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RESEARCH ARTICLE

Hearing loss and its relation to cognition in Indian cohort: A behavioral and neuroimaging study

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Abstract

INTRODUCTION: Hearing loss (HL) is an unexplored modifiable risk factor that impacts 41% of the Indian population. This study aimed to determine the prevalence of HL, investigate the association between HL and cognitive impairment in older adults, and assess neuronal structures involved in HL and cognitive impairment using behavioral and magnetic resonance imaging (MRI).

METHODS: This study assessed 589 individuals aged 45 and above using HearCheck handheld audiometry, cognitive testing, and MRI.

RESULTS: Participants with HL are 1.69 times more likely to experience cognitive impairment compared to those without HL. Neuroimaging revealed significantly less gray matter in various temporal and hippocampal regions in individuals with HL and cognitive impairment as compared with normal hearing and normal cognition.

DISCUSSION: These findings underscore the importance of exploring the link between sensory impairments, specifically HL, and cognitive impairment, emphasizing the need for preventive strategies in diverse populations.

KEYWORDS
cognition, gray matter volume, hearing loss, neuroimaging, prevalence, VBM

Highlights

- A large urban cohort provides insights into hearing and cognitive function.
- Hearing loss (HL) is associated with a 69% higher likelihood of cognitive impairment.
- Magnetic resonance imaging (MRI) reveals reduced gray matter (GM) loss in individuals with HL.
- Comprehensive cognitive and hearing evaluations strengthen findings.
- Findings align with sensory deprivation and shared risk factor hypotheses.

1 | BACKGROUND

Dementia currently affects an estimated 57 million people worldwide, with projections indicating a rapid increase to 132 million by 2050. Currently, existing pharmaceutical and treatment approaches offer, at best, limited benefits with symptom modification.[1] Therefore, exploring risk factors and implementing suitable cognitive and behavioral interventions are crucial. The 75th National Sample

Survey (NSS) defined hearing disability/hearing loss (HL) as difficulty with daily conversational speech, excluding unilateral hearing impairment. The prevalence of HL was estimated to be 0.3% of the population, with nearly half reporting difficulties in hearing only loud sounds or an inability to hear altogether.[2] The 2011 Indian Census highlighted that 2.21% of the population suffers from some disability, with hearing impairment ranking among the top three, affecting 19% of individuals.[3] The increase in prevalence can be attributed to an aging population, improved identification methods, and excessive exposure to noise.[4] In the Delhi study, the highest prevalence of HL (67%) was observed among those aged 60 years and above, followed by those aged 40 to 59 years, with a prevalence of 40.7%.[5] Recently, the association between HL and dementia has gained much momentum, as HL has been identified as the most potentially modifiable risk factor (contributing approximately 8% to the prevalence of dementia) for the development of dementia.[6]

In India, the prevalence of dementia is 7.4%,[7] and for adults who have hearing impairment, it is 1.2%.[1] Past research has extensively explored the associations between dementia and genetics,[8] vascular issues,[9] cognitive factors,[10] cardiovascular risk factors,[11,12] and genetic issues.[13] Nevertheless, a handful have HL as a factor in small studies but none in the large aging cohorts; hence, there is a need for experimental evidence obtained by testing the local population systematically on hearing and cognition. A prospective study in the United States determined that the rate of cognitive decline and the risk of mild cognitive impairment increased linearly with the severity of HL, as did the risk of developing all-cause dementia. According to one study, mild, moderate, or severe HL was related to a two-, three-, or five-fold higher risk of dementia development, respectively.[14] Meta-analysis studies have consistently reported strong associations between HL and cognitive functions, cognitive impairment, and dementia.[15] Studies also link HL and brain morphology, i.e., reduced GM volume in auditory cortices.[16] Reports suggest that GM volume reductions in the superior temporal, frontal, insular, and hippocampal brain regions are associated with poorer speech in noise understanding and hearing in older participants.[17,18]

Despite evidence of cortical atrophy, the questions remain as to whether the deprivation of auditory input exacerbates brain atrophy during aging and whether this exacerbates brain atrophy and has consequences for cortical organization. Thus, understanding the relationship between HL and structural brain volumes may provide insights into mechanistic pathways through which peripheral impairments in sensory function could contribute to brain aging. The association between HL and cognitive function or dementia has been studied mainly in high-income countries, suggesting that there is a need to assess this association in low- and middle-income countries, especially India. The outcome of this research will help bridge the gaps in developing measures for the early identification and treatment of hearing problems in older adults. The present study aimed to determine the prevalence of HL, investigate the association between HL and cognitive impairment in older adults, and assess neuronal structures involved in HL and cognitive impairment using behavioral and magnetic resonance imaging (MRI). Furthermore, we aimed to clarify whether voxel-based morphometric structural changes in GM volume in participants with HL are associated with cognitive impairment.

