

Journal of the Neurological Sciences 222 (2004) 105-107



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Short communication

Sudden deafness with vertigo as a sole manifestation of anterior inferior cerebellar artery infarction

Hyung Lee^{a,b,*}, Byung-Hoon Ahn^c, Robert W. Baloh^{d,e}

^a Department of Neurology, Keimyung University School of Medicine, Daegu, South Korea
^b Brain Research Institute, Keimyung University School of Medicine, Daegu, South Korea
^c Otorhinolaryngology, Keimyung University School of Medicine, Daegu, South Korea
^d Department of Neurology, UCLA School of Medicine, Los Angeles, CA, USA
^e Division of Surgery (Head and Neck), UCLA School of Medicine, Los Angeles, CA, USA

Received 2 March 2004; accepted 5 April 2004 Available online 24 May 2004

Abstract

Sudden deafness without associated neurological symptoms and signs is typically attributed to a viral inflammation of the labyrinth. Although sudden deafness occurs with anterior inferior cerebellar artery (AICA) infarction, the deafness is usually associated with other brainstem or cerebellum signs such as crossed sensory loss, lateral gaze palsy, facial palsy, Horner syndrome or cerebellar dysmetria. An 84-year-old woman suddenly developed right-sided tinnitus, hearing loss, vertigo and vomiting. Audiometry and electronystagmography documented absent auditory and vestibular function on the right side. T2-weighted and diffusion-weighted MRI showed a tiny infarct in the right lateral inferior pontine tegmentum. AICA occlusion can cause sudden deafness and vertigo without brainstem or cerebellar signs. © 2004 Elsevier B.V. All rights reserved.

Keywords: Sudden deafness; Anterior inferior cerebellar infarction

1. Introduction

Sudden deafness is defined as more than 20 dB sensorineural hearing loss occurring over minutes to hours [1]. About 50% of patients will have associated systemic viral infection near the time of the onset of the hearing loss [2]. Anterior inferior cerebellar artery (AICA) infarction can cause sudden deafness because the internal auditory artery, ordinarily a branch of the AICA, supplies the peripheral auditory system including the inner ear [3,4]. Recently, we reported that sudden deafness was an important sign for the diagnosis of AICA infarction [5]. Characteristically, sudden deafness due to AICA infarction is associated with multiple brainstem or cerebellar symptoms and signs [4–6].

E-mail address: hlee@dsmc.or.kr (H. Lee).

2. Report of a case

An 84-year-old woman with longstanding hypertension developed right-sided tinnitus, hearing loss and vertigo upon awakening in the morning. When she was first examined 2 days later, she was unsteady and tilting to the right when walking. She had spontaneous, left beating nystagmus with horizontal and torsional components, in primary position and on gaze to the right or left (followed Alexander's law). She did not have dysarthria, facial or limb sensory loss, facial or limb weakness, opthalmoparesis, diplopia or Horner syndrome. There was no dysmetria on finger-to-nose and heel-to-shin testing. The Weber test was lateralized to the left. Tympanic membranes were normal.

There was no prior history of temporal bone fracture, meningitis, autoimmune disease or exposure to ototoxic drugs. Pure tone audiogram showed a moderate sensorineural hearing loss of 55 dB with 80% speech discrimination on the right side (Fig. 1). Stapedial reflex thresholds were normal at multiple frequencies on both sides. Brain-

^{*} Corresponding author. Department of Neurology, Keimyung University School of Medicine, 194 Dongsan dong, Daegu 700-712, South Korea. Tel.: +82-53-250-7835; fax: +82-53-250-7840.

