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Research Article

Hearing as a Marker of Vascular Aging: Can Audiograms Predict Cardiovascular and Inflammatory Risk? A Narrative Review

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Abstract

Background: Hearing loss is typically managed within otolaryngology, yet emerging evidence suggests that specific audiometric patterns—particularly low-frequency sensorineural hearing loss (LFSNHL)—may reflect underlying vascular and inflammatory disease. We're used to thinking of hearing loss as a local problem. But what if certain hearing patterns are actually telling us about broader systemic risks? In this paper, we explore how low-frequency hearing loss (LFSNHL) and sudden sensorineural hearing loss (SSNHL) may serve as clinical clues pointing to dyslipidemia and low-grade inflammation—especially elevated hs-CRP. We propose that audiograms can serve as early warning signs of vascular aging and outline a practical algorithm for screening and management that's easy to integrate into ENT and primary care workflows.

Objective: To examine relationships between audiometric tests, dyslipidemia, and inflammatory markers (e.g., high-sensitivity Creactive protein [hs-CRP]), and to propose a clinically actionable screening framework for otolaryngologists and primary care.

Methods: Narrative review of epidemiologic, mechanistic, and interventional literature linking hearing impairment with cardiovascular risk factors, lipid profiles, and systemic inflammation; emphasis on LFSNHL and sudden sensorineural hearing loss (SSNHL).

Results: LFSNHL and SSNHL are associated with dyslipidemia, elevated inflammatory markers, and surrogate measures of vascular aging. Elevated lipids or CRP predict poorer auditory outcomes in several types of hearing loss; preliminary data suggest lipid-lowering and anti-inflammatory interventions may improve prognosis in select patients.

Conclusion: In this narrative review, audiometric findings —particularly LFSNHL and unexplained SSNHL—should prompt lipid and hs-CRP testing to detect systemic disease earlier. We offer a decision algorithm to guide screening and interdisciplinary management.

Keywords: Audiometry; Low-Frequency Hearing Loss; Sudden Sensorineural Hearing Loss; Dyslipidemia; C-Reactive Protein; Vascular Aging; Longevity

Introduction

Hearing loss affects communication, independence, and quality of life, and its prevalence rises steeply with age. Audiograms don't just help localize pathology—they can offer insight into systemic health.

The cochlea is metabolically active and totally dependent on a single artery. No backup flow. That makes it uniquely vulnerable to microvascular disease, lipid dysregulation, and chronic inflammation.

Traditionally, clinicians interpret the audiogram for site-of-lesion localization and rehabilitation planning.

When we see symmetric, unexplained low-frequency loss or sudden hearing drops, we shouldn't just think about the inner ear only—we should think about the blood vessels that feed it. Several studies suggest these hearing patterns correlate with cardiovascular risk, lipid derangements, and elevated inflammatory markers.

This paper pulls together the evidence and lays out a simple plan: when to screen, what to look for, and how treating systemic disease that might also protect hearing.

We reviewed the literature connecting hearing loss to dyslipidemia, endothelial dysfunction, and systemic inflammatory markers such as hs-CRP. We argue that the audiogram can serve as an early, non-invasive window into cardiovascular and metabolic risk, and we provide a practical algorithm intended to change practice: when clinicians see specific audiometric patterns, they should reflexively consider ordering a lipid panel and hs-CRP and collaborating with primary care and cardiology.

Cochlear microcirculation and vascular vulnerability

The labyrinthine artery, typically a branch of the anterior inferior cerebellar artery, supplies the cochlea via terminal branches lacking collateral flow, no redundancy or safety net. The stria vascularis demands continuous, high-efficiency perfusion. That means even subtle vascular aging—like reduced nitric oxide, increased stiffness, or endothelial dysfunction—can affect hearing. Histologic studies confirm capillary rarefaction and strial atrophy in age-related hearing loss. In other words, your audiogram might be an early readout of your microvasculature. Histopathology in presbycusis often reveals strial atrophy and capillary loss, consistent with a vascular contribution to age-related hearing loss.

Audiometric phenotypes that raise red flags

Low-frequency hearing (250–1000 Hz) is often overlooked, but when it's symmetric and unexplained, it deserves a closer look [1-3,6-8]. Friedland., et al. found low/flat audiograms were more predictive of cardiovascular disease than typical high-frequency patterns [1]. Quaranta., et al. linked higher cholesterol to worse mid-high frequency thresholds [2]. Other datasets (Beaver Dam, Nash SD) show that arterial stiffness, diabetes, and metabolic syndrome which are accelerators of vascular aging have worse hearing, especially in the low ranges [3,6].

