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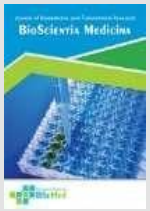

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Intratympanic Corticosteroid Salvage for Pediatric Bilateral Sudden Sensorineural Hearing Loss Complicated by Methylprednisolone-Induced Hypertensive Urgency in an Obese Adolescent

Jihan Mudrika Rahmi[1], Rossy Rosalinda[1*]

[1]Department of Otorhinolaryngology, Faculty of Medicine, Universitas Andalas, Padang, Indonesia

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***Corresponding author:**
Rossy Rosalinda

E-mail address:
rossy@med.unand.ac.id

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A B S T R A C T

Background: Bilateral sudden sensorineural hearing loss (SSNHL) is a rare otologic emergency, accounting for less than 5% of sudden deafness cases and occurring even less frequently in pediatric populations. Unlike unilateral cases, bilateral involvement strongly implicates systemic etiologies such as autoimmune inner ear disease (AIED). Systemic high-dose corticosteroids are the standard first-line therapy but pose significant risks of toxicity, including hypertensive crisis, particularly in adolescents with metabolic risk factors. **Case presentation:** We report the case of a 17-year-old male (BMI 29.0 kg/m²) presenting with acute, simultaneous bilateral hearing loss (Pure tone average [PTA]: Right 82.5 dB, Left 81.25 dB) and severe tinnitus (Tinnitus Handicap Index [THI]: 78). Initial management with high-dose intravenous methylprednisolone (500 mg/day) was complicated on Day 7 by hypertensive urgency (Blood Pressure 150/95 mmHg) and neurological symptoms, necessitating immediate cessation of systemic therapy. Diagnostic investigation was limited by resource availability; however, elevated inflammatory markers supported a presumptive immune-mediated etiology. A salvage protocol was initiated using four weekly cycles of intratympanic Triamcinolone Acetonide. Following therapy, the patient demonstrated slight audiological recovery (PTA stabilized at 65 dB bilaterally) but achieved complete resolution of tinnitus (THI: 0). **Conclusion:** This case highlights the critical role of intratympanic corticosteroids as a safe salvage modality when systemic therapy is contraindicated due to toxicity. The dissociation between modest audiometric gain and complete tinnitus resolution suggests successful mitigation of cochlear synaptopathy. The case underscores the necessity of cardiovascular monitoring and BMI-adjusted risk stratification in adolescents receiving high-dose steroids.

1. Introduction

Sudden sensorineural hearing loss (SSNHL) represents a formidable otologic emergency, characterized clinically by a rapid decline in auditory function that is often unforeseen and devastating to the patient's quality of life.[1] Defined strictly by the audiometric criterion of a sensorineural threshold shift of at least 30 decibels (dB) across three consecutive frequencies occurring within a 72-hour window, SSNHL is a pathology that demands immediate diagnostic and therapeutic attention.[2] While the annual incidence in the general population is estimated to range between 5 and 27 per 100,000 individuals, these statistics are overwhelmingly dominated by adult cases, with a peak incidence typically observed in the fifth and sixth decades of life. In the vast majority of these presentations, the hearing loss is unilateral, affecting only one ear, which allows the patient to maintain communicative competence through the unaffected side.[3] However, the

epidemiological landscape shifts dramatically when examining the pediatric and adolescent population (aged 18 years or younger). In this demographic, SSNHL is an exceptionally rare clinical entity, accounting for only 6.6% of the total caseload of sudden deafness globally. Within this already rarefied subset, the phenomenon of bilateral SSNHL—where a patient loses hearing in both ears simultaneously or sequentially within a short timeframe—is an even scarcer phenotype. Statistical analyses indicate that bilateral involvement occurs in approximately 1% to 5% of all SSNHL cases, but in children, this prevalence drops to fewer than 4% of those presenting with sudden hearing loss. The functional ramifications of bilateral loss in an adolescent are profound, threatening educational development, social integration, and psychological well-being at a critical developmental juncture.[4]

