Original Article

Can Anatomical Variations of AICA Loop be a Cause of Hearing Loss which can Affect the Laterality of Tinnitus Also?

AICA Loop Anatomik Varyasyonları Tinnitus Lateralitesini de Etkileyebilecek İşitme Kaybının Bir Nedeni Olabilir Mi?

ⓘ Eda TUNA YALÇINOZAN[1], ⓘ Yasemin KÜÇÜKÇİLOĞLU[2]

[1]Near East University Faculty of Medicine, Department of Otolaryngology, Division of Head and Neck Surgery, Nicosia, Northern Cyprus
[2]Near East University Faculty of Medicine, Department of Radiology, Lefkoşa, Northern Cyprus

ABSTRACT

Objective: We aimed to reveal whether the vascular loop variations created by the antero-inferior cerebellar artery (AICA) in the cerebellopontine angle (CPA) and the internal acoustic canal (IAC) can be a cause, that can affect laterality of tinnitus and cause hearing loss.

Methods: The data and the magnetic resonance imaging scans were collected retrospectively and 109 patients with tinnitus were included in the study. Data were recorded according to the patients' duration and laterality of tinnitus, laterality, type and frequency of hearing loss ,contact type and extension of AICA with remarking the side of the vascular loop.

Results: A statistically significant difference was found in between the left ear high frequency (HF) hearing loss (p=0.042) and extension type of the AICA. Also showed statistically significant results in between left sided tinnitus (p=0.030) and the Chavda classification. Besides the patients who had right sided tinnitus, was found to be in relation with the right sided contact type as the p value was 0.023.

Conclusion: Our results suggest that AICA variations in CPA and IAC can cause HF hearing loss as well as particularly affect the side of tinnitus. Nevertheless, this relationship must be confirmed by more advanced audiological and imaging techniques.

Keywords: Tinnitus, hearing loss, anterior inferior cerebellar artery

ÖZ

Amaç: Anterior inferior serebellar arterin (AICA), serebellopontin açıda (SPA) ve internal akustik kanalda (İAK) oluşturduğu vasküler loop varyasyonlarının, tinnitus hissedilme tarafını etkileyen ve işitme kaybı oluşturabilen bir neden olup olamayacağını ortaya koymayı amaçladık.

Yöntemler: Toplam 109 tinnitus hastasının verileri retrospektif olarak toplandı ve çalışmaya dahil edildi. Hastaların tinnitus süresi ve lateralitesi, işitme kaybının tipi ve kulak tarafı, manyetik rezonans görüntülemede AICA vasküler loop tarafı, temas tipi ve uzantısı, odyolojik ve radyolojik görüntüleme verileri taranarak kaydedildi.

Bulgular: Sol kulakta yüksek frekanslı (YF) işitme kaybı (p=0,042) ile AICA'nın ekstansiyon tipi arasında istatistiksel olarak anlamlı fark bulundu. Ayrıca sol taraflı tinnitus (p=0,030) ile Chavda sınıflandırması arasında istatistiksel olarak anlamlı sonuçlar gözlendi. Bunların yanı sıra sağ tarafta tinnitusu olan hastalar ile sağ taraflı temas tipi arasında anlamlı ilişkili saptandı (p=0,023).

Sonuç: Sonuçlarımız, SPA ve İAK'deki AICA varyasyonlarının YF'li işitme kaybına yol açabileceği gibi kulak çınlamasının tarafını da özellikle etkileyebileceğini ortaya koymaktadır. Yine de bu ilişki daha ileri odyolojik ve görüntüleme teknikleriyle onaylanmalıdır.

