

Investigation of the Relationship Between the Dynamic Properties of the Vertebrobasilar Artery and Vestibular Symptoms

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ABSTRACT

Objective: There is a large patient population presenting to the clinic with complaints of dizziness and imbalance that cannot be explained by a specific vestibular pathology (nonspecific vestibular symptoms) in routine vestibular evaluations. In the current study, it was aimed to examine the relationship between nonspecific vestibular symptoms and dynamic properties of the vertebral artery.

Methods: In this cross-sectional study involving 38 patients with nonspecific vestibular complaints and 39 healthy controls, all participants were assessed with the otoscopic examination, pure-tone audiometry, tympanometry, otoacoustic emission, vertebral artery color Doppler ultrasonography, bedside vestibular examination, videonystagmography, video head impulse tests, and functional head impulse tests.

Results: The percentage of comorbidity, presence of positional nystagmus and average air conduction hearing thresholds differed significantly between participants with vestibular complaints and healthy controls. The vertebral artery flow and velocity values were obtained lower in the group with vestibular complaints compared to the control group. The presence of spontaneous nystagmus and the total vertebral artery flow rate independent variable significantly predicted the presence of the vestibular complaint dependent variable ($\beta = -1.643$, $df = 1$, $P = .014$, $R^2 = 0.792$; $\beta = -0.026$, $df = 1$, $P = .014$, $R^2 = 0.714$)

Conclusion: Findings from this study indicate that close monitoring of the flow rate of the vertebral artery despite normal vertebral artery color Doppler ultrasonography findings in patients with nonspecific vestibular complaints may be important for the early detection of a possible vestibular pathology.

Keywords: Color Doppler ultrasonography, vertebral artery, vertigo, vestibular function tests

Introduction

The vertebrobasilar system, which supplies the brain stem, spinal cord, cerebellum, and posterior parts of the cerebrum, is an important structure, consisting of vertebral artery (VA) and carotid artery. A decrease in flow in these arteries or their branches may lead to temporary or permanent symptoms, such as vertigo, dizziness, positional nystagmus, gait disturbance, hearing loss, visual impairment, and speech impairment.¹⁻³ Although vertigo caused by VA sclerosis (VAS) is often accompanied by other neurological disturbances, isolated vertigo may also be an initial symptom of cerebellar stroke or vertebrobasilar insufficiency (VBI).⁴ In a retrospective study of 54 patients, it was reported that 12% of patients were initially afflicted with isolated vertigo.⁵ Other studies have also shown that there is a moderate prevalence of stroke (0.7%) in people with isolated vertigo, and its incidence rises as much as 3.2% in cases where vertigo and imbalance are seen together.⁶ The relationship between the dynamic properties of the VA and vestibular symptoms is complex and multifactorial. In order to identify the underlying causes of symptoms and make an accurate diagnosis, a thorough assessment of a patient's clinical history, physical examination, and diagnostic testing is necessary. The diagnosis can be further complicated by the frequency of anatomic variations of the VA and the presence of collateral circulation.⁷ Therefore, studies of anatomic variables and normal ranges of flow rate and velocity are important in the evaluation of the vertebrobasilar system and the diagnosis of its pathologies. While VAS is more commonly seen in older individuals, it can occur at any age, particularly in individuals with risk factors for the condition. Studies show conflicting data regarding the direct correlation between increased age and the development of atherosclerosis and calcification of the vertebral artery.^{8,9} There are several risk factors and comorbidities such as hypertension, diabetes mellitus, coronary artery disease, high cholesterol, cigarette

smoking, alcohol consumption, obesity, and peripheral vascular disease associated with causes of vertebral artery stenosis.¹⁰⁻¹³

