LETTER TO THE EDITOR **Open Access**

pISSN 1738-6586 / eISSN 2005-5013 / J Clin Neurol 2017;13(1):107-108 / https://doi.org/10.3988/jcn.2017.13.1.107



Bilateral Sudden Hearing Difficulty Caused by Bilateral Thalamic Infarction

Jun-Hyung Lee Sang-Soon Park Jin-Young Ahn Jae-Hyeok Heo

Department of Neurology, Seoul Medical Center, Seoul, Korea Dear Editor.

Sudden-onset bilateral hearing difficulty has various possible causes, including infectious diseases of the inner ear, ototoxic medications, and Meniere's disease. 1.2 However, there have been only rare reports of vertebrobasilar arterial infarction that extensively invades the brainstem, or bilateral middle cerebral artery infarction that simultaneously invades both auditory cortexes.³⁻⁵ Herein we describe a case of bilateral sudden hearing difficulty due to cerebral infarction of the bilateral medial geniculate bodies.

A 44-year-old male patient was admitted to Seoul Medical Center due to a 17-day history of sudden-onset hearing difficulty. About 1 year previously he had visited another hospital due to acute left-side paresthesia, and was diagnosed with and treated for diabetic neuropathy. A neurological examination revealed normal muscle strength in the bilateral upper and lower extremities, but paresthesia on his left side (both in the limbs and trunk) and hypesthesia on the right side of the face. A brain MRI scan showed a chronic cerebral infarction at the right thalamic-midbrain junction and a subacute cerebral infarction at the left thalamic-midbrain junction (Fig. 1A, B, and C). An otolaryngological examination revealed chronic otitis media without structural abnormalities. His pure-tone audiogram indicated severe sensorineural hearing loss in both ears (Fig. 1D). No response was observed in speech audiometry, and brainstem auditory evoked potentials were normal (Fig. 1D and E). The patient was treated with antiplatelet agents after hospitalization, and was discharged without improvement of his bilateral hearing difficulty. There was no significant improvement in pure-tone or speech audiometry during a 1-year follow-up.

In the auditory tract, sound stimuli reach Heschl's gyrus in the auditory cortex via the cochlear nerve, cochlear nucleus, trapezoid body, superior olivary complex, lateral lemniscus, inferior colliculus, and medial geniculate body. After leaving the cochlear nucleus, most sound stimuli from the dorsal and medial regions reach the inferior colliculus along the lateral lemniscus via the opposite superior olivary complex. However, because another nerve pathway from the ventral auditory striatum and trapezoid bodies passes through the ipsilateral superior olivary complex, hearing difficulties can occur following bilateral lesions in the superior cochlear nucleus of the pons and also following an unilateral lesion in the inferior cochlear nucleus.⁶ Thus, bilateral hearing difficulties may be due to lesions involving a wide range of brainstem sites in auditory pathways both superior and inferior to the cochlear nucleus. There has been one report of bilateral hearing loss as a symptom of unilateral thalamic hemorrhage.⁷ Those authors suggested that the hemorrhage caused deterioration of the synchronous neural responses between bilateral ear stimuli and sound location tuning.^{7,8} However, that reported hearing difficulty was limited to a partial defect in speech discrimination.

The clinical manifestations in our patient suggested that the lesion in the right thalamusmidbrain junction had occurred 1 year previously, with the hearing difficulty not present-

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April 19, 2016 Received June 12, 2016 Revised Accepted June 14, 2016

Correspondence

Iae-Hveok Heo, MD Department of Neurology, Seoul Medical Center, 156 Sinnae-ro, Jungnang-gu, Seoul 02053, Korea

Tel +82-2-2276-8669 Fax +82-2-2276-8539 E-mail drjae93@gmail.com



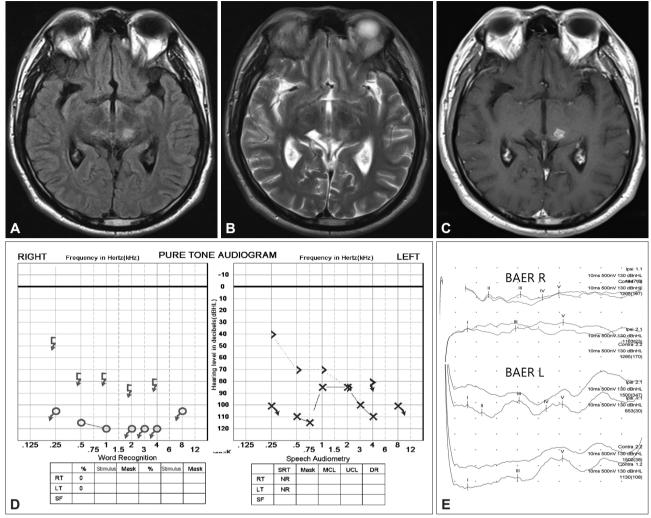


Fig. 1. Axial brain MR images (A, B, and C), pure-tone audiogram (D), and brainstem auditory evoked response (E). A and B: FLAIR and T2-weighted images showing high signal intensities in the bilateral thalamic-midbrain junction. C: T1-weighted image showing an enhanced lesion in the left medial geniculate body, suggestive of a subacute infarction. D: Pure-tone audiometry and speech audiometry showing bilateral sensorineural hearing loss. E: The brainstem auditory evoked potentials were normal. BAER: brainstem auditory evoked response, FLAIR: fluid attenuated inversion recovery.

ing at that time due to the unilateral invasion occurring at the superior cochlear nucleus in the auditory nerve. The bilateral hearing difficulty may have been delayed by 1 year due to a delay in the other side of the auditory nerve pathway being invaded. In other words, the bilateral hearing difficulty experienced by the patient was not caused by a single cerebral infarction, but by recurrent strokes occurring bilaterally at the same sites with a 1-year interval, which resulted in the invasion of the bilateral superior cochlear nuclei in the auditory nerve pathway.

This case study shows that sudden hearing difficulty may occur in rare cases via cerebral infarction at the bilateral medial geniculate bodies.

Conflicts of Interest

The authors have no financial conflicts of interest.

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