RESEARCH IN CONTEXT

1. **Systematic review**: We searched PubMed and Google Scholar from the inception of the ideas up to November 2023 using the terms hearing loss (HL), dementia, cognitively impaired, magnetic resonance imaging (MRI), and voxel-based morphometry (VBM). After screening the citation list, reading abstracts, and accessing publications, we found previous evidence on the association between HL and cognition for various cohorts but none for the Indian cohort. Also, studies comparing neuroimaging findings of normal cognition, and normal hearing versus cognitively impaired and hearing impaired are rare.
2. **Interpretation**: We found that hearing impairment is associated with an increased risk of cognitive impairment, as evidenced by cognitive, hearing, and neuroimaging findings in a large Indian cohort.
3. **Future directions**: We suggest that the causal relationship between hearing impairment and dementia can be established through functional neuroimaging, genetics, and blood biochemistry evaluations when followed up for many years.

2 | METHOD

2.1 | Participants

The participants with the age of 45 years and above were selected from the Centre for Brain Research-Tata Longitudinal Study of Aging (CBR-TLSA), an ongoing longitudinal investigation on the urban aging population in Karnataka, India. The CBR-TLSA cohort was designed to assess multiple aspects of aging, with a particular focus on identifying early cognitive impairment through yearly multimodal investigations and comprehensive health checkups. In this paper only the data of the subjects who have been assessed between 2017 (as hearing assessment started in this year) and December 2022 have been considered. Those with a history of chronic neurological conditions or psychiatric conditions were excluded. Additionally, we ensured that individuals included had no history of cognitive impairment at the baseline. After applying the exclusion criteria, a total of 589 participants with only their baseline data were considered in this current study. The study was approved by the Institutional Human Ethics Committees. All participants provided informed, written consent to participate in the study.

2.2 | Hearing assessment

The hearing status of participants was evaluated using a validated handheld device (HearCheck; Siemens) screening audiometer, following established protocol used by Ray and colleagues in their cohort study.[19] Individuals with an ear infection or a collapsed ear canal were excluded from the study and did not undergo the test. The HearCheck device tested each ear separately with a series of three consecutive tones at high frequency (3 kHz) and midfrequency (1 kHz), each at decreasing intensities: 75, 55, and 35 dB for high frequency and 55, 35, and 20 dB for midfrequency. We used participant responses to each frequency in each ear to determine hearing acuity. If participants could hear all six tones, we classified them as having good hearing (no HL). Hearing difficulty was categorized as mild to moderate if the subject heard three to five tones and moderate to severe if they could not hear two or fewer tones. We combined the best hearing test results from both ears for analysis. Hearing acuity was then classified as good (good acuity in both ears), mild difficulty (mild difficulty in both ears or either ear), or moderate to severe difficulty (moderate to severe difficulty in both ears or either ear), following classification standards from the literature.[19] HearCheck was chosen due to its accessibility and proven effectiveness, especially in older populations, where it has exhibited notable sensitivity and specificity under stringent criteria like ours, as evidenced in recent research.[20,21]

2.3 | Cognitive assessments

Cognitive functions were assessed using the Hindi Mental State Examination (HMSE) and Adenbrooke's Cognitive Examination (ACE). The HMSE, adapted from the Mini-Mental State Examination (MMSE) and validated for the Indian population,[22] evaluates cognitive function across several domains: "Orientation to time" assesses the participant's awareness of date and time, with a maximum score of five points. "Orientation to place" evaluates the participant's understanding of their current location, with a maximum possible score of five points. Registration tests the ability to repeat a short list of everyday items, with a maximum possible score of three points. "Attention and calculations" evaluate arithmetic ability by counting backward from 100 in steps of 7, with a maximum score of five points, and reciting days of the week forward and backward. The "recall" determines the participant's ability to recall items from the "Registration" section, with a maximum score of three points. Language assesses the ability to name two everyday objects, with a maximum score of two points. "Repetition" involves repeating a short phrase, with one point awarded for a correct response. Complex commands test the ability to follow instructions to perform a task or draw, with a maximum possible score of six points.

