilized for positive hearing loss identification instances of the following terms: "hearing loss," "hearing impair," or "hard of hearing." Occurrences where the terms "denies," "negative," "mother," "father," or "no " appeared within the same sentence were excluded from receiving a hearing loss diagnosis. Participants with a first mention of hearing loss prior to or within 6 months of MRI were considered to have screened positive for diagnosis of hearing loss. All hearing loss diagnoses were then confirmed by manual review of EHR from a board-certified physician, who proceeded to review medical records to confirm: (1) that hearing loss was present on the screening encounter information and (2) that corroborating evidence could be extracted from EHR in the form of an active medical problem (in past medical history and review of system), or exam finding of hearing impairment (as part of physical exam), or diagnosis based on audiogram (tracings and report were individually reviewed for confirmation). Individuals whose hearing impairment was documented to be unilateral (based on either physical exam or audiogram findings) were included among participants who received a diagnosis of hearing loss. In order to test the performance of our hearing loss diagnosis approach, we extracted from our medical records detailed hearing performance information for all study participants who underwent a formal audiogram (n=37). Our EHR-based approach (natural language processing analysis followed by manual review by physician) had sensitivity of 95% and specificity of 93% for hearing loss diagnosis (after removing audiogram data extraction from the EHR approach).

Outcomes' capture

We captured information on functional performance status by computing the modified Rankin Scale (mRS) via the simplified mRS questionnaire.[9] We administered the questionnaire and computed mRS at discharge, 3 months, and 12 months after ICH. We then subdivided participants based on changes in mRS between 3 and 12 months after ICH in the following groups: (1) functional decline (i.e., higher mRS at 12 months vs. 3 months); (2) functional stability (i.e., same mRS at 12 months vs. 3 months); (3) functional recovery (i.e., lower mRS at 12 months vs. 3 months).[10] We defined cognitive recovery by combining two sources of information. First, a board-certified neurologist conducted a manual review of all EHR to quantify cognitive performance at 3 and 12 months after ICH. Specifically, we extracted information on functional status (i.e., ability to perform activities of daily living and instrumental activities of daily living) and cognitive performance (derived from standardized testing as reported in physician, nursing and rehabilitation/therapy notes) to categorize patients as either: (1) normal cognitive performance; (2) minor neurocognitive disorder (according to DSM-5 criteria); or (3) major neurocognitive disorder (also per DSM-5 criteria).[11] Participants also underwent cognitive testing evaluation during phone-based research interviews at 3 and 12 months after ICH using the modified telephone interview for cognitive status (TICS-m), as previously described.[3] Of note, as in prior studies we included a validated phone-based hearing screen to ensure validity of results. We then utilized previously identified cut-offs to classify participants as normal cognitive performance, minor neurocognitive disorder, or major neurocognitive disorder.[12] Discrepancies in cognitive performance status adjudication between EHR and TICSm (n=16/737 participants, 2.2%) were referred to a panel of three board-certified neurologists for resolution. Among individuals with cognitive impairment at 3 months (major or minor neurocognitive disorder), we identified those who experienced cognitive recovery at 12 months based on either: (1) resolution of cognitive deficits (i.e., return to normal cognition); or (2) improvement from major to minor neurocognitive impairment). Study staff administering study questionnaires and performing EHR review were blinded to all clinical and neuroimaging information (including hearing loss status).

