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Association of Metabolic Dysfunction-Associated Steatotic Liver Disease With Sudden Sensorineural Hearing Loss Among Older Adults

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Keywords: Meniere's disease | metabolic dysfunction-associated steatotic liver disease | older adults | sudden sensorineural hearing loss

ABSTRACT

Objectives: This study examined the association between metabolic dysfunction-associated steatotic liver disease (MASLD) and the risk of sudden sensorineural hearing loss (SSNHL) in elderly individuals.

Methods: A population-based cohort study using the Korean National Health Insurance Service-Senior cohort included 189,623 individuals aged 65 and older, categorized as non-SLD or MASLD. Cox proportional hazards regression and Fine-Gray subdistribution hazard models were used to evaluate the risk of SSNHL and Ménière's disease, considering all-cause mortality as a competing event.

Results: Baseline characteristics showed that MASLD participants had higher metabolic dysfunction markers, including elevated body mass index, waist circumference, and blood pressure, compared to the non-SLD group. During 9 years of follow-up, 3803 SSNHL events occurred in the MASLD group, with an incidence rate of 2.44 per 1000 person-years. After inverse probability of treatment weighting, MASLD was associated with a significantly increased risk of SSNHL (adjusted hazard ratio: 1.05, 95% CI: 1.00–1.10; $p = 0.039$; subdistribution hazard ratio: 1.06, 95% CI: 1.01–1.11; $p = 0.016$).

Conclusion: MASLD is associated with an increased risk of SSNHL. This study provides evidence supporting a metabolic influence on auditory health, warranting further investigation into the liver-ear axis.

Level of Evidence: Step 3 (Level 3*)—Cohort study.

Eunseok Kang and Seohui Jang contributed equally.

1 | Introduction

Hearing loss (HL) is a significant public health issue with rising prevalence, impacting social, mental, and physical health outcomes [1, 2]. While HL rates continue to increase, limited health screening for inner ear conditions hampers effective follow-up, fueling interest in blood-based biomarkers for early detection [3, 4].

Sudden sensorineural hearing loss (SSNHL) is characterized by a rapid hearing decline within 72 h, with a threshold reduction of 30 dB or more across three consecutive frequencies in pure-tone audiometry [5]. SSNHL is mainly idiopathic, with most cases having no identifiable cause [6, 7]. Studies on SSNHL pathophysiology suggest possible causes, including viral infection, circulatory issues, tumors, trauma, and metabolic disorders [8–10]. Epidemiological data reveal significant age-related risk for SSNHL but no clear association with sex, with an annual incidence of 17.76 cases per 100,000 people in Korea, especially among the elderly and women [11–18].

Numerous studies have demonstrated that chronic conditions accompanied by metabolic dysfunction or liver fat accumulation are associated with an increased risk of hearing loss [19–22]. Metabolic dysfunction has been reported to negatively affect the vascular structures of the inner ear, potentially leading to hearing loss [23]. Additionally, systemic inflammation induced by fatty liver disease has been shown to increase inflammatory cytokines [24], while oxidative stress can impair mitochondrial function, acting as a key mechanism that damages the auditory nerve and inner ear tissues [25]. Given these findings, the concept of a liver-ear axis has been proposed, suggesting a potential pathophysiological link between hepatic dysfunction and auditory impairment. However, some of these studies are cross-sectional, and the lack of comprehensive data on medication use may introduce potential confounders. Therefore, further mechanistic studies are needed to elucidate the underlying mechanisms and establish the temporal relationship between liver pathology and auditory dysfunction.

In addition to SSNHL, Ménière's disease, another inner ear disorder with unclear origins, may share common metabolic or inflammatory pathways. While its exact etiology remains uncertain [26], potential contributing factors include genetic predisposition, infections, and autoimmune conditions [27–31]. Notably, approximately 10% of sudden low-frequency hearing loss cases progress to Ménière's disease [32, 33], highlighting the need to explore metabolic influences on inner ear disorders beyond SSNHL.

Metabolic dysfunction-associated steatotic liver disease (MASLD), previously termed non-alcoholic fatty liver disease (NAFLD) or metabolic dysfunction-associated fatty liver disease (MAFLD), has redefined steatotic liver disease (SLD) diagnosis to include cardiometabolic risk factors (CMRFs) [34, 35]. Understanding MASLD's influence on HL may open new prevention and treatment pathways.

This study aims to address the limitations of previous research by utilizing data from the Korean National Health Insurance Service (KNHIS)-Senior Cohort to conduct a large-scale retrospective cohort study analyzing the association between MASLD and the risk of SSNHL in an elderly population. Unlike previous cross-sectional studies, this study employs a research design that incorporates a 9-year follow-up data to more precisely evaluate the impact of MASLD on the incidence of SSNHL. Furthermore, inverse probability of treatment weighting (IPTW) and the competing risk model are applied to minimize potential confounding factors and enhance causal inference. Additionally, by integrating comprehensive health information, including medical examination data, medication history, metabolic indicators, and liver function markers, this study aims to provide more robust evidence on the association between MASLD and SSNHL, contributing to the development of preventive and therapeutic strategies.