These tests collectively assess memory, executive functions, language, attention, fluency, and visuospatial functions. For HMSE, ≤ 25 and ≤ 27 indicate cognitive impairment for older adults without education and with some level of education, respectively. For ACEs, scores ≤ 82 and ≤ 88 indicate cognitive impairment for adults with no formal education and those with some education, respectively.

During the data analysis, we considered the participants' general sociodemographic characteristics and health comorbidities. These characteristics included sex (male, female), age group (45–55, 56–64, 65 years and above), and education level (primary, secondary, graduation, postgraduate/PhD), smoking (yes, no), and alcohol consumption (yes, no). The health comorbidities considered were diabetes mellitus (DM) (yes, no), hypertension (HTN) (yes, no), cardiac illness (yes, no), depression (yes, no), and stroke (yes, no).

2.4 | MRI data acquisition

We acquired MRI data with a 3 Tesla MRI scanner (Magnetom Prisma, Siemens, Germany) equipped with a 64-channel head coil. During the MRI scan, participants lay supine, and head movement was restricted using foam pads on both sides of the head. High-resolution T1-weighted images were acquired using a magnetization-prepared rapid acquisition gradient-echo (MPRAGE) pulse sequence (repetition time [TR] = 2300 ms; echo time [TE] = 2.26 ms; inversion time [TI] = 900 ms; flip angle = 9°; field of view [FOV] = 256×240 mm^2; slice thickness = 1 mm; voxel size = $1 \times 1 \times 1$ mm^3; number of slices = 176).

2.5 | MRI data processing

2.5.1 | Voxel-based morphometry

Voxel-based morphometry (VBM) entails a voxelwise comparison of regional GM volume among groups of participants. We used the statistical parametric mapping package SPM12 (Welcome Department of Cognitive Neurology, UK; http://www.fil.ion.ucl.ac.uk/spm/) and MATLAB-based custom software for image processing. Structural data processing steps were performed per the guidelines of the Encyclopedia of Neuroscience.[23,24] The Diffeomorphic Anatomic Registration Through Exponentiated Lie Algebra (DARTEL) algorithm toolbox was employed to improve intersubject image registration. Initially, we segmented anatomical images into GM and white matter (WM) probability maps using the "new segment" option of SPM12. We subsequently executed the "DARTEL (create templates)" option to generate flow fields and a series of template images. Finally, the flow fields and final template created in the previous step were used to normalize the GM maps (modulated) and smoothed with a Gaussian filter (8 mm full width at half maximum). The representative normalized and segmented GM maps for two 55-year-old participants (Figure S1) as well as for the 88- and 91-years-old participants are shown in Figure S2, respectively.

2.5.2 | FreeSurfer analysis

FreeSurfer software (v 7.2.0) (http://surfer.nmr.mgh.harvard.edu/) was used to acquire cortical volumes for all participants. During preprocessing, we applied motion correction to all T1-weighted images. The volumetric approach was consistent with previously documented

methods. Detailed analysis, including cortical reconstruction and brain segmentation, was conducted using FreeSurfer's recon-all function (http://surfer.nmr.mgh.harvard.edu/fwiki/recon-all). Additionally, manual evaluation of the processed data ensured the inclusion of all brain areas and verified the accuracy of the neuroanatomical labels.

