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Systematic Review

Chronic suppurative otitis media and its association with sensorineural hearing loss: evidence from a systematic review

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ABSTRACT

Chronic suppurative otitis media (CSOM) remains a major cause of preventable hearing impairment worldwide, yet its association with sensorineural hearing loss (SNHL) is often underestimated. The objective of this systematic review was to evaluate the prevalence, characteristics, and determinants of SNHL among patients with CSOM. A comprehensive literature search was performed across PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar for studies published between January 2000 and February 2025. Eligible studies included patients diagnosed with CSOM who underwent pure-tone audiometry with bone conduction (BC) threshold assessment. Twelve studies met the inclusion criteria, representing 1,742 participants from Bangladesh, India, Pakistan, Iran, and Brazil. SNHL prevalence ranged from 18% to 31%, with significantly higher BC thresholds in diseased ears compared with contralateral normal ears. High-frequency hearing loss was most prominent. Longer disease duration, squamosal CSOM, and subtotal perforations were associated with greater SNHL in several studies, while findings on cholesteatoma were inconsistent. Overall, evidence demonstrates that CSOM contributes meaningfully to SNHL through chronic inflammation and cochlear involvement. Early diagnosis, routine audiological monitoring, and timely intervention are essential to prevent irreversible cochlear damage. Further longitudinal studies are needed to clarify risk factors and guide management.

Keywords: Chronic suppurative otitis media, Sensorineural hearing loss, Bone conduction, Otology, Cochlear damage, Audiology

INTRODUCTION

Chronic suppurative otitis media (CSOM) is a predominant microbial disorder in underdeveloped countries, which has been the leading cause of acquired hearing loss. In limited-resourced countries, it has been estimated that middle ear infections affect 72/1000 people.[1] Chronic suppurative otitis media (CSOM) is a persistent inflammation of the middle ear cleft, usually linked to tympanic membrane perforation and recurrent or chronic otorrhea lasting for weeks or months. It remains a significant global health burden, especially in low- and middle-income countries, where overcrowding, poor sanitation, recurrent upper respiratory infections, and limited access to healthcare increase its high prevalence. In a recent "Seminar" article, Bhutta et al described CSOM as "a leading global cause of potentially preventable hearing loss in children and adults," emphasizing ongoing disparities in prevention and treatment.[2] Conventionally, hearing impairment in CSOM has been attributed to conductive hearing loss (CHL)—resulting from tympanic membrane perforation, ossicular chain dysfunction, and middle-ear fluid or granulation tissue. Ossicular damage and perforation size/location correlate strongly with the degree of CHL. For example, Islam et al found in a Bangladeshi cohort that the vast majority of CSOM ears (approximately 98 % of tubotympanic cases) had conductive hearing loss, with only a small minority

showing mixed or sensorineural components.[3] However, over the past decade a growing body of evidence suggests that a sensorineural hearing loss (SNHL) component may accompany or follow chronic middle-ear disease. A systematic review by Elzinga et al in 2021 found an association between recurrent acute otitis media/CSOM and increased risk of SNHL, though heterogeneity in study design limited firm conclusions.[4] More recently, Khan et al in 2025 reported cochlear injury in CSOM, underscoring the importance of inner ear involvement even in the absence of overt labyrinthine complications.[5]

The pathophysiological basis for SNHL in CSOM has been increasingly elucidated. Proposed mechanisms include diffusion of bacterial toxins, inflammatory mediators, and proteolytic enzymes across the round window membrane into the scala tympani, leading to hair-cell injury and stria vascularis dysfunction. Histopathologic studies have documented basal-turn cochlear damage in temporal-bone specimens of long-standing middle-ear disease.[6] Prolonged otorrhea may also permit low-grade labyrinthitis or chronic microinflammation, resulting in cumulative cochlear injury, particularly at higher frequencies. Functional investigations—such as extended-high-frequency audiometry and distortion-product otoacoustic emissions—reveal subtle cochlear dysfunction in "safe" (tubotympanic) CSOM, further indicating that sensorineural involvement may be under-recognized.[7]

