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Is there a relationship between basilar artery tortuosity and vertigo?

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ABSTRACT

Objective: The aim of this study was to investigate the relationship between basilar artery (BA) tortuosity, hypogenesis/agenesis of the vertebral artery (VA), and vertigo, with the use of magnetic resonance imaging (MRI).
Patients and methods: This case-control study included patients admitted to the outpatient clinics, who were aged 18–80 years, without any known systemic diseases. All patients were evaluated with a 1.5-tesla MRI system. BA Tortuosity, VA agenesis, and VA asymmetry were noted. BA diameter (central) and length (longitudinal) were measured.
Results: A total of 154 vertigo patients (46 M, 108 F; mean age of 48.95 ± 17.3 years) and 346 control subjects (112 M, 234 F; mean age of 45.12 ± 17.0 years) were included. The mean age of the vertigo patients was significantly higher than that of the control group (48.95 vs 45.12 years) ($p = 0.021$). The rate of BA tortuosity was higher in patients with vertigo ($p = 0.030$). When the participants were divided into two groups according to median age (< 45 vs. ≥45 years) there was no statistically significant difference between the groups in terms of VA asymmetry ($p = 0.070$) and hypogenesis/agenesis ($p = 0.577$). There was a statistically significant difference between the groups in respect of BA tortuosity ($p = 0.033$), BA diameter ($p < 0.001$), and BA length ($p < 0.001$). When the study populations were divided into two groups according to the presence of vascular tortuosity, the mean age, BA diameter, and BA length values were higher in the tortuosity (+) group (all $p < 0.001$).
Conclusion: These results demonstrated that vertigo and BA tortuosity rates seem to increase with age. Likewise, BA diameter and length increased with age, although there was no significant relationship with vertigo. Patients with tortuosity were significantly older, and had higher rates of VA asymmetry/agenesis, and increased BA diameter compared to subjects without tortuosity.

1. Introduction

The illusory sensation of movement, known as vertigo, can occur due to dysfunction of the vestibular system. Vertigo is a widespread problem, and may be accompanied by several comorbidities such as hypertension, cerebrovascular accident, dyslipidemia, or cardiovascular diseases [1]. Lesions or variations of the circulation may result in vertigo, particularly in elderly patients, due to the complex network of arteries in the posterior circulation of the cerebral cortex [2,3]. The anatomical structures of the posterior circular system comprise the anterior inferior cerebellar artery, posterior cerebral artery, superior cerebellar artery, and labyrinthine arteries. Some of the small branches separate from the BA, and these provide the blood supply of the pons, which includes the vestibular nucleus. If the vertebrobasilar artery is increased in length or diameter, this is defined as dolichoectasia, and the associated angulation as tortuosity [3].

Previous studies have emphasized that in cases of dolichoectasia, with angulation, dilatation, and elongation of vertebral and basilar arteries (BA) due to a decreased blood supply, vertigo could be observed. As the vertebrobasilar system provides the blood supply and maintains the hemodynamics of the vestibular nuclei and connections, alterations to the hemodynamics can lead to vertigo [3]. Dominance of the vertebral artery (VA) can also result in vertigo as the arterial blood supply of the BA is affected, thus creating a higher risk of vascular damage. When blood supply is decreased, oxygen, energy, or glucose for the neuronal network associated with the vestibular structures will be limited. If sufficient collateral arteries do not develop over time, vertigo attacks occur [4]. The tortuosity of cerebral arteries is also

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frequently observed. Associated with insufficient blood supply to the brain, there can be vascular abnormalities and tortuosity or hypogenesis/agenesis in the vertebrobasilar artery, which then cause clinical symptoms including vertigo, dizziness, and nystagmus. Although mild tortuosity is generally asymptomatic, severe tortuosity can lead to vertigo [5,6].

