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Vestibulocochlear Symptoms Caused by Vertebral Arter Compression at the Cerebellopontine Angle: Is There Any Relationship?

Serebellopontin Açıda Vertebral Arter Kompresyonuna Bağlı Olası Vestibulokoklear Semptomlar: Aralarında Bir İlişki Var Mı?

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ÖZ

Amaç: Serebellopontin açıda (SPA) anormal lokalize vertebral arterin vasküler kompresyonu radyolojik olarak tespit edilen hastalarda vestibülokoklear semptomlar ve odyolojik bulgularla olan ilişkisini değerlendirmektir.
Araçlar ve Yöntem: Manyetik rözanansgörüntüleme (MRG) sekizinci sinirde açılanmış veya lateralize olmuş vertebral artere bağlı tespit edilen vasküler kompresyon görüntüsüne sahip hastalar dahil edilmiştir. Görüntülemeye göre vasküler kompresyonun derecesi radyolog tarafından not edilmiştir. Bu hastaların hastane kayıtları ise bir kulak burun boğaz uzmanı tarafından bağımsız olarak incelenmiştir. Hastaların tinnitus, işitme azlığı, başdönmesi şikayetlerinin yanısıra saf ses odyometride işitme eşikleri (Pure tone average, PTA), konuşmayı ayırt etme skorları (speech discrimination, SD) ve işitsel beyinsapı cevaplarından (auditory brain stem response, ABR) V.dalga mutlak latans ve I-V arası latans değerleri not edilmiştir.
Bulgular: 28 MRG kaydında vertebral arter ile 8. kranial sinir kompleksi arası yakın ilişki not edilmiştir. Damarın pozisyonu incelendiğinde; 5 hastada nokta pozisyon; 17 hastada yatay olarak temasta; 6 hastada sinir kompleksi üzerinde sarmal yapmış olarak değerlendirilmiştir. Bu 3 grupta vertebral arterin pozisyonu ile ipsilateral vestibülokoklear semptomlar arasında bir ilişkiye rastlanmamıştır. Gruplarda kompresyonun olduğu ipsilateral PTA, SD, ABR sonuçlar daha anormal tespit edilmekle beraber istatistiksel olarak bir anlam bulunmamıştır.
Sonuç: Bu çalışmanın bulguları, vertebral arterin vasküler kompresyonu ile kokleavestibüler semptom ve odyolojik bulgular arasında anlamlı bir ilişki olmadığını göstermektedir. Nedeni klinik olarak açıklanamayan işitme kaybı, baş dönmesi ve tinnitus şikâyeti olan hastalarda, vertebral arterin serebellopontin açıda kompresyonunun neden olduğuna karar verebilmek ve dekompresyon cerrahisinin gerekliliğini tartışabilmek ancak daha geniş hasta serileri ile mümkün olabilecektir.

Anahtar Kelimeler: işitme; MR görüntüleme; tinnitus; vasküler kompresyon; vertebral arter

ABSTRACT

Purpose: To evaluate the vestibulocochlear symptoms and audiological findings in patients with radiologically detected vascular compression of the abnormally localized vertebral artery at the cerebellopontine angle.
Materials and Methods: Patients with magnetic resonance imaging (MRI) with an image of vascular compression due to an angulated or lateralized vertebral artery included. The degree of vascular compression based on imaging was noted by the radiologist. The patients' medical records were independently reviewed and the presence of tinnitus, hearing loss, vertigo and the results of mean hearing thresholds on pure tone average (PTA), speech discrimination (SD) mean scores, V latency, and I-V interpeak latency on auditory brainstem responses (ABR) were noted.
Results: The close relationship was detected in 28 MR scans. The degree of compression was categorized as point compression on the nerve in 5 patients, longitudinal compression on the nerve in 17, and contact as vascular loop or indentation in the nerve in 6. There were no differences between vascular compression of vertebral artery and ipsilateral symptom patterns in all groups. In the groups, ipsilateral results of PTA, SD and ABR were found abnormal compared with contralateral results but failed to show statistical significance.
Conclusion: The results suggest that there was no significant clinical value of vascular compression of the vertebral artery on the cochleovestibular nerve. In patients with unexplained hearing loss, dizziness and tinnitus complaints, deciding on the cause of compression of the vertebral artery at the cerebellopontine angle and discussing the necessity of decompression surgery will only be possible with larger patient series.

