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Sudden Sensorineural Hearing Loss in a Patient with Hypertension: A Case Report

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Abstract: Sudden sensorineural hearing loss is an otologic emergency, and hypertension is often proposed as a vascular contributor to its onset and poor recovery. Published evidence is inconsistent, and accounts from settings without funded hyperbaric oxygen are limited. This report describes a hypertensive patient and compares the course with recent evidence. A 67-year-old man with treated stage II hypertension presented three days after sudden left hearing loss with tinnitus. His blood pressure was 160 over 100, and otoscopy was normal. Audiometry on day five after onset showed very severe left hearing loss with a mean air conduction threshold of 100 decibels. He received intravenous methylprednisolone, intermittent oxygen, candesartan, and a low-salt diet. Hyperbaric oxygen was not given because insurance did not cover it. By day twelve the left threshold had improved to 73.75 decibels, a gain of 26.25 decibels, which is a slight improvement by the Siegel criteria. A focused literature search found two meta-analyses with inconsistent support for hypertension as a risk or prognostic factor. This case adds an account of limited recovery in a hypertensive older adult under resource constraints and highlights blood pressure monitoring during steroid therapy.

Keywords: Sudden Sensorineural Hearing Loss, Hypertension, Corticosteroids, Hyperbaric Oxygenation, Case Report

INTRODUCTION

Sudden sensorineural hearing loss (SSNHL) is an otologic emergency that requires prompt diagnosis and management. It is defined as a sensorineural hearing loss of at least 30 decibels (dB) affecting three consecutive audiometric frequencies and occurring within 72 hours (Chandrasekhar et al., 2019). The loss is usually unilateral and may be accompanied by tinnitus, aural fullness, or vertigo. Estimated incidence differs between populations, and commonly quoted figures range from 5 to 27 cases per 100,000 people per year (Chandrasekhar et al., 2019; Lee & Chung, 2024). A cause is identified in only a minority of patients, so treatment is largely empirical. Prospective cohort data on evaluation and management are available from Asian settings (Mathews et al., 2020).

Vascular compromise is among the mechanisms most often proposed (Lee & Chung, 2024). The cochlea is supplied by the labyrinthine artery, which is an end artery without collateral circulation, so the cochlea tolerates ischemia poorly. Hypertension may contribute through endothelial dysfunction, vascular remodeling, and microthrombosis, which reduce cochlear perfusion and can lead to hair cell damage (Lee & Chung, 2024; Zheng et al., 2023). Cardiovascular risk has also been linked to hearing loss in clinical and population samples (Baiduc et al., 2023; Zheng et al., 2023).

The evidence that hypertension specifically worsens the outcome of SSNHL is mixed. A meta-analysis of 12 observational studies with 594,676 participants found that hypertension was associated with hearing loss in general, with a pooled odds ratio of 1.85 (95% confidence interval [CI] 1.55 to 2.21) (Jin et al., 2025). In contrast, a meta-analysis restricted to idiopathic SSNHL found no significant pooled association with high blood pressure, although it did for hypertriglyceridemia and high total cholesterol (Simões et al., 2023). Single-center cohorts report a worse prognosis in patients with hypertension (L. Li et al., 2025; Xu et al., 2024) and also report that overall cardiovascular risk was not significantly related to recovery (Lemons et al., 2023).

Treatment adds a second question. Systemic corticosteroids are the standard first-line therapy but can raise blood pressure (Chandrasekhar et al., 2019), and adjuncts such as hyperbaric oxygen are not available to every patient. This report describes these decisions in a hypertensive patient treated in a setting where hyperbaric oxygen is not covered by national health insurance. The clinical question was whether hypertension worsens the prognosis of hearing recovery in patients with SSNHL. The population was patients with SSNHL, the exposure was hypertension, the comparison was patients without hypertension, and the outcome was hearing recovery. This report therefore aims to discuss the management and short-term outcome of SSNHL in a patient with hypertension and to review current evidence on hypertension as a risk and prognostic factor.

