



Sudden Hearing Loss and Vertigo With Silent Pontine Infarction: A Case Report

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Most cases of sudden sensorineural hearing loss (SSNHL) occur without a specific identifiable cause, although vascular factors may serve as potential etiological contributors. Silent infarction refers to ischemic changes observed on imaging studies without accompanying clinical symptoms; however, this condition is clinically significant owing to the increased risk of future stroke. We report a case of left-sided SSNHL accompanied by dizziness in a 62-year-old male patient who was diagnosed with left pontine infarction without any other neurological symptoms. The cochlea and pons receive blood supply from the anterior inferior cerebellar artery; the cochlea lacks collateral vessels and is therefore susceptible to fluctuations in blood flow. This case report provides evidence to support the vascular hypothesis as the etiology underlying SSNHL.

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Introduction

Sudden sensorineural hearing loss (SSNHL) is defined as hearing impairment of more than a 30 dB decrease, occurring over a 72-hour period, on three consecutive frequencies of pure-tone audiometry. Its incidence is estimated at 5–20/10,000 individuals per year. Although various potential causes have been reported, including vascular events, coagulation disorders, autoimmune disease, and trauma, most cases of SSNHL are idiopathic [1]. Regardless of the etiology of the disease, the first-line treatment method for SSNHL is systemic high-dose steroids, and intratympanic steroid injections can be used as salvage treatment [2].

Silent stroke, also known as silent cerebral infarction, refers to the occurrence of cerebral infarctions that are asymptomatic or do not present with noticeable clinical symptoms [3]. These covert brain lesions are typically detected incidentally through neuroimaging techniques such as magnetic resonance imaging (MRI) [4], despite the absence of overt neurological deficits. Silent strokes are believed to result from

ischemic events in the brain, often associated with small vessel disease, microemboli, or thromboembolic events. Although they may not manifest with immediate symptoms, silent strokes have been recognized as significant contributors to long-term cognitive decline, dementia, and an increased risk of subsequent overt strokes [3,5].

The blood supply to the pons is provided by the branches of the basilar artery, including the superior cerebellar artery, anterior inferior cerebellar artery, and pontine artery. Because these vessels have well-developed anastomoses, the incidence of pontine infarction is very low [6,7]. However, the blood supply to the inner ear originates from branches of the anterior inferior cerebellar artery (AICA), which is an end artery without collateral circulation [8].

Here, we report a patient who was initially presented and treated for SSNHL with vertigo. The patient had a pontine infarction detected on MRI, but there were no neurological symptoms except for sudden hearing loss and dizziness. The diagnosis was a case of concomitant silent pontine infarction and SSNHL. Therefore, we treated with steroids and antiplatelet agents both peripheral hearing loss and silent pontine infarction, aiming to discuss the diseases and treatment outcome.

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Case Report

A 62-year-old male patient presented with sudden-onset left-sided hearing loss and vertigo, which had occurred 2 days prior to the visit. The patient had a medical history of hypertension and diabetes, and there was a family history of cerebral hemorrhage in his mother and siblings.

The patient's mental status was alert, and his vital signs were normal: blood pressure was 140/80 mm Hg, pulse rate was 82 bpm, respiration rate was 19/min, and body tempera-

ture was 36.6°C. In neurologic examination, no abnormal findings were observed including dysarthria, limb sensory loss, motor weakness, facial palsy, diplopia, ataxia, or other pathologic reflexes.

The external auditory canal and tympanic membranes appeared normal. Weber and Rinne tests demonstrated a sensorineural hearing loss on the left side.

Pure-tone audiometry showed normal hearing in the right ear, while the left side exhibited moderate sensorineural hearing loss of 70 dB with 16% speech discrimination (Fig. 1A).