Key Words: hearing; MRI; tinnitus; vascular compression; vertebral artery

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INTRODUCTION

Vascular cross compression is a clinical entity characterized by compression of various cranial nerves by vascular structures.[1]This entity has long been thought to be a cause of many clinical findings such as trigeminal neuralgia, hemifacial spasm, tinnitus, hearing loss, vertigo, hypertension, and neurological findings.[2-5]Trigeminal neuralgia was first explained with and attributed to neurovascular compression as it was due to the compression of the Vth cranial nerve by the superior cerebellar artery.[6] From that time, many clinical conditions have been described, and microvascular decompression surgery has become popular in these situations.[1]

Many studies have been performed retrospectively or prospectively to evaluate the relationship between unilateral audiological symptoms and the vascular compression of vessels, particularly the anterior inferior cerebellar artery (AICA).[7-10] Most of the studies about AICA showed that compression of this artery could not be responsible for unilateral audiological symptoms and should be considered as a normal variant on MRI.[8,10]

With the advent of magnetic resonance imaging, visualization of the anatomy of cerebellopontine angle (CPA) and evaluation of the relationship of the neurovascular structures in this area have been accurately performed.[8,9]Besides AICA, posterior inferior cerebellar artery (PICA) and vertebral artery (VA) compression to CPA have also been defined.[4,5]Among these arteries, it is well known that VA has the greatest diameter, and the compression by VA may result in more serious findings.[6]

The compression of medulla oblongata and lower cranial nerves by vertebral artery has been well recognized as a cause of many clinical findings.[2,4,5] Abnormally lateralized, enlarged, or tortuous vertebral artery can also compress to cerebellopontine angleand cause abnormal unilateral audiological findings.[2,11] This entity was called as cochlear vertebral entrapment syndrome by Liu CH and et al.[4]

Three-dimensional T2-weighted MRI (3D-T2WI-MRI) with high resolution and thin slice for imaging of the inner ear and CPA have been shown to be highly valuable in the detailed visualization of vascular loops.[12] In this study, this superior imaging technique was used, and detailed and accurate information about vascular compression of the vertebral artery was obtained.

The aims of this study were to evaluate clinical symptoms and audiological findings in patients with abnormally localized vertebrobasilar system, especially vertebral artery, as a source of vascular compression at cerebellopontine angle and to assess the relationship of this entity with ipsilateral cochleovestibular symptoms with the help of thin slice 3D-T2WI-MR Images.

MATERIALS and METHODS

This retrospective study involved the patients who have been referred from otorhinolaryngology department for MRI scanning to exclude cerebellopontine angle pathology because of asymmetric audiological symptoms and/or signs between January 2016 and December 2018. All the authors have been concerned in accordance with the principles of the Helsinki declaration. The study was approved by Institutional Review Board and supported by University Research Fund (Project No: KA 16/310).

Radiological Assessment

MR images were obtained with the imaging strategy of Three-dimensional thin slice T2-weighted MRI (Siemens®, 3 Tesla, Magnetom Skyra, Germany). Axial contiguous slices 1.0 mm in thickness were used to examine the posterior fossa and cerebellopontine angle. All MRI studies of the CPA and internal auditory canal were re-examined by one consultant radiologist, and those that demonstrate vascular compression by vertebral artery were included in the study. The relationship between vertebral artery and cochleovestibular nerve on MRI was named as type 1; point compression, type 2; longitudinal compression, type 3; loop compression and indentation (Table 1).[8]

Audiological Assessment

Clinical records of the patients were independently reviewed by an otolaryngologist. Age, sex and the presence of hearing loss, unilateral tinnitus, vertigo, and presence

of nystagmus and other symptoms and signs of compression like hemifacial spasm were determined from patient histories and hospital charts. Mean hearing thresholds pure tone average (PTA), speech discrimination (SD) mean scores,and wave V latency and I-V interpeak latency on auditory brain stem responses (ABR) were additionally noted.

