

Vertebrobasilar system computed tomographic angiography in central vertigo

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Abstract

The incidence of vertigo in the population is 20% to 30% and one-fourth of the cases are related to central causes. The aim of this study was to evaluate computed tomography angiography (CTA) findings of the vertebrobasilar system in central vertigo without stroke.

CTA and magnetic resonance images of patients with vertigo were retrospectively evaluated. One hundred twenty-nine patients suspected of having central vertigo according to history, physical examination, and otological and neurological tests without signs of infarction on diffusion-weighted magnetic resonance imaging were included in the study. The control group included 120 patients with similar vascular disease risk factors but without vertigo. Vertebral and basilar artery diameters, hypoplasias, exit-site variations of vertebral artery, vertebrobasilar tortuosity, and stenosis of $\geq 50\%$ detected on CTA were recorded for all patients. Independent-samples *t* test was used in variables with normal distribution, and Mann–Whitney *U* test in non-normal distribution. The difference of categorical variable distribution according to groups was analyzed with χ^2 and/or Fisher exact test.

Vertebral artery hypoplasia and $\geq 50\%$ stenosis were seen more often in the vertigo group ($P=0.000$, <0.001). Overall 78 (60.5%) vertigo patients had $\geq 50\%$ stenosis, 54 (69.2%) had stenosis at V1 segment, 9 (11.5%) at V2 segment, 2 (2.5%) at V3 segment, and 13 (16.6%) at V4 segment. Both vertigo and control groups had similar basilar artery hypoplasia and $\geq 50\%$ stenosis rates ($P=0.800$, >0.05).

CTA may be helpful to clarify the association between abnormal CTA findings of vertebral arteries and central vertigo.

This article reveals the opportunity to diagnose posterior circulation abnormalities causing central vertigo with a feasible method such as CTA.

Abbreviations: CTA = computed tomography angiography, DWI = diffusion-weighted imaging, MRA = magnetic resonance angiography, MRI = magnetic resonance imaging, PACS = picture archiving and communication system, TIA = transient ischemic attack.

Keywords: basilar artery, CTA, vertebral artery, vertigo

1. Introduction

The incidence of vertigo in the population is 20% to 30%.^[1] While otological, central, somatosensorial, and visual factors may cause vertigo, central causes are responsible in one-quarter of the cases.^[2] Semicircular canals, saccule, utricle, and the vestibular nerve create the peripheral vestibular system. Vestibular nucleus, cerebellum, cerebral peduncle, the spinal cord, and vestibular cortex create the central vestibular system.^[3] Central vertigo may be accompanied by neurological symptoms such as ataxia, dysarthria, diplopia, and visual disturbance or weakness.^[4] Central vertigo can be

distinguished from peripheral vertigo through history, neurological examination, and imaging findings.^[3] Most common causes of central vertigo are vertebrobasilar insufficiency, stroke, transient ischemic attack (TIA), migraine, multiple sclerosis, posterior fossa tumors, neurodegenerative disorders, some drugs, and psychiatric conditions.^[3,4]

Central vertigo related to cerebellar and cerebral peduncle strokes was presented in many studies with magnetic resonance imaging (MRI).^[5–9] However, there are no computed tomography angiography (CTA) studies investigating vertebrobasilar artery changes in central vertigo patients without stroke. Both CTA and contrast-enhanced magnetic resonance angiography (MRA) have high sensitivity and specificity for detecting $\geq 50\%$ stenosis of vertebral artery.^[10–13]

The aim of this study was to investigate the vertebrobasilar system CTA findings of central vertigo without stroke.

2. Methods

Local ethics committee approval was obtained (approval number: 2014/906).

2.1. Patient selection

Between January 2010 and January 2014 patients who had undergone CTA and MRI were retrospectively evaluated on picture archiving and communication system (PACS). Clinical

Editor: Massimo Tusconi.

The authors have no funding and conflicts of interest to disclose.

