DOI: 10.7860/JCDR/2025/80108.21334

Original Article

Ear, Nose and Throat Section

Association between Dyslipidaemia and Sensorineural Hearing Loss: A Cross-sectional Study

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ABSTRACT

Introduction: Sensorineural Hearing Loss (SNHL) is a common condition with various causes, including issues related to blood flow and metabolism. Recent studies have suggested a possible association between abnormal blood lipid levels and SNHL.

Aim: To assess the association between sensorineural hearing loss and fasting lipid profiles.

Materials and Methods: This cross-sectional study was conducted at Dr. D. Y. Patil Medical College, Pimpri, Pune, Maharashtra, India, from April 1, 2023, to October 30, 2024. It included 38 patients with SNHL and 38 age- and sex-matched patients without hearing loss. Fasting lipid levels, including Total Cholesterol (TC), Low-Density Lipoprotein (LDL), Very Low-Density Lipoprotein (VLDL), High-Density Lipoprotein (HDL), and Triglycerides (TG), were measured in both groups. Pure Tone Audiometry (PTA) was employed to assess the degree of hearing loss. Data were analysed using Chi-square and t-tests, with p-values <0.05 considered statistically significant.

Results: The mean age of participants was comparable between groups, with the SNHL group having a mean age of 52.6±11.4 years and the control group having a mean age of 51.9±10.8 years. The cases had significantly higher mean TC (215.4±32.7 mg/dL), LDL (138.2±24.5 mg/dL), and TG (175.6±40.2 mg/dL) compared to controls. HDL levels were significantly lower in cases (36.8±6.9 mg/dL) compared to controls (44.1±5.8 mg/dL). Additionally, 73.7% (n=28) of cases had HDL <40 mg/dL compared to 21% (n=8) in controls. Dyslipidaemia was significantly associated with increased hearing thresholds across all tested frequencies.

Conclusion: Dyslipidaemia was significantly associated with SNHL, suggesting the importance of monitoring lipid profiles in at-risk individuals. Early intervention may help prevent or mitigate hearing loss.

Keywords: Cholesterol, Hearing impairment, Hearing loss, Hyperlipidaemia, Pure-tone audiometry

INTRODUCTION

The SNHL is a prevalent and typically permanent form of hearing impairment caused by damage to the inner ear or auditory nerve pathways [1]. It can adversely affect an individual's communication skills, cognitive abilities and overall quality of life. While ageing, noise exposure, infections, certain medications and genetic predisposition are well-established causes, emerging evidence indicates that metabolic abnormalities—especially dyslipidaemia—may also contribute significantly to the development of SNHL [1,2]. Lipids are vital for maintaining cell membrane integrity, hormone production and cellular signalling [3]. Dyslipidaemia is a known risk factor for atherosclerosis and related vascular disorders, which may impair cochlear blood supply. The cochlea, being highly metabolically active, is particularly susceptible to ischaemia and oxidative damage [4].

Multiple studies have associated increased levels of LDL, TGs and VLDL with impaired cochlear blood flow and heightened oxidative stress, both of which can lead to damage of cochlear hair cells and auditory neurons [5-7]. In contrast, HDL is thought to offer protective benefits through its roles in cholesterol removal and anti-inflammatory mechanisms [8,9]. In a Mendelian study, Pu K et al., identified a causal relationship between low HDL-C levels and sudden onset SNHL [10]. Additionally, research by Jung W et al., and Doo JG et al., reported an inverse correlation between HDL concentrations and the severity of hearing loss [11,12].

Nevertheless, results have demonstrated variability across different population cohorts, underscoring the influence of regional, genetic and lifestyle factors on the relationship between lipid metabolism and auditory function. For instance, a study conducted by Mudhol RS and Patwegar A in an Indian population reported a statistically significant correlation between elevated serum lipid levels and SNHL, emphasising dyslipidaemia as a modifiable risk factor [5]. Similarly, a cross-sectional study by Silky S et al., investigated 150 patients aged 15 to 60 years with SNHL [6]. The authors reported that serum TC, TGs and LDL were significantly and positively correlated with the severity of hearing loss (p-value <0.05). Interestingly, HDL levels did not show a significant inverse relationship with SNHL severity in their cohort.