Sudden Sensorineural Hearing Loss, Lipids, and Inflammation

SSNHL is a known emergency, and we treat it with corticosteroids first. But elevated hs-CRP levels predict poorer recovery, as shown in Chen., *et al.* [4]. Fibrinogen may also play a role [9]. Several studies show that patients with SSNHL have worse lipid panels—higher LDL, triglycerides, and ApoB. And while we lack RCTs, retrospective data suggest that statins may help recovery in dyslipidemic patients.

This adds another layer: if you're managing SSNHL, don't stop at steroids and MRI. Check a lipid panel [10]. Check hs-CRP. If they're abnormal, treat accordingly. It's a simple addition that might change long-term outcomes.

Methods

This was a focused narrative review. We conducted a literature search of PubMed, Google up. Search terms included combinations of: "sudden sensorineural hearing loss (SSNHL)", "low-frequency sensorineural hearing loss (LFSNHL)", "lipid profile", "cholesterol", "triglycerides", "inflammation", "C-reactive protein", and "colchicine".

We included English-language studies that examined hearing loss patterns—especially LFSNHL and SSNHL—in relation to lipid profiles and markers of inflammation. Priority was given to studies with defined audiometric outcomes, biochemical markers, and either population-level or interventional data. We excluded case reports, abstracts without full text, and studies that did not directly assess hearing outcomes in relation to lipid or inflammatory markers

Results

We found nine key studies that consistently supported the association between hearing loss and systemic risk- Table 1.

Major findings

- Low-frequency loss and flat audiograms correlate with cardiovascular disease risk.
- Dyslipidemia (elevated TC, LDL-C, TG, ApoB) shows up more often in hearing-impaired populations.
- Elevated hs-CRP and fibrinogen are linked to worse SSNHL recovery.
- Statin exposure appears to improve hearing outcomes in dyslipidemic SSNHL cohorts.

Proposed clinical algorithm

We propose a simple 4-step model:

- **Trigger:** Patient presents with LFSNHL or SSNHL.
- Immediate management: Steroids (for SSNHL), MRI if indicated.
- Systemic screen: Order lipid panel (TC, LDL, HDL, TG, ApoB), hs-CRP, ± fibrinogen and HbA1c.
- Management and follow-up: Treat systemic abnormalities, counsel lifestyle, and repeat audiogram in 3–6 months to assess response.

Ref	Year	Population	Audiometric Findings	Biomarkers	Main Outcomes
1. Friedland., et al.	2009	General population, older adults	Low/flat audiograms cor- related with CVD risk	Cardiovascular risk factors	Low/flat audiograms predictive of CVD
2. Nash., et al.	2011	Population cohort	Hearing impairment, worse in low frequencies	Diabetes, meta- bolic syndrome	Metabolic risk factors associated with hearing loss
3. Quaranta., et al.	2015	SSNHL patients	Mid-high frequency thresholds	Total cholesterol	High cholesterol correlated with poorer SSNHL recovery
4. Chen., <i>et al</i> .	2023	Meta-analysis of SSNHL patients	Multiple audiometric outcomes	hs-CRP, inflamma- tory biomarkers	Elevated CRP predicted poorer SSNHL prognosis
5. Shao., <i>et al</i> .	2021	Prospective SSNHL cohort	SSNHL audiograms	Serum lipids (TC, LDL, TG)	Elevated lipid levels predicted worse prognosis
6. Bainbridge., et al.	2008	NHANES cohort	Hearing impairment prevalence	Diabetes	Diabetes significantly associated with hearing impairment
7. Gates., et al.	1993	Elderly population cohort	Audiometric thresholds	CVD and risk fac- tors	Presence of CVD linked to worse hearing
8. Kim., et al.	2021	National health screening cohort	Hearing impairment incidence	Statin use	Statin use associated with reduced hearing impairment risk
9. Kanzaki., et al.	2014	SSNHL patients	Hearing recovery out- comes	Fibrinogen	High fibrinogen correlated with poorer recovery
10. Han., et al.	2021	Type 2 diabetic pa- tients	Hearing loss incidence	Statin use	Statins protective against SNHL in diabetics

Table 1: Summary of Included Studies on Hearing Loss, Lipids, and Inflammation.

Discussion

The big idea here is that hearing loss may be your first hint of vascular trouble. We already use eye exams to find diabetic retinopathy. Why not use audiograms to detect early vascular dysfunction?

Treating dyslipidemia and inflammation may help preserve hearing—especially in sudden loss.

We're not proposing a new standard of care—yet. But we are suggesting that checking a lipid panel and CRP when you see these hearing patterns is low-cost, low-risk, and potentially high-yield. It could improve patient care beyond the ear.