Table 1. Radiological classification of MR imaging of the vertebral artery at CPA[8]

Vessel at CPA	Classification
Type 1	Present (not on nerve)
Type 2	In contact with nerve
Type 3	On the nerve as a loop

Pure tone Audiometry (Interacoustics AC 40 ®, Denmark) was applied in the quiet rooms with the standard of Industrial Acoustic Company (IAC). The mean of speech frequency between 0-20 dB, and the speech discrimination score with 92% were accepted normal. Asymmetrical sensorineural hearing loss was defined as an interaural difference of 20 dB or more at two or three consecutive frequencies.

Auditory brainstem responses were recorded using EP 15/EP 25 Operation Manual ABR System (Interacoustics®, Assens, Denmark). Stimuli were delivered monaurally through electrodes placed on each earlobe, and responses were obtained from electrodes at the vertex (active) and at mastoid (ground). For best results impedance of each electrode was kept as 3 kOhm. The responses from electrodes were amplified by a preamplifier using 50-3000 Hz bandpass filter. Click stimuli were used with a duration of 0.10 msn and a rate of 20 per second Abnormal findings in ABR were considered if I-V interpeak latencies were greater than 4.2 msn (± 0.2 msn SD) and wave V latency value was greater than 6 msn.[13]

In the patients with vertigo,the presence of spontaneous nystagmus was noted and only the results of post-head shaking test were determined. The clinical and radiological findings were analyzed to establish whether the presence of vascular loops and contact with vertebral artery to the nerves was associated with ipsilateral audiological symptoms and/or signs.

Statistical Analysis

Statistical Package for Social Sciences program (SPSS 17.0, Chicago, IL) was used for statistical analysis. The chi-squared test was used with Fisher's exact test to test any association between ipsilateral auditory symptoms and the presence of each type of loop. A p-value of<0.05 was accepted as significant.

RESULTS

During a period of about 3 years, 930 non-tumors MRIs of cerebellopontine angle were obtained from the patients with unilateral audiological symptoms and/or signs. Among them, abnormal localized and/or dilated vertebral arteryand compression at CPA specifically was present in 28 scans (3.01 %). This relationship between vertebral artery and cochleovestibular nerve in MRI was present on the left in 18 patients (64.3 %) and on the right in 10 patients (35.7 %) (Table 2).

The vertebral artery contact with the cerebellopontine angle as "point compression" was identified in 5 patients and classified as type 1 (17.8%) (Figure 1); type 2 as a "longitudinal compression" was present in 17 patients (60.7%) (Figure 2),and type 3 as a "vascular loop on nerve" and/ or "indentation in the nerve"was present in 6 patients (21.5%) (Figure 3).

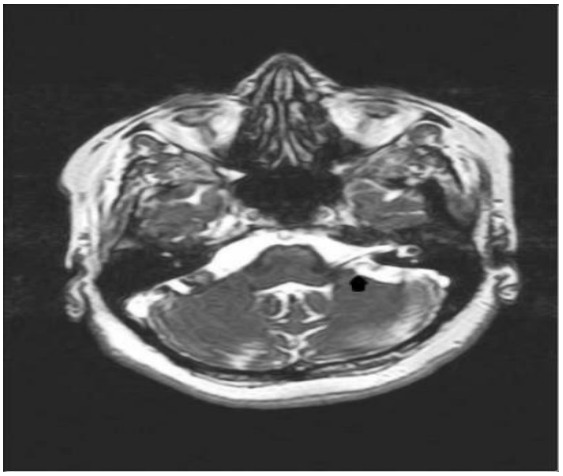

Figure 1. The vertebral artery (black arrow) and cerebellopontine angle in the relationship as type 1.