METHODS

This is a single-patient case report prepared in line with the CARE reporting guideline (Riley et al., 2017). Clinical data were taken from the medical record of a patient treated at the Department of Otorhinolaryngology-Head and Neck Surgery, Ngoerah Hospital, Denpasar, Indonesia. SSNHL was defined as in the Introduction (Chandrasekhar et al., 2019). Hearing change was graded with the Siegel criteria (Siegel, 1975), which classify the result as complete, partial, slight, or no improvement from the gain in hearing level and the final threshold. Hearing levels are the mean air conduction (AC) and bone conduction (BC) thresholds in dB as recorded in the clinical file. To protect the patient's identity, events are dated in days counted from the onset of hearing loss (day 0), and identifying details were removed from the audiograms.

The patient gave written informed consent, signed on paper, for publication of this report and its images. The report describes routine clinical care and no study-specific intervention was performed. Review or exemption by an institutional ethics committee was not obtained.

To place the case in context, a focused literature search was done in PubMed and Google Scholar for articles published between 2020 and 2025. The search terms were “sudden sensorineural hearing loss,” “SSNHL,” “hypertension,” “vascular risk factors,” and “treatment outcome,” combined with Boolean operators and Medical Subject Headings terms where available. Duplicates were removed with reference management software, and titles, abstracts, and full texts were then screened. Inclusion criteria were studies discussing SSNHL associated with hypertension, systematic reviews or meta-analyses, English-language full-text articles published between 2020 and 2025, and studies evaluating the association between hypertension and SSNHL incidence or prognosis. Exclusion criteria were studies that did not involve hypertension as a comorbidity, narrative reviews, editorials, conference abstracts, case reports, and animal or *in vitro* studies. Screening of 2,485 records left two eligible reviews. From each

review we extracted the authors, year, design, number of studies and participants, outcome definition, and the main finding on hypertension. The search was not registered and was not designed as a full systematic review. Cohort studies and trials cited in the Discussion were selected non-systematically to give clinical context.

RESULTS AND DISCUSSION

Case Presentation

A 67-year-old man was brought by his family to the emergency department of Ngoerah Hospital on day 3 after sudden hearing loss in the left ear, which had developed abruptly and was accompanied by tinnitus. There was no history of otorrhea, otalgia, fever, cough, cold, recent air travel, travel to high altitude, diving, or head trauma. The patient did not smoke or drink alcohol. He had been treated for hypertension for more than five years with candesartan 8 mg once daily.

On examination he was alert, with a blood pressure of 160/100 mmHg, pulse rate of 83 beats/minute, respiratory rate of 20 breaths/minute, and axillary temperature of 36.7 °C. Both external auditory canals were clear and both tympanic membranes were intact with a positive light reflex. The nose, throat, and neck were normal and no cervical lymph nodes were palpable. Tinnitus was no longer present at the time of examination. Tuning fork testing showed a positive Rinne test bilaterally, a Weber test lateralized to the right ear, and a shortened Schwabach test.

Admission laboratory results are shown in Table 1. The chest radiograph showed no abnormality of the heart or lungs. Pure-tone audiometry on day 5 showed normal hearing in the right ear (AC 25 dB, BC 20 dB) and a very severe mixed hearing loss in the left ear (AC 100 dB, BC 51.25 dB) (Table 2 and Figure 1). The patient was diagnosed with left SSNHL and stage II hypertension.

Table 1. Admission laboratory findings

Parameter	Result	Unit	Parameter	Result	Unit
White blood cells	7.10	$\times 10^3/\mu L$	Sodium	142	mmol/L
Neutrophils	4.48	$\times 10^3/\mu L$	Potassium	3.83	mmol/L
Hemoglobin	14.3	g/dL	Total cholesterol	173	mg/dL
Platelets	318	$\times 10^3/\mu L$	LDL	130	mg/dL
Neutrophil-to-lymphocyte ratio	4.35	ratio	HDL	39	mg/dL
Random blood glucose	93	mg/dL	Triglycerides	59	mg/dL
AST	20	U/L	Blood urea nitrogen	11.1	mg/dL
ALT	17	U/L	Serum creatinine	1.18	mg/dL

Source: patient medical record. Units are the standard units for each test and were not stated in the original record. LDL, low-density lipoprotein cholesterol. HDL, high-density lipoprotein cholesterol.