2 | Materials and Methods

2.1 | Study Population

The KNHIS is a non-profit organization that operates health insurance programs for 97% of the Korean population [36]. The KNHIS database is used for numerous nationwide population-based epidemiological studies, and its validity has been detailed in previous studies [36–39].

This study utilized the KNHIS-Senior cohort database 2.0 (NHIS-2024-11-2-054), a prospective-retrospective sample of 511,953 individuals representing approximately 6.4 million older adults (ages 60–80) as of 2008. Among those who underwent health examination between 2009 and 2010, a total of 215,354 elderly people over 65 years of age with non-SLD or MASLD were included. Participants who died ($n = 1861$), were diagnosed with HL ($n = 6915$), and were deaf ($n = 4615$) before the follow-up investigation were excluded. In addition, participants with missing information for the covariates ($n = 4995$) and those with underlying liver disease, including hepatitis virus infection ($n = 3172$), liver cirrhosis ($n = 3889$), and non-alcoholic steatohepatitis ($n = 284$), were also excluded (Figure 1). Finally, the analytic cohort consisted of 189,623 participants. The Institutional Review Board of Korea University Guro Hospital approved this study (No.: 2024-GR0-185).

2.2 | Follow-Up Investigations for Outcome

The primary outcome was SSNHL, defined by the ICD-10 code H912 [40]. Ménière's disease was defined by the ICD-10 code H810 as a secondary outcome [41]. All participants were followed up from January 1, 2011, until the occurrence of incident SSNHL, Ménière's disease, death, or December 31, 2019.

2.3 | Definition of Non-SLD, MASLD

SLD was defined by a fatty liver index (FLI) of ≥ 30, as computed by the algorithm reported in 2006 [42]. FLI is widely used in the Asian population as a non-invasive biomarker of hepatic steatosis, with an area under the receiver operating characteristic curve (AUROC) of 0.844 (95% confidence interval [CI], 0.827–0.862) [43]. Validation of FLI is well described in other previous studies [42–44].

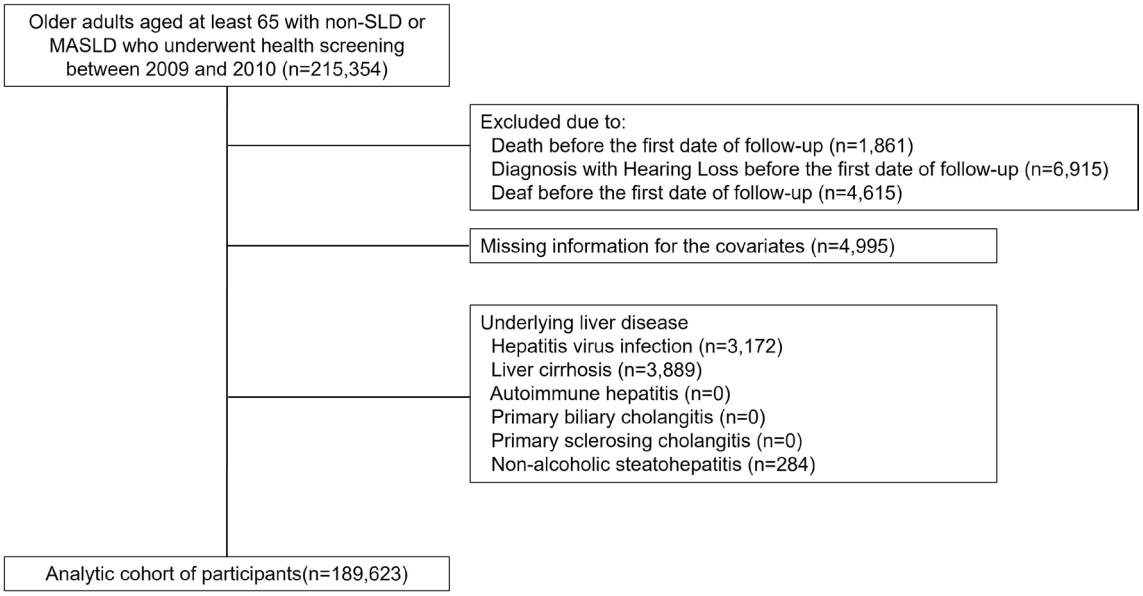

FIGURE 1 | Flow diagram for the inclusion of the older adults. Between 2009 and 2010, participants defined as non-SLD (those without hepatic steatosis), MASLD were drawn from the Korean National Health Insurance Service-Senior Cohort. Individuals who had died or been diagnosed with hearing loss prior to the follow-up period, those with hearing impairment, missing covariate data, or pre-existing liver conditions were excluded from the cohort. MASLD, metabolic dysfunction-associated steatotic liver disease; SLD, steatotic liver disease.