There are two main forms of vertigo. Central vertigo refers to the vertigo caused by a disease originating from the central nervous system such as brain stem or cerebellum infarct/hemorrhage, vertebrobasilar insufficiency, space occupying lesions, multiple sclerosis, vestibular migraine, and Chiari malformation [7]. Benign paroxysmal positional vertigo, Meniere's disease, vestibular neuritis, labyrinthitis and otitis media are among the most common causes of peripheral vertigo [8]. Peripheral vertigo is usually accompanied by symptoms such as hearing loss, ringing in the ear and it is generally acute, severe and episodic. Central vertigo typically occurs over a longer period of time and it is less severe. Peripheral vertigo can often be worsened by head movements, and the resulting nystagmus is typically associated with fatigue and horiontal nystagmus [7,8].

The compressive symptoms in dolichoectasia, elongation and distension, ensue due to pressure of the enlarged vessels to the brain stem and generally occur along the anterolateral surface [9]. Brainstem compression is a rare manifestation among dolichoectasia patients and it is asymptomatic in many patients. Compression shows slow progression and the brainstem may tolerate gradually without clinical signs [9,10]. If the corticospinal tracts in the lateral medulla are affected, contralateral or ipsilateral findings can be seen on the rostrocaudal level of compression. Vertigo, dysphagia, imbalance, and tinnitus can be caused due to the compression of lower parts of the brainstem or nucleus ambiguus [9–11].

Although the etiopathogenesis of vertebrobasilar artery tortuosity has not yet been fully clarified, connective tissue disorders, reduced elasticity, degeneration of blood vessels, and vascular wall shear stress are thought to be related to tortuosity [12]. Therefore, magnetic resonance imaging (MRI) can be used as a non-invasive and useful imaging method for the visualization and examination of the vertebrobasilar artery [13]. The determination of the relationship between vertigo and other variations, such as tortuosity and VA agenesis, may contribute to the clinical evaluation of vertigo. Therefore, the aim of this study was to utilize MRI to investigate the relationship between basilar artery tortuosity, hypogenesis/agenesis of the vertebrobasilar system, and vertigo.

2. Material and methods

2.1. Study design and participants

This cross-sectional and case control study was conducted retrospectively, between January 2016 and July 2017 at Kırıkkale University Medical School. MRIs of the patients were screened from the digital radiology database. The study included patients admitted to the outpatient clinics of neurology, otorhinolaryngology, neurosurgery and internal medicine, who were aged 18–80 years, without any known systemic diseases, any medication, or any evidence to explain vertigo after the etiological examinations. All patients were evaluated with MRI. Patients with cerebrovascular trauma, intracranial mass, carotid stenosis, migraine, multiple sclerosis, benign positional paroxysmal vertigo, vestibular neuritis, Meniere's disease, and metabolic disorders were excluded. Patients with dolichoectasia (enlargement > 4.5 mm) were also excluded.

The current study protocol was approved by the Local Ethics Committee (Date: 20.06.2017, Number: 16/04).

2.2. Magnetic resonance imaging

All patients were evaluated with a 1.5-tesla MRI system (Philips MRI Systems, Achieva Release 3.2 Level 2013-10-21, Philips Medical) A standard head and neck coil was used during the examination. Data were obtained from fat-suppressed T2-weighted images in the coronal plane (TR msn/TE msn; 6128/100, FOV 200 × 182 mm and matrix 224 × 165 mm). A total of 30 coronal sections were obtained using a 5-mm slice thickness and 1-mm intersection gap. Tortuosity, VA agenesis or hypogenesis, and VA asymmetry were noted. The BA diameter (central) and length (longitudinal) were also measured (Fig. 1).

