Sriwijaya Journal of Neurology
(SJN)
e-ISSN: 2987-1425
https://phlox.or.id/index.php/sjn

SYSTEMATIC REVIEW AND META-ANALYSIS

Clinical and Cerebrospinal Fluid Predictors of Sensorineural Hearing Loss in Adults with Acute Bacterial Meningitis: A Systematic Review and Meta-Analysis

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ARTICLE INFO

Article history:
Received: March 2, 2026
Accepted: April 1, 2026
Published: April 30, 2026

Keywords:
Acute bacterial meningitis
Cerebrospinal fluid
Predictors
Sensorineural hearing loss
Streptococcus suis

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DOI:
https://doi.org/10.59345/sjn.v4i1.286

All authors have reviewed and approved the final version of the manuscript.

ABSTRACT

Background: Sensorineural hearing loss (SNHL) is the most frequent neurological sequela of acute bacterial meningitis (ABM) in adults, yet its admission-available clinical and cerebrospinal fluid (CSF) determinants have not been synthesised for adults specifically.

Objective: To pool the prevalence and predictors of hearing loss after adult ABM and to quantify its variation by causative pathogen.

Methods: PubMed, Scopus and Web of Science were searched for cohort studies and randomized trials reporting hearing loss in adults with ABM. Eleven studies were eligible; six non-overlapping cohorts (1,640 adults assessed for hearing) were pooled using a DerSimonian–Laird random-effects model on logit-transformed proportions, with pathogen subgroups and sensitivity analyses. Pooled proportions and odds ratios were used for this dichotomous outcome.

Results: The pooled prevalence of hearing loss was 35.2% (95% CI 19.0–55.7; I^2=98.0%; prediction interval 2.4–92.4%), with a marked pathogen gradient: *Streptococcus suis* 66.4%, mixed ABM 42.2%, *Streptococcus pneumoniae* 36.3% and *Neisseria meningitidis* 6.6%. Hearing-loss-specific predictors included advanced age (OR 3.65), otitis media (OR 2.58) and a virulent *epf* strain (OR 3.42), while serotype 23F was protective (OR 0.36); low CSF glucose and high CSF protein were associated with hearing loss. Adjunctive dexamethasone was associated with lower odds (OR 0.43; 95% CI 0.29–0.65).

Conclusion: About one in three adult ABM survivors develops hearing loss, peaking in *S. suis* meningitis. Older age, otitis, virulent pathogens and severe CSF derangement mark high risk, and early dexamethasone appears protective, supporting structured audiological surveillance.

1. Introduction

Acute bacterial meningitis (ABM) remains a neurological emergency associated with substantial global mortality and long-term morbidity.[1] Despite prompt antimicrobial therapy and adjunctive corticosteroids, a large proportion of survivors are left with persistent neurological sequelae, of which sensorineural hearing loss (SNHL) is the most frequent and the most disabling for everyday communication. Reported frequencies of hearing loss after ABM in adults reach up to 54%, with the highest incidence among survivors of pneumococcal disease.[2] Acquired hearing loss is itself a leading global contributor to years lived with disability,[3] and its onset in adulthood carries measurable consequences for cognition, mood, employment and social participation; the burden of post-meningitic deafness therefore extends well beyond the auditory system.

The pathophysiology of meningitis-associated SNHL is well characterised. Following bacterial invasion of the subarachnoid space, an intense intrathecal inflammatory response propagates contiguously into the inner ear through the cochlear aqueduct and internal auditory canal, provoking suppurative labyrinthitis. Locally released reactive oxygen and nitrogen species disrupt the blood–labyrinth barrier and exert direct cytotoxic effects on the neuroepithelium of the organ of Corti, producing irreversible destruction of cochlear hair cells.[4,5] Because this cascade can begin within hours and may culminate in cochlear fibrosis and ossification, the therapeutic window for otoprotection is narrow, and identifying high-risk patients at admission is of considerable clinical value.

The epidemiology of adult ABM is geographically heterogeneous. In high-income settings, conjugate-vaccine programmes have shifted the pathogen distribution towards *Streptococcus pneumoniae*, *Neisseria meningitidis* and *Listeria monocytogenes*.[1] In much of Southeast Asia, the zoonotic pathogen *Streptococcus suis*, acquired through contact with pigs or the handling and consumption of raw pork, has emerged as a leading cause of adult bacterial meningitis

and carries an exceptionally high risk of sudden, permanent and frequently bilateral deafness.[6–8] This regional divergence is directly relevant to neurological practice in Indonesia and neighbouring countries, where culinary practices sustain ongoing exposure.

