Sudden Deafness and Anterior Inferior Cerebellar Artery Infarction

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Background and Purpose—Acute ischemic stroke in the distribution of the anterior inferior cerebellar artery (AICA) is known to be associated with vertigo, nystagmus, facial weakness, and gait ataxia. Few reports have carefully examined the deafness associated with the AICA infarction. Furthermore, previous neurological reports have not emphasized the inner ear as a localization of sudden deafness. The aim of this study was to investigate the incidence of deafness associated with the AICA infarction and the sites predominantly involved in deafness.

Methods—Over 2 years, we prospectively identified 12 consecutive patients with unilateral AICA infarction diagnosed by brain MRI. Pure-tone audiogram, speech discrimination testing, stapedial reflex testing, and auditory brainstem response were performed to localize the site of lesion in the auditory pathways. Electronystagmography was also performed to evaluate the function of the vestibular system.

Results—The most common affected site on brain MRI was the middle cerebellar peduncle (n=11). Four patients had vertigo and/or acute auditory symptoms such as hearing loss or tinnitus as an isolated manifestation from 1 day to 2 months before infarction. Audiological testings confirmed sensorineural hearing loss in 11 patients (92%), predominantly cochlear in 6 patients, retrocochlear in 1 patient, and combined on the affected side cochlear and retrocochlear in 4 patients. Electronystagmography demonstrated no response to caloric stimulation in 10 patients (83%).

Conclusions—In our series, sudden deafness was an important sign for the diagnosis of AICA infarction. Audiological examinations suggest that sudden deafness in AICA infarction is usually due to dysfunction of the cochlea resulting from ischemia to the inner ear. (*Stroke*. 2002;33:2807-2812.)

Key Words: deafness ■ ear ■ infarction, cerebral

Acute ischemic stroke in the distribution of the anterior inferior cerebellar artery (AICA) is known to be associated with vertigo, hearing loss, facial weakness, ataxia, nystagmus, and hypalgesia. In 1943, Adams[1] was the first to completely describe the syndrome associated with the AICA occlusion. In his patient, neurotologic symptoms such as vertigo, tinnitus, and bilateral hearing loss were early initial symptoms. Subsequent reports on AICA infarction have focused primarily on the brainstem and cerebellar findings without the associated neurotologic findings.[1–8] Until now, few studies have carefully investigated the nature of hearing loss that occurs with AICA infarction.[9–11]

Recently, we described a patient who had a bilateral sudden deafness as a prodrome of AICA infarction.[11] To the best of our knowledge, no consecutive clinical series has focused on hearing symptoms associated with the AICA infarction. The purpose of the present study was to characterize the nature of the deafness by applying detailed neurotologic investigation of consecutive cases to define the site of lesion associated with sudden deafness and AICA infarction.

Patients and Methods

Over 2 years, we prospectively identified 12 consecutive patients with unilateral AICA infarction diagnosed by brain MRI. In every patient, diagnostic tests were performed to determine risk factors of stroke. We determined the vascular territory using the anatomic diagrams of Amarenco and Hauw[3] (involving at least 1 of the following anatomical structures: middle cerebellar peduncle, lateral inferior pontine area, or anterior cerebellar hemisphere). The MRI studies were performed during the acute period after the onset of symptoms (1 to 4 days; mean interval, 2.8 days). For MRI with a 1.5-Tesla MR scanner (Vision, Siemens Medical), scanning was done in the axial, sagittal, and coronal planes with 6-mm-thick sections. T1-weighted images were obtained with a short spin-echo pulse sequence with a repetition time of 600 ms and an echo time of 14 ms. T2-weighted images were obtained with a long spin-echo sequence with a repetition time of 4200 ms and an echo time of 90 ms. Single-shot, spin-echo-planar imaging isotropic diffusion-weighted images were produced with *b* values of 0 and 1000 s/mm².

TABLE 1. **Baseline Clinical Profiles of 12 Patients With AICA Infarction**

Patient	Sex	Age, y	Vascular Risk Factors	Prodromata
1	M	28	Aortic valve replacement	None
2	F	62	Hypertension	Episodic unilateral tinnitus and hearing loss (10 d), isolated vertigo* (1 d)
3	F	58	Atrial fibrillation, mitral stenosis	None
4	F	71	Hypertension	Isolated vertigo* (2 mo), unilateral tinnitus and vertigo (1 d)
5	M	67	Diabetes mellitus	Bilateral hearing loss and unilateral tinnitus (10 d)
6	M	58	None	None
7	M	64	Patent foremen ovale†	None
8	M	46	Smoking, obesity	None
9	F	68	Hypertension	None
10	F	60	Hypertension, diabetes mellitus	Isolated vertigo* (3 d)
11	F	62	Hypertension	None
12	F	83	Hypertension	None

*Without tinnitus, hearing loss, or focal neurological symptoms.

