

The Association of Anterior Inferior Cerebellar Artery in Internal Auditory Canal with Tinnitus and Hearing Loss

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Background and Objectives: Tinnitus is a common disorder, but the etiology of this disorder remains unknown. The objective of this study was to assess the correlation between anatomical type and the thickness of the anterior inferior cerebellar artery (AICA) loop with tinnitus, using 3D-fast imaging employing steady state acquisition magnetic resonance image (MRI). **Materials and Methods:** 74 patients with tinnitus and 82 asymptomatic controls were included in this study. Otologic symptoms, which was measured based on the results of a pure tone audiometry, were reviewed. We evaluated the position and thickness of the AICA vascular loop in 3D-FIESTA MRI using two scoring systems. The first system was Chavda classification based on the anatomical location of the AICA loop. The second scoring system was used to measure the thickness of the AICA loop. The AICA loops were classified into two groups based on thickness, thinner than adjacent facial nerve and thicker than the facial nerve. **Results:** Ears with type I, II AICA loops showed significantly higher rates of tinnitus than those with type III. There was no association between the type of AICA loop and subtype of tinnitus (pulsatile, nonpulsatile). There was no association between the type of tinnitus and hearing loss. Ears with thinner AICA loop had a higher rate of tinnitus than those with thicker AICA loop. **Conclusions:** The type I, II and thinner AICA loop was significantly correlated with tinnitus. Compression of VIIIth cranial nerve by AICA loops at a cerebellopontine angle and impaired blood flow through the vessel may be the pathophysiology of tinnitus. **Korean J Audiol 2011;15:67-71**

KEY WORDS: Tinnitus · Vestibulocochlear nerve · Hearing loss · Magnetic resonance imaging.

Introduction

Tinnitus is a sound in the ear that occurs without an external stimulus. The lifetime incidence of tinnitus is about 7–12% in the general population,¹⁾ but the etiology of this disorder is still unknown. In 1975, Janetta suggested that anterior inferior cerebellar arterial loops around the internal acoustic canal (IAC) and cerebellopontine angle (CPA) may compress the vestibulocochlear nerve (CN VIII) and cause tinnitus, vertigo and hearing loss.²⁾ Since this initial hypothesis, several studies have evaluated the relationship between the vascular loops and otologic symptoms.^{3,4)} However, in people who do not have tinnitus, the prevalence of anterior inferior cerebellar artery (AICA) loops around the IAC and CPA have been reported to be 12% (by post-mortem dissection),⁵⁾ 7% (by computed tomography cisternography),⁶⁾ 14–34% (by magnetic resonance imaging).⁷⁾ Therefore, based on the frequency of AICA

loops in contact with CN VIII have lead researches to question the hypothesis that the AICA loop causes tinnitus.

The purpose of this study was to correlate inner ear symptoms like tinnitus and hearing loss to the type and thickness of AICA loop in CPA and IAC, using 3D-fast imaging employing steady state acquisition (FIESTA) magnetic resonance image (MRI).

Materials and Methods

We carried out a retrospective study from January, 2008 to March, 2009. During a 1-year period, 74 patients with tinnitus were enrolled in the study. All 74 patients had unexplained tinnitus (30 male, 44 female: mean age, 47.3 ± 12.7 years). 29 of 74 patients had bilateral tinnitus, and the remaining 45 patients had unilateral tinnitus. 82 patients who had undergone a MR imaging for headache, hearing loss and who were reported not to have tinnitus were included in the

control group (26 male, 56 female: mean age, 49.9 ± 17.6 years). We categorized the patients into 3 groups, tinnitus ear of patient, non-tinnitus ear of patient with unilateral tinnitus, and control ear (Table 1).

All MR imaging examinations were performed using a 3D-FIESTA. For evaluation, the anterior inferior cerebellar arteries were classified according to their location using Chavda classification⁸⁾: AICA loops lying within the CPA but not entering the IAC were classified type I. AICA loops entering the IAC but not extending more than 50% into the IAC were classified type II. AICA loops extending more than 50% into the IAC were classified type III (Fig. 1). The thickness of the AICA loop was classified into two groups: AICA loops with a diameter greater than the facial nerve were 'large' loops and loops with a diameter less than the facial nerve were defined as 'small' loops (Fig. 2).⁸⁾

The results of pure tone audiometry were analyzed [0.5 kHz (a), 1 kHz (b), 2 kHz (c)]. Sensorineural hearing loss was defined in all patients as an average pure tone audiometric threshold greater than 30 dB ($a+2b+c/4$).

