Kocatepe Tıp Dergisi
Kocatepe Medical Journal
22:445-449/ Ekim 2021 Sayısı

ARAŞTIRMA YAZISI / RESEARCH ARTICLE

NÖROTOLOJİK SEMPTOMLARI AÇIKLAMADA 7. - 8. SİNİR KOMPLEKSİ İÇİN VASKÜLER LOOP KOMPRESYONUNUN DEĞERLENDİRİLMESİ NE KADAR ETKİLİDİR ?

HOW EFFECTIVE IS THE EVALUATION OF VASCULAR LOOP COMPRESSION FOR THE 7TH-8TH NERVE COMPLEX IN EXPLAINING NEURO - OTOLOGICAL SYMPTOMS ?

Selçuk YILDIZ[1], Ayşe Özlem BALIK[2]

[1]Haydarpaşa Numune Eğitim ve Araştırma Hastanesi,
Kulak Burun Boğaz Hastalıkları ve Baş-Boyun Cerrahisi Kliniği
[2]Haydarpaşa Numune Eğitim ve Araştırma Hastanesi, Radyoloji Kliniği

ÖZET

AMAÇ: Bu çalışmanın amacı, vestibulokoklear sinirin (VCN) nörovasküler kompresyonunun radyolojik kanıtının tinnitus ve işitme kaybında patognomonik olup olmadığını manyetik rezonans görüntüleme (MRI) "3D Fast Imaging Employing Steady-State Acquisition (FIESTA)" sekansı kullanarak incelemektir.

GEREÇ VE YÖNTEM: Çalışma, sağ ve sol taraf dahil olmak üzere 85 hastada 170 temporal kemik değerlendirilmesi ile gerçekleştirildi. İnternal akustik kanal (İAK) orifisinde 1.5-Tesla MRI kullanılarak, anterior inferior serebellar arterin (AICA) sınıflandırılmış vasküler kompresyonları (Chavda sınıflandırması), AICA, superior serebellar arter (SCA) ve vertebral arter (VA) kompresyonu veya VCN'in distorsiyonu arasındaki anatomik ilişkinin noninvaziv değerlendirmesi yapıldı.

BULGULAR: Değerlendirilen 85 hastadan 36'sında (%42.4) vasküler loop izlenmedi. Chavda sınıflandırmasına göre 41'inde (%48.2) 1. derece vasküler loop, 7'sinde (%8.2) 2. derece vasküler loop ve 1'inde (%1.2) 3. derece vasküler loop saptandı. Ayrıca hastaların 6'sında (%7.1) VA kompresyonu ve 3'ünde (%3.5) SCA kompresyonu görüntülendi. Tinnitus şikayeti olan 16 (%32) hastada IAC distorsiyonu görüldü. Vasküler loop varlığı da sırasıyla tinnitus olmayan (%62.9) ve sağlıklı işiten (%51.8) olgularda yüksek insidans gösterdi. Hastaların nörotolojik semptomları için AICA loop tipleri, VA veya SCA ve IAC distorsiyonunun varlığı ve yokluğu arasında istatistiksel anlamlı farklılık saptanmadı ($p > 0,05$).

SONUÇ: Nörovasküler temas nadir bir bulgu değildir ve tinnitusla ilgili gözükmemektedir. Bununla birlikte 3D-FIESTA MRI kullanılması, VCN ve komşu vasküler varyasyonlar ile özellikle AICA varyasyonlarının ilişkisinin belirlenmesini iyi tanımlar ve mikrovasküler operasyonlar için vaka seçimine katkıda bulunur.

ANAHTAR KELİMELER: 3D-FIESTA, Nöro-otolojik semptom, Vasküler loop, Vasküler kompresyon sendromu, Vestibülokoklear sinir

ABSTRACT

OBJECTIVE: The goal of this research study was to investigate of whether the radiological proof of neurovascular compression of the vestibulocochlear nerve (VCN) was pathognomonic for hearing loss and tinnitus using "3D Fast Imaging Steady-State Acquisition (FIESTA)" magnetic resonance imaging (MRI) sequence.