†Association of right-to-left shunting at rest by contrast transesophageal echocardiogram.

MR angiogram (MRA) was performed at acute stages in all patients. The average age of the patients was 59.7 years (range, 46 to 79 years).

Each patient completed a standard dizziness questionnaire and underwent a neurotologic evaluation, including a thorough history and neurotologic examination performed by an experienced neuro-tologist. Pure-tone audiogram (PTA) was performed using air-and bone-conducted signals in an acoustic booth. The pure-tone average was obtained by averaging hearing thresholds at 500, 1000, and 2000 Hz. A pure-tone average >25 dB was regarded as indicative of hearing loss. Mild, moderate, severe, and profound hearing loss was defined as 26 to 40, 41 to 70, 71 to 90, and >90 dB, respectively. We performed additional tests to discriminate between neural and cochlear causes of hearing loss. Speech discrimination testing was done with AB Wordlists according to the Korean Hearing Services standard protocol. The stapedial reflex thresholds of each ear at frequencies of 500, 1000, 2000, and 4000 Hz were measured. Measurements of stapedial reflex were performed with a GSI 33 Middle Ear Analyzer, which allows determination of stapedial reflex thresholds up to the 110-dB hearing level. The activating stimuli were presented in 5-dB steps ipsilateral to the examined ear. Reflex threshold was defined as the lowest activator level that resulted in an observable meter deflection.

Auditory brainstem response (ABR) was performed on all patients (Medelec ER94a) with previously described techniques.[12] Rarefaction click stimuli were used. For ABR, the auditory stimulation was a click of 0.1-ms-long, 90-dB sound that was presented monaurally at a rate of 10 Hz, and broadband masking (40 dB less intense than the clicks) was presented to the contralateral ear. Each averaged response represented a composite of 2048 stimulus presentations. The reference group consisted of 30 healthy age-matched volunteers who had not been receiving drugs on a regular basis that might directly influence the ABR characteristics. PTA confirmed normal hearing in all members of the reference group. ABR was considered abnormal when it was outside the average values ±2 SD for absolute latencies and interpeak latencies.

We defined hearing loss of cochlear origin as follows[13–17]: (1) speech recognition scores corresponded well with the increasing hearing thresholds on PTA; (2) despite the hearing loss on PTA, the ABR showed no abnormalities or delay in absolute latencies of all waves, but interpeak latencies of wave I-III-V were within normal limits; and (3) stapedial reflex testing showed normal reflex thresh-olds. Vestibular function tests were performed by a computer-based electronystagmography (ENG) system (Nicolet ENG system) and included examination of oculomotor movements (saccade, smooth pursuit, and optokinetic nystagmus), spontaneous and gaze-evoked nystagmus, and caloric responses. All neurotologic evaluations were performed during the acute period.

Results

Clinical Features

Seven patients were female; 5 patients were male. Clinical data at the time of admission are summarized in Tables 1 and 2. All patients were alert and orientated on admission.

TABLE 2. **Classic features of AICA Infarction* and Findings in 12 Patients**

Signs and Symptoms	Structures Possibly Involved	Patient											
		1	2	3	4	5	6	7	8	9	10	11	12
Vertigo, nystagmus	Labyrinth, vestibular nerve, vestibular nuclei, flocculus	+	+	+	+	+	+	+	+	+	+	+	+
Tinnitus, hearing loss	Cochlea, auditory nerve, cochlear nuclei	+	+	+	+	+	+	+	+	+	+	−	+
Gait and limb ataxia	MCP, anterior inferior cerebellum	+	+	+	+	+	+	+	+	+	+	+	+
Facial hemianesthesia	Spinal trigeminal tract, nucleus	+	+	−	−	+	−	−	+	+	+	−	−
Facial paralysis	Facial nerve fascicle	+	−	−	−	+	−	−	−	+	−	−	−
Crossed sensory signs	Spinothalamic tract	−	−	−	−	+	−	−	−	+	−	−	−
Horner's syndrome	Sympathetic fibers	−	−	−	−	+	−	−	−	+	−	−	−

MCP indicates middle cerebellar peduncle.