Anahtar Sözcükler: Tinnitus, işitme kaybı, anterior inferior serebellar arter

Cite this article as: Tuna Yalçınozan E, Küçükçiloğlu Y. Can Anatomical Variations of AICA Loop be a Cause of Hearing Loss which can Affect the Laterality of Tinnitus Also? Bezmialem Science 2022;10(5):596-601

Introduction

Hearing of a nonexisting sound which is perceived without an external or internal stimuli is called tinnitus. Approximately 12% of the general population suffers from tinnitus worldwide (1-4). Tinnnitus can be both temporary or chronic and can be defined as "ringing in the ears" and as different perceptions of sound including buzzing, rustling, hissing, whistling, pulsing, roaring and clicking. Tinnitus is not a disease in itself, but rather a symptom of another health condition. In most cases, tinnitus is a sensorineural reaction that occurs in the brain and hearing system. While tinnitus is often associated with hearing loss, there is a wide variety of health problems that can cause tinnitus as a symptom however the etiology remains uncertain in some patients (5,6). To determine the possible causes of tinnitus in a patient, physical examination with detailed medical history has to be performed. Neuroradiologic imaging may also help to identify the neuro-otologic causes in mandatory cases. Vascular anatomical variants can also be considered in tinnitus etiology which can be revealed clearly with imaging methods.

Cerebellum folding around the pons and middle cerebellar peduncle forms a V-shaped cleft which is located between the superior and inferior limbs of the cerebellopontine fissure in the posterior fossa called cerebellopontine angle (CPA). The anterior inferior cerebellar artery (AICA) is a branch of the basilary artery and usually courses posterolaterally in the CPA, but various pathways of AICA are defined (7,8). The vestibulocochlear nerve (CNVIII) passes through the internal auditory canal (IAC), travels along anterolaterally in the CPA than enters the brainstem and facial nerve (CNVII) also transmits to the face via CPA through the IAC (9). Vascular compression of CNVIII can be the cause of tinnitus and accompany some other symptoms such as dizziness, hemifacial spasm, trigeminal neuralgia and vertigo. Jannetta (10) depicted that hemifacial spasm, trigeminal neuralgia, hearing loss, tinnitus and vertigo might be associated with vascular loop of AICA within the IAC compressing on cranial nerves (11). There are theories in the etiology of vascular orginated tinnitus. The primary clarification is that changes within the blood stream disturb the laminar stream and newly existing turbulence can cause an audible sound. Besides, physiologically streaming sounds of the vascular structures can be heard more escalated with bone conduction when the impact of outside sound masking effect weakens. The chronic compression of the vessel may be responsible for regional demyelination or impairment of blood flow on the nerve. These impacts can lead to dysfunction of the nerve and also development of symptoms (12).

Computed tomography and magnetic resonance imaging (MRI) have been utilized in the evaluation of microvascular compression (MVC). MRI has appeared to be a more appropriate imaging in determining the vascular loops, besides it is a supportive technique to analyze the other causes of tinnitus (11). The subject of radiological imaging still remains unclear in patients with tinnitus. The candidates can be either patients with unilateral tinnitus and hearing loss or patients with asymmetrical hearing loss or patients with unspecified audiometric results (3,6,13). Despite all these uncertainties, it is obvious that MRI is the best way to identify the AICA vascular loop. But MVC can be detected commonly in general population, so it can bring along a contradiction. Cadaveric studies also revealed that AICA loop in IAC could be found in the prevelance of 40% (7,14).

We aimed to evaluate the association between the laterality of tinnitus and hearing loss with the types of vascular loops of AICA in the CPA and IAC, examining through the MRI scans.

Methods

We carried out a retrospective study in our tertiary referral center with the approval of local ethical committee (app no: 2020/85-1195). The patients were selected from our databasa in between January 2017 and December 2019, and their medical history and physical examination notes were searched thoroughly. Patients who were under 18 years old and who had any other diagnosed otologic, neurologic or systemic diseases in their history were excluded from the study. One hundred nine patients who had tinnitus and also vascular loop in MRI were recorded according to their demographic factors (age and gender), duration and laterality of tinnitus, contact type and extension of the vascular loop, laterality and type of hearing loss (PTA and high frequency). Especially, none of the patients complained from significant pulsation, they mostly complained from buzzing or ringing in the ear.