Although previous studies have suggested a link between dyslipidaemia and SNHL, there is a paucity of cross-sectional studies from the Indian subcontinent that systematically evaluate this association using matched controls [5,6]. Previous research also lacks population-specific insights, limiting generalisability to diverse demographic settings [6]. The present study addresses this gap by employing a matched cross-sectional design in an Indian tertiary care centre, providing context-specific evidence. Its novelty lies in correlating detailed fasting lipid profiles with SNHL severity, which may help establish dyslipidaemia as a modifiable risk factor and support routine lipid screening in clinical otology practice. The present study enrolled participants aged 30-60 years to focus on those at higher risk for dyslipidaemia and early-onset SNHL while minimising confounding from age-related hearing loss in older adults.

With this background, the present study was conducted to assess the association between SNHL and fasting lipid profiles.

MATERIALS AND METHODS

This cross-sectional study was conducted in the Department of Otorhinolaryngology at Dr. D. Y. Patil Medical College, Pimpri, Pune, Maharashtra, India, over a period of 18 months from April 1, 2023, to October 30, 2024. The study commenced following ethical clearance from the Institutional Ethical Committee (IEC) (IESC/PGS/2023/118).

Inclusion criteria:

- **Cases:** Subjects aged between 30 and 60 years with SNHL were included. SNHL was confirmed using PTA, defined as a ≥25 dB loss in at least two consecutive frequencies with an air-bone gap <10 dB, based on ASHA guidelines (1990) [13].
- **Controls:** Healthy subjects without any SNHL were included as controls.
- All participants had a Body Mass Index (BMI) between 18 and 25 kg/m^2.

Exclusion criteria: Subjects with co-morbidities such as Type 2 Diabetes Mellitus, Chronic Kidney Disease (CKD), or hypertension, middle ear infections, a history of sudden SNHL, noise-induced hearing loss, or exposure to ototoxic drugs were excluded from the study.

Sample size: A total of 76 participants (38 cases and 38 controls) were selected using purposive sampling. The sample size was calculated based on an effect size of 0.70, as reported in the study by Silky S et al., ensuring a statistical power of 80% at a 5% level of significance [6].

Data collection: Data were collected using a structured proforma along with a case record form. Cases and controls were matched on age and Body Mass Index (BMI) to minimise confounding. Fasting lipid markers including TC, HDL, LDL, TG and VLDL were measured and compared across both the groups. Blood samples of 3 to 5 mL were drawn after a ≥12-hour fast and analysed on an automated enzymatic colourimetric analyser. Results were classified per National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) guidelines [14] into normal, borderline and high. Hearing loss severity was graded by PTA following ASHA (1990) [13] as mild (26-40 dB), moderate (41-55 dB), or severe (56-70 dB), and was used to assess associations with dyslipidaemia.

- TC <200 mg/dL
- LDL <100 mg/dL
- HDL >40 mg/dL for males and >50 mg/dL for females
- TG <150 mg/dL
- VLDL <30 mg/dL

The lipid values were interpreted based on the criteria outlined by the NCEP ATP III guidelines [15].

STATISTICAL ANALYSIS

The collected data were initially entered into Microsoft Excel and then analysed using statistical software including Epi Info, Primer and Winpepi. Descriptive methods were applied, wherein continuous data were reported as mean values with standard deviations and categorical data were described using frequencies and percentages. Associations between categorical variables were analysed using the Chi-square test. Differences in continuous variables between the two study groups were assessed with an independent two-tailed Student's t-test. A p-value <0.05 was considered statistically significant, and outcomes were interpreted within a 95% confidence interval.