MATERIAL AND METHODS: The research study was performed in 85 patients by evaluating 170 temporal bones, inclusive of both sides. The non-invasive assessment of the anatomical relationship between the classified vascular compression (Chavda classification) of the anterior inferior cerebellar artery (AICA) and the existence of AICA, superior cerebellar artery (SCA), vertebral artery (VA) compression or distortion of the VCN was applied by using 1.5-Tesla MRI at the internal acoustic canal (IAC).

RESULTS: Of the 85 participants examined, 42.4% (n = 36) presented no vascular loop (VL). 48.2% (n = 41) of the patients produced type 1 VL, 8.2% (n = 7) type 2 VL, and 1.2% (n = 1) type 3 VL accordingly to the Chavda classification. In addition, compressions of redundant VA and SCA were also observed in 7.1% (n = 6) and 3.5% (n = 3) of the patients respectively. Also, IAC distortion was found in 32% (n = 16) patients with tinnitus. The presence of vascular loops also showed a high incidence in patients with normal hearing (51.8%) and without tinnitus (62.9%), respectively. No statistically relevant variations were found between the existence and nonexistence of the VL of AICA forms, VA or SCA, and IAC distortion for the neurotological symptoms of patients ($p > 0.05$).

CONCLUSIONS: Neurovascular touch is not an uncommon finding. It doesn't appear to be related to tinnitus. However, the use of 3D-FIESTA MRI well defines the relationship between VCN and adjacent vascular variations and especially AICA variations and contributes to case selection for microvascular operations.

KEYWORDS: 3D-FIESTA, Neuro-otological symptom, Vascular loop, Vascular compression syndrome, Vestibulocochlear nerve

Geliş Tarihi / Received:05.09.2020
Kabul Tarihi / Accepted: 21.12.2020
Yazışma Adresi / Correspondence: Dr.Selçuk YILDIZ
Haydarpaşa Numune Eğitim ve Araştırma Hastanesi, Kulak Burun Boğaz Hastalıkları ve Baş-Boyun Cerrahisi Kliniği
E-mail: selcukyildiz60@hotmail.com
Orcid No (Sırasıyla):0000-0002-2820-8888, 0000-0002-5703-6720

INTRODUCTION

The anatomical region in which such essential structures as the trigeminal nerve, the facial nevre, and the vestibulocochlear nerve (VCN) arising from the brainstem is the cerebellopontine angle (CPA). Often, differences in the vessels underlying these structures lead to unique clinical circumstances that can be gathered into a group below the idiom "vascular compression syndrome (VCS)". This condition infers to ailments induced by a vessel's direct touch with a cranial nerve origin (1). VCN-associated VCS is also caused by the following anterior inferior cerebellar artery (AICA) (2). In this syndrome, vertigo, tinnitus, and hearing loss can occur clinically (3). Magnetic resonance imaging (MRI) is a procedure that is usually used in patients with neuro-otological symptoms of the internal auditory canal (IAC) and CPA imaging (4). Routine MRI sequences have an optimal soft-tissue resolution. On the other hand, the spatial resolution needed for the definition of cranial nerves (CN) may be deficient with this procedure (5). "Three-dimensional-fast imaging employing steady-state acquisition (3D-FIESTA)" is a high-resolution T2-weighted MRI technique. It provides more advanced spatial resolubility and a better definition of minor structures like CNs, particularly in cistern areas. The current literature datum indicates that vascular compression to the VCN can cause vertigo, tinnitus, and hearing loss (6 - 10). Nonetheless, controvertible findings have been noticed in the literature on the relation between vascular compressing to VCN and neuro-otological symptoms (1, 11-13). The goal of our research is to evaluate the compression of vertebral artery (VA), superior cerebellar artery (SCA), and vascular loop (VL) of AICA into/on VCN and the relationship of undetected neuro-otological symptoms using 3D-FIESTA sequence MRI.