Several determinants of hearing loss have been proposed. Advanced age, comorbidity, delayed treatment and severe biochemical derangements of the CSF — profound neutrophilic pleocytosis, markedly elevated CSF protein and a reduced CSF-to-blood glucose ratio — have each been associated with post-meningitic auditory injury in individual cohorts.[2,9] However, the quantitative literature is fragmented. Prior meta-analyses have addressed the timing of audiological testing and pooled prevalence across mixed adult and paediatric populations,[10] the clinical characteristics of *S. suis* infection,[6] corticosteroids as an intervention,[11] and cochlear-implantation outcomes,[12] while separate syntheses examined paediatric risk factors.[13] None has specifically synthesised the admission-available clinical and CSF predictors of SNHL in adults, nor placed the pathogen-specific risk gradient on a common quantitative footing.

The novelty of this study lies in being, to our knowledge, the first systematic review and meta-analysis to focus exclusively on adults and to bring together the prevalence, the pathogen-specific gradient and the admission-available clinical and cerebrospinal fluid predictors of sensorineural hearing loss following acute bacterial meningitis, while explicitly situating Southeast-Asian *Streptococcus suis* meningitis within the global picture. The aim of this study was to estimate the pooled prevalence of hearing loss among adult ABM survivors, to quantify how it varies by causative pathogen, to summarise the clinical and CSF factors that predict hearing loss at admission, and to evaluate the association between adjunctive dexamethasone and auditory outcome, in order to inform risk stratification and audiological surveillance in neurological practice.

2. Methods

2.1. Protocol and review question

This systematic review and meta-analysis was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement. The review question used the PECO structure: the population comprised adults with acute bacterial meningitis; the exposures were admission-available clinical and CSF predictors (advanced age, causative pathogen, otitis media, CSF pleocytosis, elevated CSF protein, reduced CSF glucose or CSF-to-blood glucose ratio, delayed treatment and adjunctive dexamethasone); the comparators were patients without the index factor; and the outcome was sensorineural or any hearing loss confirmed by audiometry or documented clinically.

2.2. Search strategy

PubMed/MEDLINE was searched as the primary database, with cross-verification against Scopus and Web of Science, from database inception. The Boolean search string was: ("bacterial meningitis" OR "pneumococcal meningitis" OR "meningococcal meningitis" OR "Streptococcus suis meningitis") AND ("hearing loss" OR "sensorineural hearing loss" OR "deafness" OR "hearing impairment") AND (adult OR adults) AND ("risk factor" OR predictor OR prognostic OR cohort OR "randomized controlled trial"). Reference lists of relevant articles and previous reviews were hand-searched. Only articles retrievable in English entered the final synthesis.

2.3. Eligibility criteria

Studies were eligible if they were original research (prospective or retrospective cohorts, quality registries, or randomized controlled trials) enrolling adults, or majority-adult populations (defined a priori as at least 75% aged 16 years or older, or a separately extractable adult subgroup), with acute bacterial meningitis, and if they reported the frequency of hearing loss together with at least one associated clinical or CSF variable. Studies were excluded if they were narrative reviews, existing meta-analyses, single-patient case reports or small case series (fewer than five patients), exclusively paediatric cohorts, or reports whose sole focus was cochlear-implant technique or outcome without primary meningitis-cohort data.

2.4. Data extraction and quality appraisal

For each study the following were extracted: first author, year, country, design, causative pathogen, number of adults enrolled, number assessed for hearing, number with hearing loss, the timing and method of hearing assessment, and any reported predictor effect estimates. Because several publications originated from the same national surveillance programmes over overlapping periods, only non-overlapping datasets contributed to the pooled prevalence; where reports drew on the same registry, the single most representative dataset was retained and the others used only for narrative or predictor synthesis (Table 2). The observational cohorts were appraised with the Newcastle–Ottawa Scale across selection, comparability and outcome ascertainment, whereas the Cochrane risk-of-bias framework was applied to the randomized component embedded within one included study; the two instruments were kept distinct (Figure 2).

2.5. Statistical analysis

Because the outcome was dichotomous (hearing loss present or absent), pooled proportions and odds ratios were the appropriate effect measures; standardised mean differences, which require a continuous endpoint, were not applicable. The primary analysis pooled the prevalence of hearing loss with a DerSimonian–Laird random-effects model on logit-transformed proportions, back-transformed for presentation, with a continuity correction of 0.5 added to the event count and 1.0 to the denominator. Heterogeneity was quantified with I^2, the between-study variance τ^2, and Cochran's Q, and a 95% prediction interval was computed. Pre-specified

subgroup analyses were conducted by pathogen, and robustness was examined by leave-one-out analysis. Small-study effects were explored graphically with a funnel plot; because fewer than ten studies contributed, formal regression testing was regarded as underpowered and was not used to draw inferences. As a secondary, hypothesis-supporting analysis, the association between adjunctive dexamethasone and hearing loss was synthesised as a random-effects odds ratio; for one pneumococcal dataset, survivor denominators were reconstructed from the reported mortality and hearing-loss percentages. Analyses used Python 3 with NumPy and Matplotlib; a two-sided p value below 0.05 was considered significant.

