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Complete Hearing Recovery in Sudden Sensorineural Hearing Loss with Type 2 Diabetes Mellitus and Steroid-Aggravated Hyperglycaemia: A Case Report

Ni Made Pasmiati Setyaningsih[1], Ni Putu Oktaviani Rinika Pranitasari[2]
[1]Department of Otorhinolaryngology Head and Neck Surgery, Faculty of Medicine, Universitas Udayana / Prof. Dr. I.G.N.G. Ngoerah General Hospital, Denpasar, Bali, Indonesia, pasmiatisetyaningsih@gmail.com
[2]Department of Otorhinolaryngology Head and Neck Surgery, Faculty of Medicine, Universitas Udayana / Prof. Dr. I.G.N.G. Ngoerah General Hospital, Denpasar, Bali, Indonesia, rinika.ps@unud.ac.id

Corresponding Author: pasmiatisetyaningsih@gmail.com[1]

Abstract: Sudden sensorineural hearing loss (SSNHL) is an otologic emergency defined as a hearing decline of at least 30 dB across three contiguous frequencies within 72 hours. Type 2 diabetes mellitus (T2DM) is a recognised risk factor and a predictor of poorer recovery, yet high-dose corticosteroid remains first-line therapy, creating a therapeutic dilemma. A 56-year-old woman with severely uncontrolled T2DM (HbA1c above 14%) and hypercholesterolaemia presented two days after sudden right-sided hearing loss. Audiometry confirmed moderate-to-severe SSNHL of the right ear. She received intravenous methylprednisolone tapered over 15 days, five sessions of hyperbaric oxygen therapy, statin therapy, and endocrinology-guided basal-bolus insulin. Hearing thresholds returned to normal by treatment day seven and remained normal at day twelve, meeting complete recovery by modified Siegel's criteria. Capillary glucose exceeded the glucometer limit of 500 mg/dL on four occasions, highest measured value 496 mg/dL. Hyperglycaemia was managed by insulin intensification without reducing or discontinuing corticosteroid, and no clinical hyperglycaemic crisis was documented. Causal attribution of recovery to treatment cannot be established from a single case because several favourable prognostic factors were present. Full-dose corticosteroid appears feasible in severely uncontrolled T2DM when structured glucose monitoring and endocrinology collaboration are available.

Keywords: Sudden Sensorineural Hearing Loss, Type 2 Diabetes Mellitus, Corticosteroid, Hyperglycaemia, Hyperbaric Oxygen Therapy.

INTRODUCTION

Sudden sensorineural hearing loss (SSNHL) is an otologic emergency that may result in permanent hearing impairment when treatment is delayed. The condition is defined as a

sensorineural hearing reduction of at least 30 dB across three contiguous audiometric frequencies occurring within 72 hours (Chandrasekhar et al., 2019). Reported incidence ranges from 5 to 20 cases per 100,000 persons per year, although the true figure is believed to be higher because a substantial proportion of patients recover spontaneously and never present to a health facility (Kuhn et al., 2011; H. A. Lee & Chung, 2024). Recognised aetiological categories include metabolic, infectious, autoimmune, traumatic, vascular, and neoplastic causes, yet no specific cause is identified in most patients, and the condition is then classified as idiopathic (Prince & Stucken, 2021; Singh & Kumar Irugu, 2020; Tripathi & Deshmukh, 2022).

Diabetes mellitus is a metabolic disorder arising from impaired insulin secretion, impaired insulin action, or both, and it produces chronic hyperglycaemia with progressive microvascular and neural injury. Its prevalence continues to rise, and Indonesia ranks among the countries with the highest absolute number of affected adults. Several mechanisms connect diabetes to cochlear dysfunction. Narrowing of the stria vascularis lumen is one of the most consistently reported histopathological findings, and it is accompanied by impaired auditory nerve transmission and by mitochondrial injury secondary to oxidative stress, which damages the outer hair cells responsible for mechanoelectrical transduction (Deng et al., 2023; Helzner & Contrera, 2016). These processes explain the higher prevalence of sensorineural hearing loss observed in people with diabetes compared with people without diabetes.