2.6 | Statistical analyses

The normality of the continuous variables was checked using the Shapiro–Wilk test. The means and standard deviations (SDs) are reported for normally distributed variables; otherwise, the medians and interquartile ranges (IQRs) are reported. Categorical variables are reported as frequencies and percentages. The socio-demographic characteristics included sex (male, female), age group (45–55, 56–64, 65 years and above), years of education (primary, secondary, graduation, postgraduate/PhD), smoking (yes, no), and alcohol consumption (yes, no). The health comorbidities considered were DM (yes, no), HTN (yes, no), cardiac illness (yes, no), depression (yes, no), and stroke (yes, no). Further, univariate binary logistic regression analysis was performed wherein cognitive impairment (no = 0, yes = 1) was considered as the outcome variable and all the sociodemographic characteristics and the health comorbidities were considered as the independent variables. Univariate analyses were performed considering variables related to the sociodemographic characteristics and the health comorbidities one at a time in the regression model. Multiple logistic regression analysis was conducted to assess the associations between hearing status (hearing acuity, NH = 0, HL = 1) and cognitive impairment (no = 0, yes = 1) after adjusting for all the factors mentioned above simultaneously in the regression model. Furthermore, we reported the results of univariate and multiple binary logistic regression analyses as unadjusted and adjusted odds ratios (ORs), respectively, along with their 95% confidence intervals (CIs).

For voxelwise GM volume differences, smoothed whole-brain GM maps of participants with normal hearing-normal cognition (NH-NC), normal hearing-cognitively impaired (NH-CI), hearing loss-normal cognition (HL-NC), and hearing loss-cognitively impaired (HL-CI) were compared using analysis of covariance (ANCOVA), with sex, DM, HTN, smoking status, and alcohol consumption as fixed factors and age and years of education (as a continuous variable) as covariates. A whole-brain analysis was performed, with a significance level of $p < 0.001$, uncorrected for multiple comparisons, a $p < 0.05$, and family-wise error (FWE) correction.

For regional GM volume differences, we constructed a general linear model (GLM) in which regional GM volume was the dependent variable and cognitive status (NH-NC, NH-CI, HL-NC, and HL-CI) was the independent variable of interest. The model was adjusted for age, sex, years of education, DM, HTN, auditory perception in background noise, and total intracranial volume (ICV). For GLM, age and years of education were treated as continuous variables. The following regional brain volumes were considered: bilateral cerebral GM volumes, hippocampus, superior temporal gyrus, middle temporal gyrus, inferior temporal gyrus, inferior parietal areas, and precuneus. Any p-value < 0.05 was treated as statistically significant. All analyses were performed in IBM SPSS Statistics version 28.0.

3 | RESULTS

3.1 | Behavioral results

The total number of participants on whom hearing tests and cognitive tests were conducted was 589 (52.3% female and 48.7% male), among whom 76.6% were 55 years and older. Among these 589 participants, 97 (16.5%) had abnormal cognitive test scores on the ACE. None of the participants scored below 26 on HMSE. A total of 241 (41%) of the participants had abnormal hearing test scores. Regarding comorbidities, 197 (33.4%) had diabetes, 389 (66.0%) had HTN, and 36 (6.1%) had cardiac illness. Although a high percentage of the participants had HTN, their hearing scores did not change to the extent that they experienced HL. Additionally, the number of participants with coexisting HTN and HL was lower. Anxiety and depression disorders were not detected in more than 6% of the total participants analyzed on the GAD and GDS, respectively. None of the participants wore hearing aids or used any other hearing interventions.

The univariate binary logistic regression model indicated that participants with HL were more likely to have cognitive impairment compared with those without HL (unadjusted OR: 1.648, 95% CI: 1.064–2.552) as shown in Table 1. Further, on using a multiple logistic regression model wherein socio-demographic and cardiovascular risk factors were adjusted, we found that only the factors diabetes and gender showed significance (adjusted OR: 1.69, 95% CI: 1.08–2.65). Thus, the chance of getting cognitive impairment increases from 64% to 69% when participants have HL with a history of diabetes (Table 2). The descriptive statistics and the unadjusted ORs for all the factors are provided in Table 1, and adjusted ORs in Table 2.

3.2 | MRI results

3.2.1 | Voxelwise GM volume differences

Normal hearing participants (NH-NC vs. NH-CI): Significantly lower GM volume in the left superior temporal gyrus, middle temporal gyrus, inferior temporal gyrus, left rolandic operculum, left insula, and left fusiform gyrus was observed in NH-CI when compared with NH-NC. This implies that participants with NH-CI had less GM volume in these regions compared to those with NH-NC (all p-value < 0.05, FWE corrected), as shown in Figure 1A,B.

