

# Acute vestibular syndrome accompanying middle cerebral artery infarctions: a single-center study

Nevin Kuloğlu Pazarıcı<sup>ORCID</sup>, Gizem Gürsoy<sup>ORCID</sup>, Necip Kürşad Çiftçi<sup>ORCID</sup>

Department of Neurology, University of Health Sciences, Ümraniye Training and Research Hospital, İstanbul, Türkiye

## ABSTRACT

**Objectives:** Acute vestibular syndrome (AVS) is a disease and disorder usually manifest with a single episode of sudden onset vestibular symptoms and signs, and may occur at any point along the vestibular pathway from the peripheral labyrinth to the vestibular cortex. This study presents the characteristics of the involved cortical area and clinical course in middle cerebral artery (MCA) territory infarction patients with central vestibular disorder.

**Methods:** Files of patients diagnosed with acute ischemic stroke in the MCA territory were reviewed retrospectively between December 2020 and March 2022. All the patients were categorized into two groups (with or without AVS) and analyzed for comorbidities as well as smoking and drinking habits, and their data pertaining to the neurological examination, bedside neuro-otological examination, and laboratory and computerized tomography angiography findings were collected.

**Results:** Seven hundred and sixty-eight patients with MCA territory infarction were included in the study. AVS incidence was calculated to be 13.3%. There was no statistically significant difference in the distribution of age and sex between patients with and without AVS. The involvement of the right side was significantly more prevalent in AVS patients. Distribution of patients with right-sided infarction: 11 with posterior superior temporal gyrus 17 with parieto-insular cortex, 16 with insular cortex and temporoparietal junction together, and 18 with more than one millimetric infarction in all three areas.

**Conclusions:** Patients with acute vestibular syndrome, may have infarctions in the anterior circulation and most of them had no pathological neuro-otologic test findings.

**Keywords:** Acute vestibular syndrome, anterior circulation, middle cerebral artery, stroke

Stroke is one of the most important causes of disability and mortality in all populations and ranks second among the causes of death globally. Treatment and rehabilitation costs and the disability it causes in patients have significant impacts on society. Symptoms of stroke depend on which brain region is affected. That said, the symptoms can generally be categorized as cognitive, motor, sensory, and autonomic

effects. Approximately 87% of all strokes are of ischemic character [1, 2]. Investigation of the epidemiology of ischemic stroke subtypes revealed 30.2% cardioembolism, 25.8% small artery occlusion, and 15.3% large artery atherosclerosis [3].

In acute stroke patients, vestibular syndrome and vertigo are rare clinical characteristics. Vertigo is the impairment of the static gravitational orientation and

**Corresponding author:** Gizem Gürsoy, MD.,  
Phone: +90 216 632 18 18, E-mail: [drgezembursoy@gmail.com](mailto:drgezembursoy@gmail.com)

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is described as a specific type of headache characterized by a sensation of spinning or tilting accompanied by nausea/vomiting and imbalance. Dizziness, on the other hand, denotes an impaired spatial orientation without distorted self-motion. Together, they are the third most common main symptom in general medical clinics where they represent 3-5% of all visits, and studies have reported the ratio of vertigo and dizziness in all stroke patients to be from 2.7% to 3.2% [1, 4]. Anatomically, acute vestibular syndrome (AVS) may occur in any point along the vestibular pathway from the peripheral labyrinth to the vestibular cortex. While isolated vertigo or dizziness supports posterior circulatory disorders, some studies have shown that anterior circulation may also be affected [5].

The vestibular cortex in humans has yet to be precisely located. It is assumed that the vestibular cortex is represented in the bilateral parieto-insular cortex with the right hemisphere dominance [6]. The 2012 coordinate-based activation likelihood estimation meta-analysis revealed vestibular activation in the bilateral insula, retroinsular cortex, inferior parietal lobe, superior temporal gyrus, caudal section of the anterior cingulate gyrus, and precuneus [7]. Its clinical characteristics in this specific localization are contralateral lateropulsion and speech impediment without nystagmus or any other brainstem symptoms [8].

AVS is relatively rare following middle cerebral artery (MCA) infarctions. MCA supplies blood to the cortical areas which are the core areas for the central vestibular symptoms. This study presents the characteristics of the involved cortical area and clinical course as shown by diffusion-weighted imaging (DWI) in MCA territory infarction patients with central vestibular disorder.