The distinction between unilateral and bilateral SSNHL is not merely numerical; it represents a fundamental divergence in pathophysiology. Unilateral SSNHL is frequently attributed to localized cochlear events. The prevailing theories for unilateral loss include microvascular occlusion (cochlear infarction), localized viral reactivation (such as Herpes Simplex or Varicella Zoster within the spiral ganglion), or mechanical insults like cochlear membrane rupture. These mechanisms act as isolated strikes against a single end-organ. In sharp contrast, the simultaneous or near-simultaneous involvement of both cochleae in bilateral SSNHL strongly implicates a systemic pathology capable of launching a coordinated assault on the inner ear. The cochlea is protected by the blood-labyrinth barrier (BLB), a selective physiological gatekeeper analogous to the blood-brain barrier, which regulates the transport of ions and fluids while restricting the entry of pathogens and large macromolecules.[5] A bilateral breach of this barrier suggests a potent systemic driver capable of overcoming these protective mechanisms on a global scale. Consequently, the differential diagnosis for bilateral SSNHL is distinct and more ominous than that of unilateral cases. It necessitates the exclusion of viral neuro-labyrinthitis, hematologic malignancies (such as leukemia or lymphoma), and potentially fatal central nervous system pathologies.

Increasingly, however, bilateral presentation is recognized as a hallmark of immune-mediated cochlear injury, often classified under the umbrella of autoimmune inner ear disease (AIED). In these scenarios, the host's immune system may generate circulating immune complexes or autoantibodies that target inner ear antigens—such as heat shock protein 70 (Hsp70) or cochlear tubulin—leading to vasculitis, stria vascularis inflammation, and subsequent ischemic hypoxia of the Organ of Corti. In pediatric patients, establishing such a definitive etiology presents a significant diagnostic challenge. The clinical presentation is often ambiguous, and specific autoimmune markers are frequently negative or unavailable in resource-limited settings, leading to a reliance on non-specific inflammatory markers like erythrocyte sedimentation rate (ESR) to guide clinical suspicion.[6]

Despite the etiological complexity, the therapeutic approach to SSNHL has remained relatively consistent over the past decades. Current clinical practice guidelines, including those from the American Academy of Otolaryngology-Head and Neck Surgery, advocate for high-dose systemic corticosteroids (oral or intravenous) as the gold standard for initial treatment.[7] The pharmacologic rationale relies on the potent anti-inflammatory and immunosuppressive properties of glucocorticoids. By downregulating the expression of pro-inflammatory cytokines (such as TNF-alpha and IL-1beta) and inhibiting the apoptotic pathways in cochlear hair cells, steroids aim to rescue the neurosensory elements of the inner ear before permanent degeneration occurs.

However, the administration of high-dose systemic steroids is not a benign intervention. The therapeutic window for hearing salvage is narrow, often necessitating aggressive dosing regimens that push the limits of physiological tolerance.[8] This is particularly pertinent in the pediatric and adolescent population. Unlike adults, adolescents are in a state

of rapid metabolic and hormonal flux, making them uniquely susceptible to the mineralocorticoid and glucocorticoid side effects of agents like methylprednisolone.

Methylprednisolone acts not only on glucocorticoid receptors to suppress inflammation but also possesses mineralocorticoid activity, acting on the distal renal tubules to increase sodium reabsorption and potassium excretion. This mechanism can lead to rapid water retention, volume expansion, and the precipitation of acute steroid-induced hypertension. Furthermore, systemic toxicity can manifest as hyperglycemia, gastric irritation, and significant behavioral changes, including mood lability, anxiety, and steroid psychosis.[9] The psychological impact of sudden deafness—already a traumatic event causing isolation and fear—is thus compounded by steroid-induced mood instability, adding a layer of complexity to patient management that is often under-recognized by clinicians focused solely on the audiogram.

When severe adverse events (SAEs) occur, the clinician is faced with a critical ethical and therapeutic dilemma: the cessation of a potentially life-saving drug for hearing preservation versus the imperative to manage life-threatening systemic toxicity. In the case of steroid-induced hypertensive urgency, continuing systemic therapy poses risks of hypertensive encephalopathy or cardiovascular strain, mandating the immediate withdrawal of the offending agent. Historically, this would have left the patient with no therapeutic recourse for their hearing loss.