*Based on Adam's[1] clinicopathological study of AICA infarction.

TABLE 3.   Audiologic Data in 12 Patients With AICA Infarction

Patient	Hearing Loss (PTA)	ABR	Stapedial Reflex Threshold	SD Scores, %	Site of Lesion
1	Severe	Absent	Absent	NR	Unknown
2	Mild	Absent	Absent	30	Retrocochlear
3	Moderate	Normal	Normal	87	Cochlear
4	Severe	Absent	Absent	15	Unknown
5	Moderate	Normal	Normal	100	Cochlear
6	Moderate	Normal	Normal	87	Cochlear
7	Moderate	Delayed*	Normal	90	Cochlear
8	Profound	Absent	Absent	NR	Unknown
9	Profound	Absent	Absent	NR	Unknown
10	Moderate	Normal	Normal	85	Cochlear
11	None	Normal	Normal	100	None
12	Mild	Normal	Normal	92	Cochlear

SD indicates speech discrimination; NR, no response.

*Delay in absolute latencies of all waves but normal interpeak latencies of wave I-III-V.

Vertigo was the initial symptom in all patients. All had accompanying nausea and/or vomiting. On neurologic examinations, all patients had a spontaneous nystagmus (horizontal-rotatory) beating toward the healthy side (ie, directed away from the side of infarction). The rotatory component was clockwise in patients with left-beating nystagmus and counterclockwise in patients with right-beating nystagmus. Lateropulsion was a common and striking subjective complaint. Only 2 patients (patients 5 and 9) had a complete AICA infarction with the classic clinical features first described by Adams.[1] Six patients had facial hypalgesia, and 3 had a peripheral seventh-nerve palsy. Crossed sensory loss and Horner syndrome were found in only 2 patients. Four patients (25%) had vertigo and/or acute auditory symptoms as an isolated manifestation from 1 day to 2 months before infarction. The isolated vertigo preceding infarction was identical in quality to the vertigo experienced at the time of infarction, and the tinnitus preceding infarction was identical to the tinnitus experienced at the time of infarction. Functional outcome was good in all patients. All were ambulatory at the time of discharge, although there were mild residual deficits such as unsteadiness, tingling sensation in the extremities, or facial palsy

MRI and MRA Findings

The middle cerebellar peduncle was affected in 11 patients, anterior inferior cerebellum in 8 patients, and lateral inferior pons in 6 patients. Complete AICA infarction involving the middle cerebellar peduncle, lateral pons, and anterior inferior cerebellum was found in only 4 patients. Eight patients had AICA territory infarction only. Patients with AICA plus infarction (n=4) had either caudal cerebellar infarction in the distribution of the medial branch of posterior inferior cerebellar artery (n=3) or ventral pontine infarction (n=1). MRA showed stenosis of the lower and/or middle basilar artery close to the origin of the AICA in 5 patients and severe dolichoectasia of the basilar artery in 2. MRA was normal in

those 5 patients. Unfortunately, MRA cannot adequately visualize smaller vessels such as AICA and its branches.