Beyond chronic hearing impairment, diabetes also increases susceptibility to SSNHL and worsens its prognosis. Population-based cohort data indicate a higher risk of developing SSNHL among people with diabetes than among matched controls (Lin et al., 2012). A recent meta-analysis of severe to profound SSNHL identified diabetes, hypertension, age above 60 years, vertigo, profound hearing loss, and delayed treatment as consistent predictors of poor recovery (H. A. Lee & Chung, 2024; S.-W. Lin et al., 2012). The labyrinthine artery supplies the cochlea without collateral compensation, so the organ is exquisitely sensitive to transient ischaemia, and microangiopathy therefore represents the leading mechanistic hypothesis linking diabetes with sudden cochlear failure (Tsuzuki & Wasano, 2024).

Systemic corticosteroid remains the mainstay of initial treatment for SSNHL, and hyperbaric oxygen therapy may be added within two weeks of onset (Chandrasekhar et al., 2019). This creates a practical dilemma in patients with poorly controlled diabetes, because high-dose glucocorticoid predictably aggravates hyperglycaemia and may precipitate hyperglycaemic emergencies. Clinicians in this situation often reduce the corticosteroid dose or withhold it entirely, and the consequences of that decision for hearing outcome are poorly documented. Published SSNHL case reports rarely present the glycaemic data that would allow other clinicians to judge how the dilemma was actually resolved.

The present report describes a patient with severely uncontrolled type 2 diabetes who achieved complete hearing recovery, and it documents the daily capillary glucose values and insulin adjustments recorded while full-dose corticosteroid was maintained. The objective is to provide clinicians facing the same dilemma with a transparent account of both the auditory outcome and the metabolic cost.

METHOD

A 56-year-old woman was referred from an otorhinolaryngology outpatient practice to the emergency department with sudden right-sided hearing loss that had begun two days earlier. The onset was abrupt, and the patient described a sensation of air entering the right ear. She denied otorrhoea, tinnitus, otalgia, vertigo, fever, cough, or rhinorrhoea. There was no history of air travel, diving, or ascent to high altitude, and no history of smoking or alcohol consumption. She had been diagnosed with type 2 diabetes mellitus four years earlier and had

been receiving insulin therapy since diagnosis, with irregular follow-up. No history of ototoxic drug exposure, head trauma, or preceding upper respiratory infection was identified.

On examination the patient was fully alert, with blood pressure 110/70 mmHg, pulse rate 80 beats per minute, respiratory rate 20 breaths per minute, and axillary temperature 36.4°C. Otoscopic examination showed patent external auditory canals bilaterally, intact tympanic membranes, and positive light reflexes. Nasal, pharyngeal, and neck examinations were unremarkable, with no cervical lymphadenopathy. Cranial nerve examination showed no focal deficit. Tuning fork testing showed a positive Rinne test bilaterally, Weber lateralisation to the left ear, and a shortened Schwabach test on the right, a pattern consistent with right-sided sensorineural hearing loss. Tympanometry demonstrated type A curves in both ears, excluding middle ear pathology (Figure 1).



Figure 1. Tympanometry at presentation showing type A curves in both ears

Pure-tone audiometry on the day of presentation showed moderate to severe sensorineural hearing loss in the right ear and normal hearing in the left ear (Figure 2). Mean thresholds are summarised in Table 2. Average thresholds reported in this manuscript were calculated manually from the air-conduction and bone-conduction values at 500, 1000, 2000, and 4000 Hz, and therefore differ slightly from the automatically generated pure-tone average displayed by the audiometer in the figures.

