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Characteristics of Hearing Loss, Treatment Course and Hearing Outcomes of *Streptococcus Suis* Meningitis: A Case Series of Four Patients

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ABSTRACT

Objective: To describe the characteristics of hearing loss, treatment course and hearing outcomes of patients with hearing loss secondary to *Streptococcus suis* meningitis.

Methods:

Design: Retrospective Case Series
Setting: Tertiary Government Training Hospital
Participants: Four Patients

Results: Three patients had microbiologically confirmed *Streptococcus suis* meningitis based on positive blood or cerebrospinal fluid cultures, while one patient was diagnosed clinically based on compatible clinical manifestations and epidemiologic linkage to contemporaneous culture-confirmed cases. All four patients developed hearing loss during the acute phase of meningitis, ranging from unilateral profound to bilateral severe-to-profound sensorineural hearing loss. Three patients experienced associated vestibular symptoms. All patients received intravenous ceftriaxone and adjunctive intravenous dexamethasone at presentation followed by oral methylprednisolone. Intratympanic dexamethasone (4 mg/mL) was administered after persistent hearing loss despite systemic corticosteroid therapy, with treatment initiated at varying intervals from hearing loss onset. Audiologic outcomes were variable: two patients demonstrated partial improvement in hearing thresholds, whereas two showed minimal or no objective recovery on follow-up pure-tone audiometry.

Conclusion: This case series described four patients with *Streptococcus suis* meningitis who developed early sensorineural hearing loss, ranging from unilateral profound to bilateral severe-to-profound hearing loss, often with vestibular symptoms. Despite treatment with antimicrobial therapy, systemic corticosteroids, and adjunctive intratympanic dexamethasone, hearing outcomes were variable, with partial recovery in two patients and persistent severe or profound hearing loss in the other two**.**

Keywords: *Streptococcus suis*; *bacterial meningitis; sensorineural hearing loss; intratympanic dexamethasone; corticosteroids; audiologic outcomes*

Acute bacterial meningitis is a medical emergency associated with significant neurologic morbidity despite advances in antimicrobial therapy. Sensorineural hearing loss is the most common permanent neurologic sequela and may develop early during illness despite prompt initiation of appropriate antibiotics. Adjunctive dexamethasone administered before or with the first dose of antibiotics is recommended to attenuate the inflammatory response associated with

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bacterial lysis and reduce the risk of hearing loss and other neurologic complications.[1,2] Nevertheless, permanent hearing impairment continues to occur in a substantial proportion of patients.

Streptococcus suis is an emerging zoonotic pathogen and an important cause of bacterial meningitis in individuals exposed to pigs or pork products particularly in Southeast Asia. Hearing impairment has been reported in approximately 53% of patients with *S. suis* meningitis, making it the most common permanent neurologic complication of the disease.[3,4] Although intratympanic corticosteroid therapy is an established treatment for idiopathic sudden sensorineural hearing loss, evidence supporting its role in hearing loss secondary to *S. suis* meningitis remains limited, with published data restricted to isolated case reports and small case series.[5,6]

This case series describes the characteristics of hearing loss, treatment course, and hearing outcomes of four patients with *Streptococcus suis* meningitis who received systemic corticosteroids followed by adjunctive intratympanic dexamethasone. It also highlights the timing of hearing loss, the clinical course following treatment, and the variability of audiologic recovery.

METHODS

With the Cagayan Valley Medical Center Research Ethics Review Committee determination that the case series was exempt from full ethics review on June 4, 2025, this retrospective case series included the records of patients admitted to the Department of Otorhinolaryngology–Head and Neck Surgery of Cagayan Valley Medical Center from October 2024 to November 2024 who met all of the following inclusion criteria: (1) diagnosis of bacterial meningitis clinically and/or microbiologically consistent with *Streptococcus suis* meningitis; (2) development of hearing loss during the acute phase of meningitis; and (3) availability of clinical and audiologic records including pure-tone audiometry and/or otoacoustic emissions. Patients were excluded if they had (1) incomplete medical records; (2) insufficient audiologic data to characterize hearing status; or (3) hearing loss documented prior to the episode of meningitis.

Hospital records including clinical charts, computed tomography and magnetic resonance imaging findings, blood and cerebrospinal fluid laboratory and microbiologic results, and audiologic evaluations, including pure-tone audiometry and otoacoustic emissions, were retrieved by Dr. Kevin Christian Roman Mari Pascua and reviewed by the co-authors, including two board-certified otolaryngologists, Dr. Marieflor Cristy M. Garcia-Elgincolin and Dr. Ronaldo S. Zabala. Audiologic findings were reviewed and compared with the corresponding clinical records and documented interpretations. The four case reports were drafted by Dr. Kevin Christian Roman Mari Pascua and reviewed by all authors.

RESULTS

Case 1

A 62-year-old government official with type 2 diabetes mellitus and hypertension, consumed *pancit batil patung*, a local noodle dish containing undercooked pork liver. Two days later, he developed severe headache, generalized malaise, nausea, vomiting, slurred speech, and altered sensorium, prompting consultation at the Emergency Department of Cagayan Valley Medical Center. He was admitted with an initial working diagnosis of cerebrovascular accident and was started on citicoline, clonazepam, and other supportive medications.