Data Collection

Radiological Evaluation

The MRIs were performed with a 1.5 T system (Magnetom Aera, Siemens Healthcare, Erlangen, Germany) with a head coil. The standart imaging protocol included transverse T2 weighted [echo time (TE) =110, repetition time (TR) =6,690 ms, slice thickness =5 mm] screening of the whole brain. For the evaluation of CPA; transverse fat-saturated (FS) T1 weighted (TE =9.7 ms, TR =404 ms, slice thickness =2 mm), T2 weighted (TE =111 ms, TR =4,540 ms, slice thickness =2 mm), T2 weighted SPACE (TE =156 ms, TR =1,400 ms, slice thickness =0.6 mm), coronal FS T1 weighted (TE =7.3 ms, TR =697 ms, slice thickness =2 mm), T2 weighted (TE =93 ms, TR =3,720 ms, slice thickness =2 mm), and following gadolinium-based contrast material injection (10 mL, 0.5 mmol/mL, gadoteric acid, Guerbet, Roissy, France), transverse FS T1 weighted (TE =9.7, TR =120 ms, slice thickness =2 mm) and coronal FS T1 weighted (TE =7.3, TR =150 ms, slice thickness =2 mm) images were used.

The previous reports of the images were not taken into consideration as MRIs were newly evaluated by a single radiologist blinded to the clinical history and findings. The radiologist examined MRIs for the location of AICA loops in the IAC and position of the loops relative to nerve(s), and also any other pathologic abnormalities in CPA, cochlea and/ or vestibulum were noted. The location of AICA loop in the IAC was graded according to the Chavda classification (Type I: lying within the CPA but not entering the IAC, Type II: entering but not extending >50% of the length of the IAC, Type III: extending >50% of the IAC) (15). The position of the

loop relative to nerve(s) was evaluated in transverse view and described in terms of the type of contact to the nerve(s) (Type A: crossing 2 (vestibular and cochlear nerve) or 4 nerves in the IAC (as a bridge), Type B: crossing only one nerve, Type C: running parallel to the nerves but not crossing them) (16).

Audiologic Tests

Auditory testing results of all patients who were included in the study were obtained by searching the audiologic archives. Tests were performed in a soundproof cabin with Radioear- 3045 earphones (Therapeutic Technologies, Bournemouth, UK). Pure tone average (PTA) was calculated from auditory thresholds recorded at 0.5, 1, and 2 kHz and high frequency (HF) hearing levels at 4 and 8 kHz were recorded also with Interacoustics AC 40 Clinical audiometer (Interacoustics, Assens, Denmark). The tresholds which were higher than 25 dB were accepted as hearing loss. According to their test results, the patients were classified as having normal hearing treshold, right sided asymmetric hearing loss, left sided asymmetric hearing treshold, bilateral symmetric hearing loss (difference at least 20 db between ears) in PTA treshold and also in HF levels (4 kHZ - 8 kHZ).

Statistical Analysis

The data were analyzed with SPSS 23.0 (IBM, Armonk, NY, USA). The descriptive data results were given as mean ± standard deviation and median (minimum-maximum). Chi-square test was used to compare the categorical variables in the groups and results were presented as frequency (n) and also in percent (%). Statistical significance level was stated as p<0.050.

Results

Demographic Data

There were 109 patients in this study consisting of 63 male and 46 female patients with a mean age of 43.6±17.5 (range 18-80). Fourty two (38.5%) patients had tinnitus symptom less than 1 year, 43 (%39.4) patients had symptom for 1 to 3 years, 14 (12.8%) patients had tinnitus for 3 to 5 years and 10 (9.2%) patients suffered from tinnitus over 5 years. While 43.1% (n=47) of patients had bilateral symmetrical tinnitus, 33% (n=36) of them had tinnitus only on the right and 23.9% (n=26) of them only on the left side. According to the gender of the patients, there was no statistically significant difference in terms of the distribution of AICA extension and contact in IAC for both ear (p>0.050).