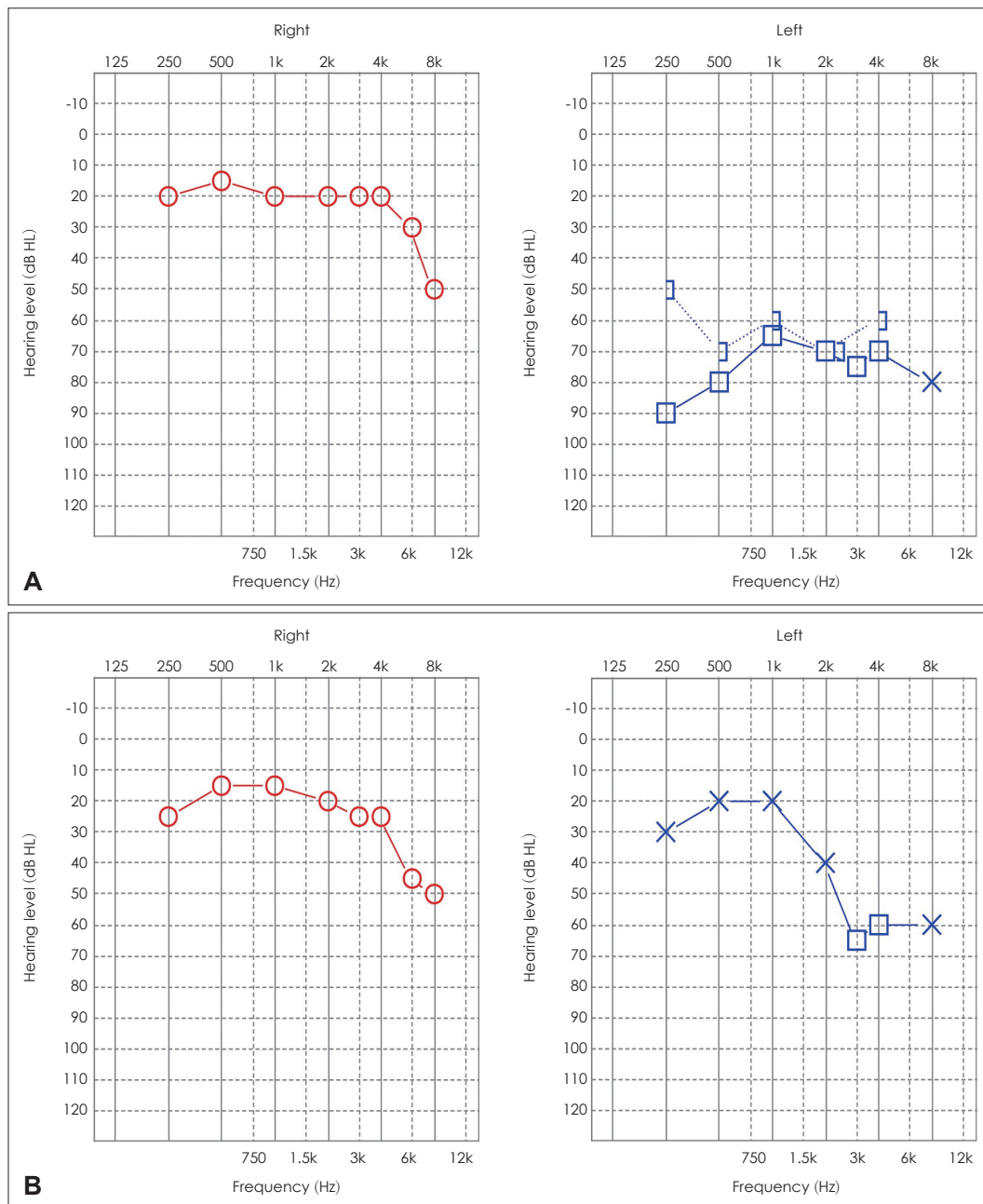


Fig. 1. Pure-tone audiometry shows the left moderately severe hearing loss on admission day (A) and much improvement at 1 month later after treatment (B).