The patient was admitted and received intravenous methylprednisolone 62.5 mg every 12 hours for 3 days, then 31.25 mg every 12 hours for 3 days. Other treatment was oxygen at 2 to 4 liters/minute for 15 minutes every 8 hours, 0.9% sodium chloride infusion at 20 drops/minute, intravenous omeprazole 40 mg every 12 hours, and oral vitamin B complex one tablet every 12 hours. He was referred to internal medicine (nephrology), who continued candesartan 8 mg once daily and prescribed a 1,900 kcal/day low-salt diet. At the patient's

request he was discharged on day 8 on oral methylprednisolone 16 mg every 12 hours for 3 days, then 8 mg every 12 hours.

Audiometry on day 8 showed a mild sensorineural loss in the right ear (AC 28.75 dB, BC 26.25 dB) and persistent very severe mixed hearing loss in the left ear (AC 100 dB, BC 81.25 dB) (Figure 2). The patient reported that left hearing was unchanged and that tinnitus persisted. At the outpatient visit on day 12 he reported that tinnitus had begun to improve. Audiometry showed normal hearing in the right ear (AC 22.5 dB, BC 15 dB) and severe mixed hearing loss in the left ear (AC 73.75 dB, BC 53.75 dB) (Figure 3). The left AC threshold had therefore improved by 26.25 dB from day 5, which is a slight improvement by the Siegel criteria (gain greater than 15 dB with a final level worse than 45 dB) (Siegel, 1975). The patient was advised to undergo hearing aid fitting and to continue hypertension management with internal medicine.

Table 2. Serial pure-tone audiometry

Day after onset	Ear	AC, dB (record)	BC, dB (record)	Printed PTA, AC/BC dB	Interpretation in record
5	Right	25	20	27/22	Normal hearing
5	Left	100	51.25	107/52	Very severe mixed
8	Right	28.75	26.25	30/30	Mild sensorineural
8	Left	100	81.25	105/80	Very severe mixed
12	Right	22.5	15	18/17	Normal hearing
12	Left	73.75	53.75	73/50	Severe mixed

Source: patient medical record and audiometer printouts. AC, air conduction. BC, bone conduction. PTA, pure-tone average. The record columns are the values stated in the clinical file. The printed PTA columns are the averages printed on each audiogram and are shown for transparency, because they differ from the record values.