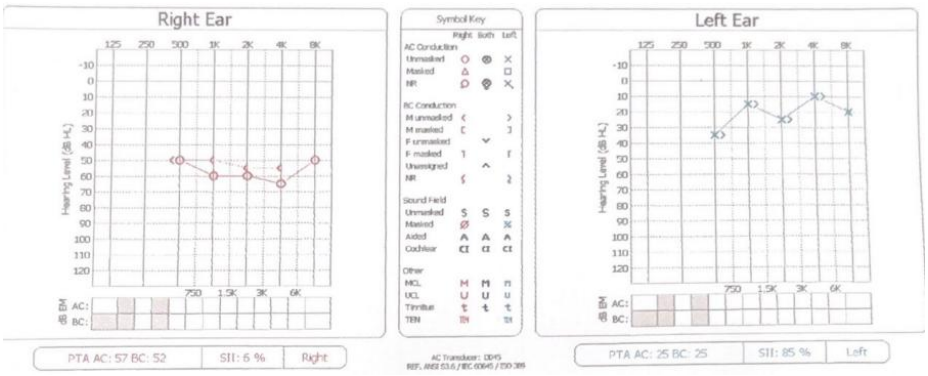


Figure 2. Pure-tone audiometry at presentation (day 1) showing moderate to severe sensorineural hearing loss in the right ear and normal hearing in the left ear

Laboratory evaluation showed a white blood cell count of 5.51 x 10^3/µL, absolute neutrophil count 3.31 x 10^3/µL, haemoglobin 12.9 g/dL, and platelet count 205 x 10^3/µL, giving a neutrophil-to-lymphocyte ratio of 1.86 and a platelet-to-lymphocyte ratio of 115.16. Aspartate aminotransferase was 15 U/L, alanine aminotransferase 12 U/L, blood urea nitrogen 8.1 mg/dL, and serum creatinine 0.78 mg/dL. Random plasma glucose was 352 mg/dL and glycated haemoglobin exceeded 14.0%. The lipid profile showed total cholesterol 342 mg/dL, low-density lipoprotein cholesterol 277 mg/dL, high-density lipoprotein cholesterol 61 mg/dL, and triglycerides 61 mg/dL. Serum sodium was 139 mmol/L and potassium 3.5 mmol/L. Chest radiography showed no cardiac or pulmonary abnormality.

The working diagnosis was moderate to severe right-sided sudden sensorineural hearing loss with type 2 diabetes mellitus and hypercholesterolaemia. Treatment comprised supplemental oxygen at 2 to 4 litres per minute for 15 minutes every 8 hours, intravenous sodium chloride 0.9% at 20 drops per minute, intravenous methylprednisolone, intravenous omeprazole 40 mg every 12 hours, and five daily sessions of hyperbaric oxygen therapy. Methylprednisolone was given as 125 mg once daily intravenously for three days, followed by 31.25 mg twice daily intravenously for three days, and then continued orally at 16 mg twice daily, 8 mg twice daily, and 4 mg twice daily for three days at each step, giving a cumulative dose of approximately 730 mg over 15 days. The patient was referred to the endocrinology division for management of diabetes, hypercholesterolaemia, and the anticipated glycaemic effect of high-dose corticosteroid. Simvastatin 20 mg once daily was started, together with basal-bolus insulin and structured capillary glucose monitoring.

Capillary glucose values, corticosteroid doses, and insulin adjustments during hospitalisation are presented in Table 1. Fasting glucose was already 328 mg/dL on the day of admission before the first corticosteroid dose was administered, which indicates that severe hyperglycaemia preceded rather than followed steroid exposure. Glucose subsequently exceeded the upper reading limit of the glucometer, corresponding to values above 500 mg/dL, on four occasions during days two, three, and five, and the highest numerically measured value was 496 mg/dL on day four. Insulin glulisine and insulin glargine were introduced and titrated by the endocrinology team. Corticosteroid dosing was neither reduced nor interrupted because of hyperglycaemia. No clinical features of hyperglycaemic crisis such as altered consciousness, Kussmaul respiration, or severe dehydration were documented, and ketone testing was not performed. Glucose values improved progressively from day five onward and were 200 mg/dL on the day of discharge.

