

Case Report

Idiopathic Sudden Deafness : A Case Report

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ABSTRACT

Background: Sudden sensorineural hearing loss (SSNHL) is an otologic emergency requiring prompt diagnosis and treatment. **Case Presentation:** A 45-year-old man presented with sudden bilateral hearing loss, worse in the right ear, accompanied by tinnitus. Audiometry confirmed profound SNHL in the right ear and moderately severe SNHL in the left ear with type A tympanograms. The patient was treated with intravenous methylprednisolone, mecobalamin, supportive therapy, oxygen therapy, and an oral steroid taper. **Conclusion:** Early recognition and corticosteroid therapy are essential to improve hearing recovery and prognosis. **Conclusion:** Early recognition and corticosteroid therapy are essential to improve hearing recovery and prognosis.

Keywords: Sudden Sensorineural Hearing Loss; SSNHL; Corticosteroid; Case Report; Audiometry.

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INTRODUCTION

Sudden deafness, or sudden hearing loss, is a condition characterized by rapid onset sensorineural hearing loss often without an immediately identifiable cause.¹ The incidence of this condition has been increasing globally. In the United States, the incidence ranges from 5–27 per 100,000 people, with approximately 66,000 new cases occurring every year.² In Japan, the incidence reached 27.5 per 100,000 in 2001 and has continued to rise. Meanwhile, in Germany, the incidence increased drastically from 20 per 100,000 in 2004 to 160–400 per 100,000 per year in 2011.³ In Indonesia, the annual incidence of SSNHL is estimated at 5 to 27 cases per 100,000 people.⁴ This upward trend highlights sudden deafness as a growing public health concern.

The exact etiology of sudden deafness remains fully unknown. Several local and systemic factors are recognized trigger, including vascular disease, viral infection, and autoimmune disease. Additionally, lifestyle factors such as stress, emotional pressure, mood alterations, irregular lifestyle, and sleep disturbances may contribute to its onset. The proposed pathophysiological mechanisms include inner-ear vasospasm, stria vascularis dysfunction, vascular embolism or thrombosis, labyrinthine membrane hydrops, and hair-cell injury.⁵

Clinically, sudden deafness is characterized by sudden hearing loss, tinnitus, a feeling of fullness in the ear, dizziness, vertigo, hypersensitivity to sound (hyperacusis), and paresthesia around the ear. In addition to these physical symptoms, some patients also experience psychological disturbances, such as anxiety and sleep disorders, which can negatively affect their quality of life.⁶

Although many studies have examined the factors associated with SSNHL, research specifically investigating the prognosis of patients with sudden deafness remains limited. Various factors may influence the rate of recovery, however, further research is needed to better understand that effect the prognosis of this condition. Therefore, this clinical study aims to identify independent prognostic factors associated with the treatment outcomes of

patients with SSNHL, thereby providing new insights into the management of this condition.

CASE REPORT

The patient was a 45-year-old, male, who was referred from Bhakti Rahayu General Hospital on 21 January 2025 with a one week history of sudden bilateral hearing loss, which was more severe in the right ear. He also reported of bilateral tinnitus. The symptoms developed suddenly upon awakening. Since then, he had experienced hearing difficulty that interfered with his daily activities. There was no history of a similar illness previously. Other complaints such as spinning dizziness, fever, cough, and runny nose were denied. There was no history of hypertension or diabetes mellitus. The patient works as a private employee.

On physical examination, the patient's general condition was good, with a fully alert (*compos mentis*) level of consciousness. Blood pressure was 135/75 mmHg, body temperature was 36.5°C, pulse was 78 beats/minute and regular, respiratory rate was 16 breaths/minute, and oxygen saturation was 99%. On ENT examination, the ear canals appeared patent without discharge, the tympanic membranes were intact, the nasal cavities were patent with pink mucosa, the turbinates were decongested without discharge, the septum showed no deviation, and the pharyngeal mucosa and tonsils appeared pink, with tonsil size T1/T1.