The MR images obtained for the 3 groups were analyzed to

Table 1. Summary of the study population

Characteristics	Patients (n=74)	Controls (n=82)
Bilateral tinnitus	29	
Unilateral tinnitus	45	
Symptomatic sides	103	0
Asymptomatic sides	45	164

evaluate the correlation of tinnitus, hearing loss with type of AICA loop, thickness of AICA loop. χ^2 tests were used to analyze the relationship between otologic symptoms and type and thickness of AICA. ANOVA tests were used to analyze the relationship between pure tone audiometric threshold and type and thickness of AICA (SPSS, Statistical Package for the Social Sciences, Version 18.0). A p value < 0.05 was considered to be a statically significant difference.

Results

Type of AICA loops

Type I AICA loops were found to be present in 68.0% (70/103) of tinnitus ears, 57.8% (26/45) of non-tinnitus ears, 57.3% (94/164) of control ears. Type II AICA loops were found to be present in 26.2% (27/103) of tinnitus ears, 28.9% (13/45) of non-tinnitus ears, and 23.8% (39/164) of control ears. Type III AICA loops were found to be present in 5.8% (6/103) of tinnitus ears, 13.3% (6/45) of non-tinnitus ears, and 18.9% (31/164) of control ears (Table 2).

The association between tinnitus and type of AICA loops

The prevalence of tinnitus according to type of AICA loops was 36.8% (70/190) for type I, 34.2% (27/79) for type II, 13.6% (6/43) for type III. There were no statistically significant differences in the prevalence of tinnitus between type I and type II AICA loops. However, the presence of type I and type II AICA

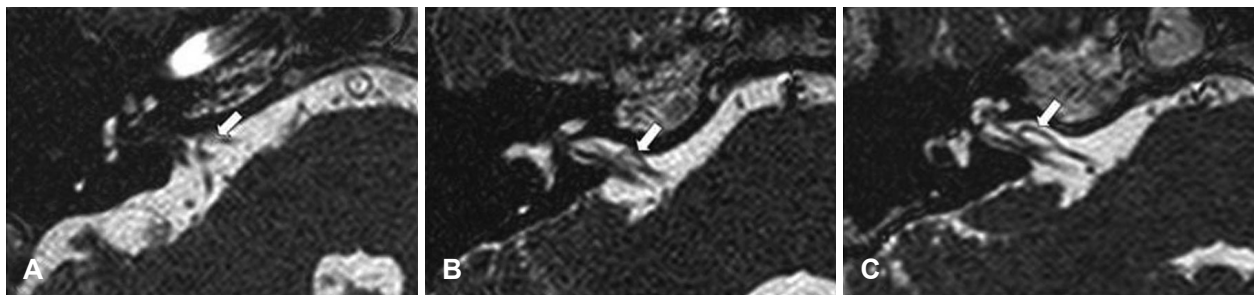


Fig. 1. Chavda classification. White arrow points to a AICA loop. A: Type I: loops in cerebellopontine angle but did not enter the internal auditory canal (IAC). B: Type II: loops entered the IAC and extended less than 50% into the IAC. C: Type III: loops extended greater than 50% into the IAC. AICA: anterior inferior cerebellar artery.

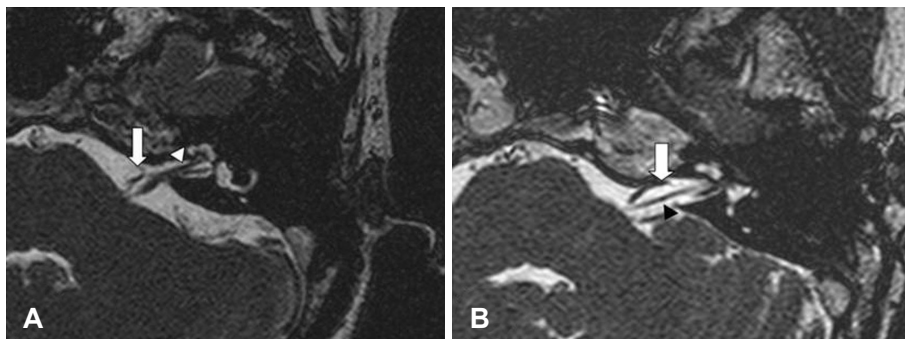


Fig. 2. Thickness of AICA loops. A: The AICA loop (white arrow) is thinner or similar than the facial nerve (white arrow head). B: The AICA loop (white arrow) is thicker than the facial nerve (arrow head). AICA: anterior inferior cerebellar artery.