On the other hand, there is a large patient population that presents to clinics with complaints of dizziness and imbalance that cannot be explained by a specific vestibular pathology in routine vestibular evaluations. The close relationship of vertebral system disorders with vestibular symptoms suggests that vestibular system variations in this group of patients should be investigated in more detail. Color Doppler ultrasonography (CDUSG), which is a noninvasive and reproducible method, is the most preferred method for evaluating the flow rate of the VA.^{14,15} The literature suggests that the range of normal VA volumes in healthy adults varies greatly. Seidel et al¹⁶ reported this range as 102.4-301 mL/min. Many studies have reported that the left VA flow rate is higher than the right.^{16,17,18} It was also reported that the minimum value of the net VA flow rate varies between 171 and 200 mL/min.^{16,19} In some cases, although the net VA flow is within normal limits, VBI symptoms may occur. Some studies also report that these symptoms were not encountered despite significant VA hypofunction, and therefore vertebrobasilar asymmetry is a normal variation.^{14,20} However, these studies had some limitations in that they consisted of healthy individuals without vestibular complaints and contained limited information about the clinical evaluation of the vestibular system. Therefore, in this study, we aim to investigate whether vertebral system anatomy, flow rate, and velocity of VA are related to nonspecific vestibular complaints in adults with nonspecific vestibular complaints who do not indicate a specific peripheral or central vestibular pathology, as well as whether these variables may result in dizziness.

Methods

This is a cross-sectional retrospective study that includes patients who were admitted to the outpatient otorhinolaryngology clinic at Istanbul Göztepe Prof. Dr. Suleyman Yalcin City Hospital/Istanbul Göztepe Prof. Dr. Suleyman Yalcin City Hospital between January 2018 and October 2020. The patients ranged in age from 18 to 65, without any known systemic diseases, medication use, or evidence to explain vertigo after the etiological examinations. All participants were evaluated with an otoscopic examination, pure-tone audiometry, tympanometry, otoacoustic emission, CDUSG, bedside examination (including Romberg's test, Unterberger's test, tandem gait test with/without vision, and cerebellar tests), videonystagmography (VNG), functional head impulse test (fHIT), and video head impulse test (vHIT). Participants were divided into 2 groups: study group and control group. The study group consisted of individuals with nonspecific dizziness, and the control group consisted of individuals who had no dizziness and had normal clinical findings in vestibular examinations. Additional criteria for the study group included having dizziness complaints for at least 1 year, and no other neurological, mental, or physical problems (including no head and neck problems). For the control group, additional inclusion criteria included having normal hearing thresholds (pure-tone thresholds ≤ 25 dB nHL at all audiometric frequencies), normal tympanometry, and no other neurological, mental, or physical problems. The demographic characteristics of participants in both groups are shown in Table 1.

The VNG test, which allows real-time recording of abnormal eye movements produced by various visual and positional stimuli, was performed with the ICS Charter 200 system (GN Otometrics, Taastrup, Denmark). The VNG test battery included the following: (i) oculomotor function tests (with fixation): saccade, tracking, and optokinetic tests; (ii) gaze stabilization tests (with or without fixation, level of alertness): gaze/spontaneous nystagmus, static position tests; (iii) dynamic positioning tests: Dix-Hallpike, supine head roll, and supine head-hanging tests; and (iv) head shake test.

The vHIT and the fHIT tests were used to test the visual fixation ability. Although both tests evaluate the vestibulo ocular reflex (VOR) system, it provides information about different aspects of the VOR. The vHIT, which measures the ability to maintain visual fixation during rapid acceleration and deceleration of the head, provides as output a gain value that summarizes the behavior of the VOR as the ratio of a measure of eye movement to the corresponding measure of head movement. In contrast, the fHIT assesses the capability to keep clear vision and to read during rapid head movements and provides a functional measure of the VOR.²¹

The vHIT test was only performed for horizontal canal function using the Eye See Cam vHIT Interacoustics® (Denmark) device in a seated position under room light. For the vHIT, a pair of lightweight goggles with a gaze-controlled high-speed digital camera system (sampling rate of 220 Hz) that recorded real-time eye movement, a motion sensor that measured head movement, and laser light for calibration were tightly fitted onto the participants' heads. Then, the examiner placed her hands on the patient's jaw and delivered at least 20 unpredictable head impulses (amplitude 15°-20°, duration 150-200 ms, target head velocity 100-200 degree/s) along the planes of the lateral semicircular canal to each side. The participants were fixated on a target located approximately 1 m straight ahead during the procedure. The presence of corrective saccades and VOR gain values on each side were examined. Corrective saccades were classified as covert saccades if they occurred during head movement and overt saccades if they occurred after head movement.