MATERIAL AND METHOD

Patients who had been referred to the Ear, Nose, and Throat clinic due to their neuro-otological symptoms between 2018 and 2019 were reviewed retrospectively with a medical history and temporal bone MRI. The study included eighty-five patients (170 temporal bones) without any other underlying cause. Patients with CPA tumors or neuritis were excluded. There were no patients undergoing CPA operation or history of temporal bone trauma. Patients were classified into groups based on their major neuro-otological symptoms. The MRI was carried out with a General Electric (GE) Signa 1.5 T MRI system (GE Healthcare, Milwaukee, WI, USA) with an eight-channel head coil. MRI was performed with T2-weighted fast spin-echo images of the entire brain (TR, 4,500 ms; TE, 104 ms; NEX, 1.5; section thickness, 5.5 mm; intersection spacing, 1.5 mm; matrix size, 352 x 352). The standard temporal bone protocol included axial and coronal 3D T1-weighted images (TR, 12.3 ms; TE, 5.4 ms; NEX, 2; section thickness, 0.8 mm; intersection spacing, 0.4 mm; matrix size, 256 x 256, field of view, 200 x 200 mm), axial and coronal 3D-FIESTA images (TR, 5.9 ms; TE, 2.3 ms; NEX, 4; flip angle, 65º; section thickness, 0.8 mm; intersection spacing, 0.6 mm; matrix size, 416 x 416, field of view, 200 x 200 mm).

Compression or distortion caused by the VL of AICA, VA and SCA on the VCN at the different levels of the orifice IAC were evaluated and classified by the same radiologist. Those were classified using the method already defined by Chavda (11). According to this, Chavda type I, which lies just in CPA but does not reach IAC; Chavda type II enters IAC but does not exceed 50 percent of IAC; and Chavda type III exceeds 50 percent of IAC were named (11), **(Figure 1 - 4)**.

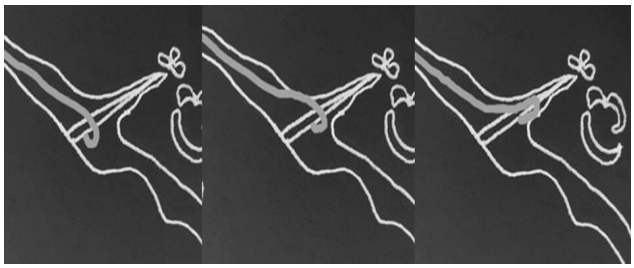

Figure 1: Schematic vascular loop of AICA types 1, 2, and 3 respectively.

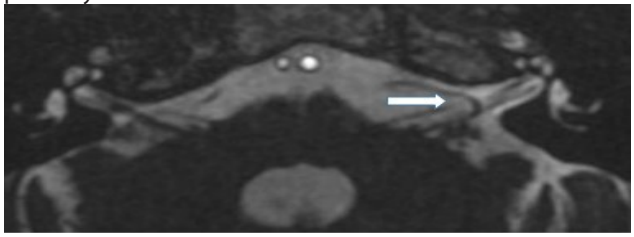

Figure 2: Vascular loop of AICA type 1, the touch of the left AICA loop with the cysternal segment of the 7th-8th nerve complex is shown (white arrow).

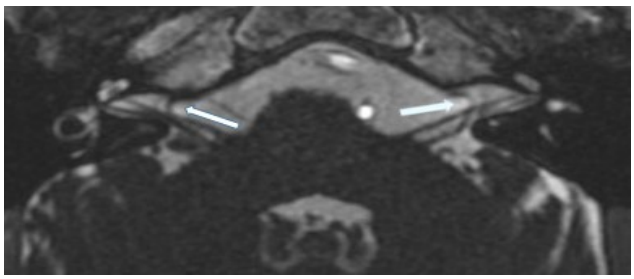

Figure 3: . Vascular loop of AICA type 2, the touch of the bilateral AICA loop with the 7th-8th nerve complex in the localization of the internal auditory canal(IAC) orifice is shown (white arrows).