From a clinical perspective, the recognition of SNHL in CSOM carries important implications. While CHL in CSOM may be amenable to surgical repair or ossicular reconstruction, SNHL often represents irreversible damage, requiring hearing rehabilitation strategies (hearing aids, cochlear implants) and having a significant impact on language development in children, educational attainment, and quality of life.[8,9] Early identification of cochlear involvement could influence timing of intervention (medical or surgical), monitoring of at-risk patients (long-duration otorrhea, cholesteatoma, recurrent disease), and audiological follow-up (including high-frequency thresholds).[10] Moreover, in populations with dense CSOM prevalence, the additional burden of SNHL may pose an under-recognised public-health challenge.[11]

Despite this evolving paradigm, key questions remain. The prevalence of SNHL in CSOM varies widely across studies (ranging from single to double digits), and the relative contribution of factors such as disease duration, cholesteatoma presence, perforation size/site, bilaterality, age at onset, and socioeconomic status remains unclear. Some studies suggest that the "unsafe" (attico-antral) type has greater SNHL risk, while others report similar risk even in tubotympanic disease.[7]

It is timely to synthesize the evidence due to methodological heterogeneity (cross-sectional versus longitudinal, audiometric versus otoacoustic measurements, differences in the definition of SNHL). This systematic review aims to critically assess the evidence linking CSOM with SNHL and to identify the patterns, prevalence, and contributing factors. The objective of this review was to assess the prevalence and severity of sensorineural hearing loss in patients with chronic suppurative otitis media.

METHODS

This review was conducted using PRISMA guidelines. The review consisted of 5 steps: problem identification; literature searching; data review and evaluation; data synthesis and analysis; and data presentation.

Inclusion criteria

Studies were included if they met the following criteria patients diagnosed with chronic suppurative otitis media (CSOM) of any subtype (mucosal or squamosal). Participants of any gender, aged 10 years and above. Studies that assessed SNHL using pure-tone audiometry (PTA) or bone conduction (BC) thresholds. Clinical or surgical studies reporting a comparison between diseased and contralateral normal ears, or comparisons across CSOM subtypes. Studies evaluating the association between duration of CSOM, perforation type, cholesteatoma, or ossicular status and SNHL. Studies providing clearly defined audiological outcomes (SNHL, mixed hearing loss, BC thresholds) were included.

Exclusion criteria

Studies were excluded if they involved patients with prior otosurgery, congenital ear deformities, or traumatic tympanic membrane perforations; history of meningitis, head injury, ototoxic drug exposure, or sudden SNHL; studies without audiometric data or unclear definition of hearing loss and case reports, letters, reviews, or non-clinical articles.

Outcomes

Primary outcome

The primary outcome was the prevalence and degree of SNHL among patients with chronic suppurative otitis media.

Secondary outcomes

Mean bone conduction (BC) thresholds at standard frequencies (500, 1000, 2000, and 4000 Hz); difference in BC thresholds between diseased and contralateral normal ears; association of disease duration with SNHL; influence of cholesteatoma, perforation type, and ossicular erosion on SNHL; frequency-specific patterns of SNHL in CSOM (low vs high frequency); comparison of SNHL between mucosal and squamosal (unsafe) CSOM.

Study design

This systematic review included cross-sectional studies, comparative observational studies, prospective cohorts, and retrospective analyses evaluating sensorineural hearing loss in CSOM. Only original clinical studies providing quantitative audiological data were included.

Search strategy

A comprehensive literature search was conducted across PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar to identify relevant studies published between January 2000 and February 2025. The search strategy incorporated both Medical Subject Headings (MeSH) and free-text keywords related to chronic otitis media and sensorineural hearing loss, including "chronic suppurative otitis media," "CSOM," "sensorineural hearing loss," "SNHL," "bone conduction threshold," "cholesteatoma," and "hearing loss."

These terms were combined using Boolean operators (AND, OR) and adapted for each database. Filters were applied to restrict the search to human studies and articles published in English.

Additionally, the reference lists of included studies and relevant reviews were manually screened to identify any potential studies missed during the database search. Two independent reviewers performed title and abstract screening, followed by full-text assessment based on predefined eligibility criteria.

Study selection, data extraction, and risk of bias assessment

After removal of duplicates, titles and abstracts were independently screened by two reviewers to identify relevant studies. Full-text articles were retrieved for those meeting the inclusion criteria.