Audiometric Findings

Audiometric results revealed that 54.1 % (n=59) of patients had normal hearing level treshold in PTA while 17.4% (n=19) of them had right sided asymmetrical, 12.8% (n=14) left sided asymmetrical and the rest 15.6% (n=17) had bilateral symmetrical hearing loss. In HF audiometry test, 49.5% (n=54) of the patients had symmetrical hearing loss while 21.1% (n=23) had normal hearing tresholds. There was no relationship between hearing loss and tinnitus (Table 1).

MRI Findings

According to the Chavda classification; the frequency of Type I AICA placement was 47.7% (n=52), the frequency of Type II was 41.3% (n=45) and the frequency of Type III was 11% (n=12) in right IAC. For the left IAC, the frequency of Type I extension of AICA was 70% (n=64.2) (Figure 1). The frequencies of Type II and Type III extensions were 29.4% (n=32) and 6.4 % (n=7), consecutively in left ear. In the MRI evaluation, Type A contact was also found in high percentages for both ears as 91.7 % (n=100) and 88.1% (n=96) (Figure 2), respectively in the right and the left ears of the patients.

The patients who had right sided tinnitus were found to be in relation with the right sided contact type (p=0.023). Among the patients who had right sided tinnitus, 94.4% (n= 34) of them had Type A (Figure 3) and 5.6% (n=2) (Figure 4) of them had Type B contacts in the right IAC. Besides, there was statistically significant relationship between the Chavda classification of the left ear and left sided tinnitus (p=0.030). Of the patients 46.2% (n=12) who had left sided tinnitus had Type I and 50% of them (n=13) had Type II extensions into the left IAC (Table 2).
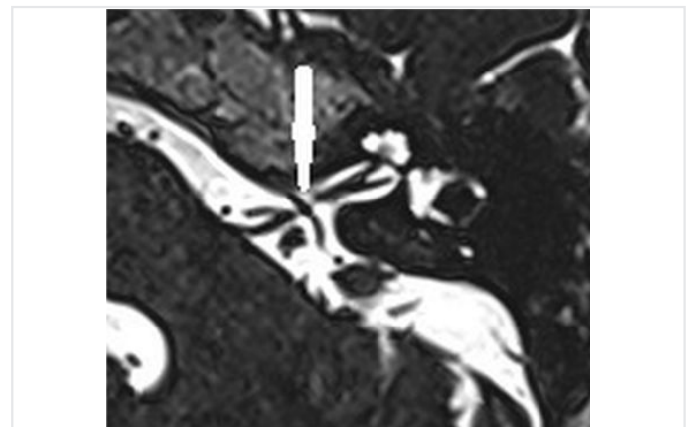

Figure 1. Transverse T2 SPACE image demonstrates vascular loop located in left cerebellopontine angle, crossing over the nerves (white arrow) (Type I A)
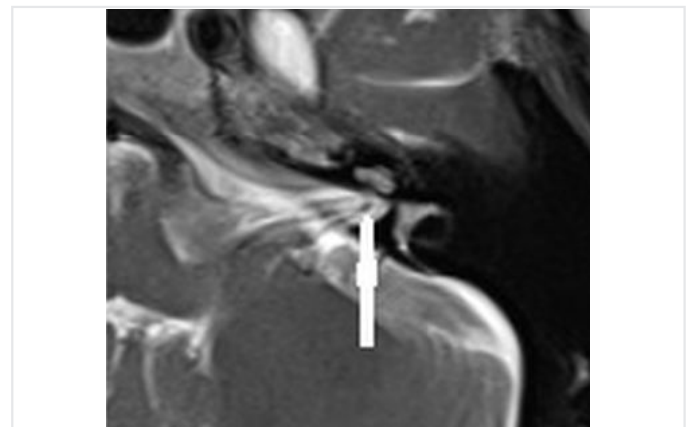