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Medicine (2017) 96:12(e6297)

Received: 15 September 2016 / Received in final form: 2 February 2017 /

Accepted: 14 February 2017

<http://dx.doi.org/10.1097/MD.0000000000006297>

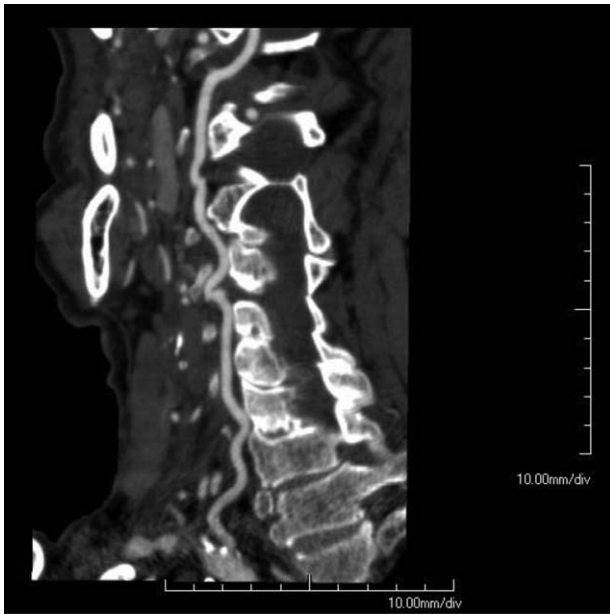


Figure 1. A 72-year-old-man with vertigo. A 50% stenosis is seen due to a noncalcified plaque on the origin of right vertebral artery on the curved multiplane reconstructed computed tomographic angiography.

data, vascular risk factors, physical examination findings, and radiological images were evaluated. The duration, intensity, and frequency of vertigo and accompanying symptoms were asked. According to the neurological examinations, horizontal, vertical, or rotational nystagmus did not decrease when the patients with abnormal skew tests focused their gaze and these patients were considered to have central vertigo. One hundred sixty-three patients with abnormal skew tests were considered to have central vertigo and included in the study.



Figure 2. A 68-year-old-woman with vertigo. Right vertebral artery hypoplasia is seen on the 3D volume rendered computed tomographic angiography.

CTA images of all patients were evaluated on PACS by an experienced radiologist. Of these 163 cases, 25 patients with central vertigo were excluded from the study because of lacking diffusion-weighted MRI (DWI). Nine patients with cerebellar or cerebral peduncle infarction detected on DWI were also excluded from the study. DWI was evaluated on PACS by the same radiologist. A total of 129 (51.8%) patients with central vertigo and 120 (48.2%) controls with similar age and vascular risk factors (diabetes, hypertension, and dyslipidemia) were included in the study. Control patients selected from the PACS had CTA examinations done for headache and evaluation of carotid and vertebral artery stenosis without vertigo.

2.2. Computed tomography angiography

CTA scans were performed with a 64-detector (Aquilion 64, Toshiba Medical Systems, Tochigi, Japan, 2011) or a 16-detector (Aquilion 16, Toshiba Medical Systems, 2005) computed tomography (CT) device. Scanning parameters of the 64-detector CT were as follows: collimation 64×0.5 , gantry rotation time 0.5 second, slice thickness 0.5 mm, step value 0.64, 120 kV, and 450 mA. Scanning parameters of the 16-detector CT were as follows: collimation 16×1.0 , gantry rotation time 0.75 second, slice thickness 1 mm, step value 0.75, 120 kV, and 300 mA.

Patients were placed in supine position and their heads immobilized with cranial coil and immobilization devices. Caudocranial scans were performed after the localization was determined on lateral topogram; the patients were told not to swallow and to breathe shallowly.

Nonionic, high-iodine concentrated, 100-mL, intravenous (350–400 mg/mL iodine concentration) contrast material was delivered from the antecubital region via 18- to 20-gauge cannula with an automated pump at 4 mL/s (Ulrich Medizin version, 2004, Germany). After the contrast material, 40 cm³ of normal saline was administered.

CTA imaging was performed with bolus tracking method, and the scanning started after the density of the aortic arch reached 120 to 130 Hounsfield units. The total duration of scanning with the 64-detector CT was 8.6 seconds, and with the 16-detector CT 9.7 seconds.