In this context, intratympanic (IT) corticosteroid injection has emerged as an essential alternative and a vital safety net. Originally developed and utilized as a salvage therapy for refractory cases—defined as patients who fail to respond to an initial course of systemic steroids—IT administration is increasingly recognized for its utility in cases of steroid intolerance. The pharmacokinetic advantage of the intratympanic route lies in its ability to bypass the systemic circulation entirely.

When corticosteroids are injected directly into the middle ear space, they contact the Round Window Membrane (RWM), a semi-permeable interface separating the middle ear from the fluid-filled cochlea. Through passive diffusion and active transport mechanisms, the steroid traverses the RWM to enter the perilymph of the scala tympani. Pharmacokinetic studies have demonstrated that this targeted delivery results in intracochlear steroid concentrations that are significantly higher—often by orders of magnitude—than those achievable by intravenous or oral delivery. Simultaneously, because the drug is not processed through the first-pass hepatic metabolism or distributed through the systemic vasculature, plasma levels remain negligible, thereby minimizing or eliminating the risk of systemic side effects such as hypertension or hyperglycemia.[10]

Despite the theoretical advantages of intratympanic therapy, literature detailing its use as a primary salvage modality in pediatric bilateral SSNHL complicated by severe systemic toxicity is sparse. Most pediatric studies focus on idiopathic unilateral loss or viral etiologies, and few address the specific metabolic risks associated with high-dose steroids in adolescents with elevated Body Mass Index (BMI). This study aims to document the clinical trajectory and management of a rare case of simultaneous bilateral SSNHL in a 17-year-old adolescent with metabolic risk factors. The novelty of this report lies in the management of a significant and sight-threatening complication—acute methylprednisolone-induced hypertension in an obese adolescent—which forced a strategic pivot from systemic to intratympanic salvage therapy. Furthermore, we provide a detailed analysis of the pharmacokinetic rationale for this switch, the diagnostic challenges in resource-limited settings where specific autoimmune serology is unavailable, and the intriguing disconnect observed between functional outcomes (complete resolution of tinnitus) and audiometric thresholds. By elucidating the management of this double-hit scenario—bilateral sensory loss complicated by iatrogenic toxicity—this report seeks to offer a roadmap for clinicians navigating the precarious balance between aggressive

immunosuppression and patient safety in the vulnerable pediatric demographic.

2. Case Presentation

Written informed consent was obtained from the patient's parents for the publication of this case report and any accompanying clinical data. The patient and his guardians were fully informed that the clinical details would be used for educational and scientific purposes, and all efforts have been made to maintain patient anonymity in accordance with ethical standards.

A 17-year-old male student presented to the emergency department of Dr. M. Djamil General Hospital, Padang, manifesting symptoms of an otologic emergency. His chief complaint was a precipitous, non-fluctuating loss of hearing affecting both ears simultaneously. The onset was acute, occurring exactly seven days prior to his admission, placing him at the outer edge of the optimal therapeutic window for sudden sensorineural hearing loss (SSNHL). Unlike the more common unilateral presentations of SSNHL, which patients often notice upon waking or attempting to use a telephone, this patient experienced a synchronous, bilateral shutting down of auditory input. This abrupt sensory deprivation was accompanied by continuous, high-pitched tinnitus that was subjectively louder in the right ear, alongside a distressing sensation of aural fullness in both ears.

A detailed history was taken to disentangle potential etiologies. The patient denied any vestibular symptoms at the onset, such as vertigo, spinning sensations, or disequilibrium, suggesting that the pathology was confined to the cochlear portion of the vestibulocochlear nerve and spared the vestibular labyrinth. There was no history of active otologic symptoms such as otorrhea (ear discharge) or otalgia (ear pain), nor was there any recent head trauma that could account for temporal bone fracture or labyrinthine concussion. A systematic review of systems was negative for prodromal symptoms of upper respiratory tract infection, including fever, cough, or rhinorrhea immediately preceding the event, which helps to differentiate this case from typical post-viral cochleitis. Crucially, the patient's history was devoid of common environmental or iatrogenic triggers. He had no prior history of acoustic trauma or noise exposure, and he denied exposure to known ototoxic agents, including aminoglycoside antibiotics or platinum-based chemotherapy agents like cisplatin. Furthermore, he had no history of previous otologic surgery. The family history was notably negative for early-onset deafness or known autoimmune disorders, reducing the likelihood of a hereditary syndromic or genetic autoimmune etiology.