On the first hospital day, cranial computed tomography angiography revealed no acute intracranial abnormalities. Lumbar puncture was subsequently performed. Empiric treatment with intravenous ceftriaxone (2 g every 12 hours) and dexamethasone (4 mg every 8 hours) were initiated and continued throughout his hospitalization.

The patient later reported that bilateral hearing loss began on the 4th hospital day although this initially went unrecognized because of his altered sensorium. During this period, he noted gradual improvement in hearing in the right ear but persistent hearing loss in the left ear accompanied by imbalance when attempting to sit upright.

On the 6th hospital day, cerebrospinal fluid (CSF) cultures yielded *Streptococcus suis*, prompting escalation of antimicrobial therapy to intravenous meropenem (2 g every 24 hours). A repeat cranial computed tomography scan demonstrated a hypodense lesion in the pons and evidence of cerebrocerebellar atrophy.

The patient's sensorium normalized on the 8th hospital day, allowing formal assessment of his hearing symptoms because of persistent bilateral hearing loss, predominantly affecting the left ear.

By the 17th hospital day, audiologic evaluation using pure tone audiometry (PTA) demonstrated mild conductive hearing loss in the right ear (average pure tone threshold (aPTT) of 35 dB HL) and profound hearing loss in the left ear (aPTT of 95 dB HL). *(Figure 1A)* Otoacoustic emissions (OAE) performed the same day showed a "Pass" result in the right ear and a "Refer" result in the left ear. Magnetic resonance imaging (MRI) with gadolinium contrast of the internal auditory canals, performed on the 18th hospital day, demonstrated minimal chronic small-vessel ischemic changes and cerebrocerebellar volume loss. There is loss of the normal T2 hyperintense fluid signal within the lateral semicircular canals bilaterally, more pronounced on the left. *(Figure 2)*

Despite approximately 17 days of systemic corticosteroid therapy, he continued to have persistent hearing loss in the left ear. Given the lack of clinical improvement, salvage intratympanic dexamethasone (4 mg) was administered into the left middle ear under otoendoscopic guidance on the 21st hospital day.

The patient was discharged on the 22nd hospital day with oral betahistine (8 mg three times daily), vitamin B complex (twice daily),

and insulin for glycemic control. He reported aural fullness for two days after the injection, followed by subjective improvement in hearing in the left ear 4 days after injection. Subsequent intratympanic dexamethasone injections were administered to the left ear on days 27, 34 and 40 from symptom onset.

A repeat pure-tone audiometry performed four months after treatment (128 days after symptom onset) demonstrated borderline normal hearing in the right ear (aPTT of 25 dB HL) and severe hearing loss in the left ear (aPTT of 75 dB HL). *(Figure 1B)* The patient reported complete resolution of dizziness and headache and was fully ambulatory without assistance.

Case 2

A 42-year-old self-employed man consumed *kinilaw* (raw pork soaked in vinegar) during a family gathering. Three days later, he developed severe headache, dizziness and numbness in all extremities, prompting consultation at the Emergency Department of Cagayan Valley Medical Center. He was subsequently admitted under the Internal Medicine service with a presumptive diagnosis of bacterial central nervous system infection. His sensorium remained clear throughout hospitalization. Notably, this case occurred nine days after the index case (Case 1), raising concern for a potential outbreak of *Streptococcus suis* meningitis.

Lumbar puncture revealed cloudy CSF consistent with purulent meningitis. A working diagnosis of bacterial meningitis was made, and empiric intravenous ceftriaxone (2 g every 12 hours) and dexamethasone (10 mg every 6 hours for 4 days) were initiated. Blood cultures subsequently yielded *Streptococcus suis*, whereas CSF cultures showed no bacterial growth.

He was referred to the ORL-HNS service on the 3rd hospital day due to sudden bilateral hearing loss associated with vertigo and persistent headache. He denied any prior hearing impairment. Oral vitamin B complex was started once daily as part of supportive management. Betahistine (16 mg three times daily) and oral methylprednisolone (24 mg twice daily for seven days) were initiated following completion of intravenous corticosteroid therapy.

By the 11th hospital day, PTA demonstrated an absence of response across all tested frequencies (250–8000 Hz) in the right ear and profound hearing loss in the left ear (aPTT of 93.3 dB HL). *(Figure 3A)* OAEs performed the same day were absent bilaterally. MRI with gadolinium contrast of the internal auditory canals demonstrated normal cochlear and vestibular anatomy without enhancement or signal abnormalities.

The patient's hearing did not improve despite 2 weeks of systemic corticosteroid therapy. Given the favorable subjective response observed in Case 1, salvage intratympanic dexamethasone (4 mg) was administered to the right ear under otoendoscopic guidance on the 16th hospital day.