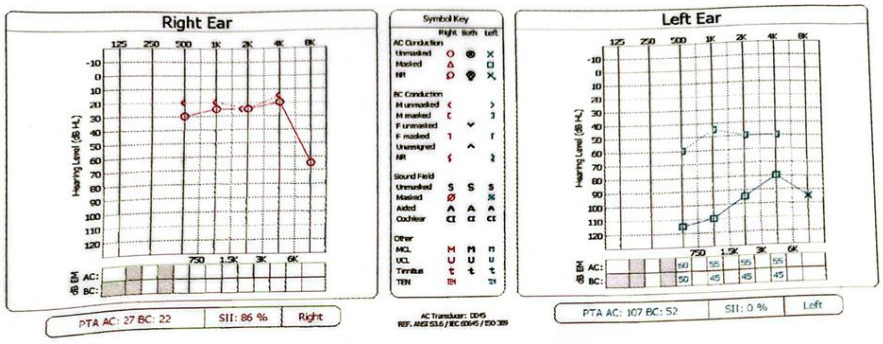


Figure 1. Pure-tone audiogram on day 5 after onset

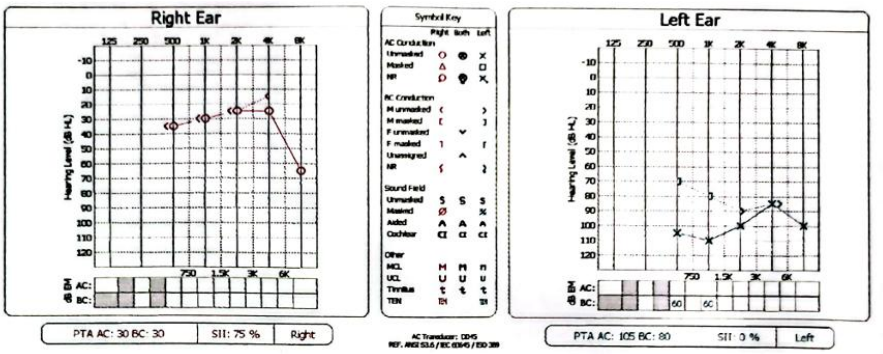


Figure 2. Pure-tone audiogram on day 8 after onset

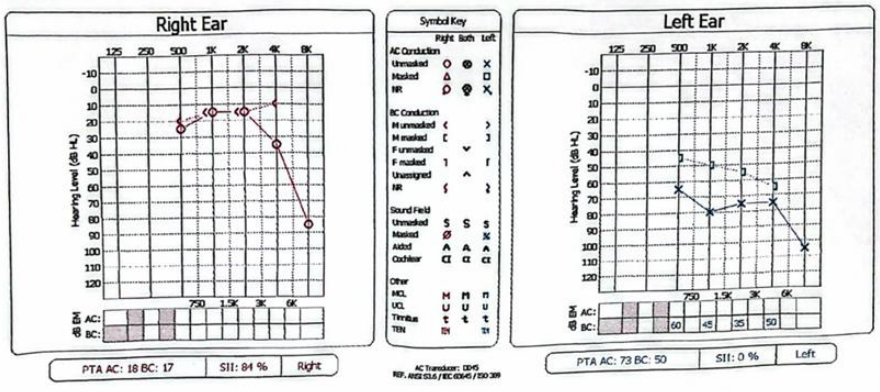


Figure 3. Pure-tone audiogram on day 12 after onset

Results of the Literature Search

Two studies fulfilled the eligibility criteria (Table 3). The first, by Jin et al. (2025), was a meta-analysis of 12 observational studies with 594,676 participants. Hypertension significantly increased the odds of hearing loss, with a pooled odds ratio of 1.85 (95% CI 1.55 to 2.21). This review addressed hearing loss in general and was not restricted to sudden onset.

The second, by Simões et al. (2023), was a systematic review and meta-analysis of 19 case-control and 2 cohort studies including 102,292 participants. Individual studies suggested a higher prevalence of hypercholesterolemia, diabetes mellitus, and higher blood pressure in patients with idiopathic SSNHL. In pooled analysis of adjusted odds ratios, however, the risk of SSNHL was significantly increased for hypertriglyceridemia (odds ratio 1.54, 95% CI 1.18 to 2.02) and high total cholesterol (odds ratio 2.09, 95% CI 1.52 to 2.87 after sensitivity analysis), but not for high blood pressure, diabetes mellitus, or low-density and high-density lipoprotein levels. The authors noted high heterogeneity.

Taken together, the two reviews do not agree. Hypertension was associated with hearing loss in general, but its association with SSNHL specifically was not confirmed in pooled data.

Table 3. Reviews included in the literature search

Study	Design and sample	Main finding relevant to hypertension
Jin et al. (2025)	Systematic review and meta-analysis of 12 observational studies, 594,676 participants, hearing loss of any type	Hypertension associated with hearing loss, pooled OR 1.85 (95% CI 1.55 to 2.21)
Simões et al. (2023)	Systematic review and meta-analysis of 19 case-control and 2 cohort studies, 102,292 participants, idiopathic SSNHL	No significant pooled association for high blood pressure. Significant for hypertriglyceridemia (OR 1.54, 95% CI 1.18 to 2.02) and total cholesterol (OR 2.09, 95% CI 1.52 to 2.87). High heterogeneity

Source: Jin et al. (2025) and Simões et al. (2023). CI, confidence interval. OR, odds ratio.