On physical examination, the patient appeared moderately ill due to the distress of sudden deafness but remained alert and cooperative (Table 1). Initial vital signs at admission were stable and within normal physiological limits for his age: Blood Pressure was 115/74 mmHg, Heart Rate 78 bpm, Respiratory Rate 18 breaths/minute, and Temperature 36.7°C. However, anthropometric evaluation revealed a critical biological variable relevant to the subsequent management of the case. The patient weighed 83 kg with a height of 169 cm. This yielded a Body Mass Index (BMI) of 29.0 kg/m². According to the World Health Organization (WHO) growth reference data for adolescents, this places the patient in the Overweight category, approaching the threshold for obesity. This finding is clinically significant as excess adipose tissue can act as a metabolically active endocrine organ, potentially contributing to a baseline inflammatory state and, more importantly, predisposing the patient to the metabolic and cardiovascular side effects of high-dose corticosteroid therapy.

The otoscopic examination was performed to rule out conductive causes of hearing loss. Inspection revealed that the bilateral external auditory canals were patent and free of debris. The tympanic membranes were intact, pearly grey in color, and exhibited a positive cone of light, indicating a healthy middle ear status. There were no signs of middle ear effusion, retraction pockets, or hemotympanum. Further assessment via anterior rhinoscopy and

oropharyngeal examination revealed no mucosal inflammation or adenoidal hypertrophy, effectively ruling out Eustachian tube dysfunction secondary to upper airway pathology. A focused neurological and vestibular assessment confirmed that the pathology was isolated to the auditory system. Cranial nerves III through XII were grossly intact, and facial nerve function was normal and symmetrical (House-Brackmann Grade I). Vestibular testing showed no spontaneous or gaze-evoked nystagmus, and the GANS Sensory Organization Performance test was normal. Tuning fork tests corroborated the suspicion of sensorineural loss. The Weber test showed no lateralization (midline localization), which is consistent with symmetrical bilateral hearing loss. The Rinne test was positive bilaterally (Air Conduction > Bone Conduction), but the Schwabach test was shortened, confirming that the reduction in hearing sensitivity was sensorineural rather than conductive.

Pure tone audiometry (PTA) performed on Day 0 of admission (Day 7 of symptom onset) quantified the severity of the deficit. The assessment confirmed severe sensorineural hearing loss in both ears with a flat configuration across all tested frequencies. Proper masking techniques were employed using the plateau method to ensure ear-specific thresholds and eliminate cross-hearing. The right ear (AD) demonstrated a Pure Tone Average (calculated at 500, 1k, 2k, and 4k Hz) of 82.5 dB HL, while the Left Ear (AS) showed a pure tone average of 81.25 dB HL. To assess the functional impact of this hearing loss, speech discrimination scores (SDS) were obtained, revealing poor speech intelligibility: 40% in the right ear and 44% in the left ear. This profound loss of clarity, combined with the threshold shift, severely compromised the patient's ability to communicate. Furthermore, the subjective burden of the condition was evaluated using the Tinnitus Handicap Index (THI). The patient scored 78, which classifies as Grade 4 or a very severe handicap. This score indicates that the tinnitus was not merely a background nuisance but a catastrophic symptom causing significant sleep disturbance and interference with daily activities.

A comprehensive laboratory panel was conducted to investigate potential systemic etiologies, tailored to the specific constraints of the resource-limited setting. Hematological analysis showed a complete blood count (CBC) that was unremarkable, with no signs of leukemia or acute infection. However, inflammatory markers provided a subtle diagnostic clue: The erythrocyte sedimentation rate (ESR) was elevated at 33 mm/hr (Reference range: <15 mm/hr). In contrast, the C-reactive protein (CRP) remained within normal limits. The metabolic profile, including random blood glucose, lipid profile, and renal function tests, was initially normal. Due to financial and resource constraints inherent to the local healthcare setting, specific advanced serological panels for autoimmune inner ear disease (such as Anti-Hsp70, Western blot for inner ear antigens) and comprehensive viral PCR panels (for CMV, HSV, VZV) could not be performed. Consequently, the diagnostic reasoning relied on the synthesis of clinical presentation (bilaterality) and available non-specific inflammatory markers (ESR). To rule out retrocochlear pathology, Magnetic Resonance Imaging (MRI) of the internal auditory canal was performed with gadolinium contrast. This imaging modality is the gold standard for detecting structural lesions affecting the vestibulocochlear nerve. The MRI revealed normal morphology of the vestibulocochlear nerve bilaterally, with no evidence of enhancing lesions, effectively excluding vestibular schwannoma, meningioma, or demyelinating diseases such as multiple sclerosis.