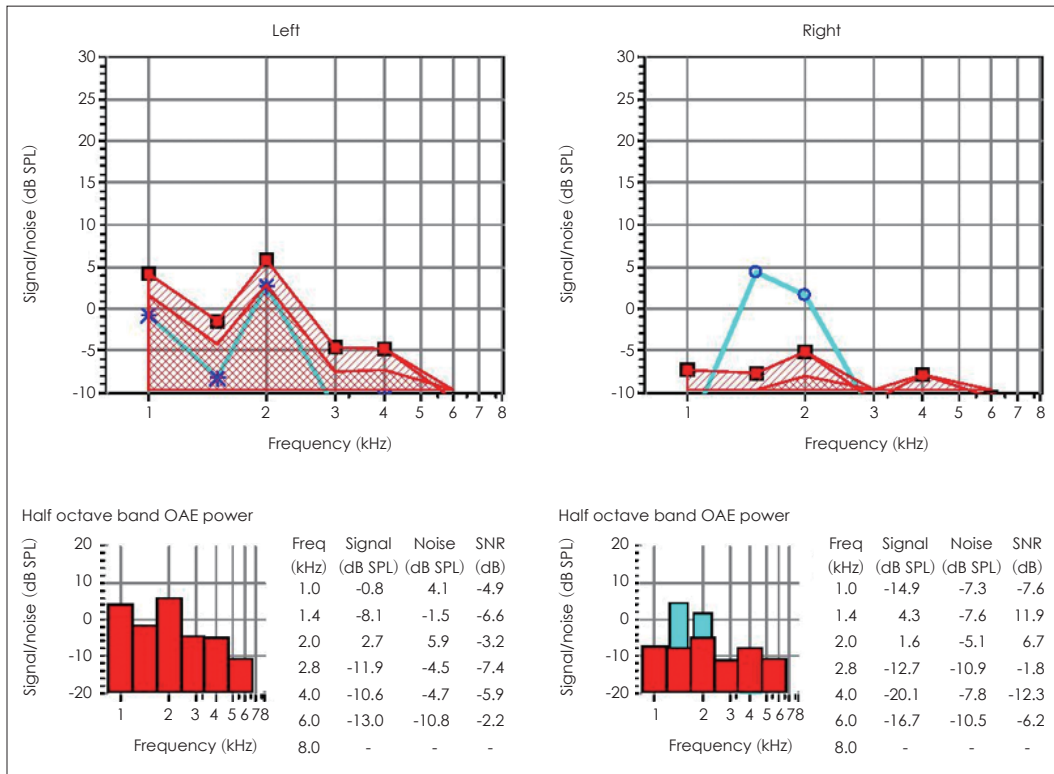


Fig. 2. Distortion product otoacoustic emission test (OAE) on admission day shows the absence of response in left ear. SNR, signal-to-noise ratio.

Distortion product otoacoustic emission (OAE) indicated no response in the left ear (Fig. 2). Videonystagmography demonstrated spontaneous right-beating horizontal nystagmus (Fig. 3A), and the bithermal caloric test revealed 85% left-sided semicircular canal paresis (Fig. 3B). Cerebellar function tests showed no dysmetria during finger-to-nose and heel-to-shin testing. The patient was diagnosed with SSNHL accompanied by vertigo.

MRI was performed to exclude retrocochlea pathologies. T1-weighted images showed no specific findings (Fig. 4A), while T2-weighted images (Fig. 4B) and diffusion-weighted images (Fig. 4C) showed increased signal intensity in the left pontine region, indicating acute pontine infarction. No additional neurological symptoms were observed during the hospitalization period.

Dual antiplatelet therapy was initiated with low-dose aspirin (100 mg/day) and clopidogrel (75 mg/day). In order to address SSNHL, intra-tympanic steroid injections were administered on days 1, 2, and 3 of the hospitalization. Daily pure-tone audiometry assessments conducted throughout the hospital stay revealed no improvement in hearing, but there was an improvement in dizziness symptoms.