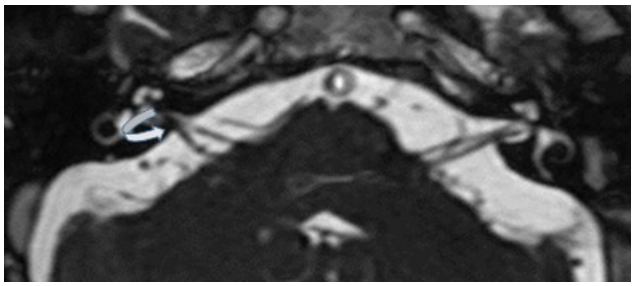

Figure 4: Vascular loop of AICA type 3, this figure shows the kinking of the right 8th nerve in the IAC orifice (curved grey arrow).

The existence of vascular contact was assessed at the contact point of the VCN as an indication of vascular compression. The Number Cruncher Statistical System software (NCSS, 2007; Kaysville, Utah, USA) was used for statistical analysis. Using illustrative statistics (e.g. standard deviation, mean, percentage, frequency, minimum, maximum) the study datum was summarized. The Pearson Chi-square test and the Fisher-Freeman-Halton Exact test were used to compare qualitative datum among study groups. The statistical significance was accepted as $p < 0.05$.

Ethical Committe

The study was accepted and performed in accordance with the ethical guidelines for medical research involving human subjects outlined in the Helsinki Declaration by the Haydarpaşa Numune Training and Research Hospital Clinical Research Ethics Committee (Approval number of the ethics committee: 2019/69). Because of the retrospective nature of the report, informed consent provisions were waived.

RESULTS

The age of the patients ranged from 12 to 82 years, with an average age of 49.74 ± 14.77 years. Of the cases, 34.1% (n=29) were female and 65.9% (n=56) were male. Tinnitus, hearing loss, and vertigo were seen in 58.8% (n = 50), 36.5% (n = 31), and 4.7% (n = 4) of the patients, respectively. Of the 85 cases examined, 42.4% (n = 36) of the patients had no VL, 48.2% (n = 41) of the patients had a type 1 VL, 8.2% (n = 7) of the patients had a type 2 VL, and 1.2% (n= 1) of the patients had a type 3 VL in accordance with the Chavda classification **(Table 1)**.

Table 1: Results of evaluating the occurence of symptoms with the existence of vascular loops and with the Chavda classification

		Chavda classification				
		No vascular loop n (%)	Chavda 1 n (%)	Chavda 2 n (%)	Chavda 3 n (%)	
Tinnitus	Absent	13 (37,1)	19 (54,3)	2 (5,7)	1 (2,9)	x^2: 2,588
	Present	23 (46,0)	22 (44,0)	5 (10,0)	0 (0)	**p: 0,476**
Hearing Loss	Absent	26(48,1)	24 (44,4)	4 (7,4)	0 (0)	x^2: 3,475
	Present	10 (32,3)	17 (54,8)	3 (9,7)	1 (3,2)	**p: 0,298**

Fisher-Freeman-Halton Exact Test

In addition, compression of redundant VA and compression of redundant SCA were determined in 7.1% (n = 6) and 3.5% (n = 3) of the patients, respectively. IAC distortion was found in 32% (n = 16) of the patients with a complaint of tinnitus **(Table 2)**.

Table 2: Evaluation of ICA Contact Presence According to the Presence of Tinnitus and Hearing Loss

		IAC Distortion		
		Absent n (%)	Present n (%)	
Tinnitus	Absent	21 (60,0)	14 (40,0)	x^2: 0,577
	Present	34 (68,0)	16 (32,0)	**p: 0,448**
Hearing Loss				x^2: 2,080
	Absent	38 (70,4)	16 (29,6)	
	Present	17 (54,8)	14 (45,2)	**p: 0,149**

Pearsonchi-square test

IAC distortion was also visualized in 45.2% (n =14) of the patients with hearing loss symptoms (Table 2). The VL of AICA also showed a high incidence in patients who have no tinnitus (62.9%) and normal hearing (51.8%) (Table 1). There were no statistically significant variations among the forms of the VL of AICA (p = 0.476). There were no statistically significant differences between the tinnitus group or hearing loss group for distortion of the VA or SCA and IAC (p = 0.298).