2.3. CTA assessment

The images obtained on CTA were evaluated using Vitrea (4.14.4, 2008, Vital Images Inc, Minnetonka, MN) or Aquarius (iNtuition edition version 4.4.11.82.6784, TeraRecon Inc, Foster, CA) software in axial, sagittal, coronal, or oblique planes, and using multiplanar reconstruction, volume rendered, and maximum-intensity projection images. HP ZR2440W (24 in., 1920 × 1200 at 60 Hz) and ASUS PB278Q (27 in., 2560 × 1440 at 60 Hz) monitors were used in the evaluation of the images.

The origins of the vertebral arteries were evaluated and the vertebral and basilar arteries were inspected along their trace on the axial images. Vertebral artery diameters on the V2 segment at the C4 vertebrae levels were measured with 200% magnification between outer contours on coronal reformatted images. In patients with atheromatous plaques the vessel diameter was measured distally from the plaque on places without plaque. Vertebral arteries were classified as normal, hypoplastic, and $\geq 50\%$ stenotic (Fig. 1). Diameters < 2 mm and the posterior inferior cerebellar artery ending termination of the vertebral arteries were accepted as hypoplastic (Fig. 2). Vertebral artery segments were

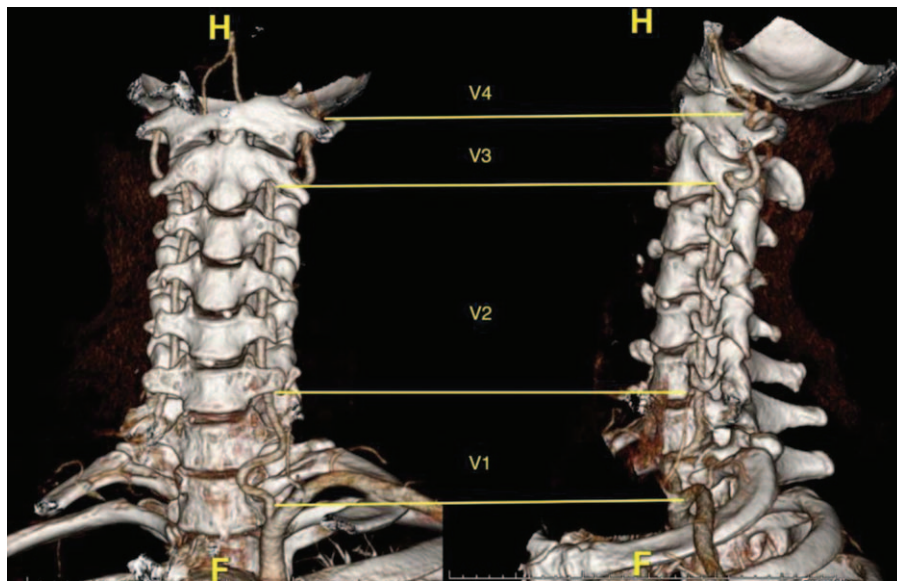


Figure 3. Vertebral artery segments are shown on the 3D volume rendered coronal and sagittal CTA images. CTA = computed tomography angiography.

classified as follows: V1, from origin to the transverse foramina of C5 or C6 vertebrae; V2, from the transverse foramina of C5 or C6 to the transverse foramina to C2; V3, from the C2 transverse foramina to dura; and V4, from dura to the confluence of 2 vertebral arteries to form the basilar artery (Fig. 3A and B). Basilar artery diameter was measured from coronal reformatted

images at midline pontine level with 200% magnification between outer contours. Basilar artery was classified as normal, hypoplastic, and $\geq 50\%$ stenotic. Diameters < 2 mm were accepted as hypoplastic (Fig. 4A and B). Curving, angulation/kinking, looping, and spiral twisting vertebral arteries were regarded tortuous (Fig. 5). If the basilar artery, throughout its course, lay lateral to the margin of the clivus or dorsum sellae or if the artery bifurcates above the plane of the suprasellar cistern, it was regarded as tortuous.