Cochleovestibular Findings

PTA detected unilateral (n=10) or bilateral (n=1) sensorineural hearing loss during the acute period after symptom onset (Table 3). Only 1 patient (patient 11) did not complain of decreased hearing and had normal hearing thresholds on PTA. Ten of the 11 patients with hearing loss were aware of decreased hearing during an attack of vertigo. One patient (patient 2) did not notice decreased hearing during the attack of severe vertigo and vomiting, but PTA showed a mild hearing loss on the affected side. None of the patients had a history of hearing loss or otologic disease. Hearing impairment was moderate in 5 patients, severe to profound in 4, and mild in 2. From the findings of the PTA, speech discrimination testing, stapedial reflex testing, and ABR, hearing loss was localized to the cochlea in 6 patients (patients 3, 5 through 7, 10, and 12). ABR showed normal waveform responses in 4 patients (patients 3, 5, 6, and 10) with a moderate hearing loss on PTA and 1 patient (patient 12) with a mild hearing loss. One patient (patient 7) showed a slight delay in absolute latencies of all waves, but interpeak latencies of wave I-III-V were normal. In contrast, 1 patient (patient 2) had a mild hearing loss on PTA and no wave responses on ABR testing, which suggested a hearing loss of the retrocochlear origin.[14,17] The other 4 patients (patients 1, 4, 8, and 9) had a severe to profound hearing loss on PTA and no wave responses on ABR testing. The hearing loss improved in 7 patients, although the extent of recovery varied. The patients themselves recognized a gradual improvement in hearing difficulty within 1 week to several months, and PTA showed improvement in hearing thresholds. ENG demonstrated no response to caloric stimulation of the affected ear in 10 patients. The caloric responses were decreased on the affected side in the other 2. In 11 patients, oculomotor testing showed asymmetric abnormalities of both smooth pursuit and optokinetic nystagmus.
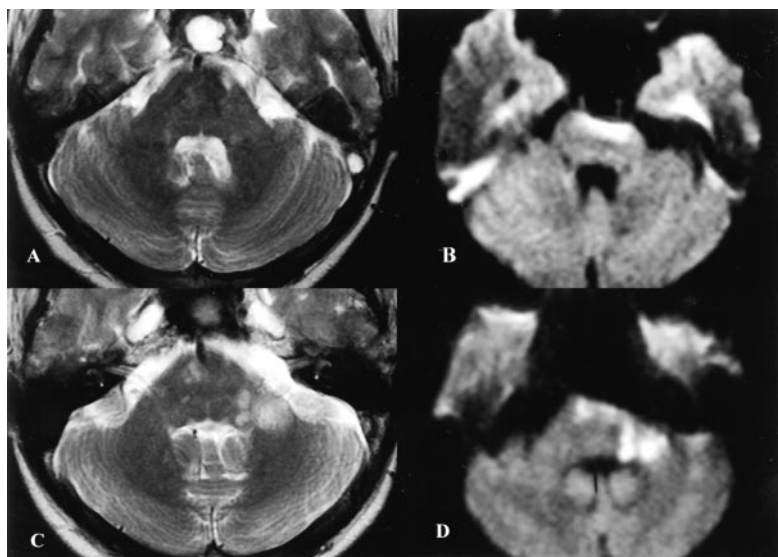
Figure 1. MRI findings in case 1 with cochlear-type hearing loss. On admission, axial T2-weighted brain MRI (A) showed a hyperintense spot in the left ventrolateral pons. Diffusion-weighted images (B) showed a possible hyperintense focus in the same area. Diffusion images also showed that the middle cerebellar peduncle and dorsolateral pons were spared. Six days later, axial T2-weighted (C) and diffusion-weighted (D) MRI of the brain demonstrated hyperintense foci in the left middle cerebellar peduncle, left dorsolateral pons, and ventral pons.

Illustrative Cases

Case 1: Patient 10 With Cochlear-Type Unilateral Hearing Loss

A 60-year-old woman with type 2 diabetes mellitus and hypertension developed the sudden onset of vertigo, nausea, vomiting, and hearing loss in the left side. On examination, she had a spontaneous right-beating horizontal nystagmus with a counterclockwise torsional component in primary position and with gaze to the right or left. PTA showed a moderate sensorineural hearing loss of 50 dB on the left side. Stapedial reflexes were recorded at normal levels from both sides. Speech discrimination scores were 85% on the left side. Normal waveform responses were evoked bilaterally on ABR testing. ENG showed no response to caloric stimulation of the left side. Axial T2- and diffusion-weighted MRI of the brain showed a small infarct in the left ventrolateral pons, but the middle cerebellar peduncle and dorsolateral pons were apparently spared. Brain MRA showed moderate stenosis of the middle third of the basilar artery. Low-dose aspirin (100 mg) therapy was started. The hearing loss persisted, but the vertigo improved steadily over a few days. Six days after the initial onset of hearing loss in the left side and vertigo, the patient complained of an exacerbation of the vertigo, combined with nausea, vomiting, and imbalance. On neurologic examination, she had a spontaneous right-beating horizontal nystagmus with a counterclockwise torsional component in primary position and rightward gaze, which changed to a left-beating horizontal nystagmus with a clockwise torsional component on leftward gaze. There was diminished left-sided facial sensation and dysmetria of the left limbs. A follow-up MRI showed new infarcts in the left middle cerebellar peduncle and left dorsolateral pons (Figure 1). Intravenous heparinization was followed by 6 months of oral anticoagulation with warfarin. Over several days of hospitalization, the vertigo, nausea, and vomiting subsided. A follow-up PTA showed that the hearing loss in the left side had improved to 35 dB.