A pure-tone audiometry performed 2 weeks after discharge showed hearing improvement to 49 dB on the left side, and

outpatient follow-up included 3 more intra-tympanic steroid injections. A pure-tone audiography performed 1 month after discharge showed improvement in the left side at 33 dB, but high-frequency hearing loss remained (Fig. 1B).

Discussion

The brainstem is composed of the medulla, pons, and mid-brain, and brainstem infarction can occur as a result of vascular disorders such as thrombosis, microembolism, and arteriosclerosis. It can present with a range of symptoms including hemiparesis, motor weakness, sensory loss, nystagmus, diplopia, dysarthria, and vertigo. Cases where there are no accompanying central symptoms other than hearing impairment are rare, and in such instances, it can be challenging to differentiate from sudden sensorineural hearing loss, acute labyrinthitis, Ménière’s disease, and other conditions [9].

The auditory pathway follows a sequential route through the cochlea, auditory nerve, cochlear nucleus, superior olivary nucleus, lateral lemniscus, inferior colliculus, medial geniculate body, and reaches the auditory cortex. Below the pons, the auditory pathway travels unilaterally, whereas above it, it travels bilaterally. Therefore, if a lesion affects the upper part of the cochlear nucleus, it can involve both sides of the audi-

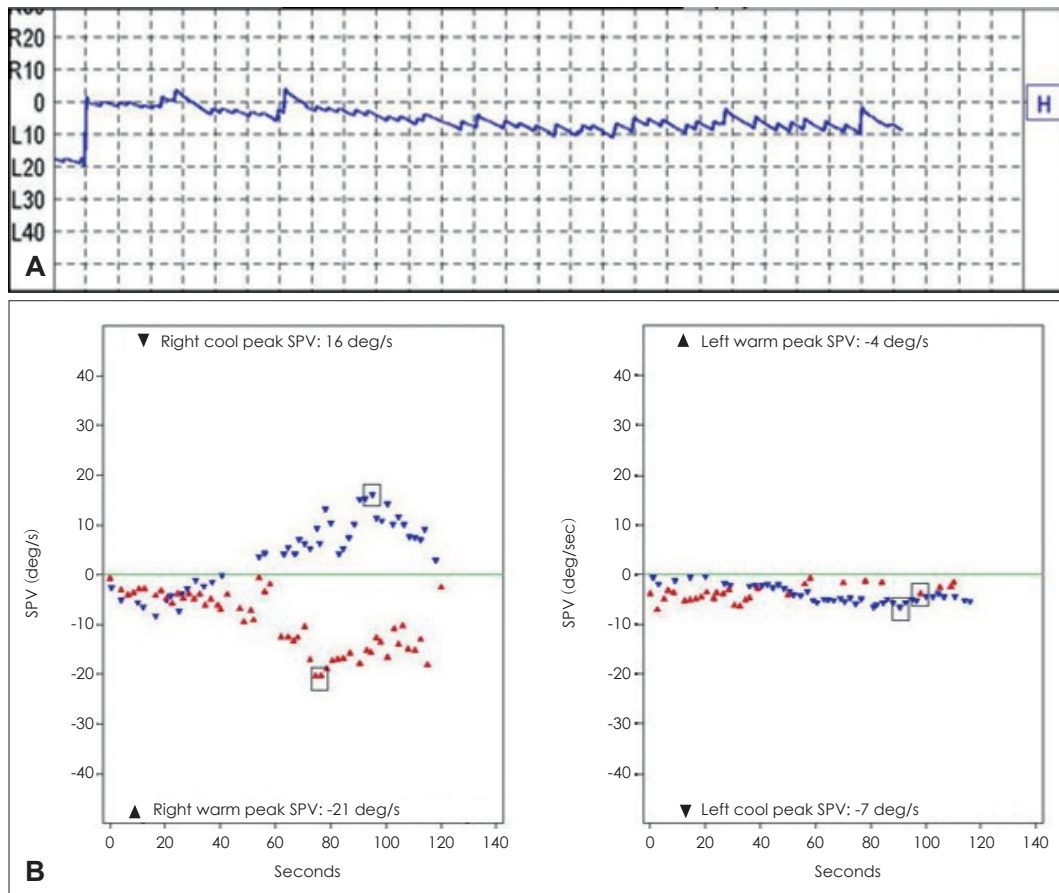


Fig. 3. The results of vestibular function test. A: Videonystagmography shows right beating spontaneous nystagmus. B: Bithermal caloric test shows 85% left-sided semicircular canal paresis. SPV, slow phase velocity.

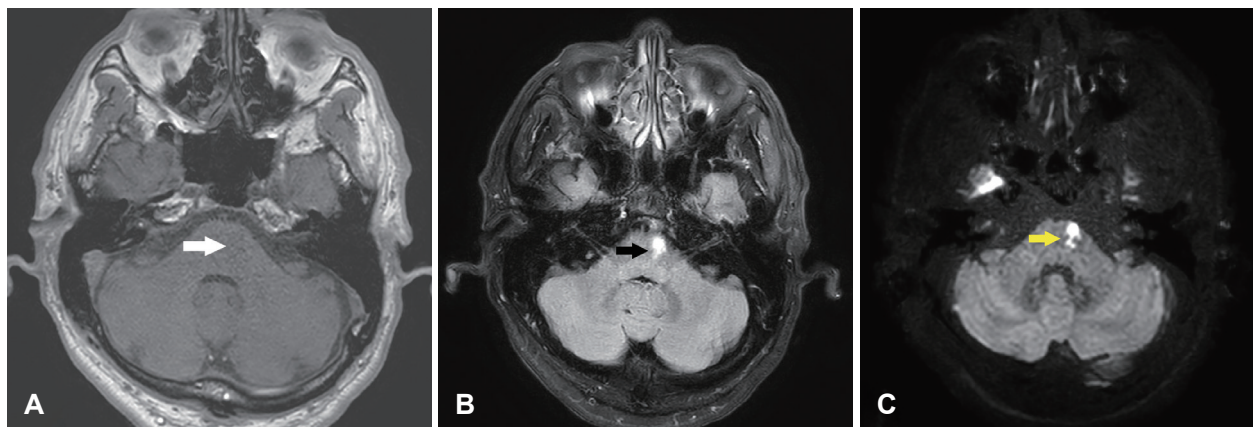


Fig. 4. Brain magnetic resonance imaging scan. T1-weighted axial image (A) shows no abnormal finding in pons (white arrow). But T2-weighted image (B) shows increased signal intensity in left pons (black arrow) and diffusion-weighted image (C) also show increased signal intensity lesion (yellow arrow) in the left pons, suggesting acute infarction.

tory pathway, resulting in bilateral hearing impairment. And if the lesion affects the lower part of the cochlear nucleus, it can lead to unilateral hearing impairment [9,10]. In cases of hearing loss resulting from pontine hemorrhage, the function of the cochlea is typically preserved, while there may be impairment in the central auditory pathway. This can lead to a

clinical presentation resembling auditory neuropathy spectrum disorders, with symptoms similar to those of sensorineural hearing loss [11]. In this case, a left pontine infarction was observed, along with left-sided hearing loss and impaired vestibular function. Decreased left-sided OAE responses suggested peripheral auditory dysfunction, and therefore, treat-

ment for SSNHL was continued.