Hypertension as a Risk and Prognostic Factor

Our patient had several features linked to a poorer outcome. He was older than 60 years, his initial loss was very severe, and his blood pressure was 160/100 mmHg at presentation despite treatment (Lee & Chung, 2024). Hypertension has been associated with a worse prognosis in cohorts of patients with severe full-frequency loss (L. Li et al., 2025) and in a cohort that also examined metabolic syndrome (Xu et al., 2024). The partial result observed here is in line with that expectation. A single case cannot separate the contribution of hypertension from that of age and severity, and another cohort found no significant relation between overall cardiovascular risk and recovery (Lemons et al., 2023).

The pooled data do not settle the question. Simões et al. (2023) did not find high blood pressure to be a significant risk factor for idiopathic SSNHL, whereas Jin et al. (2025) found a nearly two-fold higher odds of hearing loss in hypertension, but for hearing loss of any onset. The estimate of Jin et al. therefore should not be read directly as a risk estimate for sudden loss. The metabolic picture of our patient is also incomplete. His total cholesterol was 173 mg/dL and triglycerides were 59 mg/dL, values that do not match the lipid profile linked to SSNHL risk in pooled analyses (J. Li et al., 2024; Simões et al., 2023). His HDL cholesterol of 39 mg/dL was low and blood pressure was elevated, but random glucose was normal, and waist circumference and body mass index were not recorded, so metabolic syndrome cannot be established. The neutrophil-to-lymphocyte ratio of 4.35 is reported for completeness, because hematological inflammatory indices have been studied as markers of SSNHL pathogenesis and prognosis (Zhao et al., 2025), but we cannot interpret it for an individual patient.

Treatment Choices in a Hypertensive Patient

Systemic corticosteroids remain the first-line therapy for SSNHL because of their anti-inflammatory effects, reduction of cochlear edema, and improvement of microcirculation (Chandrasekhar et al., 2019). Early treatment is associated with better recovery (Lee & Chung, 2024), and in this patient treatment began within one week of onset. The guideline dose is prednisone 1 mg/kg/day to a maximum of 60 mg/day for 7 to 14 days (Chandrasekhar et al., 2019). The initial methylprednisolone dose of 125 mg/day used here corresponds to about 156 mg of prednisone and exceeds that range. The reason for the higher dose is not documented. Corticosteroids can raise blood pressure and glucose, so closer monitoring was warranted, and the lack of recorded blood pressure values during admission is a gap. Collaboration with internal medicine is therefore important to optimize cardiovascular control during treatment. Candesartan was continued. Antihypertensive choice also matters for hearing, because some drugs, such as loop diuretics, can be ototoxic (Reynard & Thai-Van, 2024).

Intermittent oxygen therapy was given to improve cochlear oxygenation. This was normobaric oxygen at a low flow, and we are not aware of strong evidence that it changes the outcome, so it should not be read as equivalent to hyperbaric oxygen. Guidelines state that hyperbaric oxygen may be offered within two weeks of onset together with steroids (Chandrasekhar et al., 2019), and the evidence has been synthesized in an umbrella review (Liu et al., 2024). A network meta-analysis of salvage regimens reported that, under a restricted definition of refractory SSNHL, intratympanic steroid combined with hyperbaric oxygen gave the largest pure-tone gain (14.5 dB, 95% CI 4.2 to 25.0), whereas intratympanic steroid alone gave a mean gain of 7.7 dB (95% CI 4.8 to 10.7) (Lin et al., 2024). An overview of systematic reviews supports intratympanic steroid as salvage therapy but rates the methodological quality of the included reviews as low (D. Li et al., 2025). Hyperbaric oxygen was not given because it is not covered by national health insurance (BPJS Kesehatan) and the patient had financial limitations. Salvage intratympanic therapy was not documented in the record available to us. This access gap, rather than clinical judgment, probably shaped the options offered, and it is a policy question as much as a clinical one.