Follow-up audiometry on treatment day seven, after completion of the fifth hyperbaric oxygen session, showed normal hearing bilaterally, and the patient reported subjective resolution of the hearing loss (Figure 3). She was discharged on oral methylprednisolone according to the tapering schedule described above, together with mecobalamin 500 micrograms every 8 hours, and was scheduled for outpatient review. Audiometry repeated on day twelve confirmed persistently normal thresholds in both ears (Figure 4). Serial audiometric results are summarised in Table 2.

Table 1. Daily capillary blood glucose, corticosteroid dose, and insulin adjustment during hospitalisation

Hospital day	Time point	Capillary glucose (mg/dL)	Methylprednisolone
1	Fasting	328	125 mg IV once daily (first dose)
2	2-hour postprandial	>500	125 mg IV once daily
3	Fasting	276	125 mg IV once daily
3	2-hour postprandial	>500	
4	Fasting	350	31.25 mg IV twice daily
4	22:00	496	
5	06:00	195	31.25 mg IV twice daily
5	22:00	>500	
6	06:00	290	31.25 mg IV twice daily
6	22:00	200	
7 (discharge)	Fasting	200	Switched to oral 16 mg twice daily

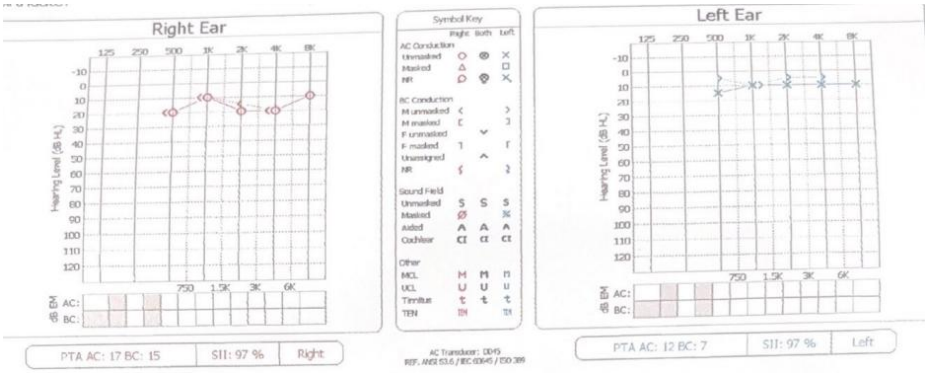


Figure 3. Pure-tone audiometry on day 7, after completion of corticosteroid and hyperbaric oxygen therapy, showing normal hearing in both ears

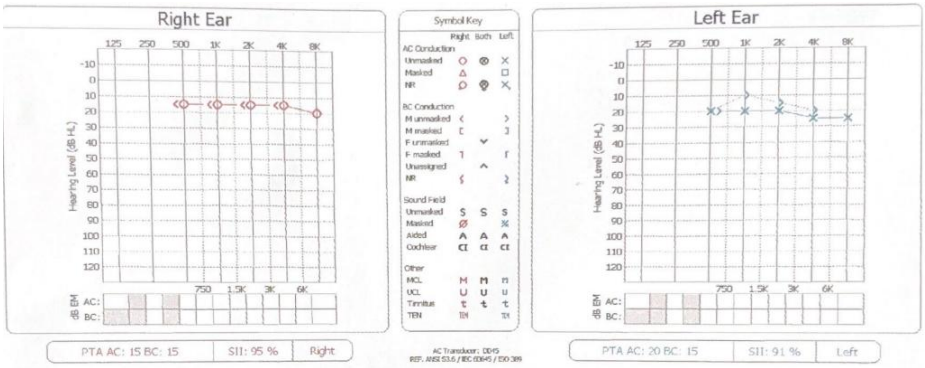


Figure 4. Pure-tone audiometry on day 12 at outpatient review, confirming persistently normal thresholds in both ears

Table 2. Serial pure-tone audiometric thresholds

Assessment	Right AC (dB)	Right BC (dB)	Left AC (dB)	Left BC (dB)	Interpretation
Day 1 (presentation)	58.75	52.50	21.25	21.25	Moderate to severe SNHL, right ear
Day 7 (end of inpatient therapy)	17.50	15.50	11.25	6.25	Normal hearing, both ears
Day 12 (outpatient review)	15.00	15.00	21.25	16.25	Normal hearing, both ears

AC, air conduction. BC, bone conduction. SNHL, sensorineural hearing loss. Values are averages of thresholds at 500, 1000, 2000, and 4000 Hz.