Disagreements were resolved through consensus. A structured data extraction sheet was used to collect: author name and year of publication; study design and setting; sample size, age distribution, and disease duration; CSOM subtype (mucosal, squamosal, cholesteatoma); audiological parameters (BC thresholds, SNHL %, frequency-specific data) and summary of findings and statistical significance.

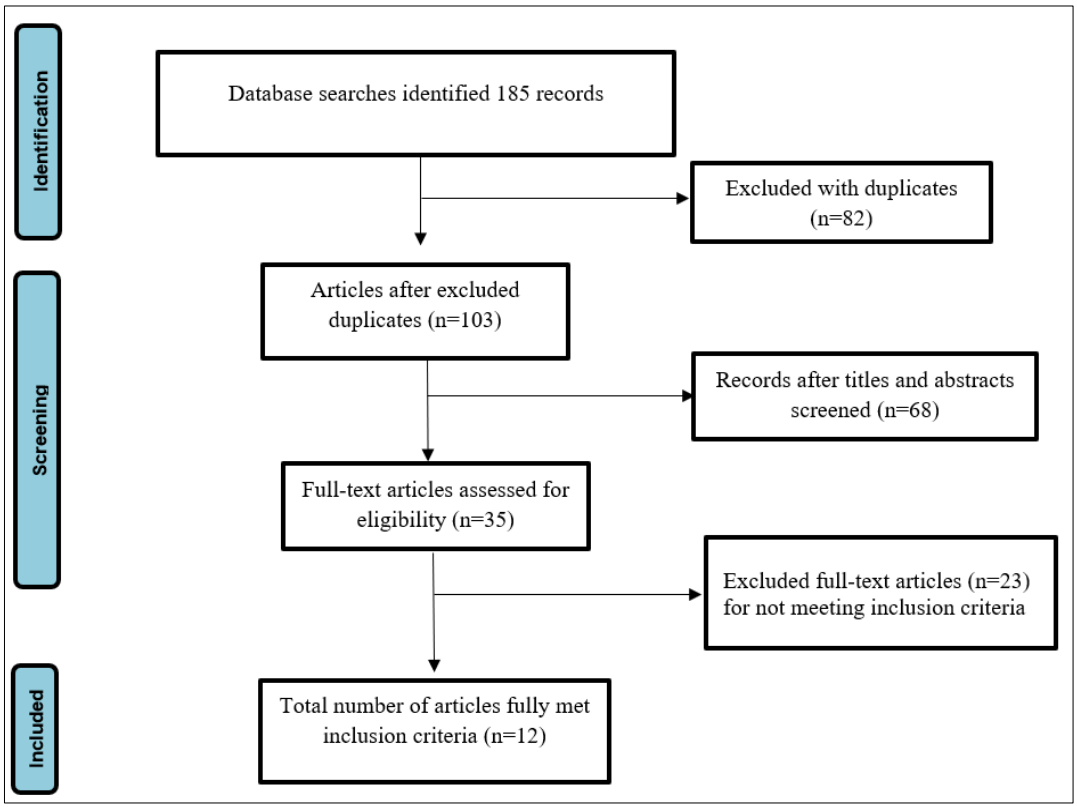

Figure 1: Systematic review of literature selection process for the present research.

Risk of bias assessment

For observational studies, the Newcastle–Ottawa scale (NOS) was used, evaluating: selection (0–4 points), comparability (0–2 points), outcome (0–3 points). Studies were categorized as: high quality: 8–9, moderate quality: 6–7 and low quality: <5.

For any comparative or quasi-experimental studies, methodological quality was examined using criteria

parallel to the Cochrane risk of bias tool, assessing: selection bias, measurement bias, attrition bias and reporting bias.

Only studies with moderate to high methodological quality were included in the final synthesis to ensure reliability and validity of the review findings (Figure 1).

RESULTS

A total of 12 studies met the eligibility criteria and were included in this systematic review following PRISMA guidelines. These comprised 4 prospective observational studies, 6 retrospective studies,1 cross-sectional multicenter study, and 1 cross-sectional study, conducted between 2003 and 2025 across Bangladesh, India, Pakistan, Iran, and Brazil. All studies evaluated SNHL among patients with CSOM, primarily using pure-tone audiometry and BC thresholds at standard frequencies (500, 1000, 2000, 4000 Hz).