The pons receives its blood supply from multiple arteries. The basilar artery is the primary supplier of blood to the pons. The pontine branches of the basilar artery include the superior cerebellar arteries, AICA [12], and pontine perforating branches. Additionally, collateral circulation between the vertebral and carotid systems contributes to the blood supply of the pons. The main arterial supply for the inner ear is the labyrinthine artery, which branches from the AICA. When an infarction affects the AICA, it can lead to symptoms such as vertigo, hearing loss, motor deficits, facial nerve paralysis, and other clinical manifestations by affecting the corticospinal, spinothalamic, and cerebellar tracts and the facial and vestibulocochlear cranial nerve nuclei as well as inner ear [11,13]. In terms of hearing, hearing loss can manifest on the contralateral side due to the decussation of the auditory pathway below the pons level. Different types of hearing impairment may occur, with peripheral auditory function being preserved while central auditory processing abilities are diminished [6,12,13].

In this case, MRI showed no abnormal finding on T1-weighted image and hyperintense lesion on T2-weighted images, localized to the ventral and mid to lateral portion of left pons which is supplied from perforating branches of the basilar artery and AICA. However, the patient did not exhibit any neurological symptoms other than hearing loss and vertigo suggesting silent pontine infarction. The hearing loss could be due to cochlear damage, as evidenced by the loss of distortion product OAE in the ipsilateral ear. The cochlea depends exclusively on the labyrinthine artery, a terminal branch of AICA, for its blood supply, and ischemia of labyrinthine artery can result in SSNHL. In consideration of the potential for SSNHL resulting from cochlear ischemia, intratympanic steroid injections were administered. Subsequent follow-up examinations revealed a notable improvement in hearing during the observation period.

Silent strokes, also known as silent cerebral infarctions, should be approached with caution due to their potential risks and implications. These strokes occur without noticeable symptoms or neurological deficits, but they can still cause brain damage and increase the risk of future strokes or cognitive decline. Recognizing and managing risk factors such as hypertension, diabetes, smoking, and high cholesterol are important in preventing and minimizing the dangers associated with silent strokes [14]. Treatment involves identifying and correcting underlying risk factors while administering anticoagulation therapy using heparin or antiplatelet agents. In this case, immediate anticoagulation therapy was initiated along with intratympanic steroid injections following the di-

agnosis of left pontine silent infarction, resulting in the recovery of hearing loss and dizziness.

There have been reports of several cases in which SSNHL was attributed to pontine infarction [6,11-13]. However, those cases were accompanied by abnormal neurological findings, including facial palsy, cerebellar ataxia, and suggesting central-type hearing loss. In specific, various forms of hearing loss have been reported in cases of pontine infarction. Bilateral hearing loss has been documented, as well as cases where hearing loss occurs on the opposite side of the pontine infarction according to central auditory pathway [6,12,13,15]. In addition, cases of hearing loss associated with pontine lesions have been reported to exhibit characteristics similar to auditory neuropathy spectrum disorder, where OAE are present but auditory brainstem response is absent, indicating preserved cochlear function [15]. In this case, the patient presented with silent pontine infarction, exhibiting ipsilateral hearing loss and dizziness, without any other neurological symptoms. In addition, absent OAE were observed, which could be attributed to concurrent pathology involving the pontine infarction and SSNHL resulting from ischemia of the AICA branches that supply both the pons and inner ear.

It is important to consider central pathology as the initial symptom of brainstem infarction can present as dizziness or hearing loss, with neurological symptoms potentially manifesting later, leading to a delayed diagnosis. These findings emphasize the need for careful consideration of central pathology in cases of pontine infarction-related hearing loss, which is crucial for accurate diagnosis and appropriate management.

Ethical Statement

This case report was approved by the local ethics review board (Hanyang University Guri Hospital Institutional Review Board, GURI 2023-10-010) and performed in accordance with the Declaration of Helsinki and good clinical practice guidelines. Informed consent was obtained from the patient.

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Conflicts of Interest

The authors have no financial conflicts of interest.

Author Contributions

Conceptualization: all authors. Data curation: all authors. Formal analysis: all authors. Investigation: Jae Yeong Jeong. Supervision: Jae Ho Chung. Validation: all authors. Visualization: all authors. Writing—original draft: Jae Yeong Jeong. Writing—review & editing: Jae Ho Chung. Approval of final manuscript: all authors.

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