Given the severity of the bilateral sensorineural deficit and the suspicion of a systemic, likely immune-mediated etiology, the clinical team opted for an aggressive initial pharmacologic strategy (phase 1). The primary objective was to achieve immediate immunosuppression to halt the presumed inflammatory cascade within the cochlea before permanent hair cell apoptosis could occur. Following a pediatric consultation to clear the patient for high-dose steroid use, he was admitted for total bed rest and initiated on a regimen of Intravenous Methylprednisolone.

Table 1. Summary of Clinical, Audiometric, and Laboratory Findings on Admission

PARAMETER	CLINICAL FINDINGS & VALUES
I. Patient Demographics & History	
Patient Profile	17-year-old Male, Student
Chief Complaint	Sudden, non-fluctuating hearing loss (Onset: 7 days prior)
Associated Symptoms	**Bilateral Aural Fullness** **Severe Tinnitus (High-pitched)**
Negative History	No vertigo, otorrhea, head trauma, URI, noise exposure, or ototoxins.
II. Anthropometry & Vital Signs	
Hemodynamics	BP: 115/74 mmHg | HR: 78 bpm | Temp: 36.7°C (Stable)
Body Mass Index (BMI)	29.0 kg/m² **Overweight** *Risk factor for steroid-induced hypertension*
III. Physical & Neurological Examination	
Otoscopy	**Normal** Bilateral intact tympanic membranes, pearly grey, patent canals.
Cranial Nerves	**Intact** CN III-XII intact. Facial Nerve: House-Brackmann Grade I.
Tuning Fork Tests	**Weber:** No lateralization (Midline) **Rinne:** Positive Bilaterally (Shortened Schwabach)
IV. Baseline Audiometric Assessment (Day 0)	
Pure Tone Average (PTA)	Right Ear: **82.5 dB HL** **Severe SNHL** Left Ear: **81.25 dB HL** **Severe SNHL**
Speech Discrimination (SDS)	Right: 40% | Left: 44% (Poor intelligibility)
Tinnitus Handicap Index (THI)	Score: **78** **Grade 4 (Very Severe Handicap)**
V. Laboratory & Imaging Workup	
Inflammatory Markers	ESR: **33 mm/hr** **Elevated** (Ref: <15 mm/hr) CRP: Normal
Metabolic Profile	**Normal** Glucose, Lipids, and Renal Function within limits.
Specific Serology	**Not Performed** (Autoimmune/Viral panels unavailable due to resource setting)
MRI (IAC w/ Contrast)	**Normal** Normal vestibulocochlear nerve morphology; no retrocochlear lesions.

The dosage selected was 500 mg intravenously daily for a planned duration of five days. While this dosage is substantial, the rationale was grounded in weight-based pharmacokinetics. For an adolescent weighing 83 kg, this regimen approximates 6 mg/kg/day, a dosage consistent with pulse therapy protocols often utilized for organ-threatening autoimmune conditions such as lupus nephritis or autoimmune hemolytic anemia. The goal was to saturate the glucocorticoid receptors rapidly and breach the blood-labyrinth barrier, which is notoriously difficult for systemic drugs to penetrate. To support cochlear metabolism during this period of hypoxic stress, a comprehensive adjuvant protocol was instituted. This included neurotrophic support with Vitamin B12 (Mecobalamin) administered three times daily to support neural regeneration, folic acid once daily, and Zinc 20 mg once daily to act as an antioxidant cofactor. Furthermore, to combat cochlear hypoxia—a key final pathway in sudden hearing loss—the patient received hyperbaric oxygen support via normobaric oxygen therapy administered at 4 Liters per minute for 15 minutes every 6 hours.