RESULTS AND DISCUSSION

Diagnostic confirmation of sudden sensorineural hearing loss

The diagnosis in this patient rested on the combination of an abrupt onset, a sensorineural pattern on tuning fork testing, type A tympanograms excluding middle ear disease, and audiometric confirmation within the recommended interval. Current guidance emphasises that audiometry should be obtained as soon as possible and within 14 days of symptom onset in order to distinguish sensorineural from conductive hearing loss (Chandrasekhar et al., 2019). Audiometry was performed on the day of presentation in this case, which is two days after symptom onset.

The right ear showed an average air-conduction threshold of 58.75 dB and an average bone-conduction threshold of 52.50 dB, giving an air-bone gap below 10 dB and confirming a sensorineural configuration. The interaural difference of approximately 37 dB satisfied the diagnostic threshold of at least 30 dB across three contiguous frequencies, using the contralateral ear as the reference in the absence of a premorbid audiogram, which is the comparison accepted when previous audiometry is unavailable (Chandrasekhar et al., 2019).

An important caveat concerns measurement precision. The unaffected left ear moved from an average air-conduction threshold of 21.25 dB at presentation to 11.25 dB on day seven and back to 21.25 dB on day twelve. A 10 dB fluctuation in an ear that was never symptomatic represents ordinary test-retest variability and indicates that differences of this magnitude in the affected ear should not be over-interpreted. The recovery observed in the right ear, a change exceeding 40 dB, comfortably exceeds that margin of uncertainty.

Diabetes mellitus, dyslipidaemia, and cochlear microvascular injury

Multiple lines of evidence link diabetes to sensorineural hearing loss. Large cross-sectional studies report a substantially higher prevalence of hearing impairment among people with diabetes compared with people without diabetes, and population-based cohort data show an elevated incidence of SSNHL specifically in the diabetic population (Deng et al., 2023; S.-W. Lin et al., 2012). Comparative series further indicate that patients with diabetes who develop SSNHL tend to present with greater initial threshold shifts and achieve lower complete recovery rates than patients without diabetes (Ju et al., 2021). Reported associations between disease duration and severity have been inconsistent, with some series describing only mild impairment in the first few years and progressively greater impairment beyond four years of disease, and others finding no clear duration effect (Deng et al., 2023; Helzner & Contrera, 2016). The present patient had a four-year history of diabetes, which places her within the range in which measurable auditory involvement has been reported, although a single case cannot support any duration-response inference.

Glycaemic control appears more informative than duration. Higher glycated haemoglobin values correlate with a greater prevalence of hearing impairment in both type 1 and type 2 diabetes, and prospective data specifically link elevated glycated haemoglobin with

SSNHL (Shen et al., 2021). This patient presented with glycated haemoglobin above 14%, which represents severe chronic hyperglycaemia and situates her at the unfavourable end of that association.

Dyslipidaemia constitutes a second vascular contributor. Hypercholesterolaemia has been associated with an increased risk of SSNHL, and the proposed mechanism is again vascular, given that the inner ear has a high metabolic demand and no collateral blood supply (Capaccio et al., 2012; Tsuzuki & Wasano, 2024). The lipid profile in this patient deserves specific comment. Total cholesterol of 342 mg/dL and low-density lipoprotein cholesterol of 277 mg/dL in the presence of triglycerides of only 61 mg/dL constitutes a pattern that should prompt evaluation for familial hypercholesterolaemia. Simvastatin 20 mg daily, the dose prescribed during admission, is unlikely to achieve an adequate reduction from a baseline of this magnitude, and high-intensity statin therapy with subsequent lipid reassessment would be the appropriate long-term strategy. This aspect of management represents a shortcoming of the present case rather than a model to follow.