In patients with atheromas, stenosis measurements were made by applying North American Symptomatic Carotid Endarterectomy Trial-style ratio calculations to the vertebral and basilar arteries.^[14]

On a curved multiplanar reconstructed image the smallest diameter of the atheromatous vessel was compared to the normal

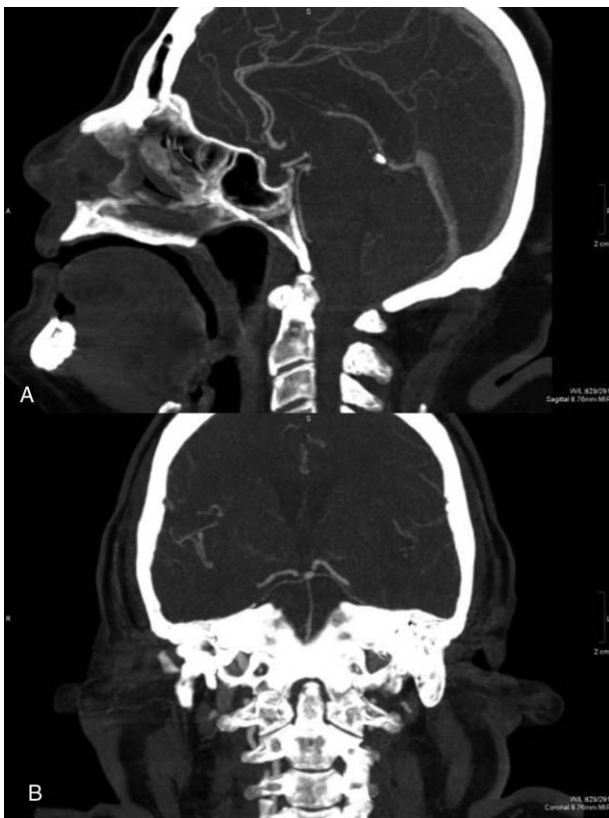


Figure 4. A 65-year-old-man with vertigo. Basilar artery hypoplasia is seen on the sagittal (A) and coronal (B) maximum-intensity projection multiplane reconstructed computed tomographic angiography images.

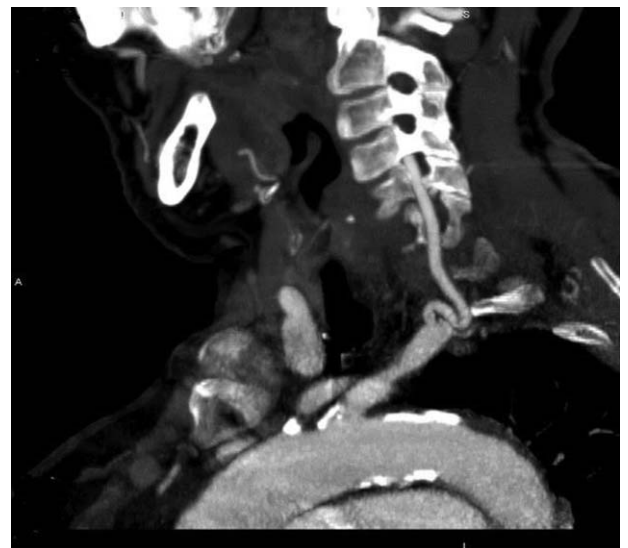


Figure 5. Sagittal oblique maximum-intensity projection (MIP) image shows the kinking and tortuosity of the V1 segment of left vertebral artery.

Table 1**Vertebral and basilar artery diameter distribution.**

	Control, mm	Vertigo, mm	P
Right vertebral artery	3.94±0.9	3.72±1.15	>0.05
Left vertebral artery	4.18±0.8	3.97±1.1	>0.05
Basilar artery	3.44±0.7	3.33±0.6	>0.05

$P > 0.05$, no statistical significance.

vessel diameter at the distal part of this segment; thus, the percentage of stenosis and the open segment was calculated.