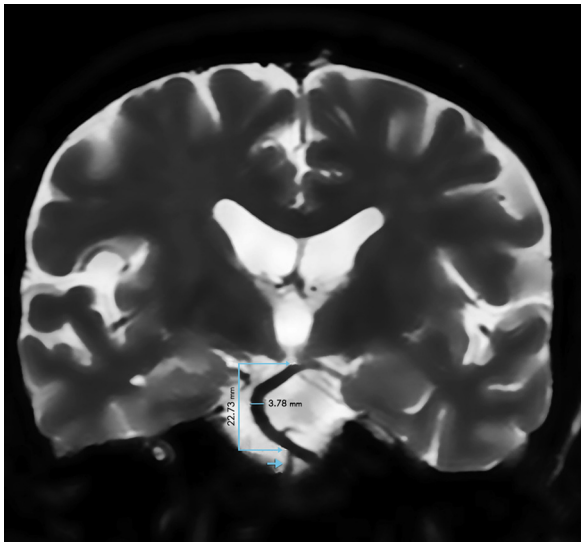

Fig. 1. 56-year-old man patient with complaints of vertigo.
On coronal T2A MRI image, basilar artery (BA) tortuosity and right hypoplasic vertebral artery (arrow head) is seen. BA diameter was measured from midpons level (3.78 mm). BA length (22.73 mm) was measured a linear distance from the confluence point of the bilateral vertebral arteries to the initial point of division of the BA into bilateral posterior cerebral arteries (distance between horizontal arrows).

2.3. Statistical analysis

SPSS 21.0 for Windows software (SPSS Inc., Chicago, IL, USA) program was used for statistical analyses. The continuous variables were stated as mean ± standard deviation (SD), and categorical variables were stated as number (n) or percentage (%). After checking the normal distribution, between-group comparisons were made by the Student's *t*-test for continuous variables. The Chi-square test was used to compare categorical variables between the groups. A value of $p < 0.05$ was accepted as statistically significant.

3. Results

Evaluation was made of a total of 154 vertigo patients (46 M, 108 F) with a mean age of 48.95 ± 17.3 years and 346 control subjects (112 M, 234 F) with a mean age of 45.12 ± 17.0 years. The clinical and demographic features of the subjects are shown in Table 1. There was no statistically significant difference between the groups in respect of the BA diameter, BA length, VA hypogenesis/agenesis, and VA asymmetry (all $p > 0.05$). The mean age of the vertigo patients was significantly higher than that of the control group (48.95 vs 45.12 years) ($p = 0.021$). There was a statistically significant difference in the presence of BA tortuosity between the groups ($p = 0.030$). The rate of BA tortuosity was higher in patients with vertigo. When the participants were divided into two groups according to median age (< 45 vs. ≥ 45 years), there was no statistically significant difference between the groups in terms of VA asymmetry ($p = 0.070$) and hypogenesis/agenesis ($p = 0.577$). There was a statistically significant difference regarding vertigo occurrence between the groups ($p = 0.033$). The rate of

Table 1
Clinical and demographical features of the patients.

Variables	Vertigo + (N = 154)	Vertigo – (N = 346)	P value
Age (yr)	48.95 ± 17.3	45.12 ± 17.0	**0.021**
Gender	46 (29.9)	112 (32.4)	0.328
Male	108 (70.1)	234 (67.6)	
Female			
BA Diameter (Central)	2.38 ± 0.53	2.38 ± 0.52	0.186
BA Length (Perpendicular)	27.03 ± 3.5	26.9 ± 3.5	0.791
BA Tortuosity	77 (50)	140 (40.5)	**0.030**
Yes	77 (50)	206 (59.5)	
No			
VA Hypogenesis/Agenesis	16 (10.4)	38 (10.1)	0.490
Yes	138 (89.6)	308 (89.0)	
No			
VA Asymmetry	62 (40.3)	129 (37.3)	0.296
Yes	92 (59.7)	217 (62.7)	
No			

BA, Basilar artery**; VA,** vertebral artery.
*Bold p values denote significance.

Table 2
Clinical data according to the median of the age.

Variables	Age < 45.5 (N = 250)	Age > 45.5 (N = 250)	P value
Vertigo	67 (26.8)	87 (34.8)	**0.033**
Yes	183 (73.2)	163 (65.2)	
No			
BA Diameter (Central)	2.20 ± 0.44	2.47 ± 0.57	< **0.001**
BA Length (Perpendicular)	26.42 ± 3.14	27.52 ± 3.87	< **0.001**
BA Tortuosity	89 (35.6)	128 (51.2)	< **0.001**
Yes	161 (64.4)	122 (48.8)	
No			
VA Hypogenesis/Agenesis	27 (10.8)	27 (10.8)	0.577
Yes	223 (89.2)	223 (89.2)	
No			
VA Asymmetry	87 (34.8)	104 (41.6)	0.070
Yes	163 (65.2)	146 (58.4)	
No			

BA, Basilar artery**; VA,** vertebral artery.
*Bold p values denote significance.