Statistical analyses

T-tests, chi-squared tests, and fisher exact tests were performed to identify univariable relationships with hearing loss diagnosis. Variables with univariable association with diagnosis of hearing loss at $P<0.20$ were included in multivariable logistic regression, with Akaike information criterion (AIC) used to determine the final model. We then performed univariable and multivariable analyses of likelihood to experience functional or cognitive recovery among study participants. Univariable analyses utilized identical methodology as described above for univariable analyses of hearing loss. For functional recovery, we created an ordinal logistic regression model quantifying likelihood of experiencing decline, stability, or improvement in functional performance, as defined by changes in mRS score between 3 and 12 months after ICH. For cognitive recovery, we created a logistic regression model quantifying likelihood of experiencing improvement in cognitive performance among participants diagnosed with minor or major neurocognitive disorder at 3 months after ICH. Specifically, cognitive recovery at 12 months was defined as: (1) return to normal cognition for participants diagnosed with minor neurocognitive disorder at 3 months or (2) return to normal cognition or improvement to minor neurocognitive disorder for participants diagnosed with major neurocognitive disorder at 3 months. We included in multivariable modeling all variables with univariable association with either functional or cognitive recovery status at $P<0.20$. We pre-specified adjustment (regardless of univariable association results) for age, sex, and self-reported race/ethnicity. Owing to the limited number of non-white participants in our study, we opted to adjust for white versus non-white race/ethnicity. We also pre-specified adjustment for discharge mRS for functional recovery multivariable modeling.[10] We then used AIC, followed by pruning of variables with association above the pre-specified threshold (i.e., $P>0.05$) to arrive at two separate final models. Variance inflation factors (VIF) were calculated with pre-defined threshold of VIF >5 for variables with univariate associations with each outcome. No variables exceeded this threshold in either model, so none were excluded. Analyses were performed using R version 4.0.2 (R Foundation for Statistical Computing, Vienna, Austria).

Ethical statement

This study was approved by the Institutional Review Board of Massachusetts General Hospital (approval number: 2021P001340). Written informed consent was obtained from all patients or their representatives.

https://doi.org/10.5853/jos.2022.00836

Supplementary References

1. Biffi A, Anderson CD, Battey TW, Ayres AM, Greenberg SM, Viswanathan A, et al. Association between blood pressure control and risk of recurrent intracerebral hemorrhage. *JAMA* 2015;314:904-912.
2. Biffi A, Murphy MP, Kubiszewski P, Kourkoulis C, Schwab K, Gurol ME, et al. APOE genotype, hypertension severity and outcomes after intracerebral haemorrhage. *Brain Commun* 2019;1:fcz018.
3. Biffi A, Bailey D, Anderson CD, Ayres AM, Gurol EM, Greenberg SM, et al. Risk factors associated with early vs delayed dementia after intracerebral hemorrhage. *JAMA Neurol* 2016;73:969-976.
4. Pasi M, Sugita L, Xiong L, Charidimou A, Boulouis G, Pongpitakmetha T, et al. Association of cerebral small vessel disease and cognitive decline after intracerebral hemorrhage. *Neurology* 2021;96:e182-e192.
5. Wardlaw JM, Smith EE, Biessels GJ, Cordonnier C, Fazekas F, Frayne R, et al. Neuroimaging standards for research into small vessel disease and its contribution to ageing and neurodegeneration. *Lancet Neurol* 2013;12:822-838.
6. Lau KK, Li L, Schulz U, Simoni M, Chan KH, Ho SL, et al. Total small vessel disease score and risk of recurrent stroke: validation in 2 large cohorts. *Neurology* 2017;88:2260-2267.
7. Charidimou A, Martinez-Ramirez S, Reijmer YD, Oliveira-Filho J, Lauer A, Roongpiboonsopit D, et al. Total magnetic resonance imaging burden of small vessel disease in cerebral amyloid angiopathy: an imaging-pathologic study of concept validation. *JAMA Neurol* 2016;73:994-1001.
8. Staals J, Makin SD, Doubal FN, Dennis MS, Wardlaw JM. Stroke subtype, vascular risk factors, and total MRI brain small-vessel disease burden. *Neurology* 2014;83:1228-1234.
9. Janssen PM, Visser NA, Dorhout Mees SM, Klijn CJ, Algra A, Rinkel GJ. Comparison of telephone and face-to-face assessment of the modified Rankin Scale. *Cerebrovasc Dis* 2010;29:137-139.
10. Murphy MP, Kuramatsu JB, Leasure A, Falcone GJ, Kamel H, Sansing LH, et al. Cardioembolic stroke risk and recovery after anticoagulation-related intracerebral hemorrhage. *Stroke* 2018;49:2652-2658.
11. American Psychiatric Association, American Psychiatric Association, DSM-5 Task Force. Diagnostic and Statistical Manual of Mental Disorders: DSM-5. 5th ed. Washington, DC: American Psychiatric Association; 2013. p. 947.
12. Knopman DS, Roberts RO, Geda YE, Pankratz VS, Christianson TJ, Petersen RC, et al. Validation of the telephone interview for cognitive status-modified in subjects with normal cognition, mild cognitive impairment, or dementia. *Neuroepidemiology* 2010;34:34-42.