Hearing impaired participants (HL-NC vs. HL-CI): Significant lesser GM volume in multiple regions, including the bilateral fusiform gyrus, bilateral hippocampus, bilateral parahippocampal gyrus, left inferior temporal gyrus, right precuneus, bilateral mid-cingulate cortex, right supramarginal gyrus, left caudate, and left putamen, was found in HL-CI when compared with HL-NC. Values of $p < 0.001$, uncorrected, are

TABLE 1 Descriptive characteristics and unadjusted OR from binary logistic regression for all sociodemographic and comorbidities factors.

Characteristics	*n* (%)	Prevalence of cognitive impairment (%)	Unadjusted OR (95% CI)
Cognitive impairment (*n* = 589)		–	–
No	492 (83.5)		
Yes	97 (16.5)		
Gender (*n* = 589)			
Female®	308 (52.3)	18.5	1
Male	281 (47.7)	14.2	0.731 (0.47–1.14)
Age groups (*n* = 589)			
45–55®	138 (23.4)	11.6	1
55–65	219 (37.2)	12.8	1.12 (0.58–2.15)
65 and above	232 (39.4)	22.8	2.26 (1.23–4.13)
Education (*n* = 572)			
Postgraduation®	169 (29.5)	5.3	1
Graduation	278 (48.6)	10.8	2.15 (0.99–4.65)
Secondary	106 (18.5)	39.6	11.67 (5.37–25.55)
Primary	19 (3.3)	73.7	49.78 (14.66–168.96)
Occupation (*n* = 589)			
Employed®	335 (57.9)	23.0	1
Unemployed	244 (42.1)	12.2	2.14 (1.37–3.32)
Smoking (*n* = 587)			
No®	487 (83.0)	17.5	1
Yes	100 (17.0)	12.0	0.65 (0.34–1.23)
Alcohol (*n* = 587)			
No®	422 (71.9)	18.5	1
Yes	165 (28.1)	11.5	0.57 (0.34–0.98)
Physical activity (*n* = 589)			
Active®	250 (58.3)	19.0	1
Inactive	179 (41.7)	18.8	1.01 (0.62–1.65)
Hearing loss (*n* = 589)			
No®	346 (58.7)	13.6	1
Yes	243 (41.3)	20.6	1.65 (1.06–2.55)
Diabetes (*n* = 589)			
No®	392 (66.6)	12.8	1
Yes	197 (33.4)	23.9	2.14 (1.38–3.34)
Hypertension (*n* = 589)			
No®	200(34.0)	12.5	1
Yes	389 (66.0)	18.5	1.590 (0.97–2.59)
Cardiac illness (*n* = 589)			
No®	553 (93.9)	16.1	1
Yes	36 (6.1)	22.2	1.49 (0.66–3.37)
Stroke (*n* = 589)			
No®	585 (99.3)	16.1	1
Yes	4.0 (0.7)	22.2	7.97 (not applicable)
Depression (*n* = 589)			
No®	546 (94.1)	16.6	1
Yes	35 (5.9)	25.0	1.85 (0.84–4.08)

Abbreviations: CI, confidence Interval; OR, odd ratio; ®, reference category.