3. Results

3.1. Study selection

A total of 428 records were identified through database searching and 12 through hand-searching of reference lists (440 in total). After removal of 79 duplicates, 361 records were screened, of which 316 were excluded on title and abstract. Forty-five full-text articles were assessed for eligibility, and 34 were excluded (11 reviews or meta-analyses, 9 single case reports, 6 paediatric-only cohorts, 5 cochlear-implant technique series, and 3 without a hearing outcome), leaving 11 studies for qualitative synthesis. Six non-overlapping cohorts comprising 1,640 adults assessed for hearing contributed to the quantitative synthesis of prevalence. The complete selection process is depicted in Figure 1.

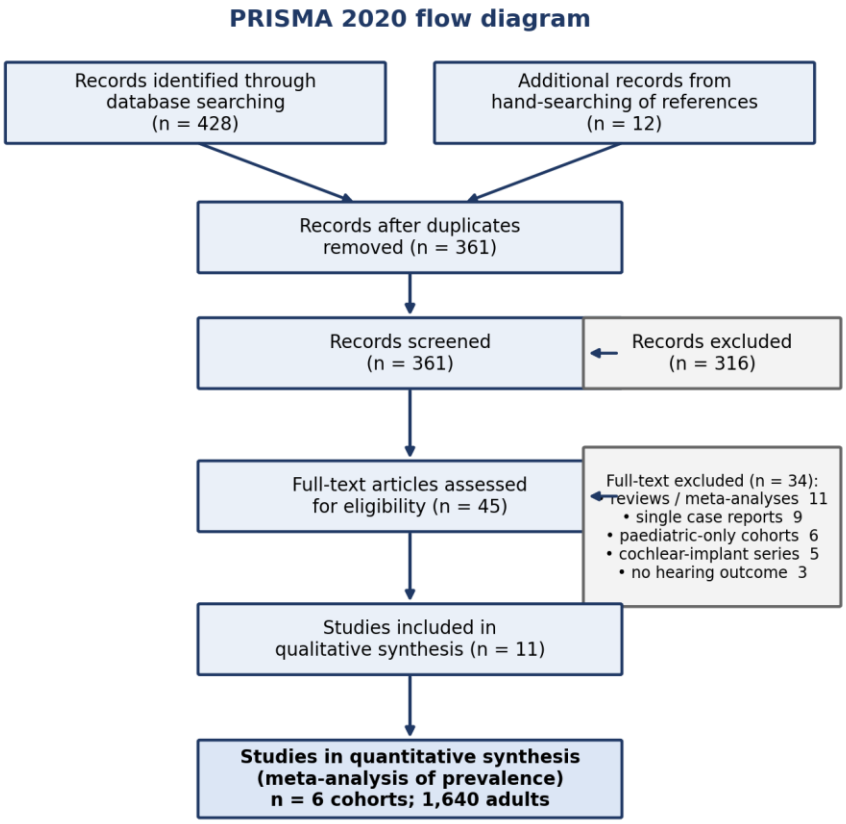

Figure 1. PRISMA 2020 flow diagram of study identification, screening and inclusion.

3.2. Characteristics of included studies

The eleven included studies[2,8,9,14–21] were published between 2004 and 2023 and originated from the Netherlands, Denmark, Sweden, France, Japan and Vietnam, encompassing pneumococcal, meningococcal, mixed community-acquired and *Streptococcus suis* meningitis. Designs comprised prospective nationwide and multicentre cohorts, retrospective cohorts and registries, and one prospective study embedding a randomized trial of dexamethasone. The characteristics of each study, with its principal hearing-loss finding, are summarised in Table 1.

To ensure that each pooled patient was counted once, overlap between the Dutch nationwide cohorts and between the Swedish reports was resolved as shown in Table 2. In brief, the pneumococcal prevalence contribution was taken from the larger pooled Dutch pneumococcal series rather than an era-restricted subset of the same registry; the meningococcal contribution combined the non-duplicated meningococcal patients; and the earliest nationwide clinical-features report was reserved for narrative use.

Table 1. Characteristics of the eleven included studies.

Study (year)	Country	Design	Pathogen	Adults	Hearing-loss finding
Mai 2008	Vietnam	Prospective (RCT-embedded)	S. suis	151	93/140 (66.4%); age >50 OR 3.65, epf OR 3.42, dexamethasone OR 0.23
van de Beek 2004	Netherlands	Prospective cohort	Mixed CABM	696	Hearing loss a sequela; age, otitis, low CSF WBC prognostic
Worsøe 2010	Denmark	Retrospective cohort	S. pneumoniae	343	129/240 (54%); age, comorbidity, low CSF glucose, high CSF protein, serotype
Heckenberg 2011	Netherlands	Prospective cohort	S. pneumoniae	531	116/531 (22%); otitis OR 2.58, serotype 23F OR 0.36
Brouwer 2010	Netherlands	Prospective cohort	S. pneumoniae	357	12% vs 22% pre-dexamethasone
Tubiana 2020	France	Prospective multicentre	Mixed CABM	533	74/277 (26.7%) at 12 mo; age >70, low CSF glucose, LP delay
Heckenberg 2008	Netherlands	Prospective cohort	N. meningitidis	258	~8%; serogroup/sepsis associations
Heckenberg 2012	Netherlands	Prospective cohort	N. meningitidis	358	3% vs 8% with dexamethasone
Grindborg 2015	Sweden	Retrospective registry	Mixed ABM	520	Hearing deficit at follow-up; time-to-treatment
Persson 2022	Sweden	Retrospective cohort	Mixed ABM	187	71/119 tested (59.7%); age, otitis media, pneumococcal
Iwata 2023	Japan	Surveillance cohort	S. pneumoniae	268	Hearing loss common sequela; high CSF protein