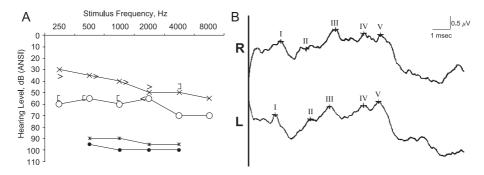


Fig. 1. Audiologic findings. (A) Pure tone air conduction audiogram showed a moderate hearing loss on the right side. Hearing levels in decibels (dB) (American National Standards Institute, 1989) are plotted against stimulus frequency on a logarithmic scale. O: air conduction on right side, ×: air conduction on left side, <: bone conduction on right side, >: bone conduction on left side, [: bone conduction (masked) on right side,]: bone conduction (masked) on left side. Stapedial reflexes (•: right, *: left) were recorded at normal levels bilaterally. (B) Brainstem auditory evoked potentials showed normal wave latencies bilaterally.

stem auditory evoked potentials (BAEP) were normal. Electronystagmography showed no response to caloric stimulation of the right ear. A brain MRI showed a tiny infarct in the tegmentum of the right lateral inferior pons (Fig. 2). A magnetic resonance angiogram (MRA) showed no abnormalities. A transesophageal echocardiography with a bubble study and electrocardiography revealed no abnormalities. The patient was treated with antiplatelet medication and the vertigo disappeared over a week. A follow-up audiogram showed persistent hearing loss of 65 dB on the right side.

3. Comment

The lateral inferior pontine tegmentum (LIPT) is typically supplied by a recurrent penetrating branch of AICA [4]. Both LIPT and the middle cerebellar peduncle are usually affected in AICA infarction, but isolated LIPT territory infarction does occur. However, in recent study of 36 patients with infarcts of the pons, no patients with

isolated LIPT infarct were described [7]. We know of three prior studies of isolated LIPT infarction due to AICA occlusion [8-10]. In 1989, Fisher [8] described a patient with a lacunar infarct of the tegmentum of the lower lateral pons with multiple AICA signs including dysarthria, staggering gait, nystagmus, facial weakness, limb dysmetria and extremity sensory loss. Interestingly, deafness and vertigo were not described. In 1998, Roquer et al. [9] reported 15 patients with AICA infarction diagnosed by brain MRI. Two patients with isolated LIPT infarct presented with vertigo, hypoacusis, ataxia and peripheral facial palsy. In 1999, Verghese and Morocz [10] described a patient with a small LIPT infarct on brain MRI who presented with vertigo and deafness without associated neurological symptoms. However, they did not perform standardized auditory and vestibular testing to localize the site of lesion.

Without pathological confirmation, it is difficult to know precisely the site of lesion producing hearing loss in our patient. However, in a patient with this degree of hearing loss, normal speech discrimination, normal stape-

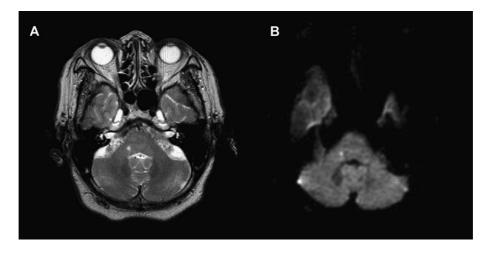


Fig. 2. Brain MRI 2 days after onset of symptoms. (A) T2-weighted and (B) diffusion-weighted axial images showed a tiny infarct in the right lateral tegmentum of the lower pons.

dial reflexes and normal BAEP responses indicate a cochlear site of injury [11,12]. Furthermore, a cochlear site for the hearing loss is consistent with its high metabolic energy requirement and lack of collateral circulation [3-5]. By contrast, the retrocochlear acoustic nerve has abundant collateral blood supply arising from the lateral medullary artery, arteries supplying adjacent dura matter and petrous bone, and the inferior lateral pontine artery [13,14]. The vertigo and unilateral canal paresis to caloric stimulation might be explained by involvement of the intrapontine vestibular nerve (i.e., vestibular fascicle) since the patient did have a small infarct on brain MRI in that region [15]. However, a labyrinthine site for the vestibular symptoms and signs seems more likely considering the cochlear damage. Presumably, the mechanism for infarction was small vessel arteriosclerosis since there was no obvious source of emboli and the large vessels were normal on MRA.

In conclusion, AICA infarction can present as sudden deafness and vertigo. Clinicians should be aware of the possibility of AICA infarction particularly in older patients with sudden deafness and vascular risk factors, even when classic brainstem or cerebellar signs are absent.

Acknowledgements

This study was supported by grants of the Oriental Medicine R&D Project (03-PJ9-PG6-SO02-0001), Ministry of health and Welfare, Republic of Korea.

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