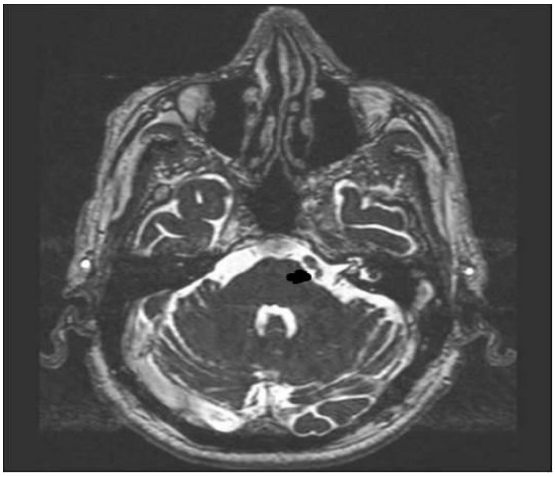

Figure 2. The vertebral artery (black arrow) near and contact with cerebellopontine angle as type 2.

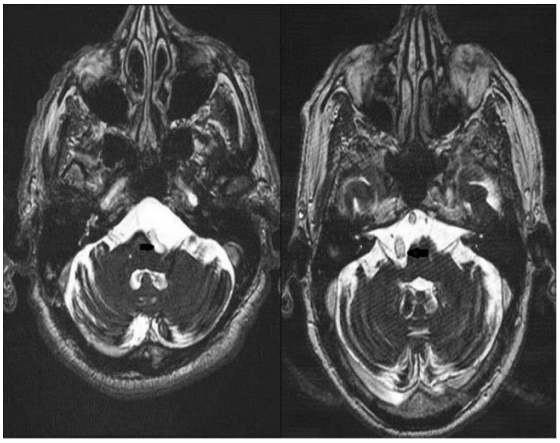

Figure 3. The vertebral artery (black arrow) contact and press the cerebellopontine angle in the area of cochlea as type 3 in two different patients.

There were 14 men and 14 women,with ages ranging from 31 to 86 (mean 55.8±14.6) years. There was no relationship between age or sex of patients and presence of compression of vessel to the nerves or any symptoms. Tinnitus was present in 23 patients (82.1%) and hearing loss was present in 20 patients (71.4%). The patients who complained about vertigo were 11 in number (39.3%), and one of them was brought to medical attention. Besides two patients with hemifacial spasms in group 3, there were not found any findings of trigeminal neuralgia, hemifacial spasm, motor deficit or any lower cranial nerves deficit in other patients.

There was no significant association between ipsilateral tinnitus and the presence and/or compression of the vertebral artery on the eighth nerve ($p>0.05$). Also, there was not found any relationship between vertigo and vascular compression.

Audiological and Vestibular Results

In the type 1 group (n=5), the mean value of ipsilateral pure tone average (PTA) was 23 dB and contralateral PTA was 30.8 dB. The mean value of speech discrimination (SD) in ipsilateral and contralateral ear with lesion were 93.6% and 91.2 %, respectively. In this group, ABR results of 4 patients were present with normal values of wave V latency and I-V interpeak latency. There was not any correlation between audiological results and the presence of vertebral artery near the nerve (in all $p>0.05$). 2 of 5 patients had complaints of vertigo. However, spontaneous nystagmus or post-head shaking nystagmus was not present.

In the type 2 group (n=17), the mean value of ipsilateral and contralateral PTA were 28.3 dB and 23 dB, respectively. The mean value of SD scores was 80% for ipsilateral ear and 90.9 % for contralateral ear. When ABR results of the patients with type 2 were regarding, wave V latency and I-V interpeak latency values were again in normal limits. Although SD score mean value at the lesion side was lower than the contralateral one, there was no significance ($p=0.316$). In this group, 5 patients had vertigo, and only one patient had spontaneous nystagmus and post-head shaking nystagmus.