The fHIT on both ears was conducted using the Beon Solutions SRL system (Zero Branco, Italy) to evaluate visual acuity during passive head movements between 3000 and 6000°/s² in the horizontal plane. For the fHIT, the test examiner delivered 20 passive head impulses in each direction in the horizontal canal plane. Participants were asked to describe the "C" optotypes that appeared on the screen during each head push.

The CDUSG examination was performed in the supine position using a Toshiba Aplio 500 (Toshiba Medical, Japan) with a broadband linear transducer (5-14 MHz). The CDUSG examination was performed for the bilateral VA of each patient. Vertebral artery origin, V1 (pre-foraminal) segment, and V2 (foraminal) segment were carefully evaluated in all cases. Arterial localization, course, and direction of blood flow were confirmed by 2-dimensional imaging. Color Doppler, power Doppler, and pulse wave functions were used to measure the hemodynamic parameters of VA (arterial diameter, arterial area, peak systolic velocity, and flow volume).

The study protocol was approved by the Istanbul Medeniyet University Göztepe Training and Research Hospital Clinical Research Ethics Committee Ethics Committee (July 1, 2020, 2020/0432), and written informed consent was obtained from patients who participated in this study. All procedures were performed in accordance with the ethical standards of the Helsinki Declaration of 2013.

Statistical Analysis

The sample sizes required for Mann-Whitney *U*-tests were calculated using the G*Power v3.1.9.4 program at a 95% power level and 5% statistical significance level. Accordingly, it would be sufficient to have 37 participants per group in comparison to any 2 groups to statistically determine a relatively large effect (relative size) of 0.954. The number of participants per group in the patient and control groups fulfilled this requirement. The data were analyzed using IBM Statistical Package for the Social Sciences Statistics for Windows, Version 20.0 (IBM Corp., Armonk, NY, USA). The Shapiro-Wilk test showed that the distribution of the data was not normal. Therefore, nonparametric tests were

Table 1. The Demographics of Participants with Vestibular Complaints and Healthy Controls

		Participants with Vestibular Complaints	Healthy Controls	P
Number of participants	n	38	39	
Age	Years (mean \pm SD)	51.73 \pm 13.98	47.31 \pm 12.51	.104
Gender	Male (n, %)	13, 34.2	16, 41	.640
	Female (n, %)	25, 65.8	23, 59	
Comorbidities	Yes (n, %)	10, 26.3	1, 2.6	<.001*
Nystagmus	Yes (n, %)	23, 60.5	11, 17.94	.006*
	No (n, %)	15, 39.5	28, 82.05	
Hearing loss	Yes (n, %)	13, 34.2	0	<.001*
	No (n, %)	25, 65.8	39, 100	
Romberg's test abnormalities	Yes (n, %)	2, 5.26	0	.149
	No (n, %)	36, 94.73	39, 100	
Unterberger's test abnormalities	Yes (n, %)	2, 5.26	0	.149
	No (n, %)	36, 94.73	39, 100	
Tandem gait test abnormalities	Yes (n, %)	3, 7.89	0	.075
	No (n, %)	35, 92.10	39, 100	
Cerebellar test abnormalities	Yes (n, %)	0	0	NA
	No (n, %)	38, 100	38, 100	
Gaze (with fixation)	R (mean \pm SD)	0	0	NA
	L (mean \pm SD)	0	0	NA
	U (mean \pm SD)	0	0	NA
	D (mean \pm SD)	0	0	NA
Gaze (without fixation)	R (mean \pm SD)	0.55 \pm 1.72	0.06 \pm 0.42	.083
	L (mean \pm SD)	0.57 \pm 1.73	0.06 \pm 0.42	.083
	U (mean \pm SD)	0.61 \pm 1.88	0.06 \pm 0.42	.146
	D (mean \pm SD)	0.11 \pm 0.71	0.06 \pm 0.42	.971
Saccade—accuracy	R (mean \pm SD)	90.41 \pm 8.25	90.75 \pm 11.76	.596
	L (mean \pm SD)	89.19 \pm 8.49	90.4 \pm 13.27	.401
Saccade—velocity	R (mean \pm SD)	391.16 \pm 46.81	390.15 \pm 39.23	.656
	L (mean \pm SD)	411.5 \pm 49.83	391.9 \pm 51.91	.056
Saccade—latency	R (mean \pm SD)	191.25 \pm 49.54	187.15 \pm 27.38	.626
	L (mean \pm SD)	198.63 \pm 38.35	195.55 \pm 39.73	.632
Tracking-velocity gain	R (mean \pm SD)	0.63 \pm 0.28	0.65 \pm 0.37	.378
	L (mean \pm SD)	0.71 \pm 0.24	0.64 \pm 0.36	.898
Optokinetic-slow phase velocity	R (mean \pm SD)	17.51 \pm 5.47	19.52 \pm 6.44	.096
	L (mean \pm SD)	18.81 \pm 5.51	19.26 \pm 6.12	.225
vHIT gain	R (mean \pm SD)	1.18 \pm 0.27	1.11 \pm 0.11	.242
	L (mean \pm SD)	1.12 \pm 0.21	1.08 \pm 0.15	.357
fHIT score	R (mean \pm SD)	91.53 \pm 14.93	98.34 \pm 4.95	.009*
	L (mean \pm SD)	90.97 \pm 16.74	97.15 \pm 4.18	.361
PTA	R (mean \pm SD)	17.03 \pm 9.59	6.59 \pm 5.31	<.001*
	L (mean \pm SD)	18.39 \pm 12.75	7.59 \pm 6.62	<.001*