Non-SLD is defined as a patient with no hepatic steatosis. MASLD is defined as a SLD with one or more of the five CMRFs [45, 46]. CMRF is associated with: (1) elevated BMI ($\geq 23\,kg/m^2$) or waist circumference (male, $\geq 90\,cm$; female, $\geq 85\,cm$), (2) elevated fasting serum glucose ($\geq 100\,mg/dL$) or a diagnosis of type 2 diabetes or treatment for type 2 diabetes, (3) elevated blood pressure (systolic $\geq 130\,mmHg$ and/or diastolic $\geq 85\,mmHg$) or specific antihypertension treatment, (4) elevated triglycerides ($\geq 150\,mg/dL$) or lipid-lowering treatment, and (5) reduced high-density lipoprotein (HDL) cholesterol (male, $\leq 40\,mg/dL$; female, $\leq 50\,mg/dL$) or lipid-lowering treatment.

2.4 | Statistical Analysis

Categorical and continuous variables were presented as number (%) and mean (standard deviation [SD]), respectively, or median (interquartile range [IQR]) if nonnormally distributed.

A Cox proportional hazards regression model was used to calculate the cause-specific adjusted hazard ratios (aHRs) with 95% confidence intervals (CIs) for incident SSNHL and Ménière's disease in MASLD patients. The minimally adjusted model included age (continuous; years) and sex (categorical; male and female) as covariates; the fully adjusted model included age, sex, household income (categorical; 1st, 2nd, 3rd, and 4th quartiles), Charlson comorbidity index (CCI; categorical; 0, 1, and ≥ 2), smoking (categorical; never, past, and current), and moderate-to-vigorous physical activity (MVPA; categorical; No, 1-2, 3-4, and ≥ 5 times/week) as covariates.

Additionally, considering the elderly study population, death without the onset of SSNHL or Ménière's disease was treated as a competing event. A multivariable-adjusted Fine and Gray regression model was employed to evaluate the association between MASLD and the risk of incident SSNHL and Ménière's disease, using subdistribution hazard ratios (SHRs) [47]. The incidence was reported as events (*n*) per 1000 person-years.

To perform statistical calibration, we applied inverse probability of treatment weighting (IPTW) based on propensity scores estimated using a logistic regression model. The propensity score model included age, sex, household income, CCI, smoking status, and MVPA as covariates. Covariate balance before and after IPTW was assessed using standardized differences.

Sensitivity analyses were conducted by excluding SSNHL and Ménière's disease cases occurring within 1, 2, and 3 years from the first date of the follow-up period. Participants were categorized based on age, sex, BMI, hypertension, diabetes, dyslipidemia, smoking status, MVPA, and CCI to examine how these factors interact with MASLD in affecting SSNHL risk in stratified analyses. Restricted cubic spline (RCS) with four knots was used to visualize the relationships between FLI and the risk of incident SSNHL among individuals with MASLD. SAS Enterprise Guide 8.3 (SAS Institute, Cary, NC, USA) was used for all data gathering, mining, and statistical analysis.

3 | Results

3.1 | Baseline Characteristics of Non-SLD and MASLD

Table 1 shows the baseline characteristics of 84,225 (44.4%) participants with non-SLD and 105,398 (55.6%) participants with MASLD. The average age of participants was 71.3 years (SD, 4.5) in the non-SLD group and 70.6 years (SD, 4.2) in the MASLD

TABLE 1 | Descriptive characteristics of the study population in the Korean NHIS-senior cohort with non-SLD and MASLD.