2.4. Magnetic resonance imaging

The study was performed with 1.5-T MRI (Optima 450W, General Electric Medical Systems, Milwaukee, WI). Head coil and immobilization devices were used. The b value was 1000/ mm^2 on diffusion-weighted imaging. Image analysis was performed on a workstation (GE Advantage Workstation AW4.2_08) using functool 2 image analysis software (GE Medical Systems).

2.5. Statistical analysis

The coherence between normal distribution with the data of the patient and the control groups was analyzed. Independent-samples *t* test was used in variables with normal distribution, and Mann–Whitney *U* test in non-normal distribution.

The difference of categorical variable distribution according to groups was analyzed with χ^2 and/or Fisher exact test.

Descriptive statistics were average \pm standard deviation, median, minimum–maximum, and percentage.

Analysis was evaluated with SPSS 11.5 package program (SPSS Inc, Chicago, IL). Margin of error was considered to be 0.05.

3. Results

A total of 249 patients, of whom 129 (51.8%) had central vertigo and 120 (48.2%) were controls, were included in the study. The control group's ages were between 36 and 85 years with an average of 59.8; the vertigo group's ages were between 31 and 81 years with an average of 62.2.

Of the cases in the vertigo group, 57 (44.2%) were female and 72 (55.8%) were male. In the control group, 54 (45%) were female and 66 (55%) were male. There was no statistically

significant difference in terms of age and gender between the vertigo group and the control group ($P > 0.05$).

The mean diameters of right vertebral arteries in vertigo and control groups were found to be 3.72 ± 1.15 and 3.94 ± 0.9 mm, respectively; the mean diameters of left vertebral arteries were found to be 3.97 ± 1.1 and 4.18 ± 0.8 mm, respectively; and the mean diameters of basilar arteries were found to be 3.33 ± 0.6 and 3.44 ± 0.7 mm, respectively. Although mean diameters of the right and left vertebral arteries and basilar arteries were less in vertigo patients, they were not statistically significant ($P > 0.05$) (Table 1).

As seen in Table 2, vertebral artery hypoplasia and $\geq 50\%$ stenosis was more common in vertigo cases ($P = 0.000$, < 0.001). Of the total 78 vertigo cases with $\geq 50\%$ stenosis, the stenotic segment in 54 (69.2%) patients was in V1 segment, in 9 (11.5%) patients in V2 segment, in 2 (2.5%) patients in V3 segment, and in 13 (16.6%) patients in V4 segment.

Dissecting aneurysm and fibromuscular hyperplasia were not detected in vertigo and control groups. In the vertigo group, 8 (6.2%) patients had vertebral artery hypoplasia associated with $\geq 50\%$ stenosis of contralateral vertebral artery (Fig. 6A–C). Five of 8 patients were treated with percutaneous transluminal angioplasty and stenting. Three patients did not accept endovascular treatment. In the control group hypoplastic vertebral artery with contralateral vertebral artery stenosis and bilateral vertebral artery stenosis was not detected.

One patient with vertigo had bilateral vertebral artery hypoplasia (Fig. 7A and B); 2 patients had basilar artery hypoplasia accompanied with right vertebral artery hypoplasia.

Five of 78 (6.4%) patients with vertigo had bilateral vertebral artery stenosis and 2 of them were treated with stenting. Surgical treatment was not performed in both groups. Antiplatelet and anticoagulant therapies were given to the 61 (78.2%) vertigo cases with $\geq 50\%$ stenosis.

Left vertebral artery originated from the aortic arch between the main carotid artery and the left subclavian artery orifice in 12 (9.3%) of the vertigo patients and in 9 (7.5%) of the controls. There was no significant difference between the groups ($P > 0.05$).

Of the vertigo cases, 9 (6.9%) had vertebral artery V1 segment tortuosity, and 17 (13.1%) had basilar artery tortuosity. Of the control group, 8 (6.6%) had vertebral artery V1 segment tortuosity, and 13 (10.8%) had basilar artery tortuosity. There was no significant difference between the 2 groups ($P > 0.05$).

Both vertigo and control groups had similar basilar artery hypoplasia and $\geq 50\%$ stenosis rates ($P = 0.800$, > 0.05) (Table 3).