## METHODS

### Patient Group

Files of the patients above 18 years of age admitted as inpatients to the Stroke Unit, at Ümraniye Training and Research Hospital, Neurology Clinic between December 2020 and March 2022 with the diagnosis of acute ischemic stroke were retrospectively reviewed. Axial MR images from the medulla to the cortex taken at admission with a slice thickness of 5 mm and fluid-

attenuated inversion recovery (FLAIR), DWI, and Apparent diffusion coefficient (ADC) sequences were scanned, and patients diagnosed with an acute cerebral ischemic lesion in the MCA territory with the help of DWI were categorized into two groups, i.e., with and without vestibular symptoms. All the patients were analyzed for comorbidities as well as smoking and drinking habits, and their data about the neurological examination, bedside neuro-otological examination, and laboratory findings were collected.

Characteristics such as age, sex, and demographics were evaluated between the groups, as well as the clinical characteristics and the involved cortical area characteristics in the group with vestibular symptoms. The computerized tomography angiography (CTA) performed at admission covers the main branches and the trunk of the anterior and posterior circulation in the cervical and cranial segments of the cerebral circulation beginning from the aortic arch, and also the whole circle of Willis. Stenosis of the symptomatic carotid artery or the vertebral basilar artery is defined as the decrease in the diameter of the carotid or vertebral basilar artery by over 50% at a location thought to be responsible for the patient's acute infarction or clinical symptoms.

Patients with previous vertigo and dizziness attacks, patients with previous ischemic or hemorrhagic stroke with concurrent infarctions in other artery watersheds, and patients with additional neurological disorders that may lead to vestibular syndrome (vertebral basilar artery stenosis, migrainous vertigo, advanced cervical spondyloarthritis, multiple sclerosis, etc.) were excluded from the study. Due to the retrospective nature of this study, the requirement of informed consent has been waived.

The Ethics Committee of the Ümraniye Training and Research Hospital approved the present study with its letter of decision no. 112 and date: 28.03.2024.

### Statistical Analysis

Data analyses were performed using the SPSS software statistical package (version 20; SPSS Inc, Chicago, IL, USA). The  $\chi^2$  test was used for comparisons of qualitative data, the Mann-Whitney U test was used for numerical data. P values <0.05 were considered to indicate statistical significance. All numeric values are presented as the means  $\pm$  standard deviation (SD).

## RESULTS

Of a total of 1,371 patients diagnosed with acute ischemic stroke, 768 patients with MCA territory infarction were included in the study. AVS incidence was calculated to be 13.3% among the included patients. There was no statistically significant difference in the distribution of age (P=0.9) and sex (P=0.62) between 102 patients with AVS (66 males: 64.7%, mean age: 67.06±1.2 years) and 666 patients without AVS (414 males: 62.16%, mean age: 67.23±0.46).

Concerning comorbidities, hypertension (HT) was found in all the patients with AVS, whereas the ratio of the presence of HT alone was 18.92% in the non-AVS group. While the presence of HT alone did not create a statistical difference, it was seen statistically

more commonly in the AVS group when it coexisted with other risk factors (P<0.001). It was further found that the presence of diabetes mellitus (DM) both alone and with other comorbidities was significantly higher in the non-AVS group (P=0.001). While smoking was higher in the non-AVS group, drinking was significantly higher in the AVS group (P=0.018).

62 patients (60.79%) in the AVS group and 308 patients (46.25%) in the non-AVS group had infarction of the right side (P=0.006), which meant that the involvement of the right side was significantly more prevalent in AVS patients. While the primary sensory cortex was the common area of involvement in only 7 out of 40 patients with infarction of the left side in the AVS group, of 62 patients with infarction of the right side, 11 had the posterior superior temporal gyrus and

**Table 1. Demographical and clinical features of patients with and without AVS**

	Patients with AVS (n=102)	Patients without AVS (n=666)	P value
Age (years)	67.06±1.2	67.23±0.46	0.6
Gender, male/female, n (%)	66/36 (64.7/36.3)	414/252 (62.2/38.8)	0.9
<b>Side of infarction</b>			
Left	40	358	<b>0.006</b>
Right	62	308	
<b>CT Angiography, n (%)</b>			
Normal	24 (23.53)	90 (13.5)	<b>0.01</b>
<50% stenosis	48 (47.06)	324 (48.65)	0.83
>50% stenosis	30 (29.41)	120 (18.05)	<b>0.007</b>
Advanced IC stenosis	0	132 (19.82)	<b>&lt;0.001</b>
<b>Size of infarction (%)</b>			
Total	0	48 (7.21)	<b>&lt;0.001</b>
Division	2 (1.96)	204 (30.63)	<b>&lt;0.001</b>
Cortical branch	52 (50.98)	246 (36.94)	
Milimetric	48 (7.21)	168 (25.23)	
<b>Comorbidities, n (%)</b>			
Hypertension	102 (100)	534 (80.18)	<b>&lt;0.001</b>
Diabetes mellitus	42 (41.18)	390 (58.56)	<b>0.001</b>
Smoking	54 (52.94)	450 (67.57)	<b>0.004</b>
Alcohol using	24 (23.53)	96 (14.42)	<b>0.018</b>