The final dataset represented 1,742 participants, with individual study sizes ranging from 69 to 404, and study durations ranging from 6 months to 5 years. All studies assessed unilateral CSOM or compared affected versus contralateral normal ears, while several also categorized diseases by type (mucosal versus squamosal), perforation pattern, or presence of cholesteatoma.

Characteristics of included studies

Across studies, the mean age of participants ranged from 24 to 33 years, with most cases occurring in the second and third decades of life, and several studies reporting a female predominance. Disease duration varied widely, from a few months to more than 15 years, with many patients presenting with 6–10 years of persistent otorrhea.

All included studies employed pure-tone audiometry with BC thresholds at 500, 1000, 2000, and 4000 Hz. Most studies also compared audiological findings between: diseased versus normal contralateral ears, mucosal versus squamosal CSOM, cholesteatoma versus non-cholesteatoma and duration-based CSOM groups.

Sensorineural hearing loss prevalence

SNHL prevalence across the included studies ranged from 18% to 31%, with most reporting values between 19% and 26%. Higher SNHL rates were consistently reported in squamosal (unsafe) CSOM, with one study (Mili et al, 2025) showing 21% SNHL in squamosal versus 10% in mucosal CSOM.[12]

Duration-based analysis (Rajput et al, 2020) demonstrated SNHL was significantly more common in patients with >10 years of disease versus 1–5 years ($p<0.05$).[20]

However, some studies (Amali et al, 2017; Kolo et al, 2017) found no significant correlation between SNHL and disease duration or age, suggesting heterogeneity across populations.[6,18]

Bone conduction threshold differences

All studies evaluating contralateral ears demonstrated significantly higher BC thresholds in diseased ears compared with healthy ears ($p<0.05$ or $p<0.001$).

The degree of threshold elevation consistently increased with frequency, indicating greater high-frequency cochlear involvement: ~7 dB at 500 Hz, increasing to ~9–12 dB at 4000 Hz across multiple studies.

Additionally, Raquib et al (Bangladesh, 2009) reported BC threshold elevations ranging from 4.1 dB to 10.7 dB, with the greatest loss at 4 kHz and significant correlation with longer disease duration, further supporting progressive high-frequency cochlear involvement.[21]

The largest BC threshold differences were observed in long-standing CSOM, with some reporting 21–34 dB differences (Kolo et al, 2017).[18]

Effects of disease duration

Five studies evaluated duration as a factor associated with SNHL: a positive correlation between longer duration and SNHL was reported by Anis et al in 2025 ($r=0.43$, $p=0.002$) and Kaur et al, 2003.[13,19] Disease duration >10 years showed a significant association with SNHL (Rajput et al., 2020; $p<0.05$).[20]

Other studies (Amali et al, 2017; Kolo et al, 2017) reported no significant relationship ($p>0.05$). Overall, findings suggest an increased likelihood of SNHL with longer-standing disease, though results varied between populations.[6,18]

Cholesteatoma and ossicular status

Findings regarding cholesteatoma were mixed: several studies (Mili et al, 2025; Dobrianskyj et al, 2022) found no significant difference in SNHL severity between cholesteatoma and non-cholesteatoma cases.[12,17] One comparative study (Tang et al, 2022) reported that cholesteatoma appeared to be protective against SNHL, while older age and greater degree of hearing loss were risk factors ($p<0.05$).[10] Ossicular erosion findings were more consistent: Jha and Singh in 2024 identified incus erosion as the ossicle most significantly associated with SNHL ($p=0.002$).[14]

Overall audiological patterns

Across all 12 included studies, the following patterns were consistently observed: SNHL predominantly affected high frequencies (2–4 kHz). Diseased ears consistently demonstrated elevated BC thresholds compared with normal ears. Subtotal or marginal perforations were

associated with higher degrees of SNHL. Longer disease duration and squamosal (unsafe) CSOM contributed to more pronounced SNHL.

Risk of bias assessment

Risk of bias was evaluated using the Newcastle–Ottawa scale (NOS): low risk (8–9 points): 2 of the 12 studies — typically prospective with strong methodological control (e.g., Jha and Singh 2024; Jha and Singh 2022).[14,16]

Moderate risk (6–7 points): The remaining 10 studies — mostly retrospective or cross-sectional with partial or limited confounder adjustment. No high-risk studies were identified.