Table 2**Distribution of artery vertebral artery hypoplasia and stenosis within the groups.**

	Vertigo		Control		P*
	n	%	n	%	
Right vertebral artery					
Normal	73	56.6	109	90.8	
Hypoplasia	16	12.4	4	3.3	
$\geq 50\%$ stenosis	40	31	7	5.9	0.000
Left vertebral artery					
Normal	80	62.1	109	90.8	
Hypoplasia	11	8.5	5	4.2	0.000
$\geq 50\%$ stenosis	38	29.4	6	5	

n = number.

* Chi-square test.

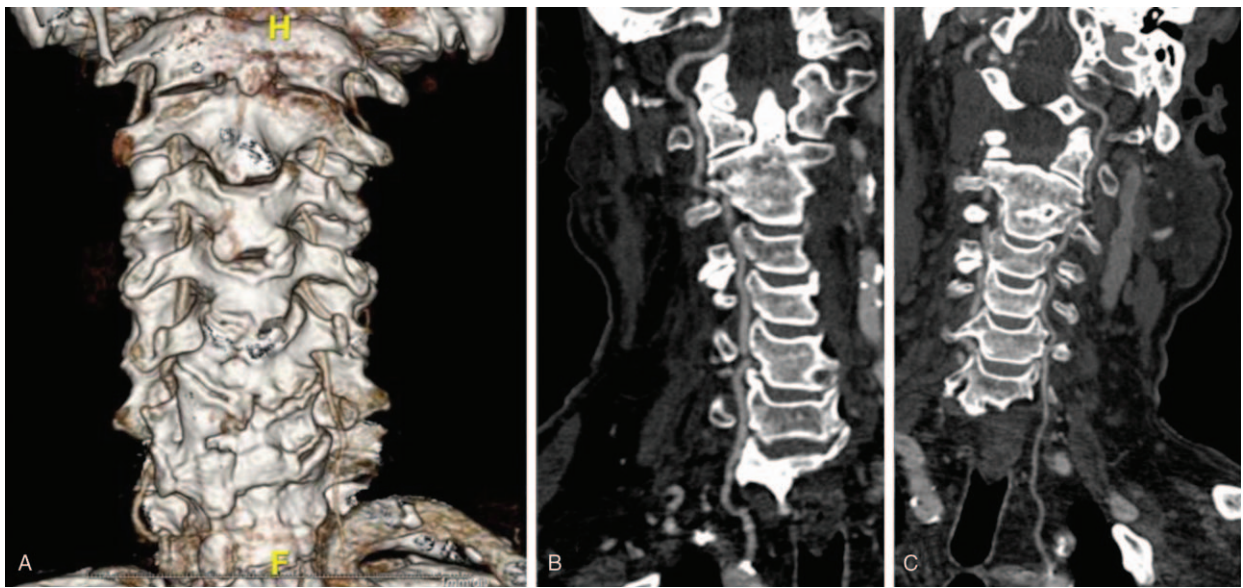


Figure 6. 3D volume rendered coronal (A) and curved multiplanar reconstruction coronal (B and C) images show right vertebral artery stenosis associated with left vertebral artery hypoplasia.

4. Discussion

Atherosclerosis is the most common cause of vertebrobasilar disorders. Stenosis of vertebral and basilar arteries secondary to atherosclerosis leads to vertebrobasilar insufficiency and poor posterior circulation. Vertigo, ataxia, dysarthria, diplopia, visual disturbances, and weakness may be seen in vertebrobasilar insufficiency and ischemia.^[4,5] In the presented study, vertebral artery hypoplasia and $\geq 50\%$ stenosis were more common in

central vertigo cases without stroke compared to in the control group. In patients without stroke signs on DWI, the cause of central vertigo was believed to be TIA or vertebrobasilar insufficiency. A single vertebral artery with normal calibration may sufficiently supply the basilar artery. However, if severe bilateral vertebral artery stenosis or occlusion is present, treatment is indicated.^[12,13] Treatment options may be medical, endovascular, or surgical. Medical treatment includes antiplatelet and anticoagulant therapy.