In the type 3 group (n=6), the mean value of PTA in both ears was 37.4 dB ipsilaterally and 27.6 dB contralaterally. The mean value of SD scores was 81.6 % and 84 % for ipsilateral and contralateral ears, respectively, and the result of ipsilateral ear was again lower than contralateral ear, and also, mean values of other groups. For ipsilateral ear, I-V interpeak latency value was 4.4 msn and higher than 4.2 msn. However, these results were not found statistically significant.Vertigo was present in 4 of 6 patients (%33.3). Although the symptom of vertigo was seen as more common, post-head shaking nistagmus was seen in only one patient.

All groups were compared with each other for all audiological findings. However, there was not found any significance ($p>0.05$). Audiological results of all groups are summarized in Table 3.

Table 2. Results of the patients with the vascular cross compression of the cochleovestibular nerve by the vertebral artery.

Types	No of Patients	R/L	Age (mean± SD)	F/M	Tinnitus	Vertigo	Hearing loss
Type 1	5(17.8%)	2/3	54.4±14.2	4/1	5	2	3
Type 2	17(60.7%)	3/14	55.5±13.8	7/10	13	5	12
Type 3	6(21.5%)	5/1	57.8±14.5	3/3	5	4	5
Total	28	10/18	55.8±14.6	14/14	23(82.1%)	11(39.3%)	20(71.4%)
p		0.380*	0.456**	0.374*	0.525*	0.329*	0.399*

R: right, L: left, F: female, M: male Differences not statistically significant (p>0.05, *Chi square test, **Student's t-test)

Table 3. Audiological results of the patients with the vascular cross compression of the cochleovestibular nerve by the vertebral artery.

Types	Ipsi PTA (dB)	Contra PTA (dB)	Ipsi SD (%)	Contra SD (%)	Ipsi I-V latency (msn)	Contra I-V latency (msn)	Ipsi V latency (msn)	Contra V latency (msn)
Type 1	23	30.8	93.6	91.2	4.12	4.07	5.59	5.43
Type 2	28.3	23	80	90,9	4.18	4.12	5.47	5.40
Type 3	37.4	27.6	81.6	84	4.40	4.16	5.59	5.42
p	0.368	0.344	0.316	0.311	0.316	0.368	0.356	0.318

(Ipsi: ipsilateral, Contra: contralateral, PTA: pure tone average, SD: speech discrimination, dB: decibel, msn: millisecond, mean values) Differences not statistically significant (p>0.05, Kruskal Wallis Test)

DISCUSSION

The knowledge of anatomy and variations of vertebrobasilar system such as the fenestration, duplication, dilatation, dolichoectasia, and tortuous or abnormally localization may explain many symptoms and signs about neurology and neuro-otology. The anatomy of vertebrobasilar system is diffused and contained many variations. Vertebral artery is a branch of subclavian artery and is combined with the opposite vertebral artery to form basilar artery. The first branch of basilar artery is anteriorinferior cerebellar artery and the end branch of basilar artery is labyrinthine artery.[14]

In regard of neurotology, many symptoms about hearing and/or balance may be accused of AICA compression of cochleovestibular nerve in the literature.[9,15,16] Compression of the medulla and cerebellopontine angle by dilated and /or tortuous vertebral artery is less known.[2] Liu CH et al.[4]reported a case of vertebral dolichoectasia as a cause of asymmetrical sensorineural hearing loss and named this condition as cochlear entrapment syndrome. Huh G. et al.[2]also claimed that the vertebral arteries could be angulated, dilated, or tortuous but not necessarily dolicoectatic to cause obvious indentation.