Therapeutically, the rationale for lipid lowering and inflammation reduction extends beyond cardiovascular event prevention to potential auditory benefits. Observational signals linking statin use to better SSNHL outcomes underscore the need for prospective trials. Meanwhile, the low risk and high systemic benefit of guideline-directed lipid therapy justify screening and treatment when indicated.

Finally, a longevity lens positions ENT practice at the forefront of preventive medicine: preserving hearing preserves cognition, social engagement, and independence—each a pillar of extended health span.

It's important to distinguish the systemic patterns of low-frequency hearing loss we describe from classic otologic causes that also affect the low-frequency range. In this paper, we focus on idiopathic, symmetric, and often subtle low-frequency sensorineural hearing loss (LFSNHL) that lacks accompanying vestibular symptoms, tinnitus, or aural fullness—patterns that may represent early microvascular compromise or chronic low-grade inflammation. These cases often present in middle-aged or older adults and can be easily missed or dismissed as benign presbycusis unless viewed through a systemic lens. In contrast, conditions such as Meniere's disease, perilymph fistula, large vestibular aqueduct syndrome (LVAS), and autoimmune inner ear disease typically present with more overt vestibular symptoms, asymmetry, fluctuating thresholds, or steroid responsiveness. Conductive mimics like otitis media with effusion or third-window phenomena (e.g., superior canal de-

hiscence) can also cause apparent low-frequency hearing loss but are distinguishable via tympanometry or imaging. By differentiating these classic local pathologies from the more subtle, systemic low-frequency patterns, clinicians can better identify patients who may benefit from lipid screening, inflammatory markers, and preventive cardiometabolic interventions.

Additionally, in my prior publication on Lyme disease and otologic manifestations, I emphasized the importance of screening for infectious etiologies—such as Lyme disease—in patients with unexplained hearing loss or vertigo [11]. While this paper focuses on vascular and inflammatory biomarkers, the broader principle remains: audiometric findings often reflect systemic pathology.

Future research directions

While current data primarily support lipid-lowering and inflammatory markers as prognostic tools, additional therapeutic strategies warrant exploration. For example, low-dose colchicine, widely used in cardiology to reduce systemic inflammation and hs-CRP, has not yet been tested in patients with LFSNHL or SSNHL. Given its favorable cardiovascular outcomes and plausible mechanistic link to cochlear microcirculation, colchicine represents a logical candidate for future clinical trials.

Beyond pharmacotherapy, lifestyle and dietary interventions that lower systemic inflammation should also be studied for their potential auditory benefits. Additionally, EHR-based alerts and artificial intelligence approaches may help flag high-risk audiograms for automated cardiovascular and metabolic screening.

Limitations and future directions

This narrative review is limited by heterogeneity in audiometric definitions, reliance on observational data, and potential confounders (e.g., noise exposure, ototoxic drugs). Randomized trials testing lipid-lowering or anti-inflammatory strategies for hearing outcomes are needed. But the signal is consistent: vascular and inflammatory markers track with certain hearing patterns. We need trials—statins, colchicine, even dietary interventions. We need EHR-based alerts when certain audiograms show up. We need AI to help flag risk. Machine learning approaches may help identify high-risk audiograms for automatic systemic screening prompts within electronic health records.

Integrating cardiovascular and otologic care

For ENT specialists, this means that an abnormal audiogram can trigger cardiovascular and inflammatory screening as reflexively as an abnormal ECG prompts troponin testing.

For primary care providers, incorporating audiometry into annual wellness visits could identify at-risk patients earlier than standard CV screening protocols.

Therapeutic implications

- Dyslipidemia: Statins may improve SSNHL recovery in dyslipidemic patients and reduce long-term hearing decline risk.
- Inflammation: Elevated CRP correlates with poorer SSNHL prognosis; systemic inflammation reduction (e.g., through anti-inflammatory diet, exercise, pharmacotherapy) may benefit both hearing and cardiovascular outcomes.
- Dual benefit: Addressing these risk factors serves both the auditory system and cardiovascular healthspan.

Conclusion

The audiogram may be more than a test of hearing—it may be a marker of vascular health. If you see low-frequency loss or SSNHL without a clear local cause, consider systemic testing. You might catch early cardiovascular disease. You might improve hearing outcomes. You might extend a patient's healthspan.

Proposed Shift in Practice

Audiograms should be interpreted not only for site-of-lesion localization but also for systemic risk stratification. In the same way that ophthalmologists screen for diabetic retinopathy, otolaryngologists and PCPs can use the audiogram as a tool for vascular and inflammatory disease detection.

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