He was discharged on the 18th hospital day with a tapering course of oral methylprednisolone and instructions to return for follow-up intratympanic steroid therapy. The patient reported persistent bilateral hearing loss and mild dizziness during his follow-up visits. Subsequent intratympanic dexamethasone injections were administered to the right ear 28, 35, and 42 days from symptom onset.

At the fourth follow-up (49 days from symptom onset), the patient reported complete resolution of dizziness and headache and was ambulatory with minimal balance support. Repeat PTA did not demonstrate improvement in the patient's hearing. *(Figure 3B)*

Case 3

A 73-year-old farmer consumed *kinilaw* during the same family gathering as his nephew (Case 2). Five days later, he developed severe back pain, headache, high-grade fever, and non-productive cough, prompting consultation at a local hospital, where he was treated with oral cefuroxime, diclofenac and eperisone. Despite treatment, his condition worsened, and he was referred to the Emergency Department of Cagayan Valley Medical Center two days after Case 2 had been admitted.

He was hemodynamically stable at the emergency room but demonstrated positive Brudzinski's sign and nuchal rigidity. A working diagnosis of bacterial meningitis was made, and empiric intravenous ceftriaxone (2 g every 12 hours) and dexamethasone (10 mg every 6 hours for 9 days) were initiated.

He was referred to the ORL-HNS service on the 2nd hospital day due to sudden bilateral hearing loss. Oral vitamin B complex was started once daily as part of supportive management. The patient was diagnosed with bacterial meningitis based on his clinical presentation and associated leukocytosis despite negative microbiologic cultures from the blood and CSF. He was managed as a case of *Streptococcus suis* meningitis because of the strong epidemiologic linkage to two microbiologically confirmed cases with shared exposure and similar clinical manifestations.

By the 9th hospital day, PTA demonstrated severe hearing loss in the right ear (aPTT of 80 dB HL) and profound hearing loss in the left ear (aPTT of 83.3 dB HL). *(Figure 4A)* OAEs were absent bilaterally. MRI with gadolinium contrast of the internal auditory canals did not demonstrate any abnormalities. Systemic corticosteroid therapy was shifted to oral methylprednisolone (24 mg twice daily) shortly after PTA confirmation.

On the 14th hospital day, salvage intratympanic dexamethasone (4 mg) was administered to the left ear (worse hearing ear) under otoendoscopic guidance. Oral corticosteroids were continued for an additional nine days. The patient was discharged on the 16th hospital day with instructions to continue vitamin B complex and return for repeat intratympanic steroid therapy.

The patient reported persistent bilateral hearing loss, dizziness

and intermittent imbalance during his follow-up visits. Subsequent intratympanic dexamethasone injections were administered to the left ear 23 and 30 days from symptom onset. Oral methylprednisolone was tapered to 16 mg twice daily for seven days after the second injection and was subsequently discontinued, while vitamin B complex was continued.

On third follow-up (37 days from symptom onset), the patient reported complete resolution of dizziness and headache, with subjective perception of sound in the left ear. However, repeat pure-tone audiometry demonstrated no objective hearing improvement, with persistent severe hearing loss in the right ear and profound hearing loss in the left ear. *(Figure 4B)* Although vestibular symptoms had resolved, he remained partially dependent for ambulation and required crutches because of residual imbalance.

Case 4

A 37-year-old government employee, without prior history of hearing loss, consumed *kinilaw* during the same family gathering as his cousin (Case 2) and uncle (Case 3). Five days later, he developed intermittent high-grade fever, chills, diffuse myalgia, progressive headache, and altered sensorium, prompting consultation at the Emergency Department of Cagayan Valley Medical Center. He was admitted on the same day as Case 3.

A working diagnosis of bacterial meningitis was made, and empiric intravenous ceftriaxone (2 g every 12 hours) and dexamethasone (10 mg every 6 hours for 9 days) were initiated. Blood cultures yielded *Streptococcus suis*. CSF obtained from lumbar puncture was clear and negative for bacterial growth. Despite the negative CSF culture, the positive blood culture together with the clinical presentation supported the diagnosis of *Streptococcus suis* meningitis.

He was referred to the ORL-HNS service on the 1st hospital day due to sudden-onset right-sided hearing loss. Oral vitamin B complex was started once daily as part of supportive management.

PTA performed on the 9th hospital day demonstrated profound sensorineural hearing loss in the right ear (aPTT of 85 dB HL) and mild hearing loss in the left ear (aPTT of 33 dB HL). *(Figure 5A)* Although the right ear demonstrated a significant air–bone gap, no tympanometry was performed to further evaluate middle ear function; therefore, the etiology of the conductive component could not be definitively determined. OAEs showed a "Refer" result in the right ear and a "Pass" result in the left ear. MRI with gadolinium contrast of the internal auditory canals did not demonstrate any abnormalities. Systemic corticosteroid therapy was shifted to oral methylprednisolone (24 mg twice daily) after PTA confirmation.

Salvage intratympanic dexamethasone (4 mg) was administered to the right ear under otomicroscopic guidance on the 14th hospital day. Oral methylprednisolone was continued post discharge, along with vitamin B complex and cinnarizine (25mg as needed for dizziness).