Data are shown as mean±standard deviation or n (%). AVS=Acute vestibular syndrome, IC=Internal carotid artery, CT=Computed tomography

17 had the parieto-insular cortex involved separately, whereas 16 had the insular cortex and the temporoparietal junction involved together, and the remaining 18 had multiple millimetric infarctions in all of these three areas.

When the infarctions of the study patients were categorized by size in the MCA territory as total, division, cortical branch, and millimetric infarctions, division, and total infarcts were significantly higher in the non-AVS group ( $P < 0.001$ ), whereas there was no such difference with other sizes.

The CT angiography images were categorized into 4 subgroups, i.e., normal,  $<50\%$  ICA stenosis,  $>50\%$  ICA stenosis, and advanced intracranial stenosis. All patients with advanced ICA stenosis were in the non-AVS group, and no difference was found between the two groups in terms of  $<50\%$  stenosis. The statistically significant differences were found in patients with normal CTA and  $>50\%$  stenosis in the AVS and non-AVS groups, and both subgroups were higher in the AVS group. These findings are summarized in Table 1.

With or without an accompanying AVS, none of the patients had any pathological neuro-otologic test findings such as a positive head impulse test, nystagmus, or skew deviation.

## DISCUSSION

While the somatosensory system regulates the proprioceptive information, the visual system provides information concerning the location of the body in relation to the outer environment, and the vestibular system supplies constant information about the movements and positions of the head and the neck. When the functions of this sensory trio are impaired, dizziness, nausea, sensation of imbalance, and sometimes nystagmus, occur.

Receiving somatosensory, visual, and vestibular inputs, the temporoparietal junction (TPJ) is defined as a large area encompassing the posterior superior temporal gyrus, angular gyrus, supramarginal gyrus, and parietal operculum. There are three posterior cortical areas processing vestibular signals: the parieto-insular vestibular cortex (PIVC), a subunit of TPJ, encodes the vestibular signals concerning the position and the movements of the head; the ventral intrapari-

etal (VIP) area integrates spatial information from various sensory modalities encoding the area on the coordinates originating from the peripheral sensory system; and the medial superior temporal (MST) area integrates the vestibular and visual signals required for the sensation of self-motion [9].

Dizziness is one of the most common complaints in the general population with a reported annual incidence of 22.9%, and approximately 30% of dizziness is non-organic [10]. According to the International Classification of Vestibular Disorders, it is defined as a sensation of disturbed or impaired spatial orientation without a false or distorted sense of motion. The ICVD defines vertigo as a sensation of self-motion (of head/body) when no self-motion is occurring or the sensation of distorted self-motion during an otherwise normal head movement. Both definitions appear under the title of symptoms and signs in the first layer of the Classification. The syndrome subheading in the second layer of the Classification discusses acute vestibular syndrome, and defines it as diseases and disorders that usually manifest with a single episode of sudden onset vestibular symptoms and signs (e.g. vestibular neuritis or acute stroke) [11].

A retrospective study using the patient records of 5 years from a single site determined the frequency of vertigo among cerebrovascular disorders to be 17% [12]. Another study investigating the presence of stroke in acute vestibular syndrome in 86 patients found that 27% of the patients with AVS also had strokes, and reported that there were no differences in DM, HT, CAD, AF, and hyperlipidemia between the peripherally and centrally induced ones [13].

Vertigo and dizziness are common signs, especially in the ischemia of the carotid region in the temporo-parietal area. A diffusion tensor tractography study in patients with MCA territory infarction observed a reduced tract volume in the core vestibular pathway leading to the parieto-insular vestibular cortex and showed that this reduction was associated with central vestibular disorder. Clinicians should give careful consideration to the investigation of anterior circulation vascular disease in patients presenting with vestibular symptoms [14, 15]. However, vestibular syndrome may not always manifest in the involvement of the cortical vestibular centers. It depends on the visual-vestibular interaction for the perception and ori-

entation of motions. The result is affected by the situation of the hemisphere in which vestibular and visual inputs are in agreement and which determines the global perception of body orientation and motion [16]. Our interpretation of the lower incidence of AVS in patients with larger areas of infarction in our study was that AVS might have been overlooked due to serious neurological signs and symptoms caused by the involvement of a large area, which might have also led to patients' failure to describe it. We believe that the fact that the infarction area was large in patients with advanced stenosis in the intracranial ICA and its branches similarly led us to find this subgroup significantly higher in the non-AVS group.