Characteristic	Non-SLD ($n=84,225$)	MASLD ($n=105,398$)
Age, years	71.3 (4.5)	70.6 (4.2)
Sex, *n* (%)		
Male	30,804 (36.6)	53,899 (51.1)
Female	53,421 (63.4)	51,499 (48.9)
Household income[a], *n* (%)		
1st quartile (lowest)	11,637 (13.8)	14,220 (13.5)
2nd quartile	12,240 (14.5)	16,351 (15.5)
34d quartile	19,350 (23.0)	25,126 (23.8)
4th quartile (highest)	40,998 (48.7)	49,701 (47.2)
Body mass index, kg/m^2	22.1 (2.4)	25.6 (2.8)
Waist circumference, cm	77.9 (6.7)	87.5 (7.0)
Systolic blood pressure, mmHg	127.7 (16.1)	132.2 (15.9)
Diastolic blood pressure, mmHg	76.9 (9.9)	79.4 (10.0)
Fasting serum glucose, mg/dL	99.3 (23.8)	107.3 (30.1)
Total cholesterol, mg/dL	194.6 (37.0)	200.9 (40.1)
HDL-cholesterol, mg/dL	55.9 (28.4)	53.0 (29.5)
LDL-cholesterol, mg/dL	117.8 (35.5)	116.8 (40.2)
Triglycerides, mg/dL	109.8 (52.4)	163.9 (89.5)
Alanine aminotransferase, IU/L	18.2 (8.0)	26.3 (19.0)
Aspartate aminotransferase, IU/L	23.7 (7.7)	28.1 (17.1)
γ-GT, IU/L	17.7 (8.0)	30 (22–46)
Alcohol consumption, *n* (%)		
No	69,615 (82.7)	72,773 (69.1)
1–2 times/week	9005 (10.7)	16,368 (15.5)
3–4 times/week	2889 (3.4)	7812 (7.4)
≥5 times/week	2716 (3.2)	8445 (8.0)
Cigarette smoking, *n* (%)		
Never	65,253 (77.5)	72,551 (68.8)
Past	9669 (11.5)	18,377 (17.4)
Current	9303 (11.1)	14,470 (13.7)
MVPA, *n* (%)		
No	52,444 (62.3)	63,471 (60.2)
1–2 times/week	8580 (10.2)	10,885 (10.3)
3–4 times/week	7601 (9.0)	9838 (9.3)
≥5 times/week	15,600 (18.5)	21,204 (20.1)
Charlson comorbidity index, *n* (%)		
0	13,694 (16.3)	14,443 (13.7)
1	20,627 (24.5)	23,797 (22.6)
≥2	49,904 (59.3)	67,158 (63.7)

Note: Continuous data are presented as mean (standard deviation) and median (interquartile range) if normally distributed and not normally distributed, respectively. Categorical data are expressed as the number (%).
Abbreviations: HDL, high-density lipoprotein; LDL, low-density lipoprotein cholesterol; MASLD, metabolic dysfunction-associated steatotic liver disease; MVPA, moderate-to-vigorous physical activity; NHIS, national health insurance service; SLD, steatotic liver disease; γ-GT, γ-glutamyl transpeptidase.
[a]Proxy for socioeconomic status based on the insurance premium of the National Health Insurance Service.

group. The majority of participants with MASLD were non-drinkers ($n=72,773$; 69.1%), non-smokers ($n=72,551$; 68.8%), and did not engage in MVPA ($n=63,471$; 60.2%).

The mean BMI and waist circumference in MASLD patients were 16% and 12% higher, respectively, compared to the non-SLD group. Additionally, the MASLD group exhibited more than a 3% increase in systolic and diastolic blood pressures, as well as total cholesterol levels, with fasting serum glucose levels being approximately 8% higher. Furthermore, serological liver function markers, including alanine aminotransferase, aspartate aminotransferase, and γ-GT, were elevated by 45%, 19%, and 69%, respectively, in MASLD patients.

3.2 | Risk of Incident SSNHL and Ménière's Disease in MASLD Patients Using the Cox Proportional Hazard Regression

The authors analyzed the risk of incident SSNHL and Ménière's disease in MASLD participants by calculating adjusted hazard ratios (aHRs) using the Cox proportional hazards regression model (Table S1). The risk of SSNHL was elevated in the MASLD group, with an aHR of 1.05 (95% CI, 0.98–1.12; $p=0.149$) after additional adjustments.

3.3 | Risk of Incident SSNHL and Ménière's Disease in MASLD Patients Using the Fine and Gray Subdistribution Hazard Model

Considering all-cause mortality, the authors investigated the risk of incident SSNHL and Ménière's disease in MASLD patients using the Fine and Gray subdistribution hazard model to account for competing events (Table S2). With additional adjustments, the risk of SSNHL in MASLD patients remained significantly higher, with a SHR of 1.06 (95% CI, 0.99–1.13; $p=0.092$).

TABLE 2 | SHRs for incident SSNHL in MASLD patients after the IPTW.

	Non-SLD	MASLD	
	(*n* = 189,533)	(*n* = 189,669)	*p*
No. of events, *n*	3576	3803	
Competing event, *n*	36,298	34,352	
Person-year	1,544,742	1,557,841	
Incidence/1000 PYs	2.31	2.44	
SHR (95% CI)[a]	1.00 (reference)	1.06 (1.02–1.11)	0.008
SHR (95% CI)[b]	1.00 (reference)	1.06 (1.01–1.11)	0.016

Note: SHRs (95% CIs) were calculated using the Fine and Gray's regression with overall death as a competing event.
Abbreviations: CI, confidence interval; IPTW, inverse probability of treatment weighting; MASLD, metabolic dysfunction-associated steatotic liver disease; *p*, probability value; PYs, person-years; SHR, subdistribution hazard ratio; SLD, steatotic liver disease; SSNHL, sudden sensorineural hearing loss.
[a]Adjusted for age and sex.
[b]Adjusted for age, sex, household income, Charlson comorbidity index, smoking, and moderate-to-vigorous physical activity.