D, down; fHIT, functional head impulse test; L, left; NA, not applicable; PTA, pure-tone average; R, right; U, up; vHIT, video head impulse test.

*Significant difference at $P=.05$ level.

performed for statistical analyses. The average and SD values of the continuous data and the frequencies of the nominal and ordinal data were calculated. The significance of the difference in continuous data between the groups was analyzed using the Mann–Whitney U -test. The significance of the difference in categorical data between the groups was analyzed using the chi-square test. Binary logistic regression models were created to analyze the predictive values of continuous and categorical independent variables on the vestibular complaint dependent variable. The significance level was given as .05 in all analyses.

Results

A total of 77 participants, 38 of whom had nonspecific vestibular complaints and 39 of whom were healthy adults, were included in the study. No abnormal finding was observed in the bedside examination tests in the control group. In the study group, 2 participants swayed

to the left during the Romberg test; during the tandem gait test, 1 participant swayed to the right, 2 participants swayed to the left, and 3 participants swayed to the right; during the Unterberger's test, 2 participants veered nearly 30° to the left. No abnormal findings were detected during the cerebellar tests. Among the participants who had abnormal findings during the bedside tests, no pathology that could be explained by clinical tests was found. There were no statistically significant differences between the groups in terms of bedside examination results. The percentage of comorbidity, positional nystagmus, and hearing thresholds differed significantly between the 2 groups. However, oculomotor functions and vHIT findings were similar. Results from the fHIT indicated that the reading scores with the right movement of the head were significantly lower in the study group compared to the control group ($P=.009$) (Table 1). The nystagmus characteristics of participants with vestibular complaints and healthy participants are presented in Table 2. The incidence of nystagmus was higher in those

Table 2. The Nystagmus Characteristics of Participants with Vestibular Complaints and Healthy Controls

Situation		Participants with Vestibular Complaints	Healthy Controls	P
		Slow Phase Velocity (degrees/s) (Mean ± SD)		
Spontaneous nystagmus (with fixation)	Center gaze	0	0	NA
Spontaneous nystagmus (without fixation)	Center gaze	0.08 ± 1.13	0.06 ± 0.42	.277
Static position tests (sitting)	Head right	0.4 ± 1.02	None	.01*
	Head left	0.37 ± 0.96	None	.01*
	Head extension	0.96 ± 1.85	0.09 ± 0.6	.002*
	Head extension with right rotation	0.52 ± 1.28	0.14 ± 0.62	.072
	Head extension with left rotation	0.31 ± 0.89	0.12 ± 0.53	.232
Static position tests (supine)	With pillow (30° flexion)	1.27 ± 2.08	0.38 ± 0.92	.034*
	Head right with pillow (30° flexion)	0.91±1.86	None	.001*
	Head left with pillow (30° flexion)	0.98 ± 1.7	0.09 ± 0.6	.001*
	Without pillow	0.93 ± 1.78	0.37 ± 1	.093
	Head right without pillow	1.08 ± 2.41	0.09 ± 0.6	.004*
	Head left without pillow	0.79 ± 1.34	0.34 ± 1.03	.019*
Head shake nystagmus		0.36 ± 1.52	0.07 ± 0.48	.289
NA, not applicable.				
*Significant difference at P= .05 level.				