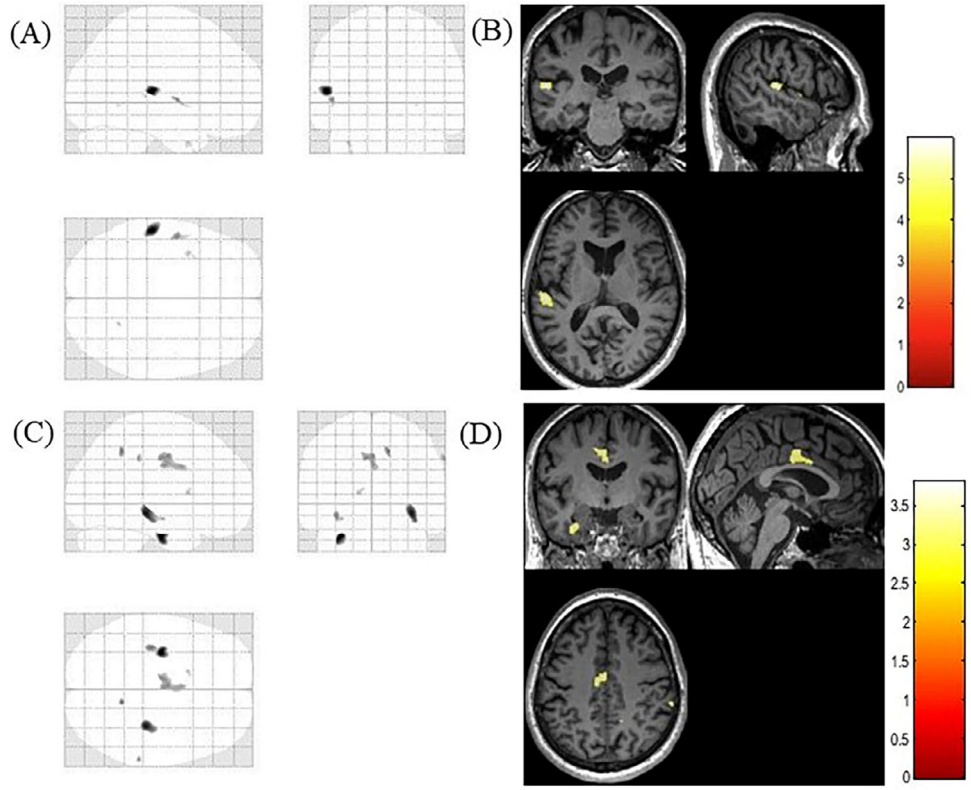

FIGURE 1 (A) Glass-brain view provided in SPM12 and (B) overlay of the peak cluster on the background image of brain regions of lesser gray matter volume in normal hearing-cognitively impaired (NH-CI) participants compared to normal hearing-normal cognition (NH-NC) control participants. (C) Glass-brain view provided in SPM12; and (D) overlay of the peak cluster on the background image of brain regions of reduced gray matter volume in hearing loss-cognitively impaired (HL-CI) participants compared to hearing loss-normal cognition (HL-NC) participants. All images are in neurological convention. Color bar indicates t-statistic values, with black/red color indicates less significance and yellow color indicates higher significance in gray matter volume differences.

TABLE 2 Adjusted OR obtained from multiple logistic regression after adjusting for diabetes and gender.

	Cognitive impairment adjusted OR (95% CI)	*p*-value
Hearing loss		
No®	1	
Yes	1.69 (1.08–2.65)	0.021

Abbreviations: CI, confidence Interval; OR, odd ratio; ®, reference category.

shown in Figure 1C,D. No other volumes or group differences achieved significance.

3.2.2 | Regional GM volume differences

Normal hearing participants

NH-NC versus NH-CI: The left superior temporal ($\beta = -680.24$, standard error (SE) = 220.88, *p*-value = 0.002) and right superior temporal ($\beta = -607.84$, SE = 165.24, *p*-value < 0.001) volumes were significantly different. This implies that participants with impaired cognition (NH-CI) exhibited reduced superior temporal volumes compared to those with normal cognition (NH-NC), as shown in Figure 2.

Hearing impaired participants

HL-NC versus HL-CI: Significant differences were observed in multiple brain regions, including the left hippocampus ($\beta = -243.97$, SE = 82.19, *p*-value = 0.003), right hippocampus ($\beta = -247.75$, SE = 90.54, *p*-value = 0.006), left superior temporal region ($\beta = -514.75$, SE = 212.30, *p*-value = 0.015), left precuneus region ($\beta = -456.738$, SE = 192.53, *p*-value = 0.018), and right temporal middle region ($\beta = -626.28$, SE = 175.09, *p*-value < 0.001). These findings indicated that HL-CI participants exhibited lesser volumes in these regions compared to HL-NC participants, as shown in Figure 2.