Figure 2. Transverse T2 weighted image demonstrates vascular loop entering and extending >50% of left internal acoustic canal, crossing over the nerves (white arrow) (Type III A)

Table 1. The comparison of PTA and HF acording to the ear with tinnitus symptom

	Right sided n (%)	Left sided n (%)	Bilateral n (%)	Total n (%)	p value
PTA					
Normal threshold levels	16 (44.4)	16 (61.5)	27 (57.4)	59 (54.1)	
Right sided asymmetrical HL	9 (25)	4 (15.4)	6 (12.8)	19 (17.4)	
Left sided asymmetrical HL	3 (8.3)	3 (11.5)	8 (17)	14 (12.8)	0.471
Bilateral symmetrical HL	8 (22.2)	3 (11.5)	6 (12.8)	17 (15.6)	
HF					
Normal threshold levels	8 (22.2)	5 (19.2)	10 (21.3)	23 (21.1)	
Right sided asymmetrical HL	7 (19.4)	4 (15.4)	4 (8.5)	15 (13.8)	
Left sided asymmetrical HL	6 (16.7)	4 (15.4)	7 (14.9)	17 (15.6)	0.844
Bilateral symmetrical HL	15 (41.7)	13 (50)	26 (55.3)	54 (49.5)	

number (% percentage), PTA: Pure tone avarage, HF: High frequency, HL: Hearing loss,
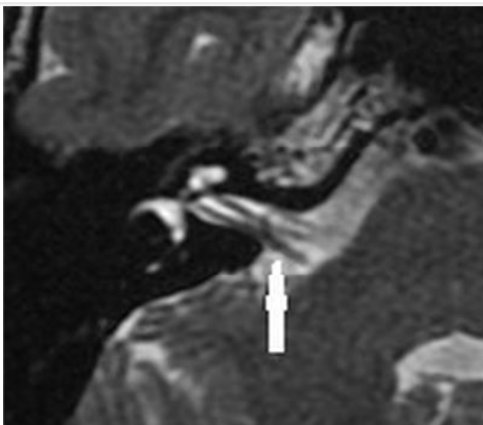*p value of chi-square test statistics which are statistically significant (p<0.050)

Figure 3. Transverse T2 weighted image demonstrates vascular loop entering and extending <50% of right internal acoustic canal, crossing over the nerves (white arrow) (Type II A)
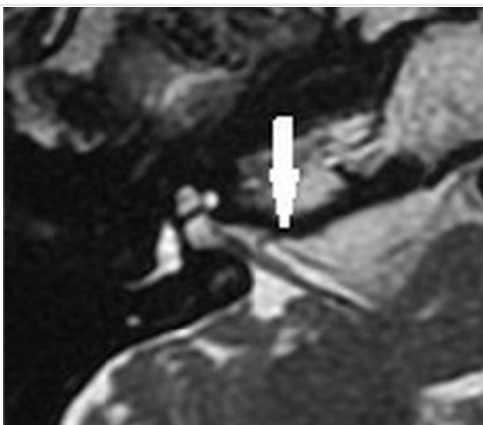

Figure 4. Transverse T2 weighted image demonstrates vascular loop entering and extending <50% of right internal acoustic canal, in contact with one nerve (white arrow) (Type II B)

Although there was no relationship between the HF audiologic findings and contact type of AICA in both ears (p>0.050), a statistically significant relationship was found between the left ear HF hearing loss and the Chavda classification (p=0.042) (Table 3).

Discussion

The neurovascular anatomical structures in CPA vary widely. These structural changes can cause several symptoms according to their relationship within surroundings. Vascular compression of the seventh and eighth cranial nerves in the CPA may lead to hearing loss, tinnitus, vertigo, dizziness, imbalance and hemifacial spasm.