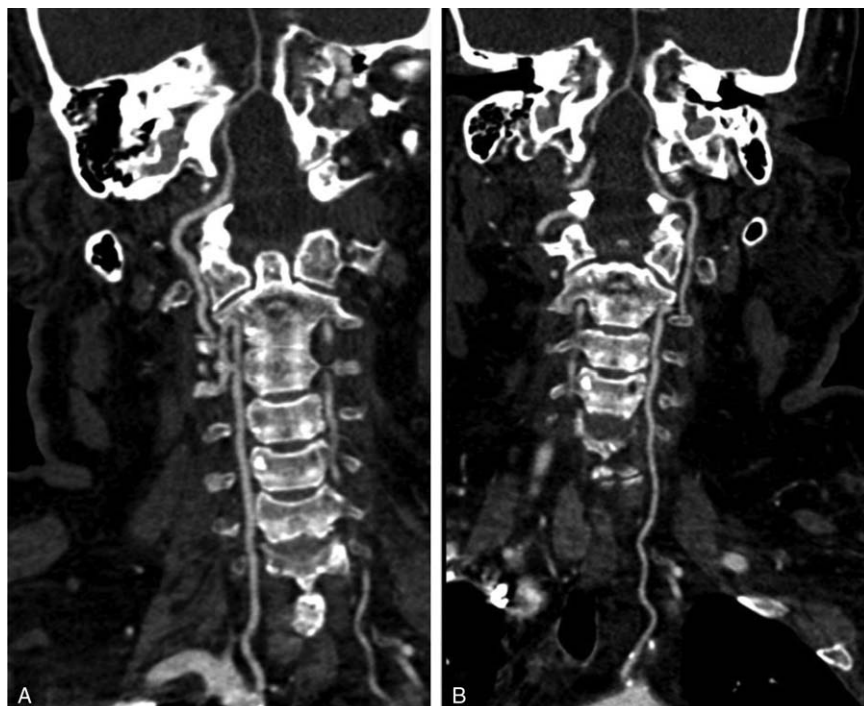


Figure 7. (A and B) Curved multiplanar reconstructed coronal CT images show bilateral hypoplastic vertebral arteries of a vertigo patient. CT = computed tomography.

Table 3**Distribution of basilar artery hypoplasia and stenosis within the groups.**

	Vertigo		Control		P*
	n	%	n	%	
Normal	118	91.4	112	93.3	0.800
Hypoplasia	10	7.8	6	5	
≥50% stenosis	1	0.8	2	1.7	

n = number.

* Fisher exact test.

Surgery is not considered in most centers because of the technical difficulties. It can be performed when medical treatment fails or the anatomy is unfavorable for endovascular treatment.^[12–15] Medical and endovascular treatments were implemented in the patients in our study. In central vertigo cases, CTA may help reveal vertebrobasilar stenosis, anatomic variations, and tortuosity. Being aware of anatomic variations and tortuosity is important before endovascular treatment and surgery because severe tortuosity can preclude safe stent placement.^[12–15]

Atherosclerotic stenosis of the posterior circulation is most often seen at vertebral artery origin. At the intracranial segment of the vertebral artery, stenosis is most commonly seen at vertebrobasilar junction level. Another common location is right before branching to posterior inferior cerebellar artery, distal from the dural penetration.^[16,17] In the presented study, the most common place of stenosis was at the origin of the vertebral artery. Patients with stenosis of vertebral artery origin were found to have a high risk for vertebrobasilar circulation ischemia.

