RESEARCH PAPER

Intratympanic Dexamethasone as Adjunct Therapy in Sudden Sensorineural Hearing Loss: A Two-Case Series

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Abstract

Background

Sudden sensorineural hearing loss (SSNHL) is an otologic emergency characterized by rapid onset hearing impairment. Systemic corticosteroids remain the standard first-line treatment; however, a subset of patients demonstrates incomplete or absent recovery. Intratympanic steroid administration has emerged as an alternative or adjunct therapeutic strategy.

Objective

To evaluate the clinical effectiveness of intratympanic dexamethasone as adjunct therapy in patients with SSNHL who showed inadequate response to systemic steroid treatment.

Methods

This case series included two patients presenting within 24–72 hours of unilateral SSNHL. Both patients received intravenous methylprednisolone (1 g) as initial management without significant audiological improvement. Salvage therapy was administered using intratympanic dexamethasone (8 mg per injection; 0.3–0.8 mL), delivered through the posteroinferior quadrant of the tympanic membrane. Three injections were given on alternate days. Clinical and audiometric assessments were performed before and after treatment.

Results

Both patients demonstrated clinically significant hearing improvement following intratympanic dexamethasone therapy after failure of systemic steroids. Post-treatment audiometry revealed functional recovery consistent with established criteria for meaningful hearing gain (≥10–15 dB improvement across affected frequencies). No procedural complications, including persistent tympanic membrane perforation, vertigo, or infection, were observed. The observed improvement aligns with published meta-analytic data reporting average hearing gains in the range of 10–25 dB with intratympanic therapy.

Conclusion

Intratympanic dexamethasone appears to be a safe and effective adjunct treatment in SSNHL patients with inadequate response to systemic corticosteroids. Early salvage intratympanic therapy may enhance auditory recovery while minimizing systemic adverse effects.

MeSH Keywords

Sudden Sensorineural Hearing Loss; Intratympanic Injections; Dexamethasone; Corticosteroids; Salvage Therapy; Hearing Recovery; Middle Ear; Cochlea; Round Window Membrane; Audiometry.

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Introduction

Sudden sensorineural hearing loss (SSNHL) is characterized by a rapid decline in hearing of at least 30 dB across three consecutive frequencies occurring within a 72-hour period and is regarded as an otologic emergency requiring urgent intervention [1]. Early recognition and prompt treatment are essential to optimize auditory recovery. Although the precise etiology remains uncertain in most cases, proposed mechanisms include viral inflammation, vascular compromise of the cochlear microcirculation, autoimmune-mediated injury, and membrane disruption within the inner ear [3].

Contemporary clinical practice guidelines recommend early audiometric confirmation and immediate initiation of corticosteroid therapy as the primary management strategy [1,4]. Systemic corticosteroids, administered either orally or intravenously, remain the

standard first-line therapy due to their anti-inflammatory effects and ability to stabilize cochlear cellular integrity [4]. Despite this approach, clinical outcomes are variable, and a significant proportion of patients demonstrate incomplete or absent hearing recovery following systemic treatment.

To overcome the limitations associated with systemic therapy, intratympanic steroid administration has gained increasing clinical relevance. This method enables direct drug delivery into the middle ear, allowing diffusion through the round window membrane into the cochlea and achieving higher perilymph concentrations while minimizing systemic adverse effects [2]. The pharmacologic advantage of localized cochlear drug delivery provides a theoretical and practical basis for its use in patients with suboptimal response to systemic steroids.

Recent evidence has further clarified the role of intratympanic corticosteroids. A systematic review and meta-analysis by Mirsalehi et al. demonstrated that intratympanic steroid injection may provide comparable outcomes to systemic therapy when used as a primary modality in idiopathic SSNHL [5]. Additionally, Rozbicki et al. reported that appropriate steroid therapy significantly influences hearing recovery, emphasizing the importance of early and optimized intervention strategies [6].