As AICA, vertebral artery is similarly located at posterior fossa and, actually, it has a greater diameter and higher blood flow rate than AICA.[6]The average outer diameter of the VA was found to be 2.8 mm on the left side and 2.35 mm on the right side while the diameter of AICA was reported to be 0.9 mm approximately. The distance between the VA and the medulla oblongata was presented to have a mean value of 3 mm.[6] Within the cervicocranial arteries, dilatative arteriopathy is usually seen in the vertebrobasilar system.[2] Thus, it is possible to suggest that VA may cause more pressure on or around the nerve than AICA, theoretically. Indeed, compression caused by VA may result in various neurological symptoms together with neurotological symptoms, as reported before.[2,5,11]

Many hypotheses have been postulated to explain the occurrence of vascular compression symptoms regarding the proximity of a blood vessel to VIIIth cranial nerve. The older theory was that the excitation and local demyelination in the root entry/exit zone (REZ) where the nerve has no glial sheet might be the cause of the symptoms.[3,16] This hypothesis is not much applicable because the central segment of VIIIth cranial nerve is more vulnerable and is more likely to face with vascular compression than its REZ or more peripheral segments.[17]

Another theory has been proposed by Applebaum and Valvassori that neurovascular cross compression of the VIIIth cranial nerve resulted in impaired blood flow through the vascular loop, reducing the perfusion of cochlea and vestibular systems.[18] However, this theory was not capable of explaining other cranial nerve deficits which might be seen together with cochleovestibular symptoms, such as hemifacial spasm.[19]

Huh G. et al.[2] said that the anterior vestibular artery, which supplies the labyrinth, is particularly vulnerable to ischemia because it lacks collateral vessels. Thus, ischemic injury at a specific location induced by vertebrobasilar compression may trigger fluctuating, aggravating, or delayed audiovestibular symptoms. This reduced blood flow could also cause thrombosis within dilated segments and obstruction of penetrating branches.[20]

Schwaber and Whetsell[21] have claimed that the symptoms seen in vascular compression were developed as a result of diffuse neuritis. According to these authors, neuritis results in swelling of the nerve and adherence of the nerve to the vessel, which, in turn, results in an image of vascular loop as a coincidental finding. This theory could actually explain that the interaction between vessel and nerve might not always cause a pathological event.[18]

In our study, the incidence of the vascular compression due to the variations of vertebral artery was 3 %. This finding was obtained from a retrospective analysis of MR scans of CPA. When the vertebral artery compressed only a limited portion of the cochleovestibular nerve (type 1) or approached the cochleovestibular nerve as both traverse parallel to each other (type 2), the radiological findings were not correlated with unilateral neurotological symptoms or audiological findings. However, when the vertebral artery compressed the cochleovestibular nerve as large loop or as to make an indentation (type 3), the speech discrimination scores were found to be lower, and the value of I-V interpeak latency in ABR was obtained longer than others. However, these results did not show statistical significance, and these audiological results showed that this relationship between the cochleovestibular nerve and vertebral artery might be incidental. Unfortunately, the number of cases in this study was not enough to prove this conclusion.

In this study, the age average of the patients in each group is well above 50 years, meaning that these patients may belong to the presbycusis population. Although many subgroups of presbycusis are present, now it is generally accepted that presbycusis is associated with age-related declines in spectral and temporal resolving power of the auditory system, and in this regard, presbycusis is also retrocochlear pathology as well as a cochlear disease.[22] In our study, in group 3; the longer value of I-V interpeak latency in ABR was detected and accepted usually as a sign of retrocochlear pathologies.[13]As a result, audiological findings in this study could not be due to vascular cross compression of the vertebral artery. However, cases of our study are too few to say these results were due to presbyacusis, not due to vascular compression.

Vertigo was detected in 11 patients in this study and one of them had taken medical attention. A Full battery of video electronystagmograghy (VNG) unfortunately could not be recorded in all patients. Two of the patients with vertigo (one had severe vertigo) had post-head shaking nystagmus with the fast phase beating toward the intact side. Although the absence of VNG test was the weakness of this study, it was lighted for new studies about the relationship between the vascular compression on cochleovestibular nerve and VNG or vestibular evoked myogenic potential (VEMP).