The therapeutic course proceeded without incident until Day 7 (Day 14 of symptom onset). During the transition from the acute pulse phase to the tapering phase, the patient's clinical status deteriorated unexpectedly. He developed a severe, pounding headache accompanied by significant dizziness. Immediate vital signs monitoring revealed a hypertensive urgency, with blood pressure spiking to 150/95 mmHg. This event presented a critical diagnostic and therapeutic pivot point. The Pediatric Department diagnosed the condition as acute pediatric hypertension secondary to methylprednisolone toxicity. The pathophysiology likely involved the saturation of the enzyme 11β-hydroxysteroid dehydrogenase type 2, which normally inactivates cortisol and methylprednisolone in the kidney. Given the high dose and the patient's elevated BMI (29.0 kg/m²), the mineralocorticoid activity of the methylprednisolone remained unchecked, leading to aggressive sodium retention and volume expansion. This complication forced an immediate cessation of intravenous steroids to prevent hypertensive encephalopathy or cardiovascular strain. To manage the acute blood pressure elevation, the patient was started on the Angiotensin-Converting Enzyme (ACE) inhibitor Captopril at 12.5 mg three times daily. Simultaneously, the clinical team faced the risk of adrenal crisis from abrupt steroid withdrawal. To mitigate this while managing the hypertension, a rapid oral methylprednisolone taper was instituted (64 mg tapering down every 2 days). Clinically, the systemic phase was considered a failure regarding auditory salvage. Interim Pure Tone Audiometry on Day 7 revealed minimal improvement, with thresholds lingering at 75 dB HL in both ears (Severe Mixed Hearing Loss). Subjectively, the patient remained in distress, with the Tinnitus Handicap Index (THI) persisting at a score of 46.

With systemic therapy now contraindicated due to toxicity and the hearing loss proving refractory to the initial pulse regimen, the management strategy shifted to a local salvage protocol (phase 2). The rationale was to deliver high concentrations of corticosteroids directly to the perilymph via diffusion across the Round Window Membrane (RWM), thereby bypassing the systemic circulation and avoiding further hypertensive events. The chosen agent was Triamcinolone Acetonide (40 mg/mL). This steroid was selected for its distinct physicochemical properties; its viscosity allows for prolonged contact time with the round window compared to more soluble solutions. An injection technique was employed to maximize patient comfort and efficacy. The steroid solution was pre-warmed to body temperature (38°C) prior to administration. This step is crucial to prevent the caloric effect—a vertigo response triggered by convection currents in the lateral semicircular canal when fluids of a different temperature are introduced into the middle ear. Under local anesthesia using 10% Xylocaine spray, 0.8 mL of Triamcinolone was injected via a spinal needle into the posteroinferior quadrant of

the tympanic membrane. This site is anatomically favorable as it sits directly over the round window niche. Following the injection, the patient remained in a supine position with his head rotated 45 degrees to the contralateral side. This position utilizes gravity to pool the steroid solution in the round window niche, maximizing the concentration gradient for diffusion into the scala tympani. To ensure adequate absorption, this position was maintained for 60 minutes. This salvage protocol was administered over four cycles, with injections occurring at one-week intervals (Days 10, 17, 24, and 31 of hospitalization).

The patient was monitored closely throughout the salvage phase. Importantly, the intratympanic procedure proved safe, with no complications such as persistent tympanic membrane perforation, otitis media, or exacerbation of vestibular symptoms observed. The trajectory of recovery revealed a fascinating dissociation between audiometric thresholds and functional symptom resolution. By the third week (following the second IT injection), the Tinnitus Handicap Index (THI) had dropped dramatically to 10 (Mild Handicap), while pure tone thresholds showed modest improvement to Right 70 dB and Left 65 dB. By the fifth week (following the fourth and final injection), the patient's status had stabilized: (1) Audiometric outcome: The final pure tone audiometry stabilized at 65 dB HL bilaterally. According to Siegel's criteria, this represents a slight improvement. The patient transitioned from severe hearing loss to the moderate-severe range. While hearing aids would likely still be required for optimal communication, this gain is functionally significant, moving the patient out of the range of profound deafness; (2) Functional Outcome (Tinnitus): The most remarkable success was the complete resolution of tinnitus. The THI score dropped to 0 (No Handicap). Given that the initial score was 78 (Catastrophic), this represents a total restoration of quality of life regarding tinnitus distress; (3) Subjective Status: One month following the final injection, the patient reported a significant subjective improvement in his ability to communicate and interact in social settings. Final diagnosis for this patient was bilateral sudden sensorineural hearing loss (SSNHL), presumed immune-mediated etiology, status post-salvage intratympanic therapy (Table 2). The case concluded with partial audiometric recovery but complete functional resolution of tinnitus, validated by the successful pivot to local therapy in the face of systemic toxicity.