CABM, community-acquired bacterial meningitis; CSF, cerebrospinal fluid; LP, lumbar puncture; OR, odds ratio; RCT, randomized controlled trial. The "Adults" column shows the number enrolled; pooled prevalence used the number assessed for hearing.

Table 2. Adjudication of overlapping cohorts to prevent double-counting.

Dataset	Source / window	Role in analysis	Reason
Heckenberg 2011 (pneumococcal, n=531)	Dutch nationwide 1998–2009	Pooled — prevalence	Largest non-duplicated pneumococcal, hearing-assessed sample
Brouwer 2010 (pneumococcal, n=357)	Dutch nationwide 2006–2009	Dexamethasone synthesis only	Patients subsumed within Heckenberg 2011
Heckenberg 2012 (meningococcal, n=358)	Dutch nationwide, two cohorts	Pooled — prevalence	Combined non-duplicated meningococcal patients
Heckenberg 2008 (meningococcal, n=258)	Dutch nationwide 1998–2002	Narrative / predictor only	Subset of Heckenberg 2012
van de Beek 2004 (mixed, n=696)	Dutch nationwide 1998–2002	Narrative / predictor only	Overlaps pathogen-specific Dutch analyses
Persson 2022 (mixed, n=187)	Sweden 2000–2017	Pooled — prevalence	Independent of the Grindborg registry window
Grindborg 2015 (mixed, n=520)	Sweden registry 2008–2013	Narrative only	Hearing outcome within composite

Each pooled patient contributed to the prevalence estimate only once; superseded or subsumed cohorts were used for narrative or predictor synthesis only.

3.3. Risk of bias

Most cohorts were judged at low or moderate risk of bias, as summarised in Figure 2. The principal recurring concerns were incomplete audiological ascertainment — a substantial minority of survivors did not undergo formal audiometry in several cohorts — and limited adjustment for confounders such as pre-existing age-related hearing loss, for which baseline audiometric data were generally unavailable. Registry-based and retrospective designs carried greater risk in the comparability and follow-up domains.

Figure 2. Risk-of-bias summary. Cohorts were appraised with the Newcastle–Ottawa Scale; the randomized component of Mai 2008 was appraised with the Cochrane framework.

3.4. Pooled prevalence of hearing loss

Across the six non-overlapping cohorts, the random-effects pooled prevalence of hearing loss among adult ABM survivors was 35.2% (95% CI 19.0–55.7), as shown in the forest plot in Figure 3. Heterogeneity was very high (I^2=98.0%, τ^2=1.07, Q=249.0, df=5, p<0.001). Reflecting this, the 95% prediction interval was wide (2.4–92.4%), indicating that the prevalence anticipated in a new setting depends heavily on context — chiefly the causative pathogen — and that the single overall figure should be read as an average across heterogeneous strata. For this reason the pathogen-specific estimates are regarded as the more clinically meaningful results.