Audiometry performed on 22 January 2025 showed that the right ear had profound-degree SNHL with AC 96.25 dB and BC 80 dB, while the left ear had moderately severe SNHL with AC 70 dB and BC 62.3 dB. Tympanometry demonstrated type A bilaterally. Repeat audiometry on 24 January 2025 revealed profound SNHL in the right ear with AC 95 dB and BC 81.25 dB, while the left ear had moderately severe SNHL with AC 65 dB and BC 55 dB. Tympanometry remained type A in both ears.

Laboratory results showed a total cholesterol level of 189 mg/dL, triglycerides 55 mg/dL, HDL cholesterol 48 mg/dL, and LDL cholesterol 141 mg/dL. Hematology examination revealed a white blood cell (WBC) count of $10.54 \times 10^3/\mu\text{L}$, with 68.6% neutrophils, 24.1% lymphocytes, 6%

reported that approximately 70% of patients with SSNHL experience tinnitus, while 30%-40% present with vertigo.¹⁷ In this case, the patient experienced sudden bilateral hearing loss accompanied by tinnitus and headache upon waking in the morning. The exact pathophysiological mechanism underlying these symptoms remains uncertain.

The diagnosis of SSNHL is established based on the patient's medical history, physical examination, and audiometry and tympanometry evaluations. The patient's audiometry demonstrated profound SNHL in the right ear and moderately severe SNHL in the left ear, while tympanometry showed type A curves bilaterally. These findings are consistent with the diagnostic criteria of SSNHL, which include a hearing loss of at least 30 dB affecting three consecutive frequencies within 72 hours.¹⁸ The patient's lipid profile showed an elevated LDL level of 141 mg/dL, which may contribute to cochlear vascular impairment.¹⁵ Other laboratory tests showed a total cholesterol level of 189 mg/dL, triglycerides of 55 mg/dL, HDL of 48 mg/dL, and hematological and biochemical parameters within normal limits. Based on these findings, the patient was diagnosed with profound Sudden Sensorineural Hearing Loss (SSNHL) in the right ear (AD) and moderately severe SNHL in the left ear (AS).

Primary management of SSNHL relies on corticosteroid administration, either orally or intratympanically, to suppress cochlear inflammation and edema. Evidence indicates that initiating corticosteroid therapy within 14 days of onset enhances hearing recovery rates by up to 60%.¹⁵ In this case, the patient was treated with intravenous Methylprednisolone at a dose of 62.5 mg every 12 hours, followed by a gradual tapering regimen upon discharge. Supportive therapies such as neurotropic vitamins (Mecobalamin), dietary sodium restriction, and hyperbaric oxygen therapy were incorporated to optimize clinical outcomes.

The prognosis of SSNHL is heavily dictated by patient age, baseline severity of hearing loss,

and the timing of therapy initiation. Studies have demonstrated that patients who begin treatment within 7 days of symptom onset have significantly better hearing recovery than those whose treatment is delayed for more than two weeks.²¹ In this case report, the patient received therapy within one week of onset, providing a better chance of recovery.

CONCLUSION

This case report describes a 45-year-old male patient with bilateral sudden sensorineural hearing loss (SSNHL) who experienced sudden hearing loss upon waking in the morning. The diagnosis was established based on medical history, physical examination, and audiometry evaluation, which demonstrated profound SNHL in the right ear and moderately severe SNHL in the left ear. Laboratory tests showed elevated LDL levels, which may represent a vascular risk factor contributing to cochlear dysfunction. The patient was treated with intravenous corticosteroids as the primary therapy, along with supportive treatments including neurotropic vitamins and hyperbaric oxygen therapy. The patient's prognosis depends on the prompt initiation of treatment and the management of underlying risk factors. Patient education regarding modifiable risk factors, including protection from excessive noise exposure and appropriate control of cardiovascular risk factors, is essential to reduce the risk of recurrence and disease progression.

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