Histopathological work supports the microvascular hypothesis. Narrowing of the stria vascularis lumen is among the most frequently reported findings in diabetic cochleae, and animal models have demonstrated marginal cell protrusion, intermediate cell swelling, and widening of intercellular spaces within the stria vascularis, together with degeneration of the hair cells responsible for converting sound vibration into neural signals (Deng et al., 2023). Impaired neural transmission and mitochondrial injury from oxidative stress act as additional contributors.

Corticosteroid therapy and steroid-aggravated hyperglycaemia

Corticosteroid remains the recommended initial pharmacological option for SSNHL, and guidance supports offering it within two weeks of symptom onset (Chandrasekhar et al., 2019). Pharmacokinetic work indicates that intravenous prednisolone at approximately 250 mg, corresponding to about 3.5 mg per kilogram of body weight, is required to achieve a substantial rise in perilymphatic cortisol concentration (Niedermeyer et al., 2003). The dose used here, 125 mg of methylprednisolone daily, was deliberately lower than that benchmark because of the anticipated glycaemic risk, and it is therefore notable that complete recovery still occurred. Intratympanic administration offers an alternative route that limits systemic exposure and is a reasonable consideration in patients with diabetes, with randomised data showing comparable outcomes to oral therapy (Rauch et al., 2011; Yang et al., 2020). Intratympanic therapy was not used in this patient, and its omission is acknowledged.

The metabolic course in this patient was not benign, and it should not be presented as though it were. Fasting glucose was already 328 mg/dL before the first corticosteroid dose, so the correct description is severe pre-existing hyperglycaemia that was further aggravated by glucocorticoid rather than hyperglycaemia induced by the drug. Glucose then exceeded the glucometer upper reading limit of 500 mg/dL on four separate occasions, with a highest measured value of 496 mg/dL. Values of that magnitude constitute a clinically significant adverse event, and they are reported here explicitly because the value of this case lies in transparency rather than in a favourable safety narrative.

Current inpatient guidance recommends basal-bolus insulin as the preferred approach for glucocorticoid-associated hyperglycaemia in non-critically ill patients, with more frequent monitoring and dose adjustment than would otherwise be applied (Lundholm & Morey-Vargas, 2023). Protocols developed during the coronavirus disease 2019 pandemic, in which large numbers of patients received high-dose corticosteroid, similarly favour early initiation of basal-bolus regimens over sliding-scale correction alone (Karol et al., 2023). The approach taken in this patient, with introduction of a rapid-acting analogue followed by a basal analogue under endocrinology supervision, is consistent with that guidance.

The practical message is that corticosteroid was neither reduced nor withheld. Glycaemic control was pursued by escalating insulin rather than by compromising the treatment directed at the ear, and hearing recovery proceeded to completion. Two important qualifications apply. Ketone testing was not performed at any point, so diabetic ketoacidosis and hyperosmolar hyperglycaemic state were never formally excluded, and monitoring was not performed on a fixed schedule, so the recorded values in Table 1 represent an incomplete sampling of the true glycaemic profile. Both shortcomings would need to be corrected before this approach could be recommended as a protocol.

Hyperbaric oxygen therapy

Reduced cochlear oxygenation is one proposed mechanism in SSNHL, and hyperbaric oxygen therapy is intended to increase oxygen delivery to the cochlea. Current guidance lists hyperbaric oxygen combined with steroid therapy within two weeks of onset as a treatment option rather than a recommendation (Chandrasekhar et al., 2019). A recent systematic review and meta-analysis of twenty studies found that patients receiving hyperbaric oxygen in combination with medical therapy had 2.61 times higher odds of hearing recovery than patients receiving medical therapy alone, with a consistent effect in the subgroup comparing hyperbaric oxygen plus systemic steroid against systemic steroid alone (Alter et al., 2026). Earlier meta-analytic work reached more cautious conclusions and highlighted the heterogeneity of treatment protocols (Joshua et al., 2022; Rhee et al., 2018).