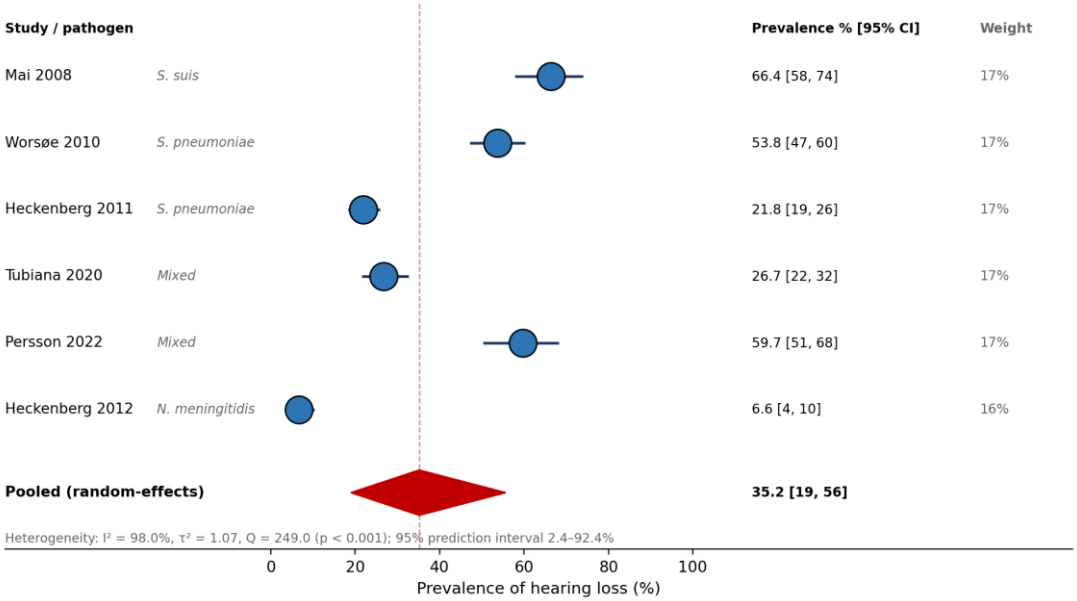

Figure 3. Forest plot of the pooled prevalence of hearing loss in adult acute bacterial meningitis survivors (random-effects, DerSimonian–Laird).

3.5. Pathogen-specific subgroup analysis

Subgrouping by pathogen revealed a pronounced gradient that accounted for much of the heterogeneity, illustrated in Figure 4. The prevalence of hearing loss was highest in *Streptococcus suis* meningitis at 66.4% (95% CI 58.6–74.3), intermediate in mixed community-acquired ABM at 42.2% (95% CI 15.7–74.1) and in pneumococcal meningitis at 36.3% (95% CI 12.4–69.6), and lowest in meningococcal meningitis at 6.6% (95% CI 3.9–9.3). The mixed stratum combined cohorts in which *S. pneumoniae* was the dominant organism (54% and 51% of isolates), so its intermediate position is coherent.

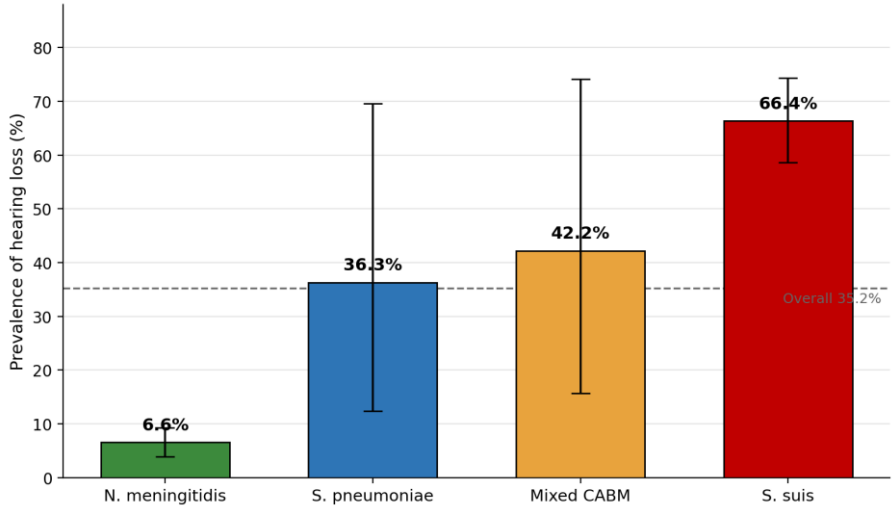

Figure 4. Pathogen-specific prevalence of hearing loss in adult acute bacterial meningitis (random-effects subgroup estimates; dashed line, overall 35.2%).

The contributing cohorts assessed hearing at different points, a source of the between-study dispersion evident in Figure 3. Estimates derived at or near discharge capture early hearing loss, a proportion of which may recover, whereas the French multicentre estimate of 26.7% was ascertained at twelve months

and better reflects persistent, rehabilitation-relevant deficit. Later, audiometrically confirmed estimates should therefore be regarded as the more reliable guide to permanent loss.

3.6. Predictors of hearing loss

For each candidate predictor, Table 3 lists the studies that examined it, the reported effect measure, whether it was adjusted, and whether the outcome was hearing-loss-specific or a composite unfavourable outcome. Considering only hearing-loss-specific estimates, advanced age (Mai 2008,[8] Worsøe 2010,[2] Persson 2022[20]), concurrent otitis media (Heckenberg 2011,[15] Persson 2022,[20] van de Beek 2004[14]) and virulent microbial genotype (Mai 2008[8]) were consistently associated with hearing loss, and pneumococcal serotype 23F was relatively protective (Heckenberg 2011[15]), while low CSF glucose and elevated CSF protein were associated with hearing loss in the retrospective pneumococcal cohort (Worsøe 2010[2]) and elevated CSF protein with unfavourable outcome in the Japanese surveillance cohort (Iwata 2023[21]). Composite-outcome estimates from the mixed cohorts (Tubiana 2020[9]) pointed in the same direction and are reported as supportive context only.

Table 3. Structured summary of predictors of hearing loss after adult acute bacterial meningitis.