Supplementary Table 1. Participants' characteristics

Variable	No hearing loss	Hearing loss	*P*
No. of Individuals	651	86	
Demographics			
Age (yr)	70.0±12.8	80.0±11.2	<0.001
Male sex	332 (51.0)	50 (58.1)	0.258
Race/Ethnicity			0.042
White or Caucasian	528 (81.1)	78 (90.7)	
Non-white race/Ethnicity	123 (18.9)	8 (9.3)	
Asian	43 (6.6)	4 (4.7)	
Black or African American	35 (5.4)	3 (3.5)	
Hispanic or Latino	42 (6.5)	1 (1.2)	
Other	3 (0.5)	0 (0)	
Social determinants of health			
Neighborhood income ($)	71,888±25,365	73,397±25,265	0.604
English as preferred language	592 (90.9)	80 (93.0)	0.661
Veteran	73 (11.2)	21 (24.4)	0.001
Education			0.238
Less than high school degree	73 (11.2)	5 (5.8)	
High school degree	317 (48.7)	41 (47.7)	
More than high school degree	261 (40.1)	40 (46.5)	
Religion affiliation			0.178
Christian	476 (73.1)	65 (75.6)	
Non-christian	56 (8.6)	11 (12.8)	
Not affiliated	119 (18.3)	10 (11.6)	
Marital status			0.133
Married	377 (57.9)	43 (50.0)	
Separated	60 (9.2)	7 (8.1)	
Single	101 (15.5)	12 (14.0)	
Widowed	113 (17.4)	24 (27.9)	
Medical visits (during year prior to ICH)	2.9 (8.3)	6.8 (11.4)	0.003
Hypertension control (before ICH)			
No. of anti-hypertensive medications			0.002
None	256 (39.3)	20 (23.3)	
One	178 (27.3)	28 (32.6)	
Two	127 (19.5)	15 (17.4)	
Three or more	90 (13.8)	23 (26.7)	
Other medical history			
Diabetes	136 (20.9)	17 (19.8)	0.920
Atrial fibrillation	109 (16.7)	21 (24.4)	0.109
Hypercholesterolemia	298 (45.8)	41 (47.7)	0.828
Prior ICH	7 (1.1)	1 (1.2)	1.000
Prior ischemic stroke	87 (13.4)	14 (16.3)	0.567
Coronary artery disease	111 (17.1)	21 (24.4)	0.127
Pre-ICH dementia	56 (8.6)	16 (18.6)	0.006