At the first follow-up (24 days from symptom onset), the patient reported persistent right-sided hearing loss and mild dizziness. A second intratympanic dexamethasone injection was administered to the right ear, and oral methylprednisolone (24 mg twice daily) was continued. On second follow-up (31 days from symptom onset), the patient reported improved balance but unchanged hearing. A third intratympanic dexamethasone injection was administered to the right ear. Oral corticosteroids were tapered to 16 mg twice daily for seven days before discontinuation.

At the third follow-up (38 days from symptom onset), the patient reported complete resolution of dizziness although right-sided hearing loss persisted. Repeat PTA demonstrated profound hearing loss in the right ear and mild hearing loss in the left ear. Long-term PTA performed 235 days after symptom onset demonstrated severe hearing loss in the right ear (aPTT of 66.6 dB HL) and normal hearing in the left ear (aPTT of 20 dB HL). *(Figure 5B)*

A summary of the key clinical features of each case can be found in *Table 1*.

DISCUSSION

In the present series, all four patients developed hearing impairment during the acute phase of meningitis despite prompt initiation of antimicrobial therapy. The severity ranged from unilateral profound sensorineural hearing loss to bilateral severe-to-profound hearing loss, and three patients experienced concomitant vestibular symptoms, including dizziness, vertigo or gait imbalance. These findings are consistent with previous reports demonstrating that auditory and vestibular dysfunction frequently coexist in *S. suis* meningitis because of inner-ear involvement.[3,4]

Although most reported cases describe bilateral sensorineural hearing loss, the degree of hearing impairment in our patients varied considerably.[3,6] One patient initially presented with unilateral profound hearing loss, whereas the remaining patients had bilateral involvement of differing severity. This variability emphasizes the importance of comprehensive audiologic assessment. Pure-tone audiometry remains the gold standard for determining hearing thresholds, while otoacoustic emissions provide an objective assessment of cochlear outer hair cell function and may serve as a useful adjunct, particularly in patients with acute meningitis who are unable to reliably undergo behavioral audiometric testing.[3,7]

The pathogenesis of hearing loss in *S. suis* meningitis is believed to involve bacterial invasion of the inner ear through the cochlear aqueduct, resulting in suppurative labyrinthitis and an intense inflammatory response.[4] Subsequent fibrosis and labyrinthine ossification may lead to irreversible cochlear injury if inflammation is not controlled promptly.[4] This mechanism may explain why severe hearing loss developed in our patients despite appropriate antimicrobial therapy and why hearing recovery remained limited in most cases.

Table 1. Clinical, audiometric, vestibular, imaging findings and hearing outcomes of four patients with *Streptococcus suis* meningitis

CASE	AGE/ SEX	SUSPECTED LATENCY FROM EXPOSURE TO SYMPTOM ONSET	COMOR BIDITY	MICRO BIOLOGIC STUDY	NO. OF ITS INJECT ION	PRE-ITS AUDIO METRY	POST-ITS AUDIO METRY	PRE-ITS OAE	POST-ITS OAE	PRE-ITS BALANCE	POST-ITS BALANCE	MRI OF BRAIN AND IAC
1	62/M	2 days	Diabetes Mellitus type 2, Hypertension stage 2	CSF Culture: Positive Blood culture: Not done	4	Right: Mild HL Left: Profound HL (17th HD)	Right: Normal Left: Severe HL † (Day 128)*	Right: Pass Left: Refer	Right: Pass Left: Refer	With support	Independent	Minimal chronic small vessel ischemic changes and cerebro cerebellar volume loss. There is loss of the normal T2 hyperintense fluid signal within the lateral semicircular canals bilaterally
2	42/M	3 days	Prediabetes	CSF Culture: Negative Blood culture: Positive	4	Right: No Response Left: Profound HL (11th HD)	Right: No Response † Left: Profound HL (Day 49)*	Right: Refer Left: Refer	Right: Refer Left: Refer	With support	Minimal support	Prominent bilateral anterior inferior cerebellar artery vascular loop (type 1)
3	73/M	5 days	Impaired Fasting Glucose	CSF Culture: Negative Blood culture: Negative	3	Right: Severe HL Left: Profound HL (9th HD)	Right: Severe HL Left: Profound HL † (Day 37)*	Right: Refer Left: Refer	Right: Refer Left: Refer	With support	Minimal Support	Normal finding on bilateral internal auditory canal
4	37/M	5 days	Impaired Fasting Glucose	CSF Culture: Negative Blood culture: Positive	3	Right: PROFOUND HL Left: Mild HL (13th HD)	Right: SEVERE HL † Left: Normal (Day 235)*	Right: Refer Left: Pass	Right: Refer Left: Pass	With support	Independent	Normal finding on bilateral internal auditory canal

HL: Hearing Loss, OAE: Otoacoustic Emission, ITS: Intratympanic Steroid, MRI: Magnetic Resonance Imaging, IAC: Internal Auditory Canal, *: From onset of disease, †: Post ITS ear