Predictor	Studies	Effect (as reported)	Adjusted?	Outcome type
Advanced age	Mai; Worsøe; Persson; (Tubiana)	OR 3.65 (1.15–11.6) age >50; sig. in others	Multivariable	HL-specific (composite in Tubiana)
Otitis / AOM	Heckenberg 2011; Persson; (van de Beek)	OR 2.58 (1.66–4.02)	Multivariable	HL-specific
Low CSF glucose	Worsøe; (Tubiana)	Significant; aOR 1.92–2.24 (composite)	Uni/adjusted	HL-specific (Worsøe); composite
High CSF protein	Worsøe; Iwata; (Tubiana)	Significant; aOR 1.09/unit (composite)	Uni/adjusted	HL-specific (Worsøe); composite
Low CSF WBC (<1500)	Tubiana	aOR 2.40 (1.42–4.03)	Adjusted	Composite
Virulent strain (epf)	Mai	OR 3.42 (1.02–11.4)	Multivariable	HL-specific
Serotype 23F (protective)	Heckenberg 2011	OR 0.36 (0.13–0.98)	Multivariable	HL-specific
Pneumococcal aetiology	Persson; (Tubiana)	Significant; aOR 4.99 (composite)	Adjusted	HL-specific (Persson); composite
Adjunctive dexamethasone	Mai; Brouwer; Heckenberg 2012	Pooled OR 0.43 (0.29–0.65)	Mixed	HL-specific

Studies in parentheses contributed composite unfavourable-outcome estimates only. AOM, acute otitis media; aOR, adjusted odds ratio; HL, hearing loss; OR, odds ratio; WBC, white-cell count.

3.7. Adjunctive dexamethasone

In the exploratory random-effects synthesis of three datasets, adjunctive dexamethasone was associated with lower odds of hearing loss, with a pooled odds ratio of 0.43 (95% CI 0.29–0.65; I^2=0%), as displayed in Figure 5. The individual estimates were 0.23 (adjusted) in *S. suis* meningitis, 0.37 in meningococcal meningitis and 0.48 in pneumococcal meningitis; the last was derived by reconstructing survivor denominators from the reported mortality and hearing-loss percentages and is interpreted cautiously. A sensitivity analysis restricted to the two datasets with directly reported counts yielded a concordant pooled odds ratio of approximately 0.31. The absence of statistical heterogeneity, despite differing pathogens, strengthens the plausibility of a genuine otoprotective effect.

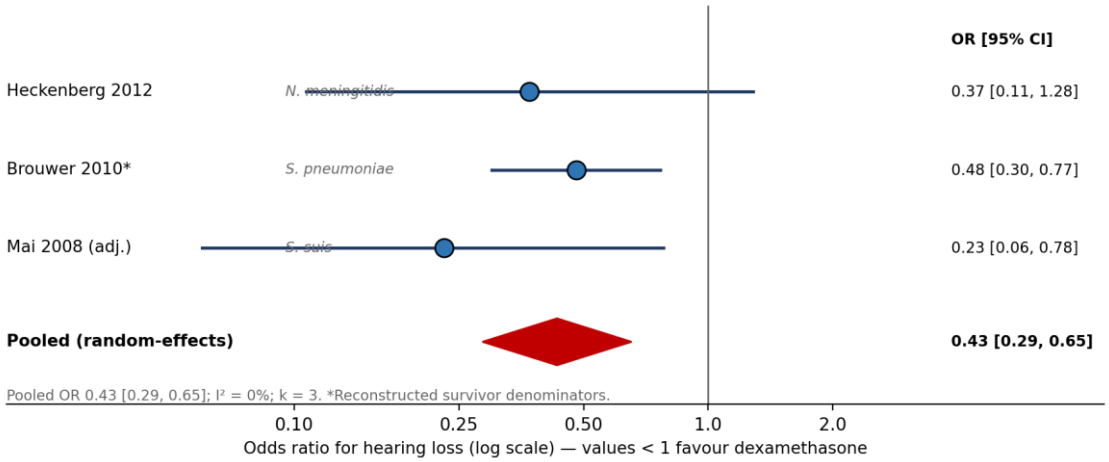

Figure 5. Exploratory forest plot of adjunctive dexamethasone and hearing loss (random-effects; *reconstructed survivor denominators).

3.8. Sensitivity analysis and small-study effects

Leave-one-out analysis confirmed robustness: the pooled prevalence ranged from 29.6% (omitting Mai 2008) to 44.5% (omitting the meningococcal cohort), heterogeneity remained high in every iteration (I^2≈97–98%), and all confidence intervals overlapped the primary estimate. Visual inspection of the funnel plot in Figure 6 did not indicate marked asymmetry; consistent with the small number of pooled studies, formal regression testing was not used to infer publication bias.