3.4 | Risk of Incident SSNHL and Ménière's Disease in MASLD Patients After the IPTW Using the Fine and Gray Subdistribution Hazard Model

The authors addressed potential confounding factors and aimed to achieve balance in covariates between the groups. Table S3 displays the standardized mean differences (SMDs) for variables before and after the IPTW in the non-SLD vs. MASLD comparison. Significant differences in logit propensity score, age, sex, and smoking status were observed before the IPTW, but SMDs for all variables were balanced after IPTW. After IPTW, the weighted pseudopopulation included 189,533 participants for non-SLD and 189,669 for MASLD. Table S4 provides comprehensive information on the changes in various demographic, metabolic, biochemical, and lifestyle factors after the IPTW.

Table S5 presents the risk of incident SSNHL and Ménière's disease in MASLD patients using the Cox proportional regression after the IPTW. In the fully adjusted model, the aHR for SSNHL was 1.05 (95% CI, 1.00–1.10; $p = 0.039$).

Table 2 assessed the risk of incident SSNHL and Ménière's disease in the MASLD group using the Fine and Gray subdistribution hazard model after the IPTW. In the MASLD group, which included 189,669 participants, there were 3803 SSNHL events and 34,352 competing events over 1,557,841 person-years, resulting in an incidence rate of 2.44 per 1000 person-years. The fully adjusted model demonstrated significant results, with an SHR of 1.06 (95% CI, 1.01–1.11; $p = 0.016$) for SSNHL. After accounting for competing risks using the Fine and Gray subdistribution hazard model, the association between MASLD and SSNHL remained statistically significant, with a similar effect size to that observed in the Cox proportional regression.

3.5 | Sensitivity Analyses

Sensitivity analyses were conducted to evaluate the risk of incident SSNHL and Ménière's disease over latent periods of 1, 2, and 3 years among MASLD patients, starting from the first date of the follow-up period. The risk of SSNHL in MASLD patients appeared to increase over time compared to the non-SLD group. With further adjustment, the SHR for SSNHL showed significant results during the 1- to 3-year latent periods, with increased risks of 1.06 (SHR; 95% CI, 1.01–1.12; $p = 0.013$), 1.07 (SHR; 95% CI, 1.02–1.13; $p = 0.008$), and 1.08 (SHR; 95% CI, 1.02–1.15; $p = 0.006$), respectively (Table S6).

3.6 | Subgroup Analysis

Subgroup analyses were performed to examine how various demographic, metabolic, and lifestyle factors affect the risk of SSNHL among participants with MASLD (Figure 2).

Subgroup analysis indicated that older adults, females, and individuals without obesity, as well as those with hypertension, type 2 diabetes, and dyslipidemia, who had MASLD exhibited a significantly higher risk of developing SSNHL. Past smokers (SHR, 1.11; 95% CI, 0.98–1.24) or current smokers (SHR, 1.14; 95% CI, 0.99–1.32) were at a higher risk of SSNHL than those who have never smoked (SHR, 1.04; 95% CI, 0.98–1.10), with a significant interaction (P for interaction = 0.319). This suggests that smoking increases the risk of SSNHL in patients with MASLD.

Furthermore, individuals who did not participate in MVPA had a higher risk of SSNHL (SHR, 1.10; 95% CI, 1.04–1.17) compared to those who engaged in MVPA (SHR, 1.00; 95% CI, 0.93–1.07), with a significant interaction (p for interaction = 0.046).

3.7 | FLI and SSNHL

RCS analysis for the association of FLI with risk of SSNHL among individuals with MASLD showed a right-upward curve (Figure 3). As shown by the RCS of the FLI, there is a significant association between hepatic steatosis and the risk of SSNHL, suggesting that chronic liver inflammation might increase the risk of SSNHL through this pathway.