Table 3
Basilar artery tortuosity and related factors.

Variables	Tortuosity + (N = 217)	Tortuosity – (N = 283)	P value
Age (yr)	49.05 ± 17.0	44.19 ± 17.0	**0.002**
BA Diameter (Central)	2.42 ± 0.57	2.26 ± 0.48	**0.003**
BA Length (Perpendicular)	27.51 ± 3.7	26.56 ± 3.3	< **0.001**
VA Hypogenesis/Agenesis	39 (18.0)	15 (5.3)	< **0.001**
Yes	178 (82.0)	268 (94.7)	
No			
VA Asymmetry	133 (61.3)	58 (20.5)	< **0.001**
Yes	84 (38.7)	225 (79.5)	
No			

BA, Basilar artery**; VA,** vertebral artery.
*Bold p values denote significance.

vertigo (34.8% vs. 26.8%) was significantly higher in patients aged 45 years and older. There was a statistically significant difference between the groups in respect of BA tortuosity ($p = 0.033$), BA diameter ($p < 0.001$), and BA length ($p < 0.001$). BA diameter and length were seen to increase with age. The clinical data according to the age groups are given in Table 2.When the study populations were divided into two groups according to the vascular tortuosity, there was a statistically significant difference between the groups in respect of age ($p = 0.002$), BA diameter ($p = 0.003$), BA length ($p < 0.001$), VA asymmetry ($p < 0.001$), and VA hypogenesis/agenesis ($p < 0.001$). The mean age, BA diameter, and BA length was higher in the tortuosity (+) group. BA tortuosity and the related factors are summarized in Table 3. There was no statistically significant difference between the tortuosity side, VA asymmetry side, VA hypogenesis/agenesis side, and vertigo (all $p > 0.05$). There was a significant positive correlation between the age and VA diameter (central) ($r = 0.295$, $p = 0.000$) or BA length (perpendicular) ($r = 0.177$, $p = 0.000$).

4. Discussion

The aim of this study was to explore the relationship between BA tortuosity, hypogenesis/agenesis of the vertebrobasilar system, and vertigo, using MRI. According to the results, there were two main findings. First, patients with vertigo were significantly older than the control group, and the rate of BA tortuosity was higher in vertigo patients. Second, BA diameter and length, and rates of VA asymmetry or hypogenesis/agenesis were higher in the patients with tortuosity compared to patients without tortuosity. Studies in literature which have evaluated the relationship between vertigo and tortuosity have mostly been conducted on patients with cerebral ischemia. Unlike previous studies, patients with cerebral ischemia were not included in the current study. Therefore, the results of this study can be considered noteworthy in the determination of the relationship between vertigo and tortuosity.