with vestibular complaints compared to the healthy participants when the head was on the right side ($P = .01$), the head was on the left side ($P = .01$), and the head was in extension ($P = .002$). Similarly, the incidence of nystagmus was higher in the study group in the supine position ($P = .034$), lying flat with the head on the right side ($P = .001$, $P = .004$), and on the left side ($P = .001$, $P = .019$) with and without a pillow. The VA CDUSG characteristics of participants with vestibular complaints and healthy participants are presented in Table 3. Among VA CDUSG findings, values for the right ($P = .007$) and left ($P < .001$) VA velocity, the left VA flow ($P = .002$), the total flow ($P < .001$), and the total velocity ($P < .001$) were found to be significantly lower in participants with vestibular complaints (Table 3).

The significance of the difference in categorical data between the groups was analyzed using the chi-square test (Table 4). Accordingly, the presence of nystagmus ($P = .006$), comorbidity ($P < .001$), hearing loss ($P < .001$), and medication usage ratios ($P < .001$) were significantly higher in participants with vestibular complaints.

Table 3. Vertebrobasilar Artery Color Doppler Ultrasonography Characteristics of Participants with Vestibular Complaints and Healthy Controls

Color Doppler Ultrasonography Parameters		Participants with Vestibular Complaints	Healthy Controls	P
		(Mean ± SD)		
Vertebrobasilar artery diameter	R	3.44 ± 0.56	3.43 ± 0.58	.717
	L	3.51 ± 0.54	3.42 ± 0.52	.759
Vertebrobasilar artery area	R	9.53 ± 3.01	9.40 ± 3.35	.599
	L	9.89 ± 3.12	9.31 ± 2.8	.595
Vertebrobasilar artery velocity	R	14.07 ± 3.99	16.71 ± 4.74	.007*
	L	14.3 ± 3.64	20.55 ± 8.96	<.001*
Vertebrobasilar artery flow	R	128.89 ± 35.66	153.71 ± 52.24	.051
	L	136.92 ± 43.62	191.97 ± 103.87	.002*
Total flow		265.81 ± 42.31	345.69 ± 101.01	<.001*
Total velocity		28.37 ± 5.61	37.27 ± 10.89	<.001*
Vertebrobasilar artery velocity differences		3.95 ± 3.29	6.88 ± 7.32	.056
Vertebrobasilar artery flow differences		45.97 ± 49.54	83.69 ± 105.48	.175

L, left; R, right.
*Significant difference at $P = .05$ level.

Two separated binary logistic regression models were created to analyze the predictive values of continuous and categoric independent variables on the vestibular complaint dependent variable. In the first model, the presence of a nystagmus independent variable significantly predicted the presence of a vestibular complaint dependent variable ($\beta = -1.643$, $df = 1$, $P = .014$, $R^2 = 0.792$). In the second model, the total VA flow independent variable significantly predicted the presence of a vestibular complaint dependent variable ($\beta = -0.026$, $df = 1$, $P = .014$, $R^2 = 0.714$).

Discussion

In this study, the aim was to investigate whether the complaint of peripheral or central vestibular system disease, syncope, or non-pre-syncope-induced vertigo is associated with hemodynamic variables of the VA. For this purpose, audio-vestibular evaluation results and VA CDUSG results of nonspecific vertigo cases were compared with healthy individuals without any vestibular and central vestibular disorders. It was observed that in the results from VA CDUSG examinations, the differences in values of the diameter of the right and left VA were not statistically significant in either group. The obtained mean values of the diameters of VA are similar to the results obtained by other authors examining these vessels.^{22,23} Mysior and Stefańczyk²⁴ reported that ≤ 0.5 mm asymmetry of the VA did not cause significant differences in measurable hemodynamic blood flow parameters.

In our study, total VA flow was found to be within normal limits in both groups. However, VA flow and velocity values were lower in the study group compared to the control group. Studies by other authors have reported lower blood flow velocity values in the VA in patients with symptoms of VBI.^{25,26} These findings suggest that close follow-up of the feeding of the VA despite normal VA CDUSG findings in patients

Table 4. The Comparisons of Categoric Variables of Participants with Vestibular Complaints and Healthy Controls

Chi-square (X^2)	P
Presence of nystagmus	.006*
Presence of comorbidity	<.001*
Medication usage	<.001*
Gender	.64
Presence of hearing loss	<.001*

*Bold indicate significant X^2 values ($P < .05$).

with nonspecific vestibular complaints may be important for the early detection of a problem.