Table 2. Types of AICA loops in the patient and control groups

Type of vascular loop	Patients group		Control group	Total (n=312)(%)
	Symptomatic sides of patients (n=103)	Asymptomatic sides of patients (n=45)(%)	Control (n=164)(%)	
Type I	70 (68.0)	26 (57.8)	94 (57.3)	190 (60.9)
Type II	27 (26.2)	13 (28.9)	39 (23.8)	79 (25.3)
Type III	6 (5.8)	6 (13.3)	31 (18.9)	43 (13.8)

Statistically significant difference between the groups for the presence of all types of vascular loops ($p=0.015$). AICA: anterior inferior cerebellar artery

Table 3. Rate of hearing loss according to the type of vascular loops

Type of vascular loop		Type of vascular loops			Total (n=312)(%)
		Type I (n=69)(%)	Type II (n=27)(%)	Type III (n=7)(%)	
Ear with tinnitus	Hearing loss (+)	12 (17.4)	6 (22.2)	0 (0)	18 (17.5)
	Hearing loss (-)	57 (82.6)	21 (77.8)	7 (100)	85 (82.5)
Ear with non-tinnitus	Hearing loss (+)	1 (3.8)	0 (0)	1 (16.7)	2 (4.4)
	Hearing loss (-)	25 (96.2)	13 (100)	5 (83.3)	43 (95.6)
Control	Hearing loss (+)	18 (19.1)	9 (23.1)	7 (22.6)	34 (20.7)
	Hearing loss (-)	76 (80.9)	30 (76.9)	24 (77.4)	130 (79.2)
Total		189 (33.0)	79 (14.4)	44 (52.6)	312

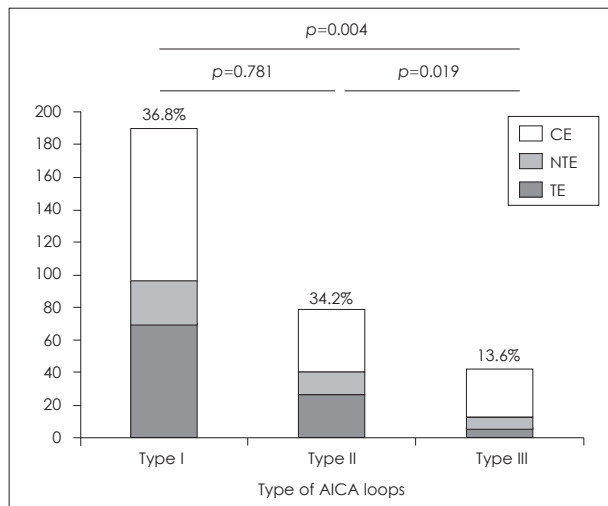


Fig. 3. Rate of tinnitus according to the types of AICA loop. TE: Ear with tinnitus of patient group, NTE: ear without tinnitus of patient group, CE: control ears, AICA: anterior inferior cerebellar artery.

loops was found to have a significantly higher association with tinnitus than those of the type III AICA loop ($p=0.004$, $p=0.019$)(Fig. 3).

The association between hearing loss and type of AICA loops

In the tinnitus ear, hearing loss was found to be present in 17.4% (12/69) of type I AICA loop, 22.2% (6/27) of type II AICA loop, and 0.0% (0/7) of type III AICA loop. In the non-tinnitus ear, hearing loss was found to be present in 3.8% (1/26) of type I AICA loop, 0% (0/13) of type II AICA loop, and

16.7% (1/6) of type III AICA loop. In the control ear, hearing loss was found to be present in 19.1% (18/94) of type I AICA loop, 23.1% (9/39) of type II AICA loop, and 22.6% (7/31) of type III AICA loop. In the tinnitus ear, no statistically significant association between hearing loss and the presence of any vascular type was observed ($p=0.428$)(Table 3).

In the tinnitus ear, the hearing threshold was 15.6 ± 11.6 dB for type I AICA loop, 15.2 ± 10.6 dB for type II AICA loop and 10.7 ± 3.4 dB for type III AICA loop. No statistically significant association between hearing threshold and the presence of any vascular type was observed ($p=0.076$).

Tinnitus and thickness of AICA loops

The ‘small’ AICA loops were found to be present in 84.5% (87/103) of tinnitus ears, 88.9% (40/45) of non-tinnitus ears, and 69.5% (114/164) of control ears. The ‘large’ AICA loops were found to be present in 15.5% (16/103) of tinnitus ears, 11.1% (5/45) of non-tinnitus ears and 30.5% (50/164) of control ears (Table 4). The prevalence of tinnitus according to thickness of AICA loops was 36.1% (87/241) in the small group and 22.5% (16/71) in the large group and these values were significantly different ($p=0.044$)(Fig. 4).