RESULTS

The comparison of age distribution between cases and controls revealed no statistically significant difference (p-value=0.645). A similar trend was observed for BMI categories, where the distribution between cases and controls was also not significantly different (p-value=0.844). However, gender distribution showed a statistically significant difference (p-value <0.001), with a higher proportion of males in the case group and females in the control group [Table/Fig-1].

SNHL cases exhibited a significantly more atherogenic lipid profile than controls, with higher mean TC, LDL, TG, and VLDL (independent-samples Student's t-test; p-value ≤0.002) and lower mean HDL

Parameters	Cases 38 (100%)	Controls 38 (100%)	p-value
Age range (years)			
30-39	8 (21.05)	10 (26.32)	0.645
40-49	12 (31.58)	14 (36.84)	
50-60	18 (47.37)	14 (36.84)	
BMI (kg/m²)			
18-20	8 (21.05)	10 (26.32)	0.844
21-23	18 (47.37)	16 (42.11)	
24-25	12 (31.58)	12 (31.58)	
Gender distribution			
Male	25 (65.79)	13 (34.21)	<0.001
Female	13 (34.21)	25 (65.79)	

[Table/Fig-1]: Distribution of age, BMI and gender among cases and controls.

(p-value <0.001), supporting an association between dyslipidaemia and auditory dysfunction [Table/Fig-2].

Lipid parameter	Cases (Mean±SD)	Controls (Mean±SD)	p-value
Total Cholesterol (TC) (mg/dL)	215.4±32.7	192.7±28.3	0.002
LDL (mg/dL)	138.2±24.5	122.5±20.3	0.001
HDL (mg/dL)	36.8±6.9	44.1±5.8	<0.001
Triglycerides (TG) (mg/dL)	175.6±40.2	152.3±35.7	0.002
VLDL (mg/dL)	35.29±12.59	25.68±10.07	<0.001

[Table/Fig-2]: Mean lipid profile values in cases and controls.

The distribution of hearing loss severity based on lipid profile status showed no statistically significant association (p-value=0.65). Among individuals with dyslipidaemia, 10 had mild, 12 had moderate, and eight had severe hearing loss. In contrast, among those with normal lipid profiles, the majority had milder forms: four mild, three moderate, and one severe case. All participants in the control group had normal hearing, including 31 with dyslipidaemia and seven with normal lipid levels. Although a higher proportion of moderate to severe hearing loss was observed among dyslipidaemic individuals, the association was not statistically significant [Table/Fig-3].

Hearing loss severity	Group	Dyslipidaemia	Normal lipid profile	p-value
Mild	Case	10	4	0.65
Moderate	Case	12	3	
Severe	Case	8	1	
Normal (no loss)	Control	31	7	

[Table/Fig-3]: Hearing loss severity (In number of patients).

Across all tested frequencies (500-4000 Hz), SNHL cases had significantly higher mean PTA thresholds than controls (e.g., 500 Hz: 35.4±5.2 vs. 15.2±4.8 dB; 4000 Hz: 55.3±8.1 vs. 16.7±5.0 dB; all p=0.001 by independent-samples Student's t-test), indicating markedly impaired hearing sensitivity in the case group [Table/Fig-4].

Frequency (Hz)	Cases (Mean±SD)	Controls (Mean±SD)	p-value
500	35.4±5.2	15.2±4.8 dB	0.001
1000	42.1±6.8	14.5±3.9 dB	0.002
2000	48.9±7.3	14.8±4.1 dB	0.003
4000	55.3±8.1	16.7±5.0 dB	0.004

[Table/Fig-4]: Mean Pure Tone Audiometry (PTA) thresholds in cases and controls.