The other symptom of vascular compression on cochleovestibular nerve is tinnitus.[17] Tinnitus is the most important symptom in the judgement of decompression surgery for vascular cross compression.[1,23] In the literature, in selected cases, microvascular decompression could cure non-pulsatile tinnitus.[17,23] The pathophysiology of tinnitus in the vascular compression could be explained by the alteration of nerve conduction in cochlear nerve or the result of audio-frequency vibrations transferred via perineural spaces to the cochlea.[3,17]

In this study, non-pulsatile tinnitus was present in 23 patients. However, the association between left or right tinnitus and the presence of any type of vascular compression was not found to be statistically significant ($p>0.05$).

Microvascular decompression surgery was first applied by Janetta, and laterby Magnan for a variety of clinical conditions like hemifacial spasm, trigeminal neuralgia, vertigo, and tinnitus.[1] For the treatment of vascular compression on cochleovestibular nerve, some authors suggested vascular decompression surgery as a successful treatment modality.[12,19,24] However, van den Berge MJC. et al.[25]have a conclusion that for microvascular decom-

pression, further validation is necessary to evaluate whether patients with combined symptoms due to the low level of evidence in their systemic review. Grasso et al.[6]claimed that perforating branches of vertebral artery could be damaged when repositioning or decompressing the vertebral artery. Schwaber and Whitsell.[21]supposed that the success of decompression surgeries might be due to a placebo effect. Nusbaum et al.[26] presented 72 years old woman with large vertebral artery aneurysm causing hearing loss and tinnitus and successfully treated symptoms of patients with surgical clip occlusion. They claimed that it is possible to cure sensorineural hearing loss and tinnitus with the management of vertebral artery pathologies in case of compression on cranial nerve VII.[26] However, Huh G et al.[2]concluded in their four case series with vertebrobasilar Dolichoectasia (VBD) that a thorough neurotological evaluation is recommended prior to treatment of VBD, and medical treatment should be firstly preferred before the surgical vascular compression.

The study of Huh G et al.[2] consisted of only four cases of vertebral artery compression. These cases were investigated with pure tone audiometry, videonystagmography, the head impulse test and MRI/MR Angiography. However, in our study, we had 28 patients with vertebral artery compression, and we also used auditory brainstem response to evaluate the hearing status of patients. We routinely have used ABR in our clinic to differentiate retrocochlear and cochlear pathologies in case of asymmetric hearing loss at PTA. If I-V inter-peak latency in ABR is longer, then we thought that retrocochlear pathology with the combining information from MRI. Unfortunately, we could not have MR Angiography unlike the study of Huh G et al.[2]

Our study has many limitations such as retrospective design, the limited number of cases and the absent of vestibular tests, particularly caloric test and the video head impulse test. However, this study was a blind study that the radiologist and the otolaryngologist have not known the patients' other signs and the different otolaryngologist has done the data analysis. This might actually be preventing the biases.This comparative study is one of the rare studies evaluating neurootologic symptoms due to vertebral artery compression. In terms of its results, our study revealed the necessity of a more detailed evaluation before making a surgical decision, as stated by Huh G in his study.

In conclusion, although the compression of the vertebral artery on the VIII. cranial nerve is well demonstrated on MRI, the clinical value of this condition is still of debate. The results of this study, however, have shown that there is no statistically significant correlation between neurootological symptoms and radiologically detected nerve compression by vertebral artery. Thus, the physician should be careful before giving the decision of decompression surgery for unexplained hearing loss, vertigo and tinnitus. However, this issue merits further neurootological investigation, and our results may need to be confirmed with more advanced studies with larger patient groups.

Conflict of Interest

The authors declare that there is not any conflict of interest regarding the publication of this manuscript.

Authors' Contributions

Concept/Design: FÖ. Data Collection and/or Processing: FÖ, ÖA. Data analysis and interpretation: CÖ. Literature Search: FÖ. Drafting manuscript: FÖ. Critical revision of manuscript: CÖ, ÖA, HY. Supervision: ÖA, HY.

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