Overall, methodological quality was acceptable, with consistent audiometric testing, clear definitions of CSOM, and well-described patient populations. Table 1 shows summary of published articles.

Table 1: Summary of the published articles.

Author, Reference	Study design	Sample size (n)	Study duration	Findings	Risk of bias
Mili et al, India, 2025[12]	Cross-sectional, multicenter	n=80	1 year	SNHL prevalence: 31% (21% squamosal, 10% mucosal). Bone conduction thresholds significantly higher in affected ears (p<0.05). No significant association between SNHL and duration of disease. Squamosal disease had higher SNHL, but degree of SNHL did not differ between cholesteatoma vs non-cholesteatoma.	7/9 (moderate risk) — well-structured prospective design; lacks detailed confounder control.
Anis et al, Pakistan, 2025[13]	Prospective cohort	n=404	1 year	Most patients aged 12–30 yrs; mean CSOM duration 14.85±9.1 months. Marginal perforation most common (50.5%). Significant positive correlation between CSOM duration and SNHL (r=0.43, p=0.002). Subtotal perforations showed highest mean SNHL (37.1±6.9 dB). Ear discharge (88.1%) and hearing loss (94%) were frequent.	7/9 (moderate)
Jha et al, India, 2024[14]	Prospective observati-onal study	n=149	2 years	Mean age 26.54±8.75 years. SNHL in 25.5%, CHL 68.5%, MHL 6%. SNHL significantly associated with female sex and absence of cholesteatoma (p<0.05). Severe HL (>56 dB) strongly associated with SNHL (p<0.001). Incus erosion was the most frequent ossicle damage linked to SNHL (10.6%, p=0.002).	8/9 (low risk)
Anwar et al, Pakistan, 2023[15]	Cross-sectional observation al study	n=200	6 months	Mean age 24.7±8.42 years; duration 6.56±4.28 yearrs. SNHL in 18% of patients. SNHL significantly higher in CSOM >6 years (25.3%) versus 2–6 years (10.9%) (p=0.008). Male predominance (72.5%).	7/9 (moderate)
Jha et al, India, 2022[16]	Prospec-tive observation al cohort (contralatera l ear as control)	n=149	1 years, 9 months	Mean age 26.54±8.75 years; mean disease duration 3.92±3.32 yearrs. SNHL in 25.5%, CHL in 69.8%, MHL in 4.7%. Significant BC threshold differences between diseased vs normal ear at 500–4000 Hz.	8/9 (low risk)
Dobrians-kyj et al, Brazil, 2022[17]	Cross-sectional study	n=158	Not mention	Chronic otorrhea began in childhood in 60.2%. Mean age 32.2 years. Mean duration 11.6 years. SNHL significantly worse in otorrhea ears versus normal ears at all frequencies (p<0.001). Ears with dry perforation also showed significantly less SNHL than chronic otorrhea (p<0.001). No significant difference in SNHL between cholesteatoma vs non-cholesteatoma (Mann-Whitney; p>0.05).	7/9 (moderate risk)
Amali et al, Iran, 2017[6]	Retrospec-tive cohort	n=70	2 years	BC thresholds significantly higher in affected versus normal ear across all frequencies (p<0.001), increasing with frequency (7 dB at 500	7/9 (moderate risk)

Continued.