Interpretation of the Mixed Audiometric Pattern

The left ear was reported as a mixed hearing loss, yet the otoscopic findings were normal and the Rinne test was positive bilaterally. A positive Rinne result in an ear with an air-bone gap of about 49 dB on day 5 is hard to reconcile, because a gap of that size would usually give a negative result. Bone conduction in the left ear also moved from 51.25 dB on day 5 to 81.25 dB on day 8 and back to 53.75 dB on day 12, and right-ear thresholds varied by up to about 10 dB between sessions. These changes are larger than expected from a true change in cochlear function. In our interpretation, the conductive component is more likely to reflect measurement variability, including inadequate masking, than a true conductive loss. Mixed patterns can also

arise from conditions that do not involve the middle ear, such as intracochlear vestibular schwannoma (Reda et al., 2021), and middle ear inflammation can accompany sensorineural loss (Soni et al., 2023). Tympanometry and retrocochlear imaging were not available in the record we reviewed, so these explanations could not be tested.

Prognosis and Follow-up

Profound or flat audiometric patterns are generally associated with poorer recovery than low-frequency or ascending patterns (Chang et al., 2005; Diao et al., 2023; Poljak et al., 2022). Although the patient improved by 26.25 dB, the left ear remained in the severe range at 73.75 dB, and complete recovery was not achieved. The printed audiometer averages give a larger gain of 34 dB (from 107 to 73 dB), and the conclusion is the same. The final level was measured only 12 days after onset, and recovery may continue or plateau afterward, so the eventual outcome is unknown. Hearing aid fitting, as advised, is appropriate for a residual loss of this degree. Because recurrence of SSNHL has been reported in about 6.7% of patients in a nationwide cohort (Ko et al., 2024), continued audiometric follow-up and blood pressure control remain sensible.

In summary, this case shows limited recovery in an older hypertensive patient with a very severe initial loss who received early systemic steroids and blood pressure control but not hyperbaric oxygen. It does not show that hypertension caused the poor result, and the published evidence is itself divided. Its practical lessons are to record blood pressure during steroid therapy, to document steroid dosing against body weight, to test the middle ear and retrocochlear pathway when the audiogram looks mixed, and to plan salvage options early for patients who cannot access hyperbaric oxygen.

CONCLUSIONS AND RECOMMENDATIONS

Conclusion

SSNHL is an otologic emergency that requires prompt diagnosis and immediate treatment to maximize hearing recovery. In this 67-year-old patient with stage II hypertension, early systemic corticosteroids, intermittent oxygen, and blood pressure control were followed by a slight improvement by the Siegel criteria, and hearing remained in the severe range. The literature does not show clearly that hypertension worsens recovery from SSNHL. One meta-analysis linked hypertension to hearing loss in general, and another found no significant pooled association with high blood pressure in idiopathic SSNHL (Jin et al., 2025; Simões et al., 2023). Management of SSNHL in hypertensive patients should address both auditory and cardiovascular factors.

Limitations

This report has several limitations. It describes one patient and cannot support causal conclusions. Follow-up ended at day 12. The first audiogram may have been obtained after steroid treatment had started. Tympanometry, retrocochlear imaging, body weight, and serial blood pressure values were not available, the steroid dose was above guideline range, and the printed audiometer averages differ from the values in the record. The literature search was not a registered systematic review, and one of the two included reviews did not isolate SSNHL.

Recommendations

Clinicians treating SSNHL in patients with hypertension should record blood pressure at each step of steroid therapy, calculate steroid dose per kilogram of body weight, and obtain tympanometry and, where a mixed pattern is unexplained, retrocochlear evaluation. Where hyperbaric oxygen is not accessible, planning for salvage intratympanic therapy and early audiologic rehabilitation is advised. Future studies should use prospective cohorts with

standardized audiometric reporting and should report hypertension control, steroid dose, and time to treatment so that the prognostic role of hypertension can be tested directly.

Author Contributions

All authors made significant contributions to this study. These contributions included clinical care and data collection, literature review, interpretation of results, manuscript preparation, scholarly revision, and final approval of the manuscript for publication. All authors confirm that they have read and approved the final version of the article.

AI Disclosure Statement

During the preparation of this manuscript, the authors used Claude (Anthropic) for translation from Indonesian to English, restructuring to the journal template, language editing, and checking of reference metadata. The authors have thoroughly reviewed and revised the content and take full responsibility for the entire publication.

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