There were many studies which aimed to investigate the effects of vascular compression on the CNVIII. We studied the relation between hearing loss and the AICA loop extension/contact to the CNVIII in tinnitus. Ultimately, a relationship was found between HF hearing loss and tinnitus, and the extension and contact types of AICA in the IAC. The Type A contact (n=100) and Type I (n=52) & Type II (n=45) extensions were the most frequent ones in right IAC. According to these, not the extension position but the type of the contact seemed to be affecting only the perception of tinnitus in the right ear. On the other hand, the left IAC imaging revealed that the Type I extension (n=70) and Type A contact (n=96) were the most common anatomic variations. But when compared to the right side findings, extension type was found to be related to both tinnitus and HF hearing loss in the left ear. Correspondingly, Di Stadio et al. (16) reported that direct contact between the vascular loop and the particular nerve could be the causal explanation of the symptoms arising and that also Type A (the most common) localization could bring out the nerve impingements. In our study, as we included the patients only with the tinnitus symptom, we revealed that Type A contact was the common anatomical variation almost in all patients for both sides. Similarly, in a meta-analysis of Chadha and Weiner (17), it was suggested that the patients with pulsatile

Table 2. Comparison of tinnitus in each ear with the extension type and contact type of AICA

	Right sided tinnitus n (%)			p value
	(+)	(-)	Total	
R extention				
Type I	37 (50.7)	15 (41.7)	52 (47.7)	
Type II	30 (41.1)	15 (41.7)	45 (41.3)	0.372
Type III	6 (8.2)	6 (16.7)	12 (11)	
R contact				
Type A	66 (90.4)	34 (94.4)	100 (91.7)	
Type B	---	2 (5.6)	2 (1.8)	**0.023***
Type C	7 (9.6)	---	7 (6.4)	
	Left sided tinnitus n(%)			
	(+)	(-)	Total	
L extention				
Type I	58 (69.9)	12 (46.2)	70 (64.2)	
Type II	19 (22.9)	13 (50)	32 (29.4)	0.030*
Type III	6 (7.2)	1 (3.8)	7 (6.4)	
L contact				
Type A	72 (86.7)	24 (92.3)	96 (88.1)	
Type B	2 (2.4)	0 (0)	2 (1.8)	0.641
Type C	9 (10.8)	2 (7.7)	11 (10.1)	

number (% percentage), (+): Means the presence of tinnitus, (-): Means absence of tinnitus,
*p value of chi-square test statistics which are statistically significant (p<0.050)

tinnitus weremore likely to have vascular abnormalities in MRI. In our results altough there was no pulsatile tinnitus complaint among the patients we encountered with the Type A contact and also Type I extension. Even the Type I extension brings along the lowest risk of nerve impengement, this placement can still lead to perception of tinnitus. Also, Yoo et al. (18) supported the idea that there was an association between the unexplained tinnitus and Type I & II extensions in IAC and even the small sized AICA loops in CPA. Nowé et al. (19) revealed that if the vascular compression on the CNVIII in cisternal segment was present, there was a possible relation between the high and low pitch tinnitus and hearing loss as well. McDermott et al. (15) reported that tinnitus was not associated with the presence of the vascular loop, but AICA loops caused unilateral hearing loss once in the IAC when extending more than 50%. Although the most common extension was not more than 50% in our study, likewise we found correlation between the HF hearing loss and extension of AICA into the IAC. In contrast, there are several studies which support the idea that the finding of tinnitus in AICA loops are incidental or may not always be a pathological finding (11,20-22). de Abreu Junior et al. (23) also stated that there was no direct relationship between MRI findings of AICA and clinical symptoms. In another retrospective study which Li et al. (20) conducted, it was reported that high prevalence of any vascular loops could be present in the asymptomatic ear so the ipsilateral symptoms might not be associated with this vascular placement.