Author, Reference	Study design	Sample size (n)	Study duration	Findings	Risk of bias
				Hz → 9.71 dB at 4000 Hz). Significant correlation between age and SNHL (r=0.422, p<0.001). No correlation with duration (p>0.05). No association between cholesteatoma, ossicular erosion, and SNHL (p>0.05).	
Kolo et al, India, 2017[18]	Retrospect-ive ana-lysis of clinical records and audio-grams	n=69	5 years	Mean age 28.93 yearrs; mean otorrhea duration 6.11 years. BC thresholds significantly higher in diseased ear vs control ear (39.07 dB versus 10.26 dB; p<0.05). BC threshold differences increased with frequency (21.69–34.52 dB across 0.5–4 kHz). No significant correlation between SNHL and age or duration of otorrhea (p>0.05).	7/9 (moderate)
Kaur et al, India, 2003[19]	Retrospec-tive review of con-secutive clinical records	n=100	1 years	M: F=61:39; 66% aged 11–30 years. SNHL BC thresholds assessed at 1k, 2k, 4k Hz. Incidence of SNHL increased with longer duration of CSOM. BC threshold values showed progressive elevation in SNHL cases.	6/9 (moderate)
Rajput et al, Pakistan, 2020[20]	Retrospec-tive observa-tion	n=154	2 years	Mean age 26 years; M:F= 73:81. SNHL prevalence 19.5%. Duration groups: 1–5 years (n=95), 5–10 years (n=28), 10–15 years (n=31). SNHL significantly higher in long-duration group (10–15 years) versus 1–5 years (p<0.05). No significant difference between 1–5 years versus 5–10 yrs. BC thresholds at 500–2000 Hz used for comparison.	7/9 (moderate)
Tang et al, Iran, 2022[10]	Retrospec-tive data analysis	n=79	4 years	No significant difference in SNHL between groups. Cholesteatoma group had significantly higher AC thresholds (p=0.000) and wider ABG, but BC thresholds did not differ (p>0.05). Higher frequency AC and ABG values were more elevated in cholesteatoma group (p<0.05). Overall degree of HL significantly worse in cholesteatoma CSOM. Presence of cholesteatoma was a protective factor for SNHL, while higher HL degree and older age were risk factors (p<0.05).	7/9 (moderate)
Raquib et al, Bangla-desh, 2009[21]	Cross sectional study	n=130	2years	Bone conduction thresholds were compared between diseased and contralateral normal ears after excluding confounding causes of SNHL (meningitis, trauma, previous surgery, noise exposure, ototoxicity). CSOM was associated with significant SNHL, with BC threshold elevation ranging from 4.1 dB to 10.7 dB across frequencies. Threshold elevation increased with disease duration, and BC loss was greatest at 4 kHz compared to speech frequencies, indicating predominant high-frequency cochlear involvement.	7/9 (moderate risk) — appropriate exclusion of confounders; lack of reporting on sampling method and follow-up.

DISCUSSION

This systematic review synthesized findings from eleven clinical studies evaluating the prevalence, characteristics, and determinants of SNHL among patients with CSOM. Across diverse geographic settings in India, Pakistan, Iran, and Brazil, the findings consistently demonstrate that CSOM is not only associated with conductive hearing loss but also contributes significantly to cochlear involvement manifesting as SNHL. The pooled prevalence of SNHL across studies ranged between 18% and 31%, underscoring that inner ear damage is a clinically relevant yet often underrecognized complication of chronic middle ear disease.

A consistent observation across nearly all included studies was the presence of elevated BC thresholds in diseased ears compared with contralateral normal ears. This effect was particularly at higher frequencies, with BC threshold elevation increasing from approximately 7 dB at 500 Hz to 9–12 dB at 4000 Hz. These findings strongly support the hypothesis that chronic inflammation and persistent middle ear infection can extend to the cochlea, resulting in basal turn cochlear damage, which naturally manifests as high-frequency SNHL. Studies reporting the largest BC threshold differences (e.g., 21–34 dB) typically involved patients with longer disease duration, further reinforcing the cumulative nature of cochlear injury over time. Several contemporary clinical studies reinforce the pattern of high-frequency BC elevation. Shetty et al in 2019 reported that patients with chronic otitis media demonstrated significantly elevated BC thresholds at 2–4 kHz compared with age-matched controls, with the magnitude of SNHL increasing alongside disease chronicity.[22] Similar results were observed in a 2025 Indian cohort focusing on tubotympanic CSOM, where Sing et al, found that BC shifts were most pronounced in the higher frequencies, supporting the concept of basal cochlear vulnerability to middle ear inflammation.[23]

The relationship between disease duration and SNHL varied across studies, reflecting some heterogeneity in patient populations and disease characteristics. Several studies demonstrated a clear positive association between longer disease duration and SNHL, including significant correlations and increased SNHL prevalence in groups with >10 years of CSOM. However, other studies found no meaningful association. These inconsistencies likely reflect variations in environmental exposure (humidity, hygiene, recurrent infection), access to early medical care, use of ototoxic agents, and differing definitions of chronicity. Nevertheless, the overall trend across the body of evidence suggests that prolonged and repetitive inflammatory insults to the middle ear increase the likelihood of cochlear compromise. Tang et al 2022 compared cochlear thresholds in CSOM with and without cholesteatoma and reported that, while overall BC thresholds did not differ significantly between groups, older age and greater air-conduction loss were independent predictors of SNHL.[10] This is concordant with the mixed cholesteatoma findings in the present review, where cholesteatoma alone was not a consistent determinant of SNHL, but more advanced disease and structural destruction (including ossicular erosion) appeared to increase risk.