The evidence base does not, however, support attributing this patient's recovery to hyperbaric oxygen. Earlier analysis suggested that benefit was greatest in patients with mean hearing loss of 70 dB or more, when hyperbaric oxygen was used as salvage therapy, and when treatment continued for at least 20 hours, while acknowledging low statistical power (Rhee et al., 2018). This patient had a mean loss of 58.75 dB, received hyperbaric oxygen as part of initial rather than salvage therapy, and completed only five sessions. The use of hyperbaric oxygen was reasonable and guideline-permitted, but any claim of a specific contribution to the outcome would exceed what the data allow. Variation in session number, pressure, and duration across published protocols remains a recognised limitation of this literature (Rozbicki et al., 2024). Reported protocols in the salvage setting typically involve considerably more sessions than the five delivered here, which further limits comparison with the published experience (J. W. Lee et al., 2024).

Inflammatory markers and prognostic assessment

Composite haematological indices have been examined as prognostic markers in SSNHL. The neutrophil-to-lymphocyte ratio and the platelet-to-lymphocyte ratio are typically higher in affected patients than in controls, and both are interpreted as surrogates of vascular inflammation during the acute phase (Diao et al., 2022). Meta-analytic evidence indicates that the neutrophil-to-lymphocyte ratio is prognostic and correlates negatively with hearing recovery.

Reported cut-off values vary considerably. One receiver operating characteristic analysis identified an optimal threshold of 6.661 for separating high responders from low responders, although that analysis was based on only twenty patients and should be regarded as exploratory (Kang et al., 2020). A threshold of 142.6 has been proposed for the platelet-to-lymphocyte ratio, with sensitivity of 51.5% and specificity of 68.4%, figures that indicate limited discriminative power (Diao et al., 2022). The values in this patient, a neutrophil-to-lymphocyte ratio of 1.86 and a platelet-to-lymphocyte ratio of 115.16, fall below both thresholds. The appropriate interpretation is the absence of an unfavourable inflammatory signal rather than the presence of a positive predictor of recovery.

Recovery was classified using modified Siegel's criteria, under which complete recovery corresponds to a final hearing level of 25 dB or better, partial recovery to a gain exceeding 15 dB with a final level of 26 to 45 dB, slight improvement to a gain exceeding 15 dB with a final level of 46 to 75 dB, no improvement to a gain below 15 dB or a final level of 76 to 90 dB, and a non-serviceable ear to a final level above 90 dB (Cheng et al., 2018). A final average threshold of 15.0 dB in the affected ear places this patient in the complete recovery category.

Interpretation of the outcome and limitations

The central limitation of this report is that causal attribution is not possible. Idiopathic SSNHL carries a high rate of spontaneous recovery, and a systematic review and meta-analysis of untreated or placebo-treated adults reported a pooled spontaneous recovery rate of 60.28%, albeit with substantial heterogeneity (Chaushu et al., 2023). This patient possessed several established favourable prognostic factors, including absence of vertigo, a non-profound degree of loss, and initiation of treatment two days after onset, which is well within the interval associated with better outcomes (H. A. Lee & Chung, 2024; W. Lin et al., 2022). Age above 60 years and diabetes were the principal unfavourable factors, and she was 56 years old, an age band in which recovery rates remain comparatively favourable (Ha et al., 2024). Recovery in this patient is therefore fully compatible with the natural history of the condition, and the corticosteroid, hyperbaric oxygen, and metabolic interventions cannot be separated from one another or from spontaneous resolution.

A second limitation concerns the diagnostic work-up. Magnetic resonance imaging and auditory brainstem response were not obtained, although current guidance recommends that patients with SSNHL be evaluated for retrocochlear pathology by one of these two modalities, and the 2019 update explicitly excludes audiometric follow-up as an acceptable substitute (Chandrasekhar et al., 2019). Imaging was not performed because of resource constraints and because recovery was rapid and complete. Retrocochlear pathology was therefore never formally excluded, and this represents a deviation from recommended practice rather than a justified omission. Complete threshold recovery lowers the pretest probability of a vestibular schwannoma but does not eliminate it.