4 | DISCUSSION

This study investigated the prevalence of HL and its correlation with cognitive impairment in an urban cohort, revealing that participants with HL are 1.69 times more likely to experience cognitive impairment than those without HL, consistent with existing research.[25] Unlike previous survey-based studies in India,[26,7] this study introduces

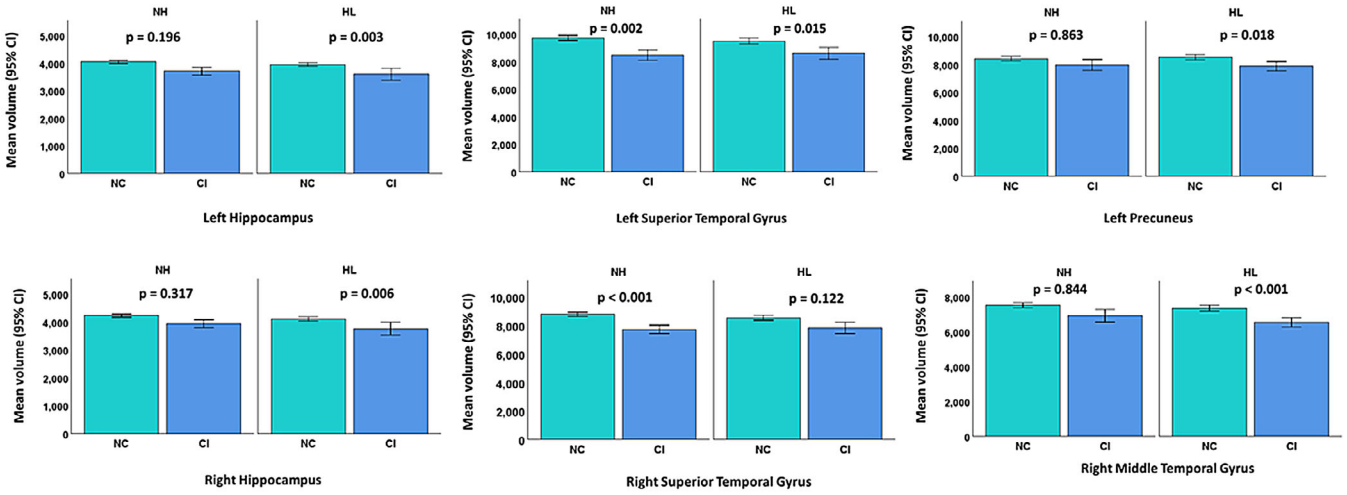

FIGURE 2 Graph showing the significant gray matter volume ($mm3$) differences between NC and CI participants with NH and HL, separately. Left cortical areas (upper panel) and right cortical areas (lower panel). CI, cognitively impaired; HL, hearing loss; NC, normal cognition; NH, normal hearing.

methodological advancements by employing comprehensive assessments, including behavioral, cognitive, hearing, and MRI examinations.

Notably, the study used an automated portable audiometer (Siemens, HearCheck) for hearing tests, in contrast to earlier prospective studies that relied on questionnaires and self-reported HL.[27] The large cohort size and stringent control of confounding variables further distinguish this study from previous research with smaller cohorts and less rigorous controls. Additionally, including both HMSE and ACE scores in the cognitive assessment surpasses earlier research that only used MMSE scores,[28] enhancing the robustness of the findings.

Neuroimaging findings revealed an association between hearing impairment and lesser GM volume, independent of cardiovascular risk factors, alcohol consumption, smoking status, or years of education, in line with previous studies linking peripheral hearing impairment with lesser cortical volumes in the primary auditory cortex,[29,30] variations in the integrity of central auditory tracks,[31,32] and lesser GM volume in other cortical regions like the precuneus and hippocampus.[33]

HL-CI participants exhibited less GM in the medial temporal lobe compared to NH-NC participants, consistent with the reports from Armstrong et al., who found reduced GM in the temporal lobe auditory regions of individuals with HL.[34] VBM analysis also revealed significant GM volume differences in multiple brain regions related to hearing in participants with coexisting HL and cognitive impairment compared to those without cognitive impairment, consistent with previous studies.[35,36]