Although some mechanisms have been previously highlighted, the etiopathogenesis leading to vertebrobasilar system tortuosity has not yet been clarified [14]. Connective tissue disorders, degenerative changes of blood vessels or decreased elasticity, and vascular wall shear stress have been reported related to tortuosity [15]. Furthermore, some vascular risk factors (e.g. hyperlipidemia/dyslipidemia, diabetes mellitus, hypertension) resulting from aggravated tortuosity are known to actively promote aging, atherosclerotic and degenerative changes of blood vessels [16]. In the present study, the presence of connective tissue disorders and cerebrovascular risk factors were not investigated. However, the rate of tortuosity and vertigo was observed to increase with age, which was consistent with the relevant data.Similarly, the relationship between VA asymmetry and vertigo has not yet been fully understood. Nevertheless, VA asymmetry is frequently seen in patients with vertigo [17]. In the present study, no significant difference was found between the groups in respect of VA asymmetry. However, previous studies have shown that VA dominancy or VA asymmetry is often determined in vertigo patients. These studies indicate that VA dominancy may be a congenital defect that has combined with an acquired predisposition to vertigo of vascular origin [5]. In a study by Hong et al. [18], the BA curvature was determined to be higher in VA dominance patients than in non-VA dominance patients. The difference between the diameters of the right and left VAs was reported to be the only independent predictor for moderate to severe BA curvature [18]. Hemodynamic changes are seen with VA dominance, and this association can be considered to lead to increased BA curvature. Thrombosis, microembolization, stretched cranial nerves, and brainstem compression may be the result of hemodynamic changes arising from BA curvature, BA tortuosity, and/or VA asymmetry or VA dominance combined with arterial elongation and enlargement [19]. In the current study, no significant relationship was found between the side of BA tortuosity, agenesis or hypogenesis of VA. Despite the increase in BA diameter and length with ageing, no correlation was determined between vertigo and BA diameter and length. Nevertheless, patients with tortuosity were older, BA diameter and BA length were significantly greater, and the rate of VA asymmetry was significantly higher in the tortuosity group.

Individuals with VA hypoplasia may have a high probability of vertigo of vascular origin, with atherosclerotic susceptibility, and ipsilateral lesions in the VA territory [20]. In general, VA asymmetry is not taken into consideration as an important finding, and it is thought to be a congenital variant if vertebrobasillar insufficiency does not occur. In contrast, recent studies have shown VA asymmetry to be a risk factor in the etiology of posterior circulation stroke [20]. These vascular anatomical structures of BA tortuosity, VA agenesis or VA dominance render the posterior circulation vulnerable to ischaemia. A wide range of central and peripheral vestibular syndromes such as vertebrobasilar insufficiency, vertigo, dizziness, nystagmus,and disequilibrium are thought to be often caused by ischaemia [21]. In the present study, an association was observed between vertigo and tortuosity in the BA. Gutierrez et al. [3] highlighted that tortuosity and hypoplasia in the vertebrobasilar arteries accelerated atherosclerosis or changed hemodynamics, thereby promoting infarctions of the posterior circulation [3].

4.1. Limitations

This study had some limitations. Although the study population size was adequate, the clinical characteristics of the participants including hypertension, diabetes mellitus, smoking status, drug use, and lipid metabolism were not questioned in detail. Second, the relationship between the severity of vertigo and vertebrobasilar arteries was not investigated. This was a retrospective study and a prospective study may include other findings attributable to basilar artery tortuosity or further posterior fossa anatomical variants. A different group comprising the patients with dolichoectasia could be formed to show whether the patients all presented with vertigo or not. The blood flow velocities in BA and VA were not determined and finally, the number of tortuosity segments in the artery (one or multiple) was not determined.

5. Conclusion

In the light of these results, it can be concluded that vertigo and BA tortuosity rates seem to increase with age. Likewise, the BA diameter and length increased with age, although there was no significant relationship with vertigo. Patients with tortuosity were significantly older, and they had higher rates of VA asymmetry/agenesis, and increased BA diameter compared to subjects without tortuosity. In conclusion, the findings of this study provide further evidence for the clinical usefulness of MRI in the determination of vertigo.

Conflict of interest

None.

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References

[1] M. von Brevern, A. Radtke, F. Lezius, M. Feldmann, T. Ziese, T. Lempert, et al., Epidemiology of benign paroxysmal positional vertigo, a population-based study, J. Neurol. Neurosurg. Psychiatry 78 (2007) 710–715.
[2] M. Karatas, Vascular vertigo: epidemiology and clinical syndromes, Neurologist 17 (2011) 1–10.
[3] J. Gutierrez, R.L. Sacco, C.B. Wright, Dolichoectasia an evolving arterial disease, Nat. Rev. Neurol. 7 (2011) 41–50.
[4] M. Cosar, M. Yaman, O. Eser, A. Songur, O.A. Ozen, Basilar artery angulation and vertigo due to the hemodynamic effect of dominant vertebral artery, Med. Hypotheses 70 (2008) 941–943.
[5] D.P. Zhang, S.L. Zhang, H.T. Zhang, S.J. Zhang, Vertebral artery dominance, brainstem auditory evoked potential, and vertigo of vascular origin, Neurol. Res. 34 (2012) 498–503.
[6] A. Dodevski, M. Lazareska, D. Tosovska-Lazarova, J. Zhivadinovik, V. Aliji, Morphological characteristics of the first part of the vertebral artery, Prilozi 32 (2011) 173–188.
[7] T.L. Tamás, T. Garai, T. Tompos, Á. Szirmai, Vertigo in the Emergency Department: new bedside tests, Orv. Hetil. 157 (March (11)) (2016) 403–409.
[8] P.M. Picciotti, D. Lucidi, E. De Corso, D. Meucci, B. Sergi, G. Paludetti, Comorbidities and recurrence of benign paroxysmal positional vertigo: personal experience, Int. J. Audiol. 55 (5) (2016) 279–284.
[9] M. Samim, A. Goldstein, J. Schindler, M.H. Johnson, Multimodality imaging of vertebrobasilar dolichoectasia: clinical presentations and imaging spectrum, Radiographics 36 (July–August (4)) (2016) 1129–1146.
[10] A. Pereira-Filho, M. Faria, C. Bleil, J.L. Kraemer, Brainstem compression syndrome caused by vertebrobasilar dolichoectasia: microvascular repositioning technique, Arq. Neuropsiquiatr. 66 (2B) (2008) 408–411.
[11] S. Passero, S. Rossi, F. Giannini, D. Nuti, Brain-stem compression in vertebrobasilar dolichoectasia: a multimodal electrophysiological study, Clin. Neurophysiol. 112 (8) (2001) 1531–1539.
[12] S.A. Morris, D.B. Orbach, T. Geva, M.N. Singh, K. Gauvreau, R.V. Lacro, Increased vertebral artery tortuosity index is associated with adverse outcomes in children and young adults with connective tissue disorders, Circulation 124 (2011) 388–396.
[13] H.S. Kim, J.H. Lee, G. Cheh, S.H. Lee, Cervical radiculopathy caused by vertebral artery loop formation: a case report and review of the literature, J. Korean Neurosurg. Soc. 48 (2010) 465–468.
[14] H.C. Han, Twisted blood vessels: symptoms, etiology and biomechanical mechanisms, J. Vasc. Res. 49 (2012) 185–197.
[15] S.H. Lee, N. Hur, S.K. Jeong, Geometric analysis and blood flow simulation of basilar artery, J. Atheroscler. Thromb. 19 (2012) 397–401.
[16] K. Ikeda, Y. Nakamura, T. Hirayama, T. Sekine, R. Nagata, O. Kano, K. Kawabe, T. Kiyozuka, M. Tamura, Y. Iwasaki, Cardiovascular risk and neuroradiological profiles in asymptomatic vertebrobasilar dolichoectasia, Cerebrovasc. Dis. 30 (2010) 23–28.
[17] G.J. Pe´rez-Carrillo, J.P. Hogg, Intracranial vascular lesions and anatomical variants all residents should know, Curr. Probl. Diagn. Radiol. 39 (2010) 91–109.
[18] J.M. Hong, C.S. Chung, O.Y. Bang, S.W. Yong, I.S. Joo, K. Huh, Vertebral artery dominance contributes to basilar artery curvature and peri-vertebrobasilar junctional infarcts, J. Neurol. Neurosurg. Psychiatry 80 (2009) 1087–1092.
[19] M. Lou, L.R. Caplan, Vertebrobasilar dilatative arteriopathy (dolichoectasia), Ann. N. Y. Acad. Sci. 1184 (2010) 121–133.
[20] J.H. Park, J.M. Kim, J.K. Roh, Hypoplastic vertebral artery: frequency and associations with ischaemic stroke territory, J. Neurol. Neurosurg. Psychiatry 78 (2007) 954–958.
[21] C. Nagashima, Surgical treatment of vertebral artery insufficiency caused by cervical spondylosis, J. Neurosurg. 32 (1970) 512–521.