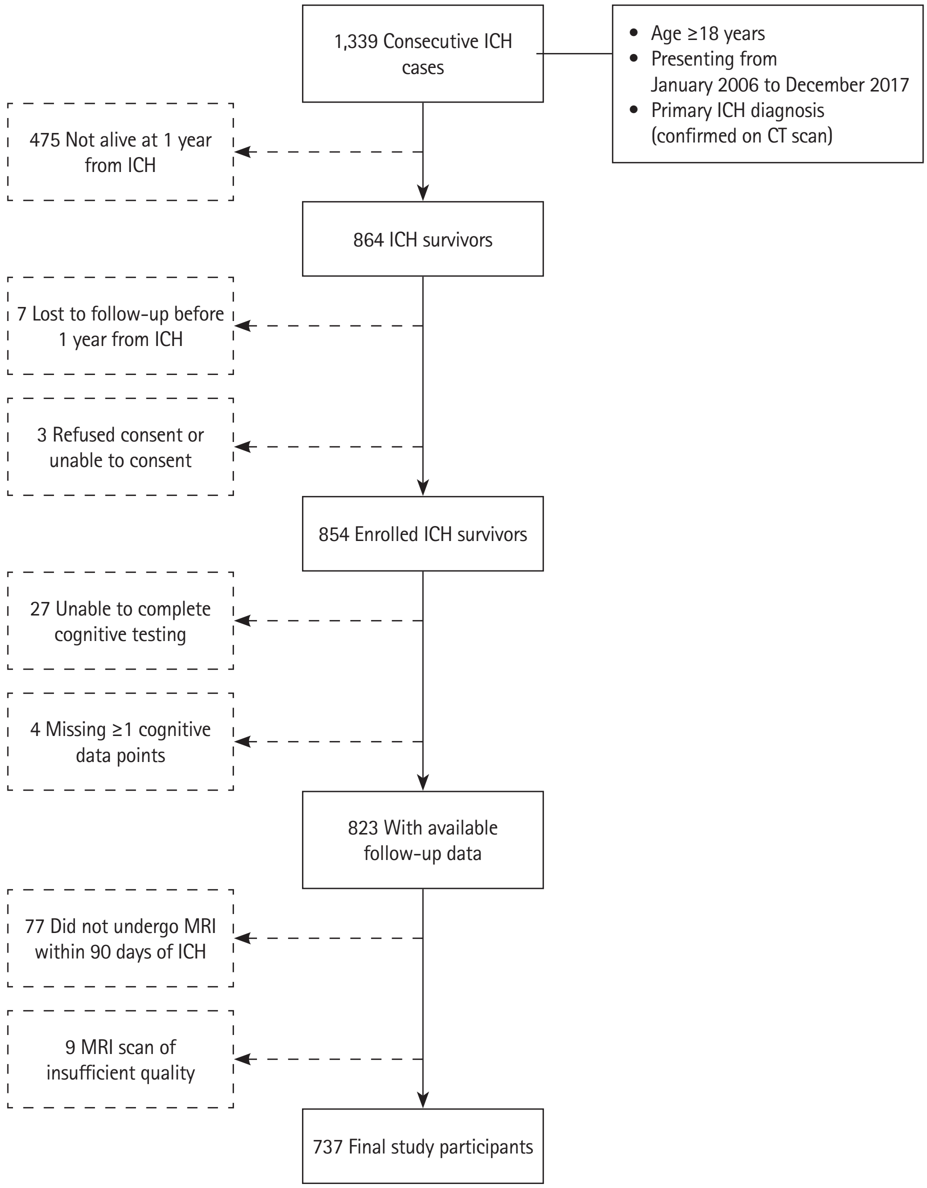
https://doi.org/10.5853/jos.2022.00836

Supplementary Table 1. Continued

Variable	No hearing loss	Hearing loss	*P*
CSVD MRI scores			
Global	2.16±1.58	2.55±1.54	0.033
HTNA	1.86±1.26	2.17±1.24	0.027
CAA	1.36±1.28	1.93±1.37	<0.001
Acute ICH characteristics			
Hematoma location			0.086
Lobar	356 (54.7)	56 (65.1)	
Non-lobar	295 (45.3)	30 (34.9)	
ICH volume (cc)	11.2 (3.6–30.1)	10.8 (2.7–22.5)	0.656
IVH volume (cc)	0.0 (0.0–1.4)	0.0 (0.0–1.1)	0.002
GCS >8 at presentation	552 (84.8)	79 (91.9)	0.111
Discharge mRS	4 (3–5)	4 (3–4)	0.146
Medication use (at time of ICH)			
Warfarin	97 (14.9)	13 (15.1)	1.000
Antiplatelet medication	35 (5.4)	8 (9.3)	0.224

Values are presented as number (%), mean±standard deviation, or median (interquartile range). Reported *P*-values represent univariable comparison between participants with and without hearing loss.

ICH, intracerebral hemorrhage; CSVD, cerebral small vessel disease; MRI, magnetic resonance imaging; HTNA, hypertensive arteriopathy; CAA, cerebral amyloid angiopathy; IVH, intraventricular hemorrhage; GCS, Glasgow Coma Scale; mRS, modified Rankin Scale.



Supplementary Figure 1. Flow chart of enrollment for study participants, indicating number of individuals initially considered for inclusion and removed based on specific exclusion criteria. ICH, intracerebral hemorrhage; CT, computed tomography; MRI, magnetic resonance imaging.

https://doi.org/10.5853/jos.2022.00836