Case 2: Patient 2 With Retrocochlear-Type Hearing Loss

A 62-year-old woman with hypertension developed a sudden onset of continuous vertigo, nausea, vomiting, and unsteadi-ness. Ten days before, she had 3 episodes of transient left-sided tinnitus and hearing loss lasting a few minutes. One day before, she had 2 episodes of transient isolated vertigo that lasted no more than several minutes. On neurologic examination, she had a spontaneous right-beating horizontal nystagmus with a counterclockwise torsional component in primary and rightward gaze, which changed to a left-beating horizontal nystagmus with a clockwise torsional component on leftward gaze. There were diminished left facial sensation, left limb dysmetria, and gait ataxia. MRI of the brain demonstrated hyperintense lesions on axial T2-weighted images situated in the left middle cerebellar peduncle and left lateral pons (Figure 2). PTA showed a mild (40 dB) sensorineural hearing loss on the left side even though she did not complain of decreased hearing in the left side during the attack of vertigo. Speech discrimination scores were 30% on the left side and 90% on the right side. No stapedial reflexes were elicited from the left side. On ABR testing, no responses were evoked by stimulation on the left side, whereas there were normal waveforms on the right side (Figure 3). ENG showed no response to caloric stimulation of the left side.
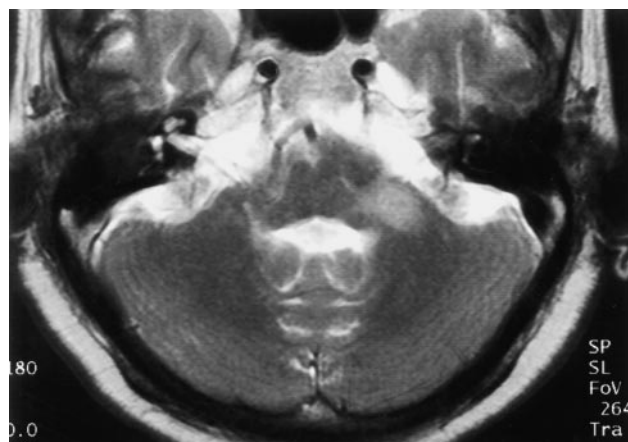

Figure 2. MRI finding in case 2 with retrocochlear-type hearing loss. Axial T2-weighted MRI showed hyperintensities involving the left middle cerebellar peduncle and left lateral pons.
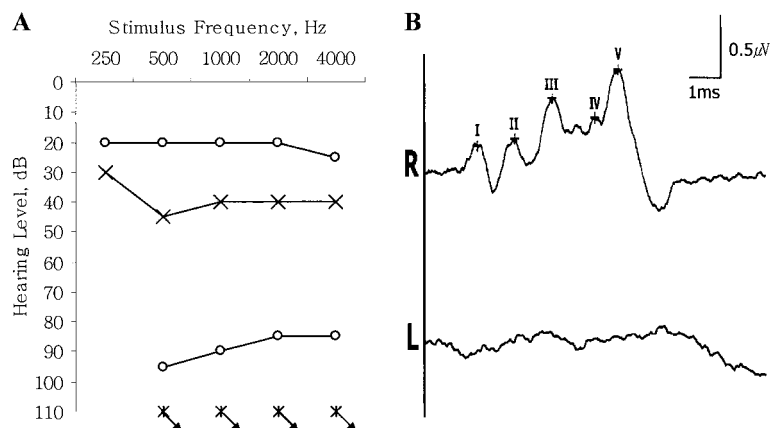
Figure 3. Audiologic findings in case 2 with retrocochlear-type hearing loss. A, PTA showed mild hearing loss on the left side. Stapedial reflexes (o, right; x, left) could be elicited from the right ear but not from the left. B, ABR recording on the left side showed an absence of all wave responses.

During several days of anticoagulation in the hospital, the patient's vertigo and nausea improved.