DISCUSSION

Jannetta et al. first suggested VCS of the VCN (14, 15). Several studies have suggested this concept by conducting microvascular decompression in patients suspected of having VCS of the VCN and showing good clinical results after surgery (7 - 9, 14 - 17). In the current lite-

rature, there is a debate over the clarification of neuro-otological complaints in patients with a vascular compression of VCN. A major concern is that, also in asymptomatic healthy individuals, the VLs of AICA are commonly found in IAC (11). Some authors have reported a powerful relationship between the involvement of the VL of AICA in IAC seen in MRI and pulsatile tinnitus (18, 19). Several MRI studies have indicated that there is no important relationship between tinnitus and vascular compression or configuration of AICA (20 - 23). In our research, there was no statistically significant correlation between tinnitus which we could not separate into tinnitus subtypes and compression of VA, SCA, and the VL's of AICA ($p = 0.448$). Also, we found no statistically relevant relation between tinnitus and the VL of AICA types according to the Chavda classification ($p = 0.476$).

Studies in the literature have shown an eloquent relationship between the VL of AICA and hearing loss (11, 24). No relationship was found between hearing loss and the VL of AICA in another study (22). There was no statistically relevant association in our study between hearing loss and compressions of VA, SCA, and the VL of AICA ($p = 0.149$). In addition, according to the Chavda classification, there was no statistically relevant association between the VL of AICA types and hearing loss ($p = 0.298$).

Møller et al. indicated that with microvascular decompression surgery, an 80% healing rate can be expected in patients with compromised positional vertigo (9). In other studies, there was no correlation between vertigo and the VL of AICA (22, 25). Only four patients with symptoms of vertigo were among the patients included in our study. VCS caused by VA, SCA, and the VL of AICA was not observed in any of these patients.

This study's major limitation is its retrospective design. Due to the retrospective nature of the study, specific information on the types and severity of tinnitus, the types and severity of hearing loss, and the types and severity of vertigo have not been provided. Neurovascular contact is not an uncommon finding, and may not cause the neuro-otological symptoms. However, VCN and adjacent vascular variations are well-identified by using 3D-FIESTA MRI, in particular by defining the relation between AICA variations and subscribing to the collection of cases of microvascular surgery.

3D-FIESTA MRI is one of the most significant methods to show vascular compression of VCN. Further studies on cases of the VL compression leading to VCN displacement or angulation can explain the etiology of neuro-otological symptoms and provide valuable evidence on the controversial need for microvascular decompression procedures.

REFERENCES

1. 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.

2. Markowski J, Gierek T, Kluczewska E, Witkowska M. Assessment of vestibulocochlear organ function in patients meeting radiologic criteria of vascular compression syndrome of vestibulocochlear nerve–diagnosis of disabling positional vertigo. Med Sci Monit. 2011; 17: CR169–73.

3. Wahlig JB, Kaufmann AM, Balzer J, Lovely TJ, Jannetta PJ. Intraoperative loss of auditory function relieved by microvascular decompression of the cochlear nerve. Can J Neurol Sci. 1999; 26: 44–7.

4. Kazawa N, Togashi K, Ito J. The anatomical classification of AICA/PICA branching and configurations in the cerebellopontine angle area on 3D-drive thin slice T2WI MRI. Clin Imaging. 2013; 37: 865–70.

5. Suzuki H, Maki H, Maeda M, Shimizu S, Trousset Y, Taki W. Visualization of the intracisternal angioarchitecture at the posterior fossa by use of image fusion. Neurosurgery. 2005; 56: 335–42.

6. Wuertenberger CJ, Rosahl SK. Vertigo and tinnitus caused by vascular compression of the vestibulocochlear nerve, not intracanalicular vestibular schwannoma: Review and case presentation. Skull Base. 2009; 19: 417–24.