4 | Discussion

This is the first study to examine the association between MASLD and SSNHL, suggesting MASLD may contribute to its development. This finding serves as an important indicator that reflects the impact of MASLD on disease progression from a prognostic perspective, rather than merely examining the occurrence of the disease itself. Specifically, the findings reveal that the incidence of SSNHL is higher in the MASLD group compared to the non-SLD group, even after controlling for various potential confounding factors, but no significant association was found between MASLD and Ménière's disease. Despite this lack of association, pathological findings in patients with

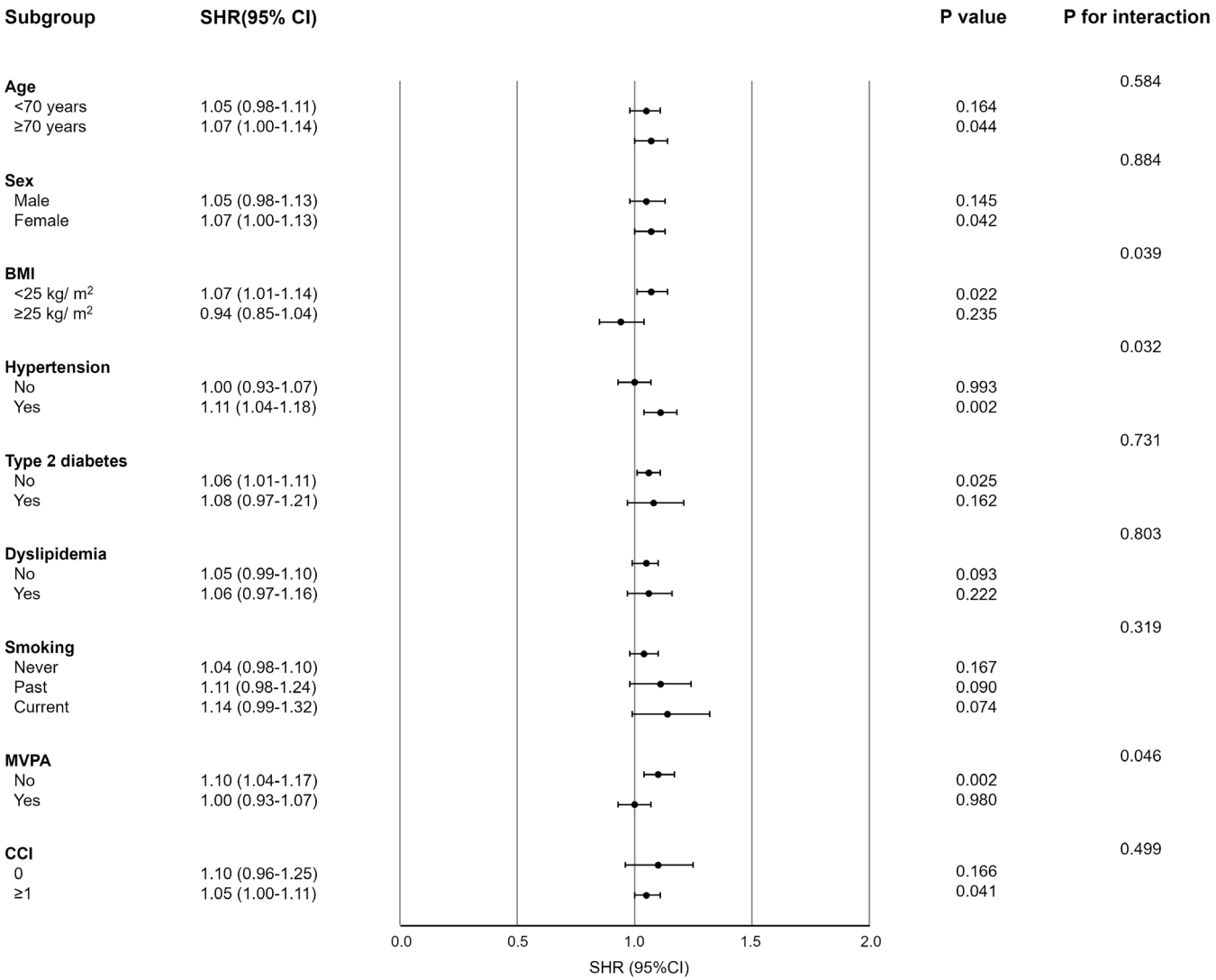

FIGURE 2 | Stratified analyses on the risk of incident SSNHL of MASLD after IPTW. The impact of MASLD and non-SLD on the risk of SSNHL and Meniere's disease was evaluated using Fine and Gray's regression model, accounting for overall mortality as a competing risk. The analysis applied IPTW and adjusted for age, sex, household income, Charlson comorbidity index, smoking, and moderate-to-vigorous physical activity. IPTW, inverse probability of treatment weighting; MASLD, metabolic dysfunction-associated steatotic liver disease; SLD, steatotic liver disease; SSNHL, sudden sensorineural hearing loss.