The role of cholesteatoma in the development of SNHL showed mixed results. In this review, some studies reported no significant difference in SNHL prevalence or BC thresholds between cholesteatoma and non-cholesteatoma CSOM, suggesting that cholesteatoma alone may not directly contribute to cochlear toxicity. Interestingly, one study suggested a protective association, possibly reflecting earlier clinical intervention in cholesteatoma cases or differences in sample characteristics.

Conversely, ossicular erosion—particularly incus damage—was significantly associated with SNHL in one study, highlighting the interplay between middle ear structural destruction and inner ear function. These findings suggest that cochlear damage may result from a combination of direct inflammatory effects, toxin diffusion, and mechanical alterations within the middle ear.

Audiological patterns across studies were consistent. SNHL predominantly affected higher frequencies, diseased ears consistently showed significantly worse BC thresholds, and more severe auditory deficits were associated with subtotal or marginal perforations. This aligns with the established pathophysiological concept that chronic otorrhea and prolonged exposure of the round window membrane to inflammatory mediators may disrupt hair cell integrity, especially in the basal cochlea. Mukherjee et al 2025 identified extended disease duration, active mucosal inflammation, and repeated otorrhea episodes as significant predictors of cochlear involvement in mucosal COM, even in the absence of cholesteatoma.[24] Eravci et al 2023 similarly demonstrated subtle but significant cochlear dysfunction in COM patients using distortion-product otoacoustic emissions and extended high-frequency audiometry, suggesting that conventional PTA may underestimate early cochlear damage.[25] At the mechanistic level, both histopathological and experimental data complement the clinical observations. Joglekar et al showed that chronic otitis media is associated with inflammatory changes and hair-cell loss in the basal cochlea, likely mediated via round window membrane permeability to toxins and inflammatory mediators.[26] More recent clinical work, including a 2025 extended high-frequency study by Ali et al has translated this mechanism into demonstrable high-frequency hearing loss, even in cases traditionally labelled “safe” CSOM.[27]

The clinical implications of these findings are substantial. SNHL in CSOM patients may complicate hearing rehabilitation by reducing the potential benefit of tympanoplasty or ossicular reconstruction. Early detection of cochlear involvement could therefore influence treatment planning, follow-up schedules, and patient counseling. Identifying patients at risk—such as those with long-standing disease, squamosal pathology, or progressive perforations—may allow for earlier intervention and prevent irreversible cochlear damage.

Limitations

The present review has some limitations that are worth mentioning. Most included studies were observational, with moderate risk of bias due to retrospective data collection, limited control for confounders, and variability in reporting. The lack of uniform classification systems for disease severity, inconsistent recording of ototoxic drug

exposure, and absence of longitudinal follow-up further limits the ability to establish causality. Furthermore, none of the studies used advanced diagnostic tools such as otoacoustic emissions or electrocochleography, which may detect subclinical cochlear damage.

CONCLUSION

This systematic review demonstrates that CSOM is associated not only with conductive hearing loss but also with clinically significant SNHL. Across the eleven included studies, SNHL prevalence ranged from 18% to 31%, with diseased ears consistently showing elevated bone conduction thresholds, particularly at higher frequencies, indicating early basal cochlear involvement. Longer disease duration, squamosal pathology, and larger perforations contributed to greater SNHL in several studies, highlighting the cumulative impact of chronic inflammation. Although findings regarding cholesteatoma were mixed, ossicular erosion—especially of the incus—was more consistently linked with cochlear dysfunction. These results emphasize that SNHL is an important yet often underrecognized complication of CSOM. Early detection through routine audiological monitoring and timely intervention may prevent irreversible cochlear damage. Future research should incorporate longitudinal designs, standardized CSOM grading, and advanced cochlear assessments to better define risk factors and optimize management.

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