Anterior Inferior Cerebellar Artery Infarction Presenting With Sudden Hearing Loss and Vertigo

Eun Jin Son, MD; Jung Hwan Bang, MD; Jae-Goo Kang, MD

A peripheral origin is typically contemplated in a patient presenting with sudden hearing loss (HL) and dizziness without other neurologic manifestations. Although symptoms of anterior inferior cerebellar artery (AICA) infarction include sudden HL and vertigo, the clinical picture usually shows ipsilateral facial anesthesia or paralysis, Horner's syndrome, contralateral body anesthesia, or cerebellar dysmetria. A 68-year-old female patient developed sudden HL in the right ear and vertigo. A left-beating horizontal torsional nystagmus was observed, and caloric weakness in the right side was noted. Diffusion- and T2weighted magnetic resonance imaging revealed cerebellar infarction in the right AICA territory. AICA infarction may present without obvious neurologic deficits, and an imaging study is advised in patients at high risk for vascular accidents. Key Words: Anterior inferior cerebellar infarction, sudden hearing loss, vertigo.

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INTRODUCTION

Sudden hearing loss (HL) and dizziness are typical symptoms of various labyrinthine disorders of both peripheral and central origins. Unilateral sensorineural HL with horizontal torsional nystagmus with a fast component directed to the same side as HL usually favors dysfunction of the peripheral auditory system between the cochlea and the auditory nerve distal to the root of entry. Anterior inferior cerebellar artery (AICA) infarction is known to be associated with HL, vertigo, facial weakness, ataxia, anesthesia, and Horner's syndrome. We report a patient who presented with only inner ear symptoms from AICA infarction.

CASE REPORT

A 68-year-old Korean female patient with a longstanding history of hypertension and diabetes mellitus

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presented with sudden HL in the right ear and dizziness for 2 days. She complained of feeling unsteady and a tendency to fall to the right side. One month ago, she had been evaluated for intermittent headache in the occipital area at another hospital, and computed tomography (CT) scans of brain revealed no abnormality. On physical examination, left-beating horizontal torsional spontaneous nystagmus was observed, and the direction did not change with gaze. She had no ophthalmoplegia, dysarthria, facial paralysis or numbness, Horner's syndrome, or cerebellar dysmetria. The eardrums appeared normal.

Pure tone audiograms showed a sensorineural HL of 60 dB, with 92% speech discrimination in the right ear (Fig. 1A). Stapedial reflex thresholds were normal. Electronystagmography confirmed spontaneous nystagmus after Alexander's law and caloric tests revealed right side vestibular weakness of 68%. Other routine laboratory findings including complete blood count, liver function tests, serum electrolytes, urea nitrogen, creatinine, and viral serology (varicella zoster virus and HIV) were insignificant. A labyrinthine origin for sudden HL and vertigo was suspected, and an oral steroid regimen and antiviral agent were started. Follow-up hearing tests revealed a slight improvement of 10 dB on day 2, which worsened back to the range of 70 to 90 dB on day 7 (Fig. 1B). Auditory brainstem responses to click stimuli at 90 dB nHL were absent in the right ear and normal in the left (Fig. 1C). Although other neurologic deficits did not develop, spontaneous nystagmus persisted to day 7, which was considered unlikely for a peripheral vertigo and necessitated further evaluation. T2-weighted and diffusionweighted gadolinium-enhanced magnetic resonance imaging (MRI) of the brain revealed acute infarction in the right middle cerebellar peduncle and dorsolateral pons, consistent with AICA territory (Fig. 2). Antiplatelet therapy was started. The patient's hearing showed no change over the next few weeks, and vertigo improved.

DISCUSSION

Acute vertigo that lasts for several days results from unilateral vestibular dysfunction, which can be from a peripheral or central origin. Sudden onset HL is usually attributed to viral labyrinthine disorders, but can also

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From the Department of Otorhinolaryngology, National Medical Center, Seoul, Korea.

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Send correspondence to Dr. Jae-Goo Kang, Department of Otorhinolaryngology, National Medical Center, 18-79 Euljiro 6-ga, Jung-gu, Seoul, Korea. E-mail: kwilly@medimail.co.kr

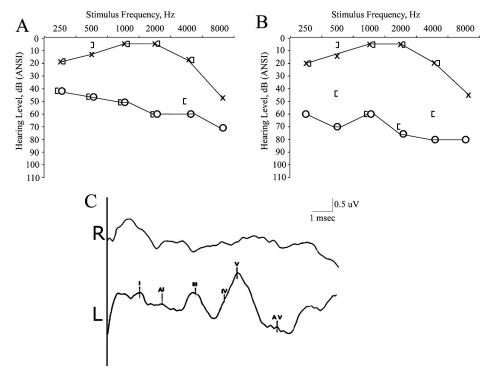


Fig. 1. Audiologic evaluations showing (A) moderate-severe sensorineural hearing loss (HL) on right side on initial pure tone audiogram, which worsened to severe HL after 7 days (B), and (C) absence of auditory brainstem responses on right side compared with normal responses on left side.