Magnetic resonance imaging (MRI) demonstrated no definite cochlear or retrocochlear abnormalities in three patients, while one patient showed findings suspicious for early labyrinthine fibrosis. MRI is useful in patients with meningitis-associated hearing loss to evaluate for inner ear inflammation, labyrinthine fibrosis or ossification, and retrocochlear pathology that may influence prognosis and subsequent management, including cochlear implantation. However, the absence of MRI abnormalities should not exclude clinically significant cochlear injury, as conventional MRI may fail to detect microscopic inflammatory changes during the early stages of labyrinthitis.[3,4] Consequently, audiologic findings remain the cornerstone for diagnosing and monitoring meningitis-associated hearing loss.[3,7]

Adjunctive corticosteroid therapy has become standard management for acute bacterial meningitis because it attenuates the inflammatory response triggered by bacterial lysis after antibiotic administration.[1,2] Administration of dexamethasone before or with the first dose of antibiotics has been associated with reduced neurologic complications, including hearing loss, particularly in pneumococcal meningitis.[1,2] Although evidence specific to *S. suis* meningitis is limited, corticosteroids are widely recommended because the inflammatory mechanisms underlying cochlear injury are similar.[3,4]

All four patients in this series received intravenous dexamethasone during the acute stage of illness followed by oral methylprednisolone. Despite systemic corticosteroid therapy, hearing recovery remained

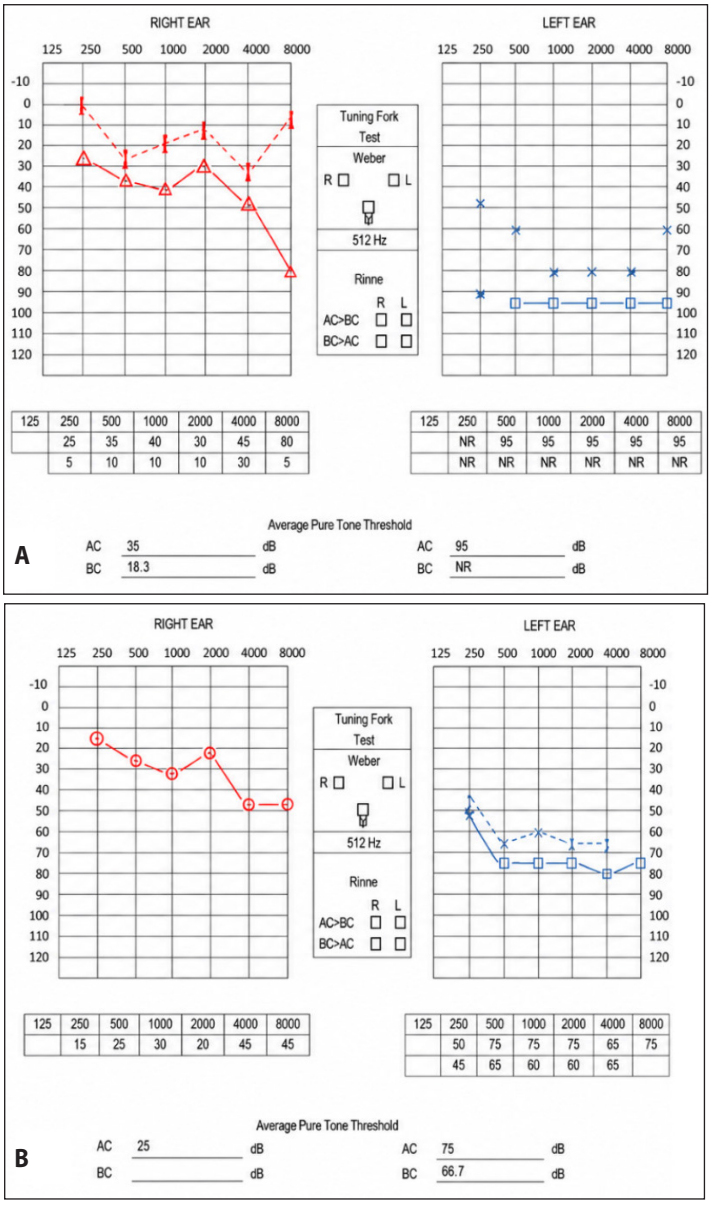


Figure 1. Initial and follow-up pure-tone audiograms of Case 1: **A.** Initial audiogram demonstrating mild conductive hearing loss in the right ear and profound hearing loss in the left ear; and **B.** Follow-up audiogram obtained four months after treatment demonstrating borderline normal hearing in the right ear and severe hearing loss in the left ear.

incomplete in some patients, suggesting that systemic treatment alone may not always prevent irreversible cochlear injury.

Hearing loss developed during the acute phase of *Streptococcus suis* meningitis in all four patients in this series. Intravenous dexamethasone was administered together with antimicrobial therapy at presentation, with treatment durations ranging from 4 to 17 days before transition to oral methylprednisolone. Persistent hearing loss despite systemic corticosteroid therapy prompted the use of adjunctive intratympanic dexamethasone, which was initiated during the second to third week of hospitalization. Audiologic outcomes remained variable.