Ménière's disease include endolymphatic hydrops, vestibular fibrosis, increased intraluminal deposits, auditory cell changes in the cochlea, and neural fiber loss in the spiral osseous lamina [48, 49]. Recently, unexplained sudden fluid shifts have been considered a possible immunological factor in Ménière's disease [50]. These factors could potentially explain the progression of Ménière's disease in patients with SSNHL. Therefore, assessing the association between Ménière's disease and fatty liver disease in affected patients is essential. Since MASLD is identified as an independent risk factor for the development of SSNHL, MASLD patients may need a closer follow-up for auditory tests. By focusing on the newly defined MASLD and its correlation with SSNHL, our research contributes to the growing body of literature that recognizes MASLD as a condition with far-reaching health implications. In addition, our results may be one of the pieces of evidence revealing the chronic low-grade liver inflammation-related liver-ear axis.

A key distinction of this study is that it is the first large-scale cohort study to comprehensively analyze the association between MASLD and auditory impairment. This reclassification underscores the systemic nature of MASLD, linking it not only to liver-related issues but also to significant cardiovascular and metabolic risks [21, 22]. Furthermore, it is important to consider that preventive management of liver health could play a crucial role in reducing the risk of HL. Since liver dysfunction can lead to metabolic imbalances that affect vascular health, maintaining liver health could serve as an essential preventive measure for protecting hearing.

The association between MASLD and an increased risk of SSNHL is supported by evidence linking related metabolic disorders to auditory impairment. MASLD shares common risk factors that have been independently associated with an increased risk of HL, including type 2 diabetes and dyslipidemia

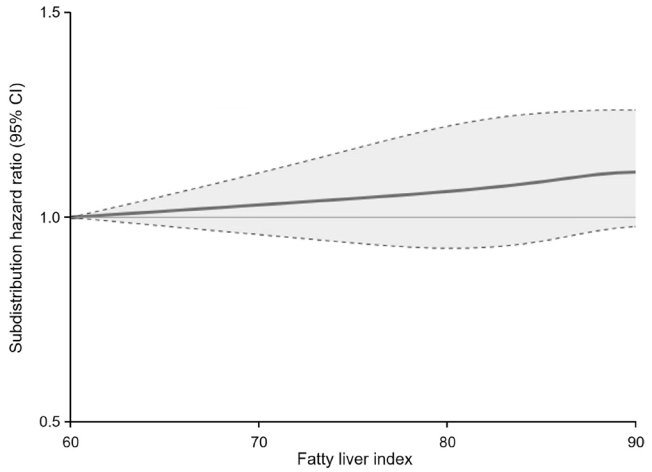

FIGURE 3 | RCS for the association of FLI with the risk of incident SSNHL among individuals with MASLD. Subdistribution hazard ratios (95% CIs) were calculated using the Fine and Gray's regression model after adjustments for age, sex, body mass index, household income, Charlson comorbidity index, smoking, and moderate-to-vigorous physical activity. The solid line typically represents the continuous trend between the predictor and outcome variables, while the dashed lines indicate the 95% CI, visually conveying the statistical reliability of the estimated relationship. CI, confidence interval; FLI, fatty liver index; MASLD, metabolic dysfunction-associated steatotic liver disease; RCS, restricted cubic splines; SSNHL, sudden sensorineural hearing loss.

[51–53]. Postmortem studies have revealed significant pathological changes in the inner ear and central auditory pathway of diabetic patients, including spiral ganglion atrophy, demyelination of the auditory nerve, and vascular lesions [54]. Furthermore, increased blood viscosity due to dyslipidemia has been shown to impair the microcirculation of the cochlea, reducing cochlear blood flow and leading to decreased oxygen and nutrient supply [55]. Additionally, hypertension, another common comorbidity in MASLD, has been linked to an increased risk of HL later in life, particularly when hypertension is present in midlife, highlighting the long-term impact of vascular health on auditory function [56]. These trends were consistent with the findings of our study, suggesting the potential existence of a shared underlying mechanism between MASLD and SSNHL. Hypertension-induced reduced blood flow has been proposed to impair the function of the stria vascularis, disrupting inner ear ionic homeostasis [57]. Such vascular alterations may lead to cochlear ischemic damage, ultimately increasing the susceptibility to SSNHL. Moreover, chronic oxidative stress can exacerbate cochlear damage by promoting sensory hair cell loss [58]. These mechanisms support our findings that hypertension, type 2 diabetes, and dyslipidemia are independent risk factors for the development of SSNHL.