Several potential mechanisms may explain the association between HL and cognitive impairment. First is the common cause hypothesis—this hypothesis suggests that common risk factors that cause HL and cognitive impairment may lead to reduced hearing and smaller brain volumes.[33,37] This study found widespread brain volume reduction, reflecting microvascular origins.[32] The GM areas identified align with previous studies reflecting on the significantly lower GM volume with age-related HL and its association with cognitive impairments.[38] Given that the medial temporal lobe and hippocampus are critical targets for Alzheimer's disease pathology,[39] lesser GM volume in these areas among HL-CI individuals supports the idea that these regions may serve as the link between HL and cognitive impairment.[40] Hippocampal atrophy, indicative of neural loss, is closely tied to accelerated volume reduction in adults with cognitive impairment[41,42] and may occur even before clinical dementia symptoms.[43] Thus, the effect of HL on cognitive impairment might be mediated through changes in the hippocampus.[44] Cognitively normal adults with HL exhibited GM volume loss compared to those with normal hearing, highlighting the regional specificity in brain volume differences linked to HL. Brain regions related to HL closely overlap with the default mode network targeted by dementia; this may indicate that hearing dysfunction could be a biomarker for the emergence of clinical dementia, consistent with the past literature.[45,46]

Another possibility is the cascade hypothesis, suggesting that change in brain volume or atrophy might be related to hearing impairment. The less GM volume in various auditory brain regions might impact proper integration of auditory information, causing auditory deficits and, in turn, poor cognitive scores. However, this explanation is challenging to justify, as the hearing assessment in this study was conducted using a handheld audiometer rather than the detailed pure tone audiometry and speech-in-noise tests suggesting that the central auditory processes could be involved.

A third hypothesis suggests that HL leads to brain atrophy due to sensory deprivation.[47] Although extensively studied in animals, particularly in deaf cats, limited research has explored its implications in humans.[48] Animal studies suggest that limited auditory input results in structural changes and atrophy in the auditory cortex, with deaf cats showing reduced neuronal activity, synaptic reorganization, and altered neuronal morphology.[49] In humans, the assumption is that HL with poor auditory input may result in synaptic pruning, decreased neural connectivity, and structural changes in the brain.[48,49]

A neuroimaging study on the impact of auditory deprivation on the human brain showed alterations in GM volume and changes in cortical thickness, providing some evidence for sensory deprivation's impact on brain structure in humans.[48] However, a comprehensive understanding of sensory deprivation-induced brain atrophy in humans still needs to be improved. It thus cannot be applied to our results directly implying that the mechanisms discussed above are not mutually exclusive and could all contribute to age-related HL and brain morphology. Longitudinal research tracking changes in brain morphology over time in individuals with varying degrees of HL is essential to confirm these findings. Advanced neurophysiological measures, such as functional MRI, can provide insights into neural activity and the consequences of reduced auditory stimulation on brain structure. Despite accounting for numerous potential confounding factors, the influence of genetic factors on age-related hearing impairment cannot be dismissed.

Additionally, the cross-sectional design of this study limits the ability to draw definitive causal conclusions, necessitating caution in establishing direct cause-and-effect relationships. Moreover, using a handheld audiometer, while practical, may provide less details than pure tone audiometry and speech-in-noise tests, potentially limiting the precision of hearing assessments. Thus, future research could explore the integration of HearCheck with complementary assessment tools like pure tone audiometry to enhance the accuracy of hearing evaluations. Future research should prioritize longitudinal studies and comparative analyses involving individuals with and without hearing aids to elucidate the causal pathways between HL and cognitive deterioration. Understanding these mechanisms will be pivotal in developing strategies to preserve cognitive health in the aging population.

In summary, our findings suggest that HL was associated with cognitive impairment, as seen from cognitive tests and MRI. We could not establish a specific causal relationship from the results obtained due to study's cross-sectional nature. Nonetheless, the study's comprehensive approach, including behavioral, cognitive, hearing, and neuroimaging assessments in a large and diverse urban population, significantly enhances the existing literature, offering valuable insights into the complex relationship between HL and cognitive impairment in the aging population.

AUTHOR CONTRIBUTIONS

All authors have made a substantial intellectual contribution to the conception and design of the study. Acquisition and management of the data (Thomas Gregor Issac), hearing and behavioral analysis (Deepashri Agrawal), MRI analysis (Sadhana Singh), and statistical analysis (Palash Kumar Malo). Drafting of the manuscript (Deepashri Agrawal). All authors approved the final version of the manuscript for publication. Authors take responsibility for data integrity and the accuracy.

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CONFLICT OF INTEREST STATEMENT

All authors declare that they have no conflicts of interest. Author disclosures are available in the supporting information.

CONSENT STATEMENT

The informed consent from all the participants was obtained before collecting the data.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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