Studies show a correlation between DM, poor glycemic control in particular, and severe stroke and a large infarct volume [17]. While the subgroup analyses conducted in our study revealed that the presence of DM among the patients' risk factors did not have a statistically significant effect on the infarction size ( $P=0.075$ ), it was found that patients with DM had mathematically larger infarction areas and more severe stroke characteristics. This may help explain our finding DM is higher in the non-AVS group. HT is a common risk factor for both cardioembolism and small artery occlusion, as well as large artery atherosclerosis, and previous studies have shown its correlation with the severity of stroke [18]. It is difficult to explain why our study found it in 80% of the non-AVS group patients while it was present alone or together with other risk factors in the AVS group; furthermore, it could not be associated with the anatomical localization of involvement. We think that this finding of our study should be verified by similar studies.

AVS induced by posterior circulation infarctions manifests with clinical signs relating to the involvement of the brainstem and cerebellum such as vertigo which lasts longer, may prevent mobilization, and is accompanied by nausea, vomiting, nystagmus, skew deviation, dysmetria, and ataxia [19]. However, we found only vertigo and dizziness accompanying the anterior circulation infarctions in the AVS patients we observed; these signs lasted up to 5 days in very few patients as we usually saw them resolve within the first 24 hours. Again, very few patients had accompanying nausea and vomiting severe enough to administer symptomatic treatment. We did not find posterior circulation or peripheral system vestibular abnormalities

in any patients.

Lesions responsible for vestibular symptoms are usually located in the right hemisphere [20]. A study found that 42.6% of the patients presenting with DWI-positive acute and episodic vestibular syndrome had acute infarcts in the anterior circulation and that the most common area of involvement was the insular cortex with 22.1% [1]. Our study, as well, found the insular cortex to be the most commonly involved area, which was affected together with the parieto-temporal area in 17 patients. It has been found that AVS is the fifth most common symptom in the patients followed up for insular cortex infarcts and it is seen in approximately 50% of the patients suffering from the involvement of the right hemisphere. This ratio is 20% for the left hemisphere, and in support of this finding, our study showed that the involvement of the right hemisphere was significantly higher [21].

### Limitations

Having been designed as a retrospective cross-sectional study limited to a single site, the study excluded patient files with incomplete data. It was not deemed necessary to perform cranial imaging in the patients again, and it was thought that some patients may have had accompanying posterior system infarctions or transient ischemic attacks.

### CONCLUSION

In conclusion, it should be noted that in patients with acute vestibular syndrome, infarctions may develop in the anterior circulation, as well, and mostly no pathological neuro-otologic test findings will be obtained. Even though our study found comorbidities to be higher in the AVS group, extensive prospective studies are needed to show the relationship between the risk factors and AVS more clearly.

### Authors' Contribution

Study Conception: NKP; Study Design: NKP, GG; Supervision: NKP; Funding: N/A; Materials: NKP, GG, NKÇ; Data Collection and/or Processing: NKP, GG, NKÇ; Statistical Analysis and/or Data Interpretation: NKP, GG, NKÇ; Literature Review: NKP, GG, NKÇ; Manuscript Preparation: NKP, GG and Critical Review: NKP, GG.

### Conflict of interest

The authors disclosed no conflict of interest during the preparation or publication of this manuscript.