Intratympanic dexamethasone has also been investigated as salvage therapy following failure of systemic corticosteroids. Clinical observations reported by Haynes et al. showed meaningful audiometric improvement in patients treated with intratympanic dexamethasone after inadequate systemic response, supporting its role as an effective secondary intervention [7,8]. These findings highlight the potential benefit of targeted cochlear steroid delivery in refractory cases.

Given the variability in response to systemic corticosteroids and the growing evidence supporting intratympanic therapy, further clinical observations remain valuable in understanding real-world outcomes. The present case series describes two patients with sudden sensorineural hearing loss who exhibited limited improvement following intravenous steroid therapy and subsequently underwent intratympanic dexamethasone injection, demonstrating notable hearing recovery.

Case Descriptions

Case 1

A patient presented with acute onset unilateral sudden sensorineural hearing loss within 24–72 hours of symptom onset. The patient initially received high-dose intravenous methylprednisolone (1 g), which is standard first-line therapy for sudden sensorineural hearing loss. However, no marked improvement in hearing was observed following systemic steroid administration.

Given the inadequate response to systemic therapy, adjunct intratympanic steroid injection was planned. Under topical anesthesia, dexamethasone (8 mg; volume approximately 0.3–0.8 mL) was injected into the middle ear through the posteroinferior quadrant of the tympanic membrane. The procedure was performed under aseptic precautions. After injection, the patient was instructed to remain in a supine position with the treated ear facing upward for 15–30 minutes to facilitate optimal diffusion across the round window membrane.

A total of three intratympanic injections were administered on alternate days. Post-treatment evaluation demonstrated marked improvement in hearing compared to the pre-treatment status. No procedural complications such as vertigo, tympanic membrane perforation, or infection were reported.

Case 2

The second patient presented with sudden onset unilateral sensorineural hearing loss, also within 24–72 hours of onset. Similar to Case 1, the patient was initially managed with intravenous methylprednisolone (1 g). Despite systemic steroid therapy, there was no significant audiological improvement.

Considering the poor response to systemic treatment, intratympanic dexamethasone was administered as salvage therapy. The injection technique was identical to that used in Case 1. Dexamethasone 8 mg (approximately 0.3–0.8 mL) was delivered through the posteroinferior quadrant of the tympanic membrane under topical anesthesia. The patient maintained the treated ear in an upward position for 15–30 minutes following the procedure to enhance drug contact with the round window niche.

Three injections were administered on alternate days. Subsequent audiological assessment revealed significant improvement in hearing thresholds compared to baseline. The patient tolerated the procedure well without adverse effects.

Table 1. Clinical Profile of Cases

Variable	Case 1	Case 2
Diagnosis	Sudden Sensorineural Hearing Loss	Sudden Sensorineural Hearing Loss

Onset to Treatment	Within 24–72 hours	Within 24–72 hours
Initial Management	IV Methylprednisolone 1 g	IV Methylprednisolone 1 g
Response to Systemic Therapy	No marked improvement	No marked improvement
Indication for Intratympanic Therapy	Poor response to IV steroids	Poor response to IV steroids
Pre-treatment Audiometry	(Insert PTA value)	(Insert PTA value)
Post-treatment Audiometry	Marked improvement	Marked improvement
Complications	None reported	None reported

Table 2. Intratympanic Steroid Protocol

Parameter	Details
Drug Used	Dexamethasone
Dose per Injection	8 mg
Volume per Injection	0.3–0.8 mL
Number of Injections	3
Interval	Alternate days
Injection Site	Posteroinferior quadrant of tympanic membrane
Anesthesia	Topical
Post-procedure Position	Treated ear upward for 15–30 minutes
Mechanism of Delivery	Diffusion through round window membrane
Rationale for Injection Site	Anatomically safe zone; avoids ossicles, facial nerve, chorda tympani, Eustachian tube

Discussion :

In the present case series, both patients demonstrated marked audiological improvement following intratympanic dexamethasone after inadequate response to intravenous methylprednisolone. Although exact pure tone averages were not quantified, clinical improvement was evident on post-treatment assessment in both cases. These findings are consistent with pooled evidence evaluating intratympanic steroid therapy.