SNHL cases had significantly more low HDL (<40 mg/dL: 73.7% vs. 21%) and elevated VLDL (>30 mg/dL: 73.7% vs. 26.4%; p-value <0.001) than controls. They also showed higher rates of hypertriglyceridaemia (TG ≥150 mg/dL: 76.3% vs. 39.5%) and LDL derangements (LDL ≥130 mg/dL: 73.6% vs. 36.8%), with severe hypercholesterolaemia (TC ≥240 mg/dL) more common in cases

(21.1% vs. 5.3%). Collectively, these derangements underscore the markedly atherogenic lipid profiles in SNHL patients, reinforcing the role of dyslipidaemia in auditory dysfunction [Table/Fig-5].

Variables	Cases (n=38)	Controls (n=38)
VLDL (mg/dL)		
≤30	10 (26.4%)	28 (73.7%)
31-40	14 (36.8%)	7 (18.4%)
>40	14 (36.8%)	3 (7.9%)
Total Cholesterol (TC) (mg/dL)		
<200	15 (39.5%)	22 (57.9%)
200-239	15 (39.5%)	14 (36.8%)
≥240	8 (21%)	2 (5.3%)
LDL (mg/dL)		
<100	2 (5.3%)	1 (2.6%)
100-129	8 (21%)	23 (60.5%)
130-159	20 (52.6%)	13 (34.2%)
160-189	7 (18.4%)	1 (2.6%)
≥190	1 (2.6%)	0
HDL (mg/dL)		
<40	28 (73.7%)	8 (21%)
40-59	10 (26.3%)	30 (79%)
Triglycerides (TG) (mg/dL)		
<150	9 (23.7%)	23 (60.5%)
150-199	23 (60.5%)	12 (31.6%)
≥200	6 (15.8%)	3 (7.9%)

[Table/Fig-5]: Range-wise distribution of lipid parameters {VLDL, Total Cholesterol (TC), LDL, HDL, and Triglycerides (TG)} among cases and controls.

DISCUSSION

This cross-sectional study investigated the correlation between fasting lipid profiles and SNHL in adults aged 30-60 years, revealing a significant association between dyslipidaemia and the presence and severity of SNHL. The findings contribute to the growing evidence that metabolic disturbances, particularly altered lipid metabolism, may play a pivotal role in cochlear dysfunction [5,6]. A striking finding of this study was the significantly higher levels of TC, LDL, TG, and VLDL observed in SNHL patients compared to matched controls, accompanied by significantly lower HDL levels. These lipid abnormalities suggest a possible link between atherogenic profiles and compromised cochlear microcirculation, leading to progressive or sudden auditory damage.

These results are consistent with the study by Silky S et al., who found that patients with SNHL had significantly elevated serum cholesterol, TG, and LDL, with greater hearing impairment corresponding to worse lipid profiles [6]. Their findings, based on a cross-sectional Indian population, underscore the relevance of lipid monitoring in otological assessment. Similarly, Quaranta N et al., reported that higher TC levels were associated with delayed or incomplete recovery in patients with idiopathic sudden SNHL, indicating a prognostic role for hyperlipidaemia [8].

The elevated LDL values seen in this study support the conclusions of Li X et al., who demonstrated that LDL is a critical metabolic factor contributing to SNHL. LDL is known to impair endothelial function and promote atherosclerosis, potentially compromising the cochlear blood supply [7]. These effects are particularly concerning in the inner ear, where oxygen demand is high and collateral circulation is limited. Kaneva AM et al., further supported this association, proposing the atherogenic index as a predictive marker of idiopathic SNHL, reinforcing the diagnostic and prognostic significance of lipid subfractions [9].

The observation of higher TG levels in cases echoes the findings of Odeh OI et al., who identified hypertriglyceridaemia as a significant predictor of SNHL in their Nigerian cohort [4]. TG-related vascular changes may impair cochlear perfusion, leading to ischaemic damage and hair cell degeneration. Similarly, Lee JS et al., found that elevated TGs and obesity were independently associated with an increased risk of sudden SNHL, reflecting a shared metabolic pathway [15].