3. Discussion

The occurrence of bilateral SSNHL in a pediatric patient is a significant red flag that necessitates a shift in diagnostic thinking from vascular causes to systemic pathology. In resource-limited settings, where specific autoimmune markers (such as Anti-Hsp70) and comprehensive viral serologies are often unavailable, the diagnosis relies heavily on clinical acumen and accessible inflammatory markers.[11] In this case, the diagnosis of a presumed immune-mediated etiology was established based on the simultaneous bilateral presentation and the elevated ESR (33 mm/hr) in the absence of active infection.

However, this presumptive diagnosis warrants rigorous scrutiny. While bilateral involvement correlates strongly with autoimmune inner ear disease (AIED), approximately 70% of AIED cases are organ-specific, meaning systemic autoantibodies (ANA, RF) may be absent even when testing is available. Furthermore, the patient's BMI of 29.0 kg/m^2 introduces a confounding variable. Adipose tissue is metabolically active, secreting proinflammatory cytokines such as IL-6, which stimulates the hepatic production of acute-phase reactants, thereby elevating ESR and CRP baseline levels.[12] Therefore, while the clinical presentation mimics AIED, the diagnosis is best categorized as idiopathic bilateral SSNHL with inflammatory features. This distinction is crucial for prognosis, as true AIED often exhibits a fluctuating course, whereas this patient showed stabilization.[13]

TABLE 2. DIAGNOSIS, TREATMENT, FOLLOW-UP, AND OUTCOMES

Comprehensive overview of therapeutic interventions and clinical trajectory

Category	Detailed Clinical Data
I. Diagnostic Profile	
Primary Diagnosis	**Bilateral Sudden Sensorineural Hearing Loss (SSNHL)** Classification: Severe, Flat configuration
Etiology	**Presumed Immune-Mediated** Based on elevated ESR (33 mm/hr) and bilateral simultaneous presentation. Specific autoimmune panel negative.
II. Phase 1: Systemic Induction (Days 1–7)	
Systemic Protocol	**Intravenous Corticosteroid** **Methylprednisolone 500 mg IV daily**. *Adjuvants:* Mecobalamin (Vit B12), Folic Acid, Zinc, Hyperbaric Oxygen Therapy.
Adverse Event	**Hypertensive Urgency** **Onset:** Day 7 of therapy. **Vitals:** BP spike to 150/95 mmHg, headache, dizziness. **Diagnosis:** Methylprednisolone-induced hypertension.
Management of AE	Immediate cessation of IV steroids. Initiation of Captopril 12.5 mg TID. Rapid oral taper to prevent adrenal crisis.
III. Phase 2: Salvage Intratympanic Protocol (Days 10–31)	
Salvage Rationale	Initiated due to systemic toxicity (HTN) and refractory hearing loss.
Protocol Details	**Intratympanic Injection** **Agent:** Triamcinolone Acetonide (40 mg/mL), warmed to 38°C. **Dosage:** 0.8 mL per ear. **Technique:** Supine, head rotated 45°, maintained for 60 minutes.
Schedule	Four cycles administered at 1-week intervals.
IV. Clinical & Functional Outcomes	
Audiometric Outcome	**Slight Improvement** **Baseline:** Right 82.5 dB | Left 81.25 dB (Severe) **Final:** Stabilized at **65 dB HL** bilaterally (Moderate-Severe). *Result:* Meets Siegel's criteria for slight improvement.
Functional Outcome	**Complete Resolution** **Baseline Tinnitus Handicap Index (THI):** 78 (Very Severe). **Final THI: 0 (No Handicap)**. *Result:* Total resolution of tinnitus symptoms despite residual hearing loss.
Follow-up	No complications (tympanic perforation, otitis media, or vertigo) observed post-injection.