7. Yap L, Pothula VB, Lesser T. Microvascular decompression of cochleovestibular nerve. Eur Arch Otorhinolaryngol. 2008;265:861–69.

8. Guevara N, Deveze A, Buza V, Laffont B, Magnan J. Microvascular decompression of cochlear nerve for tinnitus incapacity: pre-surgical data, surgical analyses, and long-term follow-up of 15 patients. Eur Arch Otorhinolaryngol. 2008; 265: 397–401.

9. Møller MB, Moller AR, Jannetta PJ, Jho HD, Sekhar LN. Microvascular decompression of the eighth nerve in patients with disabling positional vertigo: selection criteria and operative results in 207 patients. Acta Neurochir (Wien). 1993; 125: 75–82.

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

11. McDermott AL, Dutt SN, Irving RM, Pahor AL, Chavda SV. Anterior inferior cerebellar artery syndrome: fact or fiction. Clin Otolaryngol. 2003; 28: 75–80.

12. Sirikci A, Beyazıt Y, Ozer E, et al. Magnetic resonance imaging-based classification of anatomic relationship between the cochleovestibular nerve and anterior inferior cerebellar artery in patients with nonspecific neuro-otologic symptoms. Surg Radiol Anat. 2005; 27: 531–5.

13. Levy RA, Arts HA. Predicting neuroradiologic outcome in patients referred for audiovestibular dysfunction. AJNR Am J Neuroradiol. 1996; 17: 1717–24.

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

15. Jannetta PJ. Outcome after microvascular decompression for typical trigeminal neuralgia, hemifacial spasm, tinnitus, disabling positional vertigo, and glossopharyngeal neuralgia (honored guest lecture). Clin Neurosurg.1997; 44: 331–83.

16. Brackmann DE, Kesser BW, Day JD. Microvascular decompression of the vestibulocochlear nerve for disabling positional vertigo: the House Ear Clinic experience. Otol Neurotol. 2001; 22: 882–7.

17. Møller MB. Results of microvascular decompression of the eighth nerve as a treatment for disabling positional vertigo. Ann Otol Rhinol Laryngol. 1990; 99: 724–9.

18. Nowe´ V, De Ridder D, Van de Heyning PH, et al. Does the location of a vascular loop in the cerebellopontine angle explain pulsatile and nonpulsatile tinnitus? Eur Radiol. 2004; 14: 2282–9.

19. De Ridder D, De Ridder L, Nowe V, Thierens H, Van de Heyning P, Møller A. Pulsatile tinnitus, and the intrameatal vascular loop: why do we not hear our carotids? Neurosurgery. 2005; 57: 1213–7.

20. Gultekin S, Celik H, Akpek S, Oner Y, Gumus T, Tokgoz N. Vascular loops at the cerebellopontine angle: is there a correlation with tinnitus? AJNR Am J Neuroradiol. 2008; 29: 1746–9.

21. Clift JM, Wong RD, Carney GM, Stavinoha RC, Bovey KP. Radiographic analysis of cochlear nerve vascular compression. Ann Otol Rhinol Laryngol. 2009; 118: 356–61.

22. Erdogan N, Altay C, Akay E,et al. MRI assessment of internal acoustic canal variations using 3D-FIESTA sequences. Eur Arch Otorhinolaryngol. 2013; 270: 469–75.

23. Cicek ED. The Analysis of the Relationship Between Subjective Tinnitus and Vascular Loop, and Age and Gender Distribution: An MRI Study. EJMO. 2018; 2: 231–7.

24. Okamura T, Kurokawa Y, Ikeda N, et al. Microvascular decompression for cochlear symptoms. J Neurosurg. 2000; 93: 421–6.

25. Celiker FB, Dursun E, Celiker M, et al. Evaluation of vascular variations at cerebellopontine angle by 3D T2WI magnetic-resonance imaging in patients with vertigo. J Vestib Res. 2017; 27: 147-53.