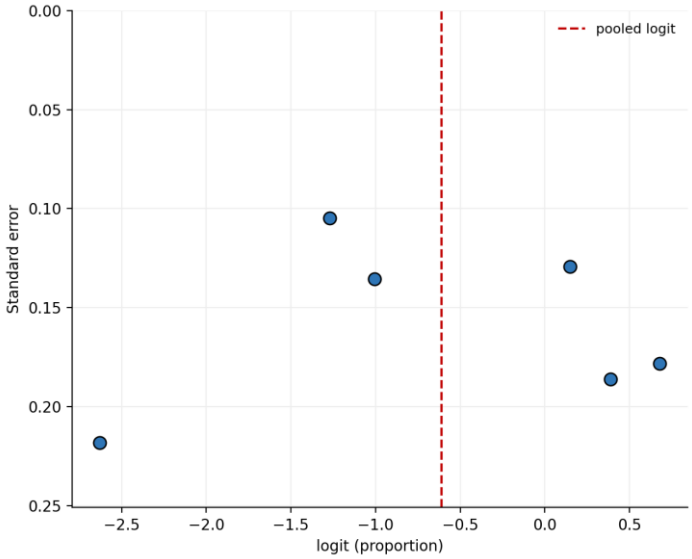

Figure 6. Funnel plot for the hearing-loss prevalence synthesis; formal small-study testing was underpowered at fewer than ten studies.

4. Discussion

In this systematic review and meta-analysis restricted to adults, approximately one in three survivors of acute bacterial meningitis experienced hearing loss, with a random-effects pooled prevalence of 35.2%. The most striking finding was a steep pathogen-specific gradient, from 66.4% in *Streptococcus suis* meningitis to 6.6% in meningococcal meningitis, with pneumococcal and mixed cohorts intermediate (Figure 4). This gradient, together with the consistent identification of advanced age, otitis media, virulent microbial genotypes and severe CSF derangement as predictors (Table 3), provides a coherent framework for identifying adults at greatest risk at admission. The wide prediction interval underscores that the pathogen-specific estimates, rather than the single pooled figure, should guide expectations in any given setting.

The exceptionally high burden in *S. suis* meningitis merits emphasis for neurological practice in Indonesia and the wider region. *Streptococcus suis* is now a leading cause of adult bacterial meningitis in pork-consuming and pig-rearing communities, and its predilection for sudden, frequently bilateral cochlear injury distinguishes it from the pyogenic meningitides more familiar in temperate settings.[6–8] The magnitude of risk observed here reinforces the case for heightened clinical suspicion in patients with relevant occupational or dietary exposure, early empirical therapy, and prompt audiological evaluation of survivors.

The present findings are complementary to, but distinct from, the existing quantitative literature. A recent global systematic review synthesised the prevalence of hearing loss by the timing of audiological testing across mixed adult and paediatric populations, reporting pooled prevalences broadly consistent with our adult estimate but not disaggregating admission-available predictors.[10] An earlier systematic review of *S. suis* meningitis reported hearing loss in roughly half of patients, a figure our *S. suis* subgroup exceeds, reflecting our focus on cohorts with formal ascertainment.[6] The Cochrane review of corticosteroids demonstrated a reduction in hearing loss across all ages, and our exploratory dexamethasone odds ratio of 0.43 aligns closely in direction and magnitude, offering adult-specific corroboration.[11] A large paediatric cohort likewise identified CSF derangement as a determinant,[22] and adult *Haemophilus influenzae* meningitis, an uncommon cause, carries an intermediate risk.[23] Meta-analyses of cochlear-implantation outcomes[12] and paediatric risk factors[13] address different questions, underscoring the gap this adult-focused predictor synthesis was designed to fill.

The very high statistical heterogeneity (I^2=98%) is unsurprising and informative rather than merely a limitation. It reflects true differences in the dominant pathogen, in whether hearing was assessed clinically or by audiometry, in the timing of assessment, and in the threshold used to define hearing loss. Pathogen subgrouping materially reduced within-group dispersion for the meningococcal and *S. suis* strata and explained much of the overall variance, indicating that causative organism is a principal driver of heterogeneity.

Because the analysis emphasises sensorineural hearing loss, the nature of the pooled outcome warrants care. The mechanism of meningogenic labyrinthitis is predominantly sensorineural, arising from cytotoxic injury to the cochlear neuroepithelium;[4,5] however, not every primary cohort distinguished sensorineural from conductive or mixed loss, and concurrent otitis media may contribute a conductive component in some patients. Otitis is best understood here in two roles: as a portal of bacterial entry and a marker of local inflammation that heightens sensorineural risk, and, occasionally, as a source of a coexisting conductive deficit. We have therefore used the broader term hearing loss where the underlying data did not permit a strict sensorineural classification.

The age association deserves particular caution. Baseline pre-meningitic audiometry was generally unavailable, so a proportion of the hearing loss attributed to meningitis in older adults may represent pre-existing presbycusis. Advanced age should therefore be regarded as a robust risk marker, but not necessarily a fully meningitis-attributable factor. Studies that corrected pure-tone thresholds for age, such as the Danish pneumococcal cohort,[2] retained a significant age effect, supporting a genuine contribution of ageing biology, but residual confounding by presbycusis remains possible.