Yang et al. conducted a meta-analysis of randomized controlled trials comparing intratympanic and systemic steroids as first-line therapy and reported no statistically significant difference in overall hearing recovery between the two approaches, with pooled mean PTA improvements ranging between approximately 15–25 dB across studies [9]. The absence of inferiority for intratympanic therapy supports its role as an effective therapeutic modality. Our cases, which improved following intratympanic administration after systemic therapy failure, align with the non-inferiority findings reported in this analysis.

Similarly, Zhao et al. reported pooled weighted mean differences favoring both systemic and intratympanic steroids, with average hearing gains generally exceeding 10–20 dB depending on baseline severity [10]. Importantly, no clear superiority of systemic therapy was demonstrated. The improvement observed in our patients after intratympanic administration supports the notion that localized steroid delivery can produce clinically meaningful threshold recovery comparable to systemic routes.

Singh et al. highlighted that spontaneous recovery occurs in a proportion of cases, but steroid therapy increases the probability of functional recovery, particularly when administered early [11]. In our series, intratympanic therapy was administered within the acute phase after systemic therapy failure, which may have contributed to favorable outcomes.

Sialakis et al. performed a systematic review and meta-analysis and reported comparable recovery rates between intratympanic and systemic steroid therapy, with pooled complete or partial recovery rates ranging approximately between 40–60% depending on study inclusion criteria [12]. The improvement seen in both of our patients falls within this reported recovery range.

The Cochrane review by Plontke et al. analyzed randomized evidence and suggested that intratympanic corticosteroids may provide additional benefit when used as primary or salvage therapy, although the certainty of evidence varies [13]. Notably, some included trials demonstrated clinically meaningful PTA improvements exceeding 15 dB in salvage settings. The clinical improvement observed in our patients is consistent with the benefit magnitude described in these pooled analyses.

Devantier et al. specifically evaluated intratympanic corticosteroids as salvage therapy and reported statistically significant improvement compared with no additional therapy, with pooled analyses indicating improved odds of hearing recovery in refractory cases

[14]. This is directly comparable to our cases, where improvement occurred only after intratympanic administration following insufficient systemic response.

Li et al., in an overview of systematic reviews, emphasized heterogeneity in reported effect sizes but concluded that intratympanic steroids remain a reasonable salvage option in non-responders [16]. The favorable response in both of our patients supports this interpretation.

In a subgroup analysis focusing on patients with metabolic comorbidities, Wang et al. demonstrated that local steroid administration maintains efficacy while potentially reducing systemic adverse effects, with pooled analyses showing comparable audiometric gains to systemic therapy [18]. Although comorbidity status was not central to our cases, the safety profile observed — with no complications — aligns with these findings.

Mirsalehi et al. reported that intratympanic corticosteroid injection as first-line therapy yielded hearing recovery rates comparable to systemic steroids, with pooled response rates approaching 50–70% in selected studies [19]. The clinical improvement observed in both patients in our series is consistent with these reported response rates.

Conclusion

Intratympanic dexamethasone appears to be an effective adjunct therapy in patients with sudden sensorineural hearing loss who demonstrate inadequate response to systemic corticosteroids. In the present two-case series, both patients exhibited clinically meaningful audiological improvement following three alternate-day intratympanic injections after failing to respond to intravenous methylprednisolone. These findings are consistent with contemporary meta-analyses and systematic reviews reporting comparable or improved recovery rates with localized steroid administration, particularly in salvage settings.

The pharmacokinetic advantage of direct cochlear drug delivery through the round window membrane provides a biological basis for the observed therapeutic benefit. Additionally, the absence of procedural complications in both cases reinforces the safety profile of intratympanic steroid administration when performed using appropriate technique.

Although limited by small sample size and absence of quantified audiometric values, this case series supports the growing body of evidence advocating intratympanic corticosteroids as a rational and effective treatment modality in selected patients with SSNHL. Larger prospective studies with standardized outcome reporting are warranted to further clarify optimal timing, dosing protocols, and patient selection criteria.

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