Discussion

Vestibulocochlear nerve compression syndrome describes a clinical entity including symptoms like tinnitus, vertigo, hearing loss characterized by compression of CN VIII by AICA loops. AICA loops that comes into contact with the CN VIII in

Table 4. Thickness of AICA loops in the patient and control groups

Thickness of AICA loops	Patients group		Control group	Total (n=312)(%)
	Symptomatic sides of patients (n=103)(%)	Asymptomatic sides of patients (n=45)(%)	Control (n=164)(%)	
Small	87 (84.5)	40 (88.9)	114 (69.5)	241
Large	16 (15.5)	5 (11.1)	50 (30.5)	71
Total	103	45	164	312

AICA: anterior inferior cerebellar artery

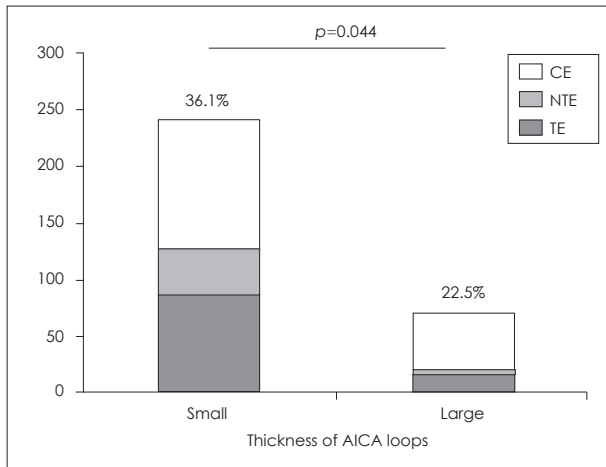


Fig. 4. Rate of tinnitus according to the thickness of the AICA loop between groups. TE: ear with tinnitus of patient group, NTE: ear without tinnitus of patient group, CE: control ears, AICA: anterior inferior cerebellar artery.

IAC or CPA can cause local demyelination, reorganization of the nerve and axonal hyperactivity, which results in otologic symptoms like tinnitus, hearing loss or vertigo.^{9,10}

The cranial nerve is composed of a central nervous system (CNS) and peripheral nervous system (PNS) segment, connected by the root entry/exit zone (REZ). Compression of CN VIII at REZ by AICA loops was thought to be the main factor causing tinnitus.¹¹ However, the results of many subsequent studies have called into question the validity of this hypothesis. In addition, many studies have reported that vascular compression at CNS of CN VIII rather than REZ is responsible for causing otologic symptoms. First, microvascular decompression of the CNS of CN VIII in patients who complain of tinnitus was highly successful in alleviating symptoms without compression at the REZ.¹¹ Second, because the peripheral segments of the cranial nerve is more resistant to compression than central segments, vascular compression at CNS has more significant effect than those of the PNS, REZ.¹¹

Unlike other cranial nerve compression syndromes, the neurovascular compression of CN VIII can cause complicated symptoms including vertigo, tinnitus and hearing loss. This complicated symptomatology makes vascular compression syndromes of CN VIII difficult to understand. Because of this, many are skeptical about vestibulocochlear compression syn-

drome. In addition, cadaveric and radiologic studies have shown that there are considerable differences regarding the occurrence and effects of vascular loops in the CPA.⁸⁾

McDermott, et al.⁸⁾ reported that tinnitus was not associated with the presence of AICA loops in CPA or ICA, although they did find a relationship between AICA loops and unilateral hearing loss. Other studies^{12,13)} have also reported that there was no relationship between nonspecific vestibulocochlear symptoms and the type of AICA loops.

The results of this study are not in agreement with these previous studies. The association between tinnitus and the presence of type of AICA loop was found to be statistically significant. Especially, the presence of type I and II AICA loops was shown to have a significantly higher association with tinnitus than those of type III AICA loop. Thus, these results are comparable to hypothesis that compression of CN VIII by AICA loops at REZ is responsible for tinnitus¹¹⁾ and vascular compression at REZ interferes with neuronal transduction in CN VIII is the cause tinnitus.^{9,14)} Our results showed the ‘small’ sized AICA loops were found to be significantly associated with tinnitus. This can be explained by Applebaum’s hypothesis¹⁵⁾ that reduced vascular perfusion and turbulent flow produced by ‘small’ AICA loops can cause inner ear dysfunction.

By using 3D-FIESTA MRI, we were able to that there was a statistically significant association between the presence of type I and type II AICA loops and tinnitus. In addition, ‘small’ sized AICA loops within the CPA were found to be significantly associated with tinnitus. The limitation of this study was that the patients had not been diagnosed as ‘Vestibulocochlear nerve compression syndrome’. Therefore, we believe that a well-designed, clinical study should be performed to further assess and verify these initial findings.

Conclusion

The results of this study showed that the presence of type I or type II AICA and ‘small’ AICA loops were correlated with unexplained tinnitus. These results are compatible with the hypothesis that compression of CN VIII by AICA loops at REZ and impaired blood flow in ‘small’ AICA loops cause tinnitus.

Therefore, it seems reasonable that diagnostic work-up should include 3D-FIESTA MRI scans of the IAC and CPA when patients complain about unexplained tinnitus. The authors also think that if further studies were conducted using patients who were diagnosed with vestibulocochlear compression syndrome, more meaningful result could be obtained.

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