Discussion

Most previous reports of AICA infarction have focused on the brainstem and cerebellar findings without detailing the associated neurotologic findings.[1–8] Several prior studies[9–11,18–21] of AICA infarction have described acute hearing symptoms. However, most were case reports,[9,11,18,19,21] and there has been no systematic study of a consecutive series of patients. In 1990, Hinojosa and Kohut[9] reported a clinicopathological study in a patient with complete AICA infarction. The temporal bone histopathology showed a loss of sensory epithelium of the cochlea and vestibular labyrinth consistent with inner ear infarction. In 1993, Matsushita et al[10] reported on 5 patients with AICA infarction, all with abrupt onset of sensorineural hearing loss. These authors suggested that the sudden deafness resulted from ischemia to the inner ear. However, the report did not include audiometric data such as the PTA, speech discrimination testing, and ABR, which are essential for localizing the sites of the sudden deafness. In 2001, we reported on a patient who had bilateral hearing loss as a prodrome of AICA territory infarction.[11] No large consecutive clinical series has focused on hearing symptoms associated with AICA infarction.

Hearing loss is a common finding in patients with AICA infarction. However, the incidence differs in the few series reported in the literature, from a low of 30%[3] to a high of 100%.[10] In our series, the sensorineural hearing loss was found in 11 of 12 patients (92%) with AICA infarction. In general, neurologists have not included the audiogram as a routine diagnostic tool for the evaluation of AICA infarction. Furthermore, patients may not be aware of unilateral hearing loss during an attack of vertigo and vomiting.[22,23] In our study, 1 patient (patient 2) had documented unilateral hearing loss on PTA, but she did not notice decreased hearing during the attack of severe vertigo. All of these factors can explain the variable incidence of sudden deafness in previous reports of AICA stroke.

In 4 patients, vertigo and/or acute auditory symptoms occurred as an isolated manifestation from 1 day to 2 months before the sudden onset of AICA stroke. These transient symptoms lasted minutes, the typical duration of transient ischemia within anterior or posterior circulation. Others have also emphasized that brief episodes of audiovestibular symptoms (minutes) can be the warning of an impending brainstem stroke.[24] The inner ear may be particularly sensitive to transient ischemia because of its high energy requirements and lack of adequate collateral blood supply.[23,24]

Without pathological confirmation, it is difficult to precisely determine the locus of injury responsible for hearing loss in our patients. However, in 5 patients with moderate hearing loss and 1 patient with mild hearing loss, speech discrimination and stapedial reflex testing were normal, and ABR had either normal wave responses (n=5) or delay in absolute latencies of all waves with no abnormalities in interpeak latencies (n=1), indicating a cochlear site of injury. Only 1 patient (patient 2) had test results indicating a retrocochlear site of lesion. The finding of absent ABR in the patients with severe to profound hearing loss is not helpful because absent ABR would be expected even with a severe to profound cochlear hearing loss. A cochlear site for hearing loss with AICA infarction is not surprising because the labyrinthine artery is an end artery without collateral circulation, whereas the retrocochlear acoustic nerve has abundant collateral blood supply.[24–26] Partial recovery of hearing was found in 2 patients (patients 1 and 4) with severe hearing loss on PTA and no wave responses on ABR. Follow-up ABR showed a delay in absolute latencies of all waves, but interpeak latencies were normal, again consistent with a cochlear site of injury. In future studies, measurement of otoacoustic emissions might help to better localize the site of hearing loss.

Because the clinical presentation of AICA infarction may mimic more common vestibular disorders, including vestibular neuritis or Ménière's disease, a detailed neurologic examination focused on additional brainstem signs such as crossed sensory loss, lateral gaze palsy, facial palsy, or Horner syndrome should be performed in all patients presenting with acute vertigo.

AICA infarction typically involves the middle cerebellar peduncle.[3,8,10] In 11 of 12 patients in our study, MRI identified a localized area of infarction in the middle cerebellar peduncle. The 1 patient with no lesion in the middle cerebellar peduncle had an infarct in the anterior inferior cerebellum. The most common mechanism of AICA infarc-

tion is formation of atheroma or thrombus in the parent basilar artery blocking the orifice of the AICA.[27,28] This was the likely mechanism in the 5 patients who had stenosis of the lower and/or mid basilar artery. Two patients had a dolicho-ectatic basilar artery, which has previously been associated with AICA stroke.[27] Of the 5 patients with normal MRA, 2 had cardiac disease. One had a likely source of cardiac emboli with mitral stenosis and atrial fibrillation, and the other had a patent foramen ovale[29] with right-to-left shunt as a possible source of cardiac embolism.

In conclusion, sudden deafness is a common sign of AICA infarction. In most cases, the hearing loss and tinnitus result from cochlear injury.

Acknowledgment

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