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## REFERENCES

1. Tong D, Chen X, Wang Y, Wang Y, Du L, Bao J. Acute and episodic vestibular syndromes caused by ischemic stroke: predilection sites and risk factors. *J Int Med Res.* 2020;48(4):300060520918039. doi: 10.1177/0300060520918039.
2. Grysiewicz RA, Thomas K, Pandey DK. Epidemiology of ischemic and hemorrhagic stroke: incidence, prevalence, mortality, and risk factors. *Neurol Clin.* 2008;26(4):871-895, vii. doi: 10.1016/j.ncl.2008.07.003.
3. Kolominsky-Rabas PL, Weber M, Gefeller O, Neundoerfer B, Heuschmann PU. Epidemiology of ischemic stroke subtypes according to TOAST criteria: incidence, recurrence, and long-term survival in ischemic stroke subtypes: a population-based study. *Stroke.* 2001;32(12):2735-2740. doi: 10.1161/hs1201.100209.
4. 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. doi: 10.1161/01.STR.0000240329.48263.0d.
5. Chakor RT, Eklare N. Vertigo in cerebrovascular diseases. *Int J Otorhinolaryngol Clin.* 2012;4(1): 46-53. doi: 10.5005/jp-journals-10003-1087
6. Hashimoto T, Taoka M, Obayashi S, Hara Y, Tanaka M, Iriki A. Modulation of cortical vestibular processing by somatosensory inputs in the posterior insula. *Brain Inj.* 2013;27(13-14):1685-1691. doi: 10.3109/02699052.2013.831128.
7. Lopez C, Blanke O, Mast FW. The human vestibular cortex revealed by coordinate-based activation likelihood estimation meta-analysis. *Neuroscience.* 2012;212:159-179. doi: 10.1016/j.neuroscience.2012.03.028.
8. Choi KD, Kim JS. Vascular vertigo: updates. *J Neurol.* 2019;266(8):1835-1843. doi: 10.1007/s00415-018-9040-3.
9. Pfeiffer C, Serino A, Blanke O. The vestibular system: a spatial reference for bodily self-consciousness. *Front Integr Neurosci.* 2014;8:31. doi: 10.3389/fnint.2014.00031.
10. Kuwabara J, Kondo M, Kabaya K, et al. Acceptance and commitment therapy combined with vestibular rehabilitation for persistent postural-perceptual dizziness: A pilot study. *Am J Otolaryngol.* 2020;41(6):102609. doi: 10.1016/j.amjoto.2020.102609.
11. Bisdorff AR, Staab JP, Newman-Toker DE. Overview of the International Classification of Vestibular Disorders. *Neurol Clin.* 2015;33(3):541-550, vii. doi: 10.1016/j.ncl.2015.04.010.
12. Okroglic S, Widmann CN, Urbach H, Scheltens P, Heneka MT. Clinical symptoms and risk factors in cerebral microangiopathy patients. *PLoS One.* 2013;8(2):e53455. doi: 10.1371/journal.pone.0053455.
13. Choi JH, Park MG, Choi SY, et al. Acute Transient Vestibular Syndrome: Prevalence of Stroke and Efficacy of Bedside Evaluation. *Stroke.* 2017;48(3):556-562. doi: 10.1161/STROKEAHA.116.015507.
14. Yeo SS, Jang SH, Kwon JW. Central vestibular disorder due to ischemic injury on the parieto-insular vestibular cortex in patients with middle cerebral artery territory infarction: Observational study. *Medicine (Baltimore).* 2017;96(51):e9349. doi: 10.1097/MD.00000000000009349.
15. Zhou Y, Lee S-H, Mantokoudis G, et al. Vertigo and Dizziness in Anterior Circulation Cerebrovascular Disease: A Systematic Review (P3. 092). *Neurology.* 2014;82(10\_supplement). doi: 10.1212/WNL.82.10\_supplement.P3.092.
16. Dieterich M, Brandt T. Why acute unilateral vestibular cortex lesions mostly manifest without vertigo. *Neurology.* 2015;84(16):1680-1684. doi: 10.1212/WNL.0000000000001501.
17. Lee SH, Jang MU, Kim Y, et al. Effect of Prestroke Glycemic Variability Estimated Glycated Albumin on Stroke Severity and Infarct Volume in Diabetic Patients Presenting With Acute Ischemic Stroke. *Front Endocrinol (Lausanne).* 2020;11:230. doi: 10.3389/fendo.2020.00230.
18. Soliman RH, Oraby MI, Fathy M, Essam AM. Risk factors of acute ischemic stroke in patients presented to Beni-Suef University Hospital: prevalence and relation to stroke severity at presentation. *Egypt J Neurol Psychiatr Neurosurg.* 2018;54(1):8. doi: 10.1186/s41983-018-0012-4.
19. Choi JH, Kim HW, Choi KD, et al. Isolated vestibular syndrome in posterior circulation stroke: Frequency and involved structures. *Neurol Clin Pract.* 2014;4(5):410-418. doi: 10.1212/CPJ.0000000000000028.
20. Eguchi S, Hirose G, Miaki M. Vestibular symptoms in acute hemispheric strokes. *J Neurol.* 2019;266(8):1852-1858. doi: 10.1007/s00415-019-09342-9.
21. Di Stefano V, De Angelis MV, Montemitto C, et al. Clinical presentation of strokes confined to the insula: a systematic review of literature. *Neurol Sci.* 2021;42(5):1697-1704. doi: 10.1007/s10072-021-05109-1.