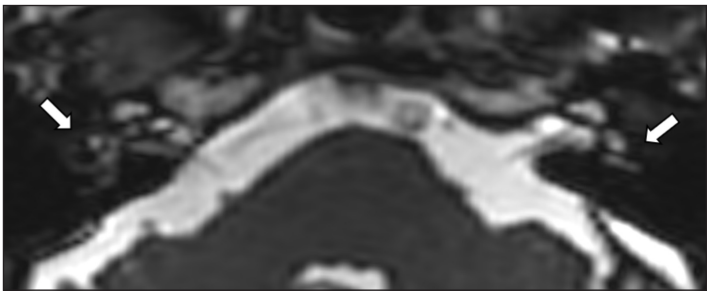
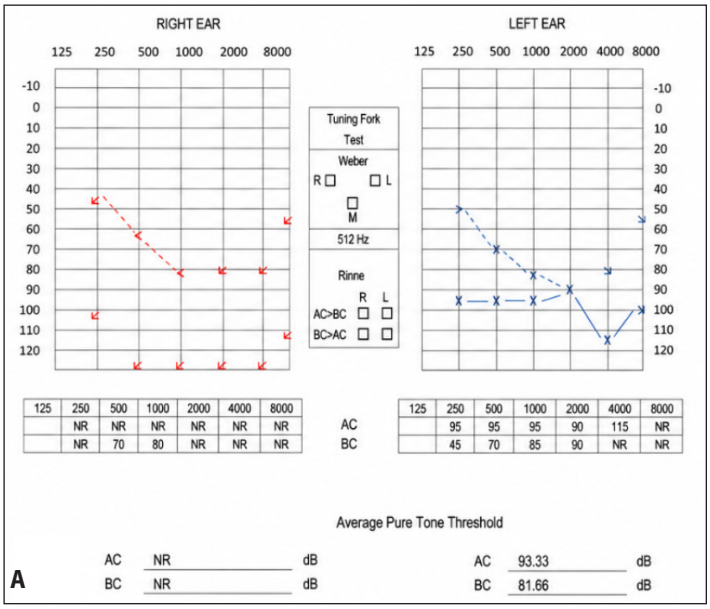


Figure 2. Axial T2-weighted MRI showing bilateral loss of the normal T2 hyperintense fluid signal in the lateral semicircular canals



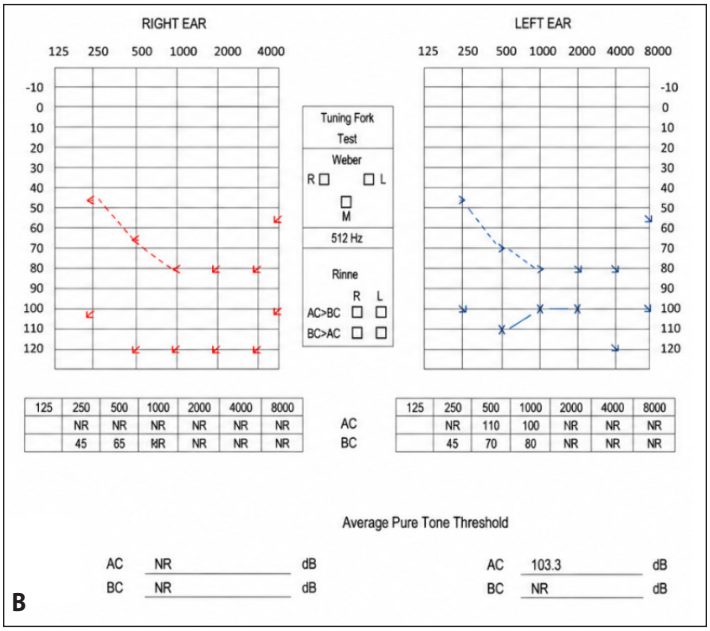
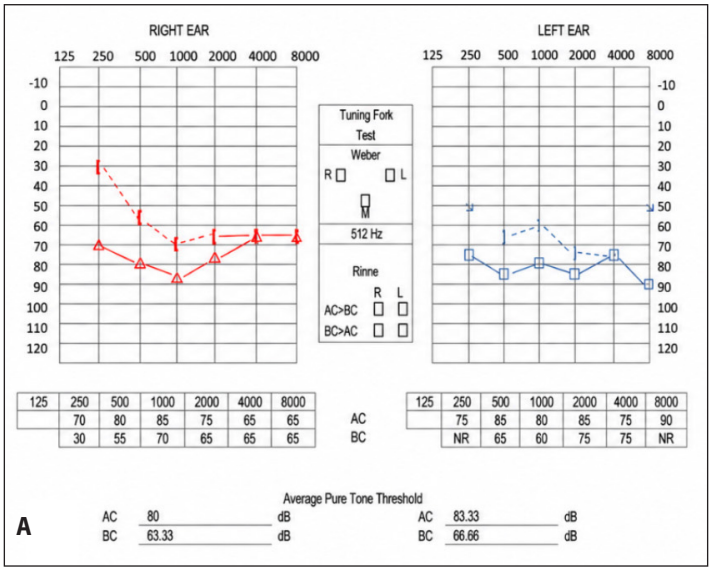


Figure 3. Initial and follow-up pure-tone audiograms of Case 2: **A.** Initial audiogram demonstrating no response across all tested frequencies in the right ear and profound hearing loss in the left ear; and **B.** Follow-up audiogram demonstrating persistent absence of response in the right ear and profound hearing loss in the left ear.