The cohorts contributing to the pneumococcal and mixed strata were predominantly European, whereas the *S. suis* data derived from Vietnam.[8] The transferability of European pneumococcal estimates to Indonesian practice is therefore uncertain, given likely differences in conjugate-vaccine coverage, circulating serotypes, comorbidity burden, time to presentation and access to adjunctive dexamethasone. The *S. suis* estimate, by contrast, arises from a setting epidemiologically similar to Indonesia and is likely to be more directly applicable to the local readership.

Clinically, the results support a pragmatic, admission-based approach to risk stratification. Adults who are older, who have concurrent or antecedent otitis media, who exhibit profound CSF derangement (a low CSF-to-blood glucose ratio, markedly elevated CSF protein, or intense pleocytosis), and who are infected with pneumococcus or, in endemic regions, *S. suis*, should be regarded as at high risk and prioritised for early, repeated formal audiometry. The consistent association between adjunctive dexamethasone and lower odds of hearing loss reinforces current guidance to administer dexamethasone with or before the first dose of antibiotics, before cochlear inflammation progresses to fibrosis and ossification that would compromise later rehabilitation. Because timely identification of profound bilateral deafness also determines candidacy and timing for cochlear implantation,[24,25] structured surveillance carries direct rehabilitative consequences. As an illustrative proposal for future validation rather than a validated instrument, these admission features could be combined into a simple count-based risk flag to prioritise audiological follow-up, concentrating scarce audiology capacity where the yield is greatest.

Several limitations should be acknowledged. First, the pooled prevalence exhibited very high heterogeneity; although largely explained by pathogen and methodological diversity and addressed through random-effects modelling, subgrouping and a prediction interval, the overall figure is an average across heterogeneous settings. Second, the number of non-overlapping cohorts with extractable numerators was modest (six), precluding adequately powered publication-bias assessment and limiting subgroup granularity. Third, hearing-loss-specific adjusted estimates were available for only a subset of predictors; several were reported only as statements of significance or within composite models. Fourth, incomplete audiological ascertainment and the absence of baseline audiometry raise the possibility that age-related hearing loss was misattributed and that hearing loss was under-ascertained where testing was not universal. Fifth, the exploratory dexamethasone synthesis incorporated reconstructed denominators for one dataset. Sixth, the non-suis evidence was predominantly European, limiting regional generalisability. Future research should prioritise prospective adult cohorts with universal, protocolised audiometry and baseline hearing status, individual-participant-data meta-analysis for adjusted predictor estimates, and dedicated multicentre studies of *S. suis* meningitis in Southeast Asia.

5. Conclusion

Hearing loss complicates approximately 35% of adult survivors of acute bacterial meningitis, with a pronounced pathogen-specific gradient that places *Streptococcus suis* meningitis, endemic to Southeast Asia, at the extreme of risk (66%) and meningococcal meningitis at the opposite pole (6.6%); the wide prediction interval emphasises that these pathogen-specific estimates, rather than the single overall figure, should guide clinical expectation. The admission-available factors most consistently associated with hearing loss were advanced age, concurrent otitis media, infection with a virulent pathogen or genotype, and severe cerebrospinal fluid derangement (a reduced CSF-to-blood glucose ratio and elevated CSF protein), whereas certain pneumococcal serotypes conferred relative protection; the age association must be read in the light of probable confounding by presbycusis, and the sensorineural label in the light of occasional conductive contribution from otitis. Adjunctive dexamethasone was associated with a clinically meaningful and statistically consistent reduction in the odds of hearing loss (pooled OR 0.43). These findings furnish neurologists with a practical basis for early risk stratification and support prompt antibiotics, early dexamethasone and a pragmatic, resource-conscious audiological surveillance pathway for high-risk adults, with the ultimate goal of reducing the durable burden of post-meningitic deafness.

6. Declarations

6.1. Ethics approval and consent to participate

Ethical approval was not required as this study is a systematic review and meta-analysis of previously published, aggregate data and involved no new collection of human participant data or identifiable material. Consent to participate was not applicable.

6.2. Consent for publication

Not applicable; no identifiable individual patient data or images are presented.

6.3. Availability of data and materials

All data analysed in this review were extracted from previously published studies, all of which are cited in the reference list. The extracted dataset and analysis scripts are available from the corresponding author on reasonable request.

6.4. Competing interests

The authors declare no conflict of interest.

6.5. Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

6.6. Author contributions

All authors contributed to the conception and design of the review. IGAAGLS conducted the literature search, study selection and data extraction and drafted the manuscript. AARS supervised the work, contributed to interpretation and critically revised the manuscript. NMS contributed to data extraction, risk-of-bias assessment and critical revision. All authors approved the final version and are accountable for the work.

6.7. Acknowledgements

The authors thank the Department of Neurology, Faculty of Medicine, Universitas Udayana, for institutional support.

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