Certainly, in our study, laterality of the most frequent type of extension correlated with the side of the HF hearing loss and the side of the tinnitus. Friede has suggested that the outer surface of the cochlear nerve is composed of the nerves that transmit the high pitched sound as they are originated from the basal turn, however the central nerves are originated from the cochlear apex which transmit the low pitched sound, so called tonotopic organization (24). This formation may also explain the HF hearing loss in this study. Nevertheless, tinnitus still can be related with the high prevalence of the Type I extension on the left side and also the HF hearing loss prevelance may also seem to be affecting our results. As all of our subjects had various types and grades of AICA, fibrosis might occur on the nerve structures due to the long standing vascular compression. But concurrently, in our study, almost half of the patients had bilateral symmetrical HF hearing loss which might also lead to tinnitus. Because this is a retrospective study in which all of the participants have AICA variations and tinnitus, we may not put forward very definitive considerations about the subject (25).

Study Limitations

Constitution of a control group from the patients who did not have neither tinnitus nor hearing loss to find a stronger audiological correlation could be the limitation of our study.

Conclusion

This study showed correlation between HF hearing loss, tinnitus and AICA placement. But since the pathophysiology of tinnitus is still unclear and there are multiple factors in the etiology of hearing loss , this relationship must be confirmed with further researches which will be conducted with improved neuroimaging techniques.

Table 3. Comparison of high frequency hearing loss with the left ear AICA extension type

HF	Left ear			Total	p value
	Type I	Type II	Type III		
Normal threshold levels	13 (18.6)	7 (21.9)	3 (42.9)	23 (21.1)	
Right sided asymmetrical HL	7 (10)	7 (21.9)	1 (14.3)	15 (13.8)	
Left sided asymmetrical HL	12 (17.1)	2 (6.3)	3 (42.9)	17 (15.6)	**0.042**
Bilateral symmetrical HL	38 (54.3)	16 (50)	---	54 (49.5)	

number (% percentage), HF: High frequency, HL: Hearing loss, *p value of chi-square test statistics which are statistically significant (p<0.050). *p value of chi-square test statistics which are statistically significant (p<0.050)

Ethics

Ethics Committee Approval: Near East University Scientific Research Ethics Committee (date: 26.11.2020/number: 1195).

Peer-review: Externally peer reviewed.

Authorship Contributions

Surgical and Medical Practices: E.T.Y., Y.K., Concept: E.T.Y., Design: E.T.Y., Data Collection or Processing: E.T.Y., Y.K., Analysis or Interpretation: E.T.Y., Y.K., Literature Search: E.T.Y., Writing: E.T.Y.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

References

1. Axelsson A, Ringdahl A. Tinnitus -- a study of its prevalence and characteristics. Br J Audiol 1989;23:53-62.

2. Chung DY, Gannon RP, Mason K. Factors affecting the prevalence of tinnitus. Audiology 1984;23:441-52.

3. Lockwood AH, Salvi RJ, Burkard RF. Tinnitus. N Engl J Med 2002;347:904-10.

4. Nondahl DM, Cruickshanks KJ, Wiley TL, Klein R, Klein BE, Tweed TS. Prevalence and 5-year incidence of tinnitus among older adults: the epidemiology of hearing loss study. J Am Acad Audiol 2002;13:323-31.

5. Crummer RW, Hassan GA. Diagnostic approach to tinnitus. Am Fam Physician 2004;69:120-6.

6. Schwaber MK. Medical evaluation of tinnitus. Otolaryngol Clin North Am 2003;36:287-92.

7. Reisser C, Schuknecht HF. The anterior inferior cerebellar artery in the internal auditory canal. Laryngoscope 1991;101:761-6.