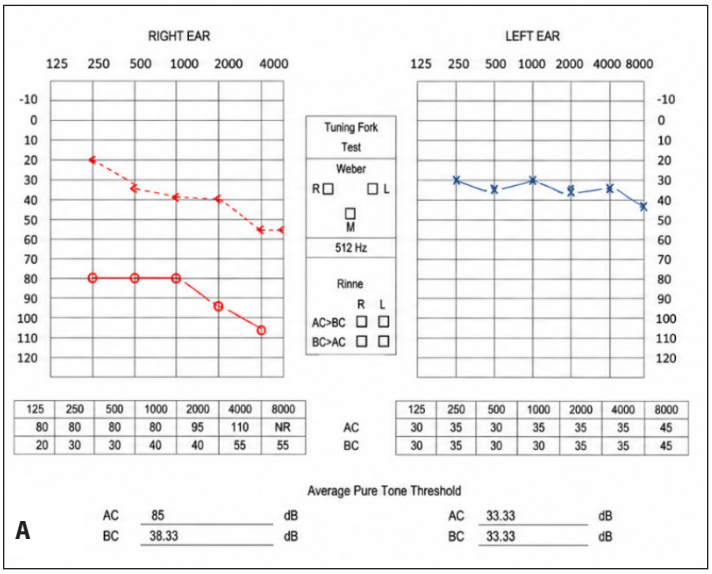
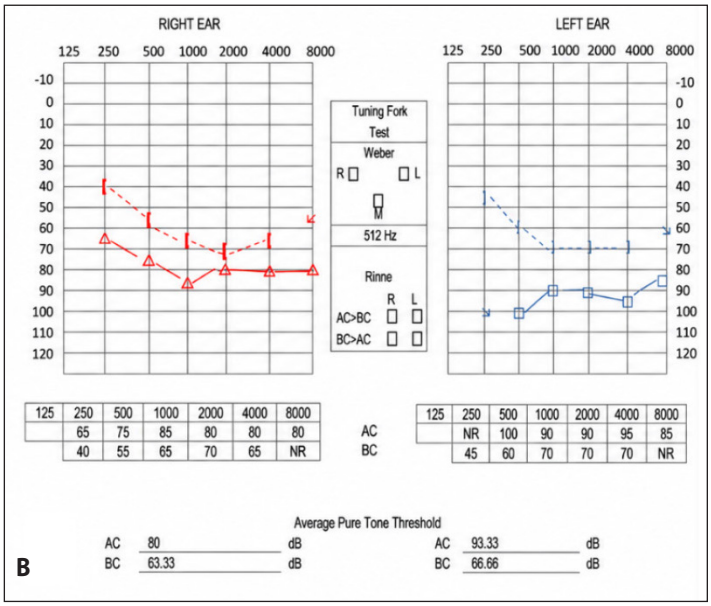


Figure 4. Initial and follow-up pure-tone audiograms of Case 3: **A.** Initial audiogram demonstrating severe hearing loss in the right ear and profound hearing loss in the left ear; and **B.** Follow-up audiogram demonstrating persistent severe hearing loss in the right ear and profound hearing loss in the left ear.



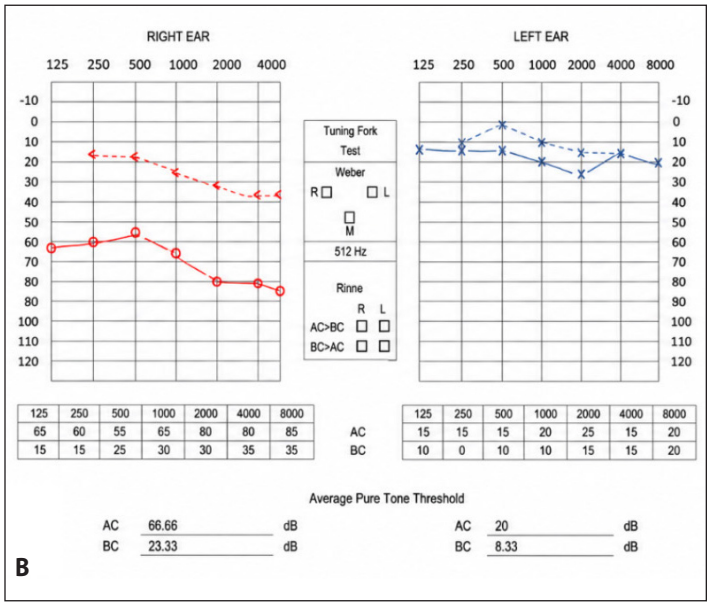


Figure 5. Initial and follow-up pure-tone audiograms of Case 4: **A.** Initial audiogram obtained on the ninth hospital day demonstrating profound sensorineural hearing loss in the right ear and mild hearing loss in the left ear; and **B.** Follow-up audiogram obtained 235 days after symptom onset demonstrating severe hearing loss in the right ear and normal hearing in the left ear.