Notably, the present study found a marked reduction in HDL levels in SNHL patients compared to controls. HDL is widely recognised for its anti-inflammatory, antioxidant and endothelial-protective functions. The findings are in concordance with the Mendelian randomisation study by Pu K et al., which established a causal link between reduced HDL and increased risk of SNHL [10]. Quaranta N et al., also reported better auditory recovery in patients with higher HDL, suggesting its protective role against cochlear injury [8].

Although few studies focus on VLDL specifically, the current study demonstrated significantly elevated VLDL levels among cases, which may reflect broader dysregulation in lipid transport and metabolism. This is partially supported by trends noted by Silky S et al., who reported overall atherogenic lipid profiles among SNHL patients, including elevated VLDL [6].

An additional finding of this study was the pattern of hearing loss severity in relation to lipid status. Patients with dyslipidaemia exhibited more severe hearing thresholds across all tested frequencies. These results support those of Mudhol RS and Patwegar A, who observed progressive SNHL associated with worsening lipid profiles, reinforcing the potential dose-dependent relationship between dyslipidaemia and hearing impairment [5].

Present study findings were further supported by large-scale studies such as those by Jung W et al., and Doo JG et al., which emphasised the role of lipid-related biomarkers in predicting hearing outcomes [11,12]. The Chinese Medical Association (2015) also included lipid profile evaluation in their clinical guidelines for managing sudden SNHL, highlighting its diagnostic relevance [16].

The novelty of this study lies in its region-specific contribution. Despite a growing body of international evidence, studies examining lipid-hearing associations in Indian populations remain limited. By employing a matched case-control design and comprehensive lipid panel analysis, this study bridges an important gap in local literature and emphasises the need to consider metabolic factors in audiological practice.

While the cross-sectional design limits causal inference, the observed associations are biologically plausible and strongly supported by external literature. Prospective longitudinal studies and interventional trials are warranted to further explore whether correction of lipid abnormalities could improve auditory outcomes or prevent the progression of SNHL.

The present study highlights a clear and clinically meaningful association between dyslipidaemia and SNHL. These findings reinforce the need for routine lipid screening in patients at risk of hearing loss and suggest that early metabolic interventions may offer a preventive strategy in audiological care. As metabolic disorders continue to rise globally, integrating otological assessment into the broader framework of lifestyle and cardiovascular health becomes increasingly relevant.

Limitation(s)

This study was limited by its cross-sectional design, which precludes establishing a causal relationship between dyslipidaemia and SNHL. The relatively small sample size and single-centre setting may affect the generalisability of the findings. Potential confounders such as dietary patterns, physical activity and cardiovascular risk factors were not fully evaluated. Additionally, only PTA was used for hearing assessment, without advanced audiological tests. Future studies with larger, multicentre cohorts and longitudinal follow-up are recommended.

CONCLUSION(S)

Present study showed a clear link between dyslipidaemia and SNHL. Compared with individuals without hearing loss, those with SNHL had higher levels of TC, LDL and TGs, and lower levels of HDL. Changes in these lipid levels were associated with hearing loss, suggesting that dyslipidaemia may play a role in causing or worsening SNHL. The findings indicate that it is important to closely monitor lipid levels in individuals at risk of hearing loss. Early detection and treatment of dyslipidaemia (through diet, lifestyle changes, or medications) may help prevent or slow the progression of hearing loss.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]

- Plagiarism X-checker: May 14, 2025
- Manual Googling: Jun 18, 2025
- iThenticate Software: Jun 17, 2025 (1%)

ETYMOLOGY: Author Origin

EMENDATIONS: 7

AUTHOR DECLARATION:

- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Apr 22, 2025**
Date of Peer Review: **May 21, 2025**
Date of Acceptance: **Jun 19, 2025**
Date of Publishing: **Aug 01, 2025**