8. Sirikci A, Bayazit Y, Ozer E, Ozkur A, Adaletli İ, Cuce MA, et al. Magnetic resonance imaging based classification of anatomic relationship between the cochleovestibular nerve and anterior inferior cerebellar artery in patients with non-specific neuro-otologic symptoms. Surg Radiol Anat 2005;27:531-5.

9. Rhoton AL Jr. The cerebellopontine angle and posterior fossa cranial nerves by the retrosigmoid approach. Neurosurgery 2000;47:93-129.

10. Jannetta PJ. Neurovascular cross-compression in patients with hyperactive dysfunction of the eighth cranial nerve. Surg Forum 1975;26:467-9.

11. Gorrie A, Warren FM 3rd, de la Garza AN, Shelton C, Wiggins RH 3rd. Is There a Correlation Between Vascular Loops in the Cerebellopontine Angle and Unexplained Unilateral Hearing Loss? Otol Neurotol 2010;31:48-52.

12. Hofmann E, Behr R, Neumann-Haefelin T, Schwager K. Pulsatile tinnitus: imaging and differential diagnosis. Dtsch Arztebl Int 2013;110:451-8.

13. Peifer KJ, Rosen GP, Rubin AM. Tinnitus: etiology and management. Clin Geriatr Med 1999;15:193-204.

14. Herzog JA, Bailey S, Meyer J. Vascular loops of the internal auditory canal: a diagnostic dilemma. Am J Otol 1997;18:26-31.

15. McDermott AL, Dutt SN, Irving RM, Pahor AL, Chavda SV. Anterior inferior cerebellar artery syndrome: fact or fiction. Clin Otolaryngol Allied Sci 2003;282:75-80.

16. Di Stadio A, Dipietro L, Ralli M, Faralli M, Della Volpe A, Ricci G, et al. Loop characteristics and audio-vestibular symptoms or hemifacial spasm: is there a correlation? A multiplanar MRI study. Eur Radiol 2020;30:99-109.

17. Chadha NK, Weiner GM. Vascular loops causing otological symptoms: a systematic review and meta-analysis. Clin Otolaryngol 2008;33:5-11.

18. Yoo HS, Lee DW, Min HJ, Lee SH, Park CW. The Association of Anterior Inferior Cerebellar Artery in Internal Auditory Canal with Tinnitus and Hearing Loss. Korean J Audiol 2011;15:67-71.

19. Nowé V, De Ridder D, Van de Heyning PH, Wang XL, Gielen J, Van Goethem J, et al. Does the location of a vascular loop in the cerebellopontine angle explain pulsatile and non-pulsatile tinnitus? Eur Radiol 2004;14:2282-9.

20. Li L, Amiraraghi N, Begbie F, Kontorinis G. The significance of vascular loops in the internal auditory meatus: a true incidental imaging finding? Eur Arch Otorhinolaryngol 2019;276:3275-80.

21. Ensari N, Gür ÖE, Selçuk ÖT, Renda L, Osma Ü, Eyigör H, et al. Is Presence of Vascular Loop in Magnetic Resonance Imaging Always Related to Tinnitus? J Craniofac Surg 2017;28:295-8.

22. Makins AE, Nikolopoulos TP, Ludman C, O'Donoghue GM. Is There a Correlation Between Vascular Loops and Unilateral Auditory Symptoms? Laryngoscope 1998;108:1739-42.

23. de Abreu Junior L, Kuniyoshi CH, Wolosker AB, Borri ML, Antunes A, Ota VK, et al. Vascular loops in the anterior inferior cerebellar artery, as identified by magnetic resonance imaging, and their relationship with otologic symptoms. Radiol Bras 2016;49:300-4.

24. Friede RL. Cochlear axon calibres are adjusted to characteristic frequencies. J Neurol Sci 1984;66:193-200.

25. Okamura T, Kurokawa Y, Ikeda N, Abiko S, Ideguchi M, Watanabe K, et al. Microvascular decompression for cochlear symptoms. J Neurosurg 2000;93:421-6.