There was no specific pathology finding that could be supported by clinical tests in either group at the bedside examinations, and no statistically significant difference was found between the groups in terms of abnormal bedside examination results. However, the error rate in the bedside examinations of the study group was higher than that of the control group. While these results do not show a definite pathology in the study group, they support the idea that the participants in the study group have more postural control and balance problems than the healthy participants in the control group. Similarly with bedside examination results, in vestibular clinic evaluations, including vHIT, fHIT, and VNG, findings also did not indicate any definite vestibular pathology in either group. All the results of these clinical tests were within normal limits when compared to normative data in the literature. However, in the static positional nystagmus examination of the VNG test, the incidence of nystagmus in the study group was statistically higher than in the control group in sitting and supine positions with the head on the right, the left, and in extension. It was thought that this may be due to the fact that the restrictive effect of neck rotation and extension on the VA flow velocity. Causse et al²⁷ have described the nystagmus obtained by extending and rotating the neck for 3 minutes as the vertebrobasilar deprivation nystagmus when every other possible cause of nystagmus has been discarded. They have advanced that it is caused by a decrease in the blood flow in the opposite vertebral artery because of the head rotation. Moubayed and Saliba⁴ reported that the physiopathology of this event is secondary to depolarization induced by ischemia of vestibular cells, such that axons become unresponsive at a late stage of ischemic injury, leading to cellular hypofunctions. However, this explanation is controversial because if the basilar artery has normal blood flow, there would be no reason for decreased perfusion of the terminal circulation. The literature findings on blood flow changes in the VA caused by neck torsion are also unclear. Some of the studies mentioned no significant hemodynamic change for those in the cervical positions,^{28,29,30,31} whereas other studies mentioned a significant hemodynamic decrease for all those in the cervical positions.^{31,33} In the current study, despite the total flow values being within normal limits in both groups, the decreased flow values compared to the control group may have caused a further decrease in flow during neck rotation in the study group and may have increased the possibility of positional/cervical nystagmus. Karlberg et al³⁴ and Niewiadomski and Kwiatkowska²⁹ reported a stable correlation between the neck torsion test and an increase in induced nystagmus. On the other hand, Niewiadomski and Kwiatkowska²⁹ reported that neck rotation and systolic and diastolic blood flow velocity values decreased more in the study group than in the control group in their study with healthy controls and participants with vertigo, hearing loss, and confirmed carotid or VA anomalies. These findings support the idea that following up on the decreased flow values in the study group with nonspecific vertigo complaints compared to the control group in our study may be important in terms of VBI. The data obtained from the regression model in the current study showed that the presence of nystagmus predicted vestibular complaints at a rate of 79%, and vestibular complaints predicted changes in total VA flow rate at a rate of 71%. These findings also contribute to demonstrating the relationship between changes in VA flow rate in the control group and positional nystagmus.

In addition, the fact that hearing thresholds were lower in the study group compared to the control group, despite normal findings, suggests that the nutrition in the inner ear may also have been affected in this group.

Although the presence of numerous confounding factors prevents us from reaching a definite conclusion, the data obtained from our study support the possibility that symptoms such as dizziness, unsteadiness, darkening of vision, and nonspecific positional nystagmus may be early signs of a transient ischemic attack. This highlights the importance of monitoring patients with nonspecific vestibular complaints using VA CDUSG and vestibular tests. However, the current study is limited in that it was carried out in a small sample group retrospectively and does not include older adults (over 65 years) who may be more at risk for VAS. In the future, conducting randomized controlled longitudinal studies with larger samples will increase the level of evidence of the results.

Conclusion

In individuals with nonspecific vertigo, the VA flow and velocity were lower than in healthy controls. In addition, the incidence of nonspecific positional nystagmus, presence of comorbid features, and hearing loss were higher. Therefore, monitoring the dynamic properties of the VA closely in individuals with nonspecific vestibular complaints, especially those accompanied by positional nystagmus, hearing loss, and comorbid features, can be important for early detection of potential problems and improvement of outcomes.