A third limitation is the duration of follow-up. Audiometry was last performed on day twelve, and no further assessment was undertaken because thresholds had normalised. Guidance nevertheless recommends follow-up audiometric evaluation at the conclusion of treatment and again within six months, and accelerated long-term threshold decline and recurrence have both been described after apparently complete recovery from SSNHL (Chandrasekhar et al., 2019; Ko et al., 2024). Longer surveillance would have strengthened the report.

Finally, ketone testing was not performed despite glucose values above 500 mg/dL, glucose monitoring did not follow a fixed schedule, and lipid management was suboptimal for the degree of hypercholesterolaemia present. These points are stated so that clinicians reading this report understand precisely which elements represent transferable practice and which do not.

What this case adds is therefore narrower than the auditory outcome might suggest, and it concerns decision-making rather than efficacy. Clinicians managing SSNHL in a patient with glycated haemoglobin above 14% face a choice between withholding or reducing corticosteroid to protect metabolic safety and maintaining it to protect the cochlea. The published literature offers little empirical guidance on that trade-off, because case reports of SSNHL in diabetes seldom publish the glycaemic data generated during treatment, and randomised comparisons of full-dose against reduced-dose corticosteroid in poorly controlled diabetes have not been performed. A proposed trial comparing intratympanic with intravenous corticosteroid

specifically in diabetic patients illustrates that the question is recognised as clinically important but remains unresolved (Yang et al., 2020).

The present report contributes an explicit account of what happened when the corticosteroid dose was preserved. Glucose rose to values that any clinician would regard as alarming, insulin requirements escalated substantially, and the metabolic disturbance resolved as the corticosteroid dose was tapered. The absence of documented clinical decompensation should not be read as evidence of safety, because a single patient cannot establish safety and because ketone testing was omitted, which means the two most serious complications of this degree of hyperglycaemia were never actively excluded. The appropriate conclusion is conditional rather than general. Preserving the corticosteroid dose may be reasonable when daily capillary glucose monitoring, ketone assessment, and same-admission endocrinology input are all available, and it should not be attempted where those three conditions cannot be met.

Two further practice points follow from this case. The first concerns lipid management, since a low-density lipoprotein cholesterol of 277 mg/dL requires high-intensity statin therapy and evaluation for familial hypercholesterolaemia rather than the moderate-intensity regimen used here. The second concerns follow-up, since guideline-concordant care would have included assessment for retrocochlear pathology and repeat audiometry within six months even after complete threshold recovery (Chandrasekhar et al., 2019).

CONCLUSION

Complete hearing recovery was achieved in a patient with moderate to severe sudden sensorineural hearing loss and severely uncontrolled type 2 diabetes mellitus, treated with a tapering course of methylprednisolone, five sessions of hyperbaric oxygen therapy, statin therapy, and endocrinology-guided basal-bolus insulin. The principal contribution of this report is the documented glycaemic course rather than the auditory outcome. Capillary glucose exceeded 500 mg/dL on four occasions with a highest measured value of 496 mg/dL, and this was managed by insulin intensification without reducing or discontinuing corticosteroid, with no documented clinical hyperglycaemic crisis. Because idiopathic sudden sensorineural hearing loss recovers spontaneously in a substantial proportion of patients and because this patient carried several favourable prognostic factors, the recovery cannot be attributed to any specific intervention. What this case does suggest is that severe baseline hyperglycaemia need not automatically lead clinicians to withhold or reduce corticosteroid, provided that structured glucose monitoring, ketone assessment, and endocrinology collaboration are available. Prospective studies comparing full-dose and dose-reduced corticosteroid regimens in patients with poorly controlled diabetes are required before this observation can inform practice.

Declarations

Consent for publication. Written informed consent for publication of this case report and the accompanying figures was obtained from the patient. All information capable of identifying the patient has been removed from the text and from the figures.

Reporting guideline. This report was prepared in accordance with the CARE guidelines for case reports.

Conflict of interest. The authors declare no conflict of interest.

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Author contributions. Both authors contributed to patient management, data collection, drafting, and critical revision of the manuscript. Both authors read and approved the final version.

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