Systemic corticosteroids remain the cornerstone of SSNHL treatment due to their ability to downregulate pro-inflammatory cytokines (TNF-alpha, IL-1beta) in the cochlea.[14] However, this case illustrates a critical limitation: the metabolic vulnerability of the adolescent patient (Figure 1). Methylprednisolone acts on the distal renal tubules via mineralocorticoid receptors, increasing sodium reabsorption and potassium excretion, leading to volume expansion. In healthy individuals, the enzyme 11β-hydroxysteroid dehydrogenase type 2 (11β-HSD2) converts cortisol and methylprednisolone into inactive metabolites, protecting the mineralocorticoid receptor.[15] We hypothesize that the high-dose regimen (500 mg/day) saturated the 11β-HSD2 capacity in this patient. Furthermore, the patient's overweight status (BMI 29) likely contributed to metabolic priming. Adipose tissue expresses 11β-HSD type 1, which regenerates active cortisol from cortisone, locally amplifying glucocorticoid signaling. This double hit of exogenous high-dose substrate and endogenous metabolic amplification likely precipitated the hypertensive urgency.[16] Clinicians must recognize that overweight adolescents are not merely small adults but possess distinct metabolic risks requiring careful dosing titration or earlier adoption of local therapy.

Figure 1. Pathophysiology of steroid-induced hypertension in adolescents.

The transition to Intratympanic Triamcinolone was the pivotal management decision. The pharmacokinetic advantage lies in bypassing the systemic circulation and the blood-labyrinth barrier.[17] When injected into the middle ear, the steroid diffuses across the round window Membrane into the perilymph of the scala tympani. We utilized Triamcinolone Acetonide (40 mg/mL). While Dexamethasone is frequently cited for its high permeability, Triamcinolone was selected for its distinct physicochemical properties.[18] Its higher viscosity and particulate nature may allow for a sustained release effect within the middle ear cleft, potentially maintaining a concentration gradient across the round window for a longer duration than non-particulate solutions. The extended supine positioning (60 minutes) employed in this case likely further maximized this diffusion gradient, contributing to the efficacy despite the delayed initiation.[19]

A key finding in this case is the divergence between the audiometric outcome and the subjective tinnitus resolution. The 17.5 dB improvement (82.5 to 65 dB) represents a slight improvement by Siegel's criteria. The patient remains hearing-impaired, while the THI score dropped from 78 (Catastrophic) to 0 (No Handicap). This dissociation is clinically profound. Prognostic studies suggest that tinnitus persistence often correlates with preserved hair cell function (the active lesion theory), whereas a dead ear is silent. Conversely, the complete resolution of tinnitus here, despite residual hearing loss, suggests that the IT steroid therapy may have successfully quelled the cochlear synaptopathy (neuro-inflammation at the inner hair cell ribbon synapse) or reduced the aberrant firing of the auditory nerve, even if the structural integrity of the outer hair cells (responsible for threshold sensitivity) was not fully restored. This validates tinnitus resolution as a distinct and valuable primary endpoint for salvage therapy.[20]

This study is limited by its design as a single case report without a control group. A significant limitation due to the setting was the inability to perform specific autoimmune and viral serology, which precludes definitive etiological confirmation. Future research should focus on validating cost-effective biomarkers for pediatric AIED that are accessible in developing healthcare systems and establishing weight-based dosing guidelines for systemic steroids to prevent toxicity.

4. Conclusion

Bilateral SSNHL in the pediatric population is a sight- and life-altering event that warrants immediate investigation for systemic etiologies. This case demonstrates that while high-dose systemic corticosteroids are the standard of care, they carry significant risks of toxicity, such as acute hypertension, particularly in adolescents with elevated BMI. Intratympanic corticosteroid injection is an effective salvage therapy when systemic steroids are contraindicated, offering a route to deliver high-concentration anti-inflammatory agents to the cochlea while bypassing systemic side effects. In settings where extensive serology is unavailable, clinical presentation (bilaterality) and basic inflammatory markers (ESR) guide the diagnosis of presumed immune-mediated loss. Clinicians must implement rigorous cardiovascular monitoring for adolescents receiving high-dose steroids, specifically screening for BMI as a risk factor for hypertensive urgency.

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