Although two patients demonstrated partial improvement in hearing thresholds, two showed minimal or no objective recovery. Although the patient who received intratympanic therapy earliest exhibited the most favorable long-term hearing outcome, this observation should be interpreted cautiously because all patients received concurrent antimicrobial therapy and systemic corticosteroids, and the small sample size precludes establishing a causal relationship between earlier intervention and improved hearing outcomes.

Intratympanic corticosteroid therapy achieves high intracochlear drug concentrations while minimizing systemic exposure and has demonstrated effectiveness in the literature for the treatment of idiopathic sudden sensorineural hearing loss.[5,8] However, its role in hearing loss secondary to *Streptococcus suis* meningitis remains uncertain because the underlying pathophysiology differs from idiopathic sudden sensorineural hearing loss, and published evidence is limited to isolated case reports and small case series.[5,6]

In the present series, intratympanic dexamethasone was administered after persistent hearing loss despite completion of systemic corticosteroid therapy, using a treatment protocol adapted from published intratympanic steroid regimens for idiopathic sudden

sensorineural hearing loss.[5,8] Audiologic outcomes were heterogeneous, with two patients demonstrating objective improvement in hearing thresholds and two experiencing no measurable recovery. Although the patient who received intratympanic therapy earliest also demonstrated the most favorable long-term hearing outcome, this observation should be interpreted cautiously. All patients received concurrent antimicrobial therapy, systemic corticosteroids, and oral corticosteroids, making it impossible to determine the independent contribution of intratympanic dexamethasone. These findings should therefore be interpreted as descriptive observations, given the limited published evidence regarding intratympanic corticosteroid therapy for hearing loss secondary to *S. suis* meningitis.[6]

Adjunctive intratympanic corticosteroid therapy was incorporated into the clinical management of all patients after persistent hearing loss despite systemic corticosteroid therapy, using a dexamethasone dose and administration protocol adapted from published intratympanic steroid regimens for idiopathic sudden sensorineural hearing loss.[8] Larger prospective studies with standardized treatment protocols and earlier intervention are needed to determine whether adjunctive intratympanic therapy offers additional benefit beyond conventional systemic treatment. Accordingly, the use of adjunctive intratympanic dexamethasone in this case series should be considered exploratory, and no conclusions regarding its therapeutic efficacy can be drawn from these observations.

The onset of hearing loss occurred within the first few days of hospitalization in all four patients, emphasizing that auditory complications may develop early during the clinical course of *S. suis* meningitis. This observation highlights the importance of routine hearing assessment in patients with bacterial meningitis, particularly those reporting new hearing loss, tinnitus, dizziness or imbalance.

Early referral for otologic evaluation, baseline audiologic testing, and serial follow-up examinations are essential for documenting hearing progression, counseling patients regarding prognosis, and identifying those who may benefit from rehabilitative interventions such as hearing aids or cochlear implantation in cases of persistent profound hearing loss.

Although this descriptive case series provides valuable clinical observations, several limitations should be considered when interpreting the findings. First, it is a retrospective case series involving only four patients from a single institution, limiting the generalizability of the findings. Second, treatment protocols were not standardized, particularly with respect to the timing of intratympanic corticosteroid administration and the duration of systemic corticosteroid therapy. Third, the concurrent administration of antimicrobial therapy, systemic corticosteroids, and intratympanic dexamethasone precludes determination of the independent therapeutic effect of intratympanic corticosteroids. Finally, one patient was diagnosed clinically based on characteristic clinical features and epidemiologic linkage to contemporaneous culture-confirmed *Streptococcus suis* cases despite negative blood and cerebrospinal fluid cultures, introducing the possibility of diagnostic uncertainty.

Accordingly, the findings should be considered hypothesis-generating rather than evidence of treatment efficacy. Prospective multicenter studies with standardized audiologic assessment and treatment protocols are needed to better define the role of adjunctive intratympanic corticosteroids in hearing loss secondary to *Streptococcus suis* meningitis.

In conclusion, this case series described the clinical presentation, audiologic characteristics, treatment course, and hearing outcomes of four patients with Streptococcus suis meningitis and highlights the variability of hearing recovery observed during follow-up. Hearing loss developed early during the acute phase of *Streptococcus suis* meningitis in all four patients and ranged from unilateral profound to bilateral severe-to-profound sensorineural hearing loss, frequently accompanied by vestibular symptoms. All patients received antimicrobial therapy, systemic corticosteroids, and adjunctive intratympanic dexamethasone as part of their clinical management. Follow-up audiologic outcomes were variable, with two patients demonstrating partial objective improvement in hearing thresholds and two showing persistent severe or profound hearing loss.

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