TIA and stroke can be seen and the risk of recurrent stroke after TIA and minor stroke was found to be as high as that of carotid stroke in vertebrobasilar stenotic disease.^[1] A prospective study revealed that the patients who have vertebral stenosis with vertebrobasilar TIA and minor stroke have a 30% risk of recurrent stroke in the first month.^[18] These patients may benefit from medical or endovascular treatment. Therefore, it is important to show stenosis of vertebral artery for starting the therapy.^[17,18]

In this study, vertebral artery hypoplasia was more commonly seen in vertigo cases than in the control group. However, in the literature, a relationship between vertebrobasilar artery hypoplasia and posterior circulatory strokes has been reported.^[19,20] It is important that in the presented study vertebral artery hypoplasia was seen more frequently in vertigo cases without stroke compared to in the control group. Hypoplasia of the vertebral arteries may cause vertigo by decrease of blood supply to the cerebellum and cerebral peduncle. It has been reported that vertebral artery hypoplasia may be commonly accompanied by basilar artery hypoplasia, thus causing posterior circulatory ischemia. Bilateral vertebral artery hypoplasia may present with episodic vertigo attacks.^[21] A single vertebral artery with normal calibration may sufficiently supply the basilar artery. However, bilateral vertebral artery hypoplasia or unilateral vertebral artery hypoplasia associated with contralateral vertebral artery stenosis may impair the sufficient blood flow through the posterior circulation.^[22]

There are studies reporting that vertigo may also be seen in basilar artery stenosis and the vertigo attacks may improve after stenting of the stenotic arteries.^[23] However, no relationship was detected between basilar artery hypoplasia or stenosis and vertigo

in our study. The small number of participants with basilar artery hypoplasia and stenosis in both vertigo and control group may have led to this result in the presented study.

In recent years, studies of vertigo related to vertebral and basilar artery tortuosity have been presented. Vertebral artery tortuosity is thought to be caused by mechanical pressure on the artery due to head position resulting in ischemia. It has been reported that basilar artery tortuosity may lead to occlusion and atherosclerosis, thus decreasing the distal blood flow in tortuous vessels.^[24,25] In the presented study there was no significant difference between vertebral and basilar artery tortuosity in the vertigo group compared to in the control group.

Vertigo patients and control participants were in the elderly group, and the presence of similar risk factors may have created this consequence.

Diagnostic approach for vertigo patients is complex. Detailed patient history, physical examination, neuro-otological tests, and imaging finding are used.^[3–26] It has been shown that MRI is useful in the detection of serious acute vertigo cases related to small posterior fossa infarctions and in the differentiation from vestibular neuritis.^[27] Contrast-enhanced MRA has frequently shown pseudostenosis of vertebral artery origin because of weak spatial resolution, intravoxel dephasing, and motion artifact caused by cardiac pulsation and respiration.^[28] Khan et al^[11] performed a systematic literature review to evaluate the accuracy of duplex ultrasound, contrast-enhanced MRA, and CTA in detecting severe vertebral artery stenosis. The sensitivity and specificity of CTA (100% and 93.9%, respectively) were higher versus those of contrast-enhanced MRA (95.2% and 94.8%, respectively) in detecting severe vertebral artery stenosis. In a recent study comparing contrast-enhanced MRA, CTA, and duplex sonography in detecting ≥50% stenosis of vertebral artery, contrast-enhanced MRA had the highest sensitivity and specificity (83% and 91%, respectively). CTA had good sensitivity (68%) and excellent specificity (92%).^[13] On the other hand, CTA had better accessibility, high spatial resolution, and short scanning time than MRA. CTA has better temporal resolution and therefore is less affected by the motion of cardiac pulsation and respiration. It is cheaper and suitable for patients with contraindications to MRI. However, CTA has problems involving radiation, and a potentially nephrotoxic contrast agent as well as inaccuracy for heavily calcified stenosis.^[28] Because CTA has higher temporal and spatial resolution than MRA and is less affected by motion artifact and pseudostenosis, we recommend evaluating patients with central vertigo with CTA after brain MRI.

Some limitations exist in the presented study. Vascular risk factors such as ischemic heart diseases and smoking were not included. The patients included in the study were elderly and with higher ischemic heart disease risks. Further limitations were small

number of patients with >50% basilar artery stenosis and basilar artery hypoplasia of the controls and vertigo patients.

This study showed that CTA may be helpful to clarify the association between abnormal CTA findings of vertebral arteries and central vertigo. It is believed that CTA may be useful in the arrangement of clinical management.

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