herald vertebrobasilar insufficiency.² Differentiation between peripheral and central lesions is important to rule out possibly life-threatening conditions. Our patient presented with sudden unilateral sensorineural HL and spontaneous unidirectional nystagmus with horizontal and torsional components, which point toward a peripheral source. Also, other focal neurologic deficits such as cerebellar dysmetria, facial paralysis or numbness, or dysphagia were not evident. Nonetheless, old age and a known history of hypertension and diabetes are sufficient to alarm the physician of the possibility of a vascular accident. The spontaneous nystagmus was observed for more than a week, which is unlikely for a peripheral vestibulopathy. Such delayed compensation also increases the suspicion for a central origin.

The inner ear and the vestibulocochlear nerve is supplied exclusively from the internal auditory artery, which stems from the AICA with few exceptions.3-5 Thus, AICA infarction can cause inner ear symptoms as well as brainstem signs such as crossed sensory loss, lateral gaze palsy, facial palsy, or Horner syndrome. Inner ear symptoms of sudden HL and vertigo as the sole manifestation of AICA infarction are rarely reported. There was only one other report of AICA infarction presenting with auditory and vestibular symptoms and no other signs.⁵ Inner ear dysfunction usually appears as prodromal signs before other neurologic signs.6 In most reported cases, the patients developed ataxia, dysmetria, diplopia, facial palsy, or other cerebellar dysfunction during the course of several days after onset when these findings were absent initially. ¹ In

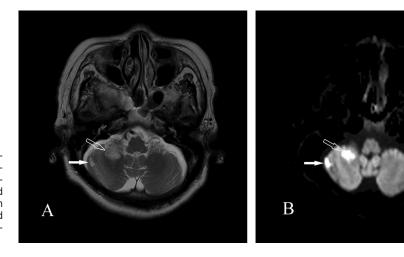


Fig. 2. Brain magnetic resonance imaging performed 9 days after onset of sudden hearing loss and vertigo. (A) T2weighted and (B) diffusion-weighted axial images revealed acute infarction in right middle cerebellar peduncle (filled arrow) and anterior cerebellar hemisphere (open arrow).

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some cases, aggravation of vestibular symptoms within few days also prompted reevaluation to confirm AICA infarction. In patients at high risk for vascular insufficiency, close observation for developing symptoms and follow-up neurologic examinations are important.

MRI scans of the brain proved invaluable to the correct diagnosis in our patient. In one report, diffusion-weighted brain MRI appeared normal initially, only to reveal AICA infarction in follow-up scans after aggravation of vertigo. Our patient had initially refused to have MRI scans taken because brain CT scans were performed 1 month before this incident during evaluation for occipital headache and reportedly showed no abnormalities. Nevertheless, unusual duration of vertigo and known risk factors alarmed the faculty to insist on further evaluation with MRI despite high expenses.

The site of injury in the auditory and vestibular systems causing sudden HL and vertigo can not be confirmed without pathologic examination. However, moderatesevere sensorineural HL with a speech discrimination score of 92% and normal stapedial reflexes suggested a cochlear site of injury in our patient. The auditory brainstem responses to the click stimuli corresponded to hearing level in the 2 to 4 kHz range, and thresholds of the patient were greater than stimulus intensity. Thus, the absence, and not the expected delayed latencies, of responses in the affected ear can be explained. The cochlear origin is supported by the fact that the inner ear lacks collateral blood flow and has a high metabolic rate. 1,8,9 Fluctuation of hearing may be explained by a transient ischemic attack followed by thrombotic occlusion of AICA. Also, unilateral loss of caloric response indicates a labyrinthine origin of vertigo, presumably caused by a circulation compromise. The internal auditory artery supplies the facial nerve as well as the cochleovestibular nerve within the internal auditory canal, and facial paralysis is also commonly described in AICA infarction. However,

unlike the inner ear, with exclusive blood flow from internal auditory artery, the intracanalicular nerves are supplied by collateral circulation from arteries supplying the adjacent dura mater and petrous bone. Depending on such collateral supply, facial nerve integrity may be preserved, as in our patient.

CONCLUSION

In conclusion, sudden HL and vertigo may be the only presenting symptoms of AICA infarction. Older patients with high risk factors of vascular disease should be evaluated carefully for the possibility of AICA infarction, even when no other neurologic signs are evident.

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