Ethics Committee Approval: Ethical committee approval was received from the Ethics Committee of İstanbul Medeniyet University, Goztepe Training, and Research Hospital (Approval no: 2020/0432, Date: July 1, 2020).

Informed Consent: Written informed consent was obtained from the patients who agreed to take part in the study.

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References

1. Back JD, Herzog RJ, Lutz GE. A rare anomaly of the course of the vertebral artery. *Spine J*. 2011;11(7):681-682. [\[CrossRef\]](#)
2. Chibbaro S, Mirone G, Yasuda M, Marsella M, Di Emidio P, George B. Vertebral artery loop – a cause of cervical radiculopathy: review article. *World Neurosurg*. 2012;78(3-4): 375.e11-13. [\[CrossRef\]](#)
3. Yamanaka T, Sawai Y, Hosoi H. Bilateral subclavian steal syndrome with vertigo. *Auris Nasus Larynx*. 2014;41(3):307-309. [\[CrossRef\]](#)
4. Moubayed SP, Saliba I. Vertebrobasilar insufficiency presenting as isolated positional vertigo or dizziness: a double-blind retrospective cohort study. *Laryngoscope*. 2009;119(10):2071-2076. [\[CrossRef\]](#)
5. Kim GW, Heo JH. Vertigo of cerebrovascular origin proven by CT scan or MRI: pitfalls in clinical differentiation from vertigo of aural origin. *Yonsei Med J*. 1996;37(1):47-51. [\[CrossRef\]](#)
6. Kerber KA, Brown DL, Lisabeth LD, Smith MA, Morgenstern LB. Stroke among patients with dizziness, vertigo, and imbalance in the emergency department: a population-based study. *Stroke*. 2006;37(10):2484-2487. [\[CrossRef\]](#)
7. Grant EG, Wong W, Tessler F, Perrella R. Cerebrovascular ultrasound imaging. *Radiol Clin North Am*. 1988;26(5):1111-1130. [\[CrossRef\]](#)
8. Katada K, Kanno T, Sano H, Shinomiya Y, Koga S. Calcification of the vertebral artery. *AJNR Am J Neuroradiol*. 1983;4(3):450-453. [\[CrossRef\]](#)
9. Solberg LA, Eggen DA. Localization and sequence of development of atherosclerotic lesions in the carotid and vertebral arteries. *Circulation*. 1971;43(5):711-724. [\[CrossRef\]](#)