Although the associations did not reach statistical significance, the trends were consistent with previous findings. The study by Seo et al. [59] demonstrates that insulin resistance, β-cell dysfunction, and impaired fasting glucose (IFG) are significantly associated with an increased risk of high-frequency hearing impairment, particularly in males under 70 years of age. Another study demonstrated a significant association between liver fibrosis (particularly related to metabolic conditions like NAFLD) and an increased risk of age-related HL and central auditory processing disorder (CAPD) [22]. In addition, several studies also sought to determine the association between metabolic syndrome and HL [60–65]. Given the aforementioned evidence, it is plausible to argue that MASLD could play a significant role in increasing the risk of HL, particularly SSNHL. The metabolic disturbances commonly associated with MASLD create a pathological environment that may exacerbate the vulnerability of the auditory system to be damaged [51–54]. Chronic liver inflammation, a hallmark feature of MASLD, can contribute to systemic vascular and metabolic dysregulation, which are known to impair microvascular health, including that of the cochlear blood supply [66]. This compromised vascular function may lead to reduced oxygenation and nutrient delivery to the auditory structures, thereby increasing the susceptibility to HL over time [67]. Moreover, insulin resistance and impaired glucose metabolism, frequently observed in MASLD patients, are linked to oxidative stress and inflammation, which can further damage the auditory system [68]. The synergistic effect of these metabolic and vascular insults might explain the observed correlation between MASLD and an increased risk of SSNHL, particularly in individuals with prolonged or severe liver involvement. Therefore, considering the metabolic pathways associated with MASLD and the previously reported associations between metabolic disorders and hearing loss, our findings suggest that MASLD and SSNHL may share common metabolic, vascular, and inflammatory pathways, providing significant support for the proposed liver-ear axis concept.

The primary strength of this study lies in the use of a large prospective cohort, enabled by the KNHIS that allowed for the inclusion of a substantial sample size. Through a 9-year follow-up, the authors enhanced the explanatory power of the association between exposure and outcome by excluding medical histories that could compromise the interpretation of this relationship. The authors also made efforts to evaluate various factors through subgroup analyses, including chronic diseases defined by ICD-10 codes, medications, and questionnaire data such as smoking and physical activity. Additionally, the study focuses on older adults aged 65 and older who often overlook the presence of SLD and metabolic dysfunction. Despite the strength of this study, there are some limitations that need to be considered. First, while the database used in this research offers many advantages compared to other big-data sources, hepatic steatosis was defined using the FLI, which is a surrogate biomarker. Although FLI is widely utilized as a surrogate marker for hepatic steatosis in epidemiological studies (AUROC, 0.844) [43], its validation in the elderly population is still limited. Additionally, since imaging modalities such as ultrasound or liver biopsy data were not available in the KNHIS database, we were unable to use these more direct measures of hepatic steatosis. Future studies incorporating liver biopsy or imaging-based assessments are needed to further validate our findings. Second, the MASLD as defined by the American Association for the Study of Liver Disease (AASLD) is not related to alcohol consumption, meaning that the study may have included patients with MetALD (Metabolic Associated Liver Disease) or ALD (Alcoholic Liver Disease), which limits the ability to accurately represent the prevalence of MASLD. Third, since patients with pre-existing liver diseases were excluded when selecting MASLD patients, the actual impact of MASLD might have been underestimated. Fourth, the study population consisted of health screening candidates who

may be healthier than the others. Fifth, the KNHIS-Senior cohort did not collect individual-level audiometry results as described in previous studies [69, 70], preventing the authors from evaluating the association between MASLD and audiometric conditions within the normal range. Future studies are warranted to confirm whether MASLD deteriorates audiometry condition within the normal range. Sixth, as this study is observational in nature, a causal relationship cannot be definitively established. Finally, given that the observed effect size is relatively small, caution should be taken not to overstate the clinical implications of the study findings. Future studies are needed to investigate whether the risk of HL becomes more pronounced by stratifying patients with metabolic dysfunction into specific risk groups in a stepwise manner.

In conclusion, this study confirmed the association between liver health and ear health, which had been previously understudied in a population-based cohort, through a large-scale cohort. Our results highlight the broader systemic health risks posed by MASLD, extending beyond liver disease to affect auditory health. In practice, these results advocate for routine auditory screenings in elderly patients with MASLD, potentially leading to earlier detection and intervention, thereby improving patient outcomes. Given the observed association, it is important that MASLD may be a modifiable risk factor for SSNHL. Identifying MASLD as a potential target for early intervention could offer new opportunities for mitigating SSNHL risk through lifestyle modifications and medical management. Future research should focus on further elucidating the mechanisms behind the liver-ear axis and exploring targeted therapeutic interventions that address both metabolic dysfunction and hearing preservation.

5 | Conclusion

Patients with MASLD exhibit an elevated risk of SSNHL compared to non-SLD individuals, with risks increasing over time and across various demographic, metabolic, and lifestyle factors. In contrast, Meniere's disease did not yield significant results in association with MASLD. The findings suggest that chronic inflammation, as indicated by elevated FLI, may contribute to the heightened risk of SSNHL in this population.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The datasets were unavailable because the database was provided for research purposes in an anonymized form under strict confidentiality guidelines to authorized personnel.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.