10. Feng Y, Liu J, Fan T, et al. Vertebral artery stenoses contribute to the development of diffuse plaques in the basilar artery. *Front Bioeng Biotechnol.* 2020;8:168. [\[CrossRef\]](#)
11. Wityk RJ, Chang HM, Rosengart A, et al. Proximal extracranial vertebral artery disease in the New England Medical Center Posterior Circulation Registry. *Arch Neurol.* 1998;55(4):470-478. [\[CrossRef\]](#)
12. Hsu CY, Cheng CY, Lee JD, et al. Clinical features and outcomes of spinal cord infarction following vertebral artery dissection: a systematic review of the literature. *Neurol Res.* 2013;35(7):676-683. [\[CrossRef\]](#)
13. Moufarrij NA, Little JR, Furlan AJ, Williams G, Marzewski DJ. Vertebral artery stenosis: long-term follow-up. *Stroke.* 1984;15(2):260-263. [\[CrossRef\]](#)
14. Schöning M, Walter J, Scheel P. Estimation of cerebral blood flow through color duplex sonography of the carotid and vertebral arteries in healthy adults. *Stroke.* 1994;25(1):17-22. [\[CrossRef\]](#)
15. Trattng S, Hübsch P, Schuster H, Pölzleitner D. Color-coded Doppler imaging of normal vertebral arteries. *Stroke.* 1990;21(8):1222-1225. [\[CrossRef\]](#)
16. Seidel E, Eicke BM, Tettenborn B, Krummenauer F. Reference values for vertebral artery flow volume by duplex sonography in young and elderly adults. *Stroke.* 1999;30(12):2692-2696. [\[CrossRef\]](#)
17. Hong JM, Chung CS, Bang OY, Yong SW, Joo IS, Huh K. Vertebral artery dominance contributes to basilar artery curvature and perivertebrbasilar junctional infarcts. *J Neurol Neurosurg Psychiatry.* 2009;80(10):1087-1092. [\[CrossRef\]](#)
18. Liu SP, Li LI, Bin SUN, et al. Two-dimensional and color Doppler ultrasound analysis of vertebral artery in pilot cadets. *Med J Chin Peoples Liberation Army.* 2012;7(4):326-329.
19. Bendick PJ, Glover JL. Vertebrbasilar insufficiency: evaluation by quantitative duplex flow measurements: a preliminary report. *J Vasc Surg.* 1987;5(4):594-600. [\[CrossRef\]](#)
20. Scheel P, Ruge C, Schöning M. Flow velocity and flow volume measurements in the extracranial carotid and vertebral arteries in healthy adults: reference data and the effects of age. *Ultrasound Med Biol.* 2000;26(8):1261-1266. [\[CrossRef\]](#)
21. Versino M, Colnaghi S, Corallo G, Mandalà M, Ramat S. The functional head impulse test: comparing gain and percentage of correct answers. *Prog Brain Res.* 2019;248:241-248. [\[CrossRef\]](#)
22. Jeng JS, Yip PK. Evaluation of vertebral artery hypoplasia and asymmetry by color-coded duplex ultrasonography. *Ultrasound Med Biol.* 2004;30(5):605-609. [\[CrossRef\]](#)
23. Yazici B, Erdoğan B, Tugay A. Cerebral blood flow measurements of the extracranial carotid and vertebral arteries with Doppler ultrasonography in healthy adults. *Diagn Interv Radiol.* 2005;11(4):195-198.
24. Mysior M, Stefańczyk L. Doppler ultrasound criteria of physiological flow in asymmetrical vertebral arteries. *Med Sci Monit.* 2007;13(1):73-77.
25. Troscák M, Pavlík V, Eiben E, et al. Cerebral circulation using transcranial and extracranial doppler ultrasonography in a patient with vertigo and ischemia in the area of the brain stem. *Cesk Neurol Neurochir.* 1990;53(5):312-320.
26. Zhao P. A clinical study of vertebrobasilar insufficiency using the ultrasonic Doppler technique. *Zhonghua Er Bi Yan Hou Ke Za Zhi.* 1991;26(2):93-95.
27. Causse JB, Conraux C, Causse J. Vertebral-basilar artery insufficiency nystagmus (author's transl). *Ann Otolaryngol Chir Cervicofac.* 1978;95(3):225-234.
28. Haynes MJ, Cala LA, Melsom A, Mastaglia FL, Milne N, McGeachie JK. Vertebral arteries and cervical rotation: modeling and magnetic resonance angiography studies. *J Manipulative Physiol Ther.* 2002;25(6):370-383. [\[CrossRef\]](#)
29. Niewiadomski W, Kwiatkowska D. Hemodynamic effects of strength exercises. *J Hum Kinet.* 2007;18:45-62.
30. Yi-Kai L, Yun-Kun b Z, Cai-Mo c L, Shi-Zhen Z. Changes and implications of blood flow velocity of the vertebral artery during rotation and extension of the head. *J Manipulative Physiol Ther.* 1999;22(2):91-95. [\[CrossRef\]](#)
31. Zmysłowska-Szmytko E, Adamczewski T, Ziaber J, Majak J. Asymetria stawu szczytowo-obrotowego jako jedna z ewentualnych przyczyn zawrotów głowy [Atlanto-axial rotation as a possible cause of cervical vertigo]. *Otolaryngologia.* 2014;13(1):58-65.
32. Mitchell JA. Changes in vertebral artery blood flow following normal rotation of the cervical spine. *J Manipulative Physiol Ther.* 2003;26(6):347-351. [\[CrossRef\]](#)
33. Sakaguchi M, Kitagawa K, Hougaku H, et al. Mechanical compression of the extracranial vertebral artery during neck rotation. *Neurology.* 2003;61(6):845-847. [\[CrossRef\]](#)
34. Karlberg M, Magnusson M. Asymmetric optokinetic after-nystagmus induced by active or passive sustained head rotations. *Acta Otolaryngol.* 